Fatal parathion poisoning in Sierra Leone

R. A. ETZEL, D. N. FORTHAL, R. H. HILL, JR., & A. DEMBY

In May and June 1986, 49 persons in Sierra Leone were acutely poisoned by the organophosphate insecticide, parathion. Fourteen people died. Illness occurred in three episodes at two different locations that were 44 km apart. A study of 21 cases and 22 household controls was undertaken to explore which factors were associated with the development of the symptoms. Cases were more likely than controls to have eaten bread in the 4 hours before becoming ill (odds ratio, 12.7; 95% confidence interval, 2.4–83.8). Scrapings of residue from the floor of the truck that had brought the wheat flour from the mill to the general store where the baker purchased it were positive for parathion, suggesting that the flour had been contaminated during transport. Pesticide poisoning is a common problem in the developing world, and public health measures such as restricting the use of parathion may help to prevent fatal poisonings.

Pesticide poisoning is a preventable cause of illness and death. WHO has estimated that about 1 million cases of unintentional acute pesticide poisoning occur each year, with an overall case fatality rate of 0.5–2.0%. Parathion, an organophosphate pesticide known for its high toxicity, has commonly been implicated in epidemics of pesticide poisoning (1), many of which occurred as a result of contamination during the transportation or storage of foodstuffs.

In June 1986, we investigated an epidemic of parathion poisoning in Kenema and Lelahun, two towns in the Eastern region of Sierra Leone. The epidemic began in Kenema, the third largest town (population approximately 39,000) in the country. On 20 May 1986, 27 persons (18 children and nine adults) presented at clinics in Kenema with an illness characterized by the sudden onset of weakness, dizziness, vomiting, and diarrhoea that began within a half hour of eating. The most severely affected persons began frothing at the mouth, became short of breath, developed pulmonary oedema, and rapidly lost consciousness. Seven persons (five children and two adults) died within a few hours of becoming ill. The others recovered completely within a day. On 21 May 1986, a similar outbreak occurred in Lelahun, a village of population 2000 located 44 km from Kenema. Thirteen persons in Lelahun became ill. Three children and two adults died. Finally, on 1 June 1986 nine persons became ill in the town of Kenema and two children died.

Our investigation, whose results are reported here, had three major goals: to identify the causative agent; to determine the vehicle of intoxication; and to specify the mechanism by which contamination occurred.

MATERIALS AND METHODS

In June 1986, a case–control study was carried out in Kenema to determine which factors were associated with the development of the observed symptoms. A case was defined as an afebrile person who was admitted to the hospital in Kenema between 20 May and 15 June 1986 with loss of consciousness and at least one of the following signs: excess salivation, frothing at the mouth, excess sweating, or muscle twitching.

Controls were selected alphabetically from the households of patients who met the case definition. To be eligible as a control, an individual had to be

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over 6 months of age, in good health during the outbreak, and available for interview on the day of the household visit. Information on cases and controls too young to be interviewed, or those who had died, was obtained from a parent or guardian. All interviews were conducted in the native language of the respondent during home visits by a bilingual male Sierra Leonean health care worker. Responses were recorded in English on questionnaire forms.

Each case and control was questioned about 18 different signs and symptoms (such as excess salivation, excess tearing, increased urination, diarrhea, convulsions, and loss of consciousness) and about foods and beverages that had been consumed during the 4 hours before becoming ill (for cases) or on the day of a case’s illness (for controls). Questions about 53 commonly used foods and beverages were included. The questionnaire also gathered information about the individual’s occupation, religion, and the use and storage of pesticides in the house.

On the basis of the results of the study, we interviewed each baker in Kenema and Lelahun to determine when he had received his last shipment of flour, how it was delivered, and where it was purchased. Samples of bread were collected from the one bakery in Lelahun and from all 15 bakeries in Kenema. Samples of bread-making ingredients, including yeast, flour, salt, and scraps from the breadboards and knives used for cutting the bread, were also collected.

Almost all the bakers used wheat flour milled in Freetown, the capital of Sierra Leone, and transported 300 km by truck from the mill to the general stores in Kenema, where it was purchased by the bakers in Kenema and Lelahun. In mid-May 1986, the flour mill introduced a requirement that an empty flour sack be exchanged for each full sack of flour purchased. The returned empty bags were subsequently fumigated with aluminium phosphide and then refilled with flour. Because the empty bags might have been contaminated and therefore, when refilled, may have contaminated the flour, a random sample of 12 returned bags (10%) was analysed for toxicants.

All the samples of bread, bread-making ingredients, and used flour bags were collected in sterile plastic containers for analysis and packed individually for shipment by overnight mail to the Division of Environmental Health Laboratory Sciences, Center for Environmental Health, Centers for Disease Control, Atlanta, GA, USA.

The samples of bread were extracted with ether and the concentrates analysed by direct probe mass spectrometry, using positive chemical ionization, and by proton nuclear magnetic resonance spectroscopy.

The unknown toxicant was identified and confirmed by gas chromatography and mass spectrometry (GC/MS) as well as by Fourier transform infrared spectroscopy. The concentrations of all samples were determined by gas chromatography using phosphorus flame photometric detection, and all positive samples were confirmed by GC/MS (2).

Odds ratios were calculated by dividing the odds of exposure in the cases by the odds of exposure in the controls. Exact 95% confidence intervals for the odds ratios were obtained using the method described by Thomas (3). P-values were obtained using Fisher’s exact test.

**RESULTS**

In both Kenema and Lelahun, children had the highest rate of illness. Table 1 shows the age-specific incidence rates for illness in Kenema. Children between 1 and 10 years of age had the highest rates of illness.

In the case–control study, we identified 21 cases and 22 controls. The signs and symptoms of the 21 cases included: loss of consciousness in 100%, shortness of breath in 75%, excess sweating in 71%, frothing from the mouth in 71%, wheezing in 67%, excess tearing in 62%, excess salivation in 62%, muscle twitching in 60%, convulsions in 52%, diarrhea in 33%, vomiting in 33%, increased urination in 24%, chest pain in 28%, and abdominal cramps in 22%. The odds ratios for demographic characteristics, such as age less than 18 years, females, Moslems, and membership of the Mende tribe, were not significantly different from unity.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Population</th>
<th>No of cases</th>
<th>Rate (per 100 000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1</td>
<td>1440</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1–4</td>
<td>4995</td>
<td>11</td>
<td>220</td>
</tr>
<tr>
<td>5–9</td>
<td>4671</td>
<td>10</td>
<td>214</td>
</tr>
<tr>
<td>10–19</td>
<td>7437</td>
<td>3</td>
<td>40</td>
</tr>
<tr>
<td>20–29</td>
<td>6691</td>
<td>5</td>
<td>75</td>
</tr>
<tr>
<td>&gt; 30</td>
<td>14 538</td>
<td>2</td>
<td>14</td>
</tr>
<tr>
<td>Unknown</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>39 772</td>
<td>36</td>
<td>91</td>
</tr>
</tbody>
</table>

*Phostoxin*
Table 2. Summary of odds ratios for consumption of various items of food in the case-control study

<table>
<thead>
<tr>
<th>Food item</th>
<th>Odds ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>0.05 (0.5)</td>
</tr>
<tr>
<td>Rice</td>
<td>0.06 (0.01, 0.4)</td>
</tr>
<tr>
<td>Dried fish</td>
<td>0.1 (0.02, 0.8)</td>
</tr>
<tr>
<td>Bouillon cubes</td>
<td>0.1 (0.02, 0.7)</td>
</tr>
<tr>
<td>Cassava leaves</td>
<td>0.3 (0.05, 1.3)</td>
</tr>
<tr>
<td>Potato leaves</td>
<td>0.7 (0.05, 6.6)</td>
</tr>
<tr>
<td>Palm oil</td>
<td>0.8 (0.2, 3.1)</td>
</tr>
<tr>
<td>Groundnuts</td>
<td>1.4 (0.2, 11.6)</td>
</tr>
<tr>
<td>Fresh fish</td>
<td>2.2 (0.1, 136.6)</td>
</tr>
<tr>
<td>Sugar</td>
<td>3.4 (0.5, 40.4)</td>
</tr>
<tr>
<td>Coffee</td>
<td>6.9 (1.7)</td>
</tr>
<tr>
<td>Bread</td>
<td>12.7 (2.4, 83.8)</td>
</tr>
</tbody>
</table>

* Figures in parentheses are the 95% confidence intervals.
* Excludes 1.

Table 2 shows the odds ratios for several food items. Water and staple foods, such as rice, dried fish, and bouillon cubes, had odds ratios less than and significantly different from unity. Only bread had an odds ratio that was significantly greater than unity. When stratified by age, the association between consumption of bread and illness was stronger for children aged under 18 years (odds ratio, 21.7; 95% confidence interval, 2.4–264.6) than for adults (odds ratio, 2.3; 95% confidence interval, 0.02–195.9).

A loaf of bread baked in the bakery in Lalehun on 21 May 1986, the day of the outbreak of illness in this town, was contaminated with 410 mg/kg of parathion. Discarded bread baked on the same day in this bakery was consumed by six ducks, who died within minutes of the ingestion. Several other items collected from the Lalehun bakery were also contaminated: the knife blade used to cut the dough had 8.2 mg/kg of parathion; samples of dough used by the baker in Lalehun to sprinkle over the tops of his loaves were also positive (0.54 mg/kg and 1.1 mg/kg); the bread-kneading board had 2.6 mg/kg of parathion; while empty flour sacks also contained traces of parathion (0.03–0.09 mg/kg). Neither the yeast nor the salt from the bakery in Lalehun was contaminated. Fifty samples of bread, flour, yeast, salt, and dough collected from the 15 bakeries in Kenema also did not contain parathion.

The baker in Lalehun supplied the name of the store in Kenema where he had bought his last shipment of flour, and the storekeeper identified the truck and driver that had delivered the load of flour from the mill in Freetown in mid-May 1986. After the truck was located on 28 June 1986, its metal floor was divided into seven sections and scraped with sterile scalpels. One section of the floor was contaminated with 0.87 mg/kg of parathion. All 12 empty bags returned to the flour mill for refilling were analysed, but none was contaminated with parathion or other toxicants.

**DISCUSSION**

In this epidemic, 49 persons were acutely poisoned and 14 died when they ingested bread baked with parathion-contaminated flour. We estimate that 10–15 ml of parathion may have spilled onto a 22.5 kg bag of flour in the truck en route from the mill to the general store in Kenema.

Parathion was detected on several items from the Lalehun bakery, primarily because it had been locked immediately after the onset of the illnesses and remained undisturbed until our investigation. However, the bakeries in Kenema remained open, and presumably any residual parathion had been washed away.

Children were more severely affected than adults, many of whom were fasting from sunrise to dusk at the time of the poisoning because of the observance of Ramadan. Thus, children, who did not fast, may have been more likely to have eaten bread during the day. Bread, however, is rarely eaten with rice and dried fish, the main meal of the day, so that fasting adults who had waited until after sunset to eat may have been more likely to have consumed rice than bread.

Parathion is well absorbed by all routes of exposure and has caused deaths by inhalation, dermal absorption, and ingestion. It is categorized by WHO as a class 1a hazard (extremely hazardous). Its median lethal dose (LD₅₀) in rats is 2–13 mg per kg body weight. Thus, if an LD₅₀ of 2 mg per kg body weight is used for a 6-year-old child weighing 20 kg, the lethal dose would be 40 mg, and if the concentration of parathion in the bread was 410 mg/kg, the child would have to consume only 100 g of bread to receive such a dose. Other workers have estimated that the lethal dose of parathion for children is 0.1 mg per kg body weight (4). If so, a 20-kg child would have to consume only 5 g of bread to ingest a lethal amount of the pesticide.

In contrast to the epidemics of pesticide poisoning in the 1960s and 1970s, which often occurred as a result of contamination of food in international...
commerce (5, 6), recent epidemics are more commonly due to contamination during transportation within a country (7). For example, in this study the detection of parathion on the floor of the delivery truck is evidence that the contamination occurred during the transport of the flour from Freetown to Kenema. Primary prevention of such incidents has already been shown to be efficacious. International regulations drawn up by organizations concerned with modes of transport, e.g., the International Civil Aviation Organization, the International Maritime Organization, and the International Air Transport Association, have effectively prevented contamination during international commerce. However, many developing countries have not yet established national laws to regulate the shipment and use of highly toxic pesticides such as parathion. Whenever possible, the importation and use of parathion in such countries should therefore be banned in favour of less toxic pesticides, such as malathion. Nine countries have already banned, and seven have severely restricted, the use of parathion (8), and the International Code of Conduct on the Distribution and Use of Pesticides (9) establishes voluntary standards of conduct for pesticide use to assist countries that have not yet established national regulatory programmes.

Secondary prevention is an important adjunct to primary prevention. People who sell or use parathion, persons involved in the food industry, and all health care workers should be aware of the symptoms of organophosphate poisoning. Medical personnel should be educated about the proper use of the antidote for parathion poisoning, pralidoxime (2-PAM), a cholinesterase reactivator, which must be used within 24–48 hours of exposure to be effective. In addition, atropine should be used to antagonize the central and muscarinic cholinergic signs of the poisoning.

Measures such as these may help prevent some of the estimated 20 000 deaths and 1 million illnesses worldwide per year that are attributable to pesticide poisonings.

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RÉSUMÉ

INTOXICATION MORTELLE PAR LE PARATHION EN SIERRA LEONE

En mai et juin 1986, 49 personnes ont été gravement intoxiquées en Sierra Leone par le parathion, insecticide organophosphoré. Quarante d’entre elles sont décédées. L’intoxication s’est déclarée en trois épisodes, en deux endroits distants de 44 km. Une étude portant sur 21 cas et 22 témoins membres des foyers des malades a été effectuée pour déterminer les facteurs qui pouvaient être liés à l’apparition des symptômes. Un cas a été défini comme une personne afébrile admise à l’hôpital entre le 20 mai et le 15 juin 1986, ayant perdu conscience et présentant au moins l’un des signes suivants: ptysalisme, écoulement par la bouche d’une mousse baveuse, sueurs profuses et myoclonies. Les cas avaient plus probablement que les témoins consommé du pain dans les quatre heures précédant le début des malaises (risque relatif. 12,7; intervalle de confiance à 95%, 2,4–83,8). L’enquête a révélé qu’un pain provenant de l’une des boulangeries locales était contaminé par le parathion à raison de 410 mg/kg. La lame du couteau ayant servi à découper la pâte à pain, la planche sur laquelle le pain avait été pétri et plusieurs sacs de farine vides provenant de la même boulangerie étaient également contaminés. L’analyse de raclettes prélevées sur le plancher du camion qui avait transporté la farine de blé du moulin au magasin où le boulanger s’était approvisionné a mis en évidence la présence de parathion, ce qui donne à penser que la farine avait été contaminée au cours du transport. L’intoxication par les insecticides est un problème courant dans les pays en développement et des mesures de santé publique telles qu’une limitation de l’emploi du parathion pourraient aider à prévenir ces décès.
REFERENCES


8. UN consolidated list of products whose consumption and/or sale have been banned, withdrawn, severely restricted or have not been approved by governments, second issue. New York, United Nations, 1987.
