Safe Applications of Pesticides and Herbicides

“Health Effects of Chronic Pesticide Exposure”

I will present to and share with City Council the understanding of long-term health effects of chronic pesticide exposure with an emphasis on Parkinson’s disease and other neurodegenerative diseases with another emphasis on the risk cup.

By: John Hamilton

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Pesticide Exposure in Children

COUNCIL ON ENVIRONMENTAL HEALTH

Pediatrics 2012;130;e1757; originally published online November 26, 2012;
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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

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

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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.[9,11,12] 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]

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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

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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

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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

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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)."

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

Data Sources for Poisoning Incidents

Table 1. SENSOR-Pesticides Program

Table 2. National Poison Data System

Table 3. California Pesticide Illness and Surveillance Program

TABLE 1

PESTICIDES MOST OFTEN IMPLICATED IN ACUTE OCCUPATIONAL PESTICIDE-RELATED ILLNESS AND INJURY CASES AND NUMBER OF CASES, SENSOR-PESTICIDES PROGRAM, 2005-2009 (N=9,906)

Rank	Pesticide Category	Number of Exposed Cases				Sum of Single + Multiple Exposure Cases* (n=9,906 individuals)	
		Exposed to Single Substance (n=6,187 individuals)		Exposed to Multiple Substances* (n=3,719 individuals)			
		n	%	n	%	n	%
1	Pyrethroids	1,368	22.10	1,479	39.80	2,847	28.70
2	Chlorinated compounds	1,174	19.00	387	10.40	1,561	15.80
3	Organophosphorous compounds	600	9.70	429	11.50	1,029	10.40
4	Pyrethrins	358	5.80	620	16.70	978	9.90
5	Glyphosate	274	4.40	203	5.50	477	4.80
6	Ammonium/ammonia	32	0.50	361	9.70	393	4.00
7	N-methyl carbamates	249	4.00	112	3.00	361	3.60
8	DEET	292	4.70	59	1.60	351	3.50
9	Sulfur compounds	145	2.30	143	3.80	288	2.90
10	Triazines	168	2.70	60	1.60	228	2.30
11	Fipronil	26	0.40	135	3.60	161	1.60
12	Naphthalene	113	1.80	22	0.60	135	1.40
13	Imidacloprid	1	0.00	118	3.20	119	1.20
14	Thiocarbamates/ Dithiocarbamates	67	1.10	31	0.80	98	1.00
15	Glutaraldehyde	51	0.80	15	0.40	66	0.70
	All other	1,269	20.50	1,287	34.60	2,556	25.80
TOTAL INDIVIDUALS		**6,187**	**100.00**	**3,719**	**100.00**	**9,906**	**100.00**

**Because some of the individuals exposed to multiple substances appear in the totals of more than one pesticide category, the sum of the pesticide categories exceeds the number of individuals.*

Source: Edward J. Kasner, MPH and Geoffrey M. Calvert, MD, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

What Are the Potential Health Effects of Pesticides?

At a Glance

- Toxicity is the ability of a chemical to cause harm to health. The amount needed to cause harm depends on the chemical.
- With most pesticides, the longer you are exposed the greater the chance of harm.
- People can be exposed by breathing a pesticide, getting it into the mouth (by eating or drinking, for example), or by contact with the skin or eyes.
- Some people are more at risk than others, depending on their age, gender, individual sensitivity, or other factors.

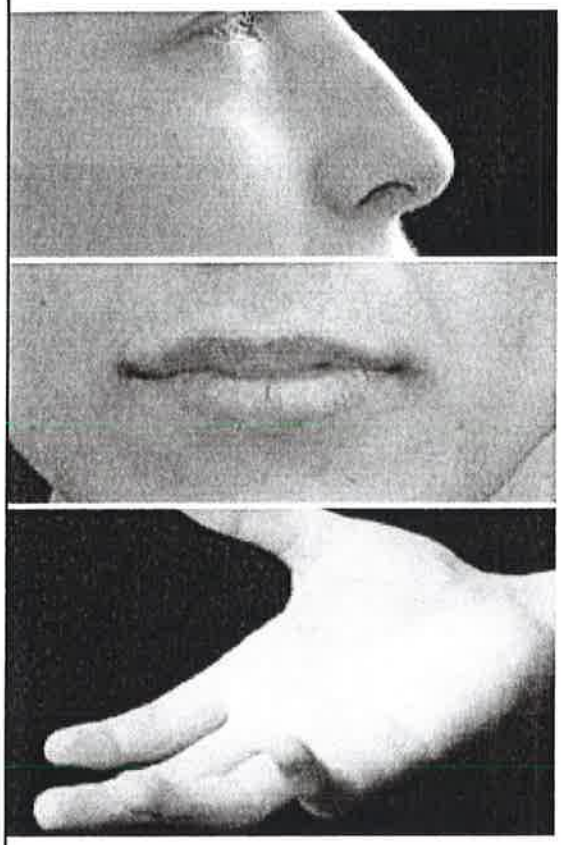
With most (but not all) pesticides, the more a person is exposed to a particular substance, the greater the chance of harm. Two aspirin may get rid of your headache, but a bottle will make you sick.

Although pesticides are intended to harm only the target pest, if not used correctly, they can also harm people or the environment.

The presence of a pesticide in the environment is not necessarily a problem, but it may be a source of exposure. As with all toxic substances, whether the exposure causes harm depends on the dose, how someone is exposed, how sensitive an individual may be to that toxin, and the toxicity of the pesticide involved.

What do you mean, "Risk = Toxicity + Exposure"?

Toxicity is the capacity of a chemical to cause harm to health. The amount needed to cause harm depends on the chemical. Like other chemicals, some pesticides are more toxic than others. A small quantity of a highly toxic pesticide



Humans can be exposed to pesticides by breathing it, getting it into their mouth, or absorbing it through the skin.

can cause great harm, but almost any substance can cause harm in large enough doses. That's why, when we consider risk, we consider both the toxicity and your exposure to it (how, how much, how long).

With most (but not all) pesticides, the more a person is exposed to a particular substance, the greater the chance of harm. Two aspirin may get rid of your headache but an entire bottle will make you sick. And for some people, aspirin may be harmful even at low doses. The degree of harm depends on the chemical, the situation, and the person. The same is true of pesticides. Very small amounts of even the most toxic materials may do no or immeasurably small harm. Less toxic materials in large amounts can cause great harm.

How are people exposed to pesticides?

People can be exposed to pesticides in three ways:

- Breathing (inhalation exposure).
- Getting it into the mouth or digestive tract (oral exposure).
- Contact with the skin or eyes (dermal exposure).

Pesticides can enter the body by any one or all three of these routes. Inhalation exposure can happen if you breathe air containing pesticide as a vapor, as an aerosol, or on small particles like dust. Oral exposure happens when you eat food or drink water containing pesticides. Dermal exposure happens when your skin is exposed to pesticides. This can cause irritation or burns. In more serious cases, your skin can absorb the pesticide into the body, causing other health effects.

Some pesticides evaporate more easily than others so they are more likely to be inhaled. Some break down quickly on surfaces; others last longer. A pesticide applied as a liquid spray may drift more easily than dry granules. A pesticide sealed in a trap is less likely to be eaten accidentally by a child or a pet. A dry pesticide plowed into the soil can be dangerous to groundwater, but is not as likely to drift through the air. All these factors affect the potential risk of human exposure and are considered when DPR makes rules for pesticide use.

Does how long you are exposed make a difference?

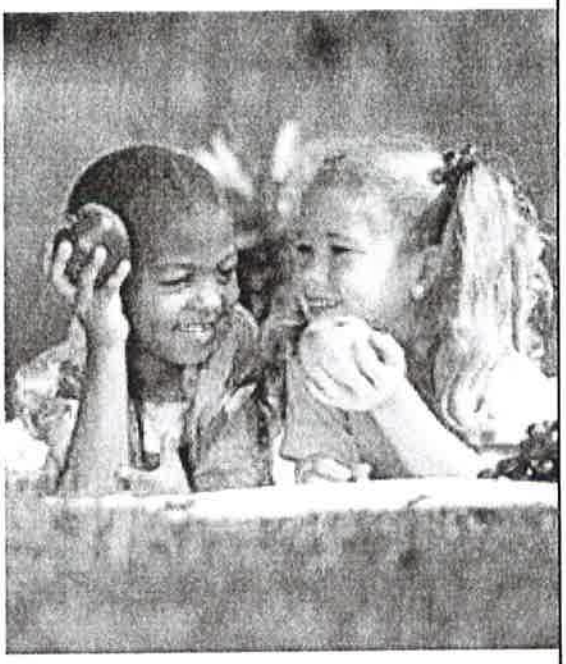
Generally, the longer or more often a person is exposed to a given amount of a pesticide, the greater the chance of harm. We at DPR consider both how long and how often someone might be exposed when we develop rules for pesticide use. For example, we know that fumigant pesticides slowly escape into the air during the hours or days after an application. (Fumigants are gaseous pesticides injected into the soil or released into buildings.) When we evaluate controls on fumigant use, we work to make sure people near an application, including workers and nearby residents, are protected over both short and long periods.

Are some people more at risk than others?

Pesticides affect different people differently. Children may be more sensitive to some pesticides than adults. Compared to adults, they breathe in more air and eat more food relative to their body size, increasing their exposure. When they play on floors or lawns or put objects in their mouths, they increase their chance of exposure to pesticides used in yards or lawns. Also, their developing bodies may not break down some chemicals as effectively as adults.

People of any age with asthma or other chronic diseases may be more likely than healthy individuals to get sick after pesticide exposure. Some individuals are also more sensitive to the odor or other irritant effects of certain pesticides.

But no matter what their individual sensitivities, people in the greatest danger of pesticide illness are those whose exposure is highest, such as workers who mix or apply pesticides. People who use pesticides in their homes may also be overexposed and become ill, especially if they do not carefully follow the directions on the product label. People living near agricultural fields are more likely than urban residents to be exposed to farm chemicals (although their exposure may not necessarily be high enough to cause harmful effects).



Because they are smaller, children get a larger dose from a given exposure to pesticides. Regulators take this into consideration when they develop restrictions on pesticide use.

Pesticide Illness Surveillance Program

Back to Pesticides and Human Health Information
Back to Worker Health and Safety Branch Programs

Since 1971, California law requires physicians to report any known or suspected illness caused by a pesticide exposure. The Pesticide Illness Surveillance Program (PISP) is tasked with collecting and evaluating these reports before they are assigned to county agricultural commissioners to investigate the exposure circumstances. Scientists then review the collected information and enter it in a database. This data not only reflects the effectiveness of the California's pesticide regulatory program but also identifies areas for improvement. PISP data is also used by the Exposure Monitoring and Health Investigation Program, Industrial Hygiene Program, Human Health Assessment and Human Health Mitigation Program. A handout, PDF (124 kb) describes the PISP program in more detail.

California PISP Annual Reports

Click arrow to select year.

California Pesticide Illness Query (CalPIQ)

- California Pesticide Illness Query - Generate customized information from the Pesticide Illness Surveillance Program database.
- Data dictionary, PDF (344 kb) for the CalPIQ and PISP Annual Reports

Related Links

- How to file a pesticide illness report - The Office of Environmental Health Hazard Assessment oversees physician reporting.
- Pesticide illness surveillance programs in other states
- Information for physicians - Resources for health care providers

For content questions, contact:
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NPIC provides objective, science-based information about pesticides and pesticide-related topics to enable people to make informed decisions about pesticides and their use. NPIC is a cooperative agreement between Oregon State University and the U.S. Environmental Protection Agency.

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2. To operate a toll-free, bi-lingual telephone information service for all callers in the United States and its territories, Monday through Friday at least 4 hours per day, with accessibility to voicemail during closed hours, and ability to address inquiries through e-mail and social media;
3. To develop and maintain English and Spanish websites accessible to broad audiences and host NPIC original content, state-of-the-art information technology tools and links to unbiased and authoritative sources of information about pesticides;
4. To collect robust pesticide incident data through systematic protocols and to disseminate the information through scheduled reporting and by request from U.S. EPA and partner agencies;
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Pesticides linked to Parkinson's Disease

* dieldrin, aldrin, endrin (OC), b-HCH(OC), lindane (OC), heptoclor(O
paraquat, paraquat dicloride (OC), *endosulfan(OC), diquat bromide
permethrin, cypermethrin, deltamethrin (P), rotenone (B),
glyphosate (OP), chlorpyrifos (OP), diazinon (OP), parathion (OP)
methomyl (C), zineb (C), mancozeb (C), ziram (C), atrazine (T),
* benomyl (O), propargite ( ), 2,4-D (Cl), pyridaben ( ), trifluralin (F)
fenazaquin ( ), fenpyroximate ( ), *maneb ( ), chlorfenvinphos (OP)
cyanazine ( ), alachor ( ), metolachlor ( ), metam ( ), broxomy(
* "furadan" (carbofuran) ( ), malathion (OP),

OC = organoclorines
OP = organophosphates
C = carbamates
B = biological

P = pyrethroids / pyrethrins

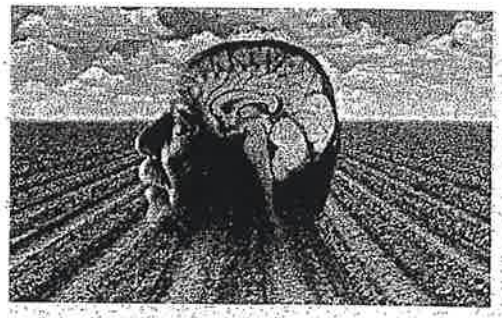
* registration cancelled

John Hamilton, PRC-NM
tel. (575) 526-2042

Pesticides and Parkinson's: UCLA researchers uncover further proof of a link

Study suggests potential new target in fight against debilitating disease

By Mark Wheeler January 03, 2013



For several years, neurologists at UCLA have been building a case that a link exists between pesticides and Parkinson's disease. To date, paraquat, maneb and ziram — common chemicals sprayed in California's Central Valley and elsewhere — have been tied to increases in the disease, not only among farmworkers but in individuals who simply lived or worked near fields and likely inhaled drifting particles.

Now, UCLA researchers have discovered a link between Parkinson's and another pesticide, benomyl, whose toxicological effects still linger some 10 years after the chemical was banned by the U.S. Environmental Protection Agency.

Even more significantly, the research suggests that the damaging series of events set in motion by benomyl may also occur in people with Parkinson's disease who were never exposed to the pesticide, according to Jeff Bronstein, senior author of the study and a professor of neurology at UCLA, and his colleagues.

Benomyl exposure, they say, starts a cascade of cellular events that may lead to Parkinson's. The pesticide prevents an enzyme called ALDH (aldehyde dehydrogenase) from keeping a lid on DOPAL, a toxin that naturally occurs in the brain. When left unchecked by ALDH, DOPAL accumulates, damages neurons and increases an individual's risk of developing Parkinson's.

The investigators believe their findings concerning benomyl may be generalized to all Parkinson's patients. Developing new drugs to protect ALDH activity, they say, may eventually help slow the progression of the disease, whether or not an individual has been exposed to pesticides.

The research is published in the current online edition of Proceedings of the National Academy of Sciences.

Parkinson's disease is a debilitating neurodegenerative disorder that affects millions worldwide. Its symptoms — including tremor, rigidity, and slowed movements and speech — increase with the progressive degeneration of neurons, primarily in a part of the mid-brain called the substantia nigra. This area normally produces dopamine, a neurotransmitter that allows cells to communicate, and damage to the mid-brain has been linked to the disease. Usually, by the time Parkinson's symptoms manifest themselves, more than half of these neurons, known as dopaminergic neurons, have already been lost.

While researchers have identified certain genetic variations that cause an inherited form of Parkinson's, only a small fraction of the disease can be blamed on genes, said the study's first author, Arthur G. Fitzmaurice, a postdoctoral scholar in Bronstein's laboratory.

"As a result, environmental factors almost certainly play an important role in this disorder," Fitzmaurice said. "Understanding the relevant mechanisms — particularly what causes the selective loss of dopaminergic neurons — may provide important clues to explain how the disease develops."

Benomyl was widely used in the U.S. for three decades until toxicological evidence revealed it could potentially lead to liver tumors, brain malformations, reproductive effects and carcinogenesis. It was banned in 2001.

The researchers wanted to explore whether there was a relationship between benomyl and Parkinson's, which would demonstrate the possibility of long-lasting toxicological effects from pesticide use, even a decade after chronic exposure. But because a direct causal relationship between the pesticide and Parkinson's can't be established by testing humans, the investigators sought to determine if exposure in experimental models could duplicate some of the pathologic features of the disease.

They first tested the effects of benomyl in cell cultures and confirmed that the pesticide damaged or destroyed dopaminergic neurons.

Parkinson's
Disease
Foundation

Environmental Factors and Parkinson's: What Have We Learned?

By Caroline M. Tanner, M.D., Ph.D.
Originally published in the Spring 2011 issue of PDF's Newsletter, News & Review.

Scientists generally agree that most cases of Parkinson's disease (PD) result from some combination of nature and nurture — the interaction between a person's underlying genetic make-up and his or her life activities and environmental exposures. A simple way to describe this is that "genetics loads the gun and environment pulls the trigger." In this formulation, "environment" has a very broad meaning — that is, it refers to any and all possible causes other than those that are genetic in origin.

The interactions between genes and environment can be quite complex. Some environmental exposures may lower the risk of PD, while others may increase it. Similarly, some people have inherited a genetic makeup that makes them more or less susceptible to the effects of toxicants, or poisonous agents, than others. The effect of a combined exposure can be greater — or lower — than a single exposure. All of this means that the particular combination of factors leading to PD is likely to be unique for each person. These combinations, in different ways, may trigger a common series of biological changes that will ultimately lead to the disease.

Scientists are beginning to tease apart the non-genetic factors that influence PD risk. In particular, epidemiologists are working to identify differences in the experiences of people who develop PD, compared to those who do not. But identifying these risk factors can be difficult. And when we do identify them, they serve only as clues. They do not provide a direct explanation for the cause of Parkinson's, so scientists must supplement these population studies with laboratory experiments.

The following is a list of some of the risk factors for which we have found some evidence of an association with PD. For the most part, it is too soon to make recommendations for how to prevent Parkinson's based on this research. However, these results may help us to understand the causes of PD, and provide direction for future research and therapy development.

Potential Risk Factors

Age. About one percent of people over age 60 have Parkinson's disease, compared with just 0.001 percent of people 45 or younger.

Gender. Parkinson's is more common in men than in women. It is not known whether this is due to genetic factors, hormones or differences in behavior.

Head Injury. Traumatic brain injury — injury that results in amnesia or loss of consciousness — has been associated with an increased risk of developing Parkinson's years after the injury. Laboratory studies suggest that such injury may provoke inflammation in the brain, which could lead to the development of PD.

Area of Residence. There are differences in the geographic distribution of PD. These could be due to differences in environmental factors, some of which are referenced below, and differences in genetic risk factors. Alternatively, they could be traced to differences in the methods that are used to count people with PD. While studies are too few to provide definitive patterns, some have been suggested. For example, Parkinson's prevalence is higher in the Inuit population in Denmark than it is among other Danes, possibly reflecting a greater dietary intake among the Inuits of persistent organic pollutants such as polychlorinated biphenyls, or PCBs (see page 7). In the agricultural California central valley, living in a home near to fields where the pesticides paraquat and maneb were used was associated with PD in one report. Another study reported greater incidence of PD in urban areas with high levels of industrial emissions of the metal manganese, and possibly copper (see more on page 7).

Occupation. Certain occupational categories or job titles have been associated with a higher incidence of PD, but results have been inconsistent. The relationship between welding (the process of fusing substances, usually metals) and PD has been a recent focus of controversy. In some reports for example, studies of people who are referred for medicolegal evaluation (an examination to determine the legal aspects of a workplace) welding has been suggested to cause Parkinson's symptoms or earlier onset of PD.

However, in most other studies, including several in large national occupational and disease registries, welding has not been associated with PD risk.

A higher frequency of PD has been associated with many other occupations, but only a few occupations have been associated with PD in multiple studies, including agricultural and industrial workers. By contrast, lower rates of Parkinson's are associated with shift work and jobs involving vigorous physical work. While we can hypothesize that the agricultural or industrial jobs may involve greater exposure to toxicant chemicals, further study in other populations is needed to understand if certain occupations are actually associated with a higher risk of PD. Some of the studies investigating specific toxicant exposures are described in the next sections.

Pesticide Exposure. Of all the chemical exposures that have been linked to Parkinson's, pesticides have been reported the most consistently. Recent research has shown higher rates of Parkinson's among people who were exposed to pesticides over a long period of time as part of their work. Investigating other types of pesticide exposure, such as home use, is more challenging. However, hobby gardening and home pesticide use have each been associated with PD in one report. Although few studies have identified specific pesticides as leading to PD, those that have been so identified include the insecticides rotenone and permethrin (used in clothing and mosquito netting to kill mosquitos); organochlorines such as beta-hexachlorocyclohexane (beta-HCH — used in the United States from the 1950s to the 1970s); and the herbicides paraquat and 2,4- dichlorophenoxyacetic acid (2,4-D). It is important to note that most people who are exposed to these pesticides do not go on to develop Parkinson's. The herbicide 2,4-D is one of the chemicals making up Agent Orange, used as a defoliant during the Vietnam war era. Although Agent Orange has not been proven to cause PD, the US Department of Veterans Affairs has ruled that veterans with PD who served in Vietnam between January 9, 1962 and May 7, 1975 are eligible to receive disability compensation from the Veterans Administration.

Exposure to Metals. Occupational exposures to various metals have been suggested to be related to the development of PD. But long-term exposure to metals is not easily measured, and the results of studies measuring PD risk and specific metals have been inconsistent. For example, high dose manganese exposure, a metal mentioned earlier, is known to cause a form of parkinsonism called manganism. Whether there is a relationship between manganese exposure and PD has been a point of interest, with focus on welders who may be exposed to it. A recent review concluded that manganese is an unlikely cause of Parkinsonism in the US population of welders. Direct measurement of lead levels in bone and blood serum suggests a link between PD and lead exposure, with greater risk associated with greater lifetime exposure.

Solvents and Polychlorinated Biphenyls (PCBs). Trichloroethylene (TCE) is a solvent used in many industries and is the most common organic contaminant in groundwater. Occupational exposure to TCE was found to be associated with Parkinson's among workers whose factory jobs resulted in long-term (eight to 33 years) exposure to the solvent. In a study of discordant twins (that is, twin pairs in which just one of the members had PD), the twin who had been occupationally exposed to TCE was more likely to develop Parkinson's than the one who had not. This link has also been observed in experiments in the laboratory.

Pesticides Trigger Parkinson's Disease

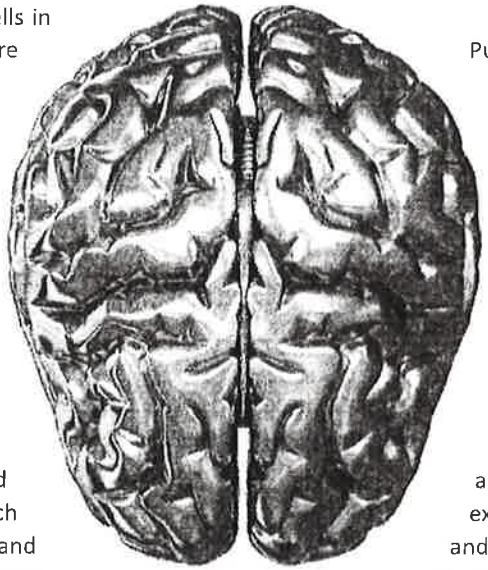
Astounding body of scientific literature finds strong evidence linking Parkinson's to normal pesticide exposure and fuels movement to phase-out numerous classes of pesticides and adopt safe management approaches.

By Kagan Owens

With less than one percent of cases caused by genetics,[1] researchers have been looking for the potential risk factors for developing Parkinson's disease (PD). What they are finding is startling. The epidemiological and toxicological evidence is repeatedly identifying exposure to pesticides, as well as specific gene-pesticide interactions, as significant adverse risk factors that contribute to PD.

What Is Parkinson's Disease?

The second most common neurodegenerative disease,[2] Parkinson's occurs when nerve cells in the substantia nigra region of the brain are damaged or destroyed and can no longer produce dopamine, a nerve-signaling molecule that helps control muscle movement. People with PD have a variety of symptoms including loss of muscle control, trembling and lack of coordination. They may also experience anxiety, constipation, dementia, depression, urinary difficulties, and sleep disturbances. Over time, symptoms intensify.



At least one million Americans have PD and about 50,000 new cases are diagnosed each year. PD affects mostly the middle-aged and elderly. Treatments are available for the symptoms, but there is currently no cure for PD.

The First Link

The suspicion that pesticides might be linked to PD was theorized in the 1980's following a wave of drug induced Parkinson's-like illnesses. The drug, MPTP, which was used as a heroin substitute, is transformed in the brain after injection. The new compound, MPP+, causes the loss of dopamine producing cells and the sudden onset of a Parkinson's-like illness.[3] The reason for the toxic effect is that MPP+ inhibits one of the enzymes in mitochondria, intracellular organelles that provide cells with energy.[4] It was later discovered that MPP+ was not only the breakdown product of an obscure drug, but also the active ingredient of the herbicide cyperquat, the closely related paraquat, and other pesticides.[5] This discovery sparked interest in studying the link between pesticides and PD, which has undercovered links to numerous pesticides and chemical families.

Pesticide Exposure Increases Risk

While some epidemiological studies and animal data linking PD with pesticides has been inconsistent[6,7] (likely due to study design issues such as control selections, study size, variety of diagnostic criteria used and statistical analysis), convincing evidence is continually emerging that demonstrates the pesticide exposure link to PD.[8,9]

Published case-control studies show a statistically significant association and elevated odds-ratios (OR) for PD (that determine the elevate disease rate above the norm of 1.0) and exposure to pesticides.[10,11] Duration of exposure[12] and level of exposure[13] is also correlated with an increase in PD risk. In a review of 40 published epidemiological case-control studies from 1983-2005, researchers from the UK evaluated the relationship between PD and pesticide exposure, finding sufficient evidence that an association exists and is strongest for exposure to herbicides and insecticides, and after long durations of exposure. In the 31 studies that show results for pesticides in an exposure category, the ORs ranged from 0.75 to 7.0 (a ¾ to 7 times greater disease rate) -- only two of those studies reported an OR less than 1.0.[14] A meta-analysis of 19 published, peer-reviewed studies done in the U.S. from 1989-1999 finds that individuals exposed to pesticides have twice the risk of developing PD than the general population.[15] A 1993 case-control study finds a positive association with insecticide exposure (OR=5.75), past residency in a fumigated house (OR=5.25), and herbicide exposure (OR=3.22) to PD.[16]

A large Harvard School of Public Health epidemiological study of more than 140,000 adults finds that those exposed to long-term, low levels of pesticides have a 70 percent higher incidence of PD

than among people who report no exposure.[17] A study of almost 3000 people in five European countries finds low level pesticide users, such as amateur gardeners, are 9% more likely to have Parkinson's, whereas high level users, like farmers, are 43% more likely. [18]

According to scientists, people exposed to chemicals that have a particular affinity for the substantia nigra region of the brain may be at particular risk for developing the disease.[19] In 2006, the preliminary results of a Centers for Disease Control and Prevention (CDC) funded study led by the University of North Dakota's Energy & Environmental Research Center, show that the areas of the brain in laboratory-tested rats affected by pesticide exposure are the same areas linked to neurological changes associated with PD.[20]

Rural Living, Well Water Consumption and Farming

Rural residency, well water consumption, and/or farming positively correlates with an increased incidence of developing PD.[21,22,23,24,25] A 2001 meta-analysis of peer-reviewed studies finds that living in a rural area, drinking well water, farming and exposure to pesticides have overall PD risk estimates between 1.26 and 1.85.[26] Early studies in Canada find the highest prevalence of PD coincides with agricultural areas with the largest amount of pesticide use.[27,28] One study discovered that many people living in rural areas, with no diagnosed neurological disorders, have lower levels of dopamine producing cells than urban populations.[29] This suggests that even in the absence of the illness, some aspect of rural life is putting people at risk for the disease. Confirming those results, another study finds that Parkinson's patients are twice as likely to be living in rural areas and drinking well water,[30] where farming pesticides often contaminate ground water. A California mortality study of individuals whose death certificates mention PD as an underlying cause of death and cross-referenced with agricultural and pesticide use data finds that the counties using restricted use pesticides (RUP) for agricultural purposes have about a 40 percent increase in PD mortality when compared to those counties reporting no RUP.[31]

Occupational Exposure

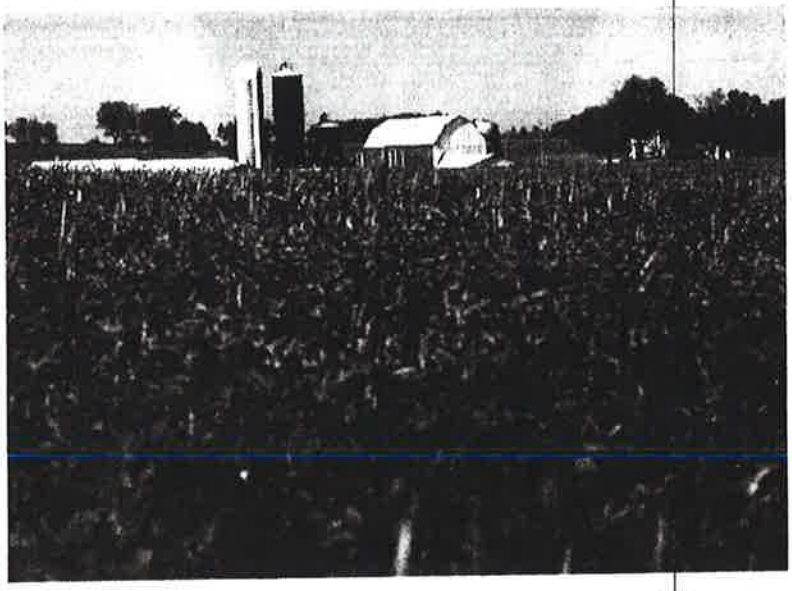
Confirmed again and again, studies find that PD is associated with occupational exposure to pesticides.[32,33,34,35,36,37,38,39,40] Studies show a two- to over a threefold increased risk of developing PD with occupational exposure, whether from working on farms,[41,42] orchards,[43] or plantations.[44] A population-based case-control study in Canada finds that a history of occupational herbicide use is associated with an estimated threefold increase in PD risk and previous insecticide use results in an estimated twofold increase in risk.[45] A case-control study in northeast Italy finds a 7.7 OR for farming as an occupation.[46]

Home Pesticide Use

A study published in the *Journal of the American Medical Association* raises concerns for residential pesticide exposure. Stanford University researchers find a 70 percent increased risk of developing PD for individuals that use pesticides in their home. Exposure to garden insecticides carries a 50 percent increased risk of developing the disease. Among herbicide users, the risk of developing PD increases as the number of days in contact with herbicides grows. Respondents who reported handling or applying herbicides for up to 30 days are 40 percent more likely to develop the disease, whereas respondents that reported 160 days exposure, have a 70 percent increase.[47]

Age-Related Risk Factors

The United Nation's World Health Organization (WHO) recently



Occupational pesticide exposure, rural living, farming, well water consumption and residential pesticide use have all been linked to elevated rates of Parkinson's disease.

Although age may contribute to Parkinson's disease, it is not considered by scientists to be a sole cause of the disease.

released a report on children's heightened vulnerability to chemical exposures at different periods of their growth and development. The report, *Principles for Evaluating Health Risks in Children Associated with Exposure to Chemicals*, highlights the fact that the stage of a child's development when chemical exposure occurs may be just as important as the magnitude of the exposure. The report states that "neurotoxic insults during development that result in no observable phenotype at birth or during childhood could manifest later in life as earlier onset of neurodegenerative diseases such as [PD]."[48] Several studies support WHO's report showing that exposure in utero, post-natal or in childhood affect the substantia nigra causing direct damage or increasing the susceptibility to additional exposures and neurodegenerative damage in adulthood.[49,50,51,52,53,54,55]

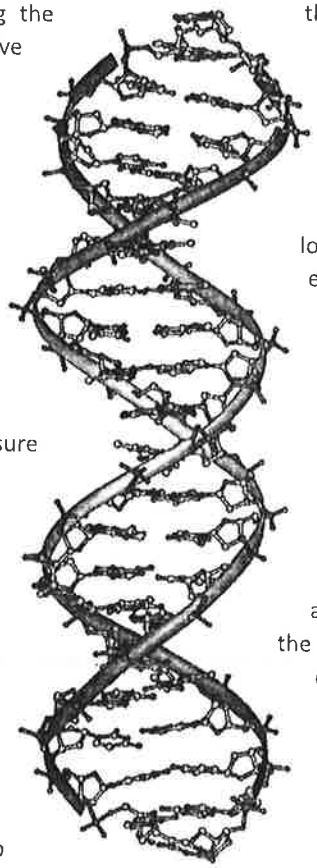
Aging is also found to be a risk factor for PD, yet researchers agree that aging alone is not a sufficient factor to explain PD. In one study, enhanced sensitivity of the aging nigrostriatal dopamine pathway to pesticides maneb and paraquat result in irreversible and progressive neurotoxicity, thus showing that exposure to pesticides combined with aging can increase the risk for developing PD.[56] University of Rochester scientists believe environmental contaminants such as pesticides make dopamine cells more vulnerable to damage from normal aging, infection, or subsequent exposure to pollutants.[57]

Genetic Risk Factors

Researchers screening twins for genetic effects and PD show that while genetic factors play a role for early-onset PD (begins at or before the age of 50), environmental factors are most important for those with late-onset PD.[58,59] Yet, genetics are not completely out of the picture for late-onset PD. A number of genes are linked to PD as they interact with toxic chemicals in such a way that they may not cause the disease directly, but cause subtle changes in the genes that can make individuals more or less likely to develop PD later in life.[60,61,62] Simply put by Kenneth Olden, Ph.D., former Director of National Institute for Environmental Health Sciences (NIEHS), "Genetics load the gun. The environment pulls the trigger."[63]

For those with a family history of the disease, exposure to certain chemicals found in pesticides may increase their risk of developing PD, according to a 2005 study. Researchers looked at specially bred fruit flies lacking both forms of the DJ-1 gene that is associated with the inherited form of PD. In the study, researchers show that flies lacking forms of the DJ-1 gene are normal under standard conditions, but when they are exposed to the herbicide paraquat and insecticide rotenone, the flies suffer from extreme oxidative or cellular stress and die. Researchers say their findings suggest that a loss of DJ-1 gene function increases sensitivity to chemicals that cause oxidative stress, thus linking a genetic cause with environmental risk factors.[64] Other research on cultured cells and in knockout mice (mice that have had a gene removed by genetic manipulation) supports these findings, showing that DJ-1 mutations can sensitize cells to the harmful effects of oxidative stress, which occurs when unstable oxygen molecules react with certain compounds like pesticides.[65]

Two other studies link family history and pesticide exposure to an increased risk of PD by looking at glutathione S-transferase P. Glutathione S-transferases (GST) are enzymes that help rid the body of toxic chemicals that generate oxidative stress. A study published in the *Lancet* finds a significant association for PD patients exposed to pesticides and having dissimilar alleles (variant forms of the same gene causing variations of inherited characteristics) at the GSTP1 locus. The scientists believe that this helps explain the susceptibility of some individuals to the parkinsonism-inducing effects of pesticides.[66] Researchers at the St. Jude Children's Research Hospital build on those findings, reporting in the *Proceedings of the National Academy of Sciences* that the GST pi detoxification enzyme that prevents damage to the substantia nigra region of the brain acts like a sentry at the crossroads of several biochemical pathways, any one of which can lead to PD. The job of the antioxidant GST pi is to protect the cell from death caused by either toxic chemicals in the environment, such as pesticides, or a self-destruction process called apoptosis, triggered by certain stressful conditions in the cell. If GST pi levels are reduced or this enzyme is overwhelmed by toxic chemicals,



these nerves are at increased risk of death. "The majority of these cases of [PD] appear to arise because individuals who have a genetic susceptibility to the disease are exposed to environmental toxins such as pesticides and herbicides, which trigger the formation of free radicals that kill dopaminergic neurons in the substantia nigra," states Richard Smeyne, Ph.D., associate member of the Department of Developmental Neurobiology at St. Jude. "We also know that GST pi blocks the process of cell suicide triggered by stresses that the cell can't overcome, such as an increase in the presence of free radicals or a loss of the cell's ability to produce energy."[67]

Enzyme deficiencies in the liver may lower resistance to pesticides, as PD patients are more likely to have a genetic deficiency in the detoxifying enzyme of the liver when compared to the normal population.[68] Scientists looking at the cytochrome P450 2D6 gene (CYP2D6) finds that this gene has a modifying effect on the risk of PD among individuals exposed to pesticides.[69] A 1998 case-control study published in *Neuroepidemiology* finds that individuals with Parkinson's who were exposed to pesticides and had the gene known as CYP2D6 29B+ allele, are three times as likely to develop dementia along with PD than those without the gene. This allele metabolizes and detoxifies chemicals that enter the body by activating liver enzymes. Those individuals who have a mutant form of the allele may be more susceptible to pesticides because of their inability to detoxify chemicals. This study finds that individuals who have a poor metaboliser CYP2D6 genotype and have also been exposed to pesticides are more likely to develop dementia.[70]

Two more genes, MnSOD and NQO1, encode enzymes that play key roles in oxidative stress and interact with pesticides to increase an individual's PD risk. Researchers show that among subjects that were exposed to pesticides, the combined MnSOD/NQO1 variant genotype is significantly associated with a four-fold increased risk of PD.[71]

"All of the evidence that has been accumulating suggests that exposure to pesticides increases the risk of PD," says Gary Miller, Ph.D., associate professor of environmental and occupational health at Emory University. "We believe that a person who is destined to get Parkinson's because of genetics or other factors at age 80 might develop symptoms when they're 65 or 70 if they have been exposed to pesticides."[72]

There is some data that shows a significant association between men, exposure to pesticides, and Parkinson's disease.

Pesticide Use Increases Risk in Men

While there is conclusive evidence that men are at an increased risk of being diagnosed with PD, how that factor comes in to play with pesticide exposure is not necessarily confirmed. There is some data that shows a significant association between men, exposure to pesticides, and PD.[73] A mouse study looking at developmental exposure to the insecticide dieldrin finds a greater effect in male offspring than in females.[74] In addition, the population-based study by Mayo Clinic researchers finds that men with PD are 2.4 times more likely to have been exposed to pesticides than those who did not have Parkinson's. Pesticide exposure did not increase the risk of Parkinson's in women, and no other household or industrial chemicals were significantly linked to the disease in either men or women.[75] Researchers suggest that men are at greater risk because male study respondents are more likely the ones that use pesticides in agriculture, in their occupation and/or around the home. The Mayo clinic researchers also suggest that "pesticide use combines with other risk factors in men's environment or genetic makeup, causing them to cross over the threshold into developing the disease."[76]

Implicating Specific Pesticides and the Mechanisms by which They Induce PD

Although the evidence showing a significant association between pesticide exposure and PD is clear, implicating specific pesticides or a group of pesticides is difficult. Exposure type, duration, product and dose are difficult to ascertain in retrospective case-control studies. Due to the possibility of recall biases, the vast number of pesticides available for use, and the fact that pesticides can work synergistically, many studies analyze pesticide exposure without regard to specifics such as product or chemical names, and, therefore, do not consistently implicate,[77,78] or estimate the PD risk associated with any particular pesticide.[79]

However, there are epidemiologic and toxicologic studies that have identified specific pesticides linked to PD. (See page 18.) Studies that identify the mechanisms by which pesticides lead to PD, such as protein aggregation (a-synuclein), effects on the striatal dopaminergic

Glossary of Common Terms

alpha-synuclein: one in a family of structurally related proteins that are prominently expressed in the central nervous system and form brain lesions that are hallmarks of some neurodegenerative diseases. Forms of hereditary PD are due to mutations and triplication in the gene for alpha-synuclein.

apoptosis: a natural process of self-destruction in certain cells that is determined by the genes and can be initiated by a stimulus or by removal of a repressor agent. Also called programmed cell death.

dopamine: a neurotransmitter formed in the brain essential to the normal functioning of the central nervous system. A reduction in its concentration within the brain is associated with Parkinson's disease.

mitochondria: spherical or rod shaped parts of the cell. Mitochondria contain genetic material (DNA and RNA) and are responsible for converting food to energy.

nigrostriatal pathway: the efferent connection between the susbtantia nigra and corpus striatum and one of the major dopamine pathways in the brain. Nigrostriatal pathway is particularly involved in the production of movement, as part of a system called the basal ganglia motor loop.

oxidative stress: an imbalance of the prooxidant antioxidant ratio in which too few antioxidants are produced or ingested or too many oxidizing agents are produced; can result in cell death.

proteasomes: a protein degradation "machine" within the cell that can digest a variety of proteins into short polypeptides and amino acids. Impaired proteasomal activity has been suggested as an explanation for Parkinson's disease.

striatum: part of the basal ganglia; a large cluster of nerve cells, consisting of the caudate nucleus and the putamen, that controls movement, balance, and walking; the neurons of the striatum require dopamine to function.

substantia nigra: a small area of the brain containing a cluster of black-pigmented nerve cells that produce dopamine, which is then transmitted to the striatum. Its destruction is associated with Parkinson's disease.

ubiquitin: a 76-amino-acid protein found in all eukaryotic cells [cells of plants, animals, protozoa, fungi and most algae] that can be covalently attached to specific lysine residues in target proteins. This often forms multimeric polyubiquitin chains, which is thought to target the protein for destruction.

system and altered dopamine levels, mitochondrial dysfunction (complex I inhibition) and oxidative stress, are discussed.

Conclusion

Although studies can have methodological limitations, overall the current review shows that there is a definitive relationship between Parkinson's disease and pesticides. The new research into PD is helping scientists better understand some of the mechanisms of this serious and disabling neurodegenerative brain disorder. Knowledge of the environmental factors and genetics of this illness has allowed investigators to create models of disease that are being used to examine potential causes of neuron disease such as pesticide exposure. While many researchers are seeking to support the development of more effective treatments of this human illness, the National Institutes of Health (NIH) has said, "[W]ith better knowledge of the role of pesticides and other environmental agents in causing [PD], effective prevention will be possible by eliminating or reducing use of specific environmental agents..."[80] Researchers that have been looking at the synergistic effects of pesticides state that, "[T]he current derivation of risk assessment guidelines needs to be reevaluated."[81] Advocates want to see the scientific knowledge support the banning of the chemical families associated with these effects. Because it is impossible to know your genetic disposition, all people should avoid contact with toxic pesticides.

Take Action

Let the U.S.EPA Administrator and Deputy Administrator know that they have a duty to alert the public to the scientific findings (laboratory and epidemiologic) that link pesticides with PD. In addition, urge these U.S.EPA officials to initiate an urgent and expedited review of pesticides' link to Parkinson's. Also let your elected members of Congress know how you feel.

Beyond Pesticides offers information on a plethora of non-toxic alternatives to pesticides. Learn how you can protect your family, community and the environment from the effects of pesticides in food and water, at home, on lawns, parks and gardens, in schools, hospitals and other public buildings. Resources are available at www.beyondpesticides.org. ***A fully cited version of this article is available at at www.beyondpesticides.org/infoservices/pesticidesandyou.***

Specific Pesticides Linked to Parkinson's Disease

The following are specific pesticides identified in the scientific literature to be linked to Parkinson's disease. However, the actual number is most likely much higher because implicating specific pesticides or a group of pesticides is difficult.

Benzimidazoles

Benomyl (Fungicide). University of North Dakota researchers found that benomyl affects rat brains, showing that mitochondrial enzymes are sensitive targets for inactivation by the pesticide.[82] Exposure to benomyl at low concentrations increases the risk of developing PD by inhibiting the ubiquitin-proteasome system.[83]

Bipyridyliums

Diquat Dibromide (Herbicide). Several days after a 72 year-old farmer was exposed to an aqueous solution of 10 percent diquat dibromide he developed severe parkinsonian syndrome.[84]

Paraquat (Herbicide). Several studies show an increased risk for PD with occupational exposure to and contact with paraquat.[85,86] A case-control study in Taiwan found that those who use paraquat are at greater risk of developing Parkinson's than those that use other pesticides.[87] A 2007 study examined a cohort of 80,000 licensed private applicators and spouses and found that farmworkers exposed to the herbicide paraquat have twice the expected risk of developing PD.[88] For those that were exposed to herbicides and could recall their exposure history, a Canadian population-based case-control study reported one individual using paraquat, between the ages of 26 and 31 years, and is the only herbicide-exposed case in the study whose onset of symptoms occurred before the age of 40.[89]

Paraquat induces dopaminergic nigral apoptosis and acts through oxidative stress-mediated mechanisms.[90] In laboratory animal studies, paraquat exposure triggers processes characteristic of early stages of dopaminergic neuron degeneration by stimulating an increase in the protein a-synuclein in the brain, likely due to preferential binding of the pesticides to a partially folded a -synuclein intermediate. The protein kills the dopamine-producing brain cells which lead to PD.[91,92,93,94,95] In 2002, researchers from the Parkinson's Institute, published that their findings "unequivocally show that selective dopaminergic degeneration, one of the pathological hallmarks of [PD], is also a characteristic of paraquat neurotoxicity."[96]

For researchers testing the role of oxidative stress in paraquat exposed mice, they find that the "initial exposure acts as a 'priming' event, enhancing neuronal vulnerability to a subsequent toxic insult," suggesting that dopaminergic cell degeneration appears to be dependent on the sequence of toxic challenges and the interaction between cell vulnerability, damaging effects and protective responses. Nigrostriatal neurons are vulnerable to oxidative processes. Depending on the paraquat exposure, oxidative stress may be reversible or lead to neurodegeneration.[97]

Botanicals

Rotenone (Insecticide). Rotenone, a naturally occurring pesticide, is used in laboratory studies to induce PD in rat and primate models to study various aspects of the disease in humans.[98,99] Laboratory studies using rats, monkeys, mice and human neuroblastoma cells find that rotenone destroys dopaminergic neurons inhibiting brain mitochondrial function, increasing excessive oxidative activity in the brain and shifting respiration to a more anaerobic state.[100,101,102,103] Rotenone can significantly stimulate the formation of a -synuclein fibrils.[104] Aging has also been found to increase the sensitivity of dopaminergic neurons to a low, systemic dose of rotenone.[105] Using rotenone *in vivo* and *in vitro* models, researchers find that chronic exposure to a pesticide and mitochondrial toxin brings into play three systems, DJ-1, a-synuclein, and the ubiquitin-proteasome system, and implies that mitochondrial dysfunction and oxidative stress link environmental and genetic forms of the disease.[106,107]

Dithiocarbamates

Diethyldithiocarbamate (Herbicide). Exposure to diethyldithiocarbamate at low concentrations increases the risk of developing PD by inhibiting the ubiquitin-proteasome system.[108] Diethyldithiocarbamate can also significantly stimulate the formation of a-synuclein fibrils, likely due to preferential binding of the pesticides to a partially folded a-synuclein intermediate.[109]

Mancozeb. (Fungicide). Mancozeb affects rat brain mitochondria, showing that mitochondrial enzymes, which are sensitive targets, are inactivated by the pesticide.[110]

Maneb (Fungicide). A case-report shows that after chronic exposure to maneb, a 37-year old man developed Parkinson's two years after the applications ceased.[111]

University of North Dakota researchers find maneb affects rat brain mitochondria.[112] Low levels of maneb can injure the antioxidant system in the dopamine neurons, especially with concurrent exposures to other environmentally relevant oxidative stressors, such as paraquat.[113]

Ziram (Fungicide/Dog and Cat Repellent). Ziram shows inhibitory effects on proteasome activities at low concentrations. This suggests that proteasome inhibition as a potential mechanism for the epidemiological association of pesticides and PD.[114]

Organochlorines

In 1996, a German study linked PD to pesticides, finding an elevated odds ratio for organochlorine pesticides.[115]

Dieldrin (Insecticide).[116,117,118,119,120] Low-level exposure to dieldrin, a banned but persistent pesticide ubiquitously distributed in the environment, appears to accelerate changes in the brain that can potentially lead to the onset of PD symptoms years or even decades before they might naturally develop, according to a research presentation at the 2006 American Chemical Society annual meeting. This finding "clearly shows that pesticides such as dieldrin appear to accelerate or exacerbate the already underlying disease," states Emory University's Gary Miller, Ph.D. "So it appears the more you are exposed to pesticides, the greater your risk of developing the disease earlier in life."

In studies looking at post-mortem brain tissue samples of Parkinson's patients, scientists find a significant association between dieldrin and the diagnosis of PD.[121,122,123] Dr. Miller and his co-researchers found levels of dieldrin three times higher in the brains of 14 people who had PD than in the brains of 12 people who did not.[124]

Endosulfan (Insecticide). A study testing 25 pesticides to see if exposure to them increases the risk of developing PD finds that endosulfan shows inhibitory effects on proteasome activities at low concentrations.[125]

Heptachlor (Insecticide). Perinatal exposure to heptachlor, another banned pesticide that persists ubiquitously, alters the dopaminergic system and may increase the vulnerability of dopamine neurons to toxic insult.[126]

Lindane (Insecticide). An autopsy case-control study finds significant levels of lindane in the brain tissues of deceased Parkinson's patients.[127]

Organophosphates

Chlorfenvinphos (Insecticide). Subchronic administration of chlorfenvinphos, a pesticide that is no longer registered by the U.S. EPA, leads to a change in the brain oxidative status in rats.[128]

Parathion (Insecticide). Although the researchers did not find a significant association between PD and pesticide exposure, their population-based case-control study in Washington state finds that among individual pesticides, the highest odds-ratio is seen with parathion, a highly toxic neurotoxic pesticide.[129]

Chlorpyrifos (Insecticide). Researchers find that dopaminergic neurotransmission is affected by exposure to chlorpyrifos in a laboratory mice study.[130]

Pyrethroids

Deltamethrin (Insecticide). One study finds that because the dopamine transporter function of the brain is affected by the vulnerability of dopamine neurons to nuerotoxicants, up-regulation (increased cellular response) of deltamethrin may increase the susceptibility of dopamine neurons to toxic insult.[131]

Permethrin (Insecticide). Studies find that permethrin affects dopaminergic neurotransmission[132] and up-regulation of permethrin may increase the susceptibility of dopamine neurons to toxic insult.[133]

Virginia Tech researchers discovered that exposure to some insecticides, such as permethrin, may cause a cascade of chemical events in the brain that can lead to PD. The researchers studied the levels of dopamine, dopamine transporter protein expression, and the levels of a-synuclein in mice exposed to various doses of permethrin. The increase in dopamine uptake indicates that the mouse's system is reacting to a neurochemical insult caused by the presence of the insecticide. In some individuals, dopamine-producing neurons may be challenged by genetic factors or by previous exposure to other neurotoxins. For individuals with a genetic predisposition, exposure to permethrin may trigger chemical events in the brain that result in an increased risk for damage to the area of the brain that is selectively damaged in PD. The researchers also find that permethrin exposure results in an overproduction of the protein a-synuclein at low doses. The accumulation of the protein is a major component of the formation of the Lewy bodies, fibrous tangles observed in the brains of patients with PD.[134]

Thiocarbamate and Chlorophenoxy Herbicides

For those that were exposed to herbicides and could recall the chemicals or trade names of the products used, a Canadian population-based case-control study found that all but one PD patient had used compounds in the thriocarbamate and chlorophenoxy and chemical groups exclusively.[135]

Triazines

Atrazine (Herbicide). A 2007 rat study found that atrazine decreases tissue dopamine levels by interfering with the vesicular storage and/or cellular uptake of dopamine.[136]

Others

Pyridaben, Fenpyroximate, Fenazaquin (Insecticides). Research at Emory University found that commonly used pesticides are toxic to the mitochondria of cells, an effect linked to PD. PD has been associated with abnormalities of mitochondria, which are the "power plants" that provide all cells with energy. The Emory scientists exposed human neuroblastoma cells to the pesticides pyridaben, fenpyroximate and fenazaquin which inhibit complex I, a mitochondrial enzyme. Pyridaben is by far the most potent toxic compound. Pyridaben is also more potent in producing "free radicals" and oxidative damage to the cells, both of which are thought to be important in causing PD.[137]

Synergistic Effects

Paraquat and Maneb. University of Rochester scientists discovered that the synergistic effects of paraquat and maneb target the nigrostriatal dopamine system and indicate progressive neurotoxicity with continuing exposure. Their findings show that while there are no or only marginal effects when these chemicals are administered individually, together they produce synergistic effects when given in combination.[138] In another study, these researchers again chronically expose mice to a low-level combination of paraquat and maneb, resulting in significant reductions in locomotor activity, levels of striatal dopamine and dopaminergic neurons in the substantia nigra, more so than when exposed individually.[139]

A laboratory study found that "prenatal exposure to the pesticide maneb produces selective, permanent alterations of the nigrostriatal dopaminergic system and enhances adult susceptibility to paraquat exposure."[140] Additional studies show that exposure to maneb and paraquat during the post-natal and juvenile period causes Parkinson-like declines in dopaminergic neurons and makes the substantia nigra more susceptible to additional exposures in adulthood,[141] "suggesting that developmental exposure to neurtoxicants may be involved in the induction of neurodegenerative disorders and/or alter the normal aging process."[142]

Endosulfan and Zineb. Researchers at Virginia Tech examining endosulfan and zineb in human cultured neuroblastoma cells found that these pesticides, individually and together, are toxic to the impulse-conducting cells of the nervous system. Mixtures of the two pesticides had greater effects.[143] Another study found that mice exposed to endosulfan and/or zineb as juveniles and then re-exposed in their adulthood result in significantly depleted striatal dopamine levels, thus concluding that exposure to pesticides such as endosulfan and zineb during critical periods of postnatal development contributes to neurotransmitter changes in adulthood.[144]

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The Link Between Pesticides and Parkinson's

By John Hamilton

Over the course of the past decade the link between pesticides and Parkinson's Disease (PD) has been demonstrated by a growing body of scientific research published in peer-reviewed professional journals both here and abroad. Our review of these studies has identified at least twenty three specific pesticides which have been used in American agriculture and are linked to PD. And of the twenty three, eleven (as of January 2012) were registered by the NMDA for use in New Mexico. That four of the eleven-glyphosate, paraquat, 2, 4-D, and Diazinon-are among the most commonly used pesticides on crops in Dona Ana and Luna counties.

This is our concern.

We believe that New Mexicans are being exposed daily to this complex of pesticides which over time are damaging the ability of their brain to produce dopamine, a key neuro-transmitter affecting body movement. Thus developing Parkinson's Disease. But, of course, this is a controversial issue and deserves explanation.

Think of pesticides as they are –complex chemicals with unique features making them effective in exterminating the target "pest", be it unwanted plant or insect. However, their apparent success with pests is compounded by their destructive impact on the dopamine producing processes of the brain.

Substantial evidence derived from various cell studies suggest that toxic pesticides likely trigger oxidative stress and damage the mitochondria which produce dopamine. That these toxins also interfere with key enzymes (i.e. PARKIN and alpha-synuclien protein) tasked with the removal of Lewy bodies which block the passage of dopamine to the next cell.

Visualize farmer and farm workers being exposed over a working life to a cluster of pesticides. While individual exposures never rise to the level creating acute distress, the individual pesticides may interact to create a multiplier effect. Further, that these toxins continue to damage brain cells by making them more vulnerable to further environmental exposures. At which time a tipping point is reached and the individual experiences the symptoms of Parkinson's Disease.

Then complicate this scenario by recognizing that the rules of exposure extend beyond applicators to include those who live within 500 meters of a sprayed field, those who drink from rural wells, and those who are particularly vulnerable-i.e. the developing fetus and young children. Their "risk cup" runneth over. And this is to say nothing about non-agriculture exposure in homes and schools, etc.

Do you understand our concern?

But it doesn't have to be that way. Farmers can dramatically reduce or eliminate their use of pesticides by employing techniques of sustainable agriculture and organic farming, and still make a profit. The evidence of this lays all around us in the success of farmers like Ramon "Dosi" Alvarez who is a certified organic farmer in Southern New Mexico.

In our judgment, it's a win-win situation. When we have fewer pesticides in our environment we will have fewer New Mexicans stricken by Parkinson's Disease and other neurological disorders.

PESTICIDES LINKED TO PARKINSON'S.

A few pesticides have been banned but remain active due to their long shelf lives which are still impacting our environment and our health today.

These pesticides are mainly for agricultural use. Yet, there is a significant urban application (lawns, bug sprays, etc.). Furthermore, while direct exposure to pesticides continues to be the most important health threat, there is a growing body of evidence that rural well water contaminated by pesticides is linked to the onset of Parkinson's symptoms." The risk of Parkinson's disease is around 90% higher for those who have private wells near fields sprayed with **propargite** or **chlorpyrifas.** However, the critical window of exposure to toxicants may have occurred years before the onset of motor symptoms" (Department of Epidemiology at UCLA).

As of January 11, 2012, the number of active pesticide ingredients registered in New Mexico exceeds 800, 90% are not tested for safety. We think we have reasonable cause for concern and need to be better informed.

PESTICIDES	TARGET	EXPOSURE
Aldrin, Endrin & Dieldrin Used on corn, cotton, termites, mosquitoes	Alters dopamine neurons; increasing vulnerability to toxins; function inhibiter	Eating fish, shellfish, dairy, breast milk and exposure
Rotenone Used to kill invasive fish	Inhibits function of cell mitochondria (energy provider)	Application and random exposure
Paraquat, paraquat dichloride Used on pecans, chile, alfalfa	Increases oxygen derivatives harming cell structure	Application and random exposure
Maneb Used on vegetables (potatoes,...), fruits (tomatoes,...), ornamentals, sod and field crops	Acts as synergist to other pesticides	Application and random exposure, residue on food crops
Ziram Used on crop foliage	Inhibits the break-down of the alpha-synuclein protein (communication)	Application and exposure
Propargite Used on nuts, corn and grapes	Oxidative cell stress, mitochondria damage	Private well water contamination, aerial spray
Methomyl Used on alfalfa	Oxidative cell stress, mitochondria damage	Private well water contamination
Chlorpyrifos Used on corn, cotton, fruit trees	Oxidative cell stress, mitochondria damage	Private well water contamination, aerial spray
2, 4-D Used as herbicide in lawns	Oxidative cell stress, mitochondria damage	Occupational exposure
Permethrin, cypermethrin Used in bug/mosquito spray	Oxidative cell stress, mitochondria damage	Open field exposure
Diazinon agriculture	Increase levels of serum amylase and glucose	Occupational exposure
Endosulfan Agricultural insecticide, cotton, potatoes, apples, tomatoes	Endocrine disruptor, acute neurotoxic	Phased out, Application and exposure
Zineb pesticide	Cholinesterase disruptor	Application and exposure
Heptachlor insecticide		Application and exposure
Lidane Pesticides used in hair products	GABA neurotransmitters disruptor	Application and exposure
Benomyl , Atrazine glyphosate	Affects the gut microbiota; inhibits the activity of cytochrome P450 enzymes	Application and exposure (Round Up)

NB: **Paraquat** is widely used in Dona Ana County (pecans, chile, alfalfa, etc.) as well as **ziram** and **maneb**.
A UCLA research team has found that residents exposed to the combination of **paraquat** & **ziram** raised the risk 80% and that of **paraquat**, **ziram** and **maneb** have a threefold risk.

Linking Pesticides and Parkinson's Disease

By John Hamilton

Parkinson's Disease is a debilitating neurological disorder characterized by a body of physical symptoms including tremors, fatigue, rigidity, and dyskinesia (jerky movements). Diagnosis is often difficult because of the complexity of factors involving PD and related motion disorders.

Classically, the etiology has been ascribed to either genetic (10 percent) or environmental (90 percent) factors. This simplified rendering of etiology has been modified to recognize a multivariate explanation. Given the preponderance of evidence linking the environment to PD, it has proven of value to look at the role of pesticides in developing Parkinson's.

Pesticide exposure as a risk factor to human health has been seen largely as a work site poisoning among farmers. However, this view is faulty on at least two accounts. First of all, there is a substantial health risk for even low-level chronic exposure. Secondly, the population at risk is much broader than simply farm workers. Substantial evidence exists to include residents living within 500 feet of a sprayed field, consumers of rural well water, and young children at home and at school.

We must connect the intervening dots by which the toxin travels along the bio-pathways of the body to the dopamine producing cells where the damage is done. Breathing, eating, and touching constitute the three principle portals of the body's exposure to pesticides. Respiration carries these toxins to the lungs; digestion moves them to the large intestine; and the lyphomatic system carries them directly to the veins. But each in turn must manage to pass these individual toxins through the blood-brain barrier before they can enter the brain. This is the most critical step in the movement of the pesticides to the brain. But rather than being a single barrier having a singular protective characteristic, it must be thought of as a complex of barriers whose unique attributes in aggregate protect the brain from a wide spectrum of toxins.

These toxic chemicals achieve their victory over the blood-brain barrier by mimicking naturally occurring molecules which are critical for the life of the brain cells in the substantia nigra where the production of the dopamine takes place. In some instances passage is determined by the size of the penetrating toxic molecule so that the toxin can push its way between b-b barrier cells. In other instances, the pesticide molecule must exhibit the appropriate electrical charge, or combine with either H_2O or lipids to effect passage across the b-b barrier. At the same time, other pesticides facilitate their passage by destroying astrocytes, the physical structures which restrict the movement of harmful chemicals through the blood-brain barrier.

As the pesticide molecule penetrates the neuron it attacks the cell's mitochondria. This structure plays a central role in the production of dopamine the chief neuro-transmitter involved with the muscle movement. As oxidative stress increases in the cell the capacity of the alpha-synuclein protein to remove the residue of the damaged cell is impaired. This leads to the accumulation of fibrils forming "Lewy Bodies" which block the movement of the mitochondria to the synapse of the cell. Thus the loss of dopamine and the onset of tremors.

PESTICIDES & PARKINSON'S COMMITTEE REPORT

by John Hamilton

EXPOSURE(S)

- Applicator
- Farm worker
- Farmer
- Rural dweller
- Consumer of rural well water.
- Residents within 500 meters of farm
- Vector control programs.
- Residents with commercial turf workers in wood processing plants.
- Students/teachers in schools.

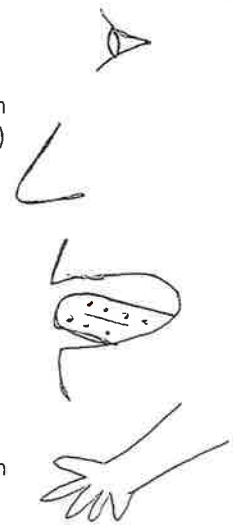
BIOLOGICAL PATHWAYS

1

Respiration (breathing)

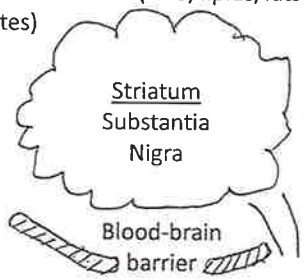
Ingestion/ digestion (eating)

Tactile (skin contact)

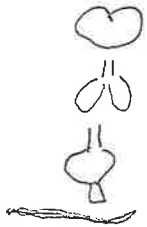


2

"Pesticides are designed to penetrate the blood-brain barrier" (H20, lipids/fats-astrocytes)



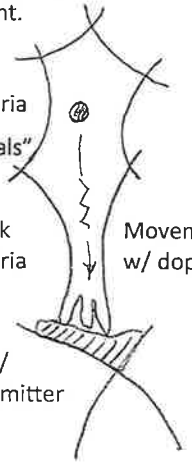
Blood circulation



3

EFFECTS IN THE NEURON / CELL

* Oxidative stress
* Damage to the mitochondria
* Inhibition of alpha-synuclein protein which removes residue of damaged cell creates "fibrils" which block dopamine movement.



PD DEVELOPMENT / SYMPTOMS

- Tremors
- Bradykinesia (slow movement)
- Fatigue
- Rigidity
- Gait (walking)
- Balance
- Dyskinesia (jerky movements)
- Speech distress
- Swallowing
- Constipation
- Dizziness
- Dementia
- Panic anxiety
- Depression

CHARACTERISTICS

Exposure may be acute, chronic, with high or low toxicity, show immediate symptoms, over extended periods of time.

SCIENTIFIC / MEDICAL STUDIES

* When dosed with rotenone neurons grown in culture become sickly and die like in PD. Zheng B. et al. Science Translation Medicine (2010).
* Consumers of contaminated (propargite, etc.) well water show increased risk of PD Ritz B. et al. Environ Health Perspective (2009).
* Mice exposed to zineb and endosulfan as juveniles are more susceptible to these chemicals in adulthood. Jia, Z. et al. Neurotoxology (2007).
* Neonatal exposition to permethrin and cypermethrin induces long-lasting reduced levels of dopamine in the striatum. Cantalassa, et al. Toxicology (2007).

CLINICAL DIAGNOSIS

Diagnosis is difficult because of the complexity of factors involving PD and related disorders. Thus the physician may diagnose Parkinson's by response to the particular medications.

Organic Land Management and the Protection of Water Quality

Organic land management, including agriculture and the production of organic food, utilizes a system that seeks to maintain and improve the environment. Organic standards, codified in the *Organic Food Productions Act* (OFPA), are subject to independent public review and oversight of practices and allowed inputs, assuring that toxic, synthetic pesticides used in conventional, chemical-intensive agriculture are replaced by methods focused on soil biology, biodiversity, and plant health. This ensures that pesticides that contaminate our water and air, hurt biodiversity, harm farmworkers, and kill bees, birds, fish and other wildlife are reduced or eliminated completely.

Current laws to protect water quality under the *Clean Water Act* (CWA) are limited by risk calculations that offer limited public health and environmental protection. Meanwhile, the nation as a whole still relies on toxic inputs to grow food and manage landscapes. These chemicals, like atrazine, 2,4-D, and glyphosate, are linked to a myriad of human and environmental health concerns, including cancer, birth defects, reproductive and sexual dysfunction, and neurological/learning problems. The high rates of cancer, learning and behavioral effects, and infertility call for a serious reevaluation of the way food is grown and waterways protected. Organic farming and landscape management provide the model for transitioning from chemical-dependent to sustainable practices.

Conventional Farming Threatens Waterways

Conventional, chemical-intensive agriculture in the U.S. and its reliance of toxic, synthetic inputs, such as insecticides, herbicides, and synthetic nitrogen and phosphorus fertilizers, have affected the quality of surface and groundwater for decades. According to data from the U.S. Geological Survey (USGS) and the Environmental Protection Agency (EPA), of the over 300 food production pesticides with tolerances registered –allowable levels of pesticide residue on food– 52 are known surface or groundwater contaminants. (See Beyond Pesticides' *Eating with a Conscience* (EWAC) at www.eatingwithaconscience.org.) The overwhelming majority of the most popular pesticides used in the U.S. have been detected in surface and groundwaters, including the popular herbicides atrazine, glyphosate, and 2,4-D. (See Table 1.)

Table 1.
Most Used Pesticides in the United States by Poundage

Chemical	Millions of Pounds Used Annually	Identified as a Surface/ Groundwater Contaminant
Glyphosate	180-185	✓
Atrazine	73-78	✓
Meta Sodium	50-55	✓
Metolachlor	30-35	✓
Acetochlor	28-33	✓
Dichlopropene	27-32	✓
2,4-D	25-29	✓
Methyl Bromide	11-15	No
Chloropicrin	9-11	✓
Chlorphyrifos	7-9	✓
Chlorothalonil	7-9	✓
Ethephon	7-9	No
Metam Potassium	7-9	✓
Pendimenthalin	7-9	✓
Copper Hydroxide	6-8	✓
Simazine	5-7	✓
Trifluralin	5-7	✓
Mancozeb	4-6	No
Propanil	4-6	No
Aldicarb	3-4	✓
Acepahte	2-4	✓
Dimethenamid	2-4	No
Diuron	2-4	No
MCPA	2-4	✓
Paraquat	2-4	✓

Ten Most Toxic Crops to Produce

Corn and soybeans are the most widely grown crops in the U.S. and as such contribute overwhelmingly to pesticide contamination, especially in the Midwest where these crops are predominantly grown, and pollution of the Mississippi River watershed and the Gulf of Mexico. Ninety percent of corn and soybeans are genetically engineered, which also correlates to an increase in herbicide use. The EWAC database indicates that of the 84 pesticides with established tolerances for corn, 22 are known to contaminate streams or groundwater. Of the 83 pesticides registered for use on soybeans, 26 contaminate streams or groundwater.

Conventional, chemical-intensive farming of bell peppers, potatoes, tomatoes, and wheat are the biggest contributors to water contamination after corn and soybeans. Asparagus, peaches, pears,

Health Effects of 30 Commonly Used Lawn Pesticides

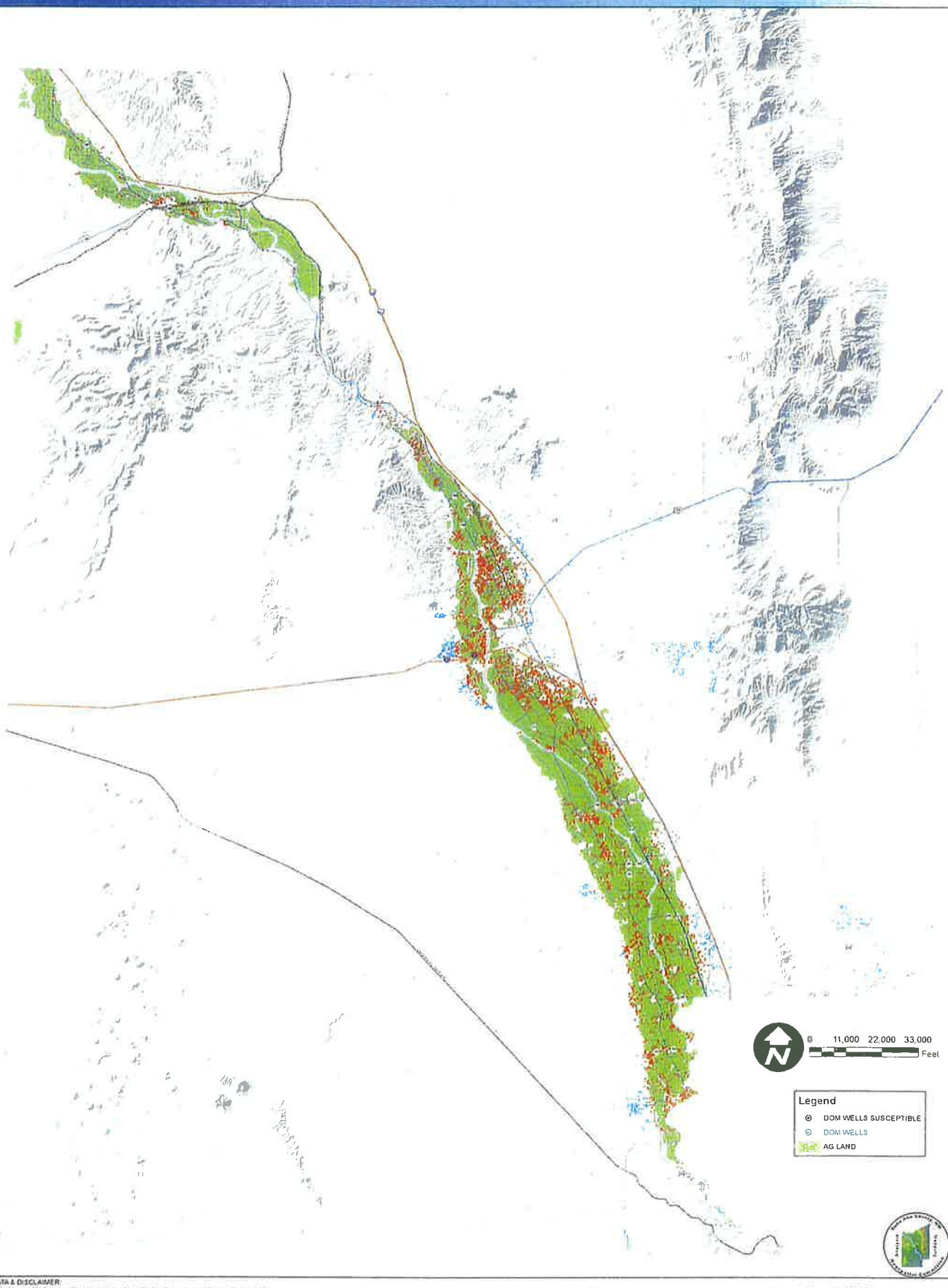
Pesticides	Health Effects						
	Cancer	Endocrine Disruption	Reproductive Effects	Neurotoxicity	Kidney/Liver Damage	Sensitizer/ Irritant	Birth Defects
Herbicides							
2,4-D*	X	X	X	X	X	X	X
Benfluralin					X	X	
Bensulide				X	X	X	
Clopyralid			X			X	X
Dachthal	Possible	X			X	X	
Dicamba*			X	X	X	X	X
Diquat Dibromide			X		X	X	
Fluazipop-p-butyl			X		X		X
Glyphosate*	X	X	X		X	X	
Isoxaben	X				X		
MCPA		X	X	X	X	X	
MCPP*	Possible	X	X	X	X	X	X
Pelargonic Acid*						X	
Pendimethalin*	Possible	X	X			X	
Pronamide	Probable	X			X	X	
Triclopyr			X		X	X	X
Trifluralin*	Possible	X	X		X	X	
Insecticides							
Acephate	Possible	X	X	X		X	
Bifenthrin*§	Possible	Suspected		X		X	X
Carbaryl	X	X	X	X	X	X	X
Dichlorvos	X	Suspected		X	X	X	
Fipronil	Possible	X	X	X	X	X	
Imidacloprid			X		X		X
Malathion*	Possible	X	X	X	X	X	X
Permethrin*§	X	Suspected	X	X	X	X	
Trichlorfon	X	X	X	X	X		X
Fungicides							
Azoxystrobin					X	X	
Myclobutanil		Probable	X		X		
Sulfur						X	
Ziram	Suggestive	Suspected		X	X	X	
Totals:	17	18	19	14	24	25	11

** These pesticides are among the top 10 most heavily used pesticides in the home and garden sector from 2006-2007, according to the latest sales and usage data available from EPA (2011).*

§ EPA lists all synthetic pyrethroids under the same category. While all synthetic pyrethriods have similar toxocological profiles, some may be more or less toxic in certain categories than others. See Beyond Pesticides' synthetic pyrethriod factsheet at bit.ly/TLBuP8 for additional information.

List of citations available online at www.beyondpesticides.org/lawn/factsheets/30health.pdf.

Domestic Wells Susceptible to Pesticides



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PARKINSON'S REGISTRY COALTION STUDY: DOMESTIC WELLS SUSCEPTIBLE TO PESTICIDES

(FACTS REGARDING GIS ANALYSIS AND RESULTING MAP)

1. Data Used in Analysis
    a. Well data for Doña Ana County provided by NM Office of the State Engineer / Interstate Stream Commission, Water Rights District IV – Las Cruces
    b. Agriculture Land Data created from 2010 Aerial Photos and provided by Doña Ana County GIS Division
2. Facts regarding well data
    a. Total of 14,054 wells in database for Doña Ana County
    b. Total of 5,335 domestic wells in database for Doña Ana County
    c. Total of 4,341 domestic wells susceptible to pesticides (analysis below)
3. GIS Analysis
    a. Selected by location the total domestic wells within a 500 meter distance of the 2010 Agriculture Land Data to determine how many domestic wells were potentially susceptible to pesticides. The total is: **4,341** domestic wells
4. Future GIS Analysis
    a. With additional data sources that may be made available or mapped in the future, including something similar to the State of California's Pesticide Use Reporting (PUR) and with addresses of known Parkinson's patients in the County through the Registry Coalition, a more detailed study like the California Study can be accomplished here in Doña Ana County in order to study links between pesticide exposure and the risk for Parkinson's disease

EPA/600/R-99/008
September 1998

PESTICIDE EXPOSURE AND HEALTH EFFECTS IN YOUNG CHILDREN

PART I: PESTICIDE DATA

by

Gerry Akland

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Post Office Box 12194
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Contract Number: 68-C5-0011

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OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
LAS VEGAS, NEVADA 89193

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Table 3-16. Doña Ana County: The Most Used Pesticides Based on 1996 Sales Data.

Chemical	Type	Pounds of Active Ingredient
Telone	Fumigant	37,995.0
Temik	Insecticide	12,030.5
Lannate	Insecticide	11,156.0
Bladex	Herbicide	6,848.2
Furidan	Insecticide	4,026.0
Diazinon	Insecticide	1,648.4
Paraquat	Herbicide	1,525.0
Counter	Insecticide	1,372.0
M. Parathion	Insecticide	1,085.0
DiSyston	Insecticide	964.0

Major Insecticides: 32281.9 lbs. of A.I.
% of all insecticide sales: 86.9%

Major Herbicides: 8373.2
% of all herbicide sales: 83.9%

Major Fungicides & Other: 37995.0
% of all Other sales: 95+%

Note: Total sales of all pesticides equal 85516.9 pounds.

Paraquat (PQ) can cause oxidative stress either intracellularly by redox cycling or by activation at cell surfaces by the NADPH oxidase. Mitochondria can be affected directly or indirectly by PQ. In neurons the effects of PQ are believed to be primarily cytosolic.

Pesticide Exposure and Risk in Young Children

RESTRICTED USE PESTICIDE

Due to acute human oral, dermal, and inhalation toxicity

For retail sale to and use only by Certified Applicators or persons under their direct supervision and only for those uses covered by the Certified Applicator's certification.

Bayer CropScience

Di-Syston® 15% Granular

Systemic Insecticide

For effective systemic crop protection.

ACTIVE INGREDIENT:

O,O-Diethyl *S*-[2-(ethylthio)ethyl]phosphorodithioate 15%

INERT INGREDIENTS: 85%

100%

EPA Reg. No. 264-723 EPA Est. No.

STOP – Read the label before use.

KEEP OUT OF REACH OF CHILDREN

DANGER POISON

PELIGRO

Si usted no entiende la etiqueta, busque a alguien para que se la explique a usted en detalle.
(If you do not understand the label, find someone to explain it to you in detail.)

For MEDICAL And TRANSPORTATION Emergencies ONLY Call 24 Hours A Day 1-800-334-7577
For PRODUCT USE Information Call 1-866-99BAYER (1-866-992-2937)

FIRST AID

Contains an organophosphate that inhibits cholinesterase.

If sw ter or doctor immediately
of water if able to swallo
nless told to do so by a
nouth to an unconscious

If on othing.
ith plenty of water for 15
er or doctor for treatme

If in e slowly and gently with w
inutes, then continue ri
er or doctor for treatme

If inh call 911 or an ambular

er or doctor for further treatment advice.

cholinesterase is an enzyme which functions to split ACh (acetylcholine) into acetate and choline. / ACh is a neurotransmitter in the caudate and putamen (striatum) circuit

neurotransmitter / basal ganglia

1996

964 lbs.

Highly Toxic

Doña Ana County

RESTRICTED USE PESTICIDE

DUE TO ACUTE ORAL TOXICITY and TO GROUND WATER CONTAMINATION

For retail sale to and use only by Certified Applicators or persons under the direct supervision of a Certified Applicator, and only for those uses covered by the Certified Applicator's certification.

TEMIK® brand 15G ALDICARB PESTICIDE

For Control of Certain Insects, Mites, and Nematodes

ACTIVE INGREDIENT: Aldicarb [2-methyl-2-(methylthio) propionaldehyde O-(methylcarbamoyl)oxime].....15%
INERT INGREDIENTS: 85%

EPA Reg. No. 264-330 EPA Est. No. 264-GA-01

KEEP OUT OF REACH OF CHILDREN

DANGER POISON

PELIGRO

Si usted no entiende la etiqueta, busque a alguien para que se la explique a usted en detalle.
(If you do not understand the label, find someone to explain it to you in detail.)

For MEDICAL And TRANSPORTATION Emergencies ONLY Call 24 Hours A Day 1-800-334-7577
For PRODUCT USE Information Call 1-866-99BAYER (1-866-992-2937)

FIRST AID

1996

12,030 lbs.

Highly Toxic

Doña Ana County

ion control center or doctor for treatment advice.
unless told to do so by a poison control center or doctor.
ss of water if able to swallow.
y mouth to an unconscious person.

se slowly and gently with water for 15-20 minutes.
s, if present, after the first 5 minutes, then continue rinsing.
enter or doctor for treatment advice.

l clothing.
y with plenty of water for 15-20 minutes.
enter or doctor for treatment advice.

air.
ing, call 911 or an ambulance, then give artificial respiration, preferably mouth-to-

center or doctor for further treatment advice.

For MEDICAL Emergencies Call 24 Hours A Day 1-800-334-7577.

Have the product container or label with you when calling a poison control center or doctor or going for treatment.

Aldicarb is a N-methyl carbamate.

MEDICAL INFORMATION

Contact a physician immediately in all cases of suspected poisoning. Illness may be produced rapidly following overexposure to TEMIK brand 15G Aldicarb Pesticide. If breathing stops, establish an airway and start artificial respiration, and provide oxygen. Make certain to remove all sources of continuing contamination. Remove clothing and wash skin and hair immediately with large amounts of water. Transport the patient to a physician or hospital immediately and SHOW A COPY OF THIS LABEL TO THE PHYSICIAN. If poisoning is suspected in animals, contact a veterinarian.

BLADEX herbicide

1996

6848.2 lbs.

Doña Ana County

Product Information

Registered uses	Peas, chickpeas, faba beans, vetch, lentils, potatoes, onions and sweetcorn
Pack size	5 kg
Formulation	WG
Active ingredient	900 g/kg CYANAZINE

BLADEX is a selective herbicide for weed control in peas, chickpeas, faba beans, vetch, lentils, potatoes, onions and sweetcorn.

Features

- Highly selective weed control in various vegetable and field crops
- Good contact activity plus 4 to 10 weeks control of a wide range of broadleaf and grass weeds
- Good crop safety
- Short persistence with low potential for plant back issues
- Compatible with many tank mix partners

Downloads

IOWA STATE UNIVERSITY
Extension and Outreach
Integrated Crop Management

Furadan
(Carbofuran)

Highly Toxic

Subscribe to ICM News updates and receive email alerts when new information is posted.

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ICM News

Cancellation of Furadan (carbofuran) for Crops Use

June 2, 2009

By Jon Tollefson and Erin Hodgson, Department of Entomology

Due to toxicity and potential environmental hazard, the Environmental Protection Agency (EPA) has announced its

1996

11,156 lbs.

Highly Toxic

Doña Ana County

DuPont™

Lannate®

Methomyl 40% SP

Insecticide

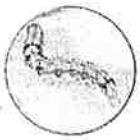
• GENERAL INFORMATION

DuPont™ Lannate® insecticide is broad-spectrum insect control product registered in more than 70 countries world wide and is effective on over 100 insect species. When Lannate® is used for the control of lepidopteran insects, it works as an effective ovicide, larvicide and adulticide.

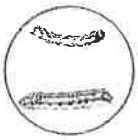
Studies shows that methomyl, the active ingredient in Lannate insecticide, has a very low impact on the environment when applied according to label direction. In addition, methomyl has been found to be non persistent in studies on plant, soil, forest and soil ecosystem, aquatic plants and animals and water. Lannate is toxic to wildlife and fish.

Lannate has been found to be particularly active on many lepidopterous insects. In addition, it is active on aphids, beetles, leafhoppers, leafminers, lygus midges and thrips.

• INSECTS CONTROLLED BY LANNATE®



Heliothis



Spodoptera

Thrips chilli

Grape Mealy Bug

• PHYSIO CHEMICAL CHARACTERISTICS

Chemical Name	Methomyl S-methyl N-[(methylcarbamoyl)oxy] thioacetimidate
Molecular Formula	$C_5H_{10}N_2O_2S$
Molecular weight	162.2
Physical state	Crystalline White Solid (technical) Granule Soluble Powder (formulation)
Acute Oral Rat	17mg/kg body wt.
Acute Dermal Rat	>2000 mg/kg body wt.

RESTRICTED USE PESTICIDE
Due to acute toxicity and carcinogenicity.
For retail sale to and use by certified applicators or persons under their direct supervision and only for those uses covered by the certified applicator's certification.

Dow AgroSciences™

00128258

Telone® C-35

Soil Fungicide and Nematicide

A multi-purpose liquid fumigant for preplant treatment of soil to control plant parasitic nematodes, symphylans and to help manage certain soil borne diseases in cropland.

Not for use in greenhouses or other enclosed areas and not for use in drip or other chemigation applications.

Active Ingredients:
1,3-dichloropropene 63.4%
chloropicrin 34.7%
Other Ingredients 1.9%
Total 100.0%

One gallon of Telone C-35 weighs about 11.2 lb at 70°F. Contains 7.10 lb of 1,3-dichloropropene and 3.89 lb of chloropicrin per gallon.

Keep Out of Reach of Children

DANGER POISON

PELIGRO

Si usted no entiende la etiqueta, busque a alguien para que se la explique a usted en detalle. (If you do not understand the label, find someone to explain it to you in detail.)

Agricultural Use Requirements
Use this product only in accordance with its labeling and with the Worker Protection Standard, 40 CFR Part 170. Refer to the label booklet under "Agricultural Use Requirements" in the Directions for Use section for information about this standard.

Refer to inside of label booklet for additional precautionary information including Directions for Use.

Notice: Read the entire label. Use only according to label directions. **Before using this product, read Warranty Disclaimer, Inherent Risks of Use, and Limitation of Remedies at end of label booklet. If terms are unacceptable, return at once unopened.**

In case of emergency endangering health or the environment involving this product, call 1-800-992-5994.

Agricultural Chemical: Do not ship or store with food, feeds, drugs or clothing.

EPA Reg. No. 62719-302 900-016808 / 00299491

®Trademark of Dow AgroSciences LLC

Produced for
Dow AgroSciences LLC
9330 Zionsville Road
Indianapolis, IN 46268

MADE IN THE USA

STORAGE TANK
Write in EPA Est. No. for producing facility.

EPA Est. No.: _____

REFILLABLE CONTAINER
Write in EPA Est. No. of repacking or retailer facility.

EPA Est. No.: _____

NET CONTENTS: _____

CUT TO OPEN

1996

37,995 lbs

Doña Ana County

Restricted Use

PESTICIDE EXPOSURE AND POTENTIAL HEALTH EFFECTS IN YOUNG CHILDREN ALONG THE U.S. - MEXICO BORDER | Science Inventory

Contact

Citation:

MCMASTER, S. B., S. HERN, AND G. L. ROBERTSON. PESTICIDE EXPOSURE AND POTENTIAL HEALTH EFFECTS IN YOUNG CHILDREN ALONG THE U.S. - MEXICO BORDER. U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-05/082, 2005.

Description:

The purpose of the Pesticides in Young Children - Border States Program is to assess the relationship between health status in children living along the United States and Mexico border and repeated pesticide exposures via multiple sources and pathways. Children's health has been highlighted by public health and environmental agencies as an area of critical concern. As environmental exposure and health scientists, we recognize that the health of children must be zealously protected, if they are to become productive members of society. Child development is a complex and continuous process of growth and change, making children likely to be susceptible to the adverse effects of pesticides and other toxins in their environment. Typical childhood behaviors, such as crawling or playing on the ground and putting non-food items in their mouths, may subject children to risks greatly exceeding those of adults in the same environment. Furthermore, although a substantial body of work exists documenting the effects of pesticides on adults, the effects of pesticides on children have not been systematically examined. Thus, the assessment of pesticide-related health outcomes in children is an essential part of efforts to enhance the health of children nationwide. The importance of this effort is even greater in areas where known factors increase risk of exposure to pesticides.

Defined by the La Paz Agreement of 1983 as the area lying 100 kilometers north and south of the border between the United States and Mexico, the US-Mexico Border Region has several characteristics that

create a potential for exposure to multiple pesticides from multiple sources. The region contains a large proportion of land devoted to year-round agriculture, creating a risk of repeated, year round exposure. Agricultural pesticides are commonly applied by aerial spraying which can contribute to exposure via multiple pathways, including air ,water and food. Residential use of pesticides is also a potential source of significant exposure as the same conditions that make the region suitable for year-round agriculture contribute to the potential for year round household pest infestations. The population of the area is

large, young and growing. The estimated population total for 2000 is 17.9 million, with 11.4 million (63.7%) living on the US side and 6.5 million (36.3%) living on the Mexico side. Almost half (47%) of the border population is under 20 years of age. The population growth rate on both sides of the border is higher than the national rates: growth from 1999 - 2000 was 3.3% for the Mexican side and 2.5% for the US side, compared with respective national rates of 1.8% and 0.9%.

Rationale

Minimal data are currently available concerning the potential health effects and cumulative risks of persistent low-level pesticide exposure in children. The present research program was designed to determine if a health threat exists and, if so, to identify the major risk factors for intervention. A three phase approach was planned to address program objectives. Phase I was designed to build capacity for subsequent studies by pilot testing methods and gathering baseline information on the potential for pesticide exposure; Phase II to identify high risk populations of children and potential health effects associated with exposure; and Phase III to describe pesticide exposure risk factors and possibly conduct a full scale study of specific exposures and health outcomes. This approach is integrated, with each subsequent phase building on the results of earlier efforts.

Purpose/Objective:

To assess the relationship between health status in children living along the United States and Mexico border and repeated pesticide exposures via multiple sources and pathways.

URLs/Downloads:

RESOURCES

- Pesticide Gateway
- Pesticide-Induced Diseases Database
- Safety Source on Pesticide Providers
- ManageSafe™
- Eating with a Conscience
- Quarterly Magazine: Pesticides and You
- Daily News
- State Pages
- YouTube Channel
- Media

New Mexico—School Policies

Select a State: NM

NM

- Overview
- Local Organizations
- Pesticide Policies
- Government Contacts
- School Policies

UPDATES FROM THE DAILY NEWS BLOG

Genetic Engineering Labeling Bill to be Introduced in U.S. Congress
February 28, 2013

Grassroots Organizers, Cutting Edge Scientists, Organic Solutions - The 31st National Pesticide Forum
February 14, 2013

New Mexico Senate Blocks GE Food Labeling Bill
February 7, 2013

Acclaimed Scientists and Activists to Convene at 31st National Pesticide Forum
January 18, 2013

Save the Date - 31st National Pesticide Forum in Albuquerque, NM
December 13, 2012

EPA Cancels Prairie Dog Poison in Four States; Moves Forward with Rodenticide Actions
September 8, 2011

Fracking Biocides Pose Danger to West
February 20, 2007

ALL RELATED STORIES >>

- STATE SCHOOL PESTICIDE LAW
- LOCAL SCHOOL PESTICIDE PROGRAMS
- a href="#localcontact">CONTACTS FOR LOCAL ORGANIZATIONS

STATE SCHOOL PESTICIDE LAW

I. Restricted Spray Zones Around School Property

Overview

Pesticides move off the target site when they are sprayed, whether inside or outside. When sprayed outside pesticides drift on to nearby property resulting in off target residues. Buffer zones can eliminate exposure from spray drift on to school property. As a result, states require buffer zones around schools. In order to adequately protect against drift, buffer zones should, at a minimum, be established in a 2 mile radius around the school's property. Aerial applications should have a larger buffer zone, at least 3 miles encircling the school. Buffer zones should be in effect at all times of the day. It is especially important for spray restrictions to be in place during commuting times and while students and employees are on school grounds.

State Information

New Mexico does not have any statewide requirements regarding restricted spray zones around school property.

II. Posting Notification Signs for Indoor Pesticide Applications

Overview

States use different approaches in providing school pesticide use information to parents, students and staff. Some forms include the posting of notification signs and/or the distribution of notices directly to the affected population. Posted notification signs warn those in the school when and where pesticides have been or are being applied. This is a vehicle for basic right-to-know if the posting occurs in an area where it is easily seen by parents, students and staff. It is important to post signs for indoor pesticide applications because of the extensive period of time students and school employees spend at school. Signs posted prior to commencement of the pesticide application, not after, are more protective. The prior notification system effectively enables people to take precautionary action. Because of the residues left behind after an application, signs should remain posted for at least 72 hours. It takes time for pesticides to start breaking down and some pesticide residues can least for weeks. Signs should also be posted at all main entrances of the building and the specific area sprayed, on the main bulletin board, and, for more comprehensive notification, in the school newspaper or on the daily announcements. Posted signs should state when and where a pesticide is applied, the name of the pesticide applied and how to get further

information, such as a copy of the material safety data sheet (MSDS) and the product(s) label.

State Information

New Mexico requires signs to be posted only for emergecy applications and it does not give any specifics on time.

III. Posting Notification Signs for Outdoor Pesticide Applications

Overview

For a wider range of protection, states should require posting pesticide notification signs for outdoor pesticide applications as well. Students who play sports or people continually on the lawns represent a high risk when applications occur on school property. Dermal exposure can occur when a football player gets tackled, a soccer player slides to make a block or a student sits on the grass to eat lunch or watch a game. Inhalation exposure can occur when a player breathes in kicked up dust and dirt and pesticide residues. Even spectators at a game or passersby face inhalation exposure to pesticides that volatilize or vaporize off the treated area.

State Information

New Mexico requires signs to be posted only for emergecy applications and it does not give any specifics on time.

IV. Prior Written Notification

Overview

Written notification of pesticide use is a good way to make sure that all parents, children and staff are aware and warned of pesticide use in the schools. Limited notification-based registries is a less effective means of notifying people and does not qualify as true right-to-know because of its limited scope. Requiring that individuals place themselves on registries, sometimes only with a doctor's letter, afford only those who already know about toxic exposure the opportunity to be informed about pesticide use in the school. Prior notification should be 72 hours in advance to make sure the information has been received, to get further information regarding the pesticide and to make arrangements to avoid the exposure, if necessary. Notification should include the name of the pesticide(s), a summary of the adverse health effects listed on the Material Safety Data Sheet (MSDS) and label, the day and time, and area of the application and how to obtain a copy of the MSDS and label.

State Information

The state of New Mexico requires schools to establish a parent registry with no time specfics on notification of application. Child care centers are required to provide universal notification to parents and staff 48 hours prior to application.

V. Prohibitions on Use

Overview

Limiting when and what pesticides are applied in and around schools is important to the reduction of pesticide exposure. Pesticides should never be applied when students or employees are in the area or may be in the area within 24 hours of the application. In reality, certain types of pesticides, such as carcinogens, endocrine disrupters, reproductive toxins, developmental toxins, neurotoxins, persistent compounds and substances, bioaccumulative compounds and substances, toxicity category 1 acutely toxic pesticides and ground water contaminants should not be used around children.

State Information

New Mexico prohibits the use of certain pesticides when areas are occupied or will be occupied for the next 6 hours. Licensed childcare centers use of pesticides are prohibited when children are on premises.

VI. Integrated Pest Management

Overview

A good integrated pest management (IPM) program can eliminate the unnecessary application of synthetic, volatile pesticides in schools. The main elements of a good IPM program include: 1) monitoring to establish whether there is a pest problem, 2) identifying the causes of the pest problem, 3) addressing the cause by changing conditions to prevent problems, 4) utilizing pest suppression techniques, if necessary, that are based on mechanical and biological controls and 5) only after non-toxic alternatives have been tried and exhausted, use the least toxic pesticide. An IPM policy should include a written policy guide and a prohibited and acceptable materials list. Material that could be considered after using other methods include boric acid and disodium octoborate tetrahydrate, silica gels, diatomaceous earth, insect growth regulators, insect and rodent baits in tamper resistant containers or for crack and crevice placement only, microbe-based insecticides, botanical insecticides (not including synthetic pyrethriods) without toxic synergists, and biological (living) control agents.

State Information

New Mexico districts and charter schools will develop procedures for the implementation of pest management with consideration for reducing the possible impact of pesticide use on human health and the environment, including people with special sensitivities to pesticides.

COPY OF STATE SCHOOL PESTICDE LAW

New Mexico Title 6, Chapter 29, Part 1, 6.29.1.9: O. (6b)

LOCAL SCHOOL PESTICIDE PROGRAMS

Santa Fe Public Schools
Date Passed: October 1996
IPM: The school's pest management program gives priority to non-chemical methods of pest control, the least-toxic-pesticides are used as a last resort.
Prohibition of Use: Products containing Chlorpyrifos, eg., Dursban are prohibited; applying chemicals to occupied areas is also prohibited.
Other: States when pesticides can be sprayed, the chemicals in the pesticides must be approved by the Federal Occupational Safety and Health hazard communication standards (OSHA).
The school district contracts The Bugman Pest Control, which utilizes IPM methods for pest control. Contact Roman Valdez for more information 1-800-644-3832 or (505) 455-3832.
Contact: Santa Fe Board of Education, 610 Alta Vista Street, Santa Fe, NM 87505

Other Schools with IPM/Notification Policies:
Albuquerque Independent School District
Clovis School District
Rio Rancho
Taos

CONTACTS FOR LOCAL ORGANIZATIONS

New Mexico Public Interest Research Group
PO Box 40173
Albuquerque, NM 87196
Phone: (505) 254-1244
Email: **nmpirg@pirg.org**
http://www.nmpirg.org/

Multiple Chemical Sensitivity Task Force of New Mexico
13 Herrada Road
Santa Fe, NM 87505
Phone: (505) 466-3622
Fax: (505) 466-2690
http://www.anapsid.org/cnd/resources/mcsnewmex.html

For more contacts for local organizations, visit our **Links to Local Organizations.**

For more information contact
Beyond Pesticides, 701 E Street, S.E., Suite 200, Washington, DC, 20003, info@beyondpesticides.org

BEYOND PESTICIDES
701 E Street, SE, Suite 200
Washington, DC 20003
Office Hours: 9 AM-5 PM EST STD
Email: info@beyondpesticides.org
Phone: 202-543-5450
Fax: 202-543-4791

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PEOPLE HELPING PEOPLE

COUNCIL WORK SESSION SUMMARY ROUTING SLIP

Meeting Date July 11, 2016

TITLE: **SAFE APPLICATIONS OF PESTICIDES AND HERBICIDES.**

Are there attachments to the Council Work Session Summary?	**Yes** ☐	**No** ☒
Will there be a Video Presentation for this item?	**Yes** ☐	**No** ☒
Will there be a PowerPoint Presentation for this item?	**Yes** ☒	**No** ☐
If "yes", will a copy of the PowerPoint Presentation be included on the Council Work Session Agenda?	**Yes** ☒	**No** ☐

DEPARTMENT / ORGANIZATION	SIGNATURE	PHONE NO.	DATE
Tim Fulton, Staff Contact	Tim Fulton	541-2586	6/28/16
Sonya Delgado, Dept. Director	Tim Fulton for Sonya Delgado	541-2550	6/28/16
Other			
Assistant City Manager/CAO (if applicable)			
Assistant City Manager/COO (if applicable)		541-2271	6-30-16
City Manager	Daniel Ortega		7-1-16

City of Las Cruces®
PEOPLE HELPING PEOPLE

Council Work Session Summary

Meeting Date: **July 11, 2016**

TITLE: SAFE APPLICATIONS OF PESTICIDES AND HERBICIDES.

PURPOSE(S) OF DISCUSSION:

☒ Inform/Update

☐ Direction/Guidance

☐ Legislative Development/Policy

BACKGROUND / KEY ISSUES / CONTRIBUTING FACTORS:

In recent months, the public has had interest in knowing what particular pesticides and herbicides the City of Las Cruces (City) applies on public property and in public buildings. The public has also expressed interest in the application methods.

This presentation will provide information regarding the use of pesticides and herbicides by the City. The presentation will review the products used by Park Operations, Street and Traffic Operations, and Facilities Management. It will explain how the pesticides and herbicides are used, how they are applied, and the locations where they are applied. The presentation will also provide information regarding what staff and/or a contractor, hired by the City, is licensed to apply a particular product.

SUPPORT INFORMATION:

1. Attachment "A", PowerPoint presentation.

(Continue on additional sheets as required)

SAFE APPLICATIONS OF PESTICIDES AND HERBICIDES

Sonya Delgado

Director, Parks & Recreation

ATTACHMENT "A"

Parks & Recreation Department

Herbicide Usage

PRODUCTS

- Roundup Pro
- Pendulum
- Speed Zone
- Battleship
- Katana

Parks & Recreation Department

Herbicide Usage

PERSONAL PROTECTIVE EQUIPMENT (PPE):

- Rubber Gloves
- Rubber Boots
- Long Sleeve Shirt
- Traffic Vest
- Eye Protection

Parks & Recreation Department

Herbicide Usage

APPLICATION SUPPLIES:

- Anemometer
- Log Book
- Proper Signage
- Safety Data Sheets (SDS)

Parks & Recreation Department

Herbicide Usage

HOW PRODUCTS ARE USED

- Products are used at manufacturer's recommended rates.
- Products are used along with horticultural best practices (i.e. regularly scheduled fertilization, aeration, mowing and irrigation).

Parks & Recreation Department

Herbicide Usage

WHERE PRODUCTS ARE USED

- Parks
- Athletic Fields
- Landscaped Medians
- Landscaped Right-of-Ways
- Municipal Buildings
- Interstate Off-Ramps

Parks & Recreation Department

Herbicide Usage

WHO APPLIES PRODUCT

- Speed Zone is the only product that requires a license – licensed staff is Baldo Garcia NM Department of Agriculture Applicator License #54786 (3A, 3B, 6B).
- Other products do not require licensed applicator, but are applied under the supervision of Mr. Garcia.

Street & Traffic Operations
Herbicide Usage

PRODUCTS

- Ranger Pro Roundup
- Sahara DG (pre-emergent)

LICENSED APPLICATORS

- Ramona Castro
- Raymond Montoya
- Various non-licensed staff spray under the direct supervision of licensed applicators

Street & Traffic Operations
Herbicide Usage

APPLICATION METHODS AND LOCATIONS

- Herbicides are used in adherence with the product's label
- STO developed a Job Safety Analysis (JSA) in coordination with the Risk Department for spraying activities
  - The JSA specifically identifies hazards and controls for all steps of the process: filling/dilution, spraying, and refueling
- Ranger Pro Roundup is currently sprayed on City roads, curbs, ponding areas, channels, and non-landscaped medians and parkways
- Sahara DG is sprayed on non-landscaped City medians and large areas

Public Works / Facilities Mgt. Pesticide Usage

- What is Used
  - BIFEN I/T Insecticide/Termiticide
  - From Control Solutions, Inc.
  - Safety Data Sheet and Instructions on file with City

- How it is Used
  - Controls a wide range of pests in and around structures
    - Ants, spiders, beetles, cockroaches, crickets, flies, etc.
    - Above list not all inclusive
  - Diluted as appropriate for the specified/intended use

Public Works / Facilities Mgt. Pesticide Usage

- Where it is Used
  - Indoors (may also be used outside)
    - Baseboards, corners, storage areas, closets, crevices
    - Around water pipes, doors, windows, attics, eves, cracks
    - Behind/under refrigerators, cabinets, sinks, furnaces
    - Stoves, underside of shelves, drawers, etc.
    - This list is not all inclusive

- Who applies the product
  - Contracted Vendor: Montoya's Pest Control