HETA 89-183-2101 FEBRUARY 1991 ANDREW JACKSON JUNIOR HIGH SCHOOL CROSS LANES, WEST VIRGINIA NIOSH INVESTIGATORS: Larry J. Elliott, M.S.P.H. Sherry Baron, M.D., M.P.H.

I. <u>SUMMARY</u>

On March 28, 1989, the National Institute for Occupational Safety and Health (**NIOSH**) received a request for a health hazard evaluation at the Andrew Jackson Junior High School in Cross Lanes, West Virginia. This request was submitted by the teachers and principal of the school. The request reported symptoms of numbness and tingling of the extremities, upper airway irritation, eye irritation, inability to concentrate, and thermal comfort complaints. The requestors believed the symptoms were related to poor indoor air quality (**IAQ**) in the school.

On April 11 - 12, 1989, an industrial hygienist and an occupational physician conducted a preliminary IAQ evaluation at the school. Follow-up industrial hygiene and medical surveys were conducted on April 16 - 17, 1989; May 9 - 11, 1989; September 13 - 14, 1989; February 28 through March 1, 1990; and, June 8, 1990. Results, findings, and recommendations from each survey were provided by letter.

The Andrew Jackson Junior High School building was built in 1969 as an "open space" classroom configuration (i.e., there were no structural barriers between classes). During 1981 to 1983, the "open space" was converted to approximately 30 classroom areas by erecting walls. The heating, ventilation, and air conditioning (HVAC) systems were modified only by adding a false ceiling return air plenum to accommodate the space conversion. Several classroom areas were noted to have very poor air circulation due to the inability of the HVAC system to adequately ventilate the room areas. The school had been treated for termite infestation by subslab injection of chlordane and direct in-room application of chlorpyrifos (Dursban) and Diazinon. Fluorescent light ballast burn-outs over several years introduced the polychlorinated biphenyl (PCB) Aroclor 1254 into the school building which was not effectively removed by the ventilation system.

Carbon dioxide (\mathbf{CO}_2), temperature, and relative humidity measurements indicated that several of the classroom areas were not adequately ventilated (insufficient fresh air) on the days when the measurements were taken. Direct measurement of ventilation rates for the classrooms was found to range from 0 to 1160 cubic feet per minute (\mathbf{cfm}), resulting in an estimated range of 0 to 13 cfm of fresh air per person in the classrooms. The American Society of Heating, Refrigerating, and Air-Conditioning Engineers (\mathbf{ASHRAE}) recommends that 15 cfm of fresh air per person be provided to classrooms. Air samples for volatile organic chemicals (\mathbf{VOCs}) were collected in classrooms and verified the indication from \mathbf{CO}_2 and ventilation measurements of deficient ventilation of the

classroom areas which allowed chemical contaminants to build up during occupancy. Carpet samples were collected to determine the extent of pesticide contamination in classrooms and found 16% by weight of chlordane in two worst case samples, Diazinon contamination ranged from trace to 76 micrograms per gram (**ug/g**) in 16 of 17 carpet samples. Aroclor 1254 (a polychlorinated biphenyl) was found to range from trace to 67 ug/g in 15 of 17 carpet samples. Detectable concentrations of chlordane, Diazinon, Dursban, and Aroclor 1254 were found on high dermal contact surfaces. Air sampling results for Diazinon and Dursban indicated concentration ranges of non-detectable (**ND**) to 2.4 micrograms per cubic meter (**ug/m³**) and ND to 0.60 ug/m³ respectively; these concentrations were considerably lower than the exposure criteria of 200 ug/m³ and 100 ug/m³ respectively. Air sampling results for chlordane and Aroclor 1254 ranged from ND to 55.3 ug/m³ and ND to 0.19 ug/m³ respectively. Since these substances are carcinogens, NIOSH recommends that exposure be reduced to the lowest feasible level.

The medical investigation consisted of measuring blood levels of chlordane and administering three medical questionnaires to the school employees; one by the County Health Department in January 1989, one by NIOSH immediately following reopening of the school after renovations in March 1990, and one by NIOSH 4 months later, just prior to the close of the school year. Blood levels of chlordane metabolites were consistent with the range of blood chlordane metabolites found in the general population. Over 90% of current employees answered each questionnaire. The Kanawha County Health Department survey found that over 30% of the school employees had several of the health complaints. The types of complaints mentioned most commonly by the employees (upper airway and mucous membrane irritation, respiratory problems, headache, muscle aches, fatigue, problems with concentration, and tension) could have been due to a variety of causes and were not suggestive of any specific environmental contaminant. These complaints were, however, similar to those found in other buildings and schools that NIOSH has investigated for IAQ problems. The prevalence of all symptoms decreased by 50% or more after the renovations. Although the prevalences increased slightly between March and June 1990, most remained much lower than prior to the renovations. Also, the rate of comfort complaints decreased significantly after the renovations.

Based on these findings, the school building was decontaminated for insecticides and Aroclor 1254. Also, the building ventilation system was replaced, the roof repaired, and the interior renovated. Post renovation measurements indicated considerable improvements in indoor air quality, non-detectable levels of pesticides, and background levels of Aroclor 1254. Questionnaire results also showed a decline in health complaints and the perceptions of improved air quality after the decontamination and renovation of the school.

Based on the environmental monitoring results, the investigators concluded that a potential hazard from dermal and inhalation exposure to chlordane and the polychlorinated biphenyl Aroclor 1254 existed in the school classroom environment. The investigators also concluded that an unnecessary inhalation and dermal exposure potential to the insecticides Dursban and Diazinon existed in the classroom environment. Based on the ventilation evaluation, the ventilation of the classroom areas was found to be deficient in the amount of fresh air provided per person, the amount of air supplied to certain classrooms, and the distribution of air throughout certain classrooms. Health complaints, though not fully specific for poor indoor air quality, decreased in prevalence following renovation of the ventilation system and decontamination of the building. Recommendations to reduce the exposure potential to chlordane and Aroclor 1254, improve the ventilation of the classrooms, and improve the indoor air quality of the school are provided in Section VIII.

KEYWORDS: SIC 8211 (Educational Facilities, Elementary and Secondary), chlordane, chlordane metabolites, chlorpyrifos, Dursban, Diazinon, indoor air quality, PCBs, Aroclor 1254.

II. <u>INTRODUCTION</u>

On March 28, 1989, the National Institute for Occupational Safety and Health (**NIOSH**) received a request for a health hazard evaluation at the Andrew Jackson Junior High School in Cross Lanes, West Virginia. The request was submitted by the teachers and the principal of the school and reported symptoms of numbness and tingling of the extremities, upper airway irritation, eye irritation, inability to concentrate, and comfort complaints of too hot and too cold. These symptoms were believed by the requestors to have resulted from poor indoor air quality (**IAQ**) in the school.

A. Environmental Monitoring and Medical Surveys

On April 11 - 12, 1989, an industrial hygienist and an occupational physician conducted a preliminary IAQ evaluation at the school. During the two-day site visit, several teachers, school officials, parents, and county health department officials were interviewed to determine the nature of the problems and what investigations had been completed. Preliminary air sampling was conducted to evaluate the efficiency of the school's heating, ventilation, and air conditioning (HVAC) system. The entire building was inspected for potential sources of chemical pollutants (i.e., science laboratories, wood shop, home economics, and art classroom areas) and microbiological contamination which could be associated with the reported symptoms. Reports of extensive use of chemical insecticides, and the possible application of chlordane for termite control, prompted a second site visit on April 16 - 17, 1989 to conduct surface wipe and air sampling for various insecticides, and measure volatile organic chemicals, temperature, and relative humidity. The medical symptom questionnaire administered by the Kanawha County Health Department was analyzed by the NIOSH investigators to determine the type and extent of health complaints. A letter (dated May 4, 1989) summarizing the observations, sampling results, and findings from these two surveys was submitted to the principal of the school and other interested parties. At the conclusion of the April 1989 survey, the NIOSH investigators recommended that classrooms 401, 402, 309, 104B, and 502 be vacated to prevent unnecessary exposure to chlordane and Diazinon. Based on this advise, the Kanawha County School Board decided on April 12, 1989, to vacate these classroom areas. Based on the May 4th NIOSH letter report, the Kanawha County School Board voted on May 4, 1989, to vacate the school by sending the students to another building and implement the recommendations to remove the pesticide contamination, improve the ventilation of the school, and repair the roof.

On May 9 - 11, 1989, a follow-up survey was conducted to collect carpet samples for determination of insecticides and polychlorinated biphenyl (**PCB**) contamination, obtain additional surface wipe and air samples for insecticides and PCBs, obtain ventilation system

measurements, and collect blood samples from the school staff for chlordane metabolite analysis. The results of this survey were reported by letters (dated June 8 and June 22, 1989) to the school principal. Recommendations on how to remove the insecticide and PCB contamination in the school were provided to Facilities Management, Kanawha County Schools by letter (dated June 30, 1989). The school was closed to the public in June 1989 for clean-up and removal of pesticide contamination, renovation and replacement of the HVAC systems for the classrooms, and repair of the roof.

On September 13 - 14, 1989, a follow-up survey was conducted to collect "clearance samples" to verify the effectiveness of the contamination removal efforts. Results of this survey effort and subsequent recommendations were provided by letter (dated October 27, 1989) to Facilities Management. The school reopened for occupancy during the week of February 12, 1990.

On February 28 and March 1, 1990, a NIOSH survey team made follow-up ventilation measurements, collected air samples for volatile organic compounds, pesticides, and PCBs, and collected surface wipe samples for pesticides and PCBs. These measurements were obtained to characterize the ventilation and environmental status of Andrew Jackson Junior High School upon re-occupancy after the extensive interior renovation and ventilation changes made at the school building. A symptom questionnaire also was administered to the school employees to determine the occupants' perception of the air quality conditions in the renovated school building. A letter (dated May 10, 1990) summarizing the results of this survey was submitted to Facilities Management and others.

On June 8, 1990, the final site visit was conducted to administer a symptom questionnaire to the staff of the school and to hold a closing conference. Re-administration of the questionnaire prior to school closing for the summer was anticipated to provide additional follow-up information on the perceived air quality conditions within the school. A closing conference was held with Kanawha County School administrators, the principal of the school, Kanawha County Health Department Officials, and representatives of the teachers, parents, and students to summarize the results of the NIOSH evaluation and respond to any questions concerning this effort.

This final report serves to summarize the various activities, observations, and findings of this evaluation. This report also serves to formally conclude and close NIOSH activity on this request for a hazard evaluation of IAQ.

III. <u>BACKGROUND</u>

A. **Description of Facility**

The Andrew Jackson Junior High School building was built in 1969 as an "open space" classroom configuration (i.e., there were no structural barriers between classes). This single-story building was heated, cooled, and ventilated by nine individual roof-mounted air handling units (AHU) with ducted air supply; the false ceiling space served as the return air duct to the HVAC. The HVAC system was not equipped with humidification or dehumidification components. During 1981 to 1983, the "open space" was converted to approximately 30 classroom areas by erecting walls. The HVAC systems were modified only by adding a false ceiling return air plenum to accommodate the space conversion. The majority of the classrooms were ventilated by four AHUs (four individual ventilation zones) which were controlled by programmable thermostats. The fresh air intake dampers on each AHU were capable of supplying 0-23% fresh air, dependent upon the position of the damper stop. Specific structural problems of the building noted during the initial evaluation included several areas where the roof was leaking during inclement weather, missing ceiling tile panels due to the leaks, and cracks in the concrete floor. Several classroom areas were noted to have very poor air circulation. This situation was possibly due to the inability of the HVAC system to adequately ventilate the room areas, since it was originally designed to ventilate an open space configuration. Also, the poor ventilation problem was compounded by missing ceiling tile panels which inhibited the HVAC systems' air distribution and mixing capabilities.

As part of the building renovation, four new AHUs, equipped with 30% efficiency prefilters and 65% efficiency final filters on the fresh air intake, were installed in the four
classroom ventilation zones. Thermostatically controlled re-heat boxes and new ductwork
were also installed. The ventilation of the entire school was designed to be remotely
monitored by computer, with warning alarms for temperature extremes, fan failure, and reheat box failure. The fresh air intake damper controls were capable of providing 0 to
100% fresh air depending on the minimum stop requirement programmed into the AHU
zone thermostat for specified time periods. During this survey, the damper controls were
set to provide a minimum of 25% fresh air during occupied hours and a setting of 0% during
a heat-up period prior to occupancy. As designed and installed, this system was viewed as
"state of the art" by the NIOSH investigators, based on the variety of air handling systems
encountered during IAQ evaluations. New carpeting, floor tile, and a roof were also
installed during the renovation.

B. **Previous Investigation Efforts**

The Kanawha-Charleston Health Department conducted a series of evaluations at the school. These evaluations occurred on October 17, 1986, January 21, 1987, and January 24 - 25, 1989. During the October 1986, IAQ survey of the building, carbon dioxide (CO₂) measurements ranged from 600 to 1100 parts per million (ppm). On January 21, 1987, the CO₂ measurements ranged from 600 to 1000 ppm. CO₂ measurements on January 25, 1989, ranged from 200 (outdoors) to 600 ppm (indoors), and air sampling detected no pesticides. Lead and mineral concentrations in water samples collected on January 24, 1989, were less than the Environmental Protection Agency's (EPA) maximum allowable concentration. A questionnaire was distributed to all employees in January 1989, which inquired about health complaints. The results of this survey are discussed in Section VI. Because of these medical and environmental evaluation results and the limitations of the Department's testing capabilities, investigators from the Health Department concluded that they were not able to identify a direct correlation between the reported health problems and the school's environment. The Health Department made several prudent recommendations to improve the IAQ of the school, reduce the potential for exposure to pesticides, and minimize the number of health problems.

IV. MATERIALS AND METHODS

A. Environmental

Ventilation Assessment

Measurements of CO₂ were made using Drager gas detection tubes and a hand-held pump, and with a Gastech direct reading portable CO₂ Monitor (Model RI 411). Temperature and relative humidity readings were collected using a Vista Scientific Corporation automatic psychrometer (model #784) and calibrated electronic thermistors.

Direct air volume measurements through ducted supply and return ceiling diffusers were made using an Alnor Balometer (Model 6465) portable ventilation meter. Classroom ventilation rates were calculated based upon the volume of air delivered to the classroom and the size of the classroom. Estimates of the amount of outside fresh air delivered to each classroom and amounts of fresh air per occupant in the room were calculated based upon an estimated 23 to 25% (depending upon survey date) fresh air portion of the total supply. The amount of outside air per person was based on an estimated classroom occupancy of 20.

Insecticides

Sampling for airborne concentrations of insecticides was performed using ORBO-42 solid sorbent tubes and portable sampling pumps calibrated at 1 liter per minute (**L/m**). The sample tubes were analyzed for chlordane, Dursban, Diazinon, Baygon, and Ficam by gas chromatography with electron capture detection (**GC/ECD**). The analytical limit of detection (**LOD**) of this analysis for these insecticides were 0.01, 0.004, 0.002, 3.0, and 3.0 micrograms per sample (**ug/sample**), respectively. Analysis of the ORBO-42 samples was conducted in the following manner: The ORBO-42 tubes were prepared for analysis according to NIOSH Method S278 with modifications. The samples were desorbed in 1 milliliter (**ml**) of toluene and sonicated for 1 hour. The front and back sections of the tubes were desorbed and analyzed separately. Matrix standards were prepared and analyzed to assure method reliability. The samples were analyzed on a HP Model 5890 GC/ECD. A 6 foot x 4 millimeter (**mm**) inside diameter (**I.D.**) glass column packed with 1.5% SP-2250/1.95% SP-2401 was used isothermally at 214°C. P-5 was used as the carrier gas.

Technical chlordane was quantitated by summing the first three peaks in the standards and comparing sums of the first three peaks in the samples. The later peaks in the standards (including alpha and gamma chlordane) did not show up in the samples. This is probably because of their lower volatility and interferences from the PCB Aroclor 1254. Chlordane and Aroclor 1254 did not quantitate in the original run which had a higher LOD. The samples and lower standards were re-run to produce results. Quality control (**QC**) samples were not re-run at the lower range because they were acceptable with the original range of standards.

Surface contamination sampling for insecticides was performed using 2" x 2" Soxhlet extracted gauze pads impregnated with 8 ml of hexane. A 0.25 square meter (**m**²) area was wiped with the pads using gloves determined not to contain PCBs. The gauze samples were prepared for analysis by extraction in 40 ml of hexane with shaking for 30 minutes. The hexane extract was transferred to a concentrator tube and the gauze pad was rinsed twice with 10 ml of hexane. The concentrated hexane eluent was brought to a final volume of 2 ml. The samples were analyzed on a Hewlett-Packard Model 5890 GC/ECD. A 6 foot x 4 mm I.D. glass column packed with 1.5 percent SP-2250/1.95 percent SP-2401 was used isothermally at 222°C. P-5 was used as the carrier gas. The LOD for chlordane using this analytical procedure was 0.09 ug/sample, with a limit of quantitation (**LOQ**) of 0.3 ug/sample.

Polychlorinated Biphenyls (PCBs)

Air sampling for PCBs was accomplished using florisil as the solid sorbent sampling media and portable sampling pumps calibrated at 1 L/m for approximately 10 hours. Analysis of these samples was performed according to NIOSH Method 5503 using GC/ECD. The front and back section of the florisil tubes were desorbed separately in 1 ml of hexane with sonication for 30 minutes. The gas chromatographic analysis was performed on a Hewlett-Packard Model 5731A GC/ECD. A 25-foot x 0.31 mm fused silica WCOT capillary column coated internally with DB-5 was used with temperature programming from 210°C (held for two minutes) to 310°C at a rate of 8°C/minute. Five percent methane in argon was used as the carrier gas. The capillary injector was operated in the splitless mode.

The presence of an Aroclor was determined by comparison with standard samples of Aroclors 1016, 1221, 1232, 1242, 1248, 1254, and 1260 obtained from the EPA. Quantitation was performed by summing the peak heights of the standards and comparing those sums to the sums of the same peaks in the sample. Aroclor 1254 was the only PCB found in the samples collected during this evaluation. The LOD for this analysis was 0.05 ug/sample, with an LOQ of 0.2 ug/sample for Aroclor 1254.

Surface contamination sampling for PCBs was conducted using the same procedure as described above for insecticides. The LOD for Aroclor 1254 in this particular analysis was 0.2 ug/sample, with an LOQ of 0.6 ug/sample.

Volatile Organic Chemicals

The volatile organic chemical sampling was conducted using activated charcoal tubes and portable sampling pumps calibrated at 1 L/m. The charcoal tubes were submitted for qualitative analysis of volatile organic compounds by gas chromatography/mass spectrometry (**GC/MS**). The charcoal tubes were desorbed with 1 ml of carbon disulfide and screened by gas chromatography with flame ionization detection (**GC/FID**), using a 30-meter DB-1 fused silica capillary column (splitless mode). Representative samples were chosen for further analysis by **GC/MS** to identify contaminants.

C. Medical

Questionnaire Surveys

A medical questionnaire was administered to all employees at the school on three occasions. On January 12, 1989, the Kanawha County Health Department distributed a

self-administered questionnaire (Questionnaire 1), which inquired about medical symptoms and perceptions of air movement and other comfort conditions. The NIOSH investigators returned to the school on March 1, 1990, a few weeks following the reopening of the school after renovations were complete. A second questionnaire (Questionnaire 2) was administered that asked questions very similar to those in Questionnaire 1. The major difference between these two questionnaires was that Questionnaire 1 asked if the specific complaints had been a problem since the person began working at the school, while the NIOSH questionnaire asked only about complaints during the past week.

A third questionnaire (Questionnaire 3) was administered on June 8, 1990, during the final week of school. The goal of this questionnaire was to determine the status of complaints after the building had been occupied for four months. In this final questionnaire, employees were asked questions identical to those in Questionnaire 2. Questionnaire 3, however, inquired about complaints both <u>during the past week</u> and <u>during the past four months</u>. The two NIOSH questionnaires used a series of questions that had previously been used in two large surveys of 9000 individuals at other worksites.²

A medical symptom was considered present if the person experienced the complaint at least once a week. For the portion of Questionnaire 3 that asked about the past four months, a complaint was considered present if it occurred sometimes, often, or always, as opposed to never or rarely. Although previous interim reports defined a potentially work-related symptom as one that improved after leaving work, this requirement was not used in the analysis for this report. This was primarily because the local health department did not include that question for each symptom and, therefore, the symptom prevalences before and after renovation could not have been compared. A comfort complaint was considered present if the problem occurred often on Questionnaire 1 or often or always on the Questionnaires 2 or 3.

The symptom prevalences were compared for all respondents on all three questionnaires. Additionally, since there was turnover of between 25% and 30% between school year 1989 and 1990, the change in prevalences only among those employees who completed all three questionnaires was examined. This allowed for the determination of the change in health complaints before and after renovation only among those who were present at all three times. This also allowed the examination of whether individual complaint rates had changed in addition to changes in the rates for the group as a whole. McNemar's test was used to assess the statistical significance of changes in symptom prevalences among those who completed all three questionnaires.³

Blood Chlordane Metabolites

All current employees and two former teachers who had worked during the previous school year were offered blood testing. The blood samples were collected by a NIOSH technician on May 10, 1989. The levels of three chlordane metabolites (oxychlordane, heptachlor epoxide, and trans-nonachlor) were measured in the blood samples of the school staff by the Centers For Disease Control Environmental Health Laboratories using a previously described method with a limit of detection of 0.2 nanograms/milliliter.⁴

V. EVALUATION CRITERIA

A. Environmental Evaluation Criteria

As a guide to the evaluation of the hazards posed by work place exposures, NIOSH field staff employ environmental evaluation criteria for the assessment of a number of chemical and physical agents. These criteria are intended to suggest levels of exposure to which most workers may be exposed up to 10 hours per day, 40 hours per week for a working lifetime without experiencing adverse health effects. It is, however, important to note that not all workers will be protected from adverse health effects if their exposures are maintained below these levels. A small percentage may experience adverse health effects because of individual susceptibility, a pre-existing medical condition, and/or a hypersensitivity (allergy). In addition, some hazardous substances may act in combination with other work place exposures, the general environment, or with medications or personal habits of the worker to produce health effects even if the occupational exposures are controlled to the level set by the evaluation criterion. These combined effects are not often considered by the evaluation criteria. Also, some substances are absorbed by direct contact with the skin and mucous membranes, and thus potentially increase the overall exposure. Finally, evaluation criteria may change over the years as new information on the toxic effects of an agent become available.

The primary sources of environmental evaluation criteria for the work place are: 1) NIOSH Criteria Documents and Recommended Exposure Limits (**RELs**)⁵, 2) the American Conference of Governmental Industrial Hygienists' (**ACGIH**) Threshold Limit Values (**TLVs**)⁶, and 3) the U.S. Department of Labor, Occupational Health and Safety Administration (**OSHA**) Permissible Exposure Limits (**PELs**)⁷. Often, the NIOSH recommendations and ACGIH TLVs are lower than the corresponding OSHA PELs. The OSHA PELs may also be required to take into account the feasibility of controlling exposures in various industries where the agents are used; the NIOSH-recommended exposure limits, by contrast, are based primarily on concerns relating to the prevention of occupational disease. In evaluating the exposure levels and the recommendations for

reducing these levels found in this report, it should be noted that industry is legally required to meet those levels specified by an OSHA PEL.

A time-weighted average (**TWA**) exposure refers to the average airborne concentration of a substance during a normal 8- to 10-hour workday. Some substances have recommended short-term exposure limits or ceiling values which are intended to supplement the TWA where there are recognized toxic effects from high, short-term exposures.

B. Carbon Dioxide and Volatile Organic Chemicals

Carbon dioxide ($\mathbf{CO_2}$) is a normal constituent of exhaled breath that can be used as a screening technique to determine if adequate quantities of fresh air are being introduced into a building. It is important to note that $\mathbf{CO_2}$ concentrations are normally higher indoors than outdoors, even in buildings with few complaints. Approximately 35% of the expired breath of building occupants is $\mathbf{CO_2}$. It is the general consensus of IAQ investigators that if the indoor concentration of $\mathbf{CO_2}$ is more than 1000 ppm, or 3 to 4 times the outdoor level, ventilation in the occupied space may be inadequate.⁸ In these situations, there may be complaints of headache, fatigue, and eye and throat irritation. The $\mathbf{CO_2}$ concentration itself is not responsible for the complaints, but the elevated $\mathbf{CO_2}$ concentrations indicate that other contaminants which can cause the symptoms may also be present at increased concentrations.

Volatile organic chemicals (**VOC**) are monitored in IAQ evaluations, in addition to CO₂, to provide a qualitative understanding of the variety of chemicals which exist in the indoor environment. Comparison of VOCs in complaint versus non-complaint areas can indicate elevated pollutant levels, point sources of chemical contamination, and/or inadequate ventilation. VOCs found in indoor environments result from the use of cleaning compounds, perfumes, waxes, paints, furnishings, and various occupant activities. Poor ventilation of occupied spaces can result in increased concentrations of VOCs.

C. <u>Temperature and Relative Humidity</u>

The American Society of Heating, Refrigeration, and Air-Conditioning Engineers (**ASHRAE**) has published guidelines describing thermal environmental conditions (ASHRAE Standard 55-1981, Thermal Environmental Conditions for Human Occupancy). These guidelines specify conditions for which 80% or more of the occupants will be expected to find the environment thermally comfortable. Figure 1 was extracted from this document and presents the acceptable ranges of temperature and humidity according to ASHRAE (73° and 77° F, or 23° to 25° C, and 20% to 60% relative humidity).

D. Ventilation

The criteria used by NIOSH investigators to judge ventilation adequacy are guidelines proposed by the ASHRAE in their ASHRAE Standard 62-1989.¹⁰ For educational facilities, ASHRAE recommends 15 cubic feet per minute (**cfm**) of fresh air per person be provided to classrooms, libraries, music rooms, and auditoriums.

E. Insecticides

Chlordane is a chlorinated cyclodiene which is very resistant to chemical destruction. At room temperature, technical grade chlordane is a thick amber liquid with a chlorine-like odor and a very low vapor pressure. Chlordane is rapidly absorbed following dermal contact, ingestion, or inhalation. Once inside the body, chlordane and its metabolites are deposited in the body fat and have a biological half-life of several weeks. Due to the widespread application of chlordane and its long half-life, chlordane is found in the tissue of the general population. ¹¹

A variety of studies have looked at the health effects of exposure to chlordane in humans and are reviewed in detail in Appendix 1. Acute poisoning associated with very high levels of exposure during spraying, manufacture, or accidental ingestion of chlordane produces central nervous system symptoms, including headache, blurred vision, dizziness, slight involuntary muscle movements, tremor, sweating, insomnia, nausea, and general malaise.¹² Studies of workers exposed to lower levels of chlordane have not found any nervous system effects; however, these studies have not looked for subtle early signs of neurologic disfunction. Animal studies indicate that the liver can be affected and may be the most sensitive organ following chronic exposure to levels not producing overt symptoms of toxicity. 13 Human studies have not found toxic effects to the liver except in a few cases of very high exposures. As with the neurological effects, some subtle signs of liver damage may have been missed because of testing that is either unavailable or too invasive (such as a liver biopsy). Chlordane was evaluated for carcinogenicity by the National Cancer Institute and found to be carcinogenic in mice, with a high incidence of hepatocellular carcinomas.¹⁴ Studies of human populations exposed to chlordane have not identified any increased incidence of cancers as compared to national rates.¹⁵

The existing OSHA PEL for chlordane is 500 micrograms per cubic meter (**ug/m³**) of air. The current ACGIH TLV for chlordane is also 500 ug/m³. Both the OSHA and ACGIH exposure criteria address skin exposure and are intended for the control of occupational exposures. According to the OSHA Cancer Policy, a chemical compound found to cause cancer in animals is to be considered a potential human carcinogen. Therefore, NIOSH recommends that exposure to chlordane be reduced to the lowest feasible level since safe

levels of exposure to carcinogens have not been demonstrated.⁵ In 1979, the National Research Council's (**NRC**) Committee on Toxicology suggested an interim guideline of 5 ug/m³ for the upper limit of airborne chlordane in military housing.¹²

Chlorpyrifos (Dursban) and Diazinon are organophosphate pesticides which can be absorbed through the skin, by inhalation, or ingestion. The principal toxic effect of these pesticides in humans and animals is a reduction in plasma and red-cell cholinesterase activity. There is no information on the chronic effects of long-term exposure of humans to these insecticides. The OSHA PEL and ACGIH TLV for Chlorpyrifos is 200 ug/m³ with attention to the skin absorption potential.^{6,7} The OSHA PEL and ACGIH TLV for Diazinon is 100 ug/m³, also with notation on the dermal absorption potential.^{6,7}

F. Blood Levels of Chlordane Metabolites

As a result of the metabolic breakdown of chlordane in the human body, the levels of blood chlordane metabolites are a more accurate reflection of chronic low-level exposure to chlordane than is the level of blood chlordane. Since chlordane is stored for many years in the fat tissues, a detectable level of chlordane in fat tissues indicates exposure to chlordane but may not indicate exposure sufficient to cause health effects. Measuring blood chlordane metabolites is a more practical means of screening for significant exposure to chlordane as compared to analysis of adipose (fat) tissue, which must be obtained by biopsy.

G. Polychlorinated Biphenyls (PCBs)

PCBs are chlorinated aromatic hydrocarbons that were manufactured in the United States from 1929 to 1977 and marketed under the trade name Aroclor. PCBs found wide use because they are heat-stable, resistant to chemical oxidation, acids, bases and other chemical agents, stable to oxidation and hydrolysis in industrial use, have low solubility in water, low flammability and favorable dielectric properties. Additionally, they have low vapor pressure at ambient temperatures and viscosity-temperature relationships which were suitable for a wide range of industrial applications. PCBs have been used commercially for insulating fluids for electrical equipment, hydraulic fluids, heat transfer fluids, lubricants, plasticizers, and components of surface coatings and inks. The PCB mixtures marketed under different trade names are often characterized by a four-digit code number. The first two digits denote the type of compound, with "12" indicating biphenyl, and the latter two digits giving the weight percentage of chlorine.

The International Agency for Research on Cancer (IARC) has concluded that the evidence for PCBs' carcinogenicity to animals and to humans is limited. IARC has reported that

"certain polychlorinated biphenyls are carcinogenic to mice and rats after their oral administration, producing benign and malignant liver neoplasms. Oral administration of polychlorinated biphenyls increased the incidence of liver neoplasms in rats previously exposed to n-nitrosodiethylamine". ²⁰

In a mortality study among workers at two capacitor manufacturing plants in the United States, a greater than expected number of observed deaths from cancer of the liver and cancer of the rectum were noted.²¹ Neither increase was statistically significant for both study sites combined. However, in a recent update of this study, with follow-up through 1982, the excess in liver/biliary tract cancer was statistically significant (5 observed versus 1.9 expected deaths); and the rectum cancer was still elevated but not statistically significant. In a mortality study among workers at a capacitor manufacturing plant in Italy, males had a statistically significant increased number of deaths from all neoplasms.²² When analyzed separately by organ system, death from neoplasms of the digestive organs and peritoneum (3 observed versus 0.88 expected) and from lymphatic and hemopoietic tissues (2 observed versus 0.46 expected) were elevated. This study was recently expanded to include all workers with one week or more of employment with vital status follow-up through 1982. In the updated results, there was a statistically significant excess in cancer among both females (12 observed versus 5.3 expected) and males (14 observed versus 7.6 expected). In both groups there were non-significant excesses in lymphatic/hematopoietic cancer and statistically significant excess in digestive tract cancer among males (6 observed versus 2.2 expected). Unfortunately, not enough information was provided to determine the risk specifically for liver cancer.

In February 1986, NIOSH reiterated its previous recommendation that exposure to PCBs in the workplace not exceed 1 ug/m³ (based upon the recommended sampling and analytical method in use at that time), determined as a TWA for up to a 10-hour workday, 40-hour workweek.²³ This recommended exposure limit was based on the findings of adverse reproductive effects in experimental animals, on the conclusion that PCBs are carcinogenic in rats and mice (and are therefore potential human carcinogens in the workplace), and on the conclusion that human and animal studies have not demonstrated a level of exposure to PCBs that will not subject the worker to possible liver injury.²⁴

In 1971, based on the 1968 ACGIH TLVs, OSHA promulgated its Permissible Exposure Limits of 1 milligrams per cubic meter (**mg/m³**) for airborne chlorodiphenyl products (PCB) containing 42% chlorine and 0.5 mg/m³ for chlorodiphenyl products containing 54% chlorine, determined as 8-hour TWA concentrations (29 CFR 1910.1000).⁷ The ACGIH TLVs and OSHA PELs which have remained unchanged through 1990, are based on the prevention of (non-carcinogenic) systemic toxicity. The ACGIH TLVs and OSHA PEL values include a "skin" notation, which refers to the potential contribution to the overall

exposure by the cutaneous route, including the mucous membranes and the eyes, by either airborne or direct skin contact with PCB.⁶

This "occupational" exposure criteria is not meant to be applicable to the general population, such as this case where large numbers of young people have potential exposures. Definite causal relationships between exposure and carcinogenic effects in humans remains unclear due to the inadequately defined study populations and the influence of mixed exposures. The overall toxicity of PCBs in humans during physiologically developmental stages is even more uncertain.

VI. <u>RESULTS AND DISCUSSION</u>

A. Environmental

Ventilation and Thermal Comfort

CO₂, temperature, and relative humidity measurements were collected during this evaluation to provide information on general ventilation effectiveness and thermal comfort conditions. Table 1 provides the CO₂, temperature, and relative humidity measurements by location as collected on April 11 and 19, 1989. These parameters provide information on the general air quality and thermal comfort conditions for these areas on these dates. CO₂ measurements collected on April 11 ranged from 700 to 1500 ppm indoors and 200 to 250 ppm outdoors. CO₂ levels obtained on April 19 ranged from 500 to 1200 ppm indoors and 200 to 300 ppm outdoors. The indoor CO₂ levels, when compared to the outdoor levels, indicate that several of the classroom areas were not adequately ventilated (insufficient fresh air) on the days when the measurements were taken. The temperature and relative humidity measurements show that conditions in some of the classroom areas fell outside the comfort ranges prescribed by ASHRAE in Figure 1. However, these are typical levels not unusual during the transition from heating to cooling seasons. Indoor relative humidity will change with outside conditions, unless the building HVAC system is capable of adding or removing moisture. The perception of comfort is related to one's metabolic heat production, the transfer of heat to the environment, physiological adjustments, and body temperature. Heat transfer from the body to the environment is influenced by environmental factors such as temperature, humidity, and air movement, as well as personal factors such as clothing and activity.

Table 2 provides the CO₂, temperature, and relative humidity measurements collected on March 1, 1990. The CO₂ measurements throughout the school

building ranged from 400 to 800 ppm, with an outdoor CO₂ concentration of 350 ppm on this date. By comparison, the indoor CO₂ levels measured on April 19, 1989, ranged from 500 to 1200 ppm. The reduction in CO₂ concentrations resulting from enhanced ventilation is shown in Figure 2 by comparing pre- (April, 1989) and post-renovation (March, 1990) concentrations found in specific rooms. The CO₂ results collected on March 1, 1990, indicate that the new ventilation system was effectively ventilating the classrooms during occupancy. The temperature and relative humidity measurements were all within the comfort range prescribed by ASHRAE.⁹

Direct measurement of classroom ventilation rates were made on May 10, 1989 (before renovation of the air handling systems) and March 1, 1990 (after the renovation), using an Alnor Balometer. The measured rates found during these two surveys, in cfm, as well as estimates of the amount of outside air delivered to each classroom and amount of fresh air per person in the room, are presented for comparison in Table 3. The amount of outside air was calculated based upon an estimated 23% of the total supply infiltrating through the dampers for each air handler. The estimate was provided by representatives of the ventilation system contractor for the school board. The amount of outside air per person was based upon an estimated classroom occupancy of 20.

In May 1989, the range of air supply rates for the classrooms was from 0 to 1160 cfm, resulting in an estimated range of 0 to 13 cfm of fresh air per person in the classrooms. The rates were quite variable throughout the classrooms surveyed. At the time of the May 1989 survey, the criteria used by NIOSH to judge ventilation adequacy were the guidelines proposed by the ASHRAE in their ASHRAE Standard 62-1981R, a proposed revision of 62-1981 later adopted as 62-1989, Ventilation for Acceptable Indoor Air Quality. For educational facilities, ASHRAE recommended that 15 cfm of fresh air per person be provided to classrooms, libraries, music rooms, and auditoriums, and 20 cfm per person for laboratories and training shops. As can be seen from Table 3, there were no classrooms in May 1989 which met the ASHRAE guideline of 15 cfm per person; although five of the 26 measurements from 1989 listed in Table 3 were between 12 and 13 cfm per person, most were well below the guideline.

Table 3 also provides the results of the classroom ventilation rates as determined on March 1, 1990. For these measured rates, the amount of outside fresh air was calculated based on an estimated 25% of total supply for the March 1, 1990, survey. This was the minimum thermostatically controlled set point for the fresh air dampers during occupancy and represents the worst possible operating conditions

in this case. Again, the amount of outside air per person was based upon an estimated classroom occupancy of 20.

It is evident by comparing the results from both surveys in Table 3 that there was a substantial improvement in the ventilation and amount of fresh air provided to a classroom. None of the classrooms met the ASHRAE requirements in May 1989 while in March 1990 there was a substantial improvement when the minimum occupied setting of the fresh air damper was used to calculate the fresh air rate. Figure 3 graphically presents the fresh air ventilation rates of selected rooms for comparison of pre- and post renovation rates.

In addition to inadequate outside air supply, there were other problems with the ventilation systems in use at Andrew Jackson Junior High School prior to the renovation. The more notable were the inability to provide suitably filtered air and an imbalance of the systems that resulted in the highly variable supply flow. The air handling units also were exceedingly dirty and corroded, which impaired their efficiency and performance. The excessive number of missing ceiling tiles also impeded the circulation and distribution of air by the ventilation system.

Volatile Organic Compounds (VOCs)

A total of eight samples for VOCs were collected in classrooms 401, 402, 203, 201, and outdoors during the April 11, 1989, survey. Examples of the qualitative analysis results of the VOCs during this sampling are depicted in Figures 4, 5, and 6. These figures represent reconstructed ion chromatograms for samples collected in classroom 401, classroom 203, and outdoors, respectively. These two classrooms represent rooms in the mid-range of ventilation rates. Figure 4 shows that classroom 401 had the highest level of contaminants and more variety of organic compounds as compared to classroom 203 and the outdoors. When compared with the outdoor VOC results, the indoor VOC sampling verified the indication from CO₂ and ventilation measurements of deficient ventilation of the classroom areas which allowed chemical contaminants to build up during occupancy. The VOC results for the other areas were similar and support this conclusion. The sources of VOCs found in an indoor air environment include as examples: cleaning compounds, perfumes, dry cleaned garments, paint, photocopying machines, office supplies, and furnishings.

A total of 10 samples for VOCs were collected in classrooms 101, 104A, 201, 203, 301, 303, 401, 402, 403 and the cafeteria during the March 1, 1990, survey. The results of the total ion chromatograms for these samples identified very low levels of toluene, limonene, trichloroethylene, isobutane, and siloxane. As examples, Figures 7 and 8 represent reconstructed ion chromatograms for the samples collected in classrooms 401 and 203, respectively, and are provided for comparison with Figures 4 and 5. While the levels were much lower in March 1990, the variety of VOCs found in these samples are similar to those

found on VOC samples collected on April 1989. The VOCs, and their respective concentrations, found on both sample sets, are typical of those in indoor air samples collected during other NIOSH indoor air evaluations. There were no VOCs found in these samples at concentrations which would be expected to result in adverse health effects. In consideration of the new paint, flooring, and extensive cleaning materials used, the extent and variety of VOCs from March 1990 sampling indicates that the new ventilation system was effectively diluting chemical contaminants in the building.

Carpet Contamination

Carpet samples collected on April 11, 1989, from classrooms 401 and 402 contained approximately 16% by weight of chlordane. These samples, however, were obtained at the injection hole in the concrete slab and thus represented a worst case sample. Table 4 presents the results of the carpet sample analyses for the pesticides chlordane, Diazinon, and Dursban. The carpet samples were collected to determine the extent of pesticide contamination in classroom areas where these pesticides were reported to have been applied by pouring and spraying on the carpet. Also reported in Table 4 are concentrations of the PCB Aroclor 1254 identified during the analysis for pesticides.

While these results indicate the carpeting of the school was not as extensively contaminated with chlordane as suspected, there was considerable Diazinon contamination of carpeting throughout the school. Detectable concentrations of Diazinon [ranging from trace concentrations to 76 micrograms of contaminant per gram of carpet (**ug/g**)] were found in 16 of 17 samples collected. Diazinon is an organo-phosphorus pesticide with a category III EPA toxicity rating (i.e., of low-level dermal and inhalation toxicity).

Of more immediate concern, because of their toxicity, were the unexpected PCB concentrations found in the carpeting. Detectable concentrations (trace to 67 ug/g) of the PCB Aroclor 1254 were found on 15 of 17 samples collected. The most likely cause of the PCBs found in the carpet appeared to be from fluorescent light ballast burnouts; there had been no known transformer spills or fires, contaminated ceiling tiles, or other PCB sources in the school.

Surface Wipe Sampling

Because of the skin absorbtion potential of pesticides and PCBs, surface sampling was conducted throughout the classrooms for these compounds. The results of the surface wipe sample analyses for the pesticides chlordane, Diazinon, and Dursban collected on May 10, 1989, are presented in Table 5. Trace concentrations of chlordane were found on high dermal contact surfaces (student desk tops) in three classrooms (401, 402, and 502). Detectable concentrations (trace to 0.2 ug/ft²) of Diazinon were found on 9 of 14 high

contact surfaces and all 5 low dermal contact surfaces. Trace to $0.14~\text{ug/ft}^2$ concentrations of Dursban were found on 5 of 14 high contact surfaces, and $0.05~\text{ug/ft}^2$ was found on 1 of 5 low contact surfaces. Also reported in this table are concentrations of the PCB Aroclor 1254 identified during the analysis for the pesticides. Aroclor 1254 was found on all 19 surfaces sampled, with concentrations ranging from 2.2 to 35.5 ug/m^2 .

Table 6 presents the results of surface wipe sampling conducted on September 13, 1989, as a follow-up survey to verify the effectiveness of the cleanup phase prior to the next phases of the renovation project. There were no detectable concentrations of chlordane, Diazinon, or Dursban found on the 19 surfaces sampled. Aroclor 1254 was found on all 19 surfaces sampled and at concentrations (3 to 676 ug/m²) generally greater than those found on May 10, 1989. There were four likely reasons for these persistent levels. First, several of the surfaces sampled appeared to still have a residue of film left from the cleanup procedure. Many of the surfaces may have had several layers of wax or polish which could absorb the PCBs and increase the difficulty in effectively removing the contaminant. Second, there was also a considerable amount of dust on many of the sampled surfaces which may also have influenced the PCB surface contamination results. Third, several surfaces were sampled which did not appear to have been cleaned (e.g., window ledges in rooms 401 and 402, the shelf top in room 502, and the counter top in the cafeteria). Finally, many of the surfaces sampled during this survey were not sampled during the May 10, 1989, survey.

To verify the effectiveness of the second decontamination cleaning effort, ten surface wipe samples were collected on March 1, 1990, from surfaces which had been previously sampled. These surfaces had been previously identified as contaminated with PCBs and the pesticide Diazinon. Table 7 provides the PCB results of March 1, 1990, surface sampling and, for comparison, results from the previous surveys. The PCB surface levels found March 1, 1990, are low-level and are considered in the "background" range.

Surface wipe sampling for pesticides on the same surfaces reported in Table 7 were non-detectable for Dursban and Diazinon. Trace concentrations (less than 0.1 micrograms per sample) of chlordane were found on a student desk top in Room 402 and a counter top in Room 105. These surfaces had no detectable levels of chlordane in September 1989. Trace concentration values are between the LOD and the LOQ for the method.

The results of the surface wipe sampling indicate that rooms 401, 402, and 502 had only trace quantities of chlordane surface contamination at the locations sampled. Low levels of Diazinon and Dursban were found on surfaces in these and several other rooms throughout the school building. Also, low level PCB concentrations were found on various surfaces sampled throughout the school. These PCB results, in conjunction with the carpet sample

results previously reported, indicate the wide extent of PCB contamination in the school. As with chlordane, NIOSH considers PCBs to be a potential human carcinogen and recommends that exposure be controlled to the lowest feasible level.

Air Sampling for PCBs and Pesticides

The results of the air sampling for the insecticides chlordane, Diazinon, and Dursban on April 19, 1989, are presented in Table 8. Sampling also was conducted for the insecticides Baygon and Ficam; however, there were no detectable levels of these two insecticides. Detectable levels of chlordane and Diazinon were found in classrooms 401, 402, 309, 502, and 104B. Dursban was found in only classrooms 401 and 402. Air concentrations of chlordane were the highest (0.02 to 55.3 ug/m³) and of the most concern of the three insecticides found. While these levels did not exceed any occupational exposure criteria, two of the chlordane levels (10.5 and 55.3 ug/m³) exceeded the National Research Council's recommended residential level of 5 ug/m³ and represented an unnecessary exposure to the teachers and students who occupied these classrooms.

Table 9 provides the results of the air sampling for chlordane and Aroclor 1254 on May 10, 1989. The results of this air sampling indicate that the potential for low-level breathing zone exposure to chlordane (0.15 to 0.33 ug/m³) existed throughout the school environment. These levels represented conditions of typical ventilation of the building and no activity in the school building during sampling. Trace quantities of PCBs were found in these air samples and were considered an indication that PCB contamination of the air was present (because the sampling media in use at the time of this survey was more suitable for pesticides than PCBs). None of the air samples from this survey contained detectable levels of Diazinon or Dursban.

On September 13 and 14, 1989, air sampling for chlordane and PCBs was conducted to verify the effectiveness of the decontamination efforts prior to the next phases of renovation (painting, installation of new AHUs and ventilation systems, and new carpeting). The results of this air sampling effort are presented in Table 10 and indicate that only trace concentrations of chlordane and Aroclor 1254 could be found after collecting a very large sample volume. The range of these trace concentrations for chlordane were 0.12 to 0.36 ug/m³, and 0.09 to 0.31 ug/m³ for Aroclor 1254. On first appearance, the high end of these sample result ranges appear as high or higher than those reported from the May 10, 1989, survey (see Table 9). It should be noted, however, that these are considered trace quantities since they fall between the analytical limits of detection and quantitation, and the sample volume was 1.5 to 2 times that collected during the May 1989 survey. It is very likely that had the sample volumes been similar to those collected in May there would have been many non-detectable results from this survey.

Air samples for PCBs and the pesticides chlordane, Diazinon, and Dursban were collected on March 1, 1990, in 12 classrooms, the library, the cafeteria commons, and the kitchen to verify the effectiveness of the decontamination and ventilation renovation. Pesticides were not detected in any air sample. Two air samples for PCBs were found to have trace concentrations (less than 0.04 micrograms per sample) with all other samples having non-detectable levels. There apparently was no substantial residual air contamination by PCBs or pesticides in the school building.

B. Medical Results

Questionnaires

The questionnaires were completed by 43 workers in January 1989 (Questionnaire 1), 42 workers in March 1990 (Questionnaire 2), and 45 workers in June 1990 (Questionnaire 3). Three of the respondents to Questionnaire 1 were not working at the school at the time and were excluded from this analysis. The exact response rate for the Kanawha County Health Department questionnaire (Questionnaire 1) was not given, but is estimated to be over 90%. The response rate for Questionnaires 2 and 3 was 100% of all employees who were present the day of the survey. The majority of respondents were teachers (approximately 70% of each survey) and the distribution of respondents by occupation is shown in Table 11. Figure 9 shows the average distribution of respondents to the questionnaire by job title. Of all the respondents, 25 of 59 individuals (42%) completed all three of the questionnaires. The major reason for not completing all three questionnaires was leaving employment at Andrew Jackson between school year 1989 and 1990; 19% were new employees in 1990 and 20% were employees who left the school after 1989.

The questionnaire data collected by the Kanawha County Health Department was initially analyzed by NIOSH investigators in June 1989 to determine if certain areas of the school had more health complaints. The school was divided into 6 areas based on the ventilation zones of the school. Custodians were considered to be in a separate area because they spent time in all parts of the school. These results are found in Table 12. This table shows that there were numerous health complaints and that these complaints were found throughout the school. The small number of workers in each zone makes it inappropriate to evaluate the distribution statistically; however, the types of complaints mentioned most commonly by the employees (upper airway and mucous membrane irritation, respiratory problems, headache, muscle aches, fatigue, problems with concentration, and tension) can be due to a variety of causes and are not suggestive of any specific environmental contaminant. These complaints are similar to those found in other buildings and schools that NIOSH has investigated for suspected IAQ problems.

Table 13 shows the prevalence of medical symptoms at each of the three questionnaire times. Prevalence rates are given both for the last week and the last 4 months for the questionnaire administered in June 1990. The prevalence of all symptoms decreased by 50% or more after the renovations. Although the prevalences increased slightly between March and June, most remained much lower than prior to the renovations. The matched analysis of the 25 who answered all three questionnaires (Table 14) also shows a consistent pattern of decrease in symptom rates after renovations. Figure 10 graphically presents the change in symptom response rates for respondents to all three questionnaires, where greater than 50% of the respondents in January, 1989, identified a problem. Statistical analysis by McNemar's test was used to compare the pre-renovation question to each of the post-renovation questionnaires, which showed that although only a few of these changes were statistically significant all of the changes were in the direction of improvement.

Table 15 shows the prevalence of comfort complaints for the three questionnaires. Table 16 shows the prevalence just for the 25 individuals who completed all three questionnaires. Figure 11 illustrates the changes in prevalence of environmental complaints as reported by these 25 individuals. There was a statistically significant decrease in complaints of too little air, air that was dusty or stuffy, and temperature complaints. There was some change in the rate of temperature complaints between March and June, which reflects the change in seasons. Tables 17 and 18 show the prevalence for complaints related to the individual's workspace. There was a significant decrease in complaints related to control of temperature and the lack of windows. In reality, the teachers had no more control of temperature, and no new windows were added. This change, however, reflects the staff's improved sense of comfort in the work environment.

Blood Chlordane Measurements

Blood levels of chlordane metabolites was determined for 49 individuals employed at Andrew Jackson Junior High School, including office staff, custodial staff, kitchen staff, and classroom teachers. This group included all but one of the current staff members and two former teachers. The results of this testing are shown in Table 19. The tests showed that forty-four (90%) of the staff members had levels that were below the limit of detection and the remaining had levels ranging from 0.23 to 1.2 nanograms per milliliter (**ng/ml**). These levels are far below any that has been shown to cause adverse health effects in medical studies reported in the literature. For example, a study undertaken by the Centers for Disease Control in a community near a toxic waste site containing chlordane, found no significant adverse health effects at blood chlordane levels up to 9 times higher than any found in the school staff. ²⁵

A study conducted by the EPA in 1976 found that 4-6% of the general population had detectable levels of blood chlordane or a chlordane metabolite. Among the Andrew Jackson School employees only one person, or 2% of the school staff, had a level that was high enough to have been detectable by the methods used during the EPA study. There was no relationship found between number of years working in the school and the level of blood chlordane metabolites. Also, those working in rooms with the highest levels of air chlordane did not consistently have higher blood chlordane levels as compared to those working in rooms with lower air chlordane levels. Thus, it is believed that these results are consistent with the variability of blood chlordane metabolites found in the general population and cannot be related to working in Andrew Jackson Junior High School.

Individual Interviews

During the initial visit on April 11 and 12 1989, interviews were held with 14 individuals, of whom 11 were teachers and 3 were parents. These individuals were chosen because they or their children were having health problems. Medical records of 11 of the individuals were requested from their physicians. The medical records from 11 individuals who were staff or students at Andrew Jackson Junior High School were reviewed. These records do not suggest any adverse health effects that were likely the result of direct toxic effects from chronic low level exposure to pesticides. Some of the medical complaints expressed by the staff members (upper airway and mucous membrane irritation, respiratory problems, headache, muscle aches, fatigue, problems with concentration, and tension) were similar to those found in the questionnaires and are discussed above.

VII. <u>CONCLUSIONS</u>

Based on the results of the environmental and ventilation measurements, there was an apparent lack of fresh air being introduced into the classroom areas of this building. The types of health complaints found in the questionnaire were similar to those found in other studies where there was a lack of fresh air. There also appeared to be poor distribution of air throughout the classroom space. Because of the deficiencies in the ventilation of the occupied spaces, common air contaminants would have increased during occupancy and may not have been effectively diluted. Unnecessary inhalation and dermal exposure potential to chlordane, Diazinon, and Dursban existed in classrooms 401, 402, 309, 502, and 104B. This exposure potential was exacerbated by the inefficiency of the ventilation system. It is important to emphasize, however, that other epidemiological studies which have looked at detrimental health effects of low-level chronic exposure to chlordane, similar to levels found in this school, have not found significant adverse health effects. Unnecessary inhalation and dermal exposure potential to the PCB Aroclor 1254 also existed throughout the school building. A lack of adequate dilution ventilation in this school building permitted elevated concentrations of this PCB.

Based on the sampling results collected afterward, the decontamination and renovation of the building was apparently successful in eliminating continued inhalation and dermal exposure potential to insecticides and PCBs.

VIII. RECOMMENDATIONS

The following recommendations were offered to **maintain** acceptable indoor air quality in this school building.

- 1. The ventilation of the occupied spaces in this school building should meet the specifications outlined in ASHRAE Standard 62-1989, Ventilation for Acceptable Indoor Air Quality.⁷
- 2. The ventilation systems should be placed on a scheduled and systematic maintenance program to ensure the systems continue to operate as designed, are efficient, and are kept clean. Filter changes, fan belt changes, cleaning, and repairs should be documented by keeping a maintenance log or work order system.
- 3. Because food is prepared in the cafeteria of this school, an effective sanitation program may require the use of insecticides. The application of insecticides should be performed by a licensed applicator who maintains records detailing the type of insecticide used, purpose for use, location, amount used, type of application procedure, and any health or safety recommendations involved to complete the application.
- 4. To minimize the dust exposure of the custodial staff during vacuuming and changing of vacuum cleaner bags, all vacuum cleaners should be equipped with disposable bags. The vacuum cleaners in use during the NIOSH survey utilized re-usable bags, which the custodians had to "shake out", resulting in unacceptably high dust exposures. These also generated dust during vacuuming because of the general condition of the bags.

The following recommendations were offered in the May 4, 1989, letter to improve the indoor air quality in this school building, reduce complaints regarding comfort and stale air, and prevent unnecessary exposure to chemical contaminants.

1. The building HVAC system should be fully evaluated by ventilation experts who are knowledgeable of the ASHRAE standards for indoor air quality and comfort. These experts should ensure proper ventilation balance, distribution of air, and adequate intake of fresh air for the various ventilation zone systems and the level of occupancy for the areas serviced by these systems. This evaluation should be in accordance with the ASHRAE Standard 62-1981R, "Ventilation for Acceptable Indoor Air Quality", which contains proposed requirements of 15 to 20 cfm/person for classroom areas and 60 cfm/person for smoking areas. Since they were not designed to do so, it is possible that the current HVAC systems cannot be balanced or modified to effectively ventilate the structural

- classroom space. The ceiling tiles should be replaced, and if additional return air grids are needed they should be positioned to effect proper mixing of room air.
- 2. The building custodian who adjusts the thermostats, and the building engineers who service the AHU on the roof, should be fully trained in the operation of these specific units by the manufacturer/installer. This training should include methods on how to adjust the delivered air temperature due to seasonal fluctuations in outdoor temperature.
- The AHUs should be thoroughly cleaned. The fiberglass sound linings in some of the units
 are deteriorating and should be removed and replaced. A log of filter changes should be
 created to document the frequency of changes and any unusual events concerning the
 HVAC systems.
- 4. The leaks in the roof should be repaired when school is not in session to avoid unnecessary exposure to fumes, dust, and noise generated by this operation. The ceiling tiles which were removed due to the roof leaks should be replaced immediately to avoid compromising the circulation and distribution efficiency of the HVAC system. Walkways should be constructed on the roof to prevent further damage to the roof resulting in more leaks.
- 5. The wood shop return air vents should be examined to determine their effect on the circulation of air in the adjacent air handling zone.
- 6. A local exhaust hood is needed over the pottery kiln in the art room to effectively remove any fumes, odors, or vapors from this operation. The current wall mounted exhaust fan is not capable of removing these contaminants and they may be recirculated in the HVAC system.
- 7. A ventilated storage cabinet (design approved by the National Fire Protection Association) should be used to store the various flammable compounds found in the art classroom and the science laboratory. This cabinet should be exhausted to the outside to prevent vapors from these materials from entering the HVAC system. The chemicals and solvents used in these two classrooms should be stored in a secure manner. The chemical storage area for the science laboratory was unlocked and unattended during our survey.
- 8. The existence of fluorescent light ballasts which contain PCBs in the building was determined during this evaluation. The ballasts are approaching the end of their service life (12 18 years) at which time they may burn out and release PCBs into the school environment. PCBs are considered by NIOSH to be potential human carcinogens; the acute effects of exposure to the smoke from a ballast burnout include headache, eye irritation, sore throat, nasal congestion, and nausea. NIOSH recommends that all light ballasts containing PCBs be identified, removed, and replaced with PCB-free light ballasts before burnout occurs. For easy identification, EPA regulations require that PCB-free light ballasts manufactured since July, 1978, be marked "No PCBs."

9. The vacuum cleaners should be equipped with disposable dust collection bags versus the re-usable bags observed during this evaluation. If these vacuum cleaners cannot accommodate disposable bags, new vacuum cleaners should be purchased. The practice of shaking out re-usable bags contributes to the exposure potential of the custodial staff.

The following recommendations were offered in the May 4, 1989, letter to prevent further exposure to residual insecticides in the school environment.

- 1. Classrooms 401, 402, 309, 104B, and 502 should be vacated immediately to prevent further unnecessary exposure to chlordane and Diazinon. The carpeting in classrooms 401, 402, 309, 104B, 502 and the hallway outside of these rooms should be removed, labeled as containing chlordane, and disposed of properly according to State requirements.
- 2. The floors, walls, and horizontal surfaces in these rooms should be thoroughly wiped down using an organic solvent such as odorless mineral spirits. The cleanup crews should wear half-face respirators with organic vapor canisters during this portion of the cleanup to minimize exposure to the solvent. This initial cleaning should be followed by cleaning with a nonionic detergent rinse containing 5% octylphenoxypolyethoxyethanol and trisodium phosphate. This wash solution will remove the grease, wax, and furniture polishes which may absorb chlordane from the air. Finally, the cleanup should be finished with a water rinse. Nitrile or neoprene rubber gloves should be worn by cleanup crews to protect them from chlordane exposure during the cleanup.
- 3. The cracks in the floors should be sealed with a silicone based caulk to prevent chlordane from entering the classroom space from the subfloor area. The cork used to seal the injection holes should be removed and the holes resealed with silicone caulk.
- 4. Prior to refurbishing the areas with new carpeting, air sampling and surface sampling for chlordane should be conducted. This sampling will verify the effectiveness of the cleanup and provide a decision point for reoccupancy.

The following recommendations were offered in the June 30, 1989, letter in response to the Kanawha County Board of Education's request on how to proceed with the cleanup of the Andrew Jackson Junior High School. The first set of recommendations were presented in a sequence believed to be a logical approach. These recommendations were provided as a guide, not as a rigid plan, and were to be modified as conditions directed during the cleanup.

Remove any insulated duct work which may possibly be contaminated with PCBs or
pesticides and replace with temporary duct work. Treat as if contaminated and dispose
according to state requirements of West Virginia. Fully open fresh air intake dampers and
operate fan units on air handlers in continuous mode to provide as much ventilation as
possible to the space during clean up. Check the filters on a daily basis and replace or
clean as needed.

- 2. Cut carpeting at juncture of wall and floors in the rooms and hallways (no need to remove the walls). Roll up carpeting, remove from the school by most direct route, and dispose of per state requirements. This should be accomplished by blocks of rooms (i.e., 401, 402, 403, 404, 309, 308, and 307) to facilitate initiating the next phase of clean up in a block of rooms while carpeting is removed from another area.
- 3. Remove dust and debris from the area by using a vacuum, not by sweeping. A high efficiency particulate air (HEPA) filtered vacuum may not be necessary for this job, but if available the HEPA vacuums would be advisable.
- 4. As a minimum, the floors, walls, and furnishings in 401, 402, and 502 should be thoroughly wiped down using odorless mineral spirits. This initial cleaning should be followed by cleaning with a nonionic detergent rinse containing 5% octylphenoxypolyethoxyethanol and trisodium phosphate. This wash solution will remove the grease, wax, and furniture polishes which may absorb chlordane and PCBs. Finally the cleanup should be finished with a water rinse. All areas where cracks are found in the floor after the carpet is removed should be cleaned using the same procedure. To fully assure that PCBs are removed from the school, the walls, and furnishings of all rooms should be cleaned by this procedure.
- 5. All cracks in the floor should be sealed with a silicone based caulk. The cork and rubber stoppers used to seal injection holes should be driven down into the holes and concrete used to completely seal the holes.
- 6. Once the carpet has been removed and the area cleaned, the electricians should identify, remove, and replace the light ballasts. For easy identification, EPA regulations require that PCB-free fluorescent light ballasts manufactured since July, 1978, be marked "No PCBs." PCB containing ballasts should be disposed of according to state requirements.
- 7. NIOSH will return to conduct air and surface wipe sampling to verify the effectiveness of the cleanup after step six has been completed throughout the school and before new flooring is put down.
- 8. After improvements have been made to the ventilation system, NIOSH will repeat the ventilation measurements to document the effectiveness of the improvements.

The following recommendations were offered in the June 30, 1989, letter, to minimize the exposure of workers during cleanup procedures to pesticides, PCBs, contaminated dust, and solvents.

1. Workers involved in removing the carpeting should wear disposable dust masks, disposable Tyvek coveralls, shoe covers, and nitrile or neoprene rubber gloves. This equipment should be removed and disposed of with the contaminated carpeting. This equipment will protect against inhalation to contaminated dust and dermal exposure to the pesticides and

- PCBs. The protective equipment should not be worn outside the building, during breaks or lunch, or taken home.
- 2. New equipment should be provided to each worker at the start of each workday with additional items available during the shift.
- 3. There should be no eating, drinking, or smoking in the cleanup area. Hands should be washed before engaging in these activities and before leaving the work site.
- 4. Workers involved in washing surfaces with mineral spirits should use the same protective garments as noted above and be provided with a half face organic vapor cartridge respirator. The respirator cartridges should be labeled with a testing and certification number approving its use for organic vapors (e.g., TC-23C-[40 through 734]). The supplier of the respirators should be consulted for the appropriate model of respirator for this job. The manufacturer's instructions for changing cartridges should be strictly followed. The selection, fitting, maintenance, and use of respirators should be in accordance with the Occupational Safety and Health Administration regulations (OSHA CFR 1910.134).

IX. <u>REFERENCES</u>

- The National Institute for Occupational Safety and Health (NIOSH). NIOSH Manual of Analytical Methods, Third Edition. Vol 1. DHHS Publication No. (NIOSH) 84-100. Cincinnati, OH: National Institute for Occupational Safety and Health, 1984.
- 2. The National Institute for Occupational Safety and Health (NIOSH). Indoor Air Quality and Work Environment Study, Library of Congress, Madison Building. Volume I: Employee Survey. December 1989. (Available from NIOSH).
- 3. Schlesselman, JJ. Case Control Studies: Design, Conduct, Analysis. Oxford University Press, New York, 1982.
- 4. Burse, VW et al. Determination of selected organochlorine pesticides and polychlorinated biphenyls in human serum, J Anal Tox, 14:137-142, 1990.
- 5. The National Institute for Occupational Safety and Health (NIOSH). NIOSH Pocket Guide to Chemical Hazards. Cincinnati, OH: National Institute for Occupational Safety and Health, 1990 Publication No. 90-117.
- 6. American Conference of Governmental Industrial Hygienists (ACGIH). Threshold Limit Values for 1990-1991. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, 1990.

- 7. Occupational Safety and Health Administration (OSHA). OSHA safety and health standards (29 CFR 1910). Washington DC: Department of Labor, 1989.
- 8. US Department of Health and Human Services, Public Health Service, Centers for Disease Control National Institute For Occupational Safety and Health, Division of Standards Development and Technology Transfer. Indoor Air Quality Selected References. Hazard Evaluations and Technical Assistance Branch; Division of Surveillance, Hazard Evaluations and Field Studies; National Institute for Occupational Safety and Health (NIOSH). Guidance for Indoor Air Quality Investigations. Cincinnati, OH. May, 1989.
- 9. American Society of Heating, Refrigerating and Air-Conditioning Engineers, (ASHRAE) Inc. ASHRAE standard 55-1981, thermal environmental conditions for human occupancy. Atlanta, Georgia: ASHRAE, 1981.
- American Society of Heating, Refrigerating and Air-Conditioning Engineers, (ASHRAE) Inc. ASHRAE standard 62-1989, ventilation for acceptable indoor air quality. Atlanta, Georgia: ASHRAE, 1989.
- 11. Strassman, SC, and Kutz, FW. Trends of organochlorine pesticide residues in human tissue. Toxicology of Halogenated Hydrocarbons, eds. Khan and Santon, Pergamon Press, 1981.
- National Research Council, Committee on Toxicology. An assessment of the health risks of seven pesticides used for termite control. Washington, DC: National Academy of Sciences, 1982.
- 13. Truhant, R. Gak, JC and Graillot C. Research on the modes and mechanisms of toxic action of organochlorinated insecticides. Comparative study of the effects of acute toxicity on the hamster and rat. Trans. J. Eur. Toxicol., 7:159-166, 1974.
- National Cancer Institute, Division of Cancer Cause and Prevention. Bioassay for chlordane for possible carcinogenicity. NCI-CG-TR-8 Bethesda, MD: National Institutes of Health. 117p. (DHEW Publ. No. (NIH) 77-808) 1977.
- 15. Wang, HH and MacMahon, B. Mortality of workers employed in the manufacture of chlordane and heptachlor. JOM, 21:745-748, 1979.
- 16. Murphy, RS, Kutz, FW, Strassman, SC, Selected pesticide residues or metabolites in blood and urine specimens from a general population survey. Envir. Hlth Perspectives, 48:81-86, 1983.
- 17. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Chlordane, ATSDR/TP-89/06, December 1989. (available from ATSDR)

- 18. Lloyd, JW, Moore, RM, Woolf, BS, and Stein, HP. Polychlorinated biphenyls. J. Occup. Med., 18:109. 1976.
- 19. Hutzinger, O, Safe, S, and Zitko, V. The chemistry of PCBs. The Chemical Rubber Company Press, Cleveland, OH. 1974.
- International Agency for Research on Cancer (IARC). IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans. Chemicals, Industrial Process, and Industries Associated with Cancer in Humans. IARC Monographs, Volumes 1 to 29. Supplement 4, Lyon, France, P. 118. 1982.
- 21. Brown, DP, and Jones, M. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Arch. Environ. Health 1981;36:120.
- 22. Bertazzi, PA, Zocchetti, C, Guercilena, S, Foglia, MD, Pesatori, A, and Riboldi, L. Mortality study of male and female workers exposed to PCBs. Presented at the International Symposium on Prevention of Occupational Cancer, April 1981, Helsinki, Finland.
- 23. Current Intelligence Bulletin No. 45 Polychlorinated Biphenyls (PCBs): Potential Health Hazards from Electrical Equipment Fires or Failures. Cincinnati, OH. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, 1986; DHHS (NIOSH) publication no. 86-111.
- 24. National Institute for Occupational Safety and Health. Criteria for a recommended standard: occupational exposure to polychlorinated biphenyls (PCBs). Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1977. DHEW publication no. (NIOSH) 77-225.
- 25. Andrews, JS et al. Health effects study of residents living near the Hollywood Dumpsite, Memphis, Tennessee. Final report of Center for Environmental Health and Injury Control, CDC, Cooperative agreement U61-CCU 400608-01, May 1988. (Available from John Andrews at Centers for Disease Control, Atlanta).

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XI. <u>DISTRIBUTION AND AVAILABILITY OF REPORT</u>

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For the purpose of informing affected employees, copies of this report shall be posted by the employer in a prominent place accessible to the employees for a period of 30 calendar days.

Table 1
Indoor Air Quality Measurements
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
April 11 and 19, 1989
HETA 89-183

Location	<u>Date</u>	Carbon Dioxide (ppm)*	Number of Occupants	Temp. <u>°F</u>	% Relative <u>Humidity</u>
Outdoors	4/11	200-250	-	63	45
	4/12	200-250	-	65	40
	4/19	200-250	-	60	60
Classroom 108	4/11	800	0	70	53
	4/19	800	10	71	45
	4/19	1200	9	73	42
Classroom 106	4/11	1100	22	70	40
Classroom 107	4/19	525	7	71	41
	4/19	850	1	73	39
Classroom 201	4/11	1100	12	74	50
Classroom 203	4/11	1300	20	71	42
	4/19	1100	20	68	54
	4/19	1100	20	72	42
Classroom 305	4/11	1100	22	70	50
Classroom 405	4/11	1000	0	70	42
Classroom 401	4/11	1000	15	74	26
Classroom 301	4/11	1400	22	76	45
Classroom 303	4/11	1500	11	76	25
	4/19	850	10	73	40
	4/19	1075	19	75	40
Admin. Office	4/11	700	5	77	23
	4/19	500	7	70	48
	4/19	575	2	77	38

^{*}ppm - parts per million; carbon dioxide levels approaching or exceeding 1000, or 3 to 4 times the outside level, indicate that inadequate amounts of fresh air are being introduced to the space.

Table 2

Indoor Air Quality Measurements
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
March 1, 1990
HETA 89-183

	Carbon Dioxide	Number of	Temp.	% Relative
Location	<u>(ppm)*</u>	<u>Occupants</u>	<u>°F</u>	<u>Humidity</u>
Outdoors	350	_	62	42
Room 101	575	14	71	52
Room 102	625	24	74	52
Room 104B	775	22	73	52
Room 105	675	14	75	52
Room 106	750	26	75	52
Room 107	700	20	75	53
Room 201	725	23	74	52
Room 202	475	0	72	50
Room 203	550	25	74	54
Room 206	800	33	75	53
Room 301	700	28	75	53
Room 302	625	17	74	52
Room 304	650	23	75	53
Room 305	650	15	76	54
Room 306	675	15	75	53
Room 307	700	22	75	55
Room 308	625	22	72	51
Room 309	600	22	74	52
Room 401	575	21	76	54
Room 402	600	22	72	52
Room 403	675	20	74	55
Room 404	675	18	74	53
Room 405	575	23	70	50
Room 502	550	2	70	50
Admin. Office	400	5	72	48

^{*}ppm - parts per million; carbon dioxide levels approaching or exceeding 1000, or 3 to 4 times the outside level, indicate that inadequate amounts of fresh air are being introduced to the space.

Table 3 Classroom Ventilation Measurements Andrew Jackson Junior High School Cross Lanes, West Virginia May 10, 1989 and March 1, 1990 HETA 89-183

Location	Total Supply Ai May 1989	r(cfm) March 1990	Outside Air S May 1989	supplied(cfm)* March 1990	Outside Air Pe May 1989	er Person(cfm)* March 1990
Room 101	410	1390	94	348	5	17
Room 102	950	1240	220	310	11	16
Room 104A	310	1050	71	263	4	13
Room 104B	290	1110	70	278	4	14
Room 105	1020	1370	235	343	12	17
Room 106	525	1350	120	338	6	17
Room 107	385	1270	90	317	5	16
Room 108	385	1050	90	262	5	13
Room 201	465	1270	105	317	5	16
Room 202	195	1005	45	251	2	13
Room 203	700	1215	160	304	8	15
Room 204	610	720	140	180	7	9
Room 206	0	1155	0	289	0	14
Room 205 (Library)	1430	4965	329	1241	16	62
Room 301	920	1250	210	313	10	16
Room 302	280	905	65	226	3	11
Room 303	480	1140	110	285	6	14
Room 304	740	1110	170	278	9	14
Room 305	860	1140	200	285	10	14

Continued (Table 3)

	Location Total S May 1989	upply Air(cfm) March 1990	Outside Air Supp May 1989	olied(cfm)* March 1990	Outside Air Per May 1989	Person(cfm)* March 1990
	Way 1767	Water 1770	Way 1767	Water 1990	Way 1707	Water 1990
Room 306	900	1170	210	293	3	15
Room 307	260	1050	60	263	3	13
Room 309	1100	1070	250	267	13	13
Room 401	950	1090	220	273	11	14
Room 402	1000	990	230	248	12	12
Room 403	1050	1080	240	270	12	14
Room 404	850	1040	95	260	5	13
Room 405	1160	1420	270	355	13	18
Room 502	600	1090	138	273	7	14

cfm - Cubic feet per minute

 $[\]ast$ - Outside air was calculated using an estimate of 23% (May 1989) and 25% (March 1990) of total supply, and outside air per person was calculated using an estimate of 20 occupants per classroom.

Table 4

Pesticide & Polychlorinated Biphenyl Contamination of Carpeting Andrew Jackson Junior High School Cross Lanes, West Virginia May 9, 1989 HETA 89-183

		Concentra	tion (ug/g)	
Sample Location	<u>Chlordane</u>	<u>Diazinon</u>	<u>Chlorpyrifos</u>	PCB*
Room 102, NE Quadrant	ND	(0.27)	ND	(1.7)
Room 104A, NE Quadrant	ND	2.0	ND	ND
Room 104B, NE Quadrant	ND	5.2	ND	ND
Room 107, East Wall	ND	76.0	ND	(3.1)
Room 201, East Wall	ND	3.7	ND	(2.8)
Room 202, North Wall	ND	41.0	ND	(1.9)
Room 301, Center of Room next to crack in floor	ND	(0.59)	ND	67.0
Room 304, near crack along South Wall	ND	8.6	ND	25.0
Room 305, near crack along South Wall	ND	17.0	ND	54.0
Room 307, next to Sink	ND	20.0	ND	12.0
Room 308, North Wall	ND	1.6	ND	11.0
Room 309, North Wall	ND	0.7	ND	44.0
Room 403, East Wall	ND	4.2	ND	46.0
Room 404, East Wall	ND	6.3	ND	29.0
Hallway outside of Room 206	ND	ND	ND	15.0
Hallway outside of Room 402	ND	2.0	ND	15.0
	ND	3.8	ND	15.0
Room 502, North Wall	ND	(0.5)	ND	12.0
Limit of Detection (LOD) Limit of Quantitation (LOQ)	0.1 0.3	0.18 0.61	0.02 0.07	1.7 5.9

^{*}PCB - polychlorinated biphenyls, Aroclor 1254 was the only congener found in these samples. ug/g - micrograms per gram of carpet.

ND - Not Detected - Values in parentheses fall between the LOD and LOQ and should should be considered as trace quantities.

Table 5

Surface Wipe Sample Results Andrew Jackson Jr. High School Cross Lanes, West Virginia May 10, 1989

HETA 89-183

	Pesticide Conc	. (ug/ft ²)	PCB Conc.(ug/m ²)	
Location, Surface	Chlordane	<u>Diazinon</u>	<u>Chlorpyrifos</u>	Aroclor 1254
Room 401, Student Desk Top	(1.1)	0.09	(0.06)	35.5
Room 402, Student Desk Top	(0.2)	ND	0.14	7.5
Room 309, Teachers' Desk Top	ND	0.05	ND	2.2
Room 309, Wall above Crack in Floor	ND	0.20	0.05	11.0
Room 402, Cover of Speech Text Book	ND	0.10	ND	7.2
Room 404, Teachers' Desk Top	ND	0.15	ND	4.3
Room 305, Teachers' Desk Top	ND	ND	ND	4.0
Room 301, Book Shelf	ND	0.05	ND	5.1
Room 204, Student Desk Top	ND	ND	(0.04)	9.7
Room 202, Student Desk Top	ND	ND	ND	7.9
Room 201, Top of Storage Locker	ND	0.03	ND	14.2
Room 104B, Student Table	ND	0.04	(0.02)	2.4
Room 104A, Teachers' Desk	ND	0.05	ND	8.6
Room 401, Text Book Cover	ND	(0.04)	ND	3.2
Hallway outside of Room 401,				
Tile Floor	ND	0.09	ND	5.1
Room 308, Students' Desk Top	ND	ND	ND	5.7
Room 502, Students' Desk Top	(0.13)	(0.02)	(.05)	8.1
Room 502, Wall above Crack in Floor	ND	(0.01)	ND	5.9
Hallway outside of Room 502,				
Tile Floor	ND	0.02	ND	16.9
LOD - Limit of Detection	0.5	0.02	0.06	0.18
LOQ - Limit of Quantitation	1.5	0.06	0.19	0.61

Surface sample results for pesticides are reported in micrograms per square foot (ug/ft^2), and for polychlorinated biphenyl (PCB) the results are reported in micrograms per square meter (ug/m^2). Aroclor 1254 was the only PCB detected in these samples by comparison with standard samples of Aroclors 1016, 1221, 1223, 1242, 1248, 1254, and 1260.

ND = Not-detected. Results contained in parenthesis indicate values between the LOD and LOQ for the analytical method. These quantities should be considered trace concentrations with limited confidence in their accuracy.

Table 6

Surface Wipe Sample Results Andrew Jackson Jr. High School Cross Lanes, West Virginia September 13-14, 1989

HETA 89-183

Location, Surface	Chlordane Conc. (ug/ft ²)	PCB Conc. (ug/m²)
Room 401, Wall Above Crack Room 401, Window Ledge* Room 401, Student Desk Top*	ND ND ND	20 158 28
Room 402, North Window Ledge* Room 402, South Window Ledge* Room 402, Student Desk Top*	ND ND ND	89 67 9
Room 309, Wall Above Crack	ND	25
Room 306, Lab Bench Top*	ND	205
Room 303, Floor Area in Doorway Room 303, Teacher's Desk Top*	ND ND	676
Room 104B, Sewing Machine Table Top*	ND	24
Room 105, Counter Top - Food Prep Area*	ND	19
Room 502, Wall Above Crack Room 502, Shelf Top Along Wall*	ND ND	3 132
Cafeteria, Counter Top - Food Prep Area* Cafeteria, Table Top in Commons Area*	ND ND	64 76
Admin. Office, Counter Top*	ND	23
Mechanical Room, Electric Transformer Shroud Mechanical Room, Electric Panel Door*	ND ND	4 4
NIOSH Investigator's Office, Credenza Top*	ND	(1)
LOD - Limit of Detection per sample LOQ - Limit of Quantitation per sample	0.09 ug/sample 0.3 ug/sample	0.2 ug/sample 0.6 ug/sample

^{*}High-skin-contact surface

Surface sample results for polychlorinated biphenyl (PCB) are reported in micrograms per square meter (ug/m²). Aroclor 1254 was the only PCB detected in these samples by comparison with standard samples of Aroclors 1016, 1221, 1223, 1242, 1248, 1254, and 1260.

ND = Not detected. Results contained in parenthesis indicate values between the LOD and LOQ for the analytical method. These quantities should be considered trace concentrations with limited confidence in their accuracy.

Table 7

PCB Surface Wipe Sample Results
Andrew Jackson Junior High School
Cross Lanes, West Virginia

HETA 89-183

Location	May 10, 1989	Aroclor 1254 ug/m ² Sept. 13, 1989	March 1, 1990
Room 401-Window Ledge	-	158	10.6
Room 402-Student Desk	7.5	9	2.2
Room 306-Lab Bench Top	-	205	1.2
Room 104A-Desk Top	2.4	24	0.6
Room 105-Counter top	-	19	1.5
Kitchen Food Prep Surf.	-	64	4.3
Admin. Office Counter	-	23	2.6
NIOSH P.O. Desk Top	-	(1)	1.0
LOD - Limit of Detection	0.18	0.2	0.03
LOQ - Limit of Quantitation	0.61	0.6	0.09

ug/m² - micrograms per square meter.

Results contained in parenthesis indicate values between the LOD and LOQ for the analytical method. This quality should be considered a trace concentration with limited confidence in accuracy of the result.

A line in the result column means that a sample was not collected at that location.

Table 8

Air Concentrations of Insecticides Andrew Jackson Jr. High School Cross Lanes, West Virginia April 19, 1989 HETA 89-183

	Sample	Conce	ntration (ug/m ³)*	
Location	Volume (L)	<u>Chlordane</u>	<u>Diazinon</u>	<u>Dursban</u>
Room 401, @ Floor Level	0.217	55.3	(0.02)	0.60
@ Breathing Zone Hgt.**	0.088	N.D.	N.D.	N.D.
Room 402, @ Floor Level	0.220	N.D.	N.D.	N.D.
@ Breathing Zone Hgt.	0.221	10.4	2.4	0.10
@ Breathing Zone Hgt.	0.157	N.D.	N.D.	N.D.
Room 403, @ Floor Level	0.232	N.D.	N.D.	N.D.
Room 309, @ Floor Level	0.234	0.34	0.02	N.D.
Room 104B, @ Breathing Zone Hgt.	0.320	(0.13)	0.02	N.D.
Room 502, @ Breathing Zone Hgt.	0.244	(0.08)	0.02	N.D.
Room 301, @ Breathing Zone Hgt.	0.230	N.D.	N.D.	N.D.
Room 107, @ Breathing Zone Hgt.	0.357	N.D.	N.D.	N.D.
Room 103, @ Breathing Zone Hgt.	0.363	N.D.	N.D.	N.D.
Room 304, @ Breathing Zone Hgt.	0.156	N.D.	N.D.	N.D.
Room 204, @ Breathing Zone Hgt.	0.147	N.D.	N.D.	N.D.
Cafeteria, @ Breathing Zone Hgt.	0.047	N.D.	N.D.	N.D.
Room 203, @ Breathing Zone Hgt.	0.152	N.D.	N.D.	N.D.
Room 206, @ Breathing Zone Hgt.	0.146	N.D.	N.D.	N.D.
Room 201, @ Breathing Zone Hgt.	0.131	N.D.	N.D.	N.D.
@ Breathing Zone Hgt.	0.137	N.D.	N.D.	N.D.
Room 105, @ Breathing Zone Hgt.	0.038	N.D.	N.D.	N.D.
Limit of Detection	0.01	0.004	0.002	
Limit of Quantitation	0.05	0.0057	0.002	
ACGIH TLV		500/skin	100/skin	200/skin
OSHA PEL		500/skin	100/skin	200/skin
NIOSH REL		Chlordane - Lowest	Feasible Level	
NRC (Air in Military Housing)		5 (air & surface)		
US Air Force (Military Housing Surface		50 (walls, ceilings),		
Standards mg/ft ²)		10 (floors)		
		7 (walls) when ch		
		ND (floors) are pr	esent	

 $[*]ug/m^3$ - micrograms per cubic meter of air.

 $N.D. \ \hbox{--Not Detected.} \ \ Values \ in parentheses \ fall \ between \ the \ LOD \ and \ LOQ \ and \ should \ be considered as trace quantities.$

^{**}Breathing zone height for seated individuals.

Table 9

Air Concentrations of Chlordane and PCBs Andrew Jackson Jr. High School Cross Lanes, West Virginia May 10, 1989 HETA 89-183

	Sample	Concentration (ug/	m ³)
Location	Volume (L)	Chlordane	Aroclor 1254
Room 101, Center @ BZ Level	393	0.15	(0.05)
Room 102, Center @ BZ Level	328	0.15	(0.06)
Room 104A, Center @ BZ Level	331	0.15	(0.03)
Room 104B, Center @ BZ Level	332	ND	ND
Room 105, Center @ BZ Level	330	0.12	(0.06)
Room 105, @ Floor level next to outside door	295	0.17	(0.07)
Room 106, Center @ BZ Level	328	0.15	(0.03)
Room 107, Center @ BZ Level	272	0.18	(0.07)
Room 108, Center @ BZ Level	335	0.15	0.12
Room 201, Center @ BZ Level	330	0.18	(0.09)
Room 202, Center @ BZ Level	323	0.15	(0.06)
Room 203, Center @ BZ Level	314	0.13	ND
Room 203, Above crack along outside wall	303	0.13	(0.03)
Room 204, Center @ BZ Level	321	0.16	(0.03)
Room 205, Center @ BZ Level	297	0.17	(0.07)
Room 206, Center @ BZ Level	307	0.16	(0.07)
Room 301, Center @ BZ Level	307	0.16	(0.07)
Room 301, Above crack in floor	307	0.23	(0.07)
Room 302, Center @ BZ Level	306	0.13	(0.07)
Room 303, Center @ BZ Level	311	0.16	(0.06)
Room 304, Center @ BZ Level	309	0.10	(0.06)

(continued)

Continued (Table 9)

	Sample	Concentration (ug	
Location	Volume (L)	Chlordane	Aroclor 1254
Room 304, Above crack in floor	309	0.13	(0.06)
Room 305, Center @ BZ Level	308	0.16	(0.06)
Room 305, Above crack in floor	309	0.13	(0.03)
Room 306, Center @ BZ Level	306	0.13	(0.07)
Room 306, Above crack in floor	306	0.10	(0.07)
Room 307, Center @ BZ Level	315	0.16	(0.03)
Room 308, Center @ BZ Level	319	0.16	(0.06)
Room 309, Center @ BZ Level	310	0.19	(0.06)
Room 401, Above crack in floor	269	0.33	(0.07)
Room 402, Center @ BZ Level	271	0.26	(0.07)
Room 402, Above crack in floor	271	0.33	(0.07)
Room 403, BZ Level @ Teachers' Desk	274	0.18	(0.07)
Room 403, Above crack in floor	274	0.11	(0.07)
Room 404, BZ Level @ Teachers' Desk	279	0.25	(0.07)
Room 404, Above crack in floor	279	0.22	(0.07)
Room 502, Center @ BZ Level	292	0.14	0.10
Room 502, Above crack in floor	308	0.11	0.11
Hallway outside of Room 107, BZ Level	247	0.16	(0.08)
Hallway outside of Room 206, BZ Level	304	0.20	0.10
Library Area, Southeast corner @ BZ Level	315	0.16	(0.06)
Library Area, Westside @ BZ Level	272	0.18	(0.07)
Cafeteria, Center @ BZ Level	315	(0.10)	0.19
LOD - Limit of Detection per sample LOQ - Limit of Quantitation per sample		0.014 0.034	0.01 0.03

ug/m³ - micrograms per cubic meter of air.

ND - Not Detected. Values in parenthesis fall between the LOD and LOQ and should be considered trace concentrations with limited confidence in their accuracy.

Table 10

Air Concentrations of Chlordane and PCBs Andrew Jackson Jr. High School Cross Lanes, West Virginia September 13-14, 1989 HETA 89-183

	Sample	Concentration	on (ug/m ³)
Location	Volume (L)	Chlordane	Aroclor 1254
D 101 C 4 6 P7 I	526		T
Room 101, Center @ BZ Level	526	- ND	Trace
Room 102, Center @ BZ Level	525	ND	- T
Room 104A, Center @ BZ Level	534	- ND	Trace
Room 104B, Center @ BZ Level	680	ND	ND
Room 105, Center @ BZ Level	528	ND	-
Room 107, Center @ BZ Level	440	Trace	-
Room 108, Center @ BZ Level	518	-	Trace
Room 108, Center @ BZ Level	690	-	Trace
Room 201, Center @ BZ Level	525	Trace	Trace
Room 203, Center @ BZ Level	640	-	Trace
Room 203, Center @ BZ Level	536	<u>-</u>	Trace
Room 204, Center @ BZ Level	525	Trace	
Room 205, East Side @ BZ Level	530	ND	Trace
Room 205, Center @ BZ Level	653	-	Trace
Room 301, Center @ BZ Level	522	Trace	-
Room 302, Center @ BZ Level	530	-	Trace
Room 303, Center @ BZ Level	523	Trace	-
Room 304, Center @ BZ Level	525	-	Trace
Room 305, Center @ BZ Level	463	Trace	-
Room 308, Center @ BZ Level	271	ND	-
Room 309, Center @ BZ Level	509	Trace	-
Room 401, Center @ BZ Level	635	Trace	Trace
Room 401, Center @ BZ Level	509	Trace	-
Room 401, @ Crack next to wall	526	Trace	-
Room 402, Center @ BZ Level	520	-	Trace
Room 402, @ Crack next to wall	522	Trace	-
Room 404, Center @ BZ Level	525	Trace	Trace
Room 502, Center @ BZ Level	489	Trace	Trace
Room 502, Center @ BZ Level	644	Trace	Trace
Cafeteria, Center @ BZ Level	386	ND	Trace
Cafeteria, Center @ BZ Level	653	-	Trace
Mechanical Room, Top of Transformer	534	-	Trace
Admin. Office, Top of Center Counter	495	-	Trace
Entrance way, Outside of Room 401, BZ Level	495	-	Trace
Range of Trace Concentrations		0.12 to 0.36	0.09 to 0.31
-		0.12 to 0.30	0.05
LOD - Limit of Detection per sample		0.20	0.03
LOQ - Limit of Quantitation per sample		0.20	0.20

ug/m³ - micrograms per cubic meter of air.

ND - Not Detected.

A line in the column indicates a sample was <u>not</u> collected at that location for that compound.

Table 11

Distribution of Job Titles for Questionnaire Respondents
Andrew Jackson Junior High School
Cross Lanes, West Virginia
HETA 89-183

	January 1989	March 1990	June 1990
Number	40	42	45
Teacher	70%*	74%	71%
Office Worker	15%	10%	16%
Custodial Staff	8%	10%	7%
Kitchen Worker	8%	7%	7%

^{*} Percentages may not add up to 100% due to rounding

Table 12 Health Complaints by Ventilation Zone Kanawha County Health Department Questionnaire Andrew Jackson Junior High School Cross Lanes, West Virginia

HETA 89-183

COMPLAINT	Zone 1	Zone 2	Zone 3	Zone 4	Custodians	Other	Total
Total # of Respondents	6	4	5	8	4	16	43
Headache	3 50%	3 75%	1 20%	4 50%	3 75%	5 30%	19 44%
Throat Irritation	2 33%	3 75%	0	1 12%	3 75%	5 30%	14 33%
Sinus Problems	3 50%	3 75%	2 40%	4 50%	3 75%	4 25%	19 44%
Cough	1 17%	0	0	0	1 25%	4 25%	6 14%
Shortness of Breath	1 17%	0	0	1 12%	1 25%	0	3 7%
Eye Irritation	1 17%	1 25%	1 20%	1 12%	2 50%	4 25%	10 23%
Rash	1 17%	0	0	0	1 25%	0	2 5%
Muscle Aches	3 50%	1 25%	0	2 25%	3 75%	1 6%	10 23%
Dizziness	3 50%	0	0	2 25%	1 25%	0	6 14%
Abdominal Pains	1 17%	1 25%	0	0	0	0	2 5%
Fatigue	4 66%	3 75%	1 20%	3 37%	3 75%	6 36%	20 46%
Problems with Concentration	3 50%	2 50%	0	4 50%	1 25%	3 18%	13 30%
Problems with Sleepy	3 50%	2 50%	0	1 12%	2 50%	1 6%	9 21%
Depression	1 17%	0	0	1 12%	2 50%	0	4 9%
Tension	4 66%	2 50%	1 20%	3 37%	1 25%	3 18%	14 30%

Zone 1 includes the following rooms: 104A, 104B, 105, 203, 204, 205 Zone 2 includes the following rooms: 303, 304, 305, 306, 307, 404, 405 Zone 3 includes the following rooms: 101, 102 106, 107, 201, 202, 206 Zone 4 includes the following rooms: 301, 302, 308, 309, 401, 402, 403

Prevalence of Medical Complaints Before and After Renovation For All Survey Respondents

Table 13

Andrew Jackson Junior High School Cross Lanes, West Virginia HETA 89-183

	Before Renovations		After Renovations	
	Since Starting Work at Andrew Jackson	During the Last Week	During the Last Week	During the Last 4 Months
Date of Survey	1/11/89	3/1/90	6/8/90	6/8/90
# of Respondents	40	42	45	45
Symptom				
Headache	65*	31	38	36
Sinus Congestion	62	33	24	40
Fatigue	62	17	22	33
Tension	50	17	27	33
Sore Throat	47	17	13	24
Eye Irritation	45	9	9	24
Problems with				
Concentration	40	5	9	16
Depression	35	2	13	22
Cough	30	17	11	15
Muscle Pains	30	19	22	22
Dizziness	25	9	7	4
Skin Irritation	15	9	9	15
Shortness of Breath	12	5	7	6

^{*}Percent of respondents reporting symptoms.

Table 14

Prevalence of Medical Complaints Before and After Renovation For Respondents Completing All Three Surveys Andrew Jackson Junior High School Cross Lanes, West Virginia HETA 89-183

	Since Starting Work at Andrew Jackson	During the Last Week	During the Last Week	During the Last 4 Months
Date of Survey	1/11/89	3/1/90	6/8/90	6/8/90
# of Respondents	25	25	25	25
Symptom				
Headache	60*	44	48	44
Sinus Congestion	56	40	32	40
Fatigue	60	16**	24**	40
Tension	52	24	36	48
Sore Throat	40	20	16**	24
Eye Irritation	52	20	16	24
Problems with				
Concentration	40	8**	12	20
Depression	44	0**	20	28
Cough	32	16	12	16
Muscle Pains	36	24	28	32
Dizziness	32	12	8	8
Skin Irritation	16	8	12	16
Shortness of Breath	16	8	8	12

^{*} Percent of respondents reporting symptoms.

^{**} Statistically significant change from 1/89 by McNemar's test (p<.05)

Table 15

Prevalence of Environmental Complaints Before and After Renovation For All Survey Respondents Andrew Jackson Junior High School Cross Lanes, West Virginia HETA 89-183

	On Most Days at Andrew Jackson	During the Last Week	During the Last Week	During the Last 4 Months
Date of Survey	1/11/89	3/1/90	6/8/90	6/8/90
# of Respondents	40	42	45	45
Complaint				
Too Dusty	48*	7	4	4
Too Little Air	45	0	4	4
Too Stuffy	43	5	11	9
Too Noisy	25	24	9	9
Too Hot	25	5	13	16
Too Cold	23	7	2	2
Too Much Air	5	7	0	7
Too Much Humidity	5	0	7	9

^{*} Percent of respondents reporting symptoms.

Table 16

Prevalence of Environmental Complaints Before and After Renovation For Respondents Completing All Three Surveys Andrew Jackson Junior High School Cross Lanes, West Virginia HETA 89-183

	On Most Days at Andrew Jackson	During the Last Week	During the Last Week	During the Last 4 Months
Date of Survey	1/11/89	3/1/90	6/8/90	6/8/90
# of Respondents	25	25	25	25
Complaint				
Too Dusty	36*	8	8**	8**
Too Little Air	52	0**	0**	0**
Too Stuffy	36	4**	12	12
Too Noisy	28	28	8	8
Too Hot	28	4**	12	8
Too Cold	16	4	4	4
Too Much Air	4	8	0	4
Too Much Humidity	5	0	4	4

^{*} Percent of respondents reporting symptoms.

^{**} Statistically significant change from 1/89 by McNemar's test (p<.05)

Table 17

Prevalence of Workplace Complaints
Before and After Renovation
For All Survey Respondents
Andrew Jackson Junior High School
Cross Lanes, West Virginia
HETA 89-183

January 1989	March 1990	June 1990
40	42	45
65*	2	16
60	17	18
15	14	7
13	12	11
10	7	13
10	2	4
	65* 60 15 13	40 42 65* 2 60 17 15 14 13 12 10 7

^{*} Percent of respondents reporting symptoms.

Table 18

Prevalence of Workplace Complaints
Before and After Renovation
For Respondents Completing All Three Surveys
Andrew Jackson Junior High School
Cross Lanes, West Virginia
HETA 89-183

	January 1989	March 1990	June 1990
# of Respondents	25	25	25
Are You Bothered By			
Lack of Control of			
the Temperature	65*	4**	16**
Lack of Windows	60	16**	20**
Lack of Privacy	15	16	8
Feeling Crowded	13	8	12
Lack of Workspace	10	4	16
Uncomfortable Chairs	10	0	4

^{*} Percent of respondents reporting symptoms.

^{*} Statistically significant by McNemar's test (p<.05)

Table 19

Blood Chlordane Metabolite Results Andrew Jackson Jr. High School Cross Lanes, West Virginia May 10, 1989 HETA 89-183

Analyte Concentration (ng/mL) Heptachlor

		Heptachlor	
Sample I.D.	Oxychlordane	<u>Epoxide</u>	<u>Trans-Nonachlor</u>
1	ND	ND	ND
2	ND	ND	ND
3	ND	ND	ND
4	ND	ND	ND
5	ND	ND	ND
6	ND	ND	ND
7	ND	ND	ND
8	ND	ND	ND
9	ND	ND	ND
10	ND	ND	0.41
11	ND	ND	ND
12	ND	ND	ND
13	ND	ND	ND
14	ND	ND	ND
15	ND	ND	ND
16	ND	0.37	ND
17	ND	ND	ND
18	ND	ND	ND
19	0.23	0.32	1.2
20	ND	ND	ND
21	ND	ND	ND
22	ND	ND	ND
23	ND	ND	ND
24	ND	ND	ND
25	ND	ND	ND
26	ND	ND	ND
27	ND	ND	ND
28	ND	ND	ND
29	ND	ND	ND
30	ND	ND	ND
31	ND	ND	ND
32	ND	ND	ND
33	ND	ND	ND
34	ND	ND	ND
35	ND	ND	ND
36	ND	ND	ND
37	ND	ND	ND
38	ND	ND	ND
39	ND	ND	ND
40	ND	ND	ND
41	ND	ND	ND
42	ND	ND	ND
43	ND	ND	ND
44	ND	0.84	0.64
45	ND	ND	0.45
46	ND	ND	ND
47	ND	ND	ND
48	ND	ND	ND
49	ND	ND	ND
72	ND	ND	MD

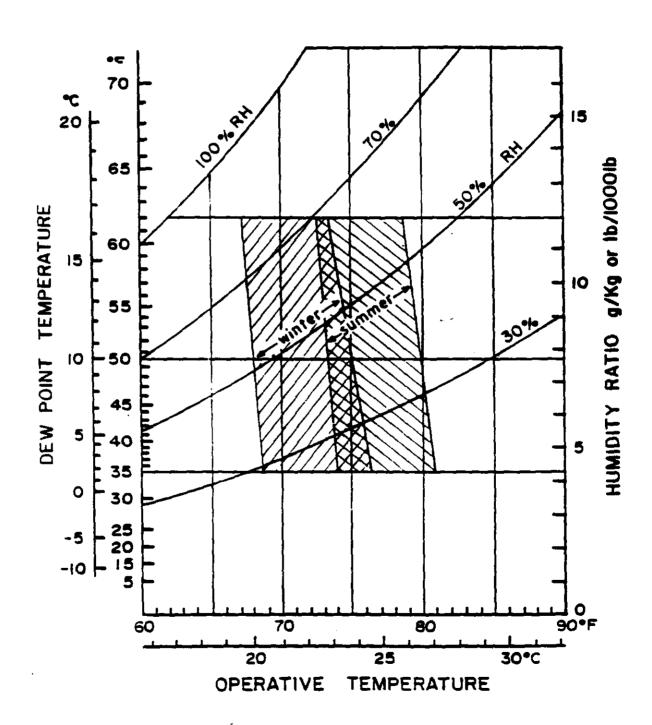
 $\mbox{ND} = \mbox{Not Detected}.$ The limit for detection with this method is $0.2~\mbox{ng/mL}$

ng/mL = nanograms/milliliter

FIGURES

Andrew Jackson Jr. High School Cross Lanes, West Virginia

HETA 89-183



are 2. Acceptable ranges of operative temperature and humidity for persons clothed in typical sumance and winter clothing, at light, mainly sedemory; activity (= 1.2 met)...

FIGURE 2 Andrew Jackson Jr. High HETA 89-183 IAQ Measurements

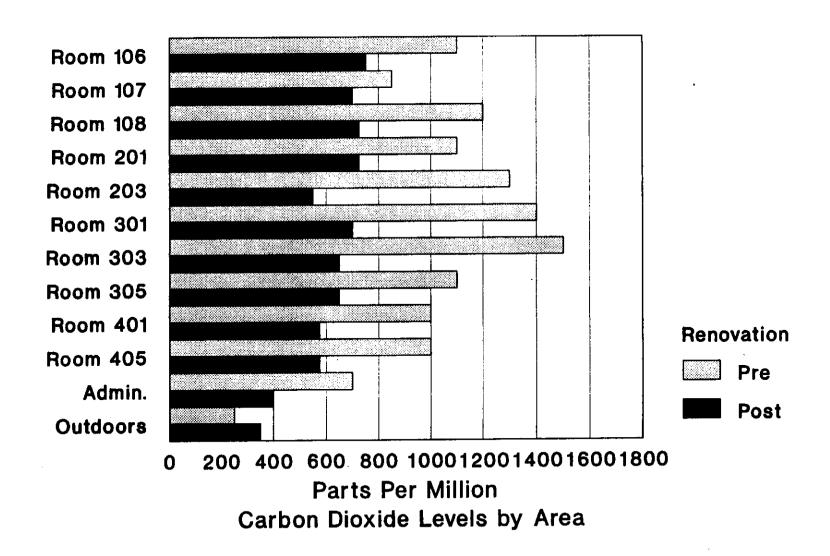


FIGURE 3 Andrew Jackson Jr. High
HETA 89-183
IAQ Measurements

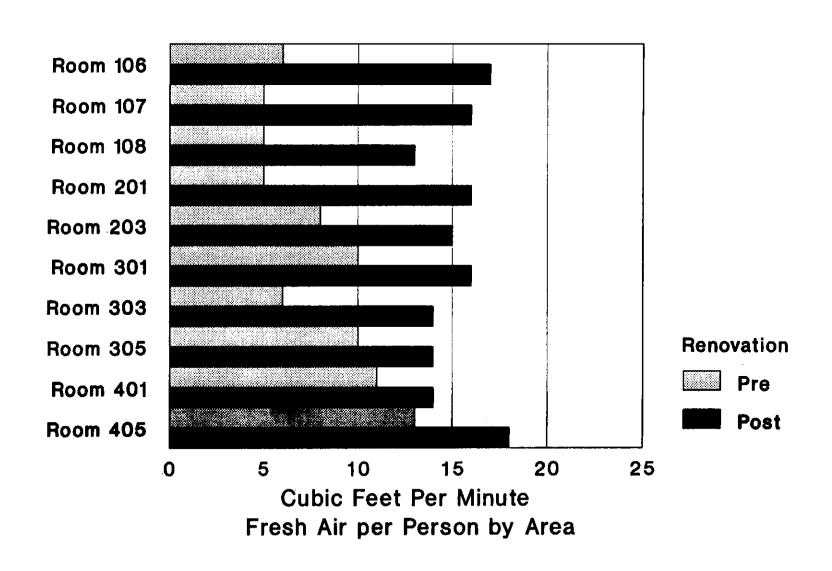


Figure 4
Volatile Organic Compounds - Room 401
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
April 11, 1989
HETA 89-183

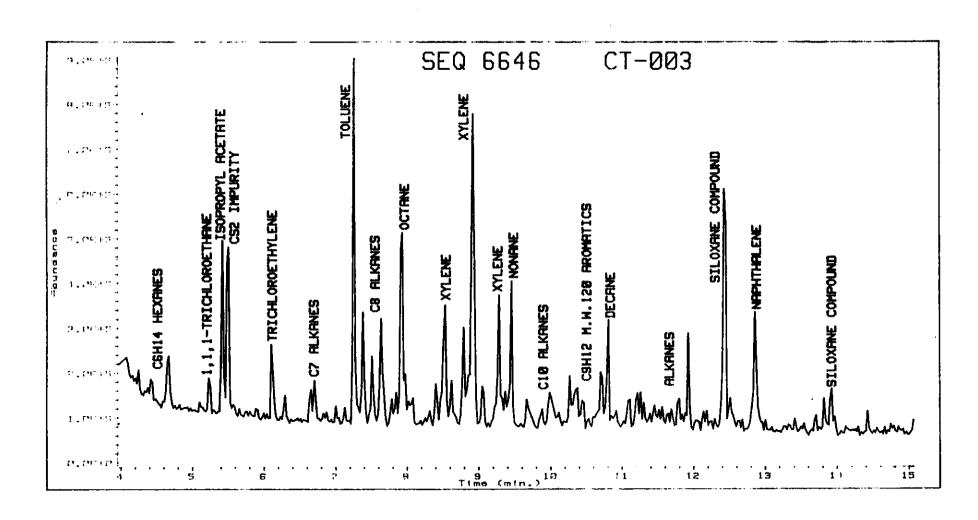


Figure 5
Volatile Organic Compounds - Room 203
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
April 11, 1989
HETA 89-183

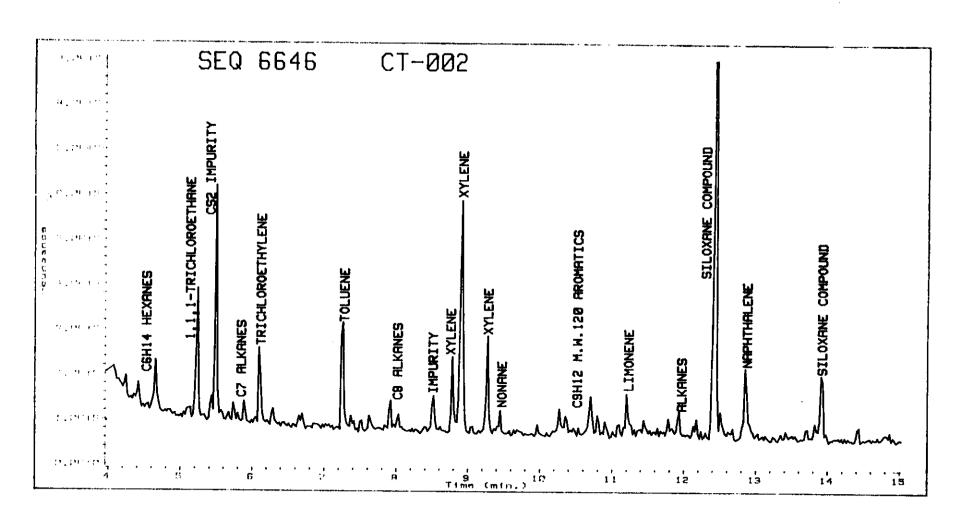


Figure 6
Volatile Organic Compounds - Outdoors
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
April 11, 1989
HETA 89-183

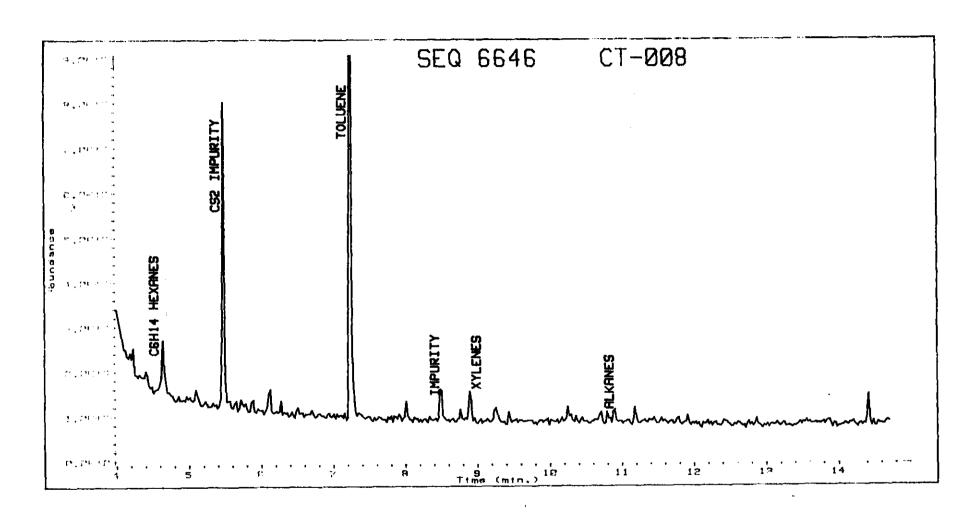


Figure 7
Volatile Organic Compounds - Room 401
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
March 1, 1990
HETA 89-183

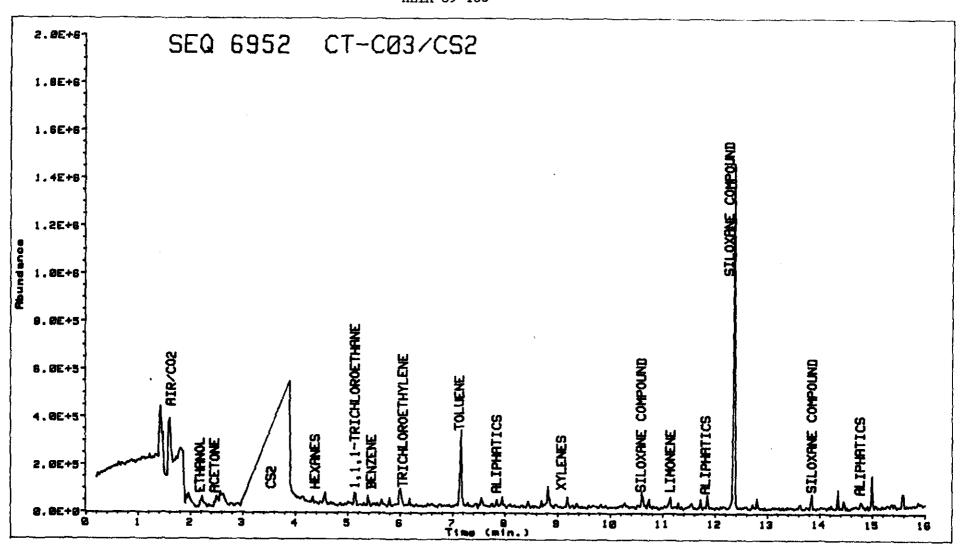


Figure 8
Volatile Organic Compounds - Room 203
Andrew Jackson Jr. High School
Cross Lanes, West Virginia
March 1, 1990
HETA 89-183

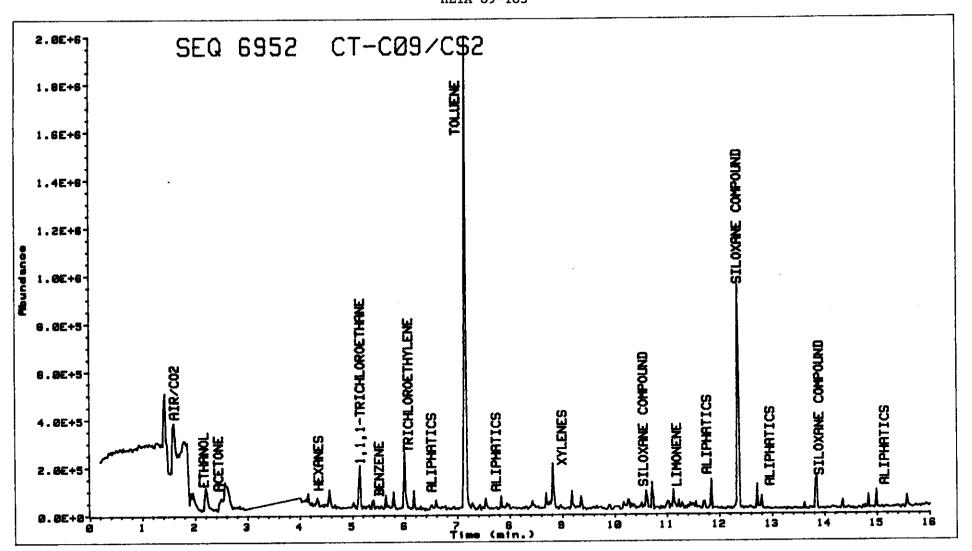
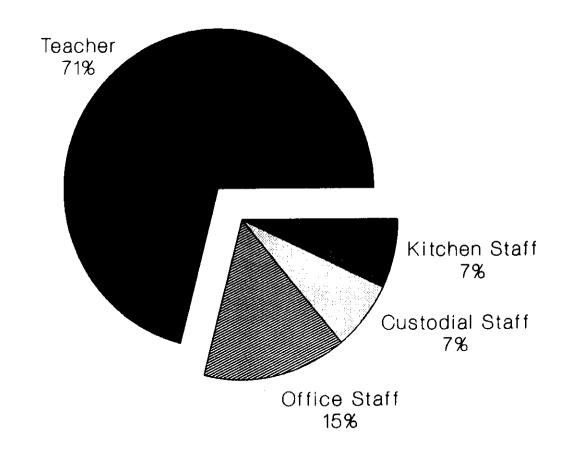
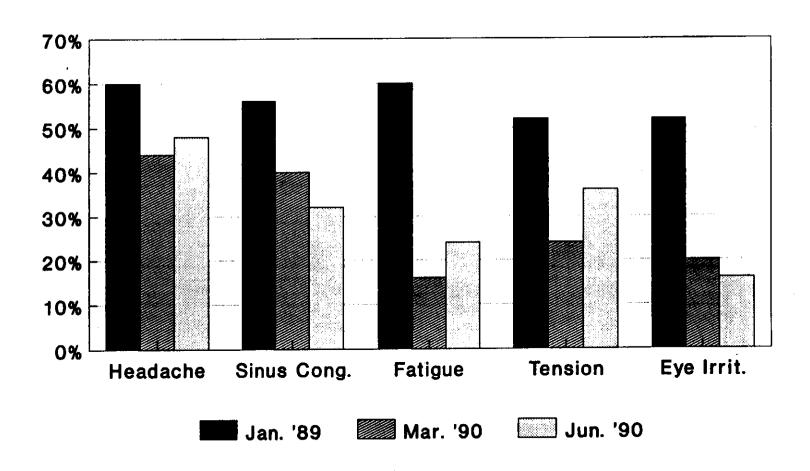


FIGURE 9 Andrew Jackson Jr. High HETA 89-183



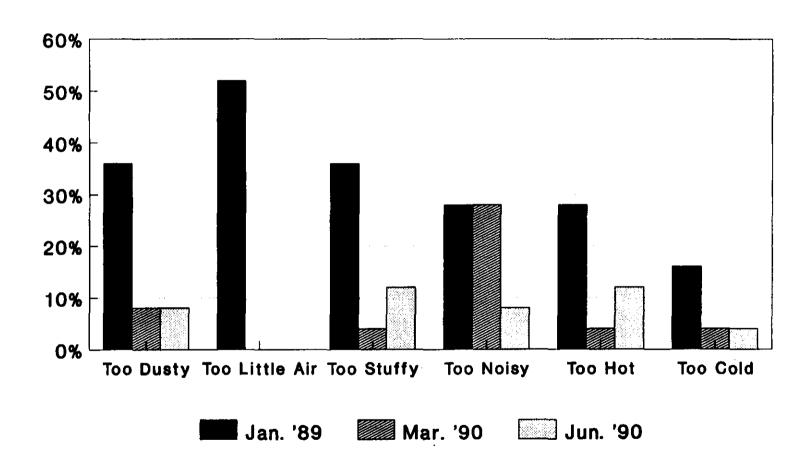
Distribution of Jobs from Questionnaire

FIGURE 10 Andrew Jackson Jr. High
HETA 89-183
Prevalence of IAQ Complaints



Respondents Completing All Three Questionnaires

FIGURE 11 Andrew Jackson Jr. High
HETA 89-183
Prevalence of Environmental Complaints



Respondents Completing All Three Questionnaires

APPENDIX 1

TOXICOLOGICAL PROFILE FOR CHLORDANE

Prepared by:

Syracuse Research Corporation Under Subcontract to:

Clement Associates, Inc. Under Contract No. 205-88-0608

Prepared for:

Agency for Toxic Substances and Disease Registry (ATSDR)
U.S. Public Health Service

In collaboration with U.S. Environmental Protection Agency (EPA)

December 1989

2.3 RELEVANCE TO PUBLIC HEALTH

Death. Absorption following ingestion of or skin contact with chlordane can be fatal to humans. WHO (1984) reported that an acute oral dose as low as 25 mg/kg can result in death, although documentation was not available for this estimation. This estimated dose is about 10 times lower than oral LD $_{50}$ values reported for animals. Mice chronically ingesting 3.9 mg/kg/day through the feed experienced increased mortality (NCI 1977), suggesting that mortality could occur in humans chronically exposed to dosages much lower than 25 mg/kg/day. The dermal dose that results in death of humans is not known, but dermal LD $_{50}$ s in animals are relatively low. A

dosage of 690 mg/kg of technical chlordane was a dermal LD50 in rats (Gaines 1960) and 1100-1200 mg/kg of "later" production chlordane (not contaminated with hexachlorocyclopentadiene) was a dermal LD50 in rabbits (Ingle 1965). Therefore, caution should be exercised when handling chlordane because of the possibility of dermal absorption. Epidemiological studies (Ditraglia et al. 1981; MacMahon et al. 1988; Shindell and Ulrich 1986; Wang and MacMahon 1979a, 1979b) and case reports of acute exposure (EPA 1980b) do not suggest that inhalation exposure is likely to result in human deaths. Also, animal studies do not suggest that mortality would be expected in humans exposed by inhalation to chlordane. Ingle (1953) reported no mortality in rats exposed to air at 25-50% of saturation with chlordane. Exposure concentrations were not further quantified. Velsicol Chemical Co. (1984) reported no mortality in rats intermittently exposed to 10 mg/m³ for 90 days. Levels of chlordane found in drinking water, air or soil near waste sites are not likely to result in non-cancer human fatality.

Respiratory Effects. Data from human or animal studies do not indicate that respiratory effects are likely in humans exposed to chlordane. Several case reports and personal reports involving acute inhalation, oral or dermal exposure (EPA 1980b; Harrington et al. 1978; NIOSH 1984a) and epidemiological studies of chlordane manufacture (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck, 1951) do not report respiratory effects in exposed humans. In one suicide case, however, pneumonia-like symptoms were reported in a woman who eventually died as a result of ingesting chlordane (Derbes et al. 1955), but these effects did not appear until 7 days into the course of the illness. Respiratory effects do not appear to be caused in rats by repeated exposure to chlordane vapor or aerosols (Ingle 1953; Velsicol Chemical Co. 1984). It is unlikely that respiratory effects would be important in humans exposed to chlordane in food, drinking water, air or soil near waste sites.

Cardiovascular Effects. Cardiovascular effects associated with exposure to chlordane were not reported in studies of occupational exposure during manufacture (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck, 1951), following inhalation and dermal exposure resulting from a chemical spill in a library room (NIOSH 1984a), in the case of a woman who eventually died following ingestion of a large dose of chlordane (Derbes et al. 1955) or in the majority of cases and personal reports of acute oral, inhalation or dermal exposure compiled by EPA (1980b). A few of the cases compiled by EPA (1980b) reported tachycardia, chest pains and shortness of breath, but exposure often involved a mixture of pesticides and vehicles such as petroleum distillates that may have caused these effects. No histopathological heart lesions were found in rats or hamsters treated acutely with oral doses of chlordane (Truhaut et al. 1974) or in rats or mice in chronic dietary (NCI 1977; Velsicol Chemical Co. 1983a, 1983b) or inhalation (Ingle 1953; Velsicol Chemical Co. 1984) studies. Thus it is unlikely that exposure to chlordane per se would result in cardiovascular effects in humans.

Gastrointestinal Effects. Gastrointestinal symptoms are an early and consistent observation in a number of case reports and personal reports of acute human oral and inhalation exposure (EPA 1980b). These symptoms include nausea, vomiting, intestinal cramps and abdominal pain, and diarrhea. NIOSH (1984a) reported gastrointestinal symptoms in library workers following inhalation and dermal exposure to high levels of chlordane resulting from a spill. Gastrointestinal symptoms were reported by humans accidently exposed to chlordane in their drinking water at levels ranging from 0.1 to 92,500 ppb (Harrington et al. 1978). Vomiting was a presenting symptom in the case of a woman who eventually died as a result of ingesting chlordane (Derbes et al. 1955). Diarrhea developed later in the course of the illness. Gastrointestinal symptoms were not reported in a number of epidemiology studies of chlordane manufacture (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck 1951) nor in a compilation of cases of acute dermal exposure (EPA 1980b).

No histopathological lesions in the gastrointestinal tract were found in rats or hamsters treated acutely with oral doses of chlordane (Truhaut et al. 1974), or in rats or mice in chronic feeding studies (NCI 1977; Velsicol Chemical Co. 1983a, 1983b), or in rats exposed by inhalation (Ingle 1953; Velsicol Chemical Co. 1984).

Necropsy of a woman who died after acute ingestion of a very large dose of chlordane (104 mg/kg estimated by the authors) revealed ulcerative inflammation of the upper gastrointestinal tract (Derbes et al. 1955). Other case reports (EPA 1980b) mention oral irritation after acute accidental ingestion. These observations suggest that chlordane, especially in large doses, is irritating to the gastrointestinal tract when ingested. The gastrointestinal symptoms (vomiting, abdominal pain, and diarrhea) reported in humans following inhalation exposure and ingestion of relatively smaller doses may reflect a primary effect on other organs or systems, such as the CNS. These data suggest that gastrointestinal symptoms would be among the earliest complaints of those exposed to chlordane at low levels in the air or drinking water. The threshold concentrations for these effects in environmental media are unknown, and the likelihood of their occurrence near waste sites cannot be predicted.

Hematological Effects. Hematological effects specifically related to exposure to chlordane were not reported in a compilation of case reports and personal reports of acute oral, inhalation or dermal exposures (EPA 1980b). Limited hematological examinations revealed no effects on workers involved in chlordane manufacture (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck 1951). Leukocytosis associated with lymphocytosis was reported in mice treated by gavage with 8 mg/kg/day for 14 days (Johnson et al. 1986), but the toxicological significance of an elevated leukocyte count is unclear. Hematological examination revealed no effects in rats exposed for 90 days by inhalation (Velsicol Chemical Co. 1984) or in rats or mice chronically exposed to chlordane in the diet (Velsicol Chemical Co. 1983a, 1983b). The available data do not suggest that hematological effects would

be likely in humans exposed to low levels of chlordane in environmental media near waste sites.

Hepatic Effects. Hepatic effects were sometimes reported in a compilation of case reports and personal reports of acute oral, inhalation or dermal exposure to chlordane (EPA, 1980b), but not in library workers acutely exposed to high levels resulting from a spill (NIOSH 1984a). Liver function tests were normal in a 20-month-old white male child at 20 hours to 3 days after ingesting chlordane (Curley and Garrettson 1969), in a nurseryman who developed neurological effects after daily handling soil which contained chlordane and other chemicals (Barnes 1967), and in workers involved in the manufacture of chlordane (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck 1951). Jaundice has been reported in humans living in homes treated with chlordane for termite control (EPA 1980b). Acute, prolonged dermal exposure of an infant to liquid chlordane was associated with jaundice, liver necrosis, fatty infiltration, and inflammation diagnosed from a biopsy sample (Balistreri et al. 1973).

Reliable acute oral and parenteral studies in animals exposed to chlordane indicate that enzyme induction, minor histochemical and histomorphological changes, and liver hypertrophy occur within hours of treatment and at very low doses (Casterline and Williams 1971; Cram et al. 1956; Den Tonkelaar and Van Esch 1974: Hart et al 1963; Johnson et al. 1986; Truhaut et al. 1974). Enzyme induction in the absence of evidence of liver damage or impaired liver function is ordinarily considered an adaptative rather than an adverse effect. In the case of chlordane, however, enzyme induction may represent an adverse response because metabolites of chlordane, such as oxychlordane, heptachlor, and heptachlor epoxide, are more toxic than the parent compound. Moreover, enzyme induction can accelerate metabolism of therapeutic drugs and hormones (Welch and Harrison, 1966; Welch et al. 1971). Prolonged inhalation (Ingle 1953; Velsicol Chemical Co. 1984) and oral exposure studies (IRDC 1973; Velsicol Chemical Co. 1983a, 1983b) identify histopathologic changes in the liver as the effect occurring at the lowest dosage associated with adverse effects in rats, mice and monkeys.

Although reports of serious liver disease in humans exposed to chlordane are uncommon, animal studies suggest that more subtle effects including altered enzyme activities and mild morphological changes may be expected from any route of exposure to low doses. These effects would be expected to occur at lower dosages and shorter durations of exposure than jaundice or other indications of severe liver dysfunction. These effects are not likely to be diagnosed routinely, however, because invasive procedures such as liver biopsy would be required. Physicians prescribing therapeutic drugs or hormones should be aware, however, that dosage may require adjustment in patients exposed to chlordane.

Renal Effects. Renal effects were not reported in a compilation of case and personal reports of acute human oral, inhalation or dermal

exposure to chlordane (EPA 1980b), in library workers exposed to high levels resulting from a spill (NIOSH 1984a) or in workers involved in chlordane manufacture (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck 1951). A woman who eventually died after ingesting a large dose of chlordane developed anuria and exhibited nephrosis at necropsy (Derbes et al. 1955). Derbes et al. (1955) also reported pathological changes in the kidneys of another woman who died within minutes after an accidental spill resulted in dermal exposure to chlordane and other pesticides.

Kidney congestion was observed in rats and hamsters given large acute oral doses of 200 and 1200 mg/kg, respectively (Truhaut et al. 1974). Kidney lesions were not reported in rats or mice exposed to chlordane in the diet (NCI 1977; Velsicol Chemical Co. 1983a, 1983b) or in rats or monkeys exposed intermittently by inhalation for 90 days (Velsicol Chemical Co. 1984). Rats in the inhalation study, however, exhibited elevated kidney weights. Kidney effects have generally been seen in humans and animals after large acute doses. It is not known whether humans could develop kidney lesions after prolonged exposure to low doses of chlordane.

Dermal/Ocular Effects. Dermal effects were not reported in a compilation of case and personal reports of acute oral and inhalation exposure to chlordane (EPA 1980b), or in a group of library workers following inhalation and dermal exposure resulting from a spill (NIOSH 1984a), or in workers involved in chlordane manufacture (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck 1951), or in a woman who died within minutes of spilling a mixture of chemicals including chlordane on the front of her clothing (Derbes et al. 1955), or in a woman who died 9 days after ingesting a large dose of chlordane (Derbes et al. 1955). EPA (1980b), however, reported burning sensations of the skin, development of a rash and pruitis in cases of accidental acute dermal exposure to chlordane. Accidental spraying of chlordane in the eyes consistently resulted in conjunctivitis (EPA 1980b). These reports of dermal and ocular effects are complicated by exposure to mixtures of chemicals including other pesticides and vehicles such as petroleum distillates that are known to be irritants.

Oral exposure of rats and mice (NCI 1977; Velsicol Chemical Co. 1983a, 1983b) and inhalation exposure of rats and monkeys (Velsicol Chemical Co. 1984) produced no skin lesions. Datta et al. (1975) reported changes in the malpighian cells of guinea pigs dermally exposed once daily for 90 days to a high dosage of chlordane (168 mg/kg). No changes were observed in the dermis.

These data suggest that both ocular and dermal exposure to liquid chlordane may cause adverse effects, but these effects are unlikely at concentrations expected in environmental media near waste sites.

Immunological Effects. No studies were located regarding immunological effects in humans following exposure to chlordane. Sensitization was not reported in guinea pigs treated once daily for 90 days with dermal

applications of chlordane in acetone (Datta et al., 1975). It is not clear, however, that the protocol would have reliably identified dermal sensitization. Studies conducted in mice suggest that chlordane depresses cell-mediated immunity as manifested by a depression in delayed-type hypersensitivity reactions and depressed mixed lymphocyte reactivity (Barnett et al. 1985a, 1985b; Menna et al. 1985; Spyker-Cranmer et al. 1982). There were no effects on humoral-mediated immunity. The mice tested were the offspring of dams treated during gestation and allowed to nurse their young. Therefore the mice had been exposed to chlordane and its metabolites during gestation and during the lactation period. The effect on delayed-type hypersensitivity occurred at very low levels in mice, but it is not possible to draw conclusions from these studies regarding the possibility of chlordane-induced effects on immune function in humans.

Neurological Effects. Chlordane clearly causes neurological effects in humans following acute oral, inhalation or dermal exposure (Aldrich and Holmes 1969; Barnes, 1967; Dadey and Kammer 1953; Derbes et al. 1955; EPA 1980b; Lensky and Evans 1952; NIOSH 1984a). Neurological symptoms are frequently the earliest symptoms reported in cases of human intoxication with chlordane and include headache, dizziness, irritability, muscle tremors, confusion, convulsions, and coma. It is possible that gastrointestinal symptoms such as nausea, abdominal pain and diarrhea may result from effects on the nervous system. Oral exposure has been associated with central nervous system effects in children (Aldrich and Holmes 1969). The threshold concentration in air for neurologic effects in humans has not been identified, but case and personal reports (EPA 1980b; NIOSH 1984a) suggest that these effects may occur at relatively low concentrations. Neurological symptoms, however, have not been reported in studies of occupational exposure (Alvarez and Hyman 1953; Fishbein et al. 1964; Princi and Spurbeck, 1951). Acute (Hrdina et al. 1974) and prolonged (Drummond et al. 1983; NCI, 1977) oral exposure in animals has been associated with neurological signs such as tremor and convulsions. Neither human nor animal studies, however, have investigated subtle neurologic or behavioral effects, which may occur at dosages lower than the obvious effects that have been associated with chlordane exposure. Therefore, it is not possible to predict the likelihood of neurological effects in humans exposed to the low levels of chlordane expected in environmental media located near waste sites.

Developmental Effects. No data were located regarding developmental effects in humans following exposure to chlordane. No developmental effects were observed in rats treated orally during gestation with dosages sufficient to cause death of 50% of the dams (Usami et al. 1986). In a screening study, no effects on viability or postnatal growth were observed in the offspring of mice treated orally during gestation (Chernoff and Kavlock 1982). Pregnant mice were treated with chlordane in order to study the immunological, behavioral, and endocrine effects on the offspring. The offspring were allowed to nurse their dams; therefore they were exposed to chlordane and metabolites during gestation and during the nursing period.

The results of immunological tests suggest that chlordane suppressed cell-mediated immunity (Barnett et al. 1985a, 1985b; Menna et al. 1985; Spyker-Cranmer et al. 1982). Results of behavioral tests indicate that chlordane depressed the acquisition of avoidance behavior, raised seizure thresholds, and increased exploratory activity (Al-Hachim and Al-Baker 1973). Increased plasma levels of corticosterone were observed in the offspring of mice treated during gestation; the change implies an effect on neuroendocrinological feedback possibly resulting from reduced ability of the liver to metabolize corticosterone (Cranmer et al. 1984). The relevance of these findings to humans is not known, and the likelihood of effects on human development resulting from exposure to chlordane near waste sites is unpredictable.

Reproductive Effects. No studies were located regarding reproductive effects in humans following exposure to chlordane. Oral administration of chlordane to male rats (19.5 mg/kg/day for 90 days) resulted in increased androgen receptor sites in the ventral prostate (Shain et al. 1977). Testicular degeneration was reported in male mice treated by gavage at 3.2 mg/kg/day for 30 days (Balash et al. 1987). On the other hand, testicular effects were not reported in rats or mice in chronic dietary studies (NCI 1977; Velsicol Chemical Co. 1983a, 1983b) or in rats or monkeys in a 90-day inhalation study (Velsicol Chemical Co. 1984). Datta et al. (1975) reported mild degenerative changes in the testes of guinea pigs treated with dermal applications of chlordane in acetone at 168 mg/kg once daily for 90 days. Ambrose et al (1953a) reported reduced fertility, reflected as a reduction in the number of mated females that delivered litters, when male and female rats were fed a diet that provided chlordane at 16 mg/kg/day. Treatment began at weaning and continued through the lactation period. None of the litters delivered survived to weaning. Reduced fertility, reflected as a reduction in the number of females that became pregnant, was observed in mice given intraperitoneal injections of chlordane (25 mg/kg) once weekly for 3 weeks before being mated to untreated males of proven fertility (Welch et al. 1971). Data indicate that exposure to chlordane can affect metabolism and circulating levels of steroid hormones (Cranmer et al. 1984; Welch et al. 1971).

The data regarding testicular effects in male animals are equivocal. Data regarding effects on the fertility of rats and mice are limited by study design. Dosage levels were not chosen to detect thresholds for effects on fertility, and the studies were not designed to investigate the mechanisms by which chlordane may interfere with fertility. The data in animals are not sufficient to indicate whether chlordane can cause reproductive effects in humans, or whether such effects would be expected in humans exposed to chlordane near waste sites.

Genotoxic Effects. Chlordane has been tested for mutagenicity in several systems. As seen from Table 2-4, mostly negative results have been obtained for reverse mutations, DNA repair, and dominant and recessive lethal assays. Chlordane induced mitotic gene conversion in Saccharomyces

TABLE 2-4. Genotoxicity of Chlordane

Assay	Indicator	Activation	Response	References
Reverse mutation	Salmonella typhimurium	•	- `	Probst and Hill 1981, Gentile et al. 1982 Encegovich and Rashid 1977 Mortelmans et al. 1986 Simmon et al. 1977 Ashby and Tennant 1988
	<u>Escherichia</u> <u>coli</u>	•	•	Probst and Hill. 1981
Mitotic gene conversion	Saccharomyces cerevisiae	+ -	+ -	Gentile et al. 1982
		NR	+	Chambers and Dutta 1976
Prophage induction	E. coli	÷	+	Houk and DeMarini 1987
DNA repair and synthesis	<u>S. typhimurium</u> <u>E. coli</u>	•	•	Rashid and Mumma 1986 Rashid and Mumma 1986
	Rat hepatocytes Rat hepatocytes	NA NA	-	Probst and Hill 1981 Haslansky and Williams 198
	Mouse hepatocytes Hamster hepatocytes	NA NA	-	 Mastansky and Witliams 198 Mastansky and Williams 198
	Human fibroblasts HeLa cells (human uterine cells)	HA NA	•/ •	Ahmed et al. 1977a Griffin and Hill 1978
	(Maneri acci inc berry)		•	Brandt et al. 1972 Blevins and Sholes 1978
Sister chromatid exchange	Human lymphoid cells	NA	+	Sobti et al. 1983
Dominant lethal	Mouse	NA	-	Arnold et al. 1977
	Mouse	NA	•	Epstein et al. 1972
Recessive lethal	<u>Drosophila</u> <u>melanogaster</u>	NA	-	Vogel 1980
Mutagenic activity	Chinese hamster ovary	NA NA	• •	Tsushimoto et al. 1983 Ahmed et al. 1977b
HGPRT ⁸	Rat hepatocytes	NA	-	Telang et al. 1981, Tong et al. 1981

 $^{^{\}rm B}$ Human cell hypoxanthine-guanine phosphoribosyl transferase mutagenesis assay NA = not applicable; NR = not reported.

<u>cerevisiae</u> in the presence, but not the absence, of metabolic activation; and in prophage in E. coli, regardless of metabolic activation. Chlordane also induced sister chromatid exchange in human lymphoid cells. The generally negative results for mutagenicity of chlordane are consistent with an epigenetic mechanism of carcinogenicity (see below).

Cancer. Chronic oral treatment with chlordane resulted in significant increases in hepatocellular carcinomas in mice (EPA 1985b, 1986c, 1987e, 1987f; Epstein 1976; IRDC 1973; NCI 1977; Reuber 1978; Velsicol Chemical Co. 1983b). Becker and Sell (1979) demonstrated that chlordane could induce hepatocellular carcinomas in a strain of mice that historically is not predisposed to liver tumors. No statistically significantly increased tumor incidence was observed in dietary studies in rats in the NCI (1977) study or in EPA (1988c) reevaluations of the Velsicol Chemical Co. (1983a) study. Negative responses in a number of mutagenicity tests suggest an epigenetic mechanism of carcinogenicity, perhaps involving a promoting effect on predisposed cells (Ashby and Tennant 1988; Maslansky and Williams 1981). Ashby and Tennant (1988) also noted that a positive carcinogenic response in the mouse liver in NCI/NTP studies may reflect an epigenetic as well as a genotoxic mechanism. Williams and Numoto (1984) presented evidence that chlordane acts as a promoter of liver tumor formation in cells initiated by diethylnitrosamine. Teland et al. (1982) observed a marked inhibition of intercellular communication in cultured rat liver cells treated with chlordane. The investigators reported that this effect is common to other organochlorine pesticides that appear to act as tumor promoting agents. EPA (1987e) considered the data in laboratory animals sufficient to classify chlordane in Group B2, that is, as a probable human carcinogen.

2.4 LEVELS IN HUMAN TISSUES AND FLUIDS ASSOCIATED WITH HEALTH EFFECTS

The most sensitive indicators of acute chlordane toxicity in humans are CNS effects including headache, confusion, behavioral aberrations and tremors. At high levels of exposure, the CNS effects include convulsions, coma, respiratory failure, and eventually death. Information on the levels of chlordane in the blood of individuals that are associated with CNS effects come from cases involving ingestion of the compound. Levels of chlordane in the blood ranging from 2.71 - 3.4 mg/L have been found to be associated with convulsions and seizures (Aldrich and Holmes 1969; Curley and Garrettson 1969). In both of these cases, the intoxicated individuals recovered following medical intervention. A blood chlordane level of 5 mg/L was found in association with seizures, respiratory failure, and coma (Olanoff et al. 1983). This individual also recovered following medical intervention. This latter value of 5 mg chlordane/L of blood may represent an upper limit for human survival following chlordane ingestion. A fatality has been reported in the recent literature (Kutz et al. 1983) in which the victim had a blood chlordane level of 4.87 μ g/g approximately 2 hours after death. Conversion of this value to units of ppm (mg/L) using a human blood density of 1.058 g/mL (Snyder et al. 1981) results in a blood chlordane concentration of 5.15 ppm. Care must be exercised in the interpretation of

REFERENCES

Al-Hachim GM, Al-Baker A. 1973. Effects of chlordane on conditioned avoidance response, brain seizure threshold and openfield performance of prenatally-treated mice. Brit J. Pharmacol 49:311-315.

Aldrich FD, Holmes JH. 1969. Acute chlordane intoxication in a child: Case report with toxicological data. Environ Health 19:129-132.

Alvarez, WC, Hyman S. 1953. Absence of toxic manifestations in workers exposed to chlordane. AMA Arch Ind Hyg Occup Med 8:480-483.

Ambrose AM, Christenson HC, Robbins DJ, et al. 1953a. Toxicological and pharmacological studies on chlordane. Ind Hyg Occup Med 7: 197-210.

Ashby J, Tennant RW. 1988. Chemical structure, Salmonella mutagenicity and extent of carcinogenicity as indicators of genotoxic carcinogenesis among 222 chemicals tested in rodents by the U.S. NCI/NTP. Mutat Res 204:17-115.

Balash KJ, Al-Omar MA, Abdul Latif BM. 1987. Effect of chlordane on testicular tissues of Swiss mice. Bull Environ Contam Toxicol 39:434-442.

Balistreri WF, Partin JC, Schubert WK. 1973. Hepatic necrosis following accidental chlordane exposure. Pediatr Res 7:319

Barnes R. 1967. Poisoning by the insecticide chlordane. Med J Austr 54:972-973.

Barnett JB, Holcomb D, Menna JH, et al. 1985a. The effects of prenatal chlordane exposure on specific anti-influenza cell-mediated immunity. Toxicol Lett 25:229-238.

Barnett JB, Soderberg LSF, Menna JH. 1985b. The effect of prenatal chlordane exposure on the delayed hypersensitivity response of Balb/c mice. Toxicol Lett 25:173-183.

Becker FF, Sell S. 1979. Alpha-fetoprotein levels and hepatic alterations during chemical carcinogenesis in C57BL/6N mice. Cancer Research 39:3491-3494.

Casterline JL, Williams CH. 1971. The effects of 28-day pesticide feeding on serum and tissue enzyme activities of rats fed diets of varying casein content. Toxicol Appl Pharmacol 18:607-618.

Chernoff N, Kavlock RJ. 1982. An in vivo teratology screen utilizing pregnant mice. J. Toxicol Environ Health 10:541-550.

Cram RL, Juchau MR, Fouts JR. 1956. Stimulation by chlordane of hepatic drug metabolism in the squirrel monkey. J Lab Clin Med 66:906-911.

Cranmer JM, Cranmer MF, Goad PT. 1984. Prenatal Chlordane Exposure: effects on plasma corticosterone concentrations over the lifespan of mice. Environ Res 35:204-210.

Curley A, Garrettson LK. 1969. Acute chlordane poisoning. Clinical and chemical studies. Arch Environ Health 18:211-215.

Dadey JL, Kammer AG. 1953. Chlordane intoxication. Jour Am Med Assoc 153:723.

Datta KK, Gupta PC, Dikshith TSS. 1975. Effect of chlordane on the skin of male guinea pigs. In: Zaida SH, ed. Environmental Pollution and Human Health. Lucknow, India, Industrial Toxicology Research Centre, pp. 608-611.

Den Tonkelaar EM, Van Esch GJ. 1974. No-effect levels of organochlorine pesticides based on induction of microsomal liver enzymes in short-term toxicity experiments. Toxicology 2:371-380.

Derbes VJ, Dent JH, Forrest WW, et al. 1955. Fatal chlordane poisoning. J Amer Med Assn 158:1367-1369.

Ditraglia D, Brown DP, Namekata T, et al. 1981. Mortality study of workers employed at organochlorine pesticide manufacturing plants. Scand J Work Environ Health 7:140-146.

Drummond L, Chetty KN, Desaiah D. 1983. Changes in brain ATPases in rats fed on chlordane mixed with iron-sufficient and deficient diet. Drug and Chem. Tox. 6:259-266.

EPA. 1980b. Summary of Reported Pesticide Incidents Involving Chlordane. Pesticide Incident Monitoring System Report No. 360. Office of Pesticide Programs, Washington, DC.

EPA. 1985b. Memorandum. 6(a) (2) Data on chlordane. Chronic mouse and rat studies for oncogenicity testing. Acc, Nos. 252267, 254665, 251815, Review No. 004635. Office of Pesticides and Toxic Substances, Washington, DC.

EPA. 1986c. Carcinogenicity Assessment of Chlordane and Heptachlor, Heptachlor Epoxide. Office of health and Environmental Assessment, Washington, DC. NTIS No. PB87-208757.

EPA. 1987e. Integrated Risk Information System (IRIS). Risk Estimate for Carcinogenicity for Chlordane. Online (Verification date 4/1/87). Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

EPA. 1987f. Drinking Water Criteria Document for Heptachlor, Heptachlor Epoxide, and Chlordane. Final. Prepared for Office of Drinking Water by Environmental Criteria and Assessment Office, Cincinnati, OH.

EPA. 1988c. Memorandum. Review of Pathology Working Group slide reevaluation of livers of rats in a 30-month oral exposure to chlordane. Accession No. 404337-01 (3 volumes). Review No. 006615. Office of Pesticides and Toxic Substances, Washington, DC.

Epstein SS. 1976. Carcinogenicity of heptachlor and chlordane. Science of the Total Env. 6:103-154.

Fishbein WI, White JV, Isaacs HJ. 1964. Survey of workers exposed to chlordane. Ind Med Surg 10:726-727.

Gaines TB. 1960. The acute toxicity of pesticides to rats. Toxicol Appl Pharmacol 2:88-90.

Harrington JM, Baker EL Jr, Folland DS, et al. 1978. Chlordane contamination of a municipal water system. Environ Res 15:155-159.

Hart LG, Shultice RW, Fouts JR. 1963. Stimulatory effects of chlordane on hepatic microsomal drug metabolism in the rat. Toxicol Appl Pharmacol 5:371-386.

Hrdina PD, Peters DA, Singhal RL. 1974. Role of Noradrenaline, 5-hydroxytryptamine and acetylcholine in the hypothermic and convulsive effects of alpha-chlordane in rats. Eur J of Pharm 26:306-312.

Ingle L. 1953. The toxicity of chlordane vapors. Science 118:213-214.

Ingle L. 1965. A Monograph on Chlordane Toxicological and Pharmacological Properties. University of Illinois, Urbona, Illinois.

IRDC. 1973. (International Research and Development Corp.) Unpublished report to Velsicol Chemical Corporation, eighteen month oral carcinogenic study in mice. December 14. (Cited in Epstein, 1976).

Johnson KW, Holsapple MP, Munson AE. 1986. An immunotoxicological evaluation of gamma-chlordane. Fundam Appl Toxicol 6:317-326.

Lensky P, Evans M. 1952. Human poisoning by chlordane. J Am Med Assoc 149:1394.

MacMahon B, Monson RR, Wang HH, et al. 1988. A second follow-up of mortality in a cohort of pesticide applicators. J Occup Med 30:429-432.

- Maslansky CJ, Williams GM. 1981. Evidence for an epigenetic mode of action in organochlorine pesticide hepatocarcinogenicity: A lack of genotoxicity in rat, mouse and hamster hepatocytes. J Toxicol Environ Health 8:121-130.
- Menna H, Barnett JB, Soderberg LSF. 1985. Influenza Type A virus infection of mice exposed in utero to chlordane; survival and antibody studies. Toxicol Lett 24:45-52.
- NCI. 1977. (National Cancer Institute). Bioassay of chlordane for possible carcinogenicity. Technical Report Series No. 8 U.S. Dept of Health, Education and Welfare, NIH PB 271 977.
- NIOSH. 1984a. (National Institute for Occupational Safety and Health). Health Hazard Evaluation Report HETA 83-444-1481. Georgetown University, Washington, DC. NIOSH. Cincinnati, OH. NTIS No. PB 85-220895.
- Princi F, Spurbeck GH. 1951. A study of workers exposed to the insecticides chlordane, aldrin, dieldrin. Ind Hyg Occup Med 3:64-72.
- Reuber MD. 1978. Carcinomas and other lesions of the liver in mice ingesting organochlorine pesticides. Clin Toxicol 13:231-256.
- Shain SA, Shaeffer JC, Boesel RW. 1977. The effect of chronic ingestion of selected pesticides upon rat ventral prostate homeostasis. Toxicol Appl Pharmacol 40:115-130.
- Shindell S and Ulrich S. 1986. Mortality of workers employed in the manufacture of chlordane: An update. J Occup Med 28:497-501.
- Spyker-Cranmer JM, Barnett JB, Avery DL, et al. 1982. Immunoteratology of chlordane: cell-mediated and humoral immune responses in adult mice exposed in utero. Toxicol Appl Pharmacol 62:402-408.
- Telang S, Tong C, Williams GM. 1982. Epigenetic membrane effects of a possible tumor promoting type on cultured liver cells by the nongenotoxic organochlorine pesticides chlordane and heptachlor. Carcinogenesis 3:1175.
- Truhaut R, Gak J-C, Graillot C. 1974. [Organochlorine insecticides; Research work on their toxic action (its modalities and mechanisms): I. Comparative study of the acute toxicity on the hamster and the rat] J Eur de Toxicol 7:159-166. (French)
- Usami M, Kawashima K, Nakaura S, et al. 1986. Effects of chlordane on prenatal development of rats. Eisei Shikenso Hokoku 104:68-73.

Velsicol Chemical Co. 1983a. Thirty-month chronic toxicity and tumorigenicity test in rats by chlordane technical. Unpublished study by Research Institute for Animal Science in Biochemistry and Toxicology (RIASBT), Japan. (Cited in EPA 1985a,b,c; EPA 1988c).

Velsicol Chemical Co. 1983b. Twenty-four month chronic toxicity and tumorigenicity test in mice by chlordane technical. Unpublished study by Research Institute for Animal Science in Biochemistry and Toxicology (RIASBT), Japan. (Cited in EPA 1985b,c).

Velsicol Chemical Co. 1984. Chlordane: A 90-day inhalation toxicity study in the rat and monkey. Unpublished study No.VCL28 conducted by Huntingdon Research Centre. (Available only in EPA 1987g).

Wang HH, MacMahon B. 1979a. Mortality of workers employed in the manufacture of chlordane and heptachlor. J Occup Med 21:745-748.

Wang HH, MacMahon B. 1979b. Mortality of pesticide applicators. J Occup Med 21:741-744.

Welch RM, Harrison Y. 1966. Reduced drug toxicity following insecticide treatment. Pharmacologist 8:217.

Welch RM, Levine W. Kuntzman R, et al. 1971. Effect of halogenated hydrocarbon insecticides on the metabolism and uterotrophic action of estrogens in rats and mice. Toxicol Appl Pharmacol 19:234-246.

Who. 1984. Environmental Health Criteria 34 Chlordane. World Health Organization, Distribution and Sales Service, 1211 Geneva 27, Switzerland, 1984, 82 p.

Williams GM, Numoto S. 1984. Promotion of mouse liver neoplasms by the organochlorine pesticides chlordane and heptachlor in comparison to dichlorodiphenyltrichloroethane. Carcinogenesis 5:1689-1696.