EPIDEMIC KEPONE POISONING IN CHEMICAL WORKERS

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From March 1974 through July 1975, 76 (57%) of 133 persons who had worked at a pesticide plant that produced Kepone, a chlorinated hydrocarbon insecticide, contracted a previously unrecognized clinical illness characterized by nervousness, tremor, weight loss, opsonolus, pleuritis and joint pain, and oligospermia. Illness incidence rates for production workers (64%) were significantly higher than for nonproduction workers (16%). The mean blood Kepone level for workers with illness was 2.53 ppm and for those without disease 0.60 ppm (P<0.001). Blood Kepone levels in current workers (mean, 3.12 ppm) were higher than those in former employees (1.22 ppm). Blood Kepone levels for workers in nearby businesses and for residents of a community within 1.6 km of the plant ranged from undetectable to 32.5 ppb. Illness attributable to Kepone was found in two wives of Kepone workers; there was no apparent association between frequency of symptoms and proximity of the plant in the survey of the community population.

epidemics; insecticides, organochlorine; Kepone; occupational diseases; pesticides; poisoning;

More than 1000 new chemical compounds are developed each year in the United States and are added to the approximately 30,000 chemicals and 2,000,000 mixtures, formulations, and blends already in commercial use (1). When the

Received for publication August 8, 1977, and in


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7 Toxicology Branch, Clinical Chemistry Division, Bureau of Laboratories, CDC, Atlanta.

toxic potential of new chemical products is not recognized, and appropriate safeguards for their handling not employed, the resultant exposure of workers may lead to new and unexpected syndromes of occupational disease (2-4). This report describes an outbreak of illness among workers in a chemical plant exposed to the chlorinated hydrocarbon insecticide Kepone. In addition to illness in workers, members of workers’ families and persons residing near the plant had documented increased absorption of Kepone, and there was widespread environmental contamination.

BACKGROUND

Production of Kepone

Kepone (1,1a,3,3a,4,4a,5b,6-decachloro-1,3,4-metheno-2H-cyclobuta (c,d) pentalen-2-one) is a chlorinated hydrocarbon insecticide closely related in structure
to Mirex. Approximately 99 per cent of the world's production of Kepone was exported to West Germany, where it served as a raw material in the formulation of another pesticide compound, Kelevan; Kelevan was used principally in Central and South America for control of banana borer weevils. In the United States the chief use of Kepone was for formulating pesticide mixtures for ant and roach traps, usually at a strength of 0.125 per cent.

Kepone was developed by Allied Chemical Corporation in the early 1950s and was registered as a pesticide in 1955. It was manufactured in relatively small amounts by Allied, and by three other firms under contract to Allied, during the 1950s and early to mid-1960s (5). Allied resumed sole production in 1966 and produced 50,000 to 200,000 kg annually in Hopewell, Virginia, from 1966 to 1973. In 1973, Allied discontinued all production of Kepone and entered into an exclusive toll contract for Kepone production with a newly established firm, Life Science Products Company (LSPC) of Hopewell. LSPC was headed by a former Allied plant manager and a former Allied researcher. Under the terms of the toll contract, Allied supplied raw materials to LSPC and then paid a tolling fee for the synthesis of each batch of raw material to finished Kepone.

Kepone is no longer manufactured, but from March 1974 through July 25, 1975, LSPC in Hopewell was the world's sole producer of Kepone. Kepone, in the form of a powder, >90 per cent pure, was synthesized at LSPC by the reaction of hexachlorocyclopentadiene (HCP) with sulphur trioxide in the presence of an antimony pentachloride catalyst. Powdered Kepone was the only product of the firm and annual output averaged over 400,000 kg. The synthesis of Kepone at LSPC was not required by law to be registered with the US Environmental Protection Agency (EPA) on the grounds that EPA considered the undiluted Kepone a chemical and not a pesticide.

**Toxicology of Kepone**

Prior to the present episode, no human data and only limited data from animal studies were publicly available regarding the toxic effects of Kepone. Subsequently, a preliminary report from the Carcinogenesis Biomassay Program of the National Cancer Institute has shown that laboratory rats and mice fed Kepone in dietary concentrations ranging from 8 ppm to 40 ppm for 80 weeks developed tremor, dermatologic changes, decreased food consumption, weight loss, and a statistically significant increase in the incidence of hepatocellular carcinoma (6). Other animal studies have noted those same findings and have also observed decreased reproductive capacity and testicular atrophy (7, 8) (table 1).

**The epidemic**

On July 11, 1975, an internist in Hopewell submitted a serum sample on a worker at the LSPC plant with severe tremors to the Center for Disease Control (CDC) Toxicology Laboratory for Kepone analysis. The result, reported by the laboratory on July 19 and transmitted on July 21 to the Virginia State Department of Health, showed a highly elevated serum Kepone level, 7.5 ppm. Follow-up field investigation on July 22 and 23 by the Epidemiology Bureau, Virginia State Health Department, revealed massive contamination of the plant site and a high incidence of unusual illness in workers. Officials of the State plant on the Virginia State that CDC a detection Age evaluation has shown that laboratory rats and mice fed Kepone in dietary concentrations ranging from 8 ppm to 40 ppm for 80 weeks developed tremor, dermatologic changes, decreased food consumption, weight loss, and a statistically significant increase in the incidence of hepatocellular carcinoma (6). Other animal studies have noted those same findings and have also observed decreased reproductive capacity and testicular atrophy (7, 8) (table 1).

**Table 1**

<table>
<thead>
<tr>
<th>Toxic effects of Kepone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Animals</td>
</tr>
<tr>
<td>---</td>
</tr>
<tr>
<td>Excitability</td>
</tr>
<tr>
<td>Tremor</td>
</tr>
<tr>
<td>Loss of weight</td>
</tr>
<tr>
<td>Dermatologic changes</td>
</tr>
<tr>
<td>Testicular atrophy</td>
</tr>
<tr>
<td>Increased liver to body weight ratio</td>
</tr>
<tr>
<td>Increase in hepatocellular carcinoma</td>
</tr>
<tr>
<td>Oesophagus</td>
</tr>
<tr>
<td>Pleuritic pain</td>
</tr>
</tbody>
</table>

The physique of the renovate workers was frequently characterised by poor clothing and health on the job. Exhaustive ventilation of the plant was installed. Workers were clothed in protective clothing and respirators. There has been a sudden increase in the number of workers affected by Kepone.

Anecdotal reports of Kepone had been reported by workers at the plant and 2 workers had been hospitalised aspowiedź. The management of Hopewell.
Epidemic Kepone Poisoning in Chemical Workers

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The epidemic

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The physical plant at LSPC consisted of a renovated gasoline service station, two outbuildings, and an open paved area between the buildings known as the "pad." Hygienic conditions were poor. A few dust masks were provided to workers, but were of poor quality and almost never used. Exhaust ventilation in the drying and filtration operations was inadequate, and workers became covered with a fine white Kepone dust. The large tanks which held Kepone in liquid form during synthesis frequently developed leaks, and a precipitate of wet Kepone flowed almost constantly onto the pad area. No protective clothing or boots were provided, and workers on the pad therefore had to walk in their own shoes in wet Kepone. No separate clean area was provided for storing food, and employees frequently ate lunch in the production area.

There had been a large labor turnover at LSPC. In the 16 months of the plant's operation, the work force had been replaced nearly five times. Reasons given by employees for this high turnover were 1) the undesirable working conditions at the plant and 2) the frequent development in workers of the symptom complex referred to as "Kepone shakes."

Anecdotal information indicated that Kepone had been discharged into both air and water outside the plant by LSPC. Odoriferous vapors from LSPC were noted frequently by workers in nearby businesses, and emissions were occasionally so thick that neighbors complained to plant management. Sewage workers in the Hopewell city waste water treatment plant, which received effluent from LSPC, began to notice noxious smelling white-flecked water swirls after the onset of LSPC's operations in March 1974. On May 30, 1975, a Hopewell sewage worker was overcome by hexachlorocyclopentadiene (HCP) fumes after a railway tank car containing HCP developed a leak on LSPC property and discharged its contents into a storm sewer.

Materials and methods

From company records, the Virginia State Health Department obtained a listing of the 148 persons who had been employed at LSPC in the 16 months of its operation (33 current employees and 115 former). One hundred thirty-five of these employees (all current and 100 former) were located (90 percent of total), and for each a questionnaire relating to general health, occupational exposure, and possible job-related illness was completed. A brief physical examination was performed on each worker, and a venous blood sample obtained for determination of Kepone level. The 15 former workers not included in the study either could not be located, had moved out of state, or refused to participate. Questionnaire information was obtained on 270 family members or household contacts of workers; 32 of these persons permitted a blood sample to be drawn.

Similar evaluations were conducted on 39 employees of Allied Chemical Corporation in Hopewell who had worked in Kepone production prior to its discontinuation by Allied in 1973.

To evaluate the possibility of Kepone absorption in persons beyond the plant premises, a community survey was undertaken. In the first phase of this survey, each of several businesses located within a block of the chemical plant was visited. Thirty-two workers in these businesses who agreed to participate were questioned regarding symptomatology in the same

\begin{table}[h]
\centering
\begin{tabular}{|c|c|}
\hline
\textbf{Symptoms} & \textbf{Frequency} \\
\hline
Nervousness & 50 \% \\
Tremor, ataxia & 40 \% \\
Loss of weight & 30 \% \\
Skin rash & 20 \% \\
Sterility & 10 \% \\
Abnormal liver function studies & 5 \% \\
Hepatomegaly & 2 \% \\
Opocelous & 1 \% \\
Joint pain & 1 \% \\
Fleuret pain & 1 \% \\
\hline
\end{tabular}
\caption{Incidence of Symptoms among Workers at LSPC in 1975.}
\end{table}
manner as the chemical workers, and a venous blood sample was obtained from each for Kepone analysis. The second phase was a survey of residents in the areas of Hopewell near the plant. Circles were drawn on a map at radii of 0.4, 0.8, 1.2, and 1.6 km around the plant. A low income housing project and a home for the elderly were within the 0.4 km circumference. In this area, the first housing unit to be surveyed was selected at random, and thereafter every fifth occupied unit was systematically chosen. An effort was made to include every member of each selected household in the survey. If the occupants of a housing unit could not be located, the unit immediately preceding or following was selected. In the home for the elderly, a somewhat less systematic procedure had to be followed, since many individuals refused to participate. In the three outer areas, survey subjects consisted of the members of one housing unit chosen at random from each residential block which fell on the circumferential line; 214 specimens were collected in this survey (112 at 0.4 km; 34 at 0.8 km; 39 at 1.2 km; and 29 at 1.6 km). Finally, included in the survey process were workers at the Hopewell waste water treatment plant. In all areas, a standard interview was conducted with each participant, concentrating on duration of residence in the area as well as on symptomatology; a venous blood sample for Kepone was also obtained.

Subsequent to identification of the problem in workers, levels of Kepone in ambient air were assessed by EPA in stored 24-hour high-volume air sample filters that had been routinely collected from January 1974 through April 1975 as part of the Virginia air monitoring network at a site 200 meters from the LSPC plant (10). Kepone levels in water, bottom sediments, sludge, fish, and shellfish were determined by EPA in samples taken from the Bailey's Creek tributary of the James River (which received the effluent from the Hopewell sewage treatment plant), from the James River itself, and from Chesapeake Bay to distances as far as 64 km downstream from the plant.

Laboratory determinations for this study were performed by three laboratories—the Toxicology Branch, Bureau of Laboratories, CDC, the Health Effects Research Laboratory, EPA, and the Division of Consolidated Laboratory Services, Commonwealth of Virginia. The EPA laboratory performed the analyses on blood specimens from Hopewell residents in the community survey as well as on multiple environmental specimens; the CDC laboratory performed the analysis of blood specimens from former LSPC employees, Allied employees and the family members of chemical workers; the Virginia laboratory analyzed blood specimens from current LSPC employees. The method used for determination of Kepone levels in whole blood was essentially the same in all laboratories and involved extraction with an organic solvent, followed by sample cleanup by elution through a Florisil column. Quantitative determination of Kepone in the extract was then accomplished in each laboratory by electron capture gas liquid chromatography. Qualitative confirmation was accomplished either by gas liquid chromatography using a Coulson Conductometric Detector or by gas liquid chromatography coupled with mass spectrometry.

The CDC and EPA laboratories participated in a blind quality control program under the auspices of the Division of Consolidated Laboratory Services of the Commonwealth of Virginia. For 10 samples, two whole blood, five "spiked" whole blood and three solvent extracts of blood, Kepone levels ranging from 0 to 9.9 ppm, the coefficient of correlation between individual CDC and EPA values was 0.97.

**Clinical Findings**

Taylor et al. (11), have described the clinical aspects of Kepone intoxication.
River itself, and from to distances as far as 64 from the plant.

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ry, EPA, and the Division Laboratory Services, Com-
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cimens; the CDC labora-
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The method used for f levels in whol-
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olved extraction with an a, followed by sample on through a Florisil col-
determination of Ke-
a was then accomplished ry by electron capture gas-
ograph. Qualitative con-
complished either by gas-
ography using a Coulom-
D or by gas liquid cou-
pled with mass spec-

EPA laboratories partic-
ul quality control pro-
 of the Division of Lab-
 Virginia. For 10 samples, five "spiked" whole blood.
nt extracts of blood, ranging from 0 to 200 ppm of correlation between the and EPA values was 0.99.

Epidemiologic findings...

A case of Kepone poisoning was defined epidemiologically in this outbreak as a worker who developed nervousness and/or tremulousness after start of employment at the LSMP pesticide plant or who at the time of our examination demonstrated objective tremor or opsoconius with or without subjective symptoms. In our case definition, we took "nervousness" to be entirely a subjective phenomenon, a sensation of unfounded anxiety, to be considered present when recounted by a subject during the questionnaire interview. We applied no further qualifying criteria, except to distinguish subjective "nervousness" from objective "tremulousness"; the latter term, either in history or on physical examination, denoted demonstrable tremor of one or more extremities, head bobbing, unsteadiness of gait, or ataxia. Using these criteria, we identified 76 past or present cases of Kepone poisoning in the 133 workers, an overall incidence rate of 57 per cent. Of the 76 cases, 27 had only subjective symptoms of nervousness while 49 demonstrated objective neurologic abnormality with or without subjective symptoms. There were no deaths.

The average latency interval between start of employment at LSMP and onset of symptoms was six weeks. Analysis of latency periods among the various categories of production personnel showed no particular trend. Average length of employment at the plant was somewhat longer for cases than for noncases: 17.8 weeks and 14.6 weeks, respectively. Among former workers, however, cases had been employed more than twice as long as noncases: 15.5 weeks vs. 7.3 weeks. Affected workers were somewhat younger than unaffected workers, with the mean age for cases being 29.4 years and for noncases 33.7 years.
Illness incidence rates by job category are shown in Table 2. There were six major job categories in the plant—secretarial/office, janitorial, maintenance, pad man, foreman, and dryer and filter operator. In general, higher attack rates were seen in production personnel than in those not directly involved in production (64 per cent vs. 16 per cent).

Laboratory analysis of blood specimens for Kepone in workers at the plant showed the range for worker cases to be 0.009 to 11.8 ppm (Table 3); for worker noncases, the range was from nondetectable to 4.1 ppm. The mean level for cases was 2.53 ppm with a median of 1.8 ppm; the mean level for noncases was 0.50 ppm with a median of 0.2 ppm. This difference in means is statistically significant at *p* < 0.001. Former LSPC workers had a mean blood Kepone level of 1.22 ppm (SD, 1.69 ppm) while current workers had a mean level of 3.12 ppm (SD, 2.88). Wide individual variation precluded the development of more precise dose-response data. In the 39 Allied workers examined, none of whom had been occupationally exposed to Kepone for at least the preceding 18 months, 30 (77 per cent) had detectable blood Kepone levels (Table 3).

In the community survey, interviews and blood specimens were obtained from 214 persons. None of the participants in this survey had ever worked in Kepone.

### Table 2

**Kepone poisoning attack rates by job category**

<table>
<thead>
<tr>
<th>Job category</th>
<th>Current workers</th>
<th></th>
<th>Former workers</th>
<th></th>
<th>Total workers</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cases Total</td>
<td>Attack rate (%)</td>
<td>Cases Total</td>
<td>Attack rate (%)</td>
<td>Cases Total</td>
<td>Attack rate (%)</td>
</tr>
<tr>
<td>Secretarial/office</td>
<td>0 5</td>
<td>0</td>
<td>0 4</td>
<td>0</td>
<td>0 9</td>
<td>0</td>
</tr>
<tr>
<td>Janitorial</td>
<td>2 4</td>
<td>50</td>
<td>1 6</td>
<td>17</td>
<td>3 10</td>
<td>30</td>
</tr>
<tr>
<td>Maintenance</td>
<td>4 7</td>
<td>57</td>
<td>14 28</td>
<td>50</td>
<td>18 35</td>
<td>51</td>
</tr>
<tr>
<td>Pad man</td>
<td>1 1</td>
<td>100</td>
<td>3 6</td>
<td>50</td>
<td>4 7</td>
<td>57</td>
</tr>
<tr>
<td>Foreman</td>
<td>7 9</td>
<td>78</td>
<td>9 13</td>
<td>68</td>
<td>16 22</td>
<td>73</td>
</tr>
<tr>
<td>Dryer and filter operator</td>
<td>6 7</td>
<td>86</td>
<td>29 43</td>
<td>67</td>
<td>35 50</td>
<td>70</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>20 33 61</strong></td>
<td><strong>56 100 56</strong></td>
<td><strong>76 133 50</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 3

**Whole blood Kepone level, by group of exposed persons**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. tested</th>
<th>No. with detectable level</th>
<th>% with detectable level</th>
<th>Range of detectable level, ppm</th>
<th>Mean of detectable level, ppm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affected LSPC* worker</td>
<td>57</td>
<td>57</td>
<td>100</td>
<td>0.009–11.8</td>
<td>2.56</td>
</tr>
<tr>
<td>Unaffected LSPC worker</td>
<td>49</td>
<td>48</td>
<td>99</td>
<td>0.003–4.1</td>
<td>0.50</td>
</tr>
<tr>
<td>Family members, LSPC worker</td>
<td>32</td>
<td>30</td>
<td>94</td>
<td>0.003–0.39</td>
<td>0.10</td>
</tr>
<tr>
<td>Allied† Kepone worker</td>
<td>39</td>
<td>30</td>
<td>77</td>
<td>0.003–0.45</td>
<td>0.06</td>
</tr>
<tr>
<td>Neighborhood worker</td>
<td>32</td>
<td>23</td>
<td>72</td>
<td>0.003–0.031</td>
<td>0.011</td>
</tr>
<tr>
<td>Sewage treatment plant worker</td>
<td>10</td>
<td>6</td>
<td>60</td>
<td>0.004–0.014</td>
<td>0.008</td>
</tr>
<tr>
<td>Cab driver</td>
<td>5</td>
<td>1</td>
<td>20</td>
<td>0.003</td>
<td>0.003</td>
</tr>
<tr>
<td>Truck driver</td>
<td>2</td>
<td>1</td>
<td>50</td>
<td>0.004</td>
<td>0.004</td>
</tr>
<tr>
<td>Hopewell resident</td>
<td>214</td>
<td>40</td>
<td>19</td>
<td>0.005–0.0225</td>
<td>0.011</td>
</tr>
</tbody>
</table>

* LSPC = Life Science Products Company.
† Allied Chemical Corporation.
This difference inially significant at \( p < 0.05 \) for PC workers had a mean el of 1.22 ppm (SD, 1.69 nt workers had a mean \( \mu \)G (SD, 2.88). Wide inter-
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dose-response
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(77 per cent) had detect-
e levels (Table 3).

ity survey, interviews ens were obtained from e of the participants in ever worked in Kepone

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Cases</th>
<th>All Kepone workers</th>
<th>Neighborhood residents</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>Rash</td>
<td>60</td>
<td>79*</td>
<td>59 67*</td>
</tr>
<tr>
<td>Nervousness</td>
<td>58</td>
<td>76*</td>
<td>56 44*</td>
</tr>
<tr>
<td>Tremor</td>
<td>61</td>
<td>65*</td>
<td>61 46*</td>
</tr>
<tr>
<td>Pleuritic pain</td>
<td>26 34*</td>
<td>32 24*</td>
<td>21 13</td>
</tr>
<tr>
<td>Weight loss</td>
<td>23</td>
<td>30*</td>
<td>27 20*</td>
</tr>
<tr>
<td>Visual difficulties</td>
<td>22 45*</td>
<td>42 32*</td>
<td>19 12</td>
</tr>
</tbody>
</table>

No. in sample 76 133 158

* \( p < 0.001 \) by Chi-square test.
\( + p < 0.05 \) by Chi-square test.

production. Among the community residents age 15 years and above (15 being the age of the youngest worker at LSPC), prevalence rates for all symptoms were significantly lower than in workers (Table 4). There was no consistent pattern of decline in frequency of symptoms with increasing distance from the plant, and no community survey participant was felt to have the clinical syndrome of Kepone poisoning manifested by LSPC workers.

In the survey of chemical workers’ family members, two wives of workers were found to have had objective tremor. Both gave a history of having washed their husbands’ work clothing.

Laboratory analyses demonstrated detectable levels of blood Kepone in 40 (19 per cent) of the 214 community residents surveyed, with levels ranging from 5.1 parts per billion (ppb) to 32.5 ppb. The majority (36 persons) of the 40 with detectable values lived within 0.4 km of the plant—34 from the low income housing project and two from the home for the elderly; two lived 0.8 km from the plant and one each lived at distances of 1.2 km and 1.6 km distances from the plant.

Kepone concentrations in high-volume air samples collected 200 meters from the LSPC plant in January 1974, prior to the start of Kepone production, averaged 0.6 nanograms/m³ air (10). From March 1974 through April 1975, the mean airborne Kepone level at the same site was 7.0 micrograms/m³ air (maximum 54.8 μg), an 11,667-fold increase. Additional high-

<table>
<thead>
<tr>
<th>Range of detectable level, ppm</th>
<th>Mean of detectable level, ppm</th>
</tr>
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<tbody>
<tr>
<td>0.009–11.8</td>
<td>2.8</td>
</tr>
<tr>
<td>0.003–4.1</td>
<td>0.6</td>
</tr>
<tr>
<td>0.003–0.39</td>
<td>0.1</td>
</tr>
<tr>
<td>0.003–0.45</td>
<td>0.0</td>
</tr>
<tr>
<td>0.003–0.031</td>
<td>0.0</td>
</tr>
<tr>
<td>0.004–0.014</td>
<td>0.0</td>
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<tr>
<td>0.003</td>
<td>0.0</td>
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<tr>
<td>0.004</td>
<td>0.0</td>
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<tr>
<td>0.005–0.0325</td>
<td>0.0</td>
</tr>
</tbody>
</table>

DISCUSSION

The data obtained in this study show that 76 (57 per cent) of 133 LSPC workers developed clinical illness, principally of the nervous system, as the result of virtually uncontrolled occupational exposure to the chlorinated hydrocarbon pesticide Kepone. Several lines of evidence support that conclusion: 1) attack rates were highest in those workers, both present and former, involved in actual production of Kepone; 2) there was a temporal relationship between start of employment at the LSPC plant and onset of symptoms; 3) there was an apparent dose-effect relation with blood Kepone levels being higher in affected workers than in nonaffected workers or in Hopewell residents, as well as being higher in workers employed at the time of our study than in former workers; 4) there was a remarkable similarity found between the effects of Kepone exposure in laboratory animals and the clinical illness in the LSPC workers (Table 1).

Although the range of blood Kepone
levels was quite wide for both case and noncase worker groups, only 33 (25 per cent) of the 133 workers examined were currently employed when the blood specimens were obtained. The remainder were past workers, some of whom had not worked in contact with Kepone for over a year. Their blood levels may therefore have been low because of excretion from the body or because of redistribution of Kepone to body compartments other than serum.

Blood levels in persons non-occupationally exposed to Kepone were much lower than those in workers, and the sources of their exposure were less well-defined. Persons not employed at the pesticide plant were presumably exposed either through environmental contamination or through dust carried home on workers' clothing. Two wives of workers gave histories of tremor. Otherwise, none of these non-occupationally exposed persons demonstrated any evidence of toxic symptoms.

There are no data at this point to suggest whether or not the present clinical findings will be reversible. Long-term follow-up of exposed workers will be instructive in this regard. A further unresolved question is whether there will develop additional delayed effects of Kepone exposure, particularly carcinogenic and reproductive sequelae, since preliminary evidence indicates that Kepone may cause hepatocellular carcinoma in laboratory rats and mice (6). Long-term prospective surveillance of both workers and community residents may be required to resolve this issue. Active planning for such surveillance is now proceeding under the direction of the Virginia State Department of Health.

In its implications for industry, labor, and governmental regulatory agencies, the Hopewell Kepone episode may be compared with the recognition of hepatic angiosarcoma in vinyl chloride workers (3) or with the documentation of lung cancer and mesothelioma among workers in the asbestos industries (13, 14). In Virginia, the Kepone episode has stimulated the development of an active, OSHA-approved occupational safety and health plan and has stimulated the passage through the State Legislature of the Virginia Toxic Substance Information Act. Hopefully, vigorous enforcement of the Occupational Safety and Health Act and close monitoring of the pre-market toxicologic testing mandated under the Toxic Substance Control Act will prevent the future occurrence of similar episodes. Effective prevention will in addition require the development of a sense of personal responsibility for occupational health and safety which goes far beyond formal compliance with the requirements of the law. Perhaps an understanding of the Kepone episode will help to foster the development of such an attitude.

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In Virginia, the stimulated the develop OSHA-approved health plan and coverage through the State Virginia Toxic Substances Act. Hopefully, vigorous enforcement of the Occupational Act and close monitoring toxicologic testing of Toxic Substances will prevent the future occurrences. Effective prevention requires the development of personal responsibility in the workplace and compliance with the law. Perhaps an incident like the Kepone episode will accelerate the development of such an

REFERENCES


