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STATEMENT OF
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ON
BILL 19-643
THE PESTICIDE EDUCATION AND CONTROL AMENDMENT ACT OF 2012
BEFORE THE
COMMITTEE ON THE ENVIRONMENT, PUBLIC WORKS, AND TRASPORTATION
COUNCIL OF THE DISTRICT OF COLUMBIA

FEBRUARY 27, 2012

Ms. Chairwoman and members of the Committee. Thank you for the opportunity to appear before the Committee today. I am Jay Feldman, Executive Director of Beyond Pesticides, a national, grassroots, membership organization that represents community-based organizations and a range of people seeking to improve protections from pesticides and promote alternative pest management strategies that reduce or eliminate a reliance on toxic pesticides. Our membership spans the 50 states, the District of Columbia, and groups around the world.

I. Support for the Legislation, Bill 19-643, with Suggested Amendments

We are here today in support of 19-643, *The Pesticide Education and Control Amendment Act of 2012*, with amendments as discussed in our testimony. We support the legislation's overall intent to restrict wide availability of hazardous pesticides, making them available only under emergency situations, and effecting a shift to least-toxic or "minimum risk" type chemicals as classified by the U.S. Environmental Protection Agency (EPA). We explain below why this approach to pesticide law is critical to the protection of children and elderly, and vulnerable population groups that suffer from compromised immune and neurological systems, cancer, reproductive problems, respiratory illness and asthma, Parkinson's, Alzheimer's, diabetes, and learning disabilities in the District of Columbia.

II. Chemical Restrictions

We suggest a framework that creates an allowable list of materials, similar to the 25(b) list created by EPA that is based on a set of criteria, protective of public health and the environment. Under this system, a defined, integrated pest management system (IPM) for buildings and organic land management for turf, playing fields, parks, and rights-of-way, will effectively meet the District's pest management needs. With the restriction of hazardous chemical use and a clear

a clear definition of IPM for buildings and organic for turf and landscape, the legislation will put in place a list of allowable materials that will incentivize the use of non-toxic practices, moving the District into the forefront of green land and building management practices.

Proposed changes to the legislation:

1. Remove toxic chemical dependency through improved IPM definition.
Define IPM to prioritize non-chemical management practices and eliminate the conditions that are conductive to pest problems. In defining IPM to first apply cultural, mechanical, and biological before the introduction of chemicals, the legislation will help to shift the current orientation in conventional pest management away from pesticide dependency. Pest management that practices effective exclusion techniques, such as eliminating and sealing pest entryways, good maintenance of plumbing, proper food and trash management, and adequate drainage, will eliminate a reliance on toxic chemicals.

We suggest the following definition of IPM to be applied to structural pest management: INTEGRATED PEST MANAGEMENT.—The term 'integrated pest management' means a managed pest control program that— (A) eliminates or mitigates economic and health damage caused by pests; (B) uses— (i) integrated methods; (ii) site or pest inspections; (iii) pest population monitoring and prevention strategies; (iv) an evaluation of the need for pest control; and (v) one or more pest prevention and management methods that incorporate exclusion techniques, such as habitat modifications, sanitation practices, entryway closures, structural repair, mechanical and biological controls, other nonchemical methods, and (if nontoxic options are unreasonable or have been exhausted) least-toxic pesticides [see Addendum A]; and C) minimizes— (i) the use of pesticides; and (ii) the hazards to human health and the environment associated with pesticide applications.

2. Add a definition for natural organic fertilizer as the focus of organic land management practices.
There is no reason to use toxic chemicals in turf and landscape management in the District. High quality turf and landscapes can be achieved through proper management of soil health through proper fertilization that eliminates synthetic fertilizers and focuses on building the soil food web and the nurturing of soil microorganisms. This approach eliminates chemicals, adopts compost fertilizers and mulching systems, and focuses on managing weeds and insects through the development of healthier plants and turf that are not vulnerable to disease and infestation.

We suggest language that establishes a clear definition of natural organic land management practices, such as the following: NATURAL ORGANIC FERTILIZER - The term 'natural organic fertilizer' means fertilizers that contain nutrients naturally derived, solely from the remains or a by-product of an organism, or from a mineral. The term includes naturally occurring materials such as cotton seed meal, bone meal, blood meal, fish emulsion, kelp, and manure, and organic compost. The term does not include any synthetic fertilizer, defined as a substance containing a plant nutrient created by a synthetic, chemical process, including triple super phosphate made by treating rock phosphate with phosphoric acid, nutrient created by a synthetic chemical process, including triple super phosphate potassium chloride, quick release synthetic fertilizers, petroleum-based fertilizers

3. Clear criteria of unacceptable hazards associated with chemical effects.
The risk management approach used by EPA to register pesticides has failed to adequately protect the public health and the environment. The approach is especially insensitive to the most vulnerable, including children and the elderly, and those who have existing illnesses associated with cancer, respiratory problems, immune and nervous system and other medical conditions cited above. Instead, the legislation should clearly state the hazards that are viewed as unacceptable and unnecessary in pest management systems in the District. Appendix A includes a list of health outcomes that are unacceptable and can be identified through existing government databases in order to screen out their associated chemical uses.

4. Public health emergency exemption.
Expanding the emergency provision beyond public health emergencies is unnecessary and will create a loophole in the statute. Including "significant economic damage" language under emergency without a clear definition and a decision making hierarchy will result in ongoing and constant emergencies that could be avoided if the language was strengthened.

5. Ensure that the burden of proof rests with the registrant or chemical manufacturer. The Department of the Environment should not have to prove that chemicals cause unacceptable hazards. Instead, the registrants should assume the burden of proving that their products can meet the standard going forward. In this context, no chemical should be introduced into a pest management system without a showing that it is necessary after all cultural, mechanical, and biological approaches have been implemented. Only then, should least-toxic or minimum risk chemicals be introduced. Too often, conventional pest management is simply trying to shift from one chemical to another deemed less toxic, despite the fact that the adoption of different practices could eliminate the need for the chemical.

III. Adverse Health Effects

In terms of overall health impact of pesticide use, Beyond Pesticides has developed a database that catalogs the range of public health diseases associated with pesticide exposure in epidemiologic studies. In fact, the *Pesticide-Induced Diseases Database* has over 300 entries with direct links to pesticides. This is not just a matter of studies on laboratory animals that characterize an adverse health outcome, there is real world epidemiologic data that establishes the connection.

The concerns associated with pesticide use cannot be understated. Given the known hazards and uncertainties or untested aspects associated with pesticide use, and in light of the availability of alternative organic practices, erring on the side of caution is sound public policy.

The vulnerability of children to pesticides is well-established, as are the problems of exposure to mixtures that are not assessed. This issue is discussed in detail in Beyond Pesticides' factsheet *Children and Pesticides Don't Mix*[1](see Addendum C), which cites peer-reviewed scientific literature on the health effects of pesticides.

[1] Beyond Pesticides, *Children and Pesticides Don't Mix*, http://www.beyondpesticides.org/lawn/factsheets/Pesticide.children.dontmix.pdf

Of 25 pesticides commonly used to manage facilities, 11 are linked to cancer, 12 are associated with neurological effects, 10 are associated with reproductive effects, 5 cause birth defects or developmental effects, 12 are sensitizers or irritants, 10 cause liver or kidney damage and 6 are suspected endocrine disruptors. Of 13 pesticides commonly used in turf and landscape management, 2 potentially leach and contaminate groundwater, 8 are toxic to birds, 8 are toxic to fish, 10 are toxic to aquatic organisms, and 3 are toxic to bees

IV. Land and Building Management without Toxic Pesticides

The support for strict adherence to a defined IPM program and natural organic turf management is driven by scientific findings of adverse effects associated with pesticide use as well as a high degree of uncertainty or unknown impacts related to chemical interactions, low-dose exposure, specific toxic endpoints or health effects, vulnerable population groups (such as children and those with existing illnesses like asthma), and synergistic effects of chemical mixtures. The identified hazards and uncertainties linked to pesticide use have formed the basis of the precautionary approach that is embodied in the approaches that have been adopted in San Francisco, Greenbelt, MD, and Highland Park Il, among dozens of other communities nationwide.

According to the former head of San Francisco's Toxics Reduction Program, now the Director Department of Toxic Substances Control for the California Environmental Protection Agency, Debbie Raphael, "Instead of asking how much harm is allowable, we ask how little harm is possible. What are the alternatives?" As Ms. Raphael explains, "IPM is more than a list of approved pesticides. It's all about prevention. . ." The City of Greenbelt, MD set a goal of "phasing out the cosmetic use of chemical pesticides on all public lands," defining cosmetic use as "any use other than the direct protection of the public and environmental health."

A. Building Management

Structural IPM techniques address the root cause of the pest problem, addressing issues related to sanitation, sealing, or structural repairs. The kinds of pest management techniques typically used by facilities include: exclusion techniques that include sealing openings (cracks and crevices), installing door sweeps, and structural repairs that include repair of leaking pipes; caulking and harborage reduction (such as elimination of storage in cardboard boxes); mechanical techniques that include the use of traps and vacuuming; and sanitation techniques that include trash management.

These techniques effectively manage the predominant structural pests, ants, cockroaches and rodents (mice and rats). Specific methods for cockroach control include vacuuming, glue boards, and crack and crevice caulking. For rodent control, techniques include removal of ivy and ground cover that provided harborage, cleaning nesting areas, and structural improvements. Effective termite control eliminates damp areas with poor drainage, and incorporates biological control or bait systems, eliminating the drilling or trenching of chemicals into and around the foundation of the building.

B. Turf Management

Toxic chemicals are not needed for successful turf management. Many communities, school districts, and state policies are now following a systems approach that is designed to put a series

of preventive steps in place that will solve pest (weed and insect) problems. The systems approach is based on three basic concepts: (i) natural, organic product where use is directed by soil testing, (ii) an understanding that the soil biomass plays a critical role in soil fertility and turf grass health, and (iii) specific and sound cultural practices.

The goal of a natural turf management program is to create turf that is both aesthetically pleasing and meets site objectives. At the same time, this turf will provide a surface that will be healthy and free from toxic chemicals. From an environmental perspective, the approach is designed to utilize materials and adopt cultural practices that will avoid any runoff or leaching of nutrients and toxic pesticides into waterways and the Chesapeake Bay watershed.

Natural turf management is a "feed-the-soil" approach that centers on natural, organic fertilization, microbial inoculants, compost (including compost teas), microbial food sources, and topdressing as needed with high quality finished compost. It is a program that supports the natural processes that nature has already in put in motion. These inputs, along with very specific cultural practices, which include mowing, aeration, irrigation, and over-seeding, are the basis of the program.

Experience finds that this approach will build a soil environment rich in microbiology that will produce strong, healthy turf that is able to withstand many of the stresses that affect turf grass. The turf system will be better able to withstand pressures from heavy usage, insects, weeds, and disease, as well as drought and heat stress, as long as good cultural practices continue to be followed and products are chosen to enhance and continually address the soil biology. While problems can arise in any turf system, they will be easier to alleviate with a soil that is healthy, and has the proper microbiology in place.

V. Need for Public Education

The importance of public education on pesticides cannot be overstated, given the misinformation that exists in the marketplace.

It cannot be suggested that pesticide product registration by EPA creates adequate assurance that the residents of the District are protected from hazards. EPA has indicated that it is unlawful for manufacturers or commercial applicators to label pesticides products or characterize them as "safe" because it is considered false and misleading. The language deemed untruthful and a violation of federal law, the *Federal Insecticide, Fungicide and Rodenticide Act*, includes, "Claims as to the safety of the pesticide or its ingredients, including statements such as "safe," "nonpoisonous," "noninjurious," "harmless" or "nontoxic to humans and pets" with or without such a qualifying phrase as "when used as directed". . . [40 CFR § 156.10(a)(5)(ix)] [2]

To give historical perspective to this problem and illustrate that this legislation is indeed on the right track, we can go back to 1986 when the U.S. Government Accountability Office (GAO) published a report entitled, *Nonagricultural Pesticides: Risks and Regulation*, and found that, "The general public receives limited and misleading information on pesticide hazards."[3] Four

[2] See http://edocket.access.gpo.gov/cfr_2010/julqtr/pdf/40cfr156.10.pdf, p63.
[3] U.S. General Accounting Office (GAO), *Nonagricultural Pesticides: Risks and*

years later, in March 1990, GAO published another report on the subject, *Lawn Care Pesticides: Risks Remain Uncertain While Prohibited Safety Claims Continue*,[4] and found "the same situation." Today, 22 years later, commercial pesticide applicators have become more sophisticated and we now call it "greenwashing." However, you can be sure that customers continue to receive the same message, the ones that GAO documented, characterizing toxic pesticides as "completely safe for humans."

This is what GAO said,

> To determine what safety information professional pesticide applicators provide to potential customers, we telephoned 21 lawn care companies in the Washington, D.C. metropolitan area (including Maryland and Virginia), requesting information and literature about the safety of their products.
>
> The lawn care company representatives we talked to provided a variety of responses when asked about the effects of their products on human, animal, and environmental health. Several representatives said they were aware of the concerns surrounding pesticide use and described the measures they take as a result. One company representative, for example, said his company did not use the pesticide diazinon because it was too toxic. [The insecticide diazinon, widely used in an around homes was finally banned from residential use at the end of 2004. That is how slow the regulatory process moves, even with highly toxic chemicals.] Another said his company used pesticides only when necessary.
>
> Many of the representatives we talked to, however, made statements that their products are safe or nontoxic. These statements included the following:
>
> "Our products are practically nontoxic; no one gets sick."
> "All [of] our products are legal and registered at EPA as practically nontoxic."
> "The only way to be affected by the [pesticide] 2,4-D would be to lay [sic] in it for a few days."
> "The safety issue has been blown out of proportion. Such a small amount of chemicals are put down directly on plants. . .[They do] not affect animals or people."
> "Dogs may get a rash or irritated [from diazinon], but they will only feel a little itchy. This is the same reaction the applicator gets when the pesticide touches their [sic] skin."

An investigation like this today, I believe, would yield the same findings. However, today, we have an added a layer of complexity to this problem that is more misleading for consumers. Risk assessments of pesticides are in full swing at EPA, and reviews of pesticides or registration reviews brand them as presenting acceptable risk, under statutory standards that blur complex toxicological questions and the limitations of the analysis. Despite rhetoric to the contrary, vulnerable population groups, such as children, the elderly, and those with neurological and immunological illnesses and cancer, are inadequately protected by these risk assessments. The testing protocol for some health endpoints, such as poisoning that disrupts the endocrine system

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Regulation, Washington, D.C., GAO/RCED-86-97.

[4]GAO, Lawn Care Pesticides: Risks Remain Uncertain while Prohibited Safety Claims Continue, GAOP/ RCED-90-134, March 1990.

(impacting human development at miniscule doses) has not been developed by EPA, as required by statute. And some issues have been simply taken off the table, such as the additive effect of chemical mixtures and the synergic effects of pesticides mixed with other pesticides or with pharmaceuticals. While all this is going on, the District's pesticide registration apparatus in the Department of the Environment is entirely dependent on a federal system that is inadequate.

In summary review, there are a number of reasons why existing regulatory standards and reviews provide inadequate protection of public health and the environment: (i) pesticide reregistration, carried out by EPA's Office of Pesticide Programs, is an ongoing process with outstanding and missing data associated with a pesticide's review; (ii) additional studies are needed to reach final decisions on the impact on children for hundreds of pesticide products; (iii) the underlying standards of the *Federal Insecticide, Fungicide and Rodenticide Act* (FIFRA) ("unreasonable adverse effects') and *Food Quality Protection Act* (FQPA) ("reasonable certainty of no harm" or "negligible risk," based on risk assessment methodology with uncertainties and risk factors) do not ensure that there will be no harm (by definition it allows levels of risk or harm to be set); (iv) "inert" ingredients (non-disclosed ingredients in pesticide products considered proprietary information) in pesticide formulations are not fully evaluated; (v) pesticide poisonings, including short- and long-term adverse effects are not tracked by EPA; (vi) endocrine disrupting effects are not currently evaluated; and, (vii) mixtures of pesticides and between pesticides and pharmaceuticals are not evaluated.

The difficulty, from a public health perspective, is that the inadequate regulatory system, allowing widespread use of poisons that are more often than not unnecessary, results in a pesticide product label that is also inadequate, or fails in restricting use or conveying hazard information. A consumer or resident may be able to glean some acute toxicity information from a product's label in some cases. Information on the chronic or long-term effects will not be found on a pesticide label –even when EPA has that information. Consumers certainly will rarely get information as to whether the pesticide will initiate or exacerbate a respiratory condition, such as asthma, or contribute to a learning disability or behavioral disorder. This information is simply not on a pesticide label. Furthermore, if there are data gaps for certain health endpoints, or if EPA has not complied with a statutory requirement to evaluate the product for endocrine disruption, a pregnant woman, for example, would have no idea about this from a label. While the Material Safety Data Sheet is better in some respects, all this information will not be found there either. Product manufacturer information usually emphasizes their lengthy product development investment and has a history of misleading, according to GAO.

VI. Pesticide Registration Fees

It is extremely reasonable to raise fees for pesticide product registration in the District to ensure an adequately funded regulatory apparatus to carry out the provision of this important legislation. When compared to other states in the region, currently, the District has among the lowest registration fees in relation to 7 nearby states that have higher fees (e.g., the per product fees for: New York $600; New Jersey $300; Virginia $160; Connecticut $188; Rhode Island $200; Massachusetts $300; and Pennsylvania $135).

VII. Conclusion

Local jurisdictions are taking up the public health issue of pesticide use in their jurisdictions with increasing frequency. Based on hazard information and regulatory deficiencies, local governments are determined to adopt a precautionary approach, eliminate the use of registered toxic pesticides, and embrace safer practices and products that effectively manage the urban environment.

While there are some who will view this legislation as burdensome and fear that their current products may be pushed out of the marketplace, in reality, the public's health and the environment will be better protected as new, safer, cost-effective pest management practices and products take hold.

Thank you for the opportunity to testify today. We appreciate your commitment to protecting and improving health and the environment for the residents of the District of Columbia.

Addendum A
Exempt Pesticides

Exempt pesticides are any pesticide or pesticide product ingredients, which, at a minimum, have not been classified as or found to have any of the following characteristics:

(1) Toxicity Category I or II by the United States Environmental Protection Agency (EPA). These pesticides are identified by the words “DANGER” or “WARNING” on the label.

(2) A developmental or reproductive toxicant as defined by the State of California Proposition 65 Chemicals Known to Developmental or Reproductive Harm.

(3) A carcinogen, as designated by EPA’s List of Chemicals Evaluated for Carcinogenic Potential (chemicals classified as a human carcinogen, likely to be carcinogenic to humans, a known/likely carcinogen, a probable human carcinogen, or a possible human carcinogen), the International Agency for Research on Cancer (IARC), U.S.National Toxicology Program (NTP), and the state of California's Proposition 65 list. Any of the following classifications shall deem the chemical a carcinogen and unacceptable:

Known to the State of California to Cause Cancer (California)
Group A: Human Carcinogen (US EPA 1986 category)
Group B: Probably Human Carcinogen (US EPA 1986 category)
Group C: Possible Human Carcinogen (US EPA 1986 category)
Known Carcinogen (US EPA 1996 category)
Likely Carcinogen (US EPA 1996 category)
Carcinogenic to Humans (US EPA 1999 category)
Likely to be Carcinogenic to Humans (US EPA 1999 category)
Suggestive Evidence of Carcinogenicity (US EPA 1999 category)
Known to be Human Carcinogens (NTP)
Reasonably Anticipated to be Human Carcinogens (NTP)
Group 1: Carcinogenic to Humans (IARC)
Group 2A: Probably Carcinogenic to Humans (IARC)
Group 2B: Possibly Carcinogenic to Humans (IARC)

(4) Neurotoxic cholinesterase inhibitors, as designated by California Department of Pesticide Regulation or the Materials Safety Data Sheet (MSDS) for the particular chemical,

(5). Known groundwater contaminants, as designated by the state of California (for actively registered pesticides) or from historic groundwater monitoring records (for banned pesticides).

(6) Pesticides formulated as dusts, powder or aerosols, unless used in a way that virtually eliminates inhalation hazard (for example, applied to cracks or crevices and sealed after the application, or as a directed spray into the entrance of an insect nest).

(7) Nervous system toxicants, including chemicals such as cholinesterase inhibitors or chemicals associated with neurotoxicity by a mechanism other than cholinesterase inhibition, or listed on:

Toxics Release Inventory (TRI), EPA EPCRA Section 313 (Identified as "NEUR" on Table 1)

EPA Reregistration Eligibility Decisions (RED)
Insecticide Resistance Action Committee (IRAC) Mode of Action Classification:
Acetylcholine esterase inhibitors;
GABA-gated chloride channel antagonists;
Sodium channel modulators;
Nicotinic Acetylcholine receptor agonists /antagonists;
Nicotinic Acetylcholine receptor agonists;
Chloride channel activators;
Octopaminergic agonists;
Voltage-dependent sodium channel blockers; or
Neuronal inhibitors (unknown mode of action).

(8) Endocrine disruptors, which include chemicals that are known to or likely to interfere with the endocrine system in humans or wildlife, based on the European Commission (EC) List of 146 substances with endocrine disruption classifications, Annex 13 (and/or any subsequent lists issued as follow-up, revisions, or extensions).

(9) (Regarding outdoor use) Adversely affect the environment/wildlife, based on:

1. Label precautionary statements including "toxic" or "extremely toxic" to bees, birds, fish, aquatic invertebrates, wildlife or other non-target organisms, unless these organisms are the target pest and/or environmental exposure can be virtually eliminated.
2. Pesticides with ingredients with moderate or high mobility in soil, according to the Groundwater Ubiquity Score (GUS), or with a soil half-life of 30 days or more (except for mineral products). Persistence and Soil Mobility procedures appear below.

a*)* If GUS (Groundwater Ubiquity Score) cannot be found, we search for the aerobic soil half-life and soil-binding coefficient Koc. GUS is then calculated from the formula: GUS = log10(half-life)*(4 – log10 (Koc)).

(10) Have data gap or missing information in EPA registration documents, including pesticide fact sheets, or EPA reregistration eligibility decisions, which EPA is requiring the registrant to fulfill.

(11) Contaminants and metabolites recognized by EPA that violate any of the above criteria.

(12) Inert or active ingredients that are Chemicals Included on EPA's former List 1 (Inerts of Toxicological Concern) or EPA former List 2: (Potentially Toxic, High Priority for Testing).

Addendum B
Background on Health Concerns

I. Typical structural use pesticides (for buidings)

Synthetic Pyrethroids

Because of the similarities to crude pyrethrum, pyrethroids may act as dermal and respiratory allergens. Exposure to pyrethroids has resulted in contact dermatitis and asthma-like reactions. Persons, especially children, with a history of allergies or asthma are particularly sensitive, and a strong cross-reactivity with ragweed pollen has been recognized.

Pyrethroids interfere with the ionic conductance of nerve membranes by prolonging the sodium current. This stimulates nerves to discharge repeatedly causing hyper-excitability in poisoned animals. The World Health Organization explains that synthetic pyrethroids are neuropoisons acting on the axons in the peripheral and central nervous systems by interacting with sodium channels in mammals and/or insects. The main systems for metabolism include breakage of the ester bond by esterase action and oxidation at various parts of the molecule. Induction of liver microsomal enzymes has also been observed (WHO, 1999).

Endocrine Disruption and Breast Cancer**.** Many pyrethroids have also been linked to disruption of the endocrine system, which can adversely affect reproduction and sexual development, interfere with the immune system and increase chances of breast cancer. Pyrethroids contain human-made, or xenoestrogens, which can increase the amount of estrogen in the body (Garey et al., 1998). When tested, certain pyrethroids demonstrate significant estrogenicity and increase the levels of estrogen in breast cancer cells (Go et al., 1999). Because increased cell division enhances the chances for the formation of a malignant tumor in the breast, artificial hormones, like those found in pyrethroids, may increase breast cancer risk (PCBR, 1996). Some pyrethroids are classified by EPA as possible human carcinogens.

In a study examining the relationship between prenatal exposure to indoor pesticides and infant growth and development in urban families,[5] researchers at the Mount Sinai School of Medicine found higher than expected levels of pyrethroid metabolites in sample urine (compared with previous National Health and Nutrition Examination Survey (NHANES) data) which, according to the authors, may be attributed to higher exposures resulting from West Nile mosquito spray programs in the subjects' communities. Given that the half-lives of the pyrethroids in question were short, high levels of metabolites in the urine indicate continuous exposures. In this study a high percentage of women (70 percent) reported being pregnant during times of pesticide exposures. This study underscores the prevalence of prenatal exposure to pyrethroid chemicals, especially in under-represented populations.

In a similar 2002 study,[6] low level concentrations of pyrethroid pesticides were frequently (47-83%) detected in pregnant minority women in New York City, indicating widespread prenatal

[5] Berkowitz GS, Obel J, Deych E, Lapinski R, Godbold J, Liu Z, et al. 2003. Exposure to indoor pesticides during pregnancy in a multiethnic, urban cohort. *Environ Health Perspect* 111:79–84.
[6] Whyatt , R et al. 2002. Residential Pesticide Use during Pregnancy among a Cohort of Urban Minority Women *Environ Health Perspect* 110:507–514

pesticide use among minority women.

In a study conducted by researchers from the Centers for Disease Control and Prevention (CDC) in a community in the Southeastern U.S., urinary pyrethroid pesticide metabolite concentrations for children, compared to those reported in the NHANES and GerES (German Enviromental Survey) studies, were significant and substantially higher than the general populations of the U**.**S**.** and Germany.[7]

Morgan et al. in a study with 127 preschool children[8] at their homes and daycare centers found that these children were exposed to low levels of the synthetic pyrethroid permethrin from several sources. 67% of the children's urine samples contained permethrin metabolites with the highest recorded concentrating, 33.8 ng/mL. The authors note that the primary route of the children's exposure was through dietary ingestion, followed by indirect ingestion (hand to mouth action). Indirect ingestion as a method of exposure is also corroborated by EPA researchers in a 2011 study,[9] which demonstrated that surface concentration of pesticide residues were 'highly influential' on the dietary intake of children. In the Morgan study, permethrin residues were detected most often in the dust (100%) and hand wipe (>78%) samples.
Several studies have determined that dietary ingestion is a main source of children's exposure to pyrethroid pesticides.[10,11] A 2011 EPA study[12] found that pesticide residues were transferred from treated surfaces to foods, stating that, "[A]s long as pesticide levels are measureable on surfaces in children's eating environment, it can be concluded that transfer of pesticides to foods will take place." The Children Pesticide Exposure Study[13] found that children are continuously exposed to pyrethroid insecticides through their diets all year long, and that chronic exposure patterns are periodically modified by episodes of relatively high exposures from residential uses. The authors conclude that the combination of the use of pyrethroid insecticides in the household, dietary intake, and seasonal differences play a significant role in predicting children's exposure to synthetic pyrethroid insecticides.

In a 2011 study[14] published in *Pediatrics,* which investigated prenatal exposure to permethrin and piperonyl butoxide (PBO) -a synergist commonly formulated with pyrethroid- and 36-month

[7] Naeher LP, et al. 2010. Organophosphorus and pyrethroid insecticide urinary metabolite concentrations in young children living in a southeastern United States city. *Sci Total Environ.* 408(5):1145-53.
[8] Morgan MK., et al. 2007. An observational study of 127 preschool children at their homes and daycare centers in Ohio: environmental pathways to cis- and trans-permethrin exposure. *Environ Res.* 104(2):266-74.
[9] Melnyk LJ, Byron MZ . et al. 2011. Pesticides on household surfaces may influence dietary intake of children. *Environ Sci Technol.* 45(10):4594-601.
[10] Schettgen T, Heudorf U, Drexler H, Angerer J. 2002. Pyrethroid exposure of the general population-is this due to diet. Toxicol Lett 134:141–145.
[11] Heudorf U, Angerer J, Drexler H. 2004. Current internal exposure to pesticides in children and adolescents in Germany: urinary levels of metabolites of pyrethroid and organophosphorous insecticides. Int Arch Occup Environ Health 77:67–72.
[12] Melnyk LJ, et al. 2011. Influences on transfer of selected synthetic pyrethroids from treated Formica to foods. J *Expo Sci Environ Epidemiol*. 21(2):186-96.
[13] Lu C., et al. 2009. The attribution of urban and suburban children's exposure to synthetic pyrethroid insecticides: a longitudinal assessment. *J Expo Sci Environ Epidemio*l. 19(1):69-78.
[14] Horton MK et al. 2011. Impact of prenatal exposure to piperonyl butoxide and permethrin on 36-month neurodevelopment. *Pediatrics*. 127(3):e699-706

neurodevelopment, found that the synergist PBO was negatively associated with 36-month cognitive and motor development in children with a history of pre-natal exposure, as measured in umbilical cord and maternal plasma.

The CDC's Fourth *National Report on Human Exposure to Environmental Chemicals*[15] - the most comprehensive assessment to date of the exposure of the U.S. population to chemicals in our environment - finds that exposure continues to be widespread, specifically for permethrin, and other synthetic pyrethroids, cypermethrin, deltamethrin, and/or their metabolites. All were all found in greater than half of the subjects tested, with levels staying relatively constant throughout the years sampled. This data also found exposures in the young (6-11 years) recorded the highest levels of pyrethroid residue in their urine for all age groups. This further highlights the continual, baseline exposure levels in the U.S. population, which should be decreased not increased by continued use, especially in children.

Other Chronic End-Points. A cumulative risk assessment is the process of combining exposure (the amount of a pesticide to which an individual is exposed) and hazard (the health effects a pesticide could cause) from all substances that share a common mechanism of toxicity. Cumulative assessments are necessary since people are exposed to multiple pesticides that behave in a similar manner in the body through various exposure pathways -air, diet, dermal absorption, etc. According to EPA's procedures, the agency identifies a cumulative assessment group (CAG) of chemicals that are to be included in the quantification of cumulative risk needs. Several of the CAG chemicals have other serious chronic endpoints that warrant special consideration when reviewing the pyrethroid class of chemicals, although they do not fall under the scope of EPA's cumulative assessment. This is especially imperative if the agency is to recommend further uses and registrations for this class of chemicals, which it did this year. In the case of synthetic pyrethroids, the neurotoxicity endpoints require the agency's serious attention, as do other endpoints not included but identified as adverse health effects associated with the majority of the synthetic pyrethroids.

Pyrethroids are neurotoxic and their neurotoxic effects are detailed in the scientific literature. However, consider the following Table 1. Several of the common mechanism pyrethroids and those not included in this group are associated with cancer, endocrine disruption and reproductive and developmental effects, which should be taken into consideration when registering these chemicals. Several studies have detailed the endocrine disrupting and reproductive effects of pyrethroids. For instance, results from a New Zealand study show that pyrethroid metabolites are capable of interacting with the human estrogen receptor, and thus pose a risk to human health and environmental well-being by adding to the overall environmental xenoestrogen load.[16] A Japanese pilot study involving men battling infertility suggests that pyrethroid exposure level and dietary habit is a significant contributor to poor semen quality.[17]

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[15] CDC. 2009. Fourth *National Report on Human Exposure to Environmental Chemicals. Available at* http://www.cdc.gov/exposurereport/pdf/FourthReport.pdf

[16] McCarthy AR, Thomson BM, Shaw IC, and Abell AD. 2006. Estrogenicity of pyrethroid insecticide metabolites. *J Environ Monit*. 8(1):197-202.

[17] Toshima, H., et al. 2011. Endocrine disrupting chemicals in urine of Japanese male partners of subfertile couples: A pilot study on exposure and semen quality. *Int J Hyg Environ Health*. [Epub ahead of print]

Table 1. Effects Observed in Scientific Literature

Chemical Name	CAS No.	Reproductive Effects	Developmental Effects	Cancer	Endocrine Disruption
Allethrin	584-79-2				X
Bifenthrin	82657-04-3	x	x	x	X
Cyfluthrin	68359-37-5	x			
Cyhalothrin	68085-85-8			x	X
Cypermethrin	52315-07-8			x	X
Cyphenothrin	39515-40-7				
Deltamethrin	52918-63-5				X
Esfenvalerate	66230-04-4				
Etofenprox	80844-07-1				X
Fenpropathrin	39515-41-8				
Fenvalerate	51630-58-1	x			X
Fluvalinate	69409-94-5	x	x		X
Imiprothrin	72963-72-5				
Permethrin	52645-53-1	x		x	X
Phenothrin/Sumithrin	26002-80-2				X
Prallethrin	23031-36-9		x	x	
Pyrethrins (not synthetic)	8003-34-7		x	x	X
Resmethrin	10453-86-8	x	x	x	X
Tetramethrin	7696-12-0			x	
Tralomethrin	66841-25-6				

Propoxur

This toxic pesticide is a classic example of the compromise that accompanies many regulatory decisions and leaves on the market a hazardous chemical that does not have a proven need, given the efficacy of alternative pest management strategies and products. Propoxur (o-isopropoxyphenyl methylcarbamate), known by the trade name Baygon, is a carbamate insecticide first registered for indoor applications and very limited outdoor applications. It is also used on pets as a spray and in flea and tick collars. According to EPA, propoxur is moderately toxic (Toxicity Category II) for oral exposure and slightly toxic (Toxicity Category III) via the dermal and inhalation routes of exposure. Propoxur is classified as a probable human carcinogen (Group B2) by EPA, and the state of California lists it as a known human carcinogen. In 1988, prompted by concern over the carcinogenic risks for occupational exposures, EPA considered initiating a Special Review for propoxur. In 1995, the agency decided not to conduct the review, indicating that the uses that posed the greatest concern (flea dips and shampoos for pets, and total-release fogger products) had been eliminated through voluntary cancellation or label amendment. Again in February 2007, the agency granted a request from the registrant for voluntary cancellation of "all propoxur indoor spray uses [including use in crack and crevices] that may result in non-occupational exposure for children." This left the chemical on the market,

with continued use in public spaces. It has created an enforcement nightmare which results in ongoing exposure. It is also instructive that EPA has allowed this class of chemical to remain on the market, given the availability of alternatives, and common mechanism of toxicity that results in multiple exposures in multiple use scenarios of chemicals in this family. While EPA's focus on children is important, the health implications of this chemical family's use are broad across all ages and those with compromised immune and neurological systems, in addition to its carcinogenicity hazard.

Also in 2007, the agency completed its cumulative risk assessment for N-methyl carbamates chemical class, of which propoxur is a member. This was done after the agency concluded in 2004 that the N-methyl carbamate pesticides share a common mechanism of toxicity - cholinesterase inhibition.

Propoxur, like others in its chemical class, is classified as highly toxic. Symptoms of propoxur poisoning include nausea, vomiting, abdominal cramps, sweating, diarrhea, excessive salivation, weakness, imbalance, blurring of vision, breathing difficulty, increased blood pressure or 'hypertension' and incontinence. Death may result from respiratory system failure associated with propoxur exposure. In rats, propoxur poisoning resulted in brain pattern and learning ability changes at lower concentrations than those which caused cholinesterase-inhibition and/or organ weight changes. Chronic (long-term) inhalation exposure has resulted in depressed cholinesterase levels, headaches, vomiting, and nausea in humans. Chronic ingestion studies in animals have reported depressed cholinesterase levels, depressed body weight, effects to the liver and bladder, and a slight increase in neuropathy. Studies have shown that propoxur is also a mutagen, a reproductive effector and it has been found to affect the immune and endocrine systems.

Dangers of Indoor Propoxur Use. The main route of exposure to propoxur is through the dermal and inhalation route. Propoxur is detected and persists in indoor air and dust. On surfaces, pesticide residues can persist for 60 hours or longer. One study found that airborne concentrations were still detectable 33.5 hours after spraying propoxur indoors. Another determined that the volatilization of propoxur from treated surfaces increased with humidity, resulting in higher air concentrations of propoxur. In a study investigating indoor-air insecticide levels in inner-city residences, propoxur was found in over 90% of indoor air samples of homes with pregnant women. Inhalation studies have found that the effects of propoxur exposure were depressions of plasma by 20 to 30%, and of erythrocyte and brain cholinesterase activities. Dermal absorption studies, which have shown that propoxur is absorbed through the skin, also find that skin moisture (affected by high temperatures and humidity) influences the dermal uptake of propoxur. A dermal LD50 study with laboratory rats found that on the day of application of propoxur on skin, muscular fasciculations suggestive of cholinesterase inhibition were observed along with decreased motor activity.

Various monitoring studies have confirmed that indoor residential pesticide applications increase the exposure and health risks of residents. Indoor residues of pesticides have been detected in carpets, hard surfaces, walls and dust. In a study with inner city underserved mothers and newborns, propoxur levels were significantly higher in the personal air of women reporting use of an exterminator, can sprays, and/or pest bombs during pregnancy compared with women reporting no pesticide use or use of lower toxicity methods. Humans exposed to indoor

concentrations of propoxur resulting from indoor application are therefore at risk for short and long term effects mentioned above, including increased risk of developing cancer**.**

Organophosphate Pesticides

Studies Link Prenatal Organophosphate Exposure to Reduced IQ. Three independent investigations published in the journal *Environmental Health Perspectives* (EHP) in 2011 have reached similar conclusions, associating prenatal exposure to organophosphate (OP) pesticides with IQ deficits in school-age children. The fact that three research groups reached such similar conclusions independently adds considerable support to the validity of the findings.

The three studies were conducted at the School of Public Health at the University of California, Berkeley,[18] the Mailman School of Public Health at Columbia University, [19]and Mount Sinai School of Medicine.[20] All three involved cohorts of women enrolled during pregnancy. The Berkeley and Mount Sinai investigators measured OP pesticide breakdown products in the pregnant women's urine, while the Columbia investigators measured the OP pesticide chlorpyrifos in umbilical cord blood. Intelligence tests were administered to children of these mothers between ages 6 and 9 years at Mount Sinai and at age 7 years at Berkeley and Columbia.

Although the study findings are not directly comparable, all three investigations found evidence linking prenatal OP pesticide exposures with adverse effects on cognitive function that continued into early childhood. "It is well known that findings from individual epidemiologic studies may be influenced by chance and other sources of error. This is why researchers often recommend their results be interpreted with caution until they are supported by similar findings in other study populations," said EHP Editor-in-Chief Hugh A. Tilson. "As a group, these papers add substantial weight to the evidence linking OP pesticides with adverse effects on cognitive development by simultaneously reporting consistent findings for three different groups of children."

The Berkeley study, examining families in the intensive agricultural region of Salinas Valley, California, found that IQ levels for children with the most OP exposure were a full seven IQ points lower than those with the lowest exposure levels. This is a very significant drop. According to USA Today[21], lead poisoning can result in a drop of less than half that amount, usually about two to three IQ points, which is still cause for grave concern. The Berkeley team also found that every tenfold increase in measures of organophosphates detected during a mother's pregnancy corresponded to a 5.5 point drop in overall IQ scores in the 7-year-olds.

The findings of the three studies support the suggestions of recent research on a phenomenon known as "invoice dose response." This refers to the idea that it is often the timing of chemical exposure that is most important, rather than the actual degree of exposure. The studies found that

[18] http://ehp03.niehs.nih.gov/article/info:doi/10.1289/ehp.1003160, Environmental Health Perspectives, Vol. 119, No. 8, August 2011.
[19] http://ehp03.niehs.nih.gov/article/info:doi/10.1289/ehp.1003160, Environmental Health Perspectives. Vol 119, No. 8, August 2011.
[20] http://ehp03.niehs.nih.gov/article/info:doi/10.1289/ehp.1003160, Environmental Health Perspectives. Vol. 119, No. 8, August 2011.
[21] http://yourlife.usatoday.com/parenting-family/pregnancy/story/2011/04/Children-exposed-to-high-pesticde-levels-in-the-womb-have-lower-IQs-new-studies-show/46340432/1, 4-21-11.

exposure to OPs while a child was still in the womb correlated to lower IQ scores, but exposures during early childhood, even at higher amounts, did not result in similar findings.

Organophosphates, derived from World War II nerve agents, are a common class of chemicals used in pesticides and are considered to be among the most likely pesticides to cause an acute poisoning. Many are already banned in several European countries. Organophosphate pesticides are extremely toxic to the nervous system, as they are cholinesterase inhibitors and bind irreversibly to the active site of an enzyme essential for normal nerve impulse transmission. In finally responding to concerns stemming from this information, EPA reached agreements with chemical manufacturers to phase out residential use of two common organophosphate pesticides, chlorpyrifos and diazinon, in 2000 and 2002 respectively. However, chlorpyrifos continues to be used on golf courses and for public health mosquito control, and both pesticides remain registered for use in agriculture.

II. Turf and Landscape Pesticides

Below is a summary of chemical concerns among some of the pesticides that are or may be used in turf and landscape management:

Clopyralid

Clopyralid is a carboxylic acid herbicide classified by EPA in acute toxicity class III as slightly toxic. Laboratory studies have shown that clopyralid is a severe eye irritant,[22,23,24] and dermal irritation has also been noted which can lead to skin sensitization for prolonged skin exposures.[25] Some developmental and reproductive effects have been observed in laboratory animals. The livers and kidneys of rats as well as the livers of dogs were affected by changes in weight and decreased red blood cell counts.[26] Another study found that weights of rabbit fetuses decreased at both low and high doses of clopyralid. Skeletal abnormalities were also observed in these fetuses at all doses.[27] Clopyralid is persistent in soil, with a recorded half-life of 2-14 months,[28] and is degraded primarily by microbial activity. However, it does not readily bind to soil and is highly soluble in water. As a result, it is very mobile in soil and has the potential to move towards groundwater and contaminate surface water. Persistence of clopyralid in vegetation was evidenced by the contamination of composts from harvested lawn and grass clippings sprayed with clopyralid which severely damaged garden crops in Washington State in 2000. In 2002, the state banned the use of clopyralid on lawns and turf in order to keep the chemical from contaminating compost supplies.[29] That same year, California found that 65% of the composts samples tested positive for clopyralid, which led to the cancellation of residential uses for clopyralid in the state.[30]

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[22] Cox, C. 1998. *Clopyralid - Herbicide Fact Sheet*. Journal of Pesticide Reform. 18(4).
[23] OSU Extension Service. 2002.*Clopyralid- Pesticide Fact Sheet: Forestry Use.*
[24] Tu, M., et al. 2001. *CLOPYRALID*. Weed Control Methods Handbook. The Nature Conservancy.
[25] Cox, C. 1998. *Clopyralid - Herbicide Fact Sheet*. Journal of Pesticide Reform. 18(4).
[26] *Ibid.*
[27] *Ibid.*
[28] Washington State University Cooperative Extension. *Clopyralid Herbicide and Compost*. Agriculture and Natural Resources Fact Sheet #538.
[29] WSDA. 2002. *Clopyralid in Compost Facts: Herbicide contamination raises problems for compost*.
[30] de la Fuente, M. *Clopyralid and Compost in California*. University of California Cooperative

Triclopyr

Triclopyr is a pyridine compound herbicide which mimics plant growth hormones called auxins, interfering with the normal plant growth response. It is readily absorbed through both roots and leaves, and is systemic, translocating throughout the plant. Triclopyr is classified as slightly toxic (class III) by EPA, though there are documented effects on reproduction, fetal development, and organ function, as well as irritation when exposed to high doses. The substance is acutely toxic to the eyes, causing severe irritation.[31] Subchronic and chronic feeding studies in dogs and rodents found damage to both the kidney and liver.[32] Data has also shown birth defects and adverse effects on reproduction associated with chronic exposure. Pregnant rodents exposed to the chemical had lower litter numbers and a higher incidence of birth abnormalities in offspring than did those which were not exposed.[33] Triclopyr is generally considered to be non-carcinogenic, but several laboratory tests have shown an increase in the incidents of breast cancer and genetic damage in rat embryos.[34] Triclopyr is also toxic to a range of wildlife, and its persistence can vary greatly depending on the application site, but some studies have shown it to persist longer than a year under certain conditions.[35]

2,4-D

2,4-D is one of the most widely used herbicides for the control of broadleaf weeds in commercial agriculture and residential landscapes in the U.S. About 46 million pounds of 2,4-D are used annually, with 16 million pounds used in non-agricultural settings, including parks, playing fields, and residential lawns.[36] Its health risks prompted a Special Review in 1985. A few years later in a unique move, several large pesticide companies with a common interest in keeping 2,4-D on the market formed a task force to keep the herbicide on the market. In 2007, EPA reversed its decision did not complete the Special Review.[37] In 2008, the Natural Resources Defense Council petitioned EPA to cancel all uses of 2,4-D. Once part of the deadly duo of chemicals that made up Agent Orange, can also be contaminated with several forms of dioxin, including 2,3,7,8-TCDD, a known carcinogen. Studies have also documented that once tracked indoors from lawns, 2,4-D can stay indoors (on carpets) for up to a year.[38,39]

Extension.

[31] C. Cox. 2000. *Triclopyr: Herbicide Fact Sheet.* Journal of Pesticide Reform 20(4).

[32] U.S. EPA. Prevention, Pesticides and Toxic Substances. 1998. Reregistration eligibility decision (RED): Triclopyr. Washington, D.C.

[33] *Ibid*.

[34] U.S. EPA. Office of Prevention, Pesticides, and Toxic Substances. 1996. Carcinogenicity peer review of triclopyr. Memo from McMahon, T.F., and E. Rinde, Health Effects Div., to R. Taylor, Registration Div. and T. Luminello, Special Review and Reregistration Div. Washington, D.C.

[35] Norris, L.A., M.L. Montgomery, and L.E. Warren. 1987. Triclopyr persistence in western Oregon hill pastures. Bull. Environ. Contam. Toxicol. 39: 134-141.

[36]USEPA. 2005 Reregistration Eligibility Decision (RED) 2,4-D Factsheet. Office of Prevention, Pesticides and Toxic Substances. Washington DC http://www.epa.gov/oppsrrd1/REDs/factsheets/24d_fs.htm

[37] Beyond Pesticides Daily News. 2007 http://www.beyondpesticides.org/dailynewsblog/?p=161

[38] Nishioka MG, Burkholder HM, Brinkman MC, Gordon SM. 1996. Measuring lawn transport of lawn applied herbicide acids from turf to home: Correlation of dislodgeable 2,4-D turf residues with carpets dust and carpet surface residues. *Environmental Sci and Tech*. 30:3313-3320.

[39] Rudel, Ruthann, et al. "Phthalates, Alkylphenols, Pesticides, Polybrominated Diphenyl Ethers, and Other Endocrine-Disrupting Compounds in Indoor Air and Dust." *Environmental Science and*

Several scientific studies have been presented to the agency that point to 2,4-D's association with cancer, for example non-Hodgkin's lymphoma.[40],[41] Nevertheless, the agency concluded that "the data are not sufficient to conclude that there is a cause and effect relationship between exposure to 2,4-D and non- Hodgkin's lymphoma."[42] 2,4-D was then classified as a Group D, not classifiable as to human carcinogenicity. Studies from the National Cancer Institute and other sources have reported an association between exposure to lawn chemicals, like 2,4-D, and adverse impacts in dogs.

There is also a wealth of relevant scientific information available indicating that 2,4-D has endocrine disrupting activity. A study by Garry et al. found a direct correlation of urinary levels of 2,4-D with serum levels of luteinizing hormone (LH) and high testosterone levels at the time of highest exposure suggest a direct effect on hormonal levels by the chlorophenoxy herbicide. [43] LH, produced by the pituitary gland, stimulates the production of testosterone and helps regulate the menstrual cycle and ovulation. Fluctuations in these hormones may affect human fertility. Others found that abnormal sperm[44] and higher rates of birth defects[45] were observed in farmers with long-time exposure to 2,4-D. Animal studies have also observed the hormone effects of 2,4-D exposure. Xie et al. (2005) observed estrogenic activity in rainbow trout[46] exposed to 2,4-D, while another study found the thyroid glands of laboratory rats were sensitive to 2,4-D as decreases in the thyroid gland transport and production functions, and impairment of hormone iodination in the thyroid were observed after acute exposure.[47] Other studies have found that 2,4-D promotes the proliferation of androgen-sensitive cells by acting synergistically with its main metabolite, 2,4-dichlorophenol (DCP), also known for its endocrine disrupting effects.[48,49]

Technology 37(20):4543-4553.

[40] Lennart Hardell, and Eriksson, M. (1999) A case-control study of non-Hodgkin lymphoma and exposure to pesticides. *Cancer* 85, 1353-1360.

[41] Ibrahim, M.A., Bond, G.G., Burke, T.A., Cole, P., Dost, F.N., Enterline, P.E., Gough, M., Greenberg, R.S., Halperin, W.E., McConnell, E., and et al.1991. Weight of the evidence on the human carcinogenicity of 2,4-D. *Environmental Health Perspectives* 96, 213-222.

[42] U.S. EPA. 2005. Reregistration Eligibility Decision for 2,4-D. Office of Prevention Pesticides and Toxic Substances. Washington DC

[43] Garry, V.F., Tarone, R.E., Kirsch, I.R., Abdallah, J.M., Lombardi, D.P., Long, L.K., Burroughs, B.L., Barr, D.B., and Kesner, J.S. 2001. Biomarker correlations of urinary 2,4-D levels in foresters: genomic instability and endocrine disruption. *Environmental Health Perspectives* 109, 495-500.

[44] Lerda, D., and Rizzi, R. 1991. Study of Reproductive Function in Persons Occupationally Exposed to 2,4-Dichlorophenoxyacetic Acid (2,4-D). *Mutation Research* 262, 47-50.

[45] Garry, V.F., Schreinemachers, D., Harkins, M.E., and Griffith, J. 1996. Pesticide Appliers, Biocides, and Birth Defects in Rural Minnesota. *Environmental Health Perspectives* 104, 394-399.

[46] Xie, L.T., Thrippleton, K., Irwin, M.A., Siemering, G.S., Mekebri, A., Crane, D., Berry, K., and Schlenk, D. 2005. Evaluation of estrogenic activities of aquatic herbicides and surfactants using an rainbow trout vitellogenin assay. *Toxicol. Sci*. 87, 391-398.

[47] Malysheva, L.N., and Zhavoronkov, A.A. 1997. Morphological and histochemical changes in the thyroid gland after a single exposure to 2,4-DA herbicide. *Bull. Exp. Biol. Med.* 124, 1223-1224.

[48] Kim, H.-J., Park, Y.I., and Dong, M.S. 2005. Effects of 2,4-D and DCP on the DHT-Induced Androgenic Action in Human Prostate Cancer Cells. *Toxicological Sciences*. 88(1), 52–59 pp. 52-59.

[49] McKinlay, R., Plant, J.A., Bell, J.N.B., and Voulvoulis, N. 2008. Endocrine disrupting pesticides: Implications for risk assessment. *Environment International* 34, 168-183.

Dicamba

Dicamba, originally registered in 1967, is a benzoic acid herbicide used in agricultural, industrial and residential settings. This neurotoxic pesticide is linked to organ damage and reproductive effects.[50] A 1992 study of farmers by the National Cancer Institute found that exposure to dicamba approximately doubled the farmers' risk of contracting the cancer non-Hodgkin's lymphoma two decades after exposure.[51] Symptoms of poisoning include muscle cramps, shortness of breath, nausea, vomiting, skin rashes, loss of voice, and swollen glands. It has attracted attention because of the toxicity of its contaminants, both dioxin and nitrosamines, and its propensity to leach through soil.

Glyphosate

Glyphosate is also cause for continued concern, as it has been linked to a number of serious human health effects, including increased cancer risk,[52] neurotoxicity, and birth defects,[53] as well as eye, skin, and respiratory irritation. One of the inert ingredients in product formulations of Roundup, polyoxyethyleneamine (POEA), kills human embryonic cells.[54]

It is also of particular concern due to its toxicity to aquatic species as well as instances of serious human health effects from acute exposure. EPA acknowledges that glyphosate has the potential to contaminate surface water because it does not readily break down in water or sunlight. Due to glyphosate's potential for water contamination, the agency has established a maximum contaminant level (MCL) for glyphosate (0.7ppm).[55] The agency lists the short- and long-term health effects for drinking water exposures: for relatively short periods of time, congestion of the lungs and increased breathing rate; for lifetime exposure at levels above the maximum contaminant level (MCL) kidney damage and reproductive effects.

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[50] Cox, Caroline. 1994. Northwest Coalition for Alternatives to Pesticides (NCAP), Pesticide Factsheets. http://www.pesticide.org/get-the-facts/pesticide-factsheets/factsheets/dicamba

[51] Cantor, K.P. 1992. Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among men in Iowa and Minnesota. *Cancer Res.* 52:2447-2455.

[52] M. Eriksson, et. al. 2008. Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis. *International Journal of Cancer*. 123(7): 647-1663 http://onlinelibrary.wiley.com/doi/10.1002/ijc.23589/abstract

[53] M. Antoniou, et. al. 2011. "Roundup and birth defects: Is the public being kept in the dark?" *Earth Open Source*. *http://www.scribd.com/doc/57277946/RoundupandBirthDefectsv5*

[54] Benachour, et. al. 2009. "Glyphosate Forumlations Induce Apoptosis and Necrosis in Human Umbilical, Embryonic, and Placental Cells. 22(1): 97-105 http://pubs.acs.org/doi/abs/10.1021/tx800218n

[55] USEPA. 1993. Reregistration Eligibility Decision (RED) Glyphosate. Office of Prevention, Pesticides and Toxic Substances. Washington DC

Addendum C

Children and Pesticides Don't Mix

Children are especially vulnerable to pesticides

- The National Academy of Sciences reports that children are more susceptible to chemicals than adults and estimates that 50% of lifetime pesticide exposure occurs during the first five years of life.[1]
- EPA concurs that children take in more pesticides relative to body weight than adults and have developing organ systems that are more vulnerable and less able to detoxify toxic chemicals.[2]
- Infants crawling behavior and proximity to the floor account for a greater potential than adults for dermal and inhalation exposure to contaminants on carpets, floors, lawns, and soil.[3]
- Children with developmental delays and those younger than six years are at increased risk of ingesting pesticides through nonfood items, such as soil.[4]
- Studies find that pesticides such as the weedkiller 2,4-D pass from mother to child through umbilical cord blood and breast milk.[5]
- Consistent observations have led investigators to conclude that chronic low-dose exposure to certain pesticides might pose a hazard to the health and development of children.[6]
- The World Health Organization (WHO) cites that over 30% of the global burden of disease in children can be attributed to environmental factors, including pesticides.[7]

Children, cancer and pesticides

- The probability of an effect such as cancer, which requires a period of time to develop after exposure, is enhanced if exposure occurs early in life.[8]
- A study published in the *Journal of the National Cancer Institute* finds that household and garden pesticide use can increase the risk of childhood leukemia as much as seven-fold.[9]
- Studies show that children living in households where pesticides are used suffer elevated rates of leukemia, brain cancer and soft tissue sarcoma.[10]
- Pesticides can increase susceptibility to certain cancers by breaking down the immune system's surveillance against cancer cells. Infants and children, the aged and the chronically ill are at greatest risk from chemically-induced immune suppression.[11]
- A study published by the American Cancer Society finds an increased risk for non-Hodgkin's lymphoma (NHL) in people exposed to common herbicides and fungicides, particularly the weedkiller mecoprop (MCPP). People exposed to glyphosate (Roundup®) are 2.7 times more likely to develop NHL.[12]
- 75 out of all 99 human studies done on lymphoma and pesticides find a link between the two.[13]
- Four peer-reviewed studies demonstrate the ability of glyphosate-containing herbicides to cause genetic damage to DNA (mutagenicity), even at very low concentration levels.[14]
- A 2007 study published in *Environmental Health Perspectives* finds that children born to mothers living in households with pesticide use during pregnancy had over twice as much risk of getting cancer, specifically acute leukemia (AL) or non-Hodgkin lymphoma (NHL).[15]
- A 2007 Canadian report shows that a greater environmental risk exists for boys, specifically when it comes to cancer, asthma, learning and behavioral disorders, birth defects and testicular dysgenesis syndrome.[16]

Commonly Used Chemicals

Chemical	Common Use	Health Effects
2,4-D	Lawns	c, ed, r, n, kl, si, bd
Dicamba	Lawns	r, n, kl, si, bd
Fipronil	Indoor/outdoor baits, pet care	c, ed, n, kl, si
Glyphosate	Lawns	c, r, n, kl, si
Permethrin	Mosquitoes, head lice, garden	c, ed, r, n, kl, si

Key: Birth/developmental defects=bd; Kidney/liver damage=kl; Sensitizer/irritant=si; Cancer=c; Neurotoxicity=n; Endocrine Disruption=ed; Reproductive effects=r

Alternatives
Reduce exposure to toxic chemicals by adopting sound organic or integrated pest mangement (IPM) practices that use cultural, mechanical and biological methods of control and least-toxic chemicals only as a last resort. An organic diet limits children's pesticide exposure and toxic body burden.