Methyl parathion (MP), an organophosphate pesticide licensed only for agricultural uses, was sprayed illegally for pest control in Mississippi and Ohio residences. To evaluate the association between MP exposure and neurobehavioral development, we assessed children 6 years or younger at the time of the spraying and local comparison groups of unexposed children using the Pediatric Environmental Neurobehavioral Test Battery (PENTB). The PENTB is composed of informant-based procedures (parent interview and questionnaires) and performance-based procedures (neurobehavioral tests for children 4 years or older) that evaluate cognitive, motor, sensory, and affect domains essential to neurobehavioral assessment. Children were classified as exposed or unexposed on the basis of urinary para-nitrophenol levels and environmental wipe samples for MP.Exposed children had more difficulties with tasks involving short-term memory and attention. Additionally, parents of exposed children reported that their children had more behavioral and motor skill problems than did parents of unexposed children. However, these effects were not consistently seen at both sites. There were no differences between exposed and unexposed children in tests for general intelligence, the integration of visual and motor skills, and multistep processing. Our findings suggest that MP might be associated with subtle changes to short-term memory and attention and contribute to problems with motor skills and some behaviors, but the results of the study are not conclusive.

Key words: children’s health, methyl parathion, neurobehavioral development, neurologic functioning, organophosphate pesticide. Environ Health Perspect 112:46–51 (2004). doi:10.1289/ehp.6430 available via http://dx.doi.org/[Online 25 September 2003]
2-year period ending in 1996 (Anonymous 1997a); in Ohio, the spraying occurred from January 1991 through November 1994 (Esteban et al. 1996). A study conducted in Lorain County, Ohio, showed that PNP was still present in the urine of some people whose homes were sprayed more than a year before the sample was collected (Esteban et al. 1996). The fact that PNP was detected in the urine after this length of time may indicate continual exposure via MP-contaminated households or personal items (Esteban et al. 1996).

Animal studies have shown that exposure to organophosphate pesticides affects neurologic functioning in developing rats by altering behavior and producing slight changes in learning ability (ATSDR 1999; Eskenazi et al. 1999). A study of 90 pesticide applicators suggested that organophosphate exposure was associated with a loss of peripheral nerve function (Stokes et al. 1995). Another cohort of pesticide applicators acutely exposed to organophosphates several years before testing were more likely to have mood changes and difficulties with memory and motor reflexes than was an unexposed comparison population (Savage et al. 1988). Agricultural workers in Nicaragua who were tested 2 years after exposure to organophosphate pesticides performed worse on tests that measured verbal and visual attention, visual memory, visuomotor speed, sequencing and problem solving, and motor steadiness and dexterity than did an unexposed comparison population (Rosenstock et al. 1991). A 1994 study examined the chronic effects of acute organophosphate poisoning using California surveillance data and found that exposed men performed significantly worse on tests that measured sustained visual attention and mood scales (Steenland et al. 1994). A study of sheep farmers with long-term exposure to organophosphates found subtle peripheral neuropathy (Beach et al. 1994). Fifty-two male workers occupationally exposed to organophosphate pesticides for at least 3 years were compared with 50 unexposed male controls; the pesticide applicators performed worse on tests that measured visuomotor speed, verbal abstraction, attention, and memory (Farahat et al. 2003). These studies suggest evidence of neurologic deficits in workers who were occupationally exposed to organophosphate pesticides.

Children may be more likely to be exposed to MP because crawling and play activities put them close to the ground, where they have increased chances of exposure to contaminated surfaces such as baseboards (ATSDR 1999; Bearer 1995; Eskenazi et al. 1999; Guzelian et al. 1992; Landrigan and Carlson 1995). Additionally, children may be more susceptible to health effects from MP exposure because of their developing organ systems (ATSDR 1999; Eskenazi et al. 1999; Kolb and Fantie 1997). No studies have examined the neurobehavioral effects of MP exposure in children.

Because children have unique characteristics that often place them at greater risk of adverse health effects when exposed to hazardous chemicals, this study examined the neurobehavioral development in children subacutely exposed to MP in doses lower than those seen in occupationally exposed workers.

**Materials and Methods**

**Study population.** Potential participants were identified using data files provided to the ATSDR by the Mississippi and Ohio state health departments. These states were selected because environmental data collection and urine testing were available. Children residing in Mississippi and Ohio who were 6 years or younger when their homes were sprayed with MP were eligible for inclusion in the study. In one Ohio county, the spraying occurred in a multifamily, subsidized housing facility that was last sprayed in 1994. In Mississippi, the spraying was more widespread and included 29 counties with residences sprayed as recently as late 1996.

Results of environmental wipe samples for MP taken from residences (household MP) and urine testing for creatinine-adjusted PNP, a metabolite of MP, were provided by the state health departments. The samples were collected for residents in areas known to be illegally sprayed with MP. Household MP samples were analyzed by laboratories approved by the U.S. EPA employing the same analytical methods, and PNP samples were analyzed by the CDC. Testing was done in Ohio in 1994 and in Mississippi from late 1996 through mid-1997. Exposure status was defined on the basis of the test results. Both household MP and urinary PNP levels were used to define exposure status.

In Mississippi, exposure was defined as at least one household MP sample ≥ 150 µg/100 cm² or urinary PNP ≥ 100 ppb for at least one person in the household. For Ohio, exposure was defined as household MP ≥ 132.9 µg/100 cm² or urinary PNP ≥ 100 ppb for at least one person in the household. To include enough exposed children in Ohio, it was necessary to set a lower cutoff value for household MP.

Comparison groups of unexposed children residing in the same communities as the exposed children were also identified. Local comparison groups were chosen to minimize confounding from sociocultural factors (e.g., regional variations in education, IQ, race, and cultural factors).

In Mississippi, unexposed children were selected through state records from houses that tested < 25 µg/100 cm² for household MP; no urine testing was done for children at these levels of MP. In Ohio, unexposed children were selected in two ways. State records were reviewed from houses that tested < 35 µg/100 cm² for household MP and where no one in the household had a urinary PNP level > 25 ppb. The cutoff value for household MP in Ohio was increased to include enough unexposed children. Because an insufficient number of unexposed children were identified through existing records, a special census was done in the sprayed complex after it was remediated and in a nearby housing complex that was not sprayed, to identify additional unexposed children.

Children whose household MP levels were between the lower and upper cutoff values for a particular site and who did not have at least one person in the household with a urinary PNP level ≥ 100 ppb were not invited to participate in the study. For example, a child in Mississippi with a household MP level of 90 µg/100 cm² and where no one in the household had a urinary PNP level ≥ 100 ppb would not be invited to participate in the study because their household MP level was not < 25 µg/100 cm² or ≥ 150 µg/100 cm² and the urinary PNP requirement was not met.

We identified 365 children in Mississippi (147 exposed and 218 unexposed) and 287 children in Ohio (104 exposed and 183 unexposed). We attempted to enroll two unexposed children for every exposed child to ensure an adequate number of unexposed children for analysis in subsequent study years. All children who participated in year 1 (summer 1999) were invited to be retested in year 2 (summer 2000) to see whether any differences in neurobehavioral testing between exposed and unexposed children persisted or disappeared.

**Data collection.** Parents/guardians of eligible children invited to take part in the study were contacted initially by letter, which was followed up with a telephone call. All parents/guardians gave written informed consent for their child to be in the study. Children 7 years and older provided assent for their participation in the study. All testing protocols were approved by the CDC’s institutional review board.

A computer-assisted personal interview was administered by trained interviewers to the parent/guardian to obtain information on potential confounders. The interview collected information on demographic and personal characteristics such as parents’ and child’s medical history, mother’s pregnancy history with regard to the index child, parental occupational histories, workplace chemical use, and child’s residential history.

The Pediatric Environmental Neurobehavioral Test Battery (PENTB) was used to assess the neurobehavioral functioning of the children (Amherl and Gibertini 1996). The PENTB consists of performance-based and informant-based tests, as described below.
**Performance-based tests.** The Developmental Test of Visual-Motor Integration (VMI) measures the integration of visual and motor skills. The Kaufman Brief Intelligence test (K-BIT) measures general intelligence, verbal ability, and nonverbal reasoning. The Purdue Pegboard tests visual-motor coordination, manual dexterity, and motor speed. The Story Memory and Story Memory-Delay from Wide Range Assessment of Memory and Learning tests verbal memory; immediate and delayed recall of both specific and general items are assessed. The Trail-Making test, Part A and Part B, assesses multistep processing involving more than one cognitive function area (visual perception, motor speed, sequential skills, and symbol recognition) and is administered to children 9 years and older. The Verbal Cancellation test measures sustained selective attention. The Visual Cancellation test measures sustained selective attention.

**Informant-based tests.** The Parenting Stress Index (PSI) estimates the occurrence of common signs and symptoms of child and family dysfunction; this test yields a child domain score composed of six subscales (adaptability, acceptability, demandingness, mood, distractibility/hyperactivity, and reinforces parent), a parent domain score composed of seven subscales (depression, attachment, restrictions of role, sense of competence, social isolation, relationship with spouse, and parent health) and a total stress score, and was completed by parents/guardians of children 1–3 years of age. The Personality Inventory for Children (PIC) assesses the child’s behavior, affect, and cognitive status. This test yields four factor scores (undisciplined/poor self-control, social incompetence, internalization/somatic symptoms, cognitive development) and is completed by the parent or guardian of children 4 years or older. The Vineland Adaptive Behavior Scales (VABS) measure communication; daily living skills such as eating and dressing, household tasks, and time and money skills; socialization; and motor skills.

Detailed information about the PENTB, including specific PENTB tests and scoring, is presented elsewhere (Zeitz et al. 2002). For the individual PENTB tests, both raw scores and age-scaled scores (where appropriate) were computed using the appropriate scoring manuals. Children were also assigned to one of four overall PENTB outcome groups (expected, equivocal, below expected, or undetermined) on the basis of the number of completed tests and their scores on the individual tests. Children classified as expected scored in the average range or better on most tests, with only one or two test scores below average; children classified as equivocal scored average or better for some tests but below average on three or four tests or well below average on one or two tests, and showed no pattern or consistency; children classified as below expected scored below average on five or more tests, well below average on three tests, or in the lower extreme on two tests; and children classified as undetermined completed too few tests.

**Data analysis.** We redefined the initial exposure status to identify a more highly exposed group of children. High exposure was defined as household MP ≥ 1,000 µg/100 cm² or a urinary PNP level ≥ 300 µg/l. Exposed children not meeting this requirement were assigned to the moderate exposure group. The unexposed group remained unchanged. Analyses were computed for both the initial exposure status and the redefined exposure status. We analyzed continuous test scores using linear regression. A higher mean difference score between exposed and unexposed children indicates better performance for the following tests: K-BIT, Story Memory and Story Memory Delay, VMI, Purdue Pegboard, Verbal Cancellation, and VABS. A lower mean difference score indicates better performance for the Story Memory Difference, Trail Making, PSI, and PIC.

We computed unadjusted and adjusted results and calculated 90% confidence intervals (CIs) for the parameter estimates. In the unadjusted models, we adjusted all raw (non-standardized) test scores for age to make the scores comparable. For each PENTB test, potential confounders were entered individually in the regression model with the exposure status. Variables that contributed to a change in the parameter estimate of the exposure status of 10% or more were included in the final adjusted model. Income (< $20,000/year compared with ≥ $20,000/year) and race (white, black, other) were included in the final adjusted model regardless of their effect on the parameter estimate of the exposure variable. Other variables adjusted for in the final model were ethnicity (Hispanic or Latino compared with non-Hispanic or non-Latino); mother’s use of chemicals at work; mother had one or more of the following conditions: diabetes or epilepsy/seizures before pregnancy, hospitalized or confined to bed during pregnancy, fever, X rays, or vaginal bleeding during pregnancy; and parent reported that a doctor told them their child had lead or mercury poisoning. A site term was included in the models. Adjusted results are presented only if the adjusted and unadjusted results differed by > 10%.

Test scores were also dichotomized to compare those children who scored in the worst 10% for a test with those children who scored in the other 90%. Dichotomized test scores were analyzed using logistic regression. Raw (nonstandardized) test scores were adjusted for age. Unadjusted odds ratios (ORs) and 90% CIs were computed. Secondary analyses were conducted for each outcome group to identify a more highly exposed group of children. High exposure was defined as household MP ≥ 1,000 µg/100 cm² or a urinary PNP level ≥ 300 µg/l. Exposed children not meeting this requirement were assigned to the moderate exposure group. The unexposed group remained unchanged. Analyses were computed for both the initial exposure status and the redefined exposure status. We analyzed continuous test scores using linear regression. A higher mean difference score between exposed and unexposed children indicates better performance for the following tests: K-BIT, Story Memory and Story Memory Delay, VMI, Purdue Pegboard, Verbal Cancellation, and VABS. A lower mean difference score indicates better performance for the Story Memory Difference, Trail Making, PSI, and PIC.

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in year 1 did not improve their classification from year 1 to year 2, compared with 60% of the unexposed children. Additionally, 61% of the exposed children who were classified as equivocal in year 1 did not improve their classification from year 1 to year 2, compared with 75% of the unexposed children. These results indicated that MP exposure was not associated with persistent deficits in year 2 among children who performed lower than expected in year 1. Overall PENTB outcome group results for years 1 and 2 were not compared for children whose parent/guardian completed informant-based tests in both years of the study because of small numbers.

Performance-based tests. No effects were seen for the K-BIT, VMI, or Trail-Making tests (ATSDR 2003). These tests measure general intelligence, the integration of visual and motor skills, and multistep processing. Table 4 presents the results of performance-based tests where exposed children performed worse than unexposed children in year 1.

For the Verbal Cancellation test, an effect was seen for the mean difference only in Ohio (highly exposed ordered form adjusted \( \beta = -0.02, 90\% \ CI, -1.12 \text{ to } -0.78\); highly exposed nonordered form adjusted \( \beta = -0.09, 90\% \ CI, -1.13 \text{ to } -0.78\)), and there was a trend with exposure. When test scores were dichotomized, an effect was seen only in Mississippi (ordered form \( OR = 2.27, 90\% \ CI, 0.52 \text{ to } 6.25\)). In Ohio, an effect was seen for PIC factor 1 when scores were dichotomized and for PIC factor 3, internalization/somatic symptoms (Table 5). A trend with exposure was present for the mean difference scores for PIC factor 3.

Discussion
Exposure to MP was not associated with poorer performance on most neurobehavioral tests, and effects were not consistently seen in both sites. Effects were seen for tasks that involve short-term memory and attention, and parents of exposed children reported that their children had more behavioral and motor skills problems than did parents of unexposed children. The reported behavioral problems included children misbehaving, acting on impulse, having problems with anger, being sad or shy, and having problems relating to other children. A comparison of overall PENTB outcome group scores indicated that MP exposure was not associated with persistent deficits in year 2 among children who performed lower than expected in year 1. This suggests that if there are neurobehavioral effects from subacute exposure to low levels of MP, they may be transient in children who were 6 years or younger when they were exposed. A previous study found major impairment of memory and concentration in workers who had exposures to various

### Table 1. Number of eligible and tested participants in year 1.

|                     | Exposed [No. (%)] | Unexposed [No. (%)] | Total [No. (%)] |
|---------------------|------------------|---------------------|----------------|
| Potential participants | 251 (100)        | 401 (100)           | 652 (100)      |
| Ineligible           | 77 (29.6)        | 141 (34.2)          | 218 (33.8)     |
| Eligible             | 174 (69.0)       | 26 (6.4)            | 200 (30.9)     |
| Refused              | 6 (2.4)          | 21 (5.2)            | 27 (4.1)       |
| Scheduled for testing | 168 (67.0)      | 219 (54.6)          | 387 (59.3)     |
| Completed or partially completed testing | 132 (75.9) | 147 (56.5) | 279 (64.5) |

### Table 2. Comparison of available demographic characteristics of participating and nonparticipating children who were scheduled for testing in year 1.

| Participant | Nonparticipant | \( \chi^2 \) |
|-------------|----------------|-------------|
| Exposed     | 132 (47.3)     | 36 (33.3)   |
| Unexposed   | 147 (52.7)     | 72 (66.7)   |
| Mean age in years at testing | 6.1 (2.5–11.5) | 6.0 (1.9–12.5) |
| Sex [n (%)] | 149 (63.4)     | 48 (48.0)   |
| Female      | 130 (46.6)     | 52 (52.0)   |
| Site [n (%)] |                |             |
| Mississippi | 179 (64.2)     | 81 (75.0)   |
| Ohio        | 100 (35.8)     | 27 (25.0)   |
| Total       | 279 (100)      | 108 (100)   |

*Age information missing for seven nonparticipating children in Mississippi. Sex information missing for eight nonparticipating children in Mississippi.

### Table 3. Overall PENTB outcome group for children who participated in year 1.

| Overall PENTB outcome group | Exposed [No. (%)] | Unexposed [No. (%)] | Total [No. (%)] |
|-----------------------------|------------------|---------------------|----------------|
| Expected                    | 72 (54.5)        | 80 (40.8)           | 152 (47.3)     |
| Equivocal                   | 41 (31.1)        | 54 (36.7)           | 95 (28.4)      |
| Below expected              | 16 (10.6)        | 15 (10.2)           | 31 (9.4)       |
| Undetermined                | 5 (3.8)          | 18 (12.2)           | 23 (7.0)       |
| Totala                      | 132 (100)        | 147 (99.9)          | 279 (100)      |

*Expected, scored in the average range or better on most tests; equivocal, scored in the average range for some tests and below average on some tests; below expected, scored below average on most tests; undetermined, did not complete enough tests to score. \( \text{aPercentages may not total } 100\% \text{ due to rounding.} \)
organophosphates for several years, but the impairments disappeared 12 months after exposure ceased (ATSDR 1999). In this study, the time between the last spraying of the pesticide and the testing was at least 2.5 years in Mississippi and at least 4.5 years in Ohio. Incoordination is a common neurologic effect of severe MP poisoning, and slowed motor processes have been associated with acute mild exposure to MP (ATSDR 1999).

Inconsistencies in the results between Mississippi and Ohio may be due to the differences in length of time between the spraying and testing in the two sites (the last spraying in Mississippi was in late 1996 and the last spraying in Ohio was in 1994; children in both sites were initially tested in 1999). Additionally, children in Ohio were older than children in Mississippi at the time of testing, and the older children may have outgrown any neurobehavioral effects that were caused by MP. Also, the exposure assessment of MP in household samples and in urine for each child represents only a snapshot in time and may not reflect the total exposure received by the child. There were also inconsistencies in results within subtests of some tests. Additionally, there was a lack of trend with exposure for most tests.

Limitations of this study include the fact that the length of time between the last MP spraying, sample collection, and neurobehavioral testing was not known and was different in the two study sites. The frequency and duration of spraying were also unknown. Results for a particular test could be lacking because the neurobehavioral effects are transient and were no longer measurable when the children were tested. It might also be possible that the PENTB was not the appropriate test battery to examine neurobehavioral effects from exposure to MP. For example, with a large sample size, the standard errors for several tests were quite large, making it difficult to find subtle effects.

Strengths of this study include the use of environmental wipe samples and urine testing to quantify the children’s individual exposure to MP. The participation rate in year 1 was 64% (76% for exposed children and 57% for unexposed children). A test battery (PENTB) that was developed by a group of experts to examine the neurobehavioral effects of environmental exposures specifically in children was used. Extensive training of PENTB examiners who were blinded to the exposure status of the child and a thorough review of the collected data also contributed to the strengths of this study.

**Conclusion**

Our findings suggest that MP might cause subtle changes to short-term memory and attention and might contribute to problems with motor skills and some behaviors. However, the results of the study are not conclusive because these effects were not seen consistently in both sites. Although some domains essential to neurobehavioral development appear to have been affected by exposure to MP, the results are largely inconsistent. The usefulness of the PENTB should be evaluated to determine whether further refinement of the battery is needed. Suggested modifications to the PENTB include adding or deleting tests, as necessary or appropriate, for the environmental exposure and outcome being examined.

**References**

Amler RW, Gibertini M (eds). 1996. Pediatric Environmental Neurobehavioral Test Battery. Atlanta, GA:U.S. Department of Health and Human Services.

[Anonymous.] 1997a. Methyl parathion alert. J Environ Health 59(7):26.

———. 1997b. Methyl parathion update. J Environ Health 60(3):42.

———. 1997c. Methyl parathion comes inside. Environ Health Perspect 105:690–691.

ATSDR. 1999. Toxicological Profile for Methyl Parathion (update). Atlanta, GA:Agency for Toxic Substances and Disease Registry.
—. 2003. Long-term Health Effects of Methyl Parathion Exposure in Children, Mississippi and Ohio, 1999–2000. Atlanta, GA:Agency for Toxic Substances and Disease Registry.

Beach JR, Spurgeon A, Stephens R, Heafield T, Calvert IA, Levy LS, et al. 1996. Abnormalities on neurological examination among sheep farms exposed to organophosphorous pesticides. Occup Environ Med 53(8):520–525.

Bearer CF. 1995. Environmental health hazards. How children are different from adults. Future Child 5(2):11–26.

Centers for Disease Control and Prevention. 1984. Epidemiologic notes and reports organophosphate insecticide poisoning among siblings—Mississippi. Morb Mortal Wkly Rep 33(42):592–594.

CERCLA. 1986. Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended by the Superfund Amendments and Reauthorization Act of 1986.

Eskenazi B, Bradman A, Castorina R. 1999. Exposures of children to organophosphate pesticides and their potential adverse health effects. Environ Health Perspect 107(suppl 3):409–419.

Esteban E, Rubin C, Hill R, Olson D, Pearce K. 1996. Association between indoor residential contamination with methyl parathion and urinary para-nitrophenol. J Expo Anal Environ Epidemiol 6(3):375–387.

Farahat TM, Abdelrasoul GM, Amr MM, Shebl MM, Farahat FM, Anger WK. 2003. Neurobehavioural effects among workers occupationally exposed to organophosphorous pesticides. Occup Environ Med 60(4):279–286.

Guzelian PS, Henry CJ, Olin SS, eds. 1992. Similarities and Differences between Children and Adults: Implications for Risk Assessment. Washington, DC:ILSI Press.

Landrigan PJ, Carlson JE. 1995. Environmental policy and children’s health. Future Child 5(2):34–52.

Morgan DP, Hetzel HT, Slach EP, Lin L. 1977. Urinary excretion of paranitrophenol and alkyl phosphates following ingestion of methyl or ethyl parathion by human subjects. Arch Environ Contam Toxicol 6:159–173.

Rosenstock L, Keiffer M, Daniel WE, McConnell R, Claypool K. 1991. Chronic central nervous system effects of acute organophosphate pesticide intoxication. Lancet 334(8761):223–227.

Savage EP, Keefe TJ, Mounce LBA, Heaton R, Lewis J, Burcar PJ. 1988. Chronic neurological sequelae of acute organophosphate pesticide. Arch Environ Health 43:38–45.

Steenland K, Jenkins B, Ames RD, O’Malley M, Chrislip D, Russo J. 1994. Chronic neurologic sequelae to organophosphate pesticide poisoning. Am J Public Health 84(5):731–736.

Stokes L, Stark A, Marshall E, Narang A. 1995. Neurotoxicity among pesticide applicators exposed to organophosphates. Occup Environ Med 52(10):648–653.

U.S. EPA Office of Pesticide Programs. 2000. Illegal Indoor Use of Methyl Parathion. Washington, DC:U.S. Environmental Protection Agency. Available: http://www.epa.gov/pesticides/factsheets/chemicals/methyl.htm [accessed 22 March 2002].

Zeitz P, Kakolewski K, Imtiaz R, Kaye W. 2002. Methods of assessing neurobehavioral development in children exposed to methyl parathion in Mississippi and Ohio. Environ Health Perspect 110(suppl 6):1079–1083.