Assessment of Human Exposure and Human Health Effects after Indoor Application of Methyl Parathion in Lorain County, Ohio, 1995–1996

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In January 1995 the U.S. Environmental Protection Agency declared methyl parathion–contaminated homes in Lorain County, Ohio, as a Superfund cleanup site. During the 2-year cleanup, the Centers for Disease Control and Prevention in collaboration with county and city health officials conducted a study of exposure and health effects among residents. We administered 254 household and 747 individual questionnaires; urine analysis for *p*-nitrophenol (PNP, a metabolite of methyl parathion) was available for 626 participants. We also reviewed medical records of 49 people who were hospitalized or died after their homes were sprayed. People living in homes sprayed <180 days previously were most likely to have the highest PNP levels (22.9% > 100 ppb PNP), but even people living in homes sprayed more than a year previously appeared to be highly exposed (8.5% > 100 ppb PNP). The National Health and Nutrition Examination Survey reference range is 0–63 ppb. Median detectable PNP levels among children younger than 3 years of age were 93.9 ppb compared with 41.6 ppb among people older than 3 years. Younger children appeared to be at greatest risk of exposure. In none of the medical records that we reviewed did a health care provider consider pesticide poisoning as a potential etiology. *Key words:* methyl parathion, organophosphate pesticide, *p*-nitrophenol. *Environ Health Perspect* 110(suppl 6):1047–1051 (2002).

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In late fall of 1994, Lorain County, Ohio, became the site of the first investigation of several large-scale incidences in which the pesticide methyl parathion was illegally applied to private residences. Public health authorities became aware of the contamination when a homeowner contacted the Ohio Department of Agriculture regarding a chemical odor that had persisted for weeks following commercial pesticide application in his home. Surface wipe sampling in the home identified methyl parathion. County authorities visited the applicator's home and confiscated containers of methyl parathion that had been purchased at an undisclosed location in another state. The applicator reported that he had been applying this product in hundreds of Lorain County residences over the course of the previous 5–7 years. The applicator did not maintain a record of client addresses, dates, volume, or strength of pesticide applied, or protocol for application. The extent of potential human exposure to this pesticide led the Ohio Department of Health to formally request technical assistance from the Centers for Disease Control and Prevention (CDC) (*1*). In January 1995 the U.S. Environmental Protection Agency (U.S. EPA) declared methyl parathion–contaminated homes in Lorain County, Ohio, a Superfund cleanup site. During the 2-year evaluation and remediation of these homes, the CDC, in collaboration with county and city health officials, conducted a study of organophosphate (OP) pesticide exposure and adverse health effects

among residents in Lorain County, Ohio, who lived in homes where methyl parathion had been inappropriately sprayed.

Methyl parathion is an OP pesticide intended for outdoor use only and is classified in U.S. EPA Toxicity Category I (i.e., most toxic). OP pesticide poisoning occurs when humans are exposed, either intentionally or unintentionally, to this class of pesticide. Although the literature reports several case examples of misuse of OP pesticides (*2*), the route of exposure is usually oral rather than dermal or inhalation (*3*). Very little is known about unintentional nonoccupational exposure, especially in children. The circumstances of methyl parathion misuse in Lorain County may be considered a unique natural experiment. This incident provided the public health community with an opportunity to further define human exposure as well as acute and short-term health effects associated with exposure. We conducted a study in Lorain County, Ohio, during the Superfund cleanup period to chronicle adverse health events that occurred during the postspray period.

Methods

A few potentially contaminated homes were identified by reviewing limited notes found in the applicator's home, but the majority of the potentially contaminated sites were identified through self-report from residents to local health officials. The residents were asked to recall approximate month(s) that the applicator had applied the pesticide and to also

report what locations in the home had been sprayed. Although the primary reason for application in most homes was roach infestation, a common application pattern was not apparent. Many residents responded that they were told by landlords or neighbors that their home had been sprayed prior to their occupancy. The U.S. EPA assigned a level of remediation to the more than 500 single- and multiple-dwelling structures (*4*) and prioritized the timing of evacuation or cleaning based on the results of environmental and biological sampling. All homes that the U.S. EPA designated as requiring priority level I or II cleaning were visited and invited to participate in our study prior to evacuation or cleaning. Once informed consent was obtained, we set up an appointment time for a motor home that had been rented for the study to park outside the home and serve as the site for questionnaire administration and urine sample collection.

We administered household questionnaires to adults who self-identified as head of the household. We collected demographic information for everyone who lived in the home, the approximate amount of time that each person had spent in the home since the spray date, the spray pattern used in the home, and the use of any product that the applicator may have left behind. In addition, we used the household questionnaire to find out if anyone living in the home or any pets kept in the home had been hospitalized or died since the last spray date. Because of the potential size of the study population and because we were conducting interviews during a time when homes were being evacuated, we did not try to collect individual questionnaire information from every resident. We did obtain individual questionnaire information for each person who completed a household questionnaire. The remaining individual questionnaires were completed by residents who were available and

willing to participate at the initial interview date. During the individual interview we asked about 23 physical symptoms, including those consistent with pesticide exposure (e.g., severe or frequent headaches, dizziness, sensitivity to light, nausea, vomiting) as well as several signs or symptoms not traditionally associated with OP exposure (e.g., fever, dental discomfort) experienced during the two weeks following methyl parathion application. We asked if these symptoms were still present and whether they improved when the participant was out of the home. All available participants were asked to provide a convenience urine sample.

Upon completion of the survey, we used the household questionnaire to identify 63 homes where the respondent answered yes to the question "Since the last spraying, has anyone in your home been hospitalized?" (*n* = 59), or to the question "Since the last spraying, has anyone in your home died?" (*n* = 9). Five homes reported both hospitalization and death. We visited each eligible home and invited residents to participate in an extended open-ended interview session and to provide consent for us to obtain medical records. After excluding hospitalizations for childbirth, chronic disease rehospitalizations, and geriatric deaths associated with preexisting chronic disease, we reviewed the medical records of 49 people. We recorded the presenting signs and symptoms, physical examination findings, vital signs, laboratory results, and discharge diagnosis for each patient visit to a health care provider. We developed a set of criteria for excluding or including a person as a potential case of organophosphate poisoning. We excluded anyone whose medical records suggested a primary diagnosis of trauma or who had a well-documented infectious disease diagnosis. For example, we excluded cases with any notation of a positive blood, sputum, or urine culture, or abnormal otoscopic exam. We also excluded cases with diagnosed abnormal imaging of the chest (e.g., pneumonia), abdomen, or head (e.g., tumor), or a gynecologic or psychiatric diagnosis. Among the remaining medical records, we considered those describing gastrointestinal complaints (e.g., abdominal pain or cramping, vomiting, or diarrhea), central nervous system (e.g., dizziness, headache) symptoms, respiratory symptoms (e.g., wheezing, shortness of breath), or evidence of increased secretions (e.g., excessive lacrimation or sweating) as being most likely to be potential cases of OP poisoning.

Of 289 homes eligible for participation, 254 (87.9%) provided complete household questionnaire information. We collected individual questionnaire information from 747 of the 1,095 people living in the participating homes, and collected at least one urine sample from 776 of the residents.

Overall, 626 participants had complete household and individual questionnaire information and also had provided sufficient urine to analyze for *p*-nitrophenol (PNP, a metabolite of methyl parathion) and creatinine (*5*). Reference range PNP values (range 0–63 ppb, mean 1.6 ppb, median < 1.0 ppb, 99th percentile 16 ppb) had previously been reported from the CDC laboratory using creatinine-adjusted urine samples collected during the Third National Health and Nutrition Examination Survey (NHANES) (*6*). We used only creatinine-adjusted urinary PNP concentration in our statistical analysis. All descriptive statistics including means, medians, and frequencies were calculated using SAS software, Version 8 (*7*). Figures were created using Microsoft Excel 2000.

Laboratory Analysis

PNP measurements were made using the method of Hill et. al. (*8*). This highly specific and accurate method involves the use of carbon 14–labeled PNP as an internal standard (isotope dilution technique). After the addition of the internal standard, urine samples were hydrolyzed with enzyme, extracted and derivatized, and then analyzed using capillary gas chromatography combined with tandem mass spectrometry. A strict quality assurance program ensured that the sample results were reliable. This method was used with samples from the NHANES to establish reference range concentrations (*9*). Urinary creatinine was determined using a Kodak 250 Analyzercolorimetric method with creatinine kinase (*10*).

Results

Household Questionnaires

The 254 homes that we evaluated varied in age, number of rooms, number of years the current resident had lived in the home, and the number of people currently living in the home. Overall, these homes were older (median 25 years), of average size (median six rooms), and exhibited typical occupancy (median four persons living in the home). Transiency of this population is suggested by a mean of 1 year of residency for the current occupants. Almost 90% of the residences were rental units. The 1,095 people who lived in the 254 homes that we evaluated ranged in age from 1 week to 83 years (median 14.5 years). Fifty-seven percent of the residents were female and 68% were non-Hispanic black. Participants 15 years of age or younger were equally divided by gender; among residents older than 16 years, two-thirds were females and one-third were males. Every household that we evaluated contributed at least one individual questionnaire (range 1–14, median 3) and at least one urine sample (range 1–10, median 2).

The household questionnaire ascertained that at some point in the past, all of the homes that we surveyed had been sprayed by the applicator who used methyl parathion, although he appeared to vary the application pattern. Baseboards and cabinets beneath sinks were consistently sprayed in most homes; optional spray areas included heating duct systems, children's sleeping areas, and outdoor areas. The residents in our study population reported that they were instructed by the applicator to stay out of their house for several hours after spraying (64%), stay away overnight (7%), and in many instances (41%) they were told to wash their dishes before using them. The applicator left product in amounts ranging from 1 to 64 cups at 12% of the homes. Among those residents who had methyl parathion left in their homes, 89% had applied the product either as a spray or a wipe.

Fifty-four (23%) of the homes reported having at least one indoor pet. In response to the question "Did you have a pet that died within two weeks of your home being sprayed?", residents reported dogs, cats, birds, and fish had died. Thirty-five of the homes with indoor pets experienced an animal death. One resident anecdotally reported that his dog slept on a carpet that was still slightly damp from spraying that had occurred the day before. The dog became ill during the night with vomiting, diarrhea, and ataxia, and his normally light-colored fur was discolored to a yellowish-green. The dog died en route to the veterinarian. This animal, and at least two others, were presumptively diagnosed as having died from pesticide poisoning. No laboratory tests were performed, and there is no record that these incidents were reported to the health department.

Individual Questionnaires

The 747 people who completed individual questionnaires ranged in age from 1 week to 83 years (median 13.0). Parents or guardians completed information for children less than 10 years of age. Respondents reported a variety of preexisting physician-diagnosed chronic physical conditions, including asthma (18%), migraine headaches (15%), hypertension (10%), diabetes (4%), and cancer (2%). When asked about signs and symptoms that originated during the 2-week period following methyl parathion spraying, they reported headaches (30%), nausea (29%), night waking (28%), diarrhea (26%), restlessness (23%), difficulty breathing (21%), dizziness (21%), abdominal cramps (20%), excessive sweating (13%), incoordination (11%), excess salivation (9%), and mental confusion (7%).

Urine Samples

The 626 people with complete household and individual questionnaire information and laboratory analysis for PNP level were similar demographically to those participants without PNP samples (Table 1). Although 213 (34%) of these people did not specify an exact date when their home was sprayed, 179 (29%) reported that spraying had occurred within the past 180 days, 116 (19%) reported spraying between 180 and 365 days previously, and the remaining 118 (19%) said their home had been sprayed more than one year before sample collection. People living in homes sprayed <180 days previously were most likely to have the highest PNP levels (23% > 100 ppb PNP), but even people living in homes sprayed more than a year previously appeared to be highly exposed (8% > 100 ppb PNP) (Figure 1). Among the 213 people who did not know the date when their home was sprayed, 74% had no detectable PNP, 16% were < 50 ppb, 7% were ≥50 to 100 ppb, and 3% were ≥100 ppb PNP. Median PNP among the 28 study participants < 3 years of age was 94 ppb, compared with median 50 ppb among the ≥3 to < 16 years of age group (n = 125), median 31 ppb among the ≥16 to 46 years of age group (n = 107), and 49 ppb among people 46 years of age or older (n = 25). Age was unknown for five people who had detectible levels of PNP. A comparison of the age distribution of the 626 people who participated in urine sample analysis to the distribution of urinary PNP by age group suggests that the youngest and the oldest residents experienced the greatest exposure to methyl parathion sprayed in their homes (Figure 2).

Follow-up of Postspray Hospitalizations and Deaths

Of the 49 residents we followed up because they were hospitalized or died after their home was sprayed, 26 were less than 17 years of age. We summarized each case history and presented them to a series of medical reviewers who, guided by the criteria described in "Methods," classified 21 individuals as potentially manifesting organophosphate poisoning. Their ages ranged from 1 month to 44 years, with 12 (57%) of them younger than 3 years of age. Only one death was reported that fit our criteria for potential organophosphate poisoning. The number of days between hospitalization and reported spray date varied, including date unknown (n = 2), <1 week (n = 3), >1 week but <3 months (n = 8), and ≥3 months but less than 1 year (n = 8). Six of the case study patients had urinary PNP results available from the household survey. Only two of the case subjects had medical records that mentioned possible toxic exposure, and in both instances the reference specified potential misuse of prescription or recreational drugs. Three of the case studies are described below.

Case 1. On the day her house was sprayed, a full-term, previously healthy girl 4 months of age became irritable and seemed congested in her upper airway, with a thick whitish nasal discharge. Over the next several days these symptoms persisted, and she began to refuse food, developed a fever, more frequent bowel movements, and decreased sleep. On day 6, she was brought to the emergency department (ED) and was given iv antibiotics, fluids, oxygen, and a series of tests to determine infection status. Results included the following abnormal elevations: white blood cell (WBC) count, 18,700 (normal range, 4,800–10,800); platelets, 873,000 (normal range, 150,000–400,000); sodium, 160 milliequivalents per liter (MEQ/L) (normal range, 139–146); and chloride, 127 MEQ/L (normal range, 95–105). Chest and abdominal X rays were normal. She was discharged with a diagnosis of upper respiratory infection and a prescription for an antibiotic.

That night the child's condition worsened; paramedics were called and she was transported by air ambulance to a pediatric intensive care unit (PICU). Physical examination revealed lethargy, moderate to severe dehydration, and heme-positive, black, foul-smelling stools. Laboratory and X-ray tests were repeated and were the same as reported by the ED. All cultures (i.e., cerebrospinal fluid, blood, urine, stool) were negative.

The patient was given a working diagnosis of dehydration and hypernatremia. She remained in the hospital for more than 1 week, receiving fluids and antibiotics. She was discharged home without medications, but she continued to have a head lag. Fourteen months later her home was evaluated for methyl parathion contamination, and her urinary PNP level was 89 ppb.

Case 2. A boy 18 months of age was brought to the ED by his mother 3 days after their home had been sprayed. The child had been crying inconsolably for several hours before he collapsed, becoming limp and difficult to arouse. He also had three apneic episodes lasting 15–20 sec each, causing his mother to apply mouth-to-mouth resuscitation. No vomiting or diarrhea was reported. In the ED the child was noted to be lethargic but would cry in response to stimulation. His temperature was 37.9°C (100.2°F), pupil size and reactivity were normal, and skin was warm and dry. Physical exam, X rays, spinal tap, and laboratory tests were normal with the exception of an elevated WBC count of 11,400 (normal range, 4,800–10,800). After the spinal tap, the
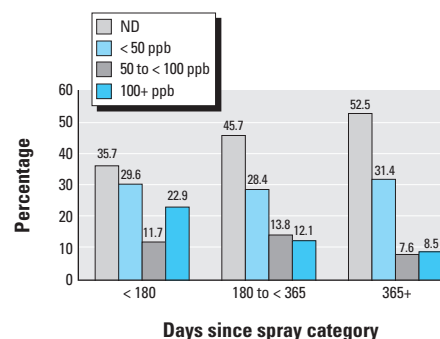
Figure 1: Percentage of people in each of four creatinine-adjusted PNP categories ranging from nondetectable to > 100 ppb, according to the number of days since a home was sprayed with a known spray for the 290 people who participated in a study of health effects following indoor application of methyl parathion in Lorain County, Ohio, 1995–1996.
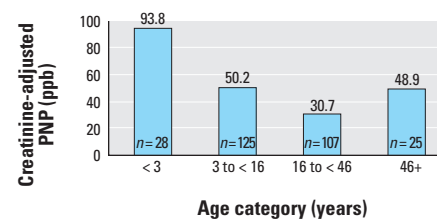
Figure 2. The median PNP level within age category of people who had complete questionnaire information and detectable levels of urinary PNP and participated in the investigation of health effects that occurred after methyl parathion was sprayed in Lorain County, Ohio, homes, 1995–1996.

Table 1. Demographic information from people with and without urine samples analyzed for PNP during the investigation of health effects related to indoor application of methyl parathion in Lorain County, Ohio, 1995–1996.

	All study residents (n = 1,095)[a]	Residents with urinary PNP results (n = 626)[b]	Residents without urine results (n = 469)[c]
Age			
Range	< 1 year–83.0 years	<1 year–83.0 years	<1 year–82 years
Median	14.0 years	14.0 years	15.0 years
Sex (female)	600 (56%)	366 (59%)	234 (53%)
Race/ethnicity (non-Hispanic black)	706 (68%)	398 (65%)	308 (73%)

[a]Information on age missing for 38 people; information on race/ethnicity missing for 63 people; information on sex missing for 30 people. [b]Information on age missing for 8 people; information on race/ethnicity missing for 17 people; information on sex missing for 4 people. [c]Information on age missing for 30 people; information on race/ethnicity missing for 46 people; information on sex missing for 26 people.

child had another apneic spell, became hypertensive, tachycardic, and developed pinpoint pupils and slowed neurologic function. A dose of naloxone (a narcotic antagonist used to treat acute intoxications) administered intramuscularly caused no improvement. The patient was transferred by helicopter to a tertiary medical center.

Upon arrival at the PICU, the child underwent a computerized tomography scan of the head, which was negative, and a suction of gastric contents, revealing no pharmacologic or toxicologic substances. The child was given a second dose of naloxone, again without effect. Physical exam was remarkable primarily for decreased responsiveness to pain, reduced deep tendon reflexes, small (2-mm) pupils with sluggish response to light, and purposeless speech. An EKG revealed occasional, spontaneously resolving sinus bradycardia. A urine toxicology screen was negative. After a 5-day hospital stay with only supportive treatment, the child was discharged home on no medications, with a diagnosis of poisoning by unspecified drug or medicine. Eighteen months later, when this child's home was evaluated for methyl parathion contamination, all surface wipe samples showed levels indicating the need for remediation. The child's urine PNP level was nondetectable.

Case 3. During a period of a year and a half of almost monthly spraying in her home, a 43-year-old female went to the ED on five different occasions. The primary complaint during three visits was coughing, wheezing, headache, and sore throat. She had no fever or abnormal vital signs during any of these visits. Her physical exams were always unremarkable, and she was diagnosed as having viral upper respiratory infection. During two additional visits, the primary complaint was nausea and vomiting. At the last visit she presented with a 2-day history of fever (101.9°F), nausea, vomiting, diarrhea, and an episode of fainting. Her physical exam was within normal limits and a pregnancy test was negative. During our investigation 1 year later, this woman had a urinary PNP level of 830 ppb.

Discussion

Illegal indoor application of methyl parathion in Lorain County, Ohio, had been going on for several years before coming to the attention of public health authorities. Once the contamination became known, many different local, state, and federal agencies became involved in evaluating and cleaning up the contaminated homes. As reported in this article, the CDC worked with local public health officials to try to determine the extent of human exposure and adverse health outcomes associated with this indoor misuse

of a pesticide. Our overall findings showed widespread and prolonged exposure to methyl parathion among the Lorain County study population. Younger children appeared to be at greatest risk of exposure. Health care providers apparently did not consider pesticide poisoning when confronted with acute generic presentations, and although veterinarians may have diagnosed pesticide poisoning in pets, this information did not reach the public health system. Our study results suggest that acute and short-term organophosphate poisoning occurred but was not recognized in Lorain County during the years that methyl parathion was sprayed in private residences.

Acute organophosphate poisoning may follow oral, dermal, or inhalation exposure to methyl parathion (*11*). After hepatic conversion, paraoxon (a more toxic form of parathion) is transported to the cholinergic nerve junction, where it inhibits acetylcholinesterase, resulting in accumulation of acetylcholine at the synapses. The effect is an initial stimulation, followed by paralysis of cholinergic transmission. Classic symptoms of acute poisoning can be divided into muscarinic (parasympathetic), nicotinic (sympathetic and motor), and central nervous system manifestations (*12*). Muscarinic signs and symptoms include chest tightness; dyspnea; increased bronchial secretions; increased sweating, salivation, and tearing; bradycardia and decreased blood pressure; nausea, vomiting, and diarrhea; miosis; and urinary incontinence. Nicotinic effects include muscular twitching, weakness, tachycardia, and increased blood pressure. Central nervous system manifestations include anxiety, restlessness, insomnia, headache, depression, confusion, slurred speech, and generalized weakness. Some researchers suggest that disturbances in thermoregulation may lead to elevated body temperature after exposure to anticholinesterase agents (*13*). Medical students are often taught to associate organophosphate poisoning with the acronym SLUDGE—salivation, lacrimation, urination, defecation, gastrointestinal distress, and emesis (*14*). Unfortunately these are all fairly generic symptoms that would not prompt an emergency caregiver to consider organophosphate poisoning unless the medical history suggested exposure (*15*).

Misdiagnosis of organophosphate poisoning may be more likely among children than adults because children are perceived as less likely to be exposed and because children may present with a less traditional array of signs and symptoms (*16*). In a retrospective review of 37 pediatric cases of confirmed organophosphate poisoning diagnosed at a tertiary care center in Dallas, Texas, 20 of 24 transferred patients were misdiagnosed by

the referring hospital (*17*). This is similar to the finding in our case review; none of the ED health care providers even considered pesticide poisoning in their diagnostic rule outs. As in the Zwiener and Ginsberg study (*17*), the Lorain children did not exhibit many of the classic signs and symptoms such as bradycardia, muscle fasciculation, and meiosis that are frequently observed in adults. It is possible that many of these children were exposed dermally, compared with the oral route of exposure that results in a more classic poisoning presentation. Dermal exposure may lead to more systemic manifestations such as bradycardia and diarrhea. However, even if a child does present with the cardinal signs of increased secretions, it is possible to confuse increased lacrimation with tears of distress, as in our second case example. Similarly, interpretation of increased salivation and incontinence is difficult in a child who wears diapers and is in the process of teething. It is also possible that children are more physiologically sensitive to pesticides compared with adults (*18*), as suggested by an investigation of indoor exposure to methyl parathion in a Mississippi home. While two adults remained symptom-free, seven siblings were hospitalized, and one child died (*3*).

The children in our study population also appeared to have a greater opportunity for acute and prolonged exposure than did other household members. The survey revealed that young children had disproportionately higher median PNP levels compared with other household members and that 12 of the 21 people who fit the criteria of potentially poisoned were children younger than 3 years of age. Children were logically at greater risk of physical contact with sprayed surfaces because very young children are more likely to spend time in their homes, and the most consistently sprayed areas were baseboards that crawling children could routinely contact.

Our finding that animals may have been missed sentinels of residential organophosphate exposure has potential public health implications. Veterinarians are more likely to diagnose and treat organophosphate poisoning because household pets are frequently intentionally exposed to this class of chemicals as part of external parasite control programs. The availability and affordability of over-the-counter as well as veterinary-supplied insecticides and pesticides means that pet owners have easy access to these products. When an animal becomes ill shortly after a pesticide application, that exposure is often captured in the history supplied to the veterinarian. An established method for timely communication between veterinarians and public health officials may have brought methyl parathion misuse in Lorain County to the attention of

public health authorities years earlier than actually occurred. The animal deaths that we and other researchers (*17*) have documented should be considered sentinels for potential human exposure.

Our follow-up study was limited by our inability to obtain pretreatment blood samples to evaluate reduced cholinesterase activity. Erythrocyte and serum cholinesterase activity assays are considered essential as biomarkers of effect for organophosphate poisoning. Given the retrospective review of medical records, we could not collect biological samples that would be representative of the poisoning incident. Another area of uncertainty in our study is the separation between the date of spraying and the onset of symptoms. When methyl parathion is applied outdoors, it degrades quickly (*1*). However, during the health survey in Lorain, we learned that methyl parathion sprayed indoors behaves very differently from the product that is applied to crops in fields. Homes that had been sprayed more than a year previously continued to exhibit surface wipe samples positive for methyl parathion or PNP (*4*). Similarly, as reported in this article, we found evidence of ongoing human exposure with elevated PNP levels in residents whose homes had been sprayed years previously.

Our data-gathering techniques and criteria for our case series probably underestimate the extent of morbidity resulting from the spraying. We actively followed up hospitalizations and deaths; we did not review medical records of participants who sought outpatient care. Our exclusion of cases where infectious processes were present ignores the possibility that infection may have occurred secondarily to the bronchorrhea that can result from organophosphate poisoning. Similarly, our exclusion of cases with psychiatric diagnoses may have missed subtle poisonings that can manifest as anxiety (*19*).

Organophosphate poisonings are almost certainly underrecognized and underreported in the United States, and this may be especially true in semiurban settings like Lorain County that do not have agricultural or other obvious occupational sources of exposure. Use of unlicensed applicators increases the risk of exposure to pesticides that are not formulated or registered for indoor use (*17*). Our investigation reinforces the importance of communication among all health care professionals in a community. If an ED physician had considered organophosphate poisoning for any of the 21 people we identified as potentially poisoned, or if a veterinarian or animal owner had reported the finding of organophosphate poisoning among indoor pets, it is possible that the contamination in Lorain County would not have reached a size and scope that required Superfund intervention.

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