T&E Item 1
March 16, 2015
Worksession

MEMORANDUM

March 12, 2015

TO: Transportation, Infrastructure, Energy and Environment Committee

FROM: Josh Hamlin, Legislative Attorney

SUBJECT: **Worksession:** Bill 52-14, Pesticides – Notice Requirements – Non-Essential Pesticides – Prohibitions

Expected Attendees

Panel 1:

Lisa Feldt, Director, Department of Environmental Protection (DEP)
Stan Edwards, Chief, DEP Division of Environmental Policy and Compliance
Carol Holko, Assistant Secretary, Plant Industries & Pest Management, Maryland Department of Agriculture
Dan Kenny, Branch Chief, Herbicide Branch, Office of Pesticide Programs, Registration Division, U.S. Environmental Protection Agency

Panel 2:

Dr. Jerome Paulson, MD, FAAP Professor Emeritus in Pediatrics at the George Washington University School of Medicine and Health Sciences and Professor Emeritus in Environmental & Occupational Health at the George Washington University Milken Institute School of Public Health
Dr. Lorne K. Garrettson, MD, FAAP, FAACT, Professor Emeritus, Emory University, Departments of Pediatrics and Environmental and Occupational Medicine
Dr. Stuart Z. Cohen, Ph.D., CGWP, Environmental and Turf Services, Inc.

Bill 52-14, Pesticides – Notice Requirements – Non-Essential Pesticides – Prohibitions, sponsored by then Council Vice President Leventhal and Councilmembers Elrich, Riemer, Floreen, and Navarro was introduced on October 28. Public hearing on the Bill began on January 15, and was continued on February 12. An additional Transportation, Infrastructure, Energy and Environment Committee worksession is tentatively scheduled for March 30, 2015 at 9:30 a.m.

Bill 52-14 would:
(1) require posting of notice for certain lawn applications of pesticide;

(2) prohibit the use of certain pesticides on lawns;
(3) prohibit the use of certain pesticides on certain County-owned property;
(4) require the County to adopt an integrated pest management program for certain County-owned property; and
(5) generally amend County law regarding pesticides.

Council Vice President Leventhal has explained the purpose of this Bill in his October 22, 2014 memorandum to Councilmembers (See ©14-64).[1]

Background

Shared Regulation of Pesticides

The regulation of pesticides is the shared responsibility of federal, state, and local governments. This shared approach, known as "environmental federalism," is consistently applied among several federal environmental protection laws,[2] and has evolved largely over the last 50 years.

At the national level, the Federal Insecticide, Fungicide and Rodenticide Act ("FIFRA") is the primary vehicle for pesticide regulation. FIFRA was enacted in 1947, and has evolved from being primarily a labeling statute to become a somewhat more broad regulation. In 1972, administration of FIFRA was transferred to the newly created Environmental Protection Agency ("EPA"), which is responsible for classifying pesticides based on a review of the scientific evidence of their safety and impact on the health of individuals and the environment. FIFRA also requires EPA to maintain a registry of all but "minimum risk" pesticides.[3] In addition to the classification and registry of pesticides, FIFRA provides a uniform national standard for labeling pesticides. FIFRA does not comprehensively regulate pesticides, however, and does not include public notice or permit requirements for the use of pesticides.

Under FIFRA, the states are the primary enforcers of pesticide use regulations, and FIFRA expressly authorizes states to enact their own regulatory measures concerning the sale or use of any federally registered pesticides in the state, provided the state regulation is at least as restrictive as FIFRA itself. In Maryland, pesticides are regulated by the Maryland Department of Agriculture, through the enforcement of Subtitles 1 and 2 of Title 5 of the Agriculture Article of the Maryland

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[1] For additional background on this Committee's recent consideration of pesticides and pesticide use in Montgomery County, see the packet for the September 9, 2013 discussion at: http://www6.montgomerycountymd.gov/content/council/pdf/agenda/cm/2013/130909/20130909_TE3.pdf. Video of the discussion is available, beginning at 22:10, at: http://montgomerycountymd.granicus.com/MediaPlayer.php?view_id=6&clip_id=5704.

[2] The 1972 Federal Water Pollution Control Act, the 1986 amendments to the Safe Drinking Water Act, the Toxic Substances Control Act, the Resource Conservation and Recovery Act, and the Oil Pollution Control Act of 1990 all provide for state and local regulatory roles.

[3] Minimum risk pesticides are a special class of pesticides that are not subject to federal registration requirements because their ingredients, both active and inert, are *demonstrably* safe for the intended use. Information about EPA's treatment of minimum risk pesticides can be found at: http://www.epa.gov/oppbppd1/biopesticides/regtools/25b/25b-faq.htm

Code.[4] Maryland law and regulations generally create a pesticide registration and labeling regime at the state level, and a licensing program for the application of certain pesticides. Title 5 does not include any express preemption language, and does not appear to generally regulate pesticides so comprehensively that preemption can be implied. As a general matter, therefore, the County may regulate pesticides, at least as restrictive as, and consistent with, federal and State law.

The authority of local governments to regulate pesticides was the subject of significant litigation in the 1980s, with a County law struck down as preempted by FIFRA. In *Maryland Pest Control Assn. v. Montgomery County, Maryland*, 646 F. Supp. 109 (D. Md. 1986), the U.S. District Court held that FIFRA preempted the County's local law imposing pesticide posting and notice requirements. The Court held that if Congress had wanted to include local governments in the regulation of pesticides, it would have expressly done so. However, in *Wisconsin Public Intervenor v. Mortier*, 111 S. Ct. 2476 (1991), the U.S. Supreme Court held, contrary to the *Maryland Pest Control Assn.* decision, that a unit of local government has the power, under FIFRA, to regulate pesticides within its own jurisdiction, provided that the local regulation is at least as restrictive as, and consistent with, FIFRA and any applicable state law. Since *Mortier* was decided, many states have expressly preempted local jurisdictions from regulating pesticides, but Maryland is one of seven states which do not preempt local regulation of pesticides.[5] The County currently imposes certain notice, storage, handling, and consumer information requirements in Chapter 33B of the County Code.

Laws in Other Jurisdictions

Due to the fact that the vast majority of states have preempted local jurisdictions from regulating pesticides, there are only two examples of local jurisdictions that have banned pesticide use on public and private property[6]: Takoma Park, Maryland[7], and Ogunquit, Maine.[8] Several local jurisdictions have enacted legislation or adopted administrative policies related to pesticide reduction on public property, integrated pest management (IPM), and pesticide free parks.[9] Locally in addition to Takoma Park, the District of Columbia enacted the Pesticide Education and Control Amendment Act Of 2012[10] which restricts the application of certain pesticides near waterways and at schools, day care centers and on District property, and imposes certain reporting and data collection requirements. Most recently, Richmond, California, which has had an IPM ordinance since 2012, passed a resolution to implement a "twelve month long ban on the use of all toxic pesticides, including those containing glyphosate, on all weed abatement activities conducted, contracted, or managed by the city . . ."[11]

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[4] Subtitle 1 is entitled the "Maryland Pesticide Registration and Labeling Law." Subtitle 2 is the "Pesticide Applicator's Law."

[5] http://www.beyondpesticides.org/lawn/activist/documents/StatePreemption.pdf

[6] http://www.telegraph.co.uk/news/worldnews/10959057/End-of-the-perfect-American-lawn-Campaigners-call-for-pesticide-ban.html

[7] http://www.takomaparkmd.gov/safegrow

[8] http://ogunquitconservation.org/ogunquitconservation.org/Pesticide_Ordinance_Overview.html

[9] http://www.beyondpesticides.org/lawn/activist/

[10] The signed Act is at: http://lims.dccouncil.us/Download/26399/B19-0643-SignedAct.pdf. The Committee report is at: http://lims.dccouncil.us/Download/2594/B19-0643-COMMITTEEREPORT.pdf

[11] Discussion of the resolution begins at page 99 of the pdf of the agenda packet found at: http://sireweb.ci.richmond.ca.us/sirepub/cache/2/mz3m1yjgzymhc5rcpuma1wre/42617103092015105517360.PDF

Perhaps the most comprehensive pesticide restriction law in North America took effect in the Canadian province of Ontario in 2009.[12] The Ontario law contains several classifications of pesticides, and generally bans the cosmetic use of over 100 pesticides.[13] Six other provinces have followed Ontario in restricting cosmetic use of pesticides.[14] British Columbia, however, considered, but did not implement a provincial ban on cosmetic pesticides.[15]

Pending legislation in the Maryland General Assembly

The Maryland General Assembly is currently considering two bills related to pesticides which have objectives similar to Bill 52-14. The bills would: (1) impose labeling requirements and future sale and use restrictions on neonicotinoid pesticides; and (2) prohibit, except in emergencies, the application of lawn care pesticides to certain areas used by children under the age of 18 years.

House Bill 605,[16] cross-filed with Senate Bill 163, would establish a labeling requirement for any seed, plant material, nursery stock, annual plant, bedding plant, or other plant that has been treated with a neonicotinoid pesticide[17] and would establish restrictions, effective January 1, 2016, on the sale and use of neonicotinoid pesticides. The future restrictions would: (1) limit the use of neonicotinoid pesticides to applicators certified by the Maryland Department of Agriculture (MDA), and farmers using the pesticide for agricultural purposes; and (2) require a seller of neonicotinoid pesticides to be permitted by MDA to sell restricted-use pesticides. House Bill 605 is scheduled in the House Environment and Transportation Committee at 1:00 pm on March 13.

House Bill 995[18] would generally prohibit the application of certain pesticides on the grounds of certain child care centers, schools, and recreation centers and on certain other recreational fields. The prohibition would apply to pesticides registered by the EPA and labeled pursuant to the FIFRA for use in lawn, garden, or ornamental sites and areas. A person would be able to apply for an emergency exemption from the prohibition when necessary to eliminate an immediate threat to human health. House Bill 995 is also scheduled in the House Environment and Transportation Committee at 1:00 pm on March 13.

Bill 52-14

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[12] http://www.davidsuzuki.org/issues/health/science/pesticides/highlights-of-ontarios-cosmetic-pesticide-ban/

[13] https://www.ontario.ca/environment-and-energy/pesticides-home-lawns-and-gardens

[14] http://news.gov.mb.ca/news/index.html?item=30526

[15] The Report of the British Columbia Special Committee on Cosmetic Pesticides, which was "convinced that further restrictions on the use and sale of pesticides in British Columbia are necessary" but was "unable to reach a consensus on the need for a provincial ban on pesticide use for cosmetic purposes" is at: https://www.leg.bc.ca/cmt/39thparl/session-4/cp/reports/PDF/Rpt-CP-39-4-Report-2012-MAY-17.pdf

[16] http://mgaleg.maryland.gov/webmga/frmMain.aspx?id=hb0605&stab=01&pid=billpage&tab=subject3&ys=2015RS

[17] The required label would read:

> "WARNING: Bees are essential to many agricultural crops. This product has been treated with neonicotinoid pesticides, found to be a major contributor to bee deaths and the depletion of the bee population."

[18] http://mgaleg.maryland.gov/webmga/frmMain.aspx?id=hb0995&stab=01&pid=billpage&tab=subject3&ys=2015RS

Bill 52-14 includes provisions related to the application of pesticides on County-owned and private property, and requires the County to adopt an Integrated Pest Management (IPM) plan. IPM is a method of pest control which minimizes the use of chemical pesticides by focusing on pest identification, monitoring and assessing pest numbers and damage, and using a combination of biological, cultural, physical/mechanical and, when necessary, chemical management tools.[19]

Bill 52-14 will:

1) Require the posting of notice when a property owner applies a pesticide to an area of lawn more than 100 square feet, consistent with the notice requirements for when a landscaping business treats a lawn with a pesticide;
2) Require the Executive to designate a list of "non-essential" pesticides including:
   - all pesticides classified as "Carcinogenic to Humans" or "Likely to Be Carcinogenic to Humans" by the U.S. EPA;
   - all pesticides classified by the U.S. EPA as "Restricted Use Products;"
   - all pesticides classified as "Class 9" pesticides by the Ontario, Canada, Ministry of the Environment;
   - all pesticides classified as "Category 1 Endocrine Disruptors" by the European Commission; and
   - any other pesticides which the Executive determines are not critical to pest management in the County.
3) Generally prohibit the application of non-essential pesticides to lawns, with exceptions for noxious weed and invasive species control, agriculture and gardens, and golf courses;
4) Require the Executive to conduct a public outreach and education campaign before and during the implementation of the Bill;
5) Generally prohibit the application of non-essential and neonicotinoid pesticides to County-owned property; and
6) Require the County to adopt an Integrated Pest Management program.

Bill 52-14 has an expiration date of January 1, 2019.

Public Hearings and Correspondence

The Committee held public hearings on the Bill on January 15 and February 12, with 38 people testifying in January, and 30 speaking in February. In addition to the public hearing testimony, the Bill has been, and continues to be, the subject of a huge amount of written correspondence. The testimony and correspondence have coalesced around several recurring themes, which frame major issues for the Committee to examine as it considers the Bill. These themes include: (1) existing regulation of pesticides, particularly at the State and federal level is, or is not, sufficient; (2) chemical pesticides pose, or do not pose, serious threats to human health; (3) pesticides threaten, or do not threaten, the health of pollinators and the Chesapeake Bay watershed; and (4) it is, or is not, possible or feasible to maintain lawns and playing fields without the use of chemical pesticides.

[19] http://www.epa.gov/opp00001/factsheets/ipm.htm

Agenda for March Worksessions

Two T&E worksessions on Bill 52-14 are scheduled for March 2015: March 16 and March 30. Council President Leventhal noted in his memorandum accompanying the Bill at introduction that this issue is extraordinarily complex, and that a thoughtful approach with input from experts in the field will be critical to a well-informed decision. With that in mind, the March worksessions will be focused on allowing the Committee to explore, with guidance from several experts, the several issues related to pesticide regulation. At the March 16th worksession, the Committee will have the opportunity to hear first from a panel of regulators working at the County, State, and federal levels of government. A second panel at this worksession will consist of physicians with expertise in environmental health and toxicology, and an environmental chemist with 39 years of experience in environmental and human risk assessment, with a focus on pesticides. The March 30 worksession will be structured to allow the Committee to engage in dialogue with experts in pollinator and Chesapeake Bay watershed health, turf management experts, and public- and private-sector landscaping professionals. Collectively, these worksessions should give the Committee the information it needs to answer the questions of *whether* there is a need to further regulate pesticides, *why* a need exists (if it exists), and *how* best to meet that need.

Panel 1: Existing Regulatory Framework

One recurring question in public hearing testimony and correspondence regarding Bill 52-14 was whether additional regulation is necessary in view of the existing work done at the State and federal levels. The first panel for this worksession will be composed of representatives of the County's Department of Environmental Protection, the Maryland Department of Agriculture, and the U.S. Environmental Protection Agency. Each of these entities plays a complementary role in the regulation of pesticides in the County, and an understanding of these roles, and the scope of the work done at each level will assist the Committee in answering that question.

County Pesticide Regulation

Chapter 33B, Pesticides, of the County Code, currently consists of four key requirements. Chapter 33B requires:

- a custom applicator[20] to provide certain information to new customers before and after the application of a pesticide;
- posting of notice after application of pesticide to a lawn by a custom applicator;
- retail sellers of pesticides to provide notice signs that are required by the County, as well as the product label required under FIFRA; and
- retail sellers of pesticides to comply with certain transport, display, and storage requirements.

State Pesticide Regulation

There are two components of Maryland law regulating pesticides that are pertinent to the consideration of a restriction on the use of pesticides for lawn care: the Maryland Pesticide

[20] "Custom applicator" is defined in Chapter 33B as "a person engaged in the business of applying pesticides."

Registration and Labeling Law, and the Pesticide Applicator's Law. Selected materials related to MDA's pesticide regulation, taken from the MDA website, are at ©82-134.

Maryland Pesticide Registration and Labeling Law

State law[21] requires the registration of each brand or product name of a pesticide before it can be distributed in the State. The registration must be made annually. The law also imposes packaging and labeling requirements on certain pesticides. The Maryland Secretary of Agriculture may suspend or cancel the registration of a pesticide if it does not comply with State law, and the law provides for several enforcement mechanisms, including administrative monetary penalties, stop-sale orders, and seizure and condemnation of noncompliant pesticides.

Pesticide Applicator's Law

State law[22] also establishes a licensing and certification regime for several practices involving pesticide application. Under this regime, pesticide business licenses, pest control consulting licenses, not-for-hire licenses, public agency permits, and certification of commercial applicators and certain private applicators are required. The law also requires certain information to be supplied by licensees to customers, and the posting of signs at the time of certain applications of pesticides. The law also requires each county board of education to implement in its schools an Integrated Pest Management system approved by MDA. A synopsis of the provisions of the Pesticide Applicator's Law and associated regulations is at ©127-134.

Federal Pesticide Regulation

Federal law requires that all pesticides must be registered by the EPA prior to sale or distribution in the United States. EPA must determine that a pesticide, used according to label directions, can be used with a reasonable certainty of no harm to human health and without posing unreasonable risks to the environment before registering it. EPA requires more than 100 different scientific studies and tests from applicants in making such determinations. EPA also sets "tolerances" (maximum pesticide residue levels), for pesticides that may be used on food or feed crops, for the amount of the pesticide that can legally remain in or on foods. Key components of EPA's role in pesticide regulation are briefly described below;[23] a more in-depth, yet still concise, discussion of EPA's pesticide regulatory programs can be found in the "Agency Response to 'Pesticides in the Air – Kids at Risk: Petition to EPA to Protect Children from Pesticide Drift (2009)'" at ©135-179.[24]

Pre-Registration Evaluation

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[21] Maryland Code, Agriculture Article, Title 5. Pesticide and Pest Control, Subtitle 1. Maryland Pesticide Registration and Labeling Law (§§5-101 through 5-211).

[22] Maryland Code, Agriculture Article, Title 5. Pesticide and Pest Control, Subtitle 2. Pesticide Applicator's Law (§§5-201 through 5-114)

[23] The information in this section is largely summarized from the EPA website. More comprehensive information about EPA's pesticide regulation can be found at: http://www2.epa.gov/pesticide-registration

[24] Specific discussion of the EPA's regulatory program is at ©146-159.

Before registering a pesticide, EPA evaluates it for impacts on human health in a process called risk assessment. EPA pesticide risk assessment is a four step process:

1. Hazard Identification: The identification of potential health effects that may occur from different types of pesticide exposure.
2. Dose-Response Assessment: Consideration of the dose levels at which adverse effects are observed in test animals, and using that data to calculate an equal dose in humans.
3. Exposure Assessment: Consideration of how a person may be exposed to a pesticide: (1) inhalation; (2) absorption through the skin (dermal); and through the mouth or digestive tract (oral).
4. Risk Characterization: Combining the hazard, dose-response, and exposure assessments to describe the overall risk from a pesticide.

In conducting risk assessments, EPA considers studies conducted over different periods of time that measure specific effects. These include measurements of:

1. Acute toxicity (short-term or single exposure);
2. Sub-chronic toxicity (exposure over an intermediate period of time, i.e., 60-90 days);
3. Chronic toxicity (long-term exposure, repeated exposure over most of the test animal's life span); and
4. Developmental and reproductive effects (effects on the fetus of an exposed female, and effects of exposure on the ability to successfully reproduce).

Registration Classifications

EPA registers pesticides and their use on specific pests and under specific circumstances. A pesticide registered for use on apples may not be used legally on grapes, or an insecticide registered for "outdoor use" may not legally be used inside a building. In some circumstances, use of a registered pesticide may be *restricted* to pesticide applicators with special training. EPA classifies pesticides into two categories: general use pesticides and restricted use pesticides. Restricted use pesticides - which make up about a quarter of total pesticides used – must only be applied by or under the direct supervision of trained and certified applicators.[25]

Registration Review

In 2006, EPA initiated a new program called registration review to reevaluate all pesticides on a regular cycle. The program's goal is to review each pesticide's active ingredient every 15 years to make sure that as the ability to assess risks to human health and the environment evolves and as policies and practices change, all pesticide products in the marketplace can still be used safely.

Reregistration & Tolerance Reassessment

EPA has completed a one-time program to review older pesticides (those initially registered before November 1984) under FIFRA to ensure that they meet current scientific and

[25] The current list of Restricted Use Products (RUPs) is at: http://www.epa.gov/opprd001/rup/rupreport.pdf

regulatory standards. This process, called reregistration, considers the human health and ecological effects of pesticides and results in actions to reduce risks that are of concern. Implementation of the decisions will continue beyond the 2008 completion of the reviews.

Pesticide Labeling

Pesticide labeling is a key component of EPA's regulation of pesticides. EPA reviews the product label as part of the registration process for pesticides. The label provides critical information about how to handle and safely use the pesticide product and avoid harm to human health and the environment.

Possible issues to be explored with Panel 1 include:

- Enforcement of existing County pesticide laws;
- The adequacy of State and federal laws in protecting the public from misuse of pesticides by non-commercial users;
- The adequacy of EPA's reliance on applicant-submitted data and animal testing in its risk assessment process; and
- The frequency with which EPA denies or cancels a pesticide registration.

Panel 2: Human Health Concerns and Risk Assessment

Health Concerns and Pesticides

There is growing evidence of harmful effects associated with long-term use of or exposure to chemical pesticides.[26] While there is not at present a consensus on causation, pesticide exposure has been linked to the following health problems: birth defects[27]; numerous cancers, including non-Hodgkins lymphoma[28]; Parkinson's disease and other neurological disorders[29]; immune system problems[30]; and male infertility.[31] In addition to potential links to human health problems, neonicotinoids, a class of insecticide chemically related to nicotine, have been linked to population declines in bees, which serve an important function in pollination.[32]

A view of the health concerns associated with pesticide exposure, and the basis for those concerns is presented in the attachments to this memorandum. Council President Leventhal has discussed many of the health issues surrounding pesticide use in his memorandum at ©14-64. Attachments to the memorandum include the Policy Statement of the American Academy of Pediatrics (AAP) on Pesticide Exposure in Children (©18-25), as well as a number of studies and articles referenced in the memorandum. The Technical Report that is the basis for the the AAP Policy Statement on Pesticide Exposure in Children is at ©180-207. A collection of peer-reviewed

[26] http://www.nrdc.org/health/kids/ocar/chap5.asp
[27] http://www.webmd.com/baby/news/20090327/do-pesticides-make-birth-defects-crop-up
[28] http://www.cfp.ca/content/53/10/1704.short
[29] http://www.scientificamerican.com/article/parkinsons-disease-and-pesticides-whats-the-connection/
[30] http://www.wri.org/publication/pesticides-and-immune-system
[31] http://weedingtech.com/new-study-suggests-exposure-to-roundup-herbicide-could-lead-to-male-infertility-2/
[32] http://usnews.nbcnews.com/_news/2012/03/29/10921493-neonicotinoid-pesticides-tied-to-crashing-bee-populations-2-studies-find

studies and relevant reports on pesticides and human and environmental health, submitted by Safe Grow Montgomery, is at ©208-212. Dr. Philip J. Landrigan, MD, MSc,[33] has submitted a letter (©213-221) to the Council expressing his views on the health effects of pesticide exposure. Dr. Garrettson, who will be part of Panel 2, has submitted several abstracts from studies demonstrating links between pesticides and health problems, with a focus on reproductive health (©224-235).

Dr. Cohen will be part of Panel 2, and will be presenting a counterpoint to the position that pesticides present unacceptable risks to human health and are inadequately regulated. He has submitted copies of the Powerpoint slides he will be presenting at the worksession, as well as a letter responding to a number of issues raised at the public hearing related to concerns about the health risks of pesticides (©236-285).

Councilmember Berliner invited a representative of the National Cancer Institute (NCI) at the National Institutes of Health (©286-287) to attend this worksession. Stephen J. Chanock, MD, Director of the Division of Cancer Epidemiology and Genetics at NCI, by letter dated March 11, respectfully declined the invitation, saying that "NCI scientists do not typically weigh in on regulatory or public policy decisions." Dr. Chanock's letter does provides some discussion of the state of science with regard to carcinogenicity of pesticides (©288-290).

Possible issues to be explored with Panel 2 include:

- Whether the relationship between pesticide use and health problems is causal, correlational, or neither;
- Whether certain pesticides present are particularly dangerous to human health;
- Whether pesticide risks are predominantly related to agricultural use/food consumption or lawn care uses; and
- Whether measures short of a prohibition can be effective in reducing pesticide exposure.

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[33] Dr. Landrigan is a pediatrician, epidemiologist, and Dean for Global Health in the Icahn School of Medicine at Mount Sinai, Professor and Chairman of the Department of Preventive Medicine. And Professor of Pediatrics.

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Bill No. 52-14
Concerning: Pesticides – Notice Requirements – Non-essential Pesticides – Prohibitions
Revised: October 22, 2014
Draft No. 9
Introduced: October 28, 2014
Expires: April 28, 2016
Enacted:
Executive:
Effective:
Sunset Date: January 1, 2019
Ch. ____, Laws of Mont. Co. ____

COUNTY COUNCIL
FOR MONTGOMERY COUNTY, MARYLAND

By: Council Vice President Leventhal and Councilmembers Elrich, Riemer, Floreen, and Navarro

AN ACT to:

(1) require posting of notice for certain lawn applications of pesticide;
(2) prohibit the use of certain pesticides on lawns;
(3) prohibit the use of certain pesticides on certain County-owned property
(4) require the County to adopt an integrated pest management program for certain County-owned property; and
(5) generally amend County law regarding pesticides.

By amending
Montgomery County Code
Chapter 33B, Pesticides
Sections 33B-1, 33B-2, 33B-3, 33B-4, 33B-5, 33B-6, and 33B-7

By adding
Montgomery County Code
Chapter 33B, Pesticides
Articles 2, 3, 4, and 5
Sections 33B-8, 33B-9, 33B-10, 33B-11, 33B-12, and 33B-13

Boldface	*Heading or defined term.*
Underlining	*Added to existing law by original bill.*
[Single boldface brackets]	*Deleted from existing law by original bill.*
Double underlining	*Added by amendment.*
[[Double boldface brackets]]	*Deleted from existing law or the bill by amendment.*
* * *	*Existing law unaffected by bill.*

The County Council for Montgomery County, Maryland approves the following Act:

Sec. 1. Sections 33B-1, 33B-2, 33B4, 33B-5, 33B-6 and 33B-7 are amended, and Sections 33B-8, 33B-9, 33B-10, 33B-11, 33B-12, and 33B-13 are added as follows:

ARTICLE 1. General Provisions

33B-1. Definitions.

In this [chapter] Chapter:

Agriculture means the business, science, and art of cultivating and managing the soil, composting, growing, harvesting, and selling sod, crops and livestock, and the products of forestry, horticulture and hydroponics; breeding, raising, or managing livestock, including horses, poultry, fish, game and fur-bearing animals, dairying, beekeeping and similar activities, and equestrian events and activities.

Custom applicator means a person engaged in the business of applying pesticides.

Department means the Department of Environmental Protection.

Director means Director of the Department of Environmental Protection[,] or the Director's designee.

Integrated pest management means a process for managing pests that:

(1) uses monitoring to determine pest injury levels;

(2) combines biological, cultural, mechanical, physical, and chemical tools and other management practices to control pests in a safe, cost effective, and environmentally sound manner that contributes to the protection of public health and sustainability;

(3) uses knowledge about pests, such as infestations, thresholds, life histories, environmental requirements, and natural control of pests; and

(4) uses non-chemical pest-control methods and the careful use of least-toxic chemical methods when non-chemical methods have been exhausted or are not feasible.

Larvicide means a pesticide designed to kill larval pests.

Lawn means an area of land, except agricultural land, that is:

(1) [Mostly] mostly covered by grass, other similar herbaceous plants, shrubs, or trees; and

(2) [Kept] kept trim by mowing or cutting.

Lawn includes an athletic playing field other than a golf course. *Lawn* does not include a garden.

Neonicotinoid means a class of neuro-active pesticides chemically related to nicotine. Neonicotinoid includes acetamiprid, clothianidin, dinotefuran, imidacloprid, nitenpyram, nithiazine, thiacloprid, and thiamethoxam.

Non-essential pesticide means a pesticide designated as a non-essential pesticide under Section 33B-4.

Pest means an insect, snail, slug, rodent, nematode, fungus, weed, or other form of plant or animal life or microorganism (except a microorganism on or in a living human or animal) that is normally considered to be a pest or defined as a pest by applicable state regulations.

Pesticide means a substance or mixture of substances intended or used to:

(1) prevent, destroy, repel, or mitigate any pest;

(2) be used as a plant regulator, defoliant, or desiccant; or

(3) be used as a spray adjuvant, such as a wetting agent or adhesive.

However, *pesticide* does not include an antimicrobial agent, such as a disinfectant, sanitizer, or deodorizer, used for cleaning that is not considered a pesticide under any federal or state law or regulation.

Private lawn application means the application of a pesticide to a lawn on property owned by or leased to the person applying the pesticide. *Private lawn application* does not include:

(1) applying a pesticide for the purpose of engaging in agriculture;

(2) applying a pesticide around or near the foundation of a building for purpose of indoor pest control;

(3) applying a pesticide to a golf course or turf farm.

Vector means an animal, insect, or microorganism that carries and transmits an infectious pathogen into another organism.

[33B-4.] 33B-2. Signs with retail purchase of pesticide.

A person who sells at retail a pesticide or material that contains a pesticide must make available to a person who buys the pesticide or material that contains a pesticide:

(a) [Notice] notice signs and supporting information that are approved by the [department] Department; and

(b) [The] the product label or other information that the federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) [, 7 U.S.C. 136 et seq.,] requires for sale of the pesticide.

The Department must enforce this Section and must annually inspect each person who sells at retail a pesticide or material that contains a pesticide.

[33B-5] 33B-3. Storage and handling of pesticides.

* * *

[33B-6] 33B-4. Regulations.

(a) The [County] Executive must adopt regulations to carry out this Chapter under method (2).

(b) The Executive must include in the regulations adopted under this [section] Section the minimum size or quantity of pesticide subject to [section 33B-4] Section 33B-2.

(c) The Executive must include in the regulations adopted under this Section a list of non-essential pesticides. The list of non-essential pesticides must include:

(1) all pesticides classified as "Carcinogenic to Humans" or "Likely to Be Carcinogenic to Humans" by the U.S. Environmental Protection Agency;

(2) all pesticides classified by the U.S. Environmental Protection Agency as a "Restricted Use Product";

(3) all pesticides classified as a "Class 9" pesticide by the Ontario, Canada, Ministry of the Environment;

(4) all pesticides classified as a "Category 1 Endocrine Disruptor" by the European Commission; and

(5) any other pesticides which the Executive determines are not critical to pest management in the County.

(d) The Executive must include in the regulations adopted under this Section a list of invasive species that may be detrimental to the environment in the County.

(e) The Executive must review and update the lists of non-essential pesticides and invasive species designated under subsections (c) and (d) by July 1 of each year.

[33B-7] 33B-5. Penalty for violating chapter.

(a) Any violation of this Chapter is a class C violation.

(b) Each day a violation continues is a separate offense.

ARTICLE 2. Notice Requirements.

[33B-2] 33B-6. Notice about pesticides to customer.

(a) In this [section] Section:

(1) Customer means a person who makes a contract with a custom applicator to have the custom applicator apply a pesticide to a lawn.

(2) New customer includes a customer who renews a contract with a custom applicator.

(b) A custom applicator must give to a new customer:

(1) [Before] before application, a list of:

[a.](A) [The] the trade name of each pesticide that might be used;

[b.](B) [The] the generic name of each pesticide that might be used; and

[c.](C) [Specific] specific customer safety precautions for each pesticide that might be used; and

(2) [After] after application, a list of:

[a.](A) [The] the trade name of each pesticide actually used; and

[b.](B) [The] the generic name of each pesticide actually used; and

(3) [A] a written notice about pesticides prepared by the [department] Department under subsection (c) [of this section].

(c) The [department] Department must prepare, keep current, and provide to a custom applicator a written notice about pesticides for the custom applicator to give to a customer under subsection (b) [of this section].

(d) The notice prepared by the [department] Department under subsection (c) [of this section] must include:

(1) [Government] government agency phone numbers to call to:

[a.](A) [Make] make a consumer complaint;

[b.](B) [Receive] receive technical information on pesticides; and

[c.] (C) [Get] get assistance in the case of a medical emergency;

(2) [A] a list of general safety precautions a customer should take when a lawn is treated with a pesticide;

(3) [A] a statement that a custom applicator must:

[a.](A) [Be] be licensed by the Maryland Department of Agriculture; and

[b.](B) [Follow] follow safety precautions; and

(4) [A] a statement that the customer has the right to require the custom applicator to notify the customer before each treatment of the lawn of the customer with a pesticide.

[33B-3] 33B-7. Posting signs after application by custom applicator.

(a) Immediately after a custom applicator treats a lawn with a pesticide, the custom applicator must [post a sign on the lawn] place markers within or along the perimeter of the area where pesticides will be applied.

(b) A [sign posted] marker required under this [section] Section must:

(1) [Be] be clearly visible [from the principal place of access to] to persons immediately outside the perimeter of the property;

(2) [Be] be a size, form, and color approved by the [department] Department;

(3) [Be] be made of material approved by the [department] Department; [and]

(4) [Have] have wording with content and dimensions approved by the [department] Department[.]; and

(5) be in place on the day that the pesticide is applied.

33B-8. Posting signs after application by property owner or tenant.

(a) A person who performs a private lawn application treating an area more than 100 square feet must place markers within or along the perimeter of the area where pesticides will be applied.

(b) A marker required under this Section must:

(1) be clearly visible to persons immediately outside the perimeter of the property;

(2) be a size, form, and color approved by the Department;

(3) be made of material approved by the Department; and

(4) have wording with content and dimensions approved by the Department; and

(5) be in place on the day that the pesticide is applied.

ARTICLE 3. Application restrictions.

33B-9. Prohibited application.

A person must not apply a non-essential pesticide to a lawn.

33B-10. Exceptions and Exemptions.

(a) A person may apply a non-esssential pesticide for the following purposes:

(1) for the control of weeds as defined in Chapter 58, Weeds;

(2) for the control of invasive species listed in a regulation adopted under Subsection 33B-4(d);

(3) for pest control while engaged in agriculture; and

(4) for the maintenance of a golf course.

(b) A person may apply to the Director for an exemption from the prohibition of Section 33B-9 for a non-essential pesticide. The Director may grant an exemption to apply a non-essential pesticide on property where application is prohibited under Section 33B-9 if the applicant shows that:

(1) effective alternatives are unavailable;

(2) granting an exemption will not violate State or federal law; and

(3) use of the non-essential pesticide is necessary to protect human health or prevent significant economic damage.

(c) A person may apply to the Director for an emergency exemption from the prohibition in Section 33B-9 if a pest outbreak poses an imminent threat to public health or if significant economic damage would result from the inability to use a pesticide prohibited by Section 33B-9. The Director may impose specific conditions for the granting of emergency exemptions.

33B-11. Outreach and Education Campaign.

The Executive must implement a public outreach and education campaign before and during implementation of the provisions of this Article. This campaign should include:

(a) informational mailers to County households;

(b) distribution of information through County internet and web-based resources;

(c) radio and television public service announcements;

(d) news releases and news events;

(e) information translated into Spanish, French, Chinese, Korean, Vietnamese, and other languages, as needed;

(f) extensive use of County Cable Montgomery and other Public, Educational, and Government channels funded by the County; and

(g) posters and brochures made available at County events, on Ride-On buses and through Regional Service Centers, libraries, recreation facilities, senior centers, public schools, Montgomery College, health care providers, hospitals, clinics, and other venues.

ARTICLE 4. County Property

33B-12. Prohibition on County-owned property.

(a) *Prohibition.* Except as provided in subsection (b), a person must not apply to any property owned by the County:

(1) a non-essential pesticide; or

(2) a nionicotinoid.

(b) *Exceptions.*

(1) A person may use any larvicide or rodenticide on property owned by the County as a public health measure to reduce the spread of disease vectors under recommendations and guidance provided by the Centers for Disease Control and Prevention, the United States Environmental Protection Agency, or the State Department of Agriculture. Any rodenticide used must be in a tamper-proof product, unless the rodenticide is designed and registered for a specific environment inaccessible to humans and pets.

(2) A person may use a non-essential pesticide or neonicotinoid for the purposes set forth in Subsection 33B-10(a).

(3) A person may use a non-essential pesticide or neonicotinoid on property owned by the County if the Director determines, after consulting the Directors of General Services and Health and Human Services, that the use of pesticide is necessary to protect

human health or prevent imminent and significant economic damage, and that no reasonable alternative is available. If a pesticide is used under this paragraph, the Director must, within 30 days after using the pesticide, report to the Council on the reasons for the use of the pesticide.

33B-13. Integrated pest management.

(a) *Adoption of program.* The Department must adopt, by a method (2) regulation, an integrated pest management program for property owned by the County.

(b) *Requirements.* Any program adopted under subsection (a) must require:

(1) monitoring the turf or landscape;

(2) accurate record-keeping documenting any potential pest problem;

(3) evaluating the site for any injury caused by a pest and determining the appropriate treatment;

(4) using a treatment that is the least damaging to the general environment and best preserves the natural ecosystem;

(5) using a treatment that will be the most likely to produce long-term reductions in pest control requirements and is operationally feasible and cost effective in the short and long term;

(6) using a treatment that minimizes negative impacts to non-target organisms;

(7) using a treatment that is the least disruptive of natural controls;

(8) using a treatment that is the least hazardous to human health; and

(9) exhausting the list of all non-chemical and organic treatments available for the targeted pest before using any synthetic chemical treatments.

(c) The Department must provide training in integrated pest management for each employee who is responsible for pest management.

Sec. 2. Initial Lists of Non-Essential Pesticides and Invasive Species. The Executive must submit the lists of non-essential pesticides and invasive species required by Subsections 33B-4(c) and (d) to the Council for approval by October 1, 2015.

Sec. 3. Effective Date. The prohibitions on use of non-essential pesticides contained in Section 33B-9 and the prohibitions on use of non-essential pesticides and neonicotinoids contained in Section 33B-12 take effect on January 1, 2016.

Sec. 4. Expiration. This Act and any regulation adopted under it expires on January 1, 2019.

Approved:

George Leventhal, President, County Council — Date

Approved:

Isiah Leggett, County Executive — Date

This is a correct copy of Council action.

Linda M. Lauer, Clerk of the Council — Date

LEGISLATIVE REQUEST REPORT

Bill 52-14

Pesticides – Notice Requirements – Non-Essential Pesticides - Prohibitions

DESCRIPTION:	This Bill would require posting of notice for certain lawn applications of pesticide, prohibit the use of certain pesticides on lawns, prohibit the use of certain pesticides on certain County-owned property and require the County to adopt an integrated pest management program for certain County-owned property.
PROBLEM:	Long term use of and exposure to certain chemical pesticides has been linked to several health problems, including birth defects, cancer, neurological problems, immune system problems, and male infertility.
GOALS AND OBJECTIVES:	To protect the health of families, especially children, from the unnecessary risks associated with the use of certain pesticides that have been linked to a wide-range of diseases.
COORDINATION:	Department of Environmental Protection
FISCAL IMPACT:	To be requested.
ECONOMIC IMPACT:	To be requested.
EVALUATION:	To be requested.
EXPERIENCE ELSEWHERE:	To be researched.
SOURCE OF INFORMATION:	Josh Hamlin, Legislative Attorney
APPLICATION WITHIN MUNICIPALITIES:	To be researched.
PENALTIES:	Class C violation

MONTGOMERY COUNTY COUNCIL
ROCKVILLE, MARYLAND

GEORGE LEVENTHAL
COUNCILMEMBER
AT-LARGE

MEMORANDUM

October 22, 2014

TO: Councilmembers

FROM: George Leventhal, Council Vice President George L Leventhal

SUBJECT: Pesticide Legislation

This coming Tuesday, October 28, I will be introducing legislation aimed at protecting the health of families – and especially children - from the unnecessary risks associated with the use of certain cosmetic pesticides that have been linked to a wide-range of diseases, and which provide no health benefits.

As you know, for the better part of the last year, I have been working towards introducing legislation on this matter. Since the September 2013 meeting of the T&E committee, I have met with countless stakeholders, on both sides of the issue, to learn more about how pesticides are being applied in the county, what other governments are doing to ensure that the public's health is being protected, and what the latest research tells us about their risks. The legislation that I am introducing on Tuesday incorporates feedback I received from proponents and opponents on the previous draft of the bill, which I shared with your offices back in May. The result is a bill that balances the rights of homeowners to maintain a beautiful lawn with the rights of residents who prefer to not be exposed to chemicals that have known health effects; I view this bill as a starting point in our discussion which can be tweaked along the way.

I want to preface my concerns by affirming the value of pesticides when they are used to protect public health, the environment, our food or our water supply, but when pesticides are used solely to improve the appearance of landscapes, they can cause more harm than good. In my view, cosmetic pesticides present a substantial threat to the health of today's children. The American Academy of Pediatrics states that children face the greatest risk from the chemicals they contain, and that epidemiologic evidence demonstrates associations between early life exposure to pesticides and pediatric cancers, decreased cognitive function and behavioral problems such as ADHD.[1] Certain toxic chemicals can cause permanent brain damage in children even at low levels of exposure that would have little to no adverse effect in an adult.[2] A child doesn't even

[1] *Pediatrics*, Pesticide Exposure in Children, Volume 130, No. 6, 1757 – 1763, December, 2012
[2] Dr. Phillippe Grandjean, MD, Dr. Phillip Landrigan, MD, *The Lancet Neurology*, Neurobehavioral Effects of Developmental Toxicity, Volume 13, Issue 3, 330-338, March 2014

have to be directly exposed to a pesticide to suffer negative health outcomes. During pregnancy, chemicals in women can cross the placenta and result in higher fetal exposure than the mother has been exposed to. Prenatal exposure to certain chemicals has been documented to increase the risk of cancer in childhood.[3] Virtually every pregnant woman in the United States is exposed to multiple chemicals during a sensitive period of fetal development that have been linked to adverse reproductive and developmental outcomes.[4]

Adults are also at risk of developing serious health problems due to pesticide exposure. Researchers at the National Institutes of Health have linked pesticide use to a wide range of diseases and conditions. Exposure to certain pesticides has been linked to Parkinson's disease, diabetes, leukemia, lymphoma, lupus, rheumatoid arthritis, dementia, reproductive dysfunction, Alzheimer's disease, and variety of cancers including breast, colon, prostate and lung cancer.[5]

In addition to the adverse health effects to humans, pesticides can also affect animals, both pets and wildlife, and our waterways. A recent study by the United States Geological Survey has found that 90% of urban area waterways now have pesticide levels high enough to harm aquatic life, and moreover, the USGS said the harm to aquatic life was likely understated in their report.[6] Terrestrial wildlife is also being harmed by the use of certain pesticides. The most concerning example involves honeybees, which pollinate nearly one-third of the food we eat, and a particular class of pesticides called neonicotinoids. Neonicotinoids have been repeatedly and strongly linked with the collapse of honey bee colonies. In just the last year, Maryland lost nearly 50 percent of its honeybee population, an increase over previous years, which averaged about a one-third loss annually.[7]

Before I describe what this bill does, let me describe what this bill does not do. This bill does not ban the use of all pesticides; it would, however, restrict the use of certain toxic chemicals that are most dangerous to human health. This bill does not prohibit the use of any pesticide for gardens. And this bill would not prohibit the use of any pesticide for agricultural use. What this bill does do is seek to limit children's exposure to harmful pesticides in places where children are most likely to be exposed to them. That being said, the major provisions of the bill are:

1) Require the posting of notice when a property owner applies a pesticide to an area of lawn more than 100 square feet, consistent with the notice requirements for when a landscaping business treats a lawn with a pesticides;
2) Require the Executive to designate a list of "non-essential" pesticides including:
    - all pesticides classified as "Carcinogenic to Humans" or "Likely to Be Carcinogenic to Humans" by the U.S. EPA;
    - all pesticides classified by the U.S. EPA as "Restricted Use Products;"

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[3] *American College of Obstetricians & Gynecologists*, Committee Opinion No. 575. American College of Obstetricians and Gynecologists. 931–5. October 2013

[4] *Environmental Health Perspectives*, Environmental Chemicals in Pregnant Women in the United States: NHANES 2003–2004, Tracey J. Woodruff, Ami R. Zota, Jackie M. Schwartz, Volume 119, No. 6, 878–885. June 2011

[5] Jan Ehrman, *NIH Record*, Pesticide Use Linked to Lupus, Rheumatoid Arthritis, http://nihrecord.nih.gov/newsletters/2011/03_18_2011/story4.htm (accessed August 3, 2014)

[6] *U.S. Geological Survey*, An Overview Comparing Results from Two Decades of Monitoring for Pesticides in the Nation's Streams and Rivers, 1992–2001 and 2002–2011, Wesley W. Stone, Robert J. Gilliom, Jeffrey D. Martin, http://pubs.usgs.gov/sir/2014/5154/pdf/sir2014-5154.pdf (accessed October 20, 2014)

[7] Tim Wheeler, Mysterious bee die-off continues, extends beyond winter, *Baltimore Sun*, http://articles.baltimoresun.com/2014-05-15/features/bal-mysterious-bee-dieoff-continues-nearly-half-maryland-hives-lost-20140515_1_bee-informed-partnership-honey-bee-beekeepers (accessed October 20, 2014)

- all pesticides classified as "Class 9" pesticides by the Ontario, Canada, Ministry of the Environment; and
- all pesticides classified as "Category 1 Endocrine Disruptors" by the European Commission

3) Generally prohibit the application of non-essential pesticides to lawns, with exceptions for noxious weed and invasive species control, agriculture and gardens, and golf courses;
4) Require the Executive to conduct a public outreach and education campaign before and during the implementation of the Bill;
5) Generally prohibit the application of a non-essential or neonicotinoid pesticide to County-owned property; and
6) Require the County to adopt an Integrated Pest Management program.
7) Sunset the act and any regulation adopted under it on January 1, 2019

The pesticide industry will respond to this legislation by saying "the science isn't there" and that "all pesticides are extensively tested and approved as safe by the EPA," but while both statements sound believable, they belie the truth. In response to the charge that the science isn't there to legislate, the absence of incontrovertible evidence does not justify inaction. As evidenced by this memo, the number of studies from respected institutions of science linking pesticides to a variety of cancers, neurodevelopmental disorders and diseases is abundant and persuasive. Furthermore, due to the inestimable number of chemical combinations possible from the thousands of products on the market and the complex interactions with the human body, the research that opponents to this legislation will demand will never be possible within the ethical confines of research. The real danger lies not in being exposed to one chemical, but a mixture of chemicals. The EPA risk assessment fails to look at the synergistic effects of multiple chemicals, even though studies show that exposure to multiple chemicals that act on the same adverse outcome can have a greater effect than exposure to an individual chemical.[8]

And to the charge that a pesticide must be safe if it has been approved by the EPA, the Government Accountability Office (GAO) has found that many pesticides are currently being approved for consumer use by the EPA without receipt and review of data that the manufacturer is required to provide on the safety of the chemicals.[9] Alarmingly, in some cases the manufacturer was given two years to submit studies on the effects of a pesticide, and ten years later no studies had been received or reviewed by the EPA.[10] What's more, the EPA itself publishes an entire manual – *Recognition and Management of Pesticide Poisonings* - for healthcare professionals that acknowledges the toxic nature and effects of many pesticides. As an educated populace, we like to think that we have a high bar for pesticide safety in this country, but sadly, when a pesticide has been approved by the EPA, it connotes little about its safety.

Lawn care does not have to be poisonous to people, pets, wildlife, or our waterways. It is simply false to say that you can't have a lush, green lawn - free of weeds - without the use of toxic pesticides. Through proper management of the soil, along with the use of natural, organic alternatives to synthetic pesticides, a high quality landscape can be achieved. And under my

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[8] *National Research Council*. Committee on Improving Risk Analysis Approaches Used by the U.S. EPA. Science and Decisions: Advancing Risk Assessment. Washington, DC: National Academies Press; 2008

[9] *United States Government Accountability Office*. Pesticides – EPA Should Take Steps to Improve its Oversight of Conditional Registrations, http://www.gao.gov/assets/660/656825.pdf (accessed October 20, 2014)

[10] *United States Government Accountability Office*, Pesticides – EPA Should Take Steps to Improve its Oversight of Conditional Registrations, http://www.gao.gov/assets/660/656825.pdf (accessed October 20, 2014)

legislation, residents will still be free to hire any lawn care professional to treat their lawn or to manage their own lawn care.

Much like the public debate that occurred in the 1950's before cigarettes were found to be cancer-causing, I believe we are approaching a similar turning point in the discourse on pesticides as the public is made more aware of the known health effects. In a poll taken earlier this year, more than three-quarters of Marylanders expressed concern about the risk that pesticides pose to them or their families, and when respondents learned of the adverse health effects that pesticides are linked to, 90% of Marylanders expressed concern.[11]

America lags behind by the rest of the developed world in recognizing the serious risks that certain pesticides pose to health and life. The GAO's report confirms that the regulatory approach taken by the EPA is broken and failing the public. In the face of mounting scientific evidence, and in the absence of action on the federal level, I find it impossible not to act now to protect the health of our children. In Montgomery County, we regularly take a precautionary approach to public health and environmental issues, such as with the forthcoming legislation on e-cigarettes and the Council's action on Ten Mile Creek. Our approach to pesticides should be no different.

I have attached all of the studies that I have cited in this memo for your reference, but I hope you will take time to review research beyond what I have provided. If, after reviewing the research, you feel compelled to act as I do, I would welcome your co-sponsorship on this bill.

This issue is among the most technically complex which the Council has ever faced. Therefore, it is critical that we approach this in a thoughtful manner and that we consult with a variety of experts who are knowledgeable in the field so we can make a well-informed decision regarding this important public health issue.

[11] *OpinionWorks*, Maryland Voter Survey on Pesticides http://www.mdpestnet.org/wp-content/uploads/2014/02/Pesticide-Poll-Memo-2-10-14.pdf (Accessed on October 20, 2014)

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Pesticide Exposure in Children

COUNCIL ON ENVIRONMENTAL HEALTH

Pediatrics 2012;130;e1757; originally published online November 26, 2012;
DOI: 10.1542/peds.2012-2757

American Academy of Pediatrics
DEDICATED TO THE HEALTH OF ALL CHILDREN™

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FROM THE AMERICAN ACADEMY OF PEDIATRICS

Organizational Principles to Guide and Define the Child Health Care System and/or Improve the Health of all Children

POLICY STATEMENT

Pesticide Exposure in Children

COUNCIL ON ENVIRONMENTAL HEALTH

KEY WORDS
pesticides, toxicity, children, pest control, integrated pest management

ABBREVIATIONS
EPA—Environmental Protection Agency
IPM—integrated pest management

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abstract

FREE

This statement presents the position of the American Academy of Pediatrics on pesticides. Pesticides are a collective term for chemicals intended to kill unwanted insects, plants, molds, and rodents. Children encounter pesticides daily and have unique susceptibilities to their potential toxicity. Acute poisoning risks are clear, and understanding of chronic health implications from both acute and chronic exposure are emerging. Epidemiologic evidence demonstrates associations between early life exposure to pesticides and pediatric cancers, decreased cognitive function, and behavioral problems. Related animal toxicology studies provide supportive biological plausibility for these findings. Recognizing and reducing problematic exposures will require attention to current inadequacies in medical training, public health tracking, and regulatory action on pesticides. Ongoing research describing toxicologic vulnerabilities and exposure factors across the life span are needed to inform regulatory needs and appropriate interventions. Policies that promote integrated pest management, comprehensive pesticide labeling, and marketing practices that incorporate child health considerations will enhance safe use. *Pediatrics* 2012;130:e1757–e1763

INTRODUCTION

Pesticides represent a large group of products designed to kill or harm living organisms from insects to rodents to unwanted plants or animals (eg, rodents), making them inherently toxic (Table 1). Beyond acute poisoning, the influences of low-level exposures on child health are of increasing concern. This policy statement presents the position of the American Academy of Pediatrics on exposure to these products. It was developed in conjunction with a technical report that provides a thorough review of topics presented here: steps that pediatricians should take to identify pesticide poisoning, evaluate patients for pesticide-related illness, provide appropriate treatment, and prevent unnecessary exposure and poisoning.[1] Recommendations for a regulatory agenda are provided as well, recognizing the role of federal agencies in ensuring the safety of children while balancing the positive attributes of pesticides. Repellents reviewed previously (eg, N,N-diethyl-meta-toluamide, commonly known as DEET; picaridin) are not discussed.[2]

SOURCES AND MECHANISMS OF EXPOSURE

Children encounter pesticides daily in air, food, dust, and soil and on surfaces through home and public lawn or garden application, household insecticide use, application to pets, and agricultural product

TABLE 1 Categories of Pesticides and Major Classes

Pesticide category	Major Classes	Examples
Insecticides	Organophosphates	Malathion, methyl parathion, acephate
	Carbamates	Aldicarb, carbaryl, methomyl, propoxur
	Pyrethroids/pyrethrins	Cypermethrin, fenvalerate, permethrin
	Organochlorines	Lindane
	Neonicotinoids	Imidacloprid
	N-phenylpyrazoles	Fipronil
Herbicides	Phosphonates	Glyphosate
	Chlorophenoxy herbicides	2,4-D, mecoprop
	Dipyridyl herbicides	Diquat, paraquat
	Nonselective	Sodium chlorate
Rodenticides	Anticoagulants	Warfarin, brodifacoum
	Convulsants	Strychnine
	Metabolic poison	Sodium fluoroacetate
	Inorganic compounds	Aluminum phosphide
Fungicides	Thiocarbamates	Metam-sodium
	Triazoles	Fluconazole, myclobutanil, triadimefon
	Strobilurins	Pyraclostrobin, picoxystrobin
Fumigants	Halogenated organic	Methyl bromide, Chloropicrin
	Organic	Carbon disulfide, Hydrogen cyanide, Naphthalene
	Inorganic	Phosphine
Miscellaneous	Arsenicals	Lead arsenate, chromated copper arsenate, arsenic trioxide
	Pyridine	4-aminopyridine

residues.[3–9] For many children, diet may be the most influential source, as illustrated by an intervention study that placed children on an organic diet (produced without pesticide) and observed drastic and immediate decrease in urinary excretion of pesticide metabolites.[10] In agricultural settings, pesticide spray drift is important for residences near treated crops or by take-home exposure on clothing and footwear of agricultural workers.[8,11,12] Teen workers may have occupational exposures on the farm or in lawn care.[13–15] Heavy use of pesticides may also occur in urban pest control.[16]

Most serious acute poisoning occurs after unintentional ingestion, although poisoning may also follow inhalational exposure (particularly from fumigants) or significant dermal exposure.[17]

ACUTE PESTICIDE TOXICITY

Clinical Signs and Symptoms

High-dose pesticide exposure may result in immediate, devastating, even lethal consequences. Table 2 summarizes features of clinical toxicity for the major pesticides classes. It highlights the similarities of common classes of pesticides (eg, organophosphates, carbamates, and pyrethroids) and underscores the importance of discriminating among them because treatment modalities differ. Having an index of suspicion based on familiarity with toxic mechanisms and taking an environmental history provides the opportunity for discerning a pesticide's role in clinical decision-making.[18] Pediatric care providers have a poor track record for recognition of acute pesticide poisoning.[19–21] This reflects their self-reported lack of medical education and self-efficacy on the topic.[22–26] More in-depth review of acute toxicity and management can be found in the accompanying technical report or recommended resources in Table 3.

The local or regional poison control center plays an important role as a resource for any suspected pesticide poisoning.

There is no current reliable way to determine the incidence of pesticide exposure and illness in US children. Existing data systems, such as the American Association of Poison Control Centers' National Poison Data System or the National Institute for Occupational Safety and Health's Sentinel Event Notification System for Occupational Risks,[27,28] capture limited information about acute poisoning and trends over time.

There is also no national systematic reporting on the use of pesticides by consumers or licensed professionals. The last national survey of consumer pesticide use in homes and gardens was in 1993 (Research Triangle Institute study).[29]

Improved physician education, accessible and reliable biomarkers, and better diagnostic testing methods to readily identify suspected pesticide illness would significantly improve reporting and surveillance. Such tools would be equally important in improving clinical decision-making and reassuring families if pesticides can be eliminated from the differential diagnosis.

The Pesticide Label

The pesticide label contains information for understanding and preventing acute health consequences: the active ingredient; signal words identifying acute toxicity potential; US Environmental Protection Agency (EPA) registration number; directions for use, including protective equipment recommendations, storage, and disposal; and manufacturer's contact information.[30] Basic first aid advice is provided, and some labels contain a "note for physicians" with specific relevant medical information. The label does not specify the pesticide class or "other"/"inert" ingredients that may have significant toxicity and can account for up to 99% of the product.

Chronic toxicity information is not included, and labels are predominantly available in English. There is significant use of illegal pesticides (especially in immigrant communities), off-label use, and overuse, underscoring the importance of education, monitoring, and enforcement.[31]

TABLE 2 Common Pesticides: Signs, Symptoms, and Management Considerations[a]

Class	Acute Signs and Symptoms	Clinical Considerations
Organophosphate and N-methyl carbamate insecticides	• Headache, nausea, vomiting, abdominal pain, and dizziness	• Obtain red blood cell and plasma cholinesterase levels
	• Hypersecretion: sweating, salivation, lacrimation, rhinorrhea, diarrhea, and bronchorrhea	• Atropine is primary antidote
	• Muscle fasciculation and weakness, and respiratory symptoms (bronchospasm, cough, wheezing, and respiratory depression)	• Pralidoxime is also an antidote for organophosphate and acts as a cholinesterase reactivator
	• Bradycardia, although early on, tachycardia may be present	• Because carbamates generally produce a reversible cholinesterase inhibition, pralidoxime is not indicated in these poisonings
	• Miosis	
	• Central nervous system: respiratory depression, lethargy, coma, and seizures	
Pyrethroid insecticides	• Similar findings found in organophosphates including the hypersecretion, muscle fasciculation, respiratory symptoms, and seizures	• At times have been mistaken for acute organophosphate or carbamate poisoning
	• Headache, fatigue, vomiting, diarrhea, and irritability	• Symptomatic treatment
	• Dermal: skin irritation and paresthesia	• Treatment with high doses of atropine may yield significant adverse results
		• Vitamin E oil for dermal symptoms
Neonicotinoid insecticides	• Disorientation, severe agitation, drowsiness, dizziness, weakness, and in some situations, loss of consciousness	• Supportive care
	• Vomiting, sore throat, abdominal pain	• Consider sedation for severe agitation
	• Ulcerations in upper gastrointestinal tract	• No available antidote
		• No available diagnostic test
Fipronil (N-phenylpyrazole insecticides)	• Nausea and vomiting	• Supportive care
	• Aphthous ulcers	• No available antidote
	• Altered mental status and coma	• No available diagnostic test
	• Seizures	
Lindane (organochlorine insecticide)	• Central nervous system: mental status changes and seizures	• Control acute seizures with lorazepam
	• Paresthesia, tremor, ataxia and hyperreflexia	• Lindane blood level available as send out
Glyphosate (phosphonate herbicides)	• Nausea and vomiting	• Supportive care
	• Aspiration pneumonia type syndrome	• Pulmonary effects may be secondary to organic solvent
	• Hypotension, altered mental status, and oliguria in severe cases	
	• Pulmonary effects may in fact be secondary to organic solvent	
Chlorophenoxy herbicides	• Skin and mucous membrane irritation	• Consider urine alkalinization with sodium bicarbonate in IV fluids
	• Vomiting, diarrhea, headache, confusion	
	• Metabolic acidosis is the hallmark	
	• Renal failure, hyperkalemia, and hypocalcemia	
	• Probable carcinogen	
Rodenticides (long-acting anticoagulants)	• Bleeding: gums, nose, and other mucous membrane sites	• Consider PT (international normalized ratio)
	• Bruising	• Observation may be appropriate for some clinical scenarios in which it is not clear a child even ingested the agent
		• Vitamin K indicated for active bleeding (IV vitamin K) or for elevated PT (oral vitamin K)

IV, intravenous; PT, prothrombin time.

[a] Expanded version of this table is available in the accompanying technical report.[1]

CHRONIC EFFECTS

Dosing experiments in animals clearly demonstrate the acute and chronic toxicity potential of multiple pesticides. Many pesticide chemicals are classified by the US EPA as carcinogens. The past decade has seen an expansion of the epidemiologic evidence base supporting adverse effects after acute and chronic pesticide exposure in children. This includes increasingly sophisticated studies addressing combined exposures and genetic susceptibility.[1]

Chronic toxicity end points identified in epidemiologic studies include adverse birth outcomes including preterm birth, low birth weight, and congenital

TABLE 3 Pesticide and Child Health Resources for the Pediatrician

Topic/Resource	Additional Information	Contact Information
Management of acute pesticide poisoning		
Recognition and Management of Pesticide Poisonings	Print: fifth (1999) is available in Spanish, English; 6th edition available 2013	http://www.epa.gov/pesticides/safety/healthcare/handbook/handbook.htm
Regional Poison Control Centers		1 (800) 222-1222
Chronic exposure information and specialty consultation		
The National Pesticide Medical Monitoring Program (NPMMP)	Cooperative agreement between Oregon State University and the US EPA. NPMMP provides informational assistance by E-mail in the assessment of human exposure to pesticides	npmmp@oregonstate.edu or by fax at (541) 737-9047
Pediatric Environmental Health Specialty Units (PEHSUs)	Coordinated by the Association of Occupational and Environmental Clinics to provide regional academically based free consultation for health care providers	www.aoec.org/PEHSU.htm; toll-free telephone number (888) 347-AOEC (extension 2632)
Resources for safer approaches to pest control		
US EPA	Consumer information documents	www.epa.gov/oppfead1/Publications/Cit_Guide/citguide.pdf
Citizens Guide to Pest Control and Pesticide Safety	• Household pest control • Alternatives to chemical pesticides • How to choose pesticides • How to use, store, and dispose of them safely • How to prevent pesticide poisoning • How to choose a pest-control company	
Controlling pests	Recommended safest approaches and examples of programs	www.epa.gov/pesticides/controlling/index.htm
The University of California Integrative Pest Management Program	Information on IPM approaches for common home and garden pests	www.ipm.ucdavis.edu
Other resources		
National research programs addressing children's health and pesticides	• NIEHS/EPA Centers for Children's Environmental Health & Disease Prevention Research	www.niehs.nih.gov/research/supported/centers/prevention
	• The National Children's Study	www.nationalchildrensstudy.gov/Pages/default.aspx
US EPA	Pesticide product labels	www.epa.gov/pesticides/regulating/labels/product-labels.htm#projects
The National Library of Medicine "Tox Town"	Section on pesticides that includes a comprehensive and well-organized list of web link resources on pesticides	http://toxtown.nlm.nih.gov/text_version/chemicals.php?id=23

anomalies, pediatric cancers, neurobehavioral and cognitive deficits, and asthma. These are reviewed in the accompanying technical report. The evidence base is most robust for associations to pediatric cancer and adverse neurodevelopment. Multiple case-control studies and evidence reviews support a role for insecticides in risk of brain tumors and acute lymphocytic leukemia. Prospective contemporary birth cohort studies in the United States link early-life exposure to organophosphate insecticides with reductions in IQ and abnormal behaviors associated with attention-deficit/hyperactivity disorder and autism. The need to better understand the health implications of ongoing pesticide use practices on child health has benefited from these observational epidemiologic data.[32]

EXPOSURE PREVENTION APPROACHES

The concerning and expanding evidence base of chronic health consequences of pesticide exposure underscores the importance of efforts aimed at decreasing exposure.

Integrated pest management (IPM) is an established but undersupported approach to pest control designed to minimize and, in some cases, replace the use of pesticide chemicals while achieving acceptable control of pest populations.[33] IPM programs and knowledge have been implemented in agriculture and to address weeds and pest control in residential settings and schools, commercial structures, lawn and turf, and community gardens. Reliable resources are available from the US EPA and University of California—Davis (Table 3). Other local policy approaches in use are posting warning signs of pesticide use, restricting spray zone buffers at schools, or restricting specific types of pesticide products in schools. Pediatricians can

play a role in promotion of development of model programs and practices in the communities and schools of their patients.

RECOMMENDATIONS

Three overarching principles can be identified: (1) pesticide exposures are common and cause both acute and chronic effects; (2) pediatricians need to be knowledgeable in pesticide identification, counseling, and management; and (3) governmental actions to improve pesticide safety are needed. Whenever new public policy is developed or existing policy is revised, the wide range of consequences of pesticide use on children and their families should be considered. The American Academy of Pediatrics, through its chapters, committees, councils, sections, and staff, can provide information and support for public policy advocacy efforts. See http://www.aap.org/advocacy.html for additional information or contact chapter leadership.

Recommendations to Pediatricians

1. Acute exposures: become familiar with the clinical signs and symptoms of acute intoxication from the major types of pesticides. Be able to translate clinical knowledge about pesticide hazards into an appropriate exposure history for pesticide poisoning.
2. Chronic exposures: become familiar with the subclinical effects of chronic exposures and routes of exposures from the major types of pesticides.
3. Resource identification: know locally available resources for acute toxicity management and chronic low-dose exposure (see Table 3).
4. Pesticide labeling knowledge: Understand the usefulness and limitations of pesticide chemical information on pesticide product labels.
5. Counseling: Ask parents about pesticide use in or around the home to help determine the need for providing targeted anticipatory guidance. Recommend use of minimal-risk products, safe storage practices, and application of IPM (least toxic methods), whenever possible.
6. Advocacy: work with schools and governmental agencies to advocate for application of least toxic pesticides by using IPM principles. Promote community right-to-know procedures when pesticide spraying occurs in public areas.

Recommendations to Government

1. Marketing: ensure that pesticide products as marketed are not attractive to children.
2. Labeling: include chemical ingredient identity on the label and/or the manufacturer's Web site for all product constituents, including inert ingredients, carriers, and solvents. Include a label section specific to "Risks to children," which informs users whether there is evidence that the active or inert ingredients have any known chronic or developmental health concerns for children. Enforce labeling practices that ensure users have adequate information on product contents, acute and chronic toxicity potential, and emergency information. Consider printing or making available labels in Spanish in addition to English.
3. Exposure reduction: set goal to reduce exposure overall. Promote application methods and practices that minimize children's exposure, such as using bait stations and gels, advising against overuse of pediculicides. Promote education regarding proper storage of product.
4. Reporting: make pesticide-related suspected poisoning universally reportable and support a systematic central repository of such incidents to optimize national surveillance.
5. Exportation: aid in identification of least toxic alternatives to pesticide use internationally, and unless safer alternatives are not available or are impossible to implement, ban export of products that are banned or restricted for toxicity concerns in the United States.
6. Safety: continue to evaluate pesticide safety. Enforce community right-to-know procedures when pesticide spraying occurs in public areas. Develop, strengthen, and enforce standards of removal of concerning products for home or child product use. Require development of a human biomarker, such as a urinary or blood measure, that can be used to identify exposure and/or early health implications with new pesticide chemical registration or reregistration of existing products. Developmental toxicity, including endocrine disruption, should be a priority when evaluating new chemicals for licensing or reregistration of existing products.
7. Advance less toxic pesticide alternatives: increase economic incentives for growers who adopt IPM, including less toxic pesticides. Support research to expand and improve IPM in agriculture and nonagricultural pest control.
8. Research: support toxicologic and epidemiologic research to better identify and understand health risks associated with children's exposure to pesticides. Consider supporting another national study of pesticide use in the home and garden setting of US households as a targeted initiative or through cooperation with existing research opportunities (eg, National Children's Study, NHANES).
9. Health provider education and support: support educational efforts to increase the capacity of pediatric health care providers to diagnose and manage acute pesticide

poisoning and reduce pesticide exposure and potential chronic pesticide effects in children. Provide support to systems such as Poison Control Centers to provide timely, expert advice on exposures. Require the development of diagnostic tests to assist providers with diagnosing (and ruling out) pesticide poisoning.

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ERRATA

Spooner. We Are Still Waiting for Fully Supportive Electronic Health Records in Pediatrics. *Pediatrics.* 2012;130(6):e1674–e1676.

An error occurred in this article by Spooner, titled "We Are Still Waiting for Fully Supportive Electronic Health Records in Pediatrics" published in the December 2012 issue of *Pediatrics* (2012;130[6]:e1674–e1676; originally published online November 19, 2012; doi:10.1542/peds.2012-2724). On page e1674, on line 33, this reads: "The alarming result from the survey was that only 3% of AAP Fellows reported that they had a system that provided all of the items listed by Leu and colleagues." This should have read: "The alarming result from the survey was that only 9.6% of AAP Fellows reported that they had or planned to adopt within 12 months a system that provided all of the five "pediatric-supportive" items listed by Leu and colleagues."

doi:10.1542/peds.2013-0134

Auger et al. Medical Home Quality and Readmission Risk for Children Hospitalized With Asthma Exacerbations. *Pediatrics.* 2013;131(1):64–70

An error occurred in this article by Auger et al, titled "Medical Home Quality and Readmission Risk for Children Hospitalized With Asthma Exacerbations" published in the January 2013 issue of *Pediatrics* (2013;131[1]:64–70; doi:10.1542/2012-1055). On page 69, in Table 2 under the heading Adjusted HR, on the line Medicaid, this reads: "0.28 (0.51–1.34)." This should have read: "0.82 (0.51–1.34)."

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Council on Environmental Health. Policy Statement: Pesticide Exposure in Children. *Pediatrics.* 2012;130(6):e1757–e1763

A couple of errors occurred in this AAP Policy Statement titled "Pesticide Exposure in Children" published in the December 2012 issue of *Pediatrics* (2012;130[6]:e1757–e1763; originally published online November 26, 2012; doi:10.1542/peds.2012-2757). In Table 2, in the second and third columns where glyphosate is discussed, the words "organic solvent" should be replaced with the word "surfactant." On page e1758, in the first paragraph of the left-hand column, immediately beneath Table 1, the first full sentence should be amended to read: "For many children, diet may be the most influential source, as illustrated by an intervention study that placed children on an organic diet (produced without most conventional pesticides) and observed drastic and immediate decrease in urinary excretion of organophosphate pesticide metabolites."

doi:10.1542/peds.2013-0576

Robert JR, Karr CJ; Council on Environmental Health. Technical Report: Pesticide Exposure in Children. *Pediatrics.* 2012;130(6):e1765–e1788

Several inaccuracies occurred in this AAP Technical Report titled "Pesticide Exposure in Children" published in the December 2012 issue of *Pediatrics* (2012;130[6]:e1765–e1788; originally published online November 26, 2012; doi:10.1542/peds.2012-2758). On page e1773 and in Tables 1 and 2 where the phosphonate herbicide glyphosate is discussed, changes should be noted. In the first paragraph of the first column on page e1773 about acute glyphosate poisoning, the word "intentional" should be substituted for the word "unintentional." In this same paragraph as well as in Tables 1 and 2, the word "surfactant" should replace the words "hydrocarbon solvent" and "organic solvent, respectively." The

Pesticide Exposure in Children

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Neurobehavioural effects of developmental toxicity

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Neurodevelopmental disabilities, including autism, attention-deficit hyperactivity disorder, dyslexia, and other cognitive impairments, affect millions of children worldwide, and some diagnoses seem to be increasing in frequency. Industrial chemicals that injure the developing brain are among the known causes for this rise in prevalence. In 2006, we did a systematic review and identified five industrial chemicals as developmental neurotoxicants: lead, methylmercury, polychlorinated biphenyls, arsenic, and toluene. Since 2006, epidemiological studies have documented six additional developmental neurotoxicants—manganese, fluoride, chlorpyrifos, dichlorodiphenyltrichloroethane, tetrachloroethylene, and the polybrominated diphenyl ethers. We postulate that even more neurotoxicants remain undiscovered. To control the pandemic of developmental neurotoxicity, we propose a global prevention strategy. Untested chemicals should not be presumed to be safe to brain development, and chemicals in existing use and all new chemicals must therefore be tested for developmental neurotoxicity. To coordinate these efforts and to accelerate translation of science into prevention, we propose the urgent formation of a new international clearinghouse.

Introduction

Disorders of neurobehavioural development affect 10–15% of all births,[1] and prevalence rates of autism spectrum disorder and attention-deficit hyperactivity disorder seem to be increasing worldwide.[2] Subclinical decrements in brain function are even more common than these neurobehavioural developmental disorders. All these disabilities can have severe consequences[3]—they diminish quality of life, reduce academic achievement, and disturb behaviour, with profound consequences for the welfare and productivity of entire societies.[4]

The root causes of the present global pandemic of neurodevelopmental disorders are only partly understood. Although genetic factors have a role,[5] they cannot explain recent increases in reported prevalence, and none of the genes discovered so far seem to be responsible for more than a small proportion of cases.[5] Overall, genetic factors seem to account for no more than perhaps 30–40% of all cases of neurodevelopmental disorders. Thus, non-genetic, environmental exposures are involved in causation, in some cases probably by interacting with genetically inherited predispositions.

Strong evidence exists that industrial chemicals widely disseminated in the environment are important contributors to what we have called the global, silent pandemic of neurodevelopmental toxicity.[6,7] The developing human brain is uniquely vulnerable to toxic chemical exposures, and major windows of developmental vulnerability occur in utero and during infancy and early childhood.[8] During these sensitive life stages, chemicals can cause permanent brain injury at low levels of exposure that would have little or no adverse effect in an adult.

In 2006, we did a systematic review of the published clinical and epidemiological studies into the neurotoxicity of industrial chemicals, with a focus on developmental neurotoxicity.[6] We identified five industrial chemicals that could be reliably classified as developmental neurotoxicants: lead, methylmercury, arsenic, polychlorinated biphenyls, and toluene. We also noted 201 chemicals that had been reported to cause injury to the nervous system in adults, mostly in connection with occupational exposures, poisoning incidents, or suicide attempts. Additionally, more than 1000 chemicals have been reported to be neurotoxic in animals in laboratory studies.

We noted that recognition of the risks of industrial chemicals to brain development has historically needed decades of research and scrutiny, as shown in the cases of lead and methylmercury.[9,10] In most cases, discovery began with clinical diagnosis of poisoning in workers and episodes of high-dose exposure. More sophisticated epidemiological studies typically began only much later. Results from such studies documented developmental neurotoxicity at much lower exposure levels than had previously been thought to be safe. Thus, recognition of widespread subclinical toxicity often did not occur until decades after the initial evidence of neurotoxicity. A recurring theme was that early warnings of subclinical neurotoxicity were often ignored or even dismissed.[11] David P Rall, former Director of the US National Institute of Environmental Health Sciences, once noted that "if thalidomide had caused a ten-point loss of intelligence quotient (IQ) instead of obvious birth defects of the limbs, it would probably still be on the market".[12] Many industrial chemicals marketed at present probably cause IQ deficits of far fewer than ten points and have therefore eluded detection so far, but their combined effects could have enormous consequences.

In our 2006 review,[6] we expressed concern that additional developmental neurotoxicants might lurk undiscovered among the 201 chemicals then known to be neurotoxic to adult human beings and among the many thousands of pesticides, solvents, and other industrial chemicals in widespread use that had never been tested for neurodevelopmental toxicity. Since our previous review, new data have emerged about the vulnerability of the developing brain and the neurotoxicity of industrial chemicals. Particularly important new evidence derives from prospective epidemiological birth cohort studies.

In this Review, we consider recent information about the developmental neurotoxicity of industrial chemicals

to update our previous report.[6] Additionally, we propose strategies to counter this pandemic and to prevent the spread of neurological disease and disability in children worldwide.

Unique vulnerability of the developing brain

The fetus is not well protected against industrial chemicals. The placenta does not block the passage of many environmental toxicants from the maternal to the fetal circulation,[13] and more than 200 foreign chemicals have been detected in umbilical cord blood.[14] Additionally, many environmental chemicals are transferred to the infant through human breastmilk.[13] During fetal life and early infancy, the blood–brain barrier provides only partial protection against the entry of chemicals into the CNS.[15]

Moreover, the developing human brain is exceptionally sensitive to injury caused by toxic chemicals,[6] and several developmental processes have been shown to be highly vulnerable to chemical toxicity. For example, in-vitro studies suggest that neural stem cells are very sensitive to neurotoxic substances such as methylmercury.[16] Some pesticides inhibit cholinesterase function in the developing brain,[17] thereby affecting the crucial regulatory role of acetylcholine before synapse formation.[18] Early-life epigenetic changes are also known to affect subsequent gene expression in the brain.[19] In summary, industrial chemicals known or suspected to be neurotoxic to adults are also likely to present risks to the developing brain.

Figure 1 shows the unique vulnerability of the brain during early life and indicates how developmental exposures to toxic chemicals are particularly likely to lead to functional deficits and disease later in life.

New findings about known hazards

Recent research on well-documented neurotoxicants has generated important new insights into the neurodevelopmental consequences of early exposures to these industrial chemicals.

Joint analyses that gathered data for lead-associated IQ deficits from seven international studies[20,21] support the conclusion that no safe level of exposure to lead exists.[22] Cognitive deficits in adults who had previously shown lead-associated developmental delays at school age suggest that the effects of lead neurotoxicity are probably permanent.[23] Brain imaging of young adults who had raised lead concentrations in their blood during childhood showed exposure-related decreases in brain volume.[24] Lead exposure in early childhood is associated with reduced school performance[25] and with delinquent behaviour later in life.[26,27]

Developmental neurotoxicity due to methylmercury occurs at much lower exposures than the concentrations that affect adult brain function.[28] Deficits at 7 years of age that were linked to low-level prenatal exposures to methylmercury were still detectable at the age of 14 years.[29] Some common genetic polymorphisms seem to increase the vulnerability of the developing brain to methylmercury toxicity.[30] Functional MRI scans of people exposed prenatally to excess amounts of methylmercury showed abnormally expanded activation of brain regions in response to sensory stimulation and motor tasks (figure 2).[31] Because some adverse effects might be counterbalanced by essential fatty acids from seafood, statistical adjustment for maternal diet during pregnancy results in stronger methylmercury effects.[32,33]

Prenatal and early postnatal exposures to inorganic arsenic from drinking water are associated with cognitive deficits that are apparent at school age.[34,35] Infants who survived the Morinaga milk arsenic poisoning incident had highly raised risks of neurological disease during adult life.[36]

The developmental neurotoxicity of polychlorinated biphenyls has been consolidated and strengthened by recent findings.[37] Although little new information has been published about the developmental neurotoxicity of toluene, much has been learned about the developmental neurotoxicity of another common solvent, ethanol, through research on fetal alcohol exposure. Maternal consumption of alcohol during pregnancy, even in very small quantities, has been linked to a range of neurobehavioural adverse effects in offspring, including reduced IQ, impaired executive function and social judgment, delinquent behaviour, seizures, other neurological signs, and sensory problems.[38]

Newly recognised developmental neurotoxicants

Prospective epidemiological birth cohort studies make it possible to measure maternal or fetal exposures in real time during pregnancy as these exposures actually occur, thus generating unbiased information about the degree and timing of prenatal exposures. Children in these prospective studies are followed longitudinally and assessed with age-appropriate tests to show delayed or deranged neurobehavioural development. These powerful epidemiological methods have enabled the discovery of additional developmental neurotoxicants.

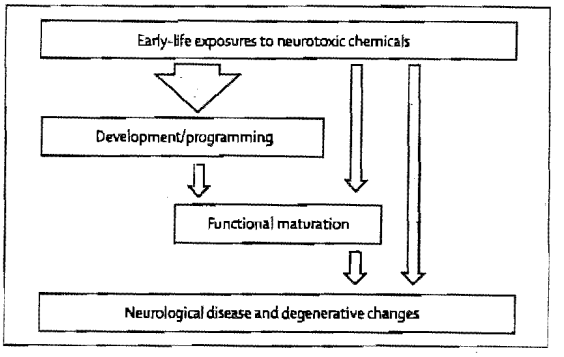

Figure 1: Effect of neurotoxicants during early brain development
Exposures in early life to neurotoxic chemicals can cause a wide range of adverse effects on brain development and maturation that can manifest as functional impairments or disease at any point in the human lifespan, from early infancy to very old age.

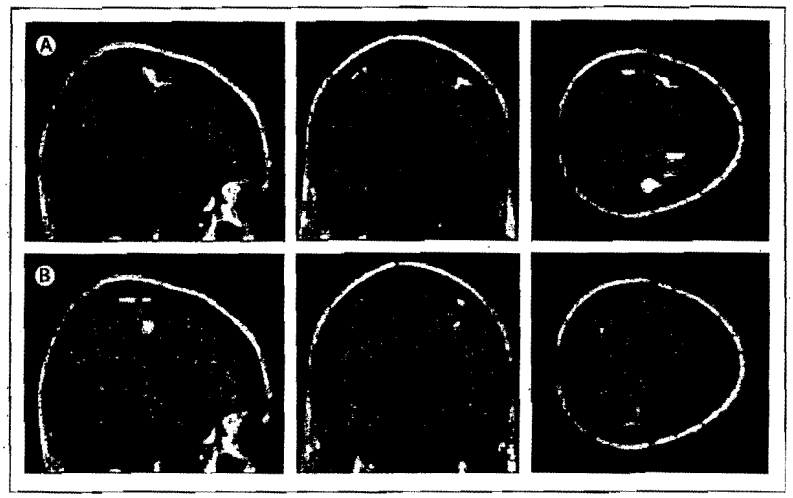

Figure 2: **Functional MRI scans show abnormal activation in the brain**
Average activation during finger tapping with the left hand in three adolescents with increased prenatal methylmercury exposure (A) and three control adolescents (B). The control participants activate the premotor and motor cortices on the right, whereas participants exposed to methylmercury activate these areas bilaterally.[37]

Cross-sectional data from Bangladesh show that exposure to manganese from drinking water is associated with reduced mathematics achievement scores in school children.[39] A study in Quebec, Canada, showed a strong correlation between manganese concentrations in hair and hyperactivity.[40] School-aged children living near manganese mining and processing facilities have shown associations between airborne manganese concentrations and diminished intellectual function[41] and with impaired motor skills and reduced olfactory function.[42] These results are supported by experimental findings in mice.[43]

A meta-analysis of 27 cross-sectional studies of children exposed to fluoride in drinking water, mainly from China, suggests an average IQ decrement of about seven points in children exposed to raised fluoride concentrations.[44] Confounding from other substances seemed unlikely in most of these studies. Further characterisation of the dose–response association would be desirable.

The occupational health literature[45] suggests that solvents can act as neurotoxicants, but the identification of individual responsible compounds is hampered by the complexity of exposures. In a French cohort study of 3000 children, investigators linked maternal occupational solvent exposure during pregnancy to deficits in behavioural assessment at 2 years of age.[46] The data showed dose-related increased risks for hyperactivity and aggressive behaviour. One in every five mothers in this cohort reported solvent exposures in common jobs, such as nurse or other hospital employee, chemist, cleaner, hairdresser, and beautician. In Massachusetts, USA, follow-up of a well-defined population with prenatal and early childhood exposure to the solvent tetrachloroethylene (also called perchlorethylene) in drinking water showed a tendency towards deficient neurological function and increased risk of psychiatric diagnoses.[47]

Acute pesticide poisoning occurs frequently in children worldwide, and subclinical pesticide toxicity is also widespread. Clinical data suggest that acute pesticide poisoning during childhood might lead to lasting neurobehavioural deficits.[48,49] Highly toxic and bio-accumulative pesticides are now banned in high-income nations, but are still used in many low-income and middle-income countries. In particular, the organochlorine compounds dichlorodiphenyltrichloroethane (DDT), its metabolite dichlorodiphenyldichloroethylene (DDE), and chlordecone (Kepone), tend to be highly persistent and remain widespread in the environment and in people's bodies in high-use regions. Recent studies have shown inverse correlations between serum concentrations of DDT or DDE (which indicate accumulated exposures), and neurodevelopmental performance.[50,51]

Organophosphate pesticides are eliminated from the human body much more rapidly than are organochlorines, and exposure assessment is therefore inherently less precise. Nonetheless, three prospective epidemiological birth cohort studies provide new evidence that prenatal exposure to organophosphate pesticides can cause developmental neurotoxicity. In these studies, prenatal organophosphate exposure was assessed by measurement of maternal urinary excretion of pesticide metabolites during pregnancy. Dose-related correlations were recorded between maternal exposures to chlorpyrifos or other organophosphates and small head circumference at birth—which is an indication of slowed brain growth in utero—and with neurobehavioural deficits that have persisted to at least 7 years of age.[52–54] In a subgroup study, MRI of the brain showed that prenatal chlorpyrifos exposure was associated with structural abnormalities that included thinning of the cerebral cortex.[55]

Herbicides and fungicides might also have neurotoxic potential.[56] Propoxur,[57] a carbamate pesticide, and permethrine,[58] a member of the pyrethroid class of pesticides, have recently been linked to neurodevelopmental deficits in children.

The group of compounds known as polybrominated diphenyl ethers (PBDEs) are widely used as flame retardants and are structurally very similar to the polychlorinated biphenyls. Experimental evidence now suggests that the PBDEs might also be neurotoxic.[59] Epidemiological studies in Europe and the USA have shown neurodevelopmental deficits in children with increased prenatal exposures to these compounds.[60–62] Thus, the PBDEs should be regarded as hazards to human neurobehavioural development, although attribution of relative toxic potentials to individual PBDE congeners is not yet possible.

Other suspected developmental neurotoxicants

A serious difficulty that complicates many epidemiological studies of neurodevelopmental toxicity in children is the problem of mixed exposures. Most populations are exposed to more than one neurotoxicant at a time, and yet

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most studies have only a finite amount of power and precision in exposure assessment to discern the possible effects of even single neurotoxicants. A further problem in many epidemiological studies of non-persistent toxicants is that imprecise assessment of exposure tends to obscure associations that might actually be present.[63] Guidance from experimental neurotoxicity studies is therefore crucial. In the assessment of potential developmental neurotoxicants, we have used a strength of evidence approach similar to that used by the International Agency for Research on Cancer for assessing epidemiological and experimental studies.

Phthalates and bisphenol A are added to many different types of plastics, cosmetics, and other consumer products. Since they are eliminated rapidly in urine, exposure assessment is complicated, and such imprecision might lead to underestimation of the true risk of neurotoxicity. The best-documented effects of early-life exposure to phthalates are the consequence of disruption of endocrine signalling.[64] Thus, prenatal exposures to phthalates have been linked to both neurodevelopmental deficits and to behavioural abnormalities characterised by shortened attention span and impaired social interactions.[65] The neurobehavioural toxicity of these compounds seems to affect mainly boys and could therefore relate to endocrine disruption in the developing brain.[66] In regard to bisphenol A, a prospective study showed that point estimates of exposure during gestation were linked to abnormalities in behaviour and executive function in children at 3 years of age.[67]

Exposure to air pollution can cause neurodevelopmental delays and disorders of behavioural functions.[68,69] Of the individual components of air pollution, carbon monoxide is a well-documented neurotoxicant, and indoor exposure to this substance has now been linked to deficient neurobehavioural performance in children.[70] Less clear is the reported contribution of nitrogen oxides to neurodevelopmental deficits,[71] since these compounds often co-occur with carbon monoxide as part of complex emissions. Tobacco smoke is a complex mixture of hundreds of chemical compounds and is now a well-documented cause of developmental neurotoxicity.[72] Infants exposed prenatally to polycyclic aromatic hydrocarbons from traffic exhausts at 5 years of age showed greater cognitive impairment and lower IQ than those exposed to lower levels of these compounds.[68]

Perfluorinated compounds, such as perfluorooctanoic acid and perfluorooctane sulphonate, are highly persistent in the environment and in the human body, and seem to be neurotoxic.[73] Emerging epidemiological evidence suggests that these compounds might indeed impede neurobehavioural development.[74]

Developmental neurotoxicity and clinical neurology

Exposures in early life to developmental neurotoxicants are now being linked to specific clinical syndromes in children. For example, an increased risk of attention-deficit hyperactivity disorder has been linked to prenatal exposures to manganese, organophosphates,[75] and phthalates.[76] Phthalates have also been linked to behaviours that resemble components of autism spectrum disorder.[77] Prenatal exposure to automotive air pollution in California, USA, has been linked to an increased risk for autism spectrum disorder.[78]

The persistent decrements in intelligence documented in children, adolescents, and young adults exposed in early life to neurotoxicants could presage the development of neurodegenerative disease later in life. Thus, accumulated exposure to lead is associated with cognitive decline in the elderly.[79] Manganese exposure may lead to parkinsonism, and experimental studies have reported Parkinson's disease as a result of developmental exposures to the insecticide rotenone, the herbicides paraquat and maneb, and the solvent trichloroethylene.[80] Any environmental exposure that increases the risk of neurodegenerative disorders in later life (figure 1) requires urgent investigation as the world's population continues to age.[81]

The expanding complement of neurotoxicants

In our 2006 review,[6] we expressed concern that additional developmental neurotoxicants might lie undiscovered in the 201 chemicals that were then known to be neurotoxic to human adults, in the roughly 1000 chemicals known to be neurotoxic in animal species, and in the many thousands of industrial chemicals and pesticides that have never been tested for neurotoxicity. Exposure to neurotoxic chemicals is not rare, since almost half of the 201 known human neurotoxicants are regarded as high production volume chemicals.

Our updated literature review shows that since 2006 the list of recognised human neurotoxicants has expanded by 12 chemicals, from 202 (including ethanol) to 214 (table 1 and appendix)—that is, by about two substances per year. Many of these chemicals are widely used and disseminated extensively in the global environment. Of the newly identified neurodevelopmental toxicants, pesticides constitute the largest group, as was already the case in

See Online for appendix

	Number known in 2006	Number known in 2013	Identified since 2006
Metals and inorganic compounds	25	26	Hydrogen phosphide[82]
Organic solvents	39*	40	Ethyl chloride[83]
Pesticides	92	101	Acetamiprid,[84] amitraz,[85] avermectin,[86] emamectin,[87] fipronil (Termidor),[88] glyphosate,[89] hexaconazole,[90] imidacloprid,[91] tetramethylenedisulfotetramine[92]
Other organic compounds	46	47	1,3-butadiene[93]
Total	202*	214	12 new substances

*Including ethanol.

Table 1: Industrial chemicals known to be toxic to the human nervous system in 2006 and 2013, according to chemical group

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	Known in 2006	Newly identified
Metals and inorganic compounds	Arsenic and arsenic compounds, lead, and methylmercury	Fluoride and manganese
Organic solvents	(Ethanol) toluene	Tetrachloroethylene
Pesticides	None	Chlorpyrifos and DDT/DDE
Other organic compounds	Polychlorinated biphenyls	Brominated diphenyl ethers
Total	6*	6

DDT=dichlorodiphenyltrichloroethane. DDE=dichlorodiphenyldichloroethylene. *Including ethanol.

Table 2: **Industrial chemicals known to cause developmental neurotoxicity in human beings in 2006 and 2013, according to chemical group**

	Number of IQ points lost
Major medical and neurodevelopmental disorders	
Preterm birth	34 031 025
Autism spectrum disorders	7 109 899
Paediatric bipolar disorder	8 164 080
Attention-deficit hyperactivity disorder	16 799 400
Postnatal traumatic brain injury	5 827 300
Environmental chemical exposures	
Lead	22 947 450
Methylmercury	1 590 000*
Organophosphate pesticides	16 899 488
Other neurotoxicants	Unknown

IQ=intelligence quotient. Data from from Bellinger.[94] *From Grandjean and colleagues.[95]

Table 3: **Total losses of IQ points in US children 0–5 years of age associated with major risk factors, including developmental exposure to industrial chemicals that cause neurotoxicity**

2006. In the same 7-year period, the number of known developmental neurotoxicants has doubled from six to 12 (table 2). Although the pace of scientific discovery of new neurodevelopmental hazards is more rapid today than in the past, it is still slower than the identification of adult neurotoxicants.

The gap that exists between the number of substances known to be toxic to the adult brain and the smaller number known to be toxic to the much more vulnerable developing brain is unlikely to close in the near future. This discrepancy is attributable to the fact that toxicity to the adult brain is usually discovered as a result of acute poisoning incidents, typically with a clear and immediate association between causative exposure and adverse effects, as occurs for workplace exposures or suicide attempts. By contrast, the recognition of developmental neurotoxicity relies on two sets of evidence collected at two different points in time: exposure data (often obtained from the mother during pregnancy), and data for the child's postnatal neurobehavioural development (often obtained 5–10 years later). Because brain functions develop sequentially, the full effects of early neurotoxic damage might not become apparent until school age or beyond. The most reliable evidence of developmental neurotoxicity is obtained through prospective studies that include real-time recording of information about exposure in early life followed by serial clinical assessments of the child. Such research is inherently slow and is hampered by the difficulty of reliable assessment of exposures to individual toxicants in complex mixtures.

Consequences of developmental neurotoxicity

Developmental neurotoxicity causes brain damage that is too often untreatable and frequently permanent. The consequence of such brain damage is impaired CNS function that lasts a lifetime and might result in reduced intelligence, as expressed in terms of lost IQ points, or disruption in behaviour. A recent study compared the estimated total IQ losses from major paediatric causes and showed that the magnitude of losses attributable to lead, pesticides, and other neurotoxicants was in the same range as, or even greater than, the losses associated with medical events such as preterm birth, traumatic brain injury, brain tumours, and congenital heart disease (table 3).[94]

Loss of cognitive skills reduces children's academic and economic attainments and has substantial long-term economic effects on societies.[4] Thus, each loss of one IQ point has been estimated to decrease average lifetime earnings capacity by about €12 000 or US$18 000 in 2008 currencies.[96] The most recent estimates from the USA indicate that the annual costs of childhood lead poisoning are about US$50 billion and that the annual costs of methylmercury toxicity are roughly US$5 billion.[97] In the European Union, methylmercury exposure is estimated to cause a loss of about 600 000 IQ points every year, corresponding to an annual economic loss of close to €10 billion. In France alone, lead exposure is associated with IQ losses that correspond to annual costs that might exceed €20 billion.[98] Since IQ losses represent only one aspect of developmental neurotoxicity, the total costs are surely even higher.

Evidence from worldwide sources indicates that average national IQ scores are associated with gross domestic product (GDP)—a correlation that might be causal in both directions.[99] Thus, poverty can cause low IQ, but the opposite is also true. In view of the widespread exposures to lead, pesticides, and other neurotoxicants in developing countries, where chemical controls might be ineffective compared with those in more developed countries,[100–101] developmental exposures to industrial chemicals could contribute substantially to the recorded correlation between IQ and GDP. If this theory is true, developing countries could take decades to emerge from poverty. Consequently, pollution abatement might then be delayed, and a vicious circle can result.

The antisocial behaviour, criminal behaviour, violence, and substance abuse that seem to result from early-life exposures to some neurotoxic chemicals result in increased needs for special educational services, institutionalisation, and even incarceration. In the USA, the murder rate fell sharply 20 years after the removal of lead from petrol,[102] a finding consistent with the idea that

exposure to lead in early life is a powerful determinant of behaviour decades later. Although poorly quantified, such behavioural and social consequences of neurodevelopmental toxicity are potentially very costly.[76]

Prevention of developmental neurotoxicity caused by industrial chemicals is highly cost effective. A study that quantified the gains resulting from the phase-out of lead additives from petrol reported that in the USA alone, the introduction of lead-free petrol has generated an economic benefit of $200 billion in each annual birth cohort since 1980,[103] an aggregate benefit in the past 30 years of over $3 trillion. This success has since been repeated in more than 150 countries, resulting in vast additional savings. Every US$1 spent to reduce lead hazards is estimated to produce a benefit of US$17–220, which represents a cost-benefit ratio that is even better than that for vaccines.[4] Furthermore, the costs associated with the late-life consequences of developmental neurotoxicity are enormous, and the benefits from prevention of degenerative brain disorders could be very substantial.

New methods to identify developmental neurotoxicants

New toxicological methods now allow a rational strategy for the identification of developmental neurotoxicants based on a multidisciplinary approach.[104] A new guideline has been approved as a standardised approach for the identification of developmental neurotoxicants.[105] However, completion of such tests is expensive and requires the use of many laboratory animals, and reliance on mammals for chemicals testing purposes needs to be reduced.[106] US governmental agencies have established the National Center for Computational Toxicology and an initiative—the Tox 21 Program—to promote the evolution of toxicology from a mainly observational science to a predominantly predictive science.[107]

In-vitro methods have now reached a level of predictive validity that means they can be applied to neurotoxicity testing.[108] Some of these tests are based on neural stem cells. Although these cell systems do not have a blood–brain barrier and particular metabolising enzymes, these approaches are highly promising. As a further option, data for protein links and protein–protein interactions can now be used to explore potential neurotoxicity in silico,[109] thus showing that existing computational methods might predict potential toxic effects.[110]

In summary, use of the whole range of approaches along with clinical and epidemiological evidence, when available, should enable the integration of information for use in at least a tentative risk assessment. With these methods, we anticipate that the pace of scientific discovery in developmental neurotoxicology will accelerate further in the years ahead.

Conclusions and recommendations

The updated findings presented in this Review confirm and extend our 2006 conclusions.[6] During the 7 years since our previous report, the number of industrial chemicals recognised to be developmental neurotoxicants has doubled. Exposures to these industrial chemicals in the environment contribute to the pandemic of developmental neurotoxicity.

Two major obstacles impede efforts to control the global pandemic of developmental neurotoxicity. These barriers, which we noted in our previous review[6] and were recently underlined by the US National Research Council,[111] are: large gaps in the testing of chemicals for developmental neurotoxicity, which results in a paucity of systematic data to guide prevention; and the huge amount of proof needed for regulation. Thus, very few chemicals have been regulated as a result of developmental neurotoxicity.

The presumption that new chemicals and technologies are safe until proven otherwise is a fundamental problem.[112] Classic examples of new chemicals that were introduced because they conveyed certain benefits, but were later shown to cause great harm, include several neurotoxicants, asbestos, thalidomide, diethylstilboestrol, and the chlorofluorocarbons.[112] A recurring theme in each of these cases was that commercial introduction and wide dissemination of the chemicals preceded any systematic effort to assess potential toxicity. Particularly absent were advance efforts to study possible effects on children's health or the potential of exposures in early life to disrupt early development. Similar challenges have been confronted in other public health disasters, such as those caused by tobacco smoking, alcohol use, and refined foods. These problems have been recently termed industrial epidemics.[113]

To control the pandemic of developmental neurotoxicity, we propose a coordinated international strategy (panel). Mandatory and transparent assessment of evidence for neurotoxicity is the foundation of this strategy. Assessment of toxicity must be followed by governmental regulation and market intervention. Voluntary controls seem to be of little value.[11]

Panel: **Recommendations for an international clearinghouse on neurotoxicity**

The main purpose of this agency would be to promote optimum brain health, not just avoidance of neurological disease, by inspiring, facilitating, and coordinating research and public policies that aim to protect brain development during the most sensitive life stages. The main efforts would aim to:

- Screen industrial chemicals present in human exposures for neurotoxic effects so that hazardous substances can be identified for tighter control
- Stimulate and coordinate new research to understand how toxic chemicals interfere with brain development and how best to prevent long-term dysfunctions and deficits
- Function as a clearinghouse for research data and strategies by gathering and assessing documentation about brain toxicity and stimulating international collaboration on research and prevention
- Promote policy development aimed at protecting vulnerable populations against chemicals that are toxic to the brain without needing unrealistic amounts of scientific proof

The three pillars of our proposed strategy are: legally mandated testing of existing industrial chemicals and pesticides already in commerce, with prioritisation of those with the most widespread use, and incorporation of new assessment technologies; legally mandated premarket evaluation of new chemicals before they enter markets, with use of precautionary approaches for chemical testing that recognise the unique vulnerability of the developing brain; and the formation of a new clearinghouse for neurotoxicity as a parallel to the International Agency for Research on Cancer. This new agency will assess industrial chemicals for developmental neurotoxicity with a precautionary approach that emphasises prevention and does not require absolute proof of toxicity. It will facilitate and coordinate epidemiological and toxicological studies and will lead the urgently needed global programmes for prevention.

These new approaches must reverse the dangerous presumption that new chemicals and technologies are safe until proven otherwise. They must also overcome the existing requirement to produce absolute proof of toxicity before action can be started to protect children against neurotoxic substances. Precautionary interpretation of data about developmental neurotoxicity should take into account the very large individual and societal costs that result from failure to act on available documentation to prevent disease in children.[114] Academic research has often favoured scepticism and required extensive replication before acceptance of a hypothesis,[114] thereby adding to the inertia in toxicology and environmental health research and the consequent disregard of many other potential neurotoxicants.[115] Additionally, the strength of evidence that is needed to constitute "proof" should be analysed in a societal perspective, so that the implications of ignoring a developmental neurotoxicant and of failing to act on the basis of available data are also taken into account.

Finally, we emphasise that the total number of neurotoxic substances now recognised almost certainly represents an underestimate of the true number of developmental neurotoxicants that have been released into the global environment. Our very great concern is that children worldwide are being exposed to unrecognised toxic chemicals that are silently eroding intelligence, disrupting behaviours, truncating future achievements, and damaging societies, perhaps most seriously in developing countries. A new framework of action is needed.

Search strategy and selection criteria

We identified studies published since 2006 on the neurotoxic effects of industrial chemicals in human beings by using the search terms "neurotoxicity syndromes"[MeSH], "neurotoxic", "neurologic", or "neuro*", combined with "exposure" and "poisoning" in PubMed, from 2006 to the end of 2012. For developmental neurotoxicity, the search terms were "prenatal exposure delayed effects"[MeSH], "maternal exposure" or "maternal fetal exchange", "developmental disabilities/chemically induced" and "neurotoxins", all of which were searched for with the limiters "All Child: 0–18 years, Human". We also used references cited in the publications retrieved.

Contributors

Both authors did the literature review, wrote and revised the report, and approved the final version.

Conflicts of interest

PG has provided paid expert testimony about mercury toxicology for the US Department of Justice. PJL has provided paid expert testimony in cases of childhood lead poisoning. We declare that we have no other conflicts of interest.

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The American College of Obstetricians and Gynecologists
WOMEN'S HEALTH CARE PHYSICIANS

COMMITTEE OPINION

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The American College of Obstetricians and Gynecologists Committee on Health Care for Underserved Women

American Society for Reproductive Medicine Practice Committee

The University of California, San Francisco Program on Reproductive Health and the Environment

This Committee Opinion was developed by the American College of Obstetricians and Gynecologists Committee on Health Care for Underserved Women and the American Society for Reproductive Medicine Practice Committee with the assistance of the University of California, San Francisco (UCSF) Program on Reproductive Health and the Environment. The Program on Reproductive Health and the Environment endorses this document. This document reflects emerging clinical and scientific advances as of the date issued and is subject to change. This information should not be construed as dictating an exclusive course of treatment or procedure to be followed.

Exposure to Toxic Environmental Agents

ABSTRACT: Reducing exposure to toxic environmental agents is a critical area of intervention for obstetricians, gynecologists, and other reproductive health care professionals. Patient exposure to toxic environmental chemicals and other stressors is ubiquitous, and preconception and prenatal exposure to toxic environmental agents can have a profound and lasting effect on reproductive health across the life course. Prenatal exposure to certain chemicals has been documented to increase the risk of cancer in childhood; adult male exposure to pesticides is linked to altered semen quality, sterility, and prostate cancer; and postnatal exposure to some pesticides can interfere with all developmental stages of reproductive function in adult females, including puberty, menstruation and ovulation, fertility and fecundity, and menopause. Many environmental factors harmful to reproductive health disproportionately affect vulnerable and underserved populations, which leaves some populations, including underserved women, more vulnerable to adverse reproductive health effects than other populations. The evidence that links exposure to toxic environmental agents and adverse reproductive and developmental health outcomes is sufficiently robust, and the American College of Obstetricians and Gynecologists and the American Society for Reproductive Medicine join leading scientists and other clinical practitioners in calling for timely action to identify and reduce exposure to toxic environmental agents while addressing the consequences of such exposure.

Reproductive Environmental Health

Robust scientific evidence has emerged over the past 15 years, demonstrating that preconception and prenatal exposure to toxic environmental agents can have a profound and lasting effect on reproductive health across the life course (1–3). Exposure to toxic environmental agents also is implicated in increases in adverse reproductive health outcomes that emerged since World War II; these changes have occurred at a rapid rate that cannot be explained by changes in genetics alone, which occur at a slower pace. For additional information, a detailed review is available at www.acog.org/goto/underserved.

Exposure to environmental chemicals and metals in air, water, soil, food, and consumer products is ubiquitous. An analysis of National Health and Nutrition Examination Survey data from 2003–2004 found that virtually every pregnant woman in the United States is exposed to at least 43 different chemicals (4). Chemicals in pregnant women can cross the placenta, and in some cases, such as with methyl mercury, can accumulate in the fetus, resulting in higher fetal exposure than maternal exposure (5–7). Prenatal exposure to environmental chemicals is linked to various adverse health consequences, and patient exposure at any point in time can lead to harmful reproductive health outcomes. For example, prenatal exposure to certain pesticides has been documented to increase the risk of cancer in childhood; adult male exposure to pesticides is linked to altered semen quality, sterility, and prostate cancer; and postnatal exposure to some pesticides can

interfere with all developmental stages of reproductive function in adult females, including puberty, menstruation and ovulation, fertility and fecundity, and menopause (8). A group of chemicals called endocrine disrupting chemicals has been shown to interfere with the role of certain hormones, homeostasis, and developmental processes (9). They represent a heterogeneous group of agents used in pesticides, plastics, industrial chemicals, and fuels. One study shows that the endocrine disrupting chemical bisphenol-A works in a fashion that is comparable to diethylstilbestrol at the cell and developmental level (10). Likewise, research has clearly shown that many industrial chemicals can affect thyroid function (9, 11). Because of deficiencies in the current regulatory structure, unlike pharmaceuticals, most environmental chemicals have entered the marketplace without comprehensive and standardized information regarding their reproductive or other long-term toxic effects (12).

Vulnerable Populations and Environmental Disparities

Although exposure to toxic environmental agents is ubiquitous among all patient populations, many environmental factors harmful to reproductive health also disproportionately affect vulnerable and underserved populations and are subsumed in issues of environmental justice. In the United States, minority populations are more likely to live in the counties with the highest levels of outdoor air pollution (13) and to be exposed to a variety of indoor pollutants, including lead, allergens, and pesticides than white populations (14). In turn, the effects of exposure to environmental chemicals can be exacerbated by injustice, poverty, neighborhood quality, housing quality, psychosocial stress, and nutritional status (14, 15).

Women with occupational exposure to toxic chemicals also are highly vulnerable to adverse reproductive health outcomes (16). For example, levels of organophosphate pesticides and phthalates measured in occupationally exposed populations are far greater than levels measured in the general population (17, 18). Furthermore, low-wage immigrant populations disproportionately work in occupations associated with a hazardous workplace environment (19, 20).

As underscored by a groundbreaking 2009 report by the National Academy of Sciences, the effects of low-dose exposure to an environmental contaminant may be quite different based on vulnerabilities, such as the underlying health status of the population and the presence of additional or "background" environmental exposure (21). Recognition of environmental disparities is essential for developing and implementing successful and efficient strategies for prevention.

Prevention

The evidence that links exposure to toxic environmental agents and adverse reproductive and developmental health outcomes is sufficiently robust, and the American College of Obstetricians and Gynecologists (the College) and the American Society for Reproductive Medicine (ASRM) join numerous other health professional organizations in calling for timely action to identify and reduce exposure to toxic environmental agents while addressing the consequences of such exposure (1, 22, 23). Reproductive care providers can be effective in preventing prenatal exposure to environmental threats to health because they are uniquely poised to intervene before and during pregnancy, which is a critical window of human development. An important outcome of pregnancy is no longer just a healthy newborn but a human biologically predisposed to be healthy from birth to old age (3, 24).

Providing Anticipatory Guidance

It is important for health care providers to become knowledgeable about toxic environmental agents that are endemic to their specific geographic areas. Intervention as early as possible during the preconception period is advised to alert patients regarding avoidance of toxic exposure and to ensure beneficial environmental exposure, eg, fresh fruit and vegetables, unprocessed food, outdoor activities, and a safe and nurturing physical and social environment. By the first prenatal care visit, exposure to toxic environmental agents and disruptions of organogenesis may have already occurred. Obtaining a patient history during a preconception visit and the first prenatal visit to identify specific types of exposure that may be harmful to a developing fetus is a key step and also should include queries of the maternal and paternal workplaces. A list of key chemical categories, sources of exposure, and clinical implications are provided in the online companion document to this Committee Opinion (www.acog.org/goto/underserved). Examples of an exposure history are available at http://prhe.ucsf.edu/prhe/clinical_resources.html. Once this exposure inventory has been completed, information should be given regarding the avoidance of exposure to toxic agents at home, in the community, and at work with possible referrals to occupational medicine programs or United States Pediatric Environmental Health Specialty Units if a serious exposure is found (25).

Reproductive care professionals do not need to be experts in environmental health science to provide useful information to patients and refer patients to appropriate specialists when a hazardous exposure is identified. Existing clinical experience and expertise in communicating risks of treatment are largely transferable to environmental health. Physician contact time with a patient does not need to be the primary point of intervention; information and resources about environmental hazards can be successfully incorporated into a childbirth class curriculum or provided in written materials to help parents make optimal choices for themselves and their children (26).

Reporting identified hazards is critical to prevention. For example, the reproductive toxicity of a common solvent used in many consumer products was first

described in a case report of a stillbirth (27). Physicians in the United States are required to report illnesses or injuries that may be work related, and reporting requirements vary by state. No authoritative national list of physician-reporting requirements by state exists. Resources for information about how to report occupational and environmental illnesses include local and state health agencies and the Association of Occupational and Environmental Clinics (http://www.aoec.org/about.htm). Illnesses include acute and chronic conditions, such as a skin disease (eg, contact dermatitis), respiratory disorder (eg, occupational asthma), or poisoning (eg, lead poisoning or pesticide intoxication) (28).

Patient-centered actions can reduce body burdens of toxic chemicals (ie, the total amount of chemicals present in the human body at any one time) (29–32). For example, research results document that when children's diets change from conventional to organic, the levels of pesticides in their bodies decrease (29, 30). Likewise, study results document that avoiding canned food and other dietary sources of bisphenol A can reduce measured levels of the chemical in children and adult family members (31), and that short-term changes in dietary behavior may significantly decrease exposure to phthalates (32).

Clinicians should encourage women in the preconception period and women who are pregnant or lactating to eat fruit, vegetables, beans, legumes, and whole grains every day, to avoid fast food and other processed foods whenever possible, and to limit foods high in animal fat, while providing information about how certain types of food affect health and how individuals can make changes. Also, patients should be advised that some large fish, such as shark, swordfish, king mackerel, and tilefish, are known to contain high levels of methylmercury, which is known to be teratogenic. As such, women in the preconception period and women who are pregnant or lactating should avoid these fish. To gain the benefits of consuming fish, while avoiding the risks of methylmercury consumption, pregnant women should be encouraged to enjoy a variety of other types of fish, including up to 12 ounces a week (two average meals) of a variety of fish and shellfish that are low in mercury. Five of the most commonly eaten seafood items that are low in mercury are shrimp, canned light tuna, salmon, pollock, and catfish. White (albacore) tuna has more mercury than canned light tuna and should be limited to no more than 6 ounces per week. Pregnant women and breastfeeding women should also check local advisories regarding the safety of fish caught in local lakes, rivers, and coastal areas. If no advice is available, they should consume no more than 6 ounces per week (one average meal) of fish caught in local waters and no other fish during that week (33).

Primary Prevention: The Role of Reproductive Care Professionals Beyond the Clinical Setting

Ultimately, evidence-based recommendations for preventing harmful environmental exposure must involve policy change (34). Action at the individual level can reduce exposure to some toxic chemicals (29, 31, 32) and informed consumer-purchasing patterns can send a signal to the marketplace to help drive societal change (35). However, individuals alone can do little about exposure to toxic environmental agents, such as from air and water pollution, and exposure perpetuated by poverty. The incorporation of the authoritative voice of health care professionals in policy arenas is critical to translating emerging scientific findings into prevention-oriented action on a large scale. Accordingly, many medical associations have taken steps in that direction (23).

For example, in 2009, the Endocrine Society called for improved public policy to identify and regulate endocrine disrupting chemicals and recommended that "until such time as conclusive scientific evidence exists to either prove or disprove harmful effects of substances, a precautionary approach should be taken in the formulation of EDC [endocrine disrupting chemical] policy" (36). Consistent with the clinical imperative to "do no harm," the precautionary principle states, "When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically" (37).

The College and the ASRM join these associations and call on their members to advocate for policies to identify and reduce exposure to environmental toxic agents while addressing the consequences of such exposure. Advancing policies and practices in support of a healthy food system should be pursued as a primary prevention strategy to ensure the health of pregnancies, children, and future generations. The College and ASRM urge the U.S. Environmental Protection Agency and other federal and state agencies to take all necessary actions when reviewing substances to guarantee health and safety. In addition, the College and ASRM fully support rigorous scientific investigation into the causes and prevention of birth defects, including linkages between environmental hazards and adverse reproductive and developmental health outcomes. Timely and effective steps must be taken to ensure the safety of all mothers and infants from toxic environmental agents. Because data are lacking on the safety of most chemicals, careful consideration of the risks posed must be given while the potential immediate and long-term health and genetic risks are evaluated. A chemical should never be released if a concern exists regarding its effect on health.

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Environmental Chemicals in Pregnant Women in the United States: NHANES 2003–2004

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BACKGROUND: Exposure to chemicals during fetal development can increase the risk of adverse health effects, and while biomonitoring studies suggest pregnant women are exposed to chemicals, little is known about the extent of multiple chemicals exposures among pregnant women in the United States.

OBJECTIVE: We analyzed biomonitoring data from the National Health and Nutritional Examination Survey (NHANES) to characterize both individual and multiple chemical exposures in U.S. pregnant women.

METHODS: We analyzed data for 163 chemical analytes in 12 chemical classes for subsamples of 268 pregnant women from NHANES 2003–2004, a nationally representative sample of the U.S. population. For each chemical analyte, we calculated descriptive statistics. We calculated the number of chemicals detected within the following chemical classes: polybrominated diphenyl ethers (PBDEs), perfluorinated compounds (PFCs), organochlorine pesticides, and phthalates and across multiple chemical classes. We compared chemical analyte concentrations for pregnant and nonpregnant women using least-squares geometric means, adjusting for demographic and physiological covariates.

RESULTS: The percentage of pregnant women with detectable levels of an individual chemical ranged from 0 to 100%. Certain polychlorinated biphenyls, organochlorine pesticides, PFCs, phenols, PBDEs, phthalates, polycyclic aromatic hydrocarbons, and perchlorate were detected in 99–100% of pregnant women. The median number of detected chemicals by chemical class ranged from 4 of 12 PFCs to 9 of 13 phthalates. Across chemical classes, median number ranged from 8 of 17 chemical analytes to 50 of 71 chemical analytes. We found, generally, that levels in pregnant women were similar to or lower than levels in nonpregnant women; adjustment for covariates tended to increase levels in pregnant women compared with nonpregnant women.

CONCLUSIONS: Pregnant women in the U.S. are exposed to multiple chemicals. Further efforts are warranted to understand sources of exposure and implications for policy making.

KEY WORDS: chemicals, environmental exposures, NHANES, pregnancy. *Environ Health Perspect* 119:878–885 (2011). doi:10.1289/ehp.1002727 [Online 14 January 2011]

Exposure to chemicals during fetal development can increase the risk of adverse health consequences, including adverse birth outcomes (e.g., preterm birth and birth defects), childhood morbidity (e.g., neurodevelopmental effects and childhood cancer), and adult disease and mortality (e.g., cancer and cardiovascular effects) (Gluckman and Hanson 2004; Stillerman et al. 2008). Biomonitoring studies report nearly ubiquitous exposure to many chemicals in the U.S. population—for example, bisphenol A (BPA), perchlorate, and certain phthalates and polybrominated diphenyl ethers (PBDEs) [Centers for Disease Control and Prevention (CDC) 2009a]. These studies, along with more geographically targeted studies of pregnant women, show that pregnant women are also exposed to many chemicals (Bradman et al. 2003; Swan et al. 2005). Chemicals can cross the placenta and enter the fetus, and a number of chemicals measured in maternal urine and serum have also been found in amniotic fluid, cord blood, and meconium (Barr et al. 2007). In some cases, such as for mercury, fetal exposures may be higher than maternal exposure (Barr et al. 2007).

Multiple chemical exposures are of increasing concern. Studies show that exposure to multiple chemicals that act on the same adverse outcome can have a greater effect than exposure to an individual chemical. This has been recognized by the National Academy of Sciences (NAS), which recommends that future efforts accounting for risks from multiple chemical exposures combine effects from chemicals acting on the same adverse health outcome (National Research Council 2008a). Subsequently, assessment of exposure to multiple chemicals has been identified as an important future research area (Kortenkamp 2007).

Because few data are available on levels of individual or multiple chemicals in pregnant women, levels in reproductive-age women have often been used as an indicator of chemical levels in pregnant women (Blount et al. 2000). Some studies have directly compared pregnant women in their cohort and reproductive-age women from the National Health and Nutritional Examination Survey (NHANES), a nationally representative sample of the U.S. population. For example, phthalates measured in pregnant women from three U.S. locations were lower than those measured in reproductive-age women from NHANES (Swan et al. 2005). Numerous physiological changes occur during pregnancy, including weight gain and increases in blood and plasma volume, which can affect concentrations of chemicals (Chesley 1972; Pirani and Campbell 1973). Chemicals may also concentrate in the fetus, which could influence maternal concentrations (Takahashi and Oishi 2000). Further, behavioral changes occurring during pregnancy, such as diet modification (e.g., quantity and food type), may also influence chemical body burdens in pregnant women (Mirel et al. 2009). Understanding whether some of these factors can influence maternal concentrations of chemicals helps inform our ability to use measurements of chemicals in nonpregnant women as a surrogate for pregnant women.

We analyzed biomonitoring data for pregnant women from NHANES to characterize exposure to individual and multiple chemicals and their metabolites in pregnant women. Additionally, we evaluated the extent to which levels measured in nonpregnant women are representative of levels in pregnant women, and what factors may explain observed differences.

Methods

Study population. NHANES, conducted by the CDC, is a nationally representative survey and physical examination assessing the health and nutritional status of the civilian, noninstitutionalized U.S. population. The survey also includes measurement of chemicals and their metabolites in blood and urine (for more information, see CDC 2010). We use the term "chemical analyte" here to describe both chemicals and their metabolites. Because of the complex stratified survey design in NHANES, separate sample weights are assigned to each survey respondent; each participant represents approximately 50,000 other U.S. residents. Pregnant women were oversampled in the NHANES survey from 2001 to 2006 (CDC

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2009b). [Protocols for oversampling pregnant women are described in Supplemental Material (doi:10.1289/ehp.1002727) and in detail elsewhere (Mirel et al. 2009).] We classified pregnancy status according to the results of the urine pregnancy test administered as part of NHANES protocols.

Most chemical analytes were measured in subsets of the total NHANES sample. Each subset included about one-third the total number of participants, so not all chemical analytes were measured in each participant. Further, not every group of chemical analytes was measured in each cycle. Therefore, we analyzed the 2003–2004 cycle, because it represents the cycle with the highest number of chemical analytes measured across the sample of pregnant women. We limited our study population to those 15–44 years of age to be consistent with the definition used by the National Center for Health Statistics for women of childbearing age (Chandra et al. 2005). Therefore, our study population includes 268 pregnant women and 1,489 nonpregnant women 15–44 years of age included in at least one subsample for chemical analyte analysis.

Environmental chemical analyte analyses. Chemical analyte analyses were conducted at the National Center for Environmental Health laboratories (CDC, Atlanta, GA). Analytical procedures and summary statistics for the general population have been described in the Fourth National Report on Human Exposure to Environmental Chemicals and in the peer-reviewed literature (Calafat et al. 2008; Caldwell et al. 2009; CDC 2009a; Sjodin et al. 2008). We assessed 163 chemical analytes across 12 chemical classes (Table 1), measured in blood, urine, and serum.

Data analysis. We conducted analyses in SUDAAN (version 10.0; Research Triangle Institute, Research Triangle Park, NC) and SAS (version 9.2; SAS Institute Inc., Cary, NC). SUDAAN calculates variance estimates after incorporating the nonrandom sampling design and the sample population weights, which account for oversampling of certain subgroups.

We examined summary statistics and distributional plots for each chemical analyte. We calculated the following descriptive statistics [for further details on analysis, see Supplemental Material (doi:10.1289/ehp.1002727)]: percentage of women with levels greater than the limit of detection (LOD), geometric mean (GM), geometric standard error (GSE), median and 95th percentile estimates, and the coefficient of variation (CV; defined as the GSE divided by the GM). The GM, GSE, and CV were calculated only for chemical analytes with > 60% detection frequency. The median and 95th percentile were calculated for all chemical analytes. Concentrations below the LOD were substituted by the CDC with LOD/$\sqrt{2}$. We present statistical results for individual chemical analytes in the main text that are representative of each chemical class [for descriptive statistics and LODs for all 163 chemical analytes, see Supplemental Material, Table 1 (doi:10.1289/ehp.1002727)]. Representative chemical analytes were chosen based on public health relevance and expectation of relatively widespread exposure.

To assess extent of multiple exposures within a chemical class, we evaluated the

Table 1. Chemical classes measured in biological tissue of pregnant women, NHANES 2003–2004.

Chemical class	No. of chemical analytes measured			
	Blood	Serum	Urine	Total
Cotinine		1		1
Environmental phenols			4	4
Metals	4			4
Organochlorine pesticides		13		13
Organophosphate insecticides			6	6
Perchlorate			1	1
Phthalates			13	13
PBDEs and other brominated flame retardants		11		11
PCBs and dioxin-like chemicals		55		55
PAHs			10	10
PFCs		12		12
VOCs	33			33

See Supplemental Material, Table 1 (doi:10.1289/ehp.1002727), for individual chemical analytes included in each chemical class.

Table 2. Characteristics of reproductive-age women by pregnancy status, NHANES 2003–2004.

Demographic characteristic	Pregnant women (n = 268)	Nonpregnant women (n = 1,489)
Age [years (mean ± SE)]**	27 ± 0.8	30 ± 0.37
Age [years (%)]**		
15–17	4	10
18–24	30	23
25–29	31	13
30–34	25	17
35–44	11	37
Race/ethnicity (%)**		
Non-Hispanic white	56	67
Non-Hispanic black	18	14
Mexican American	17	10
Other Hispanic	2	5
Other	6	5
Education (%)		
< High school diploma	26	24
High school diploma	15	22
> High school diploma	59	54
Marital status (%)**		
Married or living with partner	79	50
Divorced, separated, or widowed	2	12
Never married	19	38
Parity (%)**		
0	45	44
1	34	14
≥ 2	21	42
Smoking status (%)**		
Never	59	60
Former	31	11
Current	9	30
Trimester		
First	31	
Second	32	
Third	37	
Biochemical measurements		
Serum albumin [g/dL (mean ± SE)]**	3.46 ± 0.04	4.23 ± 0.01
Urinary creatinine [mg/dL (mean ± SE)]	127.81 ± 6.00	130.86 ± 3.27
Sampling characteristics		
Duration of food and drink fasting before blood collection [hr (mean ± SE)]**	8.40 ± 0.73	10.87 ± 0.10

Data were missing in pregnant women for parity (n = 18), education (n = 3), smoking (n = 6), trimester (n = 41), and length of fasting (n = 2) and in nonpregnant women for parity (n = 160), education (n = 46), smoking (n = 151), and length of fasting (n = 25).
**$p < 0.01$.

number of individual PBDEs, perfluorinated compounds (PFCs), organochlorine pesticides, and phthalates detected in each pregnant woman. We chose these chemical classes to represent banned persistent chemicals (organochlorine pesticides), persistent chemicals (PBDEs and PFCs), and currently used nonpersistent chemicals (phthalates).

We then evaluated the extent of multiple chemical exposures across chemical classes in three different subsamples. These three subsamples were the primary subsamples of the pregnant women. Pregnant women in subsample A were assessed for metals, cotinine, and PFCs (17 chemical analytes in 76 women); in subsample B, for metals, cotinine, organochlorine pesticides, phthalates, PBDEs, and polycyclic aromatic hydrocarbons (PAHs) (52 chemical analytes in 54 women); and in subsample C, for metals, phenols, polychlorinated biphenyls (PCBs), organophosphate insecticide metabolites, perchlorate, and cotinine (71 chemical analytes in 59 women) [for subsample composition, see Supplemental Material, Table 2 (doi:10.1289/ehp.1002727)]. Volatile organic compounds (VOCs) were measured only in a subsample of pregnant women that partially overlapped with subsamples A, B, and C. Consequently, we did not include VOCs in analyses of multiple chemical exposures.

To compare chemical analyte concentrations between pregnant and nonpregnant women, we constructed multivariate regression models, which included our main effect (binary pregnancy status variable) along with covariates. We log-transformed chemical analytes before regression analysis to account for the nonnormal distributions. From these models, we calculated the least-squares geometric means (LSGMs), which provide GM estimates after adjustment for other covariates. For every chemical analyte in the main analysis, we used the same set of covariates. Covariates were included if they were significant predictors of more than one chemical

Table 3. Descriptive statistics for chemical analytes in pregnant and nonpregnant women, NHANES 2003–2004.[a]

Chemical analyte	*n*	Reproductive status	LOD[a]	Percent > LOD	GM (GSE)	50th percentile	95th percentile	CV
Metals [blood (μg/L)]								
Cadmium**	253	Pregnant	0.14	66	0.22 (0.01)	0.2	0.8	0.07
	1,396	Nonpregnant		79	0.33 (0.01)	0.3	1.6	0.03
Lead (μg/dL)**	253	Pregnant	0.28	94	0.68 (0.04)	0.6	1.8	0.06
	1,396	Nonpregnant		99	0.96 (0.04)	0.9	2.4	0.04
Mercury (total)*	253	Pregnant	0.20	89	0.67 (0.07)	0.7	3.4	0.10
	1,396	Nonpregnant		92	0.80 (0.05)	0.8	4.4	0.06
VOCs [blood (μg/L)]								
Benzene	89	Pregnant	0.024	38	—[b]	< LOD	0.2	—[b]
	389	Nonpregnant		53	—[b]	< LOD	0.3	—[b]
1,4-Dichlorobenzene	89	Pregnant	0.12	40	—[b]	< LOD	20.0	—[b]
	373	Nonpregnant		47	—[b]	< LOD	4.1	—[b]
MTBE (methyl *tert*-butyl ether)	85	Pregnant	0.002	86	0.01 (0.01)	0.02	0.1	0.40
	373	Nonpregnant		78	0.01 (0.002)	0.01	0.1	0.20
Toluene**	90	Pregnant	0.025	94	0.07 (0.01)	0.1	0.2	0.07
	387	Nonpregnant		95	0.10 (0.01)	0.1	0.5	0.10
Cotinine [serum (μg/L)]**	249	Pregnant	0.015	66	0.07 (0.02)	0.03	68.8	0.31
	1,369	Nonpregnant		83	0.54 (0.13)	0.1	318.0	0.24
PFCs [serum (μg/L)]								
Perfluorooctanoic acid*	76	Pregnant	0.1	99	2.39 (0.24)	2.6	5.6	0.10
	400	Nonpregnant		99	3.19 (0.16)	3.2	8.4	0.05
PFOS (perfluorooctanyl sulfonate)**	76	Pregnant	0.4	99	12.29 (1.02)	12.0	21.8	0.08
	400	Nonpregnant		100	16.26 (0.84)	15.5	44.0	0.05
PBDEs [serum (ng/g lipid)]								
PBDE-47	75	Pregnant	4.2	99	23.90 (2.21)	23.7	100.0	0.09
	441	Nonpregnant		98	21.15 (2.03)	21.2	114.0	0.10
PBDE-99	75	Pregnant	5.0	87	5.51 (0.61)	5.1	21.8	0.15
	434	Nonpregnant		68	5.04 (0.42)	4.4	31.5	0.08
PBDE-100*	75	Pregnant	1.4	99	6.06 (0.91)	6.6	23.2	0.15
	443	Nonpregnant		96	4.00 (0.43)	3.8	25.2	0.11
PBDE-153	75	Pregnant	2.2	100	9.90 (3.04)	7.8	127.0	0.31
	442	Nonpregnant		93	5.18 (0.53)	4.5	43.9	0.10
PCBs [serum (ng/g lipid)]								
PCB-118	75	Pregnant	0.6	100	4.31 (0.95)	3.6	14.3	0.22
	415	Nonpregnant		100	4.46 (0.28)	4.3	16.9	0.06
PCB-138 and -158	75	Pregnant	0.4	100	7.70 (1.24)	7.3	20.2	0.16
	416	Nonpregnant		100	8.95 (0.55)	8.3	37.0	0.06
PCB-153	75	Pregnant	1.1	100	8.74 (1.29)	8.8	22.5	0.15
	415	Nonpregnant		100	11.07 (0.64)	10.2	44.0	0.06
PCB-180*	75	Pregnant	0.4	96	4.61 (0.99)	6.8	13.2	0.21
	416	Nonpregnant		98	7.42 (0.44)	7.5	33.3	0.06
Organochlorine pesticides [serum (ng/g lipid)]								
DDT (dichlorodiphenyltrichloroethane)	71	Pregnant	7.8	62	—[c]	—[c]	37.4	0.16
	426	Nonpregnant		63	—[c]	—[c]	13.3	0.06
DDE (dichlorodiphenyldichloroethylene)	71	Pregnant	7.8	100	140.39 (29.72)	99.9	850.0	0.21
	424	Nonpregnant		99	151.04 (16.03)	141.0	815.0	0.11
Hexachlorobenzene*	70	Pregnant	7.8	100	11.27 (1.08)	10.4	25.7	0.10
	428	Nonpregnant		99	14.34 (0.39)	14.3	25.7	0.03

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analyte or if their inclusion in the model changed the β-coefficient for the main effect by > 20%. The following covariates were evaluated: age (continuous), race/ethnicity (Mexican American, non-Hispanic white, non-Hispanic black, or other), education (high school diploma or less vs. more than high school diploma), marital status (married/living with a partner, divorced/separated, or never married), parity (number of pregnancies resulting in live births, nulliparous vs. one or more child), current body mass index (BMI; continuous), smoking status (never, former, or current), serum albumin (continuous), length of food and drink fasting before blood collection (0–4.5 hr, 4.5–8.5 hr, or 8.5–24 hr), and urinary creatinine (continuous). All regression models were adjusted for the same covariates except for creatinine (included in models for urinary chemicals only). We excluded 12 nonpregnant women who reported fasting times > 24 hr. We defined statistical significance as $p < 0.10$ for all analyses because of relatively small number of pregnant women sampled for each chemical analyte and, consequently, small degrees of freedom.

As a sensitivity analysis, we performed multivariate regression in women < 35 years of age, because the age distribution differed between the two groups. For this analysis, we selected model covariates separately for each individual chemical analyte using the covariate selection method described above. Thus, the covariates in the sensitivity analysis may differ from that used in the main analysis. We conducted sensitivity analyses for lead (n = 215 pregnant; n = 885 nonpregnant), BPA (n = 63 pregnant; n = 275 nonpregnant), and p,p'-dichlorodiphenyldichloroethene (DDE) (n = 65 pregnant; n = 380 nonpregnant).

Results

Although most pregnant and nonpregnant women were white, there was a higher percentage of Mexican-American pregnant women compared with nonpregnant women, reflecting higher birth rates among Hispanic women in the United States (Table 2) (Martin et al. 2007). Nonpregnant women were older, less likely to be married or with a partner, and more likely to smoke than were pregnant women (Table 2). In addition, pregnant women had lower levels of albumin and shorter fasting times before blood collection than did nonpregnant women.

Table 3 summarizes statistics for pregnant and nonpregnant women for select chemical analytes [for all 163 chemical analytes in pregnant women, see Supplemental Material, Table 1 (doi:10.1289/ehp.1002727)]. We found that 0–100% of pregnant women had a detectable level across the individual chemical analytes. Eight of 12 classes of chemicals included individual chemical analytes detected in 99–100% of pregnant women (PFCs, PBDEs, PCBs, organochlorine pesticides, phenols, phthalates, PAHs, and perchlorate). Four classes (VOCs, PFCs, PCBs, and organochlorine pesticides) included

Table 3. continued.

Chemical analyte	n	Reproductive status	LOD[a]	Percent > LOD	GM (GSE)	50th percentile	95th percentile	CV
Organophosphate insecticide metabolites [urine (µg/L)]								
Dimethylphosphate	89	Pregnant	0.5	44	—[b]	< LOD	13.7	—[b]
	483	Nonpregnant		48	—[b]	< LOD	14.3	—[b]
Diethylphosphate	89	Pregnant	0.1	33	—[b]	< LOD	10.8	—[b]
	474	Nonpregnant		49	—[b]	< LOD	14.8	—[b]
DMTP*	89	Pregnant	0.5	83	2.43 (0.43)	2.7	16.0	0.18
	483	Nonpregnant		73	1.81 (0.17)	1.7	28.3	0.09
Diethylthiophosphate	87	Pregnant	0.2	57	—[b]	0.2	2.2	—[b]
	478	Nonpregnant		46	—[b]	< LOD	2.6	—[b]
Dimethyldithiophosphate	86	Pregnant	0.1	56	—[b]	0.2	3.2	—[b]
	475	Nonpregnant		34	—[b]	< LOD	4.0	—[b]
Environmental phenols [urine (µg/L)]								
BPA	86	Pregnant	0.4	96	2.53 (0.63)	2.7	15.0	0.25
	489	Nonpregnant		96	2.89 (0.29)	3.0	17.6	0.10
Triclosan	86	Pregnant	2.3	87	17.00 (8.74)	8.2	283.0	0.51
	489	Nonpregnant		81	14.65 (0.97)	11.1	411.0	0.07
Benzophenone-3	86	Pregnant	0.3	100	25.49 (6.52)	16.9	353.0	0.26
	489	Nonpregnant		98	37.14 (6.44)	31.4	1530.0	0.17
Phthalates [urine (µg/L)]								
Monobenzyl phthalate	91	Pregnant	0.1	100	15.12 (3.79)	17.8	86.8	0.25
	497	Nonpregnant		100	14.77 (0.79)	15.5	99.9	0.05
Monoisobutyl phthalate	91	Pregnant	0.3	99	3.47 (0.84)	4.4	19.5	0.24
	497	Nonpregnant		98	4.21 (0.27)	4.5	21.1	0.06
Mono-*n*-butyl phthalate	91	Pregnant	0.4	99	18.83 (4.11)	17.1	143.8	0.22
	497	Nonpregnant		99	24.64 (1.16)	25.7	132.2	0.05
MEP	91	Pregnant	0.4	100	226.53 (79.03)	265.7	2263.0	0.35
	497	Nonpregnant		100	246.06 (29.56)	234.5	2992.6	0.12
PAHs [urine (µg/L)]								
9-Hydroxyfluorene	85	Pregnant	0.005	100	0.21 (0.04)	0.2	0.8	0.19
	478	Nonpregnant		100	0.30 (0.03)	0.2	1.1	0.11
2-Naphthol	91	Pregnant	0.031	100	2.49 (0.59)	2.4	14.7	0.24
	492	Nonpregnant		100	3.68 (0.31)	3.3	28.7	0.08
2-Hydroxyphenanthrene	87	Pregnant	0.005	100	0.06 (0.01)	0.05	0.2	0.17
	479	Nonpregnant		99	0.06 (0.004)	0.06	0.3	0.07
1-Hydroxypyrene	86	Pregnant	0.005	100	0.08 (0.02)	0.08	0.5	0.25
	481	Nonpregnant		99	0.09 (0.007)	0.09	0.6	0.07
Perchlorate [urine (µg/L)]*	89	Pregnant	0.05	100	4.17 (0.84)	4.3	34.0	0.07
	492	Nonpregnant		100	2.68 (0.21)	2.8	11.0	0.08

[a]For most chemicals, the LOD is constant across samples. However, for persistent organic pollutants (PBDEs, PCBs, and organochlorine pesticides), each individual sample has its own LOD because the available sample volume differed by sample, and a higher sample volume results in a lower LOD. For chemicals with sample-specific LODs, the maximum LOD is reported. In general, the average LOD is approximately 40–50% of the maximum LOD (CDC 2009). [b]GM ,GSE, or CV could not be calculated because detection frequency is< 60%. [c]GM or percentile estimate is not reported because it is less than the maximum LOD. *$p < 0.10$; **$p < 0.01$; calculated using univariate regression analysis.

at least one individual chemical analyte not detected in any pregnant women [see Supplemental Material, Table 1 (doi:10.1289/ehp.1002727)]. In general, organophosphate metabolites, VOCs, and dioxins and furans were less frequently detected in pregnant women than were the other chemical classes except for dimethylthiophosphate (DMTP), toluene, *m*- and *p*-xylene, and methyl *tert*-butyl ether (MTBE).

Among pregnant women, DDE had the highest GM concentration (140.4 ng/g lipid) of the persistent, lipophilic compounds measured in serum (PCBs, PBDEs, and organochlorine pesticides), whereas concentrations of most of the other measured chemical analytes in these classes were an order of magnitude lower (PCBs, 4–8 ng/g lipid; PBDEs, 5–23 ng/g lipid). Perfluorooctane sulfonic acid (PFOS) had the highest GM among the persistent chemical analytes that do not accumulate in lipids (e.g., lead, cadmium, and PFCs). Of the nonpersistent chemical analytes measured in urine (organophosphate metabolites, phenols, phthalates, PAHs, and perchlorate), triclosan, benzophenone-3, and monoethyl phthalate (MEP) had the highest GMs (17.00, 25.49, and 226.53 μg/L, respectively).

Although the GM for cotinine was < 1 μg/L, the range of concentrations spanned three orders of magnitude (CV = 0.31). Variability in other chemical analyte levels measured in pregnant women was generally low (CV < 0.25), except for some phenols (CV = 0.25–0.51), phthalates (CV = 0.22–0.35), MTBE (CV = 0.40), triclosan (CV = 0.51), and PBDE-153 (CV = 0.31).

Figure 1 shows the numbers of individual PFC, PBDE, organochlorine pesticide, and phthalate chemical analytes detected in individual pregnant women. At least two organochlorine pesticides, one PBDE, two PFCs, and four phthalates were measured in each pregnant woman. The median number of chemicals detected for organochlorine pesticides, PBDEs, PFCs, and phthalates were 6, 6, 4, and 9, respectively. For PBDEs and phthalates, 7% and 2%, respectively, had detectable levels of ≥ 90% of the chemical analytes in the class.

The median number of chemical analytes detected among women in subsamples A, B, and C were 8 (range, 4–12), 37 (range, 28–45), and 50 (range, 35–60), respectively (Figure 2). We found generally that the overall number of chemicals detected was not dominated by detects within a particular chemical class (Figure 3). For example, several participants in subsample B at the median detected level (37 chemicals) had 10 phthalates, 10 PAHs, 7 PBDEs, 6 organochlorine pesticides, 3 metals, and cotinine detected.

GM and median levels for most chemicals were similar to or lower than those in pregnant than in nonpregnant women, except for PBDEs, DMTP, triclosan, and perchlorate (Table 3). About half the LSGM estimates for pregnant women (Table 4) increased after adjusting for covariates (Tables 3 and 4). For a few chemicals, the LSGM estimates for pregnant women decreased after adjustment, such as PBDEs, some phthalates, perchlorate, and BPA. In general, adjusted LSGMs were comparable between pregnant and nonpregnant women (Table 4). Nonpregnant women had significantly higher levels of cadmium, lead, PFOS, BPA, and cotinine, but pregnant women had significantly higher levels of DDE, DMTP, MTBE, and perchlorate (Table 4). The most pronounced differences between pregnant and nonpregnant women were for MTBE and DMTP (levels in pregnant women were about two times those of nonpregnant women) and cotinine (levels in pregnant women were about half those of nonpregnant women).

Serum albumin influenced the comparison between pregnant and nonpregnant women for 28 of the 32 compounds evaluated in regression analyses (the β-coefficient changed by > 20%); however, direction of the effect varied by type of compound. In general, for chemical analytes measured in blood, effect estimates for albumin were positive, and their inclusion increased the LSGMs for pregnant women; in contrast, for nonpersistent urinary chemical analytes, the albumin effect estimates were more often negative, and their inclusion

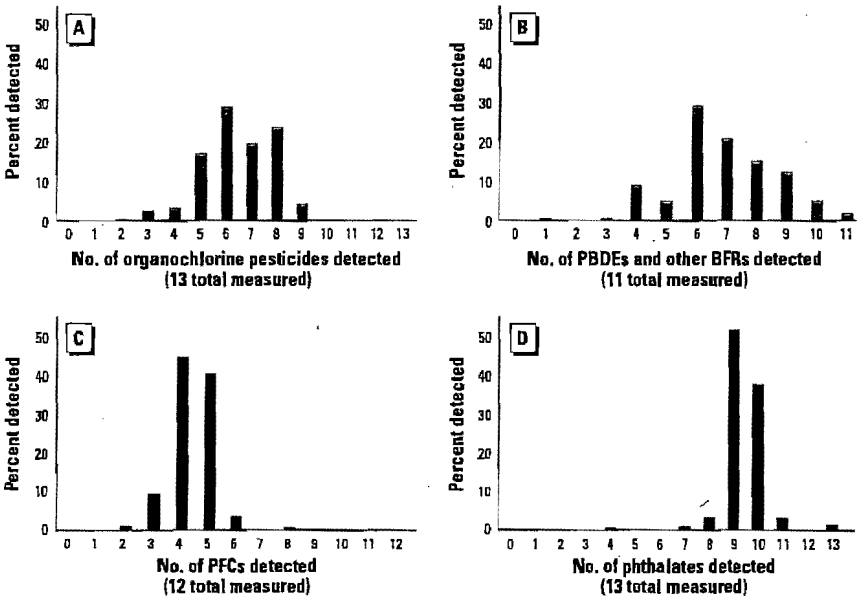

Figure 1. Distribution of the number of chemicals detected in U.S. pregnant women for four chemical classes: organochlorine pesticides (*A*; *n* = 71), PBDEs (*B*; *n* = 75), PFCs (*C*; *n* = 76), and phthalates (*D*; *n* = 91).

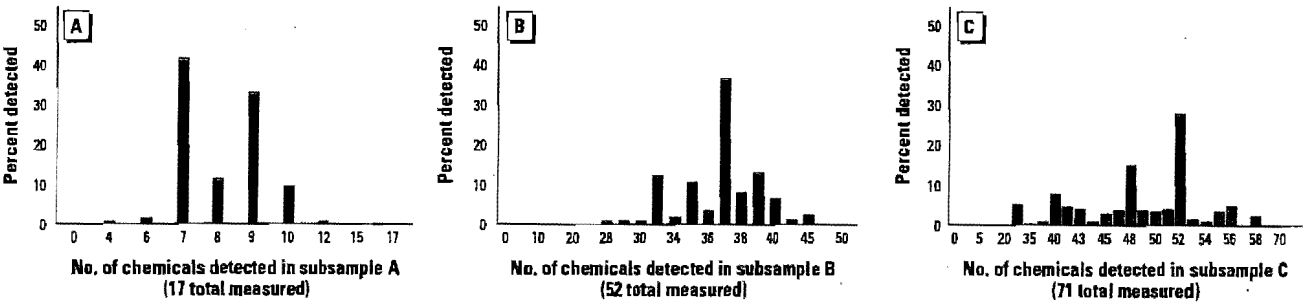

Figure 2. Distribution of the number of chemicals detected in U.S. pregnant women across multiple chemical classes. (*A*) Subsample A (metals, cotinine, and PFCs). (*B*) Subsample B (metals, cotinine, organochlorine pesticides, phthalates, PBDEs, and PAHs). (*C*) Subsample C (metals, phenols, PCBs, organophosphate insecticide metabolites, perchlorate, and cotinine).

decreased the LSGMs for pregnant women (data not shown). Smoking influenced comparison of LSGMs between pregnant and nonpregnant women for 75% of chemicals. Maternal age and BMI changed the LSGMs for persistent organic pollutants such as PCBs, and creatinine influenced LSGMs for most chemical analytes measured in urine. Other variables, such as race/ethnicity and education, were often significant predictors of chemical analyte concentrations but generally did not change LSGM comparisons in Table 4.

Compared with estimates based on women of all ages, LSGMs for lead and DDE for both pregnant and nonpregnant women were reduced when we restricted analyses to younger women (< 35 years of age). However, relative differences in adjusted estimates between pregnant and nonpregnant women were not substantially affected. LSGMs for BPA increased for both groups in the restricted analysis, and the differences in LSGM estimates between pregnant and nonpregnant women were no longer statistically significant [LSGM = 2.16 (pregnant) vs. 3.03 μg/L (nonpregnant), $p = 0.24$].

Discussion

We found widespread exposure to pregnant women in the United States to multiple chemical analytes, including both banned and contemporary contaminants. Although we did not make any direct connection to potential adverse health consequences, levels of many of these chemical analytes were similar to those measured in epidemiologic studies finding an association between prenatal chemicals exposure and adverse reproductive and developmental outcomes. These include phthalates and increased risk of adverse male reproductive outcomes (Swan et al. 2005), mercury and developmental neurological outcomes (Lederman et al. 2008), PBDEs and neurodevelopmental outcomes (Herbstman et al. 2010), and PCBs and maternal thyroid hormone disruption during pregnancy (Chevrier et al. 2008).

Additionally, pregnant women were exposed to multiple chemical analytes at one time, many of which can affect the same adverse outcomes. Examples include maternal thyroid hormone disruption [e.g., perchlorate, PCBs, PBDEs, and triclosan (Crofton 2008)], male reproductive development (multiple phthalates), and the developing brain (mercury, lead, PCBs) (National Research Council 2008a). The NAS has recommended risk assessment of multiple chemicals expand to account for chemicals acting on a common adverse outcome (National Research Council 2008a). Although the NAS focused on grouping chemicals contributing to disturbances of androgen action, they also proposed this approach for chemicals affecting brain development (National Research Council 2008a).

Levels of chemicals measured during pregnancy can be influenced by physiological (e.g., changes in BMI, plasma volume expansion, and bone mobilization) and behavioral factors. For example, previous research has found an inverse relationship between weight gain during pregnancy and levels of persistent organic pollutants in pregnant women (Bradman et al. 2006). We found that plasma volume expansion, using the level of albumin as a surrogate, may also influence chemical levels measured in pregnant women. Plasma volume begins to expand in pregnant women at around 8 weeks of gestation and increases progressively until 30–34 weeks gestation, when it plateaus. This expansion may dilute environmental chemical concentrations in blood (Faupel-Badger et al. 2007). Accurately measuring plasma volume expansion is expensive and ideally requires multiple measurements throughout pregnancy (Faupel-Badger et al. 2007). However, albumin measurements may provide a reasonable surrogate because previous studies suggest that blood volume expansion dilutes circulating levels of albumin during pregnancy (Honger 1968). We found that, in general, adjusting for albumin increased GM estimates of persistent compounds, such as DDE, in pregnant women, suggesting that the concentration is diluted by increased plasma volume. However, adjustment for albumin generally decreased estimates for nonpersistent compounds, such as BPA, in pregnant women, suggesting that lower albumin may be associated with an increased clearance of environmental contaminants. Albumin may affect metabolism and transport of chemicals by mechanisms other than plasma volume expansion. For example, previous research has shown that PFCs actually bind to albumin in the blood (Jones et al. 2003). BPA also binds to plasma proteins, such as albumin, in humans (Teeguarden et al. 2005), so reduced albumin during pregnancy may influence the amount of BPA that undergoes phase II conjugation and subsequent elimination through urine. The role of albumin, and other transport proteins, in the transport and metabolism of environmental chemicals, particularly during pregnancy, is an important topic and requires further research.

We found that, generally, the levels in pregnant women were similar to or lower than levels measured in nonpregnant women. Adjusting for physiological factors that may influence levels of chemicals in pregnant women tended to increase the levels in pregnant women compared with nonpregnant women. This suggests that generally levels of chemicals in nonpregnant reproductive-age women are reasonably representative of levels found in pregnant women. However, for several chemicals, levels in pregnant women remain lower than those in nonpregnant women. Behavioral factors may explain this difference (e.g., cotinine and smoking), or other physiological factors may be important [e.g., chemical levels concentrating in the fetus such as for BPA (Takahashi and Oishi 2000)].

The NHANES study design, where groups of chemicals were analyzed in approximate one-third–sized subsamples, meant that we could not evaluate more than 71 chemical analytes in any individual pregnant women, or about 44% of chemical analytes measured

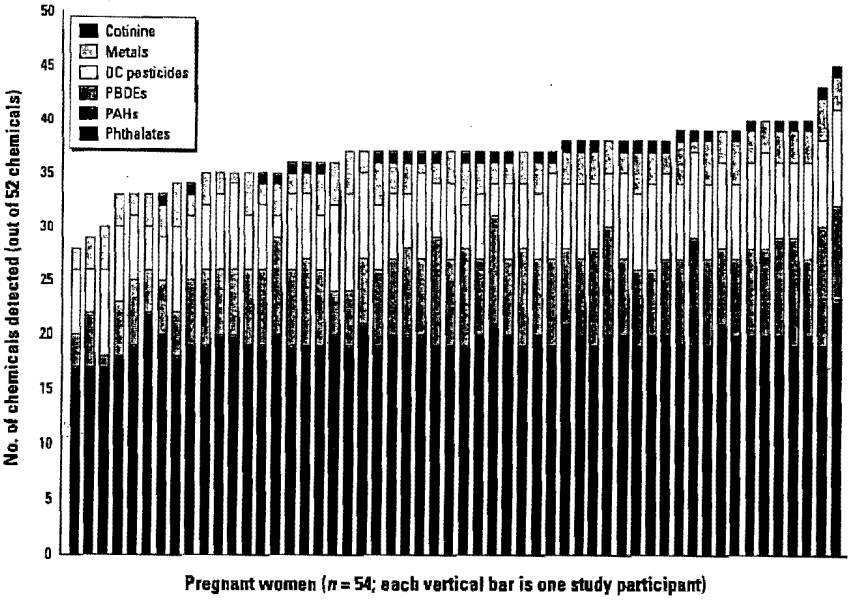

Figure 3. Number of chemicals detected by chemical class in U.S. pregnant women, NHANES subsample B [metals, cotinine, organochlorine (OC) pesticides, phthalates, brominated flame retardants (PBDEs), and PAHs], 2003–2004 ($n = 54$). Each vertical bar represents one study participant. Other subsamples showed similar results.

during 2003–2004. This also limited our ability to assess exposures to multiple chemical analytes that may be acting on the same adverse outcome (e.g., PBDEs and PCBs, which can affect neurodevelopment, were not measured in the same women). Given that several chemical analytes within each of the classes were detected almost ubiquitously, pregnant women have more detectable chemical analytes than we could assess in any individual participant in this analysis.

Other methodological changes between cycles make it challenging to compare data across NHANES cycles. For example, the number and types of chemicals sampled changes by cycle. Another challenge is that LODs vary among the cycles. Mostly they decreased, such as with PCBs, which can increase the number of chemicals detected. However, a few LODs increased; for example, certain urinary phthalate esters, such as mono-2-ethylhexyl phthalate (MEHP) and MEP, increased between 2003–2004 and 2005–2006.

Chemical analyte concentrations in NHANES participants should be representative of typical U.S. concentrations. Thus, highly exposed subpopulations may be underrepresented. For example, women living in the agricultural Salinas Valley of California had higher measurable levels of several pesticides than did NHANES pregnant women (Castorina et al. 2010). Other subpopulations may have nonrepresentative exposure patterns, such as high fish consumption or higher use of certain personal care products.

Our analysis indicates high variability in exposures for some chemical analytes, shown by the relatively high CV for phenols, phthalates, cotinine, and MTBE. For some of these analytes, with almost an order of magnitude difference between the median and the 95th percentile, variarion may reflect geographic variability in exposure sources. For example, MTBE was used in reformulated gasoline starting in 1995. Reformulated gasoline was required for use year-round in cities with significant smog problems (Energy Information Administration 2008), so it was not used in

Table 4. Comparison of chemical analyte concentrations between pregnant and nonpregnant women after adjustment for covariates,[a] calculated from multivariate regression models.

Chemical analyte	β-Coefficient (90% CI)[b]	Pregnant women		Nonpregnant women	
		LSGM	90% CI	LSGM	90% CI
Metals [blood (μg/L)]			$n = 225$		$n = 1,091$
Cadmium	−0.20 (−0.36 to −0.04)*	0.27	0.23–0.31	0.33	0.31–0.35
Lead (μg/dL)	−0.16 (−0.27 to −0.06)*	0.80	0.72–0.89	0.94	0.89–0.99
Mercury (total)	−0.11 (−0.33 to 0.10)	0.71	0.57–0.89	0.79	0.72–0.88
VOCs [blood (μg/L)]			$n = 82$		$n = 334$
MTBE	0.97 (0.03 to 1.90)*	0.02	0.01–0.06	0.008	0.005–0.01
Toluene	0.15 (−0.14 to 0.43)	0.11	0.08–0.14	0.09	0.08–0.10
Cotinine [serum (μg/L)]			$n = 225$		$n = 1,091$
	−0.94 (−1.39 to −0.48)**	0.19	0.13–0.28	0.49	0.42–0.58
PFCs [serum (μg/L)]			$n = 70$		$n = 313$
Perfluorooctanoic acid	−0.18 (−0.37 to 0.02)	2.69	2.18–3.32	3.22	2.95–3.52
PFOS	−0.23 (−0.35 to −0.12)**	12.81	11.94–13.74	16.28	15.18–17.46
PBDEs [serum (ng/g lipid)]			$n = 68$		$n = 366$
PBDE-47	0.02 (−0.32 to 0.35)	21.76	16.73–28.30	21.33	18.21–24.97
PBDE-99	−0.11 (−0.47 to 0.26)	4.62[c]	3.37–6.33	5.10	4.44–5.87
PBDE-100	0.24 (−0.22 to 0.70)	5.21	3.60–7.52	4.10	3.38–4.97
PBDE-153	0.51 (−0.10 to 1.12)	8.85	5.05–15.50	5.31	4.46–6.33
PCBs [serum (ng/g lipid)]			$n = 66$		$n = 334$
PCB-118	−0.02 (−0.31 to 0.28)	4.39	3.20–6.02	4.44	3.99–4.93
PCB-138 and -158	−0.07 (−0.33 to 0.19)	8.25	6.57–10.36	8.85	7.96–9.83
PCB-153	−0.11 (−0.39 to 0.17)	9.87	7.73–12.62	11.02	9.92–12.25
PCB-180	−0.27 (−0.65 to 0.11)	5.64	3.97–8.01	7.39	6.77–8.07
Organochlorine pesticides [serum (ng/g lipid)]			$n = 64$		$n = 354$
DDT	−0.10 (−0.32 to 0.13)	3.49[c]	2.78–4.38	3.86[c]	3.60–4.14
DDE	0.33 (0.12 to 0.53)*	198.34	160.72–244.78	142.59	126.13–161.21
Hexachlorobenzene	−0.02 (−0.14 to 0.10)	13.74	12.36–15.26	14.01	13.53–14.51
Organophosphate insecticide metabolites [urine (μg/L)]			$n = 74$		$n = 370$
DMTP	0.95 (0.34 to 1.35)*	4.39	2.74–7.05	1.88	1.60–2.20
Environmental phenols [urine (μg/L)]			$n = 72$		$n = 371$
BPA	−0.55 (−0.97 to −0.13)*	1.63	1.13–2.36	2.83	2.42–3.31
Triclosan	0.47 (−0.60 to 1.54)	23.81	8.17–69.36	15.03	13.06–17.29
Benzophenone-3	−0.07 (−1.26 to 1.12)	38.09	14.02–103.46	40.85	29.28–57.00
Phthalates [urine (μg/L)]			$n = 75$		$n = 377$
Monobenzyl phthalate	−0.02 (−0.53 to 0.50)	14.73	8.86–24.49	15.03	13.77–16.41
Monoisobutyl phthalate	−0.37 (−0.76 to 0.03)	2.83	1.89–4.23	4.06	3.65–4.50
Mono-*n*-butyl phthalate	−0.26 (−0.62 to 0.11)	18.36	12.93–26.07	23.81	21.81–25.99
MEP	−0.13 (−0.93 to 0.66)	221.41	98.85–495.90	254.68	206.36–314.30
PAHs [urine (μg/L)]			$n = 74$		$n = 372$
9-Hydroxyfluorene	−0.15 (−0.50 to 0.19)	0.20	0.14–0.28	0.23	0.21–0.26
2-Naphthol	−0.15 (−0.57 to 0.27)	3.00	1.97–4.58	3.49	3.20–3.81
2-Hydroxyphenanthrene	−0.12 (−0.27 to 0.02)	0.05	0.04–0.06	0.06	0.05–0.06
1-Hydroxypyrene	−0.14 (−0.46 to 0.19)	0.08	0.06–0.10	0.09	0.08–0.09
Perchlorate [urine (μg/L)]			$n = 74$		$n = 374$
	0.25 (0.05 to 0.45)*	3.35	2.67–4.21	2.61	2.31–2.95

CI, confidence interval. Sample sizes for chemical classes are approximate because sample sizes vary slightly by chemical.
[a]Models adjusted for age, race/ethnicity, education, smoking, parity, BMI, albumin, duration of fasting before specimen collection, and creatinine (only urinary chemical analytes adjusted for creatinine). [b]Reference group is nonpregnant women. Chemical analyte concentrations are log-transformed. [c]LSGM (least-squares geometric mean) estimates are < LOD (see Table 3). *$p < 0.10$; **$p < 0.01$.

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every U.S. location. Thus, the geographic variation in MTBE use may play a role in the wide exposure variability (Energy Information Administration 2008). PBDE-153 is another example of how geographic use variation can influence exposures levels. The 95th percentile of PBDE-153 levels is 15 times greater than the median, and previous research has found PBDE concentrations to be around two times higher in Californians than in others in the United States, likely because of California's unique flammability standard (Zota et al. 2008). Variation in exposure to chemical analytes used in consumer and personal care products (e.g., triclosan, where the 95th percentile is 35 times greater than the median) could be driven by unique product uses (Allmyr et al. 2009). Although biomonitoring studies can demonstrate variation in exposures within populations, they generally are limited in their ability to identify sources of exposures. Consequently, additional exposure assessment research is needed to identify the dominant sources of exposure among pregnant women and the general population.

Our analysis of the NHANES pregnancy data shows ubiquitous exposure to multiple chemicals during a sensitive period of fetal development. The NAS recommends accounting for both multiple exposures and exposures that occur during vulnerable developmental periods in improved approaches for assessing chemical risks across the population, which includes shifting to a risk assessment approach that presumes no threshold of effect among the population unless shown otherwise (National Research Council 2008b). Data, such as from NHANES, should be used to enhance our understanding of risks among the U.S. population and to inform further policy and research activities.

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Pesticide Use Linked to Lupus, Rheumatoid Arthritis

By Jan Ehrman

Along with what you say, be careful what you spray. Frequent or extended exposure to pesticides may increase the risk for developing autoimmune diseases such as lupus and rheumatoid arthritis, according to the results of a longterm follow-up study of thousands of postmenopausal women.

Dr. Christine G. Parks of NIEHS led a study showing that frequent or extended exposure to pesticides may increase the risk of developing such autoimmune diseases as lupus and rheumatoid arthritis.

The findings were recently presented by lead scientist Dr. Christine G. Parks of the National Institute of Environmental Health Sciences and her colleagues.

Nearly a billion pounds of pesticides, typically used to kill termites, fleas and household bugs, are spread into the environment each year, through both agricultural and nonagricultural use. According to the *2008-2009 Annual Report of the President's Cancer Panel*, nearly 1,400 pesticides have been registered and approved by the Environmental Protection Agency. However, the report notes, exposure to chemicals found in pesticides has been associated with a variety of cancers including breast, colon, prostate and lung cancer. Further, some research has shown higher rates of various cancers in farmers, pesticide applicators and manufacturers compared to the general, non-using public.

In addition, it is believed that the chemical substances found in pesticides can be toxic to the developing brain. This is backed by recent findings showing that prenatal pesticide exposure may affect intelligence and learning in children, when tested at 3 years of age. Other recent studies show that pesticide exposure may elevate the risk of Parkinson's disease.

Now it appears that a new series of conditions referred to as autoimmune rheumatic disorders— lupus and rheumatoid arthritis (RA)— may also be linked to pesticide exposure.

Parks and her associates looked at the possible relationship between self-reported household insecticide application and the development of either lupus or RA in almost 77,000 women participating in the Women's Health Initiative. The WHI Observational Study, a cohort investigation that began in 1991, was initially designed to track the most common causes of mortality, disability and poor quality of life.

"Although the hypothesis was well-founded [based on higher rates of some autoimmune diseases associated with farming], I was somewhat surprised at the findings," said Parks, who reported that the strongest association between pesticides and the two autoimmune disorders was seen in women who lived on a farm and reported personally applying insecticides. These individuals displayed nearly three times the risk for disease development, compared to women who used no pesticides whatsoever. Meanwhile, lupus/RA risk was doubled for women who underwent 20 or more years of direct exposure (personally applying pesticides) and for those who reported applying insecticides six or more times annually.

While most of the women in the study were Caucasian, no racial differences were seen and the findings were not changed in analyses that accounted for other disease risk factors.

Lupus, also known as systemic lupus erythematosus, is an autoimmune disease—a condition in which the body attacks itself—causing inflammation and damage to healthy tissues and key organs including the heart, lungs and brain. Most lupus patients are female, indicating the condition could have a hormonal or other gender-specific component. RA, another autoimmune disorder, causes joint inflammation and pain, fatigue and other symptoms that may persist for years. Affecting more than a million children and adults, the disease is more prominent in women than men.

In general, the etiology as well as the role of external factors in the development of autoimmune diseases are not well understood. Although data are scarce, most recent findings indicate that the environment may play a contributing role.

While the findings are notable, Parks' study did have a few shortcomings, she explained. For example, because of the general type of question asked "we were not able to determine which specific insecticides were applied." Also, she pointed out, the data were based on participants' long-term recall.

Still, the findings were robust, that is—"We could see a similar pattern of association for both diseases and a dose response for both increasing frequency and duration of use," said Parks. In other words, the more the exposure, the greater likelihood of developing lupus or RA. She noted that, based on previous studies of farm work, similar findings might be expected in men.

The NIEHS scientist added that a prudent approach would be to limit one's exposure to pesticides as much as possible.

The findings were reported in the February issue of *Arthritis Care and Research*.

◄ back to top of page

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An Overview Comparing Results from Two Decades of Monitoring for Pesticides in the Nation's Streams and Rivers, 1992–2001 and 2002–2011

Scientific Investigations Report 2014–5154

U.S. Department of the Interior
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Mysterious bee die-off continues, extends beyond winter

Winter losses lower, but one in three colonies lost across US in past year

May 15, 2014 | Tim Wheeler


Beekeeper Scott Seccomb holds frame from a hive covered with... (Kim Hairston, Baltimore...)

The mysterious die-off of honey bees continues, as beekeepers across the nation lost more than one in three of their colonies since last spring, researchers reported Thursday. The losses in Maryland were even more extreme, where nearly half were lost, according to the state's chief apiary inspector.

The national survey of beekeepers found that they lost one in five honey bee colonies over the winter, fewer than the winter before. But they reported seeing substantial die-off in summer as well, pushing their year-round losses to more than a third.

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The annual survey, led by a University of Maryland entomologist, is part of an effort to get to the bottom of high death rates that commercial beekeepers have experienced for nearly a decade. The losses impose high costs on beekeepers and could lead to shortages of some crops that depend on honey bees for pollination, experts say.

While many beekeepers and some researchers have linked the die-off to pesticide exposures, the team that did the survey say no single culprit is responsible for all the honey bee deaths. But Dennis vanEngelsdorp, the UM assistant professor of entomology who led the survey team, said mortality was much lower among beekeepers who treated their hives to control a common but lethal parasite, the varroa mite.

Researchers surveyed nearly 7,200 beekeepers, who collectively manage about a fifth of the nation's 2.6 million commercial honey bee colonies. It was conducted for the Bee Informed Partnership, a joint effort of the Apiary Inspectors of America and the U.S. Department of Agriculture.

In Maryland, Jerry Fischer, chief apiary inspector with the Maryland Department of Agriculture, said the state's beekeepers have been losing about a third of their colonies annually for several years. The rate increased in the past year to nearly 50 percent, he said, which he blamed on an unusually cold winter.

"It was about the worst winter we've had in the past 20 years, for bee management and surviving bees," Fischer said.

Overall, though, the state apiarist attributed 80 percent of honey bee losses in the state to the inexperience of the state's mostly part-time beekeepers, rather than pesticides, mites or any other outside factor.

Fischer said there are just four large commercial beekeeping operations in the state. The vast majority of Maryland's 1,851 registered beekeepers are what he calls "hobbyists," for whom tending honey bees is not a full-time livelihood. They manage 14,000 colonies, he said, with nearly two thirds having two or fewer colonies.

Many of the colonies he inspected late last summer appeared not to have stored up adequate honey supplies to survive the winter, he said.

But longtime beekeeper Steve McDaniel in Carroll County said he believes the pattern of die-off he's seen still implicates widespread use of pesticides containing neonicotinoids, a nerve agent. Some pesticides sold for homeowners' use contain it, he said.

Preliminary results of a survey he conducted of central Maryland beekeepers found colonies in urban and suburban areas 3.5 times more likely to die than those in rural and farm areas. Of 21 participating beekeepers, a third of their

130 colonies were lost in the past year. The loss rate reached 52 percent among suburban colonies, with only 15 percent losses in farm and rural areas.

McDaniel, a retired chemist who's been keeping bees for 36 years, said he lost two-thirds of the 20 colonies he had at this time last year. In early March, he said, he suddenly lost "a big strapping colony of bees" that appeared to be doing fine with ample food. When he returned to check the hive a week later, he saw "piles of dead bees, inside and out."

A new study conducted at the Harvard School of Public Health and published in the Bulletin of Insectology found that two widely used neonicotinoid pesticides appear to "significantly harm honey bee colonies over the winter." The colder the winter, the more severe the harm, the study's authors suggested.

The study echoed a 2012 finding of a link between low doses of one neonicotinoid compound and "Colony Collapse Disorder," which that makes bees abandon their hives and die. A second pesticide had the same effect, Harvard researchers found.

See Also

1. **Bee Keepers**
2. **Raising Bees**
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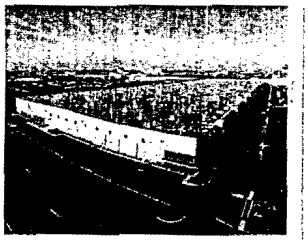

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United States Government Accountability Office

GAO

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August 2013

PESTICIDES

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OPINIONWORKS
PUBLIC SPIRITED RESEARCH

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www.OpinionWorks.com

To: **Ruth Berlin, LCSW-C, Executive Director**
Maryland Pesticide Network

From: **Steve Raabe, President**
OpinionWorks, LLC

Date: **February 10, 2014**

Subject: **Maryland Voter Survey on Pesticides**

The Maryland Pesticide Network commissioned this statewide Maryland voter survey to measure public attitudes about pesticide use and to test support for two public policy proposals. The survey found profound voter concern about health and environmental risks posed by pesticides and overwhelming support for better reporting of pesticide use.

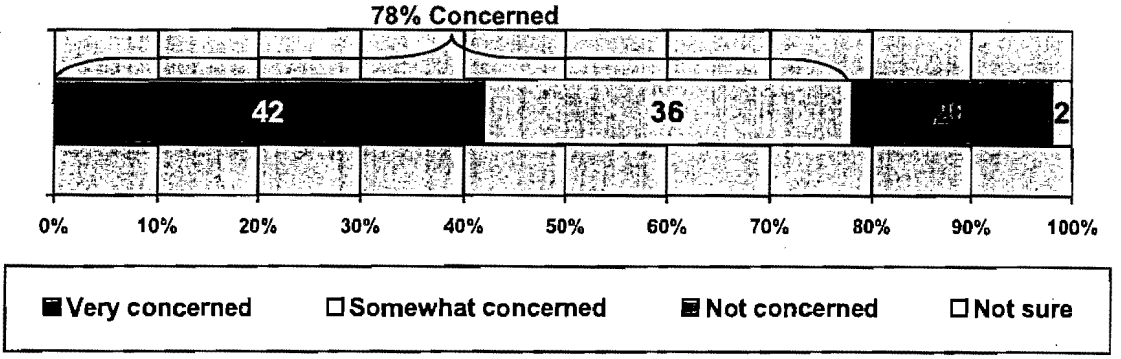
This telephone poll of 799 randomly-selected Maryland registered voters was conducted by telephone January 14-20, 2014, using trained and supervised live interviewers. The findings have a potential sampling error of no more than ± 3.4 percent at the 95% confidence level. A more detailed methodology statement is found at the end of this summary.

Findings

Profound Concern about the Impact of Pesticides on Health

Maryland voters are extremely concerned about the possible impact of pesticides on their health. More than three-quarters (78%) of Maryland voters are concerned about "the risk that pesticides pose to your own and your family's health." More than four voters in ten (42%) are ***very*** concerned about that.

Voter Concern about Health Impacts of Pesticide Use
Maryland Registered Voters Statewide



"How concerned are you about the risk that pesticides pose to your own and your family's health? Very concerned, somewhat concerned, or not concerned?"

Concern about the health impacts of pesticide use is widespread across the state and among all major demographic and political subgroups, with concern exceeding two-thirds of the public in every segment we tested. Democrats (82% concerned), Republicans (68%), and Independent voters (80%) all express high levels of concern about pesticides.

Concern is particularly acute among women, African-Americans, and in Baltimore City and County, rising as high as 92%. And concern is nearly as high in Maryland's 14 more rural counties, where 75% of voters express concern about pesticides.

This table summarizes the level of concern about pesticides in various segments of the public:

Concern about Pesticides by Subgroup

Voter Group	Very Concerned	Somewhat Concerned	Total
All Voters	**42%**	**36%**	**78%**
Women	49%	35%	84%
Men	33%	38%	71%
Whites	37%	39%	77%
African-Americans	52%	30%	82%
All Others	41%	37%	77%
Baltimore City	63%	29%	92%
Baltimore County	49%	39%	88%
Montgomery County	32%	35%	67%
Prince George's County	49%	33%	82%
Greater Baltimore (Anne Arundel, Baltimore City, Baltimore County, Carroll, Harford, Howard)	46%	36%	82%
Greater Washington (Charles, Frederick, Howard, Montgomery, Prince George's)	39%	38%	77%
Rural Counties (14 counties in Western Maryland, Southern Maryland, Eastern Shore)	36%	39%	75%
Democrats	46%	36%	82%
Republicans	33%	35%	68%
Unaffiliated Voters/Third Parties	39%	41%	80%

Numbers may not always appear to add correctly due to rounding.

(continued, next page)

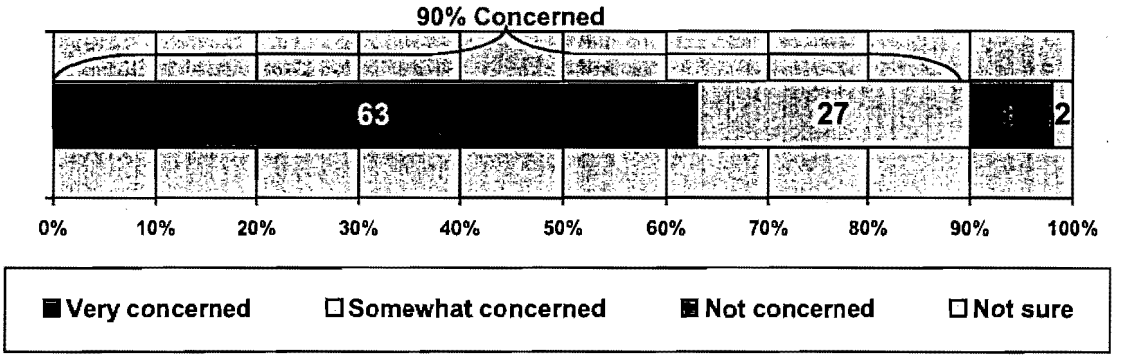
The Effect of Information about Pesticides

The intensity of concern about pesticides increases greatly when voters learn about some of the specific risks that have been linked to pesticide use.

Voter concern rises to 90% when voters hear about health risks such as asthma, autism, and cancer, as well birth defects and fertility problems; and environmental risks to Maryland's rivers and the Bay, as well as bee hive deaths. The effect of this information is a 21-point increase in the number of voters who are very concerned, for a total of 63% of voters who are *very* concerned.

Such profound public concern about a substance so widely and commonly used is extraordinary.

Informed Concern about Pesticide Risks
Maryland Registered Voters Statewide



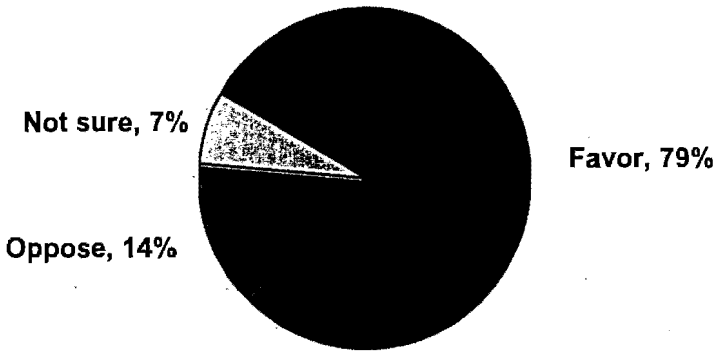
"If you knew that pesticides have been linked to many chronic illnesses including asthma, autism, and cancer, as well as to birth defects and fertility problems; and that pesticides contaminate Maryland's rivers and the Bay and have been linked to bee hive deaths and mutations in fish and fish kills, how concerned would you be about the risk that pesticides pose to your own and your family's health?
Very concerned, somewhat concerned, or not concerned?"

This Deep Concern about Pesticides Drives Support for Policy Changes

Voters' deep underlying concern about pesticides translates into overwhelming support for two specific proposals to give the public more information about pesticide use. The first proposal tested, making pesticide reporting mandatory, is overwhelmingly supported by 79% of Maryland voters, and opposed by only 14%.

(continued, next page)

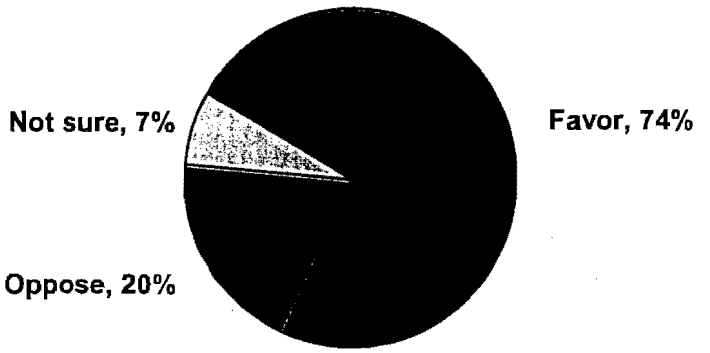
Voter Support for Mandatory Pesticide Reporting
Maryland Registered Voters Statewide



"To track pesticide use, Maryland has conducted a voluntary survey of certified pesticide applicators', agencies', and farmers' pesticide use every few years. Only a small number respond to these surveys, making it difficult to adequately track pesticides' impact on our health, honeybees, and waterways. Would you favor or oppose legislation that would require non-homeowner applicators including farmers and lawn care companies to provide this information to public health and environmental experts?"

A second proposal, charging chemical manufacturers a higher product registration fee comparable with surrounding states to cover the costs of a pesticide reporting database, is supported by 74% of voters, with only 20% opposed.

Voter Support for Higher Chemical Product Registration Fees
Maryland Registered Voters Statewide



"Like all the surrounding states, Maryland charges chemical manufacturers like Dow and Monsanto a small annual product registration fee. At $100 per product per year, Maryland's fee is the second lowest of the surrounding states. Would you favor or oppose raising this fee in line with other states to cover the costs of a modern pesticide reporting database?"

Support for each of these proposals is very broad-based, as reflected in the table on the following page. Geographically, mandatory reporting is supported by a large 70% majority in the rural counties, ranging up to 94% in Baltimore City. Support for raising product registration fees ranges from 67% in the rural counties to 78% in Prince George's County. There are no regions or voter subgroups where support for these proposals is weak.

Support for Public Policy Proposals among Subgroups

Voter Group	Mandatory Reporting	Product Registration Fees
All Voters	**79%**	**74%**
Women	84%	78%
Men	73%	68%
Whites	78%	75%
African-Americans	83%	75%
All Others	75%	68%
Baltimore City	94%	76%
Baltimore County	85%	75%
Montgomery County	80%	74%
Prince George's County	83%	78%
Greater Baltimore (Anne Arundel, Baltimore City, Baltimore County, Carroll, Harford, Howard)	79%	73%
Greater Washington (Charles, Frederick, Howard, Montgomery, Prince George's)	80%	77%
Rural Counties (14 counties in Western Maryland, Southern Maryland, Eastern Shore)	70%	67%
Democrats	83%	78%
Republicans	66%	61%
Unaffiliated Voters/Third Parties	82%	76%

How This Poll Was Conducted

OpinionWorks interviewed 799 randomly-selected registered voters across Maryland by telephone January 14–20, 2014. The interviews were conducted by trained and supervised live interviewers who are skilled in opinion research best practices.

The poll has a potential sampling error of no more than ± 3.4% at a 95% confidence level, meaning that at least 95% of the time the survey results would differ by no more than that margin if every registered voter in Maryland had been interviewed.

Interviewees were drawn randomly from the database of registered voters, supplied by the State Board of Elections and matched with landline and cellular telephone numbers by a commercial vendor. The sample was balanced geographically and by political party during interviewing, and respondents were screened to ensure that only registered voters were interviewed. Weights were applied to bring the voter sample into compliance with the demographic breakdown of the registered voter population.

Brief Background on OpinionWorks

OpinionWorks conducts frequent opinion studies in Maryland and the surrounding states. We are the polling organization for *The Baltimore Sun*, having accurately forecast the 14-point gubernatorial margin in 2010 and the Baltimore mayoral margin in 2011, and have polled for numerous other media throughout the region. We work for state and local agencies throughout the Mid-Atlantic, and for a variety of non-profit and for-profit entities within the region and nationally.

ROCKVILLE, MARYLAND

MEMORANDUM

January 26, 2015

TO: George Leventhal, President, County Council

FROM: Jennifer A. Hughes, Director, Office of Management and Budget
Joseph F. Beach, Director, Department of Finance

SUBJECT: FEIS for Bill 52-14, Pesticides -Notice Requirements -Non-Essential Pesticides Prohibitions

Please find attached the fiscal and economic impact statements for the above-referenced legislation.

JAH:fz

cc: Bonnie Kirkland, Assistant Chief Administrative Officer
Lisa Austin, Offices of the County Executive
Joy Nurmi, Special Assistant to the County Executive
Patrick Lacefield, Director, Public Information Office
Fariba Kassiri, Acting Director, Department of Environmental Protection
Joseph F. Beach, Director, Department of Finance
David Platt, Department of Finance
Matt Schaeffer, Office of Management and Budget
Alex Espinosa, Office of Management and Budget
Felicia Zhang, Office of Management and Budget
Naeem Mia, Office of Management and Budget

Fiscal Impact Statement
Bill 52-14: Pesticides – Notice Requirements – Non-Essential Pesticides -- Prohibitions

1. **Legislative Summary.**

   The bill would update county law with regard to pesticides application in the following manner:

   (1) require posting of notice for certain lawn applications of pesticide;
   (2) prohibit the use of certain pesticides on lawns;
   (3) prohibit the use of certain pesticides on certain County-owned property;
   (4) require the County to adopt an integrated pest management program for certain County-owned property;
   (5) generally amend County law regarding pesticides; and
   (6) require the creation of a media campaign to inform residents and businesses of the change in county law related to non-essential pesticides.

2. **An estimate of changes in County revenues and expenditures regardless of whether the revenues or expenditures are assumed in the recommended or approved budget. Includes source of information, assumptions, and methodologies used.**

   County revenues are not expected to be impacted by Bill 52-14. The Maryland-National Capital Park and Planning Commission (M-NCPPC) did report that there is a potential for lost revenues if playing fields are not able to be adequately maintained – this revenue has traditionally come in in the form of field rental from athletic leagues.

   County departments and agencies performed a fiscal impact analysis of the major provisions and conclude the following:

   - Section 33B-4 requires the county to develop a list of non-essential pesticides and invasive species which would be detrimental to the environment. The Department of Environmental Protection (DEP) does not envision a fiscal impact as a result of these tasks given that many jurisdictions have taken the similar action with regards to non-essential pesticides and significant documentation exists related to successful implementation of this type of prohibition. If classification becomes difficult, a consultant may need to be brought in to assist with this task.
   - Section 33B-13 requires the County Executive to create an Integrated Pest Management (IPM) program. The Department of General Services (DGS) reported no fiscal impact and is currently operating under an IPM and the Executive branch would utilize this plan across county departments under Bill 52-14.
   - Enforcement of Bill 52-14 is not clarified in great detail within the legislation. Similar to other prohibition legislation, executive staff recommends a complaint-driven enforcement model to control costs of implementation. It is likely that complaint-driven enforcement would have a minimal fiscal impact on county departments while estimates for a proactive enforcement effort include a dedicated inspector with estimated personnel costs of $75,000 and vehicle costs of approximately $40,000 for a total of $115,000 per inspector.
   - Bill 52-14 would also require county departments and agencies to convert to approved landscaping practices outside of the list of banned non-essential pesticides

in the cases wherein prohibited pesticides are being used.
Montgomery County Public Schools (MCPS) reported that it is likely that pesticides prohibited under Bill 52-14 are being used currently and that a conversion cost estimate would be available after an agreed list of prohibited pesticides is established. Based on estimates of conversion costs for M-NCPPC fields, the costs of maintaining similar fields within MCPS are expected to be significant.
Montgomery College reported no fiscal impacts as a result of Bill 52-14.
To maintain the quality of fields at the current level, M-NCPPC reported the following conversion costs associated with the move to allowable treatment methods on fields:

Athletic Fields:

- 40 athletic fields can be organically treated at the following cost:
  $648,048 in supplies and labor costs;
  $327,062 to provide a top dressing;
  $100,000 for the purchase of two aerators;
  for a total first year cost of $1,075,110.
  Additional costs in subsequent years also include:
  Sod replacement every two years at a cost of $20,440 per field or $817,600 and additional grading every four years at a total of $10,000 per field or $400,000.
- Five Bermuda playing fields cannot be organically treated and would need to be replaced with treatable sod for $102,200 per field or a total cost of $511,000.
- *Optional* replacement costs for a synthetic turf option are $1,400,000 per field with $3,700 in annual maintenance or a total capital cost of $56,000,000 and a $148,000 annual maintenance cost for all forty fields.

Regional Fields:

- 35 regional fields will need irrigation installed to maintain organic maintenance standards at the following cost:
  $3,500,000 in capital costs for system installations;
  $231,000 in annual water costs;
  $350,000 in annual maintenance costs;
  for a first year cost of $4,081,000.

Local Fields:

- 300 local fields would require manual or mechanical weed elimination at a total annual cost of $229,860.

In total, implementation costs to bring M-NCPPC fields into compliance (absent a total conversion to synthetic turf) would be:
Total first year costs to M-NCPPC would be $5,896,970.
Recurring annual costs for M-NCPPC would be $810,860.
Sod Replacement costs every two years would be $817,600.
Additional grading costs every four years for M-NCPPC would be $400,000.

3. **Revenue and expenditure estimates covering at least the next 6 fiscal years.**

Total conversion costs to allowable landscaping practices for the county would include an undetermined amount for MCPS to replace current pesticides in inventory and a six year

total of $12,804,070 for M-NCPPC as a part of converting maintenance practices on current fields to allowable practices under Bill 52-14.

M-NCPPC's six-year estimate of $12,804,070 in conversion costs consists of:
$5,896,970 in first year costs
$4,054,300 in subsequent annual expenses [$810,860 X 5 years]
$2,452,800 in sod replacement costs on athletic fields [$817,600 X 3 applications]
$400,000 in additional grading costs

If it is determined that a proactive enforcement effort is needed to enforce the bill, a dedicated inspector would be required at a personnel cost of $75,000 and a vehicle cost would of $40,000, for a total of $115,000 for the first year and a six year total of $490,000. The County Executive recommends a complaint-driven enforcement program.

Bill 52-14 also requires the County Executive to establish an awareness campaign related to the prohibitions noted in the bill. Costs related to the media campaign will depend on the scope and size of the media campaign. The County Executive recommends an education and outreach program of minimal cost to the county.

4. **An actuarial analysis through the entire amortization period for each bill that would affect retiree pension or group insurance costs.**

   Not Applicable.

5. **An estimate of expenditures related to County's information technology (IT) systems, including Enterprise Resource Planning (ERP) systems.**

   Not Applicable.

6. **Later actions that may affect future revenue and expenditures if the bill authorizes future spending.**

   Not Applicable.

7. **An estimate of the staff time needed to implement the bill.**

   The impact of implementation of Bill 52-14 on staff time will depend on the extent of the enforcement required for the provisions in the bill. Inspections on lawns, commercial sales establishments for signage, and other general enforcement actions will have an impact on various county departments similar to other countywide ban legislation.

   If Bill 52-14 requires an enforcement inspector, approximate personnel costs of an inspector would be $75,000 and a vehicle would be $40,000 for a total of $115,000 per inspector.

If enforcement of Bill 52-14 is complaint-driven, there would be an impact to current inspection operations by increasing the extent of some existing inspection protocols but would result in minimal fiscal impact to the county.

8. **An explanation of how the addition of new staff responsibilities would affect other duties.**

Depending on the enforcement model of Bill 52-14, the bill would impact the total number of inspection hours required. An inspector carrying out an inspection in a retailer for health code and other violations, for example, could be required to add on additional inspections for checks of signage and other sales requirements of pesticides to their normal inspection process.

9. **An estimate of costs when an additional appropriation is needed.**

There are three potential areas of cost related to Bill 52-14:

1) Conversion costs related to replacing old pesticides or converting contracts to include compliant pesticide application- County departments reported no fiscal impacts considering DGS already operates an IPM. MCPS reported that there would be costs associated with converting to approved pesticides from pesticides currently in use and that the extent of these conversion costs will not be known until a final list of banned pesticides has been established by DEP.

M-NCPPC estimates their conversion costs to allowable landscaping practices (excluding a conversion to artificial turf) to be $12,804,070 over the next six years. See item 3 for additional information on M-NCPPC's estimated conversion costs.

2) Costs associated with a media campaign-Bill 52-14 requires that the County Executive establish a media campaign to publicize the ban on certain non-essential pesticides. Costs related to this media campaign will vary depending on the scope and size of the campaign; and

3) Costs associated with enforcement of Bill 52-14-If dedicated enforcement personnel are needed to enforce the provisions of Bill 52-14, approximate personnel costs of an inspector would be $75,000 and a vehicle would be $40,000 for a total of $115,000 per inspector.

10. **A description of any variable that could affect revenue and cost estimates.**

See Item 9 above.

11. **Ranges of revenue or expenditures that are uncertain or difficult to project.**

M-NCPPC reports that loss of revenue is likely to occur if the spraying of certain non-essential pesticides prohibited in Bill 52-14 is eliminated as a part of the current playing field maintenance program. M-NCPPC reports that other jurisdictions have seen a loss of revenue from athletic tournaments leagues choose to take outside of the county.

12. If a bill is likely to have no fiscal impact, why that is the case.

Not Applicable.

13. Other fiscal impacts or comments.

Both M-NCPPC and the Department of Recreation (REC) are also concerned about how this prohibition will impact recreational and sport fields throughout the county. There are multiple jurisdictional studies suggesting a prohibition of this type on sport fields leads to degradation of the playing field and may lead to injury.

14. The following contributed to and concurred with this analysis:

Stan Edwards, Department of Environmental Protection
James Song, Montgomery County Public Schools
David Vismara, Maryland-National Capital Park and Planning Commission
Beryl Feinberg, Department of General Services
Matt Schaeffer, Office of Management and Budget

Jennifer A. Hughes

Jennifer A. Hughes, Director
Office of Management and Budget

1/26/15
Date

Economic Impact Statement
Bill 52-14, Pesticides – Notice Requirements - Non-Essential Prohibitions

Background:

This legislation would require the posting of a notice when a property owner applies a pesticide to an area of lawn more than 100 square feet. Bill 52-14 requires the County Executive to designate a list of "non-essential" pesticides that include the following:

- All pesticides classified as "Carcinogenic to Humans" or "Likely to Be Carcinogenic to Humans" by the United States Environmental Protection Agency (USEPA);
- All pesticides classified by USEPA as "Restricted Use Products";
- All pesticides classified as "Class 9" by the Ministry of the Environment and Climate Change, Government of Ontario, Canada
- All pesticides classified as "Category 1 Endocrine Disrupters" by the European Commission; and
- Other pesticides which the County Executive determines are not critical to pest management in the County.

The Bill would prohibit the application of non-essential pesticides to lawns, with exceptions for noxious weed and invasive species control, agriculture and gardens, and golf courses. The Bill would also require the County Executive to conduct a public outreach and education campaign during the implementation of Bill 52-14, and would prohibit the application of non-essential and neonicotinoid pesticides to County-owned property.

1. **The sources of information, assumptions, and methodologies used.**

   Department of Environmental Protection (DEP)
   SafeLawns.org
   Diffen.org
   The Fertilizer Institute (TFI)
   Grassroots Environmental Education

2. **A description of any variable that could affect the economic impact estimates.**

   The variable that could affect the economic impact estimates is the cost differential between organic pesticides and chemical pesticides. However, according to SafeLawns.org, the cost differential is comparing apples to oranges since one product provides a short-term solution while the other product aims to provide a long-term solution. Organic products "function by building up life in the soil (soil biology) and their payoff is long-term and lasting" while synthetic products, which are instantaneous, are applied frequently and in greater amounts. Therefore, SafeLawns.org indicates that the users of organic products will spend less money on lawn care over a two-year period than users of chemical or synthetic pesticides.

Economic Impact Statement
Bill 52-14, Pesticides – Notice Requirements - Non-Essential Prohibitions

According to Diffen.org, organic pesticides are much more expensive than synthetic or chemical pesticides because synthetic or chemical pesticides have more concentrated levels of nutrients per weight of product than organic pesticides. The user of organic pesticides needs several pounds of organic pesticide that would provide the same nutrient levels as synthetic or chemical pesticide. That differential in the amounts would result in a higher cost of organic pesticide.

Therefore, there is a conflict between the information provided by SafeLawns.org and Diffen.org regarding the cost differential between organic and synthetic/chemical pesticides. SafeLawns.org suggests there is less application of organic to synthetic/chemical pesticide while according to Diffen.org, one needs a higher quantity of organic pesticide to synthetic/chemical pesticide to achieve the same nutrient level.

3. **The Bill's positive or negative effect, if any on employment, spending, saving, investment, incomes, and property values in the County.**

   Because of the differences of opinions in terms of the amount of application of organic versus synthetic/chemical pesticide as stated in paragraph #2, it is uncertain whether Bill 52-14 would have economic impact on employment, spending, saving, investment, incomes, and property values in the County. Because of the specific climate and soil type endemic to Montgomery County, more consultation with the experts and research are needed to determine the economic effect on the County.

4. **If a Bill is likely to have no economic impact, why is that the case?**

   It is uncertain if Bill 52-14 has an economic impact.

5. **The following contributed to or concurred with this analysis**: David Platt and Rob Hagedoorn, Finance, and Stan Edwards, Department of Environmental Protection.

Joseph E. Beach, Director
Department of Finance

1/23/15
Date

Chapter 33B. Pesticides. [Note](1)

§ 33B-1. Definitions.

§ 33B-2. Notice about pesticides to customer.

§ 33B-3. Posting signs after application.

§ 33B-4. Signs with retail purchase of pesticide.

§ 33B-5. Storage and handling of pesticides.

§ 33B-6. Regulations.

§ 33B-7. Penalty for violating chapter.

Sec. 33B-1. Definitions.

In this chapter:

Custom applicator means a person engaged in the business of applying pesticides.

Department means the department of environmental protection.

Director means Director of the Department of Environmental Protection, or the Director's designee.

Lawn means an area of land, except agricultural land, that is:

(1) Mostly covered by grass, other similar herbaceous plants, shrubs, or trees; and

(2) Kept trim by mowing or cutting.

Pest means an insect, snail, slug, rodent, nematode, fungus, weed, or other form of plant or animal life or microorganism (except a microorganism on or in a living human or animal) that is normally considered to be a pest or defined as a pest by applicable state regulations.

Pesticide means a substance or mixture of substances intended or used to:

(1) prevent, destroy, repel, or mitigate any pest;

(2) be used as a plant regulator, defoliant, or desiccant; or

(3) be used as a spray adjuvant, such as a wetting agent or adhesive.

However, pesticide does not include an antimicrobial agent, such as a disinfectant, sanitizer, or deodorizer, used for cleaning that is not considered a pesticide under any federal or state law or regulation. (1986 L.M.C., ch. 38, § 1; 2000 L.M.C., ch. 34; § 1.)

Sec. 33B-2. Notice about pesticides to customer.

(a) In this section:

(1) *Customer* means a person who makes a contract with a custom applicator to have the custom applicator apply a pesticide to a lawn.

(2) *New customer* includes a customer who renews a contract with a custom applicator.

(b) A custom applicator must give to a new customer:

(1) Before application, a list of:

a. The trade name of each pesticide that might be used;

b. The generic name of each pesticide that might be used; and

c. Specific customer safety precautions for each pesticide that might be used; and

(2) After application, a list of:

a. The trade name of each pesticide actually used; and

b. The generic name of each pesticide actually used; and

(3) A written notice about pesticides prepared by the department under subsection (c) of this section.

(c) The department must prepare, keep current, and provide to a custom applicator a written notice about pesticides for the custom applicator to give to a customer under subsection (b) of this section.

(d) The notice prepared by the department under subsection (c) of this section must include:

(1) Government agency phone numbers to call to:

a. Make a consumer complaint;

b. Receive technical information on pesticides; and

c. Get assistance in the case of a medical emergency;

(2) A list of general safety precautions a customer should take when a lawn is treated with a pesticide;

(3) A statement that a custom applicator must:

a. Be licensed by the Maryland Department of Agriculture; and

b. Follow safety precautions; and

(4) A statement that the customer has the right to require the custom applicator to notify the customer before each treatment of the lawn of the customer with a pesticide. (1986 L.M.C., ch. 38, § 1.)

Sec. 33B-3. Posting signs after application.

(a) Immediately after a custom applicator treats a lawn with a pesticide, the custom applicator must post a sign on the lawn.

(b) A sign posted under this section must:

(1) Be clearly visible from the principal place of access to the property;

(2) Be a size, form, and color approved by the department;

(3) Be made of material approved by the department; and

(4) Have wording with content and dimensions approved by the department. (1986 L.M.C., ch. 38, § 1.)

Sec. 33B-4. Signs with retail purchase of pesticide.

A person who sells at retail a pesticide or material that contains a pesticide must make available to a person who buys the pesticide or material that contains a pesticide:

(a) Notice signs and supporting information that are approved by the department; and

(b) The product label or other information that the federal Insecticide, Fungicide, and Rodenticide Act, 7 U.S.C. 136 et seq., requires for sale of the pesticide. (1986 L.M.C., ch. 38, § 1.)

Sec. 33B-5. Storage and handling of pesticides.

Any person who sells at retail a pesticide or material that contains a pesticide must:

(a) transport, display, and store each pesticide in a secure, properly labeled container that resists breakage and leakage, and promptly clean up and either repackage or properly dispose of any pesticide that escapes from its container;

(b) display and store each pesticide separately from any food, medicine, or other product that a human being or animal may ingest;

(c) transport each pesticide separately from any food, medicine, or other product that a human being or animal may ingest unless the pesticide is in a secure container that resists breakage and leakage; and

(d) offer to each buyer of a pesticide materials approved or distributed by the Department that:

(1) explain the dangers of contamination that may occur from pesticide use; and

(2) inform buyers of the availability of alternative products.

The Department, the Health and Human Services Department, and any other agency designated by the County Executive, must enforce this Section. (2000 L.M.C., ch. 34, § 1.)

Sec. 33B-6. Regulations.

(a) The County Executive must adopt regulations to carry out this Chapter under method (2).

(b) The Executive must include in the regulations adopted under this section the minimum size or quantity of pesticide subject to section 33B-4. (1986 L.M.C., ch. 38, § 1; 2000 L.M.C., ch. 34, § 1.)

Note—Formerly, § 33B-5.

Sec. 33B-7. Penalty for violating chapter.

(a) Any violation of this Chapter is a class C violation.

(b) Each day a violation continues is a separate offense. (1986 L.M.C., ch. 38, § 1; 2000 L.M.C., ch. 34, § 1.)

Note—Formerly, § 33B-6.

Endnotes

1 (Popup - Popup)

***Editor's note**-Chapter 33B, "Pesticides," was held unconstitutional due to conflict with Federal legislation (Federal Insecticide, Fungicide and Rodenticide Act, 'FIFRA,' 7 USC 136 et seq.) which the court ruled preempted local legislation. Maryland Pest Control Ass'n. v. Montgomery County, Maryland, 646 F. Supp. 109 (D.Md., 1986), aff'd on appeal, 822 F.2d 55 (1987). Based on a later Supreme Court decision, Wisconsin Public Intervenor v. Mortier, 115 L.Ed.2d 532, 111 S.Ct. 2476 (1991), the ruling of the District Court in Civil No. JFM-86- 1688 was rescinded on April 3, 1992, and is no longer in effect.

Amendments in 1987 to State law (MD. AGRIC. CODE ANN., § 5-201, et seq.,) enacted provisions similar to those in County law. State legislation introduced in 1993 (SB 429) which would have restricted the right of local jurisdictions to regulate application of pesticides did not pass, leaving ch. 33B of the Montgomery County Code still in effect.

CHAPTER 33B. PESTICIDES — REGULATIONS

COMCOR 33B.00.01 Pesticides

COMCOR 33B.00.01 Pesticides

33B.00.01.01 General Provisions

A. Authority. In accordance with the authority conferred under Chapter 33B, Section 33B-6, of the Montgomery County Code, 1994, as amended (hereinafter referred to as the "Code"), the County Executive hereby promulgates this regulation to implement County law pertaining to public education and safety measures required of retail sellers of pesticides as set forth in Chapter 33B of the Code.

B. Applicability. This regulation applies to all pesticide retailers that are subject to Chapter 33B of the Code. Definitions

The definitions of the terms used in this regulation are provided in Chapter 33B, Section 33B-1, of the Code. For purposes of this regulation, the following additional words and phrases will have the meaning respectively ascribed to them in this regulation:

Director of Environmental Protection - The Director of the Montgomery County Department of Environmental Protection or the Director's designee.

Director of Health and Human Services - The Director of the Montgomery County Department of Health and Human Services or the Director's designee.

Food Service Facility - Any enterprise that prepares or sells food or drink for human consumption on or off the premises. Food service facility includes any restaurant, coffee shop, retail market, cafeteria, short-order café, luncheonette, tavern, sandwich stand, soda fountain; and any food service facility in an industry, institution, hospital, club, school, church, catering kitchen, or camp.

General Use Pesticide - Any pesticide classified by the U.S. Environmental Protection Agency (EPA) as a general use pesticide. General use pesticide includes any pesticide product or ingredient not listed in the EPA's Restricted Use Products Report.

Non-bulk Pesticides - Any pesticide distributed, sold, offered for sale, packaged, or repackaged in containers designed for less than 10 gallons of liquid or less than 56 pounds of dry weight.

Pesticide Producer Establishment - Any place assigned an establishment number by the U.S. Environmental Protection Agency where a pesticide or device or active ingredient

used in creating a pesticide is produced, or held, for distribution or sale.

Pesticide Retailer - A person that sells at retail non-bulk pesticides or non-bulk quantities of a material that contains a pesticide.

Properly Labeled - The written, printed, or graphic matter that appears on or is attached to a pesticide, or its immediate container, and the outside container or wrapper of any retail package of pesticide contains sufficient instructions for use and caution to satisfy the requirements of state and federal pesticide labeling laws.

33B.00.01.02 Display and Storage of Pesticides

A. A pesticide retailer must ensure that all pesticides, whenever displayed or stored in a retail establishment, are physically separated from food, medicine, beverages, or feed. The retailer must display or store the pesticides across the aisle from any food, medicine, beverages, or feed or place a solid, nonporous, physical barrier between those products and any pesticide. The retailer must take other reasonable precautions if necessary to prevent a pesticide from contaminating any product that is likely to be ingested by a human or a domestic animal. Reasonable precautions may include storing pesticide products in a locked container.

B. A pesticide display or storage area must contain only pesticide containers that are properly labeled and are free of leaks, cracks, tears, or open seams.

33B.00.01.03 Pesticide Spills

A. A pesticide retailer must promptly clean up any spilled pesticide product upon discovery of the spill.

B. Disposal

1. A pesticide retailer must not dispose of a pesticide that escapes from its container or packaging except in accordance with applicable state and federal laws.

2. A pesticide retailer must not dispose of a pesticide by discharging or dumping the pesticide or the pesticide container or packaging into a sewer, ditch, lake, or any other area that may release the pesticide into ground or surface waters.

C. Repackaging

1. A pesticide retailer may repackage a pesticide that escapes from its container for return to the distributor of that product if:

a. the retailer has an agreement that provides for the return of spilled pesticides; and

b. the procedures used by the retailer to prepare the product

for its return to the distributor comply with applicable state and federal laws.

2. A pesticide retailer must not repackage a pesticide for sale to customers unless the retailer is registered with the U.S. Environmental Protection Agency as a pesticide producer establishment.

33B.00.01.05 Inspections

A. The Director of Health and Human Services must routinely inspect food service facilities for compliance with County pesticide laws in the regular course of performing any food safety inspection required under Chapter 15 of the Code.

B. The Director of Environmental Protection must investigate each complaint alleging a violation of County pesticide laws by a retailer other than a food service facility and may conduct any other on-site visit necessary to achieve compliance with the laws.

33B.00.01.06 Public Education

A. A pesticide retailer must make written materials on general pesticide use and safety available to each purchaser of non-bulk pesticides at each site where pesticides are available for purchase. Notice of the availability of the written materials must be prominently displayed at those sites in a conspicuous place as near to the point of sale as practicable.

B. Although the written materials displayed under this Section need not be product-specific, the materials must:

1. be obtained from or have the prior approval of the Department of Environmental Protection; and

2. include information that advises the general public about opportunities for consumers to consider recommended alternative pest control measures.

33B.00.01.07 Severability

If a court holds that a portion of this regulation is invalid, the other portions remain in effect.

33B.00.01.08 Effective Date

This regulation takes effect 30 days after approval by the County Council.

(Administrative History: Reg. No. 32-01AMII (Method 2); Dept.: Environmental Protection and Health and Human Services)

BILL 52-14

5013554

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cc
SBF
LL

Maryland
Department of Agriculture

Agriculture | *Maryland's Leading Industry*

Office of the Secretary

Larry Hogan, Governor
Boyd Rutherford, Lt. Governor
Joe Bartenfelder, Acting Secretary
Mary Ellen Setting, Deputy Secretary

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February 6, 2015

RECEIVED
MONTGOMERY COUNTY
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2015 FEB 12 AM 8:56

Montgomery County Council
100 Maryland Avenue
Rockville, MD 20850

Dear Council Member:

Following up on previous correspondence regarding Montgomery County Council Bill No. 52-14 (Non-Essential Pesticides – Prohibition), I would like to emphasize some of our concerns.

At the Maryland Department of Agriculture (MDA), the Pesticide Regulation Section has delegated authority from EPA to enforce the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) in the state and also enforces the Maryland Pesticide Applicator Law, which is more restrictive than FIFRA. Our program is active and effective. Also at MDA the State Chemist Section registers all pesticide products at the state level. It is a violation of state and federal law for a person to use a pesticide product in the state that isn't registered, or in a manner inconsistent with the EPA approved label. In 2014, MDA staff registered a total of more than 12,000 pesticide products, conducted more than 2,000 inspections, and investigated 40 complaints. Nearly 500 businesses were cited for violations, and eleven civil penalties were assessed totaling more than $11,000. MDA staff conduct training, testing, licensing, certification and registration of more than 15, 000 applicators across the state.

One of our concerns with this bill is that with two levels of regulation already in place, it will cause confusion for commercial applicators as well as County residents, and compromise compliance with existing law. MDA, backed by EPA, presents a single face to pesticide enforcement. We want everyone with a concern about pesticide use or misuse in the state to report it to our Pesticide Regulation Section. MDA has the ultimate authority to revoke a pesticide license in the case of the most serious violations.

We are also concerned that there will be an increase in do-it-yourself pest control by untrained, unlicensed consumers. It is illegal for a professional to apply pesticides if they are not licensed or certified by MDA according to strict criteria. We are well engaged with these professional applicators, but untrained consumers who buy products off the shelf or mix up homemade pest control remedies are of great concern to us, as they can pose risks of their own and may unknowingly violate state and federal law.

I hope that we can work together with the County to best reach those that are not directly regulated by MDA to improve overall understanding by the public of how to properly use pesticide and to adopt IPM strategies, and in finding and reporting any potential violations of existing law. Please contact Carol Holko, Assistant Secretary, at carol.holko@maryland.gov or 410.841.5870 for additional information or with questions.

Thank you for your consideration.

Sincerely,

Joseph Bartenfelder

Joseph Bartenfelder
Acting Secretary

c: The Honorable Ike Leggett, Montgomery County Executive
The Honorable George Leventhal, President
The Honorable Nancy Floreen, Vice President
The Honorable Roger Berliner
The Honorable Marc Elrich
The Honorable Tom Hucker
The Honorable Sidney Katz
The Honorable Nancy Navarro
The Honorable Craig Rice
The Honorable Hans Riemer

AgBrief

Pesticide Regulation

Enforcing the Maryland Pesticide Applicator's Law

February 2013

Each year, many homes in Maryland are infested by termites, ants, bees and mice that threaten human health and the structural integrity of houses. Each year, invasive pests and plants threaten, damage, even destroy fruit and vegetable crops, trees and forests, ornamentals, and others plants and greenery. Each year, people and pets are sickened, even killed, by pest-borne diseases and allergies. Pesticides, when handled and applied properly, help reduce these risks. Pesticides, whether organic or traditional, can also be dangerous and toxic if applied irresponsibly or incorrectly.

The Maryland Pesticide Applicator's Law dictates how pesticides are sold, handled, stored, applied and disposed. The Maryland Department of Agriculture (MDA) Pesticide Regulation Section administers and enforces this law.

Any person or business that applies pesticides commercially – that is, anyone who gets paid to handle pesticides – must be trained, certified and registered with MDA to help ensure that only knowledgeable, competent individuals handle and apply pesticides.

Licensing, Permitting & Certification

Generally, businesses are "licensed," public agencies are "permitted" and individuals are "certified." However, before a pesticide business can be licensed or a public agency can be permitted, they must have at least one certified applicator on staff.

Training & Testing

MDA provides and approves training courses for pesticide applicators to ensure they understand and follow prescribed practices. Trainees must pass an examination before being certified. All course dates and locations are on the MDA website. All applicators must attend an approved training to be recertified periodically.

There are two broad classifications of pesticides

1. **General use pesticides** may be purchased at a retail outlet and used by the general public.
2. **Restricted use pesticides** can only be applied by (or under the direct supervision of) a certified applicator.

MDA certifies two types of pesticide applicators

1. **Private applicators** apply restricted use pesticides to their own or rented land to produce agricultural commodities. There were 3,252 certified private applicators in Maryland during FY 2012.
2. **Commercial applicators** apply general and restricted use pesticides as employees of licensed pest control businesses or public agencies. There were 7,971 commercial applicators associated with 1,755 commercial businesses and 325 agencies during FY 2012.

Applicator Reporting

All certified applicators must keep records of every pesticide application they make. That record must include the name and registration number of the pesticide, the mixing (or dilution) rate, the name of the applicator, and the location, date, time, and weather conditions of the application. By law, those records must be made available to MDA immediately upon request.

MDA issues permits to dealers who sell or distribute restricted use pesticides. Dealers, too, must keep records of every sale of restricted use pesticides. Those records include the name of the pesticide, the buyer (who the dealer must ensure is certified with MDA), and the quantity purchased. Again, those records must be made available to MDA immediately upon request. There were 148 dealers in Maryland during FY 2012.

-- Over --

Pesticide Use Survey

MDA periodically contracts with the U.S. Department of Agriculture National Agricultural Statistics Service to conduct a Pesticide Use Survey. The survey provides researchers and public policy leaders with broad information about pesticide usage in the state. The last survey, which had a 51 percent response rate, was completed in 2004. A new survey was undertaken during 2012. Results are expected this spring. The 2004 survey can be found at: http://mda.maryland.gov/Documents/2004_pesticide_use_survey.pdf

Check the MDA Databases

MDA has three online, searchable databases to help consumers ensure they do business with appropriately registered professionals. The databases can be accessed for free at: http://mda.maryland.gov/plants-pests/Pages/pesticide_db.aspx

- **The business license database** contains all businesses that are licensed to apply pesticides commercially. Consumers can search by company name, business license number or business license category.
- **The certified pesticide applicator database** contains the name of all certified individual applicators, searchable by last name, applicator number, category or location.
- **The pesticide database** -- which includes all registered pesticides that can be legally sold, distributed and used in Maryland -- provides registration data submitted by companies that sell their products in Maryland, combined with ingredients, pest and site data from the U.S. Environmental Protection Agency.

Pesticide Sensitive Individuals (PSI)

Since 1989, MDA has required licensees and permittees in the turf and ornamental pest control categories, to notify PSIs before applying a registered pesticide to properties that are contiguous or adjacent to the PSI. Although there is no fee to get on the PSI list, a physician must document that the person should not be exposed to pesticides because they have a sensitivity or diagnosed condition. MDA provides all businesses and public agencies that perform ornamental and turf pest control with the list every February and an updated list every summer. (MDA informs the relevant companies when people are added between updates.) Companies are required to notify people on the list prior to application, preferably the day before but no later than the morning of application. There were 167 people on the PSI list at the end of FY 2012.

The PSI application is available at: www.mda.maryland.gov/plants-pests/Documents/sensitiv.pdf

Enforcement

The goal of MDA's Pesticide Regulation Section is to ensure that knowledgeable and well-trained applicators use pesticides for an appropriate purpose, apply them according to label instructions, and dispose of them properly.

MDA inspects every commercial business, public agency, restricted use dealer and farmer certified to apply restricted use pesticides every 12 to 18 months. MDA also inspects locations where pesticides are manufacturered.

Inspections and Investigations: MDA cooperates with other state and federal agencies to protect public health and the environment. Inspections include a review of pesticide application, sales and training records, and safety equipment. Inspectors also observe actual pesticide applications to ensure compliance with state and federal regulations. MDA also investigates pesticide accidents, suspected misuse of pesticides and consumer complaints.

After an inspection or investigation, enforcement actions by MDA can range from a letter of reprimand to assessing civil penalties to bringing criminal charges.

In 2012, MDA performed 750 routine business inspections during which 222 businesses were cited for violations of the Pesticide Applicators Law and Regulations. Those violations are included in the agency's annual report.

Special Programs

- **Pesticide Container Recycling Program**: Between June and September 2012, MDA collected 43,050 pesticide containers weighing 39,000 pounds from 22 locations in six counties, and transported them to a plastic recycling facility.
- **Pesticide Disposal Program:** During FY 2012, MDA collected 17,866 pounds of unwanted or unusable pesticides from 54 farmers and growers in 16 counties.

For more information

See the MDA website for more comprehensive information about Pesticide Regulation: www.mda.maryland.gov/plants-pests/Pages/pesticide_regulation.aspx

Maryland Department of Agriculture
50 Harry S. Truman Parkway
Annapolis, Maryland 21114
www.mda.maryland.gov
mda.news@maryland.gov

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Pesticide Regulation

- Pesticide Regulation Home
- Searchable Pesticide Database
- Pesticide Information for Consumers
- Pesticide Information for Professionals
- Integrated Pest Management (IPM) in Schools
- Pesticide Applicator Certification and Business Licensing Requirements

Pest Control & Pesticide Information for Homeowners

The Maryland Department of Agriculture (MDA), in conjunction with the University of Maryland Cooperative Extension Service (MCES) and the

Always Identify the Pest Before Selecting the Type Of Pest Control

Governor's Pesticide Council, developed a series of brochures to assist individuals to make informed decisions on their pest control needs. The brochures provide basic information on pest control and pesticide use. There currently are four brochures available for homeowners. The first brochure "Controlling Pests - Help for the Consumer" provides background information on pest control and pesticides. The second brochure "Lawn Care Tips - Help for the Consumer" highlights some of the key issues associated with pest control and pesticide use for lawn care, with emphasis on the use of integrated pest management (IPM). The third brochure "Proper Pesticide Use - Help for the Consumer" addresses the fundamentals of proper pesticide use, and the final brochure "Using IPM - Help for the Consumer" further addresses the topic of IPM and how it is used in pest control.

The major goals of the brochures are to help consumers understand:

Always Identify the Pest Before Selecting the Type Of Pest Control

- What steps to take to control pests.
- What methods of pest control are available, including pest prevention and non-chemical control.
- How to choose pesticides and how to use, store, and dispose of pesticides properly.

Plants, insects, rodents, diseases and other organisms are a natural part of the environment. When they invade an area in which they are not wanted, they are considered to be "pests". However, there are many times that a perceived pest is actually a beneficial organism and does not need to be controlled. Termites, fleas, cockroaches, and ants are common insects often found inside dwellings and are considered pests. Weeds, potato beetles, aphids, and grubs are some of the pests associated with gardens, lawns and landscape plantings. Some of these pests can also pose health hazards to you, your family, or pets. As a result, it is very important that you know and understand the pest problem in order to choose the best pest control option, or options, that are available to you. Knowing and selecting the proper options for your particular pest situation is the key to effective, long term pest control. These options include the use of pest prevenion, non-chemical pest control, and/or the use of pesticides. Often times the most effective pest control strategy is to combine several methods of pest control. This approach is known as integrated pest management (IPM). One of the best and easiest forms of pest control is to prevent the situation from actually becoming a pest problem. No matter what pest control option you choose, it is important to follow these steps:

- Identify the pest problem
- Decide what level of pest control is necessary
- Choose the appropriate pest control option or options
- Evaluate the result to determine its effectiveness.

The brochures are designed to help the homeowner make better informed decisions regarding pest control and the use of pesticides. They also encourage individuals to seek further assistance from the

MCES Home and Garden Information Center to effectively control their pest problems. The Home and Garden Information Center is staffed with experts who can provide detailed information on lawn, tree, shrub, houseplant, and structural pests and their control. This information includes: the identification of insects, diseases, weeds, and other pests; available pest control methods, including pesticides and alternatives; choosing pest resistant plant varieties; and integrated pest management programs. Calls are answered by consultants between 8:00 a.m. and 1:00 p.m. Monday through Friday. Message tapes are also set up to provide callers with information 24 hours a day. The Home and Garden Information Center can be reached by calling (1-800342-2507. Copies of MCES publications and information sheets are also available through the Home and Garden Information Center.

COMMON MEASUREMENTS	
1 Gallon (gal.)	= 128 fluid ounces (fl. oz.) = 16 cups = 8 pints = 4 quarts
1 Quart (qt.)	= 32 fl. oz. = 4 cups = 2 pts.
1 Pint (pt.)	= 16 fl. oz. = 2 cups
1 Cup	= 8 fl. oz.
1 Tablespoon	= 1/2 fl. oz. = 3 teaspoons
1 Teaspoon	= 1/6 fl. oz.
1 fl. oz.	= 6 teaspoons = 2 tablespoons
1 Yard (yd.)	= 3 feet (ft.)
Square Feet (sq. ft.)	= length (ft) x width (ft.)

Nurseries, garden centers and pesticide retail stores are the primary distribution points that MDA is targeting for distribution of the brochures. If you would like to obtain copies of the brochures, contact the Maryland Department of Agriculture, Pesticide Regulation Section, 50 Harry S. Truman Parkway, Annapolis, Maryland 21401, or by calling (410)841-5710.

Remember, if you choose to use a pesticide for your pest control needs, it is important that you mix and use the proper amount of pesticide as specified on the product label. Always read and follow label directions. The chart above provides some of the common measurements that may be needed in the mixing and application of pesticides.

Send E-mail to Dennis Howard

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- Pesticide Information for Professionals
- Integrated Pest Management (IPM) in Schools
- Pesticide Applicator Certification and Business Licensing Requirements

Pesticides and Child Safety

Although pesticides can be beneficial to society, they can be dangerous if used carelessly or if they are not stored properly and out of the reach of children. According to data collected from the

American Association of Poison Control Centers, in 1993 alone, an estimated 80,000 children were involved in common household pesticide-related poisonings or exposures in the United States.

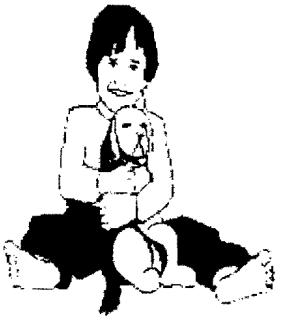
A survey by the U.S. Environmental Protection Agency regarding pesticides used in and around the home revealed some significant findings:

- Almost half, 47%, of all households with children under the age of five had at least one pesticide stored in an unlocked cabinet, less than 4 feet off the ground (i.e., within the reach of children).
- Approximately 75% of households without children under the age of five also stored pesticides in an unlocked cabinet, less than 4 feet off the ground (i.e., within the reach of children). This number is especially significant because 13% of all pesticide poisoning incidents occur in homes other than the child's home.

Bathrooms and kitchens were cited as the areas in the home most likely to have improperly stored pesticides. Examples of some common household pesticides found in bathrooms and kitchens include roach sprays; chlorine bleach; kitchen and bath disinfectants; rat poison; insect and wasp sprays, repellents and baits; and, flea and tick shampoos and dips for pets. Other household pesticides include swimming pool chemicals and weed killers.

EPA has important regulatory authority over pesticides in the United States under the pesticide law known as the Federal Insecticide, Fungicide, and Rodenticide Act, or FIFRA. Since 1981, the law has required most residential-use pesticides with a signal word of "Danger" or "Warning" to be in child-resistant packaging. These are the pesticides which are most toxic to children. Child-resistant packaging is designed to prevent most children under the age of five from gaining access to the pesticide, or at least delay their access. However, individuals must also take precautions to protect children from accidental pesticide poisonings or exposures.

RECOMMENDATIONS FOR PREVENTING ACCIDENTAL EXPOSURE OR POISONING:



- **Always** store pesticides away from children's reach in a locked cabinet or garden shed. Child-proof safety latches may also be installed on cabinets and can be purchased at your local hardware store;
- **Always** read the label first and follow the directions to the letter, including all precautions and restrictions;
- Before applying pesticides (indoors or outdoors), **always** remove children and their toys as well as pets from the area and keep them away until the pesticide has dried or as long as is recommended by the label;
- If your use of a pesticide is interrupted (perhaps by a phone call), **always** make sure to leave the container out of the reach of children while you are gone;
- **Never** transfer pesticides to other containers that children may associate with food or drink;
- Use child-resistant packaging properly by **always** closing the container tightly after use;

Never place rodent or insect baits where small children can get to them;
Alert others to the potential hazard of pesticides, especially caregivers and grandparents;
Teach children that "pesticides are poisons" and something they should not touch;
Keep the telephone number of your area Poison Control Center near your telephone. **(National Poison Center - 1-800-222-1222)**

IN CASE OF AN EMERGENCY, try to determine what the child was exposed to and what part of the body was affected before you take action, since taking the right action is as important as taking immediate action. The pesticide product label provides you with a "Statement of Treatment" to follow in emergencies. Administer the indicated initial first aid; then contact your local Poison Control Center, physician, local emergency number (911 in most areas), or the operator.

The following require immediate attention before calling for assistance - remember, act fast because speed is crucial:

- **Swallowed Pesticide** - Induce vomiting ONLY if the emergency personnel on the phone tell you to do so. It will depend on what the child has swallowed; some petroleum products or caustic poisons will cause more damage if the child is made to vomit. Always keep Syrup of Ipecac on hand (one ounce for each child in the household) to use to induce vomiting if recommended by the emergency personnel. Be sure the date on the product is current.

- **Pesticide In Eye** - Eye membranes absorb pesticides faster than any other external part of the body; eye damage can occur in a few minutes with some types of pesticides. If pesticide splashes into an eye, hold the eyelid open and wash quickly and gently with clean running water from the tap or a gentle stream from a hose for at least 15 minutes. If possible, have someone else contact a Poison Control Center for you while the victim is being treated. Do not use eye drops or chemicals or drugs in the wash water.

- **Pesticide On Skin** - If pesticide splashes on the skin, drench area with water and remove contaminated clothing. Wash skin and hair thoroughly with soap and water. Later, discard contaminated clothing or thoroughly wash it separately from other laundry.

- **Inhaled Pesticide** - Carry or drag victim to fresh air immediately. (If proper protection is unavailable to you, call for emergency equipment from the Fire Department.) Loosen victim's tight clothing. If the victim's skin is blue or the victim has stopped breathing, give artificial respiration and call rescue service for help. Open doors and windows so no one else will be poisoned by fumes.

Additional pesticide product information can be obtained from the **National Pesticide Telecommunications Network (NPTN) at 1-800-858-7378**. NPTN is a toll-free information service funded by EPA and operated by the Oregon State University Monday through Friday 9:30 a.m. - 7:30 p.m. Eastern Standard Time.

AGRICULTURE

Enter search term

Plants/Pests

- Plants/Pests Home
- Regulatory Information Center
- Emerging Invasive Plant Pests
- Forest Pest Management
- Mosquito Control
- Pesticide Regulation
- Plant Protection and Weed Management
- State Chemist
- Turf and Seed

Pesticide Regulation

- Pesticide Regulation Home
- Searchable Pesticide Database
- Pesticide Information for Consumers
- Pesticide Information for Professionals
- Integrated Pest Management (IPM) in Schools
- Pesticide Applicator Certification and Business Licensing Requirements

Citizen's Guide to Pesticide Enforcement/Complaints

To ensure pesticides are applied properly by trained and competent applicators, the Maryland Department of Agriculture (MDA) Pesticide Regulation Section (PRS):

- regulates the distribution, sale, storage, use and disposal of pesticides; and,
- establishes qualifications for the licensing of businesses and certification of persons applying or recommending pesticides or perfoming pest inspections,

MDA does not regulate matters involving contractual disputes between consumers and pest control firms.

Authority to Inspect Land, Facilities or Equipment

The Agriculture Article, Section 5-205, states that the Secretary of Agriculture may sample any pesticide and inspect any device, container, product, apparatus or equipment used or intended for use in pest control operations, any establishment from which pest control is conducted, and any pesticide applicationor treatment performed by or under the supervision of a certified applicator.

In addition, Section 15.05.01.18 A (12) of the Regulations Pertaining to the Pesticide Applicators Law states that failing to allow the Department to inspect or sample as provided in the law is grounds for issuing a civil penalty or suspending, revoking or denying a license, permit or certificate of any person.

Role of the Pesticide Inspector

- Contact all pertinent individuals to conduct investigations and/or interviews.
- Document incident information through maps, photographs, statements, pesticide labels, and on-site assessments, which are compiled into an investigative file.
- Collect physical evidence such as soil, vegetation, and water samples to aid in the determination of violations.
- Deliver samples to MDA's State Chemist Section to be analyzed for pesticide residues or to the Plant Protection Section for detection of disease, insect or pesticide damage.

Access to Information Gathered in an Investigation?

The Maryland Public Information Act, Maryland State Government Article10-611, et seq., gives the public the right to view case files once an investigation is closed. Therefore, information contained in a closed case file must be released to the public, upon written request.

Investigation Process

The goal of the Pesticide Regulation Section is to respond to the complainant within 24 hours of learning of an incident. However, there are many variables involved in an investigation that determine how long it will take to close a case. Some of the factors that delay the closure are complex lab analyses, the need to have a follow-up investigation, and backlog of legal cases under review. Because each case varies, it is impossible to provide a good estimate of how long it will take to achieve case closure. The top priority is to provide a complete and thorough investigation of any complaint.

After the inspector has gathered all of the necessary information related to an investigation, a Supervising Inspector reviews the file to determine the completeness of the documentation. After the initial review by the Supervising Inspector, the file is reviewed by the Case Review Officer for possible violations of the Maryland Pesticide Applicators Law or regulations. Once this review is complete, the staff forwards the case to the Chief of the Section for review for possible regulatory action.

In some instances no violations are noted and a report of findings is issued. However, if evidence is sufficient to prove a significant violation of the law, the Department can resolve the conflict by taking any of the following actions against the pest control business or pesticide applicator:

- Notice of warning
- Negotiated settlement agreement,
- Informal conference before the Chief of the Pesticide Regulation Section,
- Formal hearing before the Office of Administrative Hearings,
- Criminal action in court, or
- Civil penalty.

Information regarding laboratory analysis performed on samples collected from a property can be shared with the owner while an investigation is underway, but some information can not be released until a case is closed. This is to insure that decisions on any potential regulatory action are not prejudicial.

Penalties For Violating The Maryland Pesticide Applicators Law or Regulations

Any person who violates any provision of the Pesticide Applicators Law is guilty of a misdemeanor and, upon conviction, is subject to a fine of up to $1,000, or imprisonment not exceeding 60 days, or both.

In addition, a person who violates any provision of the law or regulations is subject to a civil penalty of not more than $2,500 for a first violation or $5,000 for each subsequent violation. Each day a violation occurs can be considered a separate violation. The total penalties imposed on a person for violations that result from the same set of facts and circumstances may not exceed $25,000. Violation of the law and regulations is also grounds for revocation, suspension or denial of a license, permit or certificate issued by MDA.

The Maryland Pesticide Applicators Law contains no provision for compensation to be made to individuals. All civil penalties collected by MDA go into the State's General Fund and are not used by MDA. Private civil action would be required in most instances to recover damages.

The Pesticide Regulation Section will provide notification when a decision has been made as to whether further regulatory action will be taken, and of any final action taken. Parties directly involved in a complaint can receive information by contacting the Pesticide Regulation Section at 410-841-5710.

MDA's function is to ensure compliance with the State pesticide law and to deal with the licensed firm or certified applicator, which is regulated by MDA. MDA does not assist the complainant with preparing a civil suit against the pest control firm or applicator.

For information about a specific complaint, call the Pesticide Regulation Section at 410-841-5710 and ask to speak with the inspector in charge of the investigation, the Supervising Inspector or the Case Review Officer.

If you have any questions not addressed here, please to call the MDA, Pesticide Regulation Section at 410-841-5710.

Consumer Information

- Consumer Information Home
- Consumer Awareness
- Pest Control & Pesticide Information for Homeowners
- Pesticides and Child Safety
- Termites and Ants
- Pest Control and Sanitation - What Can I Do?
- Citizens Guide to Pesticide Enforcement & Complaints
- Answers to Questions Realtors Should Know About Pesticides

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50 Harry S. Truman Parkway, Annapolis, MD 21401

(410) 841-5700

MARYLAND

PESTICIDE DATA REPORT

FOR 2013

MARYLAND
DEPARTMENT OF AGRICULTURE

Martin O'Malley
Governor

Anthony G. Brown
Lieutenant Governor

Earl F. Hance
Secretary of Agriculture

Mary Ellen Setting
Deputy Secretary

MARYLAND PESTICIDE DATA REPORT FOR 2013

I. INTRODUCTION

The Maryland Pesticide Registration and Labeling Law (Title 5, Subtitle 1, Agriculture Article 1, Agriculture Article, Ann. Code Md.), Section 5-102(D), requires the Secretary of Agriculture to develop a comprehensive pesticide data program and to provide the General Assembly, in accordance with Section 2-1246 of the State Government Article, a report on pesticide data. The annual date program is to include the number and types of enforcement actions taken and figures for the number, types, and use of pesticides in Maryland.

A pesticide as defined generally by state and federal law, is any substance, or mixture of substances intended to prevent, destroy, repel, or mitigate any pest. There are at least 21 different classes (types of pesticides based on their target pests, including algaecides = target pest is algae; avicide = birds; bactericides = bacteria; fungicide = fungi; growth regulator = insect or plant growth; herbicide = weeds; insecticide = insects; rodenticides = rodents; and slimicide = slime molds.

II. BACKGROUND

The Maryland Department of Agriculture (MDA) is the State agency responsible for regulating the distribution, sale, storage, use and disposal of pesticides in Maryland. The Department cooperates with other State agencies, institutions and federal agencies to conduct pesticide education, regulatory and enforcement programs. Departmental activities and responsibilities are described briefly, as follows:

A. Pesticide Regulation Section

1. Enforcement Program

The Pesticide Regulation Section of the Maryland Department of Agriculture enforces the Federal (Federal Insecticide, Fungicide, and Rodenticide Act FIFRA) and state (Pesticide Applicators Law) pesticide use laws and regulation. Under the enforcement program, MDA conducts routine inspections of licensed pesticide businesses, public agencies and restricted use pesticide dealers. Inspection include review of pesticide application records, restricted use pesticide sales records, safety equipment, storage areas, application equipment, vehicles and anti-siphon devices. Use observations are conducted to observe actual pesticide applications to field crops, structures, lawns and ornamental plants to ensure compliance with label directions and state and federal regulations.

Pesticide misuse, incidents, and consumer complaints are investigated. In the event of a violation, the Department has the authority to suspend, revoke or deny a license or certificate and to assess a civil penalty. As part of a Cooperative Agreement with the U.S. Environmental Protection Agency (EPA), the Pesticide Regulation Section conducts producer establishment, marketplace, worker protection, container-containment and

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pesticide import inspections. EPA also refers various complaint investigations and special initiative inspections to MDA for action.

2. Applicator Certification and Training Program

The Pesticide Regulation Section certifies private and commercial pesticide applicators to verify the competence of the applicator. Private applicators (farmers) are given closed-book written exams to become certified for a three year period. Certification authorizes them to purchase and apply restricted use pesticides on their own property for the purpose of producing agricultural commodities. Certificates are renewed by MDA after submission of proof of update training. MDA certifies commercial applicators (employees of pest control businesses and public agencies) who meet minimum standards of experience or education requirements and who have passed written exams in specific pest control categories. Commercial applicator certificates are renewed annually, after requirement training has been obtained in order to maintain their level of competency. MDA approve and monitors applicator recertification training courses and sets minimum standards for approval of courses for recertification purposes. Private and commercial applicator training sessions are coordinated with county extension agents, who are provided training materials such as slide sets, videos and educational brochures by MDA. In addition, MDA registers employees who work under the supervision of certified commercial applicators. Prior to registration with the Department, and within 30 days of employment, the employee must be trained according to standards developed by MDA.

MDA issues licenses and permits to pesticide businesses or public agencies that apply general or restricted use pesticides. Dealers who sell restricted use pesticides must obtain a permit issued by MDA to do so. MDA issues licenses to pest control consultants who either identify pests or recommend pesticides or other techniques for the purpose of controlling pests.

3. Technical Information Collection and Dissemination Program

The Pesticide Regulation Section provides information to pesticide applicators, dealers, federal, state and local agencies and the general public on issues concerning pesticide use and pesticide regulations. Training materials, informational brochures and fact sheets are developed for pesticide applicators in order to provide compliance assistance when new guidelines or regulations are implemented. A series of "Pesticide Information Sheets' was developed to provide information on pesticide issues and regulation to consumers and pesticide applicators. The Pesticide Regulation Section developed a Consumer Information Bulletin for use by licensed lawn and landscape firms for distribution to their customers. In addition, the Section has compile pesticide product label information that must be given to all pest control customers to inform them of any safety, precautions or environmental hazards associated with each pesticide used. A listing of pesticide sensitive individuals is available so that these listed individuals can receive advanced notification prior to lawn and ornamental pesticide applications being made to adjacent properties by licensed pest control businesses or public agencies. Maryland is one

of twelve (12) states that have a mandated pesticide sensitive individual notification program.

The Department provides information to applicants on where and how to obtain study material for certification and conducts certification examination session every other month in three regional locations. Private applicators (farmers applying restricted use pesticides) receive exam study material provided by the "Department and are offered certification examinations in county extension offices on an as-needed basis.

Homeowners are given information on licensing requirements for pest control firms, as well as, information on termite inspections and control, proper pesticide handling and alternatives to chemical pest control. Table top displays, brochures and "Pesticide Information Sheets" have been developed for use at various trade shows, grower meetings, and State and county fairs.

During 2013, MDA continued to expand the Pesticide Regulation Section's Homepage so that information on pesticide business licensing requirements, certification exam dates, recertification training sessions, pesticide container recycling dates, Pesticide Information Sheets, and Integrated Pest Management in Schools is available on the Internet. Consumers can now electronically file complaints, report pesticide incidents, download application forms to apply for certification, request employee I.D. cards and request additional information about pesticide regulations and management programs. The Section's website contains searchable databases of registered pesticide products, licensed pesticide businesses, certified pesticide applicators and restricted use pesticide dealers. These searchable databases allow pesticide dealers to verify a pesticide applicator's certification. In addition, pesticide applicators and homeowners can search for pesticide products by brand name, active ingredient, use site or pest controlled. In 2013, the Pesticide Regulation Section launched an online mapping application, on its website, that shows were commercial crops that are sensitive to pesticide damage are so the pesticide applicator can take extra precautions to prevent pesticide spray drift, especially form herbicides, when spraying on nearby properties. Information in the statewide map is voluntarily provided by growers of sensitive crops.

4. Water Quality Protection, Endangered Species Protection and Worker Protection Programs

MDA is involved in four Federal (EPA) regulatory programs that are being implemented through the states. The Department has developed a State water quality management plan for managing the use of pesticides to protect water resources as part of its Water Quality Protection Program. The Department monitors EPA's "Pesticides of Interest" list annually to maintain a list of "Pesticides of Concern" within Maryland. Under the Endangered Species Protection Program, the Department is responsible for programs to protect federally listed endangered species that may be harmed by the use of certain pesticides. The Department has implemented and conducts the federal Worker Protection Standard Program to protect certain pesticide users, handlers and farmworkers from exposure to pesticides. The Department also inspects agricultural facilities to ensure

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bulk pesticide storage tanks, containment structures and mixing/loading pads meet state and federal requirements.

5. Special Programs

The Pesticide Regulation Section conducts special programs relating to pesticide management, when funding is available. These special programs address specific pesticide issues, environmental concerns or regional situations that require additional focus and attention beyond routine programs. Special programs many include development of informational materials and pesticide educations programs, participation in pesticide monitoring programs, and coordination of pesticide container and unusable pesticide disposal programs.

6. Chesapeake Bay Programs

MDA is an active participant in efforts to restore the Chesapeake Bay. Pesticide management commitments were incorporated in the Toxics 2000 Strategy as part of the Chesapeake 2000 Agreement and include commitments for adoption of integrated pest management, development of programs for pesticide container recycling, unusable pesticide disposal and implementation of agricultural best management practices. These pesticide management programs conducted by the Pesticide Regulation Section have placed Maryland in a leadership role and have given MDA recognition as one of the key Bay agencies in toxics reduction. Future toxics programs will be shaped by MDA and implemented through the Section's regulatory and educational programs.

7. Integrated Pest Management in Schools

The Pesticide Regulation Section has been conducting an Integrated Pest Management in Public School Building and School Grounds since 1995, in cooperation with the Maryland State Department of Education, Maryland Association of Boards of Education, county school systems, University of Maryland, Maryland State Pest Control Association, and EPA. The purpose of the program is to review each school system's pest management practices and to provide technical assistance to Maryland public school systems to facilitate the implementation of IPM programs in order to reduce the risk of exposing students and staff members to pesticides. Mandatory IPM programs have been required in Maryland public schools and on schools ground since 2000.

B. State Chemist Section

1. Registration

The State Chemist Section (SCS) is responsible for registration of all pesticide products distributed, sold, or transported in Maryland. The purpose of pesticide product registration is to ensure the sale and distribution of commodities that are effective and safe for humans and the environment. In 2013 the Section registered 13,521 (pesticides-

12,782; fertilizer-pesticide mixtures-739) products for sale and distribution within the State as compared to 13,467 products registered in 2012.

2. Inspection

Product quality and safety are determined by chemical analysis of pesticide products sampled by the Section's inspection staff which, on a regular schedule, inspects pesticide warehouses and retail outlets. During 2013, the inspection staff collected 107 pesticide formulation products for chemical analysis. The section also collected 682 samples of fruit juice, produce, fruit and processed food for analysis by USDA/EPA in order to obtain pesticide residue data for establishing appropriate pesticide tolerances for foods consumed by children and babies.

3. Chemical Analyses

In 2013, the Section analyzed 59 MDA/EPA pesticide investigation samples and 15 EPA/MDA formulations for MDA's Pesticide Regulation Section.

4. Pesticide Data Program

The Section continued to generate pesticide data relative to determining the safeness of Maryland-grown vegetables/fruit sold at roadside stands and farmer markets. In 2013, State Chemist inspectors collected from Maryland roadside vegetable and fruit stands 80 samples. No residues above EPA tolerances were detected. The data will be sent to EPA and U. S. Department of Agriculture (USDA) for incorporation in national data banks. This project will continue and probably expand in response to monitoring Maryland grown agricultural produce for toxic materials relative to potential chemical terrorist attacks on the food supply. This project continues to indicate that produce and fruit grown in Maryland do not contain toxic levels of pesticides.

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III. PESTICIDE REGISTRATION DATA AND ENFORCEMENT

The Pesticide Registration and Labeling Law requires a distributor of pesticide products to annually register the products with MDA's State Chemist Section (SCS) before distribution in the State. The State Chemist Section utilizes a computerized registration process, which has expedited and improved the accuracy of the registration process and has enabled the Section to compile more information about registered products.

During 2013, pesticide product registration data include:

1. Number of registrants	= 1,099
2. Number of pesticide products registered	= 13,521

As a result of the State Chemist Section's enforcement and registration program (pesticide dealer inspections, product sample collection, chemical analysis and label review), the following regulatory actions were taken against pesticide products violating the State Pesticide Registration and Labeling Law:

* Market place samples collected and analyzed	= 108
* Total chemical analyses	= 271
* Non-registered product (products offered for sale but not registered with the Department) stop sale orders	= 5

In support of the Pesticide Regulation Section's enforcement activities, and for other State agencies, the SCS laboratory analyzed samples (soil, water, tissue, swabs, product, etc.) for pesticide residues. The following is a summary of the analyses:

Investigational Samples (pesticide misuse, accidents):

Samples analyzed	=	59
Total number of analyses	=	493 (for 427 different pesticides)

Food Safety Program: Monitoring of Maryland produce offered for sale from June – September (1997-2013)

Number of samples containing pesticides:

1997 - 51 samples collected	=	25 positive
1998 - 50 samples collected	=	19 positive
1999 - 51 samples collected	=	15 positive
2000 - 75 samples collected	=	19 positive
2001 - 75 samples collected	=	52 positive
2003 - 60 samples collected	=	1 positive
2004 - 72 samples collected	=	none detected above EPA tolerances

2005 - 89 samples collected	= none detected above EPA tolerances
2006 - 56 samples collected	= 24 positive
2007 - 48 samples collected	= none detected above EPA tolerances
2008 - 645 samples collected	= none detected above EPA tolerances
2009 - 64 samples collected	= none detected above EPA tolerances
2010 - 70 samples collected, discarded 8	= none detected above EPA tolerances
2011 - 92 samples collected, discarded 8	= none detected above EPA tolerances
2012 - 94 samples collected	= none detected above EPA tolerances
2013 - 80 samples collected	= to be completed in 2014

*It would appear that samples of produce grown in Maryland are free of pesticides, e.g., none detected at levels equal to or greater than the tolerance levels established by the U. S. Environmental Protection Agency.

IV. PESTICIDE USE ENFORCEMENT INSPECTION AND ACTIONS

During 2013, inspection of licensed pest control businesses and public agencies were conducted as follows:

1.	Routine business inspections	=	847
2.	Routine public agency inspection	=	101
3.	Pesticide Dealer Inspections	=	71
4.	Pesticide Use Observations	=	45
5.	Pesticide Samples collected for analysis	=	81
6.	Application records reviewed	=	1,099

Violations detected during pest control business inspection are summarized in Table 1 and include:

1.	Number of businesses and public Agencies with violations	=	257
2.	Unregistered employee violations	=	13
3.	Records incomplete or inaccurate	=	98
4.	Vehicles not properly identified	=	30
5.	No anti-siphon device	=	14
6.	No customer information	=	13

During 2013, regulatory or enforcement actions were taken against individuals or firms violating the Maryland Pesticide Applicators Law. The actions taken or penalties assessed for specific violations of the law or regulations are summarized, as follows:

1. Consumer Complaints Investigation = 53 (Investigations initiated as a result of written complaints from consumers regarding pest inspection or pesticide misuse.) See attached Table 2).
2. Investigational Conferences = ? (Informal meeting held with licensee and/or complainant to gather additional information about an ongoing investigation or to alert licensee to a situation requiring immediate action.
3. Administrative Hearing = 0 (Formal Hearing before an Administrative Law Judge, because of magnitude of violation warrants it or because of repeat violations by firm or individual).
4. Penalties Assessed:
   a. Notices of Warning = 310 (Certified Letter notifying licensee, permittee, or individual that they have committed a violation or that a situation needs to be corrected).
   b. Field Notices = 22 (Violations noted by a field inspector during routine inspection. Licensee, permittee or individual is informed of an infraction and given a compliance period to correct the infraction).
   c. Criminal Action = 0 (Action taken against an individual or company that is operating without a pesticide business license or who has repeatedly violated pesticide laws. Individual is prosecuted through county court system; violation is a misdemeanor and subject to a fine of up to $1,000, 60 days in jail, or both).
   d. Civil Penalties = 6 (A civil penalty may be assessed in lieu of or in addition to a suspension or revocation of a license, permit, certificate, or employee registration card. The Secretary may impose up to a $2,500 penalty per violation). Licensees were assessed a total of $6,090 in civil penalties.

Under the federal pesticide enforcement cooperative agreement, the following inspections were conducted by the Pesticide Regulation Section Inspector staff:

1.	Pesticide producer establishment inspections	=	30
2.	Pesticide marketplace inspections	=	31
3.	Pesticide import inspections	=	N/A
4.	EPA referrals for inspection/investigation	=	4

V. PESTICIDE APPLICATOR CERTIFICATION AND TRAINING PROGRAM

During 2013, the following licensing and certification activities were conducted, and are summarized in attached Table 3:

1. Pesticide businesses licensed = 1,728

2.	Public agencies permitted	=	323
3.	Pesticide dealer permits	=	152
4.	Pest control applicators certified	=	6,100
5.	Private applicators certified	=	3,275
6.	Commercial applicators examined	=	1,922
7.	Total examinations administered	=	2,156

In order to maintain applicator certification, private applicators must participate in Departmental approved training once every three years. Commercial applicators of pesticides must attend an annual recertification training session. The following data indicate training held in 2013:

1.	Commercial applicator training sessions held	=	468
2.	Private applicator training sessions held	=	118
3.	Commercial applicators recertified	=	3,634
4.	Private applicators recertified	=	1,594

VI. PESTICIDE USE DATA

The Pesticide Regulation Section regulates the use of pesticide in Maryland (See Section II). An essential factor in conducting effective regulatory or educations programs on pesticides is data relating to the quantity and distribution of pesticide product usage in the State. It is a costly and complicated process to collect pesticide usage data. Therefore, the Department conducted use surveys on a 3 – year cycle, beginning in 1982 and followed by annual surveys for 1985, 1988, 1991, 1994, 1997 and for 2000. Due to limited resources (funds and personnel), pesticide usage data, the Department was limited to conducting additional pesticide usage surveys in 2004 and 2011. The use data was compiled by the National Agricultural Statistics Service (NASS), an agency of the U.S. Department of Agriculture, in cooperation with MDA. The Department contracts with NASS to conduct the surveys and to provide final data, but MDA has no access to the raw data in order to protect the confidentiality of the data and privacy of the respondents.

Maryland is unique in having such extensive pesticide use data, as no neighboring state has similar data. These data meet the commitment made by Maryland as part of the Chesapeake Bay Agreement. In addition, the data has been used in a variety of ways, including as a basis for conducting surface water surveys or ground water surveys, and as a basis for developing state pesticide management plans to deal with pesticides with a potential to be a problem in water sources. MDA is planning on contracting with NASS to conduct pesticide usage surveys in 2015 for 2014 pesticide usage data and in 2016 to for 2015 pesticide usage data.

VII. WATER QUALTIY PROTECTION, ENDANGERED SPECIES PROTECTION AND WORKER PROTECTIONS PROGRAMS

MDA, as lead agency for pesticide management, is responsible for developing a Pesticide Management Plan (PMP) to protect water quality resources. The Pesticide

Regulation Section has participated in EPA sponsored ground water protection training courses on pesticide monitoring and wellhead protection in order to obtain information and guidance on developing Maryland's PMP. The PMP is one facet of an overall Comprehensive State Ground Water Protection Program (CSGWPP) which includes all state programs affecting ground water resources of the State.

MDA coordinated efforts with the Maryland Department of the Environment (MDE) and the Maryland Department of Natural Resources (DNR) to initiate development of Maryland's CSGWPP and PMP. Data collected from pesticide monitoring programs have been used to develop the generic Pesticide Management Plan. Ground water protection educational materials were developed for farmers, commercial applicators and pesticide dealers and incorporated into applicator recertification training programs. MDA has also contracted with the United States Geological Survey (USGS) in a number of monitoring projects located in the Chesapeake Bay.

MDA continues to support the endangered species protection program, initiated in 1992, to protect the Maryland darter from adverse effects due to pesticide use. Informational brochures were distributed to growers, commercial and private applicators, as well as, pesticide dealers that outlined the program and listed measures recommended for the protection of this federally listed endangered species, located in Harford County.

The federal worker protection standards (WPS) became effective in August, 1992. MDA continues to disseminate information on the federal program in pesticide applicator training sessions. Several WPS Compliance Review presentations were conducted for more than 1,000 growers throughout Maryland. MDA has conducted on-farm compliance assistance inspections to help farmers and producers comply with the WPS requirements. MDA continues to contract with Telamon Corporation, an AmeriCorps project participant, to provide pesticide safety training to farmers, farmworkers, children of farmworkers and health care providers.

VII. SPECIAL PROGRAMS

MDA continues to conduct an empty pesticide container recycling program in Maryland. During 2013, MDA Pesticide Container Recycling Program collected and recycled 42,242 empty pesticide containers weighing 36,500 pounds from growers and commercial pesticide applicators at 22 locations in seven counties and at 13 pesticide dealer/custom applicators sites.

IX. INTEGRATED PEST MANAGEMENT IN SCHOOLS

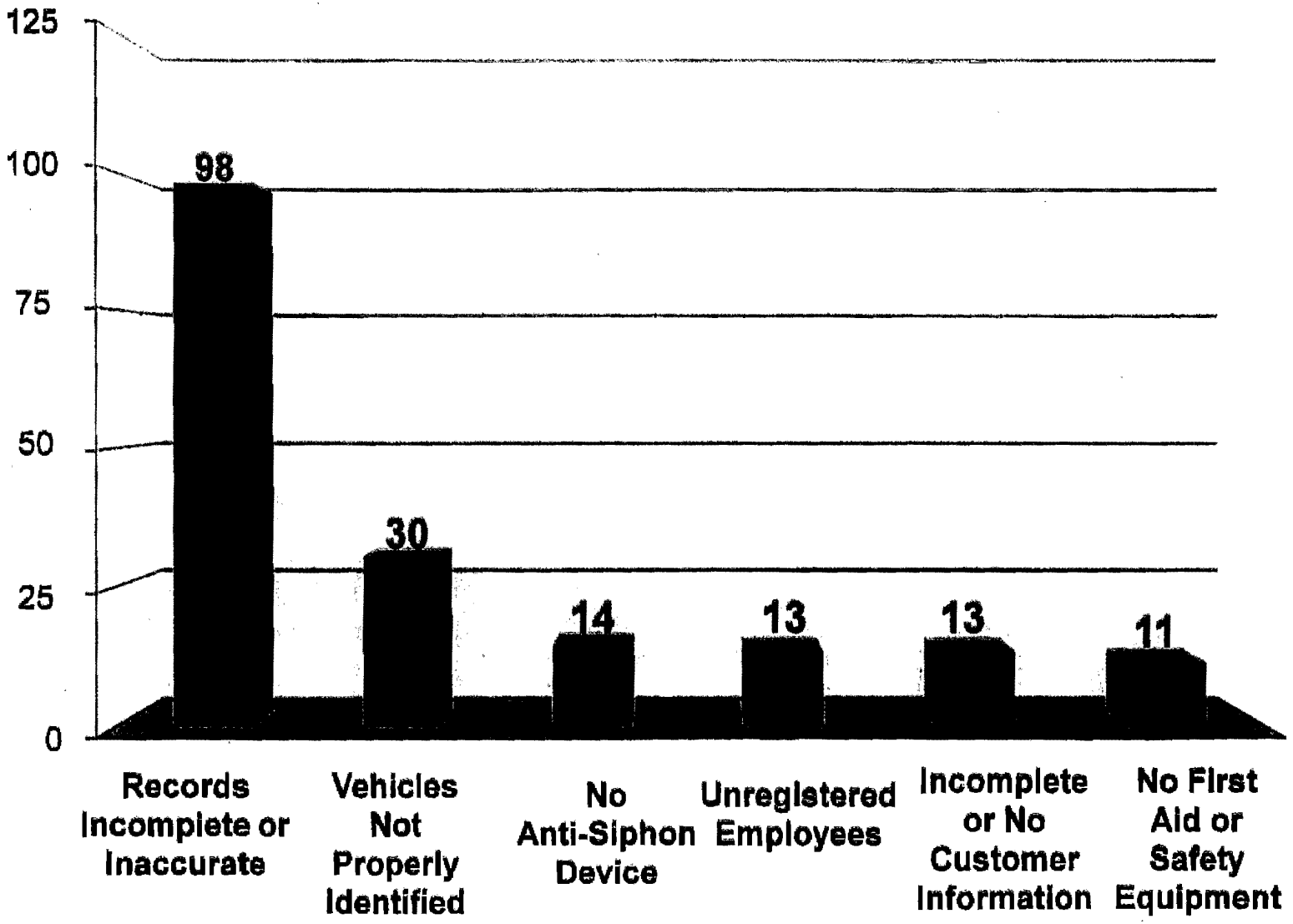
MDA continues to promote and implement the Integrated Pest Management (IPM) Program in Maryland Public Schools. Legislation was passed in 1999 that expanded the 1998 law to include pesticide use on school grounds. Schools are required to provide notification to parents, students, and staff of pesticide applications to school buildings and

on school grounds. Regulations to implement the law became effective January of 2002 and require schools to develop and implement IPM plans for school building and school grounds. MDA Pesticide Regulation Section staff reviewed and approved revised IPM plans that incorporated programs for managing pest problems on school grounds, and

provided technical assistance in the development of the plans. All of Maryland's public schools have fully implemented their IPM programs. MDA staff ensures continued compliance with these IPM regulations. A total of 60 public schools were inspected in 2013.

X. A summary and comparison of program activities conducted by the Pesticide regulation Section are provided in Appendix A

2013 Inspection Violations

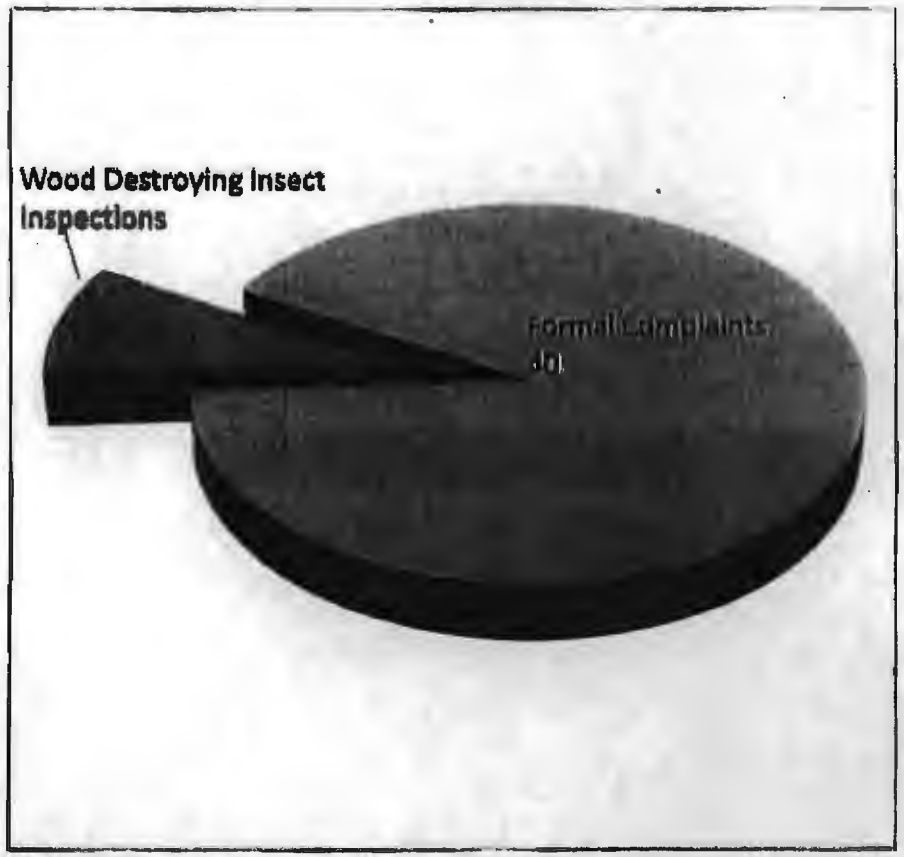


911 Businesses and Public Agencies Inspected

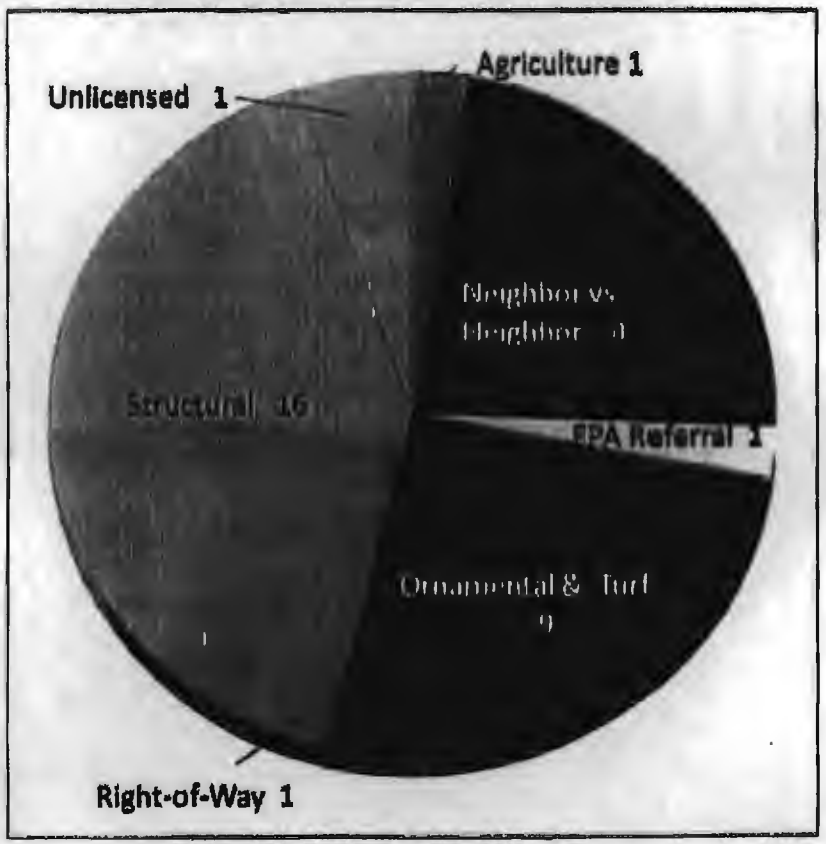
Table 1

2013 Complaint Investigations
By Pest Control Category

COMPLAINTS



COMPLAINTS BY CATEGORY



July 1, 2012 thru June 30, 2013

Table 2

Appendix A

PESTICIDE REGULATION SECTION ACTIVITIES 2011 – 2013

	2011	2012	2013
Pesticide Business Licensed	1,458	1,522	1,728
Not-For-Hire Businesses License	173	171	173
Commercial Pest Control Applicators certified in one or more Category	3,280	3,481	3,634
Registered Personnel Employed by Licensed Businesses and Public Agencies	11,372	10,266	7,533
Public Agency Permits Issued	319	325	323
Public Agency Applicators Certified in one or more category	1,051	1,102	1,042
Private Applicators Certified to Date	3,328	3,354	3,275
Dealer Permits Issued	120	141	152
Applicator Certification Examinations Sessions Held	18	18	18
Individuals Taking Certification Examinations	825	824	850
Examinations Administered in All Categories	2,130	2,158	1,922
Number of Businesses Inspected	1,050	1,099	911
Number of Businesses with Violations	276	324	344
Unregistered Employee Violations	16	24	13
Records Incomplete or Inaccurate Violations	184	110	98
Vehicles Not Properly Identified Violations	32	14	30
No Anti-siphon Device Violations	18	14	14
No First Aid/Safety Equipment Violations	8	14	11
Incomplete or No Customer Information Violations	24	49	13
Pesticide Dealer Inspections	98	89	78
Pesticide Application Records Reviewed	1,050	990	911
Hearings and Investigational Conferences	4	6	2
Consumer Complaint Investigations	37	53	
Pesticide Use Observations	65	75	71
Pesticide Samples Collected for Analysis	35	81	50
Market Place Inspections	29	61	31
Pesticide Producer Establishment Inspections	26	30	30
Container/Containment Inspections	8	8	9

MARYLAND PESTICIDE STATISTICS
for 2011

Issued cooperatively by

Maryland Department of Agriculture
Earl F. Hance, Secretary
Mary Ellen Setting, Deputy Secretary

U. S. Department of Agriculture
National Agricultural Statistics Service
Cynthia Clark, Administrator

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In consultation with
Maryland Department of Agriculture
Pesticide Regulation Section
Dennis Howard, Chief

PUBLISHED May 2013

Introduction

This publication contains estimates for specific pesticides used in Maryland during calendar year 2011. Published estimates include the combined pesticide usage of farm operators, certified private pesticide applicators, and commercially licensed businesses and public agencies.

All data were compiled by the Maryland Field Office of the National Agricultural Statistics Service (NASS) in cooperation with the Pesticide Regulation Section of the Maryland Department of Agriculture. Staff of the Pesticide Regulation Section provided technical assistance in survey planning and analysis of final summary tabulations. Data collection and summarization were completed by NASS, with access to record level data limited to NASS office personnel. All record-level data collected by NASS are confidential and protected by Title 7 of the U.S. Code.

Methodology

A survey was conducted in 2012 to estimate the amounts and types of pesticides applied in calendar year 2011 by Maryland farm operators, certified private pesticide applicators, commercially licensed businesses, and public agencies. The survey consisted of a sample of 1,501 farmers, 3,255 private applicators, 1,634 commercially licensed businesses, and 343 public agencies permitted to apply pesticides. Lists of certified applicators, businesses, and public agencies were provided by the Pesticide Regulation Section of the Maryland Department of Agriculture. The farm operator sample was selected from a comprehensive list of farm operators maintained by NASS.

In January 2012, questionnaires were mailed to all sampled operations, businesses, and agencies, with a second mailing occurring in early March 2012. Responses were received from 211 (62%) public agencies, 665 (41 %) licensed commercial applicators, 1,702 (52%) certified private applicators, and 856 (57%) of the sampled farm operators. Response was voluntary and not required by law.

Data were reviewed for completeness and accuracy and all amounts reported were converted to pounds of active ingredient. Following questionnaire review, data were keyed and summarized utilizing SAS statistical software and data analysis was conducted by NASS statisticians. Active ingredients were totaled and expanded to a State level based solely on the population of each sector, final sample sizes and survey response. The population assumed to represent total usage of the target populations.

Results

Pesticide active ingredient estimates are published only when there were a sufficient number of reports and/or amounts to deem the data reliable. Published data are listed in descending order by pounds of active ingredient and in alphabetical order. In addition, active ingredients reported but not estimated are listed. The top 20 pesticides (in terms of total pounds used), the top 10 by class (insecticides, herbicides, and fungicides) ate provided in separate tables. Common formulation and type for pesticides published comparisons tables are also provided.

State Pesticides Usage Estimates - Ranked According to Pounds Used

Rank	Pesticide Common Name	Pounds Active Ingredient
1	Chromic acid	5,145,298
2	Arsenic Pentoxide	3,718,525
3	Copper(II) oxide	2,358,000
4	Glyphosate	721,154
5	s-Metolachlor	555,807
6	2,4-D	439,538
7	Atrazine	381,321
8	Dimethoate	243,677
9	Imidacloprid	231,323
10	Simazine	200,734
11	Sulfuryl Fluoride	183,620
12	Cupric Oxide	170,007
13	Prodiamine	145,979
14	Mineral Oil	141,270
15	Paraquat dicholride	137,874
16	Glycine, N-(phosphonomethyl)-	129,218
17	Trifluralin	125,501
18	Etridiazole	118,384
19	Cypermethrin	97,844
20	MCPP	85,625
21	Chlorothalonil	61,069
22	Boric Acid	58,573
23	Permethrin	53,361
24	Alkyl poloxythylene eaters	52,057
25	Dithiopyr	52,005
26	Dicamba	51,343
27	Clomazone	47,698
28	Ethylene Oxide	45,376
29	Sulfur	38,701
30	Bifenthrin	34,527
31	Piperonyl Butoxide	32,422
32	Pendimethalin	30,957
33	Mancozeb	30,280
34	Mesotrione	25,684
35	Acetochlor	25,082
36	Thiophanate-methyl	24,138
37	Fluroxypyr	21,403
38	Fipronil	21,380
39	1H-1,2,4-Triazole-1-ethanol, .alp	21,084
40	Dimethylamine 2,4-dichlorophei	20,513
41	Potassium Salts of Fatty Acids	19,141
42	Acetic Acid	16,813
43	Dichlorvos	13,506
44	beta-Cyfluthrin	11,267
45	Diquat Dibromide	10,835
46	Petroleum Oils	10,247
47	Carbaryl	9,295
48	Chlorpyrifos	8,840
49	Dithiocarbamate	8,798
50	Triethylamine triclopyr	8,388
51	Glufosinate-ammonium	8,350
52	Metam-sodium	8,010
53	Propetamphos	7,374
54	Captan	7,127
55	Propiconazole	7,071
56	Triclopyr	6,878
57	Quinclorac	6,859
58	Ammonium Sulfate	6,829
59	Oryzalin	6,543
60	Copper Sulfate	6,462
61	Tetrachloroisophthalonitrile	6,305
62	Acephate	6,302
63	Zeta-cypermethrin	6,070
64	Mono- and di- potassium salts of	6,026
65	Maneb	5,753
66	MSMA	5,632
67	MCPA, dimethylamine salt	5,354
68	Rimsulfuron	5,313
69	Azoxystrobin	5,213
70	Dichlobenil	5,168
71	Petroleum Distillate	5,152
72	Dimethenamid-P	5,125
73	Boscalid	5,019
74	Pyraclostrobin	4,821
75	Nicosulfuron	4,805
76	Triisopropanolamine 2,4-dichlor	4,648
77	Difenoconazole	4,448
78	Iprodione	4,118
79	Paclobutrazol	4,027
80	Alachlor	3,941
81	Thifensulfuron methyl	3,502
82	Propamocarb Hydrochloride	3,415
83	Sulfentrazone	3,407
84	Copper Hydroxide	3,390
85	Diazinon	3,331
86	EPTC	2,905
87	Clopyralid	2,832
88	Mercurous Chloride	2,797
89	Methomyl	2,759
90	Phosmet	2,758
91	Phosphorus Acid	2,707
92	Pyripoxyfen	2,544
93	Triisopropanolamine	2,485
94	Phytobland Paraffinic Oil	2,365
95	Flumioxazin	2,144
96	Mecoprop	2,098

State Pesticides Usage Estimates - Ranked According to Pounds Used

Rank	Pesticide Common Name	Pounds Active Ingredient
97	Alkylphenol ethoxylate, alcohol	2,052
98	Tebuconazole	1,914
99	Ziram	1,863
100	Triadimefon	1,840
101	Potassium Bicarbonate	1,727
102	Phosphine	1,713
103	Fosetyl aluminum	1,681
104	Clethodim	1,680
105	Trichlorfon	1,651
106	Permethrin, mixed cis,trans	1,646
107	Imazethapyr	1,644
108	Tribenuron-methyl	1,594
109	Basic cupric sulfate	1,587
110	Metribuzin	1,566
111	Terbufos	1,520
112	Cyfluthrin	1,432
113	Ethalfluralin	1,376
114	lambda-Cyhalothrin	1,375
115	Deltamethrin	1,350
116	Fomesafen	1,326
117	Elemental Sulfur	1,301
118	Chlorfenapyr	1,282
119	Myclobutanil	1,261
120	Isoctyl	1,192
121	Halosulfuron-methyl	1,189
122	Kaolin clay	1,176
123	Linuron	1,174
124	Ethephon	1,056
125	Copper	1,044
126	s-Cyanomethrin	1,023
127	Endosulfan	981
128	Tefluthrin	901
129	Saflufenacil	898
130	Triisopropanolammonium salt of	875
131	Diphenylamine	867
132	Mefanoxam	828
133	Esfenvalerate	808
134	Metalaxyl-M	803
135	Poly(oxy-1,2-ethanediyl)	799
136	Prothioconazole	796
137	Flurprimidol	794
138	Mecoprop-P	750
139	Bensulide	725
140	Dinotefuran	725
141	Modified Vegetable Oil, Alkylat	700
142	Thiram	695
143	Malathion	691
144	Trinexapac-ethyl	690
145	Fosamine ammonium	673
146	Isoxaben	662
147	Phosphatidylcholine,methylaceti	657
148	3-Iodo-2-propynyl butylcarbama	656
149	Spiromesifen	625
150	Fluazifop-P-butyl	618
151	9,10- anthraquinone	609
152	Aluminum Phosphide	603
153	Canola oil	594
154	Chlorimuron-ethyl	589
155	Famoxadone	585
156	Butoxyethyl 2,4-dichlorophenox	548
157	Diuron	527
158	Azinphos-methyl	524
159	Endothall	522
160	Metconazole	489
161	Bacillus thuringiensis	477
162	Vernolate	461
163	Oxamyl	457
164	Cloransulam-methyl	421
165	Pelargonic Acid	416
166	Napropamide	411
167	Trifloxysulfuron-sodium	410
168	Imazapyr	408
169	Fenoxaprop-ethyl	408
170	Sodium Carbonate Peroxyhdrate	386
171	Crop Oil concentrate	373
172	Thiamethoxam	363
173	Diflufenzopyr	361
174	Fluthiacet-methyl	348
175	Dimethylamine (R)-2-(2-methyl-	343
176	Siduron	343
177	Prometon	325
178	Sethoxydim	315
179	Acetamiprid	312
180	Methyl Bromide	296
181	Bifenazate	289
182	Isopropyl alcohol et. Al.	285
183	Flutolanil	283
184	2,4-DP	283
185	Prohexadione- calcium	276
186	Trifloxystrobin	267
187	Silicon Dioxide	259
188	Fenhexamid	258
189	Octanic acid ester of bromoxynil	255
190	Calcium Hypochlorite	251
191	1 -Methylheptyl Ester of Fluroxy	249
192	Pyrethrins	249

State Pesticides Usage Estimates - Ranked According to Pounds Used

Rank	Pesticide Common Name	Pounds Active Ingredient
193	Chloroneb	245
194	Oxyfluorfen	242
195	Thiabendazole	241
196	Oxadiazon	238
197	Calcium polysulfide	227
198	Hydroprene	205
199	Metsulfuron-methyl	205
200	Prosulfuron	203
201	Fluoxastrobin	198
202	Cyprodinil	193
203	Copper Salts of fatty & rosin aci	191
204	Amorphous Silica	176
205	Bacillus spahericus	174
206	2-Ethylhexyl (R)-2-(2,4-dichlorc	166
207	Chlormequat	160
208	Pinoxaden	159
209	Cyazofamid	153
210	Bentazone	149
211	Borate	148
212	Indoxacarb	146
213	Manganese	146
214	Dimethylamine [N-methylmetha	144
215	Copper Oxychloride Sulfate	143
216	Flumetsulam	139
217	Mefluidide	138
218	Fenpropathrin	133
219	O, O-Dimethyl S-Phosphorodith	131
220	Hydrogen Dioxide	130
221	Hydrogen peroxide	129
222	Methoxychlor	124
223	Ericine	121
224	Aminocyclopyrachlor	118
225	Kresoxim-methyl	108
226	Polyoxin D zinc salt	103
227	Dicloran	99
228	Triticonazole	98
229	Chlorsulfuron	98
230	Imazalil	92
231	Bacillus thuringiensis subsp. Isra	90
232	Clothianidin	89
233	Chlorantraniliprole	89
234	DCPA	86
235	Imazapic	86
236	Methoprene	84
237	Sodium Chlorate	84
238	Alkyl* dimethyl benzyl ammoni	82
239	Maleic Hydrazide	80
240	Hexythiazox	77
241	Norflurazon	76
242	Hexazinone	74
243	Methoxyfenozide	73
244	Streptomycin Sulfate	62
245	Bromacil	62
246	s-Cyano-2,2 dimethylcyclopropa	58
247	Daminozide	58
248	Aminopyralid	58
249	Spinosad	55
250	Pyrimenthanil	51
251	Methylated Seed Oil	51
252	Spinetoram	49
253	Imuzamox	43
254	Copper triethanolamine complex	43
255	Glutaraldehyde	42
256	Carbendazim	42
257	Thiacloprid	41
258	Fludioxonil	41
259	Carfentrazone-ethyl	39
260	Pyrethrum	38
261	Terbacil	38
262	N-thiophosphoric triamide	38
263	Imazaquin	37
264	Fluridone	33
265	Fenbuconazole	29
266	Flumiclorac-pentyl ester	28
267	BHT (Butylated Hydroxytoluene	27
268	Methylanthranilate	27
269	Zinc Phosphide	27
270	Bacillus subtilis	24
271	Noviflumuron	23
272	beta-Cyfluthrin cyanol	22
273	Etoxazole	22
274	Calcium oxytetracycline	21
275	Abamectin	21
276	MCPA, 2-ethylhexyl ester	20
277	Mesosulfuron-methyl	20
278	Sodium metaborate (NaBO2)	20
279	N-methyl pyrrolidone	19
280	Dikegulac Sodium	19
281	s-Kinoprene	18
282	Bromoxynil	18
283	Clorantraniliprole	18
284	Zinc	18
285	Octaborate	18
286	Mandipropamide Technical	17

State Pesticides Usage Estimates - Ranked According to Pounds Used

Rank	Pesticide Common Name	Pounds Active Ingredient
287	Chorothalonil	17
288	Water conditioning agent	16
289	Quizalofop-ethyl	16
290	Anthraquinone	15
291	Spirodiclofen	14
292	Cycloate	14
293	Tartrazine	13
294	Quinoxyfen	13
295	Decanol plus related compounds	13
296	Halofenozide	13
297	Streptomycin	13
298	Kinoprene	11
299	Fenarimol	11
300	Vinclozolin	11
301	Topramezone	11
302	n-Octyl Bicyclohepten	11
303	1,3-Dichloropropene	10
304	Proiconazole	10
305	N-octyl bicycloheptene dicarbox	10
306	Novaluron	10
307	Octanol	10
308	Non-ionic surfactant blend	9
309	Cyclohexanecarboxamide	9
310	N6-Benzyladenine	9
311	s-Methoprene	8
312	Dimethomorph	8
313	Sumithrin	8
314	Fenpyroximate	7
315	Buprofezin	7
316	Triazin-3-one	6
317	Sulfometuron methyl	6
318	Spirotetramat	6
319	Chloropicrin	6
320	Diphacinone	6
321	Ethofumesate	5
322	Hydramethylnon	5
323	Azadirachtin	5
324	Fatty Acids	5
325	5-Hydroxytetracycline monohyd	5
326	Propoxur	5
327	Metiram	5
328	D-Phenothrin	4
329	Lactofen	4
330	Naptalam	4
331	Tebuthiuron	4
332	Primisulfuron-methyl	4
333	Flonicamid	3
334	Pyroxsulam	3
335	Octhilinone	3
336	Ethofenprox	3
337	Alkyl* dimethyl ethylbenzyl ami	3
338	Neem Oil	3
339	Putrescent Whole Egg Solids	2
340	Linalool	2
341	Cytokinin (as kinetin)	2
342	Carbofuran	2
343	Prallethrin	2
344	Borax	1
345	Beauveria Bassiana	1
346	d-Limonene	1
347	Flucythrinate	1
348	N,N-Dimethyl-2-?3-(4,6-dimeth	1
349	Didecyl dimethyl ammonium chl	1
350	Clodinafop-propargyl or Hexaco	1
351	Emamectin benzoate	1
352	Nicotonic Acid	1
353	Pyridaben	1
354	Gibberellic Acid	1
355	Fluopicolide	1
356	Aminoethoxyvinylglycine hydro	1
357	Fluvalinate	1
358	Phenothrin	1
359	Allethrin	1
360	Bromadiolone	1
361	Pyridalyl	1
	Total	**16,503,549**

State Pesticide Usage Estimates - Alphabetical

Rank	Pesticide Common Name	Pounds Active Ingredient
191	1 -Methylheptyl Ester of Fluroxyp	249
303	1,3-Dichloropropene	10
39	1H-1,2,4-Triazole-1-ethanol, .alpl	21,084
6	2,4-D	439,538
184	2,4-DP	283
206	2-Ethylhexyl (R)-2-(2,4-dichlorop	166
148	3-Iodo-2-propynyl butylcarbamat	656
325	5-Hydroxytetracycline monohydr	5
151	9,10- anthraquinone	609
275	Abamectin	21
62	Acephate	6,302
179	Acetamiprid	312
42	Acetic Acid	16,813
35	Acetochlor	25,082
80	Alachlor	3,941
24	Alkyl poloxythylene eaters	52,057
238	Alkyl* dimethyl benzyl ammoniu	82
337	Alkyl* dimethyl ethylbenzyl amm	3
97	Alkylphenol ethoxylate, alcohol e	2,052
359	Allethrin	1
152	Aluminum Phosphide	603
224	Aminocyclopyrachlor	118
356	Aminoethoxyvinylglycine hydrocl	1
248	Aminopyralid	58
58	Ammonium Sulfate	6,829
204	Amorphous Silica	176
290	Anthraquinone	15
2	Arsenic Pentoxide	3,718,525
7	Atrazine	381,321
323	Azadirachtin	5
158	Azinphos-methyl	524
69	Azoxystrobin	5,213
205	Bacillus spahericus	174
270	Bacillus subtilis	24
161	Bacillus thuringiensis	477
231	Bacillus thuringiensis subsp. Israe	90
109	Basic cupric sulfate	1,587
345	Beauveria Bassiana	1
139	Bensulide	725
210	Bentazone	149
44	beta-Cyfluthrin	11,267
272	beta-Cyfluthrin cyanol	22
267	BHT (Butylated Hydroxytoluene)	27
181	Bifenazate	289
30	Bifenthrin	34,527
211	Borate	148
344	Borax	1
22	Boric Acid	58,573
73	Boscalid	5,019
245	Bromacil	62
360	Bromadiolone	1
282	Bromoxynil	18
315	Buprofezin	7
156	Butoxyethyl 2,4-dichlorophenoxyace	548
190	Calcium Hypochlorite	251
274	Calcium oxytetracycline	21
197	Calcium polysulfide	227
153	Canola oil	594
54	Captan	7,127
47	Carbaryl	9,295
256	Carbendazim	42
342	Carbofuran	2
259	Carfentrazone-ethyl	39
233	Chlorantraniliprole	89
118	Chlorfenapyr	1,282
154	Chlorimuron-ethyl	589
207	Chlormequat	160
193	Chloroneb	245
319	Chloropicrin	6
21	Chlorothalonil	61,069
48	Chlorpyrifos	8,840
229	Chlorsulfuron	98
287	Chorothalonil	17
1	Chromic acid	5,145,298
104	Clethodim	1,680
350	Clodinafop-propargyl or Hexaconaz	1
27	Clomazone	47,698
87	Clopyralid	2,832
164	Cloransulam-methyl	421
283	Clorantraniliprole	18
232	Clothianidin	89
125	Copper	1,044
84	Copper Hydroxide	3,390
215	Copper Oxychloride Sulfate	143
203	Copper Salts of fatty & rosin acid	191
60	Copper Sulfate	6,462
254	Copper triethanolamine complex	43
3	Copper(II) oxide	2,358,000
171	Crop Oil concentrate	373
12	Cupric Oxide	170,007
209	Cyazofamid	153
292	Cycloate	14
309	Cyclohexanecarboxamide	9
112	Cyfluthrin	1,432
19	Cypermethrin	97,844
202	Cyprodinil	193

State Pesticide Usage Estimates - Alphabetical

	Pesticide Common Name	Pounds Active Ingredient
341	Cytokinin (as kinetin)	2
247	Daminozide	58
234	DCPA	86
295	Decanol plus related compounds	13
115	Deltamethrin	1,350
85	Diazinon	3,331
26	Dicamba	51,343
70	Dichlobenil	5,168
43	Dichlorvos	13,506
227	Dicloran	99
349	Didecyl dimethyl ammonium chlc	1
77	Difenoconazole	4,448
173	Diflufenzopyr	361
280	Dikegulac Sodium	19
72	Dimethenamid-P	5,125
8	Dimethoate	243,677
312	Dimethomorph	8
175	Dimethylamine (R)-2-(2-methyl-4	343
214	Dimethylamine [N-methylmethan	144
40	Dimethylamine 2,4-dichlorophenc	20,513
140	Dinotefuran	725
320	Diphacinone	6
131	Diphenylamine	867
45	Diquat Dibromide	10,835
49	Dithiocarbamate	8,798
25	Dithiopyr	52,005
157	Diuron	527
346	d-Limonene	1
328	D-Phenothrin	4
117	Elemental Sulfur	1,301
351	Emamectin benzoate	1
127	Endosulfan	981
159	Endothall	522
86	EPTC	2,905
223	Ericine	121
133	Esfenvalerate	808
113	Ethalfluralin	1,376
124	Ethephon	1,056
336	Ethofenprox	3
321	Ethofumesate	5
28	Ethylene Oxide	45,376
273	Etoxazole	22
18	Etridiazole	118,384
155	Famoxadone	585
324	Fatty Acids	5
299	Fenarimol	11
265	Fenbuconazole	29
188	Fenhexamid	258
169	Fenoxaprop-ethyl	408
218	Fenpropathrin	133
314	Fenpyroximate	7
38	Fipronil	21,380
333	Flonicamid	3
150	Fluazifop-P-butyl	618
347	Flucythrinate	1
258	Fludioxonil	41
216	Flumetsulam	139
266	Flumiclorac-pentyl ester	28
95	Flumioxazin	2,144
355	Fluopicolide	1
201	Fluoxastrobin	198
264	Fluridone	33
37	Fluroxypyr	21,403
137	Flurprimidol	794
174	Fluthiacet-methyl	348
183	Flutolanil	283
357	Fluvalinate	1
116	Fomesafen	1,326
145	Fosamine ammonium	673
103	Fosetyl aluminum	1,681
354	Gibberellic Acid	1
51	Glufosinate-ammonium	8,350
255	Glutaraldehyde	42
16	Glycine, N-(phosphonomethyl)- pota	129,218
4	Glyphosate	721,154
296	Halofenozide	13
121	Halosulfuron-methyl	1,189
242	Hexazinone	74
240	Hexythiazox	77
322	Hydramethylnon	5
220	Hydrogen Dioxide	130
221	Hydrogen peroxide	129
198	Hydroprene	205
230	Imazalil	92
235	Imazapic	86
168	Imazapyr	408
263	Imazaquin	37
107	Imazethapyr	1,644
9	Imidacloprid	231,323
253	Imuzamox	43
212	Indoxacarb	146
78	Iprodione	4,118
120	Isoctyl	1,192
182	Isopropyl alcohol et. Al.	285
146	Isoxaben	662
122	Kaolin clay	1,176

State Pesticide Usage Estimates - Alphabetical

	Pesticide Common Name	Pounds Active Ingredient
299	Kinoprene	11
226	Kresoxim-methyl	108
330	Lactofen	4
115	lambda-Cyhalothrin	1,375
341	Linalool	2
124	Linuron	1,174
144	Malathion	691
240	Maleic Hydrazide	80
33	Mancozeb	30,280
287	Mandipropamide Technical	17
65	Maneb	5,753
214	Manganese	146
277	MCPA, 2-ethylhexyl ester	20
68	MCPA, dimethylamine salt	5,354
20	MCPP	85,625
97	Mecoprop	2,098
139	Mecoprop-P	750
133	Mefanoxam	828
218	Mefluidide	138
89	Mercurous Chloride	2,797
278	Mesosulfuron-methyl	20
34	Mesotrione	25,684
135	Metalaxyl-M	803
52	Metam-sodium	8,010
161	Metconazole	489
90	Methomyl	2,759
237	Methoprene	84
223	Methoxychlor	124
244	Methoxyfenozide	73
181	Methyl Bromide	296
269	Methylanthranilate	27
252	Methylated Seed Oil	51
328	Metiram	5
111	Metribuzin	1,566
200	Metsulfuron-methyl	205
14	Mineral Oil	141,270
142	Modified Vegetable Oil, Alkyla	700
64	Mono- and di- potassium salts	6,026
66	MSMA	5,632
120	Myclobutanil	1,261
349	N,N-Dimethyl-2-?3-(4,6-dimetho	1
311	N6-Benzyladenine	9
167	Napropamide	411
331	Naptalam	4
339	Neem Oil	3
76	Nicosulfuron	4,805
353	Nicotonic Acid	1
280	N-methyl pyrrolidone	19
302	n-Octyl Bicyclohepten	11
305	N-octyl bicycloheptene dicarboximic	10
308	Non-ionic surfactant blend	9
241	Norflurazon	76
306	Novaluron	10
271	Noviflumuron	23
262	N-thiophosphoric triamide	38
219	O, O-Dimethyl S-Phosphorodithioate	131
285	Octaborate	18
189	Octanic acid ester of bromoxynil	255
307	Octanol	10
335	Octhilinone	3
59	Oryzalin	6,543
196	Oxadiazon	238
163	Oxamyl	457
194	Oxyfluorfen	242
79	Paclobutrazol	4,027
15	Paraquat dicholride	137,874
165	Pelargonic Acid	416
32	Pendimethalin	30,957
23	Permethrin	53,361
106	Permethrin, mixed cis,trans	1,646
71	Petroleum Distillate	5,152
46	Petroleum Oils	10,247
358	Phenothrin	1
90	Phosmet	2,758
147	Phosphatidylcholine,methylacetic aci	657
102	Phosphine	1,713
91	Phosphorus Acid	2,707
94	Phytobland Paraffinic Oil	2,365
208	Pinoxaden	159
31	Piperonyl Butoxide	32,422
135	Poly(oxy-1,2-ethanediyl)	799
226	Polyoxin D zinc salt	103
101	Potassium Bicarbonate	1,727
41	Potassium Salts of Fatty Acids	19,141
343	Prallethrin	2
332	Primisulfuron-methyl	4
13	Prodiamine	145,979
185	Prohexadione- calcium	276
304	Proiconazole	10
177	Prometon	325
82	Propamocarb Hydrochloride	3,415
53	Propetamphos	7,374
55	Propiconazole	7,071
326	Propoxur	5
200	Prosulfuron	203
136	Prothioconazole	796

State Pesticide Usage Estimates - Alphabetical

	Pesticide Common Name	Pounds Active Ingredient
339	Putrescent Whole Egg Solids	2
74	Pyraclostrobin	4,821
192	Pyrethrins	249
260	Pyrethrum	38
353	Pyridaben	1
361	Pyridalyl	1
250	Pyrimenthanil	51
92	Pyripoxyfen	2,544
334	Pyroxsulam	3
57	Quinclorac	6,859
294	Quinoxyfen	13
289	Quizalofop-ethyl	16
68	Rimsulfuron	5,313
129	Saflufenacil	898
246	s-Cyano-2,2 dimethylcyclopropan	58
126	s-Cyanomethrin	1,023
178	Sethoxydim	315
176	Siduron	343
187	Silicon Dioxide	259
10	Simazine	200,734
281	s-Kinoprene	18
311	s-Methoprene	8
5	s-Metolachlor	555,807
170	Sodium Carbonate Peroxyhdrate	386
237	Sodium Chlorate	84
278	Sodium metaborate (NaBO2)	20
252	Spinetoram	49
249	Spinosad	55
291	Spirodiclofen	14
149	Spiromesifen	625
318	Spirotetramat	6
297	Streptomycin	13
244	Streptomycin Sulfate	62
83	Sulfentrazone	3,407
317	Sulfometuron methyl	6
29	Sulfur	38,701
11	Sulfuryl Fluoride	183,620
313	Sumithrin	8
293	Tartrazine	13
98	Tebuconazole	1,914
331	Tebuthiuron	4
128	Tefluthrin	901
261	Terbacil	38
111	Terbufos	1,520
61	Tetrachloroisophthalonitrile	6,305
195	Thiabendazole	241
257	Thiacloprid	41
172	Thiamethoxam	363
81	Thifensulfuron methyl	3,502
36	Thiophanate-methyl	24,138
142	Thiram	695
301	Topramezone	11
100	Triadimefon	1,840
316	Triazin-3-one	6
108	Tribenuron-methyl	1,594
105	Trichlorfon	1,651
56	Triclopyr	6,878
50	Triethylamine triclopyr	8,388
186	Trifloxystrobin	267
167	Trifloxysulfuron-sodium	410
17	Trifluralin	125,501
93	Triisopropanolamine	2,485
76	Triisopropanolamine 2,4-dichlorophe	4,648
130	Triisopropanolammonium salt of 2-p	875
144	Trinexapac-ethyl	690
228	Triticonazole	98
162	Vernolate	461
300	Vinclozolin	11
288	Water conditioning agent	16
63	Zeta-cypermethrin	6,070
284	Zinc	18
269	Zinc Phosphide	27
99	Ziram	1,863
	Total	**16,503,549**

Pesticides Reported But Not Estimated[1]

Name	Type
1-Naphthaleneacetic Acid, Sodium Salt	Incecticide
2-Phenylethyl propionate	Incecticide
5-Chloro-2-methyl-3(2H)-isothiazolone	Bacteriocide
6-Benzyladenine	Regulator
Ancymidol	Incecticide
Bacillus thuringiensis subsp. Kurstaki	Incecticide
Benefin	Herbicide
Bentazon	Herbicide
Bioallethrin	Incecticide
Brodifacoum	Rodenticide
Bromethalin	Rodenticide
Capsaicin	Repellent
Carboxin	Fungicide
Chlorophacinone	Rodenticide
Cholecalciferol	Rodenticide
Clofentezine	Incecticide
d-Allethrin	Incecticide
Diatomaceous Earth	Incecticide
Difethialone	Rodenticide
Dimethyl Benzyl Ammonium Chloride	Fungicide
Dimethyl Ethyl-Benzyl Ammonium Chloride	Fungicide
d-trans-Allethrin	Incecticide
Fenoxaprop-P-ethyl	Herbicide
Fenoxycarb	Incecticide
gamma-Cyhalothrin	Incecticide
Garlic Oil	Repellent
Gibberellin A4 mixt. with Gibberellin A8	Plant Growth Regulator
Imazamox	Herbicide
Imibenconazole	Fungicide
Methiocarb	Incecticide
Octanoic Acid	Herbicide
Paraffinic oil	Incecticide
Peroxyacetic Acid	Herbicide
Picloram	Herbicide
Pyridine	Incecticide
Resmethrin	Incecticide
Rotenone	Incecticide
Sodium hypochlorite	Fungicide
Streptomyces lydicus	Incecticide
Sulfluramid	Incecticide
Tembotrione	Herbicide
Tert-butyl(E)-a-(1,3-dimethyl-5-phenoxyp	Incecticide
Tetramethrin	Incecticide
Thiencarbazone-methyl	Herbicide
Trichoderma harzianum Rifai	Fungicide
Tricosene	Incecticide
Triflimizole	Fungicide
Triforine	Fungicide
Uniconazole-P	Plant Growth Regulator
Warfarin	Rodenticide

[1] Reported amounts less than 1 lb.

Top 20 Reported Pesticides in 2011 Compared to 2004, 2000, 1997, 1994

Rank	Pesticide Common Name	Pounds of Active Ingredient Used					Usage Ranking for			
		2011	2004	2000	1997	1994	2004	2000	1997	1994
1	Chromic acid	5,145,298	-	-	-	-	-	-	-	-
2	Arsenic Pentoxide	3,718,525	-	-	-	-	-	-	-	-
3	Copper(II) oxide	2,358,000	-	-	-	-	-	-	-	-
4	Glyphosate	721,154	2,821,085	950,269	366,496	410,291	1	4	5	5
5	S-Metolachlor	555,807	872,768	109,566	1/	1/	5	17	-	-
6	2,4-D	439,538	199,141	225,426	168,723	226,054	10	9	9	8
7	Atrazine	381,321	1,109,475	618,515	487,837	1,166,064	3	5	4	3
8	Dimethoate	243,677	2,211	13	17,019	20,174	92	264	32	41
9	Imidacloprid	231,323	128,707	131,773	2,113	186	12	15	102	173
10	Simazine	200,734	72,883	301,427	172,911	153,240	19	7	8	12
11	Sulfuryl Fluoride	183,620	-	286	18,866	4,071	-	193	30	90
12	Cupric Oxide	170,007	46,277	1,775,876	1,026,000	1,126,997	24	2	2	4
13	Prodiamine	145,979	2,921	18,190	2,065	537	84	44	104	146
14	Mineral Oil	141,270	-	-	-	-	-	-	-	-
15	Paraquat dicholride	137,874	127,869	156,131	141,262	175,607	13	11	11	11
16	Glycine, N-(phosphonomethyl)-	129,218	-	-	-	-	-	-	-	-
17	Trifluralin	125,501	36,019	34,509	9,657	36,895	32	29	48	28
18	Etridiazole	118,384	1,217	5,325	1,368	191	116	81	127	171
19	Cypermethrin	97,844	63,871	57,280	14,983	5,637	20	22	35	78
20	MCPP	85,625	13,130	34,366	58,544	25,656	51	30	17	36

Top 10 Reported Insecticides in 2011 Compared to 2004, 2000, 1997, 1994

		Pounds of Active Ingredient Used					Usage Ranking for			
Rank	Pesticide Common Name	2011	2004	2000	1997	1994	2004	2000	1997	1994
1	Dimethoate	243,677	2,211	13	17,019	20,174	-	-	-	-
2	Imidacloprid	231,323	128,707	131,773	2,113	186	-	-	-	-
3	Cypermethrin	97,844	63,871	57,280	14,983	5,637	-	-	-	-
4	Boric Acid	58,573	488	5,194	2,817	47,992	-	-	-	-
5	Permethrin	53,361	38,038	86,681	82,730	82,985	-	-	-	-
6	Bifenthrin	34,527	1,307	2,351	304	90	-	-	-	-
7	Fipronil	21,380	15,696	78	-	-	-	-	-	-
8	Dichlorvos	13,506	-	-	-	-	-	-	-	-
9	beta-Cyfluthrin	11,267	-	-	-	-	-	-	-	-
10	Petroleum Oils	10,247	47,641	229,896	166,646	221,603	-	8	10	9

Top 10 Reported Herbicides in 2011 Compared to 2004, 2000, 1997, 1994

		Pounds of Active Ingredient Used					Usage Ranking for			
Rank	Pesticide Common Name	2011	2004	2000	1997	1994	2004	2000	1997	1994
1	Glyphosate	721,154	2,821,085	950,269	366,496	410,291	1	4	5	5
2	s-Metolachlor	555,807	872,768	109,566	1/	1/	5	1	-	-
3	2,4-D	439,538	199,141	225,426	170,559	226,054	10	9	9	8
4	Atrazine	381,321	1,109,475	618,515	487,837	1,166,064	3	5	4	3
5	Simazine	200,734	72,883	301,427	172,911	153,240	-	7	8	-
6	Prodiamine	145,979	2,921	18,190	2,065	537	-	-	-	-
7	Paraquat dichloride	137,874	127,869	156,131	141,262	175,607	-	-	-	-
8	Glycine, N-(phosphonomethyl)-	129,218	-	-	-	-	-	-	-	-
9	Trifluralin	125,501	36,019	34,509	9,657	36,895	-	-	-	-
10	MCPP	85,625	13,130	34,366	58,544	25,656	-	-	-	-

Top 10 Reported Fungicides in 2011 Compared to 2004, 2000, 1997, 1994

		Pounds of Active Ingredient Used					Usage Ranking for			
Rank	Pesticide Common Name	2011	2004	2000	1997	1994	2004	2000	1997	1994
1	Mineral Oil	141,270	-	-	-	-				
2	Etridiazole	118,384	1,217	5,325	1,368	191	-	-	-	-
3	Chlorothalonil	61,069	1,529,493	115,194	48,331	76,600	2	-	-	-
4	Sulfur	38,701	6,959	12,088	14,178	11,576	-	-	-	-
5	Mancozeb	30,280	254,254	38,107	37,343	17,572	6	-	-	-
6	Thiophanate-methyl	24,138	130,637	19,939	10,747	6,502	-	-	-	-
7	1H-1,2,4-Triazole-1-ethanol, .	21,084	-	-	-	-	-	-	-	-
8	Dithiocarbamate	8,798	-	-	-	-	-	-	-	-
9	Captan	7,127	81,816	22,095	16,412	24,694	-	-	-	-
10	Propiconazole	7,071	18,861	50,029	23,990	11,045	5	-	-	-

1/ Not reported.

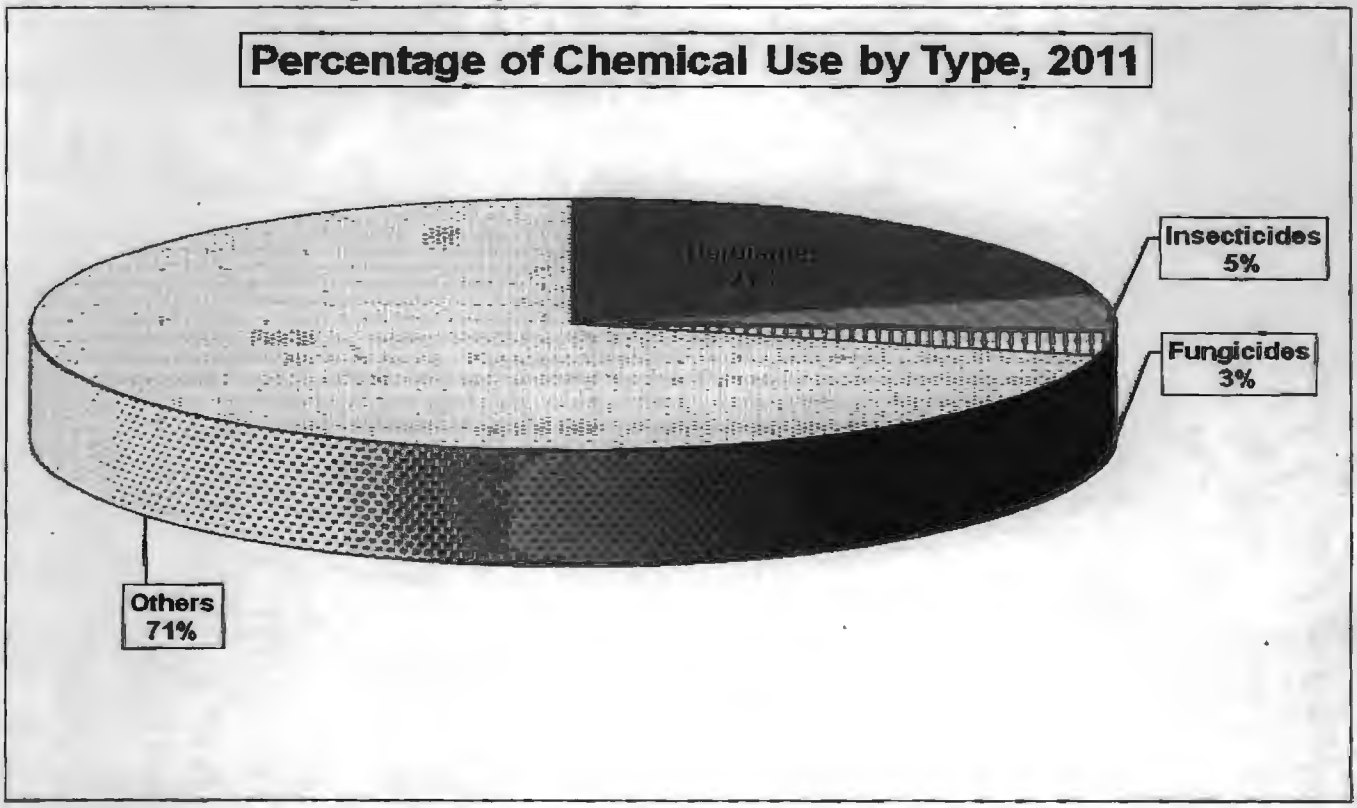
Comparison of Pesticide Use Classes Reported in 1994, 1997, 2000, 2004, 2011

Pesticide Class		1994	1997	2000	2004	2011	Percent Change 94-11	97-11	00-11	04-11
Total Usage	*pounds*	13,881,629	10,331,821	17,123,643	10,722,796	16,503,533	19%	60%	-4%	54%
Herbicides	*pounds*	5,677,775	3,195,407	4,619,656	6,310,097	3,406,867	-40%	7%	-26%	-46%
	% of Total	41%	31%	27%	59%	21%				
Insecticides	*pounds*	997,913	465,729	1,104,249	875,511	831,769	-17%	79%	-25%	-5%
	% of Total	7%	5%	6%	8%	5%				
Fungicides	*pounds*	301,612	199,373	599,556	3,387,026	538,940	79%	170%	-10%	-84%
	% of Total	2%	2%	4%	32%	3%				
Wood Preservatives	*pounds*	6,769,673	6,156,000	10,655,541	49,879	11,221,823	66%	82%	5%	22398%
	% of Total	49%	60%	62%	[3]	68%				
Antifoulants	*pounds*	27,045	1,520	3,606	46,316	[1]	------	------	------	------
	% of Total	[3]	[3]	[3]	[3]	[1]				
Others [2]	*pounds*	107,611	313,792	141,035	53,967	504,134	368%	61%	257%	834%
	% of Total	1%	3%	1%	1%	3%				

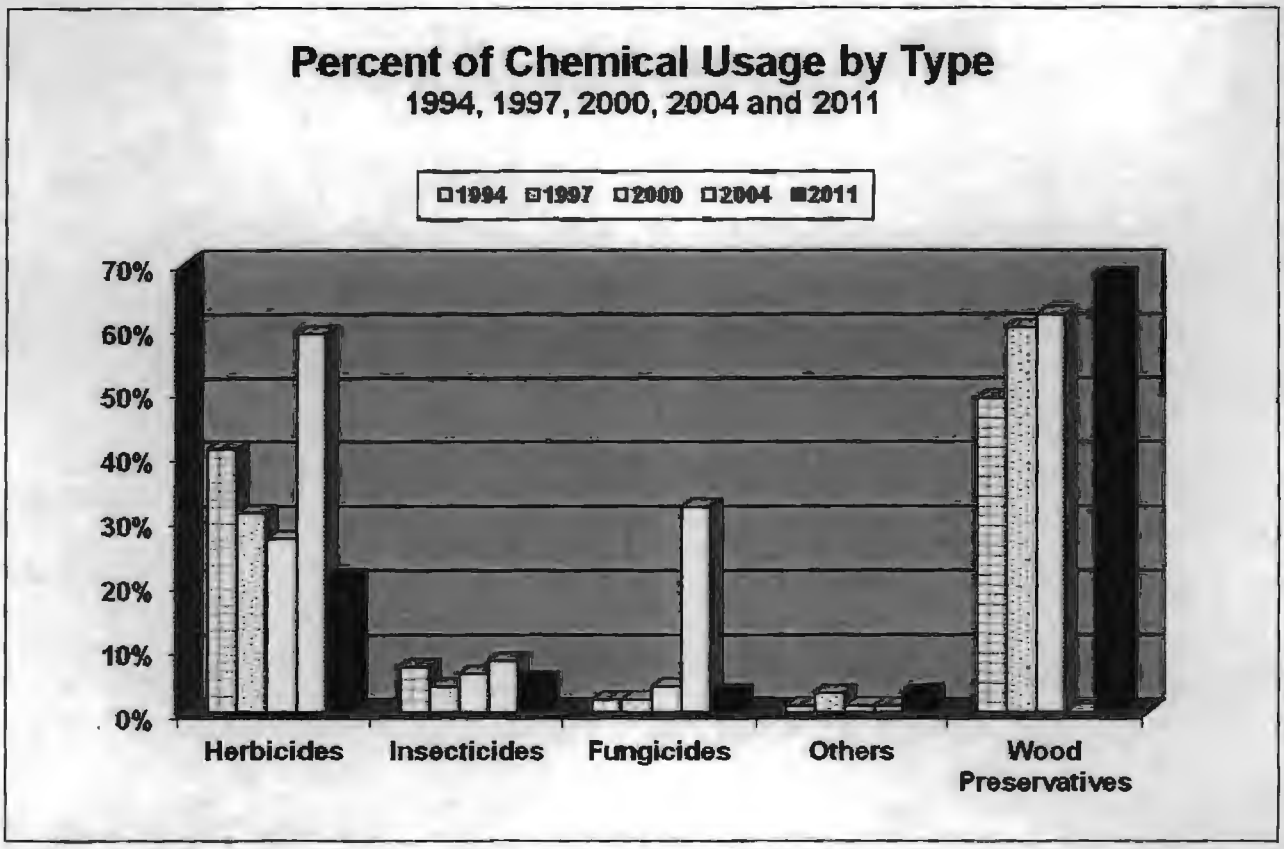
[1] Not reported.

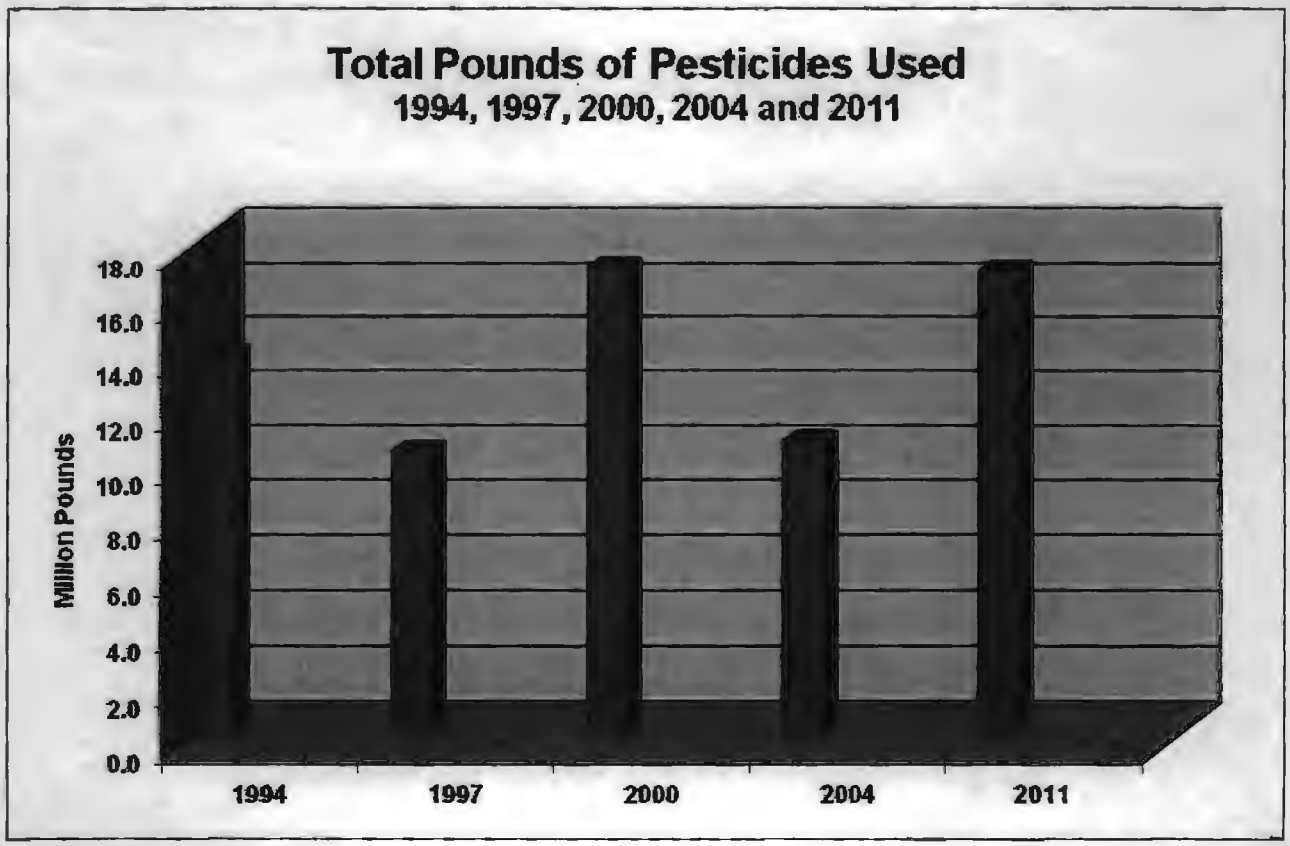
[2] Others include Algicides, Bactericides, Fumigants, Growth Regulators, Miticides, Molluscicides, Repellents, and Rodenticides.

[3] Percentage too small to publish.



[1] Others includes antifoulants and wood preservatives.


Percent of Chemical Usage by Type
1994, 1997, 2000, 2004 and 2011
1994
1997
2000
2004
2011
70%
60%
50%
40%
30%
20%
10%
0%
Herbicides
Insecticides
Fungicides
Others
Wood Preservatives


Total Pounds of Pesticides Used
1994, 1997, 2000, 2004 and 2011
Million Pounds
18.0
16.0
14.0
12.0
10.0
8.0
6.0
4.0
2.0
0.0
1994
1997
2000
2004
2011

Integrated Pest Management (IPM)

Farmer and Private Applicators

	Year	Never	Sometimes	Almost Always	Not Applicable
		percent			
Monitoring practices such as scouting for pests, soil testing, field mapping, etc	2011	13	13	56	18
	2004	10	15	49	26
Avoidance practices such as crop rotation, alternate planting dates, companion cropping, trapping, etc.	2011	11	17	54	18
	2004	8	16	51	25
Preventative practices such as mowing, burning, chopping, tillage, etc.	2011	10	25	50	15
	2004	7	21	52	20
Suppression practices such as biological pesticides, mating disruptors, beneficial organisms, genetically modified products, etc.	2011	27	24	27	22
	2004	31	26	16	27

UNIVERSITY OF MARYLAND EXTENSION
Solutions in your community

Pesticide Information Leaflet

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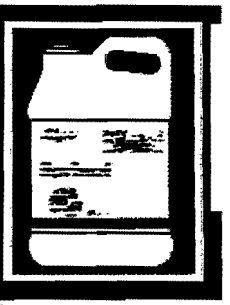
College Park, MD 20742
(301) 405-3913

College of Computer, Mathematical & Natural Sciences
College of Agriculture & Natural Resources



Check out the Pesticide Education and Assessment Program web site at http://pesticide.umd.edu

No. 35: Synopsis of the Maryland Pesticide Applicators Law and Regulations

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Revised June 2012 (orig. Sep. 2000)

PURPOSE OF THE LAW AND REGULATIONS

The Maryland Department of Agriculture (MDA) Pesticide Regulation Section is designated as the lead agency for enforcement and has the following responsibilities: regulating the use, sale, storage, and disposal of pesticides; ensuring that pesticides are applied by competent individuals; establishing guidelines for the application of pesticides; and certifying pesticide applicators. *This leaflet presents an abridged version of the regulations for quick reference of key requirements; it is not intended as a substitute for the actual regulations.* For more detailed information, readers should obtain a copy of the Code of Maryland Regulations 15.05.01, Regulations Pertaining to the Pesticide Applicator's Law, and/or Code of Maryland Regulations 15.05.02, Regulations Pertaining to Integrated Pest Management and Notification of Pesticide Use in a Public School Building or on School Grounds. Both publications are available from the Maryland Department of Agriculture, Pesticide Regulation Section, 50 Harry S. Truman Parkway, Annapolis, MD 21410, or on the internet through the Department's website at www.mda.state.md.us or directly under the Office of the Secretary of State, Division of State Documents at http://www.dsd.state.md.us/comar/subtitle_chapters/15_Chapters.aspx.

GENERAL REGULATIONS

- Pesticides must be used in strict accordance with label directions.

- Anti-siphon devices or back-flow preventers are required on all pest control equipment to prevent the flow of a pesticide into a water system.

- Pesticide applicators must consider alternative pest control measures, such as mechanical, cultural, and biological control.

- Precautions must be observed in the handling, use, storage, and disposal of pesticides and their containers to prevent off-target movement and/or harm to humans, animals, and the environment.

- Safety equipment indicated on the pesticide label must be provided for the protection of pesticide applicators.

- MDA must be notified immediately of any accident or spill involving a pesticide.

- If a pesticide concentrate is temporarily stored or transported in a service container, the container must have a securely attached label with the following information:

    - common or chemical name of pesticide;
    - U.S. EPA Registration number;
    - signal word (Danger, Warning, or Caution); and
    - percent concentration.

- If a pesticide that does not require further dilution is stored or transported in a service container as an end-use dilution, its container (excluding application equipment) must have a securely attached label listing the following information:

    - common or chemical name of the pesticide preceded by the words "Diluted" or "End-Use Concentrate";
    - U.S. EPA Registration number;
    - signal word ("Danger", "Warning", or "Caution"); and
    - percent concentration.

- A copy of the pesticide label must be on hand at the time of pesticide applications.

- Each vehicle carrying pesticides or pest control devices must have the business name and business license number displayed on both sides of the vehicle.

- It is a violation to apply a pesticide to the property of any person without the expressed permission of the property's owner or other person with authority to exercise control, management, or possession of the property

GENERAL STORAGE REQUIREMENTS

- All pesticide storage areas must meet the following minimum requirements:

  - storage areas must be locked and secured to prevent unauthorized entry;
  - pesticides should be stored in a separate building, or at a minimum physically separated by a barrier from food, feed, and fertilizer;
  - each storage area must be posted with a warning sign;
  - pesticides must be stored in a dry, well ventilated area;
  - pesticide storage areas must be kept clean;
  - all pesticide containers must be labeled and free of leaks and tears;
  - each storage area must have an appropriate fire extinguisher;
  - there must be enough absorbent material available to handle a spill of the largest container in storage; and
  - storage areas must be at least 50 feet from any water well, or have secondary containment.

TRANSPORT REQUIREMENTS

- All pesticide containers and application equipment must be secured to prevent shifting or release of pesticides.

- Pesticides shall not be placed or carried in the same compartment as the driver, food, or feed, unless in a manner that provides adequate protection for safety and health of passengers.

- A pesticide container cannot be used for any purpose other than containing the original product, unless the label states otherwise.

PESTICIDE INFORMATION FOR THE CUSTOMER

- When a pesticide is applied or at the time a customer enters into a contract for pest control, the licensee must provide the customer with the following written information:

  - name of the licensee;
  - Maryland pesticide business license number;
  - licensee's telephone number;
  - Maryland Poison Center telephone number;
  - common name of the active ingredient applied; and

  one of the following:

  - an original or legible copy of the current pesticide product label;

    or

  - an original or a legible copy of that portion of the current pesticide product label or labeling that contains precautionary statements regarding hazards to humans or animals and environmental hazards, if any;

    or

- a document containing appropriate health, safety, or precautionary information taken from the pesticide label and approved by MDA before its distribution.

PESTICIDE LICENSING AND CERTIFICATION REQUIREMENTS

- A pesticide business license is required of each business providing pest control service. The business license is obtained from MDA by completing an application, designating a certified pest control applicator, providing proof of insurance, paying a fee, and renewing annually on July 1.

- A pest control consulting license is required of any business providing pest inspections or identification of pests, or making pesticide recommendations. The consulting license is obtained from MDA by: completing an application, designating a certified pest control consultant, paying a fee, and renewing annually on January 1.

- A Not-for-Hire license is required of facilities where pest control services are performed by the owner or employees on the facility's property where the property is open to, or routinely used or enjoyed by, members of the public. This applies to private golf courses and country clubs whose employees apply either general or restricted use pesticides in the maintenance of the course. The Not-for-Hire license is obtained from MDA by: completing an application, designating a certified pest control applicator, paying a fee, and renewing annually on July 1.

- A public agency permit is required of any public agency (a unit of local, State or Federal government) whose employees apply pesticides. The permit is obtained from MDA by: completing an application, designating a certified public agency applicator, and renewing annually on July 1.

- Private applicator certification is required of any farmer, nurseryman, etc. who intends to use a restricted use pesticide on his or her own property for the purpose of producing an agricultural commodity. Private applicator certification is obtained from MDA by: passing an examination and paying a fee. Certification must be renewed every 3 years by reexamination or by participating in an MDA approved training session within 12 months before expiration of the current certificate.

- Commercial applicator certification for pest control applicators, pest control consultants, and public agency applicators is obtained from MDA by: completing an application, having 1 year experience or a degree in a science related field of study acceptable to MDA, passing an examination on core and category material, and paying a base fee plus a fee for each additional pest control category. Certification must be renewed each year on July 1, or by January 1 for consultants by participating in an MDA approved training session within the past year or by reexamination.

PEST CONTROL EMPLOYEES

- Within 30 days of employment, all employees who perform pest control services must be registered with MDA by providing the employees name, social security number, and a 1-inch color photo, and by submitting verification of training.

- Noncertified employees must complete a training program within 30 days of employment and before registration with MDA. The training program must include the following topics:

    - pesticide laws and regulations;
    - label comprehension;
    - safety and emergency procedures;
    - proper pesticide handling and storage;
    - environmental and health concerns;
    - integrated pest management (IPM) principles;
    - pest identification and control recommendations; and
    - pesticide application techniques.

An employee who has not successfully completed training in accordance with the aforementioned conditions may perform pest control services if a certified applicator or registered employee is physically present at the time and place the pesticide is applied by the untrained employee.

RECORDKEEPING REQUIREMENTS

- Private applicators, commercial applicators, pest control consultants, and public agencies must keep records on all pesticides applied or recommended. Commercial applicators, pest control consultants, and public agencies must also keep records of all pest identifications made. The records must be held for 2 years to be available to MDA on request. The following must be recorded, when applicable:

    - name of applicator or consultant;
    - date of application, recommendation, or pest identification;
    - pest and type of plant, animal or structure;
    - amount of area treated (acreage, square footage, cubic footage, linear footage, or numbers of plants or animals or a description of the area or structure treated with the acreage, square footage, cubic footage, or linear footage recorded when label instructions specify these measurements);
    - address of treated property;
    - name of owner or tenant of property;
    - common name of pesticides used or recommended;
    - rate and concentration of pesticide used or recommended;
    - total amount of pesticide used;
    - EPA registration number of the product;
    - *type of equipment used;
    - *time of day of application; and

- *wind direction and estimated velocity, and weather conditions at the site when the pesticide was applied. (This information is not required if the application consists of baits in bait stations, or is made inside or within 3 feet of a structure.)

(* Items marked with an asterisk are required to be recorded by commercial applicators, pest control consultants, and public agencies, but are not required for private applicators.)

- Dealers who sell or distribute restricted use pesticides must maintain records on the sale or distribution of restricted use pesticides for 2 years and make them available to MDA on request. The following information must be recorded:

  - name of pesticide or pesticides sold or distributed, including formulation;
  - quantity sold or distributed;
  - date of sale or distribution;
  - name and address of purchaser or receiver; and
  - name and address of certified applicator, if different from above.

ADDITIONAL REQUIREMENTS

- Signs must be posted at the time of application whenever a pesticide is applied to a lawn or exterior landscape plant. Signs must be posted at primary entrances to the property treated, or in the case of spot treatments at the site of application. There are variances for golf courses, parks, cemeteries and similar sites. The sign must be 4" x 5" in size and conform to a specific layout and design. The following information must be written on the back of the sign:

  - business name or agency name making the application;
  - date of application; and
  - business or agency telephone number.

- MDA maintains a list of individuals who have a medical condition that may be aggravated by the application of a pesticide. Individuals on the list must be notified prior to any pesticide application that is made to a contiguous or adjacent property of a registered individual. This requirement only pertains to those businesses or public agencies that are licensed or permitted in Category 3 (Ornamental and Turf).

- Pest inspections must be performed in accordance to a set of standards. Each inspection must include a visual observation and thorough examination of the readily accessible areas, objects, materials, structures, or part of structures that are inspected. The inspector must report all findings in writing and include any findings or visible evidence of the target pest. Any inspection for a wood destroying insect must include a diagram of the structure showing the locations where the pest was found. Inspections being performed for a property transfer or loan must be conducted by sounding and probing readily accessible structural members for the presence of wood destroying insects using inspection form MD-1. Copies of all inspection reports must be maintained for 2 years and made available to MDA upon request.

VIOLATIVE ACTS AND PENALTIES

- MDA may suspend, revoke or deny any license, certificate, permit or registered employee identification card for violating any provision of the Maryland Pesticide Applicators Law and Regulations, or the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). [Refer to section 15.05.01.10 (2) through 15.05.01.10 (14) of the Regulations for further violations.]

- Any person violating any provision of the Pesticide Applicators Law or Regulations is guilty of a misdemeanor and, upon conviction, is subject to a fine up to $1,000 or imprisonment up to 60 days.

- In lieu of or in addition to suspension of the license, permit, or certificate, the Secretary of Agriculture may impose a penalty of not more than $2,500 for a first violation and not more than $5,000 for each subsequent violation. The total penalties imposed on an individual for violations that result from the same set of facts and circumstances may not exceed $25,000.

INTEGRATED PEST MANAGEMENT AND NOTIFICATION REQUIREMENTS FOR PESTICIDE APPLICATIONS TO PUBLIC SCHOOL GROUNDS

- Each county board of education must implement in its schools an Integrated Pest Management (IPM) system approved by MDA. [A broad definition of IPM is a pest control program that (a) utilizes inspections and (b) incorporates different methods of pest control such as sanitation, structural repairs, and other non-chemical methods, and pesticides when warranted, to (c) keep pests from causing economic, health-related, or aesthetic damage.]

- Each school system must designate a contact person to answer questions about the pest management program and to maintain a file of pesticide product labels and Material Safety Data Sheets (MSDS).

- At the beginning of each school year, public schools must send a notice including information about pesticides used in schools and on school grounds to the parent or guardian of each student in primary and secondary schools.

- Schools must provide notification, at least 24 hours before a pesticide is applied, or within 24 hours after an emergency pesticide application is made, to:

    - all parents or guardians of elementary school students, and staff members employed by elementary schools;
    - parents or guardians of middle school or high school students, and staff members employed by these institutions, who have submitted a written request to receive notice of pesticide applications.

- The information to be provided to the above individuals includes:

    - common name of the pesticide applied;
    - location, time, and date of application;

- description of potential adverse effects listed on the Material Safety Data Sheet (MSDS) for the pesticide;
- a statement that EPA recommends that persons who are potentially more sensitive should avoid any unnecessary pesticide exposure; and
- reason for emergency application (if applicable).

- For pesticide applications made on school grounds, the notice of planned date and time of application may specify that weather conditions or other extenuating circumstances may cause the actual date of application to be postponed to a later date or dates. If the actual date of application is more than 14 days later than the original planned date of the application, a new noticed must be issued.

- Middle schools and high schools must provide in-school notification, by oral announcements or written notice, before a pesticide is applied in a school building or on school grounds. A sign or notice must be posted at the primary entrance to the school or in a central location, must remain for at least 48 hours after an application, and must include the following information:

  - the statement, "Caution - Pesticide Application";
  - common name of pesticide applied;
  - location and date of pesticide application;
  - contact person for additional information, including information of potential adverse effects.

When a pesticide application is made on school grounds, a sign must be posted at the time of the application at each primary access to the school property. If a spot or limited area pesticide application is made, a sign may be posted at the location where the pesticide application was made. The sign must remain posted for at least 48 hours following the application.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

MAR 3 1 2014

OFFICE OF CHEMICAL SAFETY
AND POLLUTION PREVENTION

VIA EMAIL AND POST

To Addressees Listed in the Enclosure to this Letter

Subject: EPA Response to "Pesticides in the Air – Kids at Risk: Petition to EPA to Protect Children from Pesticide Drift"

Dear Petitioners:

Enclosed please find the Agency's response to your petition, "Pesticides in the Air – Kids at Risk: Petition to EPA to Protect Children from Pesticide Drift," submitted in October 2009 on behalf of a number of health and environmental organizations.[1] As you may recall, EPA posted the petition to the public docket (www.regulations.gov, docket ID EPA-HQ-OPP-2009-0825) in November 2009 and opened a comment period that ran 120 days in its entirety. In summary, and as related more specifically in our response, the petition asked EPA to account for children's exposures to pesticide drift and volatilization in its risk assessments and to take certain steps to reduce the risks from these exposures.

On July 24, 2013, you filed a writ of mandamus lawsuit against EPA alleging that EPA had unreasonably delayed responding to the petition. The parties agreed to stay the case as long as EPA promised to issue a final response to the Petition by March 31, 2014.[2] The enclosed response fulfills that promise.

During the several years that passed between the time the petition was submitted and the present, the Agency was actively developing drift and volatilization assessment methodologies, applying those methodologies to both fumigant and conventional pesticides, and finding ways to mitigate the risks to adults and children posed by pesticide drift and volatilization.

Recently the Agency posted to the docket and solicited comments on the methodologies it has developed for assessing the risks from pesticide drift and volatilization. The comment periods

[1] The organizations were Pesticide Action Network of North America (PANNA), United Farmworkers, Pineros y Campesinos Unidos Del Noroeste, MomsRising, Sea Mar Community Health Center, California Rural Legal Assistance Foundation, Farm Labor Organizing Committee, and Physicians for Social Responsibility.

[2] In re: Pesticide Action Network North America, et al. v. EPA, No. 13-72616 (9th Circuit)

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for both methodologies are open at this time (at www.regulations.gov; the docket ID for the drift methodology is EPA-HQ-OPP-2013-0676; for the volatilization methodology, EPA-HQ-OPP-2014-0219).

The enclosed response discusses background on the petition, the pending lawsuit, the statutory and regulatory framework for EPA's pesticide programs, how these programs are implemented, and how EPA assesses and manages the risks associated with pesticide use, including risks from spray drift and volatilization. The response also directly addresses the three major changes to current practices that were requested by the petitioners, that the Agency 1) assess the potential risks posed by drift and volatilization for children in places where they live and play, 2) accelerate the assessment of these potential risks, and 3) adopt uniform buffers for pesticides of special concern in the interim while the risk assessments are being conducted.

EPA shares the concerns expressed by the petitioners, and agrees that the risks from drift and volatilization must be accounted for, for both children and adults, and that action must be taken to mitigate any such risks. The Agency believes that the registration review program already in place is a timely, efficient, and effective way to assess and take action on these risks, and does not believe that imposing requirements for uniform, interim buffers is scientifically supportable or defensible. Thus the Agency grants in part and denies in part the petitioners' requests.

We thank you for your interest and for your role in advancing awareness of the risks that pesticide drift and volatilization can pose to children. We hope that you will review and provide constructive comments on the newly published assessment methodologies for addressing those risks. Should you desire further information on the Agency's approach to assessing drift and volatilization, please contact Jill Bloom of my staff, at bloom.jill@epa.gov or (703) 308- 8019, and she will do her best to assist you.

Sincerely,

Martha Monell, Acting Director
Office of Pesticide Programs

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Agency Response to
"Pesticides in the Air – Kids at Risk:
Petition to EPA to Protect Children from Pesticide Drift (2009)"

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I. Executive Summary

This document presents EPA's response to a petition that asked the agency to: 1) evaluate the risks to children exposed to pesticides through drift and volatilization, 2) establish a separate process or modify its pesticide re-evaluation process to expedite assessment and management of these risks, and 3) for certain types of pesticides, require the adoption of "one size fits all" buffer zones between treated areas and places where children congregate. EPA grants in part and denies in part this petition.

EPA agrees with Petitioners that individuals including children may be exposed to pesticides through drift and/or volatilization; that these exposures can occur in places where children live, go to school, play, or are otherwise present; and that, apart from occupational activities, children (depending on certain factors, including age) may experience higher levels of pesticide exposure relative to their size than do adults. Furthermore, the Agency agrees with the petitioners that it should conduct pesticide-specific assessments of the risks that include drift and volatilization exposures, as appropriate, and that if warranted, the Agency will take action to mitigate those risks. The steps the Agency has been taking to address these exposures are discussed later in this document.

Petitioners define the term drift to include "any airborne movement of pesticides away from a target site during and/or after application, including the airborne movement of pesticide droplets, pesticide powders, volatilized vapor-phase pesticides, and pesticide contaminated soil particles." In Sections VI through VIII of this response, EPA defines drift and volatilization as they relate to our risk assessments. The definitions differ mainly because the Agency draws a distinction between the off-site movement of spray droplets or pesticide particles during and shortly after the application process ("pesticide drift") and the movement of pesticide active ingredients as a vapor or gas that can occur for longer time after application is completed ("volatilization"). Both processes describe movement of pesticides through the air. The type of pesticide drift on which this response focuses is "spray" drift, the off-site movement of aerosols originating with pesticides applied as liquids, because spray drift is more likely to occur than the drift of pesticides in solid form, and when it does, it generally poses greater risk. EPA's efforts to assess and manage pesticide drift consequently are concentrated on spray drift.

EPA denies the Petitioners' request that EPA use a process outside of the ongoing pesticide re-evaluation process, as currently scheduled, to assess and manage spray drift and volatilization risks. The Petitioners suggest that the Agency should use alternative approaches that reprioritize pesticides for registration review or speed up risk assessments. The Agency believes that such adjustments to the registration review process are not needed and do not represent an efficient use of limited Agency resources.[1]

[1] If specific and significant concerns arise about an individual pesticide not in registration review at the time, the Agency can utilize other processes as appropriate to assess and mitigate risks (as needed). These processes are described later in this document and are not intended to replace the systematic and regular actions that constitute registration review. See IV.C.iii, which discusses regulatory options for cases in which the Agency has determined that a registered pesticide no longer satisfies the statutory standard.

EPA also denies the petition as it relates to Petitioners' request that EPA immediately adopt interim prohibitions on the use of certain pesticides that they allege are toxic and may be prone to drift or volatilization, near homes, schools, parks, and daycare centers or wherever children congregate. EPA instead believes that case-by-case, chemical-specific risk assessment is a sound, science-based approach, consistent with the Agency's mandate, that yields a more realistic representation of actual risks and facilitates the identification of what, if any, mitigation measures (potentially including no-spray buffers) are needed to protect potentially exposed individuals.

The response to the petition is organized in the following manner. After this executive summary, EPA follows with two sections (II and III) that discuss background on the petition, the pending lawsuit brought by the Petitioners, and the statutory and regulatory framework as it relates to EPA's implementation of its pesticide programs. The next two sections of the response (IV and V) outline the Agency's pesticide regulatory programs and how EPA assesses and manages the risks associated with pesticide use. The following sections (VI through VIII) revisit the Agency's terminology for describing the off-site movement of pesticides through the air via spray drift and volatilization and explain how the risks of each are assessed and managed. The next section (IX) contains EPA's response to the Petitioners' request for three changes to the Agency's current practices. The last section (X) provides EPA's conclusion.

II. Background

A. Petition History and Major Claims by Petitioners

In October 2009, a group of health and environmental organizations[2] ("Petitioners") jointly filed a petition entitled, "Pesticides in the Air- Kids at Risk: Petition to EPA to Protect Children from Pesticide Drift ("Petition"). The Petition alleged that EPA has failed to address children's exposures to and potential risks from pesticide drift and volatilization. More specifically the Petitioners ask EPA to:

1. fully and expeditiously evaluate the exposure of children to pesticide drift or vapors that originate from agricultural applications and travel to areas where children congregate, such as homes, parks, schools, and daycare centers. Furthermore, the Petitioners ask that the Agency act to ensure that children are protected from aggregate pesticide exposures, including exposures to drift;
2. implement an accelerated schedule (relative to the schedule for registration review) for completing drift assessments and modifying registrations that prioritizes assessments based on the suspected degree of risk posed by the pesticide drift and volatilization; and

[2] The organizations include Pesticide Action Network North America (PANNA), United Farm Workers, Pineros Y Campesinos Unidos Del Noroeste, Moms Rising, Sea Mar Community Health Center, California Rural Legal Assistance Foundation, Farm Labor Organizing Committee, and Physicians for Social Responsibility.

3. immediately adopt interim prohibitions on the use of toxic drift-prone pesticides such as organophosphates and n-methyl carbamates and certain other pesticides that are used near homes, schools, parks, and daycare centers or wherever children congregate.[3]

On November 4, 2009, EPA issued a Federal Register Notice requesting public comment on the assertions and requests made in the October 2009 petition. The comment period for the petition closed on January 4, 2010. EPA has reviewed the comments received.[4]

B. Lawsuit

On July 24, 2013, the Petitioners filed a writ of mandamus lawsuit against EPA alleging that EPA had unreasonably delayed responding to their 2009 Petition. EPA and the Petitioners agreed to stay the case. In EPA's unopposed motion to stay the case, the Agency promised to issue a final response to the Petition by March 31, 2014.[5] This response fulfills that promise.

III. Statutory and Regulatory Background/Framework

EPA regulates pesticides under the Federal Insecticide, Fungicide, and Rodenticide Act ("FIFRA"), 7 U.S.C. §§ 136-136y, and the Federal Food, Drug, and Cosmetic Act ("FFDCA"), 21 U.S.C. §346a. FIFRA sets forth a federal licensing scheme for the sale, distribution and use of pesticides; FFDCA establishes the mechanism and standards by which EPA must set tolerances (allowable levels) for pesticide residues in food. As a general matter, under FIFRA section 3, before a pesticide can be distributed or sold in the United States, it must be registered. Petitioners' administrative petition implicates both statutes.

A. The Federal Insecticide, Fungicide, and Rodenticide Act

The principal purpose of FIFRA is to regulate the sale, distribution and use of pesticides (through registrations) while protecting human health and the environment from unreasonable adverse effects associated with pesticides. *See generally* FIFRA section 3. Under FIFRA, EPA registers a pesticide only after conducting a scientific review of the risks, and when appropriate, benefits of that pesticide to determine whether the use of the pesticide causes "unreasonable adverse effects" to human health or the environment.[6] Registration and registration review decisions under section 3, reregistration decisions under section 4, and cancellation decisions under section 6 are governed by the same statutory standard, which generally is referred to as "risk-benefit" balancing, *i.e.*, a pesticide must not pose "any unreasonable risk to man or the

[3] The petitioners believe these interim measures also should apply to "all other pesticides that are (1) registered for application by ground sprayers, broadcast equipment, and/or aerial equipment; and (2) suspected of causing acute poisonings, cancer, endocrine disruption, developmental effects, and/or reproductive effects.

[4] The Petition docket is found at http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2009-0825.

[5] In re: Pesticide Action Network North America, et al. v. EPA, No. 13-72616 (9th Circuit)

environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide." FIFRA §§ 3(c)(5) & 2(bb); 7 U.S.C. §§ 136a(c)(5) & 136(bb). If this standard is not satisfied, EPA may not register the pesticide and existing pesticides are subject to modification or cancellation. *See* FIFRA §§ 3(c)(5) & 6(b); 7 U.S.C. §§ 136a(c)(5), & 136d(b).

In order to properly evaluate pesticide applications, FIFRA and its implementing regulations generally require applicants for registration to submit or cite to a significant body of toxicity and exposure data for the pesticides for which they are seeking registration. *See* 7 U.S.C. § 136a(c)(2)(A) (directing EPA to publish guidelines for submissions by applicants); 40 C.F.R. §§ 158.1 *et seq.*161.20 *et seq.* (setting forth information to be provided by applicants).

While EPA must consider a broad range of factors in determining whether a pesticide meets this standard, the balancing of the various risks and benefits of the pesticide, and consideration of inherent policy questions, is left largely to the discretion of EPA: "[W]ithin broad limits, the [A]dministrator has latitude not merely to find facts, but also to set policy in the public interest. Like most regulatory statutes,... FIFRA confers broad discretion on the [Administrator]." *Wellford v. Ruckelshaus*, 439 F.2d 598, 601 (D.C. Cir. 1971); *See also Envtl. Def. Fund v. EPA*, 465 F.2d 528, 538 (D.C. Cir. 1972) (FIFRA empowers EPA to "take account of benefits or their absence as affecting imminency of hazard").

As part of the process of EPA's approval of a pesticide registration, the agency must review and ultimately approve proposed labeling and directions for use for each pesticide. *See* FIFRA § 3(c)(5)(B). The approved pesticide label sets forth the lawful conditions of use for a pesticide, *i.e.*, those mandated by EPA in order to ensure that the pesticide will not cause unreasonable adverse effects to human health or the environment. *See Id.* § 3(d). Indeed, it is a violation of FIFRA for any person to use a pesticide in a manner inconsistent with the EPA-approved labeling. *See Id.* § 12(a)(2)(G).

B. The Federal Food, Drug, and Cosmetic Act

In 1996, the Food Quality Protection Act ("FQPA") was enacted, amending FFDCA and FIFRA to require all pesticides the use of which results in residues on food to meet new dietary risk standards. The FQPA amended the FIFRA risk-benefit standard ("any unreasonable risk to man or the environment") to add another element to the definition of that term: "a human dietary risk from residues that result from a use of a pesticide in or on any food inconsistent with the standard under [FFDCA section 408]." *See* FIFRA § 2(bb); FFDCA § 408(b)(1). In other words, the registration of a pesticide is contingent upon its meeting the food safety standard established under FFDCA section 408, if use of the pesticide results in residues on food. Section 408 also was amended by FQPA to add protections for infants and children and to establish the estrogenic substances screening program.[7]

[7] Public Law 104– 170, 110 Stat. 1489 (1996).

Section 408 of the FFDCA authorizes EPA to establish by regulation "tolerances" setting the maximum permissible levels of pesticide residues in foods.[8] FFDCA §§ 301(a), 408(a). Tolerance setting is discussed in more detail in Section IV.B.

EPA may only promulgate a pesticide tolerance, if the tolerance is "safe." FFDCA § 408(b)(2)(A)(i). "Safe" is defined by the FFDCA section 408 to mean that "there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information." *Id.* § *408*(b)(2)(A)(ii) [emphasis added]. Section 408's reasonable certainty of no harm standard is a risk-only standard, which generally does not allow consideration of benefits.[9] Congress also amended the FFDCA to require that EPA re-assess, using the new safety standard, the existing tolerances and exemptions for all pesticide chemical residues that were in effect on August 3, 1996. *Id.* § 408(q)(1). Congress directed EPA to complete the reassessments by August 3, 2006.

Congress instructed EPA, when applying the new safety standard, to assess, among other things, the risks of pesticide chemicals based on available information concerning the special susceptibility of infants and children to pesticide chemical residues. FFDCA § *408*(b)(2)(C). To ensure that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to pesticide chemical residues, Congress mandated that EPA apply an additional ten-fold margin of safety (known as the FQPA safety factor) in setting tolerances unless reliable data show that a different margin of safety will be safe for infants and children. *Id.* The additional ten-fold margin of safety can be reduced or removed based on such a finding.

The FQPA amendments to the FFDCA also directed EPA to consider "aggregate exposure" in its decision-making. EPA has interpreted "aggregate exposure" to refer to the combined exposures to a single chemical across multiple routes (oral, dermal, inhalation) and across multiple pathways (food, drinking water, residential). As amended by FQPA, section 408(b)(2)(ii) of FFDCA requires the Agency to make a finding for each tolerance or tolerance exemption "that there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information"[emphasis added]. Section 408(b)(2)(C)(ii)(I) of FFDCA states that the Agency must find "there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to the pesticide chemical residues." Finally, section 408(b)(2)(D)(vi) directs the Agency, when making tolerance decisions, to consider "aggregate exposure levels...to the pesticide chemical residue...including dietary exposure and exposure from other non-occupational sources."

EPA reaffirms its consistent interpretation of FFDCA section 408 as requiring consideration of all exposures to pesticide residues and other related substances other than those exposures

[8] Without such a tolerance or an exemption from the requirement of a tolerance, a food containing a pesticide residue is "adulterated" under section 402 of the FFDCA and may not be legally moved in interstate commerce.

[9] Benefits may only be considered under section 408 in one very narrow circumstance not applicable in this case.

occurring in the occupational setting.[10] Relevant exposures include pesticide residues in food and water and exposures to pesticides around the home or in public from sources other than food and water.

It is important to note that Congress has expressly provided that any issue that can be raised through the FFDCA review process can only be reviewed through that process.[11] Accordingly, to the extent a petition to revoke tolerances and cancel registrations raises issues relevant to the establishment or revocation of tolerances, EPA's response to those issues may be challenged only through the administrative and judicial review procedures provided in section 408 of the FFDCA and are not reviewable under FIFRA or any other provision of law.

C. Executive Orders Cited in Petition

EPA includes the following general discussion on two Executive Orders mentioned by Petitioners as support to their claims.[12]

i. Executive Order 12898: Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations

On February 11, 1994, President Clinton issued Executive Order 12898 on Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations.[13] This Order focuses federal attention on the environmental and human health conditions of minority and low-income communities and calls on agencies to make achieving environmental justice ("EJ") part of their mission. Since the issuance of that Executive Order, EPA has been actively working to ensure that EJ issues are considered in its decision-making processes. In September 2011, EPA issued its Plan EJ 2014 strategy.[14] The strategy is the Agency's roadmap for advancing environmental justice. Based on this strategy, the Agency seeks to:

- Protect the environment and health in overburdened communities.

[10] See Imidacloprid; Order Denying Objections to Issuance of Tolerance 69 Federal Register 30042, 30073 (May 26, 2004).

[11] FFDCA § 408(h)(5); NRDC v. Johnson, 461 F.3d 164, 176 (2d Cir. 2006).

[12] These Executive Orders do not create an independent right of action against the United States.

[13] http://www2.epa.gov/laws-regulations/summary-executive-order-12898-federal-actions-address-environmental-justice

[14] See http://www.epa.gov/compliance/ej/plan-ej/for more information. The plan is not a rule or regulation. It is a strategy to help integrate environmental justice into EPA's day-to-day activities. Plan EJ 2014 has three major sections: Cross-Agency Focus Areas, Tools Development Areas, and Program Initiatives. Within these areas, EPA plans to more effectively protect human health and the environment for overburdened populations by developing and implementing guidance on incorporating environmental justice into EPA's rulemaking process. EPA also plans to enable overburdened communities to have full and meaningful access to the permitting process and to develop permits that address environmental justice issues to the greatest extent practicable under existing environmental laws. Under the two statutes at issue here, FIFRA and the FFDCA, EPA has already begun to incorporate these considerations into its licensing program.

- Empower communities to take action to improve their health and environment.
- Establish partnerships with local, state, tribal, and federal governments and organizations to achieve healthy and sustainable communities.[15]

EPA is committed to addressing risks to population groups with unique exposure pathways, such as children, farm and migrant workers, urban poor populations, rural or isolated populations, and Native Americans and Alaskan Natives [emphasis added].[16] EPA's Office of Pesticide Programs has developed an internal training program for staff that provides an overview of environmental justice issues to be considered in risk assessment. Guidance materials and the templates for risk assessment documents direct risk assessors to address environmental justice concerns specifically as a basic element of pesticide risk assessment and the Agency risk management decision-making process includes consideration of any such concerns identified for the subject pesticide.

ii. Executive Order 13045: Protection of Children from Environmental Health Risks and Safety Risks

On April 21, 1997, President Clinton signed Executive Order 13045 on the Protection of Children from Environmental Health Risks and Safety Risks.[17] This Executive Order requires all federal agencies to assign a high priority to addressing health and safety risks to children, coordinate research priorities on children's health, and ensure that their standards take into account special risks to children. While not directly cited in the Petition, it should also be noted that on October 20, 1995, EPA adopted the Policy on Evaluating Health Risks to Children (predating the Executive Order). This policy requires the EPA to consider the risks to infants and children consistently and explicitly as part of risk assessments generated during its decision making process, including the setting of standards to protect public health and the environment.[18]

In response to Executive Order 13045, EPA established the Office of Children's Health Protection ("OCHP") in May 1997. The focus of this office is to make the protection of children's health a fundamental goal of public health and environmental protection in the United States. The Agency's Office of Pesticide Programs and OCHP are committed to ensuring that the Agency's risk assessments for pesticides are protective of children's health.[19]

IV. EPA's Pesticide Regulatory Programs

[15] Petitioners claim that EPA's pesticide assessments do not adequately address environmental justice issues as directed by the 1994 EJ Executive Order (Exec. Order No. 12,898, 59 Fed. Reg. 7,629 (Feb. 11, 1994)). EPA disagrees. EPA's commitment to EJ issues is clear from its recent pronouncements on these issues.

[16] http://ajph.aphapublications.org/doi/full/10.2105/AJPH.2011.300121

[17] http://yosemite.epa.gov/ochp/ochpweb.nsf/content/whatwe_executiv.htm

[18] http://yosemite.epa.gov/ochp/ochpweb.nsf/content/policy-eval_risks_children.htm

[19] http://gao.gov/products/GAO-13-254

The following sections discuss how EPA implements its statutory obligations under FIFRA and the FFDCA.

A. EPA's Review of Pesticide Registration Applications

EPA provides an application kit[20] to assist in preparation of an application to register a pesticide. The applicant is responsible for submitting or citing all of the information and data that are required to support the registration, including proposed product labeling, and scientific data that meet the data requirements related to the specific product the applicant intends to register. In addition, the applicant provides a Confidential Statement of Formula that details the composition of the pesticide product, i.e., 1) all active ingredients, 2) all inert ingredients, 3) all impurities of toxicological significance associated with the active ingredient, and 4) all impurities found to be present at a level equal to or greater than 0.1 percent by weight of the technical grade active ingredient.[21]

The applicant will also provide EPA with draft labeling. FIFRA section 2(p)(1) defines "label" as "the written, printed or graphic material on, or attached to, the pesticide or device or any of its containers or wrappers." The term "labeling" is defined as "all labels and all other written, printed, or graphic matter –

(A) accompanying the pesticide or device at any time; or
(B) to which reference is made on the label or in literature accompanying the pesticide or device." *See* FIFRA § 2(p)(2)

Labeling includes detailed information such as the ingredients statement, warnings and precautionary statements, and directions for use. It is unlawful to sell or distribute a pesticide if any claims made for it differ from claims made on labeling required for registration. *See* FIFRA § 12(a)(1)(B). Therefore, advertising claims for a pesticide product must not contradict claims made in the product's labeling. Labeling requirements are codified in 40 CFR Part 156. EPA has developed a Label Review Manual[22] as guidance to its staff on reviewing labels.

Applicants are responsible for citing or generating all data to meet data requirements. The purpose of these data requirements is to demonstrate that the product will not cause unreasonable adverse effects on the environment. In general, these data are used to evaluate whether a pesticide has the potential to cause adverse effects on humans, non-target wildlife, and plants, as well as possible contamination of surface water or groundwater from leaching, run-off, and spray drift. For pesticides that will need a tolerance or tolerance exemption to demonstrate a reasonable certainty of no harm, additional data are needed.

Requirements may include, as applicable, data on:

- spray drift,
- residue chemistry,

[20] http://www.epa.gov/pesticides/registrationkit/

[21] http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol24/pdf/CFR-2011-title40-vol24-sec158-320.pdf

[22] http://www.epa.gov/oppfead1/labeling/lrm/

- environmental fate,
- toxicology,
- applicator exposures
- reentry protection,
- wildlife and aquatic organisms,
- plant protection,
- nontarget insects,
- product performance, and
- product chemistry.

Data requirements in support of applications for registration of a pesticide product are specified in 40 CFR Part 158.[23]

EPA's review of the application includes the assessment of the risks to human health and the environment that may be posed by the pesticide.[24]

B. Tolerance Setting

i. Process overview

A tolerance is the maximum allowable concentration of a pesticide on a particular food item. The tolerance is the residue level that triggers enforcement actions. That is, if residues are found above that level, the commodity will be subject to seizure by the government. EPA must consider a number of factors when it establishes, modifies, leaves in effect, or revokes a tolerance for a pesticide chemical residue. FFDCA § 408(b)(2)(D). The process for the establishment, modification, or revocation of tolerances, is described directly below.

A tolerance action may be initiated by EPA of its own accord[25] or in response to an administrative petition. *Id.* § 408(d)(1), (e)(1). "Any person may file with [EPA] a petition proposing the issuance of a regulation . . . establishing, modifying, or revoking a tolerance for a pesticide chemical residue in or on a food." *Id.* § 408(d)(1)(A). If EPA determines that an administrative petition meets the statutory and regulatory requirements governing petition contents, EPA publishes a notice of the administrative petition's filing. *Id.* § 408(d)(3). (The Agency will also publish a notice when the action is Agency-initiated.)

After the publication of the notice, EPA must give "due consideration" to the petition and then: i) issue a final regulation establishing, modifying, or revoking a tolerance; ii) issue a proposed regulation under the separate provisions of the FFDCA § 408(e), and thereafter issue a

[23] Data requirements are described at http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol24/pdf/CFR-2011-title40-vol24-part158.pdf; links to guidelines for the conduct of required studies are located at http://www.epa.gov/pesticides/science/guidelines.htm

[24] For further details on the pesticide registration see http://epa.gov/pesticides/factsheets/registration.htm.

[25] When, for example, the tolerance action follows a cancellation action.

final regulation after additional public notice and comment; or iii) issue an order denying the petition. FFDCA § 408(d)(4)(A). *See NRDC v. Johnson*, 461 F.3d at 173-74.

After EPA issues a regulation or order establishing, modifying, or revoking a tolerance for a pesticide chemical residue on food, any person may file objections with EPA and request an evidentiary hearing on those objections. FFDCA § 408(g)(2). After consideration of any such objections, EPA must issue a final order separately stating the action taken on each objection and whether any hearing is appropriate. *Id.* § 408(g)(2)(C). Then the Agency can conclude its deliberations and grant, modify, or deny the tolerance, as appropriate.

ii. The Tolerance Petition and Required Documentation

As discussed above, a determination on the proposed tolerance relies on the risk-only standard from FFDCA section 408. EPA must ensure that the use associated with the tolerance will not pose unreasonable risks to human health. Except in certain instances,[26] a tolerance petition request is usually accompanied by an application for registration, an application to amend the registration of a currently registered product, or an experimental use permit for the uses proposed in the petition. As risk assessments are a component of the standards for evaluating both tolerance proposals and registration actions, EPA determines whether any meaningful risks exist for the proposed uses, based on an evaluation of the applicant's petition for a tolerance. The Agency bases its tolerance decision on the aggregate exposures and risks associated with the pesticide and the use to which the petition applies. Aggregate exposure is the combined exposures to a single chemical across multiple routes (oral, dermal, inhalation) and across multiple pathways (food, drinking water, residential).[27]

iii. How the Proposed Tolerance Action is Assessed

The risks of concern that are considered in the setting of tolerances are the human health risks from aggregate exposures, which are the sum of exposures from each relevant pathway--food, drinking water, and/or residential. The assessment of human health risks is described in more detail in Section V. Aggregate risks are calculated based on varying durations of exposure. When no residential uses exist, aggregate risks are based on exposure contributions from food and drinking water for the acute and chronic durations. For residential-use pesticides, residential exposures are combined with food and drinking water exposures for each applicable duration of exposure. As discussed above, a determination on the proposed tolerance will be based on the risk-only standard from FFDCA section 408.

[26] A request for an import tolerance generally would not require an accompanying application for registration.

[27] Residential exposures include exposures associated with homes, home lawns, yards, gardens, apartments and grounds around apartment buildings, schools, schoolyards, daycare facilities, playgrounds, athletic fields, and parks and other public spaces.

In 1997, EPA issued "HED SOP 97.2 Interim Guidance for Conducting Aggregate Exposure and Risk Assessments (11/26/97)."[28] The Agency continued to work on more sophisticated methods for estimating aggregate exposures to pesticides, and in 2001, released "General Principles for Performing Aggregate Exposure and Risk Assessments,"[29] to augment the guidance document.

The aggregate risk assessment process relies on available data, assumptions designed to be protective of public health, standard analytical methods and Agency SOPs to estimate exposures to a pesticide for each potential pathway and route of exposure. EPA combines these separate exposure estimates to calculate potential aggregate exposure and risk; aggregate exposures estimated in this way reflect upper-bound or high-end of exposures for each route/pathway. The most highly exposed group, which generally also has the highest associated risk, is used as the basis for decision-making purposes. Aggregate risk assessments conducted in this manner typically can be refined by the use of additional exposure data and data specifically designed to address the uncertainties within an individual aggregate analysis, as well as more sophisticated analysis techniques.

The assumption implicit in this approach is that individuals can encounter the high-end exposures from the different pathways all at one time. In actuality, co-occurrence of high-end food, drinking water, and residential exposure scenarios is very unlikely. Thus, in using this approach, EPA is confident that aggregate exposure estimates will overstate, sometimes significantly, the amount of a pesticide to which people actually are exposed. The primary advantage to relying on these highly conservative assessments is that they require relatively fewer data and analytical resources and less time to conduct. In addition, an aggregate risk assessment of this type may be enough to indicate that a particular pesticide use satisfies the appropriate regulatory standards.

C. Pesticide Re-evaluation Processes

i. Reregistration

FIFRA requires the periodic re-evaluation of currently registered pesticides. In 1988, Congress amended FIFRA section 4 to include a specific process for the "reregistration" of pesticides containing active ingredients first registered before November 1, 1984. Pub. L. 100-532, title I, § 102(a), 102 Stat. 2655, 2683(1988). To make a Reregistration Eligibility Decision (RED),[30] EPA reviewed all the studies that were submitted by the pesticide registrants for a

[28] U.S. Environmental Protection Agency. 1997e. Memorandum from Margaret Stasikowski, Health Effects Division to Health Effects Division Staff. "HED SOP 97.2 Interim Guidance for Conducting Aggregate Exposure and Risk Assessments (11/26/97);" November 26, 1997. Office of Pesticide Programs, Office of Prevention, Pesticides, and Toxic Substances, Washington, D.C. Available upon request.

[29] http://www.epa.gov/pesticides/trac/science/aggregate.pdf

[30] In particular cases, the Agency issued a variation on the RED, i.e., a Tolerance Reassessment Decision, or, for individual active ingredients identified as belonging to a group of pesticides with a common mechanism of action, an Interim Reregistration Decision may have been issued before the RED.

pesticide active ingredient, as well as other relevant information, developed the appropriate risk assessments, and decided whether or not the pesticide active ingredient satisfied the risk-benefit standard of FIFRA section 3(c)(5). After determining eligibility of the active ingredient, EPA determined whether to reregister products containing the active ingredient.[31] *See* FIFRA § 4(g)(2)(B). In conjunction with most REDs, the Agency "called-in" active ingredient- or product-specific data considered necessary to reduce uncertainty in the RED risk assessments.

Reregistration was an open and transparent process. Before finalizing its decision on the eligibility for reregistration of a pesticide, EPA made the supporting risk assessments available for public comment, although it was not required to do so. Comments were solicited particularly on the factual basis of the risk assessments and also on options for mitigating the risks posed by the subject pesticide. These comments were considered in the Agency's decision-making.

Most of the pesticides specifically named in the Petition went through the reregistration process. Information on the reregistration status and links to the reregistration dockets for any pesticide can be accessed via the Agency's Chemsearch database.[32] And, as explained more fully below, they have also been scheduled for review early in the current re-evaluation process, known as Registration Review. *See* FIFRA § 3(g).

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ii. Registration Review

Once the reregistration decisions for all the subject active ingredients were completed, the Agency began the next re-evaluation process under FIFRA, which requires EPA to regularly review pesticides to ensure that they continue to satisfy the statutory standard for registration. The ongoing re-evaluation process is called registration review. Section 3(g) of FIFRA requires EPA to complete its initial registration review cycle by October 1, 2022, for all pesticides registered prior to October 1, 2007, and by 15 years after the date of initial registration for pesticides registered after that date. *Id.*[33] Following the initial review of the pesticide, EPA must conduct subsequent reviews of each registered pesticide every 15 years thereafter.

The registration review program[34] makes sure that, as the ability to assess risk evolves and as policies and practices change, all registered pesticides continue to meet the statutory standard of no unreasonable adverse effects. Through registration review, the Agency is ensuring that registered pesticides do not cause unreasonable risks to human health or the environment when used as directed on product labeling. Changes in science, public policy, and pesticide use practices will occur over time, and the cyclical nature of registration review will enable the

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[31] While the Agency has completed its statutory requirement to make eligibility determinations on the subject active ingredients, product reregistration is still ongoing for some reregistration cases.

[32] http://iaspub.epa.gov/apex/pesticides/f?p=chemicalsearch:1; search by active ingredient name, PC Code, or CAS number.

[33] See also 40 CFR Part 155 for the implementing regulations. An overview of the process is found at http://www.epa.gov/oppsrrd1/registration_review/highlights.htm.

[34] Unlike earlier re-evaluation programs, registration review operates continuously, and provides for the review of all registered pesticides.

Agency to consider updated information every time a pesticide comes up for registration review.[35]

In conducting the registration review program, EPA generally is reviewing pesticides in chronological order according to their baseline dates;[36] that is, older cases are being reviewed first. In addition, many pesticides that received priority scheduling for reregistration have been scheduled early in registration review. Thus, food-use chemicals[37] that were identified as or suspected of having risk concerns at the outset of reregistration generally are scheduled early in the registration review process. Additionally, within this structure, EPA plans to review certain related pesticides at the same time, particularly pesticides in the same family, with the same general structure and mode of action.

In reregistration, EPA gained experience and benefited from efficiencies in the concurrent review of pesticides in families like the organophosphates (OPs), N-methyl carbamates (NMCs), triazines, and chloroacetanilides. The rodenticides and soil fumigants were also reviewed concurrently. EPA plans to continue the practice of grouping related pesticides during registration review.[38] Potential efficiencies from this practice include:

- Technical and regulatory issues may be resolved more easily looking across an entire chemical class or group;
- Resources can be optimized within EPA, among stakeholders, and within other federal agencies; and
- In developing decisions, a "level playing field" among chemicals in the group may be assured so that EPA's actions do not inadvertently cause increased risks.

EPA completed cumulative risk assessments and reregistration risk management decisions for the OP pesticides in August 2006 and the NMC pesticides in September 2007. In recent years, EPA and stakeholders have invested significant resources in gaining a better understanding of these classes of pesticides. The registration review of the OPs began in 2008, and the N-methyl carbamate review began in 2010. The Petitioners are requesting expedited reviews for both classes of chemicals. These classes of pesticides were among the first pesticides to enter the registration review program, so the assessment of risks associated with their use (including risks to children, and risks from spray drift), and the management of those risks, should be accomplished early in the current registration review cycle, and in subsequent cycles as well. Volatilization will also be addressed based on the results of the screening analysis and the subsequent availability of pertinent data, should they be required for individual pesticides. The scheduling of these classes of pesticides in registration review reflects an understanding of the importance of addressing the toxicity, dietary and aggregate risks, and the

[35] The Agency uses the best, most recent information in any risk assessment, but the cyclical nature of registration review assures that assessments for any one pesticide will be updated at least every 15 years.

[36] The baseline date is the date when a RED was completed, or for a pesticide not subject to reregistration, the date when the pesticide was first registered.

[37] Pesticides used on food crops were given high priority because they have potential to affect the population at-large via dietary exposure.

[38] The overview at http://www.epa.gov/oppsrrd1/registration_review/highlights.htm provides links to information on the pesticide family groupings.

volume of use for these pesticides. *See* Appendix to this response for the registration review schedules for the OPs and NMCs.

EPA initiates a registration review by establishing a docket for a pesticide registration review case and opening the docket for public review and comment. The Agency publishes a Federal Register notice that announces the availability of a Preliminary Work Plan (PWP) and provides a comment period of at least 60 days. Anyone may submit data or information in response. EPA will consider information received during the comment period in conducting a pesticide's registration review. The PWP:

- explains what EPA knows about the pesticide from previous risk assessments including hazard and exposure information related to children, when available, and application of uncertainty factors including the FQPA safety factor;
- tentatively identifies what kind of risk assessments are needed;
- tentatively identifies what data will be needed to conduct the assessments;
- addresses the uncertainties in the database that will impact the risk assessments (particularly missing or unclear use parameters);
- provides basic use and usage information and other background information on the pesticide; and
- provides a proposed schedule that lays out milestones up until the registration review decision is made.

Information from registration review dockets that have opened is readily accessible via Chemical Search on a chemical-by-chemical basis.

Registration review, as set forth in the regulations, is a transparent process in which stakeholders of all types are invited to provide relevant data and comments. At the beginning of registration review, the public is asked to comment on the PWP and its supporting documents; by soliciting these comments, EPA aims to gather additional information that can enhance registration review planning and decision-making. Other registration review documents also are subject to a public comment period, such as when the preliminary risk assessments are posted to the docket later in the process. Similar approaches to public notification and comment were used during reregistration and can be used in decision-making about new active ingredients.

EPA may also, as needed, consult with registrants, the U.S. Department of Agriculture. and other stakeholders to resolve any uncertainties about how the product is used, particularly if use parameters are not clear from product labeling, or if the registrant may have data not already submitted to the Agency that could inform the process. For example, some labels are not specific about retreatment intervals or seasonal maximum application rates. Without actual use parameters, the Agency is forced to make conservative assumptions that can result in overly conservative risk assessment results. The Agency may solicit such information in a "Focus Meeting." These meetings can be held whenever in the registration process they might be helpful. To ensure transparency, materials associated with Focus Meetings are available in the pesticide-specific registration review dockets. If a focus meeting is held prior to the docket

opening, these materials are posted to separate docket.[39] Once the docket opens, a copy of the focus meeting notes will be posted to the case-specific docket.

After the close of the initial comment period, EPA revises its work plan as needed based on public input and any additional information that has become available in the interim. The Final Work Plan is then posted to the docket, and EPA prepares to call-in any data needed for the risk assessments. Once the registrants submit the required data, work on the risk assessments begins. As noted above, EPA makes the draft risk assessments available for public review and comment, and subsequent to review of public comments, the Agency posts the revised assessments. If the revised assessments indicate that there are risks of concern, EPA may invite the public to submit suggestions for mitigating those risks. These suggestions are used in the development of a proposed registration review decision.

EPA will announce the availability of a proposed registration review decision on the docket and will provide a public comment period of at least 60 days. The process culminates with a final registration review decision—EPA's determination on whether the pesticide in question meets or does not meet the standards for registration. The final registration review decision discusses any changes that are needed to the pesticide's registration or labels to address the risks of concern. If a registrant fails to take action to implement these changes, EPA may take appropriate action under FIFRA.

To meet its statutory obligations for registration review, EPA is opening 70 or more dockets annually continuing through 2017, and almost all of the pesticides registered at the start of the program will have dockets opened by 2017. The Agency is directed to complete the first round of registration reviews by October 1, 2022; during that time the Agency will complete the registration reviews of at least 744 pesticide cases comprising 1,165 active ingredients. Pesticides registered after 2007 will be folded in each year.[40]

iii. Regulatory Responses to Unacceptable Risks

If EPA determines that a pesticide product does not meet the statutory standard, the Agency may take "appropriate regulatory action." Such regulatory actions can include, but are not limited to, restricting pesticide uses or canceling the pesticide's registration. *See* FIFRA §§ 3(d) & 6(b). If EPA chooses to cancel a pesticide registration, EPA must first issue a Notice of Intent to Cancel and hold a formal administrative hearing, if one is requested by a person adversely affected by the notice. *See* FIFRA § 6(b).

If the Agency determines that an "imminent hazard" exists from the use of a pesticide (including a pesticide that was not eligible for reregistration), EPA may commence proceedings

[39] General information about focus meetings is at http://www.epa.gov/oppsrrd1/registration_review/focus-meetings.html; the docket itself is at http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2012-0778.

[40] The status of all completed and ongoing registration reviews can be found at http://www.epa.gov/oppsrrd1/registration_review/reg_review_status.htm.

to suspend the registration of a pesticide during the time needed to complete cancellation proceedings. *See* FIFRA § 6(c). Section 2(l) of FIFRA defines imminent hazard as:

> [a] situation which exists when the continued use of a pesticide during the time required for cancellation proceeding would be likely to result in unreasonable adverse effects on the environment or will involve unreasonable hazard to the survival of a species declared endangered or threatened by the Secretary pursuant to the Endangered Species Act....

If the EPA determines that an emergency exists such that the imminent hazard will occur during the period necessary to complete normal suspension proceedings, the EPA may issue an immediately-effective emergency suspension order in advance of completing suspension proceedings. *Id.* § 6(c)(3).

Courts addressing the suspension provisions have held that an imminent hazard exists if there is "a substantial likelihood that serious harm will be experienced during the year or two required in any realistic projection of the administrative process." *Love v. Thomas*, 858 F.2d 1347, 1350 (9th Cir. 1988) (quoting *Environmental Defense Fund v. EPA*, 465 F.2d 528, 540 (D.C. Cir. 1972). In the case of an emergency suspension, one court has found by analogy that suspension is appropriate if there is a "substantial likelihood that serious harm will be experienced during the three or four months required in any realistic projection of the administrative suspension process." *Dow v. Blum*, 469 F.Supp. 892, 901 (E.D. Mich. 1979). Thus, courts interpreting the FIFRA suspension standard have made clear that an imminent hazard finding requires a greater degree of likelihood, immediacy, and severity of harm than is otherwise required to take a cancellation action under FIFRA. And in evaluating the nature and extent of information before EPA, the courts have instructed the Agency to consider (1) the seriousness of the threatened harm, (2) the immediacy of the threatened harm, (3) the probability that the threatened harm will occur, and (4) the benefits to the public of the continued use of the pesticide *Id.* at 902.

EPA's review and re-evaluation processes for pesticides have been developed to account for advancements in science so that risks can be identified and managed before a pesticide is registered and at regular intervals thereafter. Through these processes, the Agency can anticipate and correct problems with the use of pesticides as time goes on, and, ideally, reduce the chances that a pesticide would pose risks of an immediacy and magnitude like those associated with an imminent harm finding.

V. How Pesticide Risks Are Assessed

The type of assessment pertinent to this Petition is the human health risk assessment[41] (ecological risk assessments are not discussed in the Petition). EPA uses a science-based risk assessment approach. Risk is a function of both the hazard associated with a pesticide and how much exposure occurs to that pesticide. Hazard is the innate ability of a pesticide or other stressor to cause an adverse effect (its toxicity). At EPA, hazards are typically identified from the results of testing on several animal species and typically the most sensitive effect is used as

[41] See http://www.epa.gov/pesticides/about/overview_risk_assess.htm

the basis for risk assessment. Exposure is the amount or concentration of a stressor with which an affected individual or group interacts. Risk is a function of both hazard and exposure and represents the likelihood that an individual or group will be adversely affected by that stressor. Both hazard and exposure can differ according to a person's age, thus EPA uses age-appropriate behaviors to determine exposures and also looks at any special sensitivity to pesticides associated with the age of the exposed parties. EPA uses the National Research Council's four-step process[42] for its human health risk assessments. The four steps include: 1) hazard identification, 2) dose-response assessment, 3) exposure assessment, and 4) risk characterization. Each of these steps is summarized below.

It is important to note that risk assessment and risk management are separate activities. Risk management relates to the ways in which the risks characterized in the assessment may be reduced or eliminated. Risk management measures can include tolerance revocation or the cancellation of registrations, termination of some uses of a pesticide, changes to a pesticide's use parameters, and risk reduction training for people who are occupationally exposed, such as pesticide applicators.

A. Hazard identification

The first step in the risk assessment is to identify potential health effects that may occur from different types of pesticide exposure. EPA considers the full spectrum of a pesticide's potential health effects.

Typically, a pesticide active ingredient is subjected to many toxicity studies, and the data from these studies (if determined to be acceptable) are used in risk assessment. Requirements for the relevant data are typically imposed on the pesticide applicant or registrant, and the studies are typically conducted by independent laboratories, with strict standards for data surety.[43] The data are evaluated for acceptability by EPA scientists. The toxicity studies primarily are performed on laboratory animals or *in vitro*, although some of the required studies are conducted in the field. The Agency will also review human toxicity studies, with qualifications, as discussed below, but does not require them. EPA evaluates pesticides for a wide range of potential toxic effects including eye and skin irritation, neurological effects, cancer, and birth defects. In addition to reviewing the required studies, EPA also consults the public literature or other sources of supporting information.

The required tests are used in the assessment of potential health effects in infants, children, and adults. They are conducted for exposures of different durations, as appropriate to represent the durations of exposure anticipated for various lifestages and behaviors. Exposures may be of single day or longer durations, up to and including durations spanning a lifetime. Additionally, EPA considers the route by which these exposures may occur—orally, e.g., through the diet or via children's mouthing behaviors; through the skin; or by inhalation.

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[42] The National Research Council produces reports for the National Academy of Sciences. The process is explained at http://epa.gov/riskassessment/health-risk.htm.

[43] Required laboratory practices for studies used to support pesticide registration are detailed at http://www.gpo.gov/fdsys/pkg/CFR-2011-title40-vol24/xml/CFR-2011-title40-vol24-part160.xml

To address risks to infants, children, and adults, EPA typically requires animal testing at multiple life stages, including during gestation and shortly after. The effects that are observed in this type of testing are fetal development (including birth defects) and reproductive success. The results of these studies are particularly applicable to the assessment of risks to the fetus and young children and, when compared to studies with adult animals, provide a basis for evaluating the relative sensitivity of the young to adults.

In order to develop a risk assessment that is protective of human health, the Agency will select the "most sensitive" endpoints and the corresponding point of departure (POD) from among these different studies for the relevant populations, taking into account the durations and routes of exposure. The endpoint can be described as the toxic effect itself. The POD is typically the dose level below the lowest dose at which the adverse effect is manifested.

As an example, consider an endpoint that was selected from a shorter-term animal study in which the animals were exposed via the dermal route, for a pesticide that is registered for use on lawns. In this example, the POD based on this endpoint would be used to estimate risks to adults and to children aged 1 to 2 years old. Children in this age group typically are the most highly exposed relative to children of all ages for pesticide residues on turf by weight and because of their behavior on lawns, as they are exposed to residues through contact with their skin when they play in their yards or in parks and playgrounds, and are also exposed orally, predominantly via hand-to-mouth behaviors.

As noted previously, the Agency also will consider relevant data from sources other than required studies, including data from prospective and retrospective epidemiologic studies, incident reports,[44] and studies in which human subjects have been exposed intentionally to a pesticide (although the latter must undergo an specialized review to ensure that the Agency does not rely on data from studies that violate established ethical standards).[45] EPA uses its draft "Framework for Incorporating Human Epidemiologic & Incident Data in Risk Assessments" when considering those types of data.[46]

B. Dose-response

The second step in the risk assessment process is to consider the dose levels at which adverse effects were observed in test animals and to extrapolate those dose levels to an equivalent dose in humans. For animal studies, the uncertainty around the extrapolation from test animals to humans and the variability of sensitivity in the human population are accounted for by uncertainty factors. The default factors assume there could be up to a ten-fold difference between animals and humans and up to a ten-fold difference between the average person and the most sensitive people in the population. That is, humans generally are assumed to be 10 times more sensitive than animals, and the most sensitive individuals generally are assumed to be 10 times more sensitive than that. These uncertainty factors create a margin of safety for protecting

[44] For example, per analysis of pesticide acute illnesses based on 1998-2006 NIOSH SENSOR data, see http://ehp03.niehs.nih.gov/article/info%3Adoi%2F10.1289%2Fehp.1002843, referenced in the Petition.

[45] http://www.epa.gov/oppfead1/guidance/human-test.htm

[46] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2009-0851-0004

people who may be exposed to the pesticides. The FFDCA requires EPA to use an extra 10-fold safety factor to protect infants and children from effects of the pesticide, unless reliable data show that a different (larger or smaller) factor would protect the safety of infants and children.[47]

C. Exposure

The third step in the risk assessment process is to address how long, and at what level people are exposed to a pesticide, a critical consideration when selecting endpoints and also for calculating risks. For spray drift, exposures are assessed for contact with previously contaminated surfaces such as lawns adjacent to treatment areas. More specifically, the Agency assesses exposures from dermal contact (e.g., children playing on lawns) and also non-dietary ingestion from mouthing behaviors of young children. Adults also are expected to have exposures from dermal contact. For volatilization exposure, the primary exposures result from inhalation since exposure is to the gas or vapor form of a pesticide.

In general, pesticide residues deposited on surfaces (such as grass or the leaves of treated crop plants) remain available for exposure to people entering treated areas, or areas in which spray drift residues have settled, for more than a single day, so subchronic studies are used to derive endpoints and PODs. Single day (acute toxicity) studies also may be considered in order to evaluate risks on the day of application (when the greatest exposures are likely). Impacts on fetuses due to the exposure of pregnant women are included in the risk assessments when information on reproductive and developmental toxicities is available. If information on reproductive and developmental toxicities is not available, an uncertainty factor may be used to account for the possibility of the special sensitivity of children not apparent from available data.

More details are provided in Sections VII and VIII below on how exposures to spray drift and volatilization are determined.

D. Risk characterization

The last step in the risk assessment process is to combine the hazard and exposure assessments to describe the overall risk from a pesticide. Risk characterization explains the assumptions used in assessing exposure as well as the uncertainties[48] that are built into the dose-response assessment, and whether or not the assumptions and uncertainties used are likely to overstate or understate potential risks. The strength of the overall database is considered, and broad conclusions are made.

In summary, the risks to human health from a pesticide depend on both the toxicity of the pesticide and the likelihood of people coming into contact with it. At least *some* exposure and *some* toxicity are required to result in a risk. For example, if the pesticide is very toxic, but no people are exposed, there is no risk. Likewise, if there is ample exposure but the chemical is non-toxic, there is no risk.

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[47] EPA may modify these uncertainty factors on a case-by-case basis when supported by chemical-specific behavior.

[48] These are uncertainties not covered by FQPA or other uncertainty factors, e.g. deriving from an incomplete database.

E. Children and pesticides

EPA has developed methods for estimating pesticide exposures to children through the diet and via non-dietary sources such as residential exposures. These methods rely on the best available scientific sources, such as EPA's "Exposure Factor's Handbook."[49] Dietary exposures are based on consumption data for children, and residential exposures are based on methods outlined in the "Standard Operating Procedures (or SOPs) for Residential Exposure Assessment."[50] The SOPs address exposures for various lifestages, and allow for the identification of the most highly exposed lifestage. The SOPs, including the concept of lifestage, have been discussed extensively by the FIFRA Scientific Advisory Panel (SAP).[51] The 2012 revision of the SOPs reflect the input of the SAP.[52]

Young children may have unique exposures that adults do not have because of age-specific behaviors, for example, picking things up from the ground and mouthing them, or putting their hands (potentially contaminated with pesticide residues) in their mouths. They may also come into contact with pesticides when crawling or at play on treated or contaminated surfaces. Children up to adolescence have a higher surface to weight ratio than adults, so they may also be proportionately more highly exposed via the dermal route. Children can also be more highly exposed via the dietary route because they consume more food and water in proportion to body size than adults, and the types of food they eat a lot of tend to contain more pesticide residues.

Available data pertinent to children's health risks are evaluated along with data on adults and the most sensitive, appropriate POD is defined (e.g., NOAEL or no observed adverse effect level) for the most sensitive critical effect(s) based on consideration of all health effects. By doing this, protection of the health of children will be considered along with that of other potentially sensitive populations. In most cases, it is appropriate to evaluate the potential hazard to children separately from the assessment for the general population or other population subgroups.

The approach used by EPA to account for pesticide exposures in children is consistent with EPA's general risk assessment methods[53] and follows the Agency's age grouping guidance.[54] The approach is also consistent with recommendations from the National Academy of Science

[49] http://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=236252.

[50] http://www.epa.gov/pesticides/science/residential-exposure-sop.html

[51] The SAP is a federal advisory committee consisting of independent, external scientific experts that advises the Agency's Office of Pesticide Programs (OPP) on technical issues.

[52] The SAP meeting report is at http://www.epa.gov/scipoly/sap/meetings/2009/100609meeting.html.

[53] EPA's risk assessment methods can be found at http://www.epa.gov/risk/guidance.htm; An overview of EPA's approach to assessing and managing these risks is provided in the 2010 report "Protecting Children's Health," found at http://www.epa.gov/pesticides/health/protecting-children.pdf.

[54] An overview of EPA's approach to assessing and managing these risks is provided in the 2010 report "Protecting Children's Health," found at http://www.epa.gov/pesticides/health/protecting-children.pdf; the 2005 paper "Guidance on Selecting Age Groups for Monitoring and Assessing Childhood Exposures to Environmental Contaminants, at http://www.epa.gov/raf/publications/pdfs/AGEGROUPS.PDF, advises risk assessors on selecting appropriate age ranges for use in implementing the Agency's initiatives on pr

1993 report, "Pesticides in the Diets of Infants and Children,"[55] cited extensively by the Petitioners. EPA has adopted many of the recommendations from that report into its current risk assessment procedures.

VI. Nomenclature Associated with Spray Drift and Volatilization

EPA uses an informative nomenclature that allows for a clear delineation between the possible forms and/or sources of pesticide movement through the air and away from treated fields. This approach provides for a means of avoiding confusion when describing the unique processes and factors that can contribute to pesticide movement. Whether or not pesticide drift occurs during or after application is an important factor, as is whether or not pesticides are applied in liquid form or as solid material.

As indicated previously, for this response, the Agency focuses on "spray" drift, the off-site movement of aerosols originating with pesticides applied as liquids, rather than dust drift, because spray drift is more likely to occur and generally poses greater risk. Also as noted previously, the Petitioners do not appear to differentiate between drift and volatilization.

Although there are similarities in the mode of transport (through the air) associated with volatilization and spray drift, EPA assesses spray drift and volatilization separately, for several reasons:

- They are two distinctly different processes. Spray drift is dependent on how applications are made and on the form in which the pesticide is applied, while volatilization is driven by the physical and chemical characteristics of the pesticide active ingredient (especially its vapor pressure).
- Spray drift occurs at the time of application and shortly thereafter, for as long as droplets remain aloft. Volatilization, on the other hand, can occur during the application process and also over longer periods of time depending upon the physical and chemical characteristics of the pesticide and how it was applied.
- The route of exposure that the Agency assesses for volatilization is inhalation. The Agency's assessment of spray drift focuses on dermal exposures and exposures from non-dietary oral ingestion (predominantly from hand-to-mouth behaviors in young children). Inhalation exposures are not included in the Agency's spray drift assessments for the following reasons:
    - Most, but not all, of the droplets in spray drift are too large to be respirable.
    - For agricultural pesticides, the Worker Protection Standard (WPS) prohibits the application of a pesticide such that it contacts, either directly or through drift, any worker or other person.[56] This WPS prohibition mitigates the potential for bystander exposure to active drift, but not the residues of drift that are deposited on surfaces to which bystanders may be exposed.

  The Agency at present lacks an assessment methodology for drift inhalation exposures.
- The ways that the risk from volatilization and spray drift can be mitigated are different.

[55] http://www.nap.edu/openbook.php?record_id=2126&page=1.
[56] http://www.epa.gov/pesticides/health/worker.htm

The operating definitions of "spray drift" and "volatilization" are discussed in detail in Sections VII and VIII, respectively.

VII. Assessing and Managing Risks from Spray Drift

EPA has been working with a broad range of public and private stakeholders to address concerns related to spray drift and the potential for adverse effects related to drift exposure.[57] EPA's goal is to assess, and if necessary, to mitigate spray drift via a science-based approach relying on case-specific information.

Spray drift is influenced primarily by environmental conditions (such as wind speed) and application parameters (such as formulation type, application method, application rate, droplet/particle size, application release height). Some degree of pesticide drift is an inevitable result of nearly all types of pesticide application. Even under the best of circumstances, a minute amount of pesticide can move out of the treatment area for a short distance. When the amount of drift is such that it poses risks of concern, the Agency will take action to mitigate those risks.[58]

Quantifying the potential risks of spray drift is a complex process that involves predicting the amount of drift associated with various types of application equipment, estimating potential exposures, and considering the potential health effects from such exposures. Managing the risks associated with spray drift can be complex as well and there are a variety of potential approaches that can be used, as discussed more thoroughly below.

A. Estimating Spray Drift and Potential Exposures to Bystanders[59]

Since the early 1980's, EPA has been working to better understand spray drift. Information key to this effort was developed by a group of pesticide registrants working collaboratively to create a database that addressed spray drift data requirements under 40 CFR 158.440.[60] This group is referred to as the Spray Drift Task Force (SDTF).[61] Since its formation in 1990, the SDTF has generated standardized data on spray drift levels associated with a variety of application methods under varying field and meteorological conditions. The database was reviewed by EPA internally, through external peer review workshops, and by the SAP.[62] Using the database, the SDTF began working with EPA's Office of Research and Development and the USDA's Agricultural Research Service and Forest Service in 2000 to develop and validate a

[57] The Pesticide Program Committee (PPDC) is a federal advisory committee that includes representatives from a broad variety of stakeholders interested in pesticide regulation. See http://www.epa.gov/oppfead1/cb/ppdc/.The PPDC's Spray Drift Workgroup has provided valuable input on the Agency's approach to assessing and mitigating spray drift. Membership of the Workgroup includes environmental advocacy groups, grower associations, registrants, and state officials. See http://www.epa.gov/oppfead1/cb/ppdc/spraydrift/members.htm.

[58] http://www.epa.gov/pesticides/factsheets/spraydrift.htm.

[59] For purposes of this response, bystanders are defined as people who, live, play, or work in areas at proximity to pesticide-treated areas.

[60] See http://www.epa.gov/ocspp/pubs/frs/publications/Test_Guidelines/series840.htm

[61] The Task Force's website is at http://www.agdrift.com/

[62] See http://www.epa.gov/scipoly/sap/meetings/1997/december/spraydrift.htm.

model for predicting the magnitude of off-target movement of pesticides via spray drift, called the "AgDrift" model.

The AgDrift model was developed to assess spray drift under a variety of different conditions for aerial, ground, and orchard air-blast applications. Input features provide the capability to alter over 30 parameters related to the aerial application method including types and numbers of nozzles, weather conditions, and terrain features. AgDRIFT also can provide empirically based predictions for ground and airblast applications made under various conditions, and can accommodate differences in use patterns that relate to crop-specific pest management needs. In addition, users can run the AgDrift model to estimate the amount of spray drift at specified distances from the application site.

Spray drift associated with aerial application has been evaluated extensively by the U.S. Army and the USDA Forest Service, so the drift database is more extensive for aerial applications compared to ground applications. For orchard airblast and groundboom sprayer estimates, AgDrift is more limited; unlike estimates for aerial applications, there is no mechanistic option for these ground applications. Rather, an empirical approach based on the available data is used, and users are more limited in the number of factors that can be considered (e.g., orchard canopy type for airblast sprayers). Even so, AgDrift is a powerful tool for quantifying spray drift for these application methods.

For aerial applications, EPA uses AgDrift to predict conservative estimates of the amount of spray drift given the conditions specified on pesticide labels (when such conditions are not specified on the proposed label, the Agency uses conservative assumptions). AgDRIFT has more limited capabilities to reflect label specifications for ground applications. AgDrift can be used to estimate the risk reduction attributable to buffer zones of specified widths (essentially by estimating the differences in the amount of spray drift at different distances from the application site). In addition to its use in assessing bystander risks, AgDrift can be used in environmental assessments to estimate the potential spray drift exposures to non-target animals and plants. It also is used to estimate the potential contribution of spray drift to pesticide residues in drinking water.

Results from the use of AgDrift represent a range of possible outcomes that are reflective of cultural practices tied to how the target crops are produced and the nature of the pesticide being applied. For example, a contact insecticide application to dense-canopy field crops may be most efficacious when the spray is composed of finer aerosols, while a systemic herbicide applied to a field crop canopy of lesser density, where complete spray coverage is not needed to achieve the desired degree of efficacy, can contain droplets of a larger size (as an aside, larger size or coarse droplets tend to drift less, all other factors being equal).

EPA acknowledges that there is some potential for drift exposures to occur indoors, but believes that the amount of drift entering enclosed structures is very small relative to the amount present out-of-doors. The Agency's efforts to understand the human health risks posed by drift are focused on outdoor exposures.

B. Assessment of Risk to Bystanders from Spray Drift

EPA has been working to refine and standardize the way it assesses the risks to bystanders from spray drift. On December 5, 2013, EPA presented its approach to assessing spray drift to the PPDC. EPA made this new methodology for assessing spray drift available for public comment on January 29, 2014. The announcement and directions on how to submit comments on the proposal can be accessed on the public docket.[63] EPA will continue to conduct spray drift risk assessments under its current process while it reviews and analyzes comments received during the public comment period, which originally was scheduled to end on March 31, 2014, but is being extended by 30 days. After reviewing public comments, EPA plans to finalize its methodology and consider it in cases that warrant spray drift risk assessment.

The methodology for assessing spray drift exposures is based in part on the methodology for assessing residential exposures to pesticides on turf, as explained below,[64] coupled with estimates of the amount of spray drift reaching the area in question, which are derived as described in Section A., above. EPA has developed methods for estimating risks for residential scenarios in which people may be exposed through their use of a pesticide or because they live, work, or play in places where pesticide use occurs. As noted previously, EPA uses its SOPs for residential exposure assessment as the basis for estimating exposures in these situations. These SOPs have undergone extensive external peer review by the SAP.[65] SOPs exist for a wide range of possible exposure scenarios.

The Agency assesses bystander risks from spray drift based on the residential turf scenario, in which people (including children) are exposed to pesticide residues on lawns.[66] If an agricultural pesticide is also registered for use on residential turf, EPA has determined that an additional drift assessment is not necessary beyond that of the residential turf. For an agricultural pesticide not also registered for use on turf, the Agency can use the screening methodology that is included in the new drift methodology, and may be able to conclude, qualitatively, that drift does not pose risks of concern for bystanders, so a quantitative assessment is not needed. When a quantitative assessment is needed, the methodology calls for the use of the AgDrift model to estimate the amount of pesticide residue available on turf for the exposures of adult and child bystanders. The estimated amount of residue and the exposure factors for adults' and childrens' time and activities on lawns are used to calculate exposures through the skin and from the mouthing behaviors (predominantly hand-to-mouth) of children of appropriate developmental age. These exposures are compared to the appropriate endpoint and POD, as discussed in V.B. "Hazard Identification" above.

The development of the SOPs for evaluating lawn pesticides considered a number of factors related to how residues should be quantified, the appropriate behavioral considerations for adults

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[63] http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2013-0676

[64] http://www.epa.gov/pesticides/science/residential-exposure-sop.html

[65] http://www.epa.gov/scipoly/sap/tools/atozindex/residentexp.htm and http://www.epa.gov/scipoly/sap/meetings/2009/100609meeting.html

[66] See particularly the Lawns/Turf SOP at http://www.epa.gov/pesticides/science/residential-exposure-sop.html. The SOP identifies default values for exposure parameters e.g., time that a child spends on the lawn, how often children will put their hands in their mouths, etc. These values have been selected so that exposure estimates overall will be conservative.

and children, development of appropriate exposure metrics, how exposures should be combined, and what age groups should be considered as the basis for risk management. EPA considers adults involved in heavy yardwork and children ages 1 to 2 (based on both body weight and play behavior) as the two groups most highly exposed to turf–applied pesticides. EPA has extensively considered children of varying ages in order to ascertain which lifestage (referred to as index lifestage in the SOPs) has the highest relative exposure given behaviors that occur at various stages of development. Children between 1 and 2 years old routinely and very actively engage in outdoor play, and they exhibit mouthing behaviors (predominantly hand-to-mouth) which contribute to the overall exposure levels.

EPA relies on a number of assumptions when using the SOPs in the calculation of risks from spray drift. Risks are based on residue levels present on the day of application when they are at their highest levels because they have not had a chance to dissipate. Risks are also estimated based on a standardized lawn width of 50 feet. The standardized lawn was derived from U.S. Census information for single- and multi-unit dwellings—it is the mean lot width for multi-unit housing and also is a reasonable representation for single-unit housing with smaller lots. A low-end lot size is used because the concentration of spray drift residues is assumed to be inversely correlated to lot size due to the effects of residue dilution in larger lot sizes.[67] Use of "day of application" residues and the standardized lawn size contribute to a data-informed conservative estimate of risk.

C. How EPA Mitigates Potential Risks Associated with Spray Drift

Unacceptable risks associated with spray drift can be mitigated in a number of ways, including changes to application parameters, use of drift reduction technologies,[68] changes to formulations, and no-spray buffer zones, either in combination or by themselves.[69] While the use of buffer zones is one of the key issues raised by the petitioners, other measures also can be used to manage potential risks associated with spray drift. Changes to application parameters and pesticide labels that may mitigate drift risks include reduced application rates; prohibition of certain application methods; soil-incorporation of pesticides at the time of application; and prescriptive, product-specific labeling that requires a particular spray quality (i.e., droplet sizes) or climatic conditions. The Agency may also undertake cancellation of specific uses, in those rare cases where spray drift causes unreasonable risks that cannot otherwise be mitigated.

There is a significant level of effort within the agricultural engineering community to develop both drift reduction technologies and best management practices to reduce spray drift. Useful drift reduction technologies include different forms of spray nozzles and other sophisticated application equipment (e.g., sensors for canopy identification that turn off nozzles

[67] More information on the standardized lawn is found in the proposed methodology at http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2013-0676.

[68] http://www.epa.gov/etop/etc_at_psdt.html and http://www.epa.gov/etop/etc_at_proppsdt.html . An instructive presentation on the Drift Reduction Technology Program is located at http://www.epa.gov/oppfead1/cb/ppdc/2012/may/session-7-drift-reduction.pdf

[69] When appropriate, EPA considers the potential consequences of mitigation measures in light of the impact on producers and on the potential for undesirable risk trade-offs.

at ends of rows), and the use of adjuvants.[70] EPA, working with academia and industry, has developed a program to rate drift reduction technologies, and plans to identify on its website tested technologies and the risk reduction potential attributable to them.[71] Other potential means of reducing exposures to spray drift are already commonly accepted,[72] for example, the adjustment of release height in ground applications. Some formulation types are less prone to drift than others; for example, switching to a dry (soil-incorporated) formulation from one that is applied as a liquid may reduce drift potential. A series of possible drift reduction measures are already included in the proposed EPA method for calculating risks from spray drift as a starting point for considering mitigation options should they be required. This element of the method will facilitate consistency in the Agency's decision-making process for managing spray drift risks.

The Agency has used the proposed assessment approach to support pesticide drift risk mitigation in past decision-making. The Agency completed its Preliminary Human Health Risk Assessment for chlorpyrifos (an organophosphate pesticide) under the registration review process in July 2011.[73] That assessment identified risk concerns for bystanders from exposure to spray drift from agricultural applications of chlorpyrifos and provided estimates of the potential risk reductions associated with various drift mitigation options.

In the Spray Drift Mitigation Decision for Chlorpyrifos (July 2012),[74] EPA announced an agreement with the chlorpyrifos registrants for implementing use restrictions intended to reduce spray drift risks to bystanders. In accordance with the agreement, risk mitigation was accomplished through amendments to chlorpyrifos product labels, which were put in place by the end of the same year. Mitigation measures were: buffer zones for groundboom, airblast, and aerial applications of chlorpyrifos around sites such as homes, sidewalks, and recreational areas; and a reduced application rate for aerial applications of chlorpyrifos (from 6 to 2 pounds active ingredient per acre).

VIII. Assessing and Managing the Risks Due to Pesticide Volatilization

Pesticide volatilization can be defined as the change of a pesticide in solid or liquid form to a gas or vapor after application has occurred.[75] Volatilized pesticides can move off-site resulting in the potential for exposure outside the treatment area. The volatilization process is complex and depends on many factors that include the innate physical and chemical properties of the pesticide, the innate characteristics of the site where it is applied, and the atmospheric conditions at the time of application. Other factors that impact volatilization, particularly those associated with application parameters, directions for use, and best management practices are under the

[70] An adjuvant is broadly defined as any non-pesticide material added to a pesticide product or pesticide spray mixture to enhance the pesticide's performance and/or the physical properties of the spray mixture.

[71] http://www.epa.gov/pesticides/factsheets/spraydrift.htm#other

[72] For example, http://pesticidestewardship.org/drift/Pages/defaultaspx provides a general overview of existing technologies.

[73] See http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0025.

[74] See http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0103

[75] See http://www.epa.gov/opp00001/about/intheworks/volatilization.htm

control of the user and can be manipulated to reduce off-site movement.[76] For example, soil incorporation of the pesticide, compaction of the soil, and (particularly in the case of fumigant pesticides) tarping and tenting can reduce volatilization from soil- applied pesticides. The use of certain adjuvants may reduce volatilization from pesticides applied to foliage.

Fumigant pesticides are highly volatile.[77] Once applied, they will change into a gaseous form that works by filling the application space or by permeating the soil to kill a wide array of pests. Efficacy is achieved by ensuring that the appropriate air concentration is maintained for the necessary time in order to control the pest of concern.[78] Practices that are used to reduce exposure to (while also improving the efficacy of) fumigant applications include the use of tarps, field conditions management (e.g., soil moisture levels), and the use of specialized application implements (e.g., specially designed shanks for closing up and compacting the soil disturbances to retain the fumigant in soil).

Conventional pesticides (pesticides other than fumigants) tend to be much less volatile than fumigants because of differences in their physical-chemical properties, although some conventional pesticides volatilize under some circumstances. Conventional pesticides also are designed to achieve efficacy via different mechanisms so volatility is not a key required characteristic. When conventional pesticides do volatilize in the field, they too can move outside of the treatment area.

A. Quantifying Volatilization For Conventional Pesticides

EPA has developed a good understanding of the volatilization of fumigant pesticides, as noted above and discussed by the SAP,[79] including an understanding of how use site conditions can impact volatilization. The volatilization of conventional pesticides has not been studied to the same extent. A number of entities are now focusing on the volatilization of conventional pesticides. Research to enhance our understanding of the volatilization of conventional pesticides could include work on the impact of a crop canopy or leaf type on the volatilization process. Air monitoring data are also important in efforts to characterize how much of a pesticide will volatilize and travel out of the treatment area.

The approach used in the past fumigant risk assessments and EPA's proposed approach for conducting volatilization exposure assessments for conventional pesticides consider both single application events and the contamination of ambient air within a local community, region, or the

[76] As in controlling drift, practices that limit the off-site movement of volatilized pesticides can improve the efficacy of an application by keeping more pesticide where it is needed to control the target pests.

[77] Background and status of the Agency's re-evaluation of the fumigants is found at http://www.epa.gov/pesticides/reregistration/soil_fumigants/soil-fum-ree-backgrnd.html

[78] The interaction of concentration and time to achieve efficacy is commonly referred to as the required concentration x time schedule (CxT).

[79] Supporting documents and the final report from the December 1-4, 2009 SAP meeting on "Scientific Issues Associated with Field Volatilization of Conventional Pesticides are located in the docket at http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2009-0687.

airshed (the air supply for a defined geographical region) from multiple applications within the same area.

Potential impacts associated with individual application events can be managed more directly through pesticide labels than potential exposures to ambient air. Also, more information can be collected to quantify the volatilization associated with a single pesticide application event than the concentrations expected in ambient air. "Flux" is the term used to describe how much volatilization (or emission) of a pesticide can occur across a given area for specific period of time. Field data can be collected for empirical use in risk assessment and also as the basis for empirically based dispersion modeling. There are a number of recognized flux methods in the peer-reviewed literature.[80] Along with air concentration measurements each method requires that detailed meteorological data be collected and that the conditions of the application are also well- documented.

A large number of flux studies have been completed for fumigants, so their behavior under various field conditions is relatively well understood. Flux data have been also generated for a limited number of conventional pesticides including chlorpyrifos,[81] but in general, they are not available for conventional pesticides. This lack of flux data for conventional pesticides was a primary focus of the 2009 SAP review. EPA presented a number of options for predicting flux in lieu of data which would allow EPA to screen conventional pesticides for potential risk concerns. Building on advice from the SAP,[82] the Agency developed a screening methodology using a preferred approach known as the Woodrow equation.[83]

EPA has recently announced the availability of this screening methodology[84] and is soliciting public input on the new methodology and the proposed approach for assessing volatilization for conventional pesticides. EPA will finalize the approach after considering public comment.

Along with distinct application events, EPA also considers potential exposures to ambient air that may represent those within a community, region, or widespread airshed depending upon where, when, and how such monitoring data were collected. Such data have been used empirically and EPA characterizes such data to the extent possible given available resources. The 2009 EPA SAP analysis provides an example of how an air monitoring result could be characterized by risk assessors based on use information and knowledge of the application site relative to where monitors are placed. To date, EPA has not attempted a modeling approach for

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[80] Relevant citations can be found in the bibliography to "Scientific Issues Associated with Field Volatilization of Conventional Pesticides Presented Jointly to the FIFRA Scientific Advisory Panel, USEPA, 2009" at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2009-0687-0006. They include: USEPA, 2008; Majewski and Capel, 1995; Lenoir *et al.*, 1999; Majewski and Baston, 2002; McConnell *et al.*, 1998; Zamora *et al.*, 2003; Glotfelty *et al.*, 1990; Schomburg *et al.*, 1991.

[81] The preliminary volatilization evaluation for chlorpyrifos is found at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0114; supporting documents posted in the same docket discuss the modeling of flux rates.

[82] The final report of the SAP is found at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2009-0687-0037.

[83] See Woodrow *et al.*, 1997 and 2001. Citations found at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2009-0687-0006.

[84] http://www.regulations.gov/#!docketDetail;D=EPA-HQ-OPP-2014-0219

predicting ambient exposures to pesticides for these types of circumstances, but plans on exploring such approaches in the future.

Air monitoring data for conventional pesticides are limited and the quality is generally lacking compared to current protocols. The data that do exist mostly come from California; data collection under authority of California's Toxic Air Contaminant statute[85] began in the mid-1980s and continues in the present time. The Washington Department of Health has collected air monitoring data with a focus on organophosphates involved in agricultural production.[86] PANNA (one of the Petitioners) has also collected air monitoring data for a number of pesticides in various locations throughout the United States.[87] The Agency does consider these "Drift Catcher" data for risk characterization, despite certain limitations, and has concluded that the data thus far are not suggestive of concentrations of pesticides in the air that pose significant risks to human health. Other sources of air monitoring data are found in the scientific literature.

B. Estimation of Risks Associated with Pesticide Volatilization

At this time, EPA has conducted at least one volatilization assessment for a conventional pesticide using the fumigant methodology approach.[88] Volatilization risk assessments for the fumigants and conventional pesticides consider distinct, individual pesticide applications as well as ambient air contamination from multiple applications of the same pesticide in the same general area. While single application event risk estimates are based on modeled values, ambient air analyses are based on empirical values; for ambient risk estimation the most representative exposure statistic is selected and compared to the appropriate toxicological value. Additionally, risk assessments for both ambient air and single application events are informed by incident information. Looking for commonalities in the incident information and the predictive risk estimates is a critical consideration for regulatory decision-making.

The Agency's approach to assessing risks associated with single application events is multi-faceted. It includes the use of information on how the pesticide is used; flux data, which are needed for use of the Woodrow equation[89] (or, if flux data are not available, information on physical and chemical properties of the pesticide); and information on the toxicology of the pesticide from the inhalation route of administration (if inhalation toxicity data are not available, other toxicity data may be used). An air dispersion model is used to estimate air concentrations

[85] http://www.cdpr.ca.gov/docs/emon/pubs/tacmenu.htm, and http://www.cdpr.ca.gov/docs/emon/pubs/tac/tacstdys.htm

[86] http://www.doh.wa.gov/ehp/Pest/drift.htm

[87] See http://www.panna.org/science/drift; the Agency considers Drift Catcher data when available and uses them in risk characterization. Unfortunately, the raw data that EPA prefers are not always available and the timing intervals for air samples under the PANNA protocol tend not to permit association with particular applications of the pesticides detected.

[88] Refer to the fumigant risk assessment documents for a detailed overview of the risk assessment process available: http://www.epa.gov/pesticides/reregistration/soil_fumigants/soil-fum-reg-backgrnd.html#information

[89] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2009-0687-0006

of volatilized pesticides surrounding treated fields.[90] Model inputs include standard assumptions about field sizes and the surrounding terrain, and weather data for representative locations. Dispersion modeling is a two-step process. First, flux information is used to characterize how much applied pesticide will volatilize from a treatment area, and then the dispersion of the volatilized pesticide around the treatment area is characterized. Changing weather patterns over time are considered. The mathematical approach to compute how volatilized residues will dissipate is based on a construct known as a Gaussian plume.[91]

EPA has data requirements for information needed to support registrations of pesticides that may result in inhalation exposures.[92] Such data are strongly preferred for use in volatilization risk assessments, but if necessary oral toxicity data can be used. Issues related to the use of oral data for inhalation exposures were discussed by the SAP in 2009.

As noted previously, EPA has recently released a screening methodology for characterizing the potential risks associated with volatilization of conventional pesticides. The Agency has used the methodology to conduct a screening level assessment of all currently-registered conventional pesticides, and found that only about 20 percent of conventional pesticides need to be evaluated further so that the Agency can better understand the potential risks associated with their volatilization and off-site movement. The results of this screening exercise will be released along with the methodology. The Agency may need additional information to perform more detailed assessments, such as data that are needed to model flux, inhalation toxicity data, and information on the pesticide's use parameters. Comments submitted by the public on the screening analysis and its conclusions will be considered as the Agency determines how to proceed. Also as noted previously, the Agency has already conducted at least one volatilization risk assessment for a conventional pesticide, and it can be viewed on the public docket.[93]

C. How EPA Mitigates Potential Risks Associated with Volatilization

After chemical risk assessments are completed, EPA must determine whether there are risks to be mitigated, and if so, how that should be accomplished. EPA has used this process to address risks posed by the volatilization of a pesticide and its off-site movement. Options that can be effective include: buffer zones, reduced application rates, low volatility formulations or adjuvants, tarping and tenting of treated fields, and crop management practices.

The fumigants assessment and mitigation measures provide a framework for considering how to manage potential volatility risks associated with conventional pesticides. For the fumigants, EPA required a suite of complementary mitigation measures to protect handlers, workers, and bystanders from risks resulting from exposure to the soil fumigant pesticides. The measures were designed to work together to address the full range of risks, but were focused on the risks of volatilization to people (workers and bystanders), and included restricted-use status,

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[90] http://www.epa.gov/scipoly/sap/meetings/2004/082404_mtg.htm and http://www.exponent.com/perfum/.

[91] Distribution based on the standard bell-shaped curve.

[92] See 40 CFR §158.500.

[93] Chlorpyrifos: http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0130

use site limitations, application rate reductions, and buffer zones with chemical-specific widths.[94] Other risk management measures implemented for the fumigants are not relevant to conventional pesticides, such as the use of tarps over treated fields, which is employed in conjunction with fumigant applications to reduce the off-gassing from soil applications. Risk mitigation measures differ among pesticides because the individual risk assessments were based on chemical-specific information.

IX. Responses to Requests Made by Petitioners

EPA reads the petition to make three specific requests for programmatic changes: 1) that EPA evaluate the risks to children exposed to pesticide drift and volatilization for all pesticides, 2) that EPA modify its pesticide re-evaluation process to expedite assessment of these risks, and 3) that, for certain types of pesticides, the Agency require the adoption of generic buffer zones between treated areas and places where children could congregate.[95] The Agency's responses to each of these elements follow.

Although this petition addresses how EPA assesses risk under the FFDCA, it does not specifically request to cancel registrations or modify or revoke specific tolerances. The Petitioners also are requesting that EPA require interim buffers for all "drift-prone" pesticides during the time EPA makes the programmatic changes they have requested. Because the Petitioners are suggesting specific changes to use practices but not requesting cancellation of registrations, we also have interpreted the petition to request that EPA attempt to procure voluntary label amendments from the registrants. However if the registrants did not agree, Agency-initiated cancellation actions would likely be needed to achieve the requested relief.

A. EPA Will Evaluate the Risks to Children Associated with Spray Drift and Volatilization Exposures.

While it is true that EPA has not always assessed the risks to children from spray drift and volatilization, the need for consistent and refined methods have led to the development of appropriate methodologies for doing so. The development of these methods is described in detail in Sections VII and VIII of this response.

The Petitioners are requesting that pesticide drift and volatilization risk assessments be conducted for all pesticides. We agree. The first step in EPA's new spray drift assessment methodology is a screening process to facilitate the identification of conventional pesticides that could pose risks of concern to bystanders through spray drift. As previously noted, the Agency released the draft spray drift assessment methodology earlier this year. Elements of the "needs

[94] http://www.epa.gov/pesticides/reregistration/soil_fumigants/implementing-new-safety-measures.html#risk

[95] As noted earlier, the Agency considers residential exposures to include exposures associated with homes, home lawns, yards, gardens, apartments and grounds around apartment buildings, schools, schoolyards, daycare facilities, playgrounds, athletic fields, and parks and other public spaces.

screening" process are detailed in materials posted to the docket on "Consideration of Spray Drift in Pesticide Risk Assessment."[96] The new volatilization methodology is designed to serve as a stand-alone screening methodology for any and all conventional pesticides. As noted above, the Agency has already used the volatilization methodology to screen all currently-registered pesticides. The screening processes include consideration of factors such as methods of application and use patterns of the subject pesticide. Using the screening procedures for both spray drift and volatilization, EPA considers the potential for exposure in a qualitative way. In the case of spray drift, pesticides for which the screen indicates there are potential risk concerns will undergo a quantitative assessment. Thus, we grant the Petitioners' request to conduct spray drift and volatilization assessments for all pesticides, while noting that some of these assessments will not provide quantitative results.

In the last several years, EPA has conducted a number of spray drift and volatilization risk assessments, even while the proposed assessment methodologies were being developed and refined. EPA conducted volatilization assessments for the fumigant pesticides during reregistration.[97] Chlorpyrifos also recently underwent a spray drift evaluation,[98] while the recently released proposed drift assessment methodology[99] was still in development (see Section VII B). The preliminary human health risk assessment for chlorpyrifos was completed in July 2011[100] and attendant risk reduction measures for spray drift exposures were implemented in July 2012.[101] EPA also recently published a draft volatilization risk assessment for chlorpyrifos[102] and is working to finalize the assessment even as work continues on the volatilization methodology. The methodologies set out standardized procedures so that the way spray drift and volatilization are assessed will be consistent between pesticides in general, but they are not substantively different from the approaches that were used to assess spray drift and volatilization for pesticides in the recent past.

Further evidence of EPA's commitment to reducing the risks to children exposed to pesticides is demonstrated by actions taken during pesticide reregistration process to terminate residential uses, as was done with many organophosphate insecticides. EPA now has better-developed tools for determining when spray drift poses risks to bystanders, and is committed to taking action on risks to bystanders, including children, during registration review.

The Petitioners also are requesting that EPA include the drift and volatilization exposures of children in its aggregate assessments. Including spray drift and volatilization in EPA's aggregate risk assessments would involve the following steps. First, EPA would look at exposures from drift and volatilization under the new policies to determine whether any non-negligible exposure

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[96] Process described at http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2013-0676-0003.

[97] Based on the findings from these assessments, EPA required the implementation of risk mitigation measures, including measures intended to protect bystanders. The risk mitigation measures are detailed at http://www.epa.gov/pesticides/reregistration/soil_fumigants/implementing-new-safety-measures.html.

[98] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0105

[99] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2013-0676-0001

[100] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0025

[101] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0103

[102] http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2008-0850-0114

due to drift or volatilization could occur. If non-negligible exposure could occur, EPA would quantitatively assess that exposure under the new policies and then aggregate that exposure with other sources of exposure consistent with existing policies on aggregate exposure. However, aggregation of drift and volatilization exposure with other sources of exposure is not specifically addressed in existing aggregate exposure policies, and thus EPA's approach may need to be modified to account for the factors involved in drift and volatilization exposure. Finally, if initial aggregate exposure estimates using conservative methodologies indicate there could be a risk of concern, EPA would need to develop efficient approaches to refining those assessments.

EPA fully agrees with the Petitioners that exposures to spray drift and volatilized pesticides should be considered in our risk assessments, and that the risks to children, with their unique biology and behaviors, must be considered separately from risks to adults. The Agency has developed the methodologies for assessing drift and volatilization and is committed to considering the comments of the public on them so that we may employ the best possible science in assessing these risks and taking action to mitigate risks as needed.

B. Expediting Assessments of Spray Drift and Volatilization Outside of the Registration Review Schedule is not Necessary

Petitioners state that EPA should accelerate its schedule for completing drift and volatilization risk assessments and prioritize pesticide reviews based on the suspected degree of risks posed to children. Petitioners suggest that this acceleration should be accomplished through modifications to the registration review process or by utilizing other authorities. Petitioners believe that registration review is too slow to be protective of drift and volatilization risks to children.[103] EPA's response to this request covers aspects of public health policy and the Agency's obligations under FIFRA and the FFDCA, and issues of efficiency.

The Agency is denying the Petitioners' request to the extent that they ask EPA to perform bystander risk assessments of the chemicals highlighted in the Petition before considering other exposures and risks. The registration review program is an appropriate and risk-protective approach for evaluating and managing the risks associated with spray drift and volatilization (and other types of risk as well). Utilizing a process outside of registration review to assess these risks to children separately from other types of risk would bypass and defer the comprehensive evaluations that allow the Agency to rely on the best and newest developments in science and to address the full complement of potential risks. The consideration of all potential risks at one time for a single active ingredient can lead to the adoption of a package of risk mitigation measures that works for all the risks of concern, or at the very least, that do not work in opposition to each other.

[103] The Petitioners also assert that because the registration review process is designed to update the reregistration risk assessments, and since the Agency did not address the risks to bystanders from drift and volatilization during reregistration, registration review will not include such assessments. In reality, the Agency updates risk assessments on the basis of available data and scientific developments, so drift and volatilization will be included in registration review, even if they were not considered during reregistration.

To set the schedule for registration review, EPA relied primarily on the baseline date for each pesticide case (usually its RED date or the date the first product containing the active ingredient was registered). Additionally, some registration review cases were grouped for program efficiency. The OPs and NMCs were among those cases.[104] For the most part, food- use pesticides subject to reregistration were given priority scheduling in reregistration, and thus, they have the earliest baseline dates. The OPs and NMCs, on which the Petitioners focus, have been scheduled at the front-end of registration review and many individual pesticides from those families are currently undergoing risk assessment. EPA believes that, insofar as the Petition requests EPA to give high priority to certain chemicals in its registration review program, the petition asks for an action that has already occurred.

The OPs and NMCs account for more than 40 *individual* active ingredients. All of these pesticides have entered registration review (or were canceled prior to entering registration review).The preliminary risk assessments for 12 of the pesticides in these two families are scheduled to be completed before October 2014.The schedule for OP and NMC registration reviews[105] is summarized in the Appendix to this response.[106] The Appendix also identifies the pesticides in these groups that were cancelled subsequent to tolerance reassessment or during the beginning stages of registration review.

With respect to conducting separate bystander exposure assessments of individual pesticides, if the Agency granted Petitioners' proposal, it would significantly reduce the efficiency of the overall registration review program. Separating the bystander drift risk assessment for children from the ongoing comprehensive evaluations for these same chemicals would require Agency resources to be redirected to the evaluation of one type of potential risk, and management of the full complement of potential risks associated with a pesticide would be deferred. In addition, the overall demand for resources and the time needed to assess first spray drift and then all other potential risks for a given pesticide would be greater in total than the time and resources needed to conduct a comprehensive assessment of that pesticide, and thus would slow Agency action on risk management. Registration review, as currently planned, is the most efficient way to achieve the Petitioners' and EPA's common goal of protecting human health, including the health of children, and the environment. Adopting the approach proposed by the Petitioners also would significantly reduce EPA's ability to meet its statutory obligation to complete registration review by 2022 or the date that is 15 years after the date on which the first pesticide containing a new active ingredient is registered. *See* FIFRA § 3(g)(1)(A)(iii).

The same logic applies to the idea of assessing chemical-specific volatilization risks separately from the comprehensive registration review assessment. Significantly, preliminary application of the volatilization screening methodology currently under development led the Agency to conclude that only 20% or so of all pesticide active ingredients have characteristics that suggest that they potentially could pose any meaningful level of volatilization risk. Thus, including the volatilization risk assessment in the registration review process as a matter of

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[104] Described in a Federal Register Notice: http://www.gpo.gov/fdsys/pkg/FR-2006-10-11/html/E6-16483.htm.

[105] http://www.epa.gov/pesticides/cumulative/ identifies the organophosphate and n-methyl carbamate pesticides by name.

[106] The full schedule is at http://www.epa.gov/oppsrrd1/registration_review/schedule.htm.

course will not appreciably affect the resources needed or timing for the vast majority of registration review decisions.

Petitioners suggest that EPA could accelerate the reviews of drift risks for children by utilizing other authorities, such as rulemaking[107] or the special review process.[108] Rulemaking is a long, resource-intensive process that can take many years to complete, and EPA believes its limited resources are better spent assessing and developing risk mitigation measures for pesticides individually than developing a regulation that could take many years to finalize. As an example, the Petitioners suggest that the WPS Rule is an appropriate model for implementing broad changes for a large number of pesticides at once. Although the WPS is an important and effective tool for reducing worker risk, it took approximately eight years to develop and promulgate the initial 1992 rule and as many years to develop a set of proposed amendments to the 1992 rule.[109]

And while Special Review served its purpose in the past, individual Special Reviews typically took many years to conclude and used Agency resources to address a narrow set of risk concerns at a time when there was no systematic re-evaluation process for pesticide registrations. Indeed, in 2009, the Agency announced that "[t]he pesticide program is moving toward closing out both the Special Review and the reregistration programs" in favor of new re-evaluations of previously registered pesticides to be conducted under registration review.[110] That is not to say that potential risk concerns that rise to the highest levels, including "emergency" or "imminent hazard" status, must wait to be addressed in registration review—the processes for such situations include those described in Section IV of this response.

Based on these considerations, for existing registrations, EPA has concluded that registration review, as currently planned, is the most appropriate, timely, and efficient way to achieve the Petitioners' and EPA's common goal of protecting human health, including the health of children, and the environment.

C. Immediate Adoption of Interim No-Spray Buffers Around Homes, Schools, Daycare Centers, and Parks to Protect Children from Drift Is Not Appropriate

Petitioners request that EPA impose interim no-spray buffers around locations where children congregate and that these measures should apply to OPs, NMCs, and all other pesticides that are: "(1) registered for application by ground sprayers, broadcast equipment, and/or aerial equipment; and (2) suspected of causing acute poisonings, cancer, endocrine disruption,

[107] Although Petitioners mention EPA's general rulemaking authority under FIFRA, EPA is not treating this petition as a specific request for rulemaking. Instead EPA understands Petitioners' statements being made as actions that could be taken by EPA. The Agency's general rulemaking authority is provided at 7 U.S.C. § 136w(a)(1).

[108] 40 C.F.R. § 154.7

[109] The revisions to the WPS were just released for public comment on February 20, 2014--http://www.regulations.gov/#!documentDetail;D=EPA-HQ-OPP-2011-0184-0002.

[110] http://epa.gov/oppfead1/cb/csb_page/updates/2009/namechange-prd.html

developmental effects, and/or reproductive effects."[111] Petitioners further state that the interim buffers should be at least 60 feet in width for ground applications and 300 feet in width for aerial applications regardless of the pesticide being applied, and that these buffer requirements should remain in place at least until case-specific drift risk assessments can be undertaken. To accomplish this request, Petitioners further suggest that the Agency could use administrative procedures rather than chemical-specific risk assessment and management to effect the generic buffers i.e., rulemaking or a Pesticide Registration (PR) Notice. The Agency's response to this request covers the usefulness of the alternate approaches, the scientific basis for pesticide decision-making, and issues of efficiency.

The Agency denies the Petitioners' request to impose a requirement for interim buffers of either 60 or 300 feet on these pesticides before EPA completes the registration reviews for these pesticides. EPA contends that drift and volatilization are not posing risks of concern for all pesticides, and that interim buffers, as suggested by the Petitioners, are not the most efficient or scientifically appropriate way to mitigate such risks for any particular pesticide or group of pesticides.[112] It is the Agency's practice to assess pesticide risks based on chemical-specific data. The Agency acknowledges that the OPs and NMCs are generally among the more acutely toxic pesticides, but risk is a function of both toxicity and exposure, so toxicity alone is not sufficient to characterize the risks these pesticides may cause to bystanders via drift or volatilization, or to determine if risk mitigation is needed. Additionally, the OPs and the NMCs were reevaluated both in reregistration and in the tolerance reassessment process and, at that time found to meet the applicable statutory safety standards. These pesticides will be reassessed again in the next few years under registration review. Because pesticides vary in environmental fate characteristics, and use sites and parameters, potential exposures also differ, not just between different active ingredients, but also between different uses of the same active ingredient.

The pesticides other than the OPs and NMCs that the Petitioners believe warrant the use of 60 and 300 foot buffers are a very large and diverse group. Without considering pesticide-specific data, it is impossible to know the risks posed by each. Again, the manner in which the Petition proposes to manage the drift and volatilization risks associated with these pesticides ignores the interaction of exposure and hazard. When these pesticides underwent reregistration, the Agency found that (with certain conditions) they met the statutory standards of FIFRA and FFDCA. In order to make the same type of determinations when spray drift and volatilization exposures are appropriately considered for each individual case, the Agency must satisfy the same standards.

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[111] While an exact count of pesticides that fit in this second category cannot be made, the effects listed are a large subset of the complete set of adverse effects the Agency takes into consideration. Furthermore, EPA notes that the Petition describes the referenced pesticides and groups of pesticides as "drift prone." The Agency rejects this notion. Because drift is influenced by factors other than the characteristics of the pesticide active ingredient, primarily the physical form of the product as applied, the application method, climate, and wind, no one active ingredient or pesticide family can be considered to be drift-prone. There are particular formulations and application methods that may make certain applications of a pesticide product prone to drift. Volatilization, on the other hand, is a direct function of the physical and chemical properties of an active ingredient, and the Agency is able to identify pesticides that tend to volatilize.

[112] EPA does not believe the Petitioners have presented adequate justification for interim, across-the-board buffers for all the pesticides they have identified as being of special concern.

Even if buffer restrictions may be appropriate to mitigate the risks to children from spray drift and volatilization for some pesticides, the EPA contends that the same buffer width will not be appropriate for each of them. Despite the Petitioners' request for across-the-board, interim buffers, the Petition itself makes the point that case-specific assessments have shown that buffers of varying widths are needed to mitigate risks associated with different pesticides. They cite examples of the different buffer widths that EPA determined were necessary for products contain different active ingredients—e.g., for the fumigants, widths ranging from 25 ft to one-half mile. EPA believes that the more scientifically defensible approach involves determining whether no-spray buffers are necessary to protect children on a pesticide-by-pesticide basis. This is the approach the Agency has taken and intends to include in consideration of bystander exposure in future risk assessments.

Finally, the Agency believes that the requests made by the Petitioners with regard to interim buffers would divert limited Agency resources from important risk assessment and risk management activities and would diminish the overall level of protection EPA is able to achieve in its pesticide re-evaluations. The alternate means that the Petitioners suggest to implement the buffer requirement are also resource-intensive, time-consuming, and not likely to result in the broad protections the Petitioners desire. The resource and time requirements of rulemaking are discussed above.

The Petitioners suggest that EPA could use a Pesticide Registration (PR) Notice[113] to inform registrants that label amendments are necessary to address drift and volatilization, and, that failure to make these changes could result in cancellation or the finding that their products are misbranded. Although EPA agrees that a PR Notice is a useful tool to communicate new policies to pesticide registrants, compliance with a PR Notice is voluntary, and the Agency believes that registrants are not likely to implement changes that lack a risk-based rationale. If EPA took action to require the amendment of pesticide registrations to mitigate spray drift via buffers of uniform width, pesticide applicants and registrants could challenge the validity and applicability of the science behind the Agency-prescribed regulatory actions. Additionally, the resources needed to pursue a cancellation proceeding are extensive, and in the absence pesticide-specific assessments, would not be an effective use of EPA's limited resources.

X. Conclusion

The Agency appreciates the concern the petitioners express for bystanders, particularly children, who may be harmed by exposure to spray drift and the off-site movement of volatilized pesticides. We share this concern. The Agency has assessed spray drift and volatilization for particular pesticides in the past and is now taking steps to formalize the assessment methodologies for future assessments. These methodologies include screening-level assessment processes for use in determining if there is a need to take a more in-depth look at any given pesticide. Thus, all pesticides will be assessed either qualitatively or quantitatively. The Agency also believes that we are addressing pesticide risks on a schedule that gives the most potentially

[113] PR Notices are issued by EPA's Office of Pesticide Programs to inform pesticide registrants and other interested persons about important policies, procedures and regulatory decisions. The Agency's PR Notice webpage is at http://www.epa.gov/PR_Notices/.

risky pesticides precedence. We will continue to use approved approaches to account for the differences between children and adults in their exposures and sensitivity to pesticides.

The Agency believes that its current program of registration review is the most comprehensive and effective way to assess and mitigate pesticide risks and to take advantage of new and emerging science. The Agency believes that looking at a particular pesticide and all the potential risks associated with its use in a comprehensive fashion provides the best opportunity to effectuate necessary protections for human health and the environment. Other means of managing the risks posed by spray drift and volatilization as suggested by the Petitioners, such as conducting drift and volatilization-only assessments or re-ordering the pesticides in registration review, are either not needed, not likely to be successful, require more resources than are available, and/or take more time than registration review.

While we understand the thinking behind the proposal to mandate generic, uniform buffer requirements on pesticides of particular concern, we do not agree that a "one size fits all" solution is appropriate or scientifically defensible. Without case-specific assessments and risk mitigation, we believe it is unlikely that registrants will voluntarily adopt generic buffers. Pesticide registrants are by law afforded specific rights and opportunities to oppose decisions by the Agency that affect their registrations. Because the evidence needed to support the cancellation or amendment of registrations within this context must be scientifically defensible and specific to the subject pesticide, we believe that Agency resources are better spent in registration review.

APPENDIX

Registration Review Timelines for Pesticide Families Specifically Cited in the Petition

Table 1. Anticipated Organophosphate Registration Review Milestones[114]

Active Ingredient	Docket Opening	Draft Human Health Risk Assessment	Final Decision
Acephate	3/18/09	2014	2015
Azinphos-methyl	All registrations cancelled 2012		
Bensulide	6/25/08	2015	2015
Chlorethoxyfos	12/17/08	2014	2015
Chlorpyrifos	3/18/09	July 6, 2011	2016
Chlorpyrifos-methyl	3/13/10	2015	2016
Coumaphos	6/25/08	2014	2015
Diazinon	6/25/08	2014	2016
Dichlorvos	6/24/09	2015	2016
Dicrotophos	6/25/08	2014	2015
Dimethoate	3/18/09	2014	2015
Disulfoton	3/18/09	All registrations subsequently cancelled	
Ethoprop	12/17/08	2014	2015
Fenamiphos	All registrations cancelled 2007		
Fenitrothion	3/18/09	2015	2015
Fenthion	All registrations cancelled 2004		
Fosthiazate	6/24/09	2013	2014
Malathion	6/24/09	2014	2016
Methamidophos	12/17/08	All registrations subsequently cancelled	
Methidathion	3/18/09	All registrations subsequently cancelled	
Methyl parathion	6/24/09	All registrations subsequently cancelled	
Naled	3/18/09	2015	2016
Oxydemeton-methyl	6/25/08	All registrations subsequently cancelled	
Phorate	3/18/09	2015	2015
Phosmet	6/24/09	2015	2015
Phostebupirim	6/24/09	2016	2017
Pirimiphos-methyl	3/18/09	2015	2016
Profenofos	6/25/08	2015	2016
Propetamphos	6/25/08	All registrations subsequently cancelled	
Phosalone	2/19/08	All registrations cancelled effective 12/30/15	
Temephos	6/25/08	All registrations subsequently cancelled	
Terbufos	6/25/08	2014	2015
Tetrachlorvinphos	6/25/08	2014	2015
Tribufos	3/18/09	2015	2016
Trichlorfon	3/18/09	2015	2016

[114] Dates in the future should be considered tentative.

Table 2. Anticipated N-Methyl Carbamates Registration Review Milestones[115]

Active Ingredient	Docket Opening	Draft Human Health Risk Assessment	Final Decision
Aldicarb	6/20/12	2015	2017
Carbaryl	9/22/10	2016	2018
Carbofuran	All registrations cancelled 2009		
Formetanate HCl	12/22/10	2016	2017
Methiocarb	6/22/10	2015	2016
Methomyl	9/22/10	2015	2016
Oxamyl	9/22/10	Early 2015	2016
Pirimicarb	All registrations cancelled 2010		
Propoxur	12/16/09	2014	2016
Thiodicarb	12/16/09	2015	2016

[115] Dates in the future should be considered tentative.

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TECHNICAL REPORT

Pesticide Exposure in Children

James R. Roberts, MD, MPH, Catherine J. Karr, MD, PhD, and COUNCIL ON ENVIRONMENTAL HEALTH

KEY WORDS
pesticides, toxicity, children, pest control, integrated pest management

ABBREVIATIONS
CDC—Centers for Disease Control and Prevention
CI—confidence interval
2,4-D—2,4-dichlorophenoxyacetic acid
DDE—*p,p'*-dichlorodiphenyldichloroethylene
EPA—Environmental Protection Agency
ES—Ewing sarcoma
GI—gastrointestinal
INR—international normalized ratio
IPM—integrated pest management
NPDS—National Poison Data System
OP—organophosphate
OR—odds ratio
PT—prothrombin time
RR—relative risk
SGA—small for gestational age
Th2—T helper 2

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abstract

FREE

Pesticides are a collective term for a wide array of chemicals intended to kill unwanted insects, plants, molds, and rodents. Food, water, and treatment in the home, yard, and school are all potential sources of children's exposure. Exposures to pesticides may be overt or subacute, and effects range from acute to chronic toxicity. In 2008, pesticides were the ninth most common substance reported to poison control centers, and approximately 45% of all reports of pesticide poisoning were for children. Organophosphate and carbamate poisoning are perhaps the most widely known acute poisoning syndromes, can be diagnosed by depressed red blood cell cholinesterase levels, and have available antidotal therapy. However, numerous other pesticides that may cause acute toxicity, such as pyrethroid and neonicotinoid insecticides, herbicides, fungicides, and rodenticides, also have specific toxic effects; recognition of these effects may help identify acute exposures. Evidence is increasingly emerging about chronic health implications from both acute and chronic exposure. A growing body of epidemiological evidence demonstrates associations between parental use of pesticides, particularly insecticides, with acute lymphocytic leukemia and brain tumors. Prenatal, household, and occupational exposures (maternal and paternal) appear to be the largest risks. Prospective cohort studies link early-life exposure to organophosphates and organochlorine pesticides (primarily DDT) with adverse effects on neurodevelopment and behavior. Among the findings associated with increased pesticide levels are poorer mental development by using the Bayley index and increased scores on measures assessing pervasive developmental disorder, inattention, and attention-deficit/hyperactivity disorder. Related animal toxicology studies provide supportive biological plausibility for these findings. Additional data suggest that there may also be an association between parental pesticide use and adverse birth outcomes including physical birth defects, low birth weight, and fetal death, although the data are less robust than for cancer and neurodevelopmental effects. Children's exposures to pesticides should be limited as much as possible. *Pediatrics* 2012;130:e1765–e1788

INTRODUCTION

Pesticides represent a broad classification of chemicals that are applied to kill or control insects, unwanted plants, molds, or unwanted animals (eg, rodents). "Pesticide" is a collective term for a wide array of products but is often inappropriately used in reference to only insecticides. The universe of pesticide types and products is broad, and

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a comprehensive review of all active ingredients is beyond the scope of this report. This review focuses on select insecticides, herbicides, and rodenticides and specific chemical classes within these groups that have the greatest acute and chronic toxicity for children on the basis of historical experience and/or emerging evidence (Table 1).

Several types of pesticides are not discussed in this report. Fumigants and fungicides, although potentially toxic, are less commonly involved in acute childhood exposure and poisoning, in general, so these are not included. Wood preservatives containing arsenic are also not included in this report. The specific compound containing arsenic, copper chromium arsenate, has been removed from the market since January 2004. Older wood structures treated with copper chromium arsenate may still be found in homes, on playgrounds, and in yards and should be treated yearly with a waterproof sealant.[1] Insect repellents, including *N, N*-diethyl-meta-toluamide and picaridin, are different from most pesticides in that they are a product purposefully applied to human skin to prevent insect bites and are, in fact, not insecticides. These compounds are unique and have been reviewed recently.[2]

Although the severity of pesticide exposures and toxicity may be greater in developing countries where regulatory oversight and information is limited, the content of this technical report is oriented toward exposures most relevant to children residing in the United States. Commonly used insecticides, including the organophosphates (OPs), carbamate, and pyrethroid classes, are discussed, as are the relatively new neonicotinoids. Other pesticides that will be discussed in some detail include the phosphonate herbicides (eg, glyphosate), chlorophenoxy herbicides, and long-acting anticoagulants (rodenticides). For a more comprehensive survey of the acute toxicity from the spectrum of pesticide active ingredients and products, see other sources.[1,3]

CHILDREN'S EXPOSURE: VULNERABILITY, MECHANISMS, AND SOURCES OF EXPOSURE

Children's Unique Vulnerabilities

Children are uniquely vulnerable to uptake and adverse effects of pesticides because of developmental, dietary, and physiologic factors. Exposure occurs through ingestion, inhalation, or dermal contact. Unintentional ingestion by children may be at a considerably higher dose than an adult because of the greater intake of food or fluids per pound of body weight. Children exhibit frequent hand-to-mouth activity, and this is an important source of increased exposure in comparison with adults.[4,5]

Residential Factors

Fortunately, acute toxicity attributable to pesticide poisoning is relatively uncommon in US children, and a pediatrician in general practice may not encounter such an event. However, subacute and chronic low-level exposure is common. Residential factors that influence chronic exposure include the use of insecticides and rodenticides in the home, and herbicide and fungicide use on lawns, as well. Indoors, broadcast applications including sprays, "flea bombs," and foggers can leave lingering residues in the air, carpet, toys, and house dust.[6–9] Typical exploratory behavior, including playing on and crawling across the floor, increases the risk of dermal, inhalation, and oral exposure to residues on surfaces or the air as it settles.[10] Repeated and cumulative incidental exposure can also occur. Pesticides can be measured in indoor air samples and persist in dust vacuumed from carpeted areas, upholstered objects, and children's toys, such as stuffed animals, and can also be brought home from the workplace.[11–14] Herbicides applied on the lawn or garden can be tracked into the home, with residues building up over time.[15] Applications of diazinon to lawns have been demonstrated to be carried indoors via the paws of pet dogs.[16] Residential pesticide residue levels also vary geographically according to the specific pesticide needs in the area. In Los Angeles, high levels of chlorpyrifos and other insecticides were found because of the large numbers of crawling insects, fleas, and termites. Conversely, in Iowa, there were high levels of the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and dicamba because of weed-control applications.[17]

Residentially related sources may be relevant in other settings where children spend time, including school, child care, a relative's home, etc, depending on indoor and outdoor pesticide use patterns and proximity to pesticide use. In a North Carolina study of 142 urban homes and preschools, chlorpyrifos was detected in all indoor air and dust samples.[18]

Biomonitoring Data for Exposure Assessment

The Centers for Disease Control and Prevention (CDC) conducts a population-based biomonitoring program associated with the NHANES.[19] The most recent report includes biomarker data for many organochlorine, OP, and carbamate insecticides; herbicides; pyrethroid insecticides; and some other pesticides. Testing of 44 pesticide metabolites revealed that 29 were detectable in most people from whom samples were analyzed (ages 6–59 years), with OP and organochlorine insecticides reported to be most prevalent in the US population.[19] Although the health implications of these "snapshot" sampling data are largely unknown, they do

TABLE 1 Major Pesticide Classes and Selected Examples

Pesticide Class	Examples	Toxicity	Comment, Uses
Organochlorines	DDT, endrin, aldrin, chlordane, lindane	• High toxicity	• Many organochlorines now banned in the United States • Lindane has been banned in California, elsewhere used for control of lice and scabies • DDT and other organochlorines have long metabolic disposition and are stored in fatty tissues and can persist in the environment
Organophosphates	Parathion, chlorpyrifos, dichlorvos, acephate, methyl-parathion, malathion, phorate	• Most OPs are highly toxic • Malathion is considered relatively less toxic than other OPs	• Parathion is banned for use in the United States • Chlorpyrifos is no longer approved for residential use • Most others are used for insect control in both agricultural and home settings • Malathion is an approved treatment of head lice
N-Methyl carbamates	Aldicarb, carbaryl, carbofuran, pirimicarb, propoxur	• Aldicarb and carbaryl are both highly toxic • Other carbamates have a relatively moderate toxicity	• Insect control in agricultural and home settings
Pyrethrins and pyrethroids	Permethrin, cyano-pyrethroids: deltamethrin, cypermethrin, fenvalerate	• Permethrin has relatively low toxicity • Other pyrethroids have moderate toxicity	• Permethrin is a common pediculicide • Most other pyrethroids are commonly used to control insects, often used in home and garden
Neonicotinoids	Imidacloprid	• Relatively newer class of insecticides • Have relatively lower toxicity than OPs and carbamates	• Selective affinity toward insect nicotinic acetylcholine receptors compared with mammalian nicotinic acetylcholine receptors • Often used as spot-on flea control for domestic animals
N-Phenylpyrazole insecticides	Fipronil	• Relatively newer class of insecticides	• Often used as spot-on flea control for domestic animals • Yard treatments for insect control
Phosphonate herbicides	Glyphosate	• Because of primary mechanism of action, has relatively low toxicity from active ingredient. • Toxicity often due to the accompanying organic solvent	• Acts on plant cell wall • Commercially available in many products
Chlorophenoxy herbicides	2,4-D, 2,4,5-T	• Moderate toxicity	• Weed control
Dipyridyl herbicides	Paraquat, diquat	• Highly toxic	• Infrequently used • Paraquat toxicity often requires lung transplant
Long-acting anticoagulants	Brodifacoum (superwarfarins)		• Rodenticides • Longer-acting than warfarin • Recently eliminated packaging as loose pellets

2,4,5-T, 2,4,5-trichlorophenoxy acetic acid.

provide a reference point on pesticide metabolite distributions. Periodic reassessment also allows for evaluations of population-level exposure trends.

As noted previously, children's unique behaviors and metabolic rate often place them at risk for absorption of higher doses from contaminated environments in comparison with adults. One example evident from the biomonitoring data is chlorpyrifos, a nonpersistent OP insecticide. Although banned in 2000 for use inside the home, it continues to be used in agriculture, including orchard fruits, such as apples and pears, and other dietary staples of children. In the CDC biomonitoring data, chlorpyrifos-specific urinary metabolites were highest for the youngest age group assessed (6–11 years) compared with older children and adults.[19] In contrast, biomonitoring of serum markers of organochlorine insecticides and their metabolites, such as DDT, dieldrin, and chlordane, many of which were banned from use in the United States in the 1970s and 1980s, revealed lower concentrations in the youngest age group monitored (12–19 years). Despite relatively lower concentrations, the ongoing detection and the higher levels with increasing age likely reflect the influence of the accumulation of these fat-soluble, persistent compounds over a lifetime.

Exposures From the Food Supply

In the general population, the food supply represents the most important source of exposure for organochlorines and OPs. For pyrethroids, both food residues and household pest control products are important sources.[20] The US Environmental Protection Agency (EPA) regulates exposure to pesticides in food by setting "tolerances," which are the maximum amount of pesticides that may legally remain in or on food

and animal feed. The US Food and Drug Administration is responsible for enforcement of these tolerances, which includes a modest monitoring program, which analyzed 7234 total samples in 2003. Among the domestically produced samples, 49% of fruit, 29% of vegetables, 26% of grain products, 24% of fish/shellfish, and 0% of milk/dairy tested had detectable but legally allowable pesticide residues. Only fruit and vegetables had residues above the legal tolerance (approximately 2% each). Overall, the detection of residues in the samples from imported fruits and vegetables tested were less, but the exceedances of legal tolerances were greater (5%–7% of imported fruits/vegetables sampled).[21] Consumption of organic food may lower pesticide exposure, as demonstrated by a study in which children were placed on an organic diet for a period of 5 consecutive days. A rapid and dramatic drop in their urinary excretion of metabolites of malathion and chlorpyrifos OP insecticides during the organic diet phase was observed.[22]

Agriculturally Related Exposures

Proximity to pesticide-treated agricultural areas or household members that work with pesticides presents another opportunity for contamination of the residential environment for some children. In a Washington State study of children of agricultural workers and nonagricultural workers in an agricultural setting, pesticide levels in carpet dust and pesticide metabolites in urine of residents increased with self-reported proximity of homes to orchard fields and during the pesticide application season.[9,23] Similarly, in an agriculture center in California, pesticide residues of 3 chemicals used recently on crops were significantly correlated with house dust samples in nearby homes and urine samples among their inhabitants. The findings were noted in both farmworkers and nonfarmworkers.[24] The presence of an agricultural worker in the home also increases pesticide levels through "take-home" exposures.[23] Children living on a farm had higher urinary pesticide metabolite levels than children not living on a farm.[25] Children themselves may participate in agricultural work that involves the use of pesticides or contact with pesticide-treated foliage.[26–28]

Exposures From Drinking Water

Contamination of drinking water presents another potential source of exposure, particularly for herbicides. A 10-year study (1992–2001) by the US Geological Survey's National Water-Quality Assessment program provided a national-scale view of pesticide occurrence in streams and groundwater. Overall, pesticides were detected in more than 50% of sampled wells from shallow groundwater tapped beneath agricultural and urban areas as well as in 33% of the deeper wells that tap major aquifers used for water supply. The concentrations associated with these detections rarely exceeded water quality health reference levels (approximately 1% of the 2356 domestic and 364 public-supply wells that were sampled). Herbicides, particularly the triazine class, were the most frequently detected pesticide group in agricultural areas. (It should be noted that atrazine and other triazine herbicides were monitored from surface water.) In urban areas, both herbicides and insecticides (particularly diazinon and carbaryl) were frequently detected. The greatest proportion of wells exceeding a health reference level was for those tapping shallow groundwater beneath urban areas. It is noteworthy that the detection of pesticides usually occurred as mixtures, and health reference levels reflected exposure to a single agent.[29]

NATIONAL DATA ON ACUTE EXPOSURE, MORBIDITY, AND MORTALITY

Although some states (eg, California and Washington) mandate the reporting of pesticide-related illness, there is no national surveillance system for pesticide exposure and poisoning. The American Association of Poison Control Centers' National Poison Data System (NPDS [formerly known as the Toxic Exposure Surveillance System]) compiles annual data on pesticide exposures. Incidents reported by the NPDS are categorized by age (<6 years, 6–19 years, and >19 years), reason (unintentional, intentional, other, adverse reaction), and outcome (none [no morbidity], minor, moderate, major, or death). However, these data represent self-reports from patients and/or family members and calls from medical treatment facilities. Although they are useful to describe trends, they do not indicate true prevalence or incidence. Data are reported annually and, since 2005, have been published in *Clinical Toxicology*.[30]

In 2009, pesticides were the tenth most frequently involved substance in human exposure (3.9% of all NPDS reports) and the ninth most common substance encountered in children (3.3% of pediatric NPDS reports). Nearly 55.8% of all single-substance pesticide exposures involved children ≤19 years of age, and 94% of all pesticide ingestions were unintentional. Twenty-one of the reports from pesticide exposure resulted in death; however, these were not categorized by age.[30] Rates (calculated by using US census data for the catchment area served by the poison control center as the denominator) of reported pesticide poisonings described as moderate, major, and fatal declined from 1995 to 2004 by approximately 42%. The sharpest declines in poisonings were from OP and carbamate insecticides,

likely reflecting EPA regulatory action to discontinue residential use of several previously widely available OP and carbamate insecticides on the basis of child health concerns.[31]

ACUTE TOXICITY MECHANISMS AND CLINICAL MANIFESTATIONS

OP and Carbamate Insecticides

OP and carbamates insecticides have been widely used for insect control in the home and in agriculture since the 1960s. During this period, OP and carbamates usage largely replaced the use of organochlorines because of environmental and human health concerns of the latter class. In the past 10 years, chemical products in the OP and carbamate group have come under scrutiny, with subsequent regulatory action based on human health concerns. Examples include 2 commonly used OPs with high acute toxicity: parathion (banned) and chlorpyrifos (no longer allowed for residential use). Other OPs that remain widely used include dichlorvos, acephate, methyl-parathion, and malathion. Malathion has relatively lower acute toxicity among the OPs and is registered for the treatment of head lice (Ovide). A well-known example of a carbamate is aldicarb, although use has largely been curtailed by regulatory action because of its high toxicity. Commonly used carbamates include carbaryl and pirimicarb.[1,3]

Toxicity, Clinical Signs, and Symptoms

OPs and carbamates exert a common mechanism of action by inhibiting the acetylcholinesterase enzyme, thereby producing accumulation of acetylcholine at the synapses, neuromuscular junction, and end organs, which results in excessive stimulation at those sites. The reaction is generally an irreversible binding by OPs and a reversible binding by carbamates, and it influences treatment approaches for each class of insecticides. Consequently, acute poisoning by OPs tends to be more severe and refractory than that of carbamates; however, variations are observed in each class. There are some notable carbamates (such as aldicarb) that have equal if not greater toxicity than some OPs.[1,3]

Acute clinical manifestations reflect the development of cholinergic crisis and can arise from stimulation of muscarinic, nicotinic, and/or central nervous system receptors (Table 2). Early findings can often mimic a flu-like illness and include hypersecretion. Miosis is a helpful diagnostic sign. The classic cardiovascular sign is bradycardia, although early on, tachycardia may be present initially because of nicotinic stimulation. Progressive symptoms lead to muscle and respiratory problems. The central nervous system may also be affected, signifying severe poisoning, particularly in children.[1,3,32–34] Reviews of case series indicate that between 20% and 30% will have seizures, and between 50% and 100% of children will have lethargy, stupor, or coma.[32–34] A high clinical suspicion plus directed and persistent environmental history taking to identify potential exposures are necessary to identify these poisonings. Reviews of pediatric poisonings note that, historically, most children were transferred to a referral center with the wrong preliminary diagnosis and parents initially denied any exposure history.[33,34]

Laboratory Evaluation and Treatment

Poisoning with OPs and carbamates can be detected on the basis of clinical findings and history of exposure. Laboratory confirmation can assist in the diagnosis by using red blood cell and plasma cholinesterase levels; both are typically depressed with acute poisoning, although there is some variation among active ingredients as well as variation in levels by severity of poisoning.[35] Measurement techniques and resultant levels vary among laboratories; therefore, clinicians will need to check with their own laboratory for reference values. Red blood cell cholinesterase levels typically are more specific for acute poisoning and will be depressed longer than plasma cholinesterase levels (often 1–3 months) until enzyme is replaced.[3] Interpretation of results can be discussed with a pediatric environmental health specialist or clinical toxicologist.

The parent active ingredient cannot typically be measured in biological specimens. These compounds undergo metabolic transformation in the liver and are excreted in the urine mostly in their metabolized form, most of which are nonspecific metabolites for all OPs.[19] Exceptions include parathion, methyl-parathion, and chlorpyrifos, all of which have their own specific metabolite in addition to the nonspecific metabolites. Urinary metabolites can be measured, and human data are available from the CDC on a nationally representative sample.[19] However, an evidence base to support clinical interpretation of urinary concentrations is lacking.

Treatment of OP poisoning (and this applies to the acute treatment of any other pesticide as well) begins with the basics of advanced life support, with any necessary airway or breathing support as needed. Gastrointestinal (GI) decontamination is controversial. The American Academy of Clinical Toxicology and the European Association of Poisons Centres and Clinical Toxicologists issued a joint statement on the use of single-dose charcoal for poisoned patients (inclusive of all types of poisonings). They stated that activated charcoal is most effective when given within 1 hour after the ingestion of a poison, but routine administration in all poisonings is not recommended.

TABLE 2 Clinical Signs and Symptoms

Class of Compounds	Signs and Symptoms	Special Notes, Laboratory Evaluations, Specific Treatments, or Antidote
Organophosphate and carbamate insecticides	• Nonspecific early symptoms: headache, nausea, vomiting, abdominal pain, and dizziness	• Red blood cell and plasma cholinesterase levels
	• Sometimes hypersecretion: sweating, salivation, lacrimation, rhinorrhea, diarrhea, and bronchorrhea	• Measure nonspecific metabolites for most OPs
	• Progressive symptoms: muscle fasciculation, muscle weakness, and respiratory symptoms (bronchospasm, cough, wheezing, and respiratory depression)	• Specific metabolites can be measured for chlorpyrifos and parathion
	• Bradycardia is typical, although early in acute poisoning, tachycardia may be present	• Atropine is primary antidote
	• Miosis	• Pralidoxime is also an antidote for OP and acts as a cholinesterase reactivator
	• Central nervous system: respiratory depression, lethargy, coma, and seizures	• Because carbamates generally produce a reversible cholinesterase inhibition, pralidoxime is not indicated in these poisonings
Pyrethroids	• Dermal: skin irritation and paresthesia	• At times have been mistaken for acute OP or carbamate poisoning and treated with atropine with potentially adverse or disastrous results
	• Nonspecific symptoms including headache, fatigue, vomiting, diarrhea, and irritability	• Symptomatic treatment
	• Similar findings found in OPs, including hypersecretion, muscle fasciculation, pulmonary symptoms and seizures	• Vitamin E oil for dermal symptoms
Neonicotinoids	• Disorientation, agitation—severe enough to require sedation, drowsiness, dizziness, weakness, and, in some situations, loss of consciousness	• Supportive care
	• Vomiting, sore throat, abdominal pain	• No available antidote
	• Ulcerations in upper GI tract	• No available diagnostic test
Fipronil (*N*-phenylpyrazole insecticides)	• Nausea and vomiting	• Supportive care
	• Aphthous ulcers	• No available antidote
	• Altered mental status and coma	• No available diagnostic test
	• Seizures	
Organochlorines	• Central nervous system: mental status changes and seizures	• Control acute seizures with lorazepam
	• Paresthesia, tremor, ataxia, and hyperreflexia	
Glyphosate (phosphonate herbicides)	• Nausea and vomiting	• Supportive care
	• Aspiration pneumonia type syndrome	
	• Hypotension, altered mental status, and oliguria in severe cases	
	• Aspiration pneumonia type syndrome	
	• Pulmonary effects may in fact be secondary to organic solvent	
Chlorophenoxy herbicides	• Skin and mucous membrane irritation	• Consider forced alkaline diuresis with sodium bicarbonate in IV fluids
	• Vomiting, diarrhea, headache, confusion	
	• Metabolic acidosis is the hallmark	
	• Renal failure, hyperkalemia, and hypocalcemia	
Long-acting anticoagulants (rodenticides)	• Bleeding: gums, nose, and other mucous membrane sites	• Consider PT (INR) or observation
	• Bruising	• Vitamin K indicated for bleeding (IV vitamin K) or for elevated PT (INR) (oral vitamin K)

IV, intravenous.

Activated charcoal is contraindicated if the patient does not have a protected or intact airway.[36] A randomized controlled trial evaluating the effect of multiple-dose charcoal for pesticide-poisoned patients in Asia found no benefit, as measured by a reduction in mortality.[37] Skin decontamination also is critically important, and clothing should be removed. Medical personnel should take measures to protect themselves from contaminated skin and clothing, because numerous cases of hospital-acquired OP poisoning have been documented.[38] Parents or other family caregivers may also be at risk for skin contamination. Seizures should be controlled with intravenous lorazepam.[3]

Atropine can be given as a nonspecific antidote in both OP and carbamate poisoning. It will reverse the muscarinic effects of the poisoning; however, it is less effective on central nervous system effects. It is given as a dose of

0.05 to 0.1 mg/kg per dose and may be given as often as every 15 minutes until respiratory secretions are controlled.[3] Notably, this dose is 10 times the usual dose given during a resuscitation situation, because the purpose is to overcome complete blockade of the muscarinic channel. Pralidoxime is also given as a specific antidote to reverse the acetylcholinesterase inhibitor complex. The use of pralidoxime continues to be of interest, particularly in developing countries, although most studies have been performed with adult patients.[39,40] The World Health Organization recommends its use for all patients who require atropine.[41] Its use is indicated for OP poisoning, because cholinesterase inhibition usually is permanent in OP poisoning. Use of pralidoxime usually is not necessary or recommended for carbamate poisoning, because this inhibition is reversible.[3]

Pyrethrins and Pyrethroid Insecticides

Pyrethrins and pyrethroids are a relatively more recent class of insecticides that have been largely replacing the use of cholinesterase-inhibiting insecticides, especially in the consumer market. These insecticides are used for structural pest control in urban areas, in gardening or agriculture for row crops and orchards, and in the home for pet sprays and shampoo.

The pyrethrins are botanically derived from pyrethrum, an extract of the chrysanthemum plant. For these consumer products, pyrethrins are usually combined with another active ingredient: either a longer-acting synthetically derived pyrethroid or one of the cholinesterase inhibitors. Pyrethrins are not stable in heat or sunlight and, therefore, are usually used more for indoor application. Permethrin is the most widely known example of a pyrethrin and is one of the few products licensed for use to apply to human skin, because it is commonly used as a pediculicide.[3,42,43]

Pyrethroids are synthetically derived compounds that have been modified to be more stable in sunlight and heat and are, therefore, used more widely for insect control, especially outdoors. Toxicity varies widely among pyrethrins and pyrethroids, and, although they are less acutely toxic as a class than the cholinesterase insecticides, there is a subgroup of these compounds that has been modified with a cyano side chain. This modification creates a compound that is significantly more resistant to degradation and potentially more acutely toxic than other pyrethroids. Commonly used chemicals in this subgroup include deltamethrin, cypermethrin, and fenvalerate—these are the insecticides to which the majority of toxic signs and symptoms in the next section apply.[43]

Toxicology, Clinical Signs, and Symptoms

Pyrethroids exert their toxic effect by blocking the sodium channel at the level of the cell membrane. Most clinical reports of poisoning occur either through excessive skin contact or through ingestion or inhalation. The result is continued hyperpolarization, effectively inhibiting cell function. Some types of pyrethroids also work at other sites, including voltage-dependent chloride channels and γ-aminobutyric acid–gated chloride channels. This appears to be one of the reasons for a variety of toxicity found among pyrethroid insecticides.[42,43] Pyrethroids with a cyano group, also known as type II pyrethroids, constitute most cases of human poisoning.[42,43] Pyrethroids are well absorbed across the GI tract, but limited penetration occurs across the skin barrier, which can limit acute toxicity.[42,44] Some pyrethroids have a high acute toxicity, usually after ingestion.[42,45] Pyrethroids are metabolized by the liver and excreted in their metabolic forms.

Pyrethroids have adverse effects on the nervous system, GI tract, and skin. Specific signs and symptoms are found in Table 2. Similar to OPs, muscle fasciculation, weakness, an altered level of consciousness, and seizures can develop after exposures to some pyrethroids.[42–45] Of note, paresthesias, including burning, tingling, stinging, and eventually numbness, are characteristic of pyrethroid exposure.[46,47] The paresthesias appear to be dose-dependent and occur at pyrethroid dosages lower than what would cause systemic toxicity, thereby acting as a warning of exposure. The paresthesias are self-limiting once exposure is eliminated.[48]

Laboratory Evaluation and Treatment

Pyrethroid toxicity is identified through clinical history and knowledge of exposure to the agent. There are no rapidly available diagnostic laboratory tests. Most pyrethroids are metabolized to 3-phenoxybenzoic acid, which can be recovered in the urine. CDC national surveys provide biomonitoring information on pyrethroid urinary metabolites and can act as comparison for background measures of exposure in the general population. However, in the clinical setting, results of metabolite levels are usually obtained from specialty laboratories and are not immediately available; therefore, these results not useful in acute clinical management.

Paresthesias are generally self-limiting and resolve within 24 hours.[46,48] If exposure is interrupted after the onset of paresthesias and other dermal findings, no additional treatment is necessary. Vitamin E oil or cream has been shown to improve the

symptoms associated with the paresthesias.[47] The mechanism is not completely clear; however, in experimental studies, vitamin E (α-tocopherol) blocked tetramethrin-modified sodium channels.[49]

Treatment of systemic pyrethroid poisoning is supportive, in general, and there are no specific antidotes. Because of the similar features of cholinesterase inhibitor poisoning, some patients have been treated erroneously with high atropine, sometimes with disastrous results.[45] Efforts have been aimed at antagonizing the sodium current resulting from the pyrethroid blockade. Several medications have been tested in the animal model, but, to date, none have been considered effective antidotes for systemic pyrethroid poisoning in humans. For significant neurologic effects, patients should have standard decontamination, including GI tract decontamination, supportive respiratory care, seizure control with diazepam or lorazepam, and careful dosing of atropine for excessive salivation.[42] Proper identification of the offending agent is imperative to distinguish these poisonings from OPs and often requires a high index of suspicion and a thorough exposure history.

Organochlorine Insecticide (Lindane)

The discussion of acute toxicity for organochlorines is focused on lindane, because most other organochlorine compounds have been banned for use in the United States. Other organochlorines, including DDT and some of the cyclodienes, including chlordane and dieldrin, are important compounds, because they can still persist in human and environmental samples. These chronic exposures are of continuing concern for developmental health effects, including immunotoxicity, endocrine disruption, and neurodevelopmental insults (see Chronic Health Effects of Pesticide Exposure).

Lindane, also known technically as the γ-isomer of hexachlorocyclohexane, is still approved in some states for control of lice and scabies. However, in a comparison of in vitro activity against lice with other pediculicides, it was the least effective.[50] It is efficiently absorbed across the skin (approximately 9%) and even more so across abraded skin, such as with severe excoriations from scabies.[51,52] Signs and symptoms are noted in Table 2. Treatment is supportive and includes decontamination and the control of seizures with lorazepam. There is no specific antidote. Lindane has been banned in California because of high levels found in the water supply.[53]

Neonicotinoids

Neonicotinoids are a new class of insecticides based on metabolic alterations of nicotine. They are used primarily in agriculture and are gaining widespread use for flea control on domestic animals. They act on the nicotinic *N*-acetylcholine receptors and selectively displace acetylcholine. They do have a relatively selective affinity for insects as opposed to mammals, although there have been a few reports of human poisoning.[54–56] The most commonly used neonicotinoid in the United States is imidacloprid. Information about toxicity and signs and symptoms can be found in Tables 1 and 2.

N-Phenylpyrazoles

Fipronil is the primary representative of this class and was developed in the mid-1990s. It is widely used in flea control on domestic pets. It is also used in ant and roach bait stations, agriculture crops, and lawn treatments. It acts by inhibiting γ-aminobutyric acid–gated chlorine channels. The inhibition will block chloride passage and result in hyperexcitability of the cell.[57–59] Signs and symptoms are reported in Table 2.

HERBICIDES

Chlorophenoxy Herbicides

Chlorophenoxy herbicide compounds are often mixed with fertilizers and are used both in agriculture and on residential lawns. These compounds are well absorbed from the GI tract but are not well absorbed after inhalational or dermal exposure.[60] Examples of commonly used chlorophenoxy herbicides are 2,4-D and 2,4,5-trichlorophenoxy acetic acid. The half-lives of these compounds range between 13 and 39 hours. They are mostly excreted unchanged in the urine; excretion can be greatly enhanced in an alkaline environment.[3,61,62] More toxic substances that can be produced during the manufacture of these herbicides include dioxins, which were contaminants of the herbicide Agent Orange and were found in the Love Canal chemical dump site.[63]

Primary initial effects are on the skin and mucous membranes. Severe poisoning will result in metabolic acidosis and possibly renal failure.[3,61,64] Specific symptoms are discussed in Tables 1 and 2. The compounds can be measured in the urine, although similar to pyrethroid insecticides, analyses are generally performed at specialty laboratories, so results are usually not immediately available to clinicians. Treatment is primarily supportive and may also include forced alkaline diuresis by adding sodium bicarbonate to the fluids and establishing a high urine pH and high urine flow.[3,61,65]

Phosphonate Herbicides (Glyphosate)

Glyphosate is a commonly used herbicide and is commercially available in

many products. Glyphosate acts on the cell wall of plants, so, theoretically, it should have no effect on human cells, at least by way of its primary mechanism of action. Despite this, there are numerous reports in the medical literature of adverse events after human exposure, particularly unintentional ingestions. Patients have presented with signs and symptoms consistent with an aspiration pneumonia–like syndrome, and the offending agent may be the hydrocarbon solvent with which the glyphosate is mixed. Treatment is primarily supportive, and providers should be vigilant for aspiration pneumonia.

RODENTICIDES (LONG-ACTING ANTICOAGULANTS)

Most currently used rodenticides belong to the class of warfarin-type anticoagulants. Unlike warfarin, the superwarfarin agents, such as brodifacoum, have a much longer half-life. Although they have traditionally been available as pellets that can be spread around or in a box that the rat can consume, the EPA has recently changed the type of products that are available to consumers. Since 2008, superwarfarins can only be sold as a child-resistant bait station instead of loose pellets.[66]

The mechanism of action is inhibition of the synthesis of vitamin K–dependent clotting factors. As such, the primary manifestations of toxicity are bleeding and easy bruisability. In severe cases, bleeding may be life-threatening. Clinicians who suspect that their patients may have ingested a superwarfarin should consider obtaining a prothrombin time (PT; also known as the international normalized ratio [INR]).[3] However, several studies that have analyzed cohorts of exposed children have found very few subjects with an elevated PT (INR) or active bleeding. Therefore, in situations in which it is unclear whether a child ingested more than a few pellets, it is reasonable to simply observe the child.[67–70] Most patients can be managed in the outpatient setting as long as the ingestion has been recognized early.[71]

Treatment is vitamin K and should be reserved for patients with elevated PT (INR) levels or active bleeding. With severe bleeding or shock, a transfusion of blood or plasma is indicated as well.[3]

CHRONIC HEALTH EFFECTS OF PESTICIDE EXPOSURE

The health implications of the nonacute, relatively low, but often repetitive and combined exposures encountered routinely by children are an ongoing focus of concern and inquiry for scientists, regulators, and parents.[72,73] Pediatricians are well placed to provide guidance to parents about potential long-term or subtle health effects from pesticide residues on food, in water, or used in homes or schools and on exposure-reduction strategies. However, surveys suggest pediatricians often feel ill-prepared with training in this topic, underscoring the importance of improving educational opportunities for clinical providers.[74–76]

The associated health effects of chronic pesticide exposure in children vary, reflecting the diversity of toxicological properties of this broad group of differing chemicals. Some of the important end points of concern include an increased risk of cancer, abnormal neurodevelopment, asthma, perturbation of gestational growth, and endocrine-mimicking effects. Health effects of pesticides and the current relative strength of the evidence base are reviewed in subsequent sections for each of these health outcomes.

Childhood Cancer

All pesticides undergo in vitro and animal testing to determine their likelihood of causing cancer. The EPA maintains a list and classification of all active ingredients in pesticides and their potential for carcinogenicity. The method of identifying potential carcinogenicity has changed. Before 1996, pesticides were assigned a letter classification (eg, pesticides with the "C" classification were considered "possibly carcinogenic"). Subsequently, pesticides have been assigned a category such as "likely to be carcinogenic to humans," "suggestive evidence of carcinogenic potential," "inadequate evidence," and "not likely." These categories are not directly comparable, so both classifications (before 1996) and categories (after 1996) continue to exist.

The pesticides that are categorized as "possibly carcinogenic" or "likely to be carcinogenic to humans" are available from the EPA via an e-mailed report.[77] Included in this report are some well-known and widely used OPs, carbamates, pyrethroids, and fungicides. Within classes of pesticides, variation in carcinogenicity potential exists. Note that a pesticide, such as cypermethrin, that has "replaced" use of cancer-causing OPs has cancer-causing potential.

A substantial amount of observational epidemiological data demonstrate a link between pesticide exposure and childhood cancers.[78–87] However, the evidence base includes studies that found no association between childhood cancers and pesticides or few associations that cannot be ruled out as a chance finding.[88,89] Overall, the most comprehensive reviews of the existing literature implicate an association of pesticides with leukemia and brain tumors.[78,79]

Leukemia

In 1998, Zahm and Ward[79] reviewed 18 studies assessing the relationship between pesticide exposure and leukemia; 13 studies found an elevated risk, and,

for 6 of those studies, the association was statistically significant. The most frequently occurring associations among the studies were between pesticide exposure and acute lymphocytic leukemia.

A 2007 review by Infante-Rivard and Weichenthal[78] summarized the 1998 review of Zahm and Ward and updated findings from recent studies. Although it was previously postulated that childhood exposure to agricultural products or proximity to an agricultural setting would present the highest risks, the most commonly associated pesticide exposure in childhood acute lymphocytic leukemia studies was household insecticide use. Cases were more likely to have had preconception exposure and/or exposures in utero in most studies. The main limitations with the studies in the 1998 review included crude exposure assessment, concern for recall bias, small numbers of exposed cases, and mixing of different leukemia types.[78]

In the updated review, 5 of 6 recent case-control studies found a statistically significant relationship between pesticide exposure and leukemia.[84,85,90–92] In particular, 2 studies included the most detailed exposure assessment to date and reported findings related to a dose/exposure–response gradient.[84,85] The primary risk factors were maternal exposure to pesticide between the periods of preconception through pregnancy. The largest of the 2 studies had 491 cases and an equal number of controls, focused only on acute lymphocytic leukemia, included a measure of frequency of use, and considered genetic susceptibility. For maternal use of herbicides, plant insecticides, and pesticides for trees during pregnancy, the odds ratio (OR) was 1.84 (95% confidence interval [CI], 1.32–2.57), 1.97 (95% CI, 1.32–2.94), and 1.70 (95% CI, 1.12–3.59), respectively. For parental use during the child's postnatal life, OR was 1.41 (95% CI, 1.06–1.86), 1.82 (95% CI, 1.31–2.52), and 1.41 (95% CI, 1.01–1.97) after exposure to herbicides, plant insecticides, and pesticides for trees, respectively.[84]

To further explore associations between pesticides and leukemia, a group of authors conducted 2 meta-analyses. They provided similar and additional support to the associations described previously. One examined studies that included parental occupational exposure (prenatally and in early childhood) and leukemia in their offspring. Maternal occupational exposure, but not paternal occupational exposure, was found to be associated with leukemia. The reported OR was 2.09 (95% CI, 1.51–2.88) for overall pesticide exposure, 2.38 (95% CI, 1.56–3.62) for insecticide exposure, and 3.62 (95% CI, 1.28–10.3) for herbicide exposure.[93] The second meta-analysis assessed pesticide exposure in the home and garden setting. In this meta-analysis, 15 studies were included, and exposures during pregnancy to unspecified pesticides, insecticides, and herbicides were all associated with leukemia (OR, 1.54 [95% CI, 1.13–2.11], 2.05 [95% CI, 1.80–2.32], and 1.61 [95% CI, 1.2–2.16], respectively).[94]

Brain Tumors

Zahm and Ward's 1998 review included 16 case-control studies examining associations between brain tumors and pesticide exposures. Of these, 12 found an increased risk estimate of brain tumors after pesticide exposure; 7 of these findings reached statistical significance. Associated exposures were most often from parental use of pesticides in the home, in the garden, and on pets. Interpretation of these studies is difficult given the inadequate exposure assessments, small numbers because of a relatively rare childhood outcome, and a mixture of brain tumor types among cases.[79]

Since 1998, 10 additional studies have been published, all but one of which demonstrated an increased risk estimate of cancer with maternal and/or paternal exposure, although not all studies demonstrated statistical significance. Some of the more robust findings come from a case-control study with 321 cases of astrocytomas. The risk estimate from maternal occupational exposure to insecticides before or during pregnancy was 1.9 (95% CI, 1.1–3.3). The risk estimates for paternal exposure for insecticides, herbicides, and fungicides were 1.5, 1.6, and 1.6, respectively. These risk estimates were just short of reaching statistical significance.[87] In a cohort study of more than 200 000 patients, paternal exposure in any occupation and in agricultural/forestry preceding conception was associated with an increased risk of central nervous system tumors (relative risk [RR], 2.36 [95% CI, 1.27–4.39] and RR, 2.12 [95% CI, 1.08–4.39], respectively).[83] For all studies, it appears that prenatal exposure to insecticides, particularly in the household, as well as both maternal and paternal occupational exposure before conception through birth represent the most consistent risk factors.[83,86,87,95–100]

Ewing Sarcoma

Two case-control studies were performed to evaluate potential parental occupational exposures and the development of Ewing sarcoma (ES). One study of 196 cases and matched controls found an association between ES in boys age 15 years or younger and household pesticide extermination (OR, 3.0; 95% CI, 1.1–9.2). There was no association between parental occupational exposure to pesticides and ES.[101] A study in Australia compared 106 cases of either ES or peripheral primitive neuroectodermal tumor with 344 population-based controls. Exposures

included prenatal exposure from conception through pregnancy and also included parental exposures through the time of the child's diagnosis. Notable elevated risks were observed for mothers who worked on farms (OR, 2.3; 95% CI, 0.5–12.0), mothers who handled pesticides (OR, 2.3; 95% CI, 0.6–8.5), patients who ever lived on a farm (OR, 2.0; 95% CI, 1.0–3.9), and farming fathers at the time of conception and/or pregnancy (OR, 3.5; 95% CI, 1.0–11.9).[102] Of note in this study, all 95% CIs include 1.0, so they did not reach statistical significance, although some ORs approached it.

In summary, there is some evidence of increased risk of developing several childhood cancers after preconception and/or prenatal exposure to pesticides. The strongest evidence appears to be for leukemia, which is a relatively more common type of childhood cancer than brain tumors. Maternal exposure to insecticides and paternal occupational exposure appear to carry the greatest risk.

Neurodevelopment/ Neurobehavioral Effects

Many pesticides have well-described acute neurotoxicant properties that have been described previously in this report in relation to human poisoning episodes and acute toxic mechanisms. However, information on the potential neurodevelopmental toxicity arising from chronic, low-level exposure in gestational or postnatal life is inadequate or lacking for most pesticides in use. There is a growing available evidence base supporting an adverse effect on neurodevelopment from 2 classes of insecticides, the organochlorines (specifically DDT and its metabolite *p,p'*-dichlorodiphenyldichloroethylene [DDE]) and, most recently, OPs. Several recent reviews of the evidence base are now available.[103–105]

Although chronic neurologic sequelae after acute OP poisoning have been observed in multiple adult studies, the epidemiological data on children are limited.[106,107] A recent neuropsychological evaluation of healthy school-aged children who had experienced hospitalization for acute OP poisoning before the age of 3 years found subtle but significant deficits in their ability to restrain and control their motor behaviors compared with both children who had no history of poisoning and children who had a history of early life poisoning with kerosene.[108]

Of greater public health concern is the potential neurotoxicity from routinely encountered chronic exposures. This is the subject of study in ongoing, large National Institutes of Health/EPA-sponsored prospective birth cohorts. Studies in 2 urban settings and a rural farmworker community have enrolled women during pregnancy with an objective assessment of exposure by the use of environmental measurements and biological monitoring.[104,109,110] Follow-up assessment of neurodevelopment and neurobehavior in their children with the use of validated tools such as the Brazelton Neonatal Assessment Scales, the Bayley Scales of Infant Development, the Child Behavior Checklist, and IQ testing at comparable intervals is being conducted. To date, remarkably similar findings relating adverse neurodevelopmental and neurobehavioral outcomes associated with prenatal OP exposure have been made in these distinct cohort studies. For example, in 2 cohorts, the Brazelton Neonatal Behavioral Assessment Scale was administered in the first weeks of life. In both, deficits in the primitive reflex domain were noted with the other 6 of 7 Brazelton Neonatal Behavioral Assessment Scale domains not associated with prenatal OP exposure.[111,112] Two of the cohorts have published their Bayley Mental and Psychomotor Developmental Index results conducted during the toddler years (ages 2–3).[113,114] Significantly poorer mental development was associated with higher OP exposure in both, whereas one of the cohorts also observed OP-associated deficits in the motor scale at 3 years of age. Results of Child Behavior Checklist assessments are also available for 2 cohorts, conducted at 2 years of age in one and 3 to 4 years of age in the other. Significantly increased scores representative of pervasive developmental disorder were associated with higher OP exposure in both.[113,114] One cohort also had increased scores for inattention and attention-deficit/hyperactivity disorder subscales.[114] All 3 cohorts have found decrements in IQ testing associated with higher prenatal exposures at the time of follow-up at 7 years of age.[115–117]

In one of the cohorts, postnatal exposure effects in the child have been investigated and reported. Interestingly, improved mental development based on Bayley's Index at 12 and 24 months of age is associated with higher contemporary child excretion of OP urinary metabolites. Explanations for this are debated but include theories that children with higher cognitive abilities may explore their environments more thoroughly and, as such, experience higher exposure.

Recently, a US-based cross-sectional analysis demonstrated that children with high urinary concentrations of OP metabolites were more likely to have a diagnosis of attention-deficit/hyperactivity disorder. This study used data from a representative sample of 8- to 15-year-old children collected as part of the NHANES conducted by the CDC.[118]

One study based in Ecuador has examined the relationship of OP exposure on neurodevelopment in school-aged children.[119] Prenatal exposure (based

on mother occupational history questionnaire) was associated with a decrease on the Stanford-Binet copying test among the study subjects at 7 years of age. Their concurrent exposure (on the basis of OP urinary metabolites) was associated with an increase in simple reaction time.

The toxicological mechanisms that underlie the adverse neurodevelopmental observations are also under investigation. Interestingly, noncholinergic mechanisms are being deciphered in animal models and in vitro studies, distinct from the well-described mechanism of acute OP toxicity (cholinesterase inhibition) and occurring at doses much lower than required to inhibit cholinesterase.[120]

Well-designed recent cohort studies and previous work including animal models suggest that OP exposures that are being experienced by US children may have adverse neurodevelopmental consequences. The plasticity of these effects and clinical implications are as yet unclear, although continued assessments as these cohorts age and enter school age are planned and may add clarity. The potential modification of these effects on the basis of genetic factors, specifically metabolic enzymes involved in pesticide detoxification pathways, are also being explored in these cohorts. For example, preliminary analyses indicate that children with a particular variant of the paraoxonase I gene, which is associated with lower levels of this OP-metabolizing enzyme, may be at higher risk of health consequences from OP exposure.[121,122]

Although DDT has not been used since the early 1970s, its persistence in the environment and fat solubility results in ongoing detection of the parent compound and breakdown product (DDE) in contemporary US populations.[19] The potential adverse neurodevelopmental consequences of prenatal DDT (2 studies) and DDE (several studies) was studied in one of the recent cohorts described previously in this report, which was a predominately Mexican American farmworker population. In this cohort, maternal serum DDT levels were negatively associated with mental development and psychomotor development at 12 and 24 months.[123] Maternal serum DDE was associated with reduced psychomotor development at 6 months and mental development at 24 months. A review of the overall evidence base reveals that studies of in utero DDE exposure and neurodevelopment are mixed, with at least 2 studies showing decrements in psychomotor function. Both of the 2 studies that have evaluated effects of DDT exposure observed cognitive deficits.[103]

In summary, the existing and recently emerging evidence base suggests that organochlorine and OP exposure in early life, particularly prenatally, may have adverse consequences on child neurodevelopment.

Physical Developmental Effects

In addition to neurodevelopmental toxicity, there is also considerable concern of physical developmental toxicity to the embryo and fetus from pesticide exposure. These concerns arise from multiple epidemiological studies that have investigated their relationship to adverse pregnancy outcomes including intrauterine growth retardation, preterm birth, fetal death, and congenital anomalies. The available studies are heterogeneous in design, are conflicting in results, and often have an insufficient exposure assessment. Nonetheless, pesticides remain one of the most common environmental exposures of concern cited in relation to adverse pregnancy outcomes and have been the focus of recent reviews on the topic, which include weight of the evidence evaluations.[124–126]

Among studies that are able to address specific types of pesticide exposures, there are more data focused on the organochlorine and OP insecticides or phenoxy or triazine herbicides. These represent the currently or historically (eg, organochlorine) most heavily used pesticides. This review summarizes the highlights of the existing evidence base with a focus on studies that incorporate direct measures of exposure for individual study subjects.

Fetal Death and Birth Defects

A California-based case-control study found an increased risk of fetal death attributable to congenital anomalies when OP application occurred in the residential area of the mother during weeks 3 through 8 of pregnancy—consistent with organogenesis.[127] One other study found an elevated risk of spontaneous abortion associated with chlorophenoxy herbicides. However, as with some studies of birth defects discussed previously, this study also relied on self-report and less reliable means of exposure assessment.[128] Results are not consistent, because other studies have not found association of parental exposure to OPs with spontaneous abortion or stillbirth.[129–131]

Birth defects will be discussed first, followed by other adverse birth outcomes. The more common birth defects include orofacial clefts, limb defects, and neural tube defects, which are generally the defects studied in relationship to pesticide exposures. Although several studies have found associations of maternal or paternal exposures with a wide variety of birth defect categories, all of the studies used indirect measures of exposure and most were ecological study designs, making interpretation of the adverse birth outcome evidence base inadequate and unreliable.[125]

A 1995 review article discussed the available evidence for associations between birth defects and potential

pesticide exposure.[132] Five studies were included that assessed various birth defects (central nervous system, oral cleft, limb defects) compared with maternal agricultural occupation. Four of those 5 reported an elevated RR or an OR ranging from 1.6 to 5.0; however, only 2 were statistically significant.[133–137] Of note, in these studies, there was not an assessment to any single pesticide; rather, the "exposure" was maternal occupation.

Six additional studies from this period evaluated maternal pesticide exposure at work and the development of birth defects. Of the 5 studies with an elevated OR or RR, ranging from 1.3 to 7.5,[138–142] 3 were statistically significant. Unfortunately, some of these studies included small numbers of cases, and others were likely to have significant exposure misclassification. The conclusion of this review was that there are some indications of elevated risk but no clearly convincing evidence.[143]

Two studies from Minnesota have reported a relationship between physical defects in children and paternal occupation of pesticide applicator. The first study compared data from a birth registry between 1989 and 1992. A geographic section of Minnesota that had the highest agriculture activity and highest frequency of use of chlorophenoxy herbicides and fungicides was also found to have the highest rate of birth defects (30.0/1000). By comparison, the general population in this same region had a birth defect rate of 26.9/1000. Interestingly, there was a seasonal effect, with the highest frequency occurring in infants who were conceived in the spring, the same time as most herbicide and some fungicide application (OR, 1.36; CI, 1.10–1.69).[144] The second study is a cross-sectional study that used a survey of licensed applicators and subsequently more in-depth interviews of either/both the applicator and female partners of licensed applicators when possible. The study eventually included live births fathered by 536 applicators. The birth defect rate in this study was 31.3/1000, which is statistically significantly higher than what the previous study found for the general population. Again, there was a significant difference in season of conception (7.6% in spring versus 3.7% in other seasons).[145]

Studies of birth defects often include all types within the analysis because of insufficient numbers of individual defects to allow adequate power of statistical analyses. A meta-analysis used 19 studies that had sufficient data to be included to estimate the effects of pesticides on orofacial clefting. Maternal occupational exposure to pesticides was associated with orofacial clefts (OR, 1.37; 95% CI, 1.04–1.81). There was a weaker association for paternal occupation (OR, 1.16; 95% CI, 0.94–1.44).[146] Studies on 3 other birth defects—cryptorchidism, hypospadias, and polythelia—will be discussed in the section on endocrine effects.

In summary, a small risk elevation is noted for birth defects and pesticide exposure, but the findings are not robust, and the data specific to pesticide subtypes are not adequate.

Adverse Birth Outcomes (Low Birth Weight, Decreased Gestational Age)

DDT (and its major metabolite DDE) is the organochlorine that has been most extensively examined in relation to birth defects, fetal death, and fetal growth, with mixed findings. Fetal exposures, as determined by maternal serum or umbilical cord blood levels, have been associated with preterm birth, decreased birth weight, and intrauterine growth retardation.[147–151] However, not all studies reported significant associations between exposure with infant birth weight or preterm birth, including a relatively recent study of Mexican American farmworking women in the United States with higher exposures in comparison with a similar group of a national sample of nonfarmworking Mexican American women.[142,152] In the largest cohort study to date (a US cohort of births between 1959 and 1966), DDE concentrations in maternal serum during pregnancy demonstrated a dose–response relationship to risk of preterm delivery and delivering small for gestational age (SGA) infants.[147]

Exposure to pesticides is associated with risk of decreased birth weight. In a study conducted before recent regulatory actions that reduced their residential use, exposure to the OPs chlorpyrifos and diazinon were associated with decreased birth weight in a New York City cohort.[110] In another New York City cohort, birth weight was reduced among mothers with higher OP exposure levels in pregnancy, but only among those with a genetic polymorphism of an OP detoxification enzyme (paraoxonase 1 or PON1).[150] In a similar longitudinal pregnancy cohort conducted among Latina farmworkers in agricultural California, no association of maternal pregnancy exposure to OPs and birth weight was determined, but a reduction in gestational age was associated.[153]

An ecological study determined that women in a rural region of Iowa with increased levels of triazine, metolachlor, and cyanazine herbicides in the drinking water had an elevated risk of delivering an infant with intrauterine growth retardation compared with women in other parts of the state.[154] A study based in France reported that atrazine levels in municipal drinking water throughout pregnancy were not associated with increased risk of delivering an SGA infant but that the

risk of delivering an SGA infant increased when the third trimester occurred in whole or in part during the period of May through September, when atrazine levels typically peak.[155]

Summary: Physical Developmental Defects

In summary, the true extent and nature of pesticide exposure on adverse fetal growth and birth outcomes is unknown despite suggestive epidemiological studies that link some of the most widely used pesticides to reduced intrauterine growth, fetal death, preterm birth, and congenital anomalies. Very little is known about many pesticide types in current use, including synthetic pyrethroids and carbamate insecticides, rodenticides, and fungicides. Studies that examine the timing and extent of exposure to pesticides and exposure to pesticide mixtures with validated exposure assessment techniques including biological markers are needed. The potential for differential vulnerabilities because of genetic polymorphisms that influence the toxicological properties of these exposures must also be explored.

ENDOCRINE EFFECTS

An emerging concern, although less well studied in humans, is the potential effects that some chemicals including pesticides may have on the endocrine system. Some of the most notable pesticides thought to have such effects are the organochlorine pesticides, such as DDT, endosulfan, methoxychlor, chlordecone, chlordane, and dieldrin. Other herbicides (atrazine, 2,4-D, and glyphosate) and fungicides (vinclozolin) also have some endocrine activity.[156–159] The associations are very complex and are primarily based on in vitro and animal studies. Estrogen-mimicking properties tend to be the most commonly reported, although effects on androgen and thyroid hormones, among others, are also reported. Feminization has been noted in alligators found in lakes highly contaminated by organochlorine pesticides.[160] Hayes et al[161] have studied the effects of atrazine on amphibians and have noted a 10-fold decrease in testosterone from exposure to 25 ppb of atrazine in mature male frogs. The mechanism of the latter appears to be activation of the enzyme aromatase, which promotes conversion of testosterone to estrogen.[162]

The human epidemiology literature is limited on endocrine effects from pesticides. One report from Macedonia noted some degree of early pubertal findings, primarily premature thelarche, which was hypothesized to be related to organochlorine pesticide exposure.[163] A study in 2000 with 48 patients, 18 of which had cryptorchidism, first raised the hypothesis about an association with organochlorine pesticides. An association between cryptorchidism and organochlorine pesticide levels has been hypothesized.[164] Since then, additional case-control studies have been conducted to examine the effects of organochlorines on endocrine-related birth outcomes, cryptorchidism, hypospadias, and/or polythelia. Two focused on fetal exposures from maternal levels of DDE alone and development of cryptorchidism and hypospadias.[165,166] Bhatia et al[165] calculated an OR of 1.34 (95% CI, 0.51–3.48) for the association of cryptorchidism and DDE and 1.18 (95% CI, 0.46–3.02) for the association of hypospadias and DDE. Longnecker et al[166] estimated an OR of 1.3 (95% CI, 0.6–2.4) for the association between DDE and cryptorchidism and an OR of 1.2 (95% CI, 0.6–2.4) the association between DDE and hypospadias. The modest association is felt to be inconclusive with the imprecision in risk estimates and suggests that a larger sample size may be needed. A third case-control study found inconclusive results on the effect of heptachlor and β-hexachlorocyclohexane levels in pregnant women on cryptorchidism. For heptachlor, the OR was 1.2 (95% CI, 0.6–2.6), and for β-hexachlorocyclohexane, the OR was 1.6 (95% CI, 0.7–3.6). The sample size in this study was 219 cases, compared with 564 controls.[167]

Two nested case-control studies have examined the possibility that multiple organochlorine compounds will have a cumulative effect on the development of urogenital abnormalities in boys.[168,169] Fernandez et al[168] reported that total xenoestrogens as well as detectable pesticide levels were associated with cryptorchidism and/or hypospadias. They found elevated ORs in the range of 2.19 for endosulfan to 3.38 for lindane. All 95% CIs were noted to be statistically significant. The study in Finland and Denmark reported a significant relationship between chlordane and cryptorchidism but no other relationships between 7 other individual organochlorines. However, combined analysis of the 8 persistent pesticides did demonstrate a statistically significant increase in cryptorchidism in exposed boys.[169]

Testing chemicals is an important and necessary step for the EPA to determine potential long-term risks from pesticide during the registration or re-registration process. There has been progress in the development of appropriate biomarkers to evaluate chemicals for the presence of endocrine-disruption qualities. The ability to measure DDE and dioxins from human milk has been developed.[170] More recently, a biomarker for xenoestrogen mixtures was developed in Spain.[171]

In summary, there is compelling basic science evidence for endocrine-mimicking effects of several pesticide chemicals that is sound and scientifically plausible. Human data

are slowly emerging but not yet conclusive.[172]

Asthma

Given the widespread use of pesticides and the high morbidity of asthma in children, questions have been raised regarding pesticides as triggers as well as risk factors for incident disease. Concern is raised by a mounting adult occupational literature associating pesticides with asthma or other measures of respiratory health. In addition, preliminary toxicological data provide mechanisms that link pesticides and asthma. An important limitation of most epidemiological studies to date is the lack of exposure specificity regarding pesticide chemicals or chemical classes. In addition, studies regarding children are few.

There is indirect evidence that pesticides skew the immune response toward the T helper 2 (Th2) phenotype associated with atopic disease. The National Institutes of Health/EPA-sponsored rural birth cohort described above regarding evaluation of neurodevelopmental effects has also observed that maternal agricultural work was associated with a 26% increase in proportion of Th2 cells in their 24-month-old infants' blood samples.[173] The percentage of Th2 cells was associated with both physician-diagnosed asthma and maternal report of wheeze in these infants. This population of largely Mexican American farmworkers was selected for study on the basis of the relatively high use of OP pesticides in this agricultural area.

Animal-based toxicological mechanistic models include OP-induced airway hyperreactivity via alteration in muscarinic receptor function in airway smooth muscle and oxidative stress induced by OP-related lipid peroxidation.[174–177]

The few epidemiological data on pesticides and respiratory health in children have mixed results. In a cohort of rural Iowan children, any pesticide use indoors or any outdoor use in the previous year was not significantly associated with asthma symptoms and prevalence.[178] Contrarily, a cross-sectional analysis of Lebanese children identified increased risk of chronic respiratory symptoms, including wheeze, among those with any pesticide exposure in the home, exposure related to parent's occupation, and use outside the home. The highest risk was observed for children whose parents had occupational exposure to pesticides (OR, 4.61; 95% CI, 2.06–10.29).[179] However, given this study's cross-sectional design, it is not possible to discern whether the pesticide exposure preceded the diagnosis of asthma.

Among exposures in the first year of life explored in a nested case-control study of the Southern California Children's Health Study, both herbicides and pesticides/insecticides had a strong association with asthma diagnosis before 5 years of age (OR, 4.58 [95% CI, 1.36–15.43] and OR, 2.39 [95% CI, 1.17–4.89], respectively).[180]

More published data are available regarding adult farmers and adult rural residents. These studies more consistently support a link between pesticides and respiratory symptoms or chronic respiratory disease, such as asthma.[181,182] For example, use of multiple individual pesticides was evaluated in relation to self-reported episodes of wheeze in the previous year in a large cohort of commercial pesticide applicators (adults) and farmers enrolled in the Agricultural Health Study.[182] Among the pesticides classes, several OPs showed associations with wheeze, including several that demonstrated a dose–response trend. Chlorpyrifos, malathion, and parathion were positively associated with wheeze among the farmers; for the commercial applicators, the OPs chlorpyrifos, dichlorvos, and phorate were positively associated with wheeze. Among commercial applicators, the strongest OR was for applying chlorpyrifos on more than 40 days per year (OR, 2.40; 95% CI, 1.24–4.65). Elevated risk for wheeze related to herbicide use was almost exclusively associated with chlorimuron-ethyl (urea-derivative class).

Similar studies addressing the respiratory health implications for children for specific pesticide chemical types or groups are rare. However, for DDT, there is some emerging evidence for a link between metabolites of DDT and asthma risk.[183,184] In a prospective cohort study of children in Spain, wheezing at 4 years of age increased with increasing levels of DDE at birth. The adjusted RR for the children with exposure in the highest quartile was 2.63 (95% CI, 1.19–4.69). The use of physician-diagnosed asthma (occurring in 1.9% of children) instead of wheezing as the outcome variable also resulted in a positive association, although it was not statistically significant.[184]

In summary, the available data regarding chronic exposure to pesticides and children's respiratory health remain limited. Studies that incorporate pesticide-specific exposure assessment and markers of biological mechanisms and consider the influence of timing of exposure across the life span are needed.

THE PESTICIDE LABEL

Pesticides for sale or use in the United States must be registered with the EPA, and this includes approval of the product label, which contains the EPA registration number. The pesticide label contains several types of information that may be important in understanding and preventing acute health consequences associated with their use.[185]

The product label identifies the active ingredient and provides the manufacturer's

contact information. The label does not specify the particular class of pesticide for the active ingredient, which may make it difficult for a physician to identify potential toxic effects. Information about "other" or "inert" ingredients, which may account for up to 99% of the product, is not required to be disclosed on the label. These constituents include chemicals with known toxicity. The physician treating a patient may request this from the manufacturer; however, delay in information may compromise optimal clinical care. The local or regional poison control center plays an important role as a resource for any suspected pesticide poisoning. The EPA is currently considering rulemaking changes that would expand the disclosure of information on inert ingredients. One of the options under consideration includes labeling 100% of the ingredients.[186]

The "directions for use" section on the label explains when, how, and where the pesticide may be applied. The label is considered the law; therefore, any use of the product in a manner inconsistent with the label is a violation of the Federal Insecticide, Fungicide, and Rodenticide Act (Pub L No. 80-104).[187] Information on recommended storage of the product and disposal of the container is also printed on the label.

The label will contain a signal word and symbol to identify acute toxicity potential: "danger" along with the word poison and the skull and crossbones symbol signifies high acute toxicity; "warning" signifies moderate acute toxicity; and "caution" represents slight acute toxicity. There is a section for precautionary statements regarding the potential hazards to people or pets and the actions that can be taken to reduce these hazards, such as wearing gloves or other protective equipment. Basic first aid advice for responding to dermal, inhalational, and/or oral exposure is provided. Some labels contain a "note for physicians" that includes specific medical information. The label does not provide any information or warnings about the potential for chronic toxicity arising from normal use or misuse of the pesticide. An example of an interactive pesticide label can be found at the EPA Web site.[188] It includes "pop-up" features that define each of the components on the pesticide label.

STATE OF PESTICIDE KNOWLEDGE AMONG PEDIATRICIANS

Self-reported medical education and self-efficacy suggests pediatricians are not well prepared to identify pesticide exposure and illness, including taking a relevant environmental history or discussing pesticide risks with their patients.[189–191] Even in agricultural areas of the Pacific Northwest, where pesticide use is heavy, a survey of health care providers who serve high volumes of agricultural farmworkers and their families found that 61% did not feel comfortable responding to patient/client questions regarding pesticides on the basis of their training, background, and experience.[75] Among academic pediatricians with an interest in pediatric environmental health, pesticides were among the topics they felt least prepared to teach to their trainees.[192] Given the widespread use of pesticides and concerns for child health, opportunities to increase pesticide competency in pediatric medical education are likely to prevent missed diagnoses and reduce exposure because of improved anticipatory guidance.

Clinicians must have a high index of suspicion to identify pesticide poisoning. Identification and treatment of acute pesticide poisoning requires familiarity with the toxic mechanisms and related signs and symptoms of the pesticide classes. For example, when evaluating a patient with status epilepticus or mental status changes, certain insecticides belong in the differential among the numerous and more common etiologies. Eliciting an environmental history will help decipher the relative importance of pesticides in further clinical decision-making. The environmental history is a general tool for addressing potentially hazardous environmental exposures and is discussed in detail in the Pediatric Environmental Health manual from the AAP.[193]

EFFORTS TO REDUCE PESTICIDE EXPOSURE

Dietary Considerations

Dietary modifications can help reduce pesticide exposure. As mentioned previously, consuming organic produce has shown a reduced amount of urinary pesticide levels in comparison with a conventional diet.[22] Because many food-based pesticide residues occur on the surface of food crops, other practical approaches may be used to reduce exposures by washing produce, peeling off outer layers of leafy vegetables, and removing peels from fruits and vegetables. Trimming fat from meat and fat and skin from poultry and fish may reduce residues of persistent pesticides, such as the organochlorines, that concentrate in animal fat.

Efforts to address and reduce chronic pesticide exposure via the food supply in children have included regulatory approaches that consider the unique vulnerability of the developing child in policy decision-making. For example, the 1996 Food Quality Protection Act (Pub L No. 104-170, Section 405) required that the EPA use an additional 10-fold margin of safety regarding limits of pesticide residues on food (unless there are data that show a less stringent residue level is safe for

prenatal and postnatal development; for description, see http://www.epa.gov/opp00001/factsheets/riskassess.htm).

Integrated Pest Management

In addition to food residues, use of pesticides in and around the home and other settings where children spend time (child care, school, and playgrounds and sports fields) is an important influence on the chronic and cumulative exposure to pesticides among US children. Most of the pest problems that occur indoors as well as control of lawn and garden pests can be addressed with least toxic approaches, including integrated pest management (IPM) techniques. IPM focuses on nontoxic and least toxic control methods to address pest problems have been promoted and adopted for residential, school, and agricultural settings (fact sheets available at http://www.epa.gov/opp00001/factsheets/ipm.htm).

"Integrated" refers to employment of complementary strategies of pest control, which may include mechanical devices; physical devices; genetic, biological, and cultural management; and chemical management. For example, to control cockroaches, a family could be counseled to keep garbage and trash in containers with well-fitted lids, eliminate plumbing leaks or other sources of moisture, store food in insect-proof containers, vacuum cracks and crevices, clean up spills immediately, and use the least-toxic insecticides, such as boric acid, in cracks and crevices or bait stations. The goal is to target the pest and limit the effect on other organisms and the environment. Although developed with a focus on agricultural insect pests, IPM programs and knowledge have extended to address weeds and pest control in residential settings and schools, commercial structures, lawn and turf, and community gardens.

Within agriculture, IPM has been recognized and promoted for decades; however, inadequate leadership, coordination, and management of US Department of Agriculture IPM programs were identified as impediments to adequate progress in a 2001 report.[194] The report provided the basis for an ongoing national roadmap effort to improve ongoing development of increased IPM in agriculture.

To protect children, IPM in schools has been recommended by the US Department of Agriculture, EPA, American Public Health Association, and National Parent Teacher Association. Many states and local municipalities have adopted programs and resources to encourage IPM in public places, in addition to homes and schools (see Table 3). IPM strategies seek to minimize insecticide use by applying strategies such as cleaning up food and water, sealing cracks and crevices, and using pesticides that are contained in baits or traps, which are far less likely to pose a health concern compared with any type of broadcast spray application. Avoiding combination products with pesticides and fertilizers (ie, "weed and feed" preparations) is advised for lawn maintenance, because these tend to result in overapplication of pesticides. Hand weeding is always a reasonable alternative to herbicides. However, if an herbicide is to be used, some (such as glyphosate) have better acute human toxicity profiles than others (such as 2,4-D). Even so, glyphosate is not without its risks. Most cases of moderate to severe toxicity have occurred after intentional (suicidal) ingestion.[195] Using safe storage practices (in a locked cabinet or building) and not reusing pesticide containers are important components toward the prevention of acute poisonings after unintentional ingestion by small children. Reliable resources for useful information on pest-control alternatives and safe use of pesticides are available from the EPA and University of California-Davis (Table 3).

Spraying in the Community: Right to Know

Although there is no federal mandate for notification of pesticide use in communities, many states, locales, or schools have implemented requirements for posting warning signs or developing registries to alert individuals of planned pesticide application (see Table 3). These are designed to allow the public to make decisions to avoid exposures during application or soon after from residues. Other local policies that have been developed include restricting spray zones that create buffers from schools or other areas or restrict specific types of pesticide products in schools. Pediatricians can play a role in the promotion of development of model programs and practices in the communities and schools of their patients. For example, in some communities, pediatricians have participated in local organizations that have successfully advocated for no pesticide application in schools.

SUMMARY

Pesticides are a complex group of chemicals with a wide range of acute and chronic toxicity. Poison control centers report lower rates of more severe poisonings but continue to report similar total numbers of acute exposures among children. There is a growing body of literature that suggests that pesticides may induce chronic health complications in children, including neurodevelopmental or behavioral problems, birth defects, asthma, and cancer. Pediatricians are a trusted source of information for families and communities, although current training focused on pesticide toxicity and environmental health, in

TABLE 3 Pesticide and Child Health Resources for the Pediatrician

Management of Acute Pesticide Poisoning		
Recognition and Management of Pesticide Poisonings	Print: fifth (1999) is available in Spanish, English (6th edition available 2013) http://www.epa.gov/pesticides/safety/healthcare/handbook/handbook.htm	
Regional Poison Control Centers	1-800-222-1222	
Chronic Exposure Information/Specialty Consultation		
The National Pesticide Medical Monitoring Program (NPMMP)	Cooperative agreement between Oregon State University and the EPA NPMMP provides informational assistance by e-mail in the assessment of human exposure to pesticides	npmmp@oregonstate.edu or by fax at 541-737-9047
Pediatric Environmental Health Specialty Units (PEHSUs)	Coordinated by the Association of Occupational and Environmental Clinics to provide regional academically based free consultation for health care providers	http://www.aoec.org/PEHSU.htm Toll-free telephone number 888-347-AOEC (2632)
Resources for Safer Approaches to Pest Control		
EPA	Consumer information documents	http://www.epa.gov/oppfead1/Publications/Cit_Guide/citguide.pdf
Citizens Guide to Pest Control and Pesticide Safety	• Household pest control • Alternatives to chemical pesticides • How to choose pesticides • How to use, store, and dispose of them safely • How to prevent pesticide poisoning • How to choose a pest-control company	
Controlling pests	Recommended safest approaches and examples of programs	http://www.epa.gov/pesticides/controlling/index.htm
The University of California Integrative Pest Management Program	Information on IPM approaches for common home and garden pests	http://www.ipm.ucdavis.edu
Other Resources		
National research programs addressing children's health and pesticides	NIEHS/EPA Centers for Children's Environmental Health & Disease Prevention Research	www.niehs.nih.gov/research/supported/centers/prevention
	The National Children's Study	www.nationalchildrensstudy.gov/Pages/default.aspx
EPA	Pesticide product labels	www.epa.gov/pesticides/regulating/labels/product-labels.htm#projects
The National Library of Medicine "Tox Town"	Section on pesticides that includes a comprehensive and well-organized list of Web link resources on pesticides	http://toxtown.nlm.nih.gov/text_version/chemicals.php?id=23

NIEHS, National Institute of Environmental Health Sciences.

general, is limited. Pediatricians should be familiar with the common pesticide types, signs and symptoms of acute toxicity, and chronic health implications. Efforts should be made to limit children's exposure as much as possible and to ensure that products released to the marketplace have been appropriately tested for safety to protect fetuses, infants, and children from adverse effects.

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ERRATA

Spooner. We Are Still Waiting for Fully Supportive Electronic Health Records in Pediatrics. *Pediatrics.* 2012;130(6):e1674–e1676.

An error occurred in this article by Spooner, titled "We Are Still Waiting for Fully Supportive Electronic Health Records in Pediatrics" published in the December 2012 issue of *Pediatrics* (2012;130[6]:e1674–e1676; originally published online November 19, 2012; doi:10.1542/peds.2012-2724). On page e1674, on line 33, this reads: "The alarming result from the survey was that only 3% of AAP Fellows reported that they had a system that provided all of the items listed by Leu and colleagues." This should have read: "The alarming result from the survey was that only 9.6% of AAP Fellows reported that they had or planned to adopt within 12 months a system that provided all of the five "pediatric-supportive" items listed by Leu and colleagues."

doi:10.1542/peds.2013-0134

Auger et al. Medical Home Quality and Readmission Risk for Children Hospitalized With Asthma Exacerbations. *Pediatrics.* 2013;131(1):64–70

An error occurred in this article by Auger et al, titled "Medical Home Quality and Readmission Risk for Children Hospitalized With Asthma Exacerbations" published in the January 2013 issue of *Pediatrics* (2013;131[1]:64–70; doi:10.1542/2012-1055). On page 69, in Table 2 under the heading Adjusted HR, on the line Medicaid, this reads: "0.28 (0.51–1.34)." This should have read: "0.82 (0.51–1.34)."

doi:10.1542/peds.2013-0187

Council on Environmental Health. Policy Statement: Pesticide Exposure in Children. *Pediatrics.* 2012;130(6):e1757–e1763

A couple of errors occurred in this AAP Policy Statement titled "Pesticide Exposure in Children" published in the December 2012 issue of *Pediatrics* (2012;130[6]:e1757–e1763; originally published online November 26, 2012; doi:10.1542/peds.2012-2757). In Table 2, in the second and third columns where glyphosate is discussed, the words "organic solvent" should be replaced with the word "surfactant." On page e1758, in the first paragraph of the left-hand column, immediately beneath Table 1, the first full sentence should be amended to read: "For many children, diet may be the most influential source, as illustrated by an intervention study that placed children on an organic diet (produced without most conventional pesticides) and observed drastic and immediate decrease in urinary excretion of organophosphate pesticide metabolites."

doi:10.1542/peds.2013-0576

Robert JR, Karr CJ; Council on Environmental Health. Technical Report: Pesticide Exposure in Children. *Pediatrics.* 2012;130(6):e1765–e1788

Several inaccuracies occurred in this AAP Technical Report titled "Pesticide Exposure in Children" published in the December 2012 issue of *Pediatrics* (2012;130[6]:e1765–e1788; originally published online November 26, 2012; doi:10.1542/peds.2012-2758). On page e1773 and in Tables 1 and 2 where the phosphonate herbicide glyphosate is discussed, changes should be noted. In the first paragraph of the first column on page e1773 about acute glyphosate poisoning, the word "intentional" should be substituted for the word "unintentional." In this same paragraph as well as in Tables 1 and 2, the word "surfactant" should replace the words "hydrocarbon solvent" and "organic solvent, respectively." The

mechanism of action for glyphosate should be changed from "acts on cell wall" to "inhibits a critical enzyme pathway for amino acid synthesis that is found only in plants" (Bradberry SM, Proudfoot AT, Vale JA. Glyphosate poisoning. *Toxicol Rev.* 2004;23[3]:159–167).

doi:10.1542/peds.2013-0577

Copeland et al. Clinical Practice Guideline: Management of Newly Diagnosed Type 2 Diabetes Mellitus (T2DM) in Children and Adolescents. *Pediatrics.* 2013;131(2):364–382

Several inaccuracies occurred in the American Academy of Pediatrics "Clinical Practice Guideline: Management of Newly Diagnosed Type 2 Diabetes Mellitus (T2DM) in Children and Adolescents" published in the February 2013 issue of *Pediatrics* (2013;131[2]:364–382).

On page 366 in the table of definitions, "Prediabetes" should be defined as "Fasting plasma glucose ≥100–125 mg/dL or 2-hour glucose concentration during an oral glucose tolerance test of ≥140 but <200 mg/dL or an HbA1c of 5.7% to 6.4%."

On page 378, middle column, under "Reducing Screen Time," the second sentence should read as follows: "The US Department of Health and Human Services reflects the American Academy of Pediatrics policies by recommending that individuals limit "screen time" spent watching television and/or using computers and handheld devices to <2 hours per day unless the use is related to work or homework."[79–81,83]

Also on page 378, middle column, in the second paragraph under "Reducing Screen Time," the fourth sentence should read: "Pending new data, the committee suggests that clinicians follow the policy statement 'Children, Adolescents, and Television' from the AAP Council on Communications and Media (formerly the Committee on Public Education)." The references cited in the next sentence should be 80–83.

Reference 82 should be replaced with the following reference: Barlow SE; Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics.* 2007;120(suppl 4):S164–S192

Finally, a new reference 83 should be added: American Academy of Pediatrics, Council on Communications and Media. Policy statement: children, adolescents, obesity, and the media. *Pediatrics.* 2011;128(1):201–208

doi:10.1542/peds.2013-0666

Springer et al. Technical Report: Management of Type 2 Diabetes Mellitus in Children and Adolescents. *Pediatrics.* 2013;131(2):e648–e664.

An error occurred in the American Academy of Pediatrics "Technical Report: Management of Type 2 Diabetes Mellitus in Children and Adolescents" published in the February 2013 issue of *Pediatrics* (2013;131[2]:e648–e664).

On page e651, third column, under "Definitions," the first sentence should read as follows: "Children and adolescents: children <10 years of age; adolescents ≥10 years but ≤18 years of age."

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Pesticide Exposure in Children

James R. Roberts, Catherine J. Karr and COUNCIL ON ENVIRONMENTAL HEALTH

Pediatrics; originally published online November 26, 2012;
DOI: 10.1542/peds.2012-2758

Peer-Reviewed Studies & Relevant Reports on Pesticides and Human & Environmental Health

1. The American Academy of Pediatrics Position on Pesticides, November 26, 2012, ***"Policy Statement - Pesticide Exposure in Children"*** http://pediatrics.aappublications.org/content/130/6/e1757.full.pdf

SUMMARY: Increasing evidence shows urban and rural children are regularly exposed to low levels of pesticides that can have serious long-term health effects, according to a report issued by the American Academy of Pediatrics.

2. The American College of Obstetricians and Gynecologists, Committee Opinion, Number 575, October 2013, ***"Exposure to Toxic Environmental Agents"***
http://www.acog.org/Resources_And_Publications/Committee_Opinions/Committee_on_Health_Care_for_Underserved_Women/Exposure_to_Toxic_Environmental_Agents

SUMMARY: Reducing exposure to toxic environmental agents is a critical area of intervention for obstetricians, gynecologists, and other reproductive health care professionals. Patient exposure to toxic environmental chemicals and other stressors is ubiquitous, and preconception and prenatal exposure to toxic environmental agents can have a profound and lasting effect on reproductive health across the life course. Prenatal exposure to certain chemicals has been documented to increase the risk of cancer in childhood; adult male exposure to pesticides is linked to altered semen quality, sterility, and prostate cancer; and postnatal exposure to some pesticides can interfere with all developmental stages of reproductive function in adult females, including puberty, menstruation and ovulation, fertility and fecundity, and menopause. Many environmental factors harmful to reproductive health disproportionately affect vulnerable and underserved populations, which leaves some populations, including underserved women, more vulnerable to adverse reproductive health effects than other populations. The evidence that links exposure to toxic environmental agents and adverse reproductive and developmental health outcomes is sufficiently robust, and the American College of Obstetricians and Gynecologists and the American Society for Reproductive Medicine join leading scientists and other clinical practitioners in calling for timely action to identify and reduce exposure to toxic environmental agents while addressing the consequences of such exposure. **[NOTE: See Page 2, Pesticides are known Endocrine Disruptors]**

3. ***"Prenatal pesticide exposure linked to attention problems in preschool-aged children"*** Council on Environmental Health, University of California, Berkeley's School of Public Health, August 19, 2013 published in *Environmental Health Perspectives* (EHP), http://newscenter.berkeley.edu/2010/08/19/pesticide/

SUMMARY: The new findings, to be published Aug. 19, 2010 in the journal Environmental Health Perspectives (EHP), are the first to examine the influence of prenatal organophosphate exposure on the later development of attention problems. The researchers found that prenatal levels of organophosphate metabolites were significantly linked to attention problems at age 5, with the effects apparently stronger among boys.

4. Mount Sinai School of Medicine in New York, ***"Environmental Illness in U.S. Kids Cost $76.6 Billion in One Year"***, Published in the May 2011 issue of the journal "Health Affairs" http://www.ens-newswire.com/ens/may2011/2011-05-04-02.html

SUMMARY: It cost a "staggering" $76.6 billion to cover the health expenses of American children who were sick because of exposure to toxic chemicals and air pollutants in 2008, according to new research by senior scientists at the Mount Sinai School of Medicine in New York. Published in the May issue of the journal "Health Affairs," three new studies by Mount Sinai scientists reveal the economic impact of toxic chemicals and air pollutants in the environment

5. ***"Potential Health Effects Related to Pesticide Use on Athletic Fields,"***
Robyn Gilden, Ph.D.,1 Erika Friedmann, Ph.D.,2 Barbara Sattler, Dr.P.H., R.N., F.A.A.N.,1,3, Katherine Squibb, Ph.D.,4 and Kathleen McPhaul, Ph.D., M.P.H., B.S.N., R.N.1 1Family and Community Health, University of Maryland School of Nursing, Baltimore, Maryland; 2Organizational Systems and Adult Health, University of Maryland School of Nursing, Baltimore, Maryland; 3Environmental Health Education Center, University of Maryland School of Nursing, Baltimore, Maryland; and 4University of Maryland School of Medicine, Baltimore, Maryland

SUMMARY: Children come in contact with athletic fields on a daily basis. How these fields are maintained may have an impact on children's potential exposure to pesticides and associated health effects. Design and Sample: This is a cross-sectional, descriptive study that utilized a survey to assess playing field maintenance practices regarding the use of pesticides. Athletic fields (N = 101) in Maryland were stratified by population density and randomly selected. Measures: A survey was administered to field managers (n = 33) to assess maintenance practices, including the use of pesticides. Analysis included descriptive statistics and generalized estimating equations. Results: Managers of 66 fields (65.3%) reported applying pesticides, mainly herbicides (57.4%). Managers of urban and suburban fields were less likely to apply pesticides than managers of rural fields. Combined cultivation practice was also a significant predictor of increased pesticide use.
Conclusions: The use of pesticides on athletic fields presents many possible health hazards. Results indicate that there is a significant risk of exposure to pesticide for children engaged in sports activities. Given that children are also often concurrently exposed to pesticides as food residues and from home pest management, we need to examine opportunities to reduce their exposures. Both policy and practice questions are raised.

6. ***"A case-control study of childhood brain tumors and fathers' hobbies: a Children's Oncology Group study,"*** Rosso AL[1], Hovinga ME, Rorke-Adams LB, Spector LG, Bunin GR; Children's Oncology Group. Cancer Causes Control.2008 Dec;19(10):1201-7

SUMMARY: A comprehensive case-control study was conducted to evaluate parental risk factors for medulloblastoma (MB) and primitive neuroectodermal tumor (PNET). This analysis was conducted to evaluate associations between fathers' hobbies and risk of their children developing MB/PNET. The hobbies chosen for study were those with similar exposures as occupations associated with childhood cancers. This study suggests that household exposures from hobbies, particularly lawn care pesticides, may increase risk of MB/PNET in children; previous research has been mostly limited to occupational exposures.

7. ***"NIEHS-funded scientists say more chemicals linked to neurodevelopmental disorders,"***

SUMMARY: Based on a review of current published research, scientists funded by NIEHS have identified several additional industrial chemicals documented in scientific literature as toxic to brain development and the human nervous system. In a new study (http://www.ncbi.nlm.nih.gov/pubmed/24556010) published in the journal Lancet Neurology, the authors suggested that compounds including metals, solvents, and pesticides may be partially responsible for the increased prevalence of neurodevelopmental disorders in children. Neurodevelopmental disorders include autism, attention-deficit hyperactivity disorder, dyslexia, and other cognitive impairments.

[Glyphosate is identified as toxic to the human nervous system**]**

8. ***"Pesticide exposure as risk factor for nonHodgkin lymphoma including histopathological subgroup analysis,"***
Eriksson M , Hardell L, Carlberg M, Akerman M., Department of Oncology, University Hospital, Lund, Sweden, Int J Cancer. 2008 Oct 1;123(7):165763

SUMMARY: We report a population based case control study of exposure to pesticides as risk factor for nonHodgkin lymphoma (NHL). Male and female subjects aged 18-74 years living in Sweden were included during December 1, 1999, to April 30, 2002. Controls were selected from the national population registry. Exposure to different agents was assessed by questionnaire. In total 910 (91 %) cases and 1016 (92%) controls participated. Exposure to herbicides gave odds ratio (OR) 1.72, 95% confidence interval (CI) 1.182.51. Regarding phenoxyacetic acids highest risk was calculated for MCPA; OR 2.81, 95% CI 1.276.22, all these cases had a latency period >10 years. Exposure to glyphosate gave OR 2.02, 95% CI 1.103.71 and with >10 years latency period OR 2.26, 95% CI 1.164.40. Insecticides overall gave OR 1.28, 95% CI 0.961.72 and impregnating agents OR 1.57, 95% CI 1.072.30. Results are also presented for different entities of NHL. In conclusion our study confirmed an association between exposure to phenoxyacetic acids and NHL and the association with glyphosate was considerably strengthened.

[Note: The herbicide 2,4-D is a phenoxyacetic acid**]**

9. College of Family Physicians of Ontario, "***Pesticides and Human Health Why Public Health Officials Should Support a Ban on Non-essential Residential Use***" Canadian Journal of Public Health, march-April 2005.
http://www.neilarya.com/wp-content/uploads/2012/01/AryaCJPHWhyHealthProfessionalsshouldsupportaPesticideBan.pdf or
http://www.national-toxic-encephalopathy-foundation.org/humanhealth.pdf

SUMMARY: The final conclusion, i.e., that exposure to all commonly used pesticides has shown positive association with adverse health effects, made headlines throughout North America. The College of Family Physicians of Ontario recently released a comprehensive report on pesticide exposure and health risk, concluding that various pesticides had adverse health effects. The pesticide industry says that pesticides are "safe" when used as directed because they are studied and approved by governmental agencies. Yet many municipalities, including Canada's three largest, and the province of Quebec have enacted bans on cosmetic use of pesticides, largely in response to health concerns. Reviewing the report, the status of regulation of pesticides and the limitations of studies and of regulation in Canada, it appears that on the basis of evidence available to date, public health officials should support a ban on cosmetic use of pesticides.

10. ***"Distribution of 2,4-D in Air and on Surfaces inside Residences after Lawn Applications: Comparing Exposure Estimates from Various Media for Young Children,"*** *Environmental Health Perspectives,* 109:1185–1191 (2001), *Marcia G. Nishioka, Robert G. Lewis, Marielle C. Brinkman, Hazel M. Burkholder, Charles E. Hines,and John R. Menkedick*

SUMMARY: We collected indoor air, surface wipes (floors, table tops, and window sills), and floor dust samples at multiple locations within 11 occupied and two unoccupied homes both before and after lawn application of the herbicide 2,4-D. We measured residues 1 week before and after application. We used collected samples to determine transport routes of 2,4-D from the lawn into the homes, its subsequent distribution between the indoor surfaces, and air concentration as a function of airborne particle size. We used residue measurements to estimate potential exposures within these homes. After lawn application, 2,4-D was detected in indoor air and on all surfaces throughout all homes. Track-in by an active dog and by the homeowner applicator were the most significant factors for intrusion. Resuspension of floor dust was the major source of 2,4-D in indoor air, with highest levels of 2,4-D found in the particle size range of 2.5-10 microm. Resuspended floor dust was also a major source of 2,4-D on tables and window sills. Estimated postapplication indoor exposure levels for young children from nondietary ingestion may be 1-10 microg/day from contact with floors, and 0.2-30 microg/day from contact with table tops. These are estimated to be about 10 times higher than the preapplication exposures. By comparison, dietary ingestion of 2,4-D is approximately 1.3 microg/day . **** [TRACK-IN and DRIFT of 2,4D]****

11. ***"PESTICIDES AND CHILDHOOD CANCER: AN UPDATE OF ZAHM AND WARD'S 1998 REVIEW",*** Department of Epidemiology, Biostatistics, and Occupational Health, Faculty of Medicine, McGill University, Montréal, Québec, Canada, *Journal of Toxicology and Environmental Health, Part B*, 10:81–99, [updated **2007**].

SUMMARY: Children are exposed to pesticides through a number of sources, including residential and agricultural applications. Parental occupational exposure to pesticides is also a concern because exposures occurring during pregnancy and carry-home residues also contribute to children's cumulative burden. A number of epidemiological studies consistently reported increased risks between pesticide exposures and childhood leukemia, brain cancer, neuroblastoma, non-Hodgkin's lymphoma, Wilms' tumor, and Ewing's sarcoma. An extensive review of these studies was published in 1998 (Zahm & Ward, 1998). Fifteen case-control studies, 4 cohort studies, and 2 ecological studies have been published since this review, and 15 of these 21 studies reported statistically significant increased risks between either childhood pesticide exposure or parental occupational exposure and childhood cancer. Therefore, one can confidently state that there is at least some association between pesticide exposure and childhood cancer.

12. ***"Glyphosate induces human breast cancer cells growth via estrogen receptors."*** Thongprakaisang S , Thiantanawat A, Rangkadilok N, Suriyo T, Satayavivad J., *Food Chem Toxicol.* 2013 Sep;59:12936.

SUMMARY: Glyphosate is an active ingredient of the most widely used herbicide and it is believed to be less toxic than other pesticides. However, several recent studies showed its potential adverse health effects to humans as it may be an endocrine disruptor. This study focuses on the effects of pure glyphosate on estrogen receptors (ERs) mediated transcriptional activity and their expressions. Glyphosate exerted proliferative effects only in human hormonedependent breast cancer, T47Dcells, but not in hormoneindependent breast cancer, MDAMB231 cells, at 10^{-12} to 10^{-6}M in estrogen withdrawal condition. The proliferative concentrations of glyphosate that induced the activation of estrogen response element (ERE) transcription activity were 513 fold of control inT47DKBluc cells and this activation was inhibited by an estrogen antagonist, ICI 182780, indicating that the estrogenic activity of glyphosate was mediated via ERs. Furthermore, glyphosate also altered both ERα and β expression. These results indicated that low and environmentally relevant concentrations of glyphosate possessed estrogenic activity. Glyphosate-based herbicides are widely used for soybean cultivation, and our results also found that there was an additive estrogenic effect between glyphosate and genistein, a phytoestrogen in soybeans.

13. ***"Reported Residential Pesticide Use and Breast Cancer Risk on Long Island, New York,"*** *American Journal of Epidemiology* Vol. 165, No. 6, Dec. 2006, Susan L. Teitelbaum1, Marilie D. Gammon2, Julie A. Britton1, Alfred I. Neugut3,4, Bruce Levin5, and Steven D. Stellman3 1 Department of Community Medicine, Mount Sinai School of Medicine, New York, NY. 2 Department of Epidemiology, School of Public Health, University of North Carolina, Chapel Hill, NC. Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, NY. 4 Department of Medicine, Columbia College of Physicians and Surgeons, New York, NY. 5 Department of Biostatistics, Mailman School of Public Health, Columbia University, New York, NY.

SUMMARY: Pesticides, common environmental exposures, have been examined in relation to breast cancer primarily in occupational studies or exposure biomarker studies. No known studies have focused on self-reported residential pesticide use. The authors investigated the association between reported lifetime residential pesticide use and breast cancer risk among women living on Long Island, New York. They conducted a population-based casecontrol study of 1,508 women newly diagnosed with breast cancer between August 1996 and July 1997 and 1,556 randomly selected, age-frequency-matched controls. Comprehensive residential pesticide use and other risk factors were assessed by using an in-person, interviewer-administered questionnaire. Unconditional logistic regression was used to calculate odds ratios and 95% confidence intervals. Breast cancer risk was associated with ever lifetime residential pesticide use (odds ratio ¼ 1.39, 95% confidence interval: 1.15, 1.68). However, there was no evidence of increasing risk with increasing lifetime applications. Lawn and garden pesticide use was associated with breast cancer risk, but there was no dose response. Little or no association was found for nuisance-pest pesticides, insect repellants, or products to control lice or fleas and ticks on pets. This study is the first known to suggest that self-reported use of residential pesticides may increase breast cancer risk.

14. ***"Household chemical exposures and the risk of canine malignant lymphoma, a model for human non-Hodgkin's lymphoma"***, Takashima-Uebelhoer BB[1], Barber LG, Zagarins SE, Procter-Gray E, Gollenberg AL, Moore AS, Bertone-Johnson ER, *Environmental Research* 112 (2012) 171–176.

SUMMARY: Epidemiologic studies of companion animals offer an important opportunity to identify risk factors for cancers in animals and humans. Canine malignant lymphoma (CML)has been established as a model for non-Hodgkin's lymphoma(NHL).Previous studies have suggested that exposure to environmental chemicals may relate to development of CML. Methods: We assessed the relation of exposure to flea and tick control products and lawn-care products and risk of CML in a case–control study of dogs presented to a tertiary-care veterinary hospital (2000–2006).Cases were 263 dogs with biopsy-confirmed CML. Controls included 240 dogs with benign tumors and 230 dogs undergoing surgeries unrelated to cancer. Dog owners completed a 10-page questionnaire measuring demographic, environmental, and medical factors. Results: After adjustment for age, weight, and other factors, use of specific lawn care products was associated with greater risk of CML. Specifically, **the use of professionally applied pesticides was associated with a significant 70% higher risk of CML** (odds ratio (OR) ¼1.7; 95% confidence interval (CI)¼1.1–2.7).Risk was also higher in those reporting use of self-applied insect growth regulators (OR¼2.7; 95%CI¼1.1–6.8).The use of flea and tick control products was unrelated to risk of CML. **Conclusions: Results suggest that use of some lawn care chemicals may increase the risk of CML. Additional analyses are needed to evaluate whether specific chemicals in these products may be related to risk of CML, and perhaps to human NHL as well.**

15. ***"Does "the Dose Make the Poison?" Extensive results challenge a core assumption in Toxicology,"*** Pete Myers, Ph.D. and Wendy Hessler, *Environmental Health News*, April 2007

SUMMARY: Because all regulatory testing has been designed assuming that "the dose makes the poison," it is highly likely to have missed low dose effects, and led to health standards that are too weak.

16. ***"Early-life Exposure to Organophosphate Pesticides and Pediatric Respiratory Symptoms in the CHAMACOS Cohort"*** *Rachel Raanan,1 Kim G. Harley,1 John R. Balmes,2,3 Asa Bradman,1 Michael Lipsett,4 and Brenda Eskenazi1* 1Center for Environmental Research and Children's Health (CERCH), School of Public Health, University of California, Berkeley, California, USA; 2Division of Environmental Health Sciences, School of Public Health, University of Califomia, Berkeley, Berkeley, California, USA; 3Divison of Occupational and Environmental Medicine, University of California, *Environmental Health Perspectives* volume 123:2, February 2015.

SUMMARY: Early-life exposure to OP pesticides was associated with respiratory symptoms consistent with possible asthma in childhood.

17. New York Times, ***"The Year the Monarch Didn't Appear"*** http://www.nytimes.com/2013/11/24/sunday-review/the-year-the-monarch-didnt-appear.html?_r=0

SUMMARY: November, 2013 - This year, for or the first time in memory, the monarch butterflies didn't come, at least not on the Day of the Dead. It is only the latest bad news about the dramatic decline of insect populations.

18. USGS Study, ***"Pesticides in the Nation's Streams and Ground Water, 1992–2001—A Summary"*** http://pubs.usgs.gov/fs/2006/3028/pdf/fs2006-3028.pdf

SUMMARY: The findings show that streams are most vulnerable to pesticide contamination, but ground water also merits careful monitoring—especially in agricultural and urban areas. Shallow ground water in some of these areas is used for drinking water and ground-water contamination is difficult to reverse once it occurs.

19. ***"Male Fish With Female Organs Packed Full of Pesticides Found in Potomac"*** Jan 19, 2007, http://www.foxnews.com/story/2007/01/19/male-fish-with-female-organs-packed-full-pesticides-found-in-potomac/

SUMMARY: Several chemicals, including one banned in the U.S., have been found in the Potomac River and its tributaries where pollution is suspected of causing some species of male fish to develop female sexual traits.

20. ***American Rivers Names Potomac River as Most Endangered*** , May 2012 http://www.americanrivers.org/assets/pdfs/mer-2012/2012-compiled.pdf

SUMMARY: If Congress puts polluters before people, our nation's river— and many other rivers nationwide— will become a threat to public health, unsafe for drinking water, wildlife, or recreation.

21. EPA, ***"America's Children and the Environment"*** Third Edition, January 2013 http://www.epa.gov/envirohealth/children/pdfs/ACE3_2013.pdf

SUMMARY: Pg 58 – On pesticides; Page 59 – pollutants come from outside; Pg 74 – lawn pesticides & runoff & RoundUp; Pg 223 – environmental links to childhood cancer; Pg 224 –Childhood leukemia & pesticides; Pgs 292-294 – Pesticides in Schools & Child Care Facilities

22. ***Glyphosate's Suppression of Cytochrome P450 Enzymes and Amino Acid Biosynthesis by the Gut Microbiome: Pathways to Modern Diseases***, http://www.mdpi.com/1099-4300/15/4/1416

SUMMARY: Glyphosate, the active ingredient in Roundup®, is the most popular herbicide used worldwide. The available evidence shows that Roundup® may rather be the most important factor in the development of multiple chronic diseases and conditions that have become prevalent in Westernized societies. In addition to autism, these include gastrointestinal issues such as inflammatory bowel disease, chronic diarrhea, colitis and Crohn's disease, obesity, cardiovascular disease, depression, cancer, cachexia, Alzheimer's disease, Parkinson's disease, multiple sclerosis, and ALS, among others.

23. ***"President's Cancer Panel Issues Sharp Pesticide Warnings"***, May 6, 2010, Safe Lawns.org http://www.safelawns.org/blog/2010/05/presidents-cancer-panel-issues-sharp-pesticide-warnings/

SUMMARY: The entire U.S. population is exposed on a daily basis to numerous agricultural chemicals, some of which are also used in residential and commercial landscaping. Many of these chemicals have known or suspected carcinogenic or endocrine-disrupting properties. Pesticides (insecticides, herbicides and fungicides) approved for used by the U.S. Environmental Protection Agency (EPA) contain nearly 900 active ingredients, many of which are toxic."

24. National Resources Defense Council (NRDC), **"Health Hazards of Pesticides"**, October 1988
http://www.nrdc.org/health/kids/farm/chap1.asp

SUMMARY: Most pesticides used today are acutely toxic to humans. Pesticides cause poisonings and deaths every year... Chronic health effects have also been reported from pesticides, including neurological effects, reproductive problems, interference with infant development, and cancer.

25. ***"What to Know before you Spray Your Lawn with Pesticides,"*** *Washington Post*, July 7, 2014,
http://www.washingtonpost.com/national/health-science/what-to-know-before-you-spray-your-lawn-with-pesticides/2014/07/07/77d719a2-f63c-11e3-a606-946fd632f9f1_story.html

26. **Further Reading on Neonicotinoids:**

a. ***"Second Silent Spring? Bird Declines Linked to Popular Pesticides,"***
http://news.nationalgeographic.com/news/2014/07/140709-birds-insects-pesticides-insecticides-neonicotinoids-silent-spring/
b. ***"Decline of wild bird population linked to use of neonicotinoid pesticides,"***
http://www.techtimes.com/articles/10105/20140710/decline-of-wild-bird-population-linked-to-use-of-neonicotinoid-pesticides-study.htm
c. ***"Neonicotinoid pesticides are bad news for everything,"*** http://www.newscientist.com/article/dn25783-neonicotinoid-pesticides-are-bad-news-for-everything.html#.VPUATkK4mRs
d. ***"Effects of neonicotinoids and fipronil on non-target invertebrates,"*** http://link.springer.com/article/10.1007%2Fs11356-014-3471-x

27. ***Busting the Myths that Pesticides are Needed for Lawn Care***
Mike McGrath, Home & Garden, WTOP, http://wtop.com/garden-plot-living/2015/01/hold-gp-hold/

Icahn
School of
Medicine at
Mount
Sinai

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January 7, 2015

Re: Bill 52-14

Dear Montgomery County Council Members:

Thank you for the opportunity to submit testimony in support of Safe Grow Montgomery's campaign to eliminate cosmetic lawn pesticides in Montgomery County.

I am a pediatrician, epidemiologist and Dean for Global Health in the Icahn School of Medicine at Mount Sinai. I am also Professor and Chairman of the Department of Preventive Medicine, Professor of Pediatrics and Director of Mount Sinai's Children's Environmental Health Center, a designated World Health Organization Collaborating Centre in Children's Environmental Health.

For many years beginning in the early 1970s at the Centers for Disease Control and Prevention (the CDC), I have conducted research in public health, and I have published this research extensively in leading peer-reviewed journals including *The New England Journal of Medicine*, *The Lancet* and *Environmental Health Perspectives*. My research has focused on understanding the impacts on children's health of exposures to toxic chemicals. I have recently edited the first ever *Textbook in Children's Environmental Health*, a volume of 700 pages and 60 chapters, authored by 85 scientists from five continents and published by Oxford University Press. My biographical sketch is attached to this testimony.

Children are uniquely vulnerable to the health effects of pesticide exposure.
Application of pesticides for cosmetic purposes results in human exposure through contact with grass, soil, and other surfaces. Additional exposure can result from drift from spray applications. Pesticide exposures can have toxic effects on health.

Children are especially vulnerable to pesticides, because their age-appropriate hand-to-mouth behaviors, their closer proximity to the ground, and their higher breathing rates place young children at increased risk for pesticide exposures compared with adults[1]. The Centers for Disease Control and Prevention has found that children age 6-11 have higher levels of common pesticides in their bodies, indicating higher exposure[2]. Furthermore, some pesticides can pass from mother to fetus during pregnancy and breastfeeding. These are very troubling findings due to the exquisite vulnerability of the fetus and early neonate to toxic exposures[3, 4].

Children's vulnerability to chemical pesticides is further magnified by the rapid growth and development of their nervous systems and other bodily organs as well as by their immature detoxification mechanisms, which make it very difficult for infant to break down and excrete pesticides after they have been exposed. These factors place infants and children at increased risk for harmful effects of pesticide exposures, which may be permanent and irreversible[5]. Additionally, because of their young age, children have more future years of life and therefore more time to develop chronic diseases that may be triggered by environmental exposures in early life.

Health Effects of Pesticide Exposure. Acute exposure to pesticides can lead to asthma exacerbations, cough, shortness of breath, nausea, vomiting, eye irritation, and headaches[6]. Additionally, pesticide exposure early in life is associated with increased risk of certain cancers[7-9], birth defects[10, 11], reproductive defects[12, 13], asthma[14, 15], and cognitive and behavioral problems[16-20].

The association between pesticide exposure and impaired neurodevelopment in children is not surprising. Pesticides are deliberately designed to be toxic chemicals. A large number of pesticides have been deliberately engineered to attack cellular targets in the nervous systems of insects. Given that many of these same cellular targets are present in the human nervous system, children are highly vulnerable. For example, children with prenatal exposure to the organophosphate pesticide chlorpyrifos show decreased intelligence, smaller head circumference at birth, which is a marker for retarded brain growth, and changes in the brain that are evident on MRI, indicating that changes in brain structure have occurred[21]. Notably, the exposure levels measured in these studies are similar to those detected in the general public, indicating that even low levels of exposure from household use can be detrimental.

Early life exposures to commonly used lawn and garden pesticides such as glyphosate, 2,4-D, and permethrin, are associated with cancer[22], neurotoxicity[23], and endocrine disruption[24,25].

Finally, greater than 95% of most pesticide formulations consist of "inert" ingredients. Recent studies suggest that these "inactive" compounds may in fact be more toxic than the active ingredient[26, 27]. Because inert ingredients are not listed on the label and testing to assess safety is minimal, the health effects of these compounds are difficult to evaluate[28].

Preventing the Health Hazards of Pesticide Exposure. The adverse health effects that result from pesticide exposures are highly preventable. A ban on the cosmetic use of pesticides in Montgomery County will have positive effects on a wide array of health outcomes.

Historically, policy changes in pesticide regulation have successfully reduced exposures among the population. For example, after the EPA ban on residential uses of chlorpyrifos, there was a ten-fold reduction in maternal and umbilical blood levels of chlorpyrifos[29].

Several U.S. states and municipalities have banned cosmetic application of lawn pesticides in public areas that are utilized by children. The ban on cosmetic herbicides across nearly 80% of Canada has contributed to significant reductions in their use without negatively affecting the lawn care industry[30]. Levels of the three most common pesticide chemicals dropped by 80% in urban streams in Ontario following the ban[31].

A 2005 analysis calculated that pesticide use in the U.S. results in $10 billion in total damages annually, of which an estimated $1.1 billion could be accounted for by impacts on public health[32]. These indirect costs greatly outweigh the expense of integrated pest management and other non-toxic lawn care methods.

Conclusion Children are at risk for pesticide exposures at daycares, schools, on playing fields, playgrounds, and other public areas where lawn pesticides are routinely applied—a risk that could easily be reduced by legislation that would restrict the use of synthetic lawn pesticides in Montgomery County. I urge you to take steps to protect the health of your constituents by supporting a ban on the cosmetic use of pesticides.

Thank you for your consideration.

Sincerely,

Philip J. Landrigan, MD, MSc

Attachment

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BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors in the order listed on Form Page 2.
Follow this format for each person. **DO NOT EXCEED FOUR PAGES.**

NAME	POSITION TITLE
Philip J. Landrigan	Ethel H. Wise Professor of Community Medicine Chairman, Department of Preventive Medicine Professor of Pediatrics Dean for Global Health
eRA COMMONS USER NAME (credential, e.g., agency login) PJLANDRIGAN	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
Boston College	A.B. (magna cum laude)	1963	Biology
Harvard Medical School	M.D.	1967	Medicine
Cleveland Metropolitan General Hospital	Internship	1967-68	Medicine/Pediatrics
Children's Hospital Medical Center, Boston	Residency	1968-70	Pediatrics
London School of Hygiene & Tropical Medicine University of London	DIH	1977	Industrial Health
	M.Sc. (with distinction)	1977	Occupational Medicine

A. Personal Statement

I am a pediatrician, epidemiologist and Dean for Global Health in the Icahn School of Medicine at Mount Sinai. I am also Professor and Chairman of the Department of Preventive Medicine, and Professor of Pediatrics. For over 30 years, my research has focused on understanding the impacts on children's health of exposures to toxic chemicals. Beginning with studies that I led at CDC in the 1970s of subclinical neurotoxicity in children exposed to lead near smelters, I have explored the health and developmental consequences of early-life exposures. I have been especially involved in studies of metals and pesticides.

I have extensive experience in building and leading transdisciplinary environmental health research programs. At CDC in the 1970s, I was the first director of the environmental epidemiology unit that grew subsequently into CDC's National Center for Environmental Health. At NIOSH in the 1980s, I led the national program in occupational epidemiology. In 1997-98 I served as Senior Scientific Advisor to the Administrator of US EPA and helped to create EPA's Office of Children's Health Protection. I was an architect of the National Children's Study (NCS), and from 2003-2005 I served on the NCS Federal Advisory Committee, and then was PI of the Queens (NY) NCS Vanguard Center. Over the past two decades, I have led development of the environmental health research and training programs at Mount Sinai that form the base for this application.

I am deeply committed to translating the results of environmental health research into evidence-based policy to protect public health. I have testified repeatedly before the US Congress and state legislatures. I was centrally involved in EPA's 1976 decision to remove lead from gasoline, an action that led to a more than 90% reduction in incidence of childhood lead poisoning in the US and to elevation of population mean IQ. I have chaired two National Academy of Sciences committees that were instrumental in translating scientific knowledge into public policy – the Committee on Neurotoxicology and Risk Assessment (1987-99) and the Committee on Pesticides in the Diets of Infants and Children (1988-93). The Pesticides report provided the blueprint for the Food Quality Protection Act of 1996, the federal law on pesticides and the only federal environmental statute in the US that contains explicit provisions to protect the health of children.

I am committed to training the next generation of leaders in environmental health science. From 2002-2007, I directed a post-residency training program in children's environmental health for pediatricians, which was supported at Mount Sinai by the Academic Pediatric Association. From 2007-2013, I served as PI of an interdisciplinary research training fellowship supported by NICHD (T32 HD049311) that is educating pediatricians and PhD-trained scientists to become independent researchers and future leaders in EHS.

I have served as Editor-in-Chief of the *American Journal of Industrial Medicine* and as Editor of *Environmental Research*. Since 2002, I have been an Associate Editor on children's environmental health for *Environmental Health Perspectives*. I am a currently Editor-in-Chief of the *Annals of Clinical Health*.

B. Positions and Honors

1970-85 Centers for Disease Control, US Public Health Service: Epidemic Intelligence Service (EIS) Officer (1970-72); Director, Research and Development, Bureau of Smallpox Eradication (1973-74); Chief, Environmental Hazards Activity, Bureau of Epidemiology (1974-79); Director, Division of Surveillance, Hazard Evaluations and Field Studies, National Institute for Occupational Safety and Health (1979-85). USPHS: LCDR (O-4) 1970-74; CDR (O-5) 1975-79 CAPT (O-6) 1980-85.

1997-98 U.S. Environmental Protection Agency: Senior Advisor to the Administrator on Children's Health and the Environment (sabbatical position).

1985- Icahn School of Medicine at Mount Sinai: Professor of Community Medicine (1985-present); Director, Division of Environmental and Occupational Medicine (1985-90); Professor of Pediatrics (1985- present); Ethel H. Wise Professor of Community Medicine, Chair, Department of Preventive Medicine (1990-present); Dean for Global Health (2010-present).

1996-05 U.S. Navy, Medical Corps: LCDR 1996-1998; CDR 1998-2004; CAPT 2004-2005. Director, Medical Services, Fleet Hospital, Fort Dix; Officer-in Charge, West Africa Training Cruise, 2004.

Other Professional Appointments

1975- Fellow, American Academy of Pediatrics
1978- Member, Society for Epidemiologic Research
1982- Elected Member, American Epidemiological Society
1982- Member, American Public Health Association; *Chair*, Occupational Health Section, 1989-1990.
1983- Fellow, Collegium Ramazzini; *President*, 1988- present
1983- Fellow, American College of Epidemiology; *Board of Directors*, 1990-1993
1985- Member, International Commission on Occupational Health
1985- Member, New York Occupational Medicine Association; *Board of Directors*, 1988-1990
1987- Elected Member, Institute of Medicine
1991 Fellow, New York Academy of Medicine
2002- Fellow, New York Academy of Sciences
2003- Fellow, American College of Preventive Medicine

Honors and Awards

1985 US Public Health Service, Meritorious Service Medal
1993 New England College of Occupational and Environmental Medicine, Harriet Hardy Award
1995 United Brotherhood of Carpenters, William Sidell Presidential Award
1995 American Public Health Association, Herbert L. Needleman Medal
1998 International Society for Occupational and Environmental Health, Vernon Houk Award
1999 American College of Preventive Medicine, Katherine Boucot Sturgis Award
2000 American Conference of Governmental Industrial Hygienists, William Steiger Memorial Award
2002 Mount Sinai School of Medicine, Jacobi Medallion
2002 Public Health Association of New York City, Haven Emerson Medal
2002 National Institute for Occupational Safety & Health, James P. Keogh Award
2003 American Public Health Association, David P. Rall Award
2003 Finnish Institute for Occupational Health, Jorma Rantanen Award
2005 Mount Sinai School of Medicine, J. Lester Gabrilove Award
2002-05 US Navy, Navy & Marine Corps Commendation Medal (3 awards), National Defense Service Medal, Secretary of Defense Medal for Outstanding Public Service.
2006 US Environmental Protection Agency, Children's Environmental Health Champion Award
2007 Mount Sinai School of Medicine, Doctor of Science (*honoris causa*)
2008 Collegium Ramazzini, Professor Irving J. Selikoff, M.D. Memorial Award
2008 Boston College, Alumni Award for Professional Excellence
2009 US Environmental Protection Agency, Region II Environmental Quality Award
2009 New York Academy of Medicine, Stephen Smith Medal for Lifetime Achievement in Public Health
2011 University of Medicine & Dentistry of New Jersey, Sen. Frank R Lautenberg Award in Public Health
2012 National Institute of Environmental Health Sciences, Invited Keynote Lecturer, 25th Annual Meeting of the Superfund Research Program

Committees

National Academy of Sciences, Assembly of Life Sciences. Board on Toxicology and Environmental Health Hazards, 1978-1987; Vice-Chair, 1981-1984.

Institute of Medicine, Committee for a Planning Study for an Ongoing Study of Costs of Environment-Related Health Effects, 1979-1980.

American Academy of Pediatrics, Committee on Environmental Hazards, 1976-1987; *Chair,* 1983-1987. National Academy of Sciences, Committee on Neurotoxicology in Risk Assessment; *Co-Chair*, 1987-1989. National Academy of Sciences, Committee on Pesticides in the Diets of Infants and Children; *Chair*, 1988-1993.

National Institutes of Health, Study Section on Epidemiology and Disease Control (II), 1986-1990. Presidential Advisory Committee on Gulf War Veterans' Illnesses, 1995-1996.

Department of Defense, Armed Forces Epidemiological Board, 2000-2002

American Journal of Industrial Medicine, Editor-in-Chief, 1987-2005.

Institute of Medicine, Chairman, Interest Group 14 Environmental and Occupational Health and Toxicology, 2009-2011.

National Institute of Environmental Health Sciences, External Clinical Advisory Council, 2009-present

National Institute of Child Health & Human Development. Scientific Vision Workshop on the Environment, 2011.

C. Selected Peer-Reviewed Publications (from 585 publications) Relevant to the Current Proposal

1. Landrigan PJ, Gehlbach SH, Rosenblum BF, Shoults JM, Candelaria RM, Barthel WF, Liddle JA, Smrek AL, Staehling NW, Sanders JF: Epidemic lead absorption near an ore smelter: the role of particulate lead. *New Engl J Med* 292:123-129, 1975.
2. Landrigan PJ, Whitworth RH, Baloh RW, Barthel WF, Staehling NW, Rosenblum BF: Neuropsychological dysfunction in children with chronic low-level lead absorption. *The Lancet* 1:708-712, 1975.
3. Landrigan PJ, Schechter CB, Lipton JM, Fahs MC, Schwartz J. Environmental pollutants and disease in American children: Estimates of morbidity, mortality and costs for lead poisoning, asthma, cancer and developmental disabilities. *Environ Health Perspect* 110:721-728, 2002. PMCID: PMC1240919
4. Fewtrell LJ, Prüss A, Landrigan P, Ayuso-Mateos JL. Estimating the global burden of disease from environmental lead exposure. *Environ Res* 94:120-133, 2004.
5. Trasande L, Schechter C, Landrigan PJ. Public Health and Economic Consequences of Environmental Methyl Mercury Toxicity to the Developing Brain. *Environ Health Perspect* 113:590-596, 2005. PMCID: PMC1257552
6. Herbert R, Moline J, Skloot G, Metzger K, Baron S, Luft B, Markowitz S, Udasin I, Harrison D, Stein D, Todd A, Enright P, Stellman JM, Landrigan PJ, Levin S. The World Trade Center Disaster and the Health of Workers: Five-Year Assessment of a Unique Medical Screening Program. *Environ Health Perspect* 114:1853-1858, 2006. PMCID: PMC1764159
7. Landrigan PJ, Trasande L, Thorpe LE, et al. The National Children's Study: A 21-year prospective study of 100,000 American children. *Pediatrics* 118(5):2173-2186, 2006.
8. Grandjean P, Landrigan PJ. Developmental Neurotoxicity of industrial chemicals: A silent pandemic. Lancet 368(9553):2167-2178, 2006.
9. Landrigan PJ, Woolf AD, Gitterman B, Lanphear B, Forman J, Karr C, Moshier EL, Steiner JF, Godbold J, Crain E. The Ambulatory Pediatric Association Fellowship in Pediatric Environmental Health: A Five-Year Assessment. *Environ Health Perspect* 115:1383-1387, 2007. PMCID: PMC2022661
10. Landrigan PJ, Goldman LR. Children's Vulnerability to Toxic Chemicals: A Challenge and Opportunity to Strengthen Health and Environmental Policy. *Health Affairs* 30(5):1-10, 2011.
11. Wisnivesky JP, Teitelbaum S, Todd A, Boffetta P, Crane M, Crowley L, Dellenbaugh C, Harrison D, Herbert R, Hyun K, Jeon Y, Kaplan J, Katz C, Levin S, Luft B, Markowitz S, Moline J, Osbay F, Pietrzak R, Shapiro M, Sharma V, Skloot G, Southwick S, Stevenson L, Udasin I, Wallenstein S, Landrigan PJ. Persistence of multiple illness in September 11 Rescue Workers. *The Lancet* 378: 888–897, 2011.
12. Ericson B, Caravanos J, Chatham-Stephens, Landrigan P, Fuller R. Approaches to systematic assessment of environmental exposures posed at hazardous waste sites in the developing world: the

Toxic Sites Identification Program. *Environ Monit Assess* May 17, 2012. PMID: 22592783.
13. Isukapalli S, Brinkerhoff CJ, Xu S, Dellarco M, Landrigan PJ, Lioy PJ, Georgopoulos PG. Exposure Indices for the National Children's Study: Application to inhalation exposures in Queens County, NY. *J Expo Sci Environ Epidemiol,* 2012. doi:10.1038/jes.2012.99 PMID: 23072768.
14. Landrigan PJ, Lambertini L, Birnbaum LS. A Research Strategy to Discover the Environmental Causes of Autism and Neurodevelopmental Disabilities. *Environ Health Perspect* 120:A258-A259, 2012.
15. Chatham-Stephens K, Caravanos J, Ericson B, Sunga-Amparo J, Susilorini B, Sharma P, Landrigan PJ, Fuller R. Burden of disease from toxic waste sites in India, Indonesia, and the Philippines in 2010. *Environ Health Perspect* 121(7):791-6, 2013. doi: 10.1289/ehp.1206127.
16. Caravanos J, Chatham-Stephens K, Ericson B, Landrigan PJ, Fuller R. The burden of disease from pediatric lead exposure at hazardous waste sites in 7 Asian countries. *Environ Res* 120:119-25, 2013. doi: 10.1016/j.envres.2012.06.006.
17. Grandjean P, Landrigan PJ. Neurobehavioural impacts of developmental toxicity. *Lancet Neurology* 13:330-338, 2014.

D. Research Support

Ongoing

Blacksmith Institute (Landrigan, PI) 1/1/12 – 12/23/17
Assessing the Disease Burden of Hazardous Waste Sites
The purpose of this contract is to support the development of a series of scientific papers that will assess the health burden associated with human exposure to hazardous waste sites in the developing world.

New York State Legislature 1/1/88 - present
Mount Sinai - Irving J. Selikoff Clinical Center in Occupational and Environmental Medicine. This Clinical Center of Excellence provides occupational medical services to working men and women in New York State with diseases and injuries of occupational origin.

Completed

T32HD049311 NICHD Landrigan (PI) 5/1/07 – 4/30/17
Research Training Program in Environmental Pediatrics
The goal of this interdisciplinary research training program is to train the next generation of physician-researchers and academic leaders in environmental pediatrics. Dr. Robert Wright succeeded me as PI on 7/1/2013.

C-010124 NYS DoH Landrigan (PI) 4/1/09 – 3/31/12; Lucchini (PI) 4/1/12 - present
World Trade Center Responders Data and Coordinating Center. This program has collected, analyzed and published medical monitoring and treatment data collected clinically on 30,000 9/11 responders evaluated at five Clinical Centers in the New York metropolitan area.

HHSN27201100002C NIH/NICHD Landrigan (PI) 9/30/05 – 9/30/12
National Children's Study, Queens Vanguard Center. The goal of this project was to recruit and follow 1250 live births in the NCS. The Queens Vanguard Center was one of the first six sites selected to pilot the NCS.

U10-OH08232 CDC Landrigan (PI) 6/1/04 – 3/31/12; Lucchini (PI) 4/1/12 - present
New York/New Jersey Education Research Center in Occupational Safety & Health. The goal of this multi-institutional program is to train professionals from multiple disciplines - medicine, nursing, industrial hygiene and industrial safety - to be future leaders in occupational health and safety.

Jerome A. Paulson, MD is an internationally recognized expert on environmental problems that impact on the health of children. He has frequently testified before Congress or participated in Congressional briefings on environmental health issues including air pollution, water pollution, lead poisoning, unconventional gas extraction (fracking). He has advised health professionals, parents, lawyers and others on a wide range of topics including, mercury exposure, damp buildings and mold, asthma, toxicants from an asphalt plant, exposures to radioactive materials, exposure to brominated flame retardants and other environmental health hazards. He has lectured in numerous venues in the US and overseas on pediatric environmental health including climate change, environmental health policy, reform of the Toxic Substances Control Act (TSCA) and other issues.

He holds a B.S. in Biochemistry with Honors and with Genera Honors from the University of Maryland, and an M.D. from Duke University. He did his residency training in pediatrics at the Johns Hopkins Hospitals and Sinai Hospital, both in Baltimore, as well as a fellowship in ambulatory pediatrics at Sinai Hospital.

Dr Paulson taught and practiced primary care pediatrics for many years at Case Western Reserve University-Rainbow Babies & Children's Hospital, at George Washington University and at Children's Pediatricians & Associates which is in Washington, DC. In 2008, he became the medical director for national and global affairs in the Child Health Advocacy Institute of Children's National Health System in Washington, DC.

In 2015, after nearly 25 years on the faculties of the George Washington University School of Medicine and Health Sciences and the George Washington University Milken Institute School of Public Health, Dr Paulson was named Professor Emeritus in Pediatrics in School of Medicine and Professor Emeritus in Environmental & Occupational Health at the School of Public Health. He is currently a consultant to non-governmental organizations, lawyers and others in matters related to children's health and the environment.

Dr. Paulson is the chair of the Executive Committee of the American Academy of Pediatrics' Council on Environmental Health; and he served 6 years as a member of the Children's Health Protection Advisory Committee for the U.S. Environmental Protection Agency (EPA). From 2000 until early 2015, he was the director of the Mid-Atlantic Center for Children's Health and the Environment (MACCHE), one of 10 pediatric environmental health specialty units (PEHSUs) in the US.

In 2014, Dr Paulson was elected to the Collegium Ramazzini, an international honorary society of 185 experts focused on environmental health issues. He also received the Hero's Award from the Healthy Schools Network, a national nongovernmental organization focused on environmental health in schools. Dr. Paulson served on the Pediatric Medical Care Committee of the National Commission on Children and Disasters and was part of the National Conversation on Public Health and Chemical Exposures organized by the Agency for Toxic Substances and Disease Registry (ATSDR). In 2011, Dr Paulson was elected to the American Pediatric Society primarily on the basis of his work in public policy and advocacy. In October 2004 he was a Dozor Visiting Professor at Ben Gurion University in Beer Sheva, Israel. He lectured there and throughout Israel on children's environmental health. Dr. Paulson was a

recipient of a Soros Advocacy Fellowship for Physicians from the Open Society Institute and worked with the Children's Environmental Health Network in the early 2000s. He also served as a special assistant to the director of the National Center on Environmental Health of the Centers for Disease Control and Prevention working on children's environmental health issues. During the 1985-86 academic year, he was a Robert Wood Johnson Health Policy Fellow and worked for a year on Capitol Hill for a member of the US House of Representatives. Dr. Paulson served on the American Academy of Pediatrics Council on Government Affairs and chaired the Public Policy Committee of the Ambulatory Pediatric Association (now the Academic Pediatric Association). He has published papers and book chapters on a number of topics related to children's health and the environment and has served on numerous boards and committees related to children's environmental health.

Lorne K. Garrettson, MD, FAAP, FAACT
Professor Emeritus, Emory University
Departments of Pediatrics and Environmental and Occupational Medicine
Board Certified: Pediatrics and Medical Toxicology

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Pomona College, BS, 1955
Johns Hopkins School of Medicine, MD, 1959

Pediatric Residency:	Johns Hopkins 1959-1961
	Children's Hospital of Boston 1961-2
Fellowship:	Duke University 1964-5
	Emory University, Clinical Pharmacology, 1965-8
Experience:	Faculty of Pediatrics, State University of New York, Buffalo 1968-73
	Director, Research Center for Children 1970-73
	Faculty of Pediatrics and Pharmacy and Pharmaceutics, Virginia Commonwealth University, 1973-88
	Director, Virginia Poison Center 1973-1988
	Faculty of Pediatrics and Environmental & Occupational Medicine, Emory University, 1988-99
	Director, Division of Clinical Toxicology and Pharmacology, 1988-99

Organizations and Editorial Boards

American Academy of Pediatrics, Vice Chair, Injury and Poison Prevention Committee1974-84
American Association of Poison Control Centers, Board of Directors, 1978-84
American Academy of Clinical Toxicology, Board of Directors, 1989-95
La Leche League International, Medical Advisory Board
Journal of Toxicology/ Clinical Toxicology, Editorial Board, 1992-98

Dr. Garrettson has worked in drug metabolism in children. He has been involved in the management of poison control centers and in the care of poisoned patients for 40 years . He has run lead clinics for the diagnosis and care of lead poisoned children in Virginia and Georgia. He developed the Georgia Poison Center as a reference center for the public and professionals on issues of drugs in human breast milk. He currently serves as an advisor to the Maryland Children's Environmental Health and Protection Advisory Committee and serves on his county's (Montgomery) child mortality committee. He serves as the MdAAP representative on the Maryland Pesticide Network board.

From Lorne K. Garrettson, MD, FAAP, FACMT

Enclosed are several abstracts which come from a welter of studies that show the rise of congenital anomalies in the USA. The linkage to pesticides comes from two lines of reasoning. Firstly, congenital anomalies don't occur equally throughout the year but are more frequent in the months when pesticide application is most frequent. The anomalies are liked by the time of conception or the period when the body is being formed in utero. Secondly, there is an increase in anomalies in areas where the mother lives closest to pesticide application.

There are no studies that would make this association iron-clad. That would require a systematic study of the pesticides found in the child or mother's blood and that the level of the pesticides was linked to the malformation.

Nonetheless, the epidemiological evidence is large and consistent. The abstracts enclosed below are a sampling of that collection of studies. There is one animal study included here but several more which support the hypothesis.

Acta Paediatr. 2009 Apr;98(4):664-9. doi: 10.1111/j.1651-2227.2008.01207.x. Epub 2009 Jan 22.

Agrichemicals in surface water and birth defects in the United States.

Winchester PD1, **Huskins J**, **Ying J**.

Author information:

- 1Section of Neonatal-Perinatal Medicine, Indiana University School of Medicine, Indianapolis, IN, USA. paul.winchester@ssfhs.org

Abstract

OBJECTIVES:

To investigate if live births conceived in months when surface water agrichemicals are highest are at greater risk for birth defects.

METHODS:

Monthly concentrations during 1996-2002 of nitrates, atrazine and other pesticides were calculated using United States Geological Survey's National Water Quality Assessment data. Monthly United States birth defect rates were calculated for live births from 1996 to 2002 using United States Centers for Disease Control and Prevention natality data sets. Birth defect rates by month of last menstrual period (LMP) were then compared to pesticide/nitrate means using logistical regression models.

RESULTS:

Mean concentrations of agrichemicals were highest in April-July. Total birth defects, and eleven of 22 birth defect subcategories, were more likely to occur in live births with LMPs between April and July. A significant association was found between the season of elevated agrichemicals and birth defects.

CONCLUSION:

Elevated concentrations of agrichemicals in surface water in April-July coincided with higher risk of birth defects in live births with LMPs April-July. While a causal link between agrichemicals and birth defects cannot be proven from this study an association might provide clues to common factors shared by both variables.

PMCID: PMC2667895

PMID: 19183116

Environ Res. 2014 Nov;135:133-8. doi: 10.1016/j.envres.2014.08.030. Epub 2014 Sep 28.

Residential agricultural pesticide exposures and risk of selected congenital heart defects among offspring in the San Joaquin Valley of California.

Carmichael SL1, **Yang W**2, **Roberts E**3, **Kegley SE**4, **Padula AM**2, **English PB**5, **Lammer EJ**6, **Shaw GM**2. 1Department of Pediatrics, Division of Neonatology and Developmental Medicine, Stanford University School of Medicine, Stanford, USA. Electronic address: **scarmichael@stanford.edu**. 2Department of Pediatrics, Division of Neonatology and Developmental Medicine, Stanford University School of Medicine, Stanford, USA. 3Public Health Institute, Oakland, CA, USA. 4Pesticide Research Institute, Berkeley, CA 94708, USA. 5California Department of Public Health, Richmond, CA, USA. 6Children's Hospital Oakland Research Institute, Oakland, CA, USA.

Abstract

BACKGROUND:

Pesticide exposures are ubiquitous and of substantial public concern. We examined the potential association of congenital heart defects with residential proximity to commercial agricultural pesticide applications in the San Joaquin Valley, California.

METHODS:

Study subjects included 569 heart defect cases and 785 non-malformed controls born from 1997 to 2006 whose mothers participated in a population-based case-control study. Associations with any versus no exposure to physicochemical groups of pesticides and specific chemicals were assessed using logistic regression adjusted for relevant covariates, for 8 heart defect phenotypes that included ≥ 50 cases and pesticide exposures with ≥ 5 exposed cases and controls, which resulted in 235 comparisons.

RESULTS:

38% of cases and controls were classified as exposed to pesticides within a 500 m radius of mother's address during a 3-month periconceptional window. Adjusted odds ratios (AORs) with 95% CIs excluding 1.0 were observed for 18 comparisons; all were >1 and ranged from 1.9 to 7.1. They included tetralogy of Fallot (n=101 cases) and neonicotinoids; hypoplastic left heart syndrome (n=59) and strobins; coarctation of the aorta (n=74) and pyridazinones; pulmonary valve stenosis (n=53) and bipyridyliums and organophosphates; ventricular septal defects (n=93) and avermectins and pyrethroids; and atrial septal defects (n=132) and dichlorphenoxy acid or esters, organophosphates, organotins, and pyrethroids. No AORs met both of these criteria for d-transposition of the great arteries (n=58) or heterotaxia (n=53).

CONCLUSIONS:

Most pesticides were not associated with increased risk of specific heart defect phenotypes. For the few that were associated, results should be interpreted with caution until replicated in other study populations.

J Pediatr Urol. 2009 Feb;5(1):17-24. doi: 10.1016/j.jpurol.2008.08.006. Epub 2008 Oct 10.

Pesticides and hypospadias: a meta-analysis.

Rocheleau CM1, Romitti PA, Dennis LK. Author information: Department of Epidemiology, College of Public Health, University of Iowa, Iowa City, IA, USA.

Abstract

OBJECTIVE:

To use meta-analytic techniques to synthesize the findings of the current body of published literature regarding the risk of hypospadias resulting from parental exposure to pesticides.

MATERIALS AND METHODS:

A search of Pub Med for original research published in English from January 1966 through March 2008 identified 552 studies, 90 of which were reviewed in detail. Nine studies met all study inclusion criteria. Two reviewers independently abstracted data from each included study. Any disagreements were resolved by consensus. Pooled risk ratios (PRRs) and confidence intervals (CIs) were calculated using both random and fixed effects models, along with statistical tests of homogeneity.

RESULTS:

Elevated but marginally significant risks of hypospadias were associated with maternal occupational exposure (PRR of 1.36, CI=1.04-1.77), and paternal occupational exposure (PRR of 1.19, CI=1.00-1.41). Subgroup analyses provided insights into needed designs for future studies. Notably, exposure assessment using a job-exposure matrix resulted in slightly higher estimated risk than agricultural occupation in fathers; but this effect was reversed in mothers, suggesting the importance of indirect and residential pesticide exposures in this group.

CONCLUSIONS:

Despite potential exposure misclassification, which would tend to diminish observed associations, the previous literature indicates a modestly increased risk of hypospadias associated with pesticide exposure.

PMID: 18848807 [PubMed - indexed for MEDLINE

Environ Health Perspect. 2004 May;112(6):703-9.

Low-dose agrochemicals and lawn-care pesticides induce developmental toxicity in murine preimplantation embryos.

Greenlee AR1, Ellis TM, Berg RL.

Author information:

- 1Reproductive Toxicology Laboratory, Marshfield Clinic Research Foundation, Marshfield, Wisconsin 54449, USA. greenlee.anne@mcrf.mfldclin.edu

Abstract

Occupational exposures to pesticides may increase parental risk of infertility and adverse pregnancy outcomes such as spontaneous abortion, preterm delivery, and congenital anomalies. Less is known about residential use of pesticides and the risks they pose to reproduction and development. In the present study we evaluate environmentally relevant, low-dose exposures to agrochemicals and lawn-care pesticides for their direct effects on mouse preimplantation embryo development, a period corresponding to the first 5-7 days after human conception. Agents tested were those commonly used in the upper midwestern United States, including six herbicides [atrazine, dicamba, metolachlor, 2,4-dichlorophenoxyacetic acid (2,4-D)], pendimethalin, and mecoprop), three insecticides (chlorpyrifos, terbufos, and permethrin), two fungicides (chlorothalonil and mancozeb), a desiccant (diquat), and a fertilizer (ammonium nitrate). Groups of 20-25 embryos were incubated 96 hr in vitro with either individual chemicals or mixtures of chemicals simulating exposures encountered by handling pesticides, inhaling drift, or ingesting contaminated groundwater. Incubating embryos with individual pesticides increased the percentage of apoptosis (cell death) for 11 of 13 chemicals ($p \leq 0.05$) and reduced development to blastocyst and mean cell number per embryo for 3 of 13 agents ($p \leq 0.05$). Mixtures simulating preemergent herbicides, postemergent herbicides, and fungicides increased the percentage of apoptosis in exposed embryos ($p \leq 0.05$). Mixtures simulating groundwater contaminants, insecticide formulation, and lawn-care herbicides reduced development to blastocyst and mean cell number per embryo ($p \leq 0.05$). Our data demonstrate that pesticide-induced injury can occur very early in development, with a variety of agents, and at concentrations assumed to be without adverse health consequences for humans.

PMCID: PMC1241965 Free PMC Article

PMID: 15121514

Environ Health Perspect. 2003 Jul;111(9):1259-64.

Birth malformations and other adverse perinatal outcomes in four U.S. Wheat-producing states.

Schreinemachers DM1.

Author information:

- 1National Health and Environmental Effects Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711, USA. schreinemachers.dina@epa.gov

Comment in

"Birth malformations and other adverse perinatal outcomes": available data sources pose a dilemma. [Environ Health Perspect. 2003]

Re: "Birth malformations and other adverse perinatal outcomes in four U.S. wheat-producing states". [Environ Health Perspect. 2003]

Abstract

Chlorophenoxy herbicides are widely used in the United States and Western Europe for broadleaf weed control in grain farming and park maintenance. Most of the spring and durum wheat produced in the United States is grown in Minnesota, Montana, North Dakota, and South Dakota, with more than 85% of the acreage treated with chlorophenoxy herbicides such as 2,4-dichlorophenoxyacetic acid (2,4-D) and 4-chloro-2-methylphenoxyacetic acid (MCPA). Rates of adverse birth outcomes in rural, agricultural counties of these states during 1995-1997 were studied by comparing counties with a high proportion of wheat acreage and those with a lower proportion. Information routinely collected and made available by federal agencies was used for this ecologic study. Significant increases in birth malformations were observed for the circulatory/respiratory category for combined sexes [odds ratio (OR) = 1.65; 95% confidence interval (CI), 1.07-2.55]. A stronger effect was observed for the subcategory, which excluded heart malformations (OR = 2.03; 95% CI, 1.14-3.59). In addition, infants conceived during April-June--the time of herbicide application--had an increased chance of being diagnosed with circulatory/respiratory (excluding heart) malformations compared with births conceived during other months of the year (OR = 1.75; 95% CI, 1.09-2.80). Musculoskeletal/integumental anomalies increased for combined sexes in the high-wheat counties (OR = 1.50; 95% CI, 1.06-2.12). Infant death from congenital anomalies significantly increased in high-wheat counties for males (OR = 2.66; 95% CI, 1.52-4.65) but not for females (OR = 0.48; 95% CI, 0.20-1.15). These results are especially of concern because of widespread use of chlorophenoxy herbicides.

PMCID: PMC1241584 **Free PMC Article**

PMID: 12842783 [PubMed - indexed for MEDLINE

Environ Health Perspect. 2002 Jun;110 Suppl 3:441-9.

Birth defects, season of conception, and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA.

Garry VF1, Harkins ME, Erickson LL, Long-Simpson LK, Holland SE, Burroughs BL.

Author information:

- 1Environmental Medicine and Pathology Laboratory, 1st Floor Stone Laboratory 1, University of Minnesota, 421 29th Avenue SE, Minneapolis, MN 55414, USA. garry001@umn.edu

Abstract

We previously demonstrated that the frequency of birth defects among children of residents of the Red River Valley (RRV), Minnesota, USA, was significantly higher than in other major agricultural regions of the state during the years 1989-1991, with children born to male pesticide applicators having the highest risk. The present, smaller cross-sectional study of 695 families and 1,532 children, conducted during 1997-1998, provides a more detailed examination of reproductive health outcomes in farm families ascertained from parent-reported birth defects. In the present study, in the first year of life, the birth defect rate was 31.3 births per 1,000, with 83% of the total reported birth defects confirmed by medical records. Inclusion of children identified with birth or developmental disorders within the first 3 years of life and later led to a rate of 47.0 per 1,000 (72 children from 1,532 live births). Conceptions in spring resulted in significantly more children with birth defects than found in any other season (7.6 vs. 3.7%). Twelve families had more than one child with a birth defect (n = 28 children). Forty-two percent of the children from families with recurrent birth defects were conceived in spring, a significantly higher rate than that for any other season. Three families in the kinships defined contributed a first-degree relative other than a sibling with the same or similar birth defect, consistent with a Mendelian inheritance pattern. The remaining nine families did not follow a Mendelian inheritance pattern. The sex ratio of children with birth defects born to applicator families shows a male predominance (1.75 to 1) across specific pesticide class use and exposure categories exclusive of fungicides. In the fungicide exposure category, normal female births significantly exceed male births (1.25 to 1). Similarly, the proportion of male to female children with birth defects is significantly lower (0.57 to 1; p = 0.02). Adverse neurologic and neurobehavioral developmental effects clustered among the children born to applicators of the fumigant phosphine (odds ratio [OR] = 2.48; confidence interval [CI], 1.2-5.1). Use of the herbicide glyphosate yielded an OR of 3.6 (CI, 1.3-9.6) in the neurobehavioral category. Finally, these studies point out that (a) herbicides applied in the spring may be a factor in the birth defects observed and (b) fungicides can be a significant factor in the determination of sex of the children of the families of the RRV. Thus, two distinct classes of pesticides seem to have adverse effects on different reproductive outcomes. Biologically based confirmatory studies are needed.

PMCID: PMC1241196 Free PMC Article

PMID: 12060842 [PubMed - indexed for MEDLINE

Environ Health Perspect. 2002 Jun;110 Suppl 3:441-9.

Birth defects, season of conception, and sex of children born to pesticide applicators living in the Red River Valley of Minnesota, USA.

Garry VF1, Harkins ME, Erickson LL, Long-Simpson LK, Holland SE, Burroughs BL.

Author information:

- 1Environmental Medicine and Pathology Laboratory, 1st Floor Stone Laboratory 1, University of Minnesota, 421 29th Avenue SE, Minneapolis, MN 55414, USA. garry001@umn.edu

Abstract

We previously demonstrated that the frequency of birth defects among children of residents of the Red River Valley (RRV), Minnesota, USA, was significantly higher than in other major agricultural regions of the state during the years 1989-1991, with children born to male pesticide applicators having the highest risk. The present, smaller cross-sectional study of 695 families and 1,532 children, conducted during 1997-1998, provides a more detailed examination of reproductive health outcomes in farm families ascertained from parent-reported birth defects. In the present study, in the first year of life, the birth defect rate was 31.3 births per 1,000, with 83% of the total reported birth defects confirmed by medical records. Inclusion of children identified with birth or developmental disorders within the first 3 years of life and later led to a rate of 47.0 per 1,000 (72 children from 1,532 live births). Conceptions in spring resulted in significantly more children with birth defects than found in any other season (7.6 vs. 3.7%). Twelve families had more than one child with a birth defect (n = 28 children). Forty-two percent of the children from families with recurrent birth defects were conceived in spring, a significantly higher rate than that for any other season. Three families in the kinships defined contributed a first-degree relative other than a sibling with the same or similar birth defect, consistent with a Mendelian inheritance pattern. The remaining nine families did not follow a Mendelian inheritance pattern. The sex ratio of children with birth defects born to applicator families shows a male predominance (1.75 to 1) across specific pesticide class use and exposure categories exclusive of fungicides. In the fungicide exposure category, normal female births significantly exceed male births (1.25 to 1). Similarly, the proportion of male to female children with birth defects is significantly lower (0.57 to 1; p = 0.02). Adverse neurologic and neurobehavioral developmental effects clustered among the children born to applicators of the fumigant phosphine (odds ratio [OR] = 2.48; confidence interval [CI], 1.2-5.1). Use of the herbicide glyphosate yielded an OR of 3.6 (CI, 1.3-9.6) in the neurobehavioral category. Finally, these studies point out that (a) herbicides applied in the spring may be a factor in the birth defects observed and (b) fungicides can be a significant factor in the determination of sex of the children of the families of the RRV. Thus, two distinct classes of pesticides seem to have adverse effects on different reproductive outcomes. Biologically based confirmatory studies are needed.

PMCID: PMC1241196 Free PMC Article

PMID: 12060842

Environ Health Perspect. 1997 Mar;105(3):308-14.

Intrauterine growth retardation in Iowa communities with herbicide-contaminated drinking water supplies.

Munger R1, Isacson P, Hu S, Burns T, Hanson J, Lynch CF, Cherryholmes K, Van Dorpe P, Hausler WJ Jr.

Author information:

- 1Department of Preventive Medicine and Environmental Health, University of Iowa, Iowa City 52242, USA.

Erratum in

- Environ Health Perspect 1997 Jun;105(6):570.

Abstract

In a statewide survey of 856 Iowa municipal drinking water supplies in 1986-1987 the Rathbun rural water system was found to contain elevated levels of triazine herbicides. Rates of low birth weight, prematurity, and intrauterine growth retardation (IUGR) in live singleton births during the period 1984-1990 by women living in 13 communities served by the Rathbun water system were compared to other communities of similar size in the same Iowa counties. The Rathbun communities had a greater risk of IUGR than southern Iowa communities with other surface sources of drinking water (relative risk = 1.8; 95% CI = 1.3, 2.7). Multiple linear regression analyses revealed that levels of the herbicides atrazine, metolachlor, and cyanzinc were each significant predictors of community IUGR rates in southern Iowa after controlling for several potentially confounding factors including maternal smoking and socioeconomic variables. The association with IUGR was strongest for atrazine, but all three herbicides were intercorrelated and the independent contributions of each to IUGR risk could not be determined. We conclude that communities in southern Iowa with drinking water supplies contaminated with herbicides have elevated rates of IUGR compared to neighboring communities with different water supplies. Because of the limitations of the ecologic design of this study, including aggregate rather than individual measures of exposure and limited ability to control for confounding factors related to source of drinking water and risk of IUGR, a strong causal relationship between any specific water contaminant and risk of IUGR cannot yet be inferred. The association between the water supplied to the Rathbun communities and the increased risk of IUGR should be considered a preliminary finding that needs to be verified by more detailed epidemiologic studies.
PMCID: PMC1470002 Free PMC Article

PMID: 9171992

Am J Epidemiol. 2001 Oct 15;154(8):702-10.

Case-cohort analysis of agricultural pesticide applications near maternal residence and selected causes of fetal death.

Bell EM1, Hertz-Picciotto I, Beaumont JJ.

Author information:

- 1Department of Epidemiology, School of Public Health, University of North Carolina, Chapel Hill, USA. belle@mail.nih.gov

Comment in

- Re: "Case-cohort analysis of agricultural pesticide applications near maternal residence and selected causes of fetal death". [Am J Epidemiol. 2002]

Abstract

The potential association between fetal death and residential proximity to agricultural pesticide applications was examined in 10 California counties for 1984. A case-cohort analysis utilized 319 cases of selected causes of fetal death other than congenital anomalies and 611 non-cases. A statewide database of all applications of restricted pesticides was linked to maternal address; residential proximity within 1 mile (1.6 km) provided a surrogate for daily exposure. Pesticides were grouped by chemical class and mechanism of acetylcholinesterase inhibition. Multivariate proportional hazards models using time-dependent exposure variables were fit for each pesticide grouping. Overall, pesticides showed no strong association with fetal death. Slightly elevated risks were observed for women who resided near applications of halogenated hydrocarbons, carbamates, estrogenic pesticides, and carbamate acetylcholinesterase inhibitors during the second trimester, with hazard ratios of 1.3 (95% confidence interval (CI): 1.0, 1.8), 1.3 (95% CI: 1.0, 1.8), 1.4 (95% CI: 0.8, 2.5), and 1.3 (95% CI: 1.0, 1.8), respectively. In a month-by-month analysis, elevated risks were observed when exposure occurred during gestational months 3 and 4 for carbamates and carbamate inhibitors and during months 4 and 5 for halogenated hydrocarbons. Since previous studies have relied on personal recall of exposure, major strengths of this study were the objective source for environmental pesticide exposure assessment and the use of data on the timing of exposure.

PMID: 11590082

Epidemiology. 2001 Mar;12(2):148-56.

A case-control study of pesticides and fetal death due to congenital anomalies.

Bell EM 1, **Hertz-Picciotto I**, **Beaumont JJ**.

Author information:

- 1Department of Epidemiology, School of Public Health, University of North Carolina, Chapel Hill, USA.

Erratum in

- Epidemiology. 2001 Sep;12(5):596.

Comment in

- **Pesticides and fetal death due to congenital anomalies: implications of an erratum.** [Epidemiology. 2001]
- **The epidemiologic study of birth defects and pesticides.** [Epidemiology. 2001]
- **Pesticides and fetal death due to congenital abnormalities.** [Epidemiology. 2001]

Abstract

We examined the association between late fetal death due to congenital anomalies (73 cases, 611 controls) and maternal residential proximity to pesticide applications in ten California counties. A statewide database of all applications of restricted pesticides was linked to maternal address to determine daily exposure status. We examined five pesticide chemical classes. The odds ratios from logistic regression models, adjusted for maternal age and county, showed a consistent pattern with respect to timing of exposure; the largest risks for fetal death due to congenital anomalies were from pesticide exposure during the 3rd-8th weeks of pregnancy. For exposure either in the square mile of the maternal residence or in one of the adjacent 8 square miles, odds ratios ranged from 1.4 (95% confidence interval = 0.8-2.4) for phosphates, carbamates, and endocrine disruptors to 2.2 (95% confidence interval = 1.3-3.9) for halogenated hydrocarbons. Similar odds ratios were observed when a more restrictive definition of nonexposure (not exposed to any of the five pesticide classes during the 3rd-8th weeks of pregnancy) was used. The odds ratios for all pesticide classes increased when exposure occurred within the same square mile of maternal residence.

PMID: 11246574

PESTICIDE RISK ASSESSMENT SCIENCE: PRESENTATION TO THE T&E COMMITTEE AND RESPONSES TO COMMENTS

Stuart Z. Cohen, Ph.D., CGWP

Attachments

1. March 16 slides (p.2) (p.2)
2. Three page tabular summary of my March 10 letter to Council President Leventhal (cc to Committee Chair Berliner) (p.13)3)
3. Letter to the Committee with an evaluation of comments (19 pp.) and a tabular summary (p.16) (p.16)
4. Condensed curriculum vitae (p.48)0)
5. US EPA pesticide data requirements (Title 40 CFR Part 158) (p.50)2)

(23)

PESTICIDE RISK ASSESSMENT SCIENCE: REGULATORY AND OPEN LITERATURE PERSPECTIVES

by
Stuart Z. Cohen, Ph.D., CGWP
Environmental & Turf Services, Inc.
Wheaton, MD

before the
Transportation, Infrastructure, Energy and Environment Committee
Chairman, Roger Berliner
Montgomery County Council

March 16, 2015

PRESENTATION OUTLINE

I. My Qualifications

II. Basic Principles of Toxicology and Environmental Risk Assessment

III. Pesticide Regulatory Risk Assessments

IV. Pesticide Epidemiology: the Good, the Bad, and the Ugly

V. Analysis of Some Public Comments Related to Pesticide Risks

2

I. MY QUALIFICATIONS (CV in Attachment 4)

- 39 years experience in environmental and human health risk assessment, with a focus on pesticides and heavy metals.
- 11 years with the US EPA.
- Ph.D. in physical organic chemistry.
- Immunology research, and coursework in chemical carcinogenesis.
- Co-chair, Public Outreach Committee of the Society of Environmental Toxicology & Chemistry NA.
- Certified Ground Water Professional.

3

II. BASIC PRINCIPLES

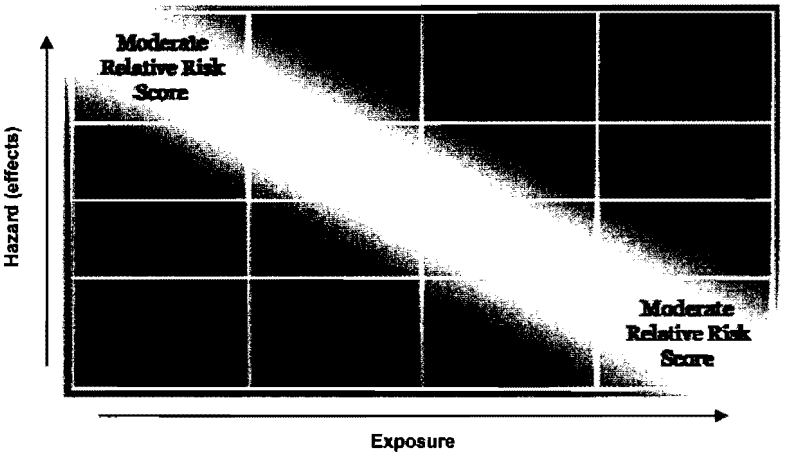
Paracelsus, Father of Modern Toxicology (16th century):

"Solely the dose determines that a thing is not a poison."

(From his Third Defense [Borzelleca, 2000])

4

RISK IS A COMBINATION OF TOXICITY (HAZARD) AND EXPOSURE



Copied from Menzie in Cohen et al., 2014]

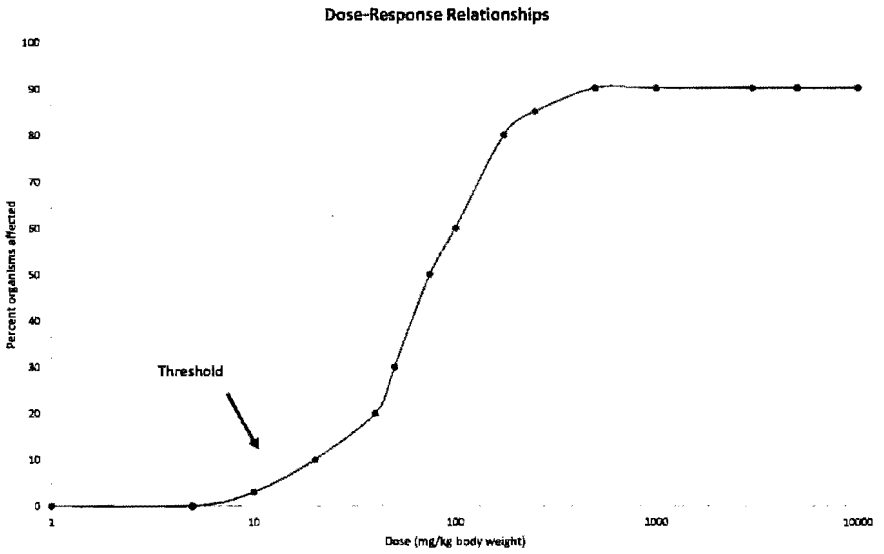
5

The Importance of the Dose

Substance	RDA or AI Dose	Benefit	Toxic Dose	Adverse Effect
Manganese	~2 mg/day	Enzyme cofactor. Essential nutrient in many chemical processes, possibly strengthens weak bones.	11 mg/d; 0.1 mg/d?? (children/chronic)	PD symptoms. Children cognitive (IQ) and neurobehavioral. (Bouchard et al. [2011] and Oulhote et al., [2014])
Selenium	0.055 mg/day	Vitamin E cofactor	0.4 mg/day	Toxic effects similar to arsenic; possibly non-melanoma skin cancer.

6

Dose-Response Relationships



7

III. PESTICIDE REGULATORY RISK ASSESSMENTS

How Does the US EPA Determine Exposure, Hazard, and Risk Prior to Product Registration?

Required Data (75-100+ studies; Attachment 6 of my packet)

- Toxicology: 15-27 studies in the areas of acute oral toxicity, 90 day neurotoxicity, prenatal developmental toxicity (2 species), *in vivo* cytogenetics, etc.
- Environmental fate (mobility and persistence): 9-15 studies
- Turf transferable residues.
- Also, ecotoxicology (bees, plants, fish, etc.), product chemistry, crop residue, spray drift, etc.
- 300+ scientist reviewers.

8

How are the Effects (Hazard) and Exposure Data Integrated into a Risk Assessment?

- Sensitive effects endpoints for people - - no observed adverse effects levels - - are lowered (divided) by a series of large uncertainty and safety factors.
- ***All significant exposure routes are conservatively modeled*** - - drinking water, food, contact between kids and turf, pollinators, workers, etc.
- The potential exposures to humans are aggregated and, for pesticides with food uses - - most turf pesticides - - a "safe" determination must be made pursuant to the Food Quality Protection Act (FQPA) of 1996 ". . . ***that there is a reasonable certainty that no harm will result*** . . ."

9

IV. PESTICIDE EPIDEMIOLOGY: THE GOOD, THE BAD, AND THE UGLY

"Epidemiology is the study of how often diseases occur in different groups of people and why." (British Medical Journal)

- Almost all epidemiology studies of pesticides have been ***case control studies***, which is a weakness: ***they may prove an association, but they do not prove causation***.
- Key weakness: surrogates for exposure are usually extremely crude.
- Key weakness: lack of control of confounding factors.
- Longitudinal (prospective/cohort) studies are more definitive: they follow a group of people for many years.
- Key weakness in both kinds of studies (as well as "ecologic"): they often don't consider disease etiology.

In a nutshell: cohort studies work from exposure to disease occurrence, case control and ecologic studies work from the disease backward to crude exposure estimates. One should not evaluate individual studies in isolation.

10

An Example of the Need to do a Comprehensive Analysis: Cancer Incidence Based on Possible Residential Exposure

Leiss and Savitz (1995): Case control study of childhood cancer

- 252 children in the Denver area diagnosed with cancer 1976-1983.
- Asked "...whether the yard...was ever treated with insecticides or herbicides...".
- The strongest association was found for soft tissue sarcomas (odds ratio (OR)=4 [with wide CIs], n=24; weak). No increased risk for total cancers, all leukemias, brain tumors, and lymphomas. BUT, the # of cases is small, and.....

11

Cancer and Residential Exposure (cont'd)

- Note: OR=0.5 for brain tumors (n=45) and 0.6 for lymphomas (n=31)!? DOES OR<1 MEAN PESTICIDE APPLICATION LOWERS THE CANCER RISK?? Not likely.....
- No information presented on potential confounding factors nor on disease etiology.
- The statistical power of the study is weak.
- "Davis et al.'s report of strong associations between use of herbicides and insecticides in the yard and brain tumors in children[7] was not corroborated by our data."

12

Does 2,4-D Cause Cancer in Humans? An Example of a Comprehensive Analysis

Not according to the US EPA, a peer-reviewed paper from a Harvard researcher, and others.

- From the US EPA's comprehensive re-re-reanalysis of 2,4-D science (2012):

"EPA's report, dated December 8, 2004, found that ***none of the more recent epidemiological and animal studies supported a conclusion that 2,4-D was a likely human carcinogen.***"

"A part of this cancer assessment was the review of data bearing on 2,4-D's potential mutagenicity. ***EPA has consistently found that these data do not support classification of 2,4-D as a carcinogen. This view was concurred in by the Joint Committee of SAB/SAP.***"

- von Stackelberg (2013) reviewed the results of 239 studies and concluded:

"Potential associations in case-control studies were based on univariate analyses without including other potential exposures and/or known risk factors, while those studies incorporating the variety of exposures experienced in the environment generally show no statistically significant role for exposures to chlorophenoxy compounds."

"(. . . t(14;18) translocations) find no association with exposure. . ."

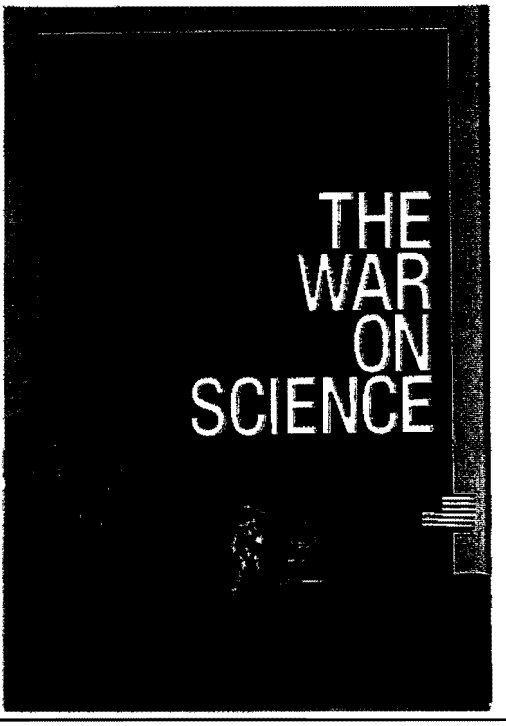
"The evidence does not support an association between exposures to 2,4-D and/or MCPA and direct DNA interaction;"

13

V. THE SCIENCE UNDERLYING SOME PUBLIC COMMENTS REALTED TO PESTICIDE RISKS

See my 19 page (+ appendices) March 10 letter to the T&E Committee for an evaluation of comments made at the two hearings.

14



15

A Scientific Basis for our Beliefs?

"Science is not a body of facts," says geophysicist Marcia McNutt, who once headed the U.S. Geological Survey and is now editor of *Science*, the prestigious journal. "Science is a method for deciding whether what we choose to believe has a basis in the laws of nature or not."

March, 2015, National Geographic, p. 40.

16

A Scientific Basis? (cont'd): The Case of Glyphosate

- Cancer - - "There is no evidence of carcinogenic potential." "Group E." [US EPA, 2000 and 2012; EU]
- Endocrine Disruption - - No adverse effects in the 11 EPA-validated *in vitro* and *in vivo* assays (Webb et al., 2013; Bailey et al., 2013)

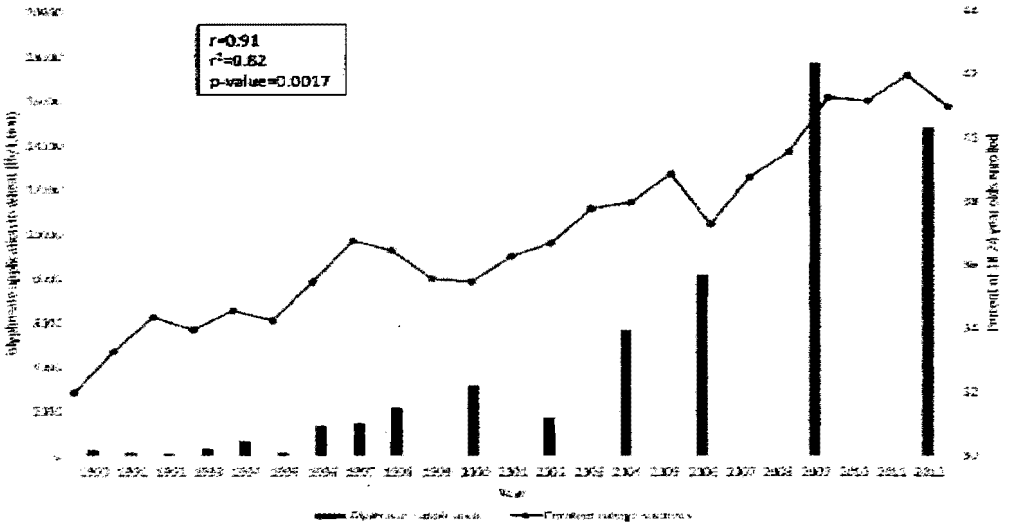
17

Comments on Glyphosate (cont'd)

- Glyphosate is a cause of ". . . gastrointestinal disorders, obesity, diabetes, heart disease, depression, autism, infertility, cancer and Alzheimer's disease" (Samsel and Seneff, 2013a and b):
  - The paper is more heavily infused with conjecture than any scientific paper I have ever seen.
  - No data were generated.
  - Lab studies they cited exposed human cells, etc. to glyphosate at 1,000X and 10,000X environmental concentrations.
  - Another fundamental flaw is the assumption that humans provide an essential amino acid internally.
  - The lead author's degrees are in electrical engineering, and she has a record of attacking vaccination programs, e.g., Seneff (2011).
  - Their graphs plotting glyphosate use on crops vs disease, are not proof, for example:

18

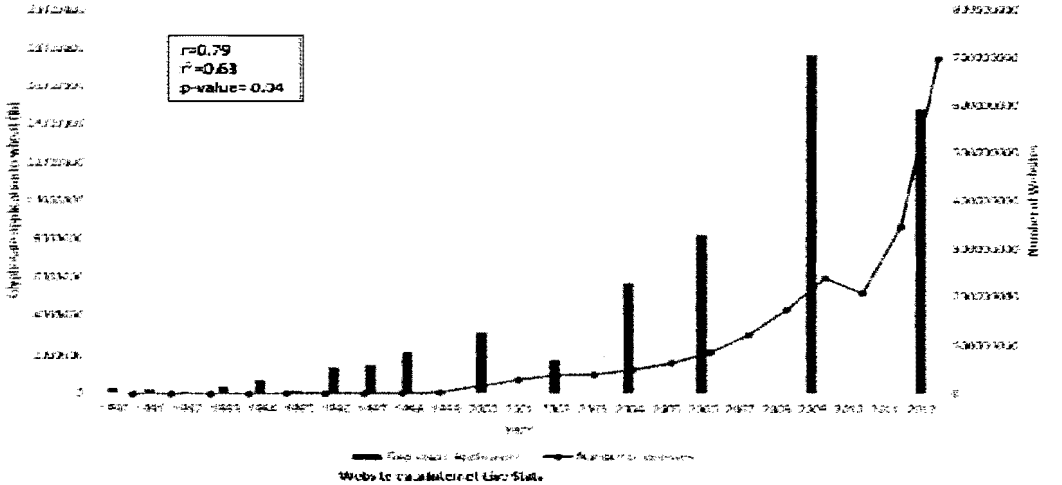
Figure 2b. Enrollment in College vs. Glyphosate Application to Wheat



Enrollment data from the National Center for Education Statistics
http://nces.ed.gov/programs/digest/d13/tables/dt13_302.60.asp (2013).
Glyphosate application data from USDA/NASS.

19

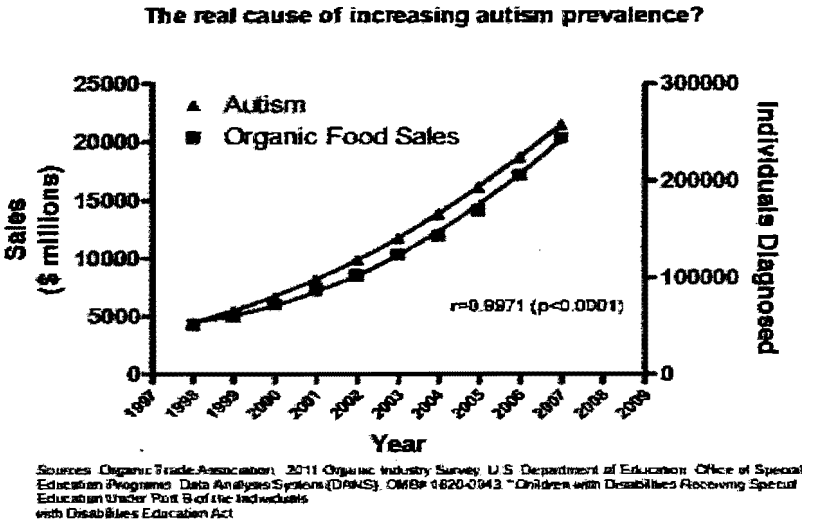
Figure 2c. Number of Websites vs. Glyphosate Application to Wheat



Web to calculate est. Geo State.
http://www.internetlivestats.com/total-number-of-websites/
Glyphosate application data from USDA/NASS.

20

Correlation of Organic Food Sales with Autism Diagnoses*



21

* Slide credit: C. Thorpe, CropLife America, Washington DC.

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Oulhote, Y., D. Mergler, B. Barbeau, D.C. Bellinger, T. Bouffard, M.-È. Brodeur, D. Saint-Amour, M. Legrand, S. Sauvé, and M.F. Bouchard. 2014. Neurobehavioral Function in School-Age Children Exposed to Manganese in Drinking Water. *Environ. Health Perspect.*, 122:1343–1350.

Samsel, A. and S. Seneff. 2013a. Glyphosate's Suppression of Cytochrome P450 Enzymes and Amino Acid Biosynthesis by the Gut Microbiome: Pathways to Modern Diseases. *Entropy* 15:1416-1463.

Samsel, A., and S. Seneff. 2013b. Glyphosate, Pathways to Modern Diseases II: Celiac Sprue and Gluten Intolerance. *Interdiscip. Toxicol.*, 6(4):159-184.

Seneff, S. 2011. Autism, Vaccines, and Cholesterol Sulfate, presented at the Wise Traditions Conference in Dallas, TX. Presentation available at http://people.csail.mit.edu/seneff/.

US EPA. April 18, 2012. "Petitions to Revoke Tolerances; Denials: Natural Resources Defense Council, 2,4-dichlorophenoxyacetic acid (2,4-D). Federal Register Vol. 77, No. 75. pp. 23125-23158.

von Stackelberg, K. 2013. A Systematic Review of Carcinogenic Outcomes and Potential Mechanisms from Exposure to 2,4-D and MCPA in the Environment. *J. Toxicol.* 2013:1.

Webb, E.G., D.A. Saltmiras, and S.L. Levine. 2013. Endocrine Disruptor Screening Program (EDSP) Tier I In Vitro Assays Indicate Glyphosate Does Not Interact With Estrogen and Androgen Receptors nor Inhibit Steroidogenesis. Poster Abstracts, Poster 500. *Int. J. Toxicol.* 32(1):58.

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Environmental & Turf Services, Inc.

Summary Chart

Evaluation of Statements made by Advocates for Council Bill 52-14

Based on March 10, 2015 Letter by S. Cohen

Statement from Bill Advocates	Fact Check
EPA often ignores the 10-fold safety factor to protect infants and children	**Incorrect** • The FQPA safety factor is in addition to an existing fact of 10X to 100X (typically 100X). • The Food Quality and Protection Act states that EPA may use a different margin of safety "...if, on the basis of reliable data, such margin will be safe for infants and children."
Of the most commonly used lawn care pesticides by homeowners and professional turf and landscape companies, 18 disrupt the endocrine hormone system, 17 are linked to cancer, 11 are linked to birth defects, 19 to reproductive effects, 24 to liver or kidney damage, and 14 to neurotoxicity (autism and learning disabilities (EPA, 1990).	**Incorrect** • While the statement references the EPA 1990 study, the only source of this statement is a Beyond Pesticides document on its website. • I peer reviewed the 1990 EPA National Pesticide Survey report and no such statement was made. • The 1990 GAO report has no data to support this statement. • Many of the pesticides listed are no longer in use for lawn care.
EPA approval doesn't mean a pesticide is safe. FIFRA is a Risk/Benefit statute.	**Incorrect** • This has not been true for most pesticides since 1996. Passage of the Food Quality Protection Act ensures that decisions for pesticides for food uses must be held to a higher standard: "a reasonable certainty of no harm" with a focus on children, evaluation of aggregate exposures and additional safety factor for pre-natal and post-natal exposures.
EPA only examines one chemical at a time	**Misleading** • EPA scientists are required to consider and combine the cumulative risks of multiple pesticides that share a common mechanism of toxicity.
Lawn care pesticides are tested for human and health effects only if they are also registered for food uses	**Misleading** • Almost all of commonly used turfgrass pesticides have food uses and are registered based, in part, on animal feeding studies.
Inert ingredients don't have to be tested as a complete package.	**Misleading** • In 1987 EPA established four categories of inert ingredients and placed a high priority on their evaluation. Evaluations concluded in 2006 and found that all food use inert ingredient tolerances and exemptions are considered safe. • The current process for submitting a food or non-food use petition for an inert ingredient include data requirements such as physical and chemical properties, acute and chronic toxicity data, reproduction and developmental data, mutagenicity data, neurotoxicity data, endocrine data, immunotoxicity data, and carcinogenicity data that will "provide a scientific explanation why (the inert ingredient) would not be carcinogenic."

EPA does no testing on its own but relies on tests paid for by chemical companies.	**Misleading** The commercial labs are subject to stringent requirements • Must follow EPA's prescribed testing guidelines; • Must follow the extensive set of EPA's Good Laboratory Practices (GLPs) • Are subject to civil and criminal penalties for violations of 40 CFR Part 160 • Are subject to periodic audits by EPA inspectors • Are subject to audits by the client companies' own quality control officers, who are trained to find and flag sloppy and fraudulent work
Health consequences of lawn care pesticides are known and real, particularly for Parkinson's Disease	**Incorrect** • The pesticides associated with Parkinson's Disease are not used in lawn care
In humans, these classes of chemicals act as carcinogens, sensitizers and endocrine disrupters, among other effects	**Incorrect** • No turf pesticide marketed in the US in the past decade is known to act as a carcinogen or disruptor in humans
The federal government General Accounting Office found that many pesticides are currently being approved for consumer use by the EPA without receipt and review of data that the manufacturer is required to provide on the safety of the chemicals. Alarmingly, in some cases the manufacturer was given two years to submit studies on the effects of a pesticides, and ten years later no studies had been received or reviewed by the EPA.	**Incomplete Information and Misleading** This statement refers only to <u>conditional registrations.</u> • A pesticide may only be registered conditionally while one or two of the required 75 to 100+ studies are being done, such that: o The database must be largely complete, i.e., almost all of the required product chemistry, environmental chemistry, ecotox, etc. studies must be complete, submitted, and reviewed o Before granting a conditional registration, the EPA must first determine that use of the pesticide would not significantly increase the risk of unreasonable adverse effects on the environment during the time needed to generate the data o A 2010 audit of conditional registrations issued between 2000 and 2010 indicated that pesticide companies (registrants) had indeed submitted required data for 533 of the 544 pesticides (98%) and the EPA had reviewed 523 of the 533 submissions (98%) (GAO, 2013) • "An EPA analysis of conditional registrations in 2012 confirmed that of the products for which the conditional registrations were examined, no conditional registration resulted in unreasonable adverse effects on the environment."
Glyphosate, the active ingredient in Roundup is an endocrine disrupter and carcinogen	**Incorrect** • Glyphosate is not a carcinogen. The carcinogenicity class for glyphosate is "Group E," meaning there is "no evidence of carcinogenic potential." • Glyphosate is not an endocrine disruptor. In 2009, the US EPA directed the manufacturers of glyphosate and 66 other compounds to test the substances for endocrine disruption. This testing was done in 11 Tier 1 (conservative), validated, *in vitro* and *in vivo* assays that evaluated glyphosate's impacts on pubertal development, thyroid function, androgen receptors, estrogen receptors, and steroidogenesis (aromatase activity). Glyphosate demonstrated no adverse effects in the 11 studies. • The EPA is scheduled to issue a comprehensive report on the Tier 1 studies shortly.

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2,4-D is a carcinogen and a major component of Agent Orange	**Incorrect** • 2,4-D is not a carcinogen. Several authoritative meta analyses reached that conclusion. EPA reached this conclusion in 2012 after an exhaustive, transparent, multiyear evaluation that included external peer review. • A Harvard researcher reached this conclusion after a thorough review that included 239 references. • 2,4-D was the only relatively benign component of the Agent Orange mixture that included the persistent and toxic ingredients 2,4,5-T, chlorinated dibenzodioxins, and chlorinated dibenzofurans.
Homeowner pesticide application rates are much higher than the rates used by farmers.	**Incorrect** • Only one published, referenced and quantitative analysis of this issue exists, authored by me in 1995 with the assistance of EPA staff. Homeowner herbicide turf use rates ranked 52, insecticide use ranked 66 and fungicide use ranked 75.

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March 11, 2015

Mr. George Leventhal, President
Montgomery County Council
100 Maryland Avenue
Rockville, MD 20850

Re: Response to testimony
on proposed bill 52-14

Dear President Leventhal:

Thanks to you and Mr. Berliner for the opportunity to testify at the January 15, 2015 hearing on the proposed bill 52-14. As I stated in my oral testimony and my lengthier written comments, I support the IPM and education provisions of the bill (if pesticides are included in the IPM component), and I object to the pesticide use ban based on the list of lists (§33-B4(c)). The basis for my comments is 30 years as a County resident and 39 years as an environmental chemist and risk assessor, with 11 of those years in the US EPA's Office of Pesticides & Toxic Substances.

There was much testimony in support of the bill at both hearings, most of which was thoughtful and clearly stated. The purpose of this letter is to respond to comments made by the bill's advocates that are within my areas of expertise. My responses occur in the approximate order the witnesses appeared at the hearings. My objective is to provide clarity, context, and the most contemporary information available.

My responses are organized as follows:

- comments made at the January 15 hearing;
- comments made at the February 12 hearing;
- comments made by Council President Leventhal regarding missing data;
- comments made by Council Member Elrich regarding the term "safe", glyphosate, and an epidemiology study; and
- some common themes.

JANUARY 15, 2015 HEARING

Rebecca Rehr, Maryland Environmental Health Network
"On the federal level, the Food Quality and Protection Act amended the Federal Insecticide, Fungicide, and Rodenticide Act and the Federal Food, Drug, and Cosmetic Act to specifically require consideration of

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an extra 10-fold safety factor to further protect infants and children. However, we found that the Office of Pesticides at the EPA does not uniformly or consistently apply this extra safety factor in their risk assessment processes that dictate the regulation of the use and labeling of pesticides. All too often, this 10-fold safety factor is ignored."

Response. It is incorrect to state "All too often, this 10-fold safety factor is ignored." It is never ignored. For example, US EPA (2002) is a 70+ page document that carefully explains when the FQPA safety factor of 10X is to be applied, and when something less - - typically 3X or 1X - - is scientifically justified.

The first sentence in Ms. Rehr's statement is correct, but a key provision of the statute is missing, i.e., the fact that the US EPA may use a safety factor (or uncertainty factor) less than 10X when it is scientifically justified. The key text from the statute is underlined for emphasis: *"In the case of threshold effects, for purposes of clause (ii)(I) an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure shall be applied for infants and children to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and toxicity to infants and children. Notwithstanding such requirement for an additional margin of safety, the Administrator may use a different margin of safety for the pesticide chemical residue only if, on the basis of reliable data, such margin will be safe for infants and children."*

Finally, it is important to note that this FQPA safety factor is applied in addition to an existing uncertainty factor of 10X to 1000X (typically 100X).

Paxson Barker, Ph.D., MS, RN, Maryland Nurses Association and Maryland Pesticide Network
"Of the most commonly used lawn care pesticides by homeowners and professional turf and landscape companies, 18 disrupt the endocrine hormone system (reproductive malformations), 17 are linked to cancer, 11 are linked to birth defects, 19 to reproductive effects, 24 to liver or kidney damage, and 14 to neurotoxicity (autism and learning disabilities (EPA, 1990))."

Response. There is much confusion about Dr. Barker's reference, "EPA, 1990". The reference she lists at the end of her testimony is *"U.S. Environmental Protection Agency (EPA). (1990. Lawn Care Pesticides: Risks Remain Uncertain While Prohibited Safety Claims Continue," National Pesticide Survey. [sic])"*. She may have combined two references and possibly failed to list the actual reference, a table on the website of Beyond Pesticides. I was the co-director of the National Pesticide Survey - - which is part of her citation - - until I departed the EPA in December, 1986, and I peer reviewed the National Pesticide Survey report when it was issued ca. 1990. I recall no such statements in any of the relevant documents. Therefore she may be referring to a report by the General Accounting Office (GAO) in 1990 with the same title. However, we could find no statements in that report that support – or even come close to support – the numbers in her statement above.

Further, the statement is largely irrelevant because the 34 pesticides listed in the GAO report - - if it is the correct report - - were those used in lawn care in 1990 and earlier, a quarter century ago. For example, four organophosphate insecticides are included that have not been applied to lawns for at least 5-10 years.

It is most likely that she obtained the information from none of the references listed above; rather, the source is likely a table called "Health Effects of 30 Commonly Used Lawn Pesticides" on the Beyond Pesticides website (http://www.beyondpesticides.org/lawn/factsheets/30health.pdf), which contains information that matches the numbers in her testimony exactly. Unfortunately, some of the

information in that table is either misleading or incorrect. An example of the former is the checked box of pendimethalin for reproductive effects, despite the fact that it demonstrated no reproductive nor developmental toxicity at doses below the large doses in which parental toxicity was observed, i.e., roughly equivalent to a human consuming a daily diet that includes 0.5% of the pure pesticide. This exceeds human exposures by orders of magnitude. An example of the latter (incorrect information) is the fact that many of the "commonly used lawn pesticides" are not even used on lawns. For example, EPA eliminated use of dichlorvos and malathion on lawns in 2006 and 2008, respectively.

Finally, there is a question about relevance in the context of toxicity/hazard vs. risk (see pp. 4-5 of my January 15 written comments). In what way were these pesticides - - those that are still used on turf in 2015 - - "linked" to these effects? Were they effects that only appeared in laboratory rodents at doses such as 1000 mg/kg bw-day, i.e., roughly equivalent to an adult eating 2½ ounces of pure pesticide active ingredient? Or was the link based on one of the epidemiology studies I addressed on pp. 6-7 and Appendix D of my January 15 submission (selected Appendix D slides resubmitted as Appendix A to this letter)?

"Glyphosate (Roundup) specifically has also been linked to Parkinson's disease, celiac disease, and gluten intolerance (Samsel & Seneff, 2013)".

Response. This reference is addressed in "Some Common Themes" near the end of this document.

Alan Cohen, Biological Pest Management

"1) EPA's approval of a pesticide does not mean a pesticide is safe. FIFRA, the Federal Insecticides, Fungicides and Rodenticides Act, is not a health-based statute. It is a Risk/Benefit statute."

Response. That statement had been true regarding human health effects prior to 1996. But the passage of the Food Quality Protection Act (FQPA) that year had the net effect of ensuring that registration decisions for pesticides with food uses are to be held to the higher standard described on pp. 2-3 of my January 15 submission, i.e., "a reasonable certainty of no harm", with a focus on children; an evaluation of aggregate risks from aggregate exposures (residues in food + drinking water + post-application contact with treated surfaces [e.g., turf]); and an additional safety factor for pre-natal and post-natal exposures. Mr. Cohen cites 2,4-D as a risk-benefit example. However, since 2,4-D has food uses, as well as turf uses, EPA must consider potential risks to children without regard to benefits. [NOTE: risk-benefit balancing is still allowed for ecological effects, unless threatened or endangered (T/E) species are involved. The EPA is extremely conservative in its pesticide risk assessments for T/E species.]

"2) In EPA toxicology reviews, only one chemical is examined at a time."

Response. This statement is mostly correct, but it is misleading. EPA scientists are required by the FQPA to consider, and combine, as appropriate, the cumulative risks of multiple pesticides with common mechanisms of toxicity. Two classes of insecticides that interact with the nervous system are examples of this approach. However, most substances do not share a common mechanism of biological interactions.

"3) Many pesticides are given conditional registrations with data gaps that are never filled. These are tests that somehow never get filed with the agency, nevertheless, the pesticide is sold for years with incomplete toxicology reviews."

Response. This is 98% false. See our response to Council President Leventhal's statement below.

"4) Lawn-care pesticides are tested for chronic human and animal health effects ONLY IF they are also registered for food uses."

Response. This is mostly correct. It is important to note that EPA scientists may require such studies for non-food use pesticides if concerns are raised in the subchronic or shorter term studies. Further, it should be noted that almost all of the most commonly used turfgrass pesticides also have food uses. (We recently found that 37 of 39 common turf pesticides are also used on food crops.) Therefore almost all of the lawn care pesticides are registered based, in part, on chronic animal feeding studies.

"5) So-called "inert" ingredients may be as toxic as the active pesticide chemical they are mixed with. But products don't have be be [sic] tested as a complete package. Some inerts are suspect carcinogens, and others have been linked to birth defects, liver damage and CNS disorders. Many of the so-called "inerts" in pesticide formulations are not inert at all, but just were not claimed to have pesticidal properties. One such inert that still concerns many today is a widely used chemical called PBO, or Piperonyl Butoxide, which slows down the degradation of pyrethrins, the most common active ingredients in general use pesticides and consumer aerosols today. It is common to see PBO at very high levels compared to a small amount of the active pyrethrin ingredient. And this PBO is classified a class C carcinogen by EPA."

Response. This is somewhat misleading. The former inert policy established four categories or 'lists' in 1987, based on the toxicological concern for the inert ingredient. This policy prioritized the inert ingredient evaluation process. Evaluations of the inert ingredients on these lists at the time the Food Quality Protection Act was passed in 1996 concluded in 2006 (Tony Britten, US EPA Inert Ingredient Assessment Branch, 1/12/2012, Personal communication), i.e., concluding with a finding that all food use inert ingredient tolerances and exemptions are considered safe (www.epa.gov/opprd001/inerts/). The list category policy developed in 1987 is no longer used by EPA to prioritize inert ingredients.

The current process for submitting a food or non-food use petition for establishment of an inert ingredient includes providing a long list of data requirements (US EPA, 2013a & b). These requirements include: physical and chemical properties, acute and chronic toxicity data, reproduction and developmental data, mutagenicity data, neurotoxicity data, endocrine data, immunotoxicity data, and carcinogenicity data that will "provide a scientific explanation why (the inert ingredient) would not be carcinogenic". In addition, information regarding human and animal metabolism, exposure, environmental fate and effects, as well as rationale indicating ecotoxicity is not a concern need to be provided. The applicant also needs to summarize how collectively this information indicates the proposed use of the chemical would be considered safe for the environment and human health.

Finally, regarding Mr. Cohen's statement about PBO, PBO is only considered by the US EPA to be an active ingredient in pesticide formulations, so discussion of its toxicity as an inert ingredient in pesticides is inaccurate (personal communication with Kerry Leifer of the Chemistry, Inerts and Toxciology Assessment Branch Registration Division, February 10, 2015).

"6) The EPA does no testing of its own but relies on commercial lab tests paid for by chemical companies. Fraud in testing is not a criteria [sic] for bouncing a chemical out of registration, but fraud occurred in the case of Industrial BioTest Labs. The amount of data generated for testing makes it difficult to assure the public that fraud will not occur data production."

Response. The first part of this statement is technically correct, but extremely misleading. ***Mr. Cohen cites a case of fraud that occurred more than 30 years ago, when I worked in the Office of Pesticide Programs.*** (Note: the lab managers were prosecuted in criminal court.) He also fails to inform the reader that these commercial labs:

- are required to follow EPA's prescribed testing guidelines;
- are required to follow the extensive set of EPA's Good Laboratory Practices (GLPs), pursuant to Title 40 of the Code of Federal Regulations (CFR) Part 160;
- are subject to civil and criminal penalties for violations of 40 CFR Part 160 (e.g., IBT lab mentioned by Mr. Cohen, see above);
- are subject to periodic audits by EPA inspectors (I have witnessed such multi-day audits); and
- are subject to audits by the client companies' own quality control officers, who are trained to find and flag sloppy and fraudulent work (My company has been audited by a pesticide-company-hired GLP auditor).

FEBRUARY 12, 2015 HEARING

Thomas Cummings

Dr. Cummings is a physician. He did not claim to have any expertise in pesticide toxicology or risk assessment. He stated the following:

"The health consequences of these lawn care pesticides are known and they are real. Pesticide exposure significantly increases the risk of developing Parkinson's Disease."

Response. As a general response, the databases and potential risks of all turf pesticides currently on the market have been thoroughly evaluated by EPA scientists. An overwhelming majority of these pesticides have had to meet the extra strict definition of "safe" pursuant to the Food Quality Protection Act of 1996 (see the comments relevant to Council Member Elrich below). A subchronic (90-day) neurotoxicity study (required; 40 CFR §158.500) and a developmental neurotoxicity study (conditionally required) can yield observations of neurobehavioral effects, if they occur.

More specifically, the science to support his statement is not strong, and it mostly does not exist for lawn pesticides (Li et al., 2005). Parkinson's Disease (PD) has a complex etiology (cause of the disease) that is not well characterized. There is clearly a genetic component, but it may only be a factor in a minority of cases (http://www.webmd.com/parkinsons-disease/guide/parkinsons-causes; Moor et al., 2005). Head trauma often appears to be a factor or co-factor (Goldman et al., 2006), and many scientists believe that multiple 'hits' to the central nervous system are required to cause PD (e.g., Cory-Slechta et al., 2005).

A pesticide component has also been reported, but the pesticides are not lawn/turf products. Richardson et al. (2009) reported that the presence, in serum, of the transformation product (β-HCH) of the long-banned insecticide lindane was associated with PD, although the number of cases was relatively low, and the spread of the 95% confidence interval was broad. Cory-Slechta (2005) found that a combination of paraquat and maneb[1] - - neither of which are applied to turf - - elicited PD-like symptoms when injected into the abdominal cavity of mice at a dose (1.3 mg/kg) that was orders of magnitude higher than would be received by humans close encountering post-use exposure. Finally, van

[1] It is likely that some or all of maneb's impact on the nervous system of rodents - - albeit at high doses relative to environmental exposures - - is due to the manganese atom in the molecule.

der Mark et al. (2012) reviewed the literature and reported associations between PD and pesticide use. However:

- most of the studies relied on gross exposure estimates *post facto* which are subject to memory bias; and
- the odds ratio only exceeded 1.0 by a very small amount (1.18; 0.86-1.63) for the three studies that only had non-occupational exposure to any pesticides.

Ryan McCalister

Dr. McCalister is a biophysics professor. He did not claim to have any expertise in pesticide toxicology or risk assessment. He made the following statements:

"In humans, these classes of chemicals act as carcinogens, sensitizers, and endocrine disruptors, among other effects."

Response. This is an amazingly broad statement, particularly for someone with scientific training. It is also not true; this may be the reason he provides no support for his statements. ***I am aware of no turf pesticide that has been marketed in the US within the last decade that is known to act as a carcinogen or an endocrine disruptor in humans.*** [I am not qualified to comment on the sensitizer issue.]

The phrase "these classes of chemicals" encompasses a highly diverse group of chemical classes that includes natural products and natural product mimics, in addition to conventional pesticide chemistry. See, for example, the various chemical structures in Figure 1. It is very difficult to believe that someone with a technical background would indict such molecules with such a broad brush.

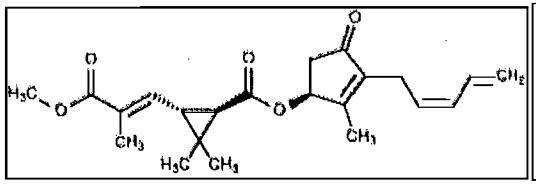
Slight differences in the molecules depicted in Figure 1 can significantly alter their physical/chemical properties and their reactivities. Such a broad indictment of such diverse structures is analogous to stating that all motor vehicles are bad.

"Further, there is no evidence that there are any harmless doses of these chemicals."

Response. This statement is contradicted by the universally accepted threshold concept, whereby most toxic effects do not occur until a particular dose is attained inside the organism. This dose, expressed as RfD (reference dose; mg pesticide/kg body wt-day), is derived as follows (US EPA, 2002):

- The no-observed-adverse-effect level (NOAEL) is the highest dose for which the test animals do not exhibit the toxic effect. It is determined either directly from observations of the test animals, or by extrapolation from the lowest-observed-adverse-effect level (LOAEL). A relatively new technique called the benchmark dose (BMD) is being used increasingly in place of the classical NOAEL.
- Then a series of safety/uncertainty factors are applied, always in the conservative (decreasing) direction, as follows for chronic effects:
  - intraspecies uncertainty factor (UF; typically 10X);
  - interspecies UF (also typically 10X);
  - UF to extrapolate from subchronic results to a chronic endpoint, if the chronic study is either missing or not acceptable;
  - database UF, when there is the absence of key data in the database for a given chemical; and
  - the FQPA safety factor of 10X for sensitivity to infants and children. (This factor can be lowered if such margin ". . . will be safe for infants and children" and based on "reliable data" (§408(b)(2)(c)i; see also response to R. Rehr above).

Figure 1. Structures of Natural Products and Synthetic Pesticides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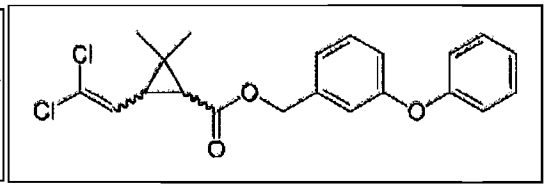
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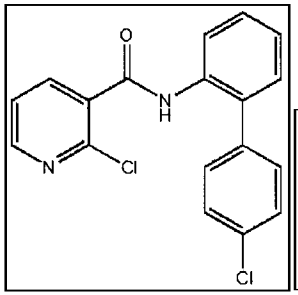
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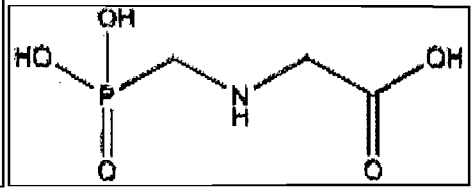
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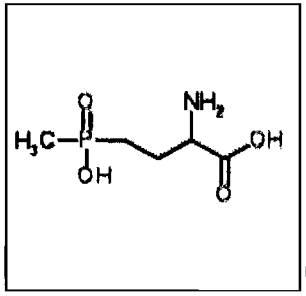
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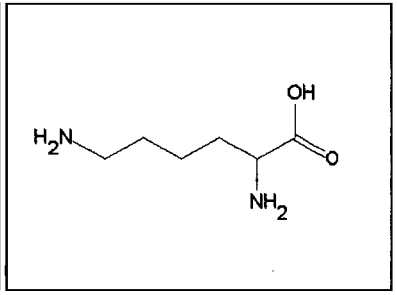
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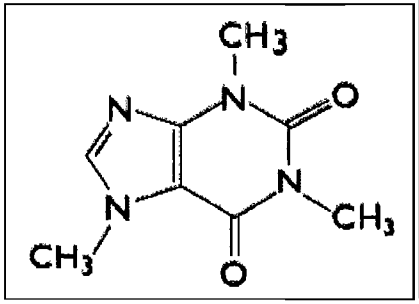
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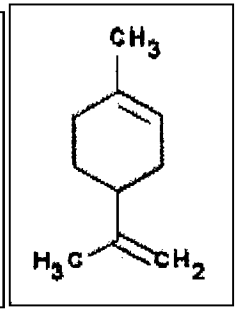
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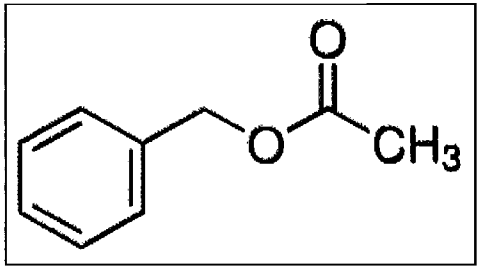
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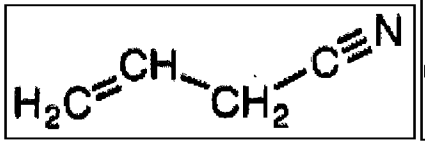
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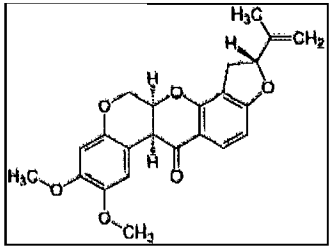
(11)

(1) = one form of the natural insecticide pyrethrin
(2) = permethrin, a synthetic pyrethroid, based on pyrethrin
(3) = the "reduced risk" fungicide boscalid
(4) = the herbicide glyphosate (see text)
(5) = the herbicide glufosinate
(6) = the natural amino acid lysine
(7) = the natural product caffeine
(8) = the natural pesticide limonene, which is a carcinogen present in black pepper, mango, and orange juice (Ames et al., 1990)
(9) = the natural pesticide benzyl acetate
(10) = the natural insecticide allyl cyanide
(11) = the naturally occurring piscicide rotenone

Take-home messages:

- pesticide molecules are highly diverse, making it difficult to make general statements that apply to all of them;
- it can be difficult to distinguish between natural and synthetic chemical structures.

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Catherine Cummings
Ms. Cummings stated:

"There are no studies showing these pesticides are safe."

Response. See the last response to Dr. McCalister above.

"Childhood and adult cancers, asthma, hormone disruption, lower IQ, birth defects, reproductive disorders, autism, Parkinson's, neurological diseases are linked to lawn pesticide exposure."

Response. The US EPA's Office of Pesticide Programs has approximately 650 employees and approximately half of those review the data from 75 to greater than 100 studies that are required for pre-sales approval. Among the endpoints that are carefully considered - - based on results from 15-27 toxicology studies - - are cancer, reproductive effects, and developmental effects.

Ms. Cummings stated that many ". . . diseases are linked to lawn pesticide exposure." What is meant by "linked" in this context? ***I am aware of no such strong relationships that even come close to cause-and-effect.*** I addressed the problems in establishing links in epidemiology studies in my January 15 testimony submission package (pp. 6-7 of the 8-page document, plus Appendix D of that package; selected slides from it are attached as Appendix A to this document). This is particularly a problem when extremely crude measures of exposure are used to establish an association, e.g., 'were pesticides ever applied to the property?', which typically occurs in the most common type of pesticide epidemiology study, case control studies.

An example of how easy it is to reach weakly supported conclusions in retrospective epidemiology studies, e.g., case control is the autism spectrum disorder association described by Shelton et al. (2014) in an agricultural region of California. Our concerns are stated in the attached letter (Appendix B), which was recently accepted by the journal Environmental Health Perspectives for publication (Burns et al., in press). (Note: our letter is lacking in some detail due to the space limitation imposed by the journal.)

COMMENTS FROM COUNCIL PRESIDENT LEVENTHAL REGARDING MISSING DATA

Advocates of the bill have been quoted as stating and/or implying that there are conventional lawn care chemicals on the market for which there are no supporting data. ***This is false***[2], as explained below.

For example, Council President Leventhal recently stated the following in a letter to Gigi Schwab, Legislative Aide to State Delegate Ben Barnes (12/17/14), and the same concept has been presented in other communications.

> "Many people assume that because these products are reviewed by the EPA and sold on store shelves that they must be safe, but the federal government's Government Accountability Office (GAO) has found that many pesticides are currently being

[2] There are possible, minor, exceptions among the FIFRA §25(b) short list of non-conventional, natural, "organic" active ingredients that are exempt from data requirements due to inherent safety. Examples are white pepper, mint oil, and rotten eggs.

approved for consumer use by the EPA without receipt and review of data that the manufacturer is required to provide on the safety of the chemicals. Alarmingly, in some cases the manufacturer was given two years to submit studies on the effects of a pesticides, and ten years later no studies had been received or reviewed by the EPA. In the absence of reform on the federal level, I believe it would be irresponsible not to act now on the local level."

This is a misleading statement at two levels. First, it is written in such a way that people may assume that ". . . ten years later no studies had been received or reviewed by the EPA" for some turf pesticides. The regulatory requirements set forth in Title 40 Code of Federal Regulations Part 158 (40 CFR Part 158) ***clearly document the minimal base set of approximately 75 studies, with the possibility of more than 100 studies, that are required to register a conventional turf pesticide*** prior to introduction into commerce. At least 15 of these studies are in toxicology; the number 15 can increase to 27 if the pesticide is also intended for use on food crops and/or if toxicologic concerns are raised in the initial base set of tests. When these study results are integrated into a series of conservative risk assessments, then EPA's pesticide scientists must find that there is "a reasonable certainty of no harm" to humans[3], with a focus on children, pursuant to the 1996 Food Quality Protection Act.

Further, the capability to review these studies by EPA's Office of Pesticide Programs (OPP) is strong. ***OPP has approximately 650 employees and approximately half of those are scientists.*** An overwhelming majority of these scientists have graduate degrees, and many of the regulatory staff have science degrees. The studies they review are generated by audited contract labs that are required to adhere to FIFRA Good Laboratory Practices (40 CFR Part 160).

Second, a better explanation is needed if readers understand that this language in the 12/17/14 letter actually refers to conditional registrations. Conditional registrations are allowed under the pesticide law, FIFRA (Federal Insecticide, Fungicide, and Rodenticide Act), §3(c)7. Someone who relies on Mr. Leventhal's statement above as a knowledge base for conditional registrations would not be aware of the following truths, that a pesticide may only be registered conditionally while one or two of the 75 to 100+ studies are being done, such that:

- the database must be largely complete, i.e., almost all of the required product chemistry, environmental chemistry, ecotox, etc. studies must be complete, submitted, and reviewed;
- before granting a conditional registration, the EPA must first determine that use of the pesticide would not significantly increase the risk of unreasonable adverse effects on the environment during the time needed to generate the data;
- a 2010 audit of conditional registrations issued between 2000 and 2010 indicated that pesticide companies (registrants) had indeed submitted required data for 533 of the 544 pesticides (98%) and the EPA had reviewed 523 of the 533 submissions (98%) (GAO, 2013); and
- "An EPA analysis of conditional registrations in 2012 confirmed that of the products for which the conditional registrations were examined, no conditional registration resulted in unreasonable adverse effects on the environment. That is, upon receipt and review of data submitted as a condition of registration, EPA's original safety determination was confirmed by

[3] The standard is "unreasonable risk" if the turfgrass pesticide has no food uses, and "a reasonable certainty of no harm" if there are food uses. But very few of the more commonly used lawn and athletic field pesticides have no food uses. For example, I recently found crop tolerances (allowable maximum limits for specific pesticide) for 37 of 39 commonly used turf pesticides.

the new information." (http://www.epa.gov/oppfead1/cb/csb_page/updates/2014/conditional-pest-reg.html)

Thus statements regarding pesticide regulatory risk assessment made in support of the bill have misled the public, and they have failed to describe the strong, conservative, comprehensive OPP/EPA assessment process and regulatory program.

COMMENTS FROM COUNCIL MEMBER ELRICH REGARDING THE WORD "SAFE", GLYPHOSATE AND AN EPIDEMIOLOGY STUDY

Council Member Elrich had informal conversations with several attendees immediately following the February 12 hearing. Three issues were discussed: the use of the word "safe" when discussing pesticides, the effect of the herbicide glyphosate on the human microbiome, and the results of a longitudinal/cohort epidemiology study.

Use of the Word "Safe". Council Member Elrich stated that the EPA does not use the word "safe" in describing pesticides. I supported his position when I told him about my tenure on the Pesticide Misuse Review Committee during my early days at the US EPA. We would recommend a civil penalty citation if a pesticide registrant were to claim a pesticide to be "safe" in a statement on the product's labeling. ***This is because we were concerned that people would not be careful to follow the label and limit their exposures.***

Both of us were technically correct, particularly for the time prior to passage of the Food Quality Protection Act (FQPA) in 1996, but both of us failed to acknowledge a key FQPA requirement. The FQPA does require that the EPA conclude that the aggregate of all exposures to a pesticide with food uses be safe, as follows:

> §408(b)(2)(A)(ii) and §408(c)(2)(A)(ii): DETERMINATION OF SAFETY.—As used in this section, the term 'safe', with respect to a tolerance for a pesticide chemical residue, means that the Administrator has determined that there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.

Thus for pesticides that also have food uses - - which is most of them[4] - - the EPA must determine that use of the pesticide is "safe" under the 1996 FQPA standard. The herbicide 2,4-D is an example of a pesticide covered by this provision (see below, "Some Common Themes").

Impact of Glyphosate on the Human Microbiome. This is addressed in the "Some Common Themes" section below.

Update of a Longitudinal/Cohort Study. In Appendix D of my January 15 testimony packet (selected slides of which are also Appendix A of this document), I briefly summarized the results of a cohort study that was based on the long term Agricultural Health Study (slide 11; Flower et al., 2004). No increased risk of childhood cancer associated with mothers who ever mixed or applied pesticides was observed.

[4] We recently researched whether crop tolerances have been established for 39 turf pesticides in 40 CFR Part 180, which denotes food uses. Only two pesticides did not have crop tolerances - - the herbicides prodiamine and MCPP.

Council Member Elrich told us there was an update to the Ag Health Study that reported an increase in prostate cancer risk associated with pesticides. We believe we found the source, http://aghealth.nih.gov/news/2014.html#p2, Koutros et al., 2013a and b, Barry et al (2012), and Koutros et al. (2010). This study focused on agricultural pesticide applicators, not children. The authors stated that the pesticide applicators had a lower overall cancer incidence compared with the general population. However, frequent users of aldrin, fonofos, malathion, and terbufos were more likely to develop aggressive prostate cancer. The first two insecticides are no longer registered for use in the US, and the latter two are not used on turf.

SOME COMMON THEMES

Glyphosate, the Active Ingredient in Roundup, is Not an Endocrine Disruptor Nor a Carcinogen

The herbicides glyphosate and 2,4-D (see below) were common targets of the bill's supporters throughout both hearings. It is important to note that glyphosate is only applied as a hand-directed spot treatment to turf. Broadcast applications would results in large areas of dead grass.

Glyphosate is Not a Carcinogen. The carcinogenicity database for glyphosate is complete and valid. Its carcinogenicity class is "Group E", "There is no evidence of carcinogenic potential" (Federal Register, 9/27/2000, vol. 65(188), 57957-57966; US EPA, 2012; and European Union/BfR [www.bfr.bund.de/en/the_bfr_has_finalised_its_draft_report_for_the_re_evaluation_of_glyphosate-188632.html] who considered the Schinasi et al. 2014 paper).

Glyphosate is Not an Endocrine Disruptor. In 2009, the US EPA directed the manufacturers of glyphosate and 66 other compounds to test the substances for endocrine disruption. This testing was done in 11 Tier 1 (conservative), validated, *in vitro* and *in vivo* assays that evaluated glyphosate's impacts on pubertal development, thyroid function, androgen receptors, estrogen receptors, and steroidogenesis (aromatase activity). ***Glyphosate demonstrated no adverse effects in the 11 studies (Webb et al., 2013; Bailey et al., 2013).*** The EPA is scheduled to issue a comprehensive report on the Tier 1 studies shortly.

The Microbiome Work Cited by Samsel and Seneff (2013a&b) is Problematic. It is highly unusual for the scientific community to witness work such as this. The authors use speculation and extrapolations liberally to imply that glyphosate is responsible for "most of the diseases and conditions associated with a western diet, which include gastrointestinal disorders, obesity, diabetes, heart disease, depression, autism, infertility, cancer and Alzheimer's disease" (2013a), as well as celiac sprue and gluten intolerance (2013). Whew.

These papers did not present any new data. But they did do the following:

- They provided more speculation, by far, than I have ever seen in a journal article in my almost 40 years as a scientist. Phrases such as "It is plausible that glyphosate could serve as a . . .", "It is conceivable that . . .", "hence, vitamin D_3 deficiency (which could be caused by glyphosate's impairment of liver CYP enzymes) . . ." (2013a), and "Thus, it is possible that glyphosate similarly impairs cobalamin function . . ." (2013b). This is just a small fraction of the speculation that populates these papers.

- In their zeal to indict glyphosate for many of the ills of mankind, the authors often chose studies to cite that yielded adverse effects at concentrations orders of magnitude larger than expected in the environment. For example, they imply that human placenta cells bathed in a 0.02% solution of glyphosate are relevant, when, in fact, it exceeds environmentally relevant concentrations by at least 4 orders of magnitude (10,000X; Richard et al., 2005, cited in Samsel and Seneff, 2013a). They committed the same error with at least two other references in that same discussion about the cytochrome P enzyme systems. ***This is not a minor point: conclusions based on irrelevant concentrations occur in the critical part of their microbiome hypothesis discussion, section 5 of Samsel and Seneff (2013a), which forms much of the basis for the two papers.***
- The authors (2013a) argue that glyphosate suppresses the synthesis of the essential amino acid typtophan in the human intestinal microbiome, which leads to obesity, etc. ***This argument has a fatal flaw: the human microbiome does not provide essential amino acids; that is why they are "essential" in the diet.***
- If such a series of terrible illnesses actually occur, they would have been observed in the multiple mammalian feeding studies.
- Samsel and Seneff (2013b) base part of their attack on glyphosate on the series of graphs that correlate glyphosate use on grains with several different adverse effects. ***Such an association does not demonstrate a cause and effect.*** See for example, figures 2a-c and 3. All four correlations are highly significant. But we think it would be irresponsible, for example, to credit increased use of glyphosate with the increasing number of websites (Figure 2c), or blame autism on the increase in organic food consumption (Figure 3).
- The authors have curious credentials. One of them is an electrical engineer, it is not clear that the other author has a relevant background either, and the engineer has been part of the pseudo science crowd that campaigns against childhood vaccinations (e.g., Seneff, 2011).

2,4-D is Not a Carcinogen

Several people stated that 2,4-D was a component of the Agent Orange defoliating agent used in Viet Nam. This is true, but misleading. It is more accurate to state that 2,4-D was a benign component of a 50:50 mixture with 2,4,5-T that was contaminated with chlorinated dibenzodioxins. (The use of 2,4,5-T in the US was cancelled in the 1970s.)

Multiple authoritative reviews have concluded that 2,4-D is not carcinogenic. (Yet speakers frequently stated that 2,4-D causes cancer.) The US EPA reached this conclusion in 2012 after an exhaustive, transparent, multiyear evaluation that included external peer review (US EPA, 2012). In addition, a Harvard researcher reached this conclusion after a thorough review that included 239 references (von Stackelberg, 2013). More detail can be found in my January 15 testimony packet; specifically, p. 7 of my eight page comments document, and slides 12-14 of Attachment D of that packet (selected slides of which are also Appendix A of this report).

Relative Pesticide Application Rates: Homeowner Turf Rates Do Not Tend to be High

At least two speakers commented that pesticide application rates are much higher than the rates used by farmers. This assumption became well publicized in the early 1990s when the New York State Attorney General's office was targeting golf courses. The AG and his staff frequently cited a Cornell professor (Pimentel) when they said pesticides are applied to golf course turf at 4X – 7X the rates used in agriculture (Abrams et al., 1991). Many people have extrapolated this conclusion to home lawns.

I am aware of only one published, referenced, quantitative analysis of this issue, which I wrote with the assistance of US EPA staff (Appendix C; Cohen, 1995). Obviously, this analysis is relatively old, but I believe its qualitative ranking conclusions are still valid, as follows: homeowner turf use rates ranked 52 in herbicide use among 90 crops, 66 in insecticide use among 88 crops ranked, and 66 in fungicide use among 75 crops ranked. See the full table in Appendix C and excerpts from the full table below.

Figures 2a-c. Correlations of Glyphosate Use on Wheat with Number of Websites, College Enrollment, and World Population

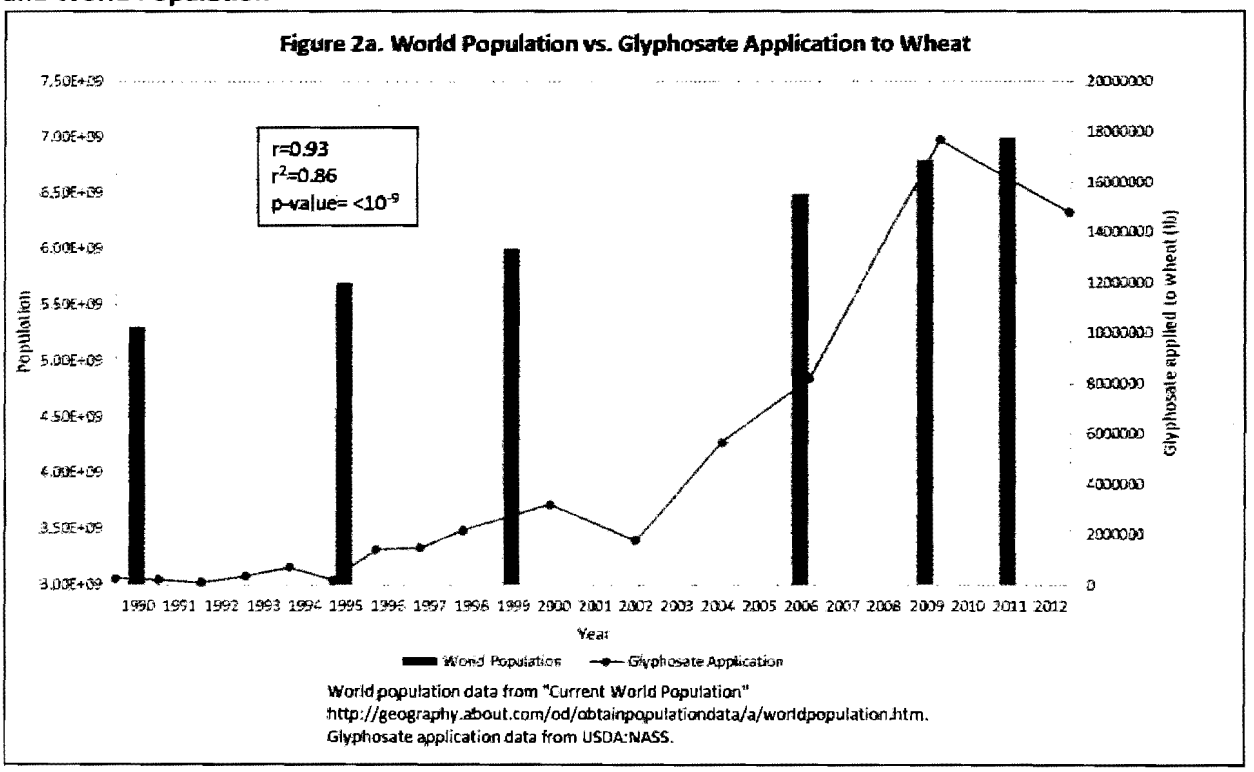
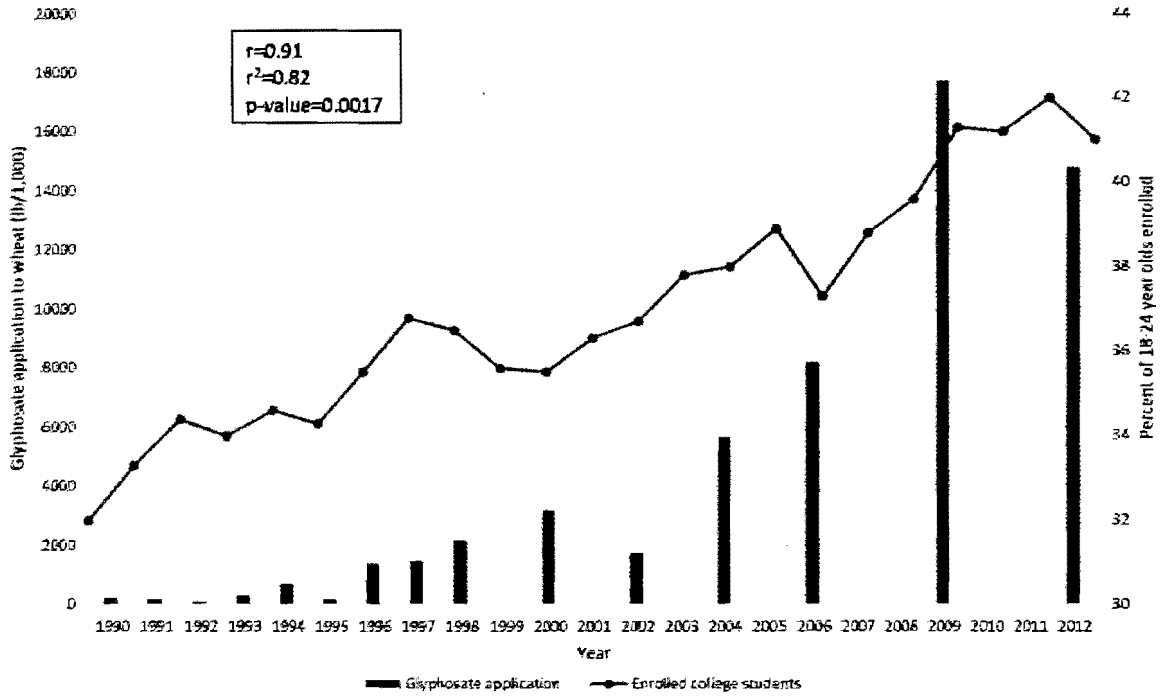
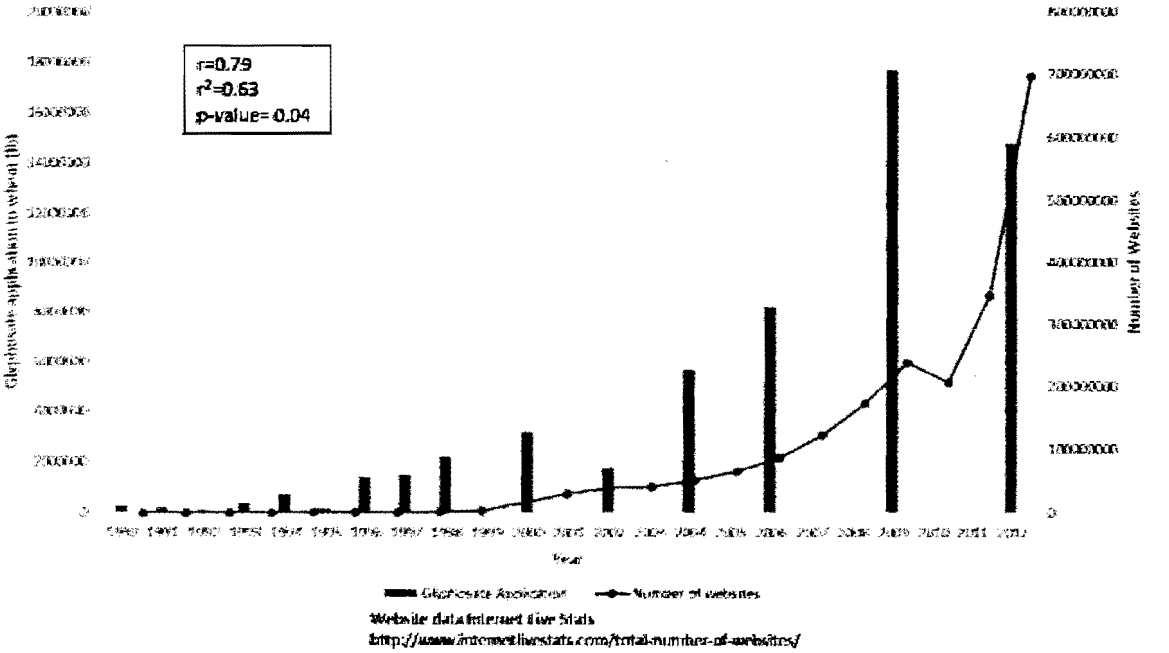


Figure 2b. Enrollment in College vs. Glyphosate Application to Wheat



Enrollment data from the National Center for Education Statistics
http://nces.ed.gov/programs/digest/d13/tables/dt13_302.60.asp (2013).
Glyphosate application data from USDA:NASS.

Figure 2c. Number of Websites vs. Glyphosate Application to Wheat



Website data Internet live Stats
http://www.internetlivestats.com/total-number-of-websites/
Glyphosate application data from USDA:NASS

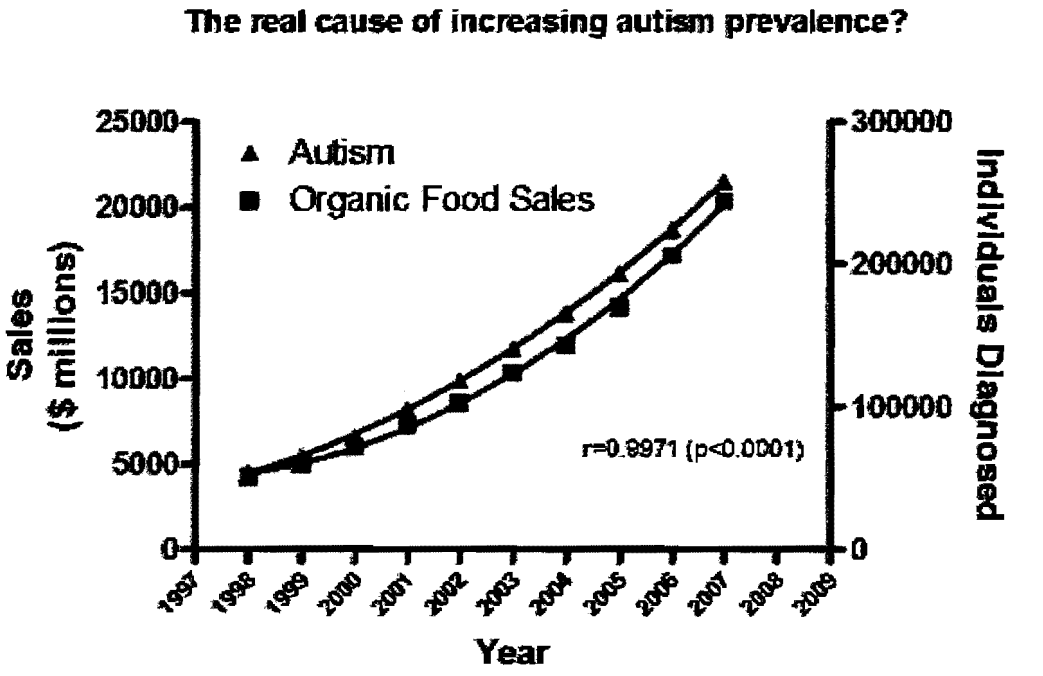
References for Figures 2a-c

2a: Rosenberg, M. Current World Population. Retrieved from http://geography.about.com/od/obtainpopulationdata/a/worldpopulation.htm.

2b: National Center for Education Statistics. 2013. Table 302.60. Percentage of 18- to 21-year-olds enrolled in degree-granting institutions, by level of institution and sex and race/ethnicity of student: 1967 through 2012. Retrieved from nces.ed.gov/programs/digest/d13/tables/dt13_302.60.asp.

2c: Internet live stats. Total number of websites. Retrieved from http://www.internetlivestats.com/total-number-of-websites/.

Figure 3. Correlation of Organic Food Sales with Autism Diagnoses*



* Slide credit: C. Thorpe, CropLife America, Washington DC.

Pesticide Use for Various Crops on a Per-Acre Basis*

Crop/Site	Acres	Pounds of Active Ingredients/Acre (Rank)		
		Herbicides (90 crops ranked)	Insecticides (88 crops ranked)	Fungicides (75 crops ranked)
Onions	151,676	6.32 (1)	1.41 (48)	6.56 (22)
Pears	72,226	1.57 (39)	77.68 (1)	13.42 (11)
Grapes	764,137	1.42 (44)	6.51 (12)	61.92 (1)
Homeowner Turf	20,900,000	1.20 (52)	0.30 (66)	0.03 (66)

*Table adapted from Cohen (1995). The numbers in this table have likely changed since 1995. However, the relative ranks have probably not changed significantly.

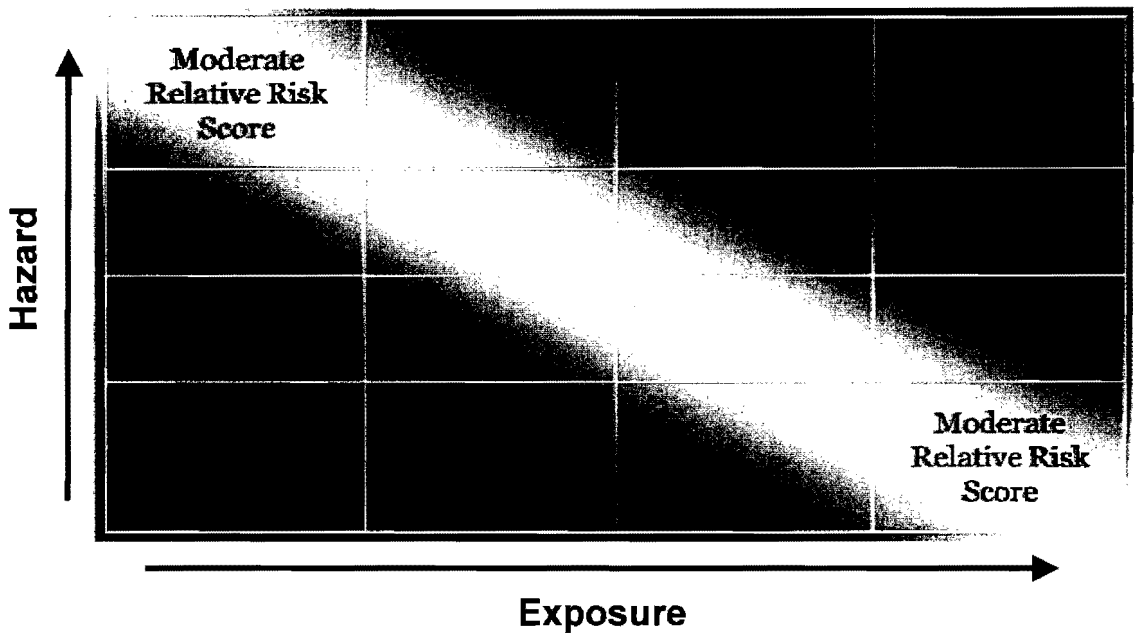
Thus home lawn pesticide application rates can be higher or lower than agricultural rates, depending on the crop, but they were lower than most of the ranked crops in this quantitative analysis.

The Relevance of Dose/Exposure: Cancers in Rodent Studies Can be Induced by High Doses

A small minority of turf pesticides are classified as possible or probable human carcinogens based on rodent studies. The fact that the EPA does not take these chemicals off the market is due to a lack of risk, which is due to minimal exposure to non workers.[5] In other words, both significant toxicity (hazard) and significant exposure are required to cause a risky condition. This is illustrated in Figure 4.

Figure 4. Risk as a Function of Exposure and Hazard (Toxicity)

[Copied from Menzie in Cohen et al., 2014]



There is an additional mitigating factor that is rarely discussed - - the fact that the methods used in these rodent studies may be artificially increasing the tumor incidence in the animals. Specifically, there is evidence that the unrealistically high doses administered to the rodents cause mitogenesis - - the induction of cell division - - which increases the probability of the creation of cancerous cells (Ames and Gold, 1991; Ames et al., 1990).

Finally, it is worth noting that there is a very large number of substances in foods and drugs that have tested positive for carcinogenicity in these rodent feeding studies (e.g., Gold et al., 1992). The authors considered risk, not just toxicity/hazard (see Figure 3 above) in their evaluation. They concluded - - based on the rodent feeding studies - - that the synthetic pesticides or environmental contaminants risks generally ranked lower than risks of the naturally occurring carcinogens.

[5] The *de minimus* cancer risk level is typically the upper 95% confidence level of the dose that yields a 1 in a million ($1x10^{-6}$) risk probability. This criterion is typically used in assessments of the general population and homeowners (e.g., lawn care).

I appreciate your patience in reading this detailed response to comments; however, it is not comprehensive in that there were additional comments made to which I could have responded. I am always available to your staff, and I plan to be available on March 16 on the second expert panel if you have questions that require an answer from within my areas of expertise.

Sincerely,

Stuart Z. Cohen, Ph.D., CGWP
President

cc: Councilmember Roger Berliner, Committee Chair

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Appendix A. Selected Epidemiology Slides Included in the January 15, 2015 Written Comments by Stuart Cohen [Slides 1,2 and 23-26; full presentation may be found at www.environmentalandturf.com]

EPIDEMIOLOGY STUDIES OF THE ADVERSE EFFECTS OF PESTICIDES IN THE CONTEXT OF THE PROPOSED MONTGOMERY COUNTY PESTICIDE BAN

by
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before the
Maryland Turfgrass Council's
Annual Turfgrass Conference

January 6, 2015
Revised January 8, 2015

PRESENTATION OUTLINE

I. Summary of Bill 52-14

II. What is Epidemiology and How is it Being Used to Justify the Proposed Ban?

III. Summary of Some Relevant Studies

Summary

1. This brief look at a complex field examined multiple cancer endpoints and two routes of exposure – via parents and more direct – and 4 neuro endpoints.

2. Most pesticide epi studies are case control, i.e., cases/effects are found, and attempts are made to document historical exposure. This study type is inferior to cohort/longitudinal studies – far fewer.

3. Neither study type proves cause and effect, but the cohort studies are typically more reliable.

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Summary (cont'd)

4. The lack of dose-response relationships – even qualitative – undermine the associations found with odds ratios significantly greater than 1.0.

5. The exposure measures in these studies – particularly the case-control studies – are usually quite crude, with little, if any documentation re: specific pesticides.

6. Most studies have targeted ag pesticides.

7. Most positive associations have been with pesticides no longer applied to lawns and athletic fields.

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Summary (cont'd)

8. These studies frequently contradict each other, probably due to failure to account for confounding factors; e.g., the positive association between pregnancy exposure to yard pesticides and childhood leukemia disappeared after adjusting for maternal X-ray exposure and antibiotic use during pregnancy (Dell, 2004, as cited in Turner et al., 2010). This is why it's important to view multiple relevant studies together.

9. The associations often lack a basis in disease etiology; e.g., does it make sense that pesticide X is associated with an increase in brain tumors if it can't cross the blood-brain barrier?

10. Associations with odds ratios significantly less than 1.0?! – that 'just goes to show ya'.....

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Appendix B. Burns, C.J., S.Z. Cohen, and C. Lunchick. *In press.* Comment on Neurodevelopment Disorders and Prenatal Residential Proximity to Agricultural Pesticides [Re: Shelton et al., 2014. Neurodevelopmental Disorders and Prenatal Residential Proximity to Agricultural Pesticides: The CHARGE Study. *Environ. Health Perspect.*, 122(10):1103–1109].

Neurodevelopmental Disorders and Agricultural Pesticides Exposures

We read with interest the analysis by Shelton et al. (2014) of the relationships between maternal proximity to insecticide application and autism spectrum disorders (ASDs) and developmental delay (DD) in children. Although we commend the investigators' efforts to identify, recruit, and enroll parents of children with ASDs or DD, absent is any confirmation of exposures or that the active ingredients drifted onto the residences or were inhaled or ingested, let alone at dose levels that might be adverse to the fetus (Williams and DeSesso 2014).

The authors note other sources of potential exposure, including diet and nonagricultural applications, that were unmeasured in their assessment. However, there are many factors that reduce the opportunity for participant exposures. Importantly, the inherent properties of each pesticide determine its volatilization and solubility. The method of application and whether the formulation is a liquid or granule also influences drift potential. For example, an orchard air-blast application has a very different exposure potential than a drip-line irrigation application of the same quantity of pesticide to the same crop at the same distance (US EPA, 2013). Weather conditions and wind direction influence whether an active ingredient is carried toward or away from a residence. Furthermore, Caldwell and Wolf (2006) found that amounts of ground-spray drift deposited 0.4 km downwind in windy conditions were 0.00001% of the applied amounts. Lastly, being inside, outside, or away from home all factor into human exposures.

Proximity to agricultural pesticide application has not been found to translate to corresponding levels of the pesticide in household dust (Curwin et al. 2005; Fenske et al. 2002; Ward et al. 2006). The California Pesticide Use Registry was evaluated by Nuckols et al. (2007). Although they confirmed agreement of pesticide applications with crop maps, they also recommended biological sampling to validate exposure assumptions for each active ingredient. Correlations of pesticide concentrations in household dust and urinary pesticide metabolite levels in children have been suggested (Lu et al. 2000) but not confirmed (Fenske et al. 2002; Morgan et al. 2008). Several studies of farmers and their families concluded that behavior patterns were more predictive of urinary pesticide concentrations than proximity to the field (Alexander et al. 2006; Arbuckle and Ritter 2005; Thomas et al. 2010).

In their recent review of geographic models in epidemiological studies, Chang et al. (2014) discuss many of these and other issues related to exposures, including of pesticides. The U.S. Environmental Protection Agency has begun to evaluate residential exposures to agricultural pesticides from spray drift and volatilization, and there is a growing understanding of off-target drift for each active ingredient. This understanding has permitted the US EPA to publish a quantitative methodologies for assessing residential exposure and risk resulting from spray drift and volatilization of conventional pesticides. (Second reference is Human Health Bystander Screening Level Analysis: Volatilization of Conventional Pesticides, US EPA, March 2014) Risk

is the result of the interaction between exposure and toxicity; unfortunately, Shelton et al. (2014) confuse the occurrence of a distant application with exposure. In light of critical weaknesses in exposure characterization in the present case, any relationship between pesticide exposure and the occurrence of ASDs and DD is unknown, and an association between exposure and occurrence is speculation.

C.J.B. and C.L. are employees of companies that manufacture and sell pesticides. S.Z.C. owns an environmental consulting firm that includes among its clients pesticides users and producers, as well as those impacted by pesticide users and producers.

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Appendix C. Cohen, S.Z. May 1995. Agriculture and the Golf Course Industry: An Exploration of Pesticide Use, *Golf Course Mgmt.*, 63(5):96-104.

Agriculture and the golf course industry: An exploration of pesticide use

To get a true picture, comparisons must focus on how use is calculated, the agricultural crops used for comparison and the different levels of golf course maintenance.

Stuart Z. Cohen, Ph.D., CGWP

The golf course industry is frequently attacked by environmental activists and others raising environmental concerns. In the majority of the 30-plus public hearings and legal proceedings in which I have testified, the opposition has had an underlying presumption that the turf industry uses large amounts of chemicals.

This presumption was quantified in 1991 by a New York State Attorney General's Office report that stated "... between four and seven times as much pesticides are used on Long Island golf courses than are applied on food crops" (1). Unfortunately, this comparison has received widespread exposure.

This brief article will attempt to set the record straight and address this emotional issue in an objective man-

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The amount of pesticide used in 1993 on agricultural crops in the United States was more than 90 times greater than the amount applied to golf courses.

Not only does the turf industry take extensive safety precautions to reduce pesticide exposure, it also ranks low in a use-comparison of crops to which chemicals are applied.

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ner. However, one thing is clear: Pesticide use is extremely complicated and is best addressed on a site-specific basis.

Following is a brief summary of some generalized agricultural and turf pesticide use scenarios.

Golf course pesticide use

• *Past analyses.* An old EPA/AARP/RTI report concluded that approximately 12 million pounds of pesticide active ingredients (a.i.) were applied to approximately 12 thousand golf courses nationwide (3) — an average of 1,000 lbs./year for each golf course. This is consistent with the New York Attorney General's Office report that approximately 50,000 pounds of pesticide active ingredients were applied annually to the 52 Long Island golf courses that responded to a survey. The report estimated that this consisted of 7 lbs./total acre per year, or 18 lbs./treated acre per year. Note that these numbers combine public and private courses.

• *Variations.* There are significant variations in pesticide use on golf courses, depending on the climate and whether the facility is public or private, high-end or minimally managed. A range that probably includes 90 percent of all golf courses is 500 to 2,000 lbs. a.i./year. These numbers will probably decrease as IPM use becomes more comprehensive.

Pesticide use on a per-acre basis

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) is the cornerstone of pesticide regulation in the United States. All pesticides distributed for sale and use must first be approved by the EPA under FIFRA and its regulations, then by each state where the pesticide is to be used under that state's equivalent laws and regulations.

FIFRA is a risk-benefit statute like the Toxic Substances Control Act, but is unlike most other environmental statutes because the EPA's Office of Pesticide Programs (OPP) must consider risks *and* benefits whenever a significant decision must be made regarding the registration status of a pesticide. Consequently, OPP's Economic Analysis Branch (EAB) has a group of experienced economists who are very familiar with pesticide use patterns.

The accompanying table presents a selection of recent pesticide use estimates compiled from data provided by EAB/OPP (Ed Brandt, personal communication) and the National Center for Food & Agricultural Policy (2). Pesticide use was ranked for various crops on a per-acre basis. Golf course pesticide use was ranked 31st of 90 for herbicides, 47th of 88 for insecticides and 38th of 75 for fungicides.

The golf course use rankings would be somewhat higher if the actual fraction of treated area were considered. This is due to the general rule that a slightly lower proportion of golf course area is treated compared with cropland, other factors being equal. But golf courses still would not be ranked in the top 10 percent, and such an adjustment would not be fair anyway.

Why? Ask someone like Tim Hiers, CGCS, who recently won GCSAA's President's Award for Environmental Leadership and a national Environmental Steward Award for integrating managed turf with a thriving ecosys-

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tem at Collier's Reserve, the first Audubon Signature Cooperative Sanctuary Golf Course. As every conscientious superintendent knows, nonchemically treated areas are an important part of every golf course. It would not make sense to separate the ecosystem into different parts just to add a few points to the pesticide use ranking. After all, regulatory permitting decisions that consider environmental impacts of land use are generally based on impacts to the whole parcel, not selected portions.

According to the EPA estimates, golf courses apply the following amounts of pesticide active ingredients to turf annually: 2,500,000 pounds of herbicides; 2,100,000 pounds of insecticides; and 4,500,000 pounds of fungicides. The total is 9,100,000 pounds, excluding relatively small amounts of nematicides and soil sterilants. This equates to 650 lbs. a.i./golf course per year, which is less than the estimates noted at the beginning of this article.

In comparison, 240,000,000 pounds of pesticide active ingredients are applied to corn annually. The total amount of pesticide used in U.S. agricultural crop production in 1993 was somewhere between 800 and 900 million pounds (2), more than 90 times the amount applied to golf courses. These numbers do not include residential pesticide use, which itself is substantial.

Note that 650 lbs. a.i./golf course per year equates to 6.5 lbs. a.i./A per year, if one assumes a 100-acre golf course. This is very close to the Long Island estimate noted at the beginning of this article. But this does not justify

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Crop/Site	Acres	lb a.i./A (rank)		
		Herbicides (90 crops ranked)	**Insecticides** (88 crops ranked)	**Fungicides** (75 crops ranked)
Onions	151,676	6.32 (1)	1.41 (48)	6.56 (22)
Citrus	878,300	6.21 (2)	25.86 (4)	6.16 (26)
Sweet corn	761,045	2.79 (13)	1.81 (42)	1.45 (49)
Pears	72,226	1.57 (39)	77.68 (1)	13.42 (11)
Cotton	11,120,700	2.65 (18)	1.79 (43)	0.19 (59)
Tomatoes	411,361	1.64 (36)	1.82 (41)	21.20 (4)
Feed corn	78,156,196	2.73 (16)	0.34 (66)	~0
Grapes	764,137	1.42 (44)	6.51 (12)	61.92 (1)
Apples	502,792	1.10 (57)	31.36 (3)	13.64 (8)
Peaches	186,388	1.38 (47)	15.15 (8)	40.11 (2)
Tobacco	784,770	1.52 (41)	4.41 (16)	0.47 (56)
Homeowner turf	20,900,000	1.20 (52)	0.30 (66)	0.03 (66)
Golf course turf	1,400,600	1.79 (31)	1.50 (47)	3.21 (38)

Pesticide use for various crops on a per-acre basis.

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the four to seven times comparison, nor does the 18 lbs./treated acre per year appear to be a valid generalization for the country's golf courses as a whole.

A national perspective on land use also helps one understand the issue better. Golf course turf area is less than 1 percent of harvested cropland area, most of which receives pesticide applications (based on data from the EPA and the Department of Commerce's Census of Agriculture). This statistic should not be used to justify unnecessary applications of pesticides. Rather, it helps lend perspective to this often emotional debate.

Now the question can be answered: Do golf courses use greater amounts of pesticides than agriculture on a per-acre basis? It should be apparent the answer is "definitely sometimes." Golf courses are in the middle range of pesticide use when one considers total acreage, and do not reach the top 10 percent when one considers actual treated acreage. Golf courses appear to account for about 1 percent of agricultural pesticide use in the United States.

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2. Gianessi, L.P. and J.E. Anderson. 1995. Pesticide use in U.S. crop production national summary report. National Center for Food and Agricultural Policy, Washington, D.C.
3. Kriner, R. 1982. National survey of pesticide usage on golf courses in the United States conducted July-September. Economic Analysis Branch, Office of Pesticide Programs, EPA, Washington, D.C.

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This article could not have been written without the assistance of Ed Brandt and Rob Esworthy, EAB/OPP/EPA; and Michael O'Connor, CGCS, Environmental & Turf Services, Vt.

Stuart Z. Cohen is president of Environmental & Turf Services Inc. of Wheaton, Md., a consulting firm that specializes in the development of integrated management plans and environmental risk assessments for golf courses. A certified groundwater professional with a doctorate in physical organic chemistry, he worked for the EPA for 11 years and was the director of the Cape Cod study.

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STUART Z. COHEN, Ph.D., CGWP

Condensed Curriculum Vitae

President, Environmental & Turf Services, Inc., Wheaton, MD
Senior Environmental Scientist

EDUCATION

Ph.D., Physical Organic Chemistry, George Washington University, Washington, DC, 1984 (dissertation research at NIADDK/National Institutes of Health).
B.A., Chemistry, University of Maryland, Baltimore County, 1975.
(Related coursework at Johns Hopkins U., Georgetown U., and Furman U.)

At least 14 short courses in habitat preservation/enhancement, chemical carcinogenesis (one full semester course, one short course), endocrine disruptors, chemical engineering, golf course drainage, soil microbiology, contaminant hydrogeology, physical organic chemistry, the CORMIX and iSTREEM point source surface water discharge models, soil ingestion in risk assessment, nanotechnology risks, and golf course design. Trained in FIFRA Good Laboratory Practices (with a focus on field studies).

Foreign Language: Limited working proficiency in German.

CERTIFICATION

Certified Ground Water Professional #196522, National Ground Water Association (since 1992).
CPR and First Aid Training-Red Cross, 1993 & 1996.
NITON XRF Spectrum Analyzer – Thermo Scientific, 1998 & 2008 (for heavy metals in soils).
Professional Fertilizer Applicator (PFA 0675), 2014-2015.

EXPERIENCE

1991 to Present: President, Environmental & Turf Services, Inc., Wheaton, MD.

Responsible for supervising and conducting field studies and computer risk assessments for pesticides and fertilizers used on golf courses and in agriculture (runoff and leaching); water quality monitoring studies; lead and arsenic contamination assessments and best management practices for firing ranges; environmental fate issues under TSCA; carbon footprint analyses; environmental site assessments in real estate transactions, including record searches, visual hazard assessments, etc.; and expert testimony.

1994 to 1999: Instructor for the NRA on environmental management at shooting ranges.

1986 to 1990: Manager, Ground Water and Environmental Programs, Biospherics Incorporated, Beltsville, MD.

Managed programs regarding ground water and soil contamination by pesticides, lead, and hazardous wastes; golf course environmental impact assessments; and real estate transactions. Programs included risk assessments, placement of monitoring wells, soil gas analysis, unsaturated zone modeling, etc. National Priority List site (Superfund) Project Manager.

1976 to 1986: Chemist and Ground-Water Team Leader, Office of Pesticides and Toxic Substances, Environmental Protection Agency, Washington, DC.

One of EPA's key scientists for the development and implementation of pesticides in ground water programs. Responsibilities/functions: management of $1.4 million budget; co-chair of the National Well-Water Survey Steering Committee; senior physical scientist on all pesticides in ground water regulatory actions; director of ground water studies by pesticide industry; main interagency and international contact on pesticides in ground water; and conducted DBCP and EDB ground water risk assessments, which led to bans of all soil uses in 1979 and 1983, respectively. Synthesized interdisciplinary risk assessments for several chemicals over a two-year period. Co-developed biorational pesticides testing guidelines. Assessment of heptachlor in human and cow milk in Hawaii. Testing guidelines and regulations for new chemicals under TSCA§ 5.

1980 to 1987: Guest Worker in organic chemistry at the Laboratory of Chemistry, NIADDK, National Institutes of Health (NIH), Bethesda, MD. (One year full-time: '81-'82, part-time: '80-'81 and '82-'87.)

1975 to 1976: Research Technician, The Johns Hopkins University School of Medicine, Department of Immunology, Baltimore, MD. Prostaglandin and antibody radioimmunoassays. Radiolabel syntheses.

HONORS & AWARDS

EPA Bronze Medal for the ethylene dibromide ground water assessment, 1984. EPA Special Achievement Awards (two cash awards) for work in ground water contamination by pesticides, 1983-1985.

MEMBERSHIPS

- Society of Environmental Toxicology and Chemistry (SETAC), North America (NA) and regional chapter (CPRC). Chair of the TSCA Reform Dialog Group, Co-Chair of the Public Outreach Committee.
- Int'l Union of Pure & Applied Chem. (IUPAC) commission member 1986-1995; Titular (voting) member, Commission on Agrochemicals, 1990-1995 (assoc. member '85-'90). Symposium co-chair for the August, 2014 Int'l Congress of Pesticide Chemistry.
- American Chemical Soc.: Environmental, Agrochemicals, and Medicinal Chem. Divisions; Chem. Soc. Wash.
- National Ground Water Assoc.: Ground Water Protection and Management Committee, Chairman, 1991-1993; committee member 1989-1996.

REGISTERED TRADEMARK

CarbonSave®: the first carbon footprint calculator and energy efficiency analyzer for golf courses.

SHORT COURSE/SEMINAR/WEBINAR INSTRUCTOR

1994-2012. Topics have included watershed resource management, hydrolysis reactions, lead management, and water quality monitoring.

INVITED INTERNATIONAL PROFESSIONAL TRAVEL

Rome (Italy, 2004), Copenhagen (Denmark, 2003), Basel (Switzerland, 2002), Vienna (Austria, 1998), London (England, 1998), São Paulo (Brazil, 1996), London (England, 1996), Budapest (Hungary, 1995), Bangkok (Thailand, 1992), Rehovot/Bet Dagan (Israel, 1991), Hamburg (Germany, 1990), Lyon (France, 1989), West Berlin (Germany, 1985)

JOURNAL & BOOK PUBLICATIONS

Baris, R.D., S.Z. Cohen, N.L. Barnes, J. Lam, and Q. Ma. 2010. Quantitative Analysis of Over 20 Years of Golf Course Monitoring Studies. *Environ. Tox. and Chem.* 29(6):1224-1236.

Plus approximately 25 others.

ABSTRACTS, POSTERS, PRESENTATIONS, AND MISCELLANEOUS ARTICLES

Gobas, F., S. Haefner, and S. Cohen. 2014. Bioaccumulation Risk Assessment of Pentachloronitrobenzene: 1. Basis for Lessons Learned (Abstract #353). Presented at the August 2014 IUPAC International Congress on Pesticide Chemistry in San Francisco, CA.

Gobas, F., S. Cohen, and S. Haefner. 2014. Bioaccumulation Risk Assessment of Pentachloronitrobenzene: 2. Lessons Learned (Abstract #354). Presented at the August 2014 IUPAC International Congress on Pesticide Chemistry in San Francisco, CA.

Haefner, S.M., S.Z. Cohen, and N.L. Barnes. August 23, 2012. Urban Stressors for Pesticide Endangered Species Assessments: Should Recent Nutrient TMDLs and Laws be Considered? Presented at the American Chemical Society (AGRO Division) 244th National Meeting in Philadelphia, PA.

Plus approximately 80 others.

Title 40 Code of Federal Regulations Part 158 – DATA REQUIREMENTS FOR PESTICIDES

- §158.310 Product Chemistry Data Requirements: 21 studies required, 10 studies conditional required in the areas of product identity and composition and physical and chemical properties.
- §158.400 Product Performance Data Requirements
  - Terrestrial Use: Food crop: 8 studies required and 1 study conditionally required; Nonfood crop: 8 studies required in the areas of efficacy of fungicides and nematicides (food only) and efficacy of vertebrate control agents.
  - Aquatic and Greenhouse Uses: Food crop: 1 study conditionally required in the area of efficacy of fungicides and nematicides.
  - Residential Outdoor Use: 8 studies required in the area of efficacy of vertebrate control agents.
  - Indoor Use: 6 studies required in the area of efficacy of vertebrate control agents.
- §158.500 Toxicology Data Requirements
  - Food Use: 20 studies required, 7 studies conditionally required, in the areas of acute testing, subchronic testing, chronic testing, developmental toxicity and reproduction, mutagenicity testing, and special testing.
  - Non-Food Use: 15 studies required, 11 studies conditionally required, in the areas of acute testing, subchronic testing, chronic testing, developmental toxicity and reproduction, mutagenicity testing, and special testing.
- §158.630 Terrestrial and Aquatic Nontarget Organisms Data Requirements
  - Terrestrial, Forestry, Residential Outdoor Uses: 9 studies required, 12 studies conditionally required, in the areas of avian and mammalian testing, aquatic organisms testing, sediment testing, and insect pollinator testing.
  - Aquatic Use: 8 studies required, 13 studies conditionally required, in the areas of avian and mammalian testing, aquatic organisms testing, sediment testing, and insect pollinator testing.
  - Greenhouse and Indoor Uses: 3 studies conditionally required in the areas of avian and mammalian testing and aquatic organisms testing.
- §158.660 Nontarget Plant Protection Data Requirements: 3 studies required, 6 studies conditionally required, in the areas of nontarget area of phytotoxicity (Tier I, Tier II, Tier III) and target area phytotoxicity.
- §158.1020 Applicator Exposure Data Requirements: occupational & residential uses: 6 studies required and 1 study conditionally required.
- §158.1070 Post-application Exposure Data Requirements
  - Occupational Use: 8 studies required and 1 study conditionally required.
  - Residential Use: 8 studies required and 2 studies conditionally required.
- §158.1100 Spray Drift Data Requirements: Terrestrial, Aquatic, Forestry Uses : 2 conditionally required studies.
- §158.1300 Environmental Fate Data Requirements
  - Terrestrial Use: 9 studies required and 6 studies conditionally required, in the areas of degradation, metabolism, mobility, dissipation (field), and ground water monitoring.
  - Aquatic Use: 6 studies required and 3 studies conditionally required, in the areas of degradation, metabolism, mobility, and dissipation (field).
  - Greenhouse Use: 3 studies required and 3 studies conditionally required, in the areas of degradation, metabolism, and mobility.
  - Indoor Use: 1 study conditionally required in the area of degradation.
  - Forestry Use: 7 studies required and 4 studies conditionally required, in the areas of degradation, metabolism, mobility, dissipation (field), and ground water monitoring.
  - Residential Outdoor Use: 4 studies required and 2 studies conditionally required, in the areas of degradation, metabolism, mobility, dissipation (field), and ground water monitoring.

MONTGOMERY COUNTY COUNCIL
ROCKVILLE, MARYLAND

ROGER BERLINER
COUNCILMEMBER
DISTRICT 1

CHAIRMAN
TRANSPORTATION, INFRASTRUCTURE
ENERGY & ENVIRONMENT COMMITTEE

March 3, 2015

Harold Varmus, M.D., Director
National Cancer Institute
MSC 2590
31 Center Drive
Bethesda, MD 20892

Dear Dr. Varmus,

Our County is proud to be the home of the National Cancer Institute, the Federal Government's principal agency for cancer research.

I am writing you because Montgomery County needs your expert help. Legislation has been introduced before our County Council that would ban the use of certain pesticides for ornamental lawn care on private property, as well as on parkland, fields, and county property generally (attached). The legislation is predicated upon the belief held by many that the exposure to pesticides poses an unacceptable risk to human health, animal health, and the environment notwithstanding that the Environmental Protection Agency permits their use.

In particular, proponents point to the following statement by the American Academy of Pediatrics issued in 2012:

> *Children encounter pesticides daily and have unique susceptibilities to their potential toxicity. Acute poisoning risks are clear, and understanding of chronic health implications from both acute and chronic exposure are emerging. Epidemiologic evidence demonstrates associations between early life exposure to pesticides and pediatric cancers, decreased cognitive function, and behavioral problems. Related animal toxicology studies provide supportive biological plausibility for these findings. Recognizing and reducing problematic exposures will require attention to current inadequacies in medical training, public health tracking, and regulatory action on pesticides.*

Not surprisingly, opponents point to the two sentences that follow in that same statement to support their position that the ban is not warranted at this time:

> *Ongoing research describing toxicologic vulnerabilities and exposure factors across the life span are needed to inform regulatory needs and appropriate interventions. Policies that promote integrated pest management, comprehensive pesticide labeling, and marketing practices that incorporate child health considerations will enhance safe use.*

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My colleagues and I are not expert in such matters, and given that there is no major jurisdiction in the country to have adopted a comparable ban, we have few resources to call upon to provide us with the scientific guidance we need to evaluate the proposal before us. Based on the NCI's rich history of research on associations between environmental exposures and cancer, it seems that the NCI is uniquely positioned to assist us in this important matter. It is my understanding that the NCI has spent years, decades even, studying the impact of pesticides on human health.

Our Council and community would greatly benefit from understanding what the NCI's research relating to pesticide exposure has concluded. Specifically, we seek your guidance as to whether the NCI believes that the exposures created by the use of pesticides for lawn care and on playing fields warrant further limitations beyond existing federal and state rules.

The Montgomery County Council's Transportation, Infrastructure, Energy & Environment Committee will meet on March 16th to hear from national experts on this matter. I would be grateful if a representative from your organization could attend that meeting and provide testimony. Please let me know if this is possible.

Thank you in advance for your consideration of this request and for your contribution to our community.

Sincerely,

Roger Berliner
Councilmember, District 1
Chair, Transportation, Infrastructure, Energy, and
Environment Committee

Attachment: Montgomery County Bill 52-14, Pesticides – Notice Requirements – Non-Essential Pesticides - Prohibitions

DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

National Institutes of Health
National Cancer Institute
Bethesda, Maryland 20892

March 11, 2015

Councilmember Roger Berliner
100 Maryland Avenue
Rockville, MD 20850

Dear Representative Berliner:

Thank you for your inquiry concerning pesticides and health risk.

The mission of the National Cancer Institute (NCI) is to conduct and support research, training, health information dissemination, and other programs with respect to the cause, diagnosis, prevention, and treatment of cancer, rehabilitation from cancer, and the continuing care of cancer patients and the families of cancer patients. Although NCI is responsible for conducting research on pesticides, it is also a part of the mission of the National Institute of Environmental Health Sciences (NIEHS), who support research to discover how the environment affects people in order to promote healthier lives. The NIEHS research portfolio includes cancer and many other outcomes, such neurological, developmental and endocrine. The Environmental Protection Agency, the federal agency responsible for regulating pesticide use taking into account cancer as well as a panoply of other health effects. For example, NIEHS and EPA have supported birth cohort studies which estimate sources, pathways of in utero and postnatal pesticide exposures of children living in their communities.

Pesticides are a diverse group of chemicals used to control pests including unwanted plants, molds, and insects. Pesticides are widely used in agricultural, commercial and residential settings, and as a result pesticides and their metabolites are detectable at low concentrations in the urine of a majority of the U.S. population (Barr et al. 2004; 2005; 2010). While pesticides are broadly known to exert adverse toxic effects to humans following high-dose acute exposures; knowledge about chronic low-dose adverse effects from exposure to specific pesticides is more limited. Assessing the health effects of specific pesticides has included a combination of laboratory studies on rodents and human epidemiological studies. Although the majority of pesticides currently registered for use in the United States are neither overly genotoxic nor carcinogenic in rodent studies, results from epidemiological studies of human cancer exposed to pesticides have shown mixed results.

The National Toxicology Program (NTP) of the NIH, and the International Agency for Research on Cancer (IARC), a component of the World Health Organization, are perhaps the two most respected scientific organizations conducting independent scientific review of the evidence for carcinogenicity of human exposures. These reviews include both the toxicologic and epidemiologic evidence for the carcinogenicity of pesticides, reviews that are periodically updated. The last such systematic review by the IARC was over 20 years ago. At that time, arsenical pesticides and dioxin (a contaminant of some herbicides) were the only two pesticides they classified as human carcinogens. However, they also indicated their opinion that "occupational exposures in spraying and application of non-arsenical insecticides" as a group could be classified as probable human carcinogens. Since a substantial body of evidence has accumulated since this report, the IARC is currently empanelling several review groups to comprehensively update this evaluation of pesticides. The NTP is an on ongoing evaluation that is frequently updated. Over time, they have assessed 506 pesticides and listed 21 as "probable human carcinogens", but have listed 141 as "possible, suggestive or likely". It should be noted that the 506 includes all pesticides, not only those for home or garden use, and also includes those used in the past, but not currently.

With this absence of definitive information on the carcinogenicity of specific pesticides, in the United States the Environmental Protection Agency has adopted a strategy to minimize non-occupational exposures to pesticides by discouraging the use of longer-lasting and broad-spectrum pesticides. The lipophilic, bioaccumulative organochlorine (OC) insecticides that were widely used in the mid-20th century were first replaced by organophosphates (OP), and have now been replaced by carbamates and pyrethroids because these compounds are more environmentally labile and do not accumulate in the food chain to the same extent as the OCs and OPs. Pyrethroids insecticides and carbamate insecticides and herbicides are generally metabolized and eliminated from the body within 24-48 hours as water soluble metabolites in urine. This policy has resulted in lower OC and OP exposures among the general public (Barr et al. 2004; 2005; 2010). Many widely-used phenoxy herbicides are also eliminated from the body within 24-48 hours.

Recent scientific advances suggest that we may be able to accelerate progress in clarifying the carcinogenicity of pesticides, as well as other chemicals and biologic agents. The revolution in molecular science over the last 20 years has given us new understanding of biology, and a set of tools to answer questions that have previously eluded us. Indeed, application of these tools in interdisciplinary studies of highly exposed human populations has recently produced hypotheses about the potential carcinogenicity of several pesticides. At this point they are simply findings that need to be tested and replicated by others to identify those with public health applications, but the NCI and other biomedical research groups world-wide are actively involved in using the new molecular science investigating the human epidemiology and the multiple

mechanisms that may be involved in pesticide-mediated carcinogenesis. However, until a more comprehensive scientific understanding of pesticide-carcinogenesis is achieved, balancing the potential, albeit uncertain, carcinogenic risk with the perceived benefits derived from the use of pesticides remains a public policy judgment rather than a strictly scientific one.

A nationwide use-reduction policy for pesticides has not yet been adopted in the United States because the scientific data concerning the carcinogenicity of specific pesticides has not been judged to be sufficient, the net benefit to health was unclear, and the economic impact was disproportionately large for some groups within the population. However, in several European countries, including Sweden, Denmark, the Netherlands, a use-reduction policy has been implemented as a precautionary measure until more definitive scientific evidence becomes available. The result in these European countries has been a substantially diminished exposure overall.

NCI scientists provide the results of their research to the public, the scientific community, and regulatory agencies. Because decisions about use of pesticides involve complex decisions involving weighing perceived risks and benefits based on local community values, as mentioned above, NCI scientists do not typically weigh in on regulatory or public policy decisions. Thus, we respectfully decline the invitation to provide testimony at the upcoming hearing.

Sincerely yours,

Stephen J Chanock, M.D.
Director
Division of Cancer Epidemiology and Genetics
National Cancer Institute

cc:
Dr. Harold Varmus
Dr. Linda Birnbaum
Dr. Lynn Austin