DEVELOPMENT OF HEALTH CRITERIA FOR SCHOOL SITE RISK ASSESSMENT PURSUANT TO HEALTH AND SAFETY CODE 901(g):

IDENTIFICATION OF POTENTIAL CHEMICAL CONTAMINANTS OF CONCERN AT CALIFORNIA SCHOOL SITES

FINAL REPORT

June 2002

Integrated Risk Assessment Section
Office of Environmental Health Hazard Assessment
California Environmental Protection Agency

Final Report

June 2002

Development of Health Criteria for School Site Risk Assessment Pursuant to Health and Safety Code, Section 901(g):

Identification of Potential Chemical Contaminants of Concern at California School Sites

Office of Environmental Health Hazard Assessment
California Environmental Protection Agency
1001 I Street, 12th Floor
P.O. Box 4010
Sacramento, California 95812-4010

LIST OF CONTRIBUTORS

Authors
Susan A. Knadle Ph.D., DABT, and David Chan, D.Env.
Kathleen Stewart

Primary Reviewers:
Jim Carlisle, DVM
David Siegel, Ph.D., DABT
George Alexeeff, Ph.D., DABT

Secondary Reviewers
Robert Schlag, MSc
Melanie Marty, Ph.D.
Andy Salmon, Ph.D.
Robert Haas, Ph.D.
Ching Hung Hsu, Ph.D.

Library Support
Jerry McGovern

Website Posting
Laurie Monserrat

Identification of Potential Contaminants

Appendices

Development of Health Criteria for School Site Risk Assessment Pursuant to Health and Safety Code, Section 901(g):

Identification of Potential Chemical Contaminants of Concern at California School Sites

Executive Summary

This report provides a status regarding the Office of Environmental Health Hazard Assessment's (OEHHA's) implementation of Health and Safety Code (HSC) Section 901(g). The report also serves a means for OEHHA to obtain interested parties' comments on the technical approach taken and on the potential chemical contaminants of concern to be evaluated for purpose of developing health guidance values.

Specifically, HSC Section 901(g) requires that:

1. On or before January 1, 2002, OEHHA, in consultation with the appropriate entities within the California Environmental Protection Agency (Cal/EPA), shall identify those chemical contaminants commonly found at school sites and determined by OEHHA to a be of greatest concern based on criteria that identify child-specific exposure and child-specific physiological sensitivities.
2. On or before December 31, 2002, and annually thereafter, OEHHA shall publish and make available to the public and other state and local environmental and public health agencies and school districts, numerical health guidance values for five of those chemical contaminants identified until the contaminants identified have been exhausted.

This report summarizes efforts undertaken to identify potential chemical contaminants of concern, and the evaluation of the first five of these contaminants will be addressed in a separate report. It should be noted that the identification of potential contaminants of concern and the development of numerical health criteria for these contaminants depends on the availability of appropriate data. As such, OEHHA underscores the use of an iterative process (rather than a definitive process) for identifying contaminants, and a feasibility study approach in developing numerical health guidance values.

In tackling the first part of the mandate, OEHHA performed a broad review of federal and state programs to identify relevant monitoring data regarding the presence of chemical contaminants at school sites. OEHHA also conducted extensive literature searches to identify applicable studies concerning child-specific physiological sensitivities of environmental contaminants. While available data did not permit OEHHA to definitively identify those chemicals that are commonly found at school sites and for which children have unique physiological sensitivities, OEHHA has identified a group of candidate chemicals that will likely include contaminants found at school sites (comprising approximately 200 chemicals), and another group that has the potential for causing adverse effects on school-age children (approximately 198 chemicals). The creation of these compilations of chemicals is solely for facilitating the development of numerical health based criteria. It is imperative that we create these compilations now so that numerical health criteria can be developed expeditiously for application in school-site risk assessment. The lead time required for developing health-based criteria certainly argues for working from these candidate chemicals, rather than waiting for complete information to create a definitive list of chemical contaminants that are found at school sites and determined to be of greatest concern based on child-specific physiological sensitivities. Under our iterative approach, these compilations will be updated as new information becomes available.

The first compilation of chemicals, which defines the boundaries of what are likely to be found at school sites, is based on the following inclusive criteria:

- Chemicals that have been targeted by federal and state agencies and are likely to be found at school sites should be included.
- Contaminants that were or are known to be present in the pertinent environmental media of California should be considered.

Using these criteria, OEHHA considers the following as candidate contaminants that are likely to be found at school sites:

- Ninety-four soil contaminants that have been reported in school site Preliminary Endangerment Assessments (PEA) and reviewed by the Department of Toxic Substances Control (DTSC). These soil contaminants are found at potential school sites and are likely to be found at existing school sites.
- Ninety Toxic Air Contaminants that were emitted to, or detected in, California's ambient air. They are used as a proxy to represent potential contaminants that may be found in the outdoor air of schools.
- Sixty potential classroom contaminants targeted for monitoring by the Department of Health Services (DHS) and Air Resources Board (ARB), as part of their Portable Classroom Program.
- Forty-seven toxic chemicals targeted by US EPA in its National Human Exposure Assessment Survey (NHEXAS) and twenty-six contaminants targeted by US EPA in its Total Exposure Assessment Methodology (TEAM) studies because of their prevalence or persistence in the environment. These

chemicals are deemed likely to be found in various environmental settings at schools, as well as their surrounding communities.

There is a fair amount of overlap among the chemicals reported or targeted by the above federal and state agencies.

The second compilation of chemicals, which outlines the boundaries for chemicals with potential child-specific sensitivities, is based on the following criteria:

- One or more citations in the scientific literature indexed by National Library of Medicine's online database, PubMed, which indicate the potential for chemically-induced adverse effects on the nervous, respiratory, reproductive, endocrine, immune system or initiated cancer during the perinatal period and childhood. These organ systems are targeted because they are characterized by having sensitive periods, or critical windows, of cell proliferation, migration, and differentiation during childhood during which chemical insults may produce adverse effects at relatively low doses. These effects may not be recognized until maturity.
- The chemical was identified as a Proposition 65 Developmental and Reproductive Toxin by OEHHA.

Of the 198 chemicals identified in the second compilation, about 87 were from the Proposition 65 list of developmental and reproductive toxicants. Chemicals on the Proposition 65 list that were not likely contaminants at school sites, such as pharmaceuticals, were excluded from the compilation of chemicals.

OEHHA's compilations must be viewed as a living document, which will require additions and deletions as more data becomes available to OEHHA in the future. While we have attempted to include all reasonable candidates on the initial lists, ongoing literature reviews and results from studies of our sister agencies may turn up additional candidate chemicals. Conversely, the list may include some chemicals that ultimately lack sufficient data to conclude that there is a differential effect on children, or may be found not to occur commonly at schools. For example, the compilation will be revised when results of the DHS-ARB "California Portable Classrooms" study become available. OEHHA will also consider additional candidates that DTSC identifies in its upcoming PEA reviews, and that DPR identifies from pesticide use notification by school districts pursuant to AB 2260 (the Healthy School Act of 2000). Furthermore, the compilation will be updated upon the completion of an in-depth evaluation of priority chemicals discussed below.

HSC 901 (g) also stipulates that by December 31, 2002, and annually thereafter, OEHHA develops numerical health-based guidance for five of the identified chemicals until the list is exhausted. The child-specific health guidance values are for use by the Department of Toxic Substances Control and other state and local environmental and public health agencies, to

assess exposures and health risks at existing and proposed schoolsites using a guidance document being prepared by OEHHA.

Table ES 1 tallies chemicals common to both compilations. As a next step, OEHHA will select a subset of chemicals from Table ES 1 for further evaluation to determine if there are sufficient data on critical toxic effects in the organ systems that are developing in children to support the creation of child-specific numerical guidance values based on a non-cancer end-point. We are currently focusing on non-cancer end-points, pending the development of cancer risk assessment guidelines for early in life exposures under a separate task pursuant to HSC Section 901(e). We will evaluate the cancer endpoint when these guidelines become available.

The following criteria are proposed for selecting chemicals from Table ES 1 for an in-depth review:

- Chemicals having strongest indication of their presence at school sites.
- Chemicals with evidence of possible adverse effects in three or more of the systems that are undergoing critical development during childhood: the neurological, immunological, respiratory, reproductive, or endocrine systems.
- Where applicable, chemical carcinogens with an existing non-cancer reference dose (RfD) that approximates the dose associated with a 10^{-4} (one in ten thousand) to 10^{-6} (one in a million) cancer risk and that is based on toxicity studies in adult animals.
- Chemicals which have been identified as a concern by other OEHHA programs.

The third criterion helps target those chemicals that could conceivably be a non-cancer risk driver when new data are considered. The premise is that if current data suggest both carcinogenic and non-carcinogenic effects of concern occur at similar dose levels in adults, and if new data show children may be more susceptible than adults for non-carcinogenic effects, the chemical should have a high priority for further evaluation because its newly observed non-cancer effect may be of great concern to children's health. Normally, an exposure level of concern for a carcinogenic chemical is much lower than for its non-carcinogenic effects, so increased susceptibility of children for its non-carcinogenic effects would not necessarily make it a priority chemical for further study.

Table ES 1
Chemicals Common to Both Compilations

Acetaldehyde
Acrolein
Aldrin
Aluminum
Arsenic
Benzene
Benzidine
Beryllium compounds
Boron
1,3-Butadiene
Cadmium
Carbon disulfide
Carbon tetrachloride
Chlordane
Chlorine
Chloroprene
Chlorpyrifos
Chromium III
Chromium VI
2,4-D Butyric acid (2,4-DB)
DDD
DDE
DDT
Diazinon
Dibromochloropropane (DBCP)
Dichlorvos
Dieldin
Diesel exhaust particles
Dimethyl sulfate (DMS)
Dioxins and Dibenzofurans (TCDD)
Diquat
Endosulfan
Epichlorohydrin
Ethylene dibromide
Ethylene glycol ethers and acetates
  Ethylene glycol monoethyl ether
  Ethylene glycol monoethyl ether acetate
  Ethylene glycol monomethyl ether
Ethylene oxide
Formaldehyde
Heptachlor
Heptachlor epoxide
Hexachlorobenzene
ã-Hexachlorocyclohexane (Lindane)
n-Hexane
Isocyanates
Lead
Malathion
Manganese
Mercury (organic and inorganic)
Methanol
Methoxychlor
Methyl chloride (Chloromethane)
Methylene chloride (Dichloromethane)
Methyl ethyl ketone
Nickel (inorganic)
PAHs
PCBs
Pentachlorophenol (PCP)
Petroleum hydrocarbons
Phthalates
  Dibutyl phthalate
  Di(2-ethylhexyl)phthalate (DEHP)
  Diethyl phthalate
  Dimethyl phthalate
  Phthalic anhydride
Propylene oxide
Pyrethroids
  Permethrin
Styrene
Tetrachloroethylene (PERC/PCE)
Toluene
Toxaphene
Triazine herbicides
  Atrazine
  Simazine
1,1,1-Trichloroethane (Methyl chloroform)
Trichloroethylene
Trifluralin
Vinyl chloride
Vinylidene chloride
Xylene

Internal reviews, an interagency advisory panel, request for data, and/or workshops will be considered as possible mechanisms for collecting input into the prioritization and selection of chemicals for the in-depth review and for developing health criteria based on available data. A minimum of five chemicals will be selected from this prioritization process for in-depth evaluation for the purpose of developing health-based numerical guidance values.

1. Introduction

A growing body of literature has indicated for some time that the fetus and children may be more susceptible to certain chemicals and physical agents than adults. The demonstration that lead, which had become pervasive in the environment, could cause irreversible decrements in intelligence quotient (IQ) and neurobehaviorial effects in children at blood lead levels less than those that caused hematopoietic toxicity in adults and children (Needleman, 1979, 1990) spurred action to reduce the exposure of children. At the same time, evidence was accumulating that chronic diseases such as asthma and certain types of childhood cancer were increasing, and that chemical contaminants in a child's environment, including pesticides, could disrupt developing nervous, immune, endocrine, and respiratory systems (Goldman, 1998). The discovery of methane gas, hydrogen sulfide and volatile organic compounds inside the almost finished new Belmont Learning Center in the Los Angeles Unified School District demonstrated that schools, even new schools, were not free of chemical contaminants.

This report represents a review of information available on a national and State level pertaining to chemicals that are environmental contaminants that may also be present at school sites. Since monitoring of actual school sites for some specific chemical classes was just beginning, this report contains a compilation of chemicals from a variety of national and State lists, as well as a list of chemicals which a literature review suggests might be more hazardous to children than to adults. The reader should be cautioned that the presentation of this compilation of over 200 chemicals does not suggest that all or any of these chemicals are present at school sites at a concentration that would be harmful to children. Since there is no actual monitoring data at this point, there is no evidence that any of the chemicals in this compilation are present. The intention of OEHHA is to utilize this compilation for further in-depth review of scientific studies on the effects of these chemicals on children, and to create child-specific health guidance values if the data on toxic effects indicates that children are more susceptible than adults.

The child-specific health guidance values are for use by the Department of Toxic Substances Control and other state and local environmental and public health agencies, to assess exposures and health risks at existing and proposed schoolsites using a guidance document being prepared by OEHHA.

1.1 Federal Activities to Protect Children's Health

The National Academy of Sciences highlighted the problem of children's susceptibility to pesticides when it released its 1994 report, "Pesticides in the Diets of Infants and Children." As a result, Congress unanimously passed landmark pesticide food safety legislation in 1996, known as the Food Quality Protection Act. The legislation established positive new directions to protect public health, especially for children, by amending two major federal statutes, the Federal Food, Drug, and Cosmetic Act (FFDCA) and the Federal Insecticide, Fungicide, and

Rodenticide Act (FIFRA), administered by United States Environmental Protection Agency (US EPA). The law explicitly required the US EPA to address risks to infants and children and to publish a specific safety finding before a pesticide tolerance level in food could be established. It also provided an additional tenfold safety factor unless reliable data could show that a lower factor would be safe.

In 1996, then US EPA Administrator, Carol Browner, directed the agency to begin to explicitly and consistently take into account environmental health risks to infants and children in all risk characterizations and public health standards set for the United States. Carol Browner initiated this act by announcing a seven-step *National Agenda to Protect Children's Health from Environmental Threats.* This agenda instructed the agency to 1) ensure that Environmental Protection Agency (EPA) standards were protective of children, 2) develop a scientific research strategy to focus on the gaps in knowledge regarding child-specific susceptibility and exposure to environmental pollutants, 3) develop new, comprehensive policies to address cumulative and simultaneous exposures faced by children, 4) expand community right-to-know, 5) encourage parental responsibility for protecting children by providing them with information, 6) encourage and expand educational efforts with health care providers and environmental professionals, and 7) provide funding to address children's environmental health as a top priority among relative health risks (US EPA, 1996).

In 1996 the Agency for Toxic Substances and Disease Registry (ATSDR) also launched a Child Health Initiative. This initiative was designed to emphasize policies and projects that promote the health of infants, children and youth. Their projects can be found on their website at http://www.atsdr.cdc.gov/child. Executive Order 13045, Protection of Children from Environmental Health Risks and Safety Risks (62 Fed. Reg. 19885, April 23, 1997), signed by President Clinton, extended the need to protect children to all federal agencies. Section 1-101 states in part:

> "A growing body of scientific knowledge demonstrates that children may suffer disproportionately from environmental health risks and safety risks. These risks arise because: children's neurological, immunological, digestive, and other bodily systems are still developing; children eat more food, drink more fluids, and breathe more air in proportion to their body weight than adults…."

This executive order established a Children's Environmental Health Task Force with the Secretary of Health and Human Services and the Administrator of US EPA as co-chairs to identify, assess, and manage risks to children from toxic substances and other environmental hazards, and it required all federal agencies to ensure that their environmental regulations adequately protect children.

1.2 California Takes New Action to Protect Children from Environmental Health Risks

Recognition of the potential for children to have increased susceptibility to some environmental contaminants and the growing concern over potential exposures in the school environment led the California Legislature and Governor Davis to meet the challenge to protect California's children from environmental health risks. In the 1999-2000 session the legislature passed, and Governor Davis signed into law, a series of legislative provisions to protect children from environmental contaminant exposure.

Health and Safety Code (HSC) Section 39669.5(a), which resulted from passage of SB 25, requires the Office of Environmental Health Hazard Assessment (OEHHA) by January 1, 2003, to 1) evaluate the adequacy of the current monitoring network to gather data to determine exposure of infants and children to air pollutants including criteria air pollutants and toxic air contaminants, and 2) identify areas where the exposure of infants and children to air pollutants is not adequately measured by the current monitoring network, and recommend changes for improvement. Pursuant to HSC Section 39669.5(a), OEHHA identified five toxic air contaminants…that may pose infants and children to be especially susceptible to illness. In developing the list, OEHHA took into account public exposures to toxic air contaminants, whether by themselves or interacting with other toxic air contaminants or criteria pollutants, and the availability of data on health effects, including potency, mode of action, and other relevant biological factors. To meet the criteria in HSC Section 39660, OEHHA, to the extent information was available, assessed 1) exposure patterns among infants and children that are likely to result in disproportionately high exposure to ambient air pollutants in comparison to the general population, 2) special susceptibility of infants and children to ambient air pollutants, 3) the effects on infants and children of exposure to ambient air pollutants and other substances that have a common mechanism of toxicity, and 4) the interaction of multiple air pollutants on infants and children, including the interaction between criteria air pollutants and toxic air contaminants.

The Healthy School Act of 2000, Education Code sections 17608-17613 and Food and Agricultural Code Sections 13180-13188 codified the Department of Pesticide Regulation's (DPR) School Integrated Pest Management Program, and added new requirements regarding pesticide use notification and reporting. These new provisions require each school district to notify all school staff and parents or guardians of students regarding pesticide use in the upcoming year, and licensed and certified pest control operators to report pesticide use and applications at school sites annually to DPR. The new requirements will facilitate OEHHA's efforts in identifying pesticides that are found at school sites.

The HSC 901(a–e) (AB 2872) contains a variety of provisions designed to protect children from excess risks from carcinogens that were identified from studies on adults. OEHHA is 1) reviewing existing federal and state cancer risk assessment guidelines for use by the office and

the other entities within Cal/EPA to consider the extent to which the guidelines address early in life exposures and to establish cancer potency values or numerical health guidance values that adequately address carcinogenic exposures to the fetus, infants, and children, 2) developing criteria for identifying carcinogens likely to have greater impact if exposures occur early in life, 3) assessing methodologies used in existing guidelines to address early-in-life exposures, 4) constructing a data base of animal studies to evaluate increases in risks from short-term early-in-life exposures, and 5) by June 30, 2004, will finalize and publish cancer guidelines that shall be protective of children's health.

Health and Safety Code 901(f) requires that on or before December 31, 2002, OEHHA publish a guidance document for use by the Department of Toxic Substances Control (DTSC) and other state and local environmental and public health agencies, to assess exposures and health risks at existing and proposed school sites, which include 1) appropriate child-specific routes of exposure unique to the school environment, in addition to those in existing exposure assessment models, 2) appropriate available child-specific numerical health effects guidance values, and plans for the development of additional child-specific numerical health effects guidance values, and 3) the identification of uncertainties in the risk assessment guidance, and those actions that should be undertaken to address those uncertainties.

Health and Safety Code 901(g) requires OEHHA by January 1, 2002, to identify those chemical contaminants commonly found at school sites and determined by the office to be of greatest concern based on criteria that identify child-specific exposures and child-specific physiological sensitivities. By December 31, 2002, OEHHA will publish and make available numerical health guidance values for five of those chemical contaminants.

OEHHA is coordinating efforts internally and working with Cal/EPA and other agencies in concert to implement these new legislations. This report summarizes the results of OEHHA's efforts in implementing statute HSC 901(g) with respect to the identification of chemical contaminants.

1.3 Methods of Approach

Health and Safety Code 901(g) calls for the identification of those chemicals that are commonly found at schools sites and determined to pose the greatest health concern based on child-specific exposures and child-specific physiological sensitivities. The Portable Classroom study authorized under HSC Section 39619.6 is the only program that directly monitors one of the environmental media at school sites, and it is designed to sample a relatively large number of schools for chemicals thought to be of greatest concern for portable classroom occupants. However, the programs of other state and federal activities help identify a greater range of contaminants that are likely to be found at school sites. Similarly, since data on child-specific physiological sensitivity is not readily available for most environmental contaminants, data on

chemically-induced adverse health effects on development, reproduction, and/or on certain critical organs will help determine the number and types of contaminants that are considered for further evaluation and assessment.

OEHHA is taking an inclusive approach by compiling two groups of "candidate" chemicals. These two groups comprise 1) lists of chemicals that other agencies have thought were of health concern and 2) chemicals that OEHHA has thought may be of special concern to children.

The first group consists of chemicals that have been targeted by federal and state agencies because they are thought to present major environmental health risks and have been found in two or more environmental media, and therefore are likely to be found at school sites. This approach reflects the views of US EPA's Science Advisory Board and Cal/EPA's Risk Assessment Advisory Committee report that the aggregate exposures to all toxic substances from all sources by all routes need to be considered to protect children.

The second group is composed of chemicals that California has already identified as Developmental and Reproductive Toxicants (DART) under Proposition 65, or for which there is some scientific data on the potential to cause cancer in childhood or adverse effects to developing nervous, respiratory, reproductive, endocrine, or immune systems. These organ systems are targeted because they are characterized by having sensitive periods, or critical windows, of cell proliferation, migration, and differentiation during childhood. This is a time during which even minimal chemical insults may produce adverse effects that may not be recognized until maturity. The criteria for inclusion in this compilation are:

- There are one or more citations when the scientific literature indexed by the National Library of Medicine's online database, PubMed, was searched for chemically-induced adverse effects on the nervous, respiratory, reproductive, endocrine or immune systems or carcinogenesis during early development and childhood.
- The chemical was identified as a Proposition 65 Developmental and Reproductive Toxin by OEHHA.

The compilations in this report include a range of chemical contaminants that have been widely found in two or more environmental media (air, water, soil, or food) and for which there are some toxicity studies suggestive of a greater effect in the developing organ systems of children than in adults. However, the information obtained to produce these compilations is not sufficient to conclude that these chemicals are found in most schools or that children have a greater sensitivity than adults to them. These compilations have been prepared to assist OEHHA scientists in selecting chemicals for further in-depth study to determine if sufficient studies exist on toxicity to developing organ systems in the young to create a health guidance value specific for children. Therefore, this report is an internal working document without any regulatory status.

2. Identification of Candidates for Chemicals Potentially Found at School Sites

This chapter provides a description of OEHHA's efforts in meeting the first part of the mandate, Health and Safety Code Section 901(g), concerning the identification of chemical contaminants commonly found at school sites.

Historically, public health and environmental impacts of chemical contaminants are evaluated on an environmental medium basis. The current trend is to take an integrated approach so that the total body burden from multiple sources and routes of chemical exposures can be considered in a health risk assessment. This shift in the risk assessment paradigm is reflected in a report of US EPA's Science Advisory Board (EPA-SAB-IAQC-95-005):

> "Regulatory efforts frequently separate contaminants by whether they are found in these different media, whether they are ingested, inhaled or absorbed through the skin, and by the environment in which the exposure occurs (e.g., work place, home). Contaminants in multi-environmental media and multi-human exposure pathways, such as pesticides, automotive fuels, polycyclic aromatic hydrocarbons, and heavy metals, are not monitored in a systematic way such that the media and pathway of exposure can be assessed or the actual exposures quantified. The focus of environmental monitoring needs to shift to an integrated approach. Exposures need to be considered across sources, media, pathways, and environments, then tied to population activity patterns."

The Cal/EPA's Risk Assessment Advisory Committee, in issuing its 1996 report: *A Review of the California Environmental Protection Agency's Risk Assessment Practices, Policies, and Guidelines*, expressed a similar view. In discussing the roles and responsibilities of Cal/EPA subdivisions in coordinating and integrating human exposure assessments, the Committee underscored that basic principle:

> "To understand environmental risks to human health, we need to know the distribution of aggregate exposures to all toxic substances from all sources by all routes (exposure pathways)."

Sharing this view of US EPA's Science Advisory Board and the Risk Assessment Advisory Committee, OEHHA is taking an integrated approach to identify chemical contaminants that have the potential to be present at school sites. The integrated approach is undertaken in the context that a school site can be a new development, a proposed expansion of an existing school, or simply an existing school. In that vein, all potential sources (e.g., past industrial and agricultural contamination of the site, present pesticide uses indoors and outdoors, emissions and discharges from neighboring facilities) and environmental media (e.g., outdoor air, indoor air, soil, groundwater) should be considered in identifying chemical contaminants at school sites.

In the absence of school-site specific data, OEHHA established the following criteria to guide the compilation of potential contaminants:

- Chemicals which have been identified by federal and state agencies as concerns and which are likely to be found at school sites should be included.
- Contaminants that were or are known to be present in the pertinent environmental media of California should be considered.

In reviewing state activities, we have identified a number of special studies and programs as relevant. For example, DHS conducted a study and issued a report regarding lead hazards in California's public elementary schools and child-care facilities. (California Department of Health Services, 1998). DHS concluded that lead levels in soil may be elevated if the soil is close to pre-1940's painted exterior walls, and that lead may be present in drinking water in up to one in five of California public elementary schools and child-care facilities. While this and other studies are useful, we found certain over-arching programs of DTSC, DHS, ARB, and DPR to be particularly applicable. DTSC, through its review of Preliminary Endangerment Assessments of school sites, is collecting information from these consultant reports, which contain soil sampling and analysis data on chemical contaminants. DHS and ARB are embarking on a study under the Portable Classroom Program, which will identify a range of chemical contaminants in the classroom environment. DPR, pursuant to AB 2260, will collect data on pesticide uses in schools. Sections 2.2, 2.3, and 2.4 provide a description of these programs. In addition, OEHHA, pursuant to the Children's Health Protection Act, has conducted a focused evaluation of 36 of the 90 Toxic Air Contaminants (TACs) that are known to be present in California's ambient air for potential for differential impacts on infants and children. A description of this OEHHA effort is given in Section 2.5.

With respect to federal activities, OEHHA has identified two US EPA studies that are especially relevant. The chemicals targeted in the National Human Exposure Assessment Survey (NHEXAS) and Total Exposure Assessment Methodology (TEAM) population-based human monitoring studies are pertinent in the context of our integrated approach.

US EPA studied the concentration of prevalent or persistent metals, pesticides, and toxic organic compounds that were found in multiple environmental media and/or human tissues. Background information regarding NHEXAS and TEAM is given in Sections 2.6 and 2.7.

In considering the above findings, OEHHA has included the following chemicals as candidate contaminants that are likely to be found at school sites:

- Ninety-four soil contaminants that have been reported in school site Preliminary Endangerment Assessments (PEAs) and reviewed by Department of Toxic Substances Control (DTSC). These soil contaminants are found at sites being evaluated for school construction and are likely to be found at existing school sites.
- Ninety Toxic Air Contaminants known to be present in ambient air of California. They serve as a proxy to represent potential contaminants that may be found in the outdoor air around schools, and consequently in indoor air of schools.
- Sixty potential classroom contaminants targeted for monitoring by Department of Health Services (DHS) and Air Resources Board (ARB), as part of their Portable Classroom Program.
- Forty-seven toxic chemicals targeted by US EPA in its NHEXAS and twenty-six in its TEAM studies because of their prevalence or persistence in the environment and likely body burden. These chemicals are deemed likely to be found in various environmental compartments at schools, as well as their surrounding communities.

In total, this compilation consists of approximately 200 candidate chemicals. In addition, we have added as Table 3.2.1, the chemicals on the Voluntary Children's Chemical Evaluation Program (VCCEP). We have also attached as Appendix A the DHS compilation of VOCs that may be emitted from building materials and products and their potential sources, and as Appendix B a report from a search on the California Department of Pesticide Regulation (DPR) website on chemicals that have been registered for use in schools. There exists a fair amount of overlap among the chemicals reported or targeted by the above federal and state agencies. The chemicals that are part of the VCCEP and the two Appendices may not all be listed on our compilation of approximately 200 chemicals, but they still may be considered available for further study if information suggests that they be of greater concern to OEHHA. These candidate chemicals must be viewed as an internal listing, which does not possess a regulatory status. It will require updates periodically. We have attempted to include all reasonable candidates on the initial list, but ongoing literature reviews and results from studies of our sister agencies may turn up additional candidate chemicals. For example, as we receive information from the Pesticide Use Notification Program in the next fiscal year, we may identify additional pesticides as contaminants on the basis of pesticide uses at schools. Conversely, we may remove certain chemicals targeted by the Portable Classroom Program when the study results

indicate they are not detected in the classroom setting. The working list will serve to direct data-gathering and in-depth literature review, and to facilitate the prioritization of chemicals for evaluation for health based criteria development.

2.1 Compilation of Chemicals Potentially Found at School Sites

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
Acetaldehyde	75-07-0		X	X		
Acetone	67-64-1	X		X		
Acrolein	107-02-8		X	X		
Acrylamide	79-06-1		X			
Acrylonitrile	107-13-1		X			
Alachlor	15972-60-8				X	
Aldrin	309-00-2	X				
Aluminum	7429-90-5			X		
Aniline	62-53-3		X			
Antimony	7440-36-0	X			X	
Arsenic	7440-38-2	X	X		X	
Asbestos	1332-21-4		X			
Barium	7440-39-3	X			X	
Benzaldehyde	100-52-7			X		
Benzene	71-43-2	X	X	X	X	X
Benzidene	92-87-5		X			
Benzylchloride	98-88-4		X			
Beryllium compounds	7440-41-7	X	X		X	
Boron	7440-42-8				X	
Bromine	7726-95-6			X		
Bromodichloromethane	75-27-4					X
Bromoform	75-25-2					X
1,3-Butadiene	106-99-0		X	X		
Butyraldehyde	123-72-8			X		
Cadmium	7440-43-9	X	X	X	X	
Calcium	7440-70-2			X		
Carbon Disulfide	75-15-0		X			
Carbon tetrachloride	56-23-5		X	X		X
Cesium	7440-46-2			X		
Chlordane	57-74-9	X	X	X	X	
Chlorine	7782-50-5		X	X		
Chlorobenzene	108-90-7		X			X
Chloroform	67-66-3	X	X	X		X

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
(Trichloromethane)						
Chloroprene	126-99-8		X			
Chlorpyrifos	2921-88-2			X	X	
Chromium compounds	7440-47-3	X			X	
Chromium VI	18540-29-9	X	X	X		
Cobalt	7440-48-4	X		May add		
Copper	7440-50-8	X		X		
Cresols/Cresylic acid	1319-77-3		X			
Crotonaldehyde	4170-30-3			X		
2,4-D	94-75-7				X	
2,4-DB	94-82-6	X				
DDD	72-54-8	X		X	X	
DDE	72-55-9	X	X	X	X	
DDT	50-29-3	X		X	X	
n-Decane	124-18-5					X
Diazinon	333-41-5				X	
Dibromochloromethane	124-48-1					X
1,2-Dibromo-3-chloropropane (DBCP)	96-12-8		X			
1,2-Dichlorobenzene	95-50-1	X				X
1,4-Dichlorobenzene	106-46-7	X	X		X	X
Dichlorodifluoromethane	75-71-8	X				
1,2-Dichloroethane	107-06-2					X
Dichlorvos	62-73-7		X		X	
Dieldrin	60-57-1	X		X	X	
Diesel exhaust PM			X			
2,5-Dimethylbenzaldehyde	5779-94-2			X		
Dimethyl sulfate	77-78-1		X			
1,4-Dioxane	123-91-1		X			X
Dioxins and dibenzofurans (TCDD)	1746-01-6	X	X			
Diquat	85-00-7	X				
Diuron	330-54-1	X				
n-Dodecane	112-40-3					X
Endosulfan	115-29-7	X				
Endosulfan I	959-98-8	X			X	

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
Endosulfan II	33213-65-9	X				
Endosulfan sulfate	1031-07-8	X				
Endrin	72-20-8	X				
Endrin aldehyde	7421-93-4	X				
Epichlorohydrin	106-89-8		X			
Ethyl acrylate	140-88-5		X			
Ethyl benzene	100-41-4	X		X		X
Ethyl chloride (Chloroethane)	75-00-3		X			
Ethylene dibromide (1,2-dibromoethane)	106-93-4		X			X
Ethylene dichloride (1,2-Dichloroethane)	107-06-2		X			
Ethylene and Diethylene Glycol Ethers and Acetates			X			
Ethylene Glycol Monobutyl Ether (EGBE)	111-76-2					
Ethylene Glycol Monoethyl Ether (2-Ethoxyethanol)	110-80-5					
Ethylene Glycol Monoethyl Ether Acetate (2-Ethoxyethyl Acetate)	111-15-9					
Ethylene Glycol Monomethyl Ether (2-Methoxyethanol)	109-86-4					
Ethylene oxide	75-21-8		X			
Ethylidene dichloride (1,1-Dichloroethane)	75-34-3		X			
Formaldehyde	50-00-0		X	X		
Heptachlor	76-44-8	X	X		X	
Heptachlor Epoxide	1024-57-3	X				
Hexachlorobenzene	118-74-1		X			
Hexachloro-cyclopentadiene	77-47-4		X			
Alpha-Hexachlorocyclohexane	319-84-6	X	X			

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
Beta-Hexachlorocyclohexane	319-85-7	X	X			
Delta-Hexachlorocyclohexane	319-86-8	X	X			
Gamma-Hexachlorocyclohexane (Lindane)	58-89-9		X	X		
Hexachloroethane	67-72-1		X			
Hexaldehyde	66-25-1			X		
N-Hexane	110-54-3		X			
2-Hexanone	30637-87-7					
HMX	2691-41-0	X				
Hydrazine	302-01-2		X			
Hydrochloric acid	7647-01-0		X			
Hydrofluoric acid	7664-39-3		X			
Hydrogen Selenide	7783-07-5		X			
Hydrogen sulfide	7783-06-4	X				
Isovaleraldehyde	590-86-3			X		
Lead	7439-92-1	X	X	X	X	
Magnesium	7439-95-4			May Add		
Malathion	121-75-5			X	X	
Maleic anhydride	108-31-6		X			
Manganese	7439-96-5		X	X	X	
MCPP	93-65-2				X	
MCPA	94-74-6				X	
Mercury and compounds	7439-97-6	X	X		X	
Metolachlor	51218-45-2				X	
Methane	74-82-8	X				
Methanol	67-56-1		X			
Methoxychlor	72-43-5	X				
Methyl bromide (Bromomethane)	74-83-9		X			
Methyl chloride (Chloromethane)	74-87-3	X				

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
Methylene chloride (Dichloromethane)	75-09-2	X	X			
Methyl ethyl ketone (2-Butanone)	78-93-3	X	X			
Methyl Isocyanate	624-83-9		X			
Methyl methacrylate	80-62-6		X			
Methylnaphthalene	1321-94-4					
¾-Methylphenol (Cresol)	1314-77-3	X				
4,4-Methylenedianiline	101-77-9		X			
Molybdenum	7439-98-7	X				
Nickel		X				
Nickel (metallic)	7440-02-0	X	X	X	X	
Nitrate	14797-55-8	X				
Nitrobenzene	98-95-3		X			
2-Nitropropane	79-46-9		X			
N-Nitrosodimethylamine	62-75-9		X			
4-Nitrotoluene	99-99-0	X				
n-Octane	111-69-5					X
PAHs - see individual PAHs for specific information						
Acenaphthene	83-32-9			May Add		
Acenaphthylene	208-96-8				X	
Anthracene	120-12-7	X		X	X	
Benz[a]anthracene	56-55-3	X		X		
Benzo[b]fluoranthene	205-99-2	X		X	X	
Benzo[ghi]perylene	191-24-2	X		X	X	
Benzo[k]fluoranthene	207-08-9	X		X	X	
Benzo[a]pyrene	50-32-8	X	X	X	X	
Benzo[e]pyrene	192-97-2				X	
Chrysene	218-01-9	X		X	X	
Dibenzo[ah]pyrene	189-64-0	X				
Fluoranthene	206-44-0	X		X	X	
Fluorene	86-73-7			X		
Indeno(1,2,3-c,d)pyrene	193-39-5	X			X	
Naphthalene	91-20-3	X	X	X		

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
Phenanthrene	85-01-8	X		May Add	X	
Pyrene	129-00-0	X		May Add	X	
Paraquat	1910-42-5	X				
PCBs	1336-36-3	X	X			
PCP (Pentachlorophenol)	87-86-5	X	X			
Phenol	108-95-2		X			
Phosphine	7803-51-2		X			
Phosphorous	7723-14-0			X		
Phthalates						
Dibutyl phthalate	84-74-2	X				
Diethylhexyl phthalate (DEHP)	117-81-7		X			
Diethyl phthalate	84-66-2	X				
Dimethyl phthalate	131-11-3					
Phthalic anhydride	85-44-9		X			
a-Pinene	7785-26-4					X
Potassium	7440-09-7			X		
Propionaldehyde	123-38-6			X		
Propylene dichloride (1,2-Dichloropropane)	78-87-5		X			
Propylene oxide	75-56-9		X			
Pyrethroids						
Permethrin	52645-53-1			X	X	
Selenium	7782-49-2	X	X	May Add		
Silicon	7440-21-3			X		
Silver	7440-22-4	X				
Sodium	7440-23-5			May Add		
Strontium	7440-24-6			X		
Styrene	100-42-5		X		X	X
Sulfur	7704-34-9			X		
Tetrachloroethane	25322-20-7					
1,1,1,2-Tetrachloroethane	630-20-6					X
1,1,2,2-Tetrachloroethane	79-34-5		X			X
Tetrachloroethylene (PERC/PCE)	127-18-4	X	X	X	X	X
Thallium	7440-28-0	X				

Chemical	CASRN	DTSC Identified Soil Contaminant	OEHHA Targeted TAC	DHS/ARB Portable Classroom Study	US EPA NHEXAS	US EPA TEAM
Tin	7440-31-5	X				
Titanium	7440-32-6			X		
o-, m-, p- Tolualdehydes	1334-78-7			X		
Toluene	108-88-3	X	X	X	X	
Toluene-2,4-diisocyanate and other diisocyanates	584-84-9		X			
Toxaphene	8001-35-2	X				
TPH-d (Diesel range hydrocarbons)		X				
TPH-g (Gasoline range hydrocarbons)		X				
TPH-mo (Motor oil range hydrocarbons)		X				
Triazine Herbicides						
Atrazine	1912-24-9				X	
Simazine	122-34-9				X	
1,2,4-Trichlorobenzene	120-82-1	X				
1,1,1-trichloroethane (Methyl chloroform)	71-55-6	X	X	X	X	X
1,1,2-Trichloroethane	79-00-5		X			
Trichloroethylene (TCE)	79-01-6	X	X		X	X
2,4,6-Trichlorophenol	88-06-2		X			
1,2,4-Trimethylbenzene	95-63-6	X				
1,3,5-Trimethylbenzene	108-67-8	X				
2,4,6-Trinitrotoluene	118-96-7	X				
n-Undecane	1120-21-4					X
Valeraldehyde	110-62-3			X		
Vanadium	7440-62-2	X		X		
Vinyl chloride	75-01-4		X			
Vinylidene chloride	75-35-4		X			
Xylenes	1330-20-7	X	X	X	X	X
Zinc	7440-66-6	X		X		

2.2 School Site Acquisition Program

DTSC is working closely with the California Department of Education (CDE) in implementing the School Site Acquisition Program. Education Code sections 17071.13, 17072.13, 17210, 17210.1, 17213.1-3, and 17268, which became effective January 1, 2000, established requirements for assessments and approvals that school districts must follow before receiving final site approval from the CDE and funds under the School Facilities Construction Program. A summary of the pertinent requirements is as follows:

- Current and historic uses on and near the proposed school site shall be investigated by a qualified consultant. The investigation, which constitutes a Phase I Environmental Site Assessment, shall be conducted according to the American Society of Testing and Materials standards (ASTM E-1527-2000).
- DTSC shall require a Preliminary Endangerment Assessment (PEA), as appropriate, upon the review of the Phase I document. The PEA includes the sampling of soils and a risk assessment to determine the extent of soil contamination and if such contaminants pose a significant health risk.

OEHHA has contracted with DTSC to capture in a database those chemical contaminants identified in school-site PEAs. This effort has helped identify chemical contaminants that may be "commonly found" in the soil medium of potential school sites. DTSC has reviewed 473 school sites and they have identified contaminants of concern from 109 sites. The Section 2.1 table shows those soil contaminants identified by DTSC.

As more PEAs are reviewed in the school-site acquisition process, DTSC may identify additional soil contaminants. OEHHA will keep abreast of this DTSC activity and update the profile of soil contaminants as appropriate in the next fiscal year.

2.3 California Portable Classroom Program

ARB and DHS are co-sponsoring a study on the environmental health conditions in portable (relocatable) classrooms. The study results will be used by the ARB and DHS in preparing a final report for the California Legislature, due June 2002, pursuant to AB 2872 (Shelley) and HSC Section 39619.6. The study will include two portable classrooms and one traditional classroom per school at about 70 schools, totaling 210 classrooms. As part of this study, dust and VOCs will be sampled and selected chemical constituents will be analyzed. Selection of these chemicals is based on previous exposure observation, known health effects, and some understanding about sources. The dust samples will be analyzed for polynuclear aromatic hydrocarbons, metals, and selected pesticides. Results from the ARB/DHS study would be available in the next fiscal year to assist OEHHA in more accurately delineating the extent and nature of those indoor contaminants that would be "commonly found" at school sites.

While the current focus is on those chemicals targeted by the Portable Classroom Study, we are also aware of two other sources of information that provide further insights on what other indoor air contaminants may be found in the school environment.

They are:

- DHS' list of VOCs that may be emitted from building materials and products.
- US EPA's Building Assessment, Survey and Evaluation Study (BASE).

DHS' list is included as Appendix A. US EPA has completed the evaluation of the indoor air quality of over 100 commercial and public buildings in its BASE study. Sampling and analytical results are expected to be available in the near future.

2.4 Pesticide Use Notification and Reporting Program

AB 2260 (the Healthy Schools Act of 2000, Education Code sections 17608-17613 and Food and Agricultural Code sections 13180-13188) codified the DPR's School Integrated Pest Management Program, and added new requirements regarding pesticide use notification and reporting. These new requirements will facilitate OEHHA's efforts in identifying pesticides that are found at school sites.

Beginning the 2001-2002 school year, each school district is required to notify all school staff and parents or guardians of students regarding pesticide use. Specifically, the school district is to identify pesticide products to be applied by district staff or an outside contractor in the upcoming year. The school district may voluntarily provide a copy of the notification to DPR. However, the law exempts the following pesticide products from the notification process:

- Pesticide product deployed in the form of a self-contained bait or trap.
- Gel or paste deployed as crack and crevice treatment.
- Any pesticide exempted from regulation by US EPA pursuant to FIFRA (7 U.S.C. Sec. 25(b)) (identified by US EPA as little or no risk products).
- Anti-microbial pesticides including sanitizers and disinfectants.

On or after January 1, 2002, licensed and certified pest control operators will also be required to report pesticide use and applications at school sites annually to DPR. In preparation, DPR is developing a database for managing data to be provided by pest control operators, as well as by school districts. Some data may be available during the next fiscal year to help OEHHA identify pesticides that are likely to be found at school sites.

In the interim, a searchable database on DPR's Web Page (www.cdpr.ca.gov) provides some insights on what pesticides may be included in school districts' notifications of proposed pesticide uses or in pest control operators' reports. This searchable database permits the query

of selected use parameters to produce a listing of pesticides according to those allowable uses. Appendix B contains results of two sample searches, with schools (indoor) (school yards); and recreational areas, tennis courts, parks, etc. (which are frequently adjacent to school sites and used by school children as if they are school grounds), respectively, as search parameters.

2.5 Toxic Air Contaminant Program for Children

The California Legislature passed the Children's Environmental Health Protection Act (SB 25, Escutia; chaptered 1999), which requires the California Environmental Protection Agency to consider children in setting Ambient Air Quality Standards and developing criteria for Toxic Air Contaminants (TACs). As an initial task, the law (Health and Safety Code Sections 39669.5(a)) requires OEHHA to evaluate available information on the TACs and develop a list of up to five TACs that "may cause infants and children to be especially susceptible to illness" by July 1, 2001. In prioritization of TACs for evaluation, OEHHA reviewed monitoring and emission data, as well as health effect information. As a result of that review, OEHHA identified 90 TACs that were either emitted or detected in California's ambient air for further prioritization. These TACs, which are referenced in Tables 1A and 1B of OEHHA's October 2001 report entitled Prioritization of Toxic Air Contaminants Under the Children's Environmental Health Protection Act, have been included as candidate contaminants that are likely to be found in outdoor air of schools.

2.6 US EPA TEAM Study

United States Environmental Protection Agency recognizes the importance of a cross-media approach to measuring the body burden of toxic chemicals in health risk assessment. As such, US EPA initiated the Total Exposure Assessment Methodology (TEAM) study to apply this approach in evaluating the exposures and body burdens of urban populations including children in several United States (U.S.) cities. US EPA focused on a number of volatile organic chemicals (see Section 2.1) and measured their levels in breath, personal air, ambient air, and drinking water. Field studies were conducted in New Jersey, North Dakota, North Carolina, and California. The California study included northern communities of Antioch and Pittsburg, and southern communities of El Segundo, Manhattan Beach, Redondo Beach, Torrance, Hermosa Beach, Carson, Lomita and West Carson.

2.7 US EPA NHEXAS Study

To further address some of the limitations of a single-chemical, and single media approach in risk assessment, US EPA initiated the National Human Exposure Assessment Survey (NHEXAS). NHEXAS focuses on the comprehensive exposure of people, including children, to multiple environmental pollutants from multiple routes and sources of exposure. In a series of

pilot studies, researchers measured chemical concentrations in 1) breathing zone air, 2) food, 3) drinking water, 4) other beverages; and 5) soil and dust around the homes. Biological measurements of residents were also made of chemicals using blood, urine, hair, and in some cases, fingernail. The list of chemicals, which NHEXAS monitored, is indicated in Section 2.1, as well as those pesticides that have been included in the Minnesota Children's Pesticide Exposure Study (MNCPES) (Quackenboss et al., 2000). MNCPES is a Phase III special study conducted as part of the NHEXAS.

3. Identification of Candidate Chemicals Based on Potential Critical Health Effects

This chapter describes OEHHA's efforts in meeting the second part of HSC 901(g) concerning the identification of chemicals of the greatest concern based on a) child-specific exposures and b) child-specific physiological sensitivities.

3.1 Child-Specific Exposures

The identification of chemicals of concern based on child-specific exposure is addressed by scientific deduction from available data. Table 3.1.1 shows that the biology and behavior of children result in exposure differences compared with adults in the same environment. Because children eat more food, drink more fluids, and breathe more air in proportion to their body weight than adults, they will be exposed to a greater amount of a chemical that contaminates these media than an adult in the same environment. Consequently, contaminants, which may be found at a school site, would be of concern because exposure of school children to any toxic chemical will result in their higher intakes per unit body weight.

Table 3.1.1 Differences between children and adults

	Children 0-12 yrs	**Adults 12-69 yrs**	**Reference**
Surface area:body mass ratio	0.067 (infant) 0.047 (young child) 0.033 (older child)	0.025 (adult)	Snodgrass, WR, ILSE Press, 1992
Respiratory minute ventilation rate (mL/kg/m^2 lung surface area/minute)	(infant) 133	(adult) 2	Silvaggio, T and Mattison, DR, 1993
Drinking water (tap) Mean intake (mL/kg/day)	(9 years)	(30 and 70 years)	Cal/EPA, 2000
Produce consumption (g/kg/day)	(0-9 years)	(0-70 yrs)	Cal/EPA, 2000
Exposed Produce	4.16 (av)-15.7 (high)	3.56 (av) – 12.1 (high)	
Leafy	2.92 (av)-10.9(high)	2.90(av)-10.6(high)	

Fruit consumption (g/kg/day)			US EPA, 1997
Citrus fruits	2.6	0.9	
Other fruits (incl apples)	5.8	1.3	
Apples	3.0	0.4	
Soil ingestion (mg/day)			US EPA, 1997
Pica child	10,000		Cal/EPA, 2000
child	200	100	
GI absorption of lead	(2-6 yrs) 30-40%	(adult) 7-15%	US EPA, 1997

3.2 Child-Specific Physiological Sensitivities

The need for scientific research on child-specific physiological sensitivities has only received attention in recent years, and only very recent information exists on the potential toxicity of environmental contaminants to school-age children. US EPA has announced the Voluntary Children's Chemical Evaluation Program (VCCEP) to ask companies to provide toxicity data for chemicals in which it is absent. Using the Chemical Right-to-Know mechanism as a vehicle to implement VCCEP, US EPA is asking companies which manufacture or import the selected 23 chemicals to collect and develop health effects and exposure data. The VCCEP objective is to ensure that there are adequate publicly available data to assess the special impact that industrial chemicals may have on children. The 23 chemicals selected by US EPA for this "data call in" are given in Table 3.2.1. In selecting the chemicals, US EPA attempted to focus on those chemicals not covered by other US EPA programs for which private companies may have data and which have been found to be present as contaminants in human tissues or fluids; food and water children may eat and drink; and air children may breathe, including residential or school air. The databases which help US EPA target chemicals include the National Health and Nutrition Examination Survey III (NHANES III), National Human Adipose Tissue Survey (NHATS), National Human Exposure Assessment Survey (NHEXAS), and Total Exposure Assessment Methodology (TEAM). OEHHA will keep abreast of the VCCEP and make use of its findings, as appropriate.

Table 3.2.1 VCCEP Chemicals

CAS No.	Chemical	CAS No.	Chemical
67-64-1	Acetone	108-88-3	Toluene
71-43-2	Benzene	108-90-7	Chlorobenzene
75-35-4	Vinylidenechloride	112-40-3	n-Dodecane
78-93-3	Methyl ethyl ketone	123-91-1	p-Dioxane
79-01-6	Trichloroethylene	124-18-5	Decane
80-56-8	Alpha-Pinene	127-18-4	Tetrachloroethylene
95-47-5	o-Xylene	541-73-1	m-Dichlorobenzene
100-41-4	Ethylbenzene	1120-21-4	Undecane
106-46-7	p-Dichlorobenzene	1163-19-5	Decabromodiphenyl ether
106-93-4	Ethylene dibromide	32534-81-9	Pentabromodiphenyl ether
107-06-2	Ethylene dichloride	32536-52-0	Octabromodiphenyl ether
108-38-5	m-Xylene		

Given the lack of data regarding potential health effects from most contaminants to school-age children, it is necessary to apply the following working definition to address the part of HSC 901(g) that pertains to the identification of chemicals of concern based on child-specific physiological sensitivities.

A chemical is considered to meet part b of the criterion if:

- it was listed as a developmental or reproductive toxicant under the Safe Drinking Water and Toxics Enforcement Act (Proposition 65); or
- a literature scan showed it to be associated with an adverse effect in nervous, respiratory, reproductive, endocrine, or immune organ systems.

These organ systems are targeted because they are still in critical development in school-age children and are, therefore, presumed to be more sensitive to "insults" by toxicants. A number of studies cited in Sections 3.1 and 3.2 support this assertion. The relative toxicity of lead illustrates the point and provides data to illustrate the rationale for targeting organ systems that are still in critical development.

Lead encephalopathy has been observed in children with blood lead levels of 80 ug/dL, compared to 100 ug/dL for adults. Children with 10 ug/dL of blood lead were shown to have IQ deficits, while an association between hearing loss and blood lead levels of 20 ug/dL was found in teenagers. However, children are not more sensitive with respect to lead's effects on the hematopoietic system.

Applying the above reasoning as the basis, we have developed a second compilation of chemicals, which is presented in Section 3.5. Similar to the Section 2.1 candidate chemicals, this compilation will be updated as more information becomes available. By and large, the candidate chemicals are identified on the basis that they are a developmental/reproductive toxicant listed under Proposition 65 and/or they have been reported in scientific journal articles as having a potential neurologic, respiratory, reproductive, endocrine, or immunologic effect. Chemicals are placed on the Proposition 65 list because an authoritative body has concluded that scientific data demonstrate that they have caused prenatal developmental effects or toxicity to male and female reproduction. In order to cover the entire temporal spectrum of development, our literature searches focused on studies of the perinatal, childhood and adulthood periods. As mentioned, few childhood or immature animal studies were found, and so we depend heavily on studies in perinatal and adulthood periods to uncover non-Proposition 65 identified chemicals that may have child-specific physiological sensitive adverse health effects.

In focusing on these critical organ effects, we have targeted non-cancer endpoints (rather than the cancer endpoint). In a separate task, OEHHA is developing a cancer evaluation methodology for children pursuant to HSC 901(e). Because that methodology will not be available until 2004, prudence dictates that we focus on identifying and evaluating the non-

cancer effects of chemicals. We will evaluate the cancer endpoint when the children's cancer methodology is developed.

3.3 Developing Systems and Critical Windows for Toxicity

Developing organ systems, which are undergoing cell proliferation, migration, and differentiation, may be more sensitive to toxic effects compared to mature adult systems. Therefore, OEHHA has been investigating chemicals that affect developing organ systems. Organ systems that may still be developing in school-age children include the nervous system, immune system, respiratory system, male and female reproductive system and endocrine system.

Developmental toxicity is defined as the occurrence of adverse effects on the developing organism that result from exposure at any time from parental exposure prior to conception, and prenatal exposure, through the time of sexual maturation of the individual (Selevan, 2000). In the past, developmental toxicity has focused on birth defects that result from prenatal exposure, such as limb deformities in babies whose mothers took the drug thalidomide during the gestational period when limbs were forming. The fact that limb malformations occurred only if thalidomide was taken 23 to 38 days after conception demonstrated that this was the time interval when cell proliferation, differentiation, and maturation to form the various tissues of the limbs was occurring. These time intervals during prenatal development are known as *sensitive periods* or *critical windows.*

Recent evidence indicates that critical windows in developing organ systems are periods of vulnerability to toxic insults that may exist at various times throughout childhood (Kimmel, 1992). Information on lead and radiation has demonstrated that exposures during critical periods in the development and maturation of organ systems can have major irreversible adverse effects on adult function (Needleman, H.L., 1990; Miller, R.W 1995; Wadsworth, M.E., 1997). Furthermore, a critical effect may occur in a child's developing organ system at a dose that is lower than the dose that caused a critical effect in another organ system in an adult. Human case reports and case control epidemiological studies on pesticide-exposed childhood cancer patients suggest that the risks are of greater magnitude than those observed in studies of pesticide-exposed adults (Daniels, J.L, 1997; Zahm and Ward, 1998). Similarly, the critical effect in lead-exposed adults was peripheral neuropathy from occupational or accidental exposure, which occurred at a blood lead concentration of 40 ug/dL (Klaassen, 1996). However, a child's brain is undergoing more cell proliferation, differentiation, and maturation than an adult brain, and adverse effects on a child's IQ were seen when the blood lead concentration was as low as 10 ug/dL. No such effects have been recorded at similar blood lead concentrations in adults. Thus, the critical effect in children was a reduction in IQ, and the adult health guidance value would not protect children from this critical effect.

Adverse developmental effects may manifest themselves at any point during the lifespan of the individual. The major manifestations of developmental toxicity before birth are death (spontaneous abortion or stillbirth), structural abnormalities as in the case of thalidomide, altered growth, and functional deficiency. Exposure during critical periods of development and maturation of organ systems during childhood may produce asthma and respiratory insufficiency, cognitive and other neurological deficits, infertility, cancer, heart disease, immune disorders, and degenerative neurological/behavioral disorders that may not become apparent for years or even decades (Selevan et al., 2000).

A major challenge to implementing this legislation is an inadequate research base on toxicity resulting from exposure during the period from birth to sexual maturity (Kimmel, 1992). Concern about structural birth defects has led to research on exposures during the fetal period to agents suspected to cause birth defects, such as thalidomide. However, there is little research on exposures during the fetal, perinatal, and growth periods that lead to adverse effects that may be expressed some years after the exposure. The peripubertal/adolescent period is especially underrepresented in toxicity studies, despite the fact that visible anatomical changes rapidly occur as many organ systems undergo significant development during this time (Selevan et al., 2000).

3.4 Developing Organ Systems

In order to identify chemicals to which children may be more susceptible, OEHHA's initial task was to review the scientific literature for information on chemical toxicity to organ systems that are undergoing cell proliferation, migration, and differentiation during childhood. An overview of critical processes and periods of vulnerability compiled for OEHHA's programs on children's health follows.

Immune System

The immune system is one of at least three important integrating and regulatory systems in humans and other animals. The other two are the nervous and endocrine systems. The immune system is a complex set of cellular, chemical, and soluble protein components designed to protect the body against foreign substances, including infectious agents and tumor cells, while not responding to self-molecules. Foreign molecules (usually proteins or carbohydrates) that evoke specific immune responses are referred to as antigens. Immune cells are located throughout the body, either in organs such as the spleen or thymus, or as diffuse accumulations of white blood cells strategically placed to monitor the entry of foreign substances. Optimal function of the immune system requires that immune cells (B lymphocytes, T lymphocytes, macrophages, and other specialized cells) and cell products interact with each other in a sequential, regulated manner (Smith and Germolec, 1999).

The development of the immune system results from a series of carefully timed and coordinated events during embryonic, fetal, and early postnatal life. Chemicals that excite or suppress the immune system during these critical periods can have a lasting effect on the ability of the system to respond to environmental challenges. There is evidence that exposure of pregnant animals to immunotoxic chemicals at doses causing only transient effects in adults produce long lasting or permanent immune deficits in their offspring. Of increasing concern is the association of a number of environmental chemical contaminants (foreign chemicals or xenobiotics) with human autoimmune disease, including mercury, iodine, vinyl chloride, canavanine, organic solvents, silica, L-tryptophan, particulates, ultraviolet radiation, and ozone (Powell et al., 1999). The concordance of autoimmune disease among identical twins is often 25 – 40 percent, and there is evidence of epidemic clustering of some autoimmune diseases following exposure to foreign chemicals, indicating that autoimmune disease is secondary to both genetic and environmental factors. The predominance of autoimmune diseases in females strongly implies that gender plays a major role in the initiation or progression of these disorders, in fact sex steroid hormones modulate many of these disorders. A major difficulty in understanding these disorders is that a long latency between exposure and observation of symptoms may impede the establishment of cause and effect (Powell at el., 1999).

There are critical stages of development of B-lymphocytes in the bone marrow and T-lymphocytes in the liver and later the thymus during prenatal and postnatal development. During these critical stages, the future immune system cells are increasing in number and becoming specialized in function. Endogenous chemicals present in the microenvironment where the future T cells develop regulate these processes. Some xenobiotic chemicals, for example, chlordane, benzo(a)pyrene, diethylstilbestrol or dioxin, are capable of crossing the placenta, and impede development of the immune system in rodents, often resulting in cases of premature thymic involution and lifelong immunosuppression (Holladay and Smialowicz, 2000; Lai et al., 2000).

Some chemical and physical agents that have been identified to cause developmental immunotoxicity in rodents include (Holladay and Smialowicz, 2000):

- Polycyclic halogenated hydrocarbons: TCDD, PCB, PBB.
- Polycyclic aromatic hydrocarbons.
- Pesticides: hexachlorocyclohexane, chlordane, diazinon, DDT, carbofuran and hexachlorobenzene.
- Metals: methyl mercury, lead, and cadmium.
- Hormonal substances: estrogens and diethylstilbestrol, testosterone and cortisone.
- Therapeutic agents: acyclovir, busulfan.
- Mycotoxin: T-2 toxin.
- X-rays.

Central Nervous System

Many of the most important environmental hazards to humans involve risks to development of the central nervous system of children. Neurodevelopmental disabilities are a group of physical, cognitive, psychological, sensory, and speech impairments arising during development and up to 18 years of age. According to the U.S. Centers for Disease Control and Prevention (CDC), some 17 percent of all U.S. children less than 18 years of age have one or more developmental disabilities (Goldman and Kodoru, 2000).

The rates of some disorders, such as autism, an impairment of social interaction along with abnormal speech development and unusual behaviors, appear to be increasing (Goldman and Kodoru, 2000). Attention deficit hyperactivity disorder (ADHD) is a disability that affects between 3-7 percent of children, with a significant number of individuals continuing to be affected into adolescence and adulthood, making ADHD a lifelong disability. There are parallels between the features of ADHD and the behavior of monkeys exposed during development to polychlorinated biphenyls (PCBs) or to lead
(Rice, 2000).

The nervous system in infants and children is more sensitive to the effects of many neurotoxins than that in adults because of rapid cell proliferation, migration, and differentiation. These processes are unidirectional and occur at very specific times for different structures. Lead, methyl mercury, and PCBs are examples of neurotoxic chemicals to which children can be very sensitive (Chang et al., 1977; Needleman, 1979, 1990; Olney, 2000; Rice and Barone, 2000).

Critical stages in the development of the central nervous system occur during embryogenesis and development of the fetus, as well as postnatally through adolescence. Two key developmental processes in brain development are 1) increase in brain mass (Dobbing and Sands, 1979) through proliferation of neurons and support cells, and 2) reinforcement of selected neuronal pathways through synaptogenesis. Prenatal events include closure of the neural tube, proliferation of neurons and migration of cortical neurons. During infancy and early childhood, proliferation and migration continue along with synaptogenesis, myelination, and development of the blood-brain barrier. New neurons are produced from stem cells and progenitor cells that appear in the central nervous system very early in development (Jacobs et al., 2000). The brain increases in mass during the last month of gestation, and grows from 350 to about 800 grams during the first year of life (Boyd, 1962). Structural maturation of neural pathways, including an increase in the diameter and myelination of axons, continues through adolescence. During adolescence the rate of synaptic pruning peaks. Chemical exposures can have profound, often irreversible, effects on all of these neurologic developmental processes (Rodier, 1994, 1995; Paus et al., 1999; Golub, 2000, Horner and Gage, 2000).

Chemicals may affect multiple processes and multiple chemicals may affect the same process (see Table 3.4.1). The effect of a chemical is dependent on the cellular process that it affects and the structures that may be undergoing that process at the time of exposure. The selection of certain synaptic connections and the elimination of others is critical for brain development. Neurotransmitters, such as GABA (ã-aminobutyric acid) and acetylcholine, are essential to this

process. Chemicals that interfere with the function of neurotransmitters can interfere with the development of synaptic connections. Such chemicals can also trigger programmed cell death or apoptosis, which results in the elimination of some neurons (Olney et al., 2000). In humans, thalidomide exposure on days 20 to 24 of gestation is linked to a 30 percent incidence of autism. This period corresponds with the production of the first neurons forming the motor nuclei of the cranial nerves; there is evidence of injury to these nerves from thalidomide exposure (Rodier et al., 1997).

The nervous system of the fetus and child is more susceptible than that of the adult to the effects of lead and methyl mercury (Chang et al., 1977). Burbacher et al. (1990) showed consistent dose-related behavioral and neuropathological effects in developing humans, monkeys and rodents. Dose-related behavioral outcomes included cognitive deficits or delayed development at low doses; cognitive and sensory effects at moderate doses; and severe sensory (blindness), cognitive, and neuromuscular abnormalities at high doses. Neuropathological effects in rodents included cellular loss and decreased brain size, cortical and cerebellar defects, and reduced myelination.

Pesticides, such as organophosphates, not only inhibit acetylcholinesterase, but may also decrease brain DNA synthesis, reduce brain weight, and downregulate muscarinic receptors. Repeated low-level exposure *in utero* and during early postnatal life to organophosphates appears to cause neurological effects such as impairment on maze performance, locomotion, and balance in rodent neonates (Eskenazi et al., 1999).

Arsenic administered orally to Wistar rats decreases acetylcholinesterase activity in the hypothalamus, cerebellum and brain stem, and slows the ability to learn and unlearn tasks. Since the effects are more pronounced in younger rats, Nagaraja and Desiraju, (1994) concluded that the developing brain is more susceptible to the neurobehavioral effects of arsenic than the adult brain.

Fetal alcohol syndrome is a recognized consequence of maternal-fetal exposure during critical periods of brain development (Clarren and Smith, 1978). Ethanol affects migration, differentiation, synaptogenesis and myelination and causes massive apoptosis during the period of synaptogenesis and brain growth of the third trimester. Ethanol causes Fetal Alcohol Syndrome in the fetus but is relatively nontoxic or even neuroprotective in the adult brain (Rice and Barone, 2000; Olney et al., 2000).

Some psychoactive drugs can interfere with the proliferation of brain cells in the hippocampus of adult nervous systems (Jacobs et al., 2000). Since there is more cell proliferation in younger brains, they may be even more sensitive to the effects of stress and foreign chemicals.

Table 3.4.1 Chemicals associated with disruption of neurodevelopmental processes. Based on Rice and Barone, 2000 and Olney, 2000, Environ. Health Perspect.

Process	Chemicals that disrupt this process in animals or humans
Proliferation	Ionizing radiation, methylazoxymethanol (MAM), ethanol, methyl mercury, chlorpyrifos
Migration	MAM, x-ray radiation, methyl mercury, ethanol
Differentiation	Ethanol, nicotine, methyl mercury, lead
Synaptogenesis	X-ray radiation, ethanol, lead, triethyltin, parathion, polychlorinated biphenyls (PCBs)
Gliogenesis and Myelination	Alterations in thyroid hormone homeostasis, ethanol, lead
Apoptosis	Ethanol, lead, methyl mercury, barbiturates, glutamine, halothane, ketamine
Neurotrophic Signaling	Aluminum, ethanol, cholinesterase inhibitors, methyl mercury

Respiratory System and Lung Development

The lungs must be functional from the moment of birth, i.e. they must be able to absorb enough oxygen from the air and deliver it to the bloodstream to support the energy requirements of the developing child. Even though the respiratory system is functional at birth, it has only a fraction of the potential that it will develop during the early years of life. Chemical toxicity to the cells that line the alveoli can result in the development of lungs that have reduced alveolar volume and surface area. Childhood exposure to ozone, sulfur dioxide, particulate matter, and nitrogen dioxide is associated with decreased lung function, increased occurrence of respiratory illnesses, and exacerbation of asthma (Bates, 1995). Exposure to environmental tobacco smoke in the home is also associated with asthma induction as well as asthma exacerbation in children (NCI, 1999). These effects may be due to interactions between effects on the immune system and respiratory system in children.

Exacerbation of asthma is of particular concern to children. Children, especially young children, are impacted by asthma morbidity more than adults (Mannino et al., 1998, CDHS, 2000). In addition, children have smaller airways than adults. Since the resistance to airflow is inversely proportional to the fourth power of the radius, bronchoconstriction and increased mucin secretion characteristic of asthma greatly increase airflow resistance in a small child more than in an adult. Thus, breathing is very difficult in young children experiencing an asthma attack. The hospitalization rate for children 0 to 4 years is greater than all other ages.

The Endocrine System

The endocrine system helps guide development, growth, reproduction, behavior and other bodily functions of animals and humans (EPA, 1997). It is composed of endocrine glands that produce and secrete hormones directly into the bloodstream. Hormones act as chemical messengers, traveling through the blood to distant tissues and organs, where they can bind to specific cell sites called receptors. By binding to receptors, hormones trigger various responses in the tissues containing the receptors. Organs with endocrine functions include the pituitary, thyroid, parathyroid, adrenals, pineal body, pancreas and male and female gonads (testis and ovary).

Autocrine, paracrine (such as growth factors) and endocrine (such as steroid) signals coordinate the direction of differentiation during critical periods in development. The differentiation of organs thus involves a complex cascade of signals whose action is dependent on being released at precise times and within a specific dose range (Bigsby et al., 1999). Because the endocrine system works together to coordinate and control development of the organism, it is critical during the first few years of life and during puberty.

Environmental chemicals known as endocrine-disruptors have been implicated in the disruption of important endocrine functions during development, including the onset and normal progression of puberty in adolescents. An endocrine disrupter is defined as an exogenous agent that interferes with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis, reproduction, development, and/or behavior (US EPA, 1997). Of particular concern are the processes or tissues stimulated by estrogens, androgens, and thyroid hormones, as well as similar chemicals that are estrogen, androgen or thyroid inhibitors or antagonists (Bixby et al., 1999). Adverse effects in females include the disruption of normal sexual differentiation, ovarian function (that is follicular growth, ovulation, corpus luteum formation and maintenance), fertilization, implantation, and pregnancy and possibly endometriosis and breast cancer. Adverse effects in males include reduced androgen levels or interference with androgen's actions, such as reduced sperm production capability and reproductive tract abnormalities. Controversy exists about the allegation that human sperm production has decreased, but there are firm data indicating an increase in human testicular cancer in young men (US EPA, 1997).

Disruption of endocrine function can also have neurodevelopmental effects, including altered reproductive behaviors, body metabolism, sexual differentiation in brain morphology, and cognitive and psychomotor development. Sexual and brain development are under the influence of estrogenic and androgenic hormones, and chemicals that interfere with these hormones, during development can adversely affect neurodevelopment. Thyroid hormones also play an important role in the development of the nervous system, and chemical-induced alterations of thyroid function during development can produce developmental neurotoxicity (Porterfield and

Hendrick, 1993). Moderate to severe alterations in thyroid hormone concentrations during development result in motor dysfunction, cognitive deficits, and other neurologic abnormalities. In addition, recent research suggests that developmental hypothyroidism in rats can cause permanent ototoxicity (Goldey et al., 1995).

There is extensive data on endocrine-related effects of PCBs. A wide variety of endocrine systems are affected by PCBs, including the estrogen and androgen system (Golden et al., 1998), thyroid hormone system (Brouwer et al., 1998), retinoid system, corticosteroid system and several other endocrine pathways. Thyroid hormones are critical to brain development *in utero* and through at least the first two years of life. Certain polychlorinated biphenyls (PCBs) and dibenzo-p-dioxins (PCDD) have been shown to alter the thyroid function during critical periods of thyroid-hormone dependent brain development resulting in neurological impairment in animal models (Porterfield, 2000). Prenatal exposure of children to PCBs from contaminated fish has been associated with lower IQ scores and decreased short- and long-term memory, and ability to focus (Jacobson and Jacobson, 1996).

Persistent chlorinated pesticides in the environment have been shown to impact children by causing sex hormone disruption. Studies indicate that DDE levels in milk fat are inversely related to duration of lactation in women in North Carolina and Mexico, possibly due to estrogenic properties of DDE (Rogan, et al., 1987, Gladen and Rogan, 1995). Boys with high pre-natal DDE exposure were heavier than other boys, and girls with higher pre-natal PCB exposures were heavier than other girls (Gladen, Ragan and Rogan, 2000).

When methoxychlor, another persistent chlorinated pesticide, was administered subchronically prior to puberty, male rats have shown a delayed onset of puberty, produce smaller seminal vesicles and ventral prostates, lower epididymal and ejaculated sperm numbers, and reduced successful matings (Gray et al., 1989; Anderson et al., 1995.) Exposing weanling male rats to DDE or vinclozlin also delayed pubertal development (Anderson et al., 1995 and Kelce et al., 1995.) In female rats, methoxychlor decreased the age at vaginal opening, first estrus, and the onset of estrous cycles (Gray et al., 1988; 1989). Lindane acts like an anti-estrogen in female rats and delays vaginal opening and the appearance of regular estrous cycles in immature rats when they are treated at the onset of puberty (Cooper et al., 1989).

The evidence for adverse effects from exposure to endocrine-disrupting chemicals has prompted a number of scientific workshops such as the Workshop on Characterizing the Effects of Endocrine Disruptors on Human Health at Environmental Exposure Levels, May, 1998. The discussions at these workshops have highlighted the need for additional scientific research and a need for the US EPA to initiate a program to test environmental chemicals for endocrine disrupter effects.

Reproductive Toxicity

Humans exhibit a high rate of infertility, low fecundity, and other reproductive disorders when compared to many other species (Lemasters et al., 2000). Since development of the reproductive system is incomplete at birth, considerable differentiation occurs during puberty. Since reproductive function cannot be assessed until pregnancy is attempted, which may be many years after exposure, the degree to which environmental contaminants affect reproduction is not known. The gender-specific differences in reproductive organs and physiology may result in gender-specific responses to a given toxicant.

Female Reproduction

At birth a human female's ovary contains about 12 million follicles containing oocytes that have been arrested in meiosis, and which remain arrested throughout childhood, and resume meiosis after puberty. The number of follicles is continuously reduced, so that only about 400,000 are present at puberty (Peters and McNatty, 1980) after which ovulation begins. The same process occurs in rodents, except that sexual maturity is reached by 34 – 36 days after birth. Once the population of oocytes is depleted, fertility ends.

Puberty in females is marked by menarche (the onset of menses), thelarche (the onset of breast development) and adrenarche (the appearance of axial hair). The changes in the reproductive tract during adolescence are pervasive and profound. The uterus increases in size and becomes anteverted, the uterine cavity is established, and epithelial secretory activities are initiated. External genitalia increase in size and the vaginal epithelium thickens and undergoes changes in pH and glycogen content (Golub, MS, 2000).

Before puberty, leutinizing hormone (LH) is secreted in a basal fashion with irregular pulses. After sexual maturing, follicular growth, ovulation, and maintenance of pregnancy are controlled by the balanced secretion of LH and follicle stimulating hormone (FSH). LH triggers follicular rupture and oocyte release, stimulates progesterone production, and stimulates the follicular theca and stromal interstitial cells to produce androgens. FSH stimulates follicular growth and causes granulosa cells to transform androgens to estrogens.

Anisimov suggested that high rates of cell proliferation and differentiation could increase susceptibility to toxicant-induced reproductive tract cancers (Anisimov, 1982). Similarly, chemicals that mimic the effect of estrogen are postulated to significantly enhance risk for breast cancer during growth and adolescence (Ardies and Dees, 1998). Until puberty, the mammary glands of males and females are essentially the same. In the female, the increase in estrogen levels at puberty stimulates growth of the rudimentary mammary gland. Over-stimulation of mammary tissues by "xenoestrogens" might increase the risk of developing breast cancer.

Male Reproduction

Sertoli cell proliferation occurs from birth to six months, and this phenomenon establishes the final number of Sertoli cells throughout adult life. Thyroid hormone is thought to regulate Sertoli cell proliferation. The testis is the site of the highest ongoing mitotic cell division in the body. Spermatogonia are the only germ cells present in the prepubertal testis, although there are reports of some spermatocytes being present (Sun, and Gondos, 1984). Between birth and 10 years of age, spermatogonia undergo 3-6 mitotic divisions, depending on the species, to become spermatocytes. Consequently there is a 3-6 fold increase in both the number of germ cells and testicular volume (Muller, J and Skakkebaek, NE, 1992). Immature Sertoli cells are the most common somatic cell type seen in the prepubertal testis, and they increase during the prepubertal period (Muller, J. and Skakkebaek, NE, 1992).

Spermarche, the age at which spermatogenesis begins, occurs early in puberty. Spermatogenesis reduces the number of chromosomes to half during the last division, called meiosis, and the cell becomes a spermatid. The early spermatids are round cells that undergo many changes to become mobile, mature, and able to fertilize ova. Many chemicals, for example carbon disulfide, dibromochloropropane (DBCP) and lead, perturb or arrest this process (Schrag and Dixon, 1985; Pryor, JL, 2000). Maturation occurs in the epididymis, which excludes proteins such as immunoglobulins and highly polar chemicals, but does not prevent the access of many chemicals (Pryor, JL, 2000). One well-known example was the chlorinated hydrocarbon insecticide, chlordecone (kepone), which caused oligospermia and altered sperm motility (Cannon et al., 1978).

The first wave of spermatogenesis is stimulated by testosterone and follicle-stimulating hormone (FSH), but testosterone appears to be the primary regulator of spermatogenesis. Testosterone exerts a negative feedback on the other hormones involved, such as Gonadotrophin Releasing Hormone (GnRH) release from the hypothalamus, and is thus involved in the regulation of both lutenizing hormone (LH) and follicle stimulating hormone (FSH) secretion.

Germ cells throughout spermatogenesis and maturation in the epididymis are susceptible to toxic chemicals, and the toxicity is often chemical and/or stage specific. Spermatids that have completed cell division and differentiation are sensitive to agents that damage chromatin (Pryor, JL, 2000). Chemicals in seminal fluid (e.g., methadone, morphine, cyclophosphamide) have been reported to be absorbed by a female partner and affect the fertilized egg (Hales and Robaire, 1997; Soyka et al., 1978; Hales et al., 1986).

Cancer from Childhood Exposures

Cancer is a multistage process and the occurrence of the first stages in childhood increases the chance that the entire process will be completed, and a cancer produced, within an individual's lifetime. Consequently, exposure to a carcinogen early in life may result in a greater lifetime risk of cancer, whether it is manifested during childhood or during adulthood. Animal models suggest that early-life exposures to carcinogens at critical periods in development can also cause adult-onset cancer at lower exposure levels than the levels required to induce cancer following adult exposure. For example, there is evidence that a brief exposure to vinyl chloride early in life can lead to a higher total lifetime incidence of tumors than a longer exposure later in life (Cogliano et al., 1996).
Differences in the metabolism of chemicals, differing rates of DNA repair, and the rapid growth and development of tissues that takes place during childhood make developing organisms especially vulnerable to carcinogenic agents (Anderson et al., 2000).

Childhood Cancer

Epidemiological data suggest that cancers which occur during childhood differ from adult cancers in several respects: they have different common sites of origin, cell type, prognosis, and responsiveness to therapy (Campleman et al., 1999). The five most common cancers in California children and adolescents, age 0-19, are leukemia, central nervous system and brain or spinal neoplasms, lymphomas and related neoplasms, germ cell and gonadal neoplasms, and soft tissue sarcomas. Cancer is more common in 0-4 year olds (22 and 18 cases per 100,000 males and females, respectively) and 15-19 year olds (20 and 19 cases per 100,000 males and females, respectively) than in the childhood ages in between (Campleman et al., 1999).

In California approximately 1,400 children and adolescents are diagnosed with cancer each year. Childhood and adolescent cases represent just over 1 percent of all invasive primary cancers among Californians, compared to the 70 percent of cases diagnosed in persons over age 60. Since 1988, the incidence rate of childhood cancer in California has not changed substantially. However, the National Cancer Institute (NCI) estimates that from 1973-1994, the incidence rate for all childhood cancers combined increased by 10.5% among children 0-14 years of age (Campleman et al., 1999).

The following table[a] shows a disturbing pattern for certain types of cancer in children:

Childhood Disease	Incidence Rate	References
Leukemia	Increasing	Gurney, J.G., Davis, S, Severson RK, et al. 1996
Wilm's Tumor	Increasing	Gurney, J.G., Davis, S, Severson RK, et al. 1996
Testicular	Increasing	Giwercman A, Carlsen E, Keiding N, et al. 1993
Brain	Increasing	Gurney, J.G., Davis, S, Severson RK, et al. 1996

[a] Schneider and Freeman, 2001

Exposures to carcinogens during fetal development and in early childhood have been suggested as possible causal factors responsible for some of the increases in leukemia, lymphoma, brain and testicular cancers (Reis et al., 1999). The rate of leukemia increased steadily from 1972 – 1990 but since 1990, the rate has decreased in boys but has continued to increase in girls. Brain cancer has shown a 39% aggregate increase in both boys and girls. Wilm's tumor has increased steadily by about 0.5% peryear. Testicular cancer has increased 68% since the early 1970s in young men between the ages of 15 and 30 years. This pattern is seen in both the United States and Europe (Schneider and Freeman, 2001).

During childhood there may be increased period of susceptibility in tissues that are undergoing rapid growth and development. Testicular cancer, for example, has been increasing in the 15 to 19 year age group and is thought to be associated with earlier onset of puberty, which involves cellular division and maturation under the stimulus of hormone production, and earlier onset of regular sexual activity (Oliver, 1996).

Numerous studies have demonstrated that exposure of pregnant animals to nitrosourea compounds results in increases in the incidences of brain and other central nervous system tumors among offspring. Nitrosourea compounds are direct acting alkylating agents (e.g., mutagens). Alkylnitrosoureas are the most potent in causing brain tumors following *in utero* exposure in rodents (Maekawa and Mitsumori, 1990). Alkylnitrosourea compounds also seem to be more carcinogenic to the central nervous system following exposure to the fetus compared to exposure of adults. For example, *in utero* exposures to ethylnitrosourea were observed to be more than 60 times more potent in causing brain tumors than exposures during adulthood (Ivankovic, 1979). It is not known why the developing animal is more sensitive to the carcinogenic effects of alkylnitrosourea compounds on the central nervous system, but it likely is due to genotoxicity of the nitroso compounds coupled with the rapid cell division and growth of the nervous system in the fetus and during the first weeks after birth (Anderson et al., 2000).

Adult-Onset Cancer

Human evidence of increased cancer risk resulting from exposures at critical periods during childhood is provided by observations of higher rates of radiation-induced breast cancer among women exposed during puberty, compared with those exposed after puberty (NRC, 1990b), and by observations of higher rates of leukemia and thyroid cancer among individuals exposed to radiation as children, compared with those exposed as adults (NRC, 1990b). Evidence in humans of increased cancer risk following *in utero* exposure is provided by increases in the incidence of clear cell adenocarcinoma of the vagina in young women exposed to diethylstilbestrol *in utero* (DES daughters), in the absence of increased risk for this cancer in DES mothers who were exposed as adults (Preston-Martin, 1989).

Epidemiological evidence indicates that exposure to tobacco smoke during puberty may increase risk of breast cancer later in life, particularly among women who have an enzyme which reduces the detoxification of chemicals which are acetylated, i.e. NAT2 slow deacetylators (Marcus et al., 2000; Morabia et al., 2000; Lash and Aschengrau, 1999).

Experimental animal studies show increased cancer risk following early in life exposure to carcinogens such as urethane, vinyl chloride, DES, tamoxifen, nitrosourea compounds (e.g., methylnitrosourea), and alkenylbenzene compounds (e.g., safrole and estragole).
Animal studies have shown that *in utero* exposure to some carcinogens can lead to mammary tumors in the offspring that do not manifest until adulthood (Napalkov, 1986; Tomatis, 1989). Numerous studies in rodents have demonstrated an increased susceptibility of the fetus, neonates and very young animals to urethane-induced cancers (Salmon and Zeise, 1991). In one study there were increased numbers and types of lung tumors when newborn mice were exposed to urethane compared to mice at maturity or 11-12 weeks of age (Kaye and Trainin, 1966). In another study, Rogers (1951) compared the tumorigenic effect of a single intraperitoneal injection of urethane in mice dosed at either two, four, six, eight or ten weeks of age. The author found that lung tumor incidence and multiplicity decreased significantly as the age at exposure increased (e.g., from 100% tumor incidence and an average of 6.1 tumors per animal in mice exposed at two weeks of age to 76% tumor incidence and an average number of tumors per animal of 2.8 in mice exposed at ten weeks of age). The basis for this increased susceptibility is thought to be due to the fact that levels of esterases responsible for the detoxification and elimination of urethane are relatively low in newborns. This results in higher blood levels of urethane for longer periods of time, and provides greater opportunity for minor routes of urethane metabolism to occur, metabolically transforming urethane to the ultimate active carcinogen.

Vinyl chloride is another carcinogen for which the effect of age at exposure on tumor outcome has been extensively studied (Maltoni et al., 1981; Drew et al., 1983). In studies comparing the tumorigenic effects of vinyl chloride in rats exposed as either newborns or 11-week old animals,

a significant increase in liver tumors was observed in the newborn group as compared with no increased incidence of these tumors in the 11-week old group (Maltoni et al., 1981). When rats, mice, and hamsters were exposed to vinyl chloride for 6- or 12-month periods starting at two, eight, or 14 months of age, the younger the age at first exposure, the greater the lifetime tumor incidence (Drew et al., 1983). Age-dependent differences in metabolism, leading to increased formation of DNA-reactive metabolites and increased DNA alkylation, combined with differences in cell proliferation rates, are thought to account for the increased susceptibility of young animals to vinyl chloride carcinogenesis. Researchers from the US EPA have suggested a risk assessment framework for vinyl chloride exposure in which exposures to children are given greater weight (i.e., risk) compared to exposures later in life (Cogliano et al., 1996).

Tamoxifen is another carcinogen with estrogenic (and antiestrogenic) effects that has been shown to cause cancers of the female reproductive tract of rats and mice when exposures occur earlier, rather than later, in life. When tamoxifen was administered to newborn female mice for the first five days of life, incidences of rare uterine adenocarcinomas of up to 50 % were observed (Newbold et al., 1997). An increase in uterine and other tumors of the reproductive tract was also observed in female mice exposed to tamoxifen *in utero* on gestation days 12-18 (Diwan et al., 1997). There was no increase in tumors of the reproductive tract when mature rats were exposed to tamoxifen in the standard long-term bioassays (IARC, 1996). However, neonatal exposure on days two through five of life resulted in the development of rare uterine adenocarcinomas and squamous cell carcinomas of the vagina and cervix (Carthew et al., 2000).

The carcinogenic mode of action of tamoxifen in young animals remains unknown, but these findings, taken together with the studies of DES, suggest that the developing reproductive tract is particularly sensitive to hormonally active carcinogens. The information obtained from studies of DES, tamoxifen, and estrogen may have value in predicting risk from early life exposures to other estrogenic compounds, based on a common mode of action.

Alkenylbenzene compounds such as safrole, estragole and methyleugenol cause a higher incidence of hepatic and other tumors in rodents exposed as newborns compared with rodents exposed as adults (Drinkwater et al., 1976; Miller et al., 1983; Wiseman et al., 1987; Vesselinovitch, 1983). The reason for this finding has not been elucidated.

To summarize, data in humans and animals for a variety of carcinogens suggest that exposures to such carcinogens early in life may result in a greater lifetime risk of cancer compared to exposures later in life. For example, humans exposed to ionizing radiation early in life have greater lifetime risk of leukemia, breast cancer and lung cancer than humans exposed later in life. Data from animal studies provide additional examples of increased sensitivity to early life exposures. These effects span a wide range of target tissues, including the liver (vinyl chloride, safrole), brain (methylnitrosourea), reproductive tract (DES, tamoxifen), and lung (urethane).

3.5 Compilation of Candidate Chemicals Based on Critical Health Effects

A review of the scientific literature for chemically-induced adverse effects in the developing immune, nervous, respiratory, endocrine and reproductive systems, or which caused carcinogenesis in children, was performed to identify candidate chemicals for further in-depth review. While we are currently focusing on the non-cancer endpoint, an attempt had been made to also search for literature pertaining to carcinogenesis in children. Carcinogenesis literature identified will be reviewed when the children's cancer risk assessment methodology becomes available. These chemicals and those identified by Proposition 65 are combined in Table 3.5.1. In total, there are about 198 candidate chemicals in this compilation. Of these, 87 were derived from the Proposition 65 list of developmental and reproductive toxicants. Chemicals on the Proposition 65 list that were obviously not likely contaminants at school sites, such as pharmaceuticals, were excluded. It should be emphasized that the chemicals in this table have not been determined to be more toxic to children than to adults. They have been identified for further in-depth review to determine if there is sufficient scientific data to develop a child-specific health guidance values.

Table 3.5.1 Candidate Chemicals Based on Critical Health Effects

Chemical	CASRN	Immune system toxicant	Nervous system toxicant	Respira-tory system toxicant	Endocrine, Neuro-endocrine or Neuro-behavioral systems toxicant	Female reproductive system toxicant or estrogen disrupter	Male reproductive system toxicant	Carcino-genesis	References	Prop 65 DART list
Acetaldehyde	75-07-0		X						Andersen et al, 2000	
Acrolein	107-02-8	X							Handzel, 2000	X
Aldrin	309-00-2	X			X	X	X		Brucker-Davis, 1998; Gellert, 1978 (a); Rao et al, 1982; Voccia et al, 1999	
Aluminum	7429-90-5		X		X				Andersen et al, 2000; Barone et al, 2000; Brucker-Davis, 1998; Golub et al, 1996; Marlowe, 1986	
Amitraz	33089-61-1									X
Arsenic	7440-38-2	X	X			X	X		Andersen et al, 2000; Berlin et al, 1983; Ihrig et al, 1997; Luster et al, 1990	X
Asbestos	1332-21-4			X		X			Hague et al, 1991; Davis et al, 1996	
Benomyl	17804-35-2						X		Bernstein, 1984	X
Benzene	71-43-2	X				X			Davis et al, 1996; Luster et al 1990; Thurson et al, 2000; Xu et al, 1998	X
Benzidine	92-87-5	X							Luster et al, 1990	
Beryllium compounds		X							Luster et al, 1989	

Chemical	CASRN	Immune system toxicant	Nervous system toxicant	Respira-tory system toxicant	Endocrine, Neuro-endocrine or Neuro-behavioral systems toxicant	Female reproductive system toxicant or estrogen disrupter	Male reproductive system toxicant	Carcino-genesis	References	Prop 65 DART list
Bisphenol-A	80-05-7	X			X	X	X		Ahmed 2000; Bigsby et al, 1999; Goldman et al, 2000; Stoker et al, 2000 (b)	
Boric Acid	10043-35-3		X			X	X		Bernstein, 1984; Chapin, 1994	
Boron	7440-42-8						X		Krasovskii et al, 1976; Lee et al, 1978; Rao et al, 1982	
Bromacil lithium salt	53404-19-6									X
2-Bromo-propane	75-26-3					X	X		Figa-Talamanca et al, 2001; Wu et al, 1999	
Bromoxynil	1689-84-5									X
Bromoxynil octanoate	1689-99-2									X
1,3-Butadiene	106-99-0					X	X		Anderson et al, 1999; Christian, 1996; Davis et al, 1996	
p-tert-Butylbenzoic acid	98-73-7						X		Bernstein, 1984	
p-tert-Butyltoluene	98-51-1						X		Bernstein, 1984	
p-tert-Butyl benzaldehyde	939-97-9						X		Bernstein, 1984	

Cadmium	7440-43-9	X	X	X	X	X	X		Andersen et al, 2000; Bellanti, 1974; Berlin et al, 1983; Bernier et al; 1995; Brucker-Davis, 1998; Clarkson et al, 1985; Copius Peereboom-Stegeman, 1989; Dan et al, 2000; DeRosa et al, 1998; Genbecev et al, 1993; Holladay 1999; Holladay et al, 2000; LaFuente et al 2000; LaFuente et al, 1997; Lesser et al, 1995; Luster 1990; Paksy et al, 1997; Ritz et al, 1998; Schroeder, 2000; Stellman, 1979	X
Captan (orthocide)	133-06-2						X		Bernstein, 1984	
Carbofuran	1563-66-2	X			X				Brucker-Davis, 1998; Holladay et al, 2000; Holladay 1999; Luster 1990	
Carbon disulfide	75-15-0	X	X		X	X	X		Andersen et al, 2000; Bernstein, 1984; Brucker-Davis, 1998; Cai et al, 1981; Cooper et al, 1997; Figa-Talamanca et al, 2001; Mattison et al, 1990; Paul et al, 1988; Pryor et al, 2000; Sever et al, 1985; Stellman, 1979; Wagner et al, 1988; Zhou et al, 1988	X
Carbon monoxide	630-08-0									X

Carbon tetrachloride	56-23-5			X			X		Boyd et al, 1980; Plopper et al, 1994; Stellman, 1979	
Chinomethionat (Oxythioquinox)	2439-01-2									X
Chlordane	57-74-9	X			X	X		X	Ahmed, 2000; Barone et al, 2000; Blyler et al, 1994; Brucker-Davis, 1998; DeRosa et al, 1998; Holladay et al, 2000; Holladay, 1999; Luster et al, 1990; Olea et al, 1998; Reigart, 1995; Spyker-Cranmer et al, 1982; Theus et al, 1992; Voccia et al, 1999; Zahm et al, 1998	
Chlordecone (Kepone)	143-50-0		X		X	X	X		Andersen et al, 2000; Cooper and Kavlock, 1997; Gellert, 1978; Gray et al, 1998; Olea et al, 1998; Paul et al, 1988; Pryor et al, 2000; Rao et al, 1982; Sever et al, 1985; Stellman, 1979	X
Chlorfenvin-phos	470-90-6						X		Bernstein, 1984	
Chlorine	7782-50-5			X					Pherwani et al, 1989	
4-Chloro-3-nitroaniline	635-22-3						X		Bernstein, 1984	
Chloroprene (2-chlorobuta-1,3-diene)	126-99-8						X		Bernstein, 1984; Sever et al, 1985; Stellman, 1979	

Chlorpyrifos	2921-88-2		X	X					Andersen et al, 2000; Barone et al, 2000; Chakraborti et al, 1993; Chanda et al, 1996; Davis et al, 1998; Eskenazi et al, 1999; Lemus et al, 2000; Slotkin et al, 2001	
Chlorsulfuron	64902-72-3									X
Chlozolinate (Vinclozolin)	50471-44-8					X	X		Gray et al, 1998; Cooper et al, 1997; Reigart, 1995; Rubin et al, 1999; Stoker et al, 2000 (b)	
Chromium III	16065-83-1						X		Clarkson et al, 1985; Rao et al, 1982	
Chromium VI	18540-29-9	X					X		Al-Tawil et al, 1983; Clarkson et al, 1985; Figa-Talamanca et al, 2001	
Ciodrin	7700-17-6						X		Bernstein, 1984	
Cyanazine	21725-46-2									X
Cycloate	1134-23-2									X
Cyclohexanol	108-93-0						X		Bernstein, 1984	X
Cycloheximide	66-81-9									X
Cyclohexylamine HCL	4998-76-9						X		Bernstein, 1984	
Cyhexatin	13121-70-5									X
2,4-DB (2,4-D butyric acid)	94-82-6									X
DDD	72-54-8				X	X			Brucker-Davis, 1998; Ottoboni, 1972	
DDE	72-55-9	X			X	X	X		Brucker-Davis, 1998; DeRosa et al, 1998; Ottoboni, 1972; Palanza et al, 1999; Rubin et al, 1999; Stoker et al, 2000 (b); Vine	

									et al, 2001	
DDT	50-29-3	X	X		X	X	X	X	Ahmed, 2000; Brucker-Davis, 1998; Cooper et al, 1997; DeRosa et al, 1998; Eriksson, 1997; Galand et al, 1987; Genbecev et al; 1993; Holladay et al, 2000; Holladay, 1999; Mattison et al, 1990; Olea et al, 1998; Ottoboni, 1972; Palanza et al, 1999; Rao et al, 1982; Voccia et al, 1999; Zahm et al, 1998	X
Dacthal	1861-32-1						X		Bernstein, 1984	
Diazinon	333-41-5	X			X			X	Davis et al, 1993; Eskenazi et al, 1999; Holladay et al, 2000; Holladay, 1999; Spyker et al, 1977	
Dibromochloropropane (DBCP)	96-12-8				X	X	X		Anderson et al, 1997; Bernstein, 1984; Davis et al, 1996; DeRosa et al, 1988; Figa-Talamanca et al, 2001; Mattison et al, 1990; Paul et al, 1988; Rao et al 1982; Sever et al, 1985; Stellman, 1979	X
Dichlorophene	97-23-4									X
Dichlorvos	62-73-7		X				X	X	Barone et al, 2000; Bernstein, 1984; Eskenazi et al, 1999; May, 2000; Zahm et al, 1998	

Diclofop methyl	51338-27-3									X
p-Dicyano-benzene	623-26-7						X		Bernstein, 1984	
Dieldrin	60-57-1	X	X		X	X	X	X	Barone et al, 2000; Brucker-Davis, 1998; DeRosa et al, 1998; Gellert 1978 (a); Good et al, 1969; Olea et al, 1998; Rao et al, 1982; Voccia et al, 1999; Zahm et al, 1998	
Diesel Exhaust Particles (DEP)		X		X		X			CA ARB, "The Report on Diesel Exhaust," 1998; Handzel, 2000; Taneda et al, 2000	
Diethylstil-bestrol (DES)	56-53-1	X			X	X	X		Bigsby et al, 1999; Cooper et al, 1997; Goldman et al, 2000; Holladay et al, 2000; Holladay 1999; Palanza et al, 1999; Sharpe 2001	X
Dimethoate	60-51-5		X		X	X			Barone et al, 2000; Brucker-Davis, 1998; Budreau et al, 1973; Eskenazi et al, 1999	
Dimethyl methylphosphonate (DMMP)	756-79-6						X		Bernstein, 1984	
Dimethyl sulfate (DMS)	77-78-1					X			Bishop et al, 1997	
m-Dinitrobenzene	99-65-0									X
o-Dinitrobenzene	528-29-0									X
p-Dinitrobenzene	100-25-4									X
Dinitrotoluene										X

(technical grade)										
2,4-Dinitro-toluene	121-14-2									X
2,6-Dinitro-toluene	606-20-2									X
Dinocap	39300-45-3									X
Dinoseb	88-85-7		X				X		Andersen et al, 2000; Bernstein, 1984	X
Dioxins and dibenzofurans (TCDD)	1746-01-6	X	X	.	X	X	X		Bigsby et al, 1999; Brucker-Davis, 1998; Cooper and Kavlock, 1997; DeRosa et al, 1998; Fan et al, 1996; Figa-Talamanca et al, 2001; Goldman et al, 2000; Guo et al, 2000; Hauser et al, 1998; Holladay et al, 2000; Holladay, 1999; Huisman et al, 1995; Luster et al, 1990; Nagayama et al, 1998; Porterfield, 2000; Pryor et al, 2000; Tilson, 1998; Weisglas-Kuperus et al, 1998	TCDD
Disodium cyano-dithioimido-carbonate	138-93-2									X
Dithio-carbamates					X	X			Brucker-Davis, 1998; Cooper et al, 1997; Tilson, 1998	
Maneb	1247-38-2		X		X		X		Bernstein, 1984; Brucker-Davis, 1998; Ferraz et al, 1988; Rao et al 1982	
Nabam	142-59-6				X				Brucker-Davis, 1998	X
Potassium	128-03-0									X

dimethyldithiocarbamate										
Sodium dimethyldithiocarbamate	128-04-1									X
Zineb	12122-67-7				X		X		Bernstein, 1984; Brucker-Davis, 1998; DeRosa et al, 1998; Rao et al, 1982	
Ziram	137-30-4				X				DeRosa et al, 1998	
Diquat	85-00-7					X	X		Rao et al, 1982	
Endosulfan	115-29-7				X	X	X		Bernstein, 1984; Brucker-Davis, 1998; Olea et al, 1998	
Environmental Tobacco Smoke		X	X	X		X			Magers et al, 1995; May, 2000; Pinkerton et al, 2000	X
Epichlorohydrin	106-89-8						X		Bernstein, 1984	X
Ethanol	64-17-5		X		X		X		Barone et al, 2000; Bernstein, 1984; Little et al, 1992; Olney et al, 2000; Stoker et al, 2000 (b)	X
Ethyl dipropylthiocarbamate	759-94-4									X
Ethylene dibromide (1,2-dibromo-ethane)	106-93-4					X	X		Bernstein, 1984; Paul et al, 1988; Rao et al, 1982	X
Ethylene and Diethylene Glycol Ethers and Acetates			X			X	X		Andersen et al, 2000; Correa et al 1996; Figa-Talamanca et al, 2001; Paul et al, 1988; Schenker et al, 1995	X

Ethylene Glycol Monoethyl Ether (2-Ethoxy - ethanol)	110-80-5	X				X	X		Almekinder et al 1997; Bernstein, 1984; Davis et al, 1996; Figa-Talamanca et al, 2001; Houchens et al, 1984	X
Ethylene Glycol Monoethyl Ether Acetate (2-Ethoxyethyl Acetate)	111-15-9						X		Figa-Talamanca et al, 2001	X
Ethylene Glycol Monomethyl Ether (2-Methoxy - ethanol)	109-86-4	X				X	X		Bernstein, 1984; Davis et al, 2001 (a); Figa-Talamanca et al, 2001; Holladay, 1999; Houchens et al, 1984; Kayama, 1991; Smialowicz et al, 1991, 1992, 1994	X
Ethylene oxide	75-21-8		X			X	X		Andersen et al, 2000; Bernstein, 1984; Bishop et al, 1997; Stellman, 1979	X
Ethylene thiourea	96-45-7									X
Fenoxaprop ethyl	66441-2-34									X
Fenthion (OP)	55-38-9					X			Budreau et al, 1973	
Fluazifop butyl	69806-50-4									X
Fluvalinate	69409-94-5									X
Formaldehyde	50-00-0	X		X		X			Handzel, 2000; Luster et al, 1989; Taskinen et al, 1999; Thrasher et al, 1990; Wantke et al, 1996	
Heptachlor	76-44-8	X				X	X	X	Nicolopoulou-Stamati et al, 2001; Rani et al, 1995; Smialowicz et al, 2001; Zahm et al, 1998	

Heptachlor Epoxide	1024-57-3	X			X				Brucker-Davis, 1998; DeRosa et al, 1998; Voccia et al, 1999	X
Hexachloro-benzene	118-74-1	X			X	X	X		Ahmed 2000; Bernstein, 1984; Brucker-Davis, 1998; DeRosa et al, 1998; Foster et al 1992, 1995; Goldey et al, 1992; Holladay et al, 2000; Holladay, 1999; Luster et al, 1989; Rao et al, 1982; Sala et al, 2001	X
gamma-Hexachloro-cyclohexane (Lindane)	58-89-9	X	X		X	X	X	X	Andersen et al, 2000; Davis et al, 1993; DeRosa et al, 1998; Goldey et al, 1992; Goldman et al, 2000; Holladay & Smialowicz, 2000; Holladay, 1999; Olea et al, 1998; Silvestroni et al, 1999; Voccia et al, 1999	
Hexachloro-phene (HCP)	70-30-4						X		Gellert et al, 1978 (b); Rao et al, 1982	
Hexafluoro-acetone (hexafluoro-propanone)	684-16-2						X		Bernstein, 1984	
Hexamethyl-phosphoramide	680-31-9						X		Bernstein, 1984	X
Hexamethylthio-phosphoramide	3732-82-9						X		Bernstein, 1984	
2,5-Hexane-dione	110-13-4						X		Bernstein, 1984	

N-Hexane	110-54-3		X						Andersen et al, 2000	
Hydramethylnon	67485-29-4									X
Isocyanates		X		X					Lesser et al, 1995; Raulf-Heimsoth et al, 1998	
Diphenyl methane diisocyanate (methylene diphenyl diisocyante - MDI)	101-68-8	X							Baur et al, 1995	
Isopropyl methane-sulfonate (IMS)	926-06-7					X			Bishop et al, 1997; Hoyer, et al, 1996	
Ketoconazole	65277-42-1				X	X	X		Bigsby et al, 1999; Cooper et al, 1997; Goldey et al, 1992	
Lead	7439-92-1	X	X		X	X	X		Adams et al, 2000; Anderson et al, 2000; Barone et al, 2000; Bellanti, 1974; Berlin et al, 1983; Bernier et al, 1995; Brucker-Davis, 1998; Clarkson et al, 1985; DeRosa et al, 1998; Figa-Talamanca et al, 2001; Holladay & Smialowicz, 2000; Holladay 1999; Luster et al, 1990; Paul et al, 1988; Pryor et al, 2000; Stellman, 1979; Tang et al, 1999; Tilson, 1998; Wennberg, 1994; Xuezhi et al, 1992	X

Linuron	330-55-2						X		Bigsby et al, 1999; Lambright et al, 2000; McIntyre et al, 2000; Stoker et al, 2000 (b)	X
Malathion	121-75-5				X		X		Bernstein, 1984; Brucker-Davis, 1998	
Manganese	7439-96-5	X	X	X			X		Berlin et al, 1983; Cawte, 1985; Ferraz et al, 1988; Figa-Talamanca et al, 2001; Lesser et al, 1995; May, 2000; Smialowicz et al, 1984; Stellman, 1979; Wennberg, 1994	
Mercury (Elemental)	7439-97-6	X		X	X	X	X		Andersen et al, 2000; Berlin et al, 1983; Bernier et al, 1995; Clarkson et al, 1985; Davis et al, 2001 (a); DeRosa et al, 1998; Etzel, 2000; Figa-Talamanca et al, 2001; Genbecev et al, 1993; May, 2000; Perlingeiro et al, 1994	
Mercury (Methyl)	22967-92-6	X	X		X	X	X		Andersen et al, 2000; Barone et al, 2000; Bernier et al, 1995; Clarkson et al, 1985; Figa-Talamanca et al, 2000; Holladay et al, 2000; Holladay, 1999; Ilback et al, 1991; May, 2000; Rice 1996; Tilson, 1998	X
Metaldehyde	9002-91-9						X		Bernstein, 1984	
Metham sodium	137-42-8									X
Methanol	67-56-1		X						Andersen et al, 2000	
Methazole	20354-26-1									X

Methoxychlor	72-43-5	X			X	X	X		Ahmed, 2000; Bigsby et al, 1999; Chapin et al, 1997; Davis et al, 2001 (b); DeRosa et al, 1998; Genbecev et al, 1993; Goldman et al, 2000; Harris et al, 1974; Palanza et al, 1999; Rao et al, 1982; Stoker et al, 2000 (b)	
Methyl chloride (Chloromethane)	74-87-3						X		Bernstein, 1984	X
Methylene chloride (Dichloro-methane)	75-09-2	X				X			Dhillon et al, 1995; Lindbohm et al, 1990	
Methyl ethyl ketone	78-93-3	X	X						Andersen et al, 2000; Stewart et al, 1992	
Metiram	9006-42-2									X
Mirex	2385-85-5				X	X			Brucker-Davis, 1998; Gellert, 1978 (a); Goldman et al, 2000; Rao et al, 1982	
Myclobutanil	88671-89-0									X
Nickel carbonyl	13463-39-3									X
Nickel (metallic)	7440-02-0	X				X	X		Budinger et al, 2000; Clarkson et al, 1985; Leonard et al, 1984; Stellman, 1979	
Nitrapyrin	1929-82-4									X
p-Nitro-benzamide	619-80-7						X		Bernstein, 1984	
Nitrogen mustard (Mechlor-ethamine)	51-75-2									X

1-Nitronaphthalene	86-57-7			X					Paige et al, 1997; Watt et al, 2000	
Octylphenol	27193-28-8	X				X			Ahmed 2000; Bigsby et al, 1999; Goldman et al, 2000; Gray et al, 1998	
Olin 1763	5135-80-8						X		Bernstein, 1984	
Organic Solvents						X			Lindbohm et al, 1990	
Organotins		X			X				Barone et al, 2000; Luster et al, 1990; Tilson, 1998	
Di-n-butyl-tindichloride	683-18-1	X							Luster et al, 1989	
Di-n-octyl-tindichloride	3542-36-7	X							Luster et al, 1989	
Tributyltin	688-73-3				X				DeRosa et al, 1998	
Tributyltin oxide – TBTO	56-35-9	X							Luster et al, 1990	
Triethyltin	997-50-2		X						Andersen et al, 2000; Barone et al, 2000; Barone et al, 1995; Freeman et al, 1994	
Trimethyltin	1631-73-8		X						Andersen et al, 2000	
Triphenyltin Chloride (TPTCL)	639-58-7					X			Ema et al, 1999	
Organophosphates - see individual pesticides for specific information		X	X	X					Erikkson et al, 2000; Eskenazi et al, 1999; Luster et al, 1989	
Oxadiazon	19666-30-9									X
Oxydemeton	301-12-2									X

methyl										
Paraquat	1910-42-5		X			X	X		Barone et al, 2000; Eriksson et al, 2000; Rao et al, 1982	
Parathion	56-38-2		X		X				DeRosa et al, 1998; Veronesi et al, 1990	
PAHs - see individual PAHs for specific information		X		X	X	X	X		Bishop et al, 1997; Brucker-Davis, 1998; Davila et al, 1995; Davis et al, 1996; Handzel, 2000; Holladay & Smialowicz, 2000; Hoyer et al, 1996; Mattison et al, 1983 (a); Plopper et al, 1993; Sram et al, 1999	X
Benzo[a]pyrene	50-32-8	X			X	X			Brucker-Davis, 1998; Davis et al, 1996; DeRosa et al, 1998; Holladay et al, 2000; Holladay 1999; Luster et al 1990; Mattison et al, 1983 (a); Paul et al, 1988	
Dimethylbenz[a] anthracene	57-97-6	X				X			Bishop et al, 1997; Davis et al, 1996; Holladay et al, 2000; Holladay, 1999	
Methylcholanthrene	56-49-5	X			X				Brucker-Davis, 1998; Holladay et al, 2000; Holladay, 1999	
Naphthalene	91-20-3			X					Fanucchi et al, 1997	
PBBs (Poly-brominated biphenyls)		X	X		X	X			Andersen et al, 2000; Blanck et al, 2000; Brucker-Davis, 1998; DeRosa et al, 1998; Holladay et al, 2000; Holladay, 1999; Luster et al 1990; Mattison et al, 1983 (b)	X

PCBs (Poly-chlorinated biphenyls)	1336-36-3	X	X		X	X	X		Ahmed, 2000; Barone et al, 2000; Bernstein, 1984; Bigsby et al, 1999; Brouwer et al, 1998 and 1999; Brucker-Davis, 1998; DeRosa et al, 1998; Erikkson, 1997; Guo et al, 2000; Hauser et al, 1998; Holladay et al, 2000; Holladay, 1999; Huisman et al, 1995; Lemasters et al, 2000; Luster et al, 1990; Mattison et al, 1983 (b); Olea et al, 1998; Osius et al, 1999; Porterfield, 2000; Rao et al, 1982; Sala et al, 2001; Tilson, 1998; Weisglas-Kuperus, 2000 and 1998	X
PCP (Pentachloro-phenol)	87-86-5	X			X	X			Brucker-Davis, 1998; Daniel et al, 2001; Daniel et al, 1995; DeRosa et al, 1998; Gerhard et al, 1999; McConnachie et al, 1991	
Perchlorate	14797-73-0				X				Brechner et al, 2000; Crump et al, 2000	
Petroleum distillates (Petroleum ether)	8030-30-6	X		X					Robledo et al, 2000	
Petroleum Hydrocarbons						X			Xu et al, 1998	

Phthalates					X	X	X		Bernstein, 1984; Bigsby et al, 1999; Brucker-Davis, 1998; Colon et al, 2000; DeRosa et al, 1998; Foster et al, 2001; Sharpe 2001	
Butyl benzyl phthalate	85-68-7					X	X		Gray Jr. et al, 1998; Olea et al, 1998	
Dibutyl phthalate	84-74-2				X		X	X	Bigsby et al, 1999; Foster et al, 2001; Sharpe 2001	
Di-(2-ethylhexyl) phthalate (DEHP)	117-81-7		X	X	X	X	X	X	Andersen et al, 2000; Brucker-Davis, 1998; Davis et al, 2001 (b); Davis et al, 1994; Foster et al, 2001; Gray Jr. et al, 1998; Oie et al, 1997; Sharpe 2001	
Diethyl phthalate	84-66-2				X		X		Bigsby et al, 1999; DeRosa et al, 1998	
Diisononyl phthalate	28553-12-0						X		Gray Jr. et al, 1998	
Dimethyl phthalate	131-11-3				X		X		Bigsby et al, 1999; DeRosa et al, 1998	
Di-n-pentyl phthalate (Diamyl phthalate)	131-18-0						X		Stoker et al, 2000 (b)	
Mono-n-butyl phthalate (MPB)	131-70-4						X		Imajima et al, 1997	
Monoethylhexyl phthalate (MEHP)	4376-20-9						X		Sharpe, 2001	
Phthalic anhydride	85-44-9	X					X		Bernstein, 1984; Luster et al, 1989	
Procymidone fungicide	3280-916-8						X		Gray et al, 1998; Hosokawa, 1993	

Propargite	2312-35-8									X
Propylene oxide	75-56-9	X					X		Bernstein, 1984; Steinkraus et al, 1994	
(n) Propyl-methane-sulfonate	1912-31-8					X			Bishop et al, 1997	
Pyrethroids		X	X		X				Brucker-Davis, 1998; Diel et al, 1999; Eriksson et al, 2000; Wagner, 2000; Wax et al, 1994	
Permethrin	52645-53-1	X							Fuortes, 1999; Punareewattana et al, 2001	
Resmethrin	10453-86-8									X
Quizalofopethyl	76578-14-8									X
Sevin (Carbaryl)	63-25-2				X	X	X	X	Bernstein, 1984; Brucker-Davis, 1998; Davis et al, 1993; Paul et al, 1988; Shtenberg et al, 1968; Stellman, 1979	
Sodium fluoroacetate	62-74-8									X
Styrene	100-42-5	X	X		X				Andersen et al, 2000; DeRosa et al, 1998; Jegaden et al, 1993; Rabovsky et al, 2001; Tulinska et al, 2000	
Terbacil	5902-51-2									X
Tetrachloro-ethylene (PERC/PCE)	127-18-4	X				X			Andrys et al, 1997; Lindbohm et al, 1990; Sallman et al; 1995	
Tetrasul (2,4,5,4'-tetrachloro-diphenylsulfide)	2227-13-6						X		Bernstein, 1984	
Thiophanate	23564-05-8									X

methyl										
Toluene	108-88-3	X	X			X			Andersen et al, 2000; Plenge-Bonig et al, 1999; Lesser et al, 1995; Luster et al, 1990; Sallmen et al, 1995	
Toxaphene	8001-35-2	X	X		X	X			Andersen et al, 2000; Brucker-Davis, 1998; DeRosa et al, 1998; Olea et al, 1998; Voccia et al, 1999	
Triadimefon	43121-43-3									X
Triazine Herbicides					X	X			Davis et al, 1996; Tilson, 1998	
Amitrole (3-amino-1,2,4-triazole)	61-82-5				X				DeRosa et al, 1998	
Atrazine	1912-24-9		X		X	X	X		Brucker-Davis, 1998; Cooper et al, 1997; DeRosa, 1998; Stoker et al, 2000 (a); Thurston et al, 2000; Tilson, 1998; Trentacoste et al, 2001; US EPA, IRIS substance file - Atrazine, 2001; Xu et al, 1998	
Simazine	122-34-9		X		X				Andersen et al, 2000; Tilson, 1998	
Trichlorfon	52-68-6		X				X		Andersen et al, 2000; Bernstein, 1984	
1,1,1-Trichloro-ethane (Methyl chloroform)	71-55-6	X	X		X				Andersen et al, 2000; Coleman et al, 1999; Jones et al, 1996; Muttray et al, 1999	
Trichloro-ethylene (TCE)	79-01-6		X		X	X			Andersen et al, 2000; Sallman et al, 1995; White et al, 1997	

Trifluralin	1582-09-8									X
Triforine	26644-46-2									X
Urethane	51-79-6									X
Vinyl Chloride	75-01-4						X	X	Bernstein, 1984; Reigart, 1995; Sever et al, 1985; Stellman, 1979	
Vinylidene chloride (1,1-dichloro-ethene)	75-35-4						X		Bernstein, 1984	
4-Vinyl-1-cyclo-hexene	100-40-3					X			Davis et al, 1996; Hoyer et al, 1996	
4-Vinylcyclo-hexene diepoxide (Vinylcyclohexe ne dioxide, VCD)	106-87-6					X			Davis et al, 1996; Lemasters et al, 2000	
Warfarin	81-81-2									X
Xylene	1330-20-7	X	X			X			Andersen et al, 2000; Goyer, 1996; Riihimaki et al, 1980; Sallmen et al, 1995	
Zearalenone	17924-92-4	X			X				Ahmed, 2000; Gray et al, 1998	

4. Next Steps

OEHHA has compiled these two candidate lists to facilitate the prioritization of chemicals for evaluation for purposes of developing child-specific numerical health criteria. The compilations will be updated as studies become available. OEHHA will consider revising the compilations when the results of the Portable Classroom Study become available, and adding candidates that DTSC identifies in its upcoming PEA reviews and that DPR identifies from pesticide use notifications from school districts pursuant to AB 2260 (the Healthy School Act of 2000). Further, the compilations will be updated upon the completion of an in-depth evaluation of the priority chemicals discussed below.

AB 2872 [HSC 901 (g)] also stipulates that by December 31, 2002, and annually thereafter, OEHHA develop numerical health-based guidance for five of the identified chemicals until the list is exhausted. Pursuant to this task, OEHHA will select a subset of chemicals from the candidate lists for further evaluation, to determine if there are sufficient data on critical toxic effects in the organ systems that are developing in children to support the creation of child-specific numerical guidance values based on a non-cancer end-point. To that end, the following selection criteria are proposed:

- Chemicals having a strong likelihood of being found at school sites.
- Chemicals with the strongest evidence of possible developmental, neurological, immunological, respiratory, reproductive, or endocrine effects.
- Where applicable, chemicals with an existing non-cancer RfD that is based on older toxicity studies in adult animals that approximates the dose associated with a 10^{-4} to 10^{-6} cancer risk. This criterion helps target those chemicals that would be of greatest concern to OEHHA because if data from recent studies on juvenile animals suggest that child-specific physiological susceptibility exists, they would pose a significant non-cancer risk compared to cancer risk.

In a separate task, OEHHA is developing a cancer evaluation methodology for children pursuant to HSC901(e). Because that methodology will not be available until 2004, our initial efforts are focused on evaluating the non-cancer effects of chemicals. We will evaluate the cancer endpoint when the children's cancer methodology is developed.

REFERENCES

1. Adams, J., Barone, S. Jr, LaMantia, A., Philen, R., Rice, D. C., Spear, L., & Susser, E. (2000). Workshop to identify critical windows of exposure for children's health: neurobehavioral work group summary. Environ Health Perspect, 108 Suppl 3, 535-44.

2. Ahmed, S. A. (2000). The immune system as a potential target for environmental estrogens (endocrine disrupters): a new emerging field. Toxicology, 150(1-3), 191-206.

3. Al-Tawil, N. G., Marcusson, J. A., & Moller, E. (1983). Lymphocyte stimulation by trivalent and hexavalent chromium compounds in patients with chromium sensitivity. An aid to diagnosis. Acta Derm Venereol, 63(4), 296-303.

4. Almekinder, J. L., Lennard, D. E., Walmer, D. K., & Davis, B. J. (1997). Toxicity of methoxyacetic acid in cultured human luteal cells. Fundam Appl Toxicol, 38(2), 191-4.

5. Andersen, H. R., Nielsen, J. B., & Grandjean, P. (2000). Toxicologic evidence of developmental neurotoxicity of environmental chemicals. Toxicology, 144(1-3), 121-7.

6. Anderson, D., Dobrzynska, M. M., & Basaran, N. (1997). Effect of various genotoxins and reproductive toxins in human lymphocytes and sperm in the Comet assay. Teratog Carcinog Mutagen, 17(1), 29-43.

7. Anderson, L. M., Diwan, B. A., Fear, N. T., & Roman, E. (2000). Critical windows of exposure for children's health: cancer in human epidemiological studies and neoplasms in experimental animal models. Environ Health Perspect, 108 Suppl 3, 573-94.

8. Anderson, R. C., & Anderson, J. H. (1999). Acute respiratory effects of diaper emissions. Arch Environ Health, 54(5), 353-8.

9. Andrys, C., Hanovcova, I., Chylkova, V., Tejral, J., Eminger, S., & Prochazkova, J. (1997). Immunological monitoring of dry-cleaning shop workers--exposure to tetrachloroethylene. Cent Eur J Public Health, 5(3), 136-42.

10. Anisimov, V. N. (1982). Carcinogenesis and aging. III. The role of age in initiation and promotion of carcinogenesis. Exp Pathol, 22(3), 131-47.

11. Ardies, C. M., & Dees, C. (1998). Xenoestrogens significantly enhance risk for breast cancer during growth and adolescence. Med Hypotheses, 50(6), 457-64.

12. Baker, T. (1972). Reproductive Biology. Amsterdam: Excerpta Medica.

13. Barone, S. Jr, Das, K. P., Lassiter, T. L., & White, L. D. (2000). Vulnerable processes of nervous system development: a review of markers and methods. Neurotoxicology, 21(1-2), 15-36.

14. Barone, S. Jr, Stanton, M. E., & Mundy, W. R. (1995). Neurotoxic effects of neonatal triethyltin (TET) exposure are exacerbated with aging. Neurobiol Aging, 16(5), 723-35.

15. Bates, D. V. (1995). The effects of air pollution on children. Environ Health Perspect , 103 Suppl 6, 49-53.

16. Baur, X. (1995). Hypersensitivity pneumonitis (extrinsic allergic alveolitis) induced by isocyanates. J

Allergy Clin Immunol, 95(5 Pt 1), 1004-10.

17. Bellanti, J. A. (1974). The susceptibility of the fetus and child to chemical pollutants. Immunologic responses to chemical pollutants. Pediatrics, 53(5), 818-9.

18. Bernier, J., Brousseau, P., Krzystyniak, K., Tryphonas, H. , & Fournier, M. (1995). Immunotoxicity of heavy metals in relation to Great Lakes . Environ Health Perspect, 103 Suppl 9, 23-34.

19. Bernstein, M. E. (1984). Agents affecting the male reproductive system: effects of structure on activity. Drug Metab Rev, 15(5-6), 941-96.

20. Bigsby, R., Chapin, R. E., Daston, G. P., Davis, B. J., Gorski, J., Gray, L. E., Howdeshell, K. L., Zoeller, R. T., & vom Saal, F. S. (1999). Evaluating the effects of endocrine disruptors on endocrine function during development . Environ Health Perspect, 107 Suppl 4, 613-8.

21. Bishop, J. B., Morris, R. W., Seely, J. C., Hughes, L. A., Cain, K. T., & Generoso, W. M. (1997). Alterations in the reproductive patterns of female mice exposed to xenobiotics. Fundam Appl Toxicol, 40(2), 191-204.

22. Blanck, H. M., Marcus, M., Tolbert, P. E., Rubin, C., Henderson, A. K., Hertzberg, V. S., Zhang, R. H., & Cameron, L. (2000). Age at menarche and tanner stage in girls exposed in utero and postnatally to polybrominated biphenyl. Epidemiology, 11(6), 641-7.

23. Blyler, G., Landreth, K. S., & Barnett, J. B. (1994). Gender-specific effects of prenatal chlordane exposure on myeloid cell development. Fundam Appl Toxicol, 23(2), 188-93.

24. Boyd E. (1962). Biological Handbooks: Growth including reproduction and morphological development (pp. 346-358). Washington: Federation of American Societies for Experimental Biology.

25. Boyd, M. R., Statham, C. N., & Longo, N. S. (1980). The pulmonary clara cell as a target for toxic chemicals requiring metabolic activation; studies with carbon tetrachloride. J Pharmacol Exp Ther, 212(1), 109-14.

26. Brechner, R. J., Parkhurst, G. D., Humble, W. O., Brown, M. B., & Herman, W. H. (2000). Ammonium perchlorate contamination of Colorado River drinking water is associated with abnormal thyroid function in newborns in Arizona. J Occup Environ Med, 42(8), 777-82.

27. Brouwer, A., Longnecker, M. P., Birnbaum, L. S., Cogliano, J., Kostyniak, P., Moore, J., Schantz, S., & Winneke, G. (1999). Characterization of potential endocrine-related health effects at low-dose levels of exposure to PCBs. Environ Health Perspect, 107 Suppl 4, 639-49.

28. Brouwer, A., Morse, D. C., Lans, M. C., Schuur, A. G., Murk, A. J., Klasson-Wehler, E., Bergman, A., & Visser, T. J. (1998). Interactions of persistent environmental organohalogens with the thyroid hormone system: mechanisms and possible consequences for animal and human health. Toxicol Ind Health, 14(1-2), 59-84.

29. Brucker-Davis, F. (1998). Effects of environmental synthetic chemicals on thyroid function. Thyroid, 8(9), 827-56.

30. Budinger, L. , & Hertl, M. (2000). Immunologic mechanisms in hypersensitivity reactions to metal ions: an overview. Allergy, 55(2), 108-15.

31. Budreau, C. H., & Singh, R. P. (1973). Effect of fenthion and dimethoate on reproduction in the mouse.

Toxicol Appl Pharmacol, 26(1), 29-38.

32. Burbacher, T. M., Rodier, P. M., & Weiss, B. (1990). Methylmercury developmental neurotoxicity: a comparison of effects in humans and animals. Neurotoxicol Teratol, 12(3), 191-202.

33. Byskov, A. G. (1974). Does the rete ovarii act as a trigger for the onset of meiosis? Nature, 252(5482), 396-7.

34. CA Air Resources Board. (1998). The Report on Diesel Exhaust. http://www.arb.ca.gov/toxics/dieseltac/de-fnds.htm: California Environmental Protection Agency.

35. Cai, S. X., & Bao, Y. S. (1981). Placental transfer, secretion into mother milk of carbon disulphide and the effects on maternal function of female viscose rayon workers. Ind Health, 19(1), 15-29.

36. Cannon, S. B., Veazey, J. M. Jr, Jackson, R. S., Burse, V. W., Hayes, C., Straub, W. E., Landrigan, P. J., & Liddle, J. A. (1978). Epidemic kepone poisoning in chemical workers. Am J Epidemiol, 107(6), 529-37.

37. Carthew, P., Edwards, R. E., Nolan, B. M., Martin, E. A., Heydon, R. T., White, I. N., & Tucker, M. J. (2000). Tamoxifen induces endometrial and vaginal cancer in rats in the absence of endometrial hyperplasia. Carcinogenesis, 21(4), 793-7.

38. Cawte, J. (1985). Psychiatric sequelae of manganese exposure in the adult, foetal and neonatal nervous systems. Aust N Z J Psychiatry, 19(3), 211-7.

39. CDHS. (2000). California county asthma hospitalizations chart book. Environmental Health Investigations Branch.

40. Chakraborti, T. K., Farrar, J. D., & Pope, C. N. (1993). Comparative neurochemical and neurobehavioral effects of repeated chlorpyrifos exposures in young and adult rats. Pharmacol Biochem Behav, 46(1), 219-24.

41. Chanda, S. M., & Pope, C. N. (1996). Neurochemical and neurobehavioral effects of repeated gestational exposure to chlorpyrifos in maternal and developing rats. Pharmacol Biochem Behav, 53(4), 771-6.

42. Chang, L. W. , Reuhl, K. R., & Spyker, J. M. (1977). Ultrastructural study of the latent effects of methyl mercury on the nervous system after prenatal exposure. Environ Res, 13(2), 171-85.

43. Chapin, R. E., Harris, M. W., Davis, B. J., Ward, S. M., Wilson, R. E., Mauney, M. A., Lockhart, A. C., Smialowicz, R. J., Moser, V. C., Burka, L. T., & Collins, B. J. (1997). The effects of perinatal/juvenile methoxychlor exposure on adult rat nervous, immune, and reproductive system function. Fundam Appl Toxicol, 40(1), 138-57.

44. Chapin, R. E., & Ku, W. W. (1994). The reproductive toxicity of boric acid. Environ Health Perspect, 102 Suppl 7, 87-91.

45. Christian, M. S. (1996). Review of reproductive and developmental toxicity of 1,3-butadiene. Toxicology, 113(1-3), 137-43.

46. Clarkson, T. W., Nordberg, G. F., & Sager, P. R. (1985). Reproductive and developmental toxicity of metals. Scand J Work Environ Health, 11(3 Spec No), 145-54.

47. Clarren, S. K., & Smith, D. W. (1978). The fetal alcohol syndrome. N Engl J Med, 298(19), 1063-7.

48. Cocco, P. L. (1997). Environmental exposure to p,p'-DDE and human fertility. Bull Environ Contam Toxicol, 59(5), 677-80.

49. Cogliano, V. J., Hiatt, G. F., & Den, A. (1996). Quantitative cancer assessment for vinyl chloride: indications of early- life sensitivity. Toxicology, 111(1-3), 21-8.

50. Coleman, C. N., Mason, T., Hooker, E. P., & Robinson, S. E. (1999). Developmental effects of intermittent prenatal exposure to 1,1,1- trichloroethane in the rat. Neurotoxicol Teratol, 21(6), 699-708.

51. Colon, I., Caro, D., Bourdony, C. J., & Rosario, O. (2000). Identification of phthalate esters in the serum of young Puerto Rican girls with premature breast development. Environ Health Perspect, 108(9), 895-900.

52. Cooper, R. L., & Kavlock, R. J. (1997). Endocrine disruptors and reproductive development: a weight-of-evidence overview. J Endocrinol, 152(2), 159-66.

53. Copius Peereboom-Stegeman JHJ . (1989). Cadmium Effects on the Female Reproductive Tract . Toxicological and Environmental Chemistry, 23, 91-99.

54. Correa, A., Gray, R. H., Cohen, R., Rothman, N., Shah, F., Seacat, H., & Corn, M. (1996). Ethylene glycol ethers and risks of spontaneous abortion and subfertility. Am J Epidemiol, 143(7), 707-17.

55. Crump, C., Michaud, P., Tellez, R., Reyes, C., Gonzalez, G., Montgomery, E. L., Crump, K. S., Lobo, G., Becerra, C., & Gibbs, J. P. (2000). Does perchlorate in drinking water affect thyroid function in newborns or school-age children? J Occup Environ Med, 42(6), 603-12.

56. Dan, G., Lall, S. B., & Rao, D. N. (2000). Humoral and cell mediated immune response to cadmium in mice. Drug Chem Toxicol, 23(2), 349-60.

57. Daniel, V., Huber, W., Bauer, K., & Opelz, G. (1995). Impaired in-vitro lymphocyte responses in patients with elevated pentachlorophenol (PCP) blood levels. Arch Environ Health, 50(4), 287-92.

58. Daniel, V., Huber, W., Bauer, K., Suesal, C., Mytilineos, J., Melk, A., Conradt, C., & Opelz, G. (2001). Association of elevated blood levels of pentachlorophenol (PCP) with cellular and humoral immunodeficiencies. Arch Environ Health, 56(1), 77-83.

59. Daniels, J. L., Olshan, A. F., & Savitz, D. A. (1997). Pesticides and childhood cancers. Environ Health Perspect, 105(10), 1068-77.

60. Davila, D. R., Davis, D. P., Campbell, K., Cambier, J. C., Zigmond, L. A., & Burchiel, S. W. (1995). Role of alterations in Ca(2+)-associated signaling pathways in the immunotoxicity of polycyclic aromatic hydrocarbons. J Toxicol Environ Health, 45(2), 101-26.

61. Davis, B. J. , Almekinder, J. L., Flagler, N., Travlos, G., Wilson, R., & Maronpot, R. R. (1997). Ovarian luteal cell toxicity of ethylene glycol monomethyl ether and methoxy acetic acid in vivo and in vitro. Toxicol Appl Pharmacol, 142(2), 328-37.

62. Davis, B. J. , & Maronpot, R. R. (1996). Chemically associated toxicity and carcinogenicity of the ovary. Prog Clin Biol Res, 394, 285-308.

63. Davis, B. J. , Maronpot, R. R., & Heindel, J. J. (1994). Di-(2-ethylhexyl) phthalate suppresses estradiol and ovulation in cycling rats. Toxicol Appl Pharmacol, 128(2), 216-23.

64. Davis, B. J. , Price, H. C., O'Connor, R. W., Fernando, R., Rowland, A. S., & Morgan, D. L. (2001). Mercury vapor and female reproductive toxicity. Toxicol Sci, 59(2), 291-6.

65. Davis, B. J. , Travlos, G., & McShane, T. (2001). Reproductive endocrinology and toxicological pathology over the life span of the female rodent. Toxicol Pathol, 29(1), 77-83.

66. Davis, D. L. , & Ahmed, A. K. (1998). Exposures from indoor spraying of chlorpyrifos pose greater health risks to children than currently estimated. Environ Health Perspect, 106(6), 299-301.

67. Davis, J. R. , Brownson, R. C., Garcia, R., Bentz, B. J., & Turner, A. (1993). Family pesticide use and childhood brain cancer. Arch Environ Contam Toxicol, 24(1), 87-92.

68. DeRosa, C., Richter, P., Pohl, H., & Jones, D. E. (1998). Environmental exposures that affect the endocrine system: public health implications. J Toxicol Environ Health B Crit Rev, 1(1), 3-26.

69. Dhillon, S., & Von Burg, R. (1995). Methylene chloride. J Appl Toxicol, 15(4), 329-35.

70. Diel, F., Horr, B., Borck, H., Savtchenko, H., Mitsche, T., & Diel, E. (1999). Pyrethroids and piperonyl-butoxide affect human T-lymphocytes in vitro. Toxicol Lett, 107(1-3), 65-74.

71. Diwan, B. A., Anderson, L. M., & Ward, J. M. (1997). Proliferative lesions of oviduct and uterus in CD-1 mice exposed prenatally to tamoxifen. Carcinogenesis, 18(10), 2009-14.

72. Dobbing J , & Sands J. (1979). The brain growth spurt in various mammalian species. Early Human Development, (3), 79-84.

73. Drew, R. T., Boorman, G. A., Haseman, J. K., McConnell, E. E., Busey, W. M., & Moore, J. A. (1983). The effect of age and exposure duration on cancer induction by a known carcinogen in rats, mice, and hamsters. Toxicol Appl Pharmacol, 68(1), 120-30.

74. Drinkwater, N. R., Miller, E. C., Miller, J. A., & Pitot, H. C. (1976). Hepatocarcinogenicity of estragole (1-allyl-4-methoxybenzene) and 1'- hydroxyestragole in the mouse and mutagenicity of 1'-acetoxyestragole in bacteria. J Natl Cancer Inst, 57(6), 1323-31.

75. Ema, M., Miyawaki, E., & Kawashima, K. (1999). Developmental toxicity of triphenyltin chloride after administration on three consecutive days during organogenesis in rats. Bull Environ Contam Toxicol, 62(3), 363-70.

76. Eriksson, P. (1997). Developmental neurotoxicity of environmental agents in the neonate. Neurotoxicology, 18(3), 719-26.

77. Eriksson, P. , & Talts, U. (2000). Neonatal exposure to neurotoxic pesticides increases adult susceptibility: a review of current findings. Neurotoxicology, 21(1-2), 37-47.

78. Eskenazi, B. , Bradman, A., & Castorina, R. (1999). Exposures of children to organophosphate pesticides and their potential adverse health effects. Environ Health Perspect , 107 Suppl 3, 409-19.

79. Etzel, R. A. (2000). The "fatal four" indoor air pollutants. Pediatr Ann, 29(6), 344-50.

80. Fan, F., Wierda, D., & Rozman, K. K. (1996). Effects of 2,3,7,8-tetrachlorodibenzo-p-dioxin on humoral

and cell- mediated immunity in Sprague-Dawley rats. Toxicology, 106(1-3), 221-8.

81. Fanucchi, M. V., Buckpitt, A. R., Murphy, M. E., & Plopper, C. G. (1997). Naphthalene cytotoxicity of differentiating Clara cells in neonatal mice. Toxicol Appl Pharmacol, 144(1), 96-104.

82. Ferraz, H. B., Bertolucci, P. H., Pereira, J. S., Lima, J. G., & Andrade, L. A. (1988). Chronic exposure to the fungicide maneb may produce symptoms and signs of CNS manganese intoxication. Neurology, 38(4), 550-3.

83. Fields, S. (2001). Caution--children at play: how dangerous is CCA? Environ Health Perspect, 109(6), A262-9.

84. Figa-Talamanca, I., Traina, M. E., & Urbani, E. (2001). Occupational exposures to metals, solvents and pesticides: recent evidence on male reproductive effects and biological markers. Occup Med (Lond), 51(3), 174-88.

85. Foster, P. M., Mylchreest, E., Gaido, K. W., & Sar, M. (2001). Effects of phthalate esters on the developing reproductive tract of male rats. Hum Reprod Update, 7(3), 231-5.

86. Foster, W. G., McMahon, A., Younglai, E. V., Jarrell, J. F., & Lecavalier, P. (1995). Alterations in circulating ovarian steroids in hexachlorobenzene- exposed monkeys. Reprod Toxicol, 9(6), 541-8.

87. Foster, W. G., Pentick, J. A., McMahon, A., & Lecavalier, P. R. (1992). Ovarian toxicity of hexachlorobenzene (HCB) in the superovulated female rat. J Biochem Toxicol, 7(1), 1-4.

88. Freeman, J. H. Jr, Barone, S. Jr, & Stanton, M. E. (1994). Cognitive and neuroanatomical effects of triethyltin in developing rats: role of age of exposure. Brain Res, 634(1), 85-95.

89. Fuortes, L. (1999). Urticaria due to airborne permethrin exposure. Vet Hum Toxicol, 41(2), 92-3.

90. Galand, P., Mairesse, N., Degraef, C., & Rooryck, J. (1987). o,p'-DDT (1,1,1-trichloro-2(p-chlorophenyl) 2-(o-chlorophenyl) ethane is a purely estrogenic agonist in the rat uterus in vivo and in vitro. Biochem Pharmacol, 36(3), 397-400.

91. Gellert, R. J. (1978). Kepone, mirex, dieldrin, and aldrin: estrogenic activity and the induction of persistent vaginal estrus and anovulation in rats following neonatal treatment. Environ Res, 16(1-3), 131-8.

92. Gellert, R. J., Wallace, C. A., Wiesmeier, E. M., & Shuman, R. M. (1978). Topical exposure of neonates to hexachlorophene: long-standing effects on mating behavior and prostatic development in rats. Toxicol Appl Pharmacol, 43(2), 339-49.

93. Genbacev, O. , White, T. E., Gavin, C. E., & Miller, R. K. (1993). Human trophoblast cultures: models for implantation and peri- implantation toxicology. Reprod Toxicol, 7 Suppl 1, 75-94.

94. Gerhard, I., Frick, A., Monga, B., & Runnebaum, B. (1999). Pentachlorophenol exposure in women with gynecological and endocrine dysfunction. Environ Res, 80(4), 383-8.

95. Giwercman, A., Carlsen, E., Keiding, N., & Skakkebaek, N. E. (1993). Evidence for increasing incidence of abnormalities of the human testis: a review. Environ Health Perspect, 101 Suppl 2, 65-71.

96. Gladen, B. C., Ragan, N. B., & Rogan, W. J. (2000). Pubertal growth and development and prenatal and lactational exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene. J

Pediatr, 136(4), 490-6.

97. Gladen, B. C., & Rogan, W. J. (1995). DDE and shortened duration of lactation in a northern Mexican town. Am J Public Health, 85(4), 504-8.

98. Golden, R. J., Noller, K. L., Titus-Ernstoff, L., Kaufman, R. H., Mittendorf, R., Stillman, R., & Reese, E. A. (1998). Environmental endocrine modulators and human health: an assessment of the biological evidence. Crit Rev Toxicol, 28(2), 109-227.

99. Goldey, E. S., Kehn, L. S., Lau, C., Rehnberg, G. L., & Crofton, K. M. (1995). Developmental exposure to polychlorinated biphenyls (Aroclor 1254) reduces circulating thyroid hormone concentrations and causes hearing deficits in rats. Toxicol Appl Pharmacol, 135(1), 77-88.

100. Goldey, E. S., & Taylor, D. H. (1992). Developmental neurotoxicity following premating maternal exposure to hexachlorobenzene in rats. Neurotoxicol Teratol, 14(1), 15-21.

101. Goldman, J. M., Laws, S. C., Balchak, S. K., Cooper, R. L., & Kavlock, R. J. (2000). Endocrine-disrupting chemicals: prepubertal exposures and effects on sexual maturation and thyroid activity in the female rat. A focus on the EDSTAC recommendations. Crit Rev Toxicol, 30(2), 135-96.

102. Goldman, L. R., & Koduru, S. (2000). Chemicals in the environment and developmental toxicity to children: a public health and policy perspective. Environ Health Perspect, 108 Suppl 3, 443-8.

103. Golub, M. S. (2000). Adolescent health and the environment. Environ Health Perspect , 108(4), 355-62.

104. Golub, M. S., & Domingo, J. L. (1996). What we know and what we need to know about developmental aluminum toxicity. J Toxicol Environ Health, 48(6), 585-97.

105. Good, E. E. , & Ware, G. W. (1969). Effects of insecticides on reproduction in the laboratory mouse. IV. Endrin and dieldrin. Toxicol Appl Pharmacol, 14(1), 201-3.

106. Goyer, R. (1996). Cassarett & Doull's Toxicology: The basic science of poisons (p. 705). New York: McGraw-Hill.

107. Gray, L. E. Jr, & Ostby, J. (1998). Effects of pesticides and toxic substances on behavioral and morphological reproductive development: endocrine versus nonendocrine mechanisms. Toxicol Ind Health, 14(1-2), 159-84.

108. Guo, Y. L., Hsu, P. C., Hsu, C. C., & Lambert, G. H. (2000). Semen quality after prenatal exposure to polychlorinated biphenyls and dibenzofurans. Lancet, 356(9237), 1240-1.

109. Gurney, J. G., Davis, S., Severson, R. K., Fang, J. Y., Ross, J. A., & Robison, L. L. (1996). Trends in cancer incidence among children in the U.S. Cancer, 78(3), 532-41.

110. Hales, B. F., Smith, S., & Robaire, B. (1986). Cyclophosphamide in the seminal fluid of treated males: transmission to females by mating and effect on pregnancy outcome. Toxicol Appl Pharmacol, 84(3), 423-30.

111. Hales BF, & Robaire B. (1996). Paternally mediated effects on development. Handbook of Developmental Toxicology (pp. 91-107). Boca Raton, FL: CRC Press.

112. Handzel, Z. T. (2000). Effects of environmental pollutants on airways, allergic inflammation, and the immune response. Rev Environ Health, 15(3), 325-36.

113. Harris, S. J., Cecil, H. C., & Bitman, J. (1974). Effect of several dietary levels of technical methoxychlor on reproduction in rats. J Agric Food Chem, 22(6), 969-73.

114. Hartwell. TD, Pellizzari, E., Perritt, R., Whitmore, R., Zelon, H., Sheldon, L., & Sparacino, C. (1987). Results from the Toatoal Exposure Assessment Methodology (TEAM) study in selected communities in Northern and Southern California. Atmospheric Environment, 21(9), 1995-2004.

115. Hauser, P., McMillin, J. M., & Bhatara, V. S. (1998). Resistance to thyroid hormone: implications for neurodevelopmental research on the effects of thyroid hormone disruptors. Toxicol Ind Health, 14(1-2), 85-101.

116. Holladay, S. D. (1999). Prenatal immunotoxicant exposure and postnatal autoimmune disease. Environ Health Perspect, 107 Suppl 5 , 687-91.

117. Holladay, S. D., & Smialowicz, R. J. (2000). Development of the murine and human immune system: differential effects of immunotoxicants depend on time of exposure. Environ Health Perspect, 108 Suppl 3, 463-73.

118. Horner, P. J., & Gage, F. H. (2000). Regenerating the damaged central nervous system. Nature, 407(6807), 963-70.

119. Hosokawa, S., Murakami, M., Ineyama, M., Yamada, T., Koyama, Y., Okuno, Y., Yoshitake, A., Yamada, H., & Miyamoto, J. (1993). Effects of procymidone on reproductive organs and serum gonadotropins in male rats. J Toxicol Sci, 18(2), 111-24.

120. Houchens, D. P., Ovejera, A. A., & Niemeier, R. W. (1984). Effects of ethylene glycol monomethyl (EGME) and monoethyl (EGEE) ethers on the immunocompetence of allogeneic and syngeneic mice bearing L1210 mouse leukemia. Environ Health Perspect, 57, 113-8.

121. Hoyer, P. B., & Sipes, I. G. (1996). Assessment of follicle destruction in chemical-induced ovarian toxicity. Annu Rev Pharmacol Toxicol, 36, 307-31.

122. Huisman, M. , Koopman-Esseboom, C., Fidler, V., Hadders-Algra, M., van der Paauw, C. G., Tuinstra, L. G., Weisglas-Kuperus, N., Sauer, P. J., Touwen, B. C., & Boersma, E. R. (1995). Perinatal exposure to polychlorinated biphenyls and dioxins and its effect on neonatal neurological development. Early Hum Dev, 41(2), 111-27.

123. IARC (International Agency for Research on Cancer). (1996). IARC monographs on the evaluation of carcinogenic risks to humans. V. 66. Some pharmaceutical drugs. pp. 253-365. Lyon, France: IARC.

124. Ihrig, M. M., Shalat, S. L., & Baynes, C. (1998). A hospital-based case-control study of stillbirths and environmental exposure to arsenic using an atmospheric dispersion model linked to a geographical information system. Epidemiology, 9(3), 290-4.

125. Ilback, N. G., Sundberg, J., & Oskarsson, A. (1991). Methyl mercury exposure via placenta and milk impairs natural killer (NK) cell function in newborn rats. Toxicol Lett, 58(2), 149-58.

126. Imajima, T. , Shono, T., Zakaria, O., & Suita, S. (1997). Prenatal phthalate causes cryptorchidism postnatally by inducing transabdominal ascent of the testis in fetal rats. J Pediatr Surg, 32(1), 18-21.

127. Ivankovic, S. (1979). Teratogenic and carcinogenic effects of some chemicals during perinatal life in

rats, Syrian golden hamsters, and minipigs. Natl Cancer Inst Monogr, (51), 103-15.

128. Jacobs BL , van Praag H , & Gage FH. (2000). Depression and the birth and death of brain cells. American Scientist, 88, 340-345.

129. Jacobson, J. L., & Jacobson, S. W. (1996). Intellectual impairment in children exposed to polychlorinated biphenyls in utero. N Engl J Med, 335(11), 783-9.

130. Jegaden, D. , Amann, D., Simon, J. F., Habault, M., Legoux, B., & Galopin, P. (1993). Study of the neurobehavioural toxicity of styrene at low levels of exposure. Int Arch Occup Environ Health, 64(7), 527-31.

131. Jones, H. E., Kunko, P. M., Robinson, S. E., & Balster, R. L. (1996). Developmental consequences of intermittent and continuous prenatal exposure to 1,1,1-trichloroethane in mice. Pharmacol Biochem Behav , 55(4), 635-46.

132. Kavlock, R. J., Daston, G. P., DeRosa, C., Fenner-Crisp, P., Gray, L. E., Kaattari, S., Lucier, G., Luster, M., Mac, M. J., Maczka, C., Miller, R., Moore, J., Rolland, R., Scott, G., Sheehan, D. M., Sinks, T., & Tilson, H. A. (1996). Research needs for the risk assessment of health and environmental effects of endocrine disruptors: a report of the US EPA-sponsored workshop. Environ Health Perspect, 104 Suppl 4, 715-40.

133. Kayama, F., Yamashita, U., Kawamoto, T., & Kodama, Y. (1991). Selective depletion of immature thymocytes by oral administration of ethylene glycol monomethyl ether. Int J Immunopharmacol, 13(5), 531-40.

134. Kaye, A. M., & Trainin, N. (1966). Urethan carcinogenesis and nucleic acid metabolism: factors influencing lung adenoma induction. Cancer Res, 26(10), 2206-12.

135. Kimmel CA , Kavlock RJ , & Francis EZ. (1992). Animal models for assessing developmental toxicity. Similarities and differences between children and adults: Implications for risk assessment (Guzelian PS // Henry CJ // Olin SS, ed., p. 43). Washington DC: ILSI Press.

136. Klaassen, ed. (1996). Cassarett & Doull's Toxicology: The basic science of poisons. (Fifth ed.,). New York: McGraw Hill.

137. Krasovskii, G. N., Varshavskaya, S. P., & Borisov, A. I. (1976). Toxic and gonadotropic effects of cadmium and boron relative to standards for these substances in drinking water. Environ Health Perspect, 13, 69-75.

138. Lafuente, A., Blanco, A., Marquez, N., Alvarez-Demanuel, E., & Esquifino, A. I. (1997). Effects of acute and subchronic cadmium administration on pituitary hormone secretion in rat. Rev Esp Fisiol, 53(3), 265-9.

139. Lafuente, A., Marquez, N., Perez-Lorenzo, M., Pazo, D., & Esquifino, A. I. (2000). Pubertal and postpubertal cadmium exposure differentially affects the hypothalamic-pituitary-testicular axis function in the rat. Food Chem Toxicol, 38(10), 913-23.

140. Lai, Z. W., Fiore, N. C., Hahn, P. J., Gasiewicz, T. A., & Silverstone, A. E. (2000). Differential effects of diethylstilbestrol and 2,3,7,8- tetrachlorodibenzo-p-dioxin on thymocyte differentiation, proliferation, and apoptosis in bcl-2 transgenic mouse fetal thymus organ culture. Toxicol Appl Pharmacol, 168(1), 15-24.

141. Lambright, C., Ostby, J., Bobseine, K., Wilson, V., Hotchkiss, A. K., Mann, P. C., & Gray, L. E. Jr.

(2000). Cellular and molecular mechanisms of action of linuron: an antiandrogenic herbicide that produces reproductive malformations in male rats. Toxicol Sci, 56(2), 389-99.

142. Lash, T. L., & Aschengrau, A. (1999). Active and passive cigarette smoking and the occurrence of breast cancer. Am J Epidemiol, 149(1), 5-12.

143. Lee, I. P., Sherins, R. J., & Dixon, R. L. (1978). Evidence for induction of germinal aplasia in male rats by environmental exposure to boron. Toxicol Appl Pharmacol, 45(2), 577-90.

144. Lemasters, G. K., Perreault, S. D., Hales, B. F., Hatch, M., Hirshfield, A. N., Hughes, C. L., Kimmel, G. L., Lamb, J. C., Pryor, J. L., Rubin, C., & Seed, J. G. (2000). Workshop to identify critical windows of exposure for children's health: reproductive health in children and adolescents work group summary. Environ Health Perspect , 108 Suppl 3, 505-9.

145. Lemus, R., & Abdelghani, A. (2000). Chlorpyrifos: an unwelcome pesticide in our homes. Rev Environ Health, 15(4), 421-33.

146. Leonard, A. , & Jacquet, P. (1984). Embryotoxicity and genotoxicity of nickel. IARC Sci Publ, (53), 277-91.

147. Lesser, S. H., & Weiss, S. J. (1995). Art hazards. Am J Emerg Med, 13(4), 451-8.

148. Lindbohm, M. L., Taskinen, H., Sallmen, M., & Hemminki, K. (1990). Spontaneous abortions among women exposed to organic solvents. Am J Ind Med, 17(4), 449-63.

149. Little, P. J., Adams, M. L., & Cicero, T. J. (1992). Effects of alcohol on the hypothalamic-pituitary-gonadal axis in the developing male rat. J Pharmacol Exp Ther, 263(3), 1056-61.

150. Luster, M. I., Ackermann, M. F., Germolec, D. R., & Rosenthal, G. J. (1989). Perturbations of the immune system by xenobiotics. Environ Health Perspect, 81, 157-62.

151. Luster, M. I., Germolec, D. R., & Rosenthal, G. J. (1990). Immunotoxicology: review of current status. Ann Allergy, 64(5), 427-32.

152. Maekawa, A., & Mitsumori, K. (1990). Spontaneous occurrence and chemical induction of neurogenic tumors in rats--influence of host factors and specificity of chemical structure. Crit Rev Toxicol, 20(4), 287-310.

153. Magers, T., Talbot, P., DiCarlantonio, G., Knoll, M., Demers, D., Tsai, I., & Hoodbhoy, T. (1995). Cigarette smoke inhalation affects the reproductive system of female hamsters. Reprod Toxicol, 9(6), 513-25.

154. Maltoni, C., Lefemine, G., Ciliberti, A., Cotti, G., & Carretti, D. (1981). Carcinogenicity bioassays of vinyl chloride monomer: a model of risk assessment on an experimental basis. Environ Health Perspect, 41, 3-29.

155. Mannino, D. M., Homa, D. M., Pertowski, C. A., Ashizawa, A., Nixon, L. L., Johnson, C. A., Ball, L. B., Jack, E., & Kang, D. S. (1998). Surveillance for asthma--United States, 1960-1995. Mor Mortal Wkly Rep CDC Surveill Summ, 47(1), 1-27.

156. Marcus, P. M., Newman, B., Millikan, R. C., Moorman, P. G., Baird, D. D., & Qaqish, B. (2000). The associations of adolescent cigarette smoking, alcoholic beverage consumption, environmental tobacco smoke, and ionizing radiation with subsequent breast cancer risk (United States). Cancer Causes Control, 11(3), 271-8.

157. Marlowe, M. (1986). Exposure to metal pollutants and behavioral disorders in children: a review of the evidence. Rev Environ Health, 6(1-4), 85-117.

158. Mattison, D. R., Nightingale, M. S., & Shiromizu, K. (1983). Effects of toxic substances on female reproduction. Environ Health Perspect, 48, 43-52.

159. Mattison, D. R., Plowchalk, D. R., Meadows, M. J., al-Juburi, A. Z., Gandy, J., & Malek, A. (1990). Reproductive toxicity: male and female reproductive systems as targets for chemical injury. Med Clin North Am , 74(2), 391-411.

160. Mattison, D. R., Shiromizu, K., & Nightingale, M. S. (1983). Oocyte destruction by polycyclic aromatic hydrocarbons. Am J Ind Med, 4(1-2), 191-202.

161. May, M. (2000). Disturbing behavior: neurotoxic effects in children. Environ Health Perspect, 108(6), A262-7.

162. McConnachie, P. R., & Zahalsky, A. C. (1991). Immunological consequences of exposure to pentachlorophenol. Arch Environ Health , 46(4), 249-53.

163. McIntyre, B. S., Barlow, N. J., Wallace, D. G., Maness, S. C., Gaido, K. W., & Foster, P. M. (2000). Effects of in utero exposure to linuron on androgen-dependent reproductive development in the male Crl:CD(SD)BR rat. Toxicol Appl Pharmacol, 167(2), 87-99.

164. Miller, R. W. (1995). Special susceptibility of the child to certain radiation-induced cancers. Environ Health Perspect, 103 Suppl 6, 41-4.

165. Morabia, A., Bernstein, M. S., Bouchardy, I., Kurtz, J., & Morris, M. A. (2000). Breast cancer and active and passive smoking: the role of the N- acetyltransferase 2 genotype. Am J Epidemiol, 152(3), 226-32.

166. Muller, J., & Skakkebaek, N. E. (1992). The prenatal and postnatal development of the testis. Baillieres Clin Endocrinol Metab, 6(2), 251-71.

167. Muttray, A. , Klimek, L., Faas, M., Schafer, D., Mann, W., & Konietzko, J. (1999). The exposure of healthy volunteers to 200 ppm 1,1,1-trichloroethane increases the concentration of proinflammatory cytokines in nasal secretions. Int Arch Occup Environ Health, 72(7), 485-8.

168. Nagaraja, T. N., & Desiraju, T. (1994). Effects on operant learning and brain acetylcholine esterase activity in rats following chronic inorganic arsenic intake. Hum Exp Toxicol, 13(5), 353-6.

169. Nagayama, J., Tsuji, H., Iida, T., Hirakawa, H., Matsueda, T., Okamura, K., Hasegawa, M., Sato, K., Ma, H. Y., Yanagawa, T., Igarashi, H., Fukushige, J., & Watanabe, T. (1998). Postnatal exposure to chlorinated dioxins and related chemicals on lymphocyte subsets in Japanese breast-fed infants. Chemosphere, 37(9-12), 1781-7.

170. NCI. (1999). Health effects of exposure to environmental tobacco smoke: The report of the California Environmental Protection Agency. Smoking and tobacco control monograph number 10. (Report No. 99-4645). Methesda, MD: US Department of Health and Human Services, National Institutes of Health, NIH.

171. Needleman, H. L., Schell, A., Bellinger, D., Leviton, A., & Allred, E. N. (1990). The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. N Engl J Med, 322(2), 83-8.

172. Newbold, R. R., Jefferson, W. N., Padilla-Burgos, E., & Bullock, B. C. (1997). Uterine carcinoma in mice treated neonatally with tamoxifen. Carcinogenesis, 18(12), 2293-8.

173. Nicolopoulou-Stamati, P., & Pitsos, M. A. (2001). The impact of endocrine disrupters on the female reproductive system. Hum Reprod Update, 7(3), 323-30.

174. NRC (National Research Council). (1990). Health effects of exposure to low levels of ionizing radiation. BEIR V, Committee on the biological effects of ionizing radiation. Washington, DC: National Academy Press.

175. Oie, L., Hersoug, L. G., & Madsen, J. O. (1997). Residential exposure to plasticizers and its possible role in the pathogenesis of asthma . Environ Health Perspect, 105(9), 972-8.

176. Olea, N., Pazos, P., & Exposito, J. (1998). Inadvertent exposure to xenoestrogens. Eur J Cancer Prev, 7 Suppl 1, S17-23.

177. Oliver, R. T. (1996). Testicular cancer. Curr Opin Oncol, 8(3), 252-8.

178. Olney, J. W., Farber, N. B., Wozniak, D. F., Jevtovic-Todorovic, V., & Ikonomidou, C. (2000). Environmental agents that have the potential to trigger massive apoptotic neurodegeneration in the developing brain. Environ Health Perspect, 108 Suppl 3, 383-8.

179. Osius, N., Karmaus, W., Kruse, H., & Witten, J. (1999). Exposure to polychlorinated biphenyls and levels of thyroid hormones in children. Environ Health Perspect, 107(10), 843-9.

180. Ottoboni, A. (1972). Effect of DDT on the reproductive life-span in the female rat. Toxicol Appl Pharmacol, 22(3), 497-502.

181. Paige, R., Wong, V., & Plopper, C. (1997). Dose-related airway-selective epithelial toxicity of 1-nitronaphthalene in rats. Toxicol Appl Pharmacol, 147(2), 224-33.

182. Paksy, K., Rajczy, K., Forgacs, Z., Lazar, P., Bernard, A., Gati, I., & Kaali, G. S. (1997). Effect of cadmium on morphology and steroidogenesis of cultured human ovarian granulosa cells. J Appl Toxicol, 17(5), 321-7.

183. Palanza, P. , Morellini, F., Parmigiani, S., & vom Saal, F. S. (1999). Prenatal exposure to endocrine disrupting chemicals: effects on behavioral development. Neurosci Biobehav Rev, 23(7), 1011-27.

184. Paul, M., & Himmelstein, J. (1988). Reproductive hazards in the workplace: what the practitioner needs to know about chemical exposures. Obstet Gynecol, 71(6 Pt 1), 921-38.

185. Paus, T., Zijdenbos, A., Worsley, K., Collins, D. L., Blumenthal, J., Giedd, J. N., Rapoport, J. L., & Evans, A. C. (1999). Structural maturation of neural pathways in children and adolescents: in vivo study. Science, 283(5409), 1908-11.

186. Perlingeiro, R. C., & Queiroz, M. L. (1994). Polymorphonuclear phagocytosis and killing in workers exposed to inorganic mercury. Int J Immunopharmacol, 16(12), 1011-7.

187. Peters, H., & McNatty, K. (1980). The Ovary. New York: Paul Elek.

188. Pherwani, A. V., Khanna, S. A., & Patel, R. B. (1989). Effect of chlorine gas leak on the pulmonary functions of school children. Indian J Pediatr, 56(1), 125-8.

189. Pinkerton, K. E., & Joad, J. P. (2000). The mammalian respiratory system and critical windows of exposure for children's health. Environ Health Perspect, 108 Suppl 3, 457-62.

190. Plenge-Bonig, A., & Karmaus, W. (1999). Exposure to toluene in the printing industry is associated with subfecundity in women but not in men. Occup Environ Med, 56(7), 443-8.

191. Plopper, C. G., Weir, A. J., Morin, D., Chang, A., Philpot, R. M., & Buckpitt, A. R. (1993). Postnatal changes in the expression and distribution of pulmonary cytochrome P450 monooxygenases during Clara cell differentiation in rabbits. Mol Pharmacol, 44(1), 51-61.

192. Plopper, C. G., Weir, A. J., Nishio, S. J., Chang, A., Voit, M., Philpot, R. M., & Buckpitt, A. R. (1994). Elevated susceptibility to 4-ipomeanol cytotoxicity in immature Clara cells of neonatal rabbits. J Pharmacol Exp Ther, 269(2), 867-80.

193. Porterfield, S. P. (2000). Thyroidal dysfunction and environmental chemicals--potential impact on brain development. Environ Health Perspect, 108 Suppl 3, 433-8.

194. Porterfield, S. P., & Hendrich, C. E. (1993). The role of thyroid hormones in prenatal and neonatal neurological development--current perspectives. Endocr Rev, 14(1), 94-106.

195. Powell, J. J., Van De Water, J., & Gershwin, M. E. (1999). Evidence for the Role of Environmental Agents in the Initiation or Progression of Autoimmune Conditions. Environ Health Perspect, 107(Suppl 5), 667-672.

196. Preston-Martin. (1989). Epidemiological studies of perinatal carcinogenesis. Napalkov NP, Rice JM, Tomatis L, & Yamasaki H (eds.) Perinatal and multigenerational carcinogenesis . Lyon, France: International Agency for Research on Cancer.

197. Pryor, J. L., Hughes, C., Foster, W., Hales, B. F., & Robaire, B. (2000). Critical windows of exposure for children's health: the reproductive system in animals and humans. Environ Health Perspect, 108 Suppl 3, 491-503.

198. Punareewattana, K., Smith, B. J., Blaylock, B. L., Longstreth, J., Snodgrass, H. L., Gogal, R. M. Jr, Prater, R. M., & Holladay, S. D. (2001). Topical permethrin exposure inhibits antibody production and macrophage function in C57Bl/6N mice. Food Chem Toxicol, 39(2), 133-9.

199. Rabovsky, J., Fowles, J., Hill, M. D., & Lewis, D. C. (2001). A health risk benchmark for the neurologic effects of styrene: comparison with NOAEL/LOAEL approach. Risk Anal, 21(1), 117-26.

200. Rani, B. E. , & Krishnakumari, M. K. (1995). Prenatal toxicity of heptachlor in albino rats. Pharmacol Toxicol, 76(2), 112-4.

201. Rao, K. S., & Schwetz, B. A. (1982). Reproductive toxicity of environmental agents. Annu Rev Public Health, 3, 1-27.

202. Raulf-Heimsoth, M., & Baur, X. (1998). Pathomechanisms and pathophysiology of isocyanate-induced diseases-- summary of present knowledge. Am J Ind Med, 34(2), 137-43.

203. Reigart, J. R. (1995). Pesticides and children. Pediatr Ann, 24(12), 663-8.

204. Reis LAG, Smith MA, Gurney JG, Linet M, Tamra T, Young JL, & Bunin GR (eds.). (1999). Cancer incidence and survival among children and adolescents: United States SEER Program 1975-1995. (Report No. NIH Publication 99-4649). Bethesda, MD: National Cancer Institute.

205. Rice, D., & Barone, S. Jr. (2000). Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. Environ Health Perspect, 108 Suppl 3, 511-33.

206. Rice, D. C. (1996). Evidence for delayed neurotoxicity produced by methylmercury. Neurotoxicology, 17(3-4), 583-96.

207. Rice, D. C. (2000). Parallels between attention deficit hyperactivity disorder and behavioral deficits produced by neurotoxic exposure in monkeys. Environ Health Perspect, 108 Suppl 3, 405-8.

208. Riihimaki, V., & Savolainen, K. (1980). Human exposure to m-xylene. Kinetics and acute effects on the central nervous system. Ann Occup Hyg, 23(4), 411-22.

209. Ritz, B., Heinrich, J., Wjst, M., Wichmann, E., & Krause, C. (1998). Effect of cadmium body burden on immune response of school children. Arch Environ Health, 53(4), 272-80.

210. Robledo, R. F., Young, R. S., Lantz, R. C., & Witten, M. L. (2000). Short-term pulmonary response to inhaled JP-8 jet fuel aerosol in mice. Toxicol Pathol, 28(5), 656-63.

211. Rodier, P. M. (1995). Developing brain as a target of toxicity. Environ Health Perspect, 103 Suppl 6, 73-6.

212. Rodier, P. M. (1994). Vulnerable periods and processes during central nervous system development. Environ Health Perspect, 102 Suppl 2, 121-4.

213. Rodier, P. M., Ingram, J. L., Tisdale, B., & Croog, V. J. (1997). Linking etiologies in humans and animal models: studies of autism. Reprod Toxicol, 11(2-3), 417-22.

214. Rogan, W. J. (1995). Environmental poisoning of children--lessons from the past. Environ Health Perspect, 103 Suppl 6, 19-23.

215. Rogan, W. J., Gladen, B. C., McKinney, J. D., Carreras, N., Hardy, P., Thullen, J., Tingelstad, J., & Tully, M. (1987). Polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethene (DDE) in human milk: effects on growth, morbidity, and duration of lactation. Am J Public Health, 77(10), 1294-7.

216. Rogers S. (1951). Age of the host and other factors affecting the production with Urethane of pulmonary adenomas in mice. J. Exp. Med., 93, 427-449.

217. Rubin, C. H., & Niskar, A. S. (1999). Endocrine disrupters: an emerging environmental health problem. J Med Assoc Ga , 88(4), 27-30.

218. Sala, M., Sunyer, J., Herrero, C., To-Figueras, J., & Grimalt, J. (2001). Association between serum concentrations of hexachlorobenzene and polychlorobiphenyls with thyroid hormone and liver enzymes in a sample of the general population. Occup Environ Med, 58(3), 172-7.

219. Sallmen, M. , Lindbohm, M. L., Kyyronen, P., Nykyri, E., Anttila, A., Taskinen, H., & Hemminki, K. (1995). Reduced fertility among women exposed to organic solvents. Am J Ind Med, 27(5), 699-713.

220. Salmon AG, & Zeise L (eds.). (1991). Risks of carcinogenesis from Urethane exposure. Boca Raton: CRC Presss, Inc.

221. Schenker, M. B., Gold, E. B., Beaumont, J. J., Eskenazi, B., Hammond, S. K., Lasley, B. L., McCurdy, S. A., Samuels, S. J., Saiki, C. L., & Swan, S. H. (1995). Association of spontaneous abortion

and other reproductive effects with work in the semiconductor industry. Am J Ind Med, 28(6), 639-59.

222. Schneider, D., & Freeman, N. (2001). Children's environmental health risks: a state-of-the-art conference. Arch Environ Health, 56(2), 103-10.

223. Schrag, S. D., & Dixon, R. L. (1985). Occupational exposures associated with male reproductive dysfunction. Annu Rev Pharmacol Toxicol, 25, 567-92.

224. Schroeder, S. R. (2000). Mental retardation and developmental disabilities influenced by environmental neurotoxic insults. Environ Health Perspect, 108 Suppl 3, 395-9.

225. Selevan, S. G., Kimmel, C. A., & Mendola, P. (2000). Identifying critical windows of exposure for children's health. Environ Health Perspect, 108 Suppl 3, 451-5.

226. Sever LE , & Hessol NA. (1985). Toxic effects of occupational and environmental chemicals on the testes. Endocrine Toxicology (p. 211). New York: Raven Press.

227. Sharpe, R. M. (2001). Hormones and testis development and the possible adverse effects of environmental chemicals. Toxicol Lett, 120(1-3), 221-32.

228. Shtenberg, A. I., & Rybakova, M. N. (1968). Effect of carbaryl on the neuroendocrine system of rats. Food Cosmet Toxicol, 6(4), 461-7.

229. Silvaggio T , & Mattison DR. (1993). Comparative approach to toxicokinetics. Occupational and environmental reproductive hazards: A guide for clinicians (Paul M. ed., pp. 25-36). Baltimore: Williams and Wilkins.

230. Silvestroni, L., & Palleschi, S. (1999). Effects of organochlorine xenobiotics on human spermatozoa. Chemosphere, 39(8), 1249-52.

231. Slotkin, T. A., Cousins, M. M., Tate, C. A., & Seidler, F. J. (2001). Persistent cholinergic presynaptic deficits after neonatal chlorpyrifos exposure. Brain Res, 902(2), 229-43.

232. Smialowicz, R. J., Riddle, M. M., Luebke, R. W., Copeland, C. B., Andrews, D., Rogers, R. R., Gray, L. E., & Laskey, J. W. (1991). Immunotoxicity of 2-methoxyethanol following oral administration in Fischer 344 rats. Toxicol Appl Pharmacol, 109(3), 494-506.

233. Smialowicz, R. J., Riddle, M. M., & Williams, W. C. (1994). Species and strain comparisons of immunosuppression by 2-methoxyethanol and 2-methoxyacetic acid. Int J Immunopharmacol, 16(8), 695-702.

234. Smialowicz, R. J., Riddle, M. M., Williams, W. C., Copeland, C. B., Luebke, R. W., & Andrews, D. L. (1992). Differences between rats and mice in the immunosuppressive activity of 2-methoxyethanol and 2-methoxyacetic acid. Toxicology, 74(1), 57-67.

235. Smialowicz, R. J., Rogers, R. R., Riddle, M. M., Luebke, R. W., Rowe, D. G., & Garner, R. J. (1984). Manganese chloride enhances murine cell-mediated cytotoxicity: effects on natural killer cells. J Immunopharmacol, 6(1-2), 1-23.

236. Smialowicz, R. J., Williams, W. C., Copeland, C. B., Harris, M. W., Overstreet, D., Davis, B. J., & Chapin, R. E. (2001). The effects of perinatal/juvenile heptachlor exposure on adult immune and reproductive system function in rats. Toxicol Sci, 61(1), 164-75.

237. Smith, D. A., & Germolec, D. R. (1999). Introduction to immunology and autoimmunity. Environ Health Perspect, 107 Suppl 5, 661-5.

238. Snodgrass WR. (1992). Physiological and biochemical differences between children and adults as determinants of toxic response to environmental pollutants. Similarities and differences between children and adults: Implications for risk assessment (Guzelian PS//Henry CJ//Olin SS ed., pp. 35-42). Washington, DC: ILSI Press.

239. Soyka, L. F., Peterson, J. M., & Joffe, J. M. (1978). Lethal and sublethal effects on the progeny of male rats treated with methadone. Toxicol Appl Pharmacol, 45(3), 797-807.

240. Spyker-Cranmer, J. M., Barnett, J. B., Avery, D. L., & Cranmer, M. F. (1982). Immunoteratology of chlordane: cell-mediated and humoral immune responses in adult mice exposed in utero. Toxicol Appl Pharmacol, 62(3), 402-8.

241. Spyker, J. M., & Avery, D. L. (1977). Neurobehavioral effects of prenatal exposure to the organophosphate Diazinon in mice. J Toxicol Environ Health, 3(5-6), 989-1002.

242. Sram, R. J. , Binkova, B., Rossner, P., Rubes, J., Topinka, J., & Dejmek, J. (1999). Adverse reproductive outcomes from exposure to environmental mutagens. Mutat Res, 428(1-2), 203-15.

243. Steinkraus, V., & Hausen, B. M. (1994). Contact allergy to propylene oxide. Contact Dermatitis, 31(2), 120.

244. Stellman, J. M. (1979). The effects of toxic agents on reproduction. Occup Health Saf, 48(3), 36-43.

245. Stewart, L. , & Beck, M. H. (1992). Contact sensitivity to methyl ethyl ketone peroxide in a paint sprayer. Contact Dermatitis, 26(1), 52-3.

246. Stoker, T. E., Laws, S. C., Guidici, D. L., & Cooper, R. L. (2000). The effect of atrazine on puberty in male wistar rats: an evaluation in the protocol for the assessment of pubertal development and thyroid function. Toxicol Sci, 58(1), 50-9.

247. Stoker, T. E., Parks, L. G., Gray, L. E., & Cooper, R. L. (2000). Endocrine-disrupting chemicals: prepubertal exposures and effects on sexual maturation and thyroid function in the male rat. A focus on the EDSTAC recommendations. Endocrine Disrupter Screening and Testing Advisory Committee. Crit Rev Toxicol, 30(2), 197-252.

248. Sun, E. L., & Gondos, B. (1984). Squash preparation studies of germ cells in human fetal testes . J Androl, 5(5), 334-8.

249. Taneda, S., Hayashi, H., Sakata, M., Yoshino, S., Suzuki, A., Sagai, M., & Mori, Y. (2000). Anti-estrogenic activity of diesel exhaust particles. Biol Pharm Bull, 23(12), 1477-80.

250. Tang, H. W. , Huel, G., Campagna, D., Hellier, G., Boissinot, C., & Blot, P. (1999). Neurodevelopmental evaluation of 9-month-old infants exposed to low levels of lead in utero: involvement of monoamine neurotransmitters. J Appl Toxicol, 19(3), 167-72.

251. Taskinen, H. K., Kyyronen, P., Sallmen, M., Virtanen, S. V., Liukkonen, T. A., Huida, O., Lindbohm, M. L., & Anttila, A. (1999). Reduced fertility among female wood workers exposed to formaldehyde. Am J Ind Med, 36(1), 206-12.

252. Theus, S. A., Tabor, D. R., Soderberg, L. S., & Barnett, J. B. (1992). Macrophage tumoricidal mechanisms are selectively altered by prenatal chlordane exposure. Agents Actions, 37(1-2),

140-6.

253. Thrasher, J. D., Broughton, A., & Madison, R. (1990). Immune activation and autoantibodies in humans with long-term inhalation exposure to formaldehyde. Arch Environ Health, 45(4), 217-23.

254. Thurston, S. W., Ryan, L., Christiani, D. C., Snow, R., Carlson, J., You, L., Cui, S., Ma, G., Wang, L., Huang, Y., & Xu, X. (2000). Petrochemical exposure and menstrual disturbances. Am J Ind Med, 38(5), 555-64.

255. Tilson, H. A. (1998). Developmental neurotoxicology of endocrine disruptors and pesticides: identification of information gaps and research needs. Environ Health Perspect, 106 Suppl 3, 807-11.

256. Trentacoste, S. V., Friedmann, A. S., Youker, R. T., Breckenridge, C. B., & Zirkin, B. R. (2001). Atrazine effects on testosterone levels and androgen-dependent reproductive organs in peripubertal male rats. J Androl, 22(1), 142-8.

257. Tulinska, J., Dusinska, M., Jahnova, E., Liskova, A., Kuricova, M., Vodicka, P., Vodickova, L., Sulcova, M., & Fuortes, L. (2000). Changes in cellular immunity among workers occupationally exposed to styrene in a plastics lamination plant. Am J Ind Med, 38(5), 576-83.

258. US EPA. (1998). EPA's Children's Environmental Health Yearbook. Washington, DC: US EPA.

259. US EPA. (1997). EPA Special report on endocrine disruption: An effects assessment and analysis. (Report No. EPA/630/R-96/012).

260. US EPA. (1997). Exposure factors handbook. Vol. 1:General factors. (Report No. EPA/600/P-95/002Fa). Washington, DC: US Environmental Protection Agency.

261. US EPA. (1986). Environmental health threats to children. (Report No. EPA 175-F-96-001). Available: http://www.epa.gov/epadocs/child.htm.

262. US EPA. (1987). IRIS Substance File - Atrazine; CASRN 1912-24-9 . http://www.epa.gov/iris/subst/0209.htm: US EPA.

263. Veronesi, B., & Pope, C. (1990). The neurotoxicity of parathion-induced acetylcholinesterase inhibition in neonatal rats. Neurotoxicology, 11(4), 609-26.

264. Vesselinovitch, S. D., Rao, K. V., & Mihailovich, N. (1979). Transplacental and lactational carcinogenesis by safrole. Cancer Res, 39(11), 4378-80.

265. Vine, M. F. , Stein, L., Weigle, K., Schroeder, J., Degnan, D., Tse, C. K., & Backer, L. (2001). Plasma 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene (DDE) levels and immune response. Am J Epidemiol, 153(1), 53-63.

266. Voccia, I., Blakley, B., Brousseau, P., & Fournier, M. (1999). Immunotoxicity of pesticides: a review. Toxicol Ind Health, 15(1-2), 119-32.

267. Wadsworth, M. E., & Kuh, D. J. (1997). Childhood influences on adult health: a review of recent work from the British 1946 national birth cohort study, the MRC National Survey of Health and Development. Paediatr Perinat Epidemiol, 11(1), 2-20.

268. Wagner, S. L. (2000). Fatal asthma in a child after use of an animal shampoo containing pyrethrin. West J Med, 173(2), 86-7.

269. Wagner, V., Wagnerova, M., Kriz, J., Kodl, M., & Wokounova, D. (1988). Relationship of blood protein levels to outdoor air pollutant concentrations in a semicohort of school-age children living in urban areas differing by quality of air. J Hyg Epidemiol Microbiol Immunol, 32(2), 121-36.

270. Wantke, F., Demmer, C. M., Tappler, P., Gotz, M., & Jarisch, R. (1996). Exposure to gaseous formaldehyde induces IgE-mediated sensitization to formaldehyde in school-children. Clin Exp Allergy, 26(3), 276-80.

271. Watt, K. C. , & Buckpitt, A. R. (2000). Species differences in the regio- and stereoselectivity of 1-nitronaphthalene metabolism. Drug Metab Dispos, 28(4), 376-8.

272. Wax, P. M., & Hoffman, R. S. (1994). Fatality associated with inhalation of a pyrethrin shampoo. J Toxicol Clin Toxicol, 32(4), 457-60.

273. Weisglas-Kuperus, N. (1998). Neurodevelopmental, immunological and endocrinological indices of perinatal human exposure to PCBs and dioxins. Chemosphere, 37(9-12), 1845-53.

274. Weisglas-Kuperus, N., Patandin, S., Berbers, G. A., Sas, T. C., Mulder, P. G., Sauer, P. J., & Hooijkaas, H. (2000). Immunologic effects of background exposure to polychlorinated biphenyls and dioxins in Dutch preschool children. Environ Health Perspect, 108(12), 1203-7.

275. Wennberg, A. (1994). Neurotoxic effects of selected metals. Scand J Work Environ Health, 20 Spec No, 65-71.

276. White, R. F., Feldman, R. G., Eviator, I. I., Jabre, J. F., & Niles, C. A. (1997). Hazardous waste and neurobehavioral effects: a developmental perspective. Environ Res, 73(1-2), 113-24.

277. Wiseman, R. W., Miller, E. C., Miller, J. A., & Liem, A. (1987). Structure-activity studies of the hepatocarcinogenicities of alkenylbenzene derivatives related to estragole and safrole on administration to preweanling male C57BL/6J x C3H/HeJ F1 mice. Cancer Res, 47(9), 2275-83.

278. Wu, X., Faqi, A. S., Yang, J., Ding, X., Jiang, X., & Chahoud, I. (1999). Male reproductive toxicity and beta-luteinizing hormone gene expression in sexually mature and immature rats exposed to 2-bromopropane. Hum Exp Toxicol, 18(11), 683-90.

279. Xu, X., Cho, S. I., Sammel, M., You, L., Cui, S., Huang, Y., Ma, G., Padungtod, C., Pothier, L., Niu, T., Christiani, D., Smith, T., Ryan, L., & Wang, L. (1998). Association of petrochemical exposure with spontaneous abortion. Occup Environ Med, 55(1), 31-6.

280. Xuezhi, J., Youxin, L., & Yilan, W. (1992). Studies of lead exposure on reproductive system: a review of work in China. Biomed Environ Sci, 5(3), 266-75.

281. Zahm, S. H. , & Ward, M. H. (1998). Pesticides and childhood cancer. Environ Health Perspect, 106 Suppl 3, 893-908.

282. Zhao, M. Y. , Ying, C. J., Shao, N., Yang, Y., Yang, C. F., Shi, L., & Liu, W. Q. (1994). The study of health effects of vinyl chloride air pollution on population. Biomed Environ Sci, 7(2), 136-43.

283. Zhou, S. Y. , Liang, Y. X., Chen, Z. Q., & Wang, Y. L. (1988). Effects of occupational exposure to low-level carbon disulfide (CS2) on menstruation and pregnancy. Ind Health, 26(4), 203-14.

APPENDIX A
Department of Health Services' compilation of VOCs that may be emitted from building materials and products and their potential sources

California Department of Health Services

Reducing Occupant Exposure to Volatile Organic Compounds (VOCs) From Office Building Construction Materials: Non-binding Guidelines

Table B1. VOCS That May be Emitted From Building Materials and Products and Their Potential Sources

Chemical Name	***Potential Sources***
Acetic acid	*Solvent for resins, caulks, sealants, glazing compounds, volatile oils*
Acetone (2-Propranone)	*Lacquer solvent*
1-Amyl alcohol (Amyl alcohol; Pentyl alcohol; 1-Pentanol)	*Solvent in organic synthesis*
Benzaldehyde	*Fiberboard, particleboard*
Benzene	*Adhesives, spot cleaners, alkyd paints, paint removers, particleboard, furniture waxes*
2-butanone (Methyl ethyl ketone)	*Floor/wall coverings, fiberboard, caulking compounds, particleboard*
n-Butyl acetate (Butyle acetate)	*Floor lacquers*
Butyl acrylate (Butyl-2-propenoate)	*Used in manufacture of polymers and resins for textile and leather finishes*
n-Butyl alcohol (1-butanol)	*Edge sealings, molding tapes, jointing compounds, cement flagstones, linoleum floor coverings, floor lacquers, industrial cleaners, paint removers*
n-Butylbenzene	*Solvent*
Camphene	*Occurs in many essential oils*
Chlorobenzene	*Solvent for paints, used in manufacture of phenol*
Cyclohexane	*Solvent for lacquers and resins, paint and varnish removers*
Cyclohexanone	*Solvent for many resins and waxes*

Table B1. VOCS That May be Emitted From Building Materials and Products and Their Potential Sources	
Chemical Name	***Potential Sources***
Dibutylphthalate (Di-n-butyl phthalate)	*Plasticizer*
Diethylamine	*Used in resins, dyes, and in manufacture of rubber*
Dimethyl. Acetaminde (N,N-Dimethyl acetamide)	*Solvent for organic reactions*
Dioxane (p-dioxane; 1-4-Dioxane)	*Solvent for many oils, waxes, dyes, cellulose acetate*
Dodecane (n-Dodecane)	*Floor varnishes, floor/wall coverings*
2-Ethoxyethanol (Cellosolve®; Ethylene glycol monoethyl ether)	*Epoxy paints, latex paints, polyurethane varnishes*
2-Ethoxyethyl acetate (Cellosolve® acetate; Ethylene glycol monoethyl ether acetate)	*Floor lacquers, epoxy paints*
Ethyl acetate	*Vinyl floor coverings, solvent for varnishes and lacquers*
Ethyl alcohol (Ethanol)	*Fiberboard, solvents*
Ethyl benzene	*Floor/wall coverings, insulation foam, chipboard, caulking compounds, jointing compounds, fiberboard, adhesives, floor lacquers, grease cleaners*
2-Ethyltoluene (o-Ethyltoluene)	*Floor waxes*
Folmaldehyde (Methanal)	*<u>Major sources</u>: MDF, plywood, particleboard, ceiling panels, fiberboard, chipboard* *<u>Minor sources</u>: Upholstery fabrics, latex-backed fabrics, fiberglass, fiberglass insulation in air ducts, urea formaldehyde foam insulation, wallpaper, caulking compounds, jointing compounds, floor and furniture varnishes, adhesives, floor lacquers, gypsum board*
Heptane (n-Heptane)	*Floor coverings, floor varnishes*
Hexachlorobenzene	*Fungicide*
Hexanal	*Polyurethane wood finish*

Table B1. VOCS That May be Emitted From Building Materials and Products and Their Potential Sources	
Chemical Name	***Potential Sources***
Hexane (N-Hexane)	*Chipboard, gypsum board, insulation board, floor coverings, wallpaper*
Isobutyl acetate (2-Methylpropyl acetate)	*Floor lacquers*
Isobutyl alcohol (Isobutanol; 2-Methyl-1-propanol)	*Edge sealings, molding tapes, jointing compounds, cement flagstone, linoleum floor coverings, floor lacquers*
Isopropyl alcohol (Isopropanol; 2-Porpanol)	*Particleboard*
Isoquinolone	*Used in synthesis of dyes and insecticides; rubber accelerator*
d-Limonene	*Paints, adhesives, chipboard, detergents, furniture polish*
Methylene chloride (Methane dichloride; Dichloromethane)	*Paint removers, aerosol paints, industrial solvents*
Methyl isobutyl ketone (MIBK;4-Methyl-2-pentanone)	*Floor/wall coverings*
2-Methylpentane (Isohexane)	*Chipboard, gypsum board, insulation foam, floor coverings, wallpaper*
Nonane (n-Nonane)	*Wallpaper, caulking compounds, floor coverings, chipboard, adhesives, cement flagstone, jointing compounds, floor varnishes, floor waxes*
Nonyl phenol isomers	*Used in manufacture of lubricating oil additives, resins, plasticizers, and surface active agents*
Pentachlorophenol (PCP)	*Wood preservative, disinfectant, fungicide, paints, wallpaper, adhesives, textiles, wood finishes, floor shampoos*
4-Phenylcyclohexene (4-PC: cyclohexylbenzene0	*Manufacturing by-product in carpets with SBR latex backing*
□-Pinene	*Cement flagstone, fiberboard, gypsum board, adhesives, insulation sheets, chipboard, wood*
n-Propyl acetate	*Plastics*
Propylbenzens (n-Propyl benzene)	*Adhesives, floor;wall coverings, chipboard, paints, caulking compounds, insulation foam*
Quinolone	*Used in the manufacture of dyes; solvent for resins*

Table B1. VOCS That May be Emitted From Building Materials and Products and Their Potential Sources	
Chemical Name	***Potential Sources***
Styrene (Vinyl benzene)	*Insulation foam, jointing compounds, fiberboard, carpets with SBR latex backing*
-Terpinene (1-Methyl-4-isopropyl-1,3-cyclohexadiene)	*Furniture polishes*
Tetrachloroethylene (Perchloroethylene)	*Widely used in the textile industry for dry cleaning, processing, and finishing of fabrics; used in metal degreasers, spot removers, adhesives, wood cleaners, and lubricants*
Tetrachlorophenol	*Wood preservative*
Toluene	*Solvent-based adhesives, water-based adhesives, edge sealings, molding tapes, wallpaper, jointing compounds, floor coverings, vinyl coated wallpaper, caulking compounds, paints, chipboard, vinyl floor coverings*
1,1,1-Trichloroethane (Methyl chloroform)	*Cleaning fluids, water and stain repellents*
Trichloroethylene (TCE)	*Solvent for paints and varnishes*
1,2,3-Trimethylbenzene	*Floor/wall coverings, floor waxes*
1,2,4-Trimethylbenzene	*Floor/wall coverings, linoleum floor coverings, caulking compounds, vinyl coated wallpaper, jointing compounds, cement flagstone, floor varnishes, chipboard, floor waxes*
1,3,5-Trimethylbenzene (Mesitylene)	*Caulking compounds, floor/wall coverings, floor waxes*
Undecane (N-Undecane)	*Wallpaper, gypsum board, floor/wall coverings, joint compounds, chipboard, floor varnishes, paints, paint removers*
Xylenes	*Adhesives, jointing compounds, wallpaper caulking compounds, floor coverings, floor lacquers, grease cleaners, varnishes*

APPENDIX B

Reports from Sample Searches on Department of Pesticide's Web database—pesticide products that may be used in schools and school grounds

S)-METOLACHLOR

1,3-DICHLORO-5, 5-DIMETHYLHYDANTOIN

1,3-DICHLORO-5-ETHYL-5-METHYLHYDANTOIN

2,4-D

2,4-D, 2-ETHYLHEXYL ESTER

2,4-D, BUTOXYETHANOL ESTER

2,4-D, DIMETHYLAMINE SALT

2,4-D, ISOOCTYL ESTER

2-(2,4-DP), DIMETHYLAMINE SALT

ACEPHATE

ALKYL (50%C12, 30%C14, 17%C16, 3%C18) DIMETHYLETHYLBENZYL AMMONIUM CHLORIDE

ALKYL (50%C14, 40%C12, 10%C16) DIMETHYLBENZYL AMMONIUM CHLORIDE

ALKYL (58%C14, 28%C16, 14%C12) DIMETHYLBENZYL AMMONIUM CHLORIDE

ALKYL (60%C14, 30%C16, 5%C12, 5%C18) DIMETHYLBENZYL AMMONIUM CHLORIDE

ALKYL (67%C12, 25%C14, 7%C16, 1%C8, C10, C18) DIMETHYLBENZYL AMMONIUM CHLORIDE

ALKYL (68%C12, 32%C14) DIMETHYLETHYLBENZYL AMMONIUM CHLORIDE

ALLETHRIN

ALLETHRIN, OTHER RELATED

AVERMECTIN

AZOXYSTROBIN

BENEFIN

BENSULIDE

BETA-CYFLUTHRIN

BIFENAZATE

BIFENTHRIN

BORAX

BORIC ACID

CACODYLIC ACID

CARBARYL

CHLORFLURENOL, METHYL ESTER

CHLOROTHALONIL

CHLORPYRIFOS

CLETHODIM

CLOPYRALID, MONOETHANOLAMINE SALT

CORN GLUTEN MEAL

CYFLUTHRIN

CYPERMETHRIN

D-ALLETHRIN

D-TRANS ALLETHRIN

DDVP

DDVP, OTHER RELATED

DELTAMETHRIN

DIATOMACEOUS EARTH

DIAZINON

DICAMBA

DICAMBA, DIMETHYLAMINE SALT

DICHLOBENIL

DIDECYL DIMETHYL AMMONIUM CHLORIDE

DIFLUBENZURON

DIHYDRO-5-HEPTYL-2(3H)-FURANONE

DIHYDRO-5-PENTYL-2(3H)-FURANONE

DIKEGULAC SODIUM

DIMETHOATE

DIOCTYL DIMETHYL AMMONIUM CHLORIDE

DIPHACINONE

DIPROPYL ISOCINCHOMERONATE

DIQUAT DIBROMIDE

DISODIUM OCTABORATE TETRAHYDRATE

DITHIOPYR

EDTA, TETRASODIUM SALT

ESBIOTHRIN

ESFENVALERATE

ETHOFUMESATE

FENARIMOL

FENOXYCARB

FENVALERATE

FIPRONIL

FLUAZIFOP-BUTYL

FLURECOL-METHYL

GLUFOSINATE-AMMONIUM

GLYPHOSATE

GLYPHOSATE, ISOPROPYLAMINE SALT

GLYPHOSATE, MONOAMMONIUM SALT

HALOSULFURON

HYDRAMETHYLNON

HYDROGEN CHLORIDE

HYDROGEN PEROXIDE

HYDROPRENE

IMAZAPYR, ISOPROPYLAMINE SALT

IMIDACLOPRID

IODINE

LAMBDA CYHALOTHRIN

LIMONENE

MALATHION

MALEIC HYDRAZIDE, POTASSIUM SALT

MCPA

MCPA, DIMETHYLAMINE SALT

MCPA, ISOOCTYL ESTER

MCPP

MCPP, DIMETHYLAMINE SALT

MCPP-P, DIMETHYLAMINE SALT

MEFLUIDIDE, DIETHANOLAMINE SALT

METALDEHYDE

METARHIZIUM ANISOPLIAE, VAR. ANISOPLIAE, STRAIN ESF1

METHOPRENE

METHYL BROMIDE

METHYL-2,7-DICHLORO-9-HYDROXYFLUORENE-9-CARBOXYLATE

METRIBUZIN

MSMA (34)

N-OCTYL BICYCLOHEPTENE DICARBOXIMIDE

NALED

NONANOIC ACID

NONANOIC ACID, OTHER RELATED

OCTYL DECYL DIMETHYL AMMONIUM CHLORIDE

OIL OF CITRONELLA

ORTHO-BENZYL-PARA-CHLOROPHENOL

ORTHO-BENZYL-PARA-CHLOROPHENOL, POTASSIUM SALT

ORTHO-PHENYLPHENOL

ORTHO-PHENYLPHENOL, POTASSIUM SALT

ORYZALIN

OXADIAZON

OXYPURINOL

PARA-TERT-AMYLPHENOL

PARA-TERT-AMYLPHENOL, POTASSIUM SALT

PCNB

PENDIMETHALIN

PERMETHRIN

PERMETHRIN, OTHER RELATED

PEROXYACETIC ACID

PETROLEUM DISTILLATES

PHENOTHRIN

PHENOTHRIN, OTHER RELATED

PHOSMET

PIPERONYL BUTOXIDE

PIPERONYL BUTOXIDE, OTHER RELATED

POTASH SOAP

PRALLETHRIN

PRODIAMINE

PROMETON

PROPOXUR

PYRETHRINS

PYRIPROXYFEN

RESMETHRIN

RESMETHRIN, OTHER RELATED

SETHOXYDIM

SIDURON

SILICA AEROGEL

SODIUM CACODYLATE

SODIUM CHLORATE

SODIUM METABORATE TETRAHYDRATE

SODIUM METASILICATE

SPINOSAD

SULFLURAMID

SULFOMETURON METHYL

SULFUR

TAU-FLUVALINATE

TEBUFENOZIDE

TETRACHLORVINPHOS

TETRAMETHRIN

TETRAMETHRIN, OTHER RELATED

THIOPHANATE-METHYL

TRALOMETHRIN

TRIADIMEFON

TRICHLORFON

TRICLOPYR, BUTOXYETHYL ESTER

TRICLOPYR, TRIETHYLAMINE SALT

TRIFLOXYSTROBIN

TRIFLURALIN

TRINEXAPAC-ETHYL

VINCLOZOLIN

XANTHINE

ZINC PHOSPHIDE

APPENDIX C

Uses of Chemicals and Likely Exposure

Chemical	CASRN	Use and environmental exposure[1]
Acetaldehyde	75-07-0	Inhalation of ambient air, esp. in urban areas; near sources of combustion (power plants, auto exh[1]aust, fire places, wood-burning stoves, tobacco smoke), emissions from manufacture of plastics and synthetic rubber.
Acetamide	60-35-5	Used as a solvent, chemical intermediate, wetting agent, penetrating agent, hygroscopic agent, and as a component in laquers, explosives, and soldering flux. Exposure mainly occupational.
Acetone	67-64-1	Used as a solvent for fats, oils, waxes, resins, rubbers, plastics, pharmaceuticals, rubber cements, and in the production of chemical products. It is also released by volcanoes, forest fires, and cigarette smoke. Exposure from use of paints, adhesives, cosmetics, and rubber cement, and from inhalation of ambient air and cigarette smoke, and ingestion of food and water.
Acetonitrile	75-05-8	Used as a solvent and chemical intermediate. Found in thermal decomposition products of flexible polyurethane foam, shale oil wastewater, and in small amounts in coal tar. Released from incineration of polyacrylonitrile polymers, automobile exhaust, tobacco smoke, manufacture of synthetic rubber, manufacture of acrylonitrile, and turbine engines. Exposure from ingestion of food and water, cigarette smoke, and dermal contact with products containing acetonitrile.
Acetophenone	98-86-2	Used in manufacture of propylene oxide, kraft bleaching, and some perfumes. Found in heavy oil fraction of coal tar, gasoline exhaust, waste incineration, residential fuel oil, coal combustion, perfume, petrochemical plant waste waters, shale oil plant, and waste liquid from kraft bleaching.
Acetylaminofluorene	53-96-3	Used in biochemistry, organic chemistry, and biomedical labs. Less than 20 lbs of Acetylaminofluorene is used per year in the US.
Acrolein	107-02-8	Used as intermediate for glycerine, methionine, glutaraldehyde; aquatic herbicide and slimicide. Released to the environment through photochemical oxidation of airborne hydrocarbons; exhaust gas from main and sidestream tobacco smoke, marijuana smoke, forest fires, residential fireplaces, burning of coal, oil, and natural gas in powerplants, turbine engines, and auto exhaust; from coffee-roasting operations, manufacture of acrylic acid, lithographic plate coating, automobile spray booths, manufacture of fish oils, laquers, varnishes, plastics, synthetic rubber, spray painting, metal paint dryers, poultry-manure dryers, and corn-starch manufacture. Volatile from white bread and chicken breast, product from heating animal fats and vegetable oil. Exposure primarily from atmospheric contact.

[1] From Hazardous Substances Data Bank except where noted.

Acrylamide	79-06-1	Exposure is mostly occupational, but exposure to the general public may occur via ingestion of contaminated drinking water.
Acrylic acid	79-10-7	Used in manufacture of plastics, paint formulations, leather finishings, paper coatings, in dental plates, artificial teeth, and orthopedic cement. Exposure from contact with polyethylene, ethylene-vinyl acetate, and ethylene-methyl acrylate resins, dental products, and consumer products such as floor polish.
Acrylonitrile	107-13-1	Used in production of acrylic and modacrylic fibers. Released from auto exhaust, cigarette smoke, fibers and plastic, outgassing from acrylic fibers, plastic containers (ie food containers). Exposure from contact with the previously mentioned sources.
Alachlor	15972-60-8	Herbicide for annual grasses and broad-leaved weeds.
Aldrin	309-00-2	Pesticide. Contaminated air, water and food (Use discontinued)
Aluminum	7429-90-5	Ingestion of food and beverages, inhalation from atmospheric dust. Used in foundries and factories, and in production of explosives and fireworks. Emitted from aluminum smelters. [Used as flocculating agent in water purification, exposure from drinking water, use of aluminum cans, containers, and cooking utensils, deodorants and medications containing aluminum (Committee on Nutrition "Aluminum toxicity in infants and children, 1996)].
4-Aminobiphenyl	92-67-1	Used as a rubber antioxidant and dye intermediate. Possibly no longer manufactured.
Amitraz	33089-61-1	[Insecticide (P65 DART agents use list)]
Aniline	62-53-3	Used in synthesis of explosives, rubber accelerators, isocyanates, herbicides, pesticides, dyes, and as a solvent. Found in tobacco smoke and effluents from shale oil recovery and oil refineries.
o-Anisidine	90-04-0	Used in production of azo and hair dyes, and guaiacol, as a corrosion inhibitor for steel storage, and as an antioxidant for some polymercaptan resins. Found in cigarette smoke. Exposure mainly from inhalation of cigarette smoke.
Antimony	7440-36-0	Lead-antimony alloys used in manufacture of storage battery grids, pewter and britannia metal, printer's type, lead shot, lead electrodes, and bearing metals.
Arochlor 1016	12674-11-2	PCB. Has not been commercially produced since 1977 in the US. Currently released from landfills containing PCB waste materials and products, incineration of municipal refuse and sewage sludge, by improper disposal of PCB materials (ie transformer fluid), and by a cycling process of other PCBs in the environment. Exposure from inhalation of ambient air, ingestion of contaminated food (especially fish) and water, and dermal contact with the compound and products containing the compound.
Arsenic	7440-38-2	Ingestion of color pigments in paints via food, cups, cigarettes, hands, ceramic work; residing near (within 12 miles) of copper, lead, and zinc smelters. [Exposure can occur from contact with CCA-treated wood used in wood chips, play sets, decks, boardwalks, fences, and framing for residential and commercial structures. (Fields, 2001)]

Asbestos	1332-21-4	Air from mining, milling and processing asbestos products, insulation, worn brake-lining; talc-containing products such as cosmetics, powders, insecticides, white shoe polish, soap filler, dusting powder for toy balloons, condoms, and contraceptive diaphragms; spackles, caulks, roofing, vinyl-asbestos flooring, furnace pipe insulation, electric irons, stoves, millboard, paint, toasters, and hairdryers; food (vegetable oil, lard, mayonnaise, ketchup, meats) and beverages (beers, soft drinks, other alcohols).
Barium	7440-39-3	Released from mining, refining and production of barium, and from combustion of coal and oil. Some areas have contaminated well water.
Benomyl	17804-35-2	Fungicide. Inhalation of dust from treated fields; ingested as food residue.
Benzaldehyde	100-52-7	Released from emissions from gasoline and diesel combustion, incinerators, cigarette smoke, and wood burning, and from photochemical oxidation of toluene and other aromatic hydrocarbons. Exposure from consumption of food (can occur naturally or as an intentional additive) and inhalation of ambient air and cigarette smoke.
Benzene	71-43-2	Inhalation of air especially in areas with heavy traffic, near gas stations, close to manufacturing plants using benzene, coke ovens, nonferrous metal manufacturing, ore mining, wood processing, coal mining, textile manufacture; from tobacco smoke; some well water; dermal contact with benzene-containing gasoline products; food (beef, eggs, fruits and vegetables, nuts, dairy, meat, fruit juice beverages, soda, and ice-tea containing benzoate).
Benzidine	92-87-5	Strictly regulated - exposure is primarily occupational; can be released as emissions in waste water from manufacturing plants (used in manufacture of azo dyes).
Benzotrichloride	98-07-7	Used in manufacture of benzotrifluoride, hydroxy benzophone, pharmaceuticals, antimicrobial agents, chlorinated toluenes, dyes, and pesticides.
Benzoyl chloride	98-88-4	Used in manufacture of dyes, resis, perfumes, pharmaceuticals, benzoyl peroxide, polymerization catalysts, benzophenone, and stabilizers. Exposure mainly occupational.
Beryllium compounds	7440-41-7	Used in alloys, microelectronics, aerospace technology, rocket fuels, aircraft brakes, x-ray windows, neutron reflectors; exposure occurs through air, food (potatoes, tomatoes, head lettuce) and water; ceramics; cigarette smoke
Biphenyl	92-52-4	Released from combustion of biomass, coal, oil, plastics, refuse, rubber, wood; from tobacco smoke, motor vehicle emissions, phthalic anhydride manufacture, coal liquefaction, textile mills, and some dyeing industries. Exposure primarily from inhalation, but also from consumption of contaminated food and water.
Bis(chloromethyl ether)	542-88-1	Used as a chemical intermediate. May be present in industrial gases and emissions.
Bisphenol-A	80-05-7	Atmospheric Bisphenol A is mostly in particulate phase. Found in effluents and emissions from its manufacture and the manufacture of epoxy, polycarbonate, and polysulfone resins; exposure can be via use of epoxy powder paints used to paint metal objects.

Boric Acid	10043-35-3	Widely distributed in nature. Minerals include Sassolite, Borax, Kernite, and Tourmaline; high levels are most likely to occur in soil from marine sediments and arid soils.
Boron	7440-42-8	See Boric acid
Bromacil lithium salt	53404-19-6	[Herbicide (P65 DART agents use list)]
Bromine	7726-95-6	Traces found in air from automobile exhaust gases.
Bromodichloromethane	75-27-4	Used as a chemical intermediate and solvent. Main release from inadvertant formation during water chlorination treatments. Exposure from inhalation of ambient air, ingestion of food and drinking water, and dermal contact with chlorinated water (ie swimming pools).
Bromoform	75-25-2	Used in fire extinguishers, shipbuilding, aerospace industry, organic synthesis, and as a solvent for waxes, greases, and oils. Byproduct of water chlorination. Exposure from inhalation of ambient air, ingestion of food and drinking water, dermal contact, and consumption of products prepared with chlorinated water.
2-Bromopropane	75-26-3	Used in the synthesis of pharmaceuticals, dyes, and other organics.
Bromoxynil	1689-84-5	[Herbicide (P65 DART agents use list)]
Bromoxynil octanoate	1689-99-2	[Herbicide (P65 DART agents use list)]
1,3-Butadiene	106-99-0	Exposure occurs mostly from atmosphere. Major emission from motor vehicles; burning of fossil fuels; manufacture, use, and disposal of petroleum, plastic and synthetic rubber; use as chemical intermediate; component of tobacco smoke. Exposure is greatest in urban areas around heavy traffic and plants manufacturing butadiene. Has been detected in drinking water, and is present in foods packaged in 1,3-Butadiene rubber-based plastic containers (Olive oil, vegetable oil, yogurt).
p-tert-Butylbenzoic acid	98-73-7	Testicular atrophy, decreased sperm motility, testicular lesions (Chapin et al, 1994); Listed (Bernstein, 1984)
p-tert-Butyltoluene	98-51-1	Can be released from manufacturing sites and from evaporation during its use as a solvent.
p-tert-Butyl benzaldehyde	939-97-9	Not in HSDB
Butyraldehyde	123-72-8	Released from combustion of gasoline and diesel engines, wood burning, and cigarettes. Found in emissions from animal waste, coffee manufacturing, fish meal manufacturing, and petroleum processing.
Cadmium	7440-43-9	Released during mining, processing and smelting of zinc and zinc-lead ores, and during recovery, refining, and maunfacture of cadmium compounds; use of cadmium compounds. Used in telephone cables, trolley wires, welding, electrodes, automatic sprinkling systems, steam boilers, fire alarms, high pressure/temperature bearings, starting switches, aircraft relays, light duty circuit breakers, low temp solder, jewelry, paint pigments for plasitics and ceramic glazes, nickel-cadmium batteries in radio portable phones, appliances, and vented cells used in stand-by power and lighting. Main route is usually air. Exposure via tobacco smoke and food.

Calcium	7440-70-2	One of the most abundant metals. Found in calcium carbonate (limestone and marble), calcium sulfate (gypsum), calcium fluoride, and calcium phosphate (apatite).
Calcium cyanamide	156-62-7	Pesticide additive.
Caprolactam	105-60-2	Used in manufacture of synthetic fibers (polyamides), and as a solvent for high molecular weight polymers. Exposure from inhalation of ambient air, particularly around new carpets.
Captan (orthocide)	133-06-2	Fungicide. Main exposure for general population is via food and drink.
Carbofuran	1563-66-2	Pesiticide. Inhalation and dermal exposure near spray sites, exposure via food and water also possible.
Carbon disulfide	75-15-0	Primary routes through ambient air, and food. Used in manufacture of rayon, carbon tetrachloride, xanthogenates, soil disinfectants, electric vacuum tubes, and as a solvent. Volatile compound released from textile floor coverings. Natural sources include oceans, marshlands, and inland soils.
Carbon monoxide	630-08-0	
Carbon tetrachloride	56-23-5	General population exposure occurs from inhalation of ambient air; ingestion of food and drinking water (widely detected in ground water); and dermal contact from carbon tetrachloride-containing products. Exposure can also occur from it's use as a solvent, cleaning agent, synthesis of nylon-7, and in fire extinguishers and grain fumigants. It is also released from iron and steel manufacturing, metal finishing, paint and ink formulations, and petroleum refining.
Carbonyl sulfide	463-58-1	Release from deciduous trees, volcanoes, coniferous trees, salt marshes, and soils; automobiles, coal-fired power plants, biomass combustion, fish processing, refuse combustion, plastic, petroleum, synthetic fiber, starch, and rubber manufacture. Used in viscose rayon industry. Exposure from background presence in atmosphere.
Catechol	120-80-9	Used in synthesis of polymerization inhibitors, antioxidants, pharmaceuticals and pesticides, and in photography and photosensitive copying papers. Found in coal conversion waste waters, crude wood tar, water from bituminous shale, effluent from coal-tar chemical production, and cigarette smoke. Exposure from ingestion of contaminated food or water.
Cesium	7440-46-2	Not in HSDB
Chinomethionat (Oxythioquinox)	2439-01-2	[Miticide, fungicide, insecticide (P65 DART agents use list)]
Chloramben	133-90-4	Herbicide for seedling grass, broadleaf weeds, velvetleaf, ragweed, redroot pigweed, and Pennsylvania smart weed. Exposure from drinking water or groundwater.
Chlordane	15789-03-6	Insecticide. No longer in use in US. Exposure via food, inhalation, and contact with treated soil. (Can persist on hands of workers for 2 years.)

Chlordecone (Kepone)	143-50-0	Insecticide. Breakdown product of Mirex. No longer approved for use as insecticide in US. Main exposure occurs via consumption of contaminated food, especially seafood and fish. Can also occur via inhalation or dermal contact. Tobacco smoke (plants having been treated previously with chlordecone). Child exposure previously occured from insect traps.
Chlorfenvinphos	470-90-6	Insecticide. Contaminated food, handling of flea-control products.
Chlorine	7782-50-5	Dermal contact from handling chlorine and its products; inhalation of ambient air; ingestion of chlorine-treated food and water; industrial accidents; school chemistry experiments; accidental release of chlorine from swimming pools; mixing cleaning agents.
Chloroacetic acid	79-11-8	Used in manufacture of cellulose ethers, herbicides, thioglycolic acid, sufactants, cyanoacetic acid, phenoxyacetic acid, glycine, and chloroacetic acid esters. Found in drinking water as a disinfectant byproduct. Exposure from drinking water.
Chloroacetophenone	532-27-4	Used in manufacture of pharmaceuticals and as an ingredient in MACE. Exposure from use of MACE.
Chlorobenzene	108-90-7	Used in manufacture of chloronitrobenzenes and as a solvent carrier for methylene diisocyanate. Found in emissions from polystyrene foam insulation and new carpets. Exposure from inhalation of ambient air, ingestion of food and drinking water, dermal contact with vapors, food and other products containing chlorobenzene.
Chlorobenzilate	510-15-6	Former acaricide. Exposure may have been from ingestion of contaminated food and water.
Chloromethyl methyl ether	107-30-2	Used as a chemical intermediate in production of ion-exchange resins.
4-Chloro-3-nitroaniline	635-22-3	Used as an intermediate in manufacture of azo dyes, pharmaceuticals, and other organic compounds.
Chloroprene (2-chlorobuta-1,3-diene)	126-99-8	Exposure from emissions from plants making polychloroprene elastomers.
Chlorpyrifos	2921-88-2	Insecticide. Inhalation of ambient air and ingestion of contaminated foods.
Chlorsulfuron	64902-72-3	[Herbicide (P65 DART agents use list)]
Chlozolinate (Vinclozolin)	50471-44-8	Fungicide. Main exposure is via ingestion of contaminated food. Dermal and inhalation exposure possible near spraying.
Chromium compounds	7440-47-3	See III and VI

Chromium III	16065-83-1	Exposure via skin, inhalation, and ingestion. Present in food. Can enter environment from fossil fuel combustion; waste and munincipal incineration; cement plants; steel, refractory, and chemical manufacturing; sewage sludge; fertilizers; road dust; asbestos dust; and paper mills. Uses include fabrication of alloys, plated products, chemical intermediates, catalyst, dyeing, silk treating, printing, moth proofing wool, tanning leather, photographic fixing baths, fuel and propellant additives, ceramics, paints, varnishes, inks, glazes, metal polishing, corrosion inhibitor. Present naturally in soils and plants. [Exposure can occur from contact with CCA-treated wood used in wood chips, play sets, decks, boardwalks, fences, and framing for residential and commercial structures The Chromium component is applied as hexavalent but converts to trivalent. (Fields, 2001)]
Chromium VI	18540-29-9	Use in chrome plating; anodizing; conversion coatings; corrosion resistance in radiator coolants, internal combustion and gas turbine engines, refrigerator and air conditioning systems, and water cooled nuclear reactors; leather finishing; photography, photoengraving, lithography, and blue printing; corrosion inhibiting and color pigment in paints, jointing pastes, inks, rubber, ceramics and color blending; dyeing of fur, leather, fabrics, wool, nylon; use in manufacture of glue (for shoes, furniture, and packaging); fungicides; preservative and fire retardant in woods; protection of textiles; batteries; manufacture of safety matches and explosives; chemistry reagent; manufacture and packaging of cement. Exposure from fossil fuel combustion; waste incineration; cooling towers; cement plants; chrome plating plants. Present naturally in plants and soil. [Exposure can occur from contact with CCA-treated wood used in wood chips, play sets, decks, boardwalks, fences, and framing for residential and commercial structures The Chromium component is applied as hexavalent but converts to trivalent. (Fields, 2001)]
Ciodrin	7700-17-6	Insecticide. Registration cancelled. Exposure primarily occupational from inhalation or dermal contact.
Cobalt	7440-48-4	In minerals, by product or coproduct of refining of other mined metals, in food (especially green leafy vegetables), and tobacco and tobacco smoke.
Coke oven emissions		Not in HSDB
Copper	7440-50-8	In minerals, near copper mines or smeliting works, may be produced by municipal incineration.
Creosote	8001-58-9	Wood preservative. Exposure from old creosote-treated lumber, which retains oil for up to 25-30 years; occupations with exposure: carpenters, railroad workers, farmers, tar distillers, glass and steel furnace attendants, engineers. Creosote is easily absorbed through skin and mucous membranes.
Cresols/Cresylic acid	1319-77-3	Used as a disinfectant, chemical intermediate, textile scouring agent, surfactant, and ore floatation device. Released in auto and diesel exhaust, tobacco smoke, during coal tar and petroleum refining, wood pulping, and during use in metal refining. Found in wastewater treatment plants.

Crotonaldehyde	4170-30-0	Released from combustion of wood, polymers, tobacco, in gasoline, diesel, or turbine engine exhausts, and in volcanic gases. Exposure from inhalation of tobacco smoke, gasoline and diesel exhausts, and wood combustion.
Cumene	98-82-8	Used in production of phenol and acetone. From evaporation of petroleum products. Released in tobacco smoke, and from the vulcanization of rubber, building materials, jet engine exhaust, outboard motors, solvent use, paint manufacture, pharmaceutical production, textile plants, leather tanning, iron and steel manufacturing, paving and roofing, paint and ink formulation, printing and publishing, ore mining, coal mining, organics and plastics manufacturing, pesticide manufacturing, electroplating, and pulp and paper production. Exposure from inhalation of ambient air and tobacco smoke, and ingestion of food (occurs naturally in many foods).
Cyanazine	21725-46-2	[Herbicide (P65 DART agents use list)]
Cyanide compounds	57-12-5	(Hydrogen cyanide) Used in manufacture of acrylates, cyanide salts, herbicides, and dyes, and was formerly used as a fumigant. Exposure from automobile exhaust, tobacco smoke, blast furnaces, gas works, coke ovens, and waste incinerators.
Cycloate	1134-23-2	[Herbicide (P65 DART agents use list)]
Cyclohexanol	108-93-0	Used in production of adipic acid and caprolactam which are used in manufacture of nylon 66 and 6; as a solvent for laquers, resins, varnishes, paint removers; in production of plasticizers and insecticides; in dry cleaning, textile cleaning, laundries, in soaps and detergents, fragrances, polishes and rubber cement. Is a volatile component of some cooked foods. Exposure from ingestion of contaminated water and food; inhalation of air; dermal contact with contaminated water.
Cycloheximide	66-81-9	[Fungicide (P65 DART agents use list)]
Cyclohexylamine HCL	4998-76-9	(Cyclohexylamine) Used in boiler water treatment; production of rubber chemicals; as a chemical intermediate in production of dyes, insecticides, and pharmaceuticals. Is a metabolite of the artificial sweetner cyclamate.
Cyhexatin	13121-70-5	[Acaricide (P65 DART agents use list)]
2,4-D	94-75-7	Used as a systemic herbicide. Exposure for general public mainly through ingestion of contaminated food and water.
2,4-DB	94-82-6	[Herbicide (P65 DART agents use list)]
2,4-DP (Dichloroprop)	120-36-5	[Pesticide - not registered (P65 DART agents use list)]
DDD	72-54-8	Insecticide, metabolite of DDT. No longer registered for agricultural use in US. Exposure from ingestion of food and drinking water, inhalation of contaminated air, and dermal contact with contaminated soil.
DDE	72-55-9	Impurity and metabolite of DDT. Exposure from ingestion of food and drinking water, inhalation of contaminated air, and dermal contact with contaminated soil. Tissue storage of DDE is almost entirely from ingestion of DDE, not from DDT conversion.

DDT	50-29-3	Broad-spectrum pesticide. Use in US only allowed for EPA-determined emergencies. Exposure from ingestion of food and drinking water, inhalation of contaminated air, and dermal contact with contaminated soil.
Dacthal	1861-32-1	Herbicide. Exposure from inhalation of indoor and outdoor air (especially in agricultural areas), carpet dust, ingestion of leafy and root vegetables, and from contaminated well water.
n-Decane	124-18-5	Found in parafin fraction of cude oil and natural gas. Released from solvent based building materials, printing pastes, paints, varnishes, adhesives and other coatings, landfills and waste incinerators, vulcanization and extrusion operations during rubber and synthetic production, and the combustion of gasoline, diesel fuels, and plastics. Exposure from inhalation of ambient air.
Diazinon	333-41-5	Insecticide and acaricide. Manufacture for indoor use products discontinued March 1, 2001; manufacture of nonagricultural outdoor use products will be discontinued June 30, 2003. Exposure during application for residential insecticide, inhalation of ambient air, and ingestion of contaminated food and water.
Diazomethane	334-88-3	Laboratory use. Prepared in situ.
Dibromochloromethane	124-48-1	Used as chemical intermediate. Formed as byproduct in water disinfection from chlorine treatment. Exposure from inhalation of ambient air, ingestion of contaminated water, and dermal contact with the compound
Dibromochloropropane (DBCP)	96-12-8	Nematocide and soil fumigant. Use is restricted. Also used in organic synthesis. Exposure from ingestion of contaminated drinking water (especially ground water). Possible emissions from use in organic synthesis.
1,2-Dichlorobenzene	95-50-1	Used as a solvent, in the manufacture of 3,4-dichloroaniline, as an insecticide, and as a deodorant in industrial wastewater treatment. Exposure from inhalation of ambient air, and ingestion of food and water.
1,4-Dichlorobenzene	106-46-7	Used as an insecticide, space deodorant, and chemical intermediate. Exposure from inhalation of ambient air, and ingestion of food and water.
3,3-Dichlorobenzidene	91-94-1	Used as an intermediate in the manufacture of pigments. Exposure is primarily occupational.
Dichlorodifluoromethane	75-71-8	Formerly produced and used as an aerosol propellant, foaming agent, and refrigerant. Exposure from inhalation of ambient air.
1,2-Dichloroethane	107-06-2	Used as a chemical intermediate, lead scavenger, extraction and cleaning solvent, diluent for pesticides, grain fumigant, and in paint, coatings, and adhesives. Released from waste water. Exposure from urban air, and drinking water from contaminated aquifers.
Dichloroethyl ether	111-44-4	Not in HSDB
Dichlorophene	97-23-4	[Pesticide - not registered (P65 DART agents use list)]
1,3-Dichloropropene	542-75-6	Used as a soil fumigant and chemical intermediate. Exposure from inhalation near source areas.

Dichlorvos	62-73-7	Insecticide in sprays, household and commercial resin strips, and flea collars. Breakdown product of Trichlorphon. Exposure from inhalation of indoor air where Dichlorvos has been used as an insecticide. Routes can be inhalation, dermal contact, and ingestion.
Diclofop methyl	51338-27-3	[Herbicide (P65 DART agents use list)]
p-Dicyanobenzene	623-26-7	m-Dicyanobenzene: occupational
Dieldrin	60-57-1	Insecticide and degradation product of Aldrin. No longer registered for agricultural use in US. Exposure from ingestion of food and drinking water, inhalation of contaminated air, and dermal contact with contaminated soil.
Diesel Exhaust Particles (DEP)		Diesel Exhaust. Mixture of >50% phenanthrenes. Also naphthalenes, pyrenes, and quinones. [Contains oxygen, nitrogen, carbon dioxide, water vapor, carbon monoxide, sulfur oxides, nitrogen oxides, alkenes, aromatic hydrocarbons, formaldehyde, 1,3-Butadiene, PAHs and derivatives, arsenic, acetaldehyde, antimony, benzene, beryllium, diethylhexyl phthalate, dioxins and dibenzofurans, lead, mercury, nickel, and styrene. About 94% of PM are <2.5 microns. (The Report on Diesel Exhaust, 1998)].
Diethanolamine	111-42-2	Used in toiletries (shampoos, soaps, cosmetics), detergents, and other surfactancts, and as a dispersing agent in agricultural chemicals. Released from disposal of consumer products, and from using lubricating liquids. Exposure from dermal contact with consumer products.
N,N-Dimethyl aniline	121-69-7	Used in dyes, intermediates, solvents, the manufacture of vanillin, stabilzers, and reagents.
Diethyl sulfate	64-67-5	Used as an ethylating agent in chemical synthesis.
Diethylstilbestrol (DES)	56-53-1	Used in biochemical research, and human and vet medicine. Exposure through contaminated beef and chicken.
Dimethoate	60-51-5	Insecticide. Exposure through ingestion of contaminated food.
3,3'-Dimethoxybenzidene (o-Tolidene)	119-93-7	Not in HSDB
Dimethyl benzaldehyde	5779-94-2	Not in HSDB
Dimethyl carbomyl chloride	79-44-7	Used in manufacture of herbicides.
Dimethyl formamide	68-12-2	Used as a solvent for manufacturing acrylic fibers, and sheets, films, and coatings for polyurethanes, specialty PVCs, polyacrylonitrile, epoxy cellulose derivatives, and polyamides; for crystallization of pharmaceutical products; and for electrolytes in high voltage capacitors and electroplating.
1,1-Dimethyl hydrazine	57-14-7	Used in jet and rocket fuels, in chemical synthesis, as a stabilizer for organic fuel additives, an absorbent for acid gases, in photography, and as a plat growth control agent. Exposure via ingestion of food.
Dimethyl methylphosphonate (DMMP)	756-79-6	Used as a flame retardant and viscosity depressant in polyester and epoxy resins and in the manufacture of bathtubs and showers.
Dimethyl sulfate (DMS)	77-78-1	Used as a methylating agent for amines and phenols and in the manufacture of dyes, agricultural chemicals, drugs and others. Also used as a solvent.
m-Dinitrobenzene	99-65-0	[Chemical intermediate (P65 DART agents use list)]
o-Dinitrobenzene	528-29-0	[Chemical intermediate (P65 DART agents use list)]

p-Dinitrobenzene	100-25-4	[Chemical intermediate (P65 DART agents use list)]
4,6-Dinitro-o-cresol and salts	534-52-1	Used as a dormant spray insecticide for fruit trees to kill locusts and other insects. Exposure from ingestion of contaminated food.
2,4-Dinitrophenol	51-28-5	Used in manufacture of dyes and diaminophenol, and as a chemical indicator.
Dinitrotoluene (technical grade)		[Manufacturing of dyes, explosives (P65 DART agents use list)]
2,4-Dinitrotoluene	121-14-2	[Manufacturing of dyes, explosives (P65 DART agents use list)]
2,6-Dinitrotoluene	606-20-2	[Manufacturing of dyes, explosives (P65 DART agents use list)]
Dinocap	39300-45-3	[Fungicide and acaricide (P65 DART agents use list)]
Dinoseb	88-85-7	Herbicide. Also used as a corn yield enhancer and a miticide.
1,4-Dioxane	123-91-1	Solvent in paints, varnishes, lacquers, cosmetics, and deodorants. Exposure from contact with products containing dioxane.
Dioxins and dibenzofurans (TCDD)	1746-01-6	Exposure primarily from incineration of municipal and chemical wastes; exhaust from automobiles using leaded gasoline; improper disposal of some chlorinated chemical wastes; contaminated fish; emissions from wood burning in the presence of chlorine. Has been detected in coffee filter papers - 20-35% passed from filter into coffee during experimental coffee brewing.
1,2-Diphenylhydrazine	122-66-7	Used in manufacture of phenylbutazone, sulfinpyrazone, and benzidene. Exposure from ingestion of fish and water.
Disodium cyanodithioimidocarbonate	138-93-2	[Microbiocide (P65 DART agents use list)]
Dithiocarbamates		See individual compounds
Maneb	1247-38-2	Fungicide. Exposure from ingestion of contaminated foods (found infrequently in US foods) and drinking water, and from dermal contact with Maneb and products containing Maneb.
Nabam	142-59-6	Fungicide. Exposure from ingestion of food and dermal contact with fungicide products containing Nabam.
Potassium dimethyldithiocarbamate	128-03-0	[Microbiocide (P65 DART agents use list)]
Sodium dimethyldithiocarbamate	128-04-1	[Microbiocide (P65 DART agents use list)]
Zineb	12122-67-7	Fungicide. No longer in commercial use in US. Exposure via ingestion of food and dermal contact with products containing Zineb.
Ziram	137-30-4	Used as a fungicide and vulcanization accelerator in rubber-processing. Exposure from its production, use in rubber industry, application as fungicide, and from ingestion of contaminated foods.
Diquat	85-00-7	Herbicide, seed dessicant, aquatic weed control agent. Exposure to individuals applying chemical, or who are near fields or water where diquat is used. May be released into wastewater or in spills during manufacture, transport and storage.
Diuron	330-54-1	Preemergent herbicide.
n-Dodecane	112-40-3	Used in organic synthesis, jet fuel research, manufacture of paraffin products, the rubber and paper processing industries, as a solvent, standardized hydrocarbon, and a distillation chaser. Also released in automobile exhaust. Exposure from inhalation of ambient air, ingestion of food and water, dermal contact with vapors, food, and other products containing dodecane.

Endosulfan	115-29-7	Insecticide. Exposure through ingestion of contaminated food. Flower inspectors and florists have been exposed to contaminated flowers. Present in tobacco smoke and tobacco products.
Endosulfan I	959-98-8	See Endosulfan
Endosulfan II	33213-65-9	See Endosulfan
Endosulfan sulfate	1031-07-8	Oxidative degradation product and biodegradative product of endosulfan.
Endrin	72-20-8	[Insecticide (P65 DART agents use list)]
Endrin aldehyde	7421-93-4	Impurity and metabolite of endrin.
Environmental Tobacco Smoke		
Epichlorohydrin	106-89-8	Used as a solvent and chemical intermediate. Used as a surfactant; stabilizer in insecticides; in epoxy resins; manufacture of metals, electrical machinery transport equipment, textile and leather, wood and furniture, paints and petroleum, non-metallic mineral products, optical instruments, plastic and boat building, and elastomers; publishing and printing; painting and carpentry; construction; and health services.
1,2-Epoxybutane	106-88-7	
Ethanol	64-17-5	Used as solvent, chemical intermediate, and in the fermentation and production of alcoholic beverages. Occurs naturally as a plant volatile, microbial degradation product, and as a product of natural fermentation of carbohydrates. Exposure from intentional ingestion, contaminated atmosphere especially near industries and cities, ingestion of pharmaceuticals and contaminated drinking water, and from natural release sources. Product of combustion (including cigarette smoke).
Ethyl acrylate	140-88-5	Used in the manufacture of emulsion polymers in surface coatings, textiles, paper, polishes, and leather, in solution polymers for surface coatings, and in acrylic fibers. Exposure from inhalation of ambient air, ingestion of food, and dermal contact.
Ethyl benzene	100-41-4	Used in production of styrene, as a resin solvent, is in automotive and aviation fuels, and crude oil. Exposure from inhalation of ambient air, ingestion of contaminated food and water, and by handling gasoline.
Ethyl chloride	75-00-3	Released from use as chemical intermediate; evaporation from use as a solvent, an aerosol, and anesthetic uses; stack emissions from plastics and refuse combustion; from chlorination treatment of landfill leachate, wastewater treatment plants, and leaching from landfills; and from use as refrigerant. Exposure from inhalation of contaminated air.
Ethyl dipropylthiocarbamate	759-94-4	[Herbicide (P65 DART agents use list)]
Ethylene dibromide (1,2-dibromoethane)	106-93-4	Used as a lead scavenger in leaded gasoline; as a solvent for resins, gums, and waxes; as an intermediate in synthesis of dyes and pharmaceuticals; and formerly as a fumigant for soil, grain, fruit, vegetables, tobacco, and seed. Exposure from evaporation of leaded gasoline (highest in areas with heavy traffic and filling stations).
Ethylene and Diethylene Glycol Ethers and Acetates		See individual compounds

Ethylene Glycol Monobutyl Ether (EGBE)	111-76-2	Used as a solvent for nitrocellulose resins, spray laquers, varnishes, enamels, dry-cleaning compounds, varnish removers, textiles, and cosmetics, and as an intermediate in acetate esters and phthalate and stearate plasticizers. Exposure from dermal adsorption (most significant route), ingestion, and inhalation (particularly from household products). Found in leachate from municipal landfills and hazardous waste sites. Found in stripping agents; printing pastes; varnishes; sterilization agents; machine cutting oils; solvent-based building materials such as silicone caulk; hydraulic fluids; water-based coatings; metal cleaners and polishers; textile lubricants; liquid wax for marble, ceramic, linoleum, plastic, and varnished wood floors; printer inks; primers; engine degreasers; rug and upholstery cleaners; all purpose cleaners; window and glass cleaners; soaps; and vinyl and acrylic paints.
Ethylene Glycol Monoethyl Ether (2-Ethoxyethanol)	110-80-5	Used as a solvent for nitrocellulose, laquers and dopes; used in emulsions, varnish removers, cleansing solutions, dye baths; used in furniture manufacturing; and in finishing leather with water pigments and dye solution. Has been detected in river water, compost exhaust, indoor air, and as a volatile component of fried bacon, pork, and beef, and several household products (including liquid wax for marble, ceramic, linoleum, plastic, and varnished wood floors). Exposure from inhalation of ambient air, ingestion of food and drinking water, dermal contact with vapors, food, and other products containing ethoxyethanol.
Ethylene Glycol Monoethyl Ether Acetate (2-Ethoxyethyl Acetate)	111-15-9	Used as a solvent for nitrocellulose, oils, and resins; used in laquers, varnish removers, wood stains, textiles, and leather. Used in semi-conductor industry, auto paint shops, airline industry, furniture factories, screen print industry (found in inks, thinners, and solvents), and in microelectronics clean rooms.
Ethylene Glycol Monomethyl Ether (2-Methoxyethanol)	109-86-4	Used as a solvent for resins and dyes. Used in nail polishes, quick drying varnishes, enamels, wood stains, and cleaning agents for removing inks and pigments. Exposure from use of consumer products containing methoxyethanol.
Ethylene imine	151-56-4	Used as a flocculating agent in water treatment, coating agent, adhesive, and chemical intermediate.
Ethylene oxide	75-21-8	Exposure from emissions and aqueous effluent from production and use in manufacture of ethylene glycol, ethoxylates, and ethanolamines. Emissions from use as fumigant and sterilant of food, cosmetics, and hospital supplies; combustion of auto and diesel exhaust; tobacco smoke.
Ethylene thiourea	96-45-7	[Neoprene production (P65 DART agents use list)]
Ethylidene dichloride (1,1-Dichloroethane)	75-34-3	Used as a chemical intermediate, coupling agent in anti-knock gasoline, paint and varnish remover, metal degreaser, ore floatation device, production of 1,1,1-trichloroethane. Also formed in groundwater from reduction of 1,1,1-trichloroethane. Exposure from inhalation of ambient air.
Ethyl methanesulfonate	62-50-0	Not in HSDB
Fenoxaprop ethyl	66441-2-34	[Herbicide (P65 DART agents use list)]

Fenthion (OP)	55-38-9	Insecticide. Exposure from ingestion of contaminated foods. When used for mosquito control, applied as thermal fog by aircraft - exposure can occur from aerial drift.
Fine mineral fibers (from glass, rock, or slag and pm1		Not in HSDB
Fluazifop butyl	69806-50-4	[Herbicide (P65 DART agents use list)]
Fluvalinate	69409-94-5	[Insecticide (P65 DART agents use list)]
Formaldehyde	50-00-0	Used in manufacture of resins and as an intermediate. Produced directly and indirectly by combustion. Exposure from ambient air in heavy traffic (especially during photochemical smog epiodes); combustion sources such as power plants, incinerators, refineries, wood stoves, smoked meats, and cigarette smo ke; and where it is used in leather tannning; and as a resin, fumigant, disinfectant, sterilant, and embalming fluid. Homes (esp. energy efficient homes) can have high levels from stoves, insulation, furniture, resin-coated rugs, and other fabrics. Found in embalming fluid, auto emissions, resin treated fabrics, rugs, paper, and materials such as particle board and plywood which use resin adhesives and foam insulation, urea-formaldehyde resin binder in furniture and floor coverings, paper plates and cups, and clothing. [Also found in cosmetics and deodorants (Handzel, 2000)]. [Might be used as preservative in water-soluble paints (Lesser and Weiss, 1995)]. Particle board generates urea-formaldehyde (which can can continue after free-formaldehyde is removed) resin bound in wood aerosol.
Heptachlor	76-44-8	Former pesticide. Currently restricted to control of fire ants in power transformers. Exposure is atmospheric, aquatic, and from food residues. Infants can be exposed from mother's and cow's milk.
Heptachlor Epoxide	1024-57-3	Formed by transformation of Heptachlor in the environment. Also found in Chlordane. Heptachlor's agricultural uses have been phased out. Still used for underground termite control. Exposure from ingesting contaminated dairy, meat and fish; and from inhalation of vapors in houses treated with Heptachlor and Chlordane.
Hexachlorobenzene	118-74-1	Used as organic synthesis reagent. Former use as fungicide. Exposure from inhalation of ambient air, and ingestion of contaminated food and water. Used in production of Tri- and Tetrachloroethylene, Carbon Tetrachloride, Chlorine, Dimethyltetrachlorophthalate, Vinyl chloride, Atrazine, Propazine, Simazine, Pentachloronitrobenzene, electrolytic Chlorine, ordinance and pyrotechnics, Sodium chlorate, Aluminum, PCP, electrodes, and synthetic rubber. Also used in seed treatment and wood preservative industries.
Hexachlorobutadiene	87-68-3	Used as a solvent for elastomers, heat transfer liquid, and transformer and hydraulic fluid. Released from refuse combustion and fly ash. Exposure from inhalation of ambient air and ingestion of contaminated food and water.
Hexachlorocyclopentadiene	77-47-4	Release from use as intermediate, application and disposal of contaminated pesticides, and the combustion of some chlorinated wastes. Exposure from ingestion of contaminated food and water and inhalation of contaminated air.

alpha-Hexachlorocyclohexane	319-84-6	Component of lindane. Banned in US. Exposure from ingestion of contaminated food.
beta-Hexachlorocyclohexane	319-85-7	Formerly used as component of the pesticide BHC. Exposure from ingestion of contaminated food and water.
delta-Hexachlorocyclohexane	319-86-8	Formerly used as component of the pesticide BHC. Exposure from ingestion of contaminated food and water.
gamma-Hexachlorocyclohexane (Lindane)	58-89-9	Insecticide. Exposure from inhalation of ambient air, ingestion of contaminated food and water, and dermal contact with medicinal products containing lindane (scabicides, pediculocides, ectoparacides). Infants may be exposed from contaminated mother's milk.
Hexachloroethane	67-72-1	Used in many products and processes. Formed during the incineration of chlorinated wastes, and the chlorination of sewage and drinking water. Exposure from inhalation of ambient air, ingestion of drinking water and possibly fish, and dermal contact with vapors and products containing hexachloroethane.
Hexachlorophene (HCP)	70-30-4	Used in germicidal soaps and other soaps and cosmetics, and as soil fumigant and seed-treatment fungicide. Exposure from dermal contact with consumer products (soaps, etc) and from contaminated drinking water.
Hexafluoroacetone (hexafluoropropanone)	684-16-2	Used in synthesis of polymers, medicinals, agriculture chemicals, and in high-performance fluoropolymers and hexafluoropropanol solvent. Used as intermediate for hexafluoroisopropanol, polyacrylates used for textile coating, polyester coatings for textiles; solvent for acetal resins and polyamides, and as a polymer adhesive.
Hexaldehyde	66-25-1	Used as a food additive, in organic synethesis of plasticizers, rubber chemicals, dyes, synthetic resins, insecticides and perfumes. Found in water samples, engine exhaust, atmosphere, food (naturally), human adipose tissue, emissions of household waste, landfills, agricultural plants, building materials, Exposure from inhalation of ambient air, ingestion of foos and water, and dermal contact.
Hexamethylene-1,6-diisocyante	822-06-0	Released from spray applications of polymer paints containing residual amounts of momomeric hexamethylene diisocyanate.
Hexamethylphosphoramide	680-31-9	Used as a UV inhibitor in plastics, a specialty solvent, an experimental chemosterilant for insects, and in promoting stereo-specific reactions. Exposure from dermal contact and inhalation of vapors.
Hexamethylthiophosphoramide	3732-82-9	Not in HSDB
N-Hexane	110-54-3	Constituent in paraffin fraction of crude oil and natural gas. Release from petroleum and gasoline industries; printing pastes; paints; varnishes; adhesives and other coatings; hazardous waste disposal sites; landfills and waste incinerators; combustion of polyvinyl chloride, and gasoline and diesel-fueled engines; glues in sandal, shoe, belt, and furniture manufacturing; polyethylene laminating; and cleaning tablets in pharmaceutical plant.
2,5-Hexanedione	110-13-4	Not in HSDB

2-Hexanone	591-78-6	Used as a solvent for laquers, resins, oils, nitrocellulose, vinyl, and alkyd coatings. Exposure from inhalation of ambient air, and ingestion of food and water containing the compound.
HMX (Cyclotetramethylenetetranitramine)	2691-41-0	Uses not given in HSDB
Hydramethylnon	67485-29-4	[Insecticide (P65 DART agents use list)]
Hydrazine	302-01-2	Used as a chemical intermediate, reducing agent, rocket fuel, and in boiler water treatment. Exposure from inhalation of cigareete smoke, ingestion of trace residues in processed foods, and dermal contact with vapors and products containing hydrazine.
Hydrochloric acid	7647-01-0	Released from the combustion of fuels (organic chlorides and gasoline); refuse incineration; secondary metals industry; pyrolysis of some wire insulation materials such as polyvinyl chloride, chlorinated acrylics, and retardant treated materials; as a by-product in dehydrohalogenation to make unsaturated compounds from chlorinated hydrocarbons; and from coal-fired power plants.
Hydrofluoric acid	7664-39-3	From the manufacture of phosphate fertilizer.
Hydrogen Sulfide	7783-06-4	Released as a byproduct in manufacture of viscose rayon and leather tanning; slurry tanks in piggeries; industrial paper plants using Kraft process; cigarette smoke; combustion of coal, fuel oil, and natural gas; and in municipal sewer air.
Hydroquinone	123-31-9	Used in photography, antioxidants, monomer inhibitors, dyes and pigments, agricultural chemicals, as stabilizers in paints and varnishes, motor fuels, and oils. Released from photographic processes, coal-gasification condensate water, and from the following industries: industrial organics, plastic materials and resins, pharmaceutical preparations, paints and allied products, cyclic crude and intermediates, chemical preparations, petroleum refining, electronic computing equipment, electronic components, and motion picture. Can also be released from production of polynivyl acetate and methy methacrylate. Exposure mainly from private film development.
Iprodione	36734-19-7	Fungicide. Exposure from ingestion of foods containing iprodione residues.
Iron	7439-89-6	Natural metal.
Isocyanates		See individual compounds
Diphenyl methane diisocyanate (methylene diphenyl diisocyante - MDI)	101-68-8	Used in polyurethane foam production. When sprayed outdoors droplets of unreacted isocyanate can be carried 40M downwind.
Methyl isocyanate	624-83-9	
Phenyl isocyanate	103-71-9	
Isophorone	78-59-1	Used as a solvent for natural and synthetic polymers, resins, waxes, fats, oils, and pesticides; as a chemical intermediate in the production of 3,5-Xylenol, 3,3,5-trimethylcyclohexanol, and plant growth retardants; and in pesticide formulations. Released from iron and steel manufature, photographic equipment and supplies manufacture, automobile tire plants, and in particulate emissions from coal-fired power plants.
Isopropyl methanesulfonate	926-06-7	Alkylating agent in chemical research.

(IMS)		
Isovaleraldehyde	590-86-3	Used as a flavoring, in perfumes, in pharmaceuticals, and in synthetic resins. Is a component of internal combustion exhaust. Exposure from inhalation of ambient air, ingestion of food and water, and dermal contact with vapors, food and other products containing compound.
Ketoconazole	65277-42-1	[Antifungal, antiandrogen, antineoplastic pharmaceutical (chemfinder.com)].
Lead	7439-92-1	Elemental lead is used in lead smelting and refining, storage battery manufacture, welding, and steel cutting and printing.
Linuron	330-55-2	Herbicide. Exposure from ingestion of contaminated water and vegetables, mostly carrots.
Magnesium	7439-95-4	Natural element.
Malathion	121-75-5	Insecticide. Used on crops, ectoparasites, human head and body lice, and human and pet fleas. Exposure from inhalation following spraying, and from dermal contact from household uses.
Maleic anhydride	108-31-6	Used as a chemical intermediate and in the production of resins, coatings, agricultural chemicals, and as an oil additive.
Manganese	7439-96-5	Elementary manganese: primary source from metallurgical processing. [In some paint pigments(Lesser and Weiss, "Art Hazards", 1995)]. Exposure from atmosphere.
Mercury (Elemental)	7439-97-6	Main exposure from consumption of fish, fish products, and seafood. From chlor-alkali plants; mining and refining of mercury; processing of cinnabar; manufacture and use of thermometers, batteries and electrical switches; fur-cutting and felt-hat industries; working and smelting of copper, gold, lead, silver, and zinc ores; coal-fired power plants; cement manufacture; sewage; and combustion of fossil fuels. [In sphygmomanometers, and thermostat switches. May be sprinkled in home by Hispanic-Americans practicing Santeria. Approximately 50% in dental amalgams. From fungicidal diaper rinse containing phenylmercury. Formerly (until 1991) phenylmercury was used as a preservative in interior latex paint (Etzel, "The 'Fatal Four' Indoor Air Pollutants," 2000)].
Mercury (Methyl)	22967-92-6	Produced in bottom sediments, rotten fish, and soil from inorganic Mercury by biological activity. Sources of inorganic Mercury are leaching from municipal wastes in sanitary landfills, emissions from refuse incineration, non-ferrous metal production, iron and steel production, fossil fuel combustion, chlor-alkali industry, and atmospheric fallout. Exposure from ingestion of contaminated fish and shellfish.
Metaldehyde	9002-91-9	Exposure from use as molluscicide in California citrus groves and from vapors when used as a fuel.
Metham sodium	137-42-8	[Herbicide (P65 DART agents use list)]

Methanol	67-56-1	Natural product from plants and biological wastes and sewage. Used as solvent in industry. Sources from paint thinners and strippers; adhesives; cleaners; degreasers; inks; exhaust from gasoline and diesel engines; combustion of biomass; refuse and plastics; maufacture of petroleum, charcoal, plastics, and starch; rendering; wood pulping; and tobacco smoke. Found in wood heel industry. Exposure from inhalation, dermal contact, and ingestion of various foods and water.
Methazole	20354-26-1	[Pesticide - not registered (P65 DART agents use list)]
Methoxychlor	72-43-5	Insecticide for field crops, animal houses, dairies, household, garden, and industrial premises. Exposure from inhalation of ambient air, ingestion of contaminated food and water, and dermal contact with methoxychlor. Sources from air emission where it is manufactured. Found in tobacco and tobacco products.
Methyl bromide (Bromomethane)	74-83-9	Used as a soil, fruit, and space fumigant; in food sterilization, organic synthesis, solvent in aniline dyes; as an extraction solvent for vegetable oils, nuts and seeds; and in wool degreasing. Released in auto exhaust.
Methyl chloride (Chloromethane)	74-87-3	Used as a chemical intermediate, solvent, propellant, and in manufacturing fumigants. Released from wood burning, field burning, backyard burning, tobacco smoke, turbine exhaust, and coal combustion. Formed in chlorination of drinking water and sewage; found in publically owned treatment works. Formed by photosynthesis by marine organisms. Exposure from inhalation of ambient air and ingestion of contaminated water.
Methylene chloride (Dichloromethane)	75-09-2	Used as solvent, chemical intermediate, grain fumigant, paint stripper and remover, metal degreaser, refrigerant. Used in plastic fill, furniture stripping, paint and ink, aluminum forming, coal mining, photographic equipment and supplies, pharmaceutical, organic chemistry, plastic, rubber processing, foundry and laundry industries; in production of Acetyl sulfonyl chloride and cellulose acetate fibers. Formed during chlorination of water. Detected in waste disposal facilities. Found in paint strippers, model and hobby glues, cosmetics, paints, lubricants, anti-rust products, soaps, and varnishes.
Methyl ethyl ketone	78-93-3	Used as a solvent for coatings, resins, rubbers, plastics, pharmaceuticals, adhesives, and rubber cement; in the manufacture of various chemical products; used in artificial leather and magnetic videotape industries. Released by forest fires. Exposure from use of products, inhalation of ambient air, and ingestion of contaminated food and water.
Methyl hydrazine	60-34-4	Used in chemical synthesis, as a solvent, and as rocket fuel.
Methyl iodide	74-88-4	Used as a methylating agent, but natural sources from ocean are more important contrubuter to exposure. Exposure from ambient air and from ingesting seafood from the ocean.

Methyl isobutyl ketone	108-10-1	Used as a solvent for vinyl, epoxy, acrylic, natural resins, nitrocellulose, and dyes; as an extracting agent in the prouduction of antibiotics, and in the removal of paraffins from mineral oil in the production of lubricating oils. Exposure from use of commercially available products such as paints, adhesives, pesticides (Pyrethrins), and rubber cement; and from inhalation and ingestion of food and water containing compound.
Methyl methacrylate	80-62-6	Used in polymethacrylate resins, in medicinal adhesives, dental technology, bone cements, water repellant on concrete surfaces. Exposure from ingestion of water and inhalation or dermal contact with resins, dental products, or artificial nail products.
Methyl naphthalene	1321-94-4	Component of crude oil, product of combustion from natural fires, released from petroleum refining, coal tar distillation, and gasoline and diesel fueled engines. Used as a chemical intermediate, a general solvent, and in Vitamin K production. Exposure from urban atmospheres, contaminated drinking water, and recreational activites in contaminated water.
Methylphenol (Cresol)	1319-77-3	Released in auto and diesel exhaust, tobacco smoke, sewage, from coal tar and petroleum refining, and metal refining. Used as a disinfectant, chemical intermediate, textile scouring agent, surfactant, ore flotation agent; used in the manufacture of organic chemicals,plastics, and resins.
4,4-Methylene bis(2-chloroaniline)	101-14-4	Used in curing urethane and epoxy resins. Exposure mainly occupational.
4,4-Methylenedianiline	101-77-9	Used in manufacture of methylenediisocyanate. Exposure mainly occupational.
Methylene diphenyl diisocyanate (MDI)	101-68-8	Used in polyurethane foam production. Exposure mainly occupational.
Methyl tertiary butyl ether (MTBE)	1634-04-4	Used as an octane booster in gasoline and in the manufacture of isobutene. Exposure from inhalation of ambient air (especially during refueling) and from ingestion of water.
Metiram	9006-42-2	[Fungicide (P65 DART agents use list)]
Metolachlor	51218-45-2	Herbicide. Exposure from inhalation of ambient air, and ingestion of contaminated food and water.
Mirex	2385-85-5	Insecticide for fire ants in SE US. Most uses banned in US. Formerly used as a flame retardant additive in thermoplastic, thermosetting, and elastomeric resin systems; paper; paint; rubber; electrical; adhesive; and textile products. May degrade into Chlordecone (Kepone) in environment. Exposure from residues in water, soil, food, and beverages..
Molybdenum	7439-98-7	Used in electrodes, welding, as a chemical reagent for lab analysis, in petroleum refining, chemical processing, as an intermediate in manfacture of corrosion inhibitors, paint pigments, pigments for laquers, coloring agents for enamels and ceramic glazes, agricultural chemicals, electroplating, leather and skin tanning, fire retardant resins, photograpy, formulation of plastics and adhesives, and as a catalyst in desulfurization of gasoline.
Myclobutanil	88671-89-0	[Fungicide (P65 DART agents use list)]

Nickel carbonyl	13463-39-3	Used in electroplating, nickel refining, and electronics industries. Inadvertant formation from industrial processes using nickel catalysts, such as coal gasification, petroleum refining, and hydrogenation of fats and oils. Exposure from inhalation, skin absorption, ingestion, and dermal contact.
Nickel (metallic)	7440-02-0	Used in mining and comminution of nickel containing ores; nickel refining and smelting; nickel electroplating; production and use of nickel catalysts; welding, flame spraying, cutting, grinding, and polishing of nickel alloys; manufacture of nickel cadmium batteries; nickel molds in glass bottle factories; paints; electrolysis shops. From recycling or disposal of nickel containing products. Released into food from food processing methods - leaching from nickel alloys in food processing equipment made from stainless steel, flour milling, and nickel catalyzed hydrogenation of fats and oils.
Nitrapyrin	1929-82-4	[Growth regulator (P65 DART agents use list)]
Nitrate	14797-55-8	(Potassium nitrate) Found in well water contaminated by runoff from nitrogen fertilizers, decaying matter, or sewage treatment. Used in food curing and preserving.
p-Nitrobenzamide	619-80-7	Not in HSDB
Nitrobenzene	98-95-3	Used in the manufacture of aniline, benzidine and quinoline; as a solvent; in the manufacture of soaps and shoe polishes. Exposure is mainly occupational.
4-Nitrobiphenyl	92-93-3	Formerly used in the manufacture of 4-diphenylamine. Exposure from contact with hazardous waste disposed of in the past.
Nitrogen mustard (Mechlorethamine)	51-75-2	[Gas warfare and drug (P65 DART agents use list)]
1-Nitronaphthalene	86-57-7	Used as a chemical intermediate in making 1-naphthaleneamine. Released in emissions from diesel fuel combustion. [Detected in urban airborne particles (Watt and Buckpitt, 2000)]. [Found in cigarette smoke. Most toxic of naphthalene derivatives (Paige et al, 1997)].
4-Nitrophenol	100-02-7	used in the manufacture of methyl and ethyl parathion, acetominophen and dyes. Used as a leather treatment agent. Released from vehicle exhaust from gasoline and diesel engines. It is a photooxidation product of nitrobenzene in air, and of benzene, toluene, and phenanthrene with nitric oxide in air. It is a degradation product and impurity of the parathion formulation Thiophos. Exposure from inhalation of ambient air and ingestion of contaminated water.
2-Nitropropane	79-46-9	Used as a solvent in coatings, inks and cellulose esters, stripping solvent for shellac and alquer, explosives, rocket propellants, additives to fuel for racing cars and diesel fuels. Exposure primarily occupational.
N-Nitrosodimethylamine	62-75-9	Currently used only in research. Formerly used in production of rocket fuels, as an antioxidant, additive for lubricants, and as a softener for copolymers. Released during the compounding, forming, and curing operations of elastomeric parts. Released in tobacco smoke. Exposure from inhalation of ambient air and cigarette smoke, and ingestion of contaminated food and water.
N-Nitroso-N-methylurea	684-93-5	Available only in small quantities for research.

N-Nitrosomorpholine	59-89-2	Released from manufacture of rubbers. Can be released from nitrosation of morpholine. May be formed in presence of high concentrations of acids, nitrates, and nitrates in process streams. Exposure from inhalation of air in new cars, or indoor air, possibly from cooking, ingestion of contaminated food items, infants can be exposure through use of rubber nipples, may be formed in vivo from morpoline and nitrite or nitrite precursors.
4-Nitrotoluene	99-99-0	Released as a byproduct in the manufacture of dinitrotoluene, trinitrotoluene, and azo and sulfur dye intermediates. May also be released from disposal of waste products containing compound. Exposure mainly occupational.
n-Octane	111-69-5	Highly volatile constituent in the parafin fraction of crude oil. Released durin manufacture, use, and disposal of products associated with petroleum and gasoline industries. Released from printing past, paints, varnishes, adhesives and other coatings, hazardous waste sites, landfills, incinerators, rubber production, and the combustion of gasoline fueld engines. exposure from inhalation of ambient air.
Octylphenol	27193-28-8	(4-Octylphenol) May be released from use as a chemical intermediate.
Olin 1763	5135-80-8	
Organic Carbon		
Organic Solvents		See individual compounds
Organotins		See individual compounds
Di-n-butyltindichloride	683-18-1	Used as an intermediate in the synthesis of other Dibutyltin compounds.
Di-n-octyltindichloride	3542-36-7	May be released from Polyvinyl chloride bottles.
Tributyltin	688-73-3	Used as a reducing agent and as a synthetic intermediate for other organotin compounds.
Tributyltin methacrylate	2155-70-6	[Pesticide (P65 DART agents use list)]
Tributyltin oxide - TBTO	56-35-9	Released in effluents and emissions from manufacturing plants, in spills during transport, during use and from disposal of marine anti-foulant paint, cooling-water system slimicides, latex and other paints, plastics, wood and stone preservatives, and disinfectants containing the compound. Exposure mostly from ingestion of contaminated fish and seafood and dermal contact with products containing the compound.
Triethyltin	997-50-2	Not in HSDB
Trimethyltin	1631-73-8	Not in HSDB
Triphenyltin Chloride (TPTCL)	639-58-7	Biocide in marine antifouling paints and as an intermediate in biocide production. Can be leaked to food from use as pesticide, and from storing liquids in polyvinyl chloride containers and cans. Can be released from organotin formulations, paint spraying (of organotin-based antifouling paints) and from PVC industry.
Organophosphates - see individual pesticides for specific information		See individual compounds
Oxadiazon	19666-30-9	[Herbicide (P65 DART agents use list)]
Oxydemeton methyl	301-12-2	[Insecticide (P65 DART agents use list)]
PAHs		See Benzo(a)pyrene as a model

Acenapthene	83-32-9	See Benzo(a)pyrene as a model
Acenaphthylene	208-96-8	See Benzo(a)pyrene as a mo del
Anthracene	120-12-7	See Benzo(a)pyrene as a model
Benz[a]anthracene	56-55-3	See Benzo(a)pyrene as a model
Benzo[b]anthracene (Naphthacene)	92-24-0	See Benzo(a)pyrene as a model
Benzo[b]fluoranthene	205-99-2	See Benzo(a)pyrene as a model
Benzo[g,h,I]perylene	191-24-2	See Benzo(a)pyrene as a model
Benzo[k]fluoranthene	207-08-9	See Benzo(a)pyrene as a model
Benzo[a]pyrene	50-32-8	From fossil fuels; product of incomplete combustion. From aluminum reduction, roofing shingle manufacturing, petroleum refineries, coal liquefaction, coal gas works, hot forging, tire manufacturing, rubber, carbon impregnation, metal arc wielding, boring of railroad ties, treated wood utility poles, railway ties, fire and car soot, coal tar and carbon black, open burning of rubber scrap tires, aluminum plant, aluminum and iron castings; silicon carbide, carbon anode, graphite, metal recycling, and bitumen paving plants; main and sidestream cigarette smoke, mainstream marijuana smoke, cigar smoke, gasoline and diesel engine exhaust, crude oil, fresh and used motor oils, gasoline, char-broiled steaks, processed foods, oils, margarine, butter, fat, fruit, vegetables, cereal, and roasted coffee; pyrolysis products of agar-agar, natural dyes, humectants, glues, starches, and logwood. Exposure from tobacco smoke, inhalation of contaminated air, ingestion of contaminated water (leaches from tar and asphalt linings on water pipelines) and char-broiled food.
Benzo[e]pyrene	192-97-2	See Benzo(a)pyrene as a model
Chrysene	218-01-9	See Benzo(a)pyrene as a model
Dibenz[a,h]acridine	226-36-8	See Benzo(a)pyrene as a model
Dibenz[a,j]acridine	224-42-0	See Benzo(a)pyrene as a model
Dibenz[a,h]anthracene	53-70-3	See Benzo(a)pyrene as a model
Dibenzo[a,e]pyrene	192-65-4	See Benzo(a)pyrene as a model
Dibenzo[a,i]pyrene	189-55-9	See Benzo(a)pyrene as a model
Dibenzo[a,l]pyrene	191-30-0	See Benzo(a)pyrene as a model
Dimethylbenz[a]anthracene	57-97-6	Synthesized for laboratory use. Not a product of combustion.
Fluoranthene	206-44-0	See Benzo(a)pyrene as a model
Fluorene	86-73-7	See Benzo(a)pyrene as a model
Indeno(1,2,3-c,d)pyrene	193-39-5	See Benzo(a)pyrene as a model
Methylcholanthrene	56-49-5	Used in biochemical research. [Petroleum (Brucker-Davis, 1998)].

Naphthalene	91-20-3	Present in fuel oil and gasoline. used as a chemical intermediate. Can be released from emissions and exhaust, and spills and leaks during storage, transport, and disposal of fuel oil and coal tar. In tobacco smoke and condensate, motor vehicle emissions, mineral oil, and petroleum waxes. Released from nonvented kerosene space heaters, municipal refuse incinerators, and smoke from tire fires. Emitted in production of beta Naphthol, celluloid, dye chemicals, fungicide, Hydronaphthalene, lampblack, Phthalic anhydride, smokeless powder, moth repellants, and textile chemicals; and from coal tar, petroleum refining, tannery, aluminum reduction, aluminum refinery, foundry, and silicon carbide plants. Exposure from air, especially near heavy traffic, near fumes from evaporating gasoline or fuel oil, or near petroleum refineries and coal coking operations.
Phenanthrene	85-01-8	See Benzo(a)pyrene as a model
Pyrene	129-00-0	See Benzo(a)pyrene as a model
Paraquat	1910-42-5	Herbicide. Exposure from inhalation, dermal contact, and ingestion.
Parathion	56-38-2	Former pesticide and acaricide. Exposure from inhalation of ambient air and ingestion of contaminated food. Widely detected in soil and surface water.
PBBs		Used as flame retardants. Extensively produced and widely distributed in environment.
PCBs	1336-36-3	No longer produced commercially. Currently released from landfills containing PCB waste materials and products, incineration of municipal refuse and sewage sludge, leaching of land-fill dumps, breakdown of PCB products, improper disposal of PCB materials (transformer fluid), from older transformers and capacitators that have not been replaced, and from PCB firers. Exposure from food (especially fish) and drinking water, inhalation of contaminated air, and swimming in contaminated waters. Indoor levels may be significantly higher (2-10 times) the levels in outdoor air due to transformers and electrical parts.
PCP (Pentachlorophenol)	87-86-5	Main use (now restricted) is as an industrial wood preservative for utility poles, cross arms, and fenceposts. Used in the manufacture of Sodium pentachlorophenolate, and for minor uses as a fungicide, bactericide, algicide, and herbicide for crops, leather, and textiles. Has been found in paints used on children's toys, effluents from a chlorinated biological sewage treatment plant, interior decorations, cotton t-shirts, fences and stakesm cable-drums, and recycling chips. Exposure from dermal contact and ingestion of contaminated food.
Pentachloronitrobenzene	82-68-8	Used as an intermediate, in slime prevention in industrial waters, an herbicide, and a fungicide for seed and soil treatment.Exposure from ingestion of contaminated food and water.
Perchlorate	14797-73-0	(Sodium and Potassium perchlorate) Used in jet fuel, explosives, pyrotechnics, flares, photography, infalting agent in automobile airbags, analytical chemistry, and as medication.
Petroleum distillates (Petroleum ether)	8030-30-6	Gasoline
Petroleum Hydrocarbons		Asphalt

Phenol	108-95-2	Used as a chemical intermediate, disinfectant, and antiseptic. Found in industrial waste water. Released by wood stoves and vehicle exhaust. Exposure from inhalation of ambient air, ingestion of food and lozenges, and dermal contact with disinfectants and other consumer products containing phenol.
p-Phenylenediamine	106-50-3	Used as an intermediate in the manufacture of diisocyanates for polyurethane, antioxidants and accelerators for rubber, and in dye mixtures for hair, fur, and photographic debeloping chemicals. Exposure from dermal contact, mainly through the use of hair dyes containing the compound.
Phosgene	75-44-5	Used as intermediate in organic synthesis, in dye manufacture, metallurgy, pesticides, and as a war gas. Naturally formed in atmosphere from the degradation of chlorinated compounds. Exposure mainly occupational and limited.
Phosphine	7803-51-2	Used in acetyl cellulose, bronze alloy, munitions, smoke bomb and incendiary, pesticide rat poison, fertilizer, electrolumiescent-coating, and semiconductor industries.
Phosphorous	7723-14-0	Used in acetyl cellulose, bronze alloy, munitions, smoke bomb and incendiary, pesticide rat poison, fertilizer, electrolumiescent-coating, and semiconductor industries.
Phthalates		See individual phthalates
Butyl benzyl phthalate	85-68-7	Used as a plasticizer and organic intermediate. Exposure from inhalation of ambient air, ingestion of water, and dermal contact with products containing compound.
Dibutyl phthalate	84-74-2	Used as a plasticizer, solvent for resins, fuel propellant, and insect repellant. Exposure from inhalation of ambient air, ingestion of contaminated food and water, and dermal contact with products containing the compound. [Plasticizer in PVC products, solvent in inks used in food packaging, nail polish. Of the phthalate esters associated with reproductive effects, DBP was shown to be the most common in the urine of woman of child-bearing age in the US (Foster et al, 2001)].
Di-(2-ethylhexyl) phthalate (DEHP)	117-81-7	Used as a plasticizer and as an insulating fluid in electrical transformers. Found in floor tiles, various furnishings for households and transportation vehicles, food packaging, industrial tubing and conduits, medical tubing, catheters and blood containers, dental material, coatings for drugs, commercial organic solvents, coal tar, common lab items, aluminum foil, raw sludge, carpets, defoaming agents in manufacturing paper, and as a vehicle for perfumes in cosmetics and lubricating oils. Exposure from inhalation of ambient air, ingestion of contaminated food (fish, seafood, and foods packaged in materials using compound as a plasticizer) and water, and dermal contact with products containing compound.
Diethyl phthalate	84-66-2	Used as a plasticizer, solvent for resins, wetting agent, and insect repellant. Exposure from inhalation of ambient air, ingestion of contaminated food and water, and dermal contact with products containing compound.
Diisononyl phthalate	28553-12-0	Low volatility plasticizer.

Dimethyl phthalate	131-11-3	Used as a plasticizer for nitrocellulose and cellulose acetate, resins, rubber, and in solid rocket propellants; in laquers, plastics, rubber, coating agents, safety glass, molding powders; insect repellant. Exposure from inhalation of ambient air, ingestion of contaminated food and water, and dermal contact with plastic products or insect repellants containing compound.
Di-n-pentyl phthalate (Diamyl phthalate)	131-18-0	Used as a plasticizer. Exposure from dermal contact with plastic products.
Mono-n-butyl phthalate (MPB)	131-70-4	Not in HSDB
Monoethylhexyl phthalate (MEHP)	4376-20-9	Principal metabolite of DEHP.
Phthalic anhydride	85-44-9	Used in manufacture of polyester resins, alkyd resins, phthaleins, phthalates, benzoic acid, synthetic indigo, artificial resins (glyptal), synthetic fibers, dyes, pigments, pharmaceuticals, insecticides, and chlorinated products. Manufacture of phthalic anhydride is from oxidation of xylenes and naphthalene. Emitted from production plants, incineration of industrial refuse and water sludges and slurries from plastic products, leachate from municipal and separate industrial wastes containing plastics.
a-Pinene	80-56-8	Used as a solvent, synthetic intermediate, fragrance, and flavoring. Exposure from inhalation or dermal contact of consumer products containing compound, from inhalation of ambient air, or from ingestion of foods where it occurs naturally or was added as flavoring.
POM (Polycyclic organic matter)		See individual compounds
Potassium	7440-09-7	Natural sources in earth.
Procymidone fungicide	3280-916-8	Not in HSDB
1,3-Propane sultone	1120-71-4	Intermediate in the manufacture of detergents, wetting agents, surfactants, dyes, soluble starches, cation-exchange resins, and insecticides. Exposure is mainly occupational.
Propargite	2312-35-8	[Miticide (P65 DART agents use list)]
b-Propiolactone	57-57-8	Used as a chemical intermediate. Exposure occupational.
Propionaldehyde	123-38-6	Used as a chemical intermediate in manufacture of propionic acid, polyvinyl and other plastics in the synthesis of rubber chemicals and as a disinfectant and preservative. Released from tobacco smoke, combustion of wood, gasoline, diesel fuel, municipal waste incinerators, and polyethylene. Exposure among coffee drinkers, or from contaminated water.
Propoxur	114-26-1	Insecticide and molluscide for cockroaches, mosquitoes, flies, lawn and turf insects, aphids, wooly aphids, bugs, and leafhoppers. Exposure can occur whereever it is used; exposure is particularly high indoors.
Propylene dichloride (1,2-Dichloropropane)	78-87-5	Used as a soil fumigant for nematodes, as a chemical intermediate for carbon tetrachlroide and perchloroethylene, as a lead scavenger fro antiknock fluids, solvent, ion exchange resin manufacture, paper coating, scouring, spotting, metal degreasing agent, and insecticide for stored grain. Can leach from landfills. Exposure from inhalation of ambient air, and contaminated water.

Propylene oxide	75-56-9	Used in manufacture of urethane polyols, Propylene glycol, surfactant polyols, di and tripropylene glycols, and glycol ethers; package fumigant for fruit products and fumigant for bulk quantities of several food products. Might be emitted from automobile exhaust and combustion of exhausts of stationary sources burning hydrocarbons. Exposure from inhalation of contaminated air, especially near areas of production or use, and consumption of contaminated foods.
1,2-Propylenimine	75-55-8	Used as a surface coating resin to improve adhesion. Exposure mainly occupational.
(n) Propylmethanesulfonate	1912-31-8	Not in HSDB
Pyrethroids		Allethrin: contact insecticide. Study showed about 55% of home fogger contents landed on floor. Phenothrin: insecticide. Exposure from dermal contact, inhalation, and ingestion.
Permethrin	52645-53-1	Insecticide. Exposure from household use in fly, mosquito, cockroach, and garden insect control, ingestion of contaminated foods, inhalation of dust, and dermal contact.
Resmethrin	10453-86-8	[Insecticide (P65 DART agents use list)]
Quinoline	91-22-5	Released from petroleum refining, coal mining, quenching and coking and release in shale oil and synthetic coal conversion wastewaters and in wood preservative wastewaters most likely due to creosote usage. In tobacco smoke. Exposure from tobacco smoke, particulates in urban air.
Quinone	106-51-4	Used as an oxidizing agent, in photography, in production of insecticides and fungicides, in pharmaceutical industry)cortisone and barbituate production), polymer and resins industries, as a toner and intensifier in photogrpahy, as a tanning agent for leather, in manufacture of quinhydrone electrodes. Found in tobacco smoke and wastewaters from coal industry. Exposure from inhalation of ambient air.
Quizalofop-ethyl	76578-14-8	[Pesticide - not registered (P65 DART agents use list)]
Radionuclides (including Radon)		From mining and fuel fabrication, tailings, nuclear waste,.
Selenium	7782-49-2	Natural mineral. Exposure from inhalation of ambient air, ingestion of food, milk, and drinking water, and dermal contact with consumer products containing selenium (anti-dandruff shampoos).
Sevin (Carbaryl)	63-25-2	Pesticide. Exposure from inhalation of ambient air, ingestion of contaminated food and water and pesticide products containing carbaryl.
Silicon	7440-21-3	Natural mineral.
Silver	7440-22-4	Released from mining and purification of ore. Used in silver nitrate for use in photography, mirros, plating, inks, dyes, and porcelain; as germicides, antiseptics, caustics, and analytical reagents, used in chemistry and medicine.
Sodium	7440-23-5	Natural element.
Sodium Cacodylate	124-65-2	Inadequate information in HSDB.
Sodium fluoroacetate	62-74-8	[Insecticide (P65 DART agents use list)]
Strontium	7440-24-6	Released from nuclear reactors or bomb tests.

Styrene	100-42-5	Emitted from automobile exhaust, exhaust from spark-ignition engines, oxy -acetylene flames, gases emitted from pyrolysis of brake linings, polystyrene food packaging, waste incineration, cigarette smoke, polyester resins (used in autobody fillers and casting plastics), styrene monomer and polymer production, reinforced plastics/composites plants. Released from manufacture of boats and yachts, truck parts; tubs, showers, and pipes using reinforced plastics. Exposure from ingestion of contaminated food and water, inhalation of contaminated air, cigarette smoke, use oof styrene-containing products (floor waxes and polishes, paints, adhesives, putty, metal cleaners, autobody fillers, and varnishes).
Styrene oxide	96-09-3	Used as a reactive plasticizer in epoxy resins and as a chemical intermediate. Exposure mainly occupational.
Sulfur	7704-34-9	Released from kraft mills, suar refining, copper and iron extraction, smelter gases, metallurgical processes, and petroleum refining.
Terbacil	5902-51-2	[Pesticide - not registered (P65 DART agents use list)]
Tetrachloroethane	25322-20-7	Used in production of trichloroethylene, tetrachloroethylene, and 1,2-dichloroethylene.
1,1,1,2-Tetrachloroethane	630-20-6	Propably not presently produced in US. May form incidentally during the manufacture of other chlorinated ethanes.
1,1,2,2-Tetrachloroethane	79-34-5	Used as a metal degreasing agent; paint, varnish, and rust remover; extractant; solvent; and chemical intermediate. Released from hazardous waste landfills. Exposure from inhalation of ambient air near industrial sources.
Tetrachloroethylene (PERC/PCE)	127-18-4	Used as a dry cleaning agent, degreasing agent, and chemical intermediate in the production of fluorocarbons. Used in textile, metal finishing, laundry, aluminum forming, organic chemical/plastics and municipal treatment plants. Exposure from inhalation of ambient air, and ingestion of contaminated food and water. Water can become contaminated by leaching from vinyl liners in asbestos-cement water pipelines. Small amounts can form as a byproduct of chlorination water treatment.
Tetrasul (2,4,5,4'-tetrachlorodiphenylsulfide)	2227-13-6	Not in HSDB
Thallium	7440-28-0	Used in manufacture of thallaphide cells, atomic beam clocks, photoelectric cells, lamps, thermometers, alloys, semiconductors and scintillation counters. Released from cement factories, metal smelters, and coal burning plants. Exposure from ingestion of food sources and contact with soil and dirt containing the common element.
Thiophanate methyl	23564-05-8	[Fungicide (P65 DART agents use list)]
Tin	7440-31-5	Present naturally.
Titanium	7440-32-6	Exposure mainly associated with metal, dioxide, carbide, or tetrachloride.
o-,m-,p-Tolualdehydes	1334-78-7	Used in perfumes and as flavoring agents. Exposure from inhalation of fragrances or ingestion of foods containing compound.

Toluene	108-88-3	Used in production of benzoic acid, benzene, explosives, dyes; as a solvent; in gasoline, petroleum fuels, solvents and thinners, motor vehicle exhaust (gasoline, diesel and rotary gasoline engines), crude oil, light oil from coal, municipal landfill gases, and mainstream unfiltered cigarette smoke. Exposure from inhalation of contaminated air, handling gasoline and other consumer products containing toluene, ingestion of contaminated food and water.
2,4-Toluene diamine	95-80-7	Used in production of diisocyanates, dyes, impact resins, polyimides, antioxidants, hydraulic fluids, urethane foams, and fungicide stabilizers.
Toluene-2,4-diisocyanate	584-84-9	Component of Toluene diisocyanate, which is used in the manufacture of polyurethane foam products and coating. Exposure mainly occupational.
o-Toluidine	95-53-4	Used as a dye and chemical intermediate. Exposure mainly occupational.
Toxaphene	8001-35-2	Insecticide composed of complex mixture of at least 177 chlorinated borans. Also formerly used in lakes as a piscicide. Banned in 1983. Existing stocks still used for emergency situations on corn, cotton, small grain, pineapples and bananas (Puerto Rico and Virgin Islands only), and for treatment of scabies on cattle and sheep. Exposure from inhalation of ambient air and ingestion of fish from contaminated water.
Triadimefon	43121-43-3	[Fungicide (P65 DART agents use list)]
Triazine Herbicides		See individual compounds
Amitrole (3-amino-1,2,4-triazole)	61-82-5	Herbicide. Exposure from herbicidal applications, ingestion of contaminated food and water.
Atrazine	1912-24-9	Pre- and early emergence herbicide. Most heavily used pesticide in US according to US EPA. Detected in groundwater, rivers, lakes, ponds, drinking water and food. Has been detected as far away as 100-300 kilometers from nearest treated corn field. Exposure from inhalation of ambient air and ingestion of food and drinking water.
Simazine	122-34-9	Herbicide. Exposure from ingestion of contaminated drinking water.
Trichlorfon	52-68-6	Insecticide for agricultural and household use. Exposure from household application.
1,2,4-Trichlorobenzene	120-82-1	Used as a solvent, organic intermediate, dielectric fluid, an insecticide, and dye carrier. Exposure from inhalation of ambient air, ingestion of food and drinking water.
Trichloroethane	25323-89-1	Used in vapor degreasing, cleaning of electrical equipment, motors, electronic components and instruments, missle hardware, paint masks, photographic film, printed circuit boards, various metal and certain plastics components. Interd=mediate in production of vinylidine chloride, and a s a solvent component of adhesives.

1,1,1-Trichloroethane (Methyl chloroform)	71-55-6	Used in vapor degreasing, metal cleaning, dry-cleaning, as a solvent, and as an aerosol. Found in fingernail polish, paint thinner, lubricants, caulking compounds, lacquers, antifreeze, gasoline, and "liquid paper" and "liquid paper thinner". Sources of indoor air pollution are cleaning agents, pesticides, painted sheetrock, glued wallpaper, and glued carpet. Released in leachates from municipal and industrial landfills, and in volatile emissions from landfills. Exposure from inhalation of air, ingestion of contaminated food and water, and dermal contact with products containing Methyl chloroform. [Exposure can also occur from chlorinated swimming pools, with main routes being inhalation and dermal absorption (Levesque et al, 2000)].
1,1,2-Trichloroethane	79-00-5	
Trichloroethylene (TCE)	79-01-6	Used in degreasing, semiconductor, plastics, rubber processing, appliances, jewellry, automobile, plumbing fixture, textile, paper, glass, dry-cleaning, furniture and fixture, fabricated metal, electric and electronic equipment, transport equipment, paint and ink formulation, and printing industries; operating rooms and dentistry. In fabric and leather treatments, oils, greases, lubricants, adhesive-related products (including some hobby glues), solvents, automotive products, household cleaning/polishing products, furniture strippers, photo developers, paint removers, some decaf coffee; spice extracts; Exposure from inhalation of ambient air, ingestion of food and water, and dermal contact with TCE and TCE-containing products.
Trichloromethane (Chloroform)	67-66-3	Used in production of hydrochlorofluorocarbon 22. Exposure from inhalation of ambient air, and ingestion of food and drinking water. Widely detected in chlorinated drinking water.
2,4,5-Trichlorophenol	95-95-4	Formerly used in the synthesis of preservatives, the herbicide 2,4,5-T, and fungicides. Released from chlorination of phenol-containing wastewater or drinking water, and from the bleaching process in pulp and paper mills. Exposure from ingestion of contaminated water.
2,4,6-Trichlorophenol	88-06-2	Used in manufacture of Prochloraz (fungicide) and Chloranile (bleaching agent). Formerly sed as a defoliant, herbicide, and fungicide. Released from chlorination of phenol-containing wastewater or drinking water, and from the bleaching process in pulp and paper mills. Exposure from ingestion of contaminated water.
Triethylamine	121-44-8	Used as a solvent and chemical intermediate. Exposure from inhalation of ambient air and ingestion of food containing compound, and through use of other products containing compound.
Trifluralin	1582-09-8	Used as an herbicide. Exposure from the ingestion of contaminated food.
Triforine	26644-46-2	[Fungicide (P65 DART agents use list)]
1,2,4-Trimethylbenzene	95-63-6	Used in the manufacture of tirmellitic anhydride, dyes, pharmaceuticals, and pseudocumidine. Exposure mainly occupational.

1,3,5-Trimethylbenzene	108-67-8	Used in production of dyestuff, solvent, paint thinner, and as a UV oxidation stabilizer for plastics. Exposure from inhalation of ambient air, ingestion of ood and water, and dermal contact with vapors, food, and other products containing compound.
2,2,4-Trimethylpentane	540-84-1	A volatile constituent of petroleum products and natural gas. Released from manufacture, use and disposal of products associated with petroleum and gasoline industries. Exposure from inhalation.
2,4,6-Trinitrotoluene	118-96-7	(TNT) Used as a military explosive and propellant. Exposure mainly occupational.
n-Undecane	1120-21-4	Used in orgainc synthesis, jet-fuel research, manufactur of paraffin products, the rubber industry, the paper processing industry, petroleum research, crude oil, as a solvent and distillation chaser, and in automobile exhaust. Exposure from inhalation of ambient air, ingestion of food and water, and dermal contact with vapors, food, and other products containing compound.
Urethane (Ethyl carbamate)	51-79-6	Alcoholic beverage byproduct
Valeraldehyde	110-62-3	Used as a chemical intermediate. Released from gasoline, diesel, turbine engines, burning logs, and some building products, such as carpet-covered pressed board and polyurethane-coated plywood. Exposure from inhalation of indoor and outdoor air and ingestion of food in which compound naturally occurs.
Vanadium	7440-62-2	Naturally occuring. Used as target material for x-rays, in the manufacture of alloy steels and vanadium compounds, and as a catalyst for sulfurinc acid and synthetic rubber. Exposure from inhalation of ambient air, ingestion of food and water containing element.
Vinyl acetate	108-05-4	Used as a monomer for making polyvinyl acetate and vinyl acetate copolymers, in the production of paints, sealants, coatings, and binders. Exposure from inhalation of contaminated air near sites of production, use, or disposal.
Vinyl bromide	593-60-2	Used as a flame retardant for acrylic fibers. Exposure from inhalation of air near areas of manufacture or use.
Vinyl Chloride	75-01-4	Manufacture of Polyvinyl chloride and other chlorinated compounds. Anaerobic biodegradation product of Tetrachloroethylene and Trichloroethylene. Monomers can migrate from PVC food wrappings to food. In PVC resins, cigarettes, and little cigars. Exposure from inhalation of ambient air, ingestion of food and water, and absorption through skin from cosmetics.
Vinylidene chloride (1,1-dichloroethene)	75-35-4	Used in manufacture of plastics, plastic wrap, adhesives, and synthetic fiber. Used as a degreasing agent in welding. Decomposition product of 1,1,1-trichloroethane, formed by anaerobic biodegradation of trichloroethylene. Can form in ground water contaminated with chlorinated solvents. Found in air of nuclear submarines and spacecraft. Exposure to low levels from inhalation of ambient air, indoor air, comtaminated water, and food which has been in contact with plastic wrap containing the residual monomer.
4-Vinyl-1-cyclohexene	100-40-3	Released from its manufacture. Formed from prolonged storage of Butadiene.

4-Vinylcyclohexene diepoxide (VCD) (Vinylcyclohexene dioxide)	106-87-6	Used in polymers, organic synthesis, diluent for diepoxides and epoxy resins derived from Bisphenol A and Epichlorohydrin.
Warfarin	81-81-2	[Rodenticide (P65 DART agents use list)]
Xylenes	1330-20-7	Commercial Xylene is a mixture of 2,3, and 4- Xylene. Produced and used in petroleum products, as a chemical solvent, as a synthesis reagent, and in agricultural spraying. From fuel emissions and exhausts. Found in paint shops, laboratories, hazardous waste facilities, and organic solvent recycling plants. Exposure from inhalation of contaminated air (especially in areas with heavy traffic, near filling stations, and near refineries), ingestion of contaminated water and food, and dermal contact with products containing xylene.
Zearalenone	17924-92-4	Intermediate for Zeranol, veterinary anabolic agent. Not produced commercially in the US. [Also a fungal contaminant - Fusarium - of corn and grains (Ahmed, 2000)].
Zinc	7440-66-6	Naturally occuring. Anthropogenic sources are greater than natural sources. Released from metal smelters and mining, prudction of brass, bronze, die castings metal, alloys, rubbers, and paints. Exposure from ingestion of food and water and inhalation of ambient air.

APPENDIX D

Glossary

GLOSSARY OF TERMS USED

Acetylcholine: A chemical found in vertebrate neurons that carries information across the synaptic cleft, the space between two nerve cells.
Acetylcholinesterase: An enzyme that breaks down unused acetylcholine in the synaptic cleft (the space between neurons); this enzyme is necessary to restore the synaptic cleft so it is ready to transmit the next nerve impulse.
Adrenal gland: This gland is found above each kidney, and it made up of an outer wall (cortex) that secretes important steroid hormones and an inner portion (medulla) that produces adrenaline (epinephrine) and noradrenaline (norepinephrine).
ALA (Aminolevulinic acid) inhibition: Inhibition of the formation of ALA, an important intermediate in the synthesis of the heme protein, a constituent of hemoglobin.
Alveolar buds (mammary): Glands that secrete milk in the lactating female breast.
Alveoli (respiratory): The air sacs at the ends of the tracheo-bronchial tree in which gases are exchanged between inhaled air and the pulmonary capillary blood.
Androgens: A class of male hormones (which includes testosterone and androsterone) that is responsible for the development of male secondary characteristics (deep voice, facial hair, etc.).
Anteverted: A body organ that is displaced so that the whole axis is directed farther forward than normal.
Apoptosis: Individual or single cell death by a process of self-destruction of the cell nucleus. In apoptosis, dying cells are not contiguous but are scattered throughout a tissue. Often referred to as "programmed cell death".
Asthma: A respiratory disease characterized by an increased responsiveness of the trachea (The cartillaginous tube which carries air from the mouth to the lungs) and bronchi to various stimuli, and manifested by widespread narrowing of the airways that changes in severity either spontaneously or as a result of treatment. Symptoms include recurrent attacks of wheezing, shortness of breath, cough, and a feeling of tightness in the chest.
Ataxia: A loss of normal muscular coordination.
Autism: A developmental disability significantly affecting verbal and nonverbal communication and social interaction, usually evident before age 3, that adversely affects a child's educational performance. Other characteristics associated with autism are engagement in repetitive activities and stereotyped movements, resistance to environmental change or change in daily routines, and unusual responses to sensory experiences.
Autocrine: A secretion that affects only the cell from which it was secreted.
Autoimmune disease: Disorder of the body's immune system in which the immune system mistakenly attacks and destroys body tissue that it believes to be foreign.
Axon: The threadlike extensions on a neuron, or nerve cell that conducts nerve impulses.
B lymphocyte: A cell produced by the bone marrow which, when stimulated by an antigen (a substance that causes an immune system response), forms antibodies (proteins that are able to combine with and neutralize an antigen).
B-EPP (Erythropoietic protoporphyrin): A disorder that causes the incorrect synthesis of the heme molecule, a constituent of hemoglobin.
Blood-brain barrier: A barrier made up of neuroglia (nerve tissue that provides support and insulation to neurons) and capillary walls that limits the movement of substances in the bloodstream into the brain.
Brain stem: The lowest part of the brain, which merges with the spinal cord.
Canavanine: An amino acid in alfalfa seeds.
Cell differentiation: The process by which cells become structurally and functionally specialized during development.
Cell migration: The movement of cells from one part to another in an organ or in the body.
Cell proliferation: The process by which cells undergo mitosis and divide into similar cells.
Cerebellar defects: Defects in the ability to coordinate muscle movement
Cerebellum: A posterior portion of the brain that is responsible for voluntary and involuntary motor activities based on memory and sensory input.

Chromatin: The genetic material of the cell consisting of DNA and protein.
Corpus luteum: A temporary endocrine gland that forms from the remains of the ovarian follicle after an ovum (egg) has been released, rupturing the follicle in the process. The gland can secrete both progesterone and estrogen, two female hormones which play a role during pregnancy. The gland disappears if pregnancy does not occur.
Cortical defects: Deficits in the thought process.
Cortical neurons: Neurons of the cerebral cortex, the wrinkled gray covering of the brain where thought processes occur.
Corticosteroid system: A system in the adrenal cortex that produces steroids.
Cranial nerve: A class of motor and sensory nerves that supply nerve signals to the head region.
Down regulate: Inhibit.
Encephalopathy: Any disease of the brain.
Endocrine: The organ system that regulates body functions by use of chemicals, known as hormones. Endocrine organs are the pituitary gland, parathyroid gland, thyroid gland, adrenal gland, thymus, pancreas, and gonads.
Endogenous chemicals: A chemical originating from within the body.
Endometriosis: Growth of the tissue lining the uterus (the endometrium) outside the uterus, usually in the lower abdomen. This mislocated tissue bleeds during the woman's period, causing pain and inflammation.
Epididymis: The tightly coiled, thin-walled tube that conducts sperm from the testicles to the vas deferens.
Epithelium: The layer(s) of cells between the organism or its tissues or organs and the environment. Examples include the skin cells, the inner linings of the lungs and the digestive tract, etc.
Estrogens: A steroid sex hormone that regulates female reproductive processes and creates feminine secondary sexual characteristics.
Exogenous chemical: A chemical originating from outside of the body.
Fecundity: A measure of fertility, such as sperm count or egg count.
Follicular theca: The wall of an ovarian follicle.
Follicles (ovarian): Small narrow sacs in the ovaries containing developing oocytes (proto-ovums).
FSH (Follicle-Stimulating Hormone): The hormone responsible for production of sperm in males and stimulation of the follicles in the ovary that produce the ova (eggs) in females.
GABA: An inhibitory neurotransmitter.
Gliogenesis (neurogliogenesis): The formation of neuroglial cells (cells in the brain which provide physical support to neurons, respond to injury, regulate the ionic and chemical composition of the brain, participate in the blood-brain and blood-retina barriers, form the myelin insulation of nervous pathways, guide neuronal migration during development, and exchange metabolites with neurons.)
Glycogen: A chain of glucose molecules that the body uses for energy storage; when the body has depleted the free glucose in the blood, the liver breaks down glycogen into more glucose.
Gonadotrophin Releasing Hormone (GnRH): The hormone produced and released by the hypothalamus that controls the pituitary gland's production and release of gonadotropins.
Gonadotropins: The hormones that control reproductive function, including follicle stimulating hormone (FSH) and lutenizing hormone (LH).
Granulosa cells: Cells of the *membrana granulosa* lining the vesicular ovarian follicle.
Hematopoietic system: The system involved in the formation of blood cells.
Hippocampus: A part of the brain that is important for learning and memory.
Homeostasis: The processes whereby the internal environment of an organism tends to remain balanced and stable.
Hypothalamus: A structure in the lower part of the brain that is connected to and controls the pituitary gland.
Hypothyroidism: The clinical syndrome that results from decreased secretion of thyroid hormone from the thyroid gland. It leads to a slowing of metabolic processes and in its most severe form causing edema; cretinism is the congenital form leading to abnormalities of intellectual and physical development.
Immunoglobulins: Proteins that are generated in response to and will bind and neutralize a specific antigen (any substance that stimulates an immune response. They are classified by structure and activity into five classes (IGA, IGD, IGE, IGG, IGM).
l-Tryptophan: The amino acid that is the biological precursor of L-5-hydroxytryptophan.
LH (Lutenizing Hormone): The pituitary hormone that causes the testicles in men and ovaries in women to manufacture sex hormones.

Macrophage: A type of large white blood cell that travels in the blood but can leave the bloodstream and enter tissue; it protects the body by digesting debris and foreign cells.
Mammary lobules: Sac-like area of the breast containing the alveolar buds.
Meiosis: A special method of cell division, occurring in the progenitors of sex cells, by means of which each daughter nucleus receives half the number of chromosomes characteristic of the somatic cells (all cells of the body except sperm and ova and their precursors) of the species.
Mitosis: The process of cell division that produces two daughter cells from one mother cell, all of which are genetically identical to each other.
Mitotic cell division: See mitosis, above.
Motor nucei: Groups of neural cell bodies in the central nervous system (CNS) that control motor impulses (impulses that move from the CNS to muscles or glands).
Mucin: The main constituent of mucus.
Muscarinic receptor: An acetylcholine receptor.
Mycotoxin: Any poisonous substance produced by fungi.
Myelination: The formation of the sheath of white, fatty protein (myelin) that covers and acts as an electrical insulator for nerve fibers.
Negative feedback: A type of regulatory process for a biochemical pathway in which the product of an earlier reaction inhibits the current reaction.
Neural tube: A tubular structure in the embryo which later develops into the central nervous system.
Neuronal pathways: Bundles of neurons connecting one part of the nervous system with another.
Neurotransmitter: Chemicals that move information across a synapse by diffusing across the synaptic junction, binding to receptors on the postsynaptic membrane, and stimulating generation of an action potential.
Neurotrophic signaling: Signaling pathways that mediate the release or actions of neurotrophins, Powerful molecules that affect the survival, growth, and differentiation of neurons, for example, Nerve Growth Factor (NGF) and Brain-Derived Neurotrophic Factor (BDNF).
Oligospermia: A low sperm count.
Oocyte: A developing female reproductive cell that divides by meiosis into four cells, forming one ovum that goes on to potentially become fertilized by a sperm cell (the others degenerate).
Organophosphates: Organic chemicals that contain a phosphate group. Many are highly neurotoxic, as they are capable of inhibition of the enzyme acetylcholinesterase at neural synapses. Many pesticides (Chlorfenvinphos, Dichlorvos, Mevinphos, Monocrotophos, Naled, Paraoxon, Phosphamidon, Tetrachlorvinphos, and Trichlorfon) and some warfare agents are organophosphate chemicals.
Ototoxicity: Ototoxicity refers to complications of or damage to the auditory (hearing) and/or vestibular systems due to an ingested/absorbed substance.
Pancreas: An organ behind the lower part of the stomach that makes insulin so that the body can use glucose (sugar) for energy. It also makes enzymes that help the body digest food. The pancreas also contains areas called the islets of Langerhans consisting of alpha, beta, and delta cells. The alpha cells make glucagon, which raises the level of glucose in the blood; the beta cells make insulin; the delta cells make somatostatin (a hormone that inhibits the release of human growth hormone; modulates important physiological functions of the kidney, pancreas, and gastrointestinal tract; and acts as a neurotransmitter in the central and peripheral nervous systems).
Paracrine: The secretion of a hormone or factor from anything other than an endocrine gland. This includes secretions from the autocrine system
Parathyroid: Four pea-size glands located behind the thyroid gland that secrete parathormone, which is involved in the regulation of plasma calcium levels (calcium in the blood).
Perinatal: The period before delivery from the 28th week of gestation through the first 7 days after delivery.
Peripheral neuropathy: Abnormal and detrimental changes to nervous tissue outside the brain or spinal cord.
Pineal body: A small, flat gland found within the brain which produces the hormones melatonin (a hormone believed to regulate the body's biological clock and reproductive cycles) and serotonin (a hormone that functions as a neurotransmitter and a vasoconstrictor - a substance that causes the blood vessels to narrow).
Pituitary gland: A pea-sized gland located at the base of the brain and secretes hormones related to growth and sexual development.
Polar chemical: A molecule that is charged or ionized. Polar substances are usually the easiest for the body to excrete.

Progenitor cell: Relatively undifferentiated cells of the same lineage (family type) that retain the ability to divide and cycle throughout postnatal life to provide cells that can become specialized and take the place of those that die or are lost.
Progesterone: A steroid hormone which is secreted by the corpus luteum of the ovary and is produced by the placenta during pregnancy; it regulates changes in the lining of the uterus (the endometrium).
Psychomotor: Relating to the mental origin of motor movement, to the production of voluntary movements.
Receptors: Specialized sensory nerve structures that respond to certain types of stimuli or sites on a cell (often on a membrane) that can combine with a specific type of molecule to alter the cell's function.
Retinoid: A biochemical closely related to vitamin A. Retinoids regulate growth of epithelial cells (skin, lung, and gut) and are often powerful antioxidants and cancer preventing agents. The early stages of some epithelial cancers can be converted back into normal tissue by some retinoids.
Sertoli cell: Supporting cells found in the seminiferous tubules of the testes in mammals. They surround and nourish developing sperm cells. They secrete androgen-binding protein and establish the blood-testis barrier.
Somatic cell: Any cell in the body except sperm and ova and their precursors.
Spermatocytes: Precursor cells to sperm.
Spermatogenesis: Process of formation and development of sperm.
Spermatogonia: The primitive differentiated male gametes which give rise to spermatocytes.
Stem cell: Relatively undifferentiated cells of the same lineage (family type) that retain the ability to divide and cycle throughout postnatal life to provide cells that can become specialized and take the place of those that die or are lost.
Stromal interstitial cell: Connective tissue cells of an organ found in the loose connective tissue.
Synaptic pruning: A process in which the brain removes ineffective connections between neurons to make the remaining connections more efficient.
Synaptogenesis: The formation of the connections between two neurons or a neuron and a connection with a muscle cell.
T lymphocyte: Lymphocytes (white blood cells involved in immune response) responsible for cell-mediated immunity. When exposed to an antigen, they divide rapidly and produce large numbers of new T cells sensitized to that antigen.
Thymic involution: The return of the thymus to normal size.
Thymus gland: A gland necessary for normal development of immunologic function early in life. By puberty, it begins to involute and much of the tissue is replaced by fat.
Thyroid: An endocrine gland found at the base of the neck which secretes hormones that regulate aspects of metabolism and growth.
Xenobiotic: A chemical foreign to the body and is not present in the body under normal conditions.
Xenoestrogen: Xenobiotics that mimic the effects of estrogens.

APPENDIX E

CALIFORNIA HEALTH AND SAFETY CODE SECTION 901

(a) As used in this section:

(1) "Center" means the Children's Environmental Health Center established pursuant to Section 900.

(2) "Office" means the Office of Environmental Health Hazard Assessment.

(b) On or before June 30, 2001, the office shall review cancer risk assessment guidelines for use by the office and the other entities within the California Environmental Protection Agency to establish cancer potency values or numerical health guidance values that adequately address carcinogenic exposures to the fetus, infants, and children.

(c) The review required by subdivision (b) shall include a review of existing state and federal cancer risk guidelines, as well as new information on carcinogenesis, and shall consider the extent to which those guidelines address risks from exposures occurring early in life.

(d) The review required by subdivision (b) shall also include, but not be limited to, all of the following:

(1) The development of criteria for identifying carcinogens likely to have a greater impact if exposures occur early in life.

(2) The assessment of methodologies used in existing guidelines to address early-in-life exposures.

(3) The construction of a data base of animal studies to evaluate increases in risks from short-term early-in-life exposures.

(e) On or before June 30, 2004, the office shall finalize and publish children's cancer guidelines that shall be protective of children's health. These guidelines shall be revised and updated as needed by the office.

(f) (1) On or before December 31, 2002, the office shall publish a guidance document, for use by the Department of Toxic Substances Control and other state and local environmental and public health agencies, to assess exposures and health risks at existing and proposed schoolsites. The guidance document shall include, but not be limited to, all of the following:

(A) Appropriate child-specific routes of exposure unique to the school environment, in addition to those in existing exposure assessment models.

(B) Appropriate available child-specific numerical health effects guidance values, and plans for the development of additional

child-specific numerical health effects guidance values.

(C) The identification of uncertainties in the risk assessment guidance, and those actions that should be taken to address those uncertainties.

(2) The office shall consult with the Department of Toxic Substances Control and the State Department of Education in the preparation of the guidance document required by paragraph (1) in order to ensure that it provides the information necessary for these two agencies to meet the requirements of Sections 17210.1 and 17213.1 of the Education Code.

(g) On or before January 1, 2002, the office, in consultation with the appropriate entities within the California Environmental Protection Agency, shall identify those chemical contaminants commonly found at schoolsites and determined by the office to be of greatest concern based on criteria that identify child-specific exposures and child-specific physiological sensitivities. On or before December 31, 2002, and annually thereafter, the office shall publish and make available to the public and to other state and local environmental and public health agencies and school districts, numerical health guidance values for five of those chemical contaminants identified pursuant to this subdivision until the contaminants identified have been exhausted.

(h) On and after January 1, 2002, and biannually thereafter, the center shall report to the Legislature and the Governor on the implementation of this section as part of the report required by subdivision (d) of Section 900. The report shall include, but not be limited to, information on revisions or modifications made by the office and other entities within the California Environmental Protection Agency to cancer potency values and other numerical health guidance values in order to be protective of children's health. The report shall also describe the use of the revised health guidance values in the programs and activities of the office and the other boards and departments within the California Environmental Protection Agency.

(i) Nothing in this section relieves any entity within the California Environmental Protection Agency of complying with Chapter 3.5 (commencing with Section 11340) of Part 2 of Division 3 Title 2 of the Government Code, to the extent that chapter is applicable to the entity on or before July 19, 2000, or the effective date of Section 57004.