POISONS ON PETS

Health Hazards from Flea and Tick Products

Authors
David Wallinga, M.D., MPA
Linda Greer, Ph.D.

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Authors
David Wallinga, M.D.
Linda Greer, Ph.D.

NRDC Director of Communications
Alan Metrick

NRDC Washington Communications Director
Elliott Negin

Layout and Production
Bonnie Greenfield

Editing
Matthew Freeman

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EXECUTIVE SUMMARY

Each year, Americans purchase and apply to their pets a vast array of toxic chemicals intended to kill fleas and ticks. These products are designed to poison insects, and they usually do just that. But they can also poison pets and the people who handle them. Moreover, when these products are combined in the home, as they often are, with other toxic chemical products in common use—pesticides, herbicides, and other products—they can pose a serious health risk, especially to children.

Adults are at risk from these flea and tick products as well—pet workers who apply pesticides to animals on a daily basis, for example. But it is children who are most vulnerable. Because children's bodies are still developing, they can be more sensitive to the effects of toxic chemicals than adults. Studies with laboratory animals have raised concerns among scientists that children exposed to certain of the pesticides in pet products—even at levels believed to be safe for adults—face much higher risks, not only for acute poisoning, but also for longer-term problems with brain function and other serious disease. Moreover, children's behavior often makes them more vulnerable than adults. In particular, toddlers' hand-to-mouth tendencies make it easy for toxics to be ingested—and not just by children who pet the family dog and then put their hands in their mouths. Children spend their time where the toxics from pet products tend to accumulate—crawling on rugs, playing with pet toys, handling accumulations of household dust, and more.

Many and perhaps most Americans believe that commercially available pesticides, such as those found in pet products, are tightly regulated by the government. In fact, they are not. Not until the passage of a 1996 law focused on pesticides in food did the Environmental Protection Agency (EPA) begin examining the risks from pesticides in pet products in earnest. To this day, the EPA allows the manufacture and sale of pet products containing hazardous insecticides with little or no demonstration that a child's exposure to these ingredients would be safe. Just because these products are on store shelves does not mean they have been tested or can be presumed safe.

Of course, as bad as these products may be for pet owners and caregivers, they often are worse for the pets themselves. Based on the very limited data available, it appears that hundreds and probably thousands of pets have been injured or killed through exposure to pet products containing pesticides. As with small children, pets cannot report when they're being poisoned at low doses.

Healthier alternatives to these pesticides are readily available. Easy physical measures like frequent bathing and combing of pets can make the use of pesticides unnecessary. Pet products containing non-pesticide growth regulators also can stop fleas from reproducing successfully. In addition, newer insecticides, sprayed or spotted onto pets, have been developed that are effective against fleas and ticks without being toxic to the human nervous system. The safety and effectiveness of these alternatives makes the continued use of older, more toxic pet products tragically unnecessary.

PET PESTICIDES AT WORK

Approximately 90 percent of American households use pesticides. According to one study, 80 percent of families surveyed have used pesticides at home even when a

woman in the household was pregnant, and 70 percent have used them during a child's first six months of life. Half of the surveyed families reported using insecticides to control fleas and ticks on pets. More than a billion dollars a year are spent on flea and tick products.

Unfortunately, the wide use of these products is no indication that they are safe. Quite the contrary, the pesticides they introduce into the home include chemicals that are hazardous to the human brain and nervous system, chemicals that may disrupt the human hormone (endocrine) system, and pesticides suspected of causing cancer.

Flea control products now on the market include seven specific "organophosphate insecticides" (OPs). OPs work by blocking the breakdown of the body's messenger chemical, acetylcholine, thereby interfering with the transmission of nerve signals in the brains and nervous systems of insects, pets and humans alike. In the presence of OPs, acetylcholine builds up in the body. The resulting interference with nerve transmissions is of such a magnitude that it actually kills insects. In overdoses, OPs can also kill people and pets. But even with normal use of flea-control products containing OPs, pets and children may be in danger.

The seven OPs are chlorpyrifos, dichlorvos, phosmet, naled, tetrachlorvinphos, diazinon and malathion. They are the active ingredients in dozens of pet products. A comprehensive list of products appears in Table 1. It includes major pet pesticide brands, such as Alco, Americare, Beaphar, Double Duty, Ford's, Freedom Five, Happy Jack, Hartz, Hopkins, Kill-Ko, Protection, Rabon, Riverdale, Sergeant, Unicorn, Vet-Kem, Victory and Zema.

Organophosphate chemicals are also used on foods and in other common household products designed to kill non-pet-borne insects. For families exposed to these toxic chemicals, however, the route into the home and the specifics of how the chemicals work are less relevant than the plain fact that they pose a health threat. From a health standpoint, a person's combined exposure to one of these OPs, irrespective of its individual uses, is what is important. Further, because the various OPs all function by attacking the same chemical in the body, acetylcholine, exposure to a variety of OPs could have a combined impact.

THE EPA'S ROLE

Actual exposure of children and adults to OPs in pet products has not been adequately measured, and such studies have not been required of manufacturers seeking to put new pet pesticide products on the market. Indeed, until passage of the 1996 Food Quality Protection Act, EPA typically assumed there were no risks from these products, often with little or no scientific basis. In other words, EPA has allowed for decades the manufacture and sale of products containing pet pesticides without demonstration that a child's exposure to the products would be safe.

The 1996 law requires something new of EPA: that it estimate the *accumulated* effect on people of particular pesticides used on food products, accounting not just for exposure from foods, but from all sources. Since OPs used in pet products also are used on food crops, the law applies to these pesticides. Another provision of the law

requires EPA to estimate the cumulative effect on a person from exposure to all pesticides and other chemicals that function in the same way. Because each OP functions by attacking the same chemical messenger in the body, home exposure to a variety of different OPs should be expected to have a cumulative health impact as well. The new law directs EPA to account for this cumulative effect in its risk assessments.

To date, EPA's compliance with the Food Quality Protection Act's provisions has been incomplete. Its risk assessments have been handicapped by flawed and inconsistent assumptions that have served to understate the risk from pet products. For example, in calculating risks of exposure to one chemical, EPA assumes that adults *never* hug their dog, and in a number of instances, EPA makes a variety of unrealistic assumptions about how long children spend in contact with their pets.

Moreover, four years after the enactment of the Act, EPA has yet to comply with the requirement that the Agency account for the cumulative impact of multiple OPs or of other chemicals that function in the same way. Here again, the result is risk assessments that understate the health hazards of exposure to the toxics in pet products. Finally, still today, EPA has never received adequate toxicity tests for these pesticide products long on the market. Of the seven chemicals that are the focus of this report, only one—chlorpyrifos—has been fully tested for its impact on a child's brain and nervous system. And, when the nervous-system testing for chlorpyrifos was recently completed, the results were so disturbing that the manufacturer itself took virtually all indoor uses of the chemical off the market.

Even with those important failings in EPA's methodology, the Agency's formal risk assessments for the seven OPs found both in pet and other products should alarm pet owners and parents: *EPA now calculates that a child's exposure to individual OPs in pet products on the day of treatment alone can exceed safe levels by up to 500 times—50,000 percent*. Exposures to children calculated over a longer period of time can exceed safe levels to an even greater degree. Were EPA to calculate the risks from these products using sound assumptions about how exposure to humans occurs in the real world, and/or were it to comply with the legal requirement that it calculate the cumulative effect of these OPs and of products that function similarly, EPA estimates of the risks from these products would be bleaker still.

The Natural Resources Defense Council is the first to put the individual risk assessments for pesticides from pet products side by side, highlighting the overall risks to children. EPA continues to look at these OP risks only one chemical at a time. The Agency has simply never gotten around to estimating the cumulative risks children face from the myriad uses of all the different OPs to which they are exposed. Once EPA does so, the cumulative risks are sure to exceed EPA's safe levels to a far greater degree.

THE RISKS

Though EPA's assessments of the risks from OPs in pet products are new, EPA has long identified OPs generally as being among the pesticides posing the highest risks to human health. Workers exposed to these chemicals, for example, have experienced

visual problems, slowed thinking, and memory deficits. In truth, however, the principal risk for humans is likely to the brain and nervous system of young children and fetuses, because their systems are still developing when they are exposed to OPs. The risks come in two forms: risks from poisoning, and risks from long-term effects on the brain and nervous system.

▶ **Children's Risk of Acute Poisoning.** OPs are considered the most dangerous pesticides for acute poisoning, particularly for children younger than six. Among incidents reported to poison control centers, children exposed to OPs were three times more likely to be hospitalized, five times more likely to be admitted to a critical care unit, and four times more likely to die, suffer life threatening illness, or develop a permanent disability, than were children who had been exposed to other types of pesticides.

▶ **Children's Long-term Health Effects.** A child's developing brain and nervous system are particularly vulnerable to the toxic effects of OPs because these systems are not fully developed at birth and must continue to form during early childhood. Brain development requires certain cells to first grow, then migrate within the brain, and then connect with one another. Chemicals such as OPs can interrupt and have irreversible effects on this development. Studies have also shown that children exposed to OPs may face increased risks for such later-in-life problems as cancer and Parkinson's disease. A recent epidemiological study, for example, showed that people with any history of in-home exposure to insecticides, like OPs, can more than double the risk of Parkinson's later in life. In addition, four OPs used in pet products increase cancers in laboratory animals, and therefore may cause cancer in humans. One epidemiological study that looked, among other things, at pregnant women who had been exposed to flea and tick products, found that their children were 250 percent more likely than those in a control group to be diagnosed with brain cancer before their fifth birthday.

Of course, it is not only children who are at risk. Pets and pet workers are vulnerable as well.

▶ **Pet Poisonings.** In recent years, hundreds, if not thousands, of pets have been poisoned by pesticide products specifically designed for use on pets. Products containing OPs are among the worst culprits. EPA finds that these pet products are frequently misused and that such misuse should be anticipated by manufacturers. Cats are particularly vulnerable, since they often lack key enzymes for metabolizing or detoxifying OPs. As with children, a cat's small size and unique behavior—in this case, grooming—work against them as well, making them particularly vulnerable to OP poisoning.

▶ **Pet Worker Poisonings.** Over a recent four-period, at least 26 adults working with pesticide pet dips were poisoned. Nearly half of these cases involved the OP, phosmet. Moreover, a survey of nearly 700 adults who worked with flea control products found that these workers were two-and-a-half times more likely to have health problems than workers not exposed to such products. The complaints included statistically significant increases in blurred vision, skin flushing and asthma.

Although each of the OPs we looked at has unsafe pet uses, the properties of these products vary, and so they pose somewhat different threats to the people exposed to them. Some examples:

▶ Pets "dipped" with phosmet. Toddlers who pet a large dog the day of its treatment and then put their fingers in their mouths will receive more than 500 times the safe level of this chemical, according to EPA estimates.

▶ Flea collars containing dichlorvos (DDVP). EPA's preliminary estimates are that toddlers exposed to pets wearing flea collars containing dichlorvos would be exposed to 21 times the safe level just from inhalation of the insecticide emitted from the collar. Adults exposed to the same product would experience exposures ten times greater than safe levels.

▶ Flea collars containing naled. EPA found no uses of naled flea collars that are safe for children ages eight and under. Toddlers' exposures were calculated to be as much as ten times more than EPA's safe level.

▶ Flea collars containing chlorpyrifos. EPA estimates that a toddler exposed to a dog wearing these collars could get more than seven times the level EPA considers to be safe merely from hugging or petting their dog.

▶ Pets sprayed or dusted with tetrachlorvinphos. EPA finds that toddlers exposed to medium- or large-sized dogs that have been sprayed or dusted with tetrachlorvinphos products could face exposures nearly twice as high as EPA's safe level.

▶ Dipping or powdering pets with tetrachlorvinphos. EPA determines that powdering or dipping a single pet with tetrachlorvinphos just twice a year would, over the course of a lifetime, pose a risk of cancer to the person dipping the pet nearly six to seven times higher than acceptable EPA levels. Dipping or powdering multiple pets, or doing so more frequently, would raise cancer risks even higher.

SAFER ALTERNATIVES

The continued exposure of children, pets and animal workers to OPs contained in pet products is all the more distressing because safer alternatives are readily available. Easy physical measures alone, like frequent washing and combing of the pet and vacuuming carpets and furniture, can bring mild flea infestations under control. Alternatives include insect growth regulators, or IGRs, which are not pesticides, but rather chemicals that arrest the growth and development of young fleas. These include methoprene, fenoxycarb and pyriproxyfen and the popular lufenuron (Program®). Alternatives also include newer pesticide products sprayed or spotted onto pets, such as fipronil (Frontline®) or imidacloprid (Advantage®). Particularly when used in combination with physical measures, the safety and effectiveness of

these newer chemical products makes the continued use of pet products containing OPs—and their attendant risks for humans and pets alike—rash and unnecessary.

RECOMMENDATIONS

The threats posed to humans and pets by OPs in pet pesticides are intolerable. The Natural Resources Defense Council recommends the following:

▸ Pet owners should begin using safer products on their pets, avoiding OP-based pet products. Safer products are best combined with such simple physical measures as brushing pets regularly with a flea comb while inspecting for fleas, and mowing frequently in areas where pets spend the most time outdoors.

▸ Pregnant women and families with children should cease using OP-based products immediately.

▸ Children should never apply flea shampoos, dusts, dips, etc. containing OPs to their pets. EPA has overlooked and underestimated the particular risks to children when evaluating the safety of these products for home use.

▸ Retailers should remove OP products from their shelves and seek to educate customers about the merits of safer alternatives.

▸ EPA should move immediately to ban the use of pet pesticides containing OPs.

▸ EPA should consider also banning pet products that contain carbamates—a class of insecticides closely related to OPs, and sharing with OPs the same basic biological mechanism of harm. Likewise, homeowners and retailers should avoid the purchase and sale of these carbamate-containing products.

▸ EPA should take steps to better inform veterinarians, pet owners and the general public about safer alternatives for the control of fleas and ticks on pets.

For most pet owners, the family dog or cat is a beloved member of the family. Unfortunately, products often used to protect pets from fleas and ticks carry serious health hazards—not just for the pets, but for the children who play with them, care for them, and love them. Safer alternatives are available—alternatives that will effectively protect pets from insects without introducing intolerable health hazards into the home. Consumers, manufacturers, veterinarians, retailers and the government all have an important role to play in eliminating these risky pet products and bringing safer alternatives into common use.

If you think you or your pet has been affected by a pet product containing pesticides, call your local poison control center if you need immediate help, and report the incident to EPA's National Pesticide Telecommunications Network, at (toll free) 1-800-858-7378.

Table 1
EPA Registered Pet Products Containing Organophosphates Insecticides

Insecticide	Dog Product	Cat Product
Chlorpyrifos	Zema 11-month collar* Sergeant's Flea + Tick Collar Sergeant's Fast-Acting Flea & Tick Collar For Dogs Hartz 330 Day Flea & Tick Collar For Dogs Sandoz Dursban Collar For Dogs (RF-9411) Methoprene/Chlorpyriphos Combination Collar For Dogs Happy Jack Tri-Plex Flea And Mange Collar Sardex Sulfodene Scratchex Flea And Tick Collar For Dogs Victory 12 Full Year Collar With Dursban For Large Dogs	*Sulfodene Scratchex Flea and Tick Collar for Cats** Happy Jack 3-X Flea, Tick And Mange Collar For Cats Victory II Full Season Cat Collar
Dichlorvos	Sergeant's Sentry Collar For Dogs Sergeant's Fast-Acting Flea & Tick Collar For Dogs Sergeant's Dual Action Flea And Tick Collar For Dogs Flea Collar For Dogs Alco Flea Collar For Dogs - Black, Clear & Glitters Freedom Clear Dog Collar	Sergeant's Sentry Collar For Cats Flea Collar For Cats Alco Flea Collar For Cats—White Alco Flea Collar For Cats—Clear Alco Flea Collar For Cats—Glitters Freedom Clear Cat Collar
Naled	Sergeant's Sentry IV Flea & Tick Collar (for dogs)* Sergeant's (R) Sentry V Flea & Tick Collar For Dogs* Sergeant's Flea + Tick Collar*	Sergeant's Sentry IV Flea & Tick Collar* Sergeant's (R) Sentry V Flea & Tick Collar For Cats*
Phosmet	Unicorn Insecticidal Dust* Vet-Kem Kemolate Emulsifiable Liquid* (for dipping)	
Tetrachlorvinphos	Hartz 2 In 1 Collar For Dogs* *Hartz 2 in 1 Flea and Tick Control Collar with 14.5% Rabon** *Hartz 2 In 1 Plus Seven Month Collar For Dogs** Hartz Rabon Collar With Methoprene Americare Rabon Flea & Tick Collar For Dogs Rabon Dust For Dogs And Cats *Hartz 2 In 1 Flea & Tick Powder For Dogs** Clean Crop Livestock 1% Rabon Dust Hartz 2 In 1 Flea & Tick Pump For Dogs II Hartz Rabon Spray With Methoprene Pump Formulation *Hartz Rabon Flea and Tick Dip for Dogs and Cats** *Hartz 2 In 1 Flea And Tick Spray With Deodorant For Dogs III** *Hartz Flea and Tick Repellent, containing 1% Rabon**	*Hartz 2 in 1 Collar for Cats** *Hartz 2 in 1 Plus Long Lasting Collar for Cats** *Hartz 2 in 1 Plus 7-month Collar for Cats** Hartz Rabon Collar With Methoprene Americare Rabon Flea & Tick Collar For Cats Rabon Dust For Dogs And Cats *Hartz 2 in 1 Flea & Tick Powder for Cats* Clean Crop Livestock 1% Rabon Dust Hartz 2 In 1 Flea & Tick Pump For Cats II* Hartz Rabon Spray With Methoprene Pump Formulation* *Hartz Rabon Flea and Tick Dip for Dogs and Cats**
Malathion	Kill-Ko Malathion Concentrate Riverdale Malathion 5 Ford's 50% Malathion Emulsifiable Concentrate SMCP 5% Malathion Dust Hopkins Malathion 57% Emulsifiable Liquid Insecticide-B 50% Malathion Emulsifiable Concentrate 55% Malathion Concentrate 50% Malathion Micro-Gro Cythion Premium Grade Malathion E-5 Fyfanon 57 EC	Kill-Ko Malathion Concentrate Riverdale Malathion 5 SMCP 5% Malathion Dust Hopkins Malathion 57% Emulsifiable Liquid Insecticide-B 50% Malathion Emulsifiable Concentrate 55% Malathion Concentrate 50% Malathion Micro—Gro Cythion Premium Grade Malathion E-5 Fyfanon 57 EC
Diazinon	Protection 150 Reflecting Flea And Tick Collar For Dogs Protection Plus 150 Flea And Tick Collar For Dogs With EFA Protection 150 Flea And Tick Collar For Dogs And Large Dogs Protection 300 Flea And Tick Collar For Dogs Diazinon-Pyriproxyfen Collar For Dogs And Puppies #1, #2, #3 Double Duty Plus Flea & Tick Collar With Nutrisorb For Dogs Double Duty Reflecting Flea & Tick Collar Freedom Five Flea And Tick Collar For Dogs Beaphar Tick & Flea Collar For Dogs Double Duty Flea & Tick Collar For Dogs	Protection 150 Reflecting Flea And Tick Collar For Cats Protection Plus Flea And Tick Collar For Cats Protection 150 Flea And Tick Collar For Cats Double Duty Plus Flea & Tick Collar With Nutrisorb For Cats Double Duty Reflecting Flea & Tick Collar For Cats Freedom Five Flea And Tick Collar For Cats

Source: James Beech, U.S. EPA Office of Pesticide Programs, Pet Products Registered for Seven Organophosphates, June 3, 2000.

Note: Products in regular type are those registered with EPA as of June 3, 2000. Asterisks indicate pet products known to form the basis for EPA's risk assessment for that chemical. Dichlorvos, diazinon and malathion risk assessments did not list particular products. Italicized products are those for which, subsequent to the risk assessment, manufacturers now indicate they will no longer maintain registration of the product. In most, if not all such cases, however, the products remain in use at the time this report was prepared.

INTRODUCTION

Approximately 90 percent of American households use pesticides.[1,2,3] In one study, 80 percent of families surveyed had used pesticides at home during a pregnancy, and 70 percent had used them during a child's first six months of life.[4] Half of the surveyed families reported using insecticides to control fleas and ticks on pets. More than a billion dollars yearly are spent on these flea and tick products.[5]

Despite popular perception to the contrary, the widespread use of these pet products is no indication that they are safe. On the contrary, the pesticides they introduce into the home include chemicals toxic to the brain and nervous system, chemicals that may disrupt the human hormone (endocrine) system, and pesticides suspected of causing cancer. In June 1999, the Centers for Disease Control and Prevention (CDC) warned that flea control shampoos, dips and other pet products containing insecticides "may pose a risk to consumers."[6] The Environmental Protection Agency (EPA) has also found exposure to some pet products to be unsafe.

Until recently, government oversight of the safety of pesticide products used on pets was virtually nonexistent. Before 1987, new pet products were not consistently required to undergo animal safety studies prior to being registered.[7] By 1996, EPA had registered nearly 1,400 such products, but typically had performed little or no scientific analysis to assess the level of risk posed to pets or their owners.[8]

This changed when Congress unanimously passed the Food Quality Protection Act (FQPA) in 1996. The law imposed a strong new health-based standard on all pesticides also used on foods. It also requires something new of EPA: that it consider the *accumulated effect* from individual pesticides used on food products, accounting not only for the direct risks from contaminated foods, but also for any other non-food use of the same pesticide.

A FOCUS ON ORGANOPHOSPHATE PRODUCTS

Because of their inherent toxicity and widespread use, seven organophosphate (OP) pesticides are the focus of this report. They are chlorpyrifos, dichlorvos, diazinon, naled, phosmet, tetrachlorvinphos and malathion.

The EPA has begun to assess the hazards of these seven chemicals (used both on pets and on food crops), as required by the FQPA in 1996. EPA's assessments include a look at acute risks; and, indeed, the Agency has long considered OPs to be among the pesticides posing the greatest risk to human health from acute poisonings.[9],[10] However, the scientific evidence also points to possible long-term effects from exposure to these pesticides, including persistent effects on the nervous system and brain, cancer, and disruption of hormone function.

EPA has conducted formal risk assessments for the seven OPs using the best science available to it. The results provide both pet owners and parents with cause for alarm: risks are often excessive, and greatest for fetuses and very young children whose nervous systems are still developing. EPA now calculates, for example, that toddlers exposed to pets treated with certain OP products during just one day can exceed safe levels by more than 500 times. When assessed over a longer period

of time, such as the entire month after a pesticide treatment, a child's exposure can be even more excessive—more than 690 times greater than EPA's safe level, in one instance.

A second new requirement of the 1996 law is that EPA consider the cumulative health impact from a person's exposure to all pesticides and other chemicals that function in the same way. OPs, along with the closely-related carbamate pesticides, are designed specifically to kill insects by disrupting the breakdown of acetylcholine in their nervous system. But EPA has yet to perform any cumulative assessment of the risks from these neurotoxic chemicals. If it had performed these assessments, the cumulative risks calculated for fetuses and children could only go higher.

ADDITIONAL CONCERNS ABOUT OP PET PRODUCTS

Pet products in general (shampoos, sprays, dips, dusts, collars, spot-ons and pills) contain many different kinds of pesticides—organophosphates, the subject of this report, as well as carbamates, pyrethrins, synthetic pyrethroids, etc. A single pet product can include several "active" ingredients; that is, manufacturers may combine different pesticides in the same product, each with a specific contribution to the overall mixture's ability to kill insects.[11] Homeowners also commonly apply multiple products to their pets to control fleas, either simultaneously or in sequence.[12] So a pet shampoo may be followed by treatment with a dip, spray, collar or all of the above. Thus, the treatment of a single pet with one or more pet products inevitably will expose family members to one or several different pesticides ingredients.

People interact intensively with their dogs and cats—they hug, pet and share living space with them. In addition, pets spend most of their time living at floor level, loafing, rolling, and sleeping on the same carpets, floors and blankets as people. As a result, those people most vulnerable to the toxic effects of insecticides—pregnant mothers, toddlers, and the elderly, probably will be exposed to these chemicals as their pets are treated.

The organophosphates found in flea and tick products are also used against lawn and garden pests, and against termites and other insects in the home (See Table 2). Depending on the particular OP, the number of treatments used to control pets' infestations can comprise anywhere from less than one percent to nearly 93 percent of all household uses of that chemical. Above and beyond direct uses in the home, however, every OP found in pet products also is used on crops; sometimes the same OPs therefore come home on food from the grocery store. As a result, pesticide exposures from treated pets are *in addition to* exposure to these same chemicals from food and other non-pet sources. Families using OP products to protect pets therefore contribute to what probably is routine exposure of their children and pets to these nervous system toxins through multiple routes.

Risks from pet products containing organophosphates, in comparison to the risks from other home uses of organophosphates, are notable for several reasons. EPA's own assessments show these risks to be quite high, often higher than the risks posed by other household insecticide products—especially for toddlers. Second, these risks

have been overlooked or ignored in the past, so the public may be less aware of them. Third, they are thoroughly unnecessary since safer, effective alternatives to flea control are readily available. Finally, risks from these products are important because they involve frequent applications to pets, which bring insecticides into close proximity to children and other family members who may be more vulnerable to them.

Table 2
Pet and Non-Pet Uses of Seven Organophosphates

OP Ingredient (Common Trade Names)[a]	Products Used on Family Pets[b]	Annual Pet & Kennel Treatments As Percentage of Entire Household Market (1990 data)[c]	Non-Pet Products Used In And Around Homes Containing the Same OP[d]	Food Uses Likely to Pose Risks to Children (Total No. of Registered Crops)[e]
Chlorpyrifos (Dursban, Lorsban)	Flea & tick collars for dogs and cats	5.6%	Ant and roach sprays, liquids, and baits; household flea sprays; wasp and hornet sprays; termites; indoor crack and crevice insecticide; houseplants; lawn and turf; spider sprays and dusts; cricket and grasshopper bait.	Apples, beans, grapes, peaches, oranges, peaches, pears, peas, tomatoes (54)
Dichlorvos (DDVP, Vapona)	Flea & tick collars for dogs and cats	1.8%	Ant, flea and roach sprays; wasp, hornet and bee sprays, indoor foggers and bombs; fly baits and sprays; no-pest-strips.	Tomatoes (5)
Naled (Dibrom, Legion)	Flea & tick collars for dogs and cats	NA	Indoor and outdoor fly sprays; fly and mosquito spray; ornamental plant insecticide.	Beans, grapes, oranges, peaches, peas, tomatoes (37)
Phosmet (Imidan, Prolate)	Dog dust, sponge-on or dip	NA	Outdoor home insecticide powder.	Apples, grapes, peaches, pears, peas, potatoes, tomatoes (23)
Tetrachlor-vinphos (Gardona, Rabon)	Flea & tick dips, powders, sprays and collars for dogs and cats	92.8%	Outdoors household insecticide use.	Apples, peaches, pears, tomatoes (8)
Malathion (Cythion)	Flea & tick dusts, dips	1.7%	"General purpose" insect sprays and dusts; ant and roach bombs; wasp, bee and hornet killer; home, lawn and garden sprays and dusts; orchard sprays.*	Apples, beans, grapes, oranges, peaches, pears, peas, potatoes, tomatoes (144)
Diazinon (Diazinon, D-Z-N)	Flea & tick collars for dogs and cats	< 1%	Ant, flea and spider sprays; roach bombs, powders and sprays; wasp and hornet bombs; indoor crack and crevice treatment; lawn and garden insecticide sprays and dusts.	Apples, beans, grapes, peaches, pears, peas, potatoes, tomatoes (63)

Sources:

[a] From USEPA, Organophosphate Pesticides: Common Names and Common Trade Names, Office of Pesticide Programs, http://www.epa.gov/oppbead1/matrices/oplist.htm; *also* from USEPA, *Recognition and Management of Pesticide Poisonings*, OPPTS, 1999.

[b] These are registered uses as of June 3, 2000; manufacturers of some pet products containing chlorpyrifos and malathion have indicated they will voluntary withdraw registration of these products, but the products remain on store shelves and in homes as this report goes to press.

[c] Compiled from data in Table D.1, Whitmore RW, Kelly JE, Reading PL, National home and garden use survey, Report prepared for US EPA, Research Triangle Institute, March 1992. This 1992 report (using 1990 data) is EPA's most recent national survey concerning home and garden use of pesticides. Phosmet and naled products were apparently not among the 77 included in the survey.

[d] *Report Of Pesticide Products For User Specified Active Ingredients And Use Site Combinations*, supplied by James Beech, USEPA, updated as of June 3, 2000.

[e] Consumers Union, *Worst First, High-Risk Insecticides, Children's Foods & Safer Alternatives*, Washington, D.C., September 1998; *also* USEPA, Staff Background Paper #5.2: Organophosphate Pesticide Crop Use Sites, Office of Pesticide Programs, http://www.epa/oppsrrdl/op/primer.htm, May 27, 1998.

OVERVIEW OF THE REPORT

It is understandable that consumers might assume these products would not be on the market unless the government had determined they were safe. This report documents the contrary: that the products are on the market because, until very recently, the government has not taken a hard look. Worse, where the government has done assessments, it has found that many products containing organophosphates are, in fact, not safe at all. Yet the products continue to be sold.

This report makes public EPA's risk calculations for organophosphate pet products and makes clear the types of hazards these chemicals pose to people and to the pets themselves. It critiques EPA's risk assessments as not being sufficient to protect families during the common practice of cuddling their pets. Finally, it describes alternatives available to control fleas from pets in the house more safely.

CHAPTER 1

The Safety of Pet Products

People are exposed to toxic chemicals in flea and tick products in many ways. They get the chemicals on their skin when they first apply the products to pets. The exposure then continues for weeks or months as they pet and cuddle their pets—until the chemical residues all wear off. Even flea and tick collars can leave high levels of chemical particles on pets. So, petting a dog or cat wearing such a collar will expose the person. People can even ingest the chemicals directly, if they pet a treated animal and then put hands in their mouth without first washing thoroughly. The chemicals can also enter the body when someone breathes in particles and fumes—either during application of the product to the pet, or as the chemicals linger on fur for days or weeks afterwards.

How do scientists calculate whether this chemical exposure is safe for families and their pets? They use a technique called "quantitative risk assessment." Risk assessments look first at the inherent hazards of the chemical, by referring to toxicity tests done on laboratory animals as well as to health studies of people who have been exposed either accidentally or at work. Following the hazard assessment, scientists study the ways and extent to which people might be exposed to the chemical in their daily lives. They then simply multiply the hazard by the exposure number and come up with a figure they can compare against "safe" levels or standards (See Spotlight on page 6, "How EPA Determines 'Safe' Pesticide Exposures").

As straightforward a process as that might seem, a major complication arises: much of the data necessary to precisely calculate both the hazard and exposure pathways to chemicals is usually lacking. EPA knows quite a bit, for example, about the effect of large doses of chemicals on laboratory animals, but usually knows little about direct effects on people exposed to the chemical at concentrations found in the home. EPA knows that people love their pets but does not actually know how frequently children might pet them and then put their hands in the mouth without washing. Finally, EPA knows that not every child is the same, but it does not know how much children of different ages and backgrounds will vary in their petting and hand-to-mouth behavior, or in their innate sensitivity to chemical exposures. In the absence of specific data such as these, regulators can complete risk assessments and calculate risk levels only by using various assumptions.

Ask any parent of a toddler how often their child puts a hand in his mouth and they are likely to roll their eyes and say "every minute." Yet EPA's risk assessments

SPOTLIGHT

How EPA Determines 'Safe' Pesticide Exposures

EPA characterizes toxic chemical risks by considering the inherent toxicity of a chemical as well as the amount of exposure people get to it.

Typically, EPA's ascertains the toxicity of a pesticide from controlled studies where laboratory animals are given specific doses—either by inhalation, ingestion or application to the skin—and then observing for toxic effects. The dose level found not to lead to an adverse effect, called the no-observed-adverse-effect-level (or NOAEL), serves as the basis for EPA's safety levels, or reference dose (RfD).[13] The reference dose expresses, in milligrams per kilogram of body weight per day (mg/kg/day) the greatest amount of pesticide EPA estimates a person can be exposed to each day and still be considered safe. In risk assessments, this "safe" dose or reference dose is also referred to as the "level of concern."

EPA may set an acute reference dose, based on adverse effects seen in laboratory animals which have been exposed for a short period of time; or it may determine a chronic reference dose based upon an average daily dose over a longer period of time. The latter typically are lower.

Often, a chemical with extreme inherent toxicity (i.e. a very low NOAEL and RfD) neverthless is calculated to pose minimal risks, if EPA determines that a person's likely exposure to that chemical is minimal or fleeting. This is done through the use of exposure scenarios that attempt to quantify the amount of exposure that is likely to occur to an "average" person under a range of conditions. In essence, very dangerous chemicals can be calculated as not a problem if EPA assumes people are rarely or never exposed to them.

EPA's exposure scenarios are often scientifically controversial because information does not exist to make them particularly accurate. For example, EPA has little information on how often toddlers put their hands in their mouths, which has important implications for the amount of pesticide they might ingest after petting their dog or cat.

generally assume that toddlers will put their hands in their mouth *less than twice an hour*. Children commonly sleep with their pets, and yet EPA generally assumes a toddler's pet contact will not exceed 2 hours per day.

EPA *says* the assumptions used in its risk assessment when actual data are lacking are "conservative," meaning that if they're wrong, they err on the side of overstating true risks, and overprotecting the public.[14] However, many outside scientific advisors have disagreed with this and are concerned that EPA's risk assessments often *underestimate* rather than overestimate risks to many children.

In the preparation of this report, the Natural Resources Defense Council obtained and reviewed EPA's risk assessments for seven organophosphate insecticides (OPs) found in pet products. NRDC researchers found that these assessments and their underlying assumptions are not "conservative" at all. To the contrary, many common assumptions in EPA's risk assessments about exposure to pets, such as how often they are petted and how often children put their hands in their mouths, instead would tend to greatly underestimate exposure.

Furthermore, NRDC found the risk assessments for these seven chemicals were extremely inconsistent. Whereas some assessments consider exposure through

accidental ingestion of pesticide residues transferred to one's hand after petting a dog, other assessments ignore this route of exposure completely, considering only absorption of the pesticide across the skin. Some assessments assume that only children hug their pets, while others assume the whole family does so. Some assume that families own only a small- or medium-sized dog, and thereby underestimate the exposure from petting a large dog. Each of these assumptions (and others) are critically important to the final risk number EPA calculates, yet NRDC found little "method to the madness" in these assessments. The inconsistencies thus very much undermine EPA's rosy-eyed view that its risk assessments are protective.

In addition, the risks identified by EPA for each OP found in pet products typically are discussed in isolation, rather than in the context of the combined risk from all uses of this same insecticide at home, on the garden, on foods, and elsewhere. Finally, EPA's risk assessments overlook the fact that families commonly use multiple pesticide products on their pet at once (such as a flea shampoo followed by a flea collar), as well as multiple different OPs around the home and garden, and that risks from these various products will also accumulate.

Most troubling is that despite all of these shortcomings in its risk assessments for pet products containing organophosphates, *EPA still calculates these risks as being extremely high, particularly for children and toddlers. Nevertheless, the Agency has not acted to take the products off the market or to notify consumers of their results.* Some of the worst problems are as follows:

► **Pets that have been "dipped" with phosmet.** Toddlers petting a large dog the day of its treatment with phosmet and then putting his or her fingers in the mouth will receive more than 500 times the safe level of this chemical, according to EPA estimates.

► **Flea collars containing dichlorvos (DDVP).** EPA's preliminary estimates are that toddlers exposed to pets wearing flea collars containing dichlorvos would be exposed to 21 times the safe level of this chemical just from inhalation of chemical particles emitted from the collar. Adult exposures to the same product result in ten-fold exceedances.

► **Flea collars containing chlorpyrifos.** EPA found *no* uses of naled flea collars that are safe for children ages eight and under. Toddler's exposures were calculated to be as much as ten times more than EPA's safe level.

► **Flea collars containing Dursban.** EPA estimates that toddlers exposed to a dog wearing these collars could get more than seven times the level EPA considers to be safe merely from hugging or petting their dog.

► **Pets sprayed or dusted with tetrachlorvinphos.** Based on EPA estimates, toddlers exposed to a medium- or large-sized dog that has been sprayed or dusted with tetrachlorvinphos products could face exposures nearly twice as high as EPA's safe level.

► **Dipping or powdering pets with tetrachlorvinphos.** EPA determines that powdering or dipping a single pet with tetrachlorvinphos just twice a year would, over the course of a lifetime, pose a risk of cancer to the person dipping the pet nearly six to

seven times higher than acceptable EPA levels. Dipping or powdering multiple pets, or doing so more frequently, would raise cancer risks even higher.

EPA'S RISK ASSESSMENTS SERIOUSLY UNDERSTATE RISKS TO CHILDREN

Historically, manufacturers have provided EPA with little or no data quantifying a person's *actual* exposure to the toxic chemicals found in pet products before putting the products on the market. Neither can this data be found in the open scientific literature. For example, the first independent study to quantify exposure to pesticide particles from flea collars has only recently been conducted, and its final results are not yet available.[15]

In the absence of such studies, EPA relies on assumptions to complete its risk assessments—typically the assumptions found in a set of 1997 guidelines, called the *Draft Standard Operating Procedures for Residential Exposure Assessments* (Residential SOPs). Many assumptions in these draft guidelines have been criticized by EPA's own Scientific Advisory Panel, as well as outside scientists, as not being protective of children.[16,17] For example, the 1997 guidelines recommend the use of single *average* values to describe certain characteristics of children—such as their weight. It would be much more protective of children to use "worst case" values—to cover situations that might arise when an unusually small child, for example, spends an unusually long time playing with a pet.

In fact, the most recent scientific review of the exposure assumptions in EPA's draft Standard Operating Procedures recommended that EPA end its reliance on all "average" values when making risk estimates. It said:

> *Medically, a screening tool is designed to be highly sensitive (e.g., few false negatives) often requiring a trade-off in being less specific (e.g. allowing more false positives). If the Standard Operating Procedures are to be used as a screening tool, they should reflect this orientation and choices should err on the side of overestimating exposures. Thus, using means and other measures of central tendency [to describe the parameters most relevant to exposure] would not be appropriate. Rather choosing "numbers" that reflect the right side of all distributions, be it the upper limits of the range of measurements when few data are available or the upper bound of a 95th or 99th percentile, is much more conservative and protective.*[18]

Some specific examples to better illustrate the problem:

▶ **EPA assumes all "toddlers," aged 1 to 6 years, weigh 33 pounds.** EPA often bases its assumption about weight for all children from ages one to six on the mean or average weight for three year-olds, 33 pounds. But among one-year olds, over 95 percent of girls and almost 90 percent of boys would be expected to weigh less than 25 pounds.[19] Because many children are smaller than the average three year-old, and thus would get more pesticide exposure per pound of weight, the use of an average weight for them is not sufficiently protective.

► **EPA assumes toddlers have contact with only one treated pet per day.** This assumption tends to understate risks to toddlers in real-world families with multiple pets, particularly when family pets are treated concurrently with flea and tick products, as would be expected in the case of a household flea infestation. EPA's assumption appears to be arbitrary, with no scientific references given.[20]

► **EPA assumes that the single treated dog will be a medium-sized dog or smaller.** By sometimes limiting its assessments to the assumption of a medium-sized dog, EPA lowers its risk estimates; that's because larger dogs simply carry more insecticide residues. This assumption therefore will understate the risks to children in homes with larger dogs.

► **EPA assumes a toddler's pet contact will not exceed 2 hours per day.** This assumption ignores children who sleep and play extensively with their pets. In its risk assessment for chlorpyrifos, EPA's assumes a range of exposure that is even less realistic than two hours per day—it assumes that a person's average daily exposure to chlorpyrifos from an 11-month flea collar will be equivalent to as little as two minutes of "vigorous" pet contact per day, and no more than 1.75 hours of such contact per day.[21]

► **EPA assumes a toddler exposed to a treated pet will put his or her hand in the mouth less than twice per hour.**[22] It will not surprise parents that EPA's scientific advisors singled out this assumption as being particularly inadequate. Though EPA describes its use of 1.56 times/hour as a "conservative" assumption, a recent study of just 30 children found an *average* hand-to-mouth rate of 9.5 times per hour—more than 6 times this frequency.[23] The children in the study with the most hand-to-mouth behavior did so 26 times per hour *on average*, with some children putting hands to mouth up to 62 times per hour.[24,25] If a study so small identifies children with 62 hand-to-mouth contacts per hour, then the vast majority of children must far exceed EPA's assumption of 1.56 such contacts per hour. EPA recently proposed to change its guidelines to replace this value with one of 20 hand-to-mouth events per hour. This would still be average value, however, and therefore not reflective of the true behavior of children who exhibit the greatest amount of hand-to-mouth behavior. Moreover, until EPA actually revises and releases the new guidelines, it presumably will continue to use the dramatically low value of 1.56 times per hour.

► **EPA assumes that children will mouth only their hands and fingers.** EPA's scientific advisors pointed out in 1999 that the Agency's current pesticide risk assessments are incomplete since they assume a child's mouthing behavior only includes hand-to-mouth exposures.[26] In reality, children put objects other than fingers in their mouth, such as toys or "feral" food, and these objects often carry pesticide particles that will be ingested. Feral food is food that has been dropped on the floor, only to pick up dust and pesticides particles from contaminated linoleum, carpet or other household surfaces.

These examples demonstrate how some of EPA's "conservative" assumptions for estimating a child's exposure to pesticides in pet products may be more than an order of magnitude too low to adequately protect many, if not most, children.

Although EPA has indicated that its draft guidelines for estimating household exposures will soon be revised, *no changes are contemplated for those assumptions most directly relevant to pesticides exposures from pet products*.[27] Therefore, the problems identified in this report will remain in effect even after EPA revises its deficient guidelines.

EPA EXAMINES EXPOSURE TO PET PRODUCTS IN ISOLATION

Every organophosphate in a pet product is also found in non-pet products (See Table 2). Seventeen OPs, including the seven that are the subject of this report, are registered by EPA for "residential" uses, including not just use on pets, but also use in or around homes and schools, on playgrounds, lawns and gardens. From the perspective of a child's safety, these non-pet uses cannot be ignored. To a child or fetus, whether OP exposure occurs through use on pets, on lawns or through contaminated food matters less than their total or aggregate exposure across all possible routes.

The real-life cumulative exposure of children to multiple organophosphates, across multiple routes, is not considered by EPA when it compares exposure to these insecticides in pet products to "safe" levels. This contributes to a very serious underestimation of the total risk a family faces from use of OP products in and around the home and should be foremost in the minds of readers.

DETAILED RISK ESTIMATES FOR INDIVIDUAL ORGANOPHOSPHATES IN PET PRODUCTS

The risks of individual OPs, and the shortcomings of EPA's approach to risk-assessment process may be best understood by examining each of the seven implicated OPs separately.

CHLORPYRIFOS (DURSBAN)

Use: dog collars

EPA estimated risk to children: At least 7 times safe levels

Chlorpyrifos, one of the nation's most heavily used insecticides overall, is found in cat and dog flea collars.[28] Until 1998, it also was marketed in pet dips, at which point Dow AgroSciences voluntarily withdrew the registration for that particular use.[29] Chlorpyrifos also is commonly used on crops, and until recently has been widely used in residential settings like homes and gardens, schools, hospitals, and child-care facilities.[30]

In addition to its short- and long-term effects on the brain and nervous system, self-reported cases suggest—although controlled studies have not confirmed—that chlorpyrifos may in some cases contribute to multiple chemical sensitivity.[31]

Mounting evidence suggests the chemical also may disrupt the hormone or endocrine system in young, developing animals.[32]

Reviewing reports from the nation's poison control centers in 1997, EPA investigators found "Chlorpyrifos is one of the leading causes of acute insecticide poisoning in the United States."[33] In April 2000, two pediatricians who formerly served as high-level EPA officials, along with ten other public health scientists, called on EPA to ban chlorpyrifos from any use around homes, schools, and childcare settings, citing research showing that the chemical is directly toxic to the developing brain in mammals, even at relatively low doses.[34]

In June 2000 Dursban's manufacturer, Dow AgroSciences, and the EPA jointly announced an agreement that the former would stop manufacturing chlorpyrifos for use on pets, along with virtually every other use around homes and schools, although products containing Dursban would continue being sold until stocks were eliminated. In making the high-profile announcement, EPA Administrator Carol Browner claimed it was "clear the time has come to take action to protect our children from exposure to this chemical."[35] NRDC has included chlorpyrifos in our report on pet products because it will continue to be sold on the market until supplies are depleted, and because many people will have chlorpyrifos products on their home shelves to use for years to come.

Concurrent with the June announcement, EPA released its final risk assessment for chlorpyrifos. The assessment concluded that use of an 11-month Dursban flea collar on dogs exposes an "average" 33 pound toddler to seven times EPA's safe level, and on cats to nearly two times its safe level (See Table 3).

Risks Could Be Higher Still

Even these official estimates of a very high risk of chlorpyrifos flea collars are likely too low. For one thing, EPA looks only at absorption through the skin and ignores the likelihood that children will ultimately put their hands in mouth and ingest any chemical residues on their hands. Overlooking these hand-to-mouth exposures is significant since children certainly will stroke their pets, and because hand-to-mouth

Table 3
Child and Adult Exposures to Chlorpyrifos Collars

Person Exposed	Source of Exposure	Exposures Considered To Be	Percentage of EPA's Safe Level (Acceptable = 100 Percent or less)	
			Dog Collar	Cat Collar
Toddler (33 lbs.)	Collar	Skin (dermal) only[a]	710	190
	Collar	50% skin, 50% inhalation[b]	12,500	3,200
Adult	Collar	Skin (dermal) only[a]	150	40
	Collar	50% skin, 50% inhalation[b]	2,600	670

[a] Based on Table 15 in USEPA, Human Health Risk Assessment: Chlorpyrifos, (Final Risk Assessment) June 8, 2000, p. 91.

[b] Based on USEPA Memorandum from D. Smegal to J. Rowland, Occupational/Residential Handler and Postapplication Residential Risk Assessment for Chlorpyrifos, (Preliminary Risk Assessment) October 5, 1999, p. 49.

exposures are among the most important routes for children involving other pet products. (See the risk assessment for phosmet, for example.)

EPA also ignores any inhalation of chlorpyrifos, despite the chemical's volatility. When EPA did include the inhalation route in its *preliminary* October 1999 risk assessment, estimates of total chlorpyrifos exposure (and risk) were much higher than for absorption across skin alone.[36] EPA's estimate in this earlier assessment was that toddlers exposed to a dog wearing a chlorpyrifos collar could get more than 125 times the level of exposure EPA considers to be safe. In its *final* assessment, EPA gives no indication of why it drops inhalation exposures, but instead offers the unsubstantiated assumption that this route of exposure is negligible.[37]

Finally, consistent with its flawed SOP guidelines, EPA estimates the risks to *average* children using an *average* daily level of exposure over the lifetime of the flea collar product. By looking only at average risks over the lifetime of the collar, EPA likely understates the risks from fresh collars; where insecticide levels in collars do not dissipate evenly over time, the risks from shorter-term exposures—over the first month, for example—will be higher, exceeding EPA safety levels by an even greater amount.

This approach also means that EPA overlooks any acute risks to children from exposure to chlorpyrifos collars. Yet Appendix A highlights the exquisitely acute sensitivity of a young child's developing brain. EPA's approach also appears inconsistent with the recent warnings of Mississippi State University toxicologist Dr. Janice Chambers that parents should keep children away from pets in the first several days that the animals are wearing a new flea collar.[38]

DICHLORVOS (DDVP)

Use: collars

EPA estimated risk to children: 21 times safe levels

Dichlorvos, known as DDVP, is used in dog and cat flea collars. First marketed more than 50 years ago, it is also used in agriculture, in commercial and institutional sites, and around the home, including in resin pest strips.[39] Besides toxicity to the nervous system, dichlorvos causes leukemia in laboratory animals, and there is suggestive evidence that it may be a human carcinogen.[40]

EPA has not taken any regulatory action on dichlorvos and only just released a preliminary risk assessment as this report was going to press. While this assessment provides few details on risks from dichlorvos collars (for example, not specifying whether the estimated exposures are from dog or cat collars), it is detailed enough to show that risks from three-month collars far exceed EPA's level of concern.[41]
For toddlers ages 1 to 2, the EPA calculated that daily dichlorvos intake *from inhalation alone*, over the long-term, was 21 times greater than its safe level.[42] Adult exposures, too, exceeded EPA safe levels by nearly tenfold.

Risks Could Be Higher Still

EPA's risk assessment for dichlorvos is remarkable because it only considers direct inhalation of the pesticide from pet collars. Yet pet collars may also release pesticide

Table 4
Child and Adult Inhalation Exposure to Dichlorvos Collars

Person Exposed	Source of Exposure	Exposures Considered	Percentage of EPA's Safe Level (Acceptable = 100 Percent or less)
Toddler (1–2 yrs)	3-Month Collar	Inhalation only	2,100
Child (3–5 yrs)	3-Month Collar	Inhalation only	2,000
Adult male	3-Month Collar	Inhalation only	900

Source: USEPA Dichlorvos preliminary risk assessment, Table 15, p. 56.

particles onto a pet's fur, especially right after the collar is applied. Thereafter, infants and toddlers cannot help but be exposed to these particles on fur, both through skin and hand-to-mouth contact.[43,44] While the manufacturer of dichlorvos collars has not measured how many of these particles get dislodged, an independent researcher finds that particles from a similar, chlorpyrifos collar remain at "very high" levels on a pet's fur for about a week after the collar is applied."[45] For dichlorvos, EPA chooses to ignore skin and hand-to-mouth exposures from collars, claiming there are no data with which to estimate skin exposures. EPA relies instead on the claim that "inhalation is of most concern" due to dichlorvos' high vapor pressure. EPA's failure to consider skin absorption and hand-to-mouth pathways could seriously underestimate the risks posed to children and families from dichlorvos collars.

EPA's approach to dichlorvos collars also is inconsistent with that for other dichlorvos products, and appears somewhat at odds with its own guidelines. EPA's draft guidelines (SOPs) for estimating pesticide exposure via the dermal and inhalation routes after applying products impregnated with pesticides states, for example: "Inhalation dose is not a concern due to several factors including (1) the pesticide is generally contained within the matrix (i.e. vinyl mattress or shower curtain), (2) pesticides usually have low vapor pressure, and (3) the concentrations used are typically low."

In EPA's assessment of dichlorvos-impregnated resin strips, the Agency finds the risks exceed safe levels by roughly the same margins as for pet collars. For pest strips, however, EPA assumes a person will be exposed for 16 hours per day, while for collars it assumes a person will have only one hour per day of close proximity to the pet, along with eight hours in the pet's general vicinity. This suggests risks from collars would be even higher if EPA assumed exposures consistent with, say, a child who sleeps with their dog. Both dichlorvos pest strips and collars, ironically, remain on the market.[46]

Cancer Risks

EPA also fails to quantify or estimate the cancer risk from dichlorvos pet products. As justification, the Agency cites the sixth internal review it recently conducted of dichlorvos' carcinogenicity, in March 2000.[47] In fact, the written summary of this review shows that the review committee initially decided that there was sufficient

cause for concern that the chemical's cancer risks should be quantified and taken into account for its regulation. After the meeting, however, two-thirds of the committee changed its mind, at the prompting of an unattributed e-mail.[48] The committee's poorly documented about-face was apparently based on an interpretation of the 1999 version of EPA's guidelines for cancer risk assessment that were still being reviewed at the time, and that have not yet been finalized.

NALED

Use: collars

EPA estimated risk to children: Up to 10 time safe levels

Naled, first registered in 1959, is used on pet collars, on food and feed crops, and in greenhouses. In December 1998, Amvac Chemical Corporation (naled's registrant) notified EPA that it voluntarily would let lapse the approvals or registrations for all residential and household uses of naled, *with the sole exception* of flea collars.[49] Besides toxicity to the nervous system, use of naled products may pose an indirect cancer risk. While naled itself does not appear carcinogenic, it degrades into dichlorvos, which causes leukemia in laboratory animals. A person's exposure to a pet treated with naled ultimately could mean exposure to dichlorvos at the cellular level.[50]

EPA completed its naled risk assessment in October 1999.[51] By its calculations, *no uses of naled flea collars are safe for children age eight and under* (See Table 5). Depending on the type of collar used, EPA estimates a toddler's exposure to naled will be up to 1,000 percent—or ten times more than—EPA's "safe" level. Yet the Agency has since taken no steps to remove the products from the market, or to notify the public of these risks.[52]

Risks Could Be Higher Still

As with dichlorvos, EPA ignores the potential for a child to ingest naled residues via hand-to-mouth behavior after petting a collar-wearing pet, or after touching the collar itself. Yet as already noted, the hand-to-mouth route is often a very significant

Table 5
Child and Adult Exposures to Naled Collars (Skin and Inhalation Exposure Only)

Grams of Naled in Collar	1	1.4	2.6	3.8
	Exposures As A Percentage of EPA's Safe Level (Acceptable = 100% or less)			
Toddler (1–2 yr.)	270	476	714	1000
Child (3–5 yr.)	196	349	500	769
Child (6–8 yr.)	135	233	349	500
Child (10–12 yr.)	85	149	229	323
Adult	45	80	120	175

Adapted from Table 11, USEPA Memorandum, Human Health Risk Assessment for Naled, Office of Pesticide Programs, October 13, 1999, p. 83.

source of exposure for children. EPA's failure to incorporate it into the naled review suggests that true risks to children, and especially toddlers, from naled flea collars may exceed EPA's safe levels by an even greater margin than was estimated.

PHOSMET

Use: dusts and dipping solutions

EPA estimated risk to children: 500 times safe level

First registered in 1966, phosmet is used in agriculture, forestry, on livestock, and on dogs. It no longer is found in pet collars but is still sold as a dust or liquid dipping treatment for dogs. Besides its toxicity to the nervous system, results in lab animals exposed to phosmet suggest that it might cause cancer in people as well.[53]

EPA's final February 2000 risk assessment of phosmet looks at direct risks to people who apply phosmet dips and dusts to pets, both as professionals and as homeowners.[54] It also examines indirect risks to adults and children exposed to pets *after* their treatment with phosmet. For toddlers, EPA properly adds together the risks from ingesting phosmet after petting or hugging a dipped or dusted dog and then putting fingers in mouth, to the risks from absorbing the chemical across the skin. (Significantly, EPA did not add such risks together in its risk assessments for other chemicals.) An earlier EPA risk review had also included risks from pet-collar products, but the manufacturer has since withdrawn phosmet dog collars and any uses on cats from the chemical's registration, so they are not considered here.

In its most recent risk assessment, EPA finds that risks to professional applicators—considering only their skin exposure from applying phosmet dips or dusts to no more than eight dogs per day, and ignoring any long-term cancer risks from these exposures—are within EPA's safe levels.[55] Similarly, EPA found that an adult homeowner's risk simply from putting phosmet dip or dust on his own pet was acceptable.[56]

However, the estimated risks to people living with their phosmet-treated dog far exceed EPA's safe levels, especially for toddlers. (See Table 6.) Exposures are excessive not only on the day the dog is treated, but for at least several days afterward.

Looking solely at how much chemical a child would eat by putting fingers in the mouth following contact with a large treated dog, EPA estimates the amount of phosmet ingested would be more than 250 times—or 25,000 percent—the Agency's safe level.

Under a more realistic scenario where at least some toddlers will *both* pet the dog and put hands in their mouths afterwards, thereby both absorbing phosmet across the skin and ingesting it, EPA estimates exposure will be more than 500 times—or 50,000 percent—the Agency's safe level. Adults, who EPA assumes will have no hand-to-mouth behavior, will be exposed to phosmet levels on pets the day of treatment more than 55 times—or 5,500 percent—the Agency's "safe" level.

Even under EPA's most optimistic scenario, in which an average toddler is exposed to phosmet residues from a single small (five-pound) treated dog, the Agency estimates that these exposures solely on the day of treatment through skin

exposure alone will be more than 11 times—or 1,100 percent—the Agency's "safe" level, or level of concern. This calculation disregards any inhalation or hand-to-mouth ingestion of phosmet.

Of course, phosmet pet products are designed to leave residues on the pet that will be effective against fleas and ticks for longer than just one day. Both children and adults therefore likely will be in contact with phosmet for a period of days or weeks, not hours. So it is important that EPA compare exposures over a longer period of time to its safe levels. However, the Agency's definition of safe also changes depending on the time frame (see box Spotlight on page 6). That's because what EPA considers to be a safe short-term level of exposure to pets treated with phosmet products will be derived from studies of the effects from higher amounts of phosmet over a short period of time. Similarly, when EPA determines a safe daily level of exposure to phosmet pet products over the course of a month, it relies upon laboratory studies of animals dosed chronically with relatively less phosmet. These procedures can result in both an acute "safe" level and a chronic "safe" level, and the two may differ.

When EPA did in fact calculate a toddler's average daily exposure to a phosmet-treated dog for the entire month after treatment, the Agency found that the toddler's exposure would be more than 690 times—or 69,000 percent—the safe level for chronic risks to the nervous system.

Of course, if EPA's review were to include inhalation exposure to phosmet as well, it would likely add to the margin by which these estimates exceed the Agency's safe levels.

Risks Could Be Higher Still

For phosmet, as for other organophosphates, EPA admits it has no data to precisely quantify human exposure and risk from pet products, whether those products

Table 6
Toddler and Adult Estimated Exposures to Phosmet-Treated Pets

Age of Person Exposed	Period of Evaluation	Route of Exposure: Petting alone – Small dog	Petting alone – Large dog	Hand-to-mouth alone – Small dog	Hand-to-mouth alone – Large dog	Combined Exposure – Small dog	Combined Exposure – Large dog
Percentage of EPA's Safe Level (Acceptable = 100% or less)							
Toddler (33 lbs.)	Day of Treatment	>1,100	25,000	>8,000	>24,000	>9,200	>50,000
	Ave. over 1 month	>790	>20,000	>16,900	>50,000	>17,700	>69,000
Adult	Day of Treatment	>242	>5,500	NA	NA	242	>5,500
	Ave. over 1 month	1,700	4,000	NA	NA	1,700	4,000

Source: USEPA Memorandum, Revised Occupational and Residential Exposure Aspects of the HED Chapter of the Re-registration Eligibility Document (RED) for Phosmet, Office of Prevention, Pesticides and Toxic Substances, January 27, 2000, Appendix D, Tables 5-7, pp. 151–53.

are applied by veterinarians or by homeowners.[57] Under the Food Quality Protection Act, however, EPA is mandated to estimate such exposures. Again, the Agency does so using a set of assumptions mostly from its flawed 1997 guidelines. For phosmet these include the assumption that professionals dip or dust just eight dogs per day,[58] and that homeowners will dip or dust no more than one dog per day. EPA further assumes that only adults weighing 154 pounds will apply phosmet to pets; an assumption that flies in the face of the only national survey on who applies pet products, completed in 1990, which found that girls under age 15 apply an estimated 855,000 pet shampoos or dips each year, while boys under age 13 apply another 466,000 treatments.[59] Children and other applicators of phosmet products weighing less than 154 pounds would experience a higher weight-adjusted dose.[60]

To its credit, EPA's phosmet review departs from the insupportable assumption in the 1997 Residential SOPs that an average child puts fingers in mouth exactly 1.56 times per hour. It assumes instead that after touching a treated dog, a child will put parts of three fingers in the mouth at a frequency of 20 times per hour.[61] The figure of 20 times per hour, however, is just another *average* value based on studies in very limited numbers of children. And the idea that only parts of three fingers, rather than the whole hand, will go into the mouth would be laughable if it did not have such important consequences. (Three fingers instead of five allows EPA to assume roughly 3/5, or 60 percent of hand-to-mouth exposure levels.) It thus again understates the behavior, and therefore the risks, for children who orally explore their environment much more than average.[62]

EPA's use of each of these assumptions in the phosmet risk assessment tends to understate risks to children, as well as risks to people who own and may treat more than one pet at a time.

Risks from Cancer

EPA assesses the risks from use of phosmet on pets based solely on its toxicity to the nervous system. EPA's failure to consider phosmet's cancer risks is both puzzling and scientifically unjustified.

There is some experimental evidence, though not considered conclusive by EPA, that phosmet may be a human carcinogen. In test-tube studies, though not in studies of whole animals, phosmet causes mutations.[63,64] Phosmet exposure also has been associated with an increased number of liver tumors in male mice, and increased breast tumors in female mice. (No increase in tumors is found in exposed rats.)[65]

Yet EPA's risk assessment assumes that if exposure to phosmet is too low to trigger any sign or symptom of acute poisoning, it also will pose no cancer risk.[66] EPA gives no scientific rationale for this position—a puzzling one, since it presumes that the lack of acute poisoning, which derives from phosmet's action on the nervous system, bears relevance to its possible impact in causing cancers of the liver, breast or other organs.[67] But as a result, EPA's risk assessment fails to quantify any cancer risk from the use of phosmet on pets.

TETRACHLORVINPHOS

Use: dips, powders, sprays, and collars

EPA estimated risk to children: nearly 2 times safe levels

Tetrachlorvinphos is registered for use in a variety of pet products, including pet dips, powders, sprays, and impregnated collars used on both cats and dogs.[68] It is also used on livestock. Like phosmet and dichlorvos, tetrachlorvinphos is toxic to the brain and nervous system, in addition to being a possible human carcinogen. Mice exposed to tetrachlorvinphos suffer significant increases in liver (male and female) and kidney (male only) tumors. EPA released its final risk assessment for tetrachlorvinphos in March 2000. In reviewing risks specifically from tetrachlorvinphos pet products, EPA examined both short-term risks to the nervous system as well as longer-term cancer risks, assuming a lifetime of exposure. It found cause for concern on both counts.

Risks to the Brain and Nervous System

Risks to the adult who applies a tetrachlorvinphos dip or powder to a dog exceed EPA's safe levels, as do risks to toddlers exposed to the family dog after its treatment with powders and sprays—according to EPA's own estimates and definition of safety (See Table 7).

True Risks Could Be Higher Still

Yet EPA's risk estimates for these tetrachlorvinphos products, even when appearing within safe limits, actually may understate true exposures and risks to toddlers and others handling tetrachlorvinphos products and treated pets. Again, this is because EPA bases its risk estimates for tetrachlorvinphos on the flawed assumptions contained in the Agency's draft 1997 guidelines—assumptions that are often unrealistic and not protective for people whose exposure may be the greatest.

In addition, EPA is inconsistent in its consideration of risks from the inhalation of tetrachlorvinphos. It calculates inhalation exposures for adults who actually apply pet *sprays*, but not for those who apply dips, powders or pet collars. For dips specifically, EPA asserts that inhalation will be a minimal route of exposure relative to skin absorption, due to the pesticide's low vapor pressure, and unspecified "conservative assumptions" on which the risk numbers for skin absorption are based.[69] EPA fails to explain, however, why inhalation exposures aren't considered in its assessment of powders or collars.

Similarly, EPA calculates inhalation exposures for adult *applicators* of tetrachlorvinphos sprays, but not for adults or toddlers *exposed to* pets that already have been sprayed. With inhalation of tetrachlorvinphos residues excluded, the only exposures EPA considers for adults exposed to already-treated pets are those through skin absorption. Even then, however, EPA assumes an adult never hugs a dog, and instead pets it with no more than a portion of the hand.

The exclusion of inhalation exposures is important for EPA's risk assessment for toddlers exposed to pets treated with tetrachlorvinphos sprays because even with the exclusion, these exposures are right on the edge of acceptability. To better protect children, risks to toddlers should reflect all possible routes of exposure.

Table 7
Estimated Exposures of Toddlers and Adults to Tetrachlorvinphos-Treated Pets

Person Exposed	Route of Exposure	Exposures Considered	Percentage of EPA's Safe Level (Acceptable = 100 Percent or less)	
Toddler (33 lbs.)			**Medium dog**	
			Average	*Maximum*
	After dip treatment	Skin & hand-to-mouth*	11	18
	After powder	Skin & hand-to-mouth*	125	175
	After spray (aerosol)	Skin & hand-to-mouth*	83	196
	After spray (pump)	Skin & hand-to-mouth*	95	144
	After collar	NA	NA	NA
Adult	After dip treatment	Skin exposure	< 1	< 1
	After powder	Skin exposure	5.2	7-3
	After spray (aerosol)	Skin exposure	3.3	8.0
	After spray (pump)	Skin exposure	3.8	5.9
	After collar	NA	NA	NA
			Small dog	**Large dog**
	Dip Application (1st scenario)[a]	Skin exposure	588	2,500
	Dip Application (2nd scenario)[b]	Skin exposure	204	217
	Powder Application[c]	Skin exposure	476	526
	Collar Application[d]	Skin exposure	NA	33–45

Source: USEPA Memorandum, *Tetrachlorvinphos: Revised Occupational and Residential Exposure and Risk Assessment for the Health Effects Division RED*, OPPTS, October 25, 1999, Tables 8–9, pp. 36–38, for risks to applicators and Tables 11-12, pp. 42–43 for post-application risks.

* As noted in the text, NRDC created these composite numbers by adding EPA's separate estimates for toddler exposures via the dermal and hand-to-mouth routes.

[a] EPA estimates dip exposures using 1997 Residential SOP guidelines; lower exposures were those calculated for dipping a single small dog in one gallon of solution, while higher exposures were those for dipping a single large dog in four gallons of solution.

[b] EPA estimates dip exposure using data from studies submitted by the manufacturer which, in turn, used water rather than tetrachlorvinphos. For that reason, and because only four animals per dose groups were studied, EPA found a lack of quality control in the manufacturer's studies. EPA also used its E-FAST model to characterize hand exposures. Treatment of small dogs involves sponging on solution made from dilution of 2 ounces of 3 percent active ingredient concentrate into one gallon. Large dogs involve dipping into four gallons of solution made from 8 ounces of 3 percent concentrated product. Exposures here reflect an average assumption about transfer of tetrachlorvinphos residues from fur to hand.

[c] Exposures estimated using 1997 Residential SOP guidelines. While EPA notes that label directions give fractions of an ounce of the product to be applied depending on the size of the pet, the Agency's review appears to use two scenarios under which the amount of tetrachlorvinphos applied varies by less than 7 percent.

[d] The range given results from the fact that EPA calculated tetrachlorvinphos exposures for a typical user (33 percent), and for a first time user who might have higher exposures as a result of greater handling of the collar. Both scenarios assumed application by an adult. EPA fails to assess risks to a child applicator.

EPA further assumes that everyone applying tetrachlorvinphos dips and powders will be an adult. EPA fails to consider risks to a child applicator. As noted earlier, the most recent national survey of home pesticide use found that an estimated 855,000 girls under age 15, and 466,000 boys under age 13 applied pet shampoos or dips every year.[70] Children, as compared to an adult, will tend to have

greater surface area relative to weight, and will suffer higher exposures when applying these products.

EPA's assessment of tetrachlorvinphos is also limited because it fails to look at cumulative risks from tetrachlorvinphos across all possible routes of exposure, across time and across multiple pets. For example, EPA acknowledges that children have multiple avenues of exposure to pets, including both skin absorption and hand-to-mouth behavior. However, the Agency calculates these risks separately, failing to combine these risks, even though they co-occur in the same child. For the sake of Table 7, NRDC has added the exposures and found that tetrachlorvinphos applied to a medium-sized dog at the maximal rate allowed by the label will result in cumulative risks to toddlers that exceed safe levels for several products.

Moreover, EPA's estimates of toddler exposures to pets treated with certain tetrachlorvinphos sprays (excluding inhalation exposure) indicate levels of exposure that are right on the edge of acceptability. Thus it is inappropriate for EPA's risk assessment to overlook this exposure. Instead, EPA's assumptions, one by one, ensure that risk estimates will be lowered.

Similarly, adults may not suck on fingers as do children, but they will pet or hug a dog and then eat without washing their hands. Nevertheless, EPA assumes that adults have *no* hand-to-mouth exposure at all to pesticides from pet products. Furthermore EPA completely divorces the risk of exposures to tetrachlorvinphos from applying pet dips, dusts and powders from the risks posed after application. Since the same adult may both apply a pesticide to a pet and then be exposed to the pet afterwards, these risks must be added together.

Finally, EPA treats exposures to tetrachlorvinphos pet products only as short- or intermediate-term events. Unlike the case of phosmet, EPA completely overlooks the impact of recurrent or chronic exposures and risks to the nervous system. Yet some tetrachlorvinphos products, like flea collars, carry tetrachlorvinphos residues for months; the label on the latter states that use is for three to seven months. This is important because longer-term or chronic insecticide exposures carry relevant health effects that may differ from those observed acutely following initial high exposures. Risk assessments therefore should consider exposure to treated pets following the application of tetrachlorvinphos products to be *both* short- and long-term events.

Risks from Cancer

Tetrachlorvinphos is another possible human carcinogen. In contrast to its assessments of phosmet, dichlorvos and malathion, however, EPA actually quantifies concerns about cancer in its risk assessment for the chemical.

EPA finds that use of certain tetrachlorvinphos pet products will elevate the risk of developing cancer above what EPA typically considers to be acceptable, for both the people applying the product to the pet as well as the family members who live with the pet. When the applicator and the family member are one and the same, the already excessive cancer risks will further compound one another. More specifically, EPA found that cancer risks were excessive (up to six or seven times the acceptable level) for persons applying tetrachlorvinphos dips and powders to large pets.

Indeed, these are probably low-ball estimates. EPA assumes, for example, that a dipper would dip *only a single pet, and no more than twice per year*, or that no more than 2 packages of powder (8-10 treatments) would be applied each year.[71] A person dipping or powdering multiple pets, or doing so more frequently, could face cancer risks even higher than EPA estimates.

For people merely exposed to a treated pet, EPA also finds the cancer risks to be excessive. For a person assumed to be exposed to a pet treated with tetrachlorvinphos sprays (pump and aerosol formulations) ten times a year for up to 50 years, EPA estimates the cancer risk will be up to 2.8-fold higher than acceptable levels.[72] Without any explanation, however, EPA fails to estimate cancer risks from tetrachlorvinphos collars, which are designed to emit pesticide residue over three to seven months.

Post-application cancer risks for people exposed to pets treated with dips or powders rather than sprays were within EPA's acceptable limits, but again the Agency's assumptions bring these results into question. EPA assumed the pet in question would receive just two dip treatments and eight powder treatments per year. It also assumed that a person's post-application exposure to the treated pet would only last seven days. Further, EPA's approach was to average the daily pesticide dose received over those seven days and then base the cancer risk assessment on this average dose. While convenient, this approach fails to reflect EPA's own admission that even a week after treatment, a treated pet may still deliver a dose of organophosphate up to 15 percent of the level the day of treatment. Moreover, averaging the dose obscures the fact that people are probably experiencing much higher levels of acute exposure to these carcinogens immediately after the pet treatment. During gestation, as well as at other points early in life, children may be especially susceptible to acutely higher levels of exposure to carcinogens.

Based on marketing data provided by Hartz, one of the nation's leading manufacturers of flea and tick products for pets, however, EPA determined several *likely* scenarios for a typical household's use of tetrachlorvinphos products. For these scenarios, the cancer risks were added both from applying the products, and then from contact with the treated pet afterwards. For example, the total lifetime cancer risk from treating a pet with one dip and two flea collars a year, plus exposure to the pet after these treatments, was around 3.8 times higher than EPA's acceptable levels.[73]

MALATHION

Use: dips and dusts

EPA estimated risks: Not calculated

Malathion is currently registered for use on pets, professional uses on food and non-feed crops, and for mosquito control, as well as for homeowner use on vegetable gardens, home orchards, ornamentals plants and lawns.

In addition to its toxicity to the brain and nervous system, EPA finds "suggestive evidence" that malathion is a carcinogen, although evidence is insufficient to quantify its cancer-causing potential in people.[74] In February 2000, the majority of an internal EPA panel of scientists determined that malathion was a "likely" human

carcinogen; the same panel reversed itself in reaching the above conclusion just two months later.[75] NRDC scientists also consider malathion to be one of the most significant pesticide disruptors of the endocrine system, because it appears toxic to a variety of endocrine glands that make and metabolize hormones.[76] In rodents exposed to malathion, for example, the levels of two critical thyroid hormones (thyroxine and triidothyronine) decrease,[77] while levels of progesterone may also fall. And in cells of the prostate gland, malathion disrupts the metabolism of androgenic male hormones by inhibiting the formation of metabolically active forms of testosterone. [78] In rodents, malathion exposure also causes atrophy of the testicles.[79]

The manufacturer of basic malathion, Cheminova, has informed EPA it does not intend to maintain registration for any indoor use of the chemical, including its uses on pets.[80] Therefore, when EPA issued its preliminary risk assessment for malathion in May 2000, it did not bother to estimate the risks from ongoing use of any malathion-containing pet products—including possible cancer risks.[81] Since EPA did not analyze the risks from these products, NRDC cannot review its risk estimates here. At this time, however, malathion pet products remain on the market and pose a risk to both adults and children.

DIAZINON

Use: collars

EPA estimated risks: Not calculated

Diazinon is currently registered for use in impregnated flea and tick collars, as well as on food crops, livestock and livestock feed crops. Around six million pounds of diazinon are used annually for "residential" uses, including indoor and outdoor uses. Most of this is accounted for by homeowner use on lawns and application by professional operators on lawns and to the home. Like dichlorvos, diazinon is an ingredient in resin pest strips.[82]

EPA issued a preliminary review of the risks from diazinon in May 2000, including risks from many indoor uses.[83] Yet EPA's review barely mentions pet uses and fails to estimate the risks from them. The EPA chemical manager for diazinon calls this an apparent oversight on the part of the Agency.[84] However, it is a critical oversight in light of the subsequent reaction by diazinon's manufacturer, Novartis, to the EPA review.

In formally responding to the EPA assessment, the Swiss multinational Novartis issued a press release asserting that the company would, as a result of "business decisions," no longer continue the registration of its diazinon products for *indoor* uses.[85] Since more than 80 percent of Novartis' sales of diazinon are for home and garden markets, this is significant. However, in contrast to the case of malathion, the diazinon manufacturer appears intent on maintaining registration of diazinon for use in pet products. In addition, the company apparently will continue selling innumerable lawn and garden products containing diazinon.

Though EPA did not look specifically at risks from pet products, its risk estimates for people handling or applying other non-pet diazinon products in residential

settings exceed EPA safe levels under every scenario, such as application to carpets or lawns. The risks to people coming into contact with diazinon after these applications (post-application exposures) also exceed safe levels, according to EPA estimates. The highest estimated diazinon exposures, and therefore the highest risks, are to toddlers through ingestion via hand-to-mouth behavior and through skin absorption after crawling on treated carpets, floors or lawns. Depending on the product and the scenario under which exposures were estimated, the risks calculated for the latter exceeded EPA's safe levels by up to 10,000-fold.[86] Excessive risks from these other products make the lack of any estimate of risks from pet products even more conspicuous by their absence.

SUMMARY OF EPA'S ESTIMATED RISKS

In sum, EPA has found that the risks from applying organophosphate products to pets—even when limited to effects on the brain and nervous system and without considering other possible effects such as cancer—typically exceed EPA's "safe" levels, sometimes by up to 500 times or more. Risks are especially high for toddlers. The highest risks to toddlers for each chemical have been summarized in Table 8.

Yet EPA's estimates of the risks from pet uses of organophosphates are likely to be low-ball estimates, because the Agency overlooked many possibilities for exposure to these chemicals, such as when children get the chemical on their hands and then put their hands in their mouth. Furthermore, EPA's estimates do not reflect the risk contribution of other real world uses of these insecticides—on foods, on gardens and lawns, and in homes. If, or when, EPA puts the risk from exposure to any single OP in a pet product in the context of a toddler's *aggregate* exposure to that chemical through all of its various uses, official estimates of risks can only grow more severe. Similarly, once EPA complies with the Food Quality and Protection Act mandate to consider risks from *all* the organophosphates and carbamates together, it will finally put the risks from any single organophosphate in their proper, real-world context. Despite the legal mandate to do so, EPA has so far failed to take these cumulative exposures into account.

In addition, EPA has assessed cancer risks only for the use of tetrachlorvinphos on pets, finding that these risks also exceed EPA's acceptable levels. Though other organophosphates in pet products are carcinogenic to animals, including dichlorvos, phosmet and malathion, EPA has failed to quantify or consider the cancer risks to people posed by their use on pets (See Table 9).

CURRENT RISK ESTIMATES DO NOT MEET FQPA MANDATE

EPA's estimated risks to the nervous system for six of the seven OPs used on pets would increase if it were to use a full tenfold margin of safety for children, as mandated under the 1996 Food Quality Protection Act when data are not complete for describing a pesticide's toxicity to children, or children's exposure to that

pesticide.[87] By incorporating a strong presumption for this safety margin into the law, Congress tried to remedy a problem highlighted by the National Academy of Sciences in 1993: namely, that EPA's testing requirements for registering pesticides, including those used in pet products, did not for the most part address possible toxicity to the young.[88] Yet among these seven OPs, only for chlorpyrifos has EPA retained the full tenfold safety margin (See Table 10).

For dichlorvos, a possible carcinogen and an even more potent neurotoxin than chlorpyrifos, EPA's preliminary decision has been to remove the tenfold margin of safety (10X), and replace it with a 3X factor instead. EPA makes this decision despite scientific evidence that in some animals, exposure to dichlorvos during pregnancy appears to shrink the size of the brain later in life.[89]

Table 8 Summary of Highest Estimated Risks To A Toddler's Nervous System from Use on Pets

Chemical	Treatment	Exposure Routes	Estimated Risk (Percentage of EPA's Safe Level)	Description
Chlorpyrifos	11 month flea collar	Dermal alone	714	Assuming only skin absorption, (no ingestion or inhalation) EPA's *mean* estimate for an average child's daily exposure to a dog wearing a long-lasting Dursban flea collar is more than 7 times higher than EPA's safe level.
	11 month flea collar	Dermal + Inhalation	12,500	Assuming a child will be exposed both through skin and inhalation, EPA's preliminary estimate for an average child's total daily exposure to a dog wearing a long-lasting Dursban flea collar is more than 125 times higher than EPA's safe level.
Dichlorvos	Collars	Inhalation Only	2,100	EPA estimates a 1–2 year-old toddler's daily dichlorvos exposure to a collar-wearing dog, from inhalation of dichlorvos residues alone, is 21 times greater than EPA's safe level.
Naled	Collars (varying amounts of naled)	Dermal + Inhalation	270 to 1,000*	A toddler's exposure to a pet wearing a long-lasting naled flea collar (from both dermal absorption and inhalation routes combined) would result in estimated average daily exposure 2.7 to 10 times as high as EPA's safe level.
Phosmet	Dips, Dusts Hand-to-Mouth	Dermal +	>50,000	Based on EPA's separate estimates for a toddler's exposure to phosmet from dermal absorption and ingestion respectively the day after a dog has been treated with a phosmet product, we calculate the toddler's combined phosmet exposure on that day is more than 500 times greater than EPA's safe level.
Tetrachlorvinphos	Aerosol spray	Hand-to-mouth alone	196	EPA estimates a toddler's hand-to-mouth exposure alone to a medium-sized dog aerosol sprayed with a tetrachlorvinphos product, would exceed EPA's safe level by nearly two-fold.
Malathion	Dips, dusts		Neglected in assessment	
Diazinon	Collars		Neglected in assessment	

Table 9
EPA Cancer Classification for Select OPs in Pet Products

Chemical	Cancer Classification	Date & Guidelines Used	Animal and Human Evidence	Estimated Cancer Risk from Use on Pets
Tetrachlorvinphos	EPA determined tetrachlorvinphos was a Class C or "possible" human carcinogen.	3/6/95– Final 1986 guidelines[a]	Tetrachlorvinphos-exposed female mice had a statistically significant increase in *liver tumors*. At higher exposure levels, male mice also had increased *liver tumors and kidney tumors*, and increases were statistically significant. Exposed rats developed increases in *thyroid and adrenal gland tumors*, but increases were not significant.	EPA quantified the cancer risk from tetrachlorvinphos pet products, estimating a cancer potency factor (Q1*) of 1.83 x 10^{-3} $(mg/kg/day)^{-1}$.
Dichlorvos	EPA labels dichlorvos a Class C or "possible" human carcinogen; however, the International Agency for Research on Cancer (IARC) labels it a "2B" or probable human carcinogen	3/27/96– Final 1986 Guidelines[b]	Dichlorvos acts directly on cells to cause mutations. It also increases incidence of mononuclear cell leukemia in exposed male rats to statistically significant levels. Increased numbers of tumors have been found in studies where animals ingested dichlorvos orally, but not in studies where animals inhaled the chemical instead.	EPA's still-not-released risk assessment fails to quantify cancer risks from dichlorvos pet products. However, in the Agency's 1996 review, EPA scientists determined a cancer potency value, or Q1*, for dichlorvos of 2.72 x 10^{-1} $(mg/kg/day)^{-1}$
Phosmet	EPA decided there was, "Suggestive evidence for carcinogenicity, but not sufficient to assess human carcinogenic potential" using these Guidelines.	10/27/99– Draft 1999 Guidelines[c]	Phosmet-exposed male mice have had statistically significant increases in the number of liver tumors. Female mice show dose-related increases in numbers of liver tumors and uncommon mammary gland (breast) tumors—however, these fall short of being statistically significant.	Most recently, EPA failed to quantify the cancer risks from phosmet use on pets. In 1994, EPA cancer scientists offered the consensus opinion that data was sufficient to quantify cancer risks from phosmet. In September 1999, however, EPA staff recommended against using the animal data to try and quantify the potential cancer risk to people, citing "low confidence that there is a potential cancer risk to humans."
	EPA determined it was a Group C or "possible" human carcinogen	5/24/94– Final 1986 guidelines[d]		
Malathion	EPA finds "suggestive evidence of carcinogenicity but not sufficient to assess human carcinogenic potential"	4/12/00– Draft 1999 guidelines[e]	Several studies of male and female rats and mice exposed to malathion over long periods of time (allowing tumors a longer time to develop) show increases in liver tumors; among male mice exposed to higher doses, nearly all the animals develop tumors. Other similar studies, however, show no cancer effect.	EPA failed to quantify cancer risks from malathion pet products. In February 2000, EPA scientists recommended that cancer risks from malathion be quantified. A cancer potency or Q_1* of 1.52 x 10-3 $(mg/kg/day)^{-1}$ was determined. 2 months later, essentially the same scientists using different guidelines stated that since malathion was now classified as having "suggestive" evidence for cancer, no quantification of the cancer risk was required.
	EPA scientists determine malathion a "likely human carcinogen"	2/2/00– Draft 1996 guidelines[f]		

Sources:

[a] US EPA Memorandum, Carcinogenicity Peer Review of Gardona (2nd), Memo from Byron Backus and Esther Rinde to George LaRocca, Office of Prevention, Pesticides and Toxic Substances (OPPTS), March 6, 1995.

[b] U.S EPA Memorandum, Report of the OPP Carcinogenicity Peer Review Committee, March 27, 1996 Meeting on Dichlorvos, signed by William Burnham and Stewart *as described in* USEPA Memorandum, Revised Preliminary HED Risk Assessment for Dichlorvos, OPPTS, February 16, 1999, obtained under Freedom of Information Request RIN-3187-99;

[c] USEPA Memorandum, Phosmet-Report of the Cancer Assessment Review Committee, OPPTS, October 27, 1999;

[d] US EPA Memorandum, Carcinogenicity peer Review of Phosmet (2nd), from Marion Copley and Esther Rinde to George LaRocca, OPPTS, May 25, 1994;

[e] US EPA Document, Evaluation of the Carcinogenic Potential of Malathion: Report of the April 12, 2000 Meeting, Cancer Assessment Review Committee, OPPTS, April 28, 2000;

[f] USEPA Memorandum, Malathion - Report of the Cancer Assessment Review Committee, Health Effects Division, OPPTS, February 10, 2000.

Table 10

Highest Estimated Risks To A Toddler's Nervous System from Use on Pets, Adjusted for Use of the Full Margin of Safety for Children

Pet Treatment	Exposure Routes Considered	Estimated Risk (As % of EPA's Safe Exposure Level)	Children's Safety Factor Used in EPA's Most Recent Assessment	Est. Risk As % of EPA Safe Levels, If Full 10X Children's Safety Factor Had Been Used
Chlorpyrifos 11-month flea collar	Dermal alone	710	10X	NA
	Dermal + Inhalation	12,500	10X	NA
Dichlorvos Collars	Inhalation Only	2,200	3X	7,400
Naled Collars Inhalation (varying amounts of naled)	Dermal +	270 to 1,000*	1X	2,700 to 10,000
Phosmet Dips, Dusts	Dermal + Hand-to-Mouth	>50,000	1X	>500,000
Tetrachlorvinphos Aerosol spray	Hand-to-mouth alone	196	1X	1,960

In addition, EPA decided to drop the children's safety factor completely for naled and tetrachlorvinphos, two of the OPs for which the Agency has issued final risk assessments. This is especially remarkable since the manufacturers' toxicity studies for both chemicals fail to measure inhibition of the nervous system enzyme cholinesterase, the exact quality that characterizes them as insecticides in the first place.[90] Despite these data gaps, and even though no test has ever been submitted to EPA on the effect of tetrachlorvinphos or naled exposure on the developing brain and nervous system, the Agency decided to drop the children's 10X safety factor for each of them.

CHAPTER 2

Health Effects of Insecticides Found in Pet Products

Pet products contain a number of different kinds of pesticides. This report focuses on organophosphates because they are of greatest concern, designed as they are to poison the brain and nervous system. The most significant potential health effects to people can be examined in two groupings: acute poisoning—that is immediate or short-term reactions to high doses of OPs; and long-term effects on the brain and nervous system. These long-term effects are of particular concern for fetuses and infants, because of the OPs' possible impact on learning, behavior and other functions of the nervous system later in life. Evidence also now strongly suggests that insecticide exposures may increase the risk of some long-term degenerative diseases of the nervous system, such as Parkinson's. Several organophosphates in pet products also pose a risk for cancer—another chronic disease. And emerging evidence links organophosphate exposures to the development of asthma in some people.

Who is at risk for these health effects? Pets are at risk from these insecticides, as will be described in detail in the next chapter. Certainly people who apply the pesticides to pets have been poisoned in significant numbers. Children more than adults, however, are vulnerable to injury from environmental chemicals, including insecticides. A child's unique diet, physiology, behavior patterns, and still-developing organ systems may all contribute to this susceptibility.[91,92,93] These factors are described in greater detail in Appendix A. Concerns about children extend to both possible short and long-term effects from exposure to OPs in pet products.

ORGANOPHOSPHATE EFFECTS ON THE BRAIN AND NERVOUS SYSTEM

Though OPs were initially developed in the 19th century, their large-scale production only began with their use as nerve warfare agents in World War II. Organophosphate insecticides were then developed, the first of which was marketed in the U.S. around 50 years ago. Individual organophosphate chemicals can vary in potency; today's insecticides, though less potent than warfare agents, still poison thousands of people each year.

OPs, as well as a related class of insecticides called carbamates, block the breakdown of a critical transmitter of nerve impulses, acetylcholine. Acetylcholine's full role in

the adult and developing nervous system is not completely understood, but cells in both the brain and peripheral nerves depend on it.[94] These so-called cholinergic cells are found in the nervous systems of people and animals, as well as in insects.

OPs and carbamates act by blocking acetylcholinesterase, an enzyme that breaks down acetylcholine. Inhibition of acetylcholinesterase results in overexpression of the nerve signals being carried by cholinergic cells. This overexpression accounts for many of the symptoms typical of acute organophosphate poisoning. These can mimic stomach flu or influenza, and commonly include nausea, vomiting, diarrhea, sweating, lightheadedness, and shortness of breath and, in more severe cases seizures, coma and death.

ADULT POISONINGS AND OPS IN PET PRODUCTS

Veterinary clinics, animal-control facilities, pet stores and pet groomers all provide flea-control services. One study surveyed nearly 700 workers who applied flea control products to animals or facilities, and found they had from 64 percent to 258 percent more health complaints involving eyes, skin and unusual tiredness than did non-applicators.[95] Pet workers who specifically used certain organophosphates and carbamates reported statistically significant increases in the occurrence of blurred vision, skin flushing, and asthma (in the case of carbaryl, a carbamate insecticide).[96]

Reports collected by the nation's poison control centers from 1993 to 1996 reveal 26 reported cases of poisoning solely among adults working with pet dips. Nearly half involved the OP, phosmet. Poisoning severity ranged from mild to moderate, the latter meaning that medical treatment was required.[97] In June 1999, the Centers for Disease Control and Prevention (CDC) warned that flea-control shampoos, dips and other products containing insecticides are particularly hazardous to workers who use them regularly (See Appendix B). At the same time, CDC noted these same products also "may pose a risk to consumers."[98]

Finally, a 1997 epidemiological study of 249 Gulf War veterans found that veterans who wore pesticide-containing flea collars during the war were nearly nine times more likely to later report symptoms of impaired thought or cognition than were veterans who did not wear collars.[99] It must be noted, however, these Gulf War veterans probably were exposed to a mixture of chemical substances, and flea collars were only one source.

A CHILD'S GREATER SENSITIVITY TO INSECTICIDES THAT BLOCK ACETYLCHOLINE BREAKDOWN

Confirmed poisonings of adults using pet products also highlight the risks to children. Human and animal data both suggest that children are more sensitive than adults, particularly to insecticides that block the breakdown of acetylcholine.

In situations where multiple people have been poisoned by organophosphates, for example, fatality rates among children often have been higher than those for adults.[100] And in immature animals, a lethal dose of organophosphate insecticides

can be just one percent of the adult lethal dose.[101] Among residential incidents of unintentional exposure to organophosphates, those involving children under age six are more likely to result in symptoms, to require medical treatment and to be considered life-threatening.[102]

Twenty-five years ago, scientists began showing that developing animals exposed to OPs were more susceptible than adults to adverse effects on the nervous system.[103,104] Rodent studies suggest that the very young may have less capacity to detoxify organophosphate insecticides generally, and chlorpyrifos in particular (See Appendix A for details). Newborn animals exposed to chlorpyrifos, for example, may be more than 20 times more sensitive to injury to the nervous system, such as effects on brain chemistry and behavior, than are adult animals. Sensitivity to chlorpyrifos may be even greater for the fetus.[105]

More recent studies suggest the nature of the greater threat posed by organophosphates to the young developing brain and nervous system. Animals exposed even to a single, low-level dose of some organophosphates, during particular times of early brain development, can suffer permanent changes in brain chemistry as well as changes in behavior, such as hyperactivity.[106,107] Chlorpyrifos, which has been the most heavily used insecticide in the nation, decreases the synthesis of DNA in the developing rodent brain, leading to a drop in the number of brain cells.[108,109] Significantly, these effects can be seen at levels of chlorpyrifos exposure too low to cause any overt signs of toxicity.

CHILDREN POISONED ACUTELY BY ORGANOPHOSPHATES

Organophosphates are the pesticides considered most dangerous for acute poisoning.[110] They pose a particular hazard to children under age six exposed in home or school environments—i.e. residential exposure.

According to a recent EPA review of pesticide-poisoning data, Americans logged nearly 63,000 reports to the nation's Poison Control Centers (PCCs) about unintentional residential exposure to OPs over a representative four-year period. Nearly 40 percent, almost 25,000 reports, involved children under age 6. At least 482 of these cases resulted in children being hospitalized.[111] The review found that children exposed unintentionally to OPs were three times more likely to be hospitalized, five times more likely to be admitted to a critical care unit, and four times more likely to die or suffer life threatening illness or permanent disability than were children exposed unintentionally to other pesticides.[112]

Taking into account that poison control centers providing these reports covered only around 80 percent of the U.S. population, the number of residential OP poisoning incidents involving children under 6 probably is closer to 31,000 than 25,000, with an average of about 150 hospitalizations per year.[113] Many poisoning cases that appear in emergency rooms and physicians' offices are never reported to Poison Control, so the true number of OP poisoning incidents likely is higher still.

The seven OPs covered in this report accounted for 72 percent of the unintentional residential OP exposures reported between 1993 and 1996 among children under age

6—about 18,000 incidents (See Table 11). Two OPs accounted for over half of these exposures: chlorpyrifos (36 percent) and diazinon (17 percent). Nearly 2,500 of these incidents resulted in a child being treated at a health care facility, and 156 involved admitting a child to an intensive care unit. Nearly five percent of the residential exposures to phosmet resulted in a child's admission to a hospital critical care unit, a much higher rate than for other OPs. This appears to be because phosmet dog dip is sold to the public as a 12-percent concentrate, with the intention that it be diluted 128-fold before use.[114] A one year-old child could die from a single teaspoonful of this concentrate.[115]

Both parents and physicians often fail to recognize or fully appreciate the symptoms of OP poisoning, or are unable to trace them back to pesticide exposures.[116] Milder or more moderate poisoning symptoms, in particular, are non-specific. But poisoning can lead to complications that are also non-specific; one study found that a third of children poisoned by pesticides may develop pneumonia.[117] Higher OP exposures can result in more severe, longer-lasting symptoms, including seizures, coma and death. But even severe OP poisoning is misdiagnosed frequently, particularly in children.[118]

Most reports of children unintentionally exposed to OPs in residential settings do not stem from routine use of these chemicals, such as pet uses. More often, they occur following direct spills or accidental ingestion of household products.[119] Of course, accidents are only possible because OP products are registered for residential use in the first place. In addition, EPA's latest review of reports to poison control centers reveals that among all organophosphates, an average of 13 percent of the unintentional incidents reported occurred after that pesticide was used as intended in a residential setting. In fact, more serious incidents—those leading to

Table 11
Unintentional Residential Exposures to Select Organophosphates Among Children Under Age Six Reported to PCCs, 1993–1996

Organophosphate Insecticide	Children Under 6	Children Under Six as a Percentage of All Ages Exposed	Approximate Number Seen in Health Care Facilities	Number Admitted for Critical Care
Chlorpyrifos	8,998	51	990	55
Diazinon	4,253	37	770	56
Dichlorvos	2,345	44	232	7
Malathion	1,352	26	247	17
Tetrachlorvinphos	495	62	24	0
Phosmet	470	38	204	21
Naled	89	60	10	0
Subtotal	**18,002**	**—**	**2,477**	**156**
Other OPs	6,887		—	—
Total	**24,889**	**40%**		

Source: USEPA Memorandum, Review of Poison Control Center Data for Residential Exposures to Organophosphate Pesticides, 1993-1996, OPPTS, February 11, 1999.

hospitalization, life-threatening illness, or death—are more likely to involve these "environmental" exposures than are less serious cases. Among the seven OPs focused on in this report, this is especially true for chlorpyrifos.[120]

Thus, reports from the nation's poison control centers clearly indicate that significant risks to children from residential use of OPs generally, even if these reports fail to single out risks from the use of pet products containing organophosphates.

LONGER-TERM EFFECTS OF ACUTE POISONING ON THE BRAIN AND NERVOUS SYSTEM

Typically, pregnant women and children exposed to pets treated with OP products would not face the same level of exposure as a professional pet worker. Indeed, NRDC's research could find no references in the medical literature to children who were poisoned following exposure to pets previously treated with OPs. However, the absence of medical reports or case studies fails to allay concerns about the risks to children posed by OP treated pets.

First, the EPA risk assessments analyzed in this report suggest that a toddler's acute risk from OP-treated pets will often exceed the safe levels set by the Agency. Second, as was previously noted, many OP poisonings are thought to be unrecognized or misdiagnosed, especially among children. Third, as discussed above, a significant percentage of the more serious incidents reported involving residential exposures to OPs involve products specifically intended for use in and around homes—just like pet products. Some experts believe that a critical segment of OP poisonings among children in the past may have occurred after the child absorbed insecticide residues from carpets or bedding that had been contaminated through indoor use of OP products.[121] Children exposed to pets treated with OPs would be expected to have this type of skin exposure as well.

A singular focus on the potential for OP pet products to poison children acutely also will miss the much larger picture: that acute exposures also pose longer-term risks to children. Together, a series of scientific studies performed over the last seven years suggest that even a single or short-term exposure to organophosphates (or other insecticides blocking the breakdown of acetylcholine, like carbamates) during particular times of early brain development, can cause permanent changes in brain chemistry and lasting changes in behavior.[122,123,124,125,126] In young mammals exposed to chlorpyrifos, the most heavily used insecticide in the nation, for example, the developing brain synthesizes less DNA, leading to a drop in the number of brain cells.[127,128] Behavioral changes observed in young animals subsequent to their brief exposure to organophosphates during early brain development include hyperactivity, a condition of obvious import to children.[129]

The links observed in these animal studies between acute insults to the nervous system and long-term effects on behavior and neurological function echo 30-year-old findings in adult workers poisoned by pesticides. Metcalf and Holmes reported on workers exposed to cholinesterase-inhibiting insecticides whose persistent com-

plaints included visual problems, impaired thinking and forgetfulness. Physical exams of the patients supported their reported symptoms, since the examiners found an imbalance in the muscles controlling the patients' eyes, slowed thinking and ability to calculate, and memory deficits.[130]

EPA's comprehensive 1997 review of the scientific literature notes four more epidemiological studies that jointly support the conclusion that persistent neurological or behavioral effects may follow acute OP poisoning among individuals whose acute symptoms have long since passed.[131] An even more recent EPA review of incidents involving unintentional residential exposure to OPs suggests that where these incidents have led to acute symptoms, there also will be health effects lasting a week or more in around one to two percent of cases.[132] The lack of long-term follow-up of incidents reported to the nation's poison control centers make it plausible that the actual percentage of acute cases with long-term symptoms is higher.

Persistent health effects following acute exposures are twice as likely with organophosphates as with other pesticides. Among OPs, chlorpyrifos seems to carry the highest rate of persistent effects, and those effects appear to occur consistently in a certain portion of the population—presumably that which has greater than average susceptibility.[133] The most common chronic complaints following an OP exposure include irritability, problems with memory and concentration, muscle weakness, confusion, depression and blurred vision.[134]

These persistent symptoms can all be plausibly traced to disrupted function of the nervous system, the intended action of both organophosphate and carbamate insecticides. The non-specific nature of the complaints is consistent with a prominent characteristic of the nervous system itself: its role of integrating and then expressing the workings of a variety of other organ systems. Of course, the lack of specificity also means that affected persons or even doctors may fail to link these symptoms to prior pesticides exposure.

LONG-TERM EFFECTS OF CHRONIC EXPOSURE TO OPS

In addition, there now is scientific cause for concern about children exposed to organophosphate insecticides even at levels too low to cause the symptoms of acute poisoning. Concern revolves around possible long-term effects on the developing nervous system, on the development of children's cancer, and possibly asthma. Prior to being put on the market, pet products containing insecticides have not been thoroughly tested for all of these possible long-term effects on developing animals or children.

LONG-TERM EFFECTS ON THE DEVELOPING NERVOUS SYSTEM

At least two recent studies found that immature animals exposed briefly to chlorpyrifos—at one day and two weeks of age, respectively—even at a single low dose, were later found to have had changes in the biochemistry and structure of their developing brains.[135,136] These changes occurred without any signs of systemic

toxicity—that is, no outward signs that the animals were being poisoned, such as acute changes in their behavior or physical appearance. The long-term effect on these animals from their brief exposures to chlorpyrifos included the loss of brain cells in certain parts of the brain, and depressed synthesis of DNA throughout the brain. These effects are at least partially attributable to a mechanism other than inhibition of cholinesterase (the enzyme that breaks down acetylcholine).

In a review of the scientific literature, EPA staff scientists cited several additional animal studies showing that exposure to low levels of chemicals that block the breakdown of acetylcholine may cause behavioral or functional effects, often in the absence of overt or clinical symptoms.[137] In some of these studies, low-dose behavioral effects were observed in conjunction with inhibition of cholinesterase that reached statistically significant levels.[138] Another study, however, observed changes on tests of behavior, as well as changes in brain-wave measurements (EEGs), following repeated exposures to very low doses of these chemicals, even when there were no detectable changes in cholinesterase activity in blood.[139]

Therefore, there is a body of science that helps to explain exactly how, at a molecular level, early-in-life exposure to organophosphate insecticides might change a person's or animal's behavior or nervous-system function, even without any overt signs or symptoms that acetylcholine is not being broken down. While the entire picture is unclear, these studies suggest that acetylcholine may have a variety of functions in the developing nervous system, some quite unrelated to the transmission of nerve signals.[140,141,142] It has been proposed, for example, that when acetylcholine is released from cells in the developing brain, it may directly regulate the proliferation and growth of both nerve and non-nerve cells, as well as their connections with one another.[143] As noted by Dr. Stephen Brimijoin of the Mayo Clinic, "If this suggestion is valid, then we must consider whether anti-cholinesterase pesticides might harm immature organisms by hindering the architectural development of their nervous systems."[144]

LACK OF PRE-MARKET TESTING FOR EFFECTS ON THE DEVELOPING NERVOUS SYSTEM

EPA has a long-validated guideline for testing pesticides for their toxicity to the developing nervous system, but such testing has never been required before putting these chemicals on the market. In fact, this kind of test has been completed for just one OP, chlorpyrifos. And the results of this test were integral to the manufacturer's subsequent decision to pull virtually all indoor uses of the chemical off the market.

Neither are insecticides that are designed to block the breakdown of acetylcholine, such as organophosphates and carbamates, required to be tested for *how well* they accomplish this task. In other words, EPA does not require that they be tested for their ability to inhibit the critical enzyme, cholinesterase. EPA fails to require that the measurement of cholinesterase inhibition for these insecticide classes even while acknowledging that cholinesterase inhibition is often the most sensitive indicator of their toxicity to people.

The bottom line for pet products is that EPA lacks some of the most basic data for assessing possible effects on people exposed to OP products. These data gaps will prove even more profound if, as the science now suggests (see Appendix A), some organophosphates adversely affect the development of the nervous system at doses too low even to inhibit cholinesterase. That being the case, consumers of pet products containing organophosphates cannot presume these products are safe.

CANCER EFFECTS

In addition to their toxicity to the nervous system, EPA has cancer concerns for at least four OPs used on pets: dichlorvos, tetrachlorvinphos, phosmet and malathion. In controlled animal studies of the sort that serve as the basis for the chemical regulatory system in the U.S., exposure to each of these OPs increased the number of tumors seen. A fifth organophosphate, naled, while not considered to be a carcinogen itself, metabolizes into dichlorvos. So a person's exposure to a pet treated with naled ultimately could mean exposure to dichlorvos at the cellular level.[145] Phosmet, malathion, and tetrachlorvinphos are found in pet dusts and dips; tetrachlorvinphos, naled and dichlorvos are registered for use in pet collars.

Among 31 epidemiological studies of the risks to children from their own or their parents' exposure to pesticides, at least two have looked specifically at risks from insecticides in pet products, such as dusts, shampoos, sprays, foggers and collars (See Spotlight). Both studies indicated an increased risk for brain cancer among children exposed to certain of these products at certain points in their development.

Besides pet products, dichlorvos is also the principal active ingredient in insecticide pest strips. In a 1995 epidemiological study, Leiss and Savitz found an association between a family's use of pest strips and both brain tumors and leukemia in children.[150] An earlier study by Davis et al. had also found an association between childhood brain cancer and exposure to No-Pest-Strips.[151]

While the few epidemiological studies to date cannot show definitively that flea and tick collars, shampoos, sprays and no-pest-strips increase a child's risk for brain tumors and other cancers, neither do they disprove this hypothesis. Rather, taken together these studies—the only ones looking at humans exposed to these household products—provide ample grounds for parents' continued concern about possible cancer risks to their children.

ORGANOPHOSPHATES AND ASTHMA

Physicians have long recognized that acute poisoning by OPs can lead to respiratory symptoms, including wheezing. Presumably, this results from direct action of the organophosphate to block the breakdown of acetylcholine in nerves regulating the diameter of the airways, causing bronchospasm. Similarly, Eskenazi et al. have noted the biological plausibility that a child's exposure to an OP insecticide, like Dursban, could contribute to the child's later development of respiratory disease, through disrupted control of the airways typically carried out by nerves using acetylcholine.[152]

SPOTLIGHT

Epidemiological Links Between Children's Cancer and Pesticides

Thirty-one epidemiological studies have looked at the question of whether a parent or child's exposure to pesticides generally, at work or at home, might increase the child's risk for developing certain kinds of cancer later in life. A recent comprehensive review of this body of research found "fairly consistent associations" between pesticide exposure during in childhood, via uses around the home or on pets, and elevated risks for later developing brain cancer and leukemia.[146] However, no study has definitively linked the use of pet pesticide products to cancer.

Two epidemiological studies have looked specifically at a child's risk for brain cancer relative to the child's exposure to pesticides in pet products—although neither study specified individual chemicals. Each study interviewed the families of children already diagnosed with brain cancer to try and determine their pre- and post-natal pesticide exposures. Both studies associated early life exposure to at least some insecticide pet products with an increased risk for developing certain types of childhood cancer.

A 1993 Missouri study matched 45 children who had already been diagnosed with brain cancer to two control groups—85 friends with no cancer and another 108 children who had non-brain cancers.[147] Using parent interviews, the researchers tried to establish whether the children had been exposed to pesticides at all, as well as specific kinds of pesticide used around the home. They also explored home pesticide use at particular points during the child's gestation and early childhood. Among the study's conclusions: children exposed from birth to six months to pets treated with insecticide shampoos and flea collars appeared to have a 4 to 5-fold higher risk of contracting brain cancer, as compared to a control group of their friends (odds ratio = 4.2 & 5.5 for shampoo and flea collars, respectively). The study did *not* find, however, that children exposed to pets sprayed, dipped or dusted with pesticides had a greater risk of developing cancer.

The second study, which looked specifically at brain cancer and pet products, involved 224 children with brain cancer matched to 218 controls. Its most important finding was that prenatal exposure to flea and tick pesticides conferred a 70 percent greater risk (odds ratio = 1.7) for developing childhood brain cancer.[148] The association was especially strong for children diagnosed with brain cancer before age 5; these children actually were 2.5 times more likely than children from the control group to have had prenatal exposure to flea and tick pesticides (odds ratio = 2.5). The study demonstrated, moreover, that the cancer risk increased according to the number of pesticide-treated pets to which the child's mother had been exposed while pregnant. The association seemed particularly strong for flea and tick sprays and foggers (odds ratio = 10.8, or a 9.8 timers greater risk, respectively).

Although both the Missouri and California studies showed an increased risk for brain cancer among some children exposed to some pet products there were significant differences. Only use of pesticide sprays and foggers on pets appeared to increase the risk of cancer in the California Study, while the much smaller Missouri study found no evidence for greater risk from these kinds of products. Moreover, the Missouri study found that cancer risks increased most in children whose exposure to treated pets occurred between birth and 6 months of age, while the California study showed that risks only increased for pre-natal exposures.[149]

The medical literature mentions at least one patient, however, who experienced recurrent asthma seemingly triggered by a single exposure to the OP, dichlorvos.[153] Scientists involved in the case speculated that the dichlorvos might have been directly toxic to the cells lining the airways and that this damage, rather than acetylcholine blockade, was responsible for the persistent asthma. Dr. Sheldon Wagner, who reviews pesticide incidents reported to the EPA-funded National Pesticide Telecommunications Network, recently reported a very similar case to EPA where a single Dursban exposure triggered new and persistent asthma in a child with no history of allergy.[154]

More than 15 years ago, physicians reported two additional cases where low-level exposure to organophosphate insecticides—exposures too low to cause any poisoning symptoms—also caused severe asthma.[155] They speculated that the patients' long-term exposures to fenthion and dichlorvos, respectively, led to development of an allergic sensitivity, and that this accounted for the persistent asthma. It's notable that Dursban exposure also has been associated with an increase in atopy, or allergic sensitization.

These scientists concluded that the public should be made aware that allergic sensitivity to insecticides can develop, and that very low-level exposures can trigger an asthmatic response.[156] Given widespread use of organophosphates on pets, pet owners and veterinarians are a critical audience for this message. Yet before approving pet products for sale, EPA does not require that they or other household products containing OPs be tested for their potential to cause asthma, or even labeled for this potential. Moreover before approving a new pesticide, EPA does not require that it be tested for potential effects on immune system function, including allergic sensitization.

A child's death provided a recent reminder that pet products containing other insecticides may also provoke asthma. In the *Western Journal of Medicine*, the same Dr. Wagner reports the case of an 11-year-old girl with a six-year history of asthma, who died just 2½ hours after giving her dog a bath and shampoo with a product containing .2 percent pyrethrin.[157] The girl had no symptoms upon starting the bath, but within 10 minutes began wheezing, became severely short of breath, and was rushed to the hospital. Dr. Wagner notes that pyrethrin products are being used with increasing frequency in homes, and are readily available to the public. He notes too, that EPA does not require pyrethrin products to be labeled as allergy-causing.

CONCLUSION

Families that use pet products containing organophosphates expose not only their pets, but also their children to the clear risk of short and long-term effects on the nervous system, as well as possible cancer, asthma and other effects. Just because these products are on store shelves does not mean they have been thoroughly tested, or can be presumed safe.

CHAPTER 3

Pet Poisonings and Organophosphates

Pet products containing chemical insecticides can be hazardous to the very population they purport to benefit, family pets. Organophosphates, as well as carbamates, are the insecticides most likely to cause adverse reactions in pets.[158] In 1990, the American Association of Poison Control Centers reported nearly 42,000 calls about animal poisonings of which more than 3,000 involved organophosphate insecticides.[159]

Like a young child, a pet's size and metabolism affects its vulnerability to OPs. Cats are more vulnerable than dogs to poisoning, probably due to their smaller size relative to dogs, their grooming habits (frequent licking of fur), and their lack of enzymes needed to detoxify certain organophosphates.[160]

As with many children, pets cannot report when they're being poisoned. And pet owners may have difficulty discerning the non-specific signs of OP poisoning, especially in cats, i.e. watery eyes, lack of appetite, excessive salivation and urination, nervous signs such as tremors, and behavioral changes such as hyperactivity. The signs are often subtle or atypical as well.[161] The lack of specific poisoning signs, as in people, contributes to misdiagnosis and underreporting of pet poisonings.[162] This, in turn, may contribute to a lack of public awareness about the problem. The fact that poison control centers consider their statistics about pet poisonings to be proprietary, and therefore reserved for paying customers, such as pet product manufacturers, also contributes to a lack of awareness among some veterinarians and the public.[163],[164]

Another problem is that EPA's safety requirements for regulating pet products containing pesticides historically have been ill-defined and inconsistently applied.[165] Specifically, pet safety studies were not required for registering pet products, or were not consistently required, prior to 1987.[166] Until around 1996, EPA did not offer manufacturers any formal guidance on how to perform such a study. After a large number of dogs and cats died or suffered ill effects from one particular product, EPA reexamined the registration requirements for pet products.[167]

Of course, subsequent changes to EPA's registration requirements do not necessarily apply to pet products registered before 1987. In addition animal toxicity studies, when they are performed, are typically carried out on individual pesticide ingredients. They therefore do not account for the impact of combinations of ingredients found in many pet products.

CHLORPYRIFOS AND PET POISONINGS

In 1995, EPA staff analyzed reports to its Incident Data System (IDS) specifically involving domestic pets that had been exposed to chlorpyrifos.[168] The analysis concluded that several hundred pets had fallen ill or died following exposure to chlorpyrifos products, including products specifically intended for use on pets as well as those intended for other uses (See Table 12). Cats were relatively more affected than dogs.

Several conclusions from the analysis are germane to the use of other OP products on pets. Most generally, pesticide incidents involving pets are underreported to EPA. Second, consumers routinely misuse pesticide products intended for residential or "premise" applications, with direct consequences for their pets.[169] Through 1995, at least 29 cats and 33 dogs died and were reported to EPA, not as a result of direct application of chlorpyrifos pet products, but due rather to the misuse on pets of chlorpyrifos products registered for other non-pet uses. The latter included scenarios where pets were left in the house during premise applications of Dursban, as well as poisonings stemming from the direct application of these products to pets. Thus, risks from organophosphate pet products cannot be considered in isolation, but instead reflect the myriad other uses of chemicals in and around the home.

Finally, EPA found that consumers often fail to follow label instructions—again, with sometimes tragic results to pets. Among chlorpyrifos pet incidents reported to EPA, 89 percent involved the direct application of *dog* dips and sprays to cats, even though the only registered use of chlorpyrifos on cats was in the form of flea collars. Thirty percent of cats in these cases died.[170] One particular dog product, Adams Flea and Tick Dip with Dursban (for Dogs), was used on cats and resulted in cat deaths despite the clear label statement, "DO NOT USE ON CATS."[171]

Subsequent to this 1996 analysis (and presumably in response to its findings), EPA revised labels on pet products to give clearer directions and warnings. In 1997, DowElanco, which manufactured Dursban, withdrew its registration for all "premise" flea products containing chlorpyrifos (i.e. those not directly applied to

Table 12
Pets Poisoned by Exposure to Chlorpyrifos (from EPA incident data)

Time Period	Chlorpyrifos Product Involved	Adverse Effect Reported				
		Dead Cats	Dead Dogs	Sick Cats	Sick Dogs	Total Pets Affected
1995 and prior[a]	Products applied to premises, not pets	29	33	129	61	252
	Flea collars	1	2	5	4	12
	Non-collar pet Products	27	12	64	33	136
1996–68[b]	Flea collars	Yes	Yes	Yes	Yes	≥26
	Totals	≥57	≥47	≥198	≥98	≥426

Sources

[a]USEPA Memorandum, Analysis of Chlorpyrifos IDS Data for Domestic Animals, Office of Pesticide Programs, January 24, 1996;

[b]USEPA Memorandum, Update of Incident Data on Chlorpyrifos for Domestic Animals, Office of Pesticide Programs, April 26, 1999.

pets), as well as registration for all flea products other than collars intended for direct application to pets.

These steps likely were insufficient to protect pets, however, since the EPA analysis identified at least 12 pets that had reacted adversely to flea collars with chlorpyrifos. Moreover, EPA scientist Dr. Virginia Dobozy later analyzed pet incidents reported to the Agency from 1996 to 1998, and found that both cats and dogs continued to die from exposure to chlorpyrifos-impregnated flea collars—although Dobozy's impression is that the number of incidents stemming from direct use of pet products was decreasing relative to pet exposures to other "residential" products containing chlorpyrifos.[172] Though her analysis did not quantify the number of pet deaths, Dobozy noted that more deaths from OP exposure still appear to occur in cats than in other pets.

OTHER ORGANOPHOSPHATES AND PET POISONINGS

Since its initial analysis of chlorpyrifos, EPA has looked at reported incidents of pets injured or killed following exposure to at least four other organophosphates—phosmet, diazinon, tetrachlorvinphos and fenthion (See Table 13). Among top generic chemicals for which the National Animal Poison Control Center received calls in 1992, phosmet ranked sixth for dogs and ninth for cats. EPA reviewed several incident reports of one particular phosmet pet dip, ProTICall Derma-Dip, which is no longer registered with the Agency.[173]

EPA also reviewed nearly 200 reports in its Incident Data System involving domestic animals exposed to diazinon, including 134 incidents with dogs and

Table 13
Adverse Effects on Pets from Select Products

Product(s)	Adverse Effect Reported				
	Dead Cat	Dead Dog	Sick Cat	Sick Dog	Total Pets Affected
Phosmet pet dip[a]	3	3	3	5	14
Tetrachlorvinphos products[b]	1	0	1	2	4
Fenthion products[c]	1	1	2	3	7
Pyrethrin-only products (five)[d]	55	10	286	70	421
Combination products including permethrin (two)[d]	39	18	302	37	396
Fipronil products (three) [e]	17	20	138	249	424
Totals	116	52	732	366	1266

Sources

[a]US EPA Memorandum, Phosmet - Review of Incident Reports for ProTICAll Derma-Dip, Office of Pesticides Programs, April 16, 1997;

[b]USEPA Memorandum, Review of Domestic Animal Incident Data for Tetrachlorvinphos, Office of Pesticide Programs, October 28, 1998;

[c]US EPA Memorandum, Fenthion—Review of Pesticide Poisoning Incident Data, Office of Pesticide Programs, January 30, 1996;

[d]Gainer JH, Post D, Feinman SE, Adverse Effects in Human Beings, Dogs, and Cats Associated with the Use of Flea and Tick Products, Pesticides, People and Nature 1(2): 135–142, 1999;

[e]US EPA Memorandum, Fipronil—Review of Incident Reports for Three Products, Office of Pesticides Programs, April 29, 1998.

54 with cats. Based on 1992 data, diazinon ranked fifth among generic chemicals in calls about dogs to the National Animal Poison Control Center, and eighth in calls about cats.[174] The diazinon incidents reported were derived both from use of flea collars, and from "premise" products. Due to the poor quality of the incident reports and of EPA's database, EPA noted that no cause-effect relationship could be drawn between specific diazinon products and types of outcomes; however, the Agency did note that "very few" incident reports had been submitted for products applied directly to pets.

EPA has reviewed incident reports of pets exposed to tetrachlorvinphos only from 1997. However, the vast majority of incidents were reported to EPA by Hartz Mountain Company, which manufacturers many pet products and puts a toll-free number on its product labels.[175] Few details of the individual incidents are available since Hartz claims such data are confidential business information, which in turn restricts EPA from entering the reports into its Incident Data System.

For 1997, Hartz reported 59 incidents, including one death, involving cats exposed to tetrachlorvinphos products; and 11 incidents and no deaths involving dogs. The National Animal Poison Control Center evaluated the incidents and determined only 21 of the 70 incidents had a moderate to high suspicion for being caused by the tetrachlorvinphos product. Later, EPA did collect and add to its Incident Data System four additional reports of pets being injured following exposure to tetrachlorvinphos products.

Fenthion is another organophosphate that has been used for flea control in dogs. This use is regulated by the Food and Drug Administration (FDA), not EPA, because fenthion is systemically absorbed.[176] For this reason, it is not included in this report. In 1996, however, EPA prepared a review of fenthion-related reports to its IDS system up to that date, and found several involving injured or dead pets. (It is unclear whether these incidents occurred after exposure to the pet products registered with FDA or exposure to the non-pet fenthion products registered with EPA.) In addition, the National Animal Poison Control Center reported 101 calls involving cats and dogs and fenthion from 1986 to 1987; EPA's analysis does not list the numbers of injured pets, but it does note that most of the calls likely derived from use of the FDA-regulated pet products.[177]

OTHER INSECTICIDES AND EFFECTS ON PETS

Another analysis recently reviewed pet poisoning incidents reported to EPA by manufacturers of certain pet sprays, shampoos and dips over a three-and-a-half-year period from 1991 to 1994.[178] The incidents involved five products that contained pyrethrins only, and two combination products that counted permethrin, a synthetic pyrethroid, among their active ingredients. The reports were not comprehensive for these products, since the incidents occurred in just 36 states.

As shown in Table 13, the study found hundreds of pet poisonings and even deaths from just these five products. It suggests that pet injuries from flea and tick products are widespread. It also suggests that conventional wisdom about pet

products containing pyrethrins and pyrethroids may need revision. While these products have generally been considered less toxic than products containing OPs or carbamates, they still may pose a significant poisoning risk to pets.

Even pet products containing the newest insecticides, like fipronil, make some pets ill. In 1997 and again in 1998, Dr. Dobozy of EPA analyzed incidents reported to EPA that possibly linked fipronil products to pet illnesses or deaths. Altogether, the incidents from the time of fipronil's registration with EPA until April 1998 involved 37 cat and dog deaths and many more illnesses.[179] However, Dobozy states that after investigation, the majority of deaths were attributed to something other than exposure to fipronil. Moreover, approximately 70 percent of the nearly 400 sick animals had some kind of skin reactions, rather than a systemic illness. Dobozy also notes that in comparison to the widespread use of fipronil, the rate of complications is very low—no more than 2 per thousand treatments.

Skin reaction are not necessarily minor, however. Dobozy found that in several cases involving dogs in particular, fipronil-related reactions more closely resembled chemical burns than a simple case of itching.[180] Dr. Dobozy further notes that while fipronil product labels warn of possible skin irritation, they fail to describe the extent of these potential reactions.

CONCLUSION

While some steps have been taken to reduce the danger, the use of OP pet products still poses significant risks to pets—especially to cats. These risks remain frustratingly difficult to quantify or characterize. Based only on the limited data available, however, it is apparent that hundreds if not thousands of pets have been injured or killed through exposure to OP products.

Unfortunately, adequate systems are not in place to collect comprehensive information on reports of incidents, including information about the specific products used, and to then provide that information freely to the public and to the veterinary community. Some affected pets are simply not reported. Another problem is that when pet illnesses are reported directly to the manufacturer of the product, the manufacturer typically sends a summary report to EPA that can obscure the number of animals affected, as well as the specific products involved.[181] Some manufacturers claim these incident reports as confidential business information, so that EPA may collect them but then cannot inform the public.[182]

Many chemical insecticides other than the organophosphates or carbamates—including the pyrethrins and synthetic pyrethroids—appear to pose risks to pets as well. Again, the lack of comprehensive monitoring for adverse events in pets leaves the extent of this risk uncertain. What is likely is that the problem of pets falling ill or dying after exposure to insecticide products is much larger than has been reported. What is certain, therefore, is that from the standpoint of the pet, the most prudent step is probably to avoid the use of chemical insecticides whenever possible.

CHAPTER 4

Least Toxic Approaches to Flea and Tick Control

Pet owners spend more than a billion dollars yearly on flea control.[183] Seven out of every ten dog owners, according to the American Pet Product Manufacturing Association, buy flea and tick products each year.[184] However, leading veterinarians now recognize a change in focus in the control of fleas on pets from the use of potent insecticides to a preference for less toxic alternatives, and from an emphasis on treatment to prevention.[185] This change in attitude makes the use of organophosphate insecticides in pet products less appropriate than ever before.

WHY THE CHANGE IN ATTITUDE?

This new attitude results from several factors. Until fairly recently, only insecticides that killed adult fleas were available on the market. Flea expert and veterinarian Dr. Michael Dryden called this the "fire engine approach," where potent chemicals are used to douse the "flea fire" only after it had gotten out of hand.[186] The hazards of these insecticides, which include the organophosphates discussed at length in this report, increasingly have been recognized.

Furthermore, aside from their health risks, pet products containing older insecticides have begun to lose their effectiveness, as pests become resistant to them.[187,188] For example, common cat fleas have been found that are resistant to organophosphates, carbamates, synthetic pyrethroids and pyrethrins, as well as other chemicals.[189] Because the government does not formally survey the insect resistance problem, the prevalence and exact nature of this resistance is unknown.[190,191]

Table 14 contains a select list of safer, effective pet products now available, none of which contains an organophosphate, carbamate, pyrethrin or pyrethroid.

USING THE LEAST TOXIC APPROACH

The Natural Resources Defense Council recommends an integrated pest management (IPM) approach to flea control. An IPM approach strives to match the appro-

priate solution to an identifiable problem. IPM depends on understanding the target, in this case the flea (see Spotlight on page 44). IPM also emphasizes methods that pose the lowest hazard to unintended targets, like pets, wildlife and people. For that reason, IPM stresses prevention over treatment, prefers physical and biological controls to insecticides, and when insecticides are necessary, emphasizes the choice of the least toxic alternative.

The initial step in using IPM for flea control is to establish whether in fact there is a real flea problem. A scratching pet may not have fleas. Before doing anything else, pet owners should visually confirm their presence. Fleas are found most often at the base of the pet's tail, around the neck, in the groin, on the back of the legs or on the middle of the back. Adult fleas are dark and wingless, with droppings that look like pepper grains and that turn red on a white background when water is added.

PHYSICAL CONTROLS

Physical controls should be the first measures used to reduce flea populations, since they are cheap, effective and non-toxic. These include bathing and combing the pet, vacuuming, washing a pet's bedding, and restricting the pet's indoor or outdoor access. Even these simple steps can effectively control a mild flea problem.

Table 14
Safer Products Registered for Use on Cats & Dogs*

Newer Spot-On Insecticides	Cat Products	Dog Products
Fipronil	Frontline Spray Treatment	Frontline Spray Treatment
	Frontline Top Spot for Cats	Frontline Top Spot for Dogs
Imidacloprid	Advantage Tm 9 (Imidacloprid) Topical Solution	Advantage TM 10, TM 20, TM 55, or TM 100 Topical Solution
	Advantage 18 (Imidacloprid) Topical Solution	Advantage TM 110
Lufenuron	Program **	Program**
Methoprene	Hartz Rabon Spray with Methoprene Aerosol Formulation	Hartz Rabon Spray with Methoprene Aerosol Formulation
	Zoecon Rf-322 Ovicidal Pump Spray	Zoecon RF-322 Ovicidal Pump Spray
	Zoecon 9207 Collar	Zoecon RF-372 Collar
	Sandoz 9116 Mousse	RF-9414 Shampoo
	Raid Flea Killer Plus	Raid Flea Killer Plus
Fenoxycarb		Raid Flea Killer IV Plus
Pyriproxyfen	Flea Ovisterilant Collar for Cats #1	Flea Ovisterilant Collar For Dogs
	Pyriproxyfen IGR Residual Ovisterilant Shampoo #1	Pyriproxyfen IGR Residual Ovisterilant Shampoo #1
	Pyriproxyfen 10% Spot On for Cats	Permethrin-Pyriproxyfen Residual Shampoo for Dogs
	Adams Flea And Tick Mist With Nylar	Adams Flea and Tick Mist with Nylar
	Mycodex Fastact WP Flea and Tick Spray with Nylar	Mycodex Fastact WP Flea and Tick Spray with Nylar

*Products are those listed in EPA databases as registered as of June 23, 2000.

**Regulated by the Food and Drug Administration, rather than EPA, as are all systemic-acting pet products.

SPOTLIGHT

Understanding the Flea

Effective application of IPM requires an understanding of how fleas develop and live. The most common and annoying flea is the cat flea (Ctenocephalides felix), which infests and bites cats, dogs, wild animals and humans. Fleas bite to feed on the blood of their host. The bite causes pets to itch, and may transmit intestinal parasites sometimes found in the flea's gut. In some pets, flea bites also provoke an uncomfortable allergic skin reaction called flea allergy dermatitis.[192] For all these reasons, it is important to reduce flea infestations on pets.

A safer, less-toxic approach to flea control results from understanding the four stages of the flea's life cycle: egg, larva, pupa and adult. The adult female lays eggs both on and off the pet; eggs laid on the pet can drop and accumulate in carpets, on floors and in household dust. Eggs become larvae. Larvae can be found indoors or outdoors, and after a period of time—generally 1 to 3 weeks—spin cocoons and develop into pupae. Pupae lay dormant until they sense a nearby host and then hatch into adults. Adult fleas immediately seek to bite a host, but can live for months with or without a single blood meal.

Flea infestations can be frustrating to control without an explicit understanding of the various life stages of the flea, particularly since there is wide variation in how long each stage may last. Depending on temperature and humidity, for example, flea larvae can take from a week to more than six months to form pupae, and pupae can lie dormant from a week up to a year. Flea control methods that only kill adult fleas, such as many insecticides, will not take care of immature fleas. The ideal flea control strategy must address all four life stages.

▶ A *soapy bath* is a good first step, since most ordinary soaps will kill fleas. Dropping a few fleas from the flea comb into a soap-and-water mixture will test its efficacy. A pet's *bedding should also be washed* once a week. Fleas tend to accumulate in bedding, so care should be taken not to spread the flea eggs and larvae contained in it.

▶ *Regular combing* of a pet also helps reduce fleas. Flea combs can be bought in pet supply stores. Fleas caught in the comb tines should be drowned in soapy water. Regular combing also helps to monitor the success of an overall flea control program, since the number of fleas on the pet can be easily counted.

▶ *Vacuuming* picks up adult fleas and eggs from carpets, floors and crevices, and from under or on furniture. Vacuuming can be effective against flea pupae since the vibrations stimulate pupae to transform into adult fleas; it is not particularly effective against flea larvae, which hang onto the base of carpet fibers. Immediately after vacuuming, bags should be sealed with tape and thrown out or burned to prevent fleas from escaping and re-infesting the area. Severe infestations may call for professional carpet cleaning with steam.

▶ *Pets should be restricted* from entering indoor areas, like bedrooms or hard-to-clean attics, where a flea infestation would be problematic. Finally, since fleas can attach to

pets in outdoor areas and then re-infest the home, one option is to simply keep a pet inside. Alternatively, outdoor strategies can be aimed at the shaded, protected outdoor areas where pets sleep or rest, and where fleas will accumulate. Also, pet owners should keep grass and shrubbery clipped short in these areas to increase dryness and sunlight, which will help reduce the flea problem. They can also use nematodes—available at garden supply stores—as a non-chemical, biological aid to help control fleas in these areas.

PREVENTION WITH INSECT GROWTH REGULATORS

Flea control can be thought of as a struggle both to eliminate adult fleas and to prevent future flea generations.[193] Insect growth regulators (IGRs) are a relatively safe way to prevent flea problems by stopping the next generation of adult fleas from developing. IGRs are available as sprays, spot-ons, collars and pills. When applied directly to the pet, IGRs have proven so effective as to make IGR "premise" products—those applied to the pet's environment—largely unnecessary. Fleas also have apparently not developed resistance to them, yet.

When used along with physical measures such as bathing and combing, the use of IGRs can be very effective in controlling fleas. However, IGRs do not kill adult fleas. They act instead against the development of eggs and larvae. So, while IGRs can help eliminate adult fleas, they take some time.

For severe flea infestations, or when pets are allergic to their flea bites, IGRs and physical measures may be insufficient. In these situations, pet owners should consider the addition of a lower-risk insecticide, like fipronil or imidacloprid (see below). Many of the lowest-risk products are available only by prescription, through veterinarians. This helps make sure they're used appropriately, and lessens the chance of resistance developing.

Lufenuron (Program®) is one popular IGR, given to dogs or cats periodically, in a tablet, as a liquid food additive, or in an injection. Along with other IGRs, like alsystin, cyromazine, and diflubenzuron, lufenuron works by inhibiting the formation of the hard covering, or exoskeleton, in the developing flea. Because this action is specific to insects, these IGRs are considered very safe for mammals (pets and people). Dr. Dryden of Kansas State University says that lufenuron is 200 times safer for animals than the more traditional insecticides.[194] Lufernuron is available over-the-counter as a tablet for dogs, or as a liquid food additive for cats. For cats, there is a prescription-only injection that is effective for up to six months. Sentinel® is a similar product that contains both lufenuron and an ingredient to prevent heartworm.

Lufenuron products can be very effective, especially for indoor pets and those with lighter flea problems.[195] Studies show that on a pet treated with a single dose of lufenuron, at least 98.2 percent of eggs or larvae produced will fail to develop for a period of 32 to 44 days.[196] Since it does not kill adult fleas, lufenuron cannot provide immediate relief from a flea infestation; but studies show that after 70 days it is 97 percent effective in ridding adult fleas from a pet.[197] Lufenuron is also available

as an injection for cats, and is up to 90 percent effective in preventing maturation of adult fleas on cats for up to six months.[198]

Since fleas have to bite for lufenuron to be effective, it is not the best choice for a pet that has an allergic reaction to flea bites.

Methoprene and pyriproxyfen (Nylar®, Biolar®) are examples of another type of IGR that act by mimicking the juvenile growth factor in fleas, thereby stopping flea eggs and larvae from maturing.[199] Again, because of the specificity of its to insects, these IGRs are considered fairly safe to pets and people. One caveat is that IGRs in general are fairly new products and therefore have not been thoroughly assessed for possible long-term health effects in people.[200]

Methoprene and pyriproxyfen products for direct application to pets include sprays, spot-ons, dips and flea collars. These can be found without prescription at pet stores. Collars with IGR ingredients are probably a better choice than collars with organophosphate and other insecticide ingredients. "Flea collars that are supposed to kill adult fleas have a mixed record of success, and generally kill very slowly," flea expert Dr. Michael Dryden is reported to have said.[201]

Methoprene and pyriproxyfen products are also available for "premise" treatment, to prevent development of immature fleas in or near where pets loaf and sleep. Methoprene sprays are for indoor use only, while pyriproxyfen (trade names, Archer®, Nylar®) is stable in sunlight and can be used both indoors and out. As already noted, however, the use of IGR premise products may be largely unnecessary, given the effectiveness of on-pet treatments, except perhaps in homes with a severe infestation or where the pet spends much time outside.

Pyriproxyfen is combined with the synthetic pyrethroid insecticide, permethrin, in a new once-a-month spot-on product called Bio Spot(, being marketed over the counter for control of both fleas and ticks in dogs only. The pyrethroid component works immediately against adult fleas and ticks, while the IGR component prevents juvenile fleas from maturing. A literature review uncovered no studies of Bio Spot's effectiveness. Moreover, as later noted, other permethrin products have been linked to pet illnesses and deaths, and there are lingering concerns about possible long-term effects of pyrethroids in humans.

Methoprene in capsule form (Hartz Flea Control Capsules®, Zodiac FleaTrol Flea Caps®) is new to the market—too new for there to be much data on its safety or efficacy.[202] Since it must be given once a week, however, it may be a less convenient IGR option than once-a-month lufenuron.

ARE INSECTICIDES NECESSARY?

NRDC does not recommend the routine use of insecticide products for flea control on pets. This especially includes products containing organophosphates, but also carbamates, pyrethrins and synthetic pyrethroids. Physical and biological measures to reduce adult and immature flea populations, combined with appropriate use of insect growth regulators to prevent the maturation of new adult fleas, can address many, if not most, flea problems.

Where pet owners feel they must use an insecticide, NRDC recommends they seek out the least toxic insecticide available (See spotlight below, "Resources for Less Toxic Flea Control.")

NEWER INSECTICIDES: IMIDACLOPRID AND FIPRONIL

Two newer insecticides, popular with pet owners and veterinarians and relatively safer than more "traditional" insecticides for use on pets, are imidacloprid and fipronil. Development of these spot-on formulations has helped to make these newer insecticides not only easier for pet owners to apply, but also more effective when compared to more traditional sprays, shampoos, or dips.[203]

Spot-ons diffuse relatively evenly over the fur coat, taking advantage of the natural oils next to the skin. Insecticide collects in, and is slowly released from, an animal's hair follicles which accounts for residual effects. In the words of one expert, "There's no reason to dip a dog anymore," because these new products control fleas and ticks far superior to what dips ever did.[204]

The use of fipronil or imidacloprid, in addition to physical controls and IGRs, may be most appropriate for an overwhelming flea infestation problem, or when a pet is suffering from an allergic reaction to flea bites.[205] They also continue to work even after a pet swims, or is bathed. Fipronil may be the best choice for a tick problem as well (see spotlight on page 48, "Tick Control").

Imidacloprid (Advantage™) is a prescription-only insecticide from the chloronicotinyl nitroguanadine class.[206] It primarily kills adult fleas, and has no effect on ticks. It can be used on puppies and kittens as young as six weeks of age. Imidacloprid acts by affecting "receptors" through which acetylcholine-carrying nerve cells in insects transmit their signals. Because these so-called nicotinic receptors are found in lower concentration in a mammal's nervous system, imidacloprid is considered to have lower neurotoxicity to people, pets and other mammals.[207] (However, it is highly toxic to bees and house sparrows, among other birds.)

SPOTLIGHT

Resources for Less Toxic Flea Control

The Rachel Carson Council, including their 1994 publication, *The Other Road to Flea Control: Mechanical, Biological and Chemical Methods for Least-Toxic Pet Protection.* For more information, write the Rachel Carson Council, 8940 Jones Mill Road, Chevy Chase, MD. 20815, ph 301-652-1877. http://members.aol.com/rccouncil/ourpage/rcc_page.htm

Northwest Coalition for Alternatives to Pesticides which has excellent fact sheets on alternative flea control methods. http://www.pesticide.org/default.htm

Kansas State University College of Veterinary Medicine's Pet Health News feature, which has online fact sheets about pet problems, including IPM use in controlling fleas and ticks. http://www.mediarelations.ksu.edu/WEB/News/NewsReleases/pethealth.html

SPOTLIGHT

Tick Control

Although this report focuses on flea control, pet owners often are also concerned about ticks. Five different kinds of ticks are commonly found on dogs and cats; different tick species predominate in different parts of the country. Some species transmit diseases to people as well as pets, including Lyme's disease, erlichosis, Rocky Mountain spotted fever, tularemia, etc.

While pet products often are marketed for use against both fleas and ticks, many are not particularly effective against ticks. Many pet collars, excepting Amitraz collars, may not provide pesticide levels on pets high enough to consistently kill ticks. Some highly recommended flea products, such as imidacloprid, simply do not work against ticks.

Experts also have observed signs of insecticide resistance among ticks. They simply are not as easy to treat as previously. Single-agent treatment of ticks in Hawaii, for example, has been described as virtually impossible.[209] As with fleas, however, there is little or no hard data on the problem, since there has been no concerted federal effort to do the research and collect the data.

Absent this research, consumers and veterinarians are often left in the dark about which products to use. Regional variation in resistance further complicates the picture. Consultation with a veterinarian can help determine the best treatment option for your pet, since they are best situated to observe local patterns of tick resistance.

Michael Dryden, a veterinarian and specialist in flea and tick problems at Kansas State University, offers the following general approach to tick problems. Insecticides are listed in Dryden's order of preference.[210] None of the products are completely effective. Efficacy will depend on tick resistance locally, as well as the pet's tick burden.

1. Fipronil spray or spot-on. Generally, fipronil is the top choice for ticks on dogs or cats. Sprays tend to work better than spot-ons for ticks, probably because they allow for a more uniform level of insecticide to be applied to the pet. Effectiveness is highest the first week, but remains around 90 percent for a month. Where there are more ticks, a single application may last only two to three weeks.
2. Amitraz (Preventic) collars. Companies are mostly selling amitraz collars through veterinarians, but they are available over the counter. Amitrazis for dogs only, and does not control fleas. It is from the formamidine class of chemicals, which kill ticks by disrupting their nervous system. It is proven effective against ticks resistant to OPs and other insecticides.[211] Amitraz kills attached ticks in less than 24 hours, making it effective in preventing the transfer of Lyme disease.
3. Selamectin (Revolution) spot-on. This newer, prescription-only insecticide is approved by FDA for treatment of ticks and fleas in dogs and cats. It controls infestations with the American dog tick on dogs, but may not kill all types of ticks.[212] Since it is absorbed into the pet's bloodstream, selamectin is also used to treat certain internal parasites, and to prevent heartworm. It should not be used on puppies or kittens less than six weeks of age.

If one of these agents does not do the trick, they may need to be combined or another insecticide product added, or steps can be taken to get rid of ticks in the environment.[213] Amitraz collars, for example, are approved for combination use with fipronil or many other topical products.

Imidacloprid is applied as a "spot-on" between a dog's shoulder blades, or on the back of a cat's neck. A single application of imidacloprid will control about 95 percent of fleas after one week. New fleas jumping on the pet will be killed within two hours. Imidacloprid does not effect immature flea forms on the pet; as they mature and emerge over a 3–5 week cycle, however, nearly all will be killed. Three monthly uses of imidacloprid will reduce flea counts by 99.5 percent.[208] In contrast to more traditional insecticides, no resistance to imidacloprid has been reported.

Fipronil (Frontline®, Topspot™), first introduced in 1996, is another prescription-only, topical insecticide that mostly kills adult fleas on dogs and cats. Unlike imidacloprid, fipronil cannot be used on puppies or kittens under 10 weeks of age.

Fipronil is from the phenylpyrazole class.[214] Like imidacloprid, it disrupts normal nerve function in insects. However, it does so by blocking the passage of chlorine through cells in the insect's nervous system and this results in paralysis.[215] Fipronil is highly toxic to fish, certain birds, and bees.

Fipronil is applied and acts much the same as imidacloprid, although it comes in a spray as well as a spot-on formulation. Its duration of action is somewhat longer than imidacloprid's—up to three months in dogs and one month in cats. One application of fipronil can provide about 97 percent control of adult fleas after one week, and 96.5 percent control after three monthly uses.[216]

MORE TRADITIONAL INSECTICIDES

Among the more traditional insecticides used for flea control, carbamates, pyrethrins and synthetic pyrethroids are considered to be somewhat less risky alternatives to organophosphates. While these chemical classes may be less acutely toxic than organophosphates, however, they typically have been less extensively tested for long-term effects, especially effects on the developing brain and nervous system. Indeed, experience suggests that the longer insecticides remain on the market, and the more testing that is done, the less safe they often appear.[217]

Each of these insecticide alternatives, therefore, has its risks. As with the organophosphate products discussed at length in this report, over-the-counter availability does not guarantee safety, either to the pet or a child.[218] For that reason, NRDC recommends against the use of pet products containing the following insecticides:

▶ **Carbamates.** NRDC strongly recommends against use of carbamate insecticides on pets. Carbaryl and propoxur are the two major carbamates used for flea control (See Table 15). Together, they account for approximately eight percent of all chemical "active ingredients" used to treat pets and their kennels.[219] Pet products with carbamate ingredients can be recognized if their label lists atropine as an antidote for poisoning.[220] Like organophosphates, carbamates are designed to block the breakdown of the critical transmitter of nerve impulses, acetylcholine.

Therefore, they also are toxic to the brain and nervous system and can result in acute poisonings.

In addition, NRDC scientists consider carbaryl one of the most significant pesticide disrupters of the endocrine system. Carbaryl causes drops in sperm count in exposed animals, interfering with both sperm structure and function—the younger the animal, the more severe the effects on sperm.[221] In exposed workers, carbaryl also has affected the shape and movement of sperm.[222] Among farm families, paternal carbaryl exposure has been found to nearly double the risk of miscarriage.[223]

Fortunately, use of pet products with carbaryl already has decreased. Rising resistance to carbaryl among fleas and ticks in certain regions of the country is thought to be responsible.[224]

▶ **Pyrethrins and pyrethroids.** Pyrethrins and pyrethroids are among the most common insecticides used in pet products. They are the insecticides most commonly found in pet shampoos, sprays and dusts (See Table 16), but also come in spot-on formulations. Pyrethroids include permethrin, allethrin, tetramethrin, resmethrin, fenvalerate and cypermethrin. According to the most recent national survey, pyrethrins and pyrethroids accounted for more than 17 percent and 10 percent, respectively, of all chemical active ingredients applied to pets and their kennels.[225]

Pyrethrins are derived from chrysanthemum flowers and related plants.[226] Pyrethroids are similar chemicals made synthetically. As with the organophosphates and carbamates, pyrethrins and pyrethroids are designed to be toxic to the nervous system, and they can impact wildlife and people, as well as pets, fleas and ticks.[227] They also are toxic to fish and beneficial insects. Since synthetic pyrethroids are more persistent than natural pyrethrins, they have greater potential for being toxic to people.[228] On the other hand, mammals often are able to metabolize pyrethroids quickly in the liver.[229] Concerns also have been raised about the potential of certain pyrethrins/pyrethroids to disrupt the hormone (endocrine) system.[230,231,232]

Table 15
Select Pet Products Containing Carbamate Insecticides*

Carbamate	Cat Products	Dog Products
Carbaryl	Kill-Ko 10% Sevin Dust	Kill-Ko 10% Sevin Dust
	Kill-Ko 5% Sevin Vegetable Dust For Garden Insects	Ortho Sevin 5 Dust, Sevin 10 Dust
	Ortho Sevin 5 Dust, Sevin 10 Dust	Green Light Sevin 5% Dust
	Green Light Sevin 5% Dust	Mycodex Pet Shampoo With Carbaryl
	Mycodex Pet Shampoo With Carbaryl	Flea Collar Rf-75 For Dogs
	Happy Jack Flea-Tick Powder II	Happy Jack Flea-Tick Powder II
	Sevin Brand Carbaryl Insecticide 5% Dust	Holiday Flea And Tick Stop For Dogs And Cats
	Drexel Carbaryl 10d (10% Sevin Dust)	Sevin Brand Carbaryl Insecticide 5% Dust
	Drexel Carbaryl 5d (5% Sevin Dust)	Unicorn Sevin Dog Dip
	Unicorn Flea & Tick Powder For Cats & Dogs	Unicorn Sevin Brand Carbaryl Insecticide 5% Dust
	Unicorn Sevin Brand Carbaryl Insecticide 5% Dust	Ritter's Tick & Flea Powder For Dogs & Cats
Propoxur	Sergeant's Dual Action Flea & Tick Collar	Sergeant's Dual Action Flea & Tick Collar
Propoxur Flea Collar For Cats RF-101		Dog Collar For Flea Control

*Products listed are those registered in EPA databases as of June 23, 2000.

Table 16
Select Products Containing Pyrethrins and Pyrethroid Insecticides Registered For Pet Use*

	Cat Products	Dog Products
Pyrethrins		
Pyrethrins	Ortho Pet Shampoo, Flea & Tick Spray or Flea & Tick Powder Purina Animal Shampoo, Purina Flea 'n Tick Mist Sergeant's Residual Flea and Tick Spray Hartz 2 in 1 Rid Flea Cat and Dog Shampoo Hartz Mountain Luster Bath for Cats with Lanolin Hartz 2 In 1 Flea and Tick Killer for Cats—Fine Mist Spray Hartz Cat Flea and Tick Killer Hartz Fast Acting Roll-On Flea and Tick Killer Hartz 2-1 Luster Bath Mousse for Cats and Dogs Hartz 2 In 1 Flea and Tick Killer For Carpets Raid Flea Killer Plus, New Formula Raid Flying Insect Killer Victory Formula Flea and Tick Pump Spray For Cats Revenge Farm and Home Fly Bomb Insect Fogger Sungro Flea-Zy Pet Shampoo Serene Companion Flea and Tick Spray for Cats Nature's Rain Flea and Tick Shampoo Four Paws Magic Coat Super Plus Zema Pyrethrins Spray for Cats Super K-Gro Pet, Flea and Tick Spray Davis Triple Pyrethrins Flea and Tick Shampoo Black Flag Pet Spray Formula Pow Herbal Flea Powder Rich Health Flea and Tick Killer Nature's Own Brand Herbal Flea and Tick Shampoo	Ortho Pet Shampoo, Flea & Tick Spray or Flea & Tick Powder Purina Dog Shampoo, Purina Flea 'n Tick Mist Sergeant's Skip-Flea Shampoo Hartz 2 in 1 Dog Flea Soap Hartz Mountain Luster Bath for Dogs with Lanolin Hartz 2 in 1 Rid Flea Cat and Dog Shampoo Hartz 2 In 1 Flea and Tick Killer for Dogs-Fine Mist Spray Hartz Dog Flea and Tick Killer Hartz 2 In 1 Rid Flea Shampoo Concentrate for Dogs Hartz Fast Acting Roll-On Flea and Tick Killer Hartz Spot Flea and Tick Remover Hartz 2 in 1 Flea and Tick Killer for Carpets Kamikaze Holiday Flea and Tick Stop for Dogs And Cats Holiday Puppy-Kitten Spray Raid Flea Killer Plus Victory Formula Flea and Tick Pump Spray For Dogs Kimberly Clark Flea and Tick Wipe Sunbugger Carpet Dust Nature's Own Brand Herbal Flea and Tick Shampoo Zema Flea and Tick Dip, Zema Pyrethrins Powder Pow Herbal Flea Powder Durham's Flea, Tick and Lice Dip Black Flag Pet Spray Formula I
Synthetic Pyrethroids		
Allethrin	Mycodex Pet Shampoo with Allethrin Hartz Cat Flea and Tick Killer with Allethrin Hartz Luster Bath for Cats—with Allethrin Hartz 2 in 1 Flea and Tick Killer for Cats/with Allethrin Sulfodene Scratchex Formula 36 Power Dip Pet Guard Flea and Tick Spray for Dogs and Cats Four Paws Magic Coat Plus II Unicorn Flea and Tick Spray IV, Pertran Aerosol Esbiothrin Flea And Tick Mist	Mycodex Pet Shampoo with Allethrin Sergeant's Skip-Flea Shampoo, Pump Soap for Dogs Hartz Dog Flea and Tick Killer with Allethrin Hartz 2 in 1 Rid Flea Dog Shampoo with Allethrin Hartz Luster Bath for Dogs—with Allethrin Hartz 2 in 1 Flea Killer for Dogs/With Allethrin Rid-A-Flea Shampoo Pet Guard Flea and Tick Spray for Dogs and Cats Unicorn Flea and Tick Spray IV, Pertran Aerosol, Ultra Pet Shampoo II Esbiothrin Flea and Tick Mist
Bioallethrin	Ptenocide Pet Spray Unicorn Pet Spray	Ptenocide Pet Spray Fastact 2 Long-Acting Flea and Tick Dip
Deltamethrin		Deltamethrin 4% Collar
Resmethrin	Fly Jinx II Gittem Gottem 0.25% Liquid Insecticide Spray Rid-A-Flea Flea and Tick Killer for Dogs and Cats Misty Aqua-Kill Insecticide Speer Flea Spray for Dogs and Cats Natra Flea Shampoo	Spray Pak Flea and Tick Killer for Cats and Dogs with Deodorant Gittem Gottem 0.25% Liquid Insecticide Spray Rid-A-Flea Flea and Tick Killer for Dogs and Cats Speer Flea Spray for Dogs and Cats Dionne Insecticide with Resmethrin Natra Flea Shampoo
Tetramethrin	Ortho Dog and Cat Flea Spray Raid Flea Killer Plus Hot Shot Flea and Tick Spray for Dogs and Cats Formula 117 Purr-R-Fect Pet Flea Spray For Cats Black Flag Flying Insect Killer Formula A	Ortho Dog and Cat Flea Spray New Formula Raid Flying Insect Killer Raid Flea Killer Plus Hot Shot Flea and Tick Spray for Dogs and Cats Formula 117 Happy Dog Flea and Tick Spray for Dogs
Phenothrin	Flea-B-Gon Flea Killer Formula II Ortho Dog and Cat Flea Spray Sergeant's Dual Action Flea and Tick Collar Hartz Cat Flea and Tick Killer with Allethrin Hartz Luster Bath for Cats—with Allethrin Hartz 2 in 1 Flea & Tick Killer for Cats/with Allethrin Hot Shot Flea and Tick Spray for Dogs and Cats Formula 117 Purr-R-Fect Pet Flea Spray for Cats	Flea-B-Gon Flea Killer Formula II Ortho Dog and Cat Flea Spray Sergeant's Skip-Flea Soap (with D-Phenothrin) Sergeant's Dual Action Flea and Tick Collar Hartz Dog Flea and Tick Killer with Allethrin Hartz Luster Bath for Dogs with Allethrin Hartz 2 In 1 Flea Killer for Dogs with Allethrin Raid Flea Killer Formula II Four Paws Magic Coat Plus II Martin's Pet Guard Super Dip
Pralethrin		Permethrin Plus Pet Spray, Permethrin Plus Pet Spray II

continued on next page

Table 16 (continued)

Select Products Containing Pyrethrins and Pyrethroid Insecticides Registered For Pet Use*

	Cat Products	Dog Products
Synthetic Pyrethroids (continued)		
Permethrin	8-In-1 14 Day Flea and Tick Dip Adams Animal Repellent Concentrate Anchor Permectrin Pet, Yard and Kennel Spray Biogroom Flea and Tick II Residual Permethrin Dip Concentrate Black Flag Pet Spray Formula I CAI Flea and Tick Spray for Dogs And Cats II CCL Flea and Tick Spray for Dog And Cats CCL Quick Breaking Insecticide Foam for Pets II Contact Flea and Tick Killer III CSA Residual F & T Spray for Dogs And Cats Elite Flea and Tick Shampoo VII Elite Permethrin Flea and Tick Dip, Flea and Tick Dip II Elite Residual Flea and Tick Mist, Flea and Tick Mist II with Aloe and Lanolin Evercide Microemulsion Pet Pump Spray 2649 Evercide Permethrin Dust 2786 Evercide Pet Dip Concentrate 2464 Evercide Pressurised Pet and Plant Spray 2561 Evercide Pressurized Pet Spray 2642 Expar Cream Rinse for Dogs And Cats Four Paws Protector Happy Jack Flea and Tick Spray Hi-Yield Garden, Pet and Livestock Dust Martin's Pet Guard Flea and Tick Spray. Mycodex Pet Shampoo with Permethrin Natura Flea and Tick Collar for Dogs and Cats Nolvacide Mist Insecticide Spray II W/Nolvasan Ptenocide Pet Spray Purina Flea 'N Tick Mist R & M Aqueous Residual Flea and Tick Spray #1, #3, #12 R & M Permethrin Flea and Tick Dip #1 R & M Permethrin Flea and Tick Dip #1, #2 R & M Permethrin Powder .25%, Synergized Permethrin Powder #1 Rigo's Best Flea and Tick Spray, Best Flea and Tick Dip Sergeant's Residual Flea and Tick Spray Sergent's Flea and Tick Dip Speer Residual Flea and Tick Dip SPI Residual Flea Tick Spray for Dogs and Cats I, II Sulfodene Scratchex Flea And Tick Killer for Dogs and Cats Sulfodene Scratchex Flea and Tick Spray for Dogs and Cats Sureco Aqueous Residual Flea & Tick Spray #2 Synerkyl Creme Rinse Unicorn Permethrin Dust Unicorn Permethrin Pet Dip II Unicorn Pet Dip Concentrate Unicorn Pet Shampoo III Unicorn Pet Spray, Aqueous Pressurized Spray, 14-Day Flea and Tick Spray, 14-Day Flea & Tick Spray II, 14-Day Flea & Tick Aerosol Spray Victory Formula Flea and Tick Pump Spray for Cats Whitmire Flea and Tick Dip Whitmire Residual Flea and Tick Spray for Dogs and Cats Whitmire Residual Pressurized Flea and Tick Spray Zema Residual Flea and Tick Spray for Dogs and Cats	8-In-1 14 Day Flea and Tick Spray, 8-in-1 14 Day Flea and Tick Dip Adams 14-Day Flea Dip AMI Flea and Tick Spray #1 Anchor Permectrin Pet, Yard and Kennel Spray Biogroom Flea and Tick II Residual Permethrin Dip Concentrate Black Flag Pet Spray Formula I CAI Flea and Tick Spray for Dogs and Cats II Cardinal Rid Flea and Tick Spray CCL Flea and Tick Spray for Dog and Cats CCL Quick Breaking Insecticide Foam for Pets II Contact Flea and Tick Killer III CSA Residual F & T Spray for Dogs and Cats Deputy Dog Flea & Tick Arrest Eagles-7 Flea and Tick Spray Ecto Flea and Tick Insecticide with Igr, Flea and Tick Insecticide Ecto-Soothe Permethrin Shampoo for Dogs Elite Flea and Tick Shampoo VII Elite Permethrin Flea and Tick Dip, Flea and Tick Dip II Elite Residual Flea and Tick Mist, Residual Equine and Pet Spray II Enforcer Flea and Tick Spray for Pets II Evercide Microemulsion Pet Pump Spray 2649 Evercide Pet Dip Concentrate 2464 Evercide Pressurised Pet and Plant Spray 2561,Pet Spray 2642 Expar Cream Rinse for Dogs and Cats Four Paws Protector Happy Jack Flea and Tick Spray Hartz One Spot Repellent for Dogs (and Puppies) Martin's Pet Guard Flea and Tick Spray. Mycodex "14" Pet Spray, Mycodex Pet Shampoo w/Permethrin Natura Flea and Tick Collar for Dogs and Cats Natura L.A. Flea and Tick Spray for Dogs One Drop Anti-Flea and Tick Treatment Permethrin—IGR # 1, #2, #3, #5 Flea and Tick Sprays for Dogs Permethrin Plus Pet Spray, Permethrin Plus Pet Spray II Permethrin-Pyriproxyfen Residual Dog Shampoo #1, #2, #3 PPP Perma-Dip Flea and Tick Dip Solution Preventic L.A. WB IGR Water-Based Long Act. Flea and Tick Spray for Dogs Ptenocide Pet Spray Purina Flea 'n Tick Mist R & M Aqueous Residual Flea and Tick Spray #1, #3, #12 R & M Permethrin Flea and Tick Dip #1, #2 R & M Synergized Permethrin Powder #1 Rigo's Best Flea & Tick Spray, Best Flea and Tick Dip Sergeant's Multipurpose Flea and Tick Killer I Sergeant's Residual Flea and Tick Spray II Sergeant's Skip Flea and Tick Shampoo Plus Conditioner for Dogs Sergent's Flea and Tick Dip, Flea and Tick Spray, Flea and Tick Powder with Pyrethrins Shield Creme Rinse for Dogs Speer Residual Flea and Tick Dip, Flea and Tick Spray III, IV, SPI Residual Flea and Tick Spray for Dogs and Cats II SPI Residual Flea Tick Spray for Dogs and Cats I Sulfodene Scratchex Flea and Tick Killer for Dogs and Cats Sulfodene Scratchex Flea and Tick Spray for Dogs and Cats Sungro Spray and Dip Sureco Aqueous Residual Flea and Tick Spray #2 Sureco Permethrin Powder Sureco Synergized Permethrin Powder #1, #2 Unicorn 14-Day Flea and Tick Spray, #2, #3 Unicorn 14-Day Flea and Tick Aerosol Spray Unicorn 30 Day Flea and Tick Treatment Unicorn Permethrin Pet Dip II, Permethrin WB Spray Unicorn Pet Dip Concentrate, Pet Shampoo III, Pet Spray Victory Formula Flea and Tick Pump Spray for Dogs Whitmire Flea and Tick Dip Whitmire Residual Flea and Tick Spray for Dogs and Cats Whitmire Residual Pressurized Flea and Tick Spray

*«Products listed are those registered in EPA databases for use on cats and dogs as of June 23, 2000

Pyrethrin products are nearly always formulated with a synergist—a compound that enhances their toxicity—such as piperonyl butoxide.[233] Pyrethroids, too, are often combined in pet products with other compounds that may influence their toxicity.

Conventional wisdom is that pyrethrins and pyrethroids are less risky insecticides than the organophosphates or carbamates, which have been in use longer. However, as use of these products on dogs and cats has increased, pet poisonings have as well.[234,235] Permethrin spot-on products for dogs, for example, are purchased directly by consumers, and their misuse on cats (which can not tolerate this formulation) has led to the death or illness of hundreds of pets.[236] Undoubtedly some of the confusion stems from the fact that imidacloprid and fipronil spot-ons are labeled for use on both cats and dogs. In fact, cats can be so sensitive to permethrin spot-ons that they will become ill simply from being in proximity to dogs treated with it.[237]

OTHER FLEA CONTROL PRODUCTS

▶ **D-limonene** (Demize) or Linalool. These citrus oil extracts are botanical insecticides, found in flea shampoos, dips and sprays. They must be used with care; d-limonene, for example, acts similarly to pyrethrins. Some pets, especially certain breeds of cat, are sensitive.[238] Poisoned pets can salivate, stagger or lose body heat, and even die; some hypersensitive pets will develop skin rashes, particularly on their sex organs.[239] Though marketed as "natural" products, these appear to offer no benefit over other pet products.

▶ **Rotenone.** Rotenone is extracted from the derris root plant. It paralyzes the insect's respiratory system, and inhibits energy production. It does not persist in the environment. While considered safe for dogs, rotenone is highly toxic to fish and some other animals, and has been associated with an allergic skin response and stomach upset in pets if swallowed. Reports identify rotenone as being suspected of various health effects including cancer, birth defects, and damage to the liver and kidney.

Table 17
Registered Pet Products Containing Rotenone and Limonene

Other Chemicals		
Rotenone	Dragon Rotenone Pyrethrin Insect Spray	Dragon 1% Rotenone Dust
	Green Light Rotenone	Green Light Rotenone
	Magic Guard With Rotenone/Pyrethrins	Magic Guard With Rotenone/Pyrethrins
	Hi-Yield Rotenone 100 Insecticide Dust	Whitmire Foam-Off Flea Killer Shampoo for Dogs
		Unicorn Rotenone Dip
Limonene	Holiday Flea and Tick Shampoo for Dogs, Cats, Puppies, and Kittens	Holiday Flea and Tick Shampoo for Dogs, Cats, Puppies, and Kittens
	Holiday Flea and Tick Dip for Dogs, Cats, Puppies and Kittens	Holiday Flea and Tick Dip for Dogs, Cats, Puppies and Kittens
	Holiday Pet Spray	Mr. Christal's Kills Fleas

*Products listed are those registered in EPA databases as of June 23, 2000.

▶ **Boric Acid.** Products containing borates, including Borax laundry detergent, sodium polyborate and boric acid, have been much used for treating carpets for fleas because they seem effective, are easily obtained and last up to a year.[240] Some are marketed specifically for flea control while others are not. But veterinarians remain wary of recommending borates for this use. And the largest borax mining company in the country, US Borax, recommends that customers and distributors not sell or use borate products for flea control purposes.[241] Used as a carpet treatment, borates are more expensive than other premise products containing IGRs.

At the right dose, boric acid is known to be acutely toxic. Officials of the National Animal Poison Control Center have confirmed cases where pets exposed to borates suffered subsequent liver and kidney damage, as well as diarrhea, anorexia, depression and vomiting.[242] Meanwhile, rat and dog studies show that chronic, high-level exposure to boric acid can cause reproductive disorders, including sterility and atrophy of the testicles.[243]

▶ **Diatomaceous Earth.** Diatomaceous earth also is marketed as a "natural" method of flea control, on carpets and in households. It consists of the skeletal remains of diatoms, one-celled organisms, the chief component of which is silica. As with borates, there are no long-term studies of the safety of using diatomaceous earth in the home, especially risks to children. But miners of the substance have an increased risk of developing lung cancer and other respiratory disease, while inhalation of other silica compounds is connected with the chronic lung disease, silicosis. One prominent veterinary doctor notes: "I am strongly against use of diatomaceous earth for flea control because silica is a known fibrogenic compound that will induce lung pathology on inhalation."[244]

CONCLUSION

In short, a range of products are available on the market to control fleas and ticks on pets. Many of these products should be avoided by consumers and commercial pet operations, because of the threat they pose to the health of pets and humans both. In addition, some of these products are losing their effectiveness over time, because insects are becoming resistant to them. By contrast, pet owners can easily take non-chemical steps such as washing and grooming their pet often, to help control mild flea problems, and to prevent new infestations. Pet areas should be kept clean and dry as well. If chemical control becomes necessary, pet owners should opt for the least toxic alternative available to them. Several chemical alternatives to control fleas and ticks appear to be safer, at least based on existing research. If pet owners choose to buy chemical products, they should read the ingredients carefully and select the least toxic alternative based on the information in this chapter.

CHAPTER 5

RECOMMENDATIONS

The simple truth is that many of the flea- and tick-control products on the market today expose humans and pets to toxic chemicals at levels far in excess of those believed safe. Indeed, the Environmental Protection Agency estimates that exposure to a pet treated with a single product can exceed acceptable levels by more than 690-fold. When the effects of these pet products are combined in the home, as they often are, with a range of other common toxic products, their impact increases further still. Children are at particular risk from these pesticides, because their behavior tends to increase exposure, their bodies are still developing, and their tolerance to even brief pesticide exposures may be much lower.

Despite the widespread presumption that products this risky are carefully regulated by the government, just the opposite is true. The Environmental Protection Agency has only recently begun to examine the issue in a meaningful way.

The result is that potentially harmful products are routinely available in grocery, hardware, pet-supply stores—even on the Internet. More important, they are routinely, sometimes carelessly, used on pets, and thereby transmit pesticides to humans. While the full scope of these products' effect on children's health has not been thoroughly studied,[245,246] the plain reality is that the organophosphate chemicals that enable many of these products to kill fleas and ticks in the first place are nerve system poisons. Moreover, evidence is rapidly accumulating that these products might cause subtler long-term effects on the nervous system, especially following exposures very early in life.

So what is to be done? NRDC recommends a number of actions by industry, retailers, consumers and government regulators. All of these recommendations are aimed at a single purpose: discouraging the use of certain pesticides on pets. Such action need not leave pets and homes unprotected against fleas and ticks, because safer and effective alternatives are readily available. Specifically, NRDC recommends:

▸ Pet owners should begin using safer products on their pets, avoiding OP-based pet products. Safer products are best combined with such simple physical measures as brushing pets regularly with a flea comb while inspecting for fleas, and mowing frequently in areas where pets spend the most time outdoors.

▸ Pregnant women and families with children should cease using OP-based products immediately.

▸ Retailers should remove OP products from their shelves and seek to educate customers about the merits of safer alternatives.

▶ Children should never apply flea shampoos, dusts, dips, etc. containing OPs to their pets; EPA routinely overlooks and underestimates the particular risks to children when evaluating the safety of these products for home use.

▶ EPA should move immediately to ban the use of pet pesticides containing OPs.

▶ EPA should consider also banning pet products that contain carbamates—a class of insecticides closely related to OPs, and sharing with OPs the same basic biological mechanism of harm. Likewise, homeowners and retailers should avoid the purchase and sale of these carbamate-containing products.

▶ EPA must take steps to better inform veterinarians, pet owners and the general public about safer alternatives for the control of fleas and ticks on pets.

▶ EPA's methods for assessing the risks from other, non-OP pesticides should fully account for risks to children from use on pets, considering children's unique patterns of behavior, metabolism, and periods of vulnerability during growth and development.

For most pet owners, the family dog or cat are beloved members of the family. Unfortunately, products often used to protect pets from fleas and ticks carry serious health hazards—not just for the pets, but for the children who play with them, care for them, and love them. Safer alternatives are available, alternatives that will effectively protect pets from insects without introducing intolerable health hazards into the home. Consumers, manufacturers, veterinarians, retailers and the government all have an important role to play in eliminating these risky pet products and bringing safer alternatives into common use.

APPENDIX A

CHILDREN'S VULNERABILITY TO ORGANOPHOSPHATES

Children are more vulnerable to injury from environmental chemicals in general than adults. A child's pattern of behavior, unique diet, physiology, and still-developing organ systems can make him or her highly susceptible to the toxic effects of pesticides, in particular.[247,248,249] Families that use known nerve poisons on pets are exposing their children to a clear risk, since it is now widely accepted that among a child's developing organs, the brain—as well as the developing immune, reproductive and endocrine systems—are particularly sensitive to chemical injury.[250]

THE DEVELOPING BRAIN'S GREATER SENSITIVITY

As the National Research Council (NRC) acknowledged in its seminal 1993 report, *Pesticides in the Diets of Infants and Children*:

> *The data strongly suggest that exposure to neurotoxic compounds at levels believed to be safe for adults could result in permanent loss of brain function if it occurred during the prenatal or early childhood period of brain development.*[251]

Research on the most-studied compounds toxic to the brain and nervous system—lead, methylmercury and PCBs—supports this NRC conclusion.[252,253,254] Meanwhile, other studies in both humans and animals also buttress the conclusion that a child's developing brain and nervous system may be particularly vulnerable to toxic insult from organophosphates and other pesticides.[255,256,257,258,259]

Immature protective mechanisms in children may contribute to their greater sensitivity to neurotoxins. For example, scientists recognize that fetuses, infants and children, more often than not, are less able to detoxify chemicals than are adults.[260,261,262,263,264] And the blood brain barrier, which protects the adult brain from many toxins circulating in the bloodstream, does not develop fully in children until 18 months to two years of age.[265]

But the brain or nervous system in a fetus or child also is more vulnerable because of the process of development itself. As the nervous system develops in the fetus, 100 billion nerve cells and one trillion glial cells are produced; once produced, cells migrate to permanent locations within the brain and form connections with one

another. Brain cell function is not fixed at birth. As the infant gets older, connections continue to form between neurons—connections that will be critical to the function of the brain and nervous system throughout life. Brain development depends upon this complex and little understood ballet of brain cell proliferation, growth, migration, and the formation and fine-tuning of connections between these cells. Development only occurs in one direction.

For a child to be able to learn, read, and reach his or her full potential as an adult, each neurological event in the ballet must take place at the proper time and in the proper sequence. Timing is especially critical during the earliest developmental stages.[266] Since the ballet can only run in the forward direction, the brain has limited ability to compensate for any early disruptions.[267] Chemical insults leading to an early loss of brain cells or other event can therefore have irreversible effects on brain development at later stages. In oversimplified terms, a child's sensitivity to neurotoxins is due not simply to the fact that their brains will suffer the same injuries as an adult at lower level of exposure. The problem also is that a fetus or child's still-undeveloped nervous system creates windows of opportunity for different kinds of toxic effects—opportunities that no longer exist in an adult's fully-developed brain.

A CHILD'S GREATER SENSITIVITY TO INSECTICIDES THAT BLOCK ACETYLCHOLINE METABOLISM

Both human and experimental data suggest that children are more sensitive to insecticides in particular that block the breakdown of acetylcholine, a critical transmitter of nerve signals. Among incidents of unintentional residential exposures to organophosphates, those involving children under age six are more likely to result in symptoms, to require medical treatment and to be considered life threatening.[268] And in cases involving multiple persons poisoned with OPs, fatality rates among children have often been higher than those for adults.[269]

Research on laboratory animals, too, demonstrated more than 20 years ago that developing animals exposed to OPs are more susceptible than adults to effects on the nervous system.[270],[271] In immature animals, a lethal dose of organophosphate insecticides can be just one percent of the adult lethal dose.[272] Studies of newborn rats dosed with several different OPs suggested that immature animals may have less physiologic capacity to detoxify these nerve poisons.[273,274,275] (Interestingly, females have been found to have less detoxification capacity than male animals, as well.[276]) Moser et al., in their review of a series of studies, found animal evidence suggesting that the relative lack of detoxification enzymes may lead to an immature animal's greater sensitivity to chlorpyrifos, specifically.[277]

Relatively more recent studies have helped to quantify this greater sensitivity among the young (see Table 18). Moser et al. (1998) reported that 17-day old rat pups dosed with chlorpyrifos showed changes in behavior and brain chemistry at doses five-fold lower than those found in adults.[278] Chakraborti et al. (1993) found that week-old rats were more than six times more sensitive to a high dose of chlorpyrifos

Table 18
Relative Sensitivity of Immature and Adult Animals to Select OPs

Study	OP	Age of Dosed Animal	Sensitivity of Immature Nervous System Relative to Adult's
Moser et al. (1998)	Chlorpyrifos	17 days	5 times greater
Chakraborti & Pope (1993)	Chlorpyrifos	7 days	6 times greater
Mendoza & Shields (1977)	Malathion	1 day	9 times greater (relative to 17-day old animals)
Whitney et al. (1995)	Chlorpyrifos	1 day	at least 20 times greater*
Whitney et al. (1995)	Chlorpyrifos	fetus	Higher still?

*Whitney et al. found 1 day-old rats to be more than four times sensitive than week-old rats to doses of chlorpyrifos inducing death, while Chakraborti and Pope found week-old rats to be more 6X more sensitive, and Moser et al. found 17 day-old animals to be 5X more sensitive, than adult rats (5 x 4 = 20 or 6 x 4 = 24).

than were adults, while Whitney et al. (1995) found that one-day old rats were four times more sensitive than even the week-old animals.[279,280] This comparison, though across studies with different experimental conditions, suggest that a day-old rat may be more than 20 times more sensitive to chlorpyrifos than an adult animal. Whitney and his co-authors further conclude that, from the time between dose and response in their study, a fetal animal may be even more sensitive to chlorpyrifos than a newborn animal. This suggests caution "in establishing standards for acceptable levels of chlorpyrifos exposure during pregnancy."[281] Mendoza and Shields' (1977) research affirms that among the OPs used on pets, the greater sensitivity exhibited by neonatal animals is not limited to chlorpyrifos.[282]

Other recent studies suggest the nature of the adverse effects induced by organophosphates in a more sensitive, very young, developing brain. Exposure to even a single, low-level dose of organophosphates, during particular times of early brain development, can cause permanent changes in brain chemistry as well as changes in behavior, such as hyperactivity.[283,284] Chlorpyrifos, the most heavily used insecticide in the nation, decreases the synthesis of DNA in the developing brain for example, leading to drops in the number of brain cells.[285,286]

A CHILD'S GREATER EXPOSURE TO ORGANOPHOSPHATE INSECTICIDES

A child's vulnerability to OPs stems not only from the often greater sensitivity of their developing brain. Children also tend to have greater levels of exposure than adults to OPs and other insecticides that disrupt acetylcholine. This tendency derives from a child's behavior, unique diet and environment, as well as the multitudinous uses of OPs in the places where children spend most of their time. These factors ensure that for a child, OP exposure is a daily routine.

Though EPA has collected little hard data to help quantify a child's daily exposure to the individual OPs used on pets, the scientific literature indicates that overall OP exposure is ubiquitous. Carpets, furniture, house dust—even toys—have all been identified as long-term sinks for pesticides.[287,288,289] Some pesticide residues persist

in carpets for as long as a year.[290] A Jacksonville, Florida study found chlorpyrifos and diazinon in the carpet dust of at least 82 percent of homes sampled; the average home's carpet dust contained at least 12 pesticides, while an average of 7.5 pesticide residues were found in the air of these same homes. Simcox et al. detected chlorpyrifos in 95 percent of the homes of 59 families studied in the Yakima valley.[291] Another study of 362 homes found chlorpyrifos in the carpet dust of 67 percent of them.[292] Eskenazi et al. also cite preliminary results from an Arizona study that detected traces of chlorpyrifos (TCP, chlorpyrifos' chief metabolite) in the urine of all 40 children sampled in a population-based survey. EPA cites preliminary evidence from a survey of 87 Minnesota children that found 92 percent had TCP in their urine.[293]

To a substantial degree, it cannot matter to the child whether the OPs in his urine came directly from foods, from house dust or from the family pet. The important fact is that continued use of these chemicals on pets only adds to a child's background of daily exposure.

A CHILD'S BEHAVIOR

Children's behavioral patterns in environments contaminated with OPs put them at greater risk than an adult. Young children especially spend far more of their time indoors at home.[294,295] Infants and toddlers (18 months to 2 years of age) sit and crawl at ground level, on the floors and carpets where pets dwell and where house dust is found in which pesticide residues are known to concentrate.[296,297] Heavier-than-air pesticides also concentrate at floor level.

Children's habits also mean they may inhale or ingest more of these indoor OP residues. Children explore their pesticide-contaminated environments with fingers, hands and mouth, especially toddlers under age 2. Some toddlers may have up to 62 or more hand-to-mouth contacts per hour.[298] Children will also hug, kiss and sleep with their pets.

From a treated pet or a contaminated household surface children therefore can be exposed to pesticides via inhalation, ingestion or skin absorption. Even if they are aware of these potential hazards, parents cannot be expected to prevent normal childhood behaviors such as breathing or playing with pets.

A CHILD'S DIET

Pound for pound, children drink more water and consume more of certain foods than do adults.[299,300] Higher rates of intake mean that children will receive higher doses of whatever pesticides are present in their food and water. In total, 37 OPs are registered for use on foods (See Table 3). Each OP used in pet products also is used on at least one of nine foods that, according to a recent report by Consumers Union, are most likely to contribute to a child's total exposure to insecticides from the diet.[301] These include apples, pears, peaches, grapes, oranges, green beans, peas, potatoes, and tomatoes.

Water is the most common item in an infant's diet, where it is drunk alone, in formula and in reconstituted juices.[302] On a per-pound basis, infants and children drink more than two-and-a-half times as much water each day as adults.[303]

Organophosphates frequently contaminate streams and wells that may serve as sources of drinking water. From 1992 to 1996, the U.S. Geological Survey (USGS) took 8,200 ground and surface water samples to assess water quality in 20 major watersheds across the country. More than 95 percent of samples collected from streams and rivers had at least one pesticide. Diazinon, chlorpyrifos and carbaryl were the most frequently detected insecticides in streams.[304] Among the more than 320 samples from urban streams, diazinon, carbaryl, chlorpyrifos, and malathion were found in 75, 45, 41, and 20 percent, respectively. These insecticides rank nationally 1st, 8th, 4th, and 13th in frequency of use by homeowners in homes and gardens.[305]

A CHILD'S ENVIRONMENT

The seven OPs used on pets have other uses in or around homes and schools, on playgrounds, lawns and gardens. Ten additional OPs are also registered for these "residential" uses.[306] Since young children may spend virtually their entire day at home, the use of OPs on pets only adds to a household environment routinely contaminated with these nerve poisons. Moreover, some organophosphates like chlorpyrifos are semi-volatile, meaning they may change from solid to gas and then redeposit in areas of the home far from the point of application and onto carpets, countertops, bedding, and even children's toys.[307] Thus, OP residues in a pet product will not necessarily stay on that pet. At the same time, recent EPA studies suggest that active children and pets walking on a carpet will act to re-suspend pesticide particles, thus contaminating the indoor air as well.[308]

EPA has not required that registrants submit data on OP residues in home, daycare or school environments that would help quantify this exposure. Studies in the scientific literature, however, suggest that children's exposures in residential settings are routine. One study of indoor pesticide exposures across three seasons in Jacksonville, Florida led to estimates that detectable levels of malathion, diazinon, and chlorpyrifos would be found in the homes of at least 17, 83 and 88 percent of the population, respectively.[309] Chlorpyrifos also has been found to concentrate in indoor air to levels nearly four times greater at floor level than at a point two feet off the floor.[310]

Other studies have found levels of diazinon, chlorpyrifos and malathion to be much higher in the dust of farmworkers' homes than other homes. Fenske et al. have found that during the pesticide spraying season, children in the homes of agricultural workers have levels of organophosphate metabolites in their urine about four times higher than children in other communities.[311] Toddlers living in some farmworker homes had detectable levels of diazinon and chlorpyrifos on their hands.[312] It was estimated that these hand levels could result in oral ingestion of diazinon residues that would exceed EPA's acute reference dose—the most pesticide that EPA estimates a person can ingest in a day without raising concerns.[313] Organophosphate residues in carpet dust or on floors, or transferred to those surfaces from

treated pets, may serve therefore as a relatively important source of exposure for infants and toddlers through skin and hand-to-mouth contact.[314,315,316]

In another study, the homes of farmworkers directly using chlorpyrifos had average house dust concentrations fully three-fold higher than homes of non-farmworkers in the same community.[317] A recent study further suggests that non-OP pollutants (PAHs—polycyclic aromatic hydrocarbons) found abundantly in house dust may add directly to the ability of certain OPs to block the critical nervous system enzyme, acetylcholinesterase.[318]

OPs need not be registered for indoor use to be found indoors. Household contamination may occur due to air drift from adjacent yards or fields, or from residues tracked home on the shoes or clothes of children or anyone walking or playing in areas where OPs have been applied. Contaminated drinking water also poses a household risk to children through showering, bathing and swimming, where there may be significant skin absorption and inhalation of chemical contaminants.

The aforementioned studies all contribute to a picture suggesting that a child's exposure to OPs in the home environment may be a significant piece of overall pesticide exposure.

APPENDIX B

CDC Report, June 1999

Reproduced below is the full text from the Center for Disease Control and Prevention's Morbidity Mortality Weekly Report (MMWR) for June 4, 1999, raising concerns about symptoms or illnesses in persons applying insecticides to pets. It can also be found on the CDC website at http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4821a3.htm.

June 04, 1999/48(21);443-447

Illnesses Associated with Occupational Use of Flea-Control Products — California, Texas, and Washington, 1989–1997

Dips, shampoos, and other insecticide-containing flea-control products can produce systemic illnesses or localized symptoms in the persons applying them. Although these products may pose a risk to consumers, they are particularly hazardous to pet groomers and handlers who use them regularly. Illnesses associated with flea-control products were reported to the California Department of Pesticide Regulation, the Texas Department of Health, and the Washington State Department of Health, each of which maintains a surveillance system for identifying, investigating, and preventing pesticide-related illnesses and injuries.* This report describes cases of occupational illnesses associated with flea-control products, summarizes surveillance data, and provides recommendations for handling these products safely.

CASE REPORTS

Case 1. In April 1997, a 35-year-old female pet groomer treated a dog for fleas by placing the animal in a tub containing water to which was added a concentrated phosmet solution. During application, the dog shook and sprayed the product on the exposed hands and arms of the groomer; a nearby open soft drink can, from which the groomer reported drinking, may have been contaminated. Within an hour after exposure, she developed skin flushing and irritation, shortness of breath, chest pain, accelerated heart rate and respiration, abdominal cramping, and nausea. She sought care at a hospital emergency department, where she was released without treatment after her clothes were discarded, and she showered with soap and ethanol. Plasma and red blood cell (RBC) cholinesterase levels were 4584 U/L (normal: 2900–7100 U/L) and 32 U/g hemoglobin (normal: 24-40 U/g hemoglobin), respectively;

however, no baseline or subsequent postexposure cholinesterase levels were available for comparison. The case-patient had been a pet groomer for 1 year and did not use personal protective equipment (PPE) (e.g., gloves, gowns, or goggles). She reported that she regularly applied insecticides with her bare hands and that her clothing was often wet with water and flea-control dips or shampoos. Previous exposures had not made her ill. No analysis of the concentration of the phosmet product was performed.

Case 2. A female pet store employee (age unknown) became ill and sought attention at a medical clinic in September 1993 after she inadvertently sprayed her face and eyes with a pyrethrin/piperonyl butoxide solution while spraying a flea-infested cat house. Despite immediately flushing her eyes with water, she developed eye irritation with reddened conjunctiva and a burning sensation. Mild, diffuse wheezing was noted on examination, although its relation to her exposure is unknown; information about preexisting asthma or respiratory infection was unavailable. An allergic reaction and chemical conjunctivitis were diagnosed, and she received epinephrine, oral antihistamines, and oral steroids. At the time of exposure, she had not been wearing goggles or other PPE. She had not received training for safe handling of pesticides.

Case 3. A 21-year-old female veterinary assistant became ill in April 1992 after applying a phosmet-containing dip to a dog. She reported using a chemical-resistant apron, but no other PPE. A pruritic rash developed on her hands and arms approximately 2 hours after exposure. Later that evening, she developed systemic symptoms, including malaise, chest pains, nausea, vomiting, dizziness, diarrhea, stomach cramps, tremors, blurred vision, and excess salivation. Approximately 48 hours after exposure, she sought care at an urgent-care facility. Cholinesterase levels were not reported; she was treated with antihistamines. The case-patient had been a veterinary assistant for 8 months and had treated animals daily using several flea-control products. Whether she previously had used phosmet-containing products is unknown.

SURVEILLANCE DATA

During 1989–1997, 16 cases of pesticide-related illness attributable to occupational use of flea-control products were reported in California (13), Washington (two), and Texas (one). The median age of the case-patients was 26 years (range: 16–73 years). Of the 16, eight (all in women) involved systemic illnesses caused by exposure to phosmet (five cases); pyrethrin/piperonyl butoxide (two cases); or a product containing carbaryl, malathion, and pyrethrin/piperonyl butoxide (one case). The other eight (four in women) involved localized symptoms (i.e., chemical conjunctivitis) caused by flea-control products splashing into the case-patients' eyes. In seven of these cases the products contained pyrethrin/piperonyl butoxide, and in one case a phosmet-containing product was used.

After receiving these data in 1998, U.S. Environmental Protection Agency (EPA) staff searched for similar cases in the Toxic Exposure Surveillance System (TESS). In 1993, TESS, maintained by the American Association of Poison Control Centers, began collection of poisoning reports that included symptom information submitted by approximately 85 percent of the poison control centers in the United States (1996 is the latest year data are available) (1). Poisonings involving intentional suicides, intentional malicious use, non-workplace exposures, and unknown intention were excluded from the search.

Symptomatic occupational exposures involving flea-control dips were identified in 20 women and six men. Responsible active ingredients were phosmet (12 cases); pyrethrin/piperonyl butoxide (five cases); rotenone/pyrethrin (five cases); rotenone, malathion, chlorpyrifos, and unknown (one case each). Eight workers developed moderate health effects that required some form of treatment, and 18 developed minor health effects (minimally bothersome symptoms that resolved rapidly). Among the workers with moderate symptoms, the responsible ingredients were phosmet (five cases), rotenone/pyrethrin (two cases), and pyrethrin/piperonyl butoxide (one case).

Reported by: L Mehler, MD, Dept of Pesticide Regulation, California Environmental Protection Agency. J Shannon, Ph.D., Environmental and Occupational Epidemiology Program, Texas Dept of Health. L Baum, Office of Toxic Substances, Washington Dept of Health. Office of Pesticide Programs, US Environmental Protection Agency. Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

EDITORIAL NOTE

Pyrethrins are plant-derived insecticides and are common ingredients in flea-control dips and shampoos (2). Although pyrethrins have low toxicity in humans (EPA classified as acute toxicity category III compounds**), exposures have caused dermatitis and upper respiratory tract irritation (3). Allergic contact dermatitis and asthma, sometimes resulting in death, also have been reported (1,3). Piperonyl butoxide, an EPA acute toxicity category IV compound, frequently is added to pyrethrins to slow chemical metabolism. No published reports of eye injury involving pyrethrins or piperonyl butoxide were identified.

Phosmet is an organophosphate insecticide and an EPA acute toxicity category II compound. The primary target in humans is the nervous system. Organophosphate exposure is associated with many of the symptoms reported by the first and third case-patients. In animals, phosmet is mildly irritating to the eyes but not irritating to the skin (4); no published reports of skin or eye irritation in humans after exposure have been identified.

The findings in this report are subject to at least three limitations. First, although 76 percent of the cases described were in women, evidence suggests that this distribution may reflect workforce demographics (more women than men are employed as pet groomers and handlers [5,6]) rather than greater sensitivity to these

toxins. Second, these surveillance data may not represent all workers with these illnesses. Third, this report describes only workplace-related illnesses following product exposure. Consumers using these products may experience similar illnesses; however, they were not included in this report.

Despite reports of the toxicity of flea-control products (7-9), including a high prevalence of symptoms among pet groomers and handlers (5,9), illnesses continue to occur among workers using these products. A survey of establishments using flea-control products found that groomers and handlers often were not provided with adequate safety training and PPE (9). When using pesticide products, label directions should be followed precisely. For phosmet-containing flea-control products, the label cautions users to wear safety glasses, long-sleeved shirts, long pants, elbow-length waterproof gloves, waterproof aprons, and unlined waterproof boots. For eye safety, CDC's National Institute for Occupational Safety and Health recommends goggles designed to provide splash protection.

Although the EPA does not require PPE for toxicity category III and IV compounds, the findings in this report suggest that PPE may be needed during pyrethrin/piperonyl butoxide use. Workers should be trained in the safe handling of flea-control products and in personal hygiene practices (e.g., washing before eating and prohibition of eating, drinking, food storage, and smoking where flea-control products are used), and should be instructed about insecticide dangers and taught to recognize the symptoms of overexposure. In California, agricultural workers who apply organophosphates on 7 days in any 30-day period are required to have plasma and RBC cholinesterase tests before commencing exposure and periodically thereafter (8). Similar testing of workers handling organophosphate-containing flea-control products may be prudent; substitution of safer, less toxic pesticides also should be considered.

This report provides an example of how state-based pesticide poisoning surveillance systems and TESS complement one another; however, both systems are affected by lack of adequate clinical recognition of pesticide-related illness and injury. A new EPA publication may assist health-care professionals to gain expertise in recognizing and managing these conditions (10). Free copies are available from EPA; telephone (800) 490-9198.

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