Appendix C

Results of the EPA Study Related to the Habitability Decisions

Summary

Until there is agreement about the possible level of chemicals in samples that contained no detectable concentrations, **it is pointless to dwell on the quantitative aspects of health risk posed by chemicals from Love Canal. If the concentrations are in the parts-per-billion (ppb) range, the risk has to be judged to be very low and probably acceptable. If the concentrations for some chemicals are 1,000 times higher, in the parts-per-million (ppm) range, the risks are probably not acceptable. According to the Environmental Protection Agency (EPA), the concentrations are in the ppb range; according to the National Bureau of Standards (NBS), they could near 1 ppm.**

OTA does not agree that the ppm estimate is realistic for all chemicals, and it tends to accept EPA's estimates, but OTA does agree with NBS that further documentation from EPA is necessary to settle the matter. A resolution between EPA and NBS might be reached by an examination of a subset of EPA's records. Also, if additional monitoring is carried out before or during rehabitation of the emergency declaration area (EDA), EPA should consult with NBS to ensure that quality control measures are adequate.

Basis of the Habitability Decision

The major input to the habitability decision was data generated by the EPA monitoring study.[1] The Department of Health and Human Services (DHHS) used absolute concentrations of chemicals found in EDA and assessed the relationship of these concentrations to potential health problems. DHHS also reviewed data about health problems observed in EDA and Love Canal residents and used professional judgments about possible human health effects resulting from exposure to chemicals deposited in the canal landfill.

The OTA review concentrated on the EPA monitoring data and possible health effects associated with Love Canal chemicals. OTA inspected but did not evaluate the validity of reported health problems of residents nor question the professional judgments of the DHHS officials. The results of the OTA analysis indicate three areas where uncertainties in the data could have a major impact on the DHHS decision. These areas include:

1. the range of variability associated with values reported for chemicals detected, nondetected, and trace;
2. uncertainties in potential health effects associated with Love Canal chemicals; and
3. problems associated with comparing data for the EDA with data in control areas.

Problems With Statistical Comparisons of EPA Results

A major statistical problem is related to the small numbers of controls used in the EPA analysis.[2] The power to detect differences in contamination between the EDA and control areas and between Love Canal and control areas has been questioned.[3] These criticisms that the canal cannot be distinguished from the control are accepted as valid by EPA. This creates uncertainty for a conclusion that the EDA is as habitable as the control areas to which it was compared. Silbergeld has attacked the EPA monitoring study on insufficiency of statistical power:

> The small number of control area sampling sites seriously reduced the ability to detect differences in chemical contamination between the Declaration Area and the control area.

The absence of power to distinguish between the canal and the control areas seriously compromises any conclusions to be drawn from comparing Love Canal to the EDA and EDA to the control areas because, in most cases, statistically there are no differences between Love Canal and the control areas. The absence

*D. Rail, National Institute of Environmental Health Science, Research Triangle Park, N. C., and B. Paigen, Children's Hospital, Oakland, Calif., personal communications, May 1983. See *Environmental Monitoring at Love Canal: Interagency Review*, comments by DHHS, NBS, and EPA (Washington, D. C.: U.S. Environmental Protection Agency, Office of Research and Development, May 1982).

[2]E. Silbergeld, Environmental Defense Fund (EDF), testimony before the Subcommittee on Commerce, Transportation, and Tourism, Committee on Energy and Commerce, U.S. House of Representatives, 97th Cong., serial No. 97-197, August 1982, pp. 68-103.

[3]R. J. Cook, testimony before the Joint Public Hearing on Future Uses of the Love Canal Hazardous Waste Site and Adjacent Property, State of New York, Assembly Standing Committee on Environmental Conservation, Assembly Subcommittee on Toxic and Hazardous Substances, Feb. 17, 1983.

of statistical power to distinguish between the Love Canal and control areas results from the small number of control area samples, and nothing can be done at this time to make up for that deficiency. It is important to remember that differences almost certainly exist in chemicals actually present in the Love Canal and control areas. But the differences cannot be shown because of too few control area samples.

Independent analysis of the EPA data shows that the greatest number of samples analyzed from any one medium in the control area was 33, compared to 539 in the EDA.[4] For every chemical tested there were fewer than 10 samples in a majority of the media. Table C-1 describes the number of samples needed to have a good chance of detecting differences between two areas. Formally, this table gives required sample size for a one-sided, alpha 0.10, Z-test on two proportions to achieve a power of **0.90.** In less formal language, if the real frequency of positive detections of chemicals in the control sample is equal to 5 percent (0.05) and the positive detection rate in the EDA is equal to **20** percent, a minimum of **61** samples from each region must be analyzed to have a 90-percent chance of detecting this (fourfold) difference. Because the maximum number of samples analyzed in the control region was 33 from any one medium and most of the time it was only 10 samples, even a fourfold difference in chemical detection rates would not be recognized. Because differences in detection rates between the EPA and control area are much smaller than these values, statistical significance between the EDA and controls could not be expected.

OTA concludes that any decision based on differences in detection frequencies between the Love Canal, EDA, and control area must be discounted because of the weak statistical basis of the EPA study. EPA apparently agrees with this assessment and asserted that making such comparisons is not the normal way to judge whether an area is contaminated.[5] Rather, EPA would rely on measured absences of chemicals to show the area is not contaminated. Some type of baseline data, however, are needed to make such judgments. These baseline measurements could be either control area analyses or established environmental standards. Unfortunately, few of the chemicals disposed in the canal landfill have established environmental standards.

Range of Variability for Reported Values

Except for some compounds detected in sumps and storm sewer systems, concentrations of chemicals reported for the Love Canal region were generally quite low, as illustrated in tables C-2 and C-3. The maximum values reported for organic chemicals detected in the EDA, Love Canal, and control regions range from **0.05** to **263** ppm. In the Love Canal, very high concentrations were reported for sump sediment **(16,500** ppm); however, these samples were taken from sumps of homes that had been built directly adjacent to the canal landfill. Table C-3 provides reported maximum values for those media where dioxin was detected. These values were 672 ppb found in storm sewer sediment within the EDA and 37 ppb detected in surface water sediment, also in the EDA. For all other environmental media, results of dioxin analyses were below EPA's reported detection limits (20 ppt). In the Love Canal, very high values of dioxin were reported for sump and storm sewer sediment. No values were reported for the control areas.

Table C-1 .—Number of Samples Required To Detect Actual Differences Between the EDA and Control Areas

Detection rates		Number of samples/per medium to produce a W-percent chance of detecting a statistical difference
Control	EDA	
0.03[a]	0.06[b]	625
0.03	0.09	203
0.03	0.12	110
0.05	0.10	362
0.05	0.15	115
0.05	0.20	61

[a]The frequencies of detection in control area samples was between 3 and 5 percent

[b]Assumed detection rate in the EDA.

SOURCE: Cupples, op. cit.

Although these measures appear low, their absolute values for organic compounds can be questioned. The EPA monitoring study used 19 different analytical laboratories, each with varying capabilities.' NBS was asked by EPA to review the quality control protocols used in the study.[7] While NBS accepted the protocols as adequate, the Bureau could not verify the certainty associated with performance of the different laboratories. As stated in a letter to Senator A.M. D'Amato:[8]

[4]L. A. Cupples, Boston University School of Public Health, report submitted to OTA, Industry, Technology, and Employment Program, May 1983.

[5]Statements made by EPA officials during a meeting with OTA on May 12, 1983.

[6]*Environmental Monitoring at Love Canal* (Washington, D. C.: Environmental Protection Agency, vol. I, pp. 36-37.

[7]*Environmental Monitoring at Love Canal: interagency Review,* op. cit.

[8]R. G. Kammer, letter to Senator A. M. D'Amato, August 1982.

Table C-2.-Maximum Vaiues (in ppm) Reported for Organic Compounds

Shallow well	EDA	0.048 di-n-octylphthalate
	Control	0.150 di-n-octylphthalate
	Love Canal	3.300 3-chlorotoluene
Deep well	EDA	0.230 phenol
	Control	0.105 xylene
	Love Canal	0.050 acrolein
Soil	EDA	3.120 chrysene
	Control	0.420 benzene
	Love Canal	10.485 1,2,3,4-tetrachlorobenzene
Sump water	EDA	0.586 1,4dichlorobenzene
	Control	0.002 Aroclor 1254
	Love Canal	8.500 2,4-dichlorophenol
Sump sediment	EDA	—
	Control	—
	Love Canal	16,523 2,4dichlorotoluene
Storm sewer water	EDA	0.062 1,2,4,5-tetrachlorobenzene
	Control	0.0001 gammma-BHC
	Love Canal	0.120 hexachlorobutadiene
Storm sewer sediment	EDA	123.000 di-(2-ethylhexyl)phthalate
	Control	0.012 1,2-dichloroethane
	Love Canal	263.000 Aroclor 1254
Surface water sediment	EDA	20.000 delta-BHC
	Control	23.645 di-(2-ethylhexyl)phthalate
	Love Canal	—

SOURCE: U.S. Environmental Protection Agency, op. cit., vol. III

Tabie C-3.–Maximum Vaiues Reported for Dioxin, ppb

Media	EDA	Love Canal	Control area
Sump water	—	.6	–
Sump sediment	—	9570	–
Storm sewer sediment	672	329	–
Surface water sediment		37.4	— —

SOURCE: U.S. Environmental Protection Agency, op. cit., vol. III.

As we reported in our May 10, 1982 review, unless measured values, including non-detected, are accompanied by estimates of uncertainty, they are incomplete and of limited usefulness for further interpretation and for drawing conclusions.

EPA's response to NBS was to provide a "worst case" range for selected chemicals based on performance of the worst laboratory.[9] While this provides some idea about the variability for these particular chemicals, it does not allow estimation of confidence limits for the total set of 150 chemicals used in the study.

As mentioned in appendix B, a major failing of the EPA effort was the improper use of replicates. For most of the environmental media, no replicate samples were taken at individual sites. In those few instances where replication was obtained, EPA treated them as separate samples. Thus, there is no way to determine if the absolute values reported for any one chemical varies by twofold, tenfold, or 100-fold.

This concern is not trivial. All samples collected from environmental media will vary to some extent. Because of the inherent variability of ecosystems and the variations in interactions between chemicals and elements of the environment, a minimal level of uncertainty can never be overcome. An additional level of variability results during the analytical phase of a monitoring study. Such analytical variations arise when different people perform the same procedure in the same laboratory. Even greater variability is introduced when different laboratories with different capabilities, experience, and equipment* are used in the same study.

In addition to uncertainties associated with the absolute values reported for chemicals detected within the EDA, there is uncertainty associated with the detection limits of the various laboratories. Within the EDA, 90 percent of the analytical measurements were below the laboratory detection limits. The obvious question is raised. Are the low values real or could they result from limitations of the various laboratories? If detection limits were insensitive (i.e., too high), concentrations significant for potential health problems may be overlooked. If detection limits were too variable, then extent of contamination may not be accurately identified. The adequacy of EPA's reported detection limits for analytical methods is difficult to

[9] *"Environmental Monitoring at Love Canal: Interagency Review,* op. cit.

● Although the same brand name of equipment can be used by different laboratory=, performance **differences between similar equipment can be expected.**

evaluate, in part because of the conceptual complexity of detection limits.

Method detection limits (MDL) were used by EPA. The MDL is the lowest concentration of a substance that can be detected with a 99 percent confidence that the reported concentration is greater than zero.[10] EPA regarded detections reported as "trace" to be above the MDL for a particular analytical laboratory .[11] Therefore, the upper limit for the frequency of reporting false positive (or false negative) readings should be 1 percent, i.e., one might not be able to determine with 99 percent confidence that an undetected substance is, indeed, absent in a sample at concentrations below the MDL. OTA finds that this is subject to uncertainty.

EPA reported detection limits for only a sample of its target substances, on the grounds that this select group was representative of each structural class present among the target substances. However, it is difficult to determine whether MDLs accurately reflect routine practices of particular analytical laboratories. As EPA acknowledged, except in the case of air samples, analytical laboratories knew which samples were performance evaluation samples.[12] Performance evaluation samples could, therefore, have been analyzed more carefully than on the field samples.

Moreover, a different MDL was reported for particular substances representing more than one analytical method, and up to six analytical laboratories. In some cases, MDLs were estimated for several laboratories based on the performance of only one. These factors contribute to uncertainty about detection limits.

Reported MDLs for metals were estimates only, and were reported as aggregated value, which obscures variability among laboratories. Also, analysis for pesticides have only a single reported MDL, again obscuring laboratory variability. In contrast, a MDL for many organic substances was reported as a measured limit for specific laboratories.

Although most reported detection limits are quite low relative to health standards (where available), the issue of uncertainty of detection limits is relevant to the issue of the validity of EPA results. For example, beta-BHC is reported to have an overall (low) MDL of 0.006 ppb for analytical Method 608 in reagent water. The same substance is reported to **have** MDLs that range from 4.2 to 9.5 ppb using analytical Method 625. The actual value depends on which laboratory performed the analysis. Thus, if different methods or different laboratories were used to analyze for beta-BHC, then intended comparisons of frequencies of beta-BHC detections among components of the environment might represent comparisons of methods and of laboratory performance. Although, in conversation, EPA has asserted that this should not happen, no mechanism for reliably preventing it was presented. It should be emphasized that neither of these values may be relevant to detection limits of actual samples as the Love Canal samples which would contain competing contaminants that possibly lower analytical power.

Such variability in MDLs is not atypical. Detection limits for other compounds also varied widely. For example, reported MDLs for 1,2,3,4-tetrachlorobenzene ranged from **0.5** to 17 ppb, varying by a factor of **34** for different laboratories employing the same analytical method. Detection limits for several closely related substances, the alpha, beta, delta, and gamma isomers of BHC, varied from 0.004 to 0.009 ppb, a factor of **2.25.** Likewise, detection limits for DDT, DDD, and DDE varied by a factor of 3 (0.004 to **0.12** ppb). MDLs for endosulfan 1, endosulfan 2, and endosulfan sulfate varied from 0.004 to 0.066 ppb (a factor of 16.5), and for heptachlor and heptachlor epoxide MDLs varied by a factor of 27.7 (from 0.003 to 0.083 ppb).

MDLs were reported for only a subset (about one-third) of the total 150 chemicals; EPA considered that the subset of compounds spanned the range of compound classes used in the study .[13] Consequently, EPA asserts that it should be possible to determine approximate detection limits for all substances:[14]

> Similarly, it is reasonable to assume that the method detection limits of most of the organic analytes . . . fall into the same range of **0.5 to 79 micrograms per liter.**

Nevertheless, none are provided for substances known to have been disposed into Love Canal.

This variability in detection limits introduces uncertainties in interpreting the meaning of the many samples reported to be below the limits of detection. This uncertainty in turn casts doubt on any conclusions about the levels of contamination of the EDA or the control area.

All reported MDLs are in or below the range of 0.5 to 79 ppb. The observed variability of MDLs across methods as well as for similar compounds and the lack of MDLs for most of the target chemicals calls into question the ability to detect hazardous concentrations

[10] J. A. Glaser, et al., "Trace Analyses of Wastewaters," *Environmental Science and Technology*, vol. 15, 1981, pp. 1426-1435.
[11] *Environmental Monitoring at Love Canal*, op. cit.
[12] Statements made by EPA officials at a meeting with OTA, May 12, 1983.
[13] Deegan, op. cit., "it is true that MDL's were determined for a subset of the target compounds, and the subset included model compounds for the complete set of target compounds . . a valid methodology . . . accepted widely in scientific research," p. 147.
[14] *Environmental Monitoring at Love Canal*, op. cit., vol. I, p. 228.

of the target substances. Were the detection limits for each compound sufficiently low and were actual laboratory performances sufficiently high to allow a conclusion that those chemicals not detected would be present in such low levels as not to pose a threat to human health? Is the range of variability for MDLs sufficiently low **to be certain that estimates of variance for absolute concentrations are not within hazardous concentrations for all target substances? Until MDL values are reported with estimates of variances for each, uncertainties about the meaning of none detected and trace, remain.**

What If EPA's Numbers Are Wrong?

Most of the samples in which EPA detected measurable amounts of chemicals revealed concentrations in the ppb range. If those numbers are accurate, the assumption can be made that the samples in which only traces of chemicals were detected or in which not even traces were detected contain even lower concentrations of chemicals. Looking at the data reported later in tables C-5 and C-6, it can be seen that if the trace measurements are in the ppb range, the levels of chemicals in the EDA are indeed so low as to pose an acceptable health risk (except for hexachlorobenzene and dioxin).

NBS was asked to comment on the amount of chemicals that might have gone undetected in EPA's monitoring program at Love Canal. NBS was not convinced that the absence of detectable levels of chemicals in the EPA analysis **was** consistent with concentrations as low as parts per billion.[15] Instead, it is confident that the concentration of a chemical reported to be below detection is no more than 1 part per million.

OTA asked officials of DHHS who participated in making the habitability decision if they would persist in their conclusion that the declaration area **was** habitable if many chemicals were present in the near 1 ppm range. The response was that they would stick by their earlier decision with a demur about certain chemicals.

OTA would not be so sanguine about the safety of the EDA if the concentration of all or most of the 150 chemicals approached 1 ppm. If the NBS estimate that the "no detectable limit" might be as high as 1 ppm is applied to monitoring of drinking water, then every limit shown on table C-5 would be exceeded in the EDA. If the conservative NBS estimate is not applied to drinking water because it is to be expected that drinking water would be cleaner than other waters and soils, finding concentrations in the 1-ppm range in other media would still show that contamination of the EDA was widespread. In that case, the chance of human exposure would have to be reckoned as substantial.

Some toxic chemicals exhibit "synergism," i.e., the toxic effect of simultaneous or sequential exposure to two (or more) chemicals greatly exceeds the toxic effects predicted from adding together the effects of the individual chemicals. Without consideration of synergism and with consideration of only additive effects of chemicals, OTA would not consider the EDA habitable if many of the 150 chemicals were present at concentrations near 1 ppm. For instance, if 10 carcinogens are present in concentrations such that each one poses a 1 in 100,000 chance of a person developing cancer, then the 10 together may pose a 1 in 10,000 risk, which may well be so high as to be unacceptable. For the very reason that so little is known about carcinogenic potentials and other toxic potentials, OTA would come to the conclusion that the uncertainties about health effects from many chemicals being present at near 1 ppm each would preclude considering the declaration area to be habitable.

However, it is impossible to interpret the NBS opinion as supporting the idea that all chemicals for which MDLs were reported might be present in concentrations near 1 ppm. First, the ability of laboratories to detect chemicals varies from substance to substance. The basis of the NBS conclusion, that no concentrations higher than 1 ppm would have gone undetected must be based on consideration of the properties of the chemicals most difficult to detect and measure. Therefore, the MDLs for chemicals that are more easily detectable must be lower, perhaps in the low ppb range claimed by EPA. The second reason is that there is little reason to believe that all 150 chemicals monitored by EPA were actually present in significant amounts in the Love Canal dump. Therefore, to assume that all the 150 chemicals could be present at concentrations of up to 1 ppm poses an immediate question about the origin of all these chemicals. OTA's concentration on a subset of chemicals known to be present in the landfill eliminates the problems associated with assigning a possible concentration to chemicals that are not present.

It would be a tedious task for EPA to supply estimates of variance to support the contention that all MDLs were in the low ppb range. However, it might be a manageable job for EPA to examine the records for the 16 or so chemicals known to be in the landfill in significant amounts. Because the argument about possible health effects hangs on knowing the absolute concentrations of chemicals in the EDA, further analysis of the EPA data *seems* worthwhile.

[15] Krammer, op. cit.

Uncertainties in Potential Health Effects

Adverse health effects from exposures to toxic substances are conveniently divided into two broad groups: acute and chronic. *Acute* effects are observed soon after exposure, typically to relatively large concentrations of toxic materials. For example, when a ruptured train car spilled nitric acid and the wind carried fumes into residential areas of Denver, Colo., people experienced choking and difficulty in breathing. Less spectacular and more common effects include eye irritations from air pollutants during periods of poor air quality.

Acute toxic effects are marked by the body's responding to an insult from ingestion, inhalation, or dermal exposure to a substance. When the insult is removed, the affected cells and organ systems of the body may recover, may die, or maybe replaced. Importantly, new body cells, those formed after the insult has passed, are not affected.

Chronic toxic effects encompass three dread events: mutations, cancer, and birth defects. These may result from exposure to extremely low concentrations and may not be observed until years after the exposure occurred. (Neurological disorders, which may be caused by low doses of some materials, e.g., lead, are also chronic health effects, but they are not discussed in this paper.) Mutations and cancer differ from acute toxic effects in that cells are altered genetically and the damage caused is perpetuated in progeny cells formed from the one originally harmed. In contrast, birth defects that result from in utero exposure of the fetus to chemicals do not necessarily involve genetic alterations. Some may result from biochemical changes in critical developmental processes. Because most concerns about health risks to EDA residents has centered on chronic effects, the OTA review focused on mutations, cancer, and birth defects.

Mutagenesis and Cancer

Mutagenesis —the causation of mutations—is the best understood chronic effect, from the standpoint of mechanism. An environmental contaminant interacts with the DNA of a germ (reproductive) cell and alters the genetic information within it. If that germ cell, an egg or a sperm, is involved in the formation of an organism, every cell in the new organism will bear the alteration, the mutation. If this organism has progeny, half of those progeny, on average, will bear the mutation. Thus, mutations are chronic in the sense that once introduced into a population they may be propagated in every succeeding generation. Some mutations are beneficial, but most of those that are detected in humans are associated with deleterious effects.[16]

Cancer also involves an interaction between a contaminant and DNA, but the mutational event occurs in a somatic, or body, cell rather than in a germ cell. Thus, the mutation is not passed on to the next generation. Instead, a mutation that results in cancer causes rapid proliferation of cells. The rapidly growing cells, all of which may derive from a single mutated cell, in turn, produce a tumor.[17]

Many mutational events, whether they occur in somatic or germ cells, may have no effect because they cause changes in DNA without biological consequences. Others may produce small but undetected changes, either beneficial or detrimental. Although it is likely that only a few DNA changes produce a detectable mutation or tumor, our awareness of mutational events in humans has been heightened by increasing knowledge of their sometimes devastating effects.

Methods for Identifying Health Effects

During recent years, much effort has been expended in identifying carcinogens, agents that cause cancer.[18] The methods used for identifying cause-effect relationships between manmade or natural substances and toxic effects in humans can be illustrated by a discussion of the methods used to identify carcinogens. Effects from carcinogens (and toxic substances, in general) can be identified through results of epidemiology—the study of diseases and their causes in human populations—and various laboratory tests.

Epidemiology is the only method that establishes associations between a substance and human toxicity. However, it is limited as a technique for identifying chronic effects that appear years or decades after exposure, because people are difficult to study, move from place to place, change their work environment, and change their living habits. Also, it is hard to locate those people who may have been exposed to a particular carcinogen several years previously. Estimating past exposures to suspect agents is very difficult.

Testing suspected chemicals in laboratory animals, generally rats and mice, is the backbone of current toxic substances identification. A chemical is administered to animals either in their food, water, air, or (less frequently) by force feeding, skin painting, or injection.

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[16]For examples see *The Rok of Genetic Testing in the Prevention of@cupational* Disease (Washington, D. C.: U.S. Congress, Office of Technology Assessment, OTA-BA-195, April 1983).

[17]*Assessment of Technologies for Determining Cancer Risks* from *the Environment* (Washington, D. C.: U.S. Congress, Office of Technology Assessment, OTA-H-138, June 1981.

[18]Ibid.

The animals are observed over a specified time period to identify acute or chronic effects.

The reliability of animal tests, bioassays, depends on their design and execution. Guidelines for cancer bioassays were published by the National Cancer Institute in 1976. Bioassays now cost between $100,000 and $1 million and require up tos years to complete. Clearly such expensive tools can be used only to test highly suspect chemicals, and much effort is devoted to selecting chemicals for testing.

Molecular structure analysis and examination of basic chemical and physical properties are used to make preliminary decisions about the likelihood of a chemical being toxic and whether or not to test it. For instance, greater suspicion is attached to chemicals that share common features with identified toxic substances. "Paper chemistry" and "paper toxicology" are used most extensively to estimate properties of new chemicals.[19] Unfortunately, not all chemicals within a single structural class behave similarly; thus, limits are placed on the use of these approaches.

New developments in laboratory testing has resulted in the greater use of short-term (a few days to months) tests. Test costs range from a few hundred dollars to a few thousand. Such tests depend *on* measuring biological interactions between chemical and DNA. The best known test, the "Ames test," measures mutations in bacteria. Other short-term tests use nonmammalian laboratory animals, as well as cultured human and animal cells. Some tests measure mutagenicity; others measure either the capacity of a chemical to alter DNA metabolism or to transform a normal cell into one with abnormal growth characteristics. Problems in interpreting mutagenicity tests arise from the ease of doing them; the possibility of false-positive tests increases with the number of tests that are done, and since negative tests frequently are not reported, there is some danger of overrelying on positive test results. Further complicating interpretation, a substance may test out as a mutagen in one assay system and not in another.

A critical problem in estimating human health effects is the need to extrapolate results from studies involving large concentrations of chemicals to expected results from exposure to low levels actually seen in the environment. The idea of dose response (that the percentage of people suffering adverse effects will decrease at lower exposures) is well accepted. However, the exact relationship between dose (exposure levels) and responses (numbers of affected people) is disputed. In particular, some knowledgeable observers argue that there are doses of chemicals so low that they will cause no disease. In other words, a threshold has to be exceeded before any adverse effects will be seen. In general, thresholds are better accepted for acute effects than for chronic effects. In particular, if the interaction between a single molecule of a chemical and DNA is sufficient to produce a mutation, no threshold value is likely for mutagenic and carcinogenic effects.

The problems of extrapolation are more complex when data from laboratory studies are the only available information. In those cases, a method must be chosen to translate the meaning of a toxic effect in the animal to an expected toxic effect in humans. Almost everyone accepts that animal results are important to predicting human effects; toxicology is based on that premise. However, there can be endless arguments about the applicability of a particular animal test.

A 1979 IARC report summarized the agency's analysis of 354 chemicals and chemical processes that it had reviewed in its program, which began in **1971.**[20] IARC found sufficient epidemiologic information to evaluate carcinogenicity in humans for fewer than 100 chemicals. For 18 of those, IARC considered that the evidence was sufficient to support a conclusion that the chemical causes cancer in humans. For an additional 18, the evidence was sufficient to support a conclusion that the agent was a probable human carcinogen. In the cases of the remaining 318 chemicals and chemical processes, the data from human studies were insufficient to support a conclusion that the substance or process is a human carcinogen or a probable human carcinogen.

IARC also reviews the worldwide literature about the testing of chemicals for carcinogenicity in animals. About animal tests, it says:

> . . . **in the absence of adequate data in humans it is reasonable, for practical purposes, to regard chemicals for which there** is *sufficient evidence* **of** carcinogenicity (i.e., a causal association) **in animals** *as if they presented a carcinogenic risk for humans.*

IARC has reviewed the literature about the testing of 354 chemicals in animals. For **142** of those, IARC considered that the animal evidence was "sufficient," and that those substances should be considered to pose a carcinogenic risk for humans. In 1980, scientists at the National Cancer Institute (NCI) reviewed all of the cancer tests carried out in animals there.[21] In the cases where both IARC and NCI evaluated data about the same chemicals, the results of the two organizations' analysis were generally consistent .22

[19] **OTA, The *Information Content of Premanufacture Notice,* OTA-BP-H-17, Government Printing Office, Washington, D. C.: 1983.**

[20] Chemicals *and Industrial Processes Associated With Cancer in Humans,* **Monographs, Supplement 1, (Lyon, France: IARC, 1979).**

[21] **R. A. Griesemer and C. Cuets, Jr., "Toward a Classification Scheme for Degrees of Experimental Evidence for Carcinogenicity of Chemicals for Anireals, " in *Molecular and Cellular Aspects of Carcinogen Screening Tests, I-I.* Bartsh and L. Tomatis (eds.), Lyon France, 1980.**

[22] **OTA, 1981, op. cit.**

Comparing the relatively small number of chemicals for which human evidence is available to the larger number of chemicals which has been tested in animals illustrates the importance of animal tests. The number of tested chemicals is much smaller than the number of all chemicals, and there are major efforts underway to use "short-term tests, " most of which measure mutagenicity to provide information about carcinogenicity.

The IARC review provides an example of scientists and policymakers wrestling with the problems of extrapolating results of epidemiology and animal tests about toxicity to estimates of human effects. Although findings of IARC are not binding on governments, they are generally accepted as authoritative and provide an example of a successful ongoing effort to evaluate scientific evidence. A number of approaches to evaluating evidence about carcinogenicity and other toxicities for regulatory purposes, all of which involve a centralized panel of experts to consider the toxicity of substances, have been advanced by Government agencies,[23] Members of Congress, and by trade associations.[24] A recent National Academy of Sciences committee document argued against a central committee for making decisions about carcinogenicity for all Government agencies, but urged that a central committee be formed and charged with developing guidelines for making those decisions.[25]

To a major extent, the interest in expert review stems from a desire to grapple with uncertainty. Uncertainties in estimating the hazards posed by chemicals result from difficulties with test design and execution, the scantiness of data, and methods for extrapolation. The activities of an expert panel (e.g., IARC) to review data and conclusions reduce the uncertainties in a few cases involving carcinogenicity. In the absence of such expert review, the reader or scientist interested in toxicity must develop a critical eye, inspect and evaluate the evidence presented by others, and discuss opinions with other interested parties.

Health Effects Associated With Love Canal Chemicals

Toxic effects associated with **18** chemicals known to have been deposited in the canal landfill are listed in table C-4. The minimum lethal doses for these chemicals are much greater than environmental concentrations reported or expected within the EDA (see tables C-5 and C-6). For example, maximum environmental concentrations of chlorobenzene were on the order of 3 to 5 micrograms per cubic meter of air (table C-6), an amount less than 1/1,000,000 the level needed to kill the most sensitive laboratory animal (table C-4).

Data for birth defects or reproductive effects (called here "teratogenic effects") are not available for most of these compounds. Only pentachlorobenzene and hexachlorobenzene were reported to have been tested for teratogenicity.[26] Both were found to be positive in at least one test, but the quality of the data was not evaluated.

Mutagenic test results are reported on **12** of the substances, and of these, **8** were positive in at least one test. In addition, additional mutagenicity tests are planned for lindane and hexachlorobenzene.[27] The chemical 1,4-dichlorobenzene provides an example of a chemical that was mutagenic in one test and not in another. It has been shown to cause mutations in bacteria, but not in a test involving the use of Chinese hamster ovary (CHO) cells. Nevertheless, whatever caveats are attached to finding a positive response in testing for a mutational effect, the finding serves to warn of a possible hazard.

The fact that IARC found adequate animal data to evaluate the carcinogenicity of five of the chemicals listed in table C-4 indicates that there had been concern about the carcinogenicity of those chemicals. In addition, five chemicals, including three of the IARC-reviewed chemicals, are currently under test at the National Toxicology Program (NTP). In other words, 7 of the **18** Love Canal chemicals have been tested or are being tested for carcinogenicity, This level of effort does not mean that many or most of the chemicals are carcinogens, but it does mean that scientists have expressed sufficient concern about them that tests are necessary to provide more information.

For three of the five chemicals that IARC reviewed, data were sufficient to conclude that the chemicals are carcinogenic in laboratory animals. In the other two cases, IARC reached the conclusion that there was "limited" evidence rather than "sufficient" evidence to support a conclusion that the chemicals were carcinogens.

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[23] OSTP, "Identification, Characterization, arid Control of Potential Human Carcinogens: A Framework for Federal Decision-Making," *J. National Cancer Institute*, 64:169-176, 1980.

[24] *AIHC Proposal for a Science Panel* (Searsdale, N. Y.: AIHS, 1980).

[25] National Research Council, *Risk Assessment in the Federal Government: Managing the Process* (Washington, D. C.: National Academy Press, 1983).

[26] The Department of Health and Human Services, *Report of the Subcommittee on the Potential Health Effects of Toxic Chemicals Dumps of the DHEW Committee to Coordinate Environmental and Related Programs*, undated.

[27] National Toxicology Program, *National Toxicology Program: Fiscal Year 1983 Annual Plan*, Research Triangle Park, N. C., 1982.

Table C-4.–Summary of Test Results Available on Health Effects of Chemicals Disposed in Love Canal and Monitored by EPA

Substance	Minimum lethal dose[a]	Mutagenicity [b]	Carcinogenicity [c]	Regulation or standard[d] ACGIH	OSHA	EPA
Lindane (gamma-hexachlorohexane) . .	Ingestion, animal, 180 mg/kg	Cytogenic changes to be tested (NTP) negative (CCERP)	Animal + (IARC) Animal - (NCI)	*Yes*	Yes	National Drinking Water Standard W.Q.C.[e]
Chlorobenzene	Inhalation, animal, 15 g/m^3	—	Under test (NTP)	*Yes*	Yes	W.Q.C.
1,2-Dichlorobenzene	Inhalation, animal, 821 ppm	Positive (CCERP)	Animal ? (IARC) Animal - (NTP)	*Yes*	*Yes*	W.Q.C.
1,3-Dichlorobenzene	—	Positive (CCERP)		—	—	W.Q.C.
1,4-Dichlorobenzene	Man, 221 mg/kg	Point mutagen, negative, CHO test (NTP)	Animal ? (IARC) Under test (NTP)	*Yes*	*Yes*	W.Q.C.
1,2,3-Trichlorobenzene	—	Negative (CCERP)		—	—	W.Q.C. insufficient data
1,2,4-Trichlorobenzene	Ingestion, animal, 758 mg/kg	Negative (CCERP)	—	*Yes*	—	W.Q.C. insufficient data
1,3,5-Trichlorobenzene	Implant, animal LD_{50} 20 mg/kg	Negative (CCERP)		—	—	W.Q.C. insufficient data
1,2,3,4-Tetrachlorobenzene	—			—	—	W.Q.C.
1,2,4,5-Tetrachlorobenzene	Ingestion, animal, 1,035 mg/kg	Positive (CCERP)		—	—	W.Q.C.
Pentachlorobenzene[f]	Ingestion, animal, 2,000 mg/kg	Positive, CHO (NTP)	Under test (NTP)	—	—	W.Q.C.
Hexachlorobenzene[f].	Man, 220 mg/kg	Positive (CCERP) to be tested (NTP)	Animal + (IARC)	—	—	W.Q.C.
2-Chloronaphthalene	Ingestion, animal 888 mg/kg	—		—	—	W.Q.C. insufficient data
Alpha-Chlorotoluene.	Ingestion, animal, LD_{50} 1,200 mg/kg	Point mutagen DNA replication positive (CCERP)	Animal + (IARC) Under test (NTP)	*Yes*	Yes	
2-Chlorotoluene.	Inhalation, animal, 175,000 ppm			*Yes*	*Yes*	—
3-Chlorotoluene.	—			—	—	—
4-Chlorotoluene.	—	Negative (CCERP)		—	—	—
2.4-Dichlorotoluene.	—			—	—	—

[a]All data in *this* column are from 1980 *Registry of* Toxic Effects *of* Chemicals Substances (NIOSH, 1982).

[b]References for data in this column are from NIOSH (1982) unless otherwise indicated: NTP is National Toxicology *Program: Fiscal Year 1983* Annual *Plan* (NTP, 1982); and CCERP is Report *of the Subcommittee on the Potentia/ Health Effects* of Toxic *Chemica/ Dumps of the DHEW* Committee to Coordinate Environmental and Related Programs (Department of Health and Human Services, undated).

The appearance of a test name means that the chemical was found to cause the named effect: cytogenic changes: microscopically visible chromosomal changes; *point* mutagen: chemical altered a specific gene in the test organism, a standard test; CHO test: a test of the capacity to alter growth patterns of Chinese hamster ovary cells, a standard test; DNA replication: a test of the capacity to alter DNA replication, a standard test; *positive:* CCERP reported that at least one test has shown the chemical is a mutagen; the quality of the data was not reviewed; and *negative:* CCERP reported that the chemical had been tested and none of the results showed the chemical to be a mutagen; the quality of the data was not reviewed.

[c]References: IARC is IARC Monographs Supplement 1 (IARC, 1979); NCI is R. A. Griesemer, and C. Cueto, Jr. in Molecular and Cellular Aspects of Carcinogen Screening Tests (IARC, 1980); and NTP is same as under (b). + means the agency judged the substance to be an animal carcinogen; ? means the evidence about carclnogenicity was limited; and – means the evidence was negative.

[d]Acronyms: ACGIH—American Council of Government Industrial Hygienist, Osha-Occupational Safety and Health Administration, EPA—Envimnmental Protection Agency, IARC—International Agency for Research on Cancer, NCI—National Cancer Institute, and NTP—National Toxicology Program.

[e]W.Q.C.: Water Quality Criteria Document (45 F. R., 11/28/80). W.Q.C. means EPA recommended a standard; W.Q.C. insufficient data means that there *were* insufficient data to *base* a standard.

[f]At least one test result indicates that this substance has teratogenic properties.

Table C-5.—Comparison of Regulated Exposure Limits to Detected Maximum Concentrations in the EDA: Water

Substance	Regulated limit (exposure through ingestion)	Maximum concentration found in EDA[a] [concentration (medium)]	Ratio: maximum detected level/ standard
One substance regulated under National Drinking Water Standard:			
Lindane	4.0 ppb[b]	5.3 ppb (sanitary sewer)	1.3
		3.4 ppb (storm sewer)	0.85
Substances for which water quality criteria have been published:			
Lindane	No safe limit (carcinogen) 10^{-5} risk,		
	water and fish, 0.186 ppb	5.3 ppb (sanitary sewer)	28.5
		3.4 ppb (storm sewer)	18.3
	fish only, 0.625 ppb	5.3 ppb (sanitary sewer)	8.5
		3.4 ppb (storm sewer)	5.4
Chlorobenzene	488 ppb	Trace[c] (shallow well)	
1,2-Dichlorobenzene	400 ppb	15 ppb (deep well)	0.04
1,3-Dichlorobenzene	400 ppb	80 ppb (sump)	0.2
1,4-Dichlorobenzene	400 ppb	586 ppb (sump)	1.46
1,2,4,5-Tetrachlorobenzene	Water and fish, 38 ppb	62 ppb (storm sewer)	0.61
	fish only, 48 ppb	62 ppb (storm sewer)	0.77
Hexachlorobenzene	No safe limit (carcinogen) 10^{-5} risk,		
	water and fish, 7.2 ppt[b]	Trace (sump water, sanitary sewer)	
	fish only, 7.4 ppt	Trace (sump water, sanitary sewer)	

[a]Much higher concentrations, sometimes in excess of 10,000 ppb, were found in surface water sediment near sewer outfalls and in sewer sediments. Those two contaminated media are to be cleaned up in the remediation process.
[b]ppb: parts per billion, 1 µg of chemical/liter water; ppt: parts per trillion, 0.001 µg of chemical/liter water.
[c]Trace: detectable, but not measurable concentrations.

SOURCE: From several published sources.

Table C-6.—Comparison of Regulated Exposure Limits to Detected Maximum Concentrations in the EDA: Air

Substance	Regulated exposure limit[a] (exposure through inhalation)	Maximum concentration found in EDA [concentration (medium)]	Ratio: maximum detected level/ standard
Lindane	500,000 µg/m³ (OSHA, ACGIH)[b]	0.098 µg/m³ (living area)	<0.001
Chlorobenzene	350,000 µg/m³ (OSHA, ACGIH)	3.5 µg/m³ (basement)	<0.001
1,2-Dichlorobenzene	300,000 µg/m³ (OSHA, ACGIH)	68 µg/m³ (living area)	<0.001
1,4-Dichlorobenzene	450,000 µ/m³ (OSHA, ACGIH)	25 µg/m³ (living area)	<0.001
1,2,4-Trichlorobenzene	40,000 µg/m³ (ACGIH)	Trace[c] (living area)	<0.001
2-Chlorotoluene	250,000 µg/m³ (ACGIH)	8 µg/m³ (living area)	<0.001

[a]Limits are generally expressed in units of milligrams/cubic meter. To facilitate comparison of limits and maximum concentrations, limits are converted to units of micrograms/m³ here.
[b]OSHA—Occupational Safety and Health Administration; ACGIH—American Council of Government Industrial Hygienists.
[c]Trace: detectable, but not measurable concentrations.

SOURCE: From several published sources.

The National Cancer Institute's (NCI) evaluation of the carcinogenicity of lindane differed from that of IARC.[28] Data from NCI did not support the idea that it is an animal carcinogen. IARC concluded that lindane was a carcinogen.

For 1,2-dichlorobenzene, IARC found that there was only limited information about carcinogenicity. A subsequent test by NTP reveals that the substance does not cause cancer in either rats or mice. Therefore, ad-

[28]Griesemer, op. cit.

ditional evidence has reduced the level of concern about possible carcinogenic effects for that chemical. A related chemical, l,4-dichlorobenzene is now under test at NTP as is alpha-chlorotoluene. Data about the carcinogenicity of chlorobenzene and pentachlorobenzene under test at NTP, have not been reviewed by IARC.

Table C-4 also shows that workplace and environmental exposures to many of these chemicals are regulated. The Occupational Safety and Health Administration (OSHA) regulates workplace exposure, and restrictions on workplace exposures have been recommended by a group of industrial health experts, the American Conference of Government Industrial Hygienists (ACGIH). EPA has published water quality criteria for **13** of the **18** chemicals. In addition, a National Drinking Water Standard regulates exposure to lindane.

The data summarized in table C-4 show that the chemicals deposited in the canal landfill include some recognized as presenting hazards to human health. An immediate objection to drawing any conclusions about health effects from these chemicals at Love Canal derives from EPA's observations that the concentrations within the EDA were very low.

Comparison of Regulated Exposure Levels and Environmental Concentrations

EPA has published National Drinking Water Standards for **16** inorganic chemicals, **6** pesticides (including lindane), 1 group of organic chemicals, and total dissolved solids. As is shown on table C-5, the maximum concentration of lindane found in one sample each from sanitary sewers and storm sewers slightly exceeded that limit. It should be emphasized that while this concentration is higher than the *drinking* water standards, the water in both systems is not likely to be ingested by humans.

Water quality criteria documents have been published for **64** chemicals to serve as guidelines for acceptable concentrations in drinking and fishing waters. There was some emphasis on protecting against carcinogenic risks in the criteria documents, and, in keeping with the idea that there is no dose of a carcinogen below which there is no risk, the Agency declared that there was no safe limit for carcinogens. Instead, it calculated the amount of the substance, that if ingested over the course of a lifetime, would cause an incremental risk of cancer equal to 1 case of cancer in 100,000 people. The magnitude of that risk can be judged by comparison to the figure that about **20** percent of Americans (or 20,000 out of every 100,000) die from cancer. As is shown in table C-6, the maximum detected concentrations of lindane in sewers exceeded the standards for water to be used for drinking *or fishing.* The measured levels of **1,2-** and 1,3-dichlorobenzene and 1,2,4,5-tetrachlorobenzenes were less than the limits established by the water criteria documents. Only 1,2-dichlorobenzene, found at 0.04 **of** the recommended guideline, was detected in deep wells and likely would be associated with human ingestion. No numerical measurements were reported for chlorobenzene and hexachlorobenzene, and EPA claims that concentrations of those chemicals would be in the low ppb range. If the concentrations are that low or lower, the level of chlorobenzene would be below that recommended by EPA.

Hexachlorobenzene presents an analytical problem. The water quality criteria document associates a 10^{-5} cancer risk with a 7 ppt concentration of hexachlorobenzene. However, that chemical cannot be measured at concentrations lower than a few ppb. Thus, hexachlorobenzene could be present in water samples in the Love Canal study in concentrations up to 1 ppb, **130** times the level associated with a 10^{-5} cancer risk. But such concentrations are possible in all water; methods are not available to measure this chemical at 7 ppt. The fact that hexachlorobenzene was detected only in waters that humans do not drink*or* fish means that opportunities for exposure are limited.

To summarize, measured concentrations of some chemicals found in sewer and sump waters in the EDA approached or exceeded levels recommended for drinking and fishing waters. However the contaminated waters in the EDA are not likely to be consumed, and exposure through ingestion is unlikely.

OSHA or ACGIH or both have established limits on workplace exposure to six of the chemicals. In the workplace, concern is about exposure by inhalation or through the skin. In the case of lindane, the exposure limit shown on table C-6, based on inhalation, is to be further lowered if there is any chance of the chemical reaching the worker's skin. The workplace exposure limits are based on consideration of acute toxic effects and are designed to protect against workers' becoming ill soon after exposure. They are not designed to protect against any chronic health effect-cancer, birth defects, or mutations. Therefore, it is no surprise that the maximum levels of airborne contamination found in the EDA is much less than the workplace limits. Although there is little reason to believe that the workplace limits protect against chronic health risks, the airborne concentrations at Love Canal are much lower, and are as low **as** levels detected in many areas of the country.

EPA can consider chronic health effects in setting limits for air pollutants under the Clean Air Act. To

date, it has not published nor made public any consideration of possible regulations of the chemicals listed in table **C-6.** Therefore, there are no standards or guidelines to compare to the detected levels. EPA did compare airborne concentrations of chemicals in the Love Canal area to concentrations in cities around the country, and there were no striking differences reported.

The Special Case of Dioxin

Dioxin (more precisely, 2,3,7,8-tetrachlorodibenzo-p-dioxin, or 2,3,7,8-TCDD, or TCDD) is one of the most toxic substances. It is known to cause cancer in laboratory animals, and it has been associated with tumors in humans. The "buyout" of Times Beach, Me., was based on the premise that dioxin present at concentrations greater than 1 ppb in the soil presented a health threat.

There is no Federal standard that restricts environmental exposure to dioxin. EPA has carried out an assessment of the health risk posed by the presence of dioxin in incinerators. The Agency concluded that **0.0004** micrograms/m^3 of stack air, which would be diluted 100,000-fold by the time it reached ground level, where it might be inspired, would not "present a public health hazard. "[29] The government of the Providence of Ontario has set a permissible limit for dioxin in air equal to **0.00003** micrograms/m^3, which is about 1/10 the level found by EPA in the incinerator stacks. (An average person breathes about **20** m^3 of air daily. Over a 70-year lifetime, a person breathing the maximum limit permitted by Ontario would inhale **219** micrograms **of** dioxin.)

One of the surprises from Times Beach is the observation that dioxin is very stable in soil. The stability is probably related to the fact that dioxin binds very firmly to particles in the soil, and being bound protects it from degradation. It is so difficult to extract dioxin from soil, that it may be that the chemical is not removed from soil particles that are inhaled or ingested. If that is the case, soil-bound dioxin would pose little threat to human health. NTP is currently conducting studies about the bioavailability of soil-bound dioxin and expects to have results by the end of the summer of **1983.** It has already been reported that root crops, such as carrots, that were grown in dioxin-containing soil did not take up appreciable quantities of dioxin .[30] The low levels found with the carrots might have resulted from contaminated soil sticking to the outside of the root.

EPA has been working on a water quality criteria document for dioxin for some time, Although it is not known what level the final document will recommend, a draft suggested that dioxin levels should not exceed 1 part in 10^{16} parts of water. This very low concentration presents analytical difficulties. Although both EPA and the Canadian Government have perfected methods to measure low levels of dioxin in water, the minimum detection level are now 100-fold higher, about 1 part of dioxin in 10^{14} parts of water. In other words, water with no detectable levels of dioxin might harbor 100 times the concentration that may be recommended as a guideline to protect health. (See discussion above, also of hexachlorobenzene.)

EPA claims that it was able to detect 1 to **20** ppt (0.001 to 0.020 ppb) dioxin in the samples taken at Love Canal. OTA has serious reservations about the intensity of sampling for dioxin, but laying those aside for the moment, EPA reported dioxin in sumps in the Love Canal homes and in storm sewers. The Agency reported finding no dioxin in air, which is not surprising because it has not been reported in air except in stack gases. And no dioxin was reported in waters. The Ring 1 homes are not being considered for rehabitation, and the storm sewers are to be cleaned up as part of the remediation effort. Therefore, the known sources of exposure to dioxin are going to be eliminated.

OTA is concerned that few samples were analyzed for dioxin in the EPA study. If the decision is made to rehabitate the EDA, and, if, as part of that effort, monitoring is carried out, dioxin should be included as a monitored substance more frequently than it was in the EPA study reported in 1982. It may be that there are good reasons for the sketchy sampling carried out earlier, but it would not reassure the public to sample this important contaminant any less frequently than other chemicals.

Studies of Health Effects in the Love Canal Population

Although the habitation decision was based on consideration of the extent of chemical contamination in the EDA, there are other data that also bear on the question of health effects. Those data derive from observations made on the population of people who lived near the Canal. Love *Canal, A Special Report to the Governor and Legislature,* in **1981** summarizes evidence collected by New York Department of Health officials until that time.[31]

[29] D. Barnes, U.S. EPA, personal communication, May 1983.

[30] R Kimbrought, Center for Disese Control, Atlanta, Ga., personal communication, May 1983.

[31] NYS/DOH, *Love Canal,* a *Special Report to the Governor and Legislature,* 1981. See also, Love *Canal: Public Health Time Bomb,* NYS/DOH, 1979.

Briefly, pregnant women who lived near the canal were found to be at greater risk of suffering a miscarriage or of delivering a 'low birth-weight baby." More focused research into the location of residences associated with these adverse effects revealed that the women at greatest risk lived either on 99th Street, directly adjacent to the canal, or in formerly wet areas just east of the canal. The New York State analysis of these data led to the conclusion that the frequency of spontaneous abortion (miscarriage) reached a peak in the 1960's and early 1970's. In addition, the percentage of children born with birth defects was larger among those delivered by women who lived on 99th Street or in formerly wet areas as compared to women who lived beyond what is now the declaration area. The excess of birth defects was not, however, statistically significant. (Neither the study of reduced birth weight nor birth defects has been published in a peer-review scientific journal.)

Cancer rates of residents of the census tract in which Love Canal is located have been compared to cancer rates in other census tracts in Niagara Falls. The female population around Love Canal was found to have experienced twice the number of respiratory cancers as a control female population; the excess among men was less, respiratory cancer among male Love Canal census tract residents was 1.7 times that observed in the control population. "Scientists differ in what importance to attach to excesses of cancer that are less than 2, but most agree that a twofold excess, as was seen for respiratory cancer in women, merits attention. Further analysis showed that the incidence of respiratory cancer within the Love Canal census tract does not vary with distance from the canal, which weakens the argument that substances in the canal were associated with the excess cancers. The incidence of some cancers, lymphoma, leukemia, and liver cancer among men living in the Love Canal census tract was only half that observed in the control areas. The incidence of genital organ and urinary cancers among women was lower among Love Canal area residents than among women in other areas of Niagara Falls.

Any conclusions to be drawn from the study of cancer incidence around Love Canal are weakened by the small number of cancers that occurred during the period (1966-77) over which the study was conducted. Furthermore, since cancers may develop years or decades after exposure, the study done by New York State may have been too early to detect an effect, if there is one. A better answer to whether or not living near the canal is associated with higher cancer rates may become available as more people who lived in the canal area are located and studied.[33]

In May of **1983,** the DHHS released a study of chromosomal abnormalities in a small population of people who had lived near the canal.[34] The results of that study were negative; that is, the frequency of unusual chromosomes among the canal area residents was no greater than the frequency found in a control population. There are some problems with this study. (There may never have been a study without some problems.) In particular, in the opinion of many scientists, a chromosomal abnormality caused by exposure to toxic chemicals may be short lived and may be repaired over time. Therefore, since the exposures occurred years ago, there might be no discernible effect from them now. Despite that reservation (so far as OTA can determine, that reservation is based on technical opinion and not upon prolonged observation of human populations) and other technical reservations, the consensus is that the study does not show any chromosomal abnormalities as a result of living near Love Canal. This negative study provides evidence that an earlier study, which detected a high frequency of chromosomal abnormalities among Love Canal area residents might have been in error.

Also in May **1983,** Beverly Paigen and coworkers presented a paper at a meeting of the Society for Pediatric Research.[35] They compared health effects observed in the population of people who had lived in the EDA to effects observed in groups of people who lived in control areas. They reported that low birth-weight babies were more common among the EDA population and that the average weights of babies born there were lighter at each week of gestation. Furthermore, children born and raised (for at least 75 percent of their lives to date) in the EDA were shorter than children in the control areas, and the parents reported that these children had more episodes of six different medical complaints than control area children.

Paigen confirmed that low birth weights were confined to families who had lived in the formerly wet parts of the declaration area.[36] The medical complaints in children were not restricted to families living in the wet areas, but decreased with distance from the canal. She thinks that the play of children might bring them into closer contact with contaminated areas, and dis-

[32] Janerich, et al., "Cancer Incidence in the Love Canal Area, " *Science* 212, 1981, pp. 1404-1407.

[33] C. W. Heath, Jr. *Assessment of Health Risks at Love Canal,* presented at the Fourth Annual Symposium on Environmental Epidemiology, Pittsburgh, Pa., May 1983.

[34] C. W. Heath, Jr., et al., A Study *of Cytogenetic Patterns in Persons Living* Near *the* Love *Canal,* Center for Disease Control, Atlanta, Ga., May 1983.

[35] B Paigen, et al., Growth *and Health in Children Living Near a Hazardous Waste Site,* presented at Society for Pediatric Research, May 1983.

[36] B. Paigen, Children's Hospital, Oakland, Calif., personal communication, May 1983.

tance from the canal would decrease the frequency of the children reaching such areas.

To accumulate the data in the paper was difficult. The 220 births reported in the declaration area and the 697 in the control areas occurred over a 15-year interval. Analyzing such data requires careful attention to detail, and reviewing the data and analysis requires an opportunity to review details. At the present time, the results presented by Paigen, et al., are being prepared for publication, and final evaluation of their work must wait until the paper, with more detailed descriptions of the study and data, is complete.

A major study of health effects is expected sometime this fall. The NYS/DOH has located as many as possible of all the people who lived in the canal area since the 1940's.[37] Those people were interviewed about their health status, and the results of that study will provide important information about the health of former residents of the Love Canal region, and current and former residents of the EDA.

The completed studies are sufficient to show that the Love Canal population has not experienced any adverse health effects at rates more than twice those experienced by control area populations. Spontaneous abortions and low birth-weight babies were statistically more frequent among the populations in the Love Canal region, and birth defects, although not statistically more frequent, were observed more often in that population. The incidence of some cancers in the area has been higher than in control areas; the incidence of other cancers has been lower.

As more data become available in the near future, it may become possible to draw firmer conclusions about the health impacts of living near Love Canal. Unsatisfying as it is, the consensus of opinion probably is that the studies of the Love Canal area population have produced more leads to follow up than strong conclusions about the safety of the EDA. Evidence is more convincing that serious health effects were associated with living in the Love Canal homes.

[37] N. Vianna, NYS/DOH, personal communication, May 1983.