# Methyl Parathion in Residential Properties: Relocation and Decontamination Methodology

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**In November 1994 methyl parathion (MP), a restricted agricultural pesticide, was discovered to have been illegally sprayed within hundreds of residences in Lorain County, Ohio. Surface levels and air concentrations of MP revealed detectable levels of the pesticide 3 years after spraying. Because of the high toxicity of MP (lethal dose to 50% of rats tested [$LD_{50}$] = 15 mg/kg) and long half-life indoors, risk-based relocation and decontamination criteria were created. Relocation criteria were derived based on levels of *p*-nitrophenol in urine, a metabolic byproduct of MP exposure. In Ohio, concentrations of MP on surfaces and in the air were also used to trigger relocations. The criteria applied in Ohio underwent refinement as cases of MP misuse were found in Mississippi and then in several other states. The MP investigation (1994–1997) was the largest pesticide misuse case in the nation, ultimately involving the sampling of 9,000 residences and the decontamination of 1,000 properties. This article describes the methodology used for relocation of residents and decontamination of properties having MP. *Key words:* decontamination, methyl parathion, risk assessment. *Environ Health Perspect* 110(suppl 6):1061–1070 (2002).**
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In November 1994 methyl parathion (MP) was found to have been illegally sprayed by an unlicensed pesticide applicator in more than 400 residential properties of Lorain and Elyria, Ohio (*1*). By May 1997 MP was believed to have been applied in 4,500 properties in Mississippi and Louisiana. Hundreds of properties in Illinois and Tennessee were also contaminated with MP (*2*). Spraying was generally conducted in poor, urban communities primarily to control cockroaches (*1,2*). MP is a restricted organophosphate (OP) pesticide for use only on agricultural crops by certified applicators (*3*). Outdoors, MP breaks down within a few days because of biodegradation and contact with water (*4*). As found in Ohio and other locations, inside homes, MP can remain for years. MP is highly toxic, with a lethal dose in 50% of rats tested ($LD_{50}$) of 12–24 mg/kg (*4,5*). Deaths have occurred from oral ingestion of MP and from a combination of dermal and inhalation exposure (*6*). Two children, 4 and 11 years of age, died when a 4% MP spray was illegally applied to the interior of a Mississippi home (*7*).

The pesticide applicator in Ohio had treated properties with MP solutions averaging 6.5% as determined by analyses of MP solutions left with residents in juice jars and cleaning bottles. Spraying occurred in kitchens, including inside cabinets, bathrooms, living rooms, and bedrooms. Sofas and chairs were sometimes sprayed. MP collected from floor baseboards and splashboards of kitchen countertops in 28 properties averaged 100 µg MP/100 $cm^2$ (*8*). Surface levels of up to 890 µg MP/100 $cm^2$ were found on kitchen counter splashboards. A child's food dish was found to have 6,000 µg MP. Personal clothing and home furnishings (e.g., furniture, drapes, and carpeting) were found to have detectable levels of MP. Air levels averaged 7.5 µg/$m^3$, with a maximum value of 30 µg/$m^3$. The U.S. Centers for Disease Control and Prevention (CDC) detected *p*-nitrophenol (PNP), an MP metabolic byproduct, in the urine of residents whose properties had been sprayed with MP. Urine PNP levels as high as 4,800 µg/L (11,000 µg/L, creatinine adjusted) were found in a 4-year-old child (*9*). These values are in the range of those found in children who became ill or died of MP exposure in Mississippi (*7*).

Based on the high acute toxicity of MP, environmental levels within residences, and known exposures, in November 1994 an interagency group was formed by the U.S. Environmental Protection Agency (U.S. EPA), CDC, Agency for Toxic Substances and Disease Registry (ATSDR), National Institute for Occupational Safety and Health (NIOSH), Ohio Department of Health and Department of Agriculture, Lorain County and Lorain City health departments, Elyria Health Department, U.S. Coast Guard National Strike Force, Lorain County Human Services, Salvation Army, and American Red Cross (*1,4,5*). This group was charged with developing health criteria to determine which residents should be temporarily relocated, how to handle relocations, which properties should be decontaminated, and what were the most effective decontamination methods. The criteria developed were initially based on conventional risk assessment techniques that required many assumptions. As more information was obtained on the relationships between environmental levels and exposure in Ohio and then in other states, the decision-making criteria were refined. This process of data collection, health risk evaluation, and decision making is described in this article.

## Methods

***Sampling.*** An environmental protocol was developed for the purpose of gathering each sprayed residence in MP data to *a*) assess potential exposures and health risks, *b*) provide a basis for enforcement actions, and *c*) determine properties and areas within properties in need of decontamination. In each sprayed residence in Lorain, a total of six 100-$cm^2$ surface wipe samples were taken, one from each of the following areas: the kitchen baseboard immediately adjacent to the refrigerator, kitchen counter splashboard in a food preparation area, under the kitchen sink adjacent to the drain pipe, living room under a heat register, bathroom baseboard under the lavatory, and bedroom baseboard (based on descending priority of children < 5 years of age, pregnant women, and adults). Samples for MP and other OPs (e.g., chloropyrifos) were collected by wiping the area inside a 10 × 10 cm template with gauze

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pads charged with isopropyl alcohol, which were then placed in glass vials. Field blanks were also collected. A single 4-hr air sample was collected from the top of the kitchen refrigerator using sampling pumps (Mine Safety Appliance, Pittsburgh, PA, USA) precalibrated at 1 L/hr, fitted with an SKC XAD-2 sampler tube (SKC, Inc., Eighty Four, PA, USA). Environmental PNP wipe samples were collected from a very small subset of properties by swabbing a 10 × 10 cm area, first with a gauze pad charged with isopropyl alcohol and then with a second gauze pad charged with distilled water.

Both gauze wipe samples and XAD air absorbent material were extracted for 1 hr in acetone, and the residue was dissolved with 10% acetone in hexane. Analysis was performed with a Hewlett Packard 5890 Series II gas chromatograph (Hewlett Packard, Palo Alto, CA, USA) with a DB-1, DB-5 (J&W Scientific, Folsom, CA, USA), or HP-1 (Hewlett Packard) capillary column and a flame photometric (detector D) or a mass-selective detector (Hewlett Packard). Quality control steps included ensuring that replicate standards were within ±15%, running blanks every 10 samples, and confirming the OP with a second detector.

PNP samples were extracted with ethyl acetate acidified with sulfuric acid, and the residues were washed with methylene chloride followed by hexane. The final residue was diluted with acetonitrile and analyzed by high-performance liquid chromatography (Waters 600-MS) coupled to a photo diode array detector, fitted with a Nova-Pak $C_{18}$ 60 A4 µm column (Waters Corp., Milford, MA, USA). Quality control steps required that replicate standards be within ±15%, and blanks were run every 10 samples.

***Toxicology and risk evaluation.*** A review of MP toxicology was necessary to assess acute and chronic hazards and to develop a risk-based approach to establish criteria for multiple routes of exposure. The evaluation below is not intended to be an exhaustive literature review but to reflect the actual evaluation process used to address MP contamination in residences. Extensive literature reviews are available elsewhere (*6*).

Short-term acute exposures to children from ingestion or dermal exposure to MP liquids in Ohio were of particular concern. Direct ingestion of 1.8–3.6 mL of 6.5% MP by a 10-kg, 1-year-old child would yield oral doses in the range of $LD_{50}$ levels reported in animal studies. $LD_{50}$ studies for dermal exposure to MP range from 67 to 120 mg/kg in rats (*4,5*). Extrapolating, dermal contact with 10–35 mL of a 6.5% MP solution, as found in Ohio residences, would be potentially lethal to a 10-kg child. As a result of these findings, hazardous materials teams conducted searches of Ohio properties where MP had been sprayed, removing a total of 15 unmarked containers left by the applicator after treating the properties. Another 20 containers were later removed by decontamination teams.

Because there was a potential for acute poisoning, questionnaires and interviews were developed and conducted by health agencies to assess adverse health effects and the need for medical follow-up. Although not used for a primary relocation criterion, an OP symptom list was developed. Medical follow-up was recommended when two or more symptoms (e.g., severe headache, incoordination, muscle twitching/tremors, difficulty breathing, vomiting, diarrhea, involuntary urination) were self-reported.

Voluntary, low-dose oral studies from the 1960s provided effects information on MP (*8,9*). At oral dosages of 6.5–7.0 mg/day, or approximately 0.1 mg MP/kg/day, plasma cholinesterase (ChE) was found to be depressed by 10% relative to controls in a 59-day study (*10*). At dosages averaging 7.0–9.0 mg/day (0.1 mg MP/kg/day) for 30 days, red blood cell (RBC) count and plasma ChE were depressed by 20% relative to controls (*11*). The dosages eliciting reduced ChE depression are about 3-fold lower than the no-observed-adverse-effect levels (NOAELs) found from animal studies, although such differences could be due to experimental variation. Mild OP poisoning is indicated at ChE depression of 20–50%, and in agricultural applications workers are often removed from exposures when ChE depression reaches 50% of normal baseline (*12,13*). Although many issues related to the use of ChE as a criterion, including lack of baseline measurements and laboratory variation, an emergency (48-hr) relocation criterion of 20% RBC ChE depression was applied. A 20% RBC depression was defined as 20% below lower end normal values or values of a particular laboratory.

Residues of MP were detected in homes up to 3 years after spraying, requiring risk assessments and resulting criteria development to consider potential subchronic and chronic effects. A 13-week, subchronic study in dogs reported a lowest-observed-adverse-effect level (LOAEL) at 3.0 mg MP/kg/day and an NOAEL of 0.3 mg MP/kg/day for systemic effects and for RBC, plasma, and brain ChE levels (*14*). A 1-year dog study also found a NOAEL of 0.3 mg/kg/day (*15*).

In addition to dose studies in humans, the subchronic dog studies were selected as a key basis on which to assess potential human health effects. Very little difference exists in MP toxicity between species. A 90-day rat study submitted to the U.S. EPA to support the registration of MP reported a NOAEL of 0.25 mg MP/kg/day, including RBC and plasma and brain ChE levels (*16*). This dosage translates to a subchronic reference dose (RfD) of $2.5 \times 10^{-3}$ mg MP/kg/day using a uncertainty factor of 10 for extrapolation from animals and humans and another factor of 10 to account for variation in human sensitivity (*17*). An RfD is an exposure level derived by the U.S. EPA at which adverse health effects are unlikely (*18*). A subchronic RfD is often appropriate for assessing exposures that are less than 10% of a lifetime (7 years), such as in the case of chemical degradation (*18*). In a 2-year rat study, a dosage of 2.5 mg MP/kg/day elicited tremors and peripheral neuropathy, and 0.25 mg MP/kg/day elicited abnormal gait (*19*). The NOAEL was 0.025 mg MP/kg/day, resulting in a chronic RfD of $2.5E^{-4}$ mg MP/kg/day, appropriate for assessing exposures greater than 10% (7 years) of lifetime (*20*). Subchronic information was considered more relevant than chronic, lifetime information because residents would not be exposed to MP for a lifetime.

There was extensive discussion on the establishment an MP dose considered to be unacceptable and requiring relocation of residents and decontamination of properties. There were concerns that long-term, low-level exposure to MP could cause chronic neurological impairments, to both the central and peripheral nervous systems, below levels eliciting either symptoms of poisoning or ChE depression. Clear evidence has been found that chronic adverse effects occurred after acute poisonings with OPs, although the results were often subclinical. Case–control studies of agricultural workers reported significant differences in memory, abstraction, or vibrotactile responses relative to controls, ranging from 2 to 9 years after poisoning (*21–23*). Studies of long-term, low-level exposure to OPs reported mixed results, but some did demonstrate subclinical findings. A case–control study of 45 applicators with at least an episode of ChE inhibition but with no poisoning found no central or peripheral nervous system effects (*24*). In contrast, a study of pesticide applicators with a mean exposure of 20 years showed significant decreases in vibrotactile sensitivity relative to controls (*25*). In a case–control study of persons exposed for an average of 15 years to pesticides in sheep dipping, significant effects were found in attention and information processing (*26*). Animal studies of MP and other OPs also suggested that changes in the nervous system may occur at doses at the threshold of ChE inhibition (0.3 mg/kg/day) (*27,28*).

Dose levels based on the subchronic MP RfD (0.0025 mg/kg/day), with two uncertainty and safety factors of 10 from a NOAEL of 0.25 mg/kg/day was considered unnecessary to protect public health because

MP levels were declining and disappeared within 3 years. Dose levels in the range of 0.03 mg MP/kg/day or about 10 times below the subchronic NOAEL and 3 times below the threshold of human Cholinesterase (CE) depression were ultimately selected for relocation and decontamination criteria for Ohio properties.

***Exposure assessment.*** Field studies and oral dosing studies provided information on PNP excretion resulting from MP or ethyl parathion (EP) exposures. In workers applying EP, PNP was detected in urine and was roughly correlated with RBC and ChE depression (*29*). A 10% CE depression was associated with urinary PNP levels of 2,000 ppb, and no ChE depression was observed below 300 ppb. PNP was considered a more sensitive indicator of EP exposure than were ChE measurements. In assessing worker exposure to MP, which was dominated by the dermal pathway, urinary PNP was also considered a more reliable exposure indicator of exposure than was ChE depression (*30,31*). With more data on the metabolism of MP, PNP was considered to potentially provide for a quantitative measurement of exposure. In a later 5-day study by Morgan et al. on four human subjects who were administered oral MP dosages of 2 and 4 mg/day, MP metabolites in the urine were evaluated (*32*). About 90% of the PNP was excreted within 8 hr, whereas longer times were required for dimethylphosphate (DMP). Seventy-six percent of the PNP was recovered from urine samples compared with standards. Quantification problems were experienced with DMP. By doubling the mass of PNP observed to convert to MP dose, 29% of the MP dose was recovered within 24 hr for both 2 and 4 mg/day dosage groups.

In Lorain, Ohio, daily MP exposure could be estimated from PNP urine data, as found in the study by Morgan et al. (*32*). Although there were recognized uncertainties in the method, including using a method based on an oral dose when the environmental exposures are likely to be dermally driven, the approach was considered less uncertain than using conventional risk assessment approaches. Initially, risk assessment techniques were used to establish surface levels of concern. Converting pesticide surface concentrations into estimates of dermal exposure values is particularly difficult because exposure estimates are based on many assumptions, including the time spent within the contaminated area, the frequency of contact, the age of the person, transfer to clothing and skin, and absorption. Yet dermal exposure in this MP case was considered to be the dominant route of exposure. PNP urine data provided a better estimate of total MP exposure from dermal, inhalation, and direct ingestion pathways. The collection of 24 total urine samples from young children was impractical, leading to the use of twice-daily (morning and evening) urine samples and data on urine production rates. To provide a conservative assessment, an upper range of urine production of 29 mL/kg of body weight per day from 17 studies on children was applied (*33*). Because this rate was similar to the upper end found for adult males (1.75 L/day), it was applied to all populations.

Four levels of priority were established in Ohio based on MP environmental levels and exposures. Concentrations of MP in the indoor air and on surfaces and PNP levels in the urine were independently applied to trigger actions (Table 1). An estimated MP dose greater than or equal to 0.1 mg/kg/day, the threshold of ChE depression, was selected as the criterion for rapid relocation (category L-1, within 48 hr). A dose of 0.1 mg MP/kg/day was converted into the equivalent of 600 ppb of PNP in the urine using the following equation, based on the work of Morgan et al. and measured rates of daily urine production per body weight (*32,33*). For children and adults 6 years of age and older, a urine concentration of 600 ppb PNP triggered relocation within 48 hr.

$$\text{Value of PNP in urine} = \frac{0.1\,\text{mg MP/kg/day}}{0.29\,\text{l/kg} \times 2\ \text{MP/PNP} \times 0.29\ \text{recovery}}$$

An additional safety factor of 4 was provided for infants 1 year or younger, resulting in a PNP rapid relocation action level of 150 ppb or a dose of approximately 0.025 mg MP/kg/day. A safety factor of 2 was established for pregnant women and children between 1 and 6 years of age, yielding an action level of 300 ppb PNP, or an MP dose of approximately 0.050 mg MP/kg/day.

An L-2 category for priority relocation (1 week) was established for individuals having PNP urine levels one-third those of L-1: ≥50 < 150 ppb for infants younger than 1 year, 100–300 ppb for pregnant women and children 1–5 years of age, and ≥200 < 600 ppb for adults and children older than 5 years. This categorization yielded protection from estimated MP doses of 0.008 mg/kg or greater for infants 1 year or younger, 0.016 mg/kg/day for pregnant women and children 1–5 years of age, and 0.033 mg/kg/day for all others. An L-3 category (limited or no action) designated persons having urine levels 3 times lower than L-2 (10 times lower than L-1). An L-4 category (no action) designated individuals with PNP urine levels 30 times or more below L-1 levels.

Relocation decisions were based on PNP values unadjusted for creatinine within the first few weeks of the project, then later on creatinine-adjusted PNP levels. A morning and evening urine sample was taken from each person, and relocations were based on whether the average of the two measurements exceeded PNP health criteria. Although urine information was considered ideal because it integrated exposure routes, obtaining urine results took several weeks until a more rapid analytical method was developed by the CDC. In addition some persons were reluctant to provide urine samples, and collecting urine from very young children and infants was not always successful. In Ohio, the use of environmental data, which were available within a few days after collection, was considered necessary to protect public health.

Simple regressions were performed of surface and air concentrations versus urine PNP levels, unadjusted for creatinine (Figures 1, 2). An average of MP surface levels versus PNP urine levels from 22 properties and 35 people, including children, revealed a very rough correlation ($r$ = 0.67) between surface and unadjusted urine levels (Figure 1). Urine PNP concentrations were

**Table 1.** Action levels at Lorain, Ohio.[a]

| Parameter | Category L-1 Rapid relocation | L-2 Priority | L-3 Minimal/no action | L-4 No action |
|---|---|---|---|---|
| Urine PNP (μg/L)[b] | | | | |
| <1 year of age | ≥150 | ≥50 < 150 | ≥5 < 50 | <5 |
| 1–5 years of age and pregnant women | ≥300 | ≥100 < 300 | ≥10 < 100 | <10 |
| >5 years of age | ≥600 | ≥200 < 600 | ≥20 < 200 | <20 |
| Wipe (μg/100 cm$^2$) | ≥150 | ≥50 < 150 | ≥15 < 50 | <15 |
| Air (μg/m$^3$) | ≥10 | ≥3 < 10 | <3 | <3 |

[a]Data from Clark (*8*). [b]Creatinine adjusted.

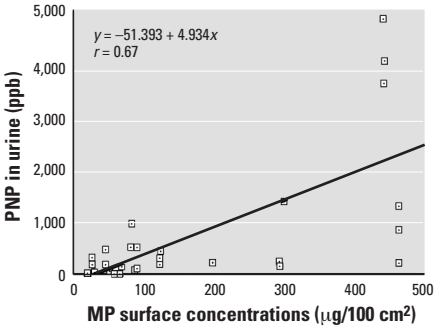


**Figure 1.** PNP in urine versus mean surface MP.

approximately 5 times the average surface concentrations. From this regression, an average surface concentration of 120 µg MP/100 $cm^2$ or greater corresponded to average unadjusted PNP urine levels of about 600 ppb for all groups. Only 1 of 12 children 1–5 years of age from 10 households with average MP levels below 150 µg/100 $cm^2$ had urine levels above the 300 ppb PNP trigger for emergency relocation of young children. The child had a PNP level of 530 ppb. Six individuals in 3 of 5 households with surface MP levels greater than 150 µg/100 $cm^2$ (an emergency relocation criterion) had PNP urine levels above the 600 ppb PNP criterion for emergency relocation (L-1).

Average surface levels of 40 µg MP/100 $cm^2$ were associated with PNP concentrations of 200 ppb for all populations, a value selected for rapid relocation (within 2 weeks) of persons 6 years of age and older. Two of seven children, 2–5 years of age, from 8 households with MP averaging below 50 µg/100 $cm^2$ had urine levels over the 100-ppb PNP criterion used for rapid relocation (L-2) for this age group and pregnant women. The two children had PNP urine concentrations of 200 ppb and 110 ppb, the latter being just marginally above the 100 ppb trigger for rapid relocation. Seven of 12 persons from 10 households with surface concentrations of 50–150 µg/100 $cm^2$ had PNP urine levels less than the 200 ppb L-2 trigger. An MP value ≥50 µg/100 $cm^2$ and <150 µg/100 $cm^2$ was selected to categorize properties for decontamination and persons for priority relocation (L-2).

Residential properties were placed in an L-3 rating (minimal or no action) if surface MP levels were between ≥15 µg/100 $cm^2$ but <50 µg/100 $cm^2$. At MP surface levels of 15 µg/100 $cm^2$ or lower, designated as L-4 or no action, no individuals were found with PNP urine concentrations above 100 ppb. Although the relationships between surface and urine levels were recognized as being very crude and the number of observations small, there was also a recognized need to protect public health using available information.

A regression of MP air concentrations versus PNP in urine found an $r$ value of 0.64 (Figure 2). Urine levels of PNP were approximately 70 times air concentrations. An emergency relocation trigger value (L-1) of 10 µg MP/$m^3$ was selected, approximating PNP urine concentrations of 600 ppb. At air levels below 10 µg MP/$m^3$, for children between 2 and 5 years of age, 1 in 5 had urine levels above 300 ppb, an emergency relation criterion for this group. This child had 530 ppb PNP. A rapid relocation trigger (L-2) of 3 µg MP/$m^3$ and greater was selected because the air concentration corresponded to PNP urine values of about 200 ppb. At air levels below 3 µg MP/$m^3$, which categorized properties as L-3, 1 child in 4 had PNP concentrations above 100 ppb, the rapid relocation criterion for children 1–5 years old. At MP air concentrations of 1 µg/$m^3$ or less, no persons had PNP urine levels greater than 100 ppb.

A regression ($r$ = 0.69) of MP air and surface concentrations provided additional support for the selection of criteria (Figure 3). Although substantial variability existed, surface levels of 150 µg/100 $cm^2$ were associated with MP air concentrations of about 7 µg/$m^3$, close to the 10 µg/$m^3$ L-1 criterion derived from urine data, whereas surface levels of 50 µg/100 $cm^2$ were associated with MP air levels of about 2 µg/$m^3$, compared with the 3 µg/$m^3$ L-2 criterion.

In Lorain, Ohio, each surface, air, urine, and ChE criterion was applied independently for L-1 and L-2 groups. That is, an exceedance of any one criterion in the groups would trigger temporary relocation of families and cleanup. Surface data, which became available sooner than air or urine data, were the primary drivers for relocations in Ohio. Urine PNP was the sole determinant in relocation and decontamination decisions involving L-3 properties.

***Decontamination of residential properties.*** The U.S. EPA Region 5 Office of Superfund in Chicago became involved in the cleanup process because of unwillingness and inability of the pesticide applicator in Ohio to decontaminate residences. U.S. EPA actions, which included relocation of residents, decontamination, and restoration of properties, were conducted. After meeting with local, state, and federal agencies, a command and control system was established by U.S. EPA on-scene coordinators.

On finding that either environmental levels of MP or urine levels of PNP exceeded health criteria, a written notification was provided by local health departments to owners and residents that described potential health risks and the need to evacuate the properties. Immediately on issuance of the communication, teams consisting of public affairs specialists and local health personnel met directly with residents and property owners. Affected persons received information on health risks of MP, relocation, MP decontamination of clothing and linens, and packing instructions (*34*). To remove MP residues, residents were instructed to wash clothing 2 or 3 times with liquid detergent and hot water (*35,36*). Residents were also provided information recommending that all items (excluding clothes and linens) used for babies and small children; drapes/window treatments, rugs, and carpets; children's toys; food products; kitchen wood and plastic utensils; personal products; and any contaminated furniture should to be left in the home to be disposed of by the decontamination crews. The cost of all contaminated furniture (including mattresses); infant furniture (including strollers, car seats, cribs, crib mattresses, walkers, and playpens); and children's toys would be considered for reimbursement by the U.S. EPA. Other items would not be considered for reimbursement.

The U.S. EPA obtained access agreements from the residents and the owners of each residence before decontamination. U.S. EPA determined that decontamination would impact residents requiring their temporary relocation (*1*). A direct communication link between the cleanup operations and the residents was developed. This was especially important for working with the local Hispanic/Latino community, many of whom required translations of materials.

Before decontamination, documentation of each residence was conducted with videotape and photographs of the interior and exterior of the residence, noting the condition of the dwelling and all sensitive possessions that remained on site. If friable asbestos was suspected or observed, a certified asbestos inspector collected samples and an asbestos contractor conducted asbestos removal before MP decontamination. Household belongings were removed to storage containers. High-contact items, such as toys, were disposed of on the basis of the decontamination procedures and the terms of the access agreement.

As was the case in creating the MP action levels, the decontamination protocol under-

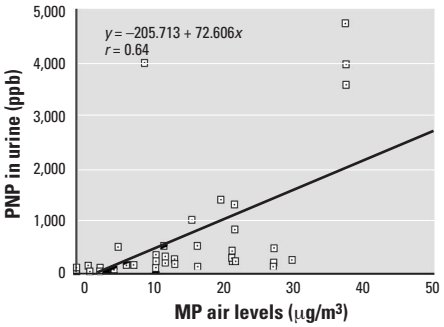


**Figure 2.** PNP in urine versus MP in air.

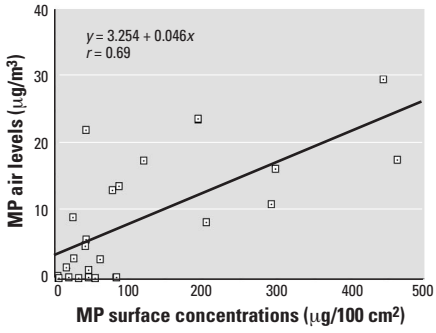


**Figure 3.** MP air levels versus MP mean surface levels.

went significant evolution. Decontamination criteria were developed that required that the residence meet an average of 50 µg MP/100 $cm^2$, with no values higher than 50 µg MP/100 $cm^2$ in bedrooms, living rooms, and bathrooms. A goal of 5 µg MP/100 $cm^2$ was established but not required for living rooms and bedrooms. Areas of low contact, such as utility rooms and under the sink, were permitted to have up to 150 µg MP/100 $cm^2$. All air samples were required to have less than 3 µg MP/$m^3$. Four homes with the highest surface levels of MP were selected for decontamination tests. Initial attempts were made to decontaminate surfaces with an ammonia fog ($NH_4OH$ in humidifiers) at temperatures of 80°F. This technique was successful on hard surfaces such as metal in ductwork, but it was unsuccessful on porous surfaces such as wood and wallboards or for fabrics and carpeting (*1*). Attempts were then made to clean surfaces with a mixture of ammonium hydroxide, water, and isopropyl alcohol. After decontamination and postdecontamination sampling, residences often did not meet the decontamination target criteria and required recleaning 2 and often 3 times. Because of the ineffectiveness of the initial decontamination solution, the decontamination protocol was modified.

The new decontamination protocol included the removal of all carpets, window treatments, furnace filters, furniture, mattresses, and food products from the residence (*1*). A commercially available solution, UltraKleen (UK; Sterilux Corp., Owings Mill, MD, USA), was used as the primary decontamination solution. The product is a caustic solution with a pH of about 12 and was found to rapidly hydrolyze MP to PNP. UK was developed as a food-contact hard surface cleaner decontaminant, primarily used for cleaning equipment at meat and poultry plants. All decontamination work was completed in Level C personal protective equipment (air purifying respirator and chemical-resistant suit). The decontamination protocol was a stepped process. First, the entire unit was sprayed with a dilute 2% UK solution (Figure 4). The resulting yellow color from PNP was used by the field crews to identify spray patterns of the exterminator, help concentrate their cleanup efforts, and thus aid in the decontamination process. The second step was to thoroughly scrub a 15% UK solution into and over all surfaces. This was followed by a sponge wipe of all surfaces with a 2% UK solution, which in heavily stained areas was followed by another 15% UK wash. These steps were repeated as often as necessary until all visible yellow PNP coloration was gone.

After completion of the UK wash, all surfaces were wiped with a vinegar and water solution to remove any residual PNP (*1*). The final step was to wipe all surfaces with clean water to remove all UK and vinegar residues. In addition, all appliances, cookware, dishes, and drinking glasses were washed with a 5% UK solution and thoroughly rinsed with fresh water to remove any residues. After the decontamination procedure, a high-efficiency particulate air (HEPA) filter vacuum unit was used to remove all particulates from the residence and to complete the decontamination. Furnaces were set at 80°F and a 12-hr ammonia fog ($NH_4OH$) in humidifiers was used to reach areas such as ductwork that were difficult to reach.

However, shortly after beginning to use this new protocol, decontamination teams observed the recontamination of some surfaces that previously had been cleaned. Some heavily contaminated surfaces (e.g., wood baseboards, drywall, plaster, wood cabinets) had absorbed the pesticide and became recontaminated as the UK solution wicked the pesticide to the surface.

After a detailed cost evaluation of repeated decontamination with solutions versus replacement of contaminated materials, the decontamination protocol was later amended to include the removal of baseboards, lower portions of the dry wall, kitchen cabinets, ceilings, and any other portion of the house structure that remained contaminated after an initial decontamination attempt (Figure 5). After these procedural changes, the time required to complete the decontamination of the residence was reduced, as were costs.

Sampling was also conducted 10–12 hr after the decontamination teams completed their work, and indoor temperatures were returned to 72°F (*1*). The sampling protocol after decontamination consisted of both wipe and air samples collected to verify the effectiveness of the residential cleanup and to allow the initiation of the restoration phase. A minimum of two and a maximum of four air samples were collected from each residence, at least one sample from each floor of the residence. Between 8 and 12 wipe samples were collected from each home, depending on the number of rooms in the residence. Air and wipe samples were analyzed by Roy F. Weston, Inc., Laboratories (Edison, NY, USA) using NIOSH method 5600 for wipe samples and modified 5600 method for air samples.

The analytical results usually were received within 48 hr, and the data were reviewed against the preestablished target cleanup criteria. If the home met cleanup criteria, the residence would be immediately referred to the restoration phase. If the analytical data did not meet target cleanup levels, the unit was returned to the decontamination phase for "spot" decontamination of the "hot" areas, as identified in the sampling results. The spot decontamination was continued until target cleanup levels were achieved and the residence could be referred to the restoration program.

In early February 1995 U.S. EPA contractors began restoring the decontaminated homes in Ohio (*1*). Restoration of each unit was designed to return each unit to preexisting condition and to repair damage caused by the destructive decontamination procedures. Restoration included sealing walls and ceilings and portions of the floors with an alcohol-based primer, hanging new and repairing existing drywall, painting ceilings and walls, installing baseboards and cabinets in the kitchen, hooking up all appliances and plumbing, and installing carpeting and padding. The alcohol-based primer sealant was used to create a 2- to 3-mm thick barrier that was found to prevent MP from leaching through to the drywall surface. Before the return of the residents, a video documentation of each residence was made. Stored belongings and furniture not found to be contaminated were moved back into the residence and photographed, and their condition was documented and decontaminated or disposed.

**Figure 4.** Decontamination of surfaces.

**Figure 5.** Residences with drywall removed.

## Results

***Environmental and urine data.*** A total of 480 properties in Ohio were investigated and sampled for MP contamination by the

State of Ohio. Ultimately, the Ohio Department of Agriculture analyzed more than 7,000 samples for MP and other OPs within a 6-month period. Another 250 properties were independently investigated by the U.S. Department of Housing and Urban Development. None of these properties were found to have MP levels above the criteria. The distributions of individuals by categories of PNP, mean MP surface levels (L-1, L-2, L-3, L-4), and age categories are presented in Figure 6. Most individuals had PNP concentrations below levels of potential concern, especially in L-4 and L-3 categories. However, the data show that as MP surface concentrations increase, the number of individuals above PNP exposure criteria increases.

Once the CDC established a rapid method for urine analysis, this measure became the dominant criterion for evaluating persons in L-3 properties. Several small studies were conducted on L-2 and L-3 properties and persons to assess short-term variation in urine levels (Table 2), changes in exposure over time, MP concentrations on high-contact surfaces, and PNP in the environment (Table 3). Variation in exposure to MP was believed to be highly dependent on amount of time spent inside the property, age of the individual, amount of contact with hot spots, degree of clothing contamination, and individual behavior. To assess variation in PNP, repeated urine testing was conducted on a subset of persons in 20 L-3 properties over 2 days. In eight children from whom four urine samples were collected (2 A.M. and 2 P.M.), the range in highest to lowest urine levels averaged about 2-fold (Table 2). Samples collected in the morning tended to have higher PNP concentrations and less variability than those collected in the afternoon. In 3 (15%) of the 20 properties, average urine levels were above the MP exposure protocol when MP concentrations were less than the 50 μg/100 cm$^2$ L-3 criterion. The highest single PNP urine level found in a child in L-3 properties was 254 ppb compared with the 100 ppb L-3 criterion. To assess exposure over time and to ensure that more highly exposed individuals were not missed, persons in L-3 properties were retested about 4 months after initial testing. Overall, retesting found that average PNP urine levels had declined from 87 to 42 ppb, or a reduction of about 50% in 4 months. Ten children of 101 persons tested after 4 months still had urine levels > 100 ppb PNP, resulting in decontamination of a few L-3 properties.

The protocol applied baseboard surface samples from lower-contact areas as an rough index of potential exposure. A study of eight L-3 properties was conducted to assess potential exposure from higher-contact surfaces such as floors and carpets. Wipe samples were taken from both floors and carpets (wet and dry wipes) in living rooms, bedrooms, kitchen, and hallways. Although these higher-contact areas averaged 1.2 μg MP/100 cm$^2$, 32 times less than the average samples taken from baseboards, they likely represented a dermal exposure route as reported in other OP studies (*37,38*). Clothing likely represented another major exposure route (*35*). Most carpet wipe samples had nondetectable levels of MP (<1 μg/100 cm$^2$). Two carpets from L-2 properties were evaluated for potential bioavailability data via wipe samples versus the total amount of MP in the carpet. Wet wipes of carpeting from the center of the rooms and near walls revealed MP concentrations ranging from nondetectable to a maximum of 4 μg/100 cm$^2$, with a mean of 0.75 μg/100 cm$^2$. Total amount of MP in the total carpet samples were considerably higher, ranging from nondetectable to 169 μg/100 cm$^2$, with a mean of 76 μg/100 cm$^2$. The highest levels were found near walls. The data clearly indicated that carpets were significant MP sinks. The removal of all carpeting and decontamination of surfaces in L-1 and L-2 homes ensured that future MP exposures would be minimized. Because the pesticide levels were very low and near the detection limit on these higher-contact surfaces, an improved exposure protocol was not developed.

The amount of exposure via ingestion of dust was evaluated in four L-3 homes that had MP baseboard concentrations averaging 30 μg/100 cm$^2$. Dust from the houses collected from vacuum cleaners averaged 3.5 mg MP/kg. Assuming 1,000 mg of soil/dust intake (90th percentile) per day, the MP dose would have been about 3.5 μg of MP/day (*39*). Urine concentrations of PNP in children averaged 86 ppb, unadjusted for creatinine, yielding a estimated dose of 0.27 mg MP/day using urine production rates per body weight and the equation from Morgan et al. (*32*). Based on this analysis, oral exposure to MP in dust constituted about 1% of that observed from urine. Although relatively high detection limits were applied, these findings are consistent with the NIOSH study conducted in December 1994 on 30 children 18 months or younger (*40*). An entire hand (front, back, between fingers) was washed with alcohol on gauze. At a limit of detection of 8–30 μg, 29 wipe samples were negative and one was positive at 10 μg.

***Environmental PNP.*** MP is known to break down in soils, with PNP being a potential transitory intermediate product (*41*). Other information has been reported that MP may be biodegraded directly to carbon dioxide or a volatile compound (*42*). Human exposure to environmental PNP results in urinary excretion of PNP that would be indistinguishable from metabolically derived PNP resulting from MP exposure. Six L-3 houses in Ohio were evaluated to assess MP and PNP environmental levels. If the environmental PNP average concentrations were found to be low relative to MP or found only in isolated areas of the households, then there would be no change

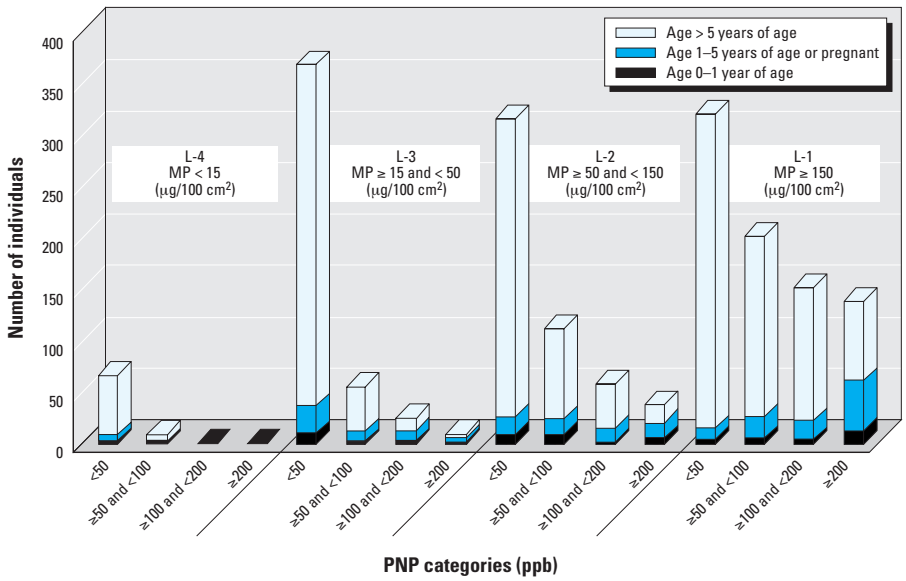


**Figure 6.** Number of individuals by PNP, MP mean surface level, and age group.

**Table 2.** Variation in PNP urine levels.[a]

| Age (years) | PNP range (ppb) | Range (multiplier) |
|---|---|---|
| 6 | 63–120 | 1.9 |
| 9 | 26–34 | 1.3 |
| 8 | 38–95 | 2.5 |
| 7 | 38–68 | 1.8 |
| 11 | 40–130 | 3.2 |
| 6 | 130–254 | 1.9 |
| 5 | 120–240 | 2.0 |
| 8 | 81–120 | 1.5 |

[a]Based on four samples per child, creatinine adjusted, from L-3 residences (≤50 μg/100 cm$^2$).

in the protocol. If the environmental PNP were found to be significant or if it were found throughout the residence, then an approach would be developed to correct the urinary PNP values based on the ratio of environmental PNP to MP and comparative toxicities of PNP and MP.

L-3 Ohio homes were tested for MP and PNP by obtaining seven side-by-side baseboard samples from each home. Dust samples were also collected from vacuum cleaners and analyzed for MP and PNP. All samples from all homes had detectable levels of MP (Table 3). Two homes had no detectable environmental PNP, whereas two had detectable levels in only a single location (under the sink, bathroom). The locations where PNP was found are those where water or ammonia-based cleaning agents are most likely to be applied. Both water and particularly bases hydrolyze the DMP group of MP, resulting in the PNP derivative. One house had a detectable level of PNP only in the living room. One house had detectable PNP levels by the sink, under the sink, and in a bathroom. Dust samples had no detectable levels of PNP but did have MP. Although the average of environmental PNP greatly exceeded MP in one house and was about equal in another, the areas affected with PNP were small compared with the total property area. Also, no PNP was found in bedrooms of any home, where exposures were likely to occur. Because PNP environmental levels were generally low and in small areas in Ohio properties, no changes in the protocol were made.

***Decontamination results.*** In Ohio, 233 properties were decontaminated. It took about 6 weeks for the entire process of relocation, decontamination, restoration, and reoccupancy to take place. Properties being decontaminated without removal of baseboards and drywall had resultant MP surface concentrations averaging 21 μg/100 $cm^2$ and air levels of 0.9 μg/$m^3$. These surface concentrations were about 5-fold lower than concentrations before decontamination, whereas air levels were about 8-fold lower than initial MP concentrations. Postdecontamination results in homes where contaminated baseboards and drywall were removed had average surface levels of 3 μg MP/100 $cm^2$ and average air levels of 0.7 μg/$m^3$. These MP surface and air concentrations were, respectively, approximately 35 and 10 times less than initial surface and air concentrations. To ensure that the decontamination process was completely effective, eight properties that had among the highest MP concentrations found before decontamination were selected for short-term and longer-term monitoring. Wipe and air samples showed no significant differences in MP concentrations over time, which is likely related to concentrations being near the detection limit. MP surface concentrations averaged 1.2, 2.0, and 2.0 μg/100 $cm^2$ and MP air levels averaged 0.7, 0.7, and 0.6 μg/$m^3$, respectively, at 7, 30, and 90 days after restoration. Urine tests from 34 persons from 12 L-1 decontaminated residences revealed very low PNP exposures. Twenty individuals, mostly young children, had nondetectable (<25 ppb) PNP urine levels. Ten individuals had detectable urine levels averaging 44 ppb PNP (creatinine adjusted), with a maximum value of 110 ppb, slightly over the 100 ppb L-3 criterion for children 1–5 years of age. Laboratory data were unavailable on four individuals. The single high urine value was linked to a child's mattress that had not been removed during decontamination.

***Evaluation of the protocol used at Lorain, Ohio.*** After completion of the Ohio MP investigation and decontamination of 233 residences, the entire MP and urine PNP database was examined to assess the outcomes resulting from the decision-making protocol. Of particular interest was how well the process, using rough correlations on initially collected data, successfully categorized exposure groups. For example, a correct positive decision would have been one where mean surface MP concentrations were ≥ 50 μg MP/100 $cm^2$ and PNP levels were ≥ 50 ppb in infants younger than 1 year. All individuals in residences having concentrations greater than these criteria would be subject to relocation and their properties would be decontaminated. A false negative was characterized as residences with surface levels < 50 μg MP/100 $cm^2$ but with urine PNP > 50 ppb in infants. Results by age group and MP surface concentrations are presented in Figure 7.

Categorizations were correct for 72% of infants and 64% of children 1–5 years of age and pregnant women, whereas false negatives were 9 and 7% and false positives were 9 and 28%, respectively, for these two groups. With the >5 year age group, correct categorization fell to 30% and false positives increased to 65%; false negatives were less than 1%.

On a household basis the correct categorization was made 54% of the time, with 46% false positives (Figure 8). Therefore, almost half the properties did not have individuals with PNP exposures that exceeded their PNP criteria based on average PNP levels from two urine samples. About 1% of the properties had average MP surface levels below 50 μg MP/100 $cm^2$ L-3 criterion (no-action) but had individuals with PNP urine levels above L-2 (decontamination) criteria. A portion of the false

**Table 3.** MP and PNP in homes.

| Home | MP positive[a] | Mean MP[b] | PNP positive[a,c] | Mean PNP[b] |
|---|---|---|---|---|
| 1 | 6/6 | 63.7 | 0/6 | <1 |
| 2 | 6/6 | 49.9 | 0/6 | <1 |
| 3 | 6/6 | 42.9 | 1/6 (s) | 16 |
| 4 | 6/6 | 18.8 | 1/6 (us) | 18 |
| 5 | 6/6 | 11.7 | 1/6 (l) | 3.8 |
| 6 | 6/6 | 35.4 | 3/6 (s, us, l) | 77.9 |

[a]Number of samples positive out of total. [b]Values in μg/100 $cm^2$. [c]s = sink, us = under sink, l = living room.

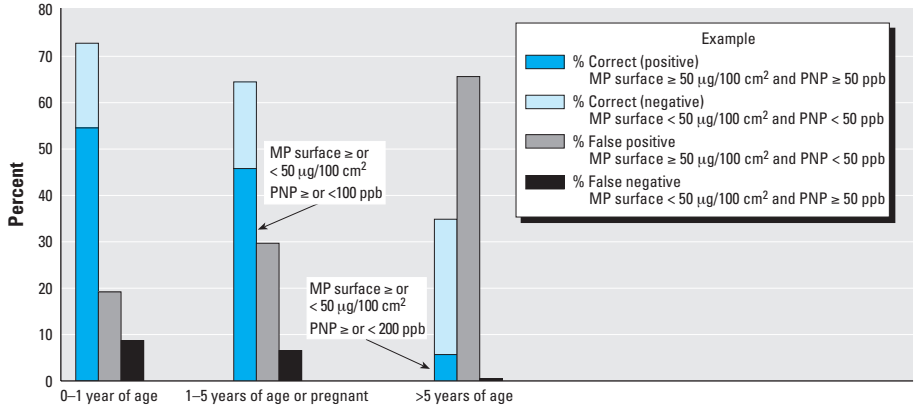


**Figure 7.** Decision outcome by MP surface level, PNP, and age group.

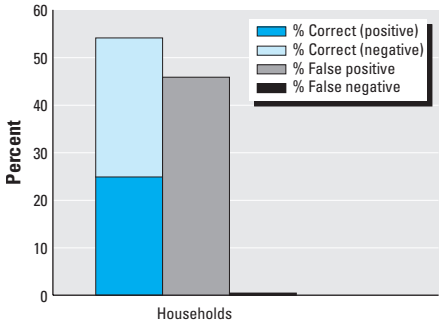


**Figure 8.** Decision outcome by household (by MP surface level, PNP, and age group).

positive results in households may have been due to variation in spot urine samples. A study of L-3 children had found that spot urine PNP levels could vary by about 2-fold (Table 2).

Regressions of all available PNP data versus mean surface MP levels demonstrated variation in the data and were different from those performed before most data were collected (Figure 9). However, the regressions of PNP to MP were statistically significant ($p < 0.0001$) for the 1- to 5-year and >5-year age groups and also provided support for selecting 50 µg/100 cm$^2$ as a relocation criterion in Ohio. For the 1- to 5-year age group, the resultant linear regression equation was PNP = 0.34 (MP) + 101.8, with a correlation of $r = 0.52$. The intercept was also significant ($p < 0.0001$). Forcing the line through the intercept improved the linear relationship ($r = 0.66$) and could be justified by the extremely low levels of PNP in persons not exposed to MP. However, the linear equation with the statistically significant intercept also had better residual analysis results and had a lower root mean square error.

Correlations were weaker ($r = 0.16$) for infants and for individuals older than 5 years ($r = 0.38$, $p < 0.0001$). Weaker correlations would be expected for persons 5 years of age and older because of increased time spent outside households. As in the case of the 1- to 5-year age group, the intercept for the >5-year age group was statistically significant ($p < 0.0001$). Because the positive intercepts (1- to 5-year and >5-year age groups) were unlikely due to chance, it suggests that there were other sources of MP exposure. Surface samples from baseboards and under the sink were only indicators of potential exposure. Higher-contact surfaces such as carpets, clothing, linens, and furniture were also found to be contaminated with MP, probably due to MP volatilization, and they would be expected to be a dominant exposure route. Inhalation and oral (e.g., dishes or food) MP exposures also would have occurred.

***Development of a national methyl parathion protocol.*** After MP was found in residential and commercial properties in Pascagoula, Mississippi, and soon after in Louisiana, Alabama, and Tennessee, concerns were raised about whether the Lorain, Ohio, protocol, which was based on an initial evaluation of the relationship between environmental MP and urine PNP in northern properties treated by one sprayer, would be fully appropriate in a situation involving southern properties where multiple applicators were involved. In the South, homes generally were not as extensively sprayed as they were in Ohio. In addition, only weak correlations were found in Ohio.

A Methyl Parathion Health Sciences Steering Committee, consisting of state and federal health officials, was established by the U.S. EPA Office of Emergency and Remedial Response to evaluate these more recent incidents and determine if the Lorain, Ohio, protocol should be modified. Scientists who had developed the Lorain, Ohio protocol also participated. Toxicological literature and the usefulness of using urine PNP as an exclusive basis for relocation or in conjunction with environmental MP was extensively re-reviewed. In contrast to the early findings in Ohio, ATSDR found virtually no correlations with environmental MP and urine MP in Louisiana (*43*). Therefore, in the absence of reasonable correlations, the committee recommended that urine PNP and not environmental MP should drive future relocation decision making. The committee also concluded that urine-based PNP relocation criteria for adults and older children could be raised without significantly increasing health risks. These PNP relocation levels more closely matched L-1 emergency relocation values used in Ohio but were lowered to 50 ppb PNP to provide greater protection of the fetuses for pregnant women exposed to MP. Infants younger than 1 year were also protected at 50 ppb PNP. The enhanced level of protection for fetuses and young infants was based on information indicating that their MP metabolism would be more restricted (*44–46*). The ATSDR developed an improved questionnaire that obtained more information on the amount of time each individual spent in the home and their activities 24 hr before urine collection. More emphasis was placed on environmental sample collection from areas of higher contact. For instance, under the kitchen sink samples were eliminated. As in the Lorain, Ohio protocol, the committee concluded that cleanup levels derived from chronic RfDs were not needed because MP levels declined to nondetectable levels within a few years. After an external peer review process overseen by the ATSDR (*44,45*), a national MP protocol was finalized (*47*) (Table 4). Overall, nearly 7,000 properties were sampled during the 1994–1997 MP investigation and about 1,000 residences were decontaminated (Table 5) (*2*). The pesticide misuse investigation and decontamination effort was the largest project of its type ever conducted by federal and state agencies.

## Discussion

The risk assessment–risk management approach used in Ohio, although based on crude regressions using limited data collected early in the assessment process, yielded an appropriate degree of protection for infants younger than 1 year. Assuming one infant per household, about 90% (correct plus false positives) of the residences having infants younger than 1 year would be considered safe for occupancy using only the surface MP criterion. Infants born into homes or new families with infants moving into properties having less than 50 µg MP/100 cm$^2$ would be protected. Such approaches and outcomes are consistent with risk assessment and risk management approaches under Superfund procedures, which apply risk characterizations and decontamination criteria to protect the most vulnerable populations, often at 95th exposure percentiles (*18*). For instance, in the case of lead in residential soils, decontaminated targets

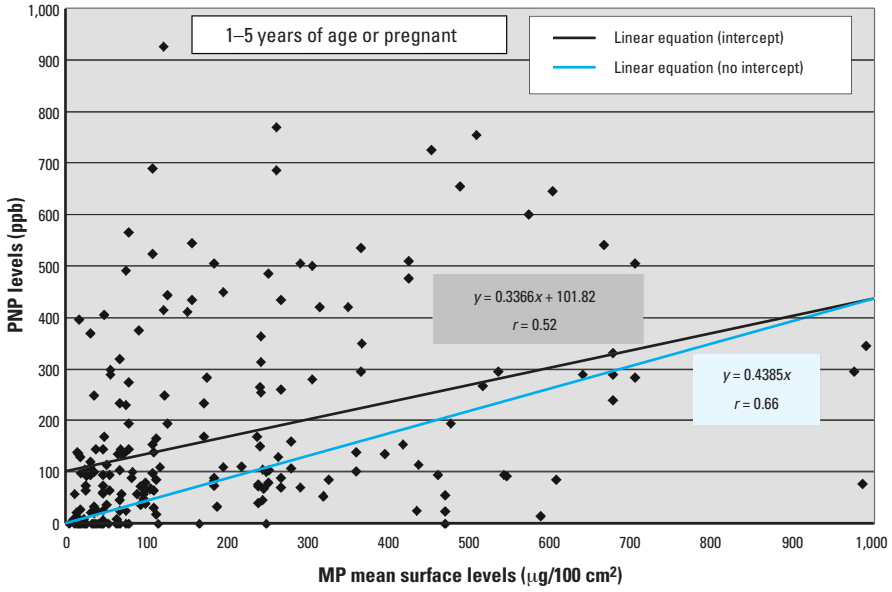


**Figure 9.** Regression of PNP in urine versus mean surface MP.

are established such that there is only a 5% chance that a young child will exceed the lead blood level health criterion of 10 µg/dL (*48*).

Modifications in the Ohio protocol were necessary when MP exposures in Mississippi were found to be considerably lower than those in Ohio. Differences in housing, climate, and spraying practices were believed responsible for these differences. By placing emphasis exclusively on actual exposures as measured by urine PNP, there were fewer relocations. The quarterly monitoring of residents who were not relocated provided a protective approach, although there were limitations. For example, because MP residues could last several years and urine monitoring was required for only a year, PNP levels could be above the 50 ppb criterion for women who became pregnant or infants born into households after a year. However, it is highly unlikely that persons were at significant risk because exposure to environmental PNP may have been occurring.

In Chicago, Illinois, testing of 13 properties found mean surface levels of PNP almost 3 times the MP levels (*49*). In contrast to the Illinois findings, tests on Ohio properties found PNP levels averaging 70% of MP levels. For Chicago properties, there appeared to be no clear relationship between the ratio of PNP to MP and the length of time (11–66 months) after spraying or the concentration of MP. Unlike the findings in Lorain, Ohio, every living area in every Chicago home tested was positive for PNP.

The Chicago results, which came late in the national assessment process, strongly suggest that urine PNP levels were based on exposures to both MP and environmental PNP. Although this raises doubts about urine PNP as an exposure criterion, the approach may have been appropriate. First, urine PNP did provide a measure of total exposure to two toxic chemicals, MP and PNP. PNP causes increases in methemoglobin and death in rats at doses of 70 mg/kg body weight per day (*50*). PNP also is a respiratory, eye, and skin irritant. Although the oral NOAEL (25 mg/kg body weight per day) for PNP is 100 times higher than that reported from MP subchronic studies, it has a relatively low RfD (0.008 mg PNP/kg body weight per day) because of uncertainties in its toxicology (*50*).

Second, there is evidence that dermal exposure to environmental PNP could enhance MP absorption, as is the case for EP (*43*). Finally, although definitive dermal studies need to be conducted, as recommended by the ATSDR expert panel, in the absence of such data it would not be an inappropriate risk management approach to assume that all urine PNP has been derived from MP exposure. However, the Chicago findings of extensive environmental PNP in properties demonstrate the need to better understand environmental PNP formation and to develop an assessment approach that is based on either *a*) ratios of environmental PNP and MP levels and toxicities or *b*) methods that could differentiate between exposures resulting from environmental MP and PNP. Urine testing of methyl phosphates, a derivative of MP metabolism, could provide an approach to distinguish between metabolic PNP and environmental PNP. More sophisticated exposure approaches are also needed that better determine or model dermal exposures to MP and other OPs (*51*). The MP exposure studies in Chicago applied a more refined approach using composite surface samples from high-contact areas such as floors (*48*).

**Table 4.** National creatinine protocol.[a,b]

| Age group | Recommended action | | |
|---|---|---|---|
| | No further action | Urine monitoring | Relocation |
| 0 < 1 year of age and pregnant women | <25 ppb in urine and <50 µg/100 cm$^2$ on surfaces | 25–50 ppb in urine or <25 ppb in urine and ≥50 µg/100 cm$^2$ on surfaces | >50 ppb in urine |
| ≥1 year < 16 years of age | <100 ppb in urine and <50 µg/100 cm$^2$ on surfaces | 100–300 ppb in urine or <100 ppb in urine and ≥50 µg/100 cm$^2$ on surfaces | >300 ppb in urine |
| ≥16 years of age | <300 ppb in urine and <50 µg/100 cm$^2$ on surfaces | 300–600 ppb in urine and <300 ppb in urine and ≥50 µg/100 cm$^2$ on surfaces | >600 ppb in urine |

[a]Data from the U.S. EPA (*2,47*). [b]Urine values are creatinine adjusted. Surface levels are based on averages. The higher of two (A.M. and P.M.) urine samples was used for decisions. Infants were monitored quarterly until reaching 1 year of age. Other groups were monitored for at least 1 year.

**Table 5.** Number of residences sampled and decontaminated.[a]

| | State | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | OH | MI | IL | MS | LA | TN | AR | Total |
| Residences sampled | 728 | 140 | 897 | 2,655 | 2,050 | 396 | 58 | 6,924 |
| Residences decontaminated | 233 | 4 | 110 | 452 | 201 | 6 | 8 | 1,014 |

Data from the U.S. EPA (*2*).

In response to the extensive illegal use of MP, the U.S. EPA required modifications in the registration of MP products and initiated a residential pesticide control program (*52,53*). Specifically, the U.S. EPA required Cheminova, the sole manufacturer of MP, to *a*) use no more than 5 pounds of MP per gallon; *b*) have returnable, refillable containers that have tamper-resistant openings and are designed for use only with agricultural spray equipment; *c*) include a bar coding system to track the sale and disposal of containers; and *d*) add a stenching agent in the formulation that would alert persons if the product were used inside properties (*52*). In 1999, because of MP residues in some foods and potential risks, the U.S. EPA canceled MP use on crops such as apples, grapes, carrots, lettuce, and tomatoes (*53*). Uses on nonfood crops such as ornamentals and nursery stock were also eliminated. To help prevent future misuse, training of pesticide applicators and alerts to state inspectors and retailers of MP products were initiated (*52*). Those responsible for the MP spraying in Ohio, Illinois, and Mississippi were convicted of felonies, and some users served time in jail. However, solving the problem of illegal pesticide use will clearly require constant vigilance and aggressive enforcement.

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