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EXPERT JUDGMENTS OF ENVIRONMENTAL RISKS

by

Samantha Nicole Rizak

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

in

Environmental Engineering

Department of Civil and Environmental Engineering

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "Expert Judgments of Environmental Risks" submitted by Samantha Nicole Rizak in partial fulfillment of the requirements for the degree of Master of Science in Environmental Engineering.

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ABSTRACT

Although much progress has been made in improving risk communication by understanding the differing risk judgments between the lay public and the scientific community, there is increasing awareness that discrepancies in risk judgments between experts themselves also hinder effective risk communication. To further address this issue a survey was conducted to determine the extent to which members of various environmental disciplines share similar beliefs and conceptual frameworks concerning several basic assumptions and concepts in environmental health risk assessment.

This study focuses on exploring the perspectives concerning risk judgments of two disciplinary groups whose members often become involved in studying risk issues and/or conveying risk information to others. These include a group of environmental epidemiologists as well as a group of environmental engineering professors. In addition, previous published findings on toxicologists in the United States and Canada provide a comparison for some of our analyses.

The results of this survey indicate that divergent interpretations do exist among respondents for several of the statements provided. Although no sharp distinctions were found between disciplinary perspectives, differences in opinion were apparent within each group itself. Furthermore, the qualitative responses provided from this survey suggest that there are areas in which a certain level of misunderstanding among some respondents is apparent. Areas which seem to offer the greatest opportunity for improved understanding for these groups include carcinogenic risk assessment, the validity of animal studies for predicting human health effects, and causal and statistical inference in environmental health risk assessment. The results from statements evaluating uncertainty and confidence in health risk estimates also reflect the potential need for improved understanding of the substantial uncertainties and limitations in environmental health risk assessment.

In light of the apparent difficulties in evaluating and communicating risk, the findings from this study provide a possible basis for which risk judgments can be more informed. Because many environmental scientists and engineers often become involved in environmental risk controversies regardless of their direct involvement and expertise with risk assessment, it is necessary that these experts evaluate their own knowledge and understanding of these concepts and be fully aware of the strengths and limitations of the methods used for risk assessment.

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INTRODUCTION

The practice of environmental health risk assessment has developed as a means to analyze what we know about chemical risks to guide the selection of risk management options. Risk assessment has been defined as (ATSDR 1990):

> "the evaluation of the toxic properties of a chemical and the conditions of human exposure to it in order both to determine the likelihood that exposed humans will be adversely affected, and to characterize the nature of the effects they may experience."

The process of risk assessment has been formalized into four stages: hazard identification, dose-response assessment, exposure assessment and risk characterization. Hazard identification is the initial step of the process. It examines all the available evidence that associates exposure to a chemical and an adverse effect, and provides a qualitative judgment about the strength of that evidence. This stage relies on knowledge of the basic properties of chemicals as well as the findings from toxicology and epidemiology. Dose-response assessment seeks to determine the quantitative relationship between the magnitude of dose and the probability of occurrence of an adverse health effect from a chemical during a specified time. Exposure assessment seeks to determine the human exposure to a chemical and identifies the populations exposed, describes their composition and size, and examines the routes, magnitudes, frequencies, and durations of such exposures. Risk characterization combines the dose-response and exposure assessment information to estimate the incidence of an adverse effect in humans and/or determine allowable exposure concentrations. Risk characterization also evaluates the magnitude of the uncertainties involved and the major assumptions that were used.

Assessments of risk have been widely acknowledged to be inherently subjective, relying heavily on judgment (National Academy of Sciences 1983, Kraus et al. 1992, Slovic 1997, Pidgeon et al. 1992). Although there may be substantial evidence collected for a risk analysis (i.e. hazardous properties of a substance, exposure conditions, dose-response relationships, etc.), translating this evidence into risk estimates requires substantial inference. So while evidence may seem objective, the inferences required to interpret the evidence are dependent on judgment.

Because judgment is inherent in all assessments of risk, both technical and non-technical, exploring the subjective elements of risk judgments has become important research for determining perceptions of risk, and for improving communication of risk information in light of widely differing perceptions. Although much progress has been made in improving the communication of risk information by understanding the disparities between expert and lay risk judgments, there is increasing awareness that discrepancies in risk judgments between experts themselves are also a major difficulty in achieving effective risk communication.

Two studies, "Intuitive Toxicology: Expert and Lay Judgments of Chemical Risks" by Kraus et al. (1992) and Slovic et al. (1995), examined the different attitudes, beliefs and perceptions regarding basic toxicological concepts, assumptions and interpretations of both toxicologists and the lay public in the United States and Canada. These studies were motivated by the premise that different assumptions, concepts and values, as well as disagreements about facts, underlie many of the discrepancies between expert and lay risk judgments. The results from these studies indicated that toxicologists and laypeople do differ greatly in their attitudes and perceptions regarding many toxicological concepts. In both studies, it was found that the public had much more negative attitudes towards chemicals in general than the toxicologists. The public was also found to be much less sensitive to considerations of dose and exposure. Another important finding from these studies was that both the public and toxicologists

lacked confidence in the value of animal studies for predicting human health risks. However, the public was much more likely to see positive evidence of carcinogenicity in animal studies as implying danger to humans.

An important observation arising from these studies was that toxicologists and others involved in risk assessment need to be fully aware of the strengths and limitations of the methods used to generate risk information. Furthermore, they should play a greater role in interpreting the health implications of the data for the public and in doing so, they should also acknowledge the subjective elements, judgments and assumptions inherent in the analyses as well as the degree of uncertainty in the conclusions (Kraus et al. 1992).

Although the findings from these studies demonstrated that toxicologists and laypeople do greatly differ in their views on toxicological concepts, another major finding was that, in some cases, there was as much disagreement over concepts within the toxicologist group as there was between the toxicologists and the public (Kraus et al. 1992, Slovic et al. 1995). Thus it is apparent that the differences in judgments between experts themselves may be a significant cause of the public's dissatisfaction and distrust with risk assessment.

This finding is important in that it suggests that risk communication may be hindered as much by the conflicting and contradictory messages emanating from the scientific community as by differing public perceptions. Therefore, although it is useful to understand the similarities and differences between expert and lay risk judgments, it is apparent that effective communication of risk, both with the public and between scientists, also rests with understanding the discrepancies in risk judgments among experts themselves.

To further explore this issue, a survey was conducted to determine the extent to which members of various environmental disciplines share similar beliefs and conceptual frameworks concerning several underlying assumptions and concepts in

environmental health risk assessment. These will inevitably influence their risk judgments.

RESEARCH OBJECTIVES

The objectives of this research are:

- to determine if members of some major environmental disciplines exhibit divergent interpretations of several underlying assumptions and concepts in environmental health risk assessment,
- to reveal, where possible, the nature of any misunderstandings and/or disagreements on these issues,
- to compare and contrast any apparent perspectives of these disciplines, and
- to determine if some differences may be attributable to demographic factors such as age, employer, gender or experience.

This research was designed to reveal some perspectives concerning risk judgments among members of various environmental disciplines. In addition, the survey, which was designed to solicit both quantitative and qualitative responses, should provide some insight into underlying reasons for some of these perspectives. Specifically, for the case of disagreements, qualitative responses will be interpreted to judge whether the disagreements are due to misunderstandings, misinformation or honest disagreements. These findings will be explored as a possible basis for improving scientific analyses and communication regarding the assessment and management of environmental risks.

METHODOLOGY

Survey Groups

Because risk issues extend far beyond the practice of risk assessment, the scientific and technical community will often become involved in environmental risk controversies regardless of their direct involvement and expertise with risk assessment. Consequently, it is important to understand the judgments of environmental scientists and engineers as they often play a role in studying risk issues, participating in risk management decisions, or conveying risk information to others such as the public.

Risk assessment is inherently multidisciplinary. Therefore, we chose to survey two major disciplines whose members contribute knowledge to the risk assessment process and to the applications of risk assessment findings to environmental management. The first group was comprised of the participants at an international conference on environmental epidemiology and the second group was comprised of environmental engineering professors.

Although not surveyed with our questionnaire, the groups of toxicologists and the public surveyed in the previous studies, "Intuitive Toxicology: Expert and Lay Risk Judgments of Chemical Risks" (Kraus et al. 1992), "Intuitive Toxicology. II. Expert and Lay Risk Judgments of Chemical Risks in Canada" (Slovic et al. 1995) and "Health Risk Perception in Canada II: Worldviews, Attitudes and Opinions" (Krewski et al. 1995) provide a comparison for some of our analysis because several of the statements in our questionnaire were replicated from the surveys of these groups.

Four of the statements in our survey were also included in a Danish survey of medical students and students attending a postgraduate course in risk assessment (Grandjean and Nielson 1996). We also surveyed students in a postgraduate course in

public health science at the University of Alberta. These results provide some additional perspectives on these issues from health and medical students as well as risk assessment students.

Survey Content

The questionnaire consisted of 20 statements as well as a general demographic section detailing the respondent's personal and educational background, affiliation, and experience. The statements were developed from several underlying assumptions and concepts in environmental health risk assessment. These statements addressed many different issues which encompass environmental health risk assessment such as:

- conceptions of toxicity (dose-response relationships and exposure) and carcinogenic risk assessment,
- trust in animal studies,
- causal inference in epidemiological studies,
- statistical inference in environmental health risk assessment,
- uncertainty and confidence in health risk analyses, and
- objectivity and values in scientific analyses.

The statements to determine views on toxicity and carcinogens are presented in Table 1. These statements were designed to assess the sensitivity to the relationship of chemical concentration, dose and exposure, and risk to human health. Table 2 contains statements to determine the value one places on toxicological studies for determining the health effects of chemicals on humans. Since the discipline of risk assessment relies heavily on animal studies, these statements were put forth to examine how much confidence these disciplines have in data sources such as these.

Table 1. Statements on Conceptions of Toxicity (Dose-Response Relationships and Exposure)/ Carcinogenic Risk Assessment
1. A chemical is either safe or dangerous. There is really no in between.
2. There is no safe level of exposure to a cancer-causing agent.
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.

Table 2. Statements on Trust in Animal Studies
3. The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.

Tables 3 and 4 contain statements designed to assess the respondents' understanding of cause-effect relationships between exposure to chemicals and human health, and the statistical significance of such findings.

Table 3. Statements on Causal Inference in Epidemiological Studies
14. A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.
19. Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.

Table 4. Statements on Statistical Inference in Environmental Health Risk Assessment
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.

Table 5 presents statements which were designed to determine the confidence and understanding one has in the predictive methods of risk analysis. Two statements were also included in the questionnaire to determine the degree to which experts recognize the subjective elements which enter into their scientific work. These statements are listed in Table 6.

Table 5. Statements on Uncertainty and Confidence in Health Risk Analyses
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.

Table 6. Statements on Objectivity and Values in Scientific Analyses
11. Environmental epidemiology/science is an applied science (i.e., not a basic science).
12. Applied sciences are rarely value-free or value-neutral.

Additional miscellaneous statements were added to the questionnaire to elicit the respondents' perceptions of various risks in the environment and to determine their attitudes towards natural and synthetic chemicals, and chemicals in the environment. These statements are listed in Table 7. The final question, which will be reported separately, addressed the sources and reliability of information upon which respondents rely on for information about health risks from chemicals and other health hazards.

Table 7. Statements on Perceptions of Risk
6. Fruits and vegetables contain natural substances that can cause cancer.
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.
8. Natural chemicals are not as harmful as man-made chemicals.
9. The land, air and water around us are, in general, more contaminated now than ever before.

Twelve of the statements used in the questionnaire were replicated from the previous published surveys on "Intuitive Toxicology: Expert and Lay Judgments of Chemical Risks" by Kraus et al. (1992) and Slovic et al. (1995). These are listed in Table 8. The remaining 8 questions were developed in consultation with researchers of the Eco-Research Chair in Environmental Risk Management at the University of Alberta.

Table 8. Replicated Statements

1. A chemical is either safe or dangerous. There is really no in between.
2. There is no safe level of exposure to a cancer-causing agent.
3. The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.
6. Fruits and vegetables contain natural substances that can cause cancer.
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.
8. Natural chemicals are not as harmful as man-made chemicals.
9. The land, air and water around us are, in general, more contaminated now than ever before.
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.
19. Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.

The information requested in the demographic section included the respondent's age, gender, country of residence, complete educational background, employer, current held position(s), and years of experience in the respective discipline. This information was requested to provide insight into the influence of these factors on the responses given. The questionnaires for the environmental epidemiologists and environmental engineering professors are included in Appendix A and B.

Survey Design

To encourage a high response rate, the length and format of the questionnaire were important design considerations. In order for completion of the questionnaire to be quick (5 - 15 minutes), we limited the number of questions to only 20 plus a section on demographics. This length constraint fixed the complexity of the questions asked. Furthermore, because English may not have been a first language for some respondents, clear and simple wording of the statements was required.

The survey was patterned after the studies by Kraus et al. (1992) and Slovic et al. (1995). In the questionnaire, a scale of agreement, including strongly agree, agree, disagree and strongly disagree, was provided for each statement. The statements were given as absolute statements to provoke a response and respondents were explicitly asked to indicate their level of agreement to each statement by selecting the category that most closely reflected their view concerning the statement. A "don't know" category was also included for each statement. "Don't know" was explicitly intended to mean "unable to respond due to lack of knowledge" and not to be equivalent to a "neither agree nor disagree" category. This latter category was intentionally excluded in order to force an opinion to the statement based on the respondent's educated intuition. However, to overcome any discomfort respondents might have with this forced choice, a comment section was provided after each statement and respondents were encouraged to include any comments and/or an elaboration detailing the reasons for their response. The comment sections were also designed to aid in interpreting responses by providing some insight into the underlying reasons for a respondent's choices.

Respondents were also instructed to think of the term "chemicals" as including "...all chemical elements and compounds, including pesticides, food additives,

industrial chemicals, household cleaning agents, prescription and non-prescription drugs, etc." (Kraus et al. 1992).

The questionnaire was pretested informally within the Department of Public Health Sciences at the University of Alberta. Based on the responses and feedback from this pretest, minor modifications were made to some statements prior to administration of the survey. The questionnaire was also reviewed by the Research Ethics Board. The request for ethical review and the ethics approval form are included in Appendix C.

Survey Administration and Response

Both the environmental epidemiology group and the environmental engineering professors were administered the same questionnaire with slight differences of wording in a few areas to account for the differences in background discipline between the two groups. The questionnaires for these groups are included in Appendix A and B.

For the environmental epidemiology group, the questionnaire was included in the registration package of the 386 registrants at the International Society for Environmental Epidemiology (ISEE) Conference held in Edmonton, Canada in August of 1996. Of these, 196 were completed and returned providing an overall response rate of 50.8%.

The environmental engineering professors were surveyed from the 1995 Association of Environmental Engineering Professors (AEEP) membership directory. In all, 702 questionnaires were mailed in October of 1996 and 413 were completed and returned providing an overall response rate of 58.8%.

RESULTS

Data Analysis

The statistical software package, SPSS, was used to analyze the results from the survey. The Population Research Laboratory, Department of Sociology at the University of Alberta performed the SPSS programming, data cleaning, creation of the data set, and determining the frequency distributions for the quantitative responses.

Statistical analysis was not conducted for the quantitative responses because the respondents were not randomly selected nor was it feasible to replicate the surveys with equivalent populations to determine variability in survey response. Although statistical analysis would have been useful for distinguishing what levels of differential response were statistically significant, this would provide only limited insight concerning the practical significance of the results.

Demographic Profile

Of the 196 respondents from the International Society for Environmental Epidemiology Conference (1997), 61.5% were male; 53.4% were between the ages of 25 and 44, 41.9% were between ages 45 and 64; and 4.7% were younger than 25 or older than 64. Over half of these respondents were affiliated with an academic institution (54.1%), 24.2% with a government office, 6.7% with a consulting firm, 3.1% with private industry, and 11.9% with other affiliations such as public interest. Survey respondents were highly experienced: 46.7% had more than 5 years experience with environmental epidemiology, 31.8% had 1 to 5 years experience, 11.8% had

limited experience (less than 1 year), and 9.7% had no experience with environmental epidemiology.

The respondents from the Association of Environmental Engineering Professors were predominantly male (87.6%) with 48.3% of the 413 respondents between the ages of 25 and 44, 45.9% were between ages 45 and 64, and 5.8% were older than 65. The majority of respondents from this group were affiliated with an academic institution (90.8%), 2.2% were affiliated with a government office, 4.4% with consulting, 1.5% with private industry, and 1.2% with other affiliations such as public interest. Over 90% of respondents had greater than 5 years experience with environmental engineering; 9.7% had 1 to 5 years, and 0.2% had less than 1 year experience.

Internal Validity of Respondent Sample

Information on the entire sampling frame for the environmental engineering professors indicate that response from this group was representative of the sample chosen indicating no bias resulted from non-response. From the information provided in the membership directory of the Association of Environmental Engineering Professors (AEEP) it was estimated that of the 702 members surveyed overall, 88.6% were male and 11.4% were female. These gender percentages were consistent with the 413 members who responded (87.6% male, 12.4% female). Furthermore, geographic residency was also found to be consistent. Of the 702 members surveyed, 90.7% resided in the United States while 4.3% lived in Canada and 5.0% resided outside North America. These percentages were fairly consistent with the 413 members who responded to the survey: 91.6% resided in the United States, 4.4% resided in Canada and 3.9% resided outside North America.

Information on gender and geographic residency for the participants at the International Society for Environmental Epidemiology conference was not available.

External Validity of Surveyed Population

As mentioned previously, the groups surveyed included two major environmental disciplines. The first group was composed of the participants at an international conference on environmental epidemiology (ISEE '97). The participants at this conference came from numerous countries from around the world with approximately half of the respondents being from North America. These participants had diverse experience with environmental epidemiology with some respondents (9.7%) having no epidemiology experience. As well, respondents had diverse areas of study with most respondents holding graduate degrees from the health sciences with medicine, epidemiology and/or public health science being the most common. The majority of respondents in this group were affiliated with an academic institution.

The second group was composed of environmental engineering professors taken from the 1995 membership directory of the Association of Environmental Engineering Professors (AEEP). These members were predominantly North American (95%) and almost all were affiliated with an academic institution with greater than 5 years experience with environmental engineering. The large majority of the respondents from this group held graduate degrees in civil and/or environmental engineering.

Because many of the respondents from the ISEE '97 Conference had a range of experience with environmental epidemiology, the results from this survey may not be generalizable to environmental epidemiologists on the whole. This is not a well defined discipline and it is not clear that any particular professional society would completely

represent the discipline. The results for environmental engineering professors are likely generalizable to academics in this field but may not be generalizable to environmental engineering professionals who are not academics. These results are also not specific to professionals having direct involvement and expertise with risk assessment. However, the respondents from both these groups do often play a role in studying risk issues, participating in risk management decisions and/or conveying risk information to others. Therefore, these results are more generalizable to any environmental professionals who are users of risk assessments rather than those directly involved with conducting risk assessments.

Statement Responses

The responses to statements 1 through 19 for the environmental epidemiologists and environmental engineering professors are shown in Tables 9 and 10. Table 9 presents the results for the replicated statements. For comparison in the discussion, Table 9 also includes the results from the groups of toxicologists and public, as well as for the different groups of students surveyed. Table 10 presents the results for the non-replicated statements for the environmental epidemiologists and environmental engineering professors, as well as for the students in a postgraduate public health science course at the University of Alberta.

Table 9. Responses to Replicated Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
1. A chemical is either safe or dangerous. There is really no in between.	EE[b]	51.8[a]	37.8	7.3	3.1	0.0
	EP[c]	70.0	26.4	2.2	1.0	0.5
	T2[d]	62.7	31.3	4.7	0.0	1.3
	P2[e]	20.2	27.6	25.0	24.6	2.6
	PH[f]	28.1	43.8	15.6	6.3	6.3
2. There is no safe level of exposure to a cancer-causing agent.	EE	16.6	40.4	24.4	10.4	8.3
	EP	26.0	47.6	12.6	2.2	11.7
	T1[g]	27.7	47.0	13.9	4.8	6.6
	P1[h]	6.6	28.1	35.5	18.4	11.3
	R[i]	2.9	27.5	57.8	7.8	3.9
	M[j]	4.1	28.9	44.3	13.4	9.3
	PH	12.9	29.0	19.4	32.3	6.5
3. The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.	EE	13.1	46.6	29.8	1.6	8.9
	EP	8.4	44.0	28.6	1.0	18.0
	T1	1.9	38.9	50.3	5.1	3.8
	T2	4.0	26.0	56.7	9.3	4.0
	P1	5.5	40.2	40.2	3.5	10.6
	P2	14.9	21.7	39.6	20.7	3.1
	R	13.7	58.8	23.5	1.0	2.9
	M	17.5	53.6	27.8	1.0	0.0
	PH	40.6	43.8	12.5	0.0	3.1
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	EE	9.7	46.9	29.1	2.6	11.7
	EP	5.7	38.3	39.6	2.5	14.0
	T1	10.3	47.3	39.4	1.2	1.8
	T2	7.3	36.0	44.0	7.3	5.3
	P1	1.9	22.9	64.0	5.4	5.8
	P2	4.1	11.8	47.5	34.7	1.9
	R	4.9	54.9	37.3	1.0	2.0
	M	3.1	35.1	50.5	7.2	4.1
	PH	9.4	43.8	40.6	3.1	3.1

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] T2 - Canadian Toxicologists (n = 150; Slovic et al. 1995)
[e] P2 - Canadian Public (n = 1500; Krewski et al. 1995)
[f] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)
[g] T1 - American Toxicologists (n = 170; Kraus et al. 1992)
[h] P1 - American Public (n = 262; Kraus et al. 1992)
[i] R - Danish students in a risk assessment postgraduate course (n = 102; Grandjean and Nielson 1996)
[j] M - Danish medical students (n = 97; Grandjean and Nielson 1996)

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	EE	43.1	41.5	8.2	0	7.2
	EP	35.4	48.8	4.6	0.2	10.9
	T1	25.7	62.3	7.8	0.6	3.6
	T2	34.7	49.3	7.3	3.3	5.3
	P1	5.1	42.6	25.4	9.0	17.2
	P2	6.7	27.0	38.7	23.0	2.7
	R	24.5	58.8	15.7	0.0	1.0
	M	7.2	44.3	42.3	5.2	1.0
	PH	16.1	58.1	6.5	0.0	19.4
6. Fruits and vegetables contain natural substances that can cause cancer.	EE	5.7	16.1	45.8	14.1	18.2
	EP	2.4	8.3	48.2	15.1	26.0
	T2	8.0	11.3	48.0	25.3	7.3
	P2	34.9	33.6	19.9	5.8	5.9
	PH	3.1	28.1	34.4	6.3	28.1
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	EE	2.1	12.6	40.0	28.9	16.3
	EP	1.7	5.8	45.0	36.7	10.7
	T2	2.7	7.3	37.3	47.3	5.3
	P2	12.6	24.2	38.8	20.1	4.3
	PH	6.3	21.9	34.4	15.6	21.9
8. Natural chemicals are not as harmful as man-made chemicals.	EE	21.9	43.8	13.5	0.0	20.8
	EP	23.5	40.3	22.0	3.4	10.8
	T1	45.6	40.2	11.2	2.4	0.6
	T2	54.0	34.0	6.7	2.7	2.7
	P1	10.8	34.0	37.8	7.3	10.0
	P2	14.1	24.1	33.0	23.1	5.7
	PH	31.3	43.8	12.5	0.0	12.5
9. The land, air and water around us are, in general, more contaminated now than ever before.	EE	4.1	29.9	37.1	18.6	10.3
	EP	10.1	37.3	36.3	10.9	5.4
	T1	3.6	24.8	53.3	13.9	4.2
	P1	1.5	8.1	45.2	43.2	1.9
	P2	1.9	4.3	20.8	72.6	0.5
	PH	3.1	6.3	56.3	34.4	0.0

Table 9 continued. Responses to Replicated Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	EE	13.4	38.1	22.2	0.5	25.8
	EP	5.4	30.6	20.1	1.0	42.9
	T2	19.3	52.0	18.0	4.7	6.0
	P2	18.7	32.6	31.5	12.5	4.8
	PH	19.4	51.6	12.9	3.2	12.9
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.	EE	11.4	45.1	35.2	1.0	7.3
	EP	13.2	49.8	27.1	0.5	9.5
	T2	7.3	46.0	40.7	2.7	3.3
	P2	11.2	25.0	47.3	13.4	3.0
	PH	9.7	51.6	22.6	3.2	12.9
19. Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.	EE	19.1	47.4	8.2	0.5	24.7
	EP	11.2	44.9	9.3	0.2	34.4
	T1	22.2	59.3	4.3	1.2	13.0
	P1	3.9	23.4	39.5	9.0	24.2
	R	16.7	52.9	18.6	0.0	11.8
	M	4.1	21.6	60.8	6.2	7.2
	PH	10.0	13.3	30.0	3.3	43.3

Table 9 continued. Responses to Replicated Statements

Table 10. Responses to Non-replicated Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
11. Environmental epidemiology/science is an applied science (i.e., not a basic science).	EE[b]	0.5[a]	10.5	64.4	19.4	5.2
	EP[c]	5.2	19.5	58.6	12.1	4.7
	PH[d]	3.2	25.8	32.3	9.8	29.0
12. Applied sciences are rarely value-free or value-neutral.	EE	6.3	15.7	42.9	20.9	14.1
	EP	7.1	22.8	43.8	5.6	20.8
	PH	6.7	16.7	23.3	26.7	26.7
14. A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.	EE	30.4	52.6	13.4	1.5	2.1
	EP	24.0	53.2	11.8	1.5	9.6
	PH	40.0	53.3	6.7	0.0	0.0
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.	EE	12.0	36.1	23.6	1.6	26.7
	EP	15.1	40.6	25.0	2.5	16.8
	PH	6.5	25.8	16.1	3.2	48.4
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	EE	5.7	30.4	45.9	7.7	10.3
	EP	7.8	37.2	30.4	5.1	19.5
	PH	6.5	3.2	41.9	16.1	32.3
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	EE	31.1	46.1	14.0	0.0	8.8
	EP	14.5	40.5	22.1	2.0	20.9
	PH	29.0	32.3	9.7	0.0	29.0
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.	EE	20.2	36.8	31.6	1.6	9.8
	EP	7.3	33.4	25.4	2.0	32.0
	PH	10.0	30.0	16.7	3.3	40.0

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] PH - Students in a public health science postgraduate course (n = 32)

ANALYSIS AND DISCUSSION

Epidemiological and toxicological methods are the two main approaches used in environmental health risk assessment to identify, characterize and quantify risks to human health from chemicals in the environment. Epidemiology is the study of the distribution of disease in populations and the factors associated with that distribution (Spivey 1994). In environmental health risk assessment, epidemiology involves identifying associations between potential risk factors and a disease (or other effect), and evaluating those associations for evidence of causality. These methods use real world observations and hypothesis testing to compare groups of exposed and unexposed individuals to determine whether there is a difference in the occurrence of disease in relation to exposure (Sowers 1994).

Toxicology is the study of the adverse effects of chemicals (Gallo and Doull 1991). In environmental health risk assessment, the principles of toxicology are utilized to characterize the adverse effects of chemicals on living organisms, namely experimental animals, define the conditions of exposure required to produce the adverse effects, and understand the mechanisms by which chemicals produce toxicity (Klaassen and Kershaw 1994).

Although epidemiological methods are conceptually the best for health risk assessment in that they provide directly relevant information about the health effects of exposed humans, they often provide inconclusive evidence because of the numerous limitations and methodological difficulties of such studies. Epidemiological methods have several disadvantages such as:

- their observational nature limits control,
- accurate exposure data is limited and difficult to obtain,

- unrecognized bias and confounding cannot be fully accounted for, and
- they cannot provide toxicity predictions prior to human exposure (Harrison and Hoberg 1994).

Therefore, experimental evidence of animal toxicological studies is more commonly used in environmental health risk assessment to help establish causation between a chemical and a health effect.

Toxicological methods offer many advantages over epidemiological methods in that they can:

- be more controlled in the laboratory setting,
- reveal the mechanisms of toxic action through invasive monitoring and post-mortem examination,
- provide information on all target sites which may be adversely affected by a chemical, and most importantly for risk assessment
- provide toxicity information prior to human exposure and therefore can be predictive (Rodricks 1992).

Although toxicological methods compensate for the numerous difficulties associated with epidemiological methods, they also have the great disadvantage that the population health risk estimates they produce have an even higher degree of uncertainty than those obtained from epidemiological studies because of the numerous additional inferences and extrapolations between species and across enormous dose ranges. Therefore, in order to provide any guidance for environmental risk management, scientists must use all the evidence from both epidemiological and toxicological studies, and combine the available information to produce the most reliable risk estimates possible.

Conceptions of Toxicity (Dose-Response Relationships and Exposure)/Carcinogenic Risk Assessment

Dose-Response Assessment

Dose-response assessment is often the most controversial aspect of environmental health risk assessment because of the substantial uncertainties and many inferences involved in determining the relationship between the amount of toxic substance administered and the resulting toxic response(s). The dose-response relationship is based on the premise that as the quantity of exposure to a substance increases within its harmful range, the likelihood and/or severity of adverse health effects will also increase (Hrudey 1996). Although the dose-response relationship is a critical component in quantitative health risk assessment and provides the quantitative basis for regulating toxic compounds, there are many difficulties associated with both determining and interpreting the form of the relationship within the relevant range for environmental exposures.

Because reliable exposure and outcome data are rarely available for the general human population, dose-response relationships are usually determined through animal experiments by exposing laboratory animals to various dose levels of a substance and then observing the adverse health effects that develop at each dose level. Aside from using animal data to draw inferences to human populations, the dose levels used in animal experiments are another source of controversy for dose-response determination. Because of the extreme costs of such tests, a standard animal carcinogen bioassay is usually restricted to only 3 dose groups with 50-100 animals in each group (Graham and Rhomberg 1996; Harrison and Hoberg 1994). With this limit on the number of animals tested, there are severe restrictions to the ability to detect statistically significant effects to small proportions of the dose group. Therefore, to increase the sensitivity of the test and compensate for the limited number of animals used, dose levels are usually

increased to levels far greater (often several orders of magnitude greater) than what humans would typically experience in the environment (Graham and Rhomberg 1996; Foster et al. 1993).

For example, in carcinogen testing, dosing is usually conducted chronically at doses near the maximum that the animal can tolerate, the maximum tolerated dose (MTD) (Ames and Gold 1990a). The MTD is defined for chronic (lifetime) carcinogen bioassays as the highest dose level predicted that produces no irreversible damage, serious incapacity, or altered longevity or growth from effects other than cancer (Hrudey 1995). A typical carcinogen bioassay consists of using dose levels of MTD, 1/2 MTD and 1/4 MTD (plus a control group) with 50-100 animals of each sex per dose level.

Once testing is complete, the severity and/or frequency of response at each dose level of a chemical is plotted to obtain a dose-response curve. The evaluation and interpretation of the shape and slope of this curve is a critical component in determining the risk calculation for a carcinogen. Because animal experiments are frequently performed at exposure concentrations much higher than the levels encountered in any environmental exposures, the cancer risk to humans from environmental exposures must be estimated by extrapolating the dose-response curve downwards to doses much lower than those used for generating the experimental data. Consequently, the further a dose-response extrapolation reaches beyond the experimental data, the greater the uncertainty is of the shape and slope of the curve in the region being questioned (Hrudey and Lambert 1994).

Faced with this uncertainty, regulatory agencies and many scientists assume that, for carcinogenic compounds capable of reacting directly with DNA and causing genotoxic effects like mutation (genotoxic carcinogens), the dose-response curve goes through the origin (zero dose - zero excess response) and is linear in the low dose region (Foster et al. 1993; Hrudey 1995). The assumption of zero-intercept, low dose

linearity for genotoxic carcinogens is based on the premise that there is no threshold dose below which no adverse response is expected. Effectively, the no threshold hypothesis assumes that any exposure above zero may produce some risk above zero (Hrudey and Krewski 1995). This theory refers to the possibility that a single molecule of a DNA reactive carcinogen has some finite, non-zero probability of altering DNA in a single cell which through cell replication can multiply and ultimately become the initial event in the development of cancer (Hrudey and Krewski 1995). However, this possibility is obviously not certain nor even probable as we have all been exposed to many carcinogens and yet we will not all die of cancer. For example, whereas up to 90% of lung cancer is attributable to tobacco smoking (Zaridze and Peto 1986), only a small minority of smokers actually develop lung cancer.

Although it is observed that some carcinogenic compounds, which do not react directly with DNA (nongenotoxic carcinogens), do exhibit a threshold dose below which no adverse effects are observed, the absence of evidence for genotoxic carcinogens and the uncertainty in the detailed mechanisms of carcinogenesis has made the zero-intercept, low dose linearity, no-threshold hypothesis a conservative default assumption for carcinogenic risk assessment.

The no-threshold theory is a controversial issue in current carcinogenic risk assessment with plausible arguments both for and against a threshold for genotoxic substances (Hart and Fishbein 1985; Maynard et al. 1995). Many scientists who dispute the no-threshold hypothesis suggest that many of the mechanisms which may cause a carcinogenic response are entirely the result of administering highly toxic doses, such as the MTD, which saturate the biological processes necessary for defending against toxic effects. The mechanisms of carcinogenesis induced by these high doses may not operate in a similar manner at more realistic low dose levels to which humans are generally exposed in the environment (Ames and Gold 1993; Marx 1990; Koshland 1994). Furthermore, because cells have extremely efficient DNA

repair mechanisms to deal with the large numbers of naturally occurring DNA damage, an effective threshold level may exist because any trace level damage to DNA from low level exposures to environmental contaminants could also be easily repaired.

However, regardless of these arguments, even if evidence suggests that thresholds do exist, there would still be the difficulty of establishing what the threshold level is for a substance as it likely varies with species and/or individuals within the same species. Because the human population is a very genetically diverse heterogeneous group with varying degrees of responses to a variety of toxicants, the assumption of one threshold is unrealistic given that not all members of a population will have the same one (Hart and Fishbein 1985).

A more profound implication of the no-threshold assumption is that it may reinforce a prevalent belief that there is no safe level of exposure to a carcinogen (Hrudey and Krewski 1995). Many people believe that any exposure, no matter how small, to a carcinogen means they will get cancer and that safety can only be achieved if there is zero risk (Fisher 1991; Pidgeon et al. 1992; Kraus et al. 1992; Slovic et al. 1995). However, this definition of safety is unrealistic because zero risk is unattainable. Obviously we cannot impose zero risk as our standard of safety as nothing would warrant being considered safe. Furthermore, the failure to distinguish safety from zero risk hinders the ability to achieve safety because it intrinsically implies that any infinitesimal, non-zero risks are unsafe. Thus, saying that there is no safe level of exposure in effect may be interpreted as meaning any exposure is unsafe. However, Hrudey and Krewski (1995) demonstrated that, even using the conservative, upper bound cancer risk estimation procedure, the cancer risk associated with a chronic lifetime exposure to the smallest indivisible unit of the most potent carcinogen is so insignificantly small that it is arguably safe. Therefore, provided that safety is not equated with zero risk, there must exist a safe level of exposure to any known

carcinogen. Consequently, a more realistic and effective representation of safety would be an acceptable degree of risk rather than zero risk.

Exposure

In addition to the relationship between dose and response, exposure is a key determinant of dose. Regardless of the concentration of a substance in the environment, if there is no exposure to it, then human health is obviously not harmed. Thus, exposure and adverse effects are greatly influenced by the fate and behavior (transport) of a substance in the environment.

Furthermore, the consequences of exposure to any substance is dependent on many factors. Adverse effects from exposure are dependent on the exposure route (ingestion, inhalation or dermal exposure), the exposure level (dose), and the length of exposure duration. They also depend on the properties of the substance and the nature of the toxic action in the body (toxicokinetics and toxicodynamics). Finally, adverse effects from exposure to a chemical are also dependent on an individual's susceptibility and other risk factors which are influenced by numerous factors such as genetic predisposition, age, race, nutritional status, immune response, diet, lifestyle choices, etc. (Hrudey and Chen 1996)

Analysis of Statements

Table 11 presents the results for Statements 1, 2 and 5 for the groups of environmental epidemiologists, environmental engineering professors, toxicologists and the public, as well as for the public health and Danish medical students and the Danish students participating in a postgraduate course in risk assessment. These statements were included in the survey to determine the respondents' views of toxicity

and their sensitivity to the relationship between chemical dose and exposure, and risk to human health.

Table 11 - Responses to Statements 1, 2 and 5

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
1. A chemical is either safe or dangerous. There is really no in between.	EE[b]	51.8[a]	37.8	7.3	3.1	0.0
	EP[c]	70.0	26.4	2.2	1.0	0.5
	T2[d]	62.7	31.3	4.7	0.0	1.3
	P2[e]	20.2	27.6	25.0	24.6	2.6
	PH[f]	28.1	43.8	15.6	6.3	6.3
2. There is no safe level of exposure to a cancer-causing agent.	EE	16.6	40.4	24.4	10.4	8.3
	EP	26.0	47.6	12.6	2.2	11.7
	T1[g]	27.7	47.0	13.9	4.8	6.6
	P1[h]	6.6	28.1	35.5	18.4	11.3
	R[i]	2.9	27.5	57.8	7.8	3.9
	M[j]	4.1	28.9	44.3	13.4	9.3
	PH	12.9	29.0	19.4	32.3	6.5
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	EE	43.1	41.5	8.2	0	7.2
	EP	35.4	48.8	4.6	0.2	10.9
	T1	25.7	62.3	7.8	0.6	3.6
	T2	34.7	49.3	7.3	3.3	5.3
	P1	5.1	42.6	25.4	9.0	17.2
	P2	6.7	27.0	38.7	23.0	2.7
	R	24.5	58.8	15.7	0.0	1.0
	M	7.2	44.3	42.3	5.2	1.0
	PH	16.1	58.1	6.5	0.0	19.4

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] T2 - Canadian Toxicologists (n = 150; Slovic et al. 1995)
[e] P2 - Canadian Public (n = 1500; Krewski et al. 1995)
[f] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)
[g] T1 - American Toxicologists (n = 170; Kraus et al. 1992)
[h] P1 - American Public (n = 262; Kraus et al. 1992)
[i] R - Danish students in a risk assessment postgraduate course (n = 102; Grandjean and Nielson 1996)
[j] M - Danish medical students (n = 97; Grandjean and Nielson 1996)

Statement 1, "*A chemical is either safe or dangerous. There is really no in between.*" was designed to assess the sensitivity to the relationship between dose and response. Perhaps the most fundamental principle in toxicology is that "the dose makes the poison" (Klaassen 1996). As Paracelus (1493-1541) noted:

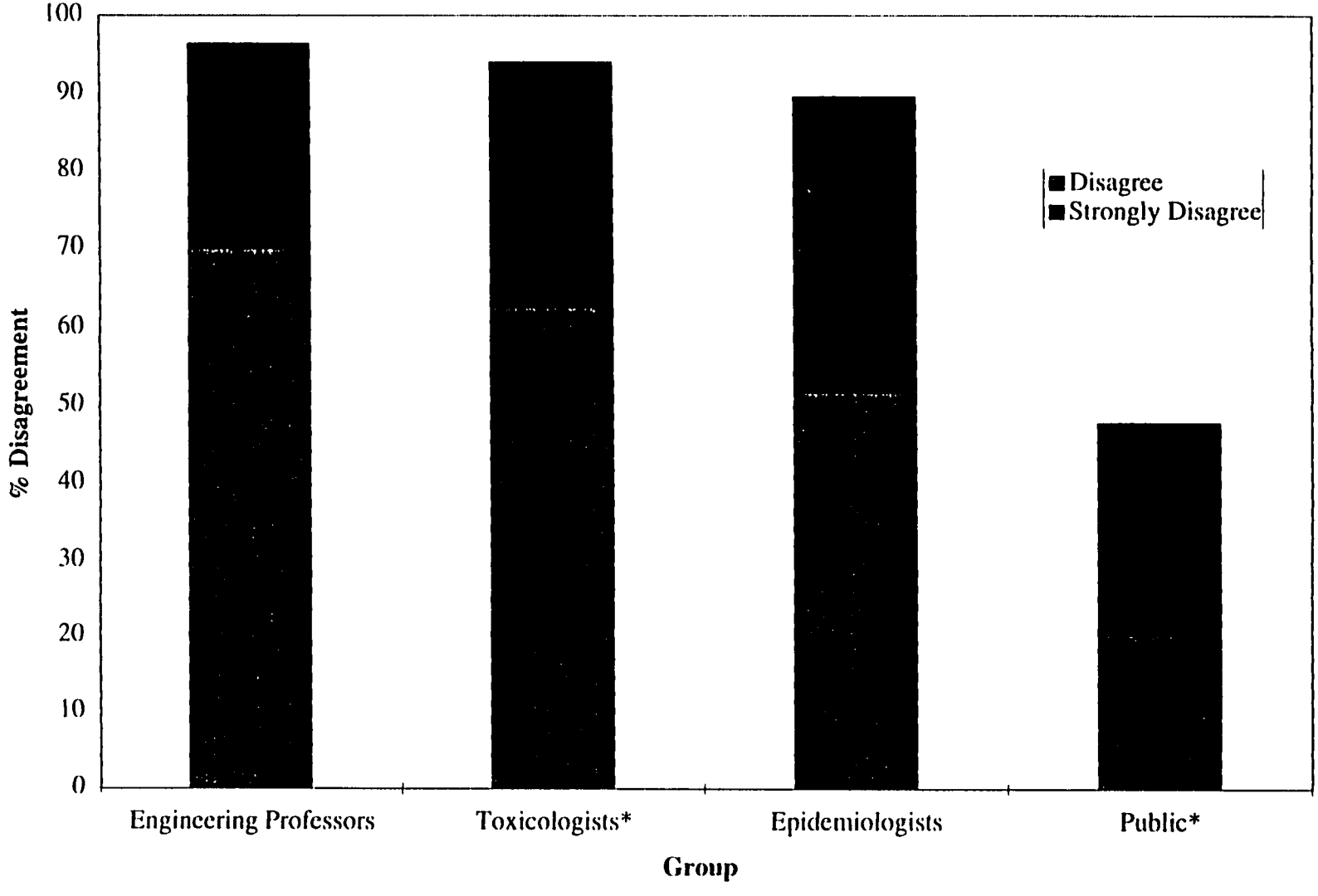
> *"All substances are poisons; there is none which is not a poison.*
> *The right dose differentiates a poison from a remedy."*

In other words, any substance can cause a toxic effect if the dose is great enough. This is important in that it implies that substances do not have "all or nothing" toxic properties but exhibit more of a continuum where low concentrations can be safe, sometimes even beneficial, while high concentrations may be dangerous. For example, many nutrients that are essential in trace amounts can become toxic at high concentrations.

Responses to this statement indicate that there was little divergence in opinion both within and between the three disciplinary professions regarding this statement (Table 11). Figure 1 shows that there was substantial disagreement with Statement 1 among all three disciplinary groups. The environmental engineering professors showed the greatest total disagreement (96.4 %) as well as the strongest disagreement (70%). The group of Canadian toxicologists were also in strong disagreement to this statement (94.0% total disagreement; 62.7% strong disagreement) (Slovic et al. 1995). This substantial disagreement for the three groups suggests that these respondents appreciate the view that the dose makes the poison.

Although the majority of environmental epidemiologists disagreed with this statement (89.6%), this group was more likely to "agree" and "strongly agree" than the other two groups (10.4% agreement overall: 7.3% agreement and 3.1% strong agreement) (Table 11). The environmental engineering professors were the least likely group to agree with this statement (2.2% agreement and 1.0% strong agreement).

Figure 1. Responses to Statement 1:
"A chemical is either safe or dangerous. There is really no in between."



* Canadian survey (Krewski et al. 1995; Slovic et al. 1995)

The Canadian public responses to this statement indicate that the public is much less sensitive to considerations of dose-response and are much more likely to view chemicals as either safe or dangerous having "all-or-nothing" toxic properties than the scientists surveyed. 49.6% of the public agreed with his statement and 47.8% disagreed (Figure 1) (Krewski et al. 1995). These findings show that the public exhibits much less appreciation of the toxicological principle that "the dose makes the poison." The public health science students showed more appreciation than the public but much less appreciation than the disciplinary groups (71.9% total disagreement with only 28.1% in strong disagreement) that toxic effects of chemicals are dependent on the dose.

Approximately one fifth of the respondents from both the environmental engineering professors and the environmental epidemiologist group provided qualitative responses for this statement. The comments that were provided verified that numerous respondents from both groups recognize dose-response relationships and the view that the dose makes the poison. Additional qualitative responses also indicated that a chemical's safety not only is related to dose but also "depends on exposure (contact) and exposure length."

The lack of qualitative responses makes it difficult to interpret the reasoning behind any agreement to this statement; particularly, the higher percentage of environmental epidemiologists who responded this way. Only two qualitative responses were provided for this category. These were both from environmental epidemiologists who responded "strongly agree." Their responses were that the "dose makes the poison" and "depends on concentration." These comments were similar to ones provided from those in disagreement with the statement. Considering these comments, it is difficult to interpret why these two respondents agreed. One explanation may be in how they define "safe" and "dangerous" which was not explained. Another explanation may be that they misread the statement and were

agreeing to there being an in between for a chemical rather than disagreeing that there really is no in between. This seems most likely.

Overall, comments to this statement did not indicate many difficulties with the wording or interpretation. A few respondents indicated that response depends on how one defines "safe" or "dangerous." Although a few respondents expressed concern with these terms, overall, the statement did not appear to cause any difficulties in interpretation because of its wording.

While qualitative responses were useful in explaining the reasoning of those who were in disagreement with the statement, unfortunately they did not provide much insight as to the rationale behind "agreement responses."

Statement 2, "*There is no safe level of exposure to a cancer-causing agent.*" was intended to assess how respondents view safety and whether they equate safety with zero risk. Agreement or disagreement to this statement strongly depends on one's definition of what a "safe" level is: what an individual considers safe involves some degree of value judgment (Whittemore 1983). Agreement that there is no safe level of exposure to a cancer-causing agent suggests that one defines safety as "absolute" safety (i.e. zero risk). However, this definition of safety is unrealistic as zero risk is unattainable. Furthermore, considering the basic principles of dose-response and that we are constantly exposed to carcinogens, there clearly must be some level of exposure (greater than zero) that can still be considered safe. Therefore, if safety is defined as an acceptable degree of risk rather than zero risk, disagreement with this statement would be likely.

Figure 2 displays the results to this statement for the environmental engineering professors, environmental epidemiologists, toxicologists and the public (American sample). Although the majority in all three disciplinary groups were in disagreement with this statement, there was still a moderate proportion who agreed that there is no

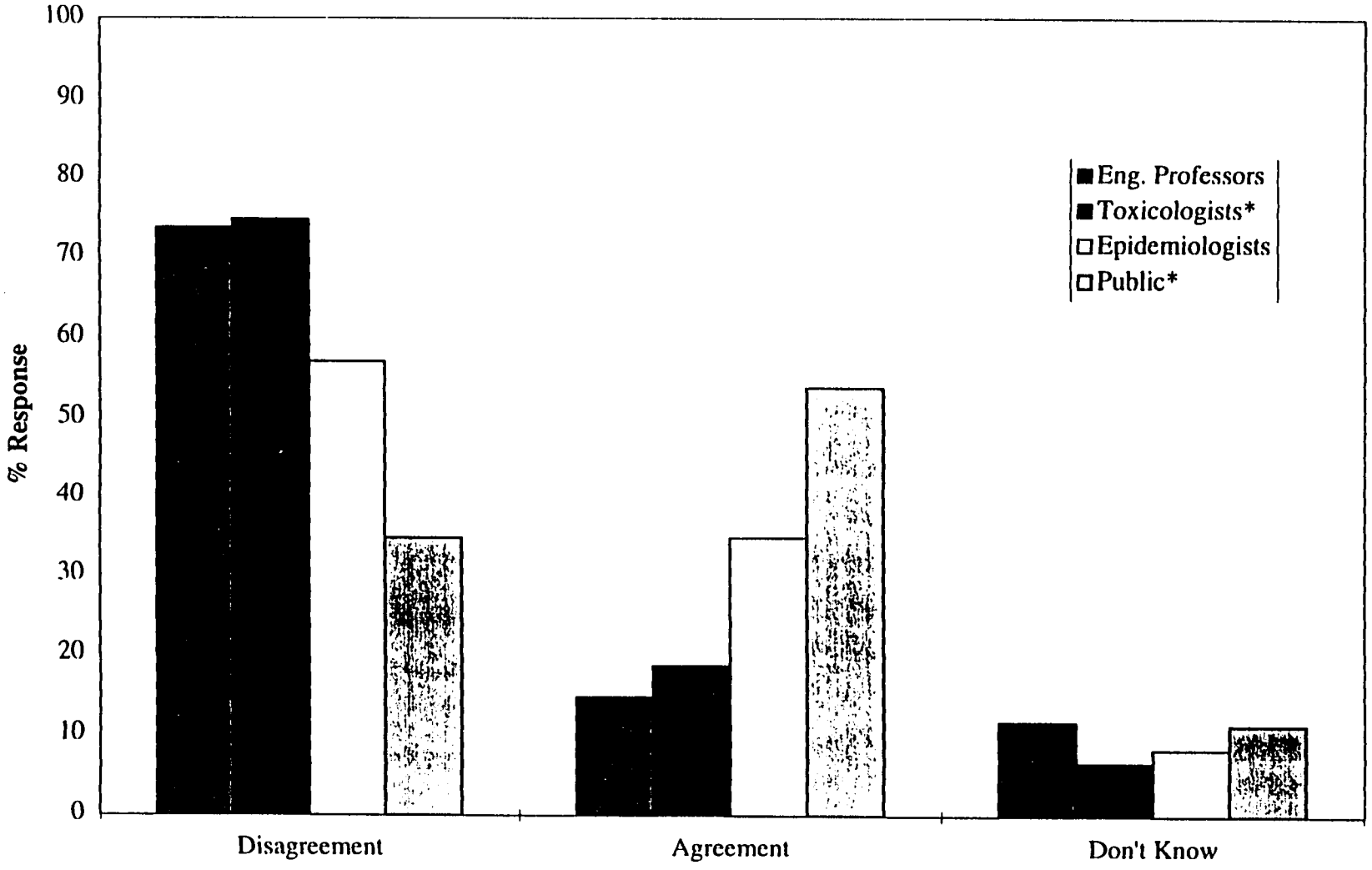
safe level of exposure to a cancer-causing agent. Whereas in Statement 1 a substantial portion in all three groups recognized that a chemical's toxic effects depend on the dose, many respondents were less inclined to agree that a safe level of exposure to a cancer-causing agent exists.

The environmental epidemiologists were more likely than the environmental engineering professors or toxicologists to agree that there is no safe level of exposure to a cancer-causing agent (34.8% agreement versus 14.8% for the environmental engineering professors and 18.7% for the toxicologists (Kraus et al. 1992)) (Figure 2). The group of environmental epidemiologists were also most likely to "strongly agree" with 10.4% in strong agreement with the statement. This group was twice as likely as the group of toxicologists and almost five times as likely than the environmental engineering professors to "strongly agree". This suggests that this group may be more inclined to equate safety with zero risk.

As might be expected, a majority of the public was in agreement that there is no safe level of exposure to a cancer-causing agent (53.9% agreement versus 34.7% disagreement) (Kraus et al. 1992). Of these, 18.4% were in strong agreement.

Of further interest, over half of the public health science students and Danish medical students agreed to this statement. The public health students also had 32.3% in strong agreement. The Danish students of a postgraduate course in risk assessment had the greatest agreement at 65.6%. This finding suggests there may be something in the way these students have been informed about carcinogenic risk which makes them inclined to doubt the possibility of there being a safe level of exposure to a carcinogen. Similar to the public, these responses suggest that many people equate safety with zero risk and believe that there is no safe level of exposure to a cancer-causing agent.

Figure 2. Responses to Statement 2:
"There is no safe level of exposure to a cancer-causing agent."



* American survey (Kraus et al. 1992)

From these results, there are obviously varying opinions concerning whether or not safe levels of exposure exist for carcinogens both between and within the various groups surveyed. The discrepancies between the scientific disciplines and the public, and between the scientific disciplines themselves, suggest that environmental scientists may need to carefully evaluate their definition of safety and recognize that "absolute" safety (zero risk) is unachievable. Regardless of whether a safe level can actually be agreed upon, a safe level can still exist. Perpetuating the belief that safety can only be achieved at zero risk and that there is no safe level of exposure to a carcinogen hinders the ability of achieving effective public policies by possibly sending misleading messages to the public. If they have unrealistic expectations, they may not trust the safety of the regulations that are made.

The qualitative responses to this statement provided the opportunity to explore the respondents' definitions of safety. For the environmental epidemiologists and environmental engineering professors in disagreement with this statement, the comments provided indicate that many are aware of the issue of thresholds and stated that a safe level depends on the agent and whether or not it is a genotoxic or non-genotoxic carcinogen. Others stated that they believe safe levels exist for all practical purposes provided that safety doesn't imply zero risk. Many indicated that safe levels must exist since "we are constantly exposed to many carcinogens all the time." Further explanations include that a safe level is relative to an individual, dependent on individual susceptibility, and dependent on genetic predisposition. Some respondents included that mechanisms of carcinogenesis are unclear and that many natural repair mechanisms exist.

Qualitative responses provided from the respondents (environmental epidemiologists and environmental engineering professors) in agreement with Statement 2 indicate that many generally feel that unless a threshold is known, there is no safe level of exposure to a carcinogen. Some comments stated explicitly that the

only safe level is if it is zero and that there is no "absolute" safe level. Others explained because of the long latency and seriousness of the disease that people should avoid any exposure to a risk factor. Some respondents (both in disagreement and agreement with the statement) recognized the fact that zero risk is impossible to achieve in any circumstance and that some risk is unavoidable. Overall, the qualitative responses did not indicate any divergent perspectives between these two groups for this statement.

Furthermore, judging from the available comments, the moderate proportion of "don't know" responses for this statement can be attributed to the difficulties respondents had with interpreting what "safe" meant. Many of the environmental epidemiologists and environmental engineering professors who responded "don't know", as well as other category responses, included in their comments that response strongly depends on one's definition of "safe." Thus it is likely that some divergence in opinion for this statement may be related to the wording of the question and the definition of what a "safe" level is. This difficulty may have been avoided had the statement explicitly defined a safe level. For example, the additional statement, *"Zero is the only safe level of exposure to a cancer-causing agent."* or *"Safety can only be achieved if there is zero risk."* may have provided better information on the respondents' definitions of safety. However, such wordings may have lead the respondents to disagree without really evaluating their own definitions of safety.

Statement 5, "*If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.*" was designed to assess the respondents' sensitivity to degree of exposure and if they equate any amount of exposure with harm. Given that we are constantly exposed to carcinogens yet the majority of individuals do not develop cancer, it seems unlikely that any amount of exposure will cause cancer because the consequences of exposure is dependent on

numerous other factors such as the degree of exposure, the exposure duration, the exposure route as well as an individual's susceptibility.

The findings from both "Intuitive Toxicology" studies showed that the public was much less sensitive to considerations of exposure than the toxicologists surveyed (Kraus et al. 1992; Slovic et al. 1995). The public was found to have much stronger concerns regarding any exposure and tended more to equate any amount of exposure to a carcinogenic substance, no matter how small, with almost certain harm (Table 11) (Kraus et al. 1992; Slovic et al. 1995).

All three disciplinary groups, however, substantially disagreed with this statement (Table 11). The environmental epidemiology group showed the strongest disagreement (84.6% total disagreement; 43.1% strong disagreement). Only 8.2% of epidemiologists agreed. Over 80% of the toxicologists (Kraus et al. 1992; Slovic et al. 1995) and the environmental engineering professors disagreed with this statement. The environmental engineering professors were again the least likely to agree (4.8% agreement). However, almost 11% responded "don't know."

The responses from the Danish students in a postgraduate risk assessment course and the students from public health science are similar to those of the disciplines with a vast majority disagreeing that if a person is exposed to a chemical that can cause cancer then that person will get cancer some day. However, almost half of the Danish medical students (47.5%) agreed that a person will probably get cancer if there is exposure to a cancer-causing chemical.

The similar comments provided from the environmental engineering professors and environmental epidemiologists suggest that both groups have similar perspectives concerning this statement. The most common theme in responses was that the risk from exposure to a chemical is dependent on many factors such as the agent(s), dose, duration of exposure and individual susceptibility. Some respondents recognized that we are constantly exposed to cancer-causing agents but we do not all die from cancer.

Example comments such as "some people who smoke never get cancer" were common responses for those in disagreement with the statement. Other respondents who disagreed stated that an exposed person will be at increased risk of getting cancer but will not get it with any certainty.

Similar comments were also provided for those in agreement to the statement. Some respondents agreed but included comments such as "assuming relevant exposure" and "depends on dose and duration of exposure." These comments help to clarify that some of the respondents in agreement with the statement may not necessarily think that any exposure implies harm. Comments such as these suggest that their focus was more on higher dose exposure than low dose exposure since they agreed to the statement even though they recognized that risk from exposure is dependent on many factors.

Overall, the comments provided indicate that most respondents are sensitive to the degree of exposure and other considerations. However, some respondents, particularly those who responded "don't know" or "disagree" indicated difficulty with the words "probably" or "exposed." Other respondents equated "probably" with "probability" and stated, for example, that "probability is 0 to 1." Other respondents questioned what was meant by "exposed." It appears from some comments, that "exposed" may have been interpreted only as "high dose exposure" rather than possibly any exposure, be it high or low, as was intended. To improve interpretation and understanding of this statement, the statement might have been posed as "*If a person has any exposure to a chemical that can cause cancer then that person will likely get cancer some day.*" Providing this less ambiguous statement may have been more effective in determining the respondents' sensitivity to the degree of exposure. An additional question asked in the previous surveys that may have been useful for examining whether respondents believe "any" exposure, no matter how small, implies

harm is: "*The body can usually repair the damage caused by exposure to a cancer-causing agent so that cancer does not occur.*" (Krewski et al. 1995; Slovic et al. 1992).

Trust in Animal Studies

Because of the inherent difficulties in assessing environmental hazards to human health by means of epidemiological studies, toxicological data derived from animal studies have become the primary foundation of regulatory policy governing environmental contaminant hazards, particularly environmental carcinogens (Marx 1990; Rodricks 1992). Although the biological similarities between animals and humans are the basis for reliance on animal testing to assess human health risks, the substantial uncertainties and extrapolations associated with animal studies has promoted debate concerning the relevance of these studies for predicting human health effects.

Dose Extrapolation

One of the greatest uncertainties associated with predicting human health effects from animal studies, particularly for assessing carcinogens, is the extrapolation of effects from high dose levels to low dose levels. This extrapolation is necessary because, as mentioned previously, in order to increase the sensitivity of the experiment and to compensate for the limited number of laboratory animals used, scientists typically test doses far greater (as much as hundreds of thousands of times greater) than what humans would be typically exposed to (Ames and Gold 1990b).

The theory behind high to low dose extrapolation is that a chemical that causes effects such as cancer at high doses will likely do so at low doses; although with less frequency at lower doses (Graham and Rhomberg 1996). The most widely used method of extrapolation for carcinogens has been the linearized multistage model because of its adoption as a regulatory default (U.S. EPA 1986). This model holds the no-threshold hypothesis and assumes a linear dose-response function passing though the origin (zero dose-zero excess response).

The no-threshold assumption for carcinogens is a controversial aspect in extrapolation of effects between high and low dose levels because of the many uncertainties which exist in the knowledge of the mechanisms of carcinogenesis. Many scientists argue that the health effects, particularly cancer, experienced by animals at these extremely high dose levels are a consequence of the toxic mechanisms which occur only at high dose levels such as the MTD, and would not be meaningful at lower dose levels because different biological mechanisms would result (Ames and Gold 1990a; Marx 1990; Ames and Gold 1993). Animal carcinogenesis testing is further complicated by the many additional uncertainties about the mechanisms of carcinogenesis itself (Ames and Gold 1993; Rodricks 1992; Graham and Rhomberg 1996).

Exposure Extrapolations

In determining health risks to humans, toxicological data derived from animal studies also involves extrapolating from carefully controlled artificial exposure conditions to variable and complex human exposures with alternate routes of exposure. As opposed to experimental animals, humans experience multiple and cumulative exposures to a variety of chemicals where the interactions between many chemicals are also uncertain.

Interspecies Extrapolation

The validity of animal studies for predicting human health effects is also compromised because of the many uncertainties in extrapolating from animals to humans. Species differences in toxicological response are a major problem creating much uncertainty in interspecies extrapolation. Although mammalian species have

similar biological functioning, animals are obviously not humans. Humans have many important differences in anatomy, physiology, metabolism and biochemistry as well as a much larger spectrum of determinants (including physical, mental and social factors) which influence their health. Therefore, humans can have much different toxicological responses to chemicals than laboratory animals.

Furthermore, there are many examples where different species, and even different strains and sexes, of animals differ widely in their toxicological response to chemicals. For example, acute exposure animal studies involving 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) indicate that as little as 0.0006 mg/kg body weight kills half of guinea pigs tested whereas other experiments show that the hamster is 5000 times less sensitive, requiring 3 mg/kg body weight to kill half the animals. Therefore, to make matters worse, if even rodents can differ so significantly from one another, extrapolating to humans is obviously difficult because of uncertainties about which species, if any, and under what conditions and which chemicals, humans might experience similar toxicological responses. (Gots 1993) Meaningful extrapolation requires some understanding of the toxic mechanisms involved so that interspecies comparisons can be based on an appreciation of relative susceptibility of the species.

Animal studies on carcinogens also indicate that sites of tumor formation often do not match across species, strains and even sexes of test animals. Some experimental animals appear to be uniquely susceptible to carcinogens (Rodricks 1992). For example, the kidney of male rats undergoes a predictable series of quick degenerative changes as they age that seem not to occur in other species, including humans (Rodricks 1992). Likewise, mice tend to be prone to liver cancer which is an uncommon cancer in humans in developed countries.

Interindividual Variation

In addition to interspecies variation, human heterogeneity and interindividual variation also creates much uncertainty in interspecies extrapolation. Human variation in response is generally much greater than that observed in commonly used animal species (Calabrese 1988). Whereas test animals, particularly rodents, are often inbred under identical environmental conditions for genetic homogeneity (Gots 1993), the human population is vastly heterogeneous. Humans follow a broad variety of dietary patterns and divergent lifestyle orientations which all contribute to the highly varied susceptibility to environmental hazards (Calabrese 1988). Furthermore, biological factors such as age, sex, genetic composition, nutritional status, metabolism and preexisting disease conditions inevitably may enhance one's susceptibility to experience adverse health effects from exposure to toxic substances.

Despite the uncertainties associated with extrapolations from animals to humans, toxicological data from animal experiments can provide useful information on a chemical's toxic effects. With the exception of arsenic, all proven human carcinogens have been shown to be carcinogenic in at least one animal species (Harrison and Hoberg 1994). However, this correspondence does not necessarily imply the reverse: that every substance found carcinogenic in one or more animal species will be carcinogenic in humans. There are numerous substances for which there is evidence of carcinogenicity in animal studies but, as yet, no evidence of carcinogenicity in humans. As well, substances which show no evidence of carcinogenicity in animal tests cannot be absolutely rejected as possible human carcinogens (Rodricks 1992).

The many uncertainties in extrapolation from animal to humans indicates that laboratory animals cannot be viewed nondiscriminantly as surrogates for humans.

When evaluating the relevance of animal studies for predicting human health effects, animal studies need to be carefully scrutinized with these and many other considerations in mind especially in the absence of relevant human data. Obviously a chemical is more likely to be a human carcinogen if there is evidence that it produces large excesses of tumors at several sites in several species and strains of test animals in both sexes and at multiple doses than if it produces only a small excess of tumors at a single site in one species and sex, and produces no other excesses of tumors in other species and strains. Similarly, the greater the number of clearly negative outcomes in animal experiments, the more convinced we can become that the substance is not carcinogenic to humans. (Rodricks 1992).

Analysis of Statements

Table 12 presents the results for Statements 3 and 4 for the environmental epidemiologists, environmental engineering professors, toxicologists, public as well as the students from public health science and the Danish students from medicine and a postgraduate course in risk assessment. These statements were included in the survey to determine the value respondents place on animal studies for predicting the effects of chemicals on human health. Since the discipline of risk assessment relies heavily on animal studies, these statements were put forth to examine how much confidence these disciplines have in these data sources.

Table 12 - Responses to Statements 3 and 4

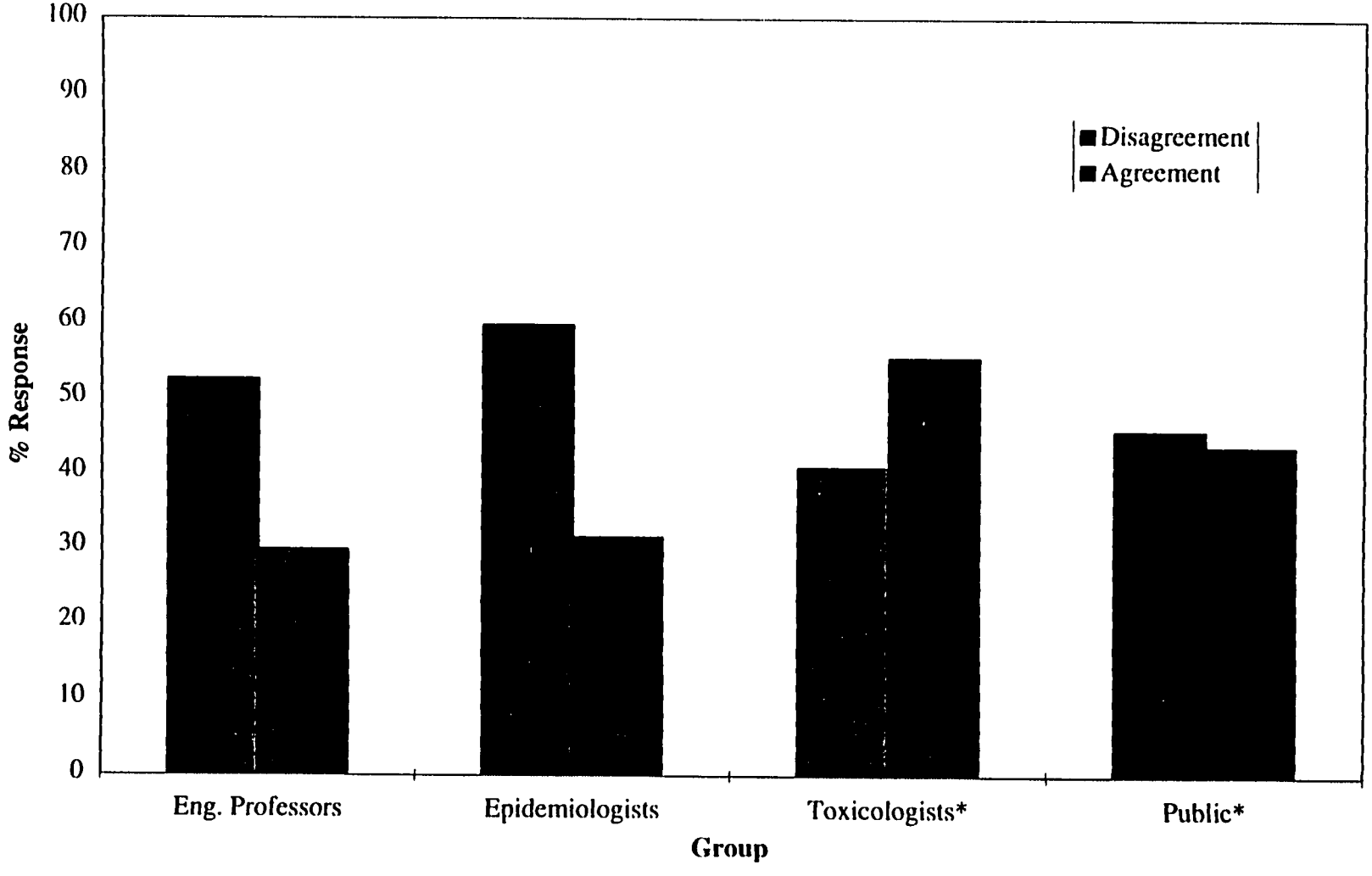
		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
3. The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.	EE[b]	13.1[a]	46.6	29.8	1.6	8.9
	EP[c]	8.4	44.0	28.6	1.0	18.0
	T1[d]	1.9	38.9	50.3	5.1	3.8
	T2[e]	4.0	26.0	56.7	9.3	4.0
	P1[f]	5.5	40.2	40.2	3.5	10.6
	P2[g]	14.9	21.7	39.6	20.7	3.1
	R[h]	13.7	58.8	23.5	1.0	2.9
	M[i]	17.5	53.6	27.8	1.0	0.0
	PH[j]	40.6	43.8	12.5	0.0	3.1
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	EE	9.7	46.9	29.1	2.6	11.7
	EP	5.7	38.3	39.6	2.5	14.0
	T1	10.3	47.3	39.4	1.2	1.8
	T2	7.3	36.0	44.0	7.3	5.3
	P1	1.9	22.9	64.0	5.4	5.8
	P2	4.1	11.8	47.5	34.7	1.9
	R	4.9	54.9	37.3	1.0	2.0
	M	3.1	35.1	50.5	7.2	4.1
	PH	9.4	43.8	40.6	3.1	3.1

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] T1 - American Toxicologists (n = 170; Kraus et al. 1992)
[e] T2 - Canadian Toxicologists (n = 150; Slovic et al. 1995)
[f] P1 - American Public (n = 262; Kraus et al. 1992)
[g] P2 - Canadian Public (n = 1500; Krewski et al. 1995)
[h] R - Danish students in a risk assessment postgraduate course (n = 102; Grandjean and Nielson 1996)
[i] M - Danish medical students (n = 97; Grandjean and Nielson 1996)
[j] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)

Statement 3 states "*The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.*" Although this statement provides no absolute answer, as a general statement without any specifics, I would disagree that animal studies are a reliable predictor of human health effects. Appreciating the substantial uncertainties associated with animal studies such as interspecies, exposure and dose extrapolations, as well as interindividual variation, makes it difficult to generalize animal health effects to humans. However, if animal data were supported by similar human data from epidemiological studies, then an increased confidence in animal studies would result and I would be more inclined to agree.

The responses to this statement from the environmental epidemiologists and environmental engineering professors (Table 12) indicate that both groups were greatly divided in their beliefs about the ability to predict a chemical's effect on human health on the basis of animal studies. Figure 3 demonstrates the divergence in opinion for the environmental engineering professors and the environmental epidemiologists to this statement. There was relatively little difference in the distributions for these two groups. About 59% of environmental epidemiologists and 52% of environmental engineering professors were in disagreement with this statement. There was about 30% agreement overall for both these groups. The environmental engineering professors also had a high percentage of "don't know" responses.

Figure 3. Responses to Statement 3:
"The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical."



* American survey (Kraus et al. 1992)

These findings are consistent with those for the groups of toxicologists in the United States and Canada in that their opinions were also greatly divided (Kraus et al. 1992; Slovic et al. 1995). However, the toxicologist groups showed an opposite pattern of response with the majority of the group agreeing with the statement rather than disagreeing. Of the toxicologists surveyed in the United States, 55% agreed and 41% disagreed with this statement (Kraus et al. 1992). The Canadian toxicologists were found to have more confidence in the validity of animal studies for predicting human harm than their American counterparts (66% agreement and 30% disagreement) (Slovic et al. 1995). Although toxicologists showed a more favorable view of animal studies, there was still a large percentage of toxicologists who doubt the validity of the animal studies that form the backbone of their science.

Of further interest, the public was also found to have divided opinions regarding their trust in extrapolating effects from animals to humans: 43.7% of the American public sampled (Kraus et al. 1992) and 60.3% of the Canadian public sampled (Krewski et al. 1995) agreed with the reliability of animal studies for predicting human harm. There was substantial disagreement from all the students (medical, public health science and students in a postgraduate course in risk assessment) to this statement with the public health science students most strongly disagreeing in the reliability of animal studies for predicting human health effects (84.4% total disagreement with 40.6% strong disagreement).

This statement resulted in many diverging opinions among members of the various disciplinary groups surveyed. Although all three disciplinary groups had divided opinions to this statement, the environmental engineering professors and the environmental epidemiologists were more likely to disagree with this statement than the toxicologists. This suggests that these two groups have less confidence in these types of studies for predicting human harm than the toxicologists. The fact that many respondents in all three of these groups, in addition to the public and the students, lack

confidence in the value of animal studies for predicting human health effects is especially noteworthy considering that toxicological data from animal experiments are often the basis for policy making concerning the regulation of possible hazardous substances, particularly, carcinogenic substances. It seems likely that such discrepancies inevitably contribute to the public's confusion and distrust in risk assessment and risk management.

Qualitative responses to this statement for the environmental epidemiologists and environmental engineering professors indicate that both groups tended to have similar perspectives on the issue raised. Many respondents from both groups indicated awareness of the difficulties associated with extrapolation from animals to humans, and recognized that the relevance of such toxicological data is dependent on many factors such as species differences, exposure conditions and the substance being tested. However, other qualitative responses also indicate that some respondents may not fully appreciate the limitations of animal testing and believe that animal studies are "often", "more often than not" or "for most chemicals" applicable to humans.

While many respondents expressed that animal studies cannot be generalized as reliable predictors for human health, they explain that animal studies can be a "good indicator" and are "sometimes reliable." Many respondents appreciated that animal studies are the best indicator humans have and that a better alternative does not really exist. Only a few of the respondents who provided qualitative responses indicated the need for animal studies to be supported with additional human data from epidemiological studies to make them "reliable predictors."

Although there was a relatively high percentage of "don't know" responses to this statement, particularly from the environmental engineering professors, the comments provided for this category, such as "depends on animal and chemical", "case by case question", "extrapolation is uncertain", "the animal/human connection has not been well established", suggest that some of these respondents used the category not as

"don't know" but rather for "no opinion." Many of these comments were similar to those provided for disagreement and/or agreement. That these respondents were uncomfortable making a general opinion suggests that the issues raised regarding animal studies may not be well thought out or fully understood.

From the available comments, the wording of this statement did not seem to present problems for interpretation. A couple of respondents, however, did indicate that "reliable" was unclear. Another respondent noted that in epidemiology, reliability is equivalent to replicability rather than validity. However, for the most part, interpretation did not appear to be problematic for responses.

Although some qualitative responses (particularly for those in agreement with the statement) suggested that awareness of the limitations of animal studies may not be great, overall, they reflected that most respondents from both groups are familiar with the limitations of animal studies. Unfortunately however, the comments were not extremely useful in evaluating the depth of understanding of these issues. Because of the apparent diversity in opinion regarding the validity of animal studies for predicting human health effects, it is important that environmental scientists carefully evaluate their understanding of the uncertainties and limitations concerning animal studies so as to ensure that misunderstandings are not relayed to others.

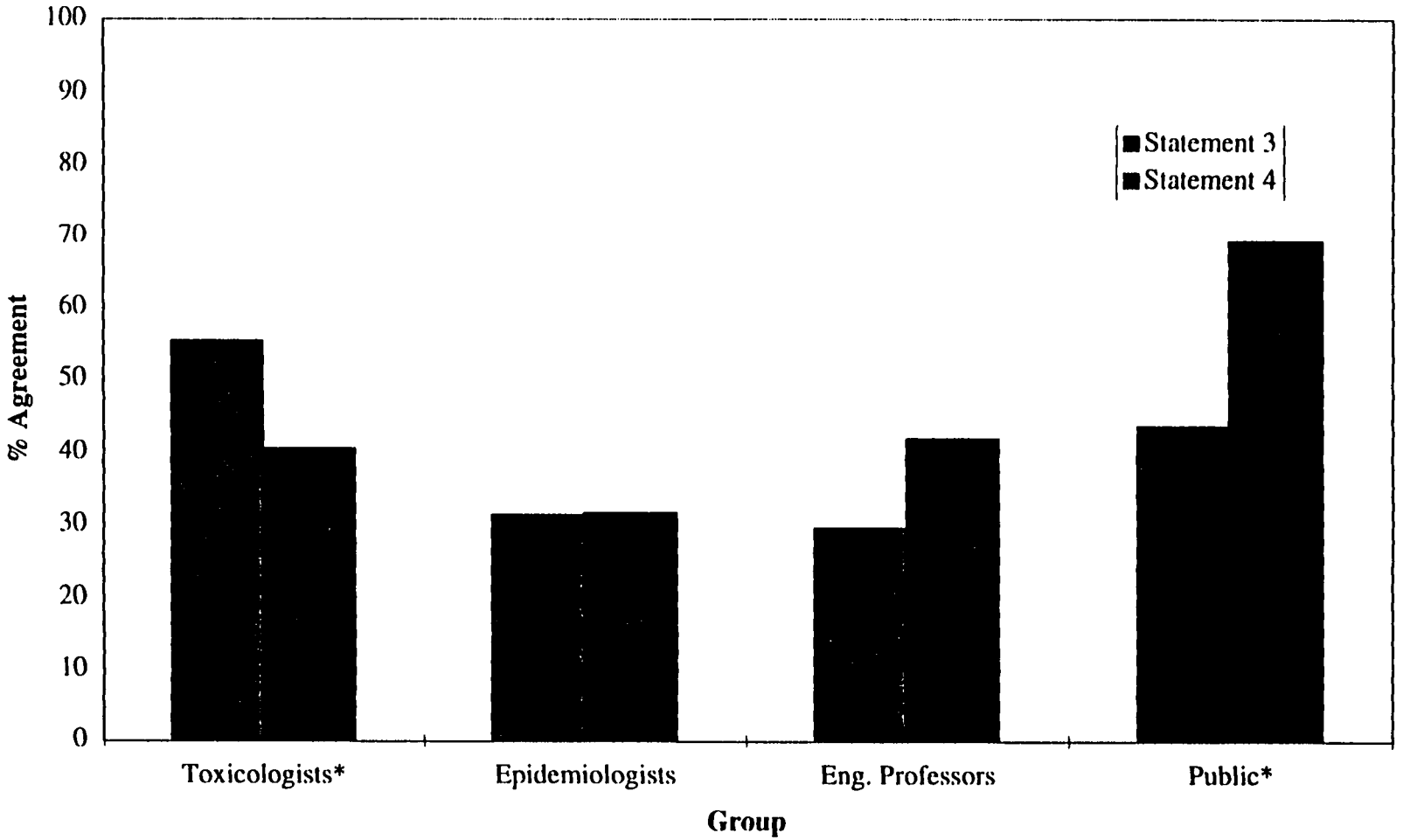
Statement 4 is similar to Statement 3 except it is more specific in that the chemical being tested is shown to cause cancer in animals. The statement reads, "*If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.*" Again this statement has no absolute answer but because cancer testing is even more uncertain with debates about the use of the MTD, the mechanisms of carcinogenesis and the within species variation of carcinogenic effects, the confidence in animal studies is even less for these types of studies than other chronic toxicity tests.

The responses to this statement (Table 12) are similar to those from the previous statement in that all three disciplinary groups are still divided in their opinions regarding the implications of animal studies to human health. However, when animal tests imply cancer, the groups differ in their patterns of responses.

The previous studies by Kraus et al. (1992) and Slovic et al. (1995) showed important differences between the public and toxicologists for this statement when contrasted to the previous one. When an animal study provided evidence of carcinogenicity in animals, many respondents of both these groups changed their opinions concerning the reliability of animal testing to predict human health effects. When a chemical was found to be a carcinogen in animals, the public became much more certain in the prediction of harm to humans, and overall agreement rose substantially from 43.7% to 69.4% for the American sample (Figure 4) (Kraus et al. 1992) and 60.3% to 82.2% for the Canadian sample (Slovic et al. 1995). Although difficult to interpret, the increase in the public's confidence in animal studies showing positive evidence of carcinogenicity can possibly be explained by their familiarity with chemicals being designated as carcinogens or by the dread factor that is often associated with the disease.

Opposite to the public, many toxicologists changed their opinions from agree/strongly agree (or don't know) to disagree/strongly disagree, and the overall percent agreement decreased from 55.4 to 40.6 for the American sample (Figure 4) (Kraus et al. 1992) and from 66.0 to 51.3 for the Canadian sample (Slovic et al. 1995). The finding that the toxicologists became even less confident that similar health effects (cancer) will occur in humans may reflect that these respondents have a greater understanding of the many additional uncertainties associated with carcinogen testing such as use of the MTD and the limited knowledge on the mechanisms of carcinogenicity itself.

Figure 4. Change in agreement for Statement 4:
"If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans."



* American survey (Kraus et al. 1992)

Although this statement caused virtually no change in overall responses from the previous statement for the group of environmental epidemiologists, the environmental engineering professors displayed a pattern of response opposite to that of the toxicologists and similar to the public (Figure 4). When a chemical was found to be a carcinogen in animals, environmental engineering professors became more confident in animal studies for determining similar health effects in humans and overall agreement rose from 29.6% to 42.1% (Figure 4). The fact that this group is more convinced in the validity of animal tests for predicting human harm when the chemical causes cancer in animals is interesting considering this group was the least likely to agree that there is no safe level of exposure to a cancer-causing agent, and also least likely to agree that any exposure, no matter how small, will likely cause cancer. This increased confidence in cancer animal tests suggests that this area may be one in which greater understanding needs to be developed.

The patterns of response for all three groups of students (medical, health and risk assessment) were also similar to that of the public and the environmental engineering professors. Again overall percent agreement rose substantially (Table 12).

Qualitative responses to this statement from the environmental epidemiologists and the environmental engineering professors indicate that not much difference exists between the perspectives of the two groups. For the most part, the qualitative responses for this statement were similar to those from the previous statement and many of the respondents recognized difficulties associated with animal studies, particularly dose extrapolation. Numerous respondents who were in disagreement with Statement 4 felt that "reasonably sure" was too strong a statement while others who were in agreement stated that we can be "reasonably sure but not certain."

Although there was not many comments provided for those who responded "don't know", some of these comments suggest that some respondents did not want to

take an opinion. Only few of the comments provided for "don't know" indicated that the respondent was unsure because of their lack of knowledge.

Particularly interesting was the large number of respondents who felt that, regardless of the uncertainties, positive evidence of carcinogenicity in animals raises a flag for humans, and it is best to err on the side of safety and assume that carcinogenicity will also occur in humans.

From the comments provided, there did not appear to be any difficulty in understanding or interpreting this statement except for the fact that interpreting "reasonably sure" involves some value judgment. Overall, Statements 3 and 4 were effective in evaluating the trust respondents have in animal studies and provided some interesting perspectives.

However, the comments were not particularly useful for explaining the change in patterns of response for the groups. Unfortunately, except for one respondent indicating that "animal studies are better for cancer than other endpoints.", the comments provided offered no further insight as to why so many respondents changed their opinions and became more confident in animal studies when positive evidence of carcinogenicity was the result. One possible explanation may be that "reliable" and "reasonably" may have vastly different interpretations by some of these respondents. The responses may have shown less difference between the two statements had we used similar adjectives in both statements. However, as in the earlier statements, it appears that the discussion of cancer sets off an alarm for some people, most likely for those whose knowledge of cancer and its mechanisms is superficial. A statement evaluating the understanding of carcinogenesis and/or worry people have with developing cancer may have been helpful in discerning these patterns of responses. One such statement from a previous survey is: "*A cancer-causing substance turns normal cells into cancer cells through contact, much like the spread of a highly contagious disease.*" (Krewski et al. 1995). This statement may have been useful in

providing further insight into responses to Statements 3 and 4 as well as for the exposure statements of the previous section.

Causal Inference in Epidemiological Studies

Inferring causation in environmental health risk assessment is a challenging task. For a population, adverse health outcomes due to exposures to hazards are typically determined by epidemiological methods. These methods attempt to develop evidence of an association between some factor and a health outcome by comparing groups of individuals having different exposure scenarios and determining whether there is any differential frequency of a disease (Sowers 1994). Essentially, environmental epidemiology (also known as observational or risk factor epidemiology) tries to distinguish the meaningful from chance correlations between exposure to a hazardous substance and an adverse outcome, and to determine those that may indicate cause and effect.

Applying environmental epidemiology to judge whether an association between exposure and effect is causal is difficult for many reasons. Firstly, unlike infectious diseases which are specific to a single, causative agent (such as a specific virus or bacteria), many of the chronic diseases and other health effects studied in environmental epidemiology (such as heart disease, stroke and cancer) are non-specific and invariably have multiple factors all contributing to cause (web of causation) (Stehbens 1992). Thus, no single factor can be clearly defined as a sole specific cause of a health outcome.

Secondly, the web of causation for a health effect is influenced by other factors which may increase the individual risk of developing a health problem or disease. Risk factors which can contribute to causation for a health effect can be described in four categories (Beaglehole et al. 1993):

- predisposing factors (e.g. age, gender, race),
- enabling factors (e.g. economic class, nutritional status, living conditions, health care),

- precipitating factors (e.g. exposure to a specific harmful agent), and
- reinforcing factors (e.g. repeated exposure).

Thirdly, obtaining persuasive causal evidence by epidemiological methods is also difficult because of the many methodological limitations inherent in environmental epidemiological methods. Because of their observational rather than experimental nature, environmental epidemiological studies suffer from bias and confounding. Bias represents anything that might influence the magnitude and direction of observed results and lead a study to reach the wrong answer, to postulate the existence of a causal association that does not exist, or vice versa (Maldonado 1994). Confounding is a particular source of bias resulting from failure to account for confounding factors other than the hypothetical cause which may increase or decrease the rate of disease.

Because bias and confounding are inherent in epidemiological methods, they must be thoroughly evaluated when inferring associations between exposures and adverse health effects. Therefore, to correctly conduct and interpret epidemiological studies, the first critical step is to recognize and understand the important potential sources of bias and confounding so that they can be eliminated, or where elimination is not possible, they can be evaluated for their influence on the observed results.

Bias

Biases are often imbedded within the study designs. Two important types of bias that can occur in epidemiological studies are classified as selection bias and information bias (Beaglehole et al. 1993). Selection bias arises from the design of an epidemiological study. Environmental epidemiological studies often use the case-control design whereby cases of a disease are identified and then information on their exposure is gathered retrospectively. This design requires matched controls (individuals without the disease) who are also assessed for their exposures. Selection

bias can arise according to the manner in which cases or controls are selected for study. The process of choosing an appropriate population of controls can easily lead to an apparent difference between cases and controls that has nothing to do with the hypothesized cause of the disease. Biased sampling can also arise from differential migration, susceptibility, and low response or loss of follow-up (Hrudey and Chen 1996).

Information bias is caused by errors in measuring (or classifying) the study variables dealing with either exposure or health outcome. This often occurs in the data collection stage of a study. Since there are many sources of measurement error, the effect of information bias can be large. Various types of information bias are misclassification, interviewer bias, and recall or reporting bias (Maldonado 1994).

Recall or reporting bias can be the most difficult to control for. Because accurate exposure measurements are difficult to obtain, exposure data often comes from memory of the subjects (Foster et al. 1993). Recall bias is especially strong among patients diagnosed with the disease in question. For instance, when searching for a possible relationship between fat intake and breast cancer, people who have just recently been diagnosed with breast cancer may recall their past intake of fat differently than a person without cancer who is selected from a random sample (Taubes 1995). People diagnosed with a disease are more likely to give complete information regarding their exposure.

Identified biases are often difficult, if not impossible, to assess. Information bias can only be handled by minimizing measurement error or by making inferences about the magnitude and direction of bias (Maldonado 1994).

Confounding

Confounding factors are factors, other than the hypothetical risk factor being studied, that can substantially influence the observed results in an epidemiological study. For example, cigarette smoking can confound studies examining a causal association between cancer and coffee or alcohol consumption because coffee or alcohol consumption may be linked to smoking behavior. Failure to consider whether coffee or alcohol consumers are more likely to be smokers will risk having the observed cancer causation confounded by smoking behavior. When interpreting relationships between exposure and health effects, it is always essential to consider possible alternative explanations for the observations.

For confounding factors that have been measured, study design (such as randomization, restriction and matching) and data analysis techniques (such as stratified and multivariate analysis) can be used to adjust for confounding factors (Maldonado 1994). However, measuring all possible confounders is impossible and evaluating confounding caused by unmeasured confounders is a problem.

Criteria for Causal Inference

Because associations could be the result of biases, confounding factors, or even random chance, inferring cause and effect relationships from epidemiological evidence requires careful and thoughtful evaluation of all available evidence. Therefore, to help in assessing the strength of evidence supporting causality various criteria have developed within epidemiology. These include the Surgeon General of the United States criteria (SGACSH 1964) and Hill's criteria (Hill 1965). More recent criteria such as Susser's criteria (1991) have made alterations and extensions to the earlier criteria.

Some of the more important and commonly used criteria for evaluating the evidence for causation are (Beaglehole et al. 1993):

- temporality,
- strength of association,
- consistency among studies,
- plausibility,
- dose-response relationship,
- reversibility, and
- study design.

Temporality

The temporal relationship of the observed association is an important consideration in assessing causality. To be a cause, the suspected factor must precede the effect. Although this criterion is conceptually simple, the time sequence of exposure and effect is often ambiguous; especially for diseases with long latency periods such as cancer. For this reason, despite its critical importance, temporality is often difficult to assess (Spivey 1994; Hill 1965).

Strength of Association

This criterion considers how large and statistically significant the apparent relationship between exposure and effect appears to be. The strength of an observed association is usually measured by the relative risk. An association which shows a substantial increase in risk for a disease when there is exposure to the proposed cause will obviously be much more convincing than a case where there is only a small increase in risk. One of the strongest associations documented is the large increase in risk for lung cancer among smokers versus non-smokers. Smokers have been found to

have an increased risk of lung cancer ranging from 4 to 20 times higher than the risk for non-smokers (Beaglehole et al. 1993). This increased factor is termed the relative risk rate ratio or risk ratio (RR). Unfortunately, most causal hypotheses have generally much weaker associations, with RR values often less than 2.

Because environmental epidemiological studies are inherently limited in their ability to detect a small RR in a population, it is difficult to support a causal hypothesis for a subtle effect.

Consistency on Replication

Another important criterion is consistency among studies. Because observational epidemiological studies are not controlled and there is great potential for systematic errors, no single study, regardless of how strong the association is determined to be, can provide sufficiently definitive evidence to prove causation (Rodricks 1992; Rothman 1988). Rather, evidence of causality becomes more compelling when several studies of a relationship present similar consistent findings. However, consistency among findings is only persuasive for causality if the observed relationship is repeatedly found by different investigators using a variety of study designs with different populations, in different locations, circumstances and time (Hill 1965). As with the other criterion, lack of consistency is not sufficient to reject a cause-effect relationship because of the variability in quality and strength of study design and performance.

Plausibility

One of the strongest criteria for causality is if there is toxicological evidence on the biological mechanisms of an effect. Biological plausibility is very supportive of causality if there is some reasonable hypothesis for a mechanism of action. Unfortunately, causation cannot be ruled out if there is no evidence of mechanisms

because it may be that the mechanism is not yet understood. This criteria is perhaps most useful where strong experimental evidence is available to counter the plausibility of a hypothesized causal relationship (Hrudey and Chen 1996).

Dose-Response Relationship

Demonstration of a clear relationship between exposure to a proposed cause and response in individuals also provides strong evidence in support of causation. However, revealing a dose-response relationship does not exclude the possible role of confounding factors. Furthermore, failure to demonstrate a dose-response relationship may be a function of the quality of data and may not be useful for rejecting causation. (Hrudey and Chen 1996)

Reversibility

Removal of a hypothesized cause resulting in reduction or elimination of the adverse effect will also strongly support the existence of a cause-effect relationship.

Study Design

Because the nature of the study design dictates what conclusions can be drawn, it must also be considered when judging the causal evidence obtained. When determining the possible existence of a cause-effect relationship, there is a hierarchy of study designs for epidemiology (Sowers 1994):

- experimental approaches,
- observational analytical approaches, and
- descriptive approaches.

The choice of study design depends primarily on feasibility, efficiency (cost, time, etc.) and purpose of investigation.

Experimental approaches such as the randomized trial are the strongest and most powerful study designs in epidemiology for establishing associations or supporting cause-effect relationships. In a randomized trial, subjects are selected at random to test and control groups. As the test group is exposed under experimental conditions to the suspected risk factor, both exposure and control groups are followed to obtain information on outcomes. These approaches are rare in environmental epidemiology because they are usually neither practical (cost, time, etc.) nor ethical.

Observational analytical approaches such as cohort and case-control studies are the most common in environmental epidemiology. As noted previously, case-control studies involve a group of individuals that have been diagnosed with a particular disease (or other effect) which is compared to a control group who are free of the disease to determine the exposures of each group to the risk factor(s) for the disease. A cohort study is one in which a large population is assembled. The cohort is then followed over time to determine their individual exposure to specified risk factors and their individual health outcomes. Cohort studies are potentially more reliable, but also more costly, than case-control studies.

At the bottom of the hierarchy are descriptive approaches such as ecological studies. These approaches are the weakest study designs for establishing associations or supporting cause-effect relationships. Unlike the hypothesis testing approaches of experimental and observational methods, descriptive approaches are only useful for generating hypotheses. These types of studies can only generate causal hypotheses because they use aggregate data for population exposure and health outcome rates. They do not collect data on individuals so it is not possible to know if any individual who has the disease was also exposed to the hypothesized cause. Therefore, these approaches cannot provide any substantial evidence to support causal inferences.

Evaluation of the above criteria is useful for judging causality from epidemiological methods. However, it is important to realize that, except for temporality, these criteria are only guidelines for causal inference and hence, cannot be used as indisputable evidence for or against a cause-effect hypothesis. If temporality is clearly shown to be violated, then there is no evidence for causation. Obviously, the more criteria that are satisfied, the more compelling is the evidence for causality between a factor and an effect. However, unless there has been a serious attempt to control and evaluate the effects of bias and confounding, positive evidence of these criteria may still be meaningless. Furthermore, failure to meet these criteria only indicates a failure to provide positive evidence in support of a hypothesized cause; it does not prove the absence of a causal contribution of the proposed factor.

When evaluating the epidemiological evidence for causality, scientists need to examine all the evidence with these criteria in mind. Furthermore, it is important to recognize that because of the numerous uncertainties and methodological difficulties, epidemiological studies, strictly by themselves, cannot prove causation (Hrudey 1996). Because epidemiological methods cannot establish the biological mechanisms necessary to demonstrate causation, they are only capable of demonstrating an association between an exposure and an effect. However, when epidemiological studies are supported by highly plausible and specific biological mechanisms determined from toxicological studies, and various other causation criteria are met, such as strong consistent associations, confidence in the demonstration of causation can be increased to reasonable certainty.

Analysis of Statements

Table 13 presents the results for Statements 14 and 19 for the environmental epidemiologists, environmental engineering professors and public health students.

Results for Statement 19 from the toxicologists and public (American sample) as well as the Danish medical and risk assessment students are also included. These statements were included in the survey to assess how respondents interpret epidemiological evidence regarding cause-effect relationships.

Table 13 - Responses to Statements 14 and 19

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
14. A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.	EE[b]	30.4[a]	52.6	13.4	1.5	2.1
	EP[c]	24.0	53.2	11.8	1.5	9.6
	PH[d]	40.0	53.3	6.7	0.0	0.0
19. Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.	EE	19.1	47.4	8.2	0.5	24.7
	EP	11.2	44.9	9.3	0.2	34.4
	T1[e]	22.2	59.3	4.3	1.2	13.0
	P1[f]	3.9	23.4	39.5	9.0	24.2
	R[g]	16.7	52.9	18.6	0.0	11.8
	M[h]	4.1	21.6	60.8	6.2	7.2
	PH	10.0	13.3	30.0	3.3	43.3

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)
[e] T1 - American Toxicologists (n = 170; Kraus et al. 1992)
[f] P1 - American Public (n = 262; Kraus et al. 1992)
[g] R - Danish students in a risk assessment postgraduate course (n = 102; Grandjean and Nielson 1996)
[h] M - Danish medical students (n = 97; Grandjean and Nielson 1996)

Statement 14, "*A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.*" was designed to assess whether respondents recognize the importance of evaluating various causal criteria, the most important of which are strength of association, consistency and a highly plausible biological mechanism. Because epidemiological studies are often not

controlled in the sense of an experimental study and thus there is great potential for systematic errors such as bias and confounding, no single study, regardless of how strong the association is, can provide sufficiently definitive evidence to prove causation. Furthermore, because epidemiological studies cannot establish the biological mechanisms necessary to demonstrate causation, they are only capable of demonstrating an association. Strictly by themselves, epidemiological studies cannot prove causation.

Strong disagreement was found for both the environmental engineering professors and the environmental epidemiologists to the statement "*A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.*" (Table 13). The epidemiologists showed slightly more disagreement (83.0% disagreement overall - 30.4% strong disagreement) than the engineering professors (77.2% disagreement overall - 24.0% strong disagreement). About 15% of the respondents in both groups agreed that a single epidemiology study can be sufficient to establish a cause-effect relationship. Almost 10% of the engineering professors responded "don't know" to this statement. There was almost unanimous disagreement to this statement from the students in public health science (93.3% total disagreement - with 40.0% strongly disagree).

The qualitative responses indicated similar perspectives from the two groups. The most common theme among disagreement responses was that causation cannot be concluded from only one study, and that consistency in replication is important for making causal inferences. Only four environmental epidemiologists and one environmental engineering professor indicated understanding that epidemiology can never establish causation's but can only demonstrate associations. As well, only two respondents (in disagreement category) indicated the need for evidence of biological mechanisms from toxicological investigations to support an epidemiological study when inferring causation. Another common response for this category from

respondents in both groups was that generally a single study cannot establish causation but can under "very rare circumstances", and "depending on the quality of study" (such as study design and size of sample) or "if results are overwhelming." "Depends on the strength (of association)" between the risk factor and health effect was also a repeated comment for those in disagreement with Statement 14, particularly for the group of environmental epidemiologists.

These comments suggest that, while most recognized that an isolated epidemiological study cannot prove causation, awareness that biological mechanisms are necessary for establishing causation may not be as great among these respondents. Thus many of these respondents may have agreed with the statement that an epidemiological study can prove causation had other criteria such as a strong, consistent association and/or a good study design were satisfied.

Similarly, the majority of qualitative responses for those in categories of agreement with the statement or "don't know" stated that if the study is a good, quality study (e.g. properly designed, executed and interpreted) then it can establish causation. Some indicated a single study can determine causation "if the effect is very high" and/or "if there is a large sample population." Some respondents in this category also indicated that establishing causation from a single study only happens under very rare circumstances such as in an occupational setting. There was almost no recognition from these respondents in either group of the need for plausible biological mechanisms for establishing causation. Only one respondent (environmental epidemiologist) stated that epidemiological studies can establish causation "when confirmed by animal studies."

Although the qualitative responses were useful for indicating that the majority of respondents from both groups are familiar (to some extent) with various criteria for judging causality, they were not helpful for interpreting whether the respondents appreciate that epidemiological approaches can only demonstrate an association between

exposure to a risk factor and an effect and can never by themselves establish causation. However, the comments provided (and lack of comments) suggest that a small but substantial percentage of respondents from both groups may not appreciate this. Furthermore, some engineering professors indicated they did not have much knowledge with environmental epidemiology. Therefore, this area may be one in which greater understanding may be beneficial.

This statement did not appear to pose any difficulties for the respondents. Furthermore, it appeared to be effective in determining whether respondents recognize the importance of evaluating various criteria or causal inference. However, it was not as effective for interpreting whether respondents have an appreciation that epidemiological studies can only support causation by demonstrating an association but cannot solely establish causation. This is likely because of the context of the statement which addressed two issues: a single study and epidemiology establishing causation. As the statement was worded, it may have lead people to focus only on the phrase "single epidemiological study" and not so much on "causes a specific human health effect." If this was the case then it is reasonable that so many respondents' comments focused on the criteria of consistency.

It may have been more effective if we had two separate statements addressing the key issues presented. A possible improvement over the statement may have been "*A single epidemiology study can be sufficient to establish an association between a contaminant in the environment and a specific human health effect.*" or "*A strong association can be sufficient to establish an association between a contaminant in the environment and a specific human health effect.*" Statements for evaluating whether respondents believe epidemiology can by itself provide enough evidence to establish causality or whether they recognize the important criteria of biological plausibility for causation may have included: "*Epidemiological studies, in some cases, can provide sufficient evidence to establish that a contaminant in the environment causes a specific*

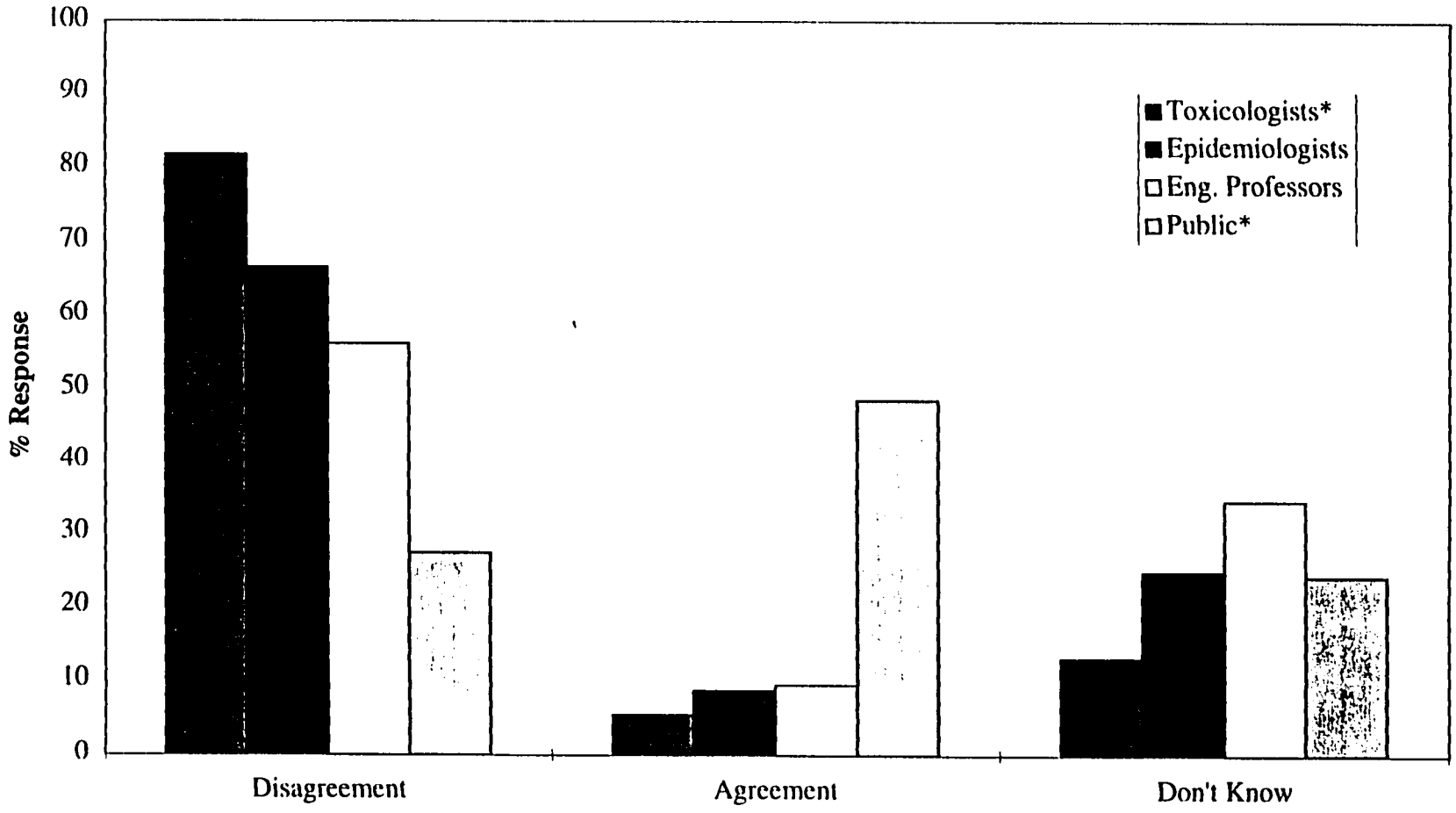
human health effect." and "*A strong consistent association can be sufficient to establish that a contaminant in the environment causes a specific human health effect.*"

Statement 19, "*Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.*", was put forth to assess how respondents interpret evidence regarding cause-effect relationships. While the hypothesized association may warrant further epidemiological investigation, the evidence provided in this statement is insufficient to make any such linkage between pesticide use and malformed children.

The findings from "Intuitive Toxicology" to Statement 19 indicate that the public was much more likely than the toxicologists to view an association between pesticide use and birth defects as a causal relationship. Almost half (48.5%) of the public agreed with this statement versus only 5.5% of the toxicologists surveyed (Kraus et al. 1992). 27.3% of the public disagreed and 24.2% responded "don't know" (Figure 5).

The majority in all three disciplinary groups were in disagreement with this statement. Of the three disciplinary groups, the toxicologists group was most likely to disagree (81.5% overall - 22.2% strongly disagree) and the least likely to agree (5.5%) with this statement (Kraus et al. 1992). In contrast, the environmental engineering professors were the least likely of the three groups to disagree (only 56.1% overall; 11.2% strongly disagree) and the most likely to agree (9.5%) with the statement. The environmental epidemiologists fit in between with percent disagreement at 66.5% (19.1 % strongly disagree) and percent agreement at 8.7% (Figure 5).

Figure 5. Responses to Statement 19:
"Residents of a small community observed that malformed children had been born there during each of the past few years. The town is in a region where pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations."



* American survey (Kraus et al. 1992)

Similar to the public, there was a high proportion of "don't know" responses from both the environmental epidemiologists and the environmental engineering professors. Both these groups were just as likely, if not more, to respond "don't know" as the public was (24.2%) (Kraus et al. 1992). "Don't know" responses for the toxicologists, epidemiologists and the engineering professors were 13.0% (Kraus et al. 1992), 24.7% and 34.4% respectively (Figure 5).

The responses from the students were more similar to the public in that they indicate much greater agreement to this statement than the disciplinary groups. A substantial portion of the Danish medical students (67.0%) agreed that it is very likely that pesticide use was the cause of the malformations.

The most common comment for those who disagreed with this statement (from both the environmental engineering professors and the environmental epidemiologists) was that the statement contained insufficient information/evidence to conclude an association or causal linkage. Some respondents specifically indicated that you need information on other surrounding communities, background rates of malformations and/or exposures before making any causal claims. Many respondents stated that the wording "very likely" is too strong for such a claim. However, some indicated that it was "possible" or "not very likely" or that the hypothesis warrants an extensive investigation. Many other respondents indicated that there are too many potential variables to test and that the malformations could be caused by other important factors such as smoking, drugs, genetics, diet, lifestyle, etc.

There were few qualitative responses for those respondents in agreement with the statement. However, of the ones provided, some felt that the hypothesis is reasonable and it is "likely" (often not "very likely") that the pesticides contributed to the malformations and indicated a need to investigate.

For those who responded "don't know" to whether it is very likely the pesticides caused the malformations this statement, the comments provided are similar

to the comments for those who disagreed with the statement. Comments such as "insufficient information to conclude", "need a study done", "could be other factors", "likely but not very likely", etc. were provided for this category. This suggests that the high percentage of "don't know" responses for these two groups may be attributable to respondents interpreting "don't know" as "unable to conclude from the statement given" rather than meaning "unable to respond due to lack of individual knowledge" as was intended. Thus, it appears that many of these responses would be more accurately reflected as "disagree" or "strongly disagree."

Overall, the results for these two statements did not show large differences in interpretation between the two groups. Both groups appeared to have reasonable understanding of some limitations of environmental epidemiology. However, the comments provided indicate that there may be some information that may not be well understood. The discrepancies between the responses for the scientific disciplines and the public suggest that this area may be one which is likely to lead to serious misunderstanding in a public forum. Therefore, to improve understanding and communication, it is important for environmental scientists to recognize all the limitations of environmental epidemiology and the need for careful evaluation of the evidence using all criteria when inferring causation.

Statistical Inference in Environmental Health Risk Analyses

Epidemiological and toxicological methods are 2 main approaches used for identifying and evaluating potential health risks from environmental contaminants. Because statistical analyses are inherent in both these methods, it is imperative to understand the important concepts of statistical inference in order to accurately interpret their findings.

Tests of Significance

A common statistical technique used in both epidemiological and toxicological methods is significance testing. Tests of significance are used to determine whether any observed differences in groups could have occurred solely by random chance alone, i.e. by sampling error rather than because of true differences in measurements. Tests of significance evaluate the observed results by testing the null hypothesis (the observed differences are due to random chance) against an alternate hypothesis (the observed differences are due to factors other than random variation).

P-value

Significance tests are usually measured by the probability value or P-value of the data. The P-value is determined through statistical analysis and expresses the probability that observed differences in results could have occurred strictly by chance alone. Therefore, a low P-value means a low probability this has happened while a high P-value is more indicative that the differences in observation are due to chance (Cohn 1989).

Understanding the P-value is critical for interpreting statistical findings in environmental health risk assessment. Firstly, it is important to recognize that the P-value only refers to the probability that the null hypothesis may still be true (Walter 1995). Thus, the P-value can never falsify the null hypothesis. Secondly, the P-value is dependent on both the size of the samples being compared. Therefore, if the size of samples is inadequate, testing the null hypothesis for a given P-value may result in a "non-significant" finding simply because there were too few subjects to detect an effect which may have been evident in a larger sample. Conversely, a highly "significant" P-value can sometimes result from negligible differences in very large samples yielding a result of no practical significance. Thirdly, the P-value is also dependent on magnitude of the differences found between groups. Statistical significance testing has a limited ability to detect small, subtle effects among a population. Finally, the P-value selected for significance testing is a trade-off between making Type I and Type II errors. The Type I error is the chance of concluding there is an effect when there is not (false positive) while a Type II error is the chance of concluding there is no effect when there is one (false negative). Conventionally this trade-off has been set at a P-value of 0.05, but this value has no more fundamental meaning for the interpretation of whether the observed effect is real (Hrudey and Chen 1996).

Confidence Intervals

As an alternative to the P-value, statistical significance of an association can also be expressed in terms of a confidence interval. Confidence intervals attempt to account for uncertainty by giving a range of the observed results in which the null hypothesis (absence of a real effect) is not expected to fall. Although advanced statistical techniques attempt to correct for known confounders and control for theoretical effects of biases, the calculation of confidence intervals only takes into account the random

variation in the data and not the systematic errors, such as bias and confounding, which may have a great influence on the statistical variation (Hrudey and Chen 1996). Therefore, regardless of the significance level achieved by the P-value and its corresponding confidence interval, the findings will not be valid or reliable if the data available are subject to severe bias or confounding. Furthermore, similar to the P-value, confidence intervals are also influenced by the size of the sample with a larger sample producing narrower confidence limits.

Statistical Significance

Because the concept of significance may also present difficulties when interpreting environmental health information, understanding what is meant by the terms statistically "significant" or "non-significant" is important. Whereas in common language the word "significant" is logically understood as "important" (Jardine and Hrudey 1997), in statistics, the term "significant" has a much different meaning. When the P-value calculated for the study results is found to be less than a specified significance level, the hypothesized association is said to be statistically "significant" at that level. Using the conventional P-value of 0.05 means that if the P-value calculated for the observed results was less than 0.05, the observed results would be deemed statistically significant at the 5% level. This test means that if the null hypothesis is true (no effect) there would be less than a 5% chance of observing results that inconsistent with the null hypothesis. This suggests it is unlikely, but clearly not impossible, that the null hypothesis is true. Thus, any observed P-values less than or equal to the specified significance level are deemed "significant" while those greater are "non-significant".

Thus, statistically speaking, the term "significant" only indicates that it is unlikely to some degree that observed differences are due to random chance. Unlike

what logically may come to mind, the term says nothing about the importance of the differences observed. Furthermore, "non-significant" findings do not disprove an association because it may just be the case that the sample size was inadequate to detect something that might have shown to be significant with a larger sample (Type II error).

Causal Inference

In addition to these foregoing concepts, it is also important to distinguish statistical inference from causal inference. Although statistical analysis is essential for interpreting the data of epidemiological and toxicological studies, it has a very limited role in explaining causation. It is important to recognize that statistical testing and corresponding references to confidence intervals provide only a narrow interpretation about the meaning of results and that statistical significance does not reflect the biological or practical significance, nor does it confirm the existence of a cause-effect relationship (Jardine and Hrudey 1997). In fact, significance testing only provides information on whether observed differences are caused strictly by random variation. Although this analysis may provide evidence that observed differences are real and because of factors other than random variation, it cannot provide any insight into whether the magnitude of observed differences is important. Furthermore, even when there is a great probability that differences are due to factors other than random variation, significance testing does not actually prove whether the hypothesized factor is in fact the cause of the observed differences. Other causes for statistically significant differences which cannot be ruled out include systematic bias (errors in measurement) and confounders (factors other than those suspected which unknowingly influence causality).

Analysis of Statements

Table 14 presents the results for Statements 17 and 18 for the environmental epidemiologists, environmental engineering professors and the public health science students. These statements were designed to assess the respondents' understanding of statistical inference in environmental health risk assessment. These statements were not included in the surveys of the toxicologists and the public nor in the surveys of the Danish students. Therefore, no comparisons can be made with these groups.

Table 14 - Responses to Statements 17 and 18

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	EE[b]	31.1[a]	46.1	14.0	0.0	8.8
	EP[c]	14.5	40.5	22.1	2.0	20.9
	PH[d]	29.0	32.3	9.7	0.0	29.0
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.	EE	20.2	36.8	31.6	1.6	9.8
	EP	7.3	33.4	25.4	2.0	32.0
	PH	10.0	30.0	16.7	3.3	40.0

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)

Statement 17, "*A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.*", was included in the survey to assess the value respondents place on using statistics to establish causation. Because statistics are widely used for explaining environmental health data, it is important to understand the meaning of statistical concepts in order to avoid making common errors when interpreting them. Firstly, while statistical analysis

can aid in supporting a causal hypothesis (similar to the other causal criteria), statistical significance alone can never confirm a causal hypothesis. Statistical significance only provides information on whether observed differences are caused strictly by random variation. Furthermore, even if statistical analysis indicates the observed differences are real and not caused by random variation, it cannot provide insight into whether the magnitude of observed differences is important nor can it actually prove that the risk factor being studied is in fact the cause of the differences.

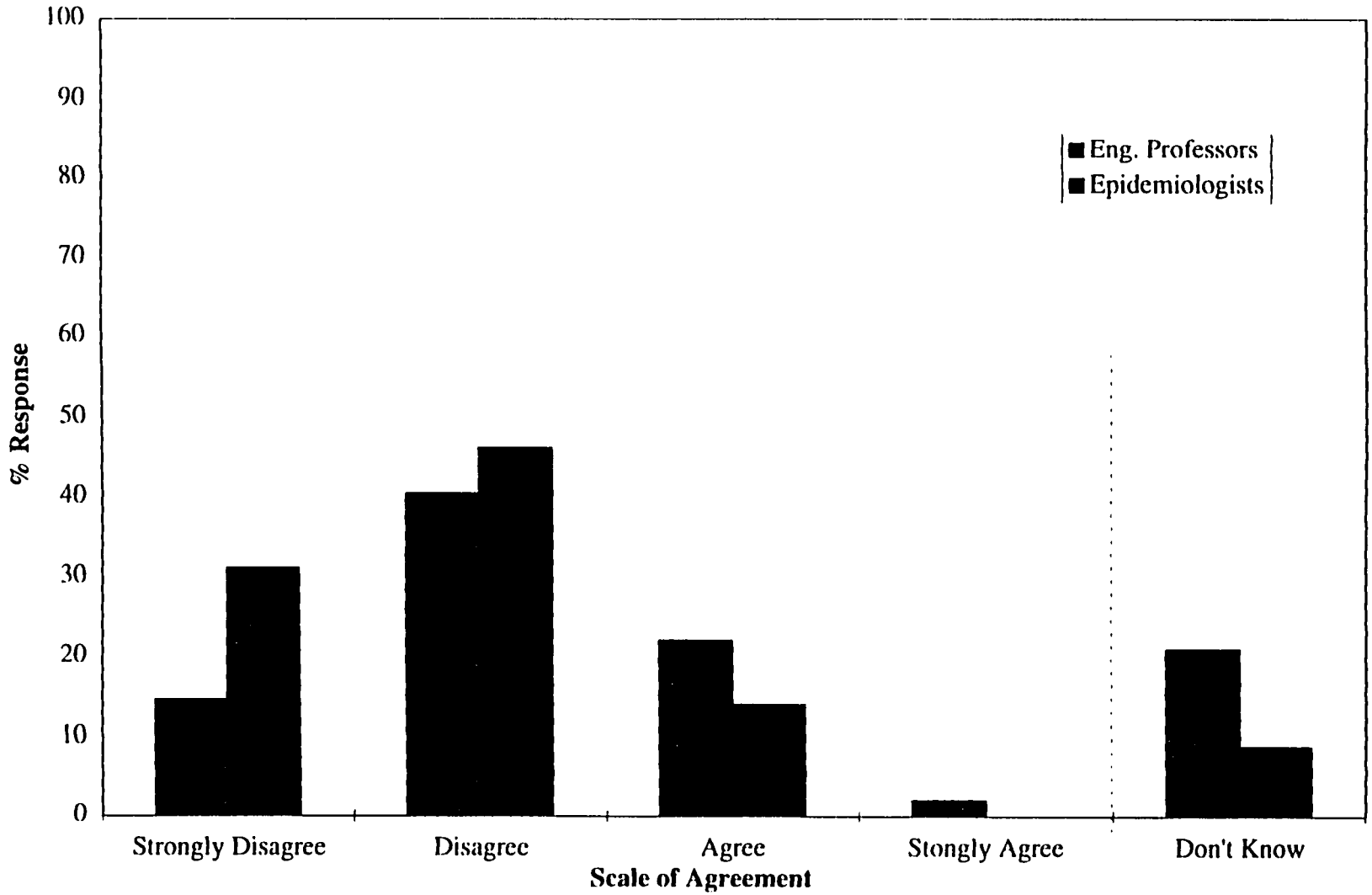
Responses for Statement 17 (Table 14) indicate that the majority of respondents in both the environmental engineering professors and the environmental epidemiologists groups disagreed that a statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis (Figure 6). The environmental epidemiologists were much more likely to disagree with the statement than the engineering professors (77.2% versus 55.0%). Epidemiologists also had a much greater percentage of "strongly disagree" responses (31.1% versus 14.5%). It seems reasonable that the greater disagreement among environmental epidemiologists is because this group may possibly have more opportunity for understanding the role of statistical inference in environmental health risk assessment.

Despite the majority disagreement, there was still a relatively large number of respondents in agreement with this statement. The group of environmental of engineering professors showed 24.1% agreement. The engineering group was also more likely to respond "don't know" to whether a statistically significant association between an environmental contaminant and a health effect can confirm a causal hypothesis (20.9% versus 8.8%) (Figure 6). The fact that a substantial portion (45%) of environmental engineering professors was in agreement or unsure of this statement suggests that the role of statistical inference in environmental health risk assessment may not be fully understood by this group. This is an important finding suggesting the

opportunity to inform environmental engineering professors regarding statistical inferences in environmental health risk assessment.

There was also a relatively high percentage of "don't know" response from the students in public health science (29.0%). The majority of these respondents were in disagreement with Statement 17.

Figure 6. Responses to Statement 17:
"A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can <u>confirm</u> a causal hypothesis."



For respondents who were in agreement with this statement, few qualitative responses were provided. However, these comments along with the moderate percentage of agreement suggests that many may be making common errors when interpreting statistics. Some felt this statement to be true if the study conducted was of good quality and design, and if confounders and biases were accounted for. Agreement was also explained in situations where "other studies showed similar associations." While these factors will aid in supporting causal inference, these responses are overly optimistic about the ability to account for biasing and confounding. Similar to Statement 14, it appears these respondents neglect the understanding that association does not mean causation, and that statistical significance alone can never ascertain or confirm a cause-effect hypothesis.

A number of respondents who responded "don't know" and provided comments to this statement indicated that they had "little understanding in this area" or that this was "outside (their) area of expertise." Other responses in this category were similar to those respondents in agreement such as "depends on the study design", "depends on the size of population", "if other influences have been ruled out", etc. Comments such as these also indicate that this area may be one in which some misunderstanding is apparent.

The comments provided from those in disagreement indicate that many appreciate statistics as a narrow analysis of results. Many of the comments encompassed the concepts presented earlier in the discussion. The most common response was that "statistics can only support a causal hypothesis but can never confirm one." Other comments stated the importance of other criteria, such as Hill's criteria, for causal inference. Some comments explicitly stated the difficulties that factors such as bias and confounding and sample size present in interpreting statistics.

An apparent problem with this statement may have been that the word "confirm" was underlined. As indicated by a few comments, some respondents disregarded the

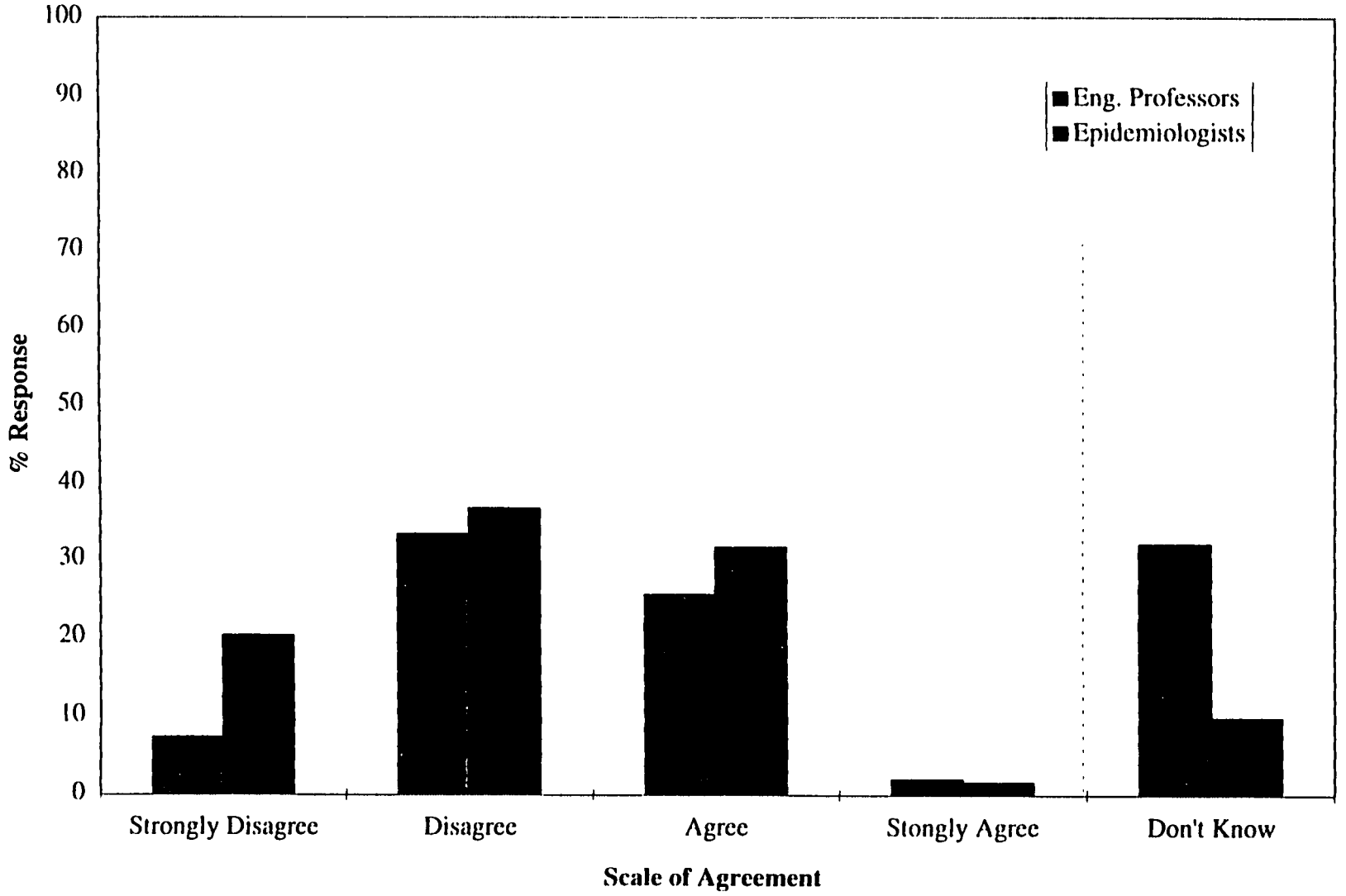
statistical aspect of this statement and focused only on the "confirmation of a causal hypothesis" aspect stating that "causality can never be confirmed or proved." For these specific responses, it is difficult to detect the understanding respondents have of statistical inferences. Furthermore, it is likely that underlying this word may have led respondents to disagree. While we were attempting to highlight that word in the statement we did not intend it to be the focus. Thus, the statement would likely have been more effective had we not underlined "confirm."

Statement 18, "*Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.*", was designed to evaluate the interpretation and understanding respondents have of the presentation of statistical findings in environmental epidemiology. Specifically, the recognition that while confidence intervals account for uncertainty due to random variation, they cannot account for all the sources of uncertainty such as bias and confounding which can have a much greater influence on the statistical variation. Although some bias and confounding can be handled through data analysis techniques, identifying all sources and accounting for them is difficult if not impossible.

Both groups were quite divided in their opinions to Statement 18 (Table 14). Figure 7 displays moderate disagreement for both groups (57.0% disagreement for epidemiologists; 40.7% disagreement for engineering professors). Again the environmental epidemiologists were over twice as likely to strongly disagree with this statement that the engineering professors. However, there was still a substantial portion of epidemiologists in agreement (33.2%) that statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study. There was less agreement to this statement from the environmental engineering professors (27.4%). However, 32.0% from this group responded "don't know."

A substantial portion of the public health science students also responded "don't know" to this statement (40%) while another 40% were in disagreement and 20% were in agreement that statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.

Figure 7. Responses to Statement 18:
"Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study."



As might be expected, the comments of the environmental epidemiologists in disagreement with Statement 18, suggested that this group displayed much more understanding of interpreting confidence intervals in environmental health risk assessment. Whereas the engineering professors did not provide many explanations for their responses, many of the epidemiologists stated that confidence intervals do not reflect uncertainty associated with bias and confounding. Furthermore, some respondents indicated that confidence intervals only address uncertainty due to random variation.

A common response from respondents from both groups (for agreement as well as disagreement) indicated that confidence intervals are dictated by the size of the sample with larger samples enabling narrower confidence intervals. For these specific responses, it is difficult to interpret the understanding respondents have of the uncertainty accounted for in confidence intervals for epidemiological studies.

Very few of those in agreement provided comments regarding random variation or effects from bias and confounding. Several respondents believed that confidence intervals do accurately represent "most but not all" of the uncertainty associated with epidemiological findings. Others felt this to be true if "possible bias is accounted for" or because of the definition of "rigorous." However, only one respondent provided a definition for rigorous: "the study is designed to eliminate the influence of external factors." These comments suggest that numerous respondents may not adequately appreciate the many sources of uncertainty (bias, confounding, exposure assessment) which confidence intervals cannot encompass and which statistical analysis cannot account for.

The high percentage of "don't know" responses from the environmental engineering professors for this and the last statement suggests that many of these respondents may be unclear when interpreting statistical presentation of health risk findings. Several of the qualitative responses for those in the "don't know" category

explicitly state that they have "little experience with this" and that they "don't understand well enough." These comments, as well as many from those in agreement, reflect a potential need and an opportunity to improve the understanding by these groups, particularly the environmental engineering professors, on this issue.

The greatest difficulty this statement posed was likely due to the use of "rigorous." Many believed this statement to be true providing the study was truly rigorous and if all biases are accounted for. However, although a "rigorous" study will attempt to minimize measurement error and to account for as much bias and confounding as possible, identifying all the possible sources or even evaluating any identified ones is difficult if not impossible.

Uncertainty and Confidence in Health Risk Analyses

Uncertainty in Health Risk Analyses

Although risk assessment is a useful tool and does provide essential guidance for risk decision making, it has many limitations. Because it is inherently predictive in nature (i.e. it attempts to estimate risks before adverse outcomes arise), risk assessment inevitably involves substantial uncertainty. There are two types of uncertainties involved in risk assessment: uncertainty due to variability (type A uncertainty) and uncertainty due to lack of knowledge (type B uncertainty). Type A uncertainty represents true variability or heterogeneity in values for a variable, i.e. the actual distribution of values in time, space or among individuals. This type of uncertainty cannot be reduced or eliminated in a quantitative risk assessment; it can only be characterized or understood. Type B uncertainty, "true uncertainty," represents the lack of knowledge about what the true value is for some parameter. This uncertainty can be reduced by gaining knowledge to improve how much you know about the value.

Variability and uncertainty surround all aspects of quantitative risk assessment. Major sources of variability (type A uncertainty) include exposure variability and interindividual variability in susceptibility (Finkel 1990). Exposure variability results from many factors in various stages of the exposure process including emission variability, transport and transformational variability, micro-environmental and personal-activity variability (NRC 1994). Human interindividual variability includes differences in genetic predisposition, biological function and behavior.

Sources of true uncertainty include parameter uncertainty, modeling uncertainty and decision-rule uncertainty (Finkel 1990). True uncertainty is inherent in all stages of risk assessment such as hazard identification, exposure assessment, dose-response assessment and risk characterization. The largest quantitative sources of uncertainty

likely occur in exposure assessment and dose-response assessment. Uncertainty in exposure assessment is dependent on the quality of the sources of the information on exposure parameters including both variability and knowledge uncertainty. Sources of variability and uncertainty in exposure assessment include the intensity, duration and frequency of exposures, exposure route, bioavailability, exposure source characteristics, the nature, size and make-up of the exposed population, and cumulative multiple exposures to various other chemicals.

Although the exposure assessment stage of quantitative risk assessment contains large elements of uncertainty, there is substantially more uncertainty associated with dose-response assessment because of the numerous assumptions and inferences which must be made regarding extrapolation of tested doses to estimated human doses, extrapolation between species, and the approaches and model selections for these. However, in the few cases where epidemiology can contribute dose-response information for quantitative risk assessment, exposure assessment may be a greater source of uncertainty.

Confidence in Health Risk Analyses

When dealing with scientific methods and quantitative analyses there is often a tendency for overconfidence to develop in the accuracy of predictions (NRC 1989a; Otway 1992; Freudenburg 1988; Pidgeon et al. 1992). The same is true for the estimations made in risk assessment. Although the many limitations and uncertainties underlying risk assessment methods are readily acknowledged, many scientists, including those directly involved with risk assessment, underestimate the weaknesses and may express excessive confidence in the risk estimates that are generated through risk assessment. To develop a realistic perspective on the estimates generated by risk

assessment, it is necessary to understand the numerous limitations of risk assessment methods.

To gain a better understanding of the risk estimates generated through risk assessment it is first necessary to understand what is meant by risk in the context of environmental health risk assessment. Although many definitions of risk have been put forth, one developed from the quantitative definition by Kaplan and Garrick (1981) defines risk as a prediction of the future likelihood (probability) of an event or set of circumstances (hazard) leading to adverse consequences all with reference to a specified time period (Hrudey and Chen 1996). Because risks, by definition, are always a prediction or a forecast of what might happen, and predictions are influenced by the beliefs of an individual, risk is fundamentally a statement of belief about what will happen. As such it cannot be measured when the prediction is made. Subsequent observations of events rarely satisfy the assumptions of the prediction so that verification of a risk prediction is usually not possible.

Aside from being inherently predictive in nature, the final estimates of risk are unreliable and inaccurate for many reasons. Firstly, because most of the qualitative and quantitative factors underlying a risk assessment are uncertain (Jardine and Hrudey 1997; Otway 1992), scientists must make assumptions and judgments at every stage in an analysis from initially identifying a hazardous substance to deciding which endpoints or consequences to include and which methods to use, to identifying and estimating exposures, choosing appropriate species and dose-response relationships, selecting which models to use and their input parameters, to extrapolating between dose levels and between species, as well as a multitude of many other judgments (Slovic 1997). An important result of this is that analysts often provide widely differing, yet equally justified, risk estimates. In fact, the implications of different sets of assumptions can be very significant. For example, the sole choice of a dose-response extrapolation model can have an enormous effect on the risk estimates produced: two scientifically

plausible dose-response extrapolation models for the risk associated with aflatoxin in peanuts or grain showed risk levels differing by a factor of 40,000 (Freedman and Zeisel 1988). Similarly, comparison studies of health risk to people living near liquified natural gas terminals were found to vary over a factor of 100 million, even though the technical facilities were similar (Mandl and Lathrop 1983). These differences in risk estimates are mostly due to the assumptions made by analysts and the methods and models they chose to use in making their quantitative assessments. Therefore, because there are many choices and approaches available for generating quantitative risk estimates, there is no unique value which can be deemed reliable and accurate.

Secondly, because of the high degree of uncertainty, it has become common practice in risk assessment procedures to use cautious or conservative assumptions, models and inputs to minimize the likelihood that the resulting risks are understated. The rationale for the use of the "conservative assumption" approach is best summarized by the premise "better safe than sorry" (Jardine and Hrudey 1997).

Examples of some common conservative assumptions often relied on in environmental health risk assessment include using data/evidence from the most sensitive animal species and sex tested, assuming no-thresholds and linear low dose relationships for carcinogenic substances, using the extrapolation model that produces the highest risk estimate, reporting statistical upper confidence limits rather than most likely estimates, and making cautious assumptions (often worst case scenarios) about exposure (Harrison and Hoberg 1994).

The major concern associated with the principle of erring on the side of safety is that each individual conservative assumption is compounded in the final risk estimate. Often when the most conservative alternative is selected at each inference juncture, these overprotective assumptions compound to give an impossible scenario as the

outcome. In fact, the cumulative effect of the conservative assumptions may be very large.

Thirdly, the manner in which risk estimates are represented may also contribute to overconfidence and a failure to recognize the weaknesses in risk estimates. Although a range of risk estimates are usually calculated, risk estimates are often presented as one single numerical estimate of risk representing a plausible upper limit value (U.S. EPA 1986). This practice is misleading because it gives the impression of more scientific certainty than what truly exists (NRC 1996). As stated in the U.S. EPA Carcinogen Assessment Guidelines (1986): "Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero. The range of risks, defined by the upper limit given by the chosen model and the lower limit which may be as low as zero, should be explicitly stated."

There are also many examples of risk estimates expressed to three or more significant figures. Stating risk estimates in such a precise fashion can clearly give an impression of certainty and thus misrepresent the confidence they warrant. The degree of precision and accuracy in the quantitative cancer risk estimates do not warrant more than one significant figure being presented (U.S. EPA 1986).

Furthermore, since risk estimates are derived from analysis assuming population responses without accounting for much for interindividual variability, the meaning of a single value of risk for any specified individual is open to a very wide interpretation and should never be taken as an accurate estimate of the risk to any specific individual. The full range of risk estimates, conveying both the most probable estimate and richer information on the extent of uncertainty, should be reported (Finkel 1989).

Analysis of Statements

Table 15 presents the results for Statements 10, 13, 15 and 16 for the environmental epidemiologists, environmental engineering professors, toxicologists and the public (Canadian sample) as well as the students in public health science. These statements were included in the questionnaire to determine:

- the respondents' confidence and understanding of the predictive methods of risk assessment,
- what respondents recognize as the major source of uncertainty associated with environmental health risk assessment, and
- the respondents' attitudes towards regulation of chemical hazards and how they resolve uncertainty.

These statements were not included in the surveys for the American sample of toxicologists and public nor for the Danish medical and risk assessment students. Therefore, no comparisons can be made with these groups.

Table 15 - Responses to Statements 10, 13, 15 and 16

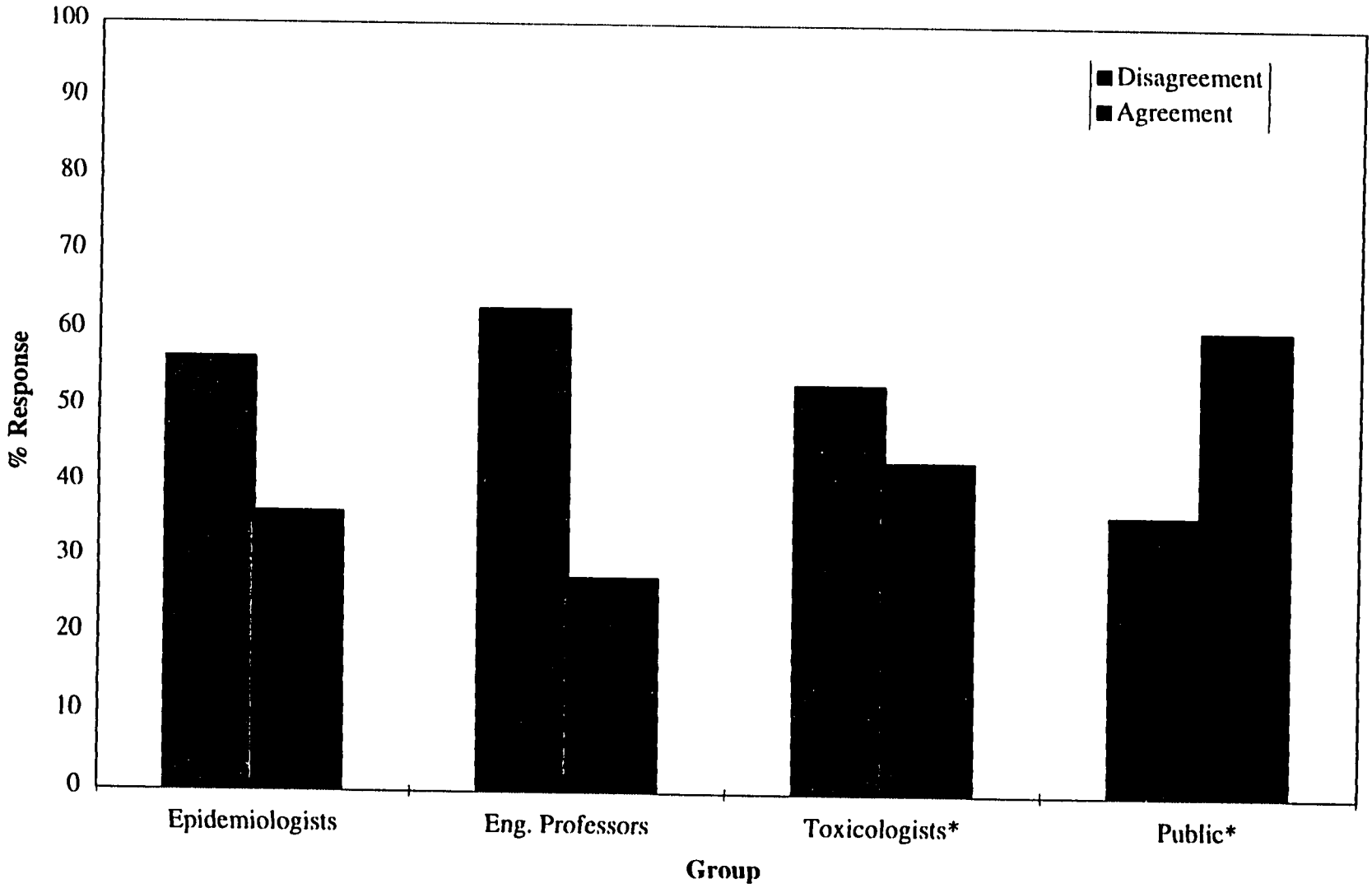
		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	EE[b]	13.4[a]	38.1	22.2	0.5	25.8
	EP[c]	5.4	30.6	20.1	1.0	42.9
	T2[d]	19.3	52.0	18.0	4.7	6.0
	P2[e]	18.7	32.6	31.5	12.5	4.8
	PH[f]	19.4	51.6	12.9	3.2	12.9
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.	EE	11.4	45.1	35.2	1.0	7.3
	EP	13.2	49.8	27.1	0.5	9.5
	T2	7.3	46.0	40.7	2.7	3.3
	P2	11.2	25.0	47.3	13.4	3.0
	PH	9.7	51.6	22.6	3.2	12.9
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.	EE	12.0	36.1	23.6	1.6	26.7
	EP	15.1	40.6	25.0	2.5	16.8
	PH	6.5	25.8	16.1	3.2	48.4
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	EE	5.7	30.4	45.9	7.7	10.3
	EP	7.8	37.2	30.4	5.1	19.5
	PH	6.5	3.2	41.9	16.1	32.3

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] T1 - American Toxicologists (n = 170; Kraus et al. 1992)
[e] T2 - American Public (n = 262; Kraus et al. 1992)
[f] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)

Statement 13, "*Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.*", was included in the questionnaire to evaluate the respondents' confidence and understanding of the substantial uncertainty in risk estimates. Many people have a tendency for overconfidence in the accuracy of risk estimations. However, because of the numerous assumptions and judgments made at every stage in a health risk analysis, the final estimates of risk are unreliable and inaccurate. For example, because of different sets of assumptions, scientists often provide widely differing, yet equally justified, risk estimates. Therefore, because there are many choices and approaches available for generating quantitative risk estimates, there is no unique value which can be deemed accurate and reliable. Furthermore, because the most conservative assumptions, models and inputs have often been used at each stage in a policy-driven risk analysis, the final estimate of risk is vastly overestimated.

The responses to this statement for the environmental epidemiologists and environmental engineering professors show that both groups were divided in their opinions concerning whether or not scientific experts are able to make accurate estimates of health risks from chemicals in the environment (Table 15). This was also consistent with the responses from the survey of the toxicologists (Slovic et al. 1995). Figure 8 presents the responses for this statement for the three disciplinary groups as well as the Canadian public.

Figure 8. Responses to Statement 13:
"Scientific experts are able to make accurate estimates of health risks from chemicals in the environment."



* Canadian survey (Krewski et al. 1995; Slovic et al. 1995)

The majority in all three disciplinary groups disagreed with the statement with the environmental engineering professors disagreeing and strongly disagreeing more often then the other 2 groups (63.0% disagreement overall; 13.2% strong disagreement). Slightly more than half of the toxicologists and environmental epidemiologists disagreed with this statement (53.3% (Slovic et al. 1995) and 56.5% disagreement respectively). The Canadian public were also somewhat divided in their opinions; however, they showed an opposite pattern to the 3 disciplinary groups in that the majority were in agreement with the statement (60.7% agreement overall; 13.4% strong agreement versus 36.2% disagreement) (Slovic et al. 1995).

Although disagreement was the view held by the majority in all three disciplinary groups, there was still a relatively large percentage of respondents who agreed with the accuracy of expert risk estimates. The toxicologists were the most likely to agree (43.4% agreement overall) (Slovic et al. 1995). The environmental epidemiologists agreed 36.2% and the environmental engineering professors agreed 27.6%.

The most common qualitative response for the environmental epidemiologists and the environmental engineering professors who disagreed with the statement was that while experts can make estimations, the estimates are often not accurate. These comments along with others such as "(there is) too much estimation involved", "assumptions and limitations introduce error", "too much uncertainty remains", suggest that many respondents appreciate the enormous uncertainty in risk estimates. Other respondents disagreed because "(there is) no proof of accuracy" or "can't measure the true value." Other respondents stated that the accuracy of estimates depend on the evidence and data available. Only a few respondents indicated that health risk estimates can vary by orders of magnitude and that health risks may be vastly different for different people.

Numerous respondents who agreed that scientific experts are able to accurately estimate health risks from chemicals in the environment stated that it depends on one's interpretation of "accurate"; several of these respondents felt that "accurate" was too strong a word but that experts are "able to make useful estimates." Some in this category believed this statement to be true only under certain circumstances and for specific chemicals. A few respondents stated that risk estimates are usually accurate within an order of magnitude. Some respondents indicated that generally this is true and that risk estimates are accurate "sometimes," "more often than not," and that scientists can "accurately rank relative risks, but not make accurate absolute estimates." Another comment indicated the statement to be true at the population level but not at the individual level.

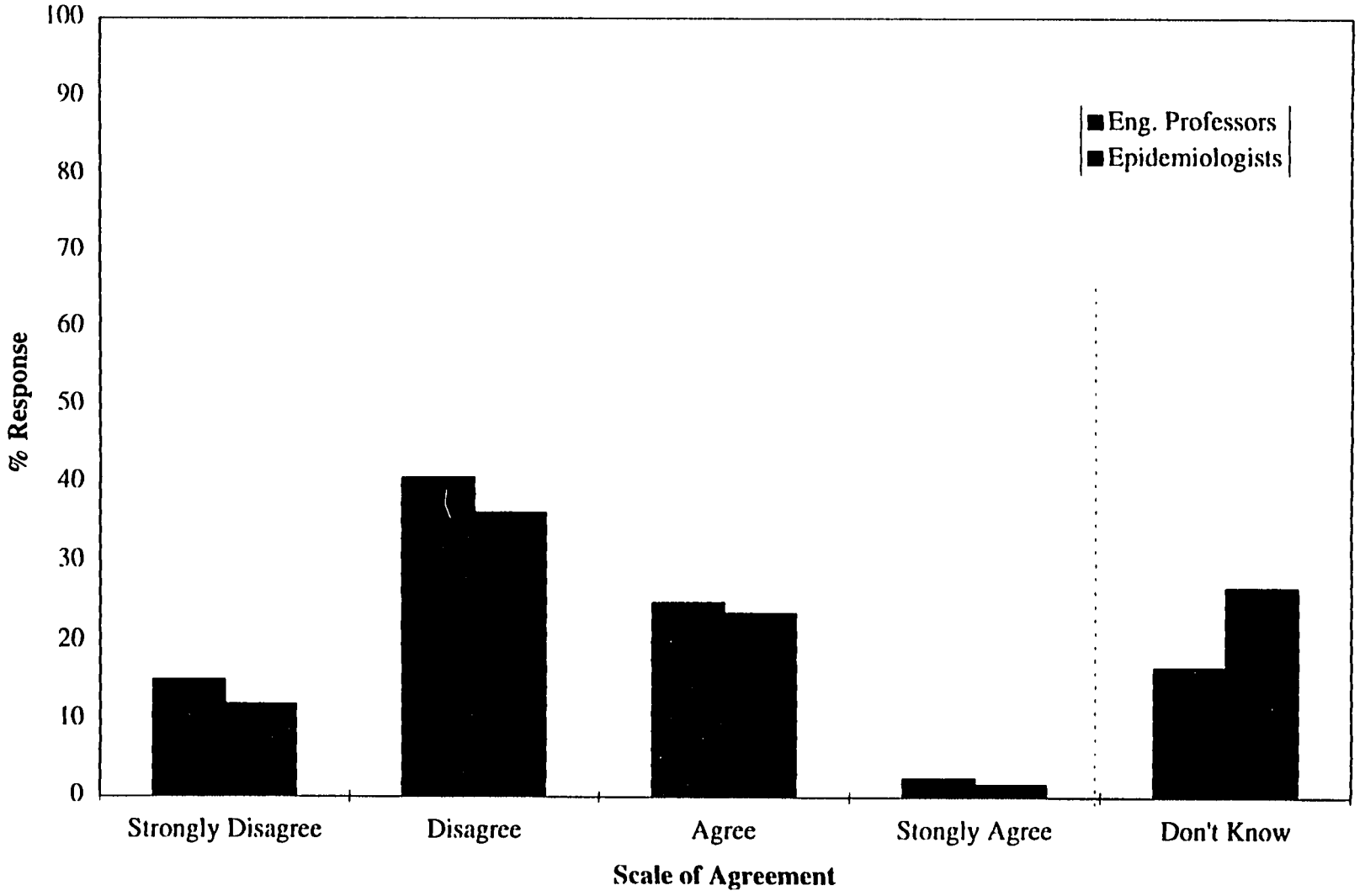
Many of the respondents in the "don't know" category for this statement indicated "sometimes" this statement is true or indicated difficulty in interpreting the word "accurate." Although the majority of respondents in the disciplinary groups may appreciate the substantial uncertainties in health risk estimates, the high percent agreement suggests that some may not be fully aware of the limitations in health risk analysis. Furthermore, the difference between the scientific disciplines and the public on this issue is interesting. Apparently the public has more faith in the ability of the scientists than do the scientists. There may be some potential for scientists to foster greater understanding with the public if they are able to admit to the public that the tools of science are very limited in this field.

Statement 15, "*A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.*", was included in the survey to determine whether respondents recognize the substantial uncertainty associated with

quantitative cancer risk assessment and that risks, by definition, are predictions of what might happen and thus cannot be measured or known when the prediction is made.

Similar to Statement 13, the environmental epidemiologists and the engineering professors each had divided opinions to Statement 15 (Figure 9). Both groups showed moderate disagreement to this statement (48.1% of environmental epidemiologists and 55.7% of environmental engineering professors). Over 25% of the respondents from each group agreed that a "one chance in a million lifetime cancer risk can be known for a given level of exposure to a carcinogen." A relatively large percentage of respondents indicated "don't know" to this statement (26.7% of environmental epidemiologists and 16.8% of environmental engineering professors. Additionally, almost half (48.4%) of the students in public health science responded "don't know" to this statement.

Figure 9. Responses to Statement 15:
"A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen."



The most common theme for the qualitative responses for both groups was that a lifetime cancer risk as low as one chance in a million "can be estimated but not known." Some of those in disagreement with this statement recognized the numerous uncertainties in cancer risk estimation, particularly with interspecies and low dose extrapolation methods. Others stated that one chance in a million is too low a number to achieve high confidence and that the enormous individual variation makes these values unreliable. A few more respondents indicated that risk estimates are based on numerous assumptions and that different sets of assumptions affect the final risk estimates.

Many of the respondents in agreement with this statement who provided qualitative responses indicated that they interpreted "can be known" as "can be calculated." Others stated that a lifetime cancer risk as low as one chance in a million can be known for a given level of exposure "in some cases," "roughly," "for some classes of chemicals/carcinogens," or "if the potency slope is accurate."

For those who responded "don't know", many indicated difficulty in interpreting that statement and what was meant by the word "known." Others indicated that they doubt estimates could be so precise, and that the risk can be computed but the "true risk can never be known."

As noted in the comments, the phrase "can be known" created numerous difficulties in interpretation and response to this statement. One possible modification of this phrase is "can be measured." This statement reworded in this manner likely would not have posed as many problems for respondents when interpreting the statement.

Overall, the quantitative responses to Statement 13 and 15 suggest that some of the respondents from both groups, although not the majority, may be overconfident in the risk estimates produced through risk assessment and don't fully appreciate that risk assessment deals with substantial uncertainty and is an inherently subjective and

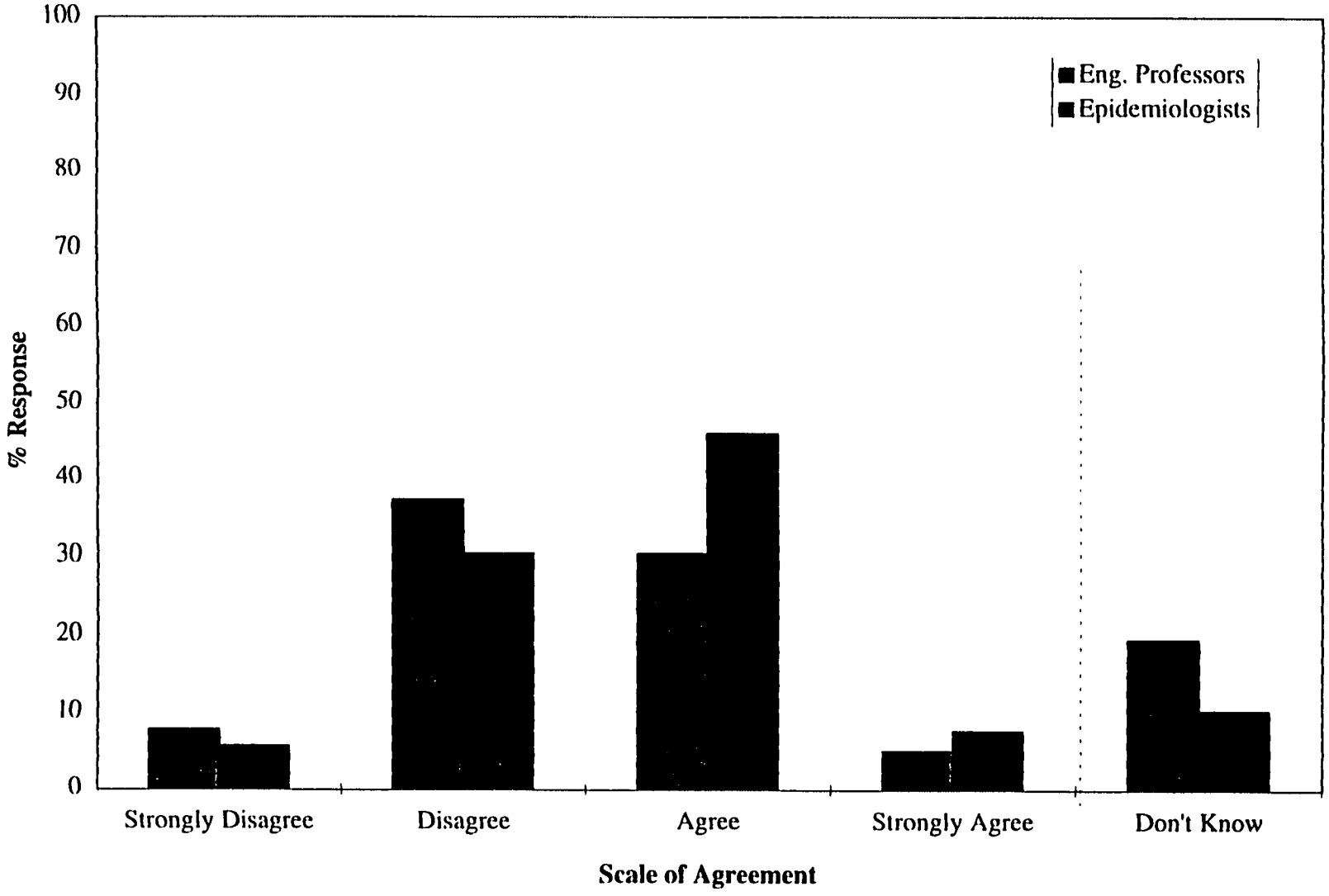
assumption-laden process. However, the qualitative responses (particularly for Statement 15) are helpful in interpreting some of these quantitative responses. For example, if the respondents in agreement with Statement 15 had not explained their interpretation of "known", the substantial number who agreed would indicate a large number of respondents were overconfident in the ability of quantitative risk assessment for predicting cancer risk. Even so, these results suggest an opportunity for developing better understanding among these disciplines about the limitation to quantitative cancer risk assessment.

Statement 16, "*The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.*", was included to determine what respondents recognize as the major sources of uncertainty associated with environmental health risk assessment. A health risk assessment includes all evidence from both epidemiological and toxicological studies and although the degree of exposure is usually the largest element of uncertainty in an epidemiological study, in a health risk assessment there is substantially more uncertainty in dose-response assessment because of the numerous additional assumptions and inferences regarding extrapolation of doses, extrapolation between species, and the approaches and models used for these which can vary considerably for the same substance depending on the model used. Therefore, unless dose-response information can be provided from epidemiology, the degree of exposure is not likely to be the largest element of uncertainty in a health risk assessment.

Responses to Statement 16 showed both the environmental epidemiologists and the environmental engineering professors divided in their opinions (Table 15). Figure 10 indicates that over half (53.6%) of the respondents in the environmental epidemiologist group agreed with this assertion, 36.1% disagreed and 10.3% responded "don't know". The environmental engineering professors had an opposite

pattern of response with the majority of the group disagreeing with the statement rather than agreeing (45.0% disagreement versus 35.5% agreement). Almost 1 in 5 (19.5%) of the respondents in this group answered "don't know" to this statement (Figure 10). The students in public health science also had a high percentage of "don't know" responses (32.3%); the majority, however, were in agreement with the statement (58.0%).

Figure 10. Responses to Statement 16:
"The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment."



Qualitative responses for the environmental epidemiologists who disagreed with the statement included a variety of comments such as: "(exposure) is just one of the many uncertainties," "response to exposure is the greatest uncertainty," "potency estimation is the greatest uncertainty," "most of the elements of uncertainty are the confounding factors," and "interindividual variability creates bigger uncertainties." One respondent indicated that the degree of exposure is the largest uncertainty in an epidemiology study but not in a health risk assessment. This last explanation likely explains why the majority of epidemiologists agreed with the statement. Epidemiologists are more likely to be aware of the limitations of exposure assessment to epidemiological studies. However, they may be unaware how few health risk assessments are based on epidemiological evidence.

The numerous comments from the environmental engineering professors who disagreed with this statement suggested that this group may be more sensitive to the substantial uncertainty associated with the dose-response relationship. Most of the respondents stated that the dose-response relationship and the degree of human response to exposure is much more uncertain than the degree of exposure. Other comments indicated large uncertainties in interspecies extrapolation.

Respondents in agreement that the degree of exposure is the largest uncertainty in any health risk assessment (particularly the environmental epidemiologists) believed this statement to be true "often" and "for most studies." Some indicated that the "actual dose was more uncertain." Although some of these respondents may have been considering only epidemiological studies when responding, the high percent agreement, particularly for the environmental epidemiologists, suggests that improved understanding of what a health risk assessment entails and its limitations may be needed.

Comments from those in the "don't know" category included "there are many sources of uncertainties," "often exposure is (the largest uncertainty)," there is also

huge uncertainty in dose-response," and "also much uncertainty about past and other exposures." These comments suggest that while some respondents are familiar with sources of uncertainty in environmental health risk assessment, they may be unsure of which is greatest.

There appeared to be no difficulty with this statement other than the possibility that some epidemiologists interpreted it considering only epidemiological studies rather than a health risk assessment which includes both epidemiological and toxicological studies. Modifying the statement to, "*In any health risk assessment, the degree of exposure to an environmental contaminant is usually the largest element of uncertainty.*" may have made a difference in responses.

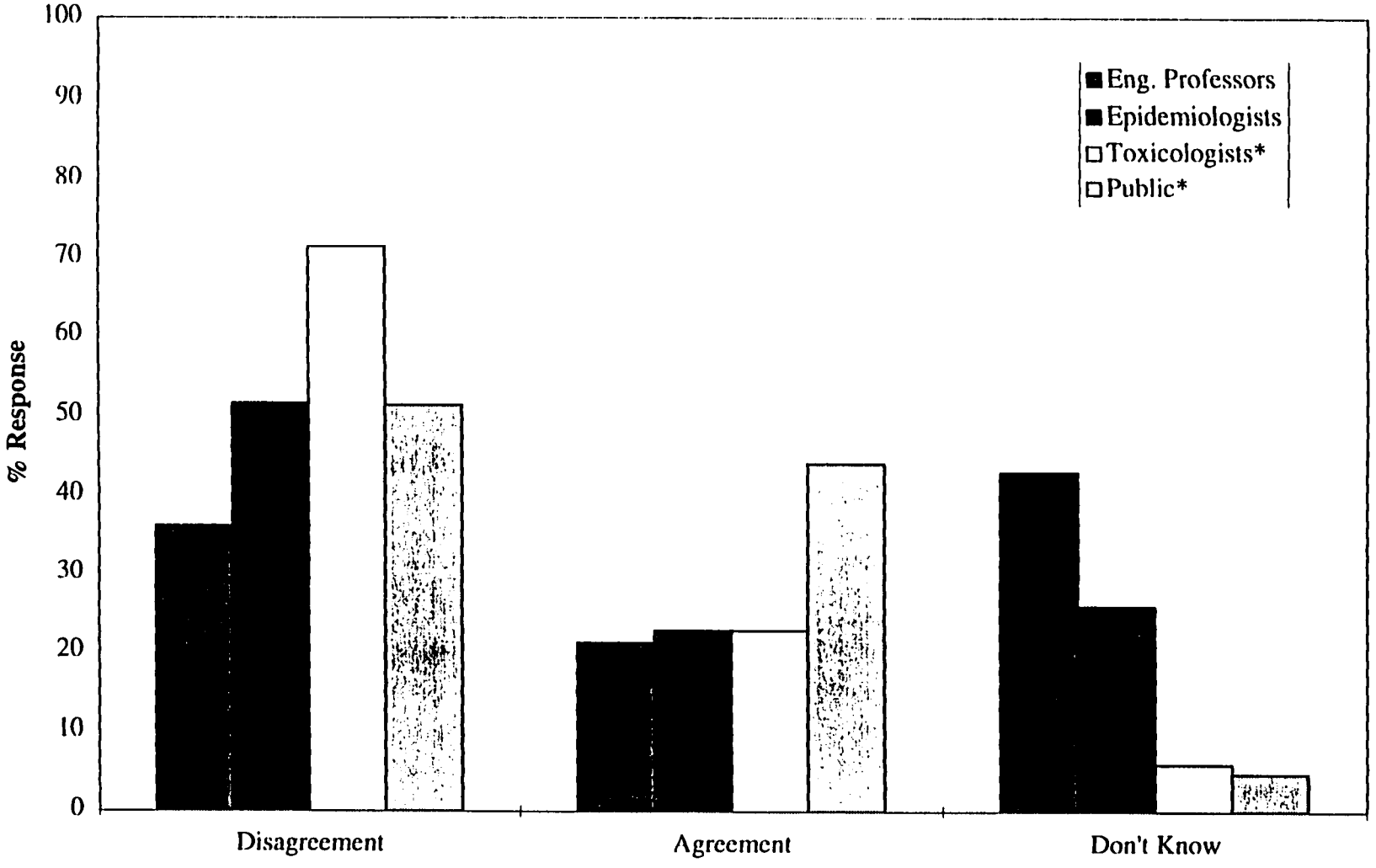
Statement 10, "*A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.*", was included in the questionnaire to examine the respondents' attitudes toward regulation of chemicals and their confidence in animal testing protocols versus human experience. Considering the many limitations of animal testing protocols used for current regulatory guidelines, I would be more inclined to agree with this statement and accept that the experience of wide use of a prescription drug over 20 years in a human population would be more relevant than the bioassays which use a limited number of animals.

Responses for the environmental epidemiologists, environmental engineering professors, Canadian toxicologists and public as well as the public health students are listed in Table 15. All groups surveyed tended more to disagree with the statement rather than agree (Figure 11). The group of toxicologists surveyed was the most likely group to disagree (71.3% disagreement overall; 19.3% strong disagreement) (Slovic et al. 1995). The environmental epidemiologists disagreed 51.5% and the environmental engineering professors disagreed 36.0%. About 1 in every 5 respondents from each

disciplinary group agreed with this statement (Figure 11). It is interesting that so few respondents (approximately 20%) from both the environmental epidemiologists and engineering professors agreed that the older drug was safer than the new "tested" drug given that the majority of respondents (over 50%) did not trust the validity of animal studies for predicting human health effects from chemicals (Statement 3). However, there was a substantial portion of "don't know" responses from these groups: 42.9% of the environmental engineering professors and 25.8% of the environmental epidemiologists were included in this category (Figure 11).

As can be seen from Figure 11, the Canadian public were also divided in their opinions to this statement: there was 44.0% agreement and 51.3% disagreement (Krewski et al. 1995). The group of public health science students had similar results to the toxicologists with 71.0% disagreement (Table 15).

Figure 11. Responses to Statement 10:
"A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines."



* Canadian survey (Krewski et al. 1995; Slovic et al. 1995)

The comments provided for this statement indicated that there was not much difference in perspectives between the environmental epidemiologists and environmental engineering professors. Of those who disagreed that a prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines, the most repeated comment was that some serious long term effects may take longer than 20 years to be seen and that epidemiology studies related to use are needed to link effects to the chemical in question. Others indicated that they have more confidence in current testing protocols because "current guidelines are fairly strict" whereas "prior testing rules were much more lax." Another repeated comment was that response is "situation specific" and depends on the drug in question and on the results (both anecdotal and experimental) obtained.

Of the respondents inclined to accept years of human experience over animal testing protocols, many believed this statement to be true provided that post marketing surveillance was conducted and no adverse effects were observed in the 20 years of use. Others stated that they have "more faith in human epidemiology than animal studies" and that there is "no substitute for extensive human experience." Another common theme among qualitative responses was that current testing protocols do not test drugs in large numbers of subjects prior to use and that the knowledge gained from experience with the drug in a large population (used widely for 20 years) is much greater than tests done on a new drug. Other respondents indicated that any adverse effects over 20 years from a widely used drug would have become apparent and reportable.

Overall these results indicate that the majority of respondents have more faith in the current testing protocols for chemicals than accepting the knowledge gained through years of human experience. The large proportion of respondents who were in disagreement with the statement or responded "don't know" suggests that most are not

comfortable with accepting the safety of prescription drugs/chemicals solely on the basis that they are familiar and appear to be safe. Many stated concern that adverse effects may have escaped unnoticed or unmeasured.

Of those who provided comments for "don't know" responses, several stated they were "not familiar with the current guidelines." Numerous respondents also stated they needed more information and that it depended on which drugs and if adverse effects of the older drug were reported and/or studied. Some respondents felt that it was "impossible to know" which one is safer. Despite the difficulty this question poses, the very large percentage of "don't know" responses from these two groups suggests there is scope for developing greater understanding of the limitations of any drug testing protocol. While neither alternative offers assurances of complete safety, that the number of humans exposed over 20 years would greatly exceed the number of humans exposed in clinical trials and the few rodents tested suggests that the long term human experience is likely to be more relevant.

Other than the absence of specifics such as which drugs and if post-surveillance was conducted, this statement did not appear to pose any interpretation problems for respondents. An additional statement that may have been useful in providing information on the attitudes toward regulation of chemicals may have been: "*I believe current regulatory guidelines are adequate in determining risks from prescription drugs and other chemicals.*"

Objectivity and Values in Scientific Analyses

Objectivity in Scientific Analyses

Scientific analysis is often presumed to be a neutral and objective search for truth. While good scientific analysis can be objective in the sense that any scientist who knows the rules of observation of the particular field of study can, in principle, obtain the same results i.e. reproducibility (NRC 1996), there is a strong subjective element in the practice of science which may not be recognized by many scientists.

Firstly, science is neither neutral nor objective in its ways of framing problems. Choosing what to investigate and which outcomes to observe ultimately depend on one's beliefs and value judgments of what is deemed worthy or important to study. For example, analyses of the cancer risks of industrial synthetic chemicals divert attention from the possibly comparable or greater risks from naturally occurring chemicals in foods, or analyses of the risks of drunk driving that highlights drivers' behaviors as a cause of traffic fatalities draw attention away from equally significant factors of automobile and highway design (NRC 1996).

Secondly, judgment is a defining characteristic of all interpretations of scientific knowledge. As the role of judgment increases in science, results become increasingly subjective (NRC 1989a). Because most science involves uncertainty and incomplete information, scientists must often make compensating or simplifying assumptions. These assumptions are inevitably influenced by the objectives, values and biases held by the scientist. Thus, in cases where results are derived from numerous assumptions, results are better regarded as educated estimates involving subjective influence, rather than "objective" truth.

Values in Scientific Analyses

Although there is often an attempt to separate facts from values, a complete separation is not possible (NRC 1989a). Facts do not exist independently of people; the articulation of facts requires a human element (Otway 1992). In essence, our values determine what facts we produce and use. Knowledge is usually created only if someone believes that it is worth knowing (NRC 1989a). Furthermore, the facts we produce from science shape our values. Because values are created and influenced through education, experience and our perspective of the world, so long as the world is revealed to us through science, the facts it reveals will ultimately shape our values.

Analysis of Statements

Table 16 presents the responses from the environmental epidemiologists, environmental engineering professors and public health students to the Statements 11 and 12. These statements were included in the questionnaire to determine the degree to which scientists recognize the subjective elements which enter into their work. These statements were not included in the surveys of toxicologists and public nor the surveys of the Danish students. Therefore, no comparisons can be made with these groups.

Table 16 - Responses to Statements 11 and 12

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
11. Environmental epidemiology/science is an applied science (i.e., not a basic science).	EE[b]	0.5[a]	10.5	64.4	19.4	5.2
	EP[c]	5.2	19.5	58.6	12.1	4.7
	PH[d]	3.2	25.8	32.3	9.8	29.0
12. Applied sciences are rarely value-free or value-neutral.	EE	6.3	15.7	42.9	20.9	14.1
	EP	7.1	22.8	43.8	5.6	20.8
	PH	6.7	16.7	23.3	26.7	26.7

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)

Most environmental epidemiologists agreed with Statement 11 that "*Environmental epidemiology is an applied science.*" (83.8% agreement versus 11.0% disagreement). About 5% responded "don't know." There was not as much agreement with the environmental engineering professors that "*Environmental science is an applied science (i.e. not a basic science).*" 70.7% of environmental engineering professors were in agreement (12.1% strong agreement) with this assertion. 4.7% responded "don't know." A large proportion of public health science students also responded "don't know" to this statement (26.7%). (Table 16).

The most common response (from those in all response categories - agree, disagree, don't know) was that environmental epidemiology/environmental science has elements of "both" applied sciences and basic sciences. For those in agreement, some respondents indicated "both applied and basic science" because applied sciences build on basic sciences and encompass many diverse disciplines. For the disagreement category, qualitative responses indicating "both applied and basic science" suggest that

these respondents disagreed because they interpreted the statement as one or the other, i.e., either an applied science or a basic science but not both. This was likely due to the wording of the question with some respondents interpreting it as applied sciences being distinctly separate from the basic sciences. Others who were in disagreement, particularly the environmental engineering professors, indicated that "some aspects are applied and some are not." Most respondents who selected "don't know" indicated "could be both." Other respondents comments indicated confusion with the definitions of "applied" and "basic."

This statement created many difficulties in interpretation for the respondents. The phrase in parenthesis, "i.e. not a basic science" was intended to help clarify what was meant by an "applied science." However, as was mentioned, it apparently created more confusion for the respondents and many may have interpreted the statement as stating "environmental science/environmental epidemiology is an applied science but not a basic science" and thus disagreed. Therefore, these respondents may not have been disagreeing that environmental epidemiology/environmental science is an applied science but rather, that it is not a basic science.

From the qualitative feedback to this statement, the most effective modification to this statement would likely be to have not included the phrase in parenthesis. Interpreting the statement may have been more uniform had the statement read, "*Environmental epidemiology/environmental science is an applied science.*"

Statement 12, "*Applied sciences are rarely value-free or value-neutral.*," specifically assessed whether respondents recognize that there is a strong subjective element in all science. Because most science involves uncertainty, the assumptions often made by scientists are inevitably influenced by the objectives, values and biases held by the scientist.

The responses to this statement indicate that both the environmental epidemiologists and environmental engineering professors had fairly similar distribution (Table 16). There was moderate agreement from the group of environmental epidemiologists. This group had 63.8% agreement and of these, 20.9% were in strong agreement. Over 1 in 5 (22.0%) environmental epidemiologists surveyed disagreed with the statement that applied sciences can be value-free. Also, a relatively large portion responded "don't know" (14.1%). The environmental engineering professors were more divided in their opinions (49.4% agreement versus 29.9% disagreement) regarding whether applied sciences are value-free or value-neutral. There were much fewer "strongly agree" responses in this group than the environmental epidemiologists (5.6% versus 20.9%) and there was 9% more disagreement. Furthermore, 20.8% environmental engineering professors responded "don't know." The same pattern of response was also found for the students in public health science (Table 16).

The qualitative responses indicate that both groups had similar perspectives for this statement. Numerous comments from both groups show that most respondents in agreement with the statement recognize that all sciences have some degree of subjectivity and that no sciences are completely free of value judgments.

There were not many qualitative responses for those in disagreement that applied sciences are rarely value-free. However, of the ones provided, comments such as, "many aspects of applied sciences are value-free", "depends on the investigator and method used", applied sciences "can be value-neutral" and "if it is a true science then values should not affect it", suggest that some of these scientists may not recognize the subjective element of science. A few of the environmental engineering professors who disagreed that applied sciences are rarely value-free distinguished policy from science and qualified "policy decisions are never value-neutral" and "science and policy are not the same." The distinction between science and policy is important because it indicates

that while these respondents understand the role of values in policy decisions, developing better understanding of the roles of values in scientific analyses may be important.

The comments indicate that there was great difficulty in interpreting this statement. The high percentage of "don't know" responses can be attributed to the problems respondents had in understanding the statement. A substantial number of comments indicated that they did not understand the statement or that it was unclear. Some respondents specifically did not know what was meant by "value-free" and "value-neutral." Furthermore, a few respondents who disagreed that applied sciences are rarely value-free or value-neutral provided comments such as "this is true of all science", "no science is absolutely value-free", "all sciences have elements of values", etc. These comments suggest that these respondents may have misread the statement and meant to agree rather than disagree. An improved statement which may have avoided these difficulties is, "*Applied sciences are objective and value-free.*"

An additional statement which may have provided useful information on whether these respondents recognize the many policy driven, and thus value driven, elements in risk assessment could have asked, "*Risk assessment is largely objective and value-free.*" This statement may have also helped in assessing the respondents' confidence in risk assessment and whether they recognize the subjectivity and numerous judgments that are made in a risk analysis.

Perceptions of Risk

Natural and Synthetic Chemicals

The premise that synthetic chemicals are bad compared to natural ones is commonly expressed in society. Fears of man-made chemicals may be attributed to events involving accidental releases of industrial chemicals in the environment, in addition to widespread, and sometimes sensational, media coverage. Many environmentalists, as well as authors such as Rachel Carson, have also been a major influence behind the view that natural chemicals are not as harmful as synthetic chemicals (Carson 1962; Epstein 1979). The focus of attention in environmental health risk assessment has been directed to substances which are strictly or primarily man-made, including a wide range of synthetic organic chemicals (halogenated solvents and pesticides), and reaction by-products (polychlorinated dioxins). While this may be justified on the premise that exposures to man-made chemicals are preventable, such pragmatic justification does not imply that man-made chemicals are inherently more dangerous.

The chemical dioxin (2,3,7,8-tetrachlorodibenzo-p-dioxin), a by-product of various industrial processes, is highly toxic and is generally characterized as man-made. Because dioxins are produced by combustion processes, some production of dioxin may be considered natural (i.e. forest fires). However, most evidence suggests that the majority of dioxin present in the environment has arisen from man-made sources (Brzuzy and Hites 1996). No human deaths, even at high exposures, have been explicitly documented but dioxin is commonly accepted as a human carcinogen at high doses. Dioxin causes deaths in some species of animals at extremely small doses (0.0006 mg/kg body weight in female guinea pigs) (Rodricks 1992). Because of this, dioxin has developed a reputation as being the most toxic substance known to

humanity. However, in terms of acute toxicity, many natural toxins are substantially more acutely toxic and lethal than dioxin. For example, botulinum toxin produced from the common soil bacterium, *Clostridium botulinum*, is regarded among toxicologists as the most acutely toxic of all substances. Although the bacterium produces its deadly toxins only under certain conditions, this "supertoxic" substance can be lethal at a single dose in the range of 0.00001 mg/kg body weight (Rodricks 1992). Botulinum toxins target the nervous system and can progress to paralysis of the muscles controlling breathing causing death due to respiratory failure (Rodricks 1992). Many human deaths have been explicitly attributed to botulinum toxin poisoning.

Some other supertoxic chemicals in nature and their sources include tetrodotoxin from pufferfish, the poisons in the tissues of certain species of animals such as saxitoxin (found in certain shellfish), and the venoms of poisonous snakes such as the cobra (Rodricks 1992). These toxins have also caused human deaths.

Another natural source of some unusually toxic chemicals is plants. Although the specific chemicals involved are in many cases not known, the plant kingdom produces many life threatening poisons. For example, poisons from common house plants such as philodendrons, and amatoxins, the toxins associated with some mushrooms, when ingested can be life threatening. Even small intakes of amatoxins can cause serious liver injury that can ultimately lead to death. Also, solanaceous plants, which include many species of wild and cultivated plants (the latter including potatoes, tomatoes and eggplants) contain certain natural toxicants called solanine alkaloids which can also produce serious adverse effects. However, the levels of these toxicants found in the varieties used for food are usually below the toxic level. (Rodricks 1992)

Unlike the acute toxicity and lethality of poisons in nature, environmental health risk assessment usually examines the more chronic effects due to chronic exposures to typical levels of chemicals found in the environment. The potential environmental

exposures to chemicals is often greatest through the diet. Although there is often much concern with synthetic chemicals in water and residual man-made pesticides in foods, the largest mass fraction of chemicals to which we are exposed is comprised of the natural plants, animals, beverages, and herbs and spices that make up our diet (Ames and Gold 1993).

For instance, a cup of coffee contains hundreds of different organic chemicals which are natural components of the coffee bean that are extracted into water. Likewise, spices and herbs also contain numerous organic compounds. Most of these chemicals impart flavors, aromas and colors (Rodricks 1992).

Fruits and vegetables also contain numerous different natural chemicals with some being quite toxic. For instance, plants (including fruits and vegetables) have biochemical mechanisms that enable them to produce natural pesticides to defend themselves against insects and fungi. Tens of thousands of these natural pesticides have been discovered and every species of plant contains its own sets of different toxins. Ames et al. (1990a) have proposed that about 99.99% of all pesticides in the human diet are natural pesticides from plant foods. According to this perspective, we consume far greater concentrations of natural pesticides than we do synthetic ones. The concentrations of natural pesticides is usually measured in parts per million (ppm) whereas the usual concentrations of synthetic pesticide residues and water pollutants are measured in parts per billion (ppb). Ames and Gold (1993) estimate that an average person eats about 1500 mg/d of natural pesticides, which is 10000 times more than the average daily consumption of synthetic pesticide residues. They also estimated that an average person ingests about 5000 to 10000 different natural pesticides and their breakdown products.

Available evidence shows that many of the chemicals occurring naturally in foods are carcinogenic. These natural carcinogens are found in a variety of sources.

Common are the natural pesticides occurring in plant foods. Some natural pesticides that have been tested and found to be carcinogenic in animals occur naturally at concentrations greater than 10000 ppb in fruits and vegetables such as apples, brussel sprouts, cabbage, carrots, cauliflower, celery, cherries, eggplant, grapes, grapefruit juice, lettuce, mangoes, mushrooms, orange juice, pears, plums and potatoes. Numerous herbs and spices also contain many natural pesticides that have been found to be rodent carcinogens (Ames and Gold 1993).

Besides natural pesticides, foods may contain other types of carcinogens. Cooked food is another major dietary source of substances that cause cancer in rodents. It is estimated that an average person consumes about 2000 mg per day of browned and burned material which contain many substances that are rodent carcinogens and countless other substances that have not yet been tested (Ames and Gold 1993). Roasted coffee contains more than a thousand natural chemicals. Although only 26 of these have been tested, 19 have been found to be carcinogenic in rodents (Ames and Gold 1993). Formaldehyde, also a known animal carcinogen, is found in bread and shrimp (Gots 1993) while broccoli, cauliflower and cabbage all contain the carcinogen indole carbinol. Additionally, strawberries and eggs both contain benzene (Ames and Gold 1993; Gots 1993) and peanuts and peanut butter may contain aflatoxin, a potent carcinogen produced by the mold *Aspergillus flavus* (Rodricks 1992).

Undoubtedly, these naturally occurring chemicals represent only a small fraction of the total chemicals tested. Although the greater part of the chemicals we consume are natural, only few natural chemicals have been tested systematically. Rather, the large majority of chemicals that have been tested for carcinogenicity in animals are synthetic. However, of the few natural chemicals that have been investigated, a large portion has proven capable of producing the same range and forms of toxicity that has been found for synthetic chemicals (Rodricks 1992). Consequently, it seems likely that if natural chemicals were subjected to the same chronic testing now

used for synthetic chemicals, a large fraction would also be found to produce serious forms of toxicity, including cancer (Rodricks 1992).

Therefore, because the vast majority of chemicals we are exposed to are natural components of our diet and many have been found to be carcinogenic, the possible carcinogenic hazards from synthetic chemicals are unlikely to exceed the background of possible carcinogenic hazards of natural chemicals except in specific contamination episodes. However, this is not to say that dietary exposures to natural carcinogens increase human cancer risk or are even harmful. In fact, there is epidemiological evidence that fruits and vegetables are an important component to reduce the risk of cancer (NRC 1989b). Fruits and vegetables contain numerous anticarcinogens such as nutrients, vitamins and other antioxidants that are essential in protecting humans against both synthetic and natural carcinogens (Ames et al. 1995). Also, because humans have protective mechanisms against low doses of toxins, we should not be alarmed by the presence of low doses of synthetic and natural toxins (Ames and Gold 1993).

Despite the large number of naturally occurring chemicals we are exposed to, some believe that because they are part of human evolutionary history, whereas industrial chemicals are not, we have become acclimatized to natural chemicals and have evolved defense mechanism to cope with their toxicity. For example, Rachel Carson observed that although "...natural cancer-causing agents are still a factor in producing malignancy...they are few in number and they belong to that ancient array of forces to which life has become accustomed from the beginning" (Carson 1962 p195). She contrasts these natural chemicals with synthetic chemicals against which "man had no protection" (p196). As explained previously, the argument that natural carcinogens are few in number is not correct. Furthermore, the evolution argument does not explain why natural poisons like botulinum toxin or strychnine are able to kill humans

While humans have evolved general and specific defense mechanisms to protect against some natural toxicity, the toxicology of many natural chemicals is not different from that of synthetic chemicals (Ames et al. 1990b). Thus it is likely that the general and specific defense mechanisms humans have evolved over time to protect against toxicity offer protection against both natural and synthetic chemicals. General defenses include things such as the continuous shedding of the surface layer of our skin, stomach, mouth, intestine, colon and lungs which may be exposed to toxins, the ability of cells to respond to stress by using a wide variety of detoxifying enzymes such as antioxidant enzymes, the ability of cells to repair damaged DNA and numerous other mechanisms (Ames and Gold 1993).

Causes of Cancer

Approximately 1/3 of all North Americans will contract cancer at some time during their life and 1/4 will die of some form of the disease (Hrudey and Krewski 1995). Because of this, determining the causes of cancer has become a common concern. The lack of knowledge and understanding of cancer and its causal mechanisms make it difficult for scientists to determine with certainty the specific causes of cancer in individuals. However, various factors contributing to cancer incidence in large populations have been determined. Differences in the cancer rates of certain types of cancers in different populations for different regions of the world, as well as in the same population at different times, provide some indication of the relative importance of various factors (Rodricks 1992). Toxicological and epidemiological investigations have also provided substantial information on the factors contributing to cancer.

Several reports examining the causes and trends of cancer in the United States have indicated that somewhere between 70% and 90% of human cancers appear to be of "environmental origin" and are potentially avoidable or preventable (Higginson 1968; Davis 1985; WHO 1964; Doll and Peto 1981). At a 1968 International Conference in Israel, Dr. John Higginson stated:

> "While we do not know the etiological factors for many cancers, we are in a position to estimate on theoretical grounds the proportions of all cancers that may be of environmental origin. Calculations would indicate that in the United States approximately 80% of all malignant tumors are likely to be environmentally conditioned and thus theoretically preventable." (Higginson 1968).

Although the words "environmental origin" contributed to a widespread public belief that exposures to man-made chemicals in the environment are a major factor in the causation of cancer, the word "environmental" was not intended to be equated with "chemical or industrial pollution." "Environmental" referred broadly to any factors extrinsic to genetic predisposition (Gots 1993). Examples of "environmental factors" include not only industrial chemicals and pollution, but also factors such as diet, stress, smoking habits, reproductive and sexual behavior, occupation, and natural and man-made radiation (Doll and Peto 1981; Gots 1993; Rodricks 1992).

Although many people still believe that exposures to man-made chemical pollutants in the environment are a major factor in the causation of cancer, epidemiology and toxicology provide no convincing evidence that pollution is a substantial cause of cancer. Rather, estimates suggest that the most important contributors to cancer risk are associated with certain 'lifestyle' factors; that is, with personal choices and not with the environment in general (Davis 1985; Doll and Peto 1981). Lifestyle factors have been viewed to be primarily dietary, reproductive and other habits that are assumed to be largely under the control of individuals. These are

distinguishable from factors that are less directly in the control of individuals such as occupations and consumer products, and those over which individuals have little or no control such as food additives, pesticides and environmental pollutants (Rodricks 1992).

In a study of the causes of cancer in the United States by Sir Richard Doll and Richard Peto (1981), it was estimated that only 2% (within a range from less than 1 to 5%) of human cancer causes arise from all sources of "pollution" (as distinct from tobacco, alcohol, diet, food additives, occupation, industrial products, medicines, and sunlight, UV light, and other radiation). Occupational exposures were estimated to contribute approximately 4% (within a range from 2 to 8%) to the total human cancer deaths in the United States.

The most important findings in this study were the contribution of tobacco and diet to the total human cancer deaths. Approximately 30% (within a range from 25 to 40%) of human cancer deaths are due to habitual cigarette smoking which has been said to chronically inflame the lungs, expose tissues throughout the body to numerous cancer-causing agents and decrease antioxidant defenses (Ames and Gold 1993). Also, another 35% (within a range from 10 to 70%) of cancer deaths in the United States was attributed to various dietary factors; in particular, to consuming too much fat (high fat intake) and too few fruits and vegetables which contain vitamins, nutrients and other antioxidants that are essential in defending against oxidation and DNA damage (Ames et al. 1995).

Alcohol was estimated to contribute 3% (within a range from 2 to 4%) to total human cancer deaths while 7% (within a range from 1 to 13%) was attributable to reproductive and sexual behavior. Other environmental related factors, such as industrial products, food additives, medicines and medical procedures, and sunlight, UV light and other radiation, were said to contribute to the remainder of avoidable human cancer deaths (approximately less that 6%) (Doll and Peto 1981).

In summary, these estimates, as well as others, suggest that directly controllable "lifestyle" factors such as dietary patterns, tobacco and alcohol usage, reproductive and sexual habits, and sunlight exposure account for the majority (approximately 3/4) of human cancer deaths. Furthermore, while high occupational exposures may cause a small percentage of cancers (approximately 4%), environmental pollution appears to be an insignificant risk factor for cancer.

Despite these estimates, there is still much attention and funding given to studying chemical carcinogens in the environment. Critics of Doll and Peto's estimates argue that since cancer can take several decades to develop, the full effect of the massive increase in industrial chemical production, usage and waste disposal that occurred following World War II is not reflected in the cancer rate statistics relied upon by Doll and Peto, which were collected primarily in the 1970s. However, cancer rate statistics for the 1990s do not support an increase in cancer mortality once the effect of lung cancer is accounted for. (National Cancer Institute of Canada 1996).

Perhaps the high level of concern towards the effects of environmental exposures to chemical pollutants is also attributed to the fact that people feel they have little or no personal control over these products and their usage. Lifestyle factors are to greater or lesser degrees within an individual's personal control and are voluntary risks (Rodricks 1992). However, exposure to many industrial chemicals is seen as involuntary and it has been widely recognized that people do not tolerate involuntary risks even if these risks are insignificantly small.

Environmental Contaminants

Although chemical manufacturing had its beginnings in the 19th century, its dramatic expansion after World War II combined with the increasing understanding of

the hazards of chemicals since this time heightened awareness of contamination in the environment. Aside from major toxic incidents involving high level exposure of humans to chemical contaminants, evidence of mortality, reproductive failure and genetic defects in wildlife have raised much concern over low level exposure to toxic contaminants in the environment.

Some of the more serious chemical threats to the environment come from various groups of toxic chemicals such as chlorinated organic compounds, dioxins and furans, heavy metals, and hydrocarbons. Chlorinated organic compounds such as DDT were widely used as pesticides in Canada in the 1950s and 1960s. Because pesticides are usually applied as a fine spray or mist, they readily enter the environment. PCBs, another class of chlorinated organic compounds, were used in a broad range of industrial applications. The most common use of PCBs in Canada was as insulators and coolants in electrical transformers and capacitors. For these applications, PCBs were enclosed and thus entered the environment via leaks and spills or at decommissioning of equipment. Some products which are easily released into the environment and have contained PCBs include paints and pesticides. Evidence of negative effects from high level exposure and low level chronic exposure led to tight restrictions on the use of these chemicals during the 1970s and 1980s. In particular, some chemicals such as DDT and PCBs were banned from use, although PCBs used in sealed equipment applications could continue use until the equipment was decommissioned. (Government of Canada 1991a)

Polychlorinated dioxins and furans are unintended by-products of various industrial technologies. Principal sources in Canada today include chlorophenol wood treatment agents, phenoxy herbicides, effluents from pulp and paper mills using chlorine bleaching processes, tobacco smoke, and older municipal incinerators and other sources of incomplete combustion. Because of evidence of toxicity and carcinogenic effects in animals, polychlorinated dioxins and furans are closely

regulated. Restrictions are being imposed to eliminate emissions from pulp and paper mills using chlorine bleaching by modifying the chlorine bleaching process. Also, changes in production processes have resulted in a decrease in dioxin and furan contaminants released by the production and use of chlorophenols and phenoxy herbicides. (Government of Canada 1991a)

Heavy metals such as lead and mercury are naturally present in the environment but human activities have greatly increased the chance of exposure to harmful concentrations of them. Lead sources include the mining and smelting of lead-bearing ores and metals, lead plumbing and solder, paints and ceramic glazes, the careless disposal of lead-zinc batteries in landfill sites, and the use of leaded gasoline for motor vehicles. Lead releases have been greatly decreased through the use of emission-reducing technologies and the phasing out of some applications such as in motor fuels and paints. Until leaded gasoline was phased out in 1990, motor fuels were the largest single source of lead to the environment in Canada. In 1980, these fuels accounted for 60% of the lead emission in Canada. (Government of Canada 1991a)

Sources of mercury releases to the environment include the recovery of metals, the burning of coal, the use of paint, and the production of chlorine and caustic soda. Mercury has also been widely used in electrical applications, in thermometers and barometers, in seed treatment, in chemical production and in dental fillings. Since the 1970s, there has been a substantial decrease in the amount of mercury released to the environment. Closer regulation of emissions and effluent discharges, control of mercury waste from solid waste disposal sites, control and process improvements, and the replacement of mercury with alternative materials have all contributed to the decline. Furthermore, the use of organic mercury compounds for seed treatment has been banned and the use of mercury in chlorine and caustic soda production has largely been phased out. (Government of Canada 1991a)

Other toxic contaminants in the environment include hydrocarbon compounds such as benzene and polycyclic aromatic compounds (PAHs). Benzene is used mostly in the production of other chemicals. Most of the benzene which enters the environment comes from the burning of motor fuels. Other sources include industrial effluent, spills and leakage from landfill sites and storage tanks. The benzene derivatives, PAHs, are primarily a by-product of incomplete combustion from gasoline and diesel engines and from the burning of wood and coal. (Government of Canada 1991a)

Aside from their intrinsic toxic properties, these groups of substances have other important characteristics that, over time, can greatly enhance their capacity to do biological damage. Firstly, many of these substances are very stable which allows them to persist in the environment for the long periods without decomposing into often less harmful by-products. Likewise, these stable compounds may resist metabolic decomposition by living organisms. Thus, when absorbed or ingested by a living organism, they can gradually accumulate in its tissues possibly to harmful levels. Another result of this stability is that a contaminant can increase in concentration at higher levels within the food chain. Most chlorinated organic compounds have this characteristic and can easily accumulate in the food chain, a process referred to as biomagnification. Another important characteristic is that some chemicals may undergo transformations in the environment that make them much more biologically active or that ease their entry into the food chain. (Government of Canada 1991a)

The concern over toxic contamination in the environment has led to some contaminants being closely studied and monitored since the 1970s. However, monitoring the presence of these contaminants in the environment is difficult because of the low concentrations found in the air, water and soil. Therefore, monitoring of contaminants is usually done in those parts of the environment where they accumulate

such as in aquatic sediments and in living organisms such as algae, fish, birds and other wildlife.

Because the greatest contamination has occurred in heavily populated and industrialized areas, monitoring environmental contaminant levels in Canada has centered on the Great Lakes region. These monitoring programs have provided a good indication of the changes in contaminant levels of various toxic chemicals. The most consistent trend observed in studies monitoring contaminant levels in the Great Lakes region is the decrease in levels of chlorinated organic compounds because of the increasingly tight restrictions and/or bans on the use of some of these substances since the 1970s. There has been a significant decline in the levels of DDT and derivative compounds such as DDE in all areas surveyed. Although not as great as DDT and DDE, the levels of PCBs have also declined in most areas. However, for most other organochlorines that have been monitored, studies have shown considerable local variation with no clear trends observed. (Noble 1990; Government of Canada 1991a)

Some studies of dioxin concentrations in Herring Gull eggs in the Lake Ontario area have shown substantial decreases in levels of 2,3,7,8-TCDD, the most toxic form of dioxin, up to 1989 (Government of Canada 1991b). However, other studies in different locales have shown no clear trends and some have even indicated slightly elevated levels. Although studies have indicated no obvious trends in environmental levels of polychlorinated dioxins and furans, some decrease in the quantity of these contaminants entering the environment has likely occurred as a result of the reduced usage and stricter regulations on the use of chemicals such as chlorophenols and PCBs containing these substances. Furthermore, more recent efforts to reduce emissions of these contaminants through modifying technologies and production processes will also contribute to a decrease in polychlorinated dioxin and furan contaminants in the environment.

Studies have also found significant decreases in levels of heavy metals in the environment. Largely as a result of the phasing out of leaded gasoline, lead emissions have decreased dramatically in Canada. This, along with emission-reducing technologies, has decreased the average ambient air levels of lead particles by over 90% between the years 1974 and 1989 (Government of Canada 1991a). Studies of various fish have also indicated substantial decreases in mercury levels since the 1970s. One study of walleye collected from Lake St. Clair, Ontario between 1970 and 1989 found a significant decline with mercury levels in 1989 being only 25% of the 1970 levels (Government of Canada 1991b). This decline has largely been attributed to the significant decrease in industrial discharges of mercury as its use in many industries has been phased out.

In general, the evidence suggests that most Canadians are now less exposed to toxic contaminants in the environment than they were in the 1960s and 1970s. This decrease in environmental contamination in Canada is attributed to the efforts made to control or eliminate specific contaminant releases to the environment. However, although the groups of toxic chemicals commonly studied and monitored appear to pose the most serious threat to the environment, they only represent a small portion of the total number of chemicals currently used. There are numerous other chemicals which enter the environment that are neither monitored nor regulated which also contribute to contamination.

Furthermore, in many parts of the world, namely many Third World countries, toxic chemicals such as organochlorine pesticides and PCBs are still widely used. Although there has been a recent decline in their use globally, these chemicals still greatly contribute to the contamination of the environment in many areas (Government of Canada 1991a). Likewise, because toxic contaminants are readily dispersed and can be carried long distances by wind and water currents and by wildlife that have absorbed

them, some remote areas, such as the Canadian arctic, have become increasingly contaminated even though they are far from the sources of toxic pollution.

Analysis of Statements

Table 17 presents the results to Statements 6, 7, 8 and 9 for the environmental epidemiologists, environmental engineering professors, toxicologists and the public, as well as for the students surveyed from public health science. These statements were included in the questionnaire to evaluate the respondents' perceptions of various risks in the environment and to determine their attitudes towards natural and synthetic chemicals, and contaminants in the environment.

Table 17 - Responses to Statements 6, 7, 8 and 9

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
6. Fruits and vegetables contain natural substances that can cause cancer.	EE[b]	5.7[a]	16.1	45.8	14.1	18.2
	EP[c]	2.4	8.3	48.2	15.1	26.0
	T2[d]	8.0	11.3	48.0	25.3	7.3
	P2[e]	34.9	33.6	19.9	5.8	5.9
	PH[f]	3.1	28.1	34.4	6.3	28.1
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	EE	2.1	12.6	40.0	28.9	16.3
	EP	1.7	5.8	45.0	36.7	10.7
	T2	2.7	7.3	37.3	47.3	5.3
	P2	12.6	24.2	38.8	20.1	4.3
	PH	6.3	21.9	34.4	15.6	21.9
8. Natural chemicals are not as harmful as man-made chemicals.	EE	21.9	43.8	13.5	0.0	20.8
	EP	23.5	40.3	22.0	3.4	10.8
	T1[g]	45.6	40.2	11.2	2.4	0.6
	T2	54.0	34.0	6.7	2.7	2.7
	P1[h]	10.8	34.0	37.8	7.3	10.0
	P2	14.1	24.1	33.0	23.1	5.7
	PH	31.3	43.8	12.5	0.0	12.5
9. The land, air and water around us are, in general, more contaminated now than ever before.	EE	4.1	29.9	37.1	18.6	10.3
	EP	10.1	37.3	36.3	10.9	5.4
	T1	3.6	24.8	53.3	13.9	4.2
	P1	1.5	8.1	45.2	43.2	1.9
	P2	1.9	4.3	20.8	72.6	0.5
	PH	3.1	6.3	56.3	34.4	0.0

[a] Cell entries are percentages
[b] EE - Environmental Epidemiologists (n = 196)
[c] EP - Environmental Engineering Professors (n = 413)
[d] T2 - Canadian Toxicologists (n = 150; Slovic et al. 1995)
[e] P2 - Canadian Public (n = 1500; Krewski et al. 1995)
[f] PH - Students in a public health science postgraduate course at the University of Alberta (n = 32)
[g] T1 - American Toxicologists (n = 170; Kraus et al. 1992)
[h] P1 - American Public (n = 262; Kraus et al. 1992)

Statement 8, "*Natural chemicals are not as harmful as man-made chemicals.*", was included in the questionnaire to elicit the respondents' attitudes towards natural versus synthetic chemicals. Although many people believe that man-made chemicals are inherently more dangerous than natural chemicals, some of the most acutely toxic and lethal chemicals are natural toxins. For example, botulinum toxin is regarded as one of the most acutely toxic substances in existence. While the majority of toxicity testing is conducted for synthetic chemicals, which may give the impression that synthetic chemicals are more dangerous, evidence suggests that many of the natural chemicals found in foods are just as harmful, if not more harmful because of the greater quantity consumed, than synthetic chemicals such as synthetic pesticides. Furthermore, although some people believe humans have better coping mechanisms for natural chemicals, evidence shows that the toxicology of many natural chemicals is not inherently different from that of synthetic chemicals. Therefore, while specific individual comparisons of natural and synthetic chemicals may vary in relative toxicity, as a general statement, natural chemicals do not have inherent properties which make them safer than man-made chemicals.

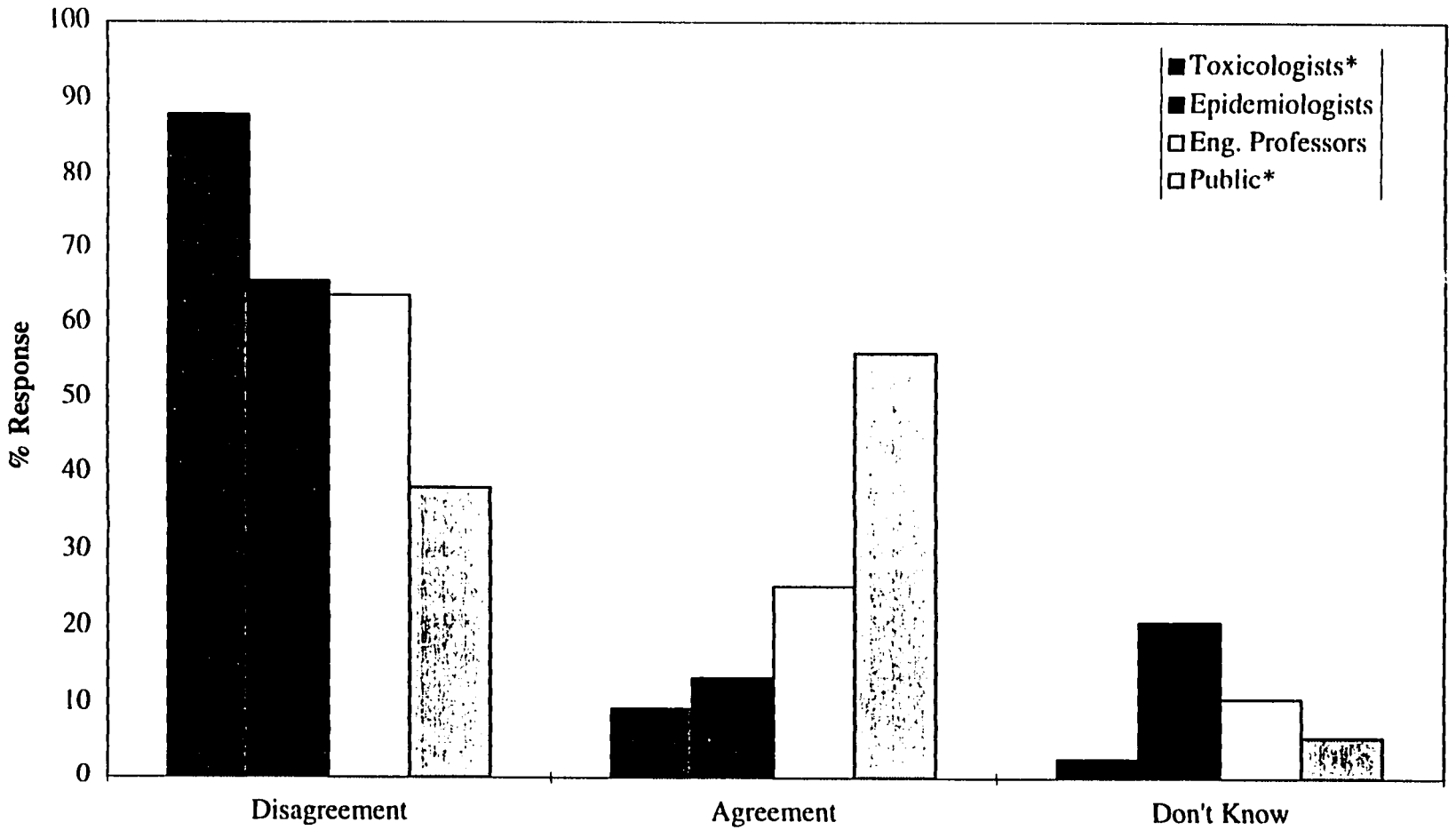
The responses to this statement are listed in Table 17. Figure 12 displays the responses for the environmental epidemiologists and the environmental engineering professors as well as for the groups of Canadian toxicologists and the public sampled from the previous surveys.

Responses to this statement indicate that most of the respondents of the three disciplinary groups disagreed with the assertion that natural chemicals are not as harmful as man-made chemicals. There was substantial disagreement from the groups of toxicologists in both Canada and the United States (88.0% and 85.8% disagreement respectively) (Slovic et al. 1995; Kraus et al. 1992). The environmental epidemiologists and the environmental engineering professors showed a majority level of disagreement (65.7% and 63.8% disagreement respectively). The toxicologists also

had the highest proportion of "strongly disagree" responses. 54.0% of the Canadian toxicologists (Slovic et al. 1995) responded "strongly disagree" as compared to only 23.5% of the environmental engineering professors and 21.9% of the environmental epidemiologists. A large percentage (20.8) of the environmental epidemiologists responded "don't know" to this statement. Similar results were found for the students in public health science (Table 17).

Results for this statement from the public confirm the commonly held belief that the public has more confidence in the safety of natural chemicals than synthetic chemicals. The public showed strong agreement that natural chemicals are not as harmful as synthetic chemicals (56.1% agreement overall with a substantial portion - 23.1% in strong agreement (Krewski et al. 1995)) (Figure 12). Although there was not strong agreement from any of the three disciplinary groups, the group of environmental engineering professors were more likely to agree with this statement than the other two groups. 25.4% of the engineering professors surveyed indicated that natural chemicals are less harmful than man-made chemicals. This group agreed with this statement almost twice as often as the environmental epidemiologists and almost three times as often as the Canadian toxicologists. These results suggest that although the majority of engineering professors disagreed that natural chemicals are not as harmful as synthetic chemicals, many of them have more confidence in the safety of natural chemicals as opposed to man-made chemicals.

Figure 12. Responses to Statement 8:
"Natural chemicals are not as harmful as manmade chemicals."



* Canadian survey (Krewski et al. 1995; Slovic et al. 1995)

The majority of qualitative responses provided for this statement from the environmental epidemiologists and environmental engineering professors were from those respondents in disagreement. Many respondents indicated awareness that some of the most toxic and deadly substances are natural. Many gave references to deadly natural toxins with botulism and aflatoxin being the most common. Others included comments that "both can be equally dangerous" and there is "not much difference toxicologically for some compounds."

Although not many respondents qualified their "agreement" responses, the most common response was that natural chemicals are not as harmful as synthetic chemicals was that this was "true as a general statement but exceptions exist." Another comment indicated that "some natural chemicals are very harmful but there are more man-made ones." A few others believed this statement to be true because "humans encounter lower concentrations of natural chemicals." Other comments suggest that some are in agreement with views such as those from Rachel Carson that natural chemicals are not as harmful because of acclimation to natural chemicals. One response stated that "the body has no defense against artificial chemicals such as dioxin."

Another common response (particularly for those who responded "don't know") was that this statement was too broad of a generalization and needed specifics to answer. Other "don't know" responses included that both natural and synthetic chemicals are significant and can be dangerous, and it is dependent on the specific chemical and the level of exposure. For these comments, the respondents should have responded "disagree" because the statement asked them to make the generalization.

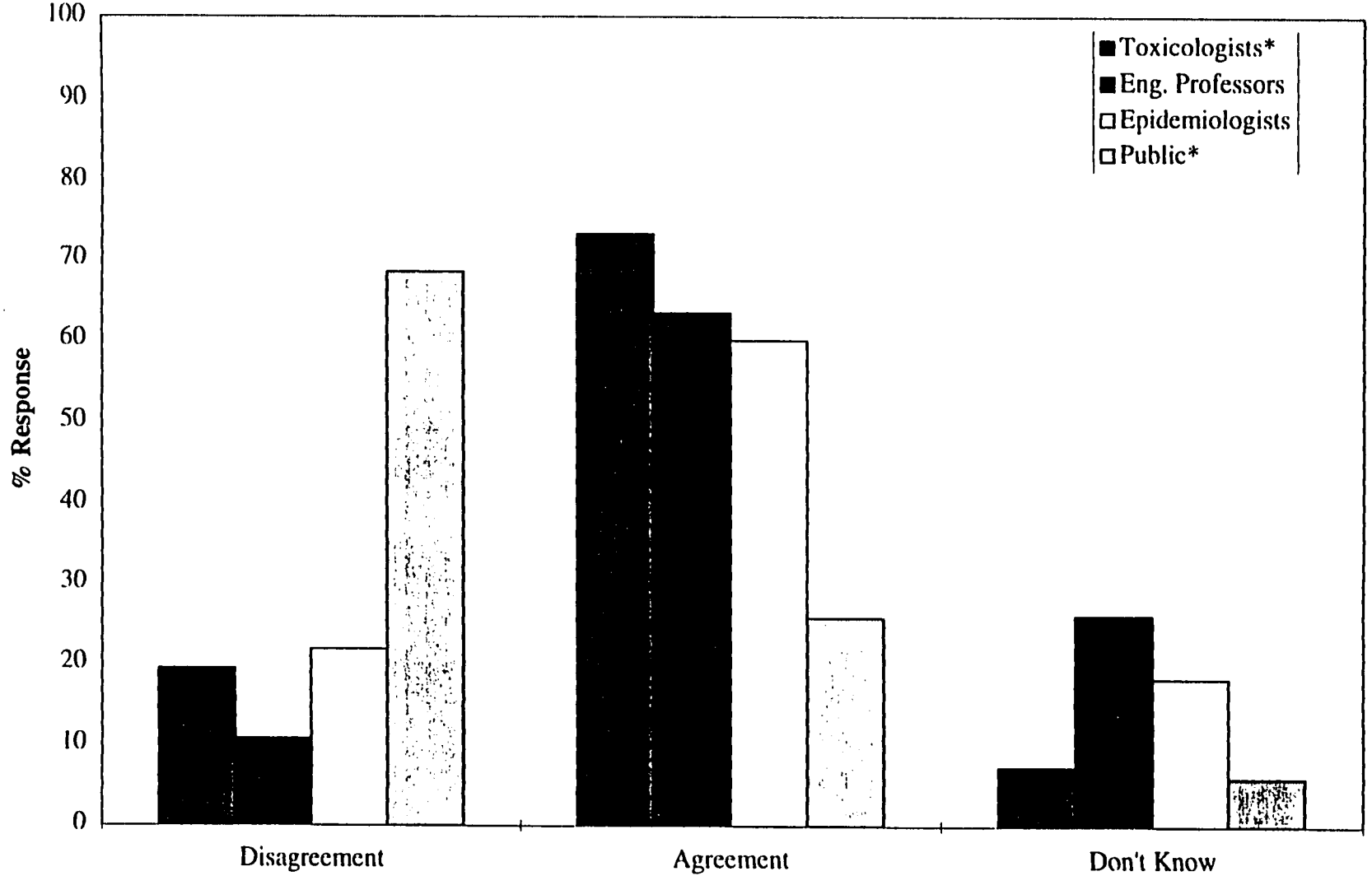
This statement was fairly straight forward and the comments provided did not indicate that respondents had any difficulty in interpreting it. However, one respondent was not sure what was meant by natural chemicals.

Statement 6, *"Fruits and vegetables contain natural substances that can cause cancer."*, was also included in the questionnaire to determine the respondents attitudes towards chemicals. Specifically, their awareness that fruits and vegetables contain numerous different natural chemicals, many of which have been shown to be carcinogenic. For example, all plants (including fruits and vegetables) have biochemical mechanisms that enable them to produce natural pesticides to defend themselves against insects and fungi. Thousands of these natural pesticides have been discovered and of the ones tested using current cancer bioassays, many have been found to be carcinogenic. Furthermore, because potential environmental exposures to chemicals is often greatest through the diet and because almost all of the chemicals and pesticides in the diet are natural rather than synthetic, the risk of cancer from natural substances in food is likely to be far greater than that from synthetic chemicals.

Responses to Statement 6 for the environmental engineering professors, environmental epidemiologists, toxicologists and public (Canadian sample) are displayed in Figure 13. Responses for the Canadian public (68.5 disagreement versus 25.6% agreement) indicate that most of these respondents do not share the view that fruits and vegetables contain natural substances that can cause cancer. Furthermore, many of the public in disagreement displayed great certainty that fruits and vegetables do not contain carcinogenic substances (34.9% strongly disagreed). Only 5.9% expressed that they "don't know". (Krewski et al. 1995)

Figure 13 indicates that the three disciplinary groups were much less likely than the public to disagree that fruits and vegetables contain natural substances that can cause cancer: 19.3% of toxicologists (Slovic et al. 1995), 21.8% of epidemiologists, and 10.7% of environmental engineering professors disagreed that fruits and vegetables contain natural carcinogens.

Figure 13. Responses to Statement 6:
"Fruits and vegetables contain natural substances that can cause cancer."



* Canadian survey (Krewski et al. 1995; Slovic et al. 1995)

The environmental epidemiologist group showed majority agreement (59.9% agreement overall; 14.1% strongly agree) to the statement that fruits and vegetables contain natural substances which can cause cancer. Environmental engineering professors also showed majority agreement (63.3% agreement overall; 15.1% strongly agree). Toxicologists were the most likely to agree that fruits and vegetables contain natural carcinogens: 48.1% of toxicologists surveyed were in agreement with this statement and another 25.3% were in strong agreement (73.4% agreement overall) (Slovic et al. 1995). The public health science students were more split in their opinions (31.2% disagreement versus 40.7% agreement). 28.1% of respondents from this group responded "don't know" to whether fruits and vegetables contain natural substances that can cause cancer (Table 17).

Also noteworthy is the relatively large percentage of "don't know" responses from the environmental engineering professors and environmental epidemiologists (26.0% and 18.2% respectively). Comments from these respondents included: "fruits and vegetables are generally thought to protect against cancer", "evidence incomplete at this point", "not sure but probably", "maybe but probably not", "no one knows", and other similar comments. These responses suggest that a substantial portion of respondents may not be aware of the evidence of natural carcinogenic substances found in fruits and vegetables.

Several respondents of the two groups surveyed disagreed that fruits and vegetables contain carcinogenic substances "unless they are exposed to chemicals", "only if they are contaminated", or "unless they include things like aflatoxin in peanuts." Others stated that some fruits and vegetables "possibly do (contain natural carcinogens) but not always." Another repeated comment for those in disagreement was that there is no risk at doses that are consumed.

From the comments of those respondents in agreement with the statement, it appears that many recognize that natural carcinogens exist in fruits and vegetables.

Many of these respondents provided specific examples of potential carcinogens found in fruits and vegetables such as natural pesticides and substances such as indole carbinol, benzene and molds. Many comments also indicated respondents' awareness that fruits and vegetables also contain anticarcinogenic properties. Numerous comments suggest that although fruits and vegetables contain natural carcinogens the risk depends on dose and these do not occur in dangerous concentrations, i.e. the "concentration present probably does not cause cancer." As well, one person indicated that carcinogenic substances can also occur from cooking. For some others in agreement with this statement, comments such as "if not properly handled", "yes, because of additives-pesticides/herbicides", and "yes, in rare instances, e.g. aflatoxin" indicate that even some who agreed with the statement may not be informed of the presence of natural carcinogens in fruits and vegetables.

Overall, the qualitative and quantitative responses to this statement suggest that a substantial portion of respondents may not be aware of the evidence of naturally occurring carcinogenic substances found in fruits and vegetables. Furthermore, that many respondents have more confidence in the safety of natural chemicals over man-made chemicals (Statement 8) suggests that this area may be one in which greater understanding is needed.

Statement 7, "*The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.*", was included in the questionnaire to examine the respondents' perception of the causes of human cancer and whether they believe chemicals in the environment to be significant factor. Although many may believe that exposures to chemical pollutants are a major factor in the causation of cancer, estimates suggest that only approximately 2% of human cancer in the Unites States arises from all sources of "pollution" (as distinct from tobacco, alcohol, diet, occupation, etc.). Occupational exposures were estimated

to contribute approximately 4% to the total human cancer deaths. Estimates also suggest that the most important contributors to cancer risk are associated with directly controllable "lifestyle" factors such as dietary patterns, tobacco and alcohol usage, reproductive and sexual habits, and sunlight exposure. These factors have been estimated to account for approximately 3/4 of all human cancer deaths. Therefore, while risks from environmental pollution still exist, they appear to be insignificant relative to the risks from lifestyle factors such as smoking and diet.

There was substantial agreement to this statement for the toxicologists (84.6%) (Slovic et al. 1995) and the environmental engineering professors (81.7%) (Table 17). Only 68.9% of the environmental epidemiologists agreed that lifestyle factors posed much greater cancer risks than do chemicals in the environment. The toxicologists were the most likely of the groups to "strongly agree" with this assertion: almost half the respondents of this group "strongly agreed" (47.3%). The environmental epidemiologists had the largest percentage of disagreement (14.7%) as well as "don't know" responses (16.3%).

The greater disagreement from the epidemiologists is interesting given that the main evidence in support of the statement comes from epidemiology. This may be a case of those most familiar with an issue being more cautious than those who are working with second hand knowledge. This finding may be similar to the difference between toxicologists and the other disciplines on the reliability of animal testing for carcinogens compared with other health effects.

The Canadian public had split opinions to this statement with the majority in agreement (58.9% agreement versus 38.8% disagreement). This is supportive of the belief that many in the public perceive environmental pollution to be a major contributor to human cancer. The students in public health science also had split opinions to this statement (50.0% agreement versus 28.2% disagreement) but many of the respondents answered "don't know" (21.9%). (Table 17).

The qualitative responses to this statement indicate that there was not much difference in perspectives between the environmental engineering professors and the environmental epidemiologists. Although disagreement with Statement 7 was a minority overall, qualitative responses for these respondents (and many who responded "don't know") suggest that some view chemicals in the environment to be a serious risk for cancer. Some respondents also stated that lifestyle factors and pollutants in the environment are "both significant contributors to cancer" and that risk is dependent on the chemical - "some chemicals are worse than tobacco", "bad lifestyle is a risk but many chemicals are worse," etc. Many respondents stated that risk is dependent on exposure and the environment in which you live, i.e. whether you "live in a heavily polluted area" or "at or near a hazardous waste site." Similar comments were also included as responses for some respondents who agreed with this statement.

The most common response for those in agreement was "smoking-yes but not necessarily diet." Many of the respondents felt this statement to be true especially for smoking but stated "not sure" or "it's unproven" that diet is a major contributor to cancer. Numerous respondents stated lifestyle factors are the greater risk except in cases such as "high occupational exposures" or "living close to a major source."

Several respondents in all categories also made the distinction between individual characteristics and population. A repeated response was that while this statement may be true for the "general population as a whole, it may not necessarily be true for as individual" or "for most of the population but not all." Others felt this statement to be true "generally" but depends on other factors such as environment (geographic location), the chemical and the level of exposure.

One apparent difficulty in responding to this statement was if respondents agreed that smoking was an important factor in cancer causation but that diet was not. Some of these respondents avoided making a forced choice and responded "don't know" with their qualification. However, most of the respondents with this view still

agreed to the statement but indicated they were not sure about diet. Although this difficulty could have been avoided by just using the term "lifestyle factors" by itself without any clarification, this wording was effective for assessing specifically what respondents believed to be major contributors to cancer. The fact that many respondents are not convinced that dietary patterns are a greater risk factor for cancer than environmental pollution is informative and indicates an opportunity for developing better understanding of the causes of cancer.

Another apparent difficulty with this statement was in the interpretation of "chemicals in the environment." Although it was intended as "industrial chemical pollution", a few comments suggest that some respondents had a larger perspective of environmental chemicals and interpreted the phrase to include smoking and diet as major sources of chemicals in the environment (i.e. the xenobiotic chemicals in tobacco and food). This difficulty would have likely been avoided had the statement been modified to "industrial chemicals in the environment."

Statement 9, "*The land, air and water around us are, in general, more contaminated now than ever before.*" was included in the questionnaire as a general perspective statement to determine the attitudes of respondents towards chemicals in the environment and their overall general perception of the state of the environment. Although perhaps not in some specific locales, from my perspective in a global sense, I would agree with Statement 9 that the land, air and water around us are, in general, more contaminated now than ever before. While many of the western countries have reduced some contaminant levels in the environment by imposing strict controls and regulations of certain toxic chemicals, many countries still widely use these chemicals which contribute to increases in contaminant levels. Furthermore, it seems likely that with increasing deforestation and desertification, along with increasing population

growth and chemical use, the environment is less capable of handling contaminants and they inevitably build up in the environment.

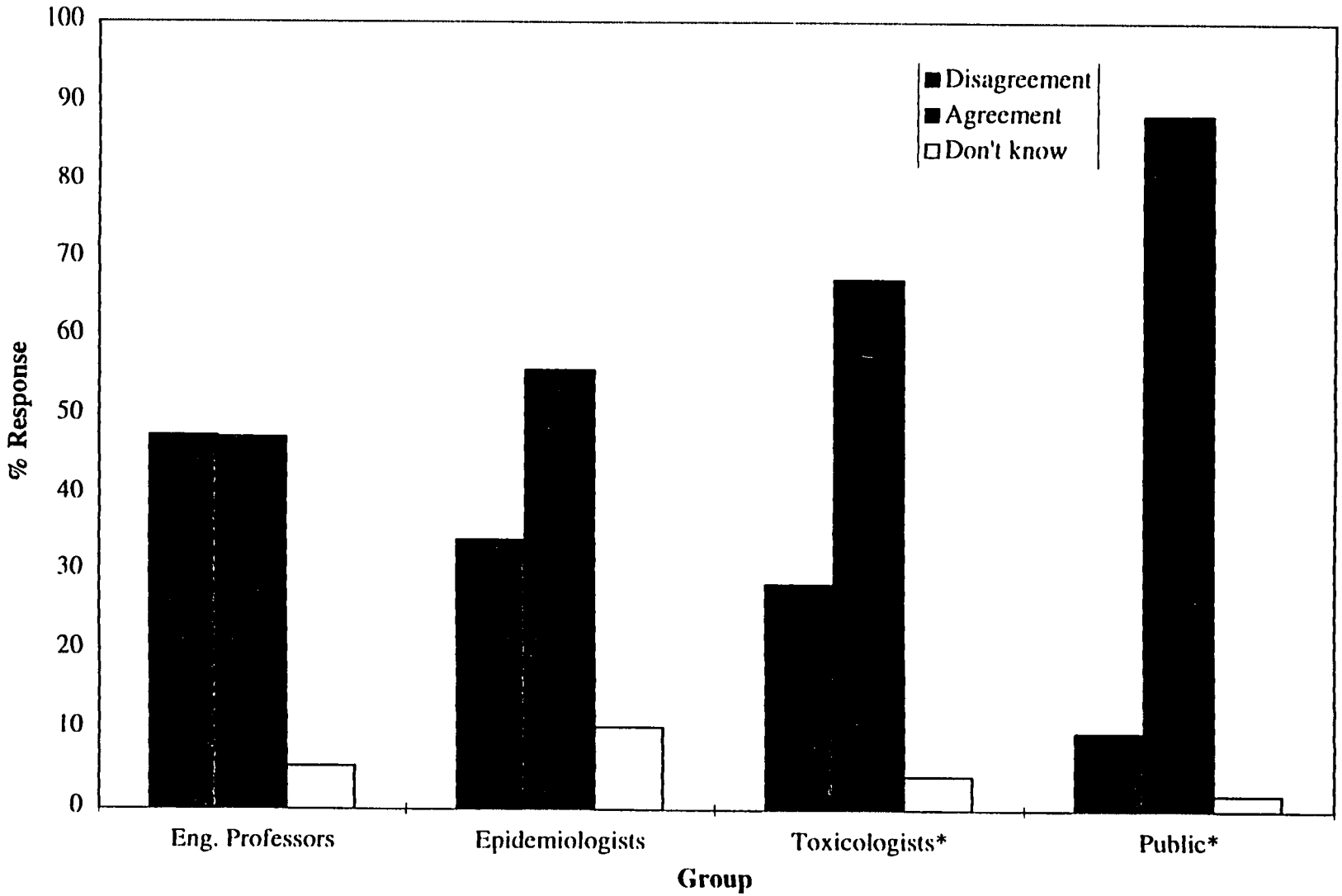
The previous studies show there was almost unanimous agreement (93.4%) from the Canadian public that contamination of the environment is greater now than ever before; over 70% were in strong agreement (Krewski et al. 1995). There were similar findings for the American public with 88.4% agreement overall (43.2% strongly agree) (Figure 14) (Kraus et al. 1992).

Responses to this statement for the toxicologists and the environmental epidemiologists indicate that while opinions are somewhat divided regarding the present contamination of the environment, the majority of respondents from both disciplines were in agreement that the land, air and water around us are, in general, more contaminated now than ever before (Table 17). The toxicologists were the most likely to agree with this statement (67.2% agreement) (Kraus et al. 1992). This group showed 28.4% disagreement (Figure 14). The environmental epidemiologists had slightly less agreement (55.7% agreement versus 34.0% disagreement (Figure 14). This group also had the largest percentage of "don't know" responses.

The environmental engineering professors were equally divided in their opinions to this statement with 47.4% in disagreement and 47.2% in agreement that contamination of the environment is greater now than ever before (Figure 14). This group showed the strongest disagreement to this statement.

The responses from the students in public health science were similar to those of the public in that there was almost complete agreement that contamination of the environment is at its greatest (Table 17).

Figure 14. Responses to Statement 9:
"The land, air and water around us are, in general, more contaminated now than ever before."



* American survey (Kraus et al. 1992)

Qualitative responses do not indicate any divergent perspectives between the environmental epidemiologists and the environmental engineering professors. The most common theme for the qualitative responses to this statement from both groups was that it "depends on location": a substantial number of respondents (from all response categories) believed this statement to be true in a global sense but not necessarily locally. Many agreed with this statement for developing countries but would disagree for developed countries.

Aside from "where" contamination takes place, "when" was also an important consideration. Time references and comparisons to the Industrial Revolution and the last 20 - 30 years were common responses. A large portion of comments for those in disagreement that contamination is greatest now indicated that many feel regulatory and clean-up efforts have significantly improved the environment (at least some areas) in the last 30 years and that the environment was much worse a few decades ago. Other comments from the "disagree" category indicated that some aspects of the environment are worse now while others are better. A few references were made to the air and water stating that air and/or water are cleaner now than past.

A repeated comment for those in agreement that contamination is greater now was that contamination is the inevitable consequence of population growth and human behavior, i.e. "more people means more pollution." References to man-made chemicals and their persistency in the environment (long half lives, etc.) were also made.

Similar to other responses, the most repeated comment for "don't know" category was that response depends on location. Also, many indicated confusion over the time scale and what was meant by the words "now" and "ever before."

The qualitative response provided for this statement indicate that response to this statement largely depends on whether respondents considered the environment to include only their "local" surroundings or whether it included the environment as a

whole. Response may also have depended on how respondents viewed "contaminated" and whether their perspective of contamination was larger than only chemical pollutants. Unfortunately, the comments available did not provide any insight for determining how contamination was considered.

Summary

A major objective of this research was to determine if members of some major environmental disciplines exhibit divergent interpretations of several underlying assumptions and concepts in environmental health risk assessment. These environmental disciplines included environmental engineering professors, environmental epidemiologists and toxicologists. If divergent interpretations were apparent, the research sought to characterize the difference.

The results to the statements presented indicated that there were not any sharp distinctions in perspectives between these three disciplines. For the most part, these disciplinary groups often exhibited very similar patterns of response. However, divergent interpretations were found for several of the statements within the groups themselves indicating that even within a particular field of study, members are not a homogeneous group.

Overall, the majority of these respondents appeared to have a reasonable understanding of the assumptions and concepts presented. However, results suggest that there are areas in which a certain level of misunderstanding among some respondents is apparent. Therefore, it is instructive to identify those issues which seem to offer the greatest opportunity for improved understanding among one or more of these groups.

Responses for statements concerning dose-response and exposure relationships indicate that these concepts are well understood among respondents in all three disciplinary groups. There was substantial disagreement among respondents to the statements that a chemical is either safe or dangerous with no in between (Statement 1), and any exposure to a cancer-causing agent will probably cause harm (Statement 5). However, contrary to the basic principles of dose-response recognized in these former

statements, there were varying opinions concerning whether or not safe levels of exposure exist for carcinogens (Statement 2). Although the majority of respondents in all three disciplinary groups were in disagreement with this statement, there was a moderate proportion of respondents, particularly in the environmental epidemiology group, who agreed that a safe level of exposure to a carcinogen does not exist. Over 1/3 of environmental epidemiologists were in agreement with this statement.

The increase in agreement for Statement 2 among all three groups but particularly the environmental epidemiologists suggests that respondents may need to carefully evaluate their definition of safety and recognize that while zero risk is unattainable, there can still be some level of exposure that can be considered safe. Perpetuating the belief that there is no safe level of exposure to a carcinogen may possibly send misleading messages to the public who then may not trust the safety of the regulations made for carcinogens.

Responses to Statement 3, "*The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of that chemical.*", and Statement 4, "*If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.*", also indicate that there may be a need for greater understanding among some respondents concerning the limitations of cancer testing in animals and carcinogenesis in general.

When asked about their confidence in the ability of animal studies to predict human health effects, all three disciplinary groups as well as the public had diverging opinions. Over half of the environmental engineering professors and environmental epidemiologists surveyed were in disagreement that the health effects from exposure to a chemical experienced by an animal are a reliable predictor of the human health effects from that chemical. The toxicologists showed a more favorable view of animal studies.

Overall, this statement indicated that numerous respondents (in all groups) lack confidence in the value of animal studies for predicting human health effects. However, when asked a similar statement about the validity of animal studies showing evidence of cancer in animals for predicting cancer in humans (Statement 4), many respondents changed their opinions concerning the reliability of animal testing to predict human health effects. Whereas the mention of cancer provoked many in the public to become more certain in the prediction of harm to humans (overall percent agreement rose over 25% - Kraus et al. 1992), the toxicologists became less confident that similar health effects (cancer) will occur in humans; likely reflecting that these respondents have more appreciation for the numerous uncertainties associated with animal cancer testing.

Although there was no change in overall response between Statements 3 and 4 for the environmental epidemiologists, the environmental engineering professors displayed a pattern of response opposite to that of the toxicologists and similar to that of the public. When a chemical was found to be a carcinogen in animals, some environmental engineering professors became more confident in animal studies for determining health effects in humans and overall agreement rose 12.5%. Although this may appear insignificant overall, it is important to recognize that even small groups of individuals can be very influential on the decisions of others particularly where their interaction with others is great.

It is difficult to interpret why the mention of cancer provokes a different response pattern among some respondents. However, it suggests that a better appreciation of the limitations and additional uncertainties of animal cancer testing, such as the use of the maximum tolerated dose (MTD), within species variation of carcinogenic effects as well as the mechanisms of carcinogenesis itself, may be needed.

Another area in which increased awareness may be required is the ability of epidemiological methods for establishing causation. While it is encouraging that quantitative and qualitative responses to statements on causal inference (Statements 14 and 19) indicate that a large majority of respondents from both the environmental epidemiology group and the environmental engineering professors are familiar (to varying extents) with various criteria for judging causality between exposure to a risk factor and an effect, there were very few respondents who indicated understanding that epidemiological studies are only capable of demonstrating an association between an exposure and an effect and that a plausible biological mechanism from toxicological investigation is necessary to support an epidemiological study when inferring causation. Several responses suggested that a small but notable percentage of respondents from both groups may not appreciate this.

Therefore, to improve understanding and communication of health risk findings, it is important for environmental scientists to recognize that because of the numerous uncertainties and methodological difficulties, epidemiological studies, strictly by themselves, cannot establish causation. Furthermore, because epidemiological methods cannot establish the biological mechanisms necessary for demonstrating causation, they are only capable of demonstrating an association between an exposure and an effect.

The high percentage (45%) of agreement and "don't know" responses among the environmental engineering professors to statements on statistical inference (Statements 17 and 18) suggests that the role of statistical inference in environmental health risk assessment may not be well understood by this group. Furthermore, although understanding of statistical concepts in environmental health risk assessment appeared to be more apparent in the environmental epidemiology group, some of the

comments provided reflect a potential need to improve the understanding on this issue for some of these respondents as well.

Because statistical analyses are inherent in methods used for identifying and evaluating potential health risks from environmental contaminants, it is important for those people who use environmental health data to understand the concepts of statistical inference in order to accurately interpret the findings.

One important area which appears to deserve increased awareness among respondents of these groups, particularly the environmental engineering professors, is that of distinguishing statistical inference from causal inference. Although statistical analysis is essential for interpreting the data of epidemiological and toxicological studies, it has a very limited role in explaining causation. While statistical analysis such as significance testing can aid in supporting a causal hypothesis, it does not reflect the biological or practical significance, nor does it confirm the existence of a cause-effect relationship. Statistical significance only provides information on whether observed differences are caused strictly by random variation. Furthermore, even if statistical analysis indicates the observed differences are real and not caused by random variation. it cannot provide insight into whether the magnitude of observed differences is important nor can it actually prove that the risk factor being investigated is in fact the cause of the differences.

The responses to statements evaluating uncertainty and confidence in health risk analysis (Statements 10, 13, 15 and 16) also reflect the potential need for improved understanding of the substantial uncertainties and limitations in environmental health risk assessment among the disciplinary groups.

Both the environmental engineering professors and the environmental epidemiology groups were divided in their opinions regarding the accuracy of expert risk estimates (Statement 13) and whether a lifetime cancer risk as low as one chance in

a million can be known (Statement 15). Although disagreement to these statements was the view held by the majority, there was still a substantial number of respondents from both groups who expressed confidence in the ability of experts to determine reliable health risk estimates. This result suggests that some respondents may have unrealistic expectations of what risk assessment can accomplish and that an awareness of the uncertainties and limitations in risk estimation, particularly cancer risk estimation, may be lacking. Recognizing that variability and uncertainty surround all aspects of quantitative risk assessment, and that there are numerous assumptions and judgments made at every stage in a health risk analysis are essential for developing a mature perspective of the estimates generated through risk assessment.

Analysis of Individual Responses

Although overall responses did not often indicate large discrepancies within and between the disciplinary groups, analysis of individual responses validates how internally consistent responses are. To develop better understanding of individual responses, selected statements were cross-tabulated and examined for consistency. Individual consistency in response is important in that it suggests respondents have examined the issues presented and have responded logically and consistently. Inconsistent responses suggest that there are likely misunderstandings concerning some of the issues. Furthermore, statements with large inconsistencies indicate that responses may not have been well thought out and may be less useful for determining the knowledge respondents have of risk assessment concepts. For example, if a respondent was in disagreement with Statement 1, "*A chemical is either safe or dangerous. There is really no in between.*" then logically the respondent should also be in disagreement with Statement 2, "*There is no safe level of exposure to a cancer-causing agent.*"

Both the environmental epidemiologist group and the environmental engineering professors showed large inconsistencies between their response to Statement 3, "*The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.*" and their response to Statement 4 "*If a scientific study produces evidence that a chemical causes cancer in animals, then we can reasonably sure the chemical will cause cancer in humans.*" For consistent individual responses, respondents in disagreement (both disagree and strongly disagree responses) with Statement 3 should likely have also been in disagreement with Statement 4 or responded "don't know" because of the even greater uncertainties and inferences associated with animal cancer testing.

For the environmental epidemiologists, although overall percent agreement or disagreement changed very little between these two statements, examination of individual responses indicate some inconsistencies. 24.6% of respondents in disagreement with Statement 3 changed their response to either agreement (agree or strongly agree responses) or "don't know." Furthermore, 41.7% of respondents in agreement with Statement 3 and 52.9% of respondents who answered "don't know" to Statement 3 changed their view for Statement 4.

In further detail, the majority of environmental epidemiologists were in disagreement with Statement 3 (114 of 191 valid responses or 59.7% overall). However, of these 114 respondents in disagreement with Statement 3 the majority (86 respondents or 75.4%) were also in disagreement with Statement 4. The remaining respondents who were in disagreement with Statement 3 were either in agreement or responded "don't know" to Statement 4. Additionally, a few respondents who answered "don't know" to Statement 3 responded agree to Statement 4. Therefore, a total of 12.6% of environmental epidemiologists changed their view for Statement 3 from either disagreement or "don't know" to agreement for Statement 4. However, these individual changes were not reflected in the overall numbers because there was also approximately 12% who changed their responses from agree or "don't know" to disagree when animal tests showed evidence of cancer. Overall this analysis of individual response indicates that although many respondents became more confident in animal testing when there was evidence of cancer, many also recognize the substantial uncertainties associated with cancer testing.

Inconsistency in overall response was much more apparent in the sample of environmental engineering professors. Although the majority of respondents (52.4% overall) were in disagreement with Statement 3, there was a large increase in percent agreement for Statement 4 when animal testing showed evidence of cancer (percent agreement increased from 29.6% to 42.1%).

For this group, only 67.3% of the 211 respondents in disagreement with Statement 3 also disagreed with Statement 4. 77.3% (92 of 119 respondents) of those respondents in agreement with Statement 3 remained in agreement with Statement 4 and only 38.9% (28 of 72 respondents) of those in the "don't know" category for Statement 3 also responded "don't know" to Statement 4.

Of those in disagreement with Statement 3, 22.7% changed their view to agreement when answering Statement 4 while 10% changed to "don't know" to Statement 4. Many of those responding "don't know" to Statement 3 also responded agree (or strongly agree) to Statement 4 (38.9%). For these respondents, the aspect of cancer testing apparently increased their confidence in the meaning of results.

The inconsistencies for these statements, particularly the increased faith in animal testing when the substance is carcinogenic for the environmental engineering professors, suggest that there may be some misunderstandings of carcinogenesis, and animal carcinogenic testing in general, which may be the driving factor for response to these statements. Clearly, analysis of the patterns of individual responses suggests that the mention of cancer provokes a different response pattern for many respondents.

To further examine the distinction respondents make between cancer and any other adverse effect, Statement 1, "*A chemical is either safe or dangerous. There is really no in between.*" and Statement 2, "*There is no safe level of exposure to a cancer-causing agent.*" were also cross-tabulated. Disagreement that a chemical is either safe or dangerous with no in between (i.e. recognition that toxicity is a continuum rather than all or nothing) should correlate with disagreement that there is no safe level of exposure to a cancer causing agent (i.e. recognition of a continuum of cancer risks rather than an absolute cancer risk). Likewise, disagreement with Statement 2 may correlate with disagreement with Statement 5, "*If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.*"

For the environmental epidemiologists, large inconsistencies in response were found for Statements 1 and 2. While a substantial majority (89.5% overall) of respondents disagreed that chemicals are either safe or dangerous with all-or-nothing toxic properties (Statement 1), many agreed that there is no safe level of exposure if the chemical is a carcinogen. Only 59.4% (101 respondents) who were in disagreement with Statement 1 also disagreed with Statement 2. 31.8% (54 respondents) in disagreement with Statement 1 agreed with Statement 2; again suggesting a distinction in how cancer risk was viewed.

Individual consistency in response for this group was much more apparent for Statements 2 and 5. Of those respondents in disagreement that there is no safe level of exposure to a cancer-causing agent, i.e. agree that a safe level exists, almost 90% were also in disagreement that if a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day (Statement 5).

The environmental engineering professors did not show inconsistencies between Statements 1 and 2 as large as those shown by the environmental epidemiologists. For Statement 1, there was almost unanimous disagreement (96.4% or 397 respondents) among this group. Of these respondents, 74.6% were also in disagreement with Statement 2. Only 13.9% of those in disagreement with Statement 1 were in agreement with Statement 2; 11.6% changed their response to "don't know."

For the environmental engineering professors, there was also large consistency in response to Statements 2 and 5. Of those who were in disagreement that there is no safe level of exposure to a cancer-causing agent, almost 90% also disagreed that any exposure to a cancer causing agent will probably result in harm.

Similar to the inconsistencies found for Statements 3 and 4 in trust in animal testing, the results for these two disciplinary groups suggest that respondents make a distinction between carcinogens and other chemicals. When statements include

carcinogenic compounds, it triggers some effect and some respondents appear to be more inclined to rate any exposure, no matter how small, as dangerous.

Cross-tabulations of Statements 6 and 8 also provide information on individual consistency in respondents' answers. Respondents who recognize that *"Fruits and vegetables contain natural substances that can cause cancer."* (Statement 6) should also recognize the fallacy that *"Natural chemicals are not as harmful as man-made chemicals."* (Statement 8). Thus agreement with Statement 6 should correlate with disagreement with Statement 8.

For the environmental epidemiologists, some inconsistencies were apparent. Overall responses indicate that the majority of respondents (59.9%) agreed that fruits and vegetables contain natural carcinogens and disagreed that natural chemicals are not as harmful as man-made chemicals (65.7%). However, of those respondents in agreement with Statement 6 (113 respondents), only 74.3% (84 respondents) were also in disagreement with Statement 8. The remaining 14.2% and 11.5% responded "don't know" and agree (or strongly agree) respectively.

The overall responses for the environmental engineering professors show consistent results with 63.3% agreeing that fruits and vegetables contain natural substances that can cause cancer (Statement 6) and 63.8% disagreeing that natural chemicals are not as harmful as man-made chemicals (Statement 8). However, similar to the environmental epidemiologists, only 181 (70.4%) of the 257 respondents in agreement with Statement 6 were in disagreement with Statement 8. 23.3% of those respondents in agreement that fruits and vegetables contain natural carcinogens also agreed that natural chemicals are not as harmful as synthetic chemicals. This group of respondents (60 respondents) appear to recognize that natural carcinogens exist yet they still recognize some inherent danger in synthetic chemicals.

Statements 10 and 3 can also be cross-tabulated to determine the consistency in individual response. If respondents disagree with Statement 10 that 20 years of human experience with a prescription drug is safer than current regulatory testing protocols, i.e. they believe that animal testing provides more accurate information regarding the possible effects of a prescription drug, then the respondents should be in agreement with Statement 3 that the health effects from animal testing are a reliable predictor of human harm from that chemical. Logically, those people with more faith in present regulatory evaluation should also have faith in animal testing which underlies the current regulatory scheme.

For both disciplinary groups, there was much greater disagreement than agreement for Statement 10. However, the majority of respondents in both groups were in disagreement with Statement 3 regarding the validity of animal testing for predicting human harm. Both groups showed large inconsistencies in response to these statements. Of the environmental epidemiologists who were in disagreement that an untested but widely used prescription drug is safer than a new prescription drug tested and approved for use under present regulatory guidelines, only 36.4% trusted animal tests for predicting human health effects. The majority of these respondents (58.6%) disagreed with the reliability of animal testing and the remaining 5.1% responded "don't know."

Similar results were found for the environmental engineering professors. Of these respondents who disagreed with Statement 10, only 32.9% were in agreement with Statement 3; 55.2% were in disagreement and 11.9% responded "don't know."

Similarly, if respondents have trust in current testing protocols (i.e. disagreement with Statement 10) then they may also believe in the accuracy of health risk estimates made by scientists (i.e. agreement with Statement 13). There were large inconsistencies in response to these statements for both disciplinary groups. Of the

environmental epidemiologists in disagreement with Statement 10 (51.6% overall), only 35.4% responded in agreement that scientific experts are able to make accurate estimates of health risks from chemicals in the environment (Statement 13); 63.6% of these respondents were in disagreement.

The environmental engineering professors had a similar pattern of response with 33.6% of those in disagreement with Statement 10 agreeing with Statement 13; 57.5% were in disagreement and 8.9% responded "don't know."

These large inconsistencies in response evident between Statements 10 and 3 and between Statements 10 and 13 for both groups suggest that many respondents may not fully appreciate the heavy reliance on animal testing for current regulatory protocols. Although current drug approvals will usually involve clinical trials with human populations, they are strongly influenced by evidence from animal experiments. Consequently, this inconsistency suggests a possible misunderstanding about the evidence used for regulatory approval of drugs.

Responses to statements concerning confidence in health risk estimates should also correlate with each other. For instance, those in agreement with Statement 15, "*A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.*", should likely have also agreed that scientific experts are able to make accurate health risk estimates (Statement 13).

Again both groups showed large inconsistencies. Of those in agreement that a lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen (Statement 15), 53.2% of the environmental epidemiologists and 40.0% of the environmental engineering professors agreed with Statement 13 that scientific experts are able to make accurate health risk estimates. However, it is possible that these inconsistencies can partly be attributed to the wording and interpretation of the statements.

The responses to the statements which evaluate how respondents interpret evidence regarding cause-effect relationships indicate that most respondents were consistent in their responses. If respondents recognize that a single epidemiological study cannot alone establish causation between a contaminant in the environment and a human health effect (disagreement with Statement 14) then it is likely they would also recognize that causal inference cannot be made between pesticide use and malformations in children (disagreement with Statement 19). "Don't know" may also have been a valid response to Statement 19 if respondents interpreted this category for "cannot be known from the information provided."

Both disciplinary groups had the large majority of respondents disagreeing with Statement 14 that "*A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.*" Of these respondents approximately 90% of both groups were either in disagreement (58.5% of environmental epidemiologists and 68.1% of environmental engineering professors) or responded "don't know" (31.6% of environmental epidemiologists and 22.5% of environmental engineering professors) to Statement 19 that it is very likely that agricultural pesticide use in a small community contributed to malformed children.

Statement 14, "*A single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.*" can also be cross-tabulated with Statement 17, "*A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.*" and Statement 18, "*Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.*" These cross-tabulations provide further insight on whether respondents interpret statistical inference as causal inference. Misunderstanding of the role of

statistical inference may be a large contributor to respondents not considering other important criteria necessary for establishing causation. This may possibly be indicated by those in agreement with Statements 17 and 18 also agreeing with Statement 14 that a single epidemiological study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.

However, the results for both the environmental epidemiologists and the environmental engineering professors did not indicate this. For the environmental epidemiologists in agreement with Statements 17 and 18, the vast majority (74.1% and 82.8% for Statements 17 and 18 respectively) disagreed with Statement 14. Only 25.9% of those in agreement with Statement 17 and 14.1% of those in agreement with Statement 18 also agreed with Statement 14.

Of the environmental engineering professors in agreement with Statement 17, 75% were in disagreement with Statement 14 while 16.7% were in agreement. Similarly, 80.0% of the environmental engineering professors who agreed with Statement 18, disagreed with Statement 14 and 11.8% agreed.

In summary, although overall responses for statements did not often show large discrepancies between or within these two disciplinary groups, analysis of consistency in individual responses indicate that there may be misunderstandings on some of these concepts. Particularly for statements concerning cancer versus any other adverse effect, validity of animal studies for predicting human health effects, and confidence in risk estimates, inconsistent responses suggest that these areas may require developing better understanding.

Analysis of Demographics

Assessing the influence of demographic factors such as gender, experience, age or affiliation also provides further insight into the responses given to these statements.

Gender Effects

Previous studies on "Intuitive Toxicology" by Kraus et al. (1992) and Slovic et al. (1995) suggested that within the public sample, women were consistently more concerned about chemical risks than men and that they had less favorable attitudes regarding the benefits of chemicals (Kraus et al. 1992). These studies also found that some questions showed a high percentage of "don't know/no opinion" responses among women. For the samples of toxicologists, gender differences were much less evident. However, female toxicologists did appear to be more concerned about chemical risks and less favorably impressed with the benefits of chemicals than were male toxicologists (Kraus et al. 1992).

Within the sample of environmental epidemiologists, differences in opinion between men and women were not great. Statements for which gender differences were largest are shown in Table 18. Overall disagreement to Statements 1, 2 and 5 indicate no differences in opinion between men and women. However, although the majority of women disagreed, there was a much stronger reaction from the male respondents to these statements. Table 18 illustrates that women were much less inclined to "strongly disagree" that:

- a chemical is either safe or dangerous with no in between (Statement 1),
- there is no safe level of exposure to a cancer-causing agent (Statement 2), and

- if a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day (Statement 5).

In these cases, the difference between men and women was mainly evident in the scale of disagreement. When the "strongly disagree" and "disagree" responses were combined, there was essentially no difference between male and female responses.

With regard to the reliability and validity of animal tests for predicting human harm (Statement 3), there was little difference in agreement between genders. Even when an animal study produced evidence of cancer (Statement 4), agreement responses did not change much for either gender.

Examination of Table 18 also indicates that women were much less likely than men to:

- agree that lifestyle factors pose a much greater risk for cancer than chemicals in the environment (Statement 7),
- disagree that an untested prescription drug that has been widely used for 20 years is safer than a new drug that has been tested and approved under current regulatory guidelines (Statement 10), and
- disagree that statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study (Statement 18).

Note, however, that these views, though more common among men, were still held by the majority of women sampled. Also of interest is the higher percentage of "don't know" responses among women for Statements 5, 7 and 10 (Table 18).

Table 18. Gender Differences Among Environmental Epidemiologists to Selected Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
1. A chemical is either safe or dangerous. There is really no in between.	M[b]	**56.9**[c]	31.0	9.5	2.6	0.0
	F[c]	**43.8**	49.3	2.7	4.1	0.0
2. There is no safe level of exposure to a cancer-causing agent.	M	**20.3**	36.4	27.1	8.5	7.6
	F	**9.9**	47.9	18.3	14.1	9.9
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	M	**53.4**	33.9	8.5	0.0	4.2
	F	**27.4**	53.4	6.8	0.0	**12.3**
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	M	1.8	11.4	**40.4**	**34.2**	12.3
	F	2.8	12.5	**40.3**	**20.8**	**23.6**
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	M	**14.4**	**42.4**	22.9	0.8	19.5
	F	**12.5**	**30.6**	19.4	0.0	**37.5**
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.	M	**20.5**	**42.7**	28.2	0.9	7.7
	F	**18.1**	**26.4**	38.9	2.8	13.9

[a] Cell entries are percentages
[b] Male environmental epidemiologists (n = 118)
[c] Female environmental epidemiologists (n = 74)

Table 19 presents the statements for which gender differences among environmental engineering professors were the largest. However, interpreting these results is difficult as there was a large discrepancy in the number of female and male respondents. While 353 (85.5%) respondents were male only 50 (12.1%) who responded were female.

For Statements 1, 2 and 5, the pattern of response for men and women environmental engineering professors was similar to those for the environmental epidemiologists. Again, although there was little difference in overall disagreement percentages between men and women, women were much less inclined to "strongly disagree" that (Table 19):

- a chemical is either safe or dangerous with no in between (Statement 1),
- there is no safe level of exposure to a cancer-causing agent (Statement 2), and
- if a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day (Statement 5).

Table 19. Gender Differences Among Environmental Engineering Professors to Selected Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
1. A chemical is either safe or dangerous. There is really no in between.	M[b]	**71.1**[a]	25.5	2.0	1.1	0.3
	F[c]	**58.0**	36.0	4.0	0.0	2.0
2. There is no safe level of exposure to a cancer-causing agent.	M	**27.6**	46.9	11.9	2.0	11.6
	F	**10.0**	60.0	14.0	4.0	12.0
3. The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.	M	**6.9**	**43.8**	30.5	0.9	17.9
	F	**12.2**	**49.0**	18.4	0.0	20.4
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	M	**5.5**	**39.7**	39.1	2.6	13.2
	F	**4.1**	**30.6**	44.9	0.0	20.4
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	M	**36.4**	47.7	4.8	0.3	10.8
	F	**24.0**	62.0	4.0	0.0	10.0
8. Natural chemicals are not as harmful as man-made chemicals.	M	**22.3**	**39.3**	23.2	3.4	11.7
	F	**32.0**	**48.0**	14.0	0.0	6.0
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	M	**5.2**	**28.1**	20.9	1.1	**44.7**
	F	**4.1**	**44.9**	18.4	0.0	**32.7**

[a] Cell entries are percentages
[b] Male environmental engineering professors (n = 353)
[c] Female environmental engineering professors (n = 50)

Although not the majority view, men were somewhat more favorably inclined towards animal testing for predicting human health effects (Statement 3). Although there was not great difference between the genders, women were more likely to disagree that the health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical (61.2% disagreement

versus 50.7% for male respondents). Statement 4 also indicated some difference in response between male and female environmental engineering professors. However, when animal studies show positive evidence of cancer in animals, women were much more inclined to change their response and agree that similar health effects will occur in humans. Whereas percent agreement from men increased from 31.4 to 41.7, percent agreement from women increased substantially from 18.4 to 44.9 (Table 19). Thus, the majority view for women in Statement 3 became a minority view when animal tests showed evidence of carcinogenicity (Statement 4).

On other statements, male environmental engineering professors were much less likely to (Table 19):

- disagree that natural chemicals are not as harmful as man-made chemicals (Statement 8), and
- disagree that an untested prescription drug that has been widely used for 20 years is safer than a new drug that has been tested and approved under current regulatory guidelines (Statement 10).

Note that for Statement 10, the largest fraction of male respondents (44.7%), as well as a high percentage of female respondents, answered "don't know".

Experience Effects

Years of experience was also found to be a possible factor in explaining response to specific statements. Of the 196 respondents from the environmental epidemiology group, the majority (91 respondents - 46.4%) indicated greater than 5 years experience with environmental epidemiology. 62 (31.6%) respondents indicated 1 to 5 years experience and 42 (21.4%) indicated less than 1 year experience with environmental epidemiology.

Table 20 presents selected statements for which differences between the number of years of experience with environmental epidemiology were the greatest. Overall disagreement did not indicate any difference between respondents from the various experience categories to the statements concerning dose-response and exposure relationships (Statements 1, 2 and 5). However, responses to these statement indicate that respondents with greater experience with environmental epidemiology (> 5 years) had the strongest reaction to these statements. Respondents with less experience (0 to 5 years) were much less inclined to "strongly disagree" that (Table 20):

- a chemical is either safe or dangerous with no in between (Statement 1),
- there is no safe level of exposure to a cancer-causing agent (Statement 2), and
- if a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day (Statement 5).

Table 20. Experience Differences Among Environmental Epidemiologists to Selected Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
1. A chemical is either safe or dangerous. There is really no in between.	<1[b]	**42.9**[a]	45.2	9.5	0.0	0.0
	1-5[c]	**41.0**	44.3	8.2	6.6	0.0
	>5[d]	**62.2**	30.0	5.6	2.2	0.0
2. There is no safe level of exposure to a cancer-causing agent.	<1	**11.9**	52.4	23.8	4.8	7.1
	1-5	**13.1**	37.7	24.6	8.2	16.4
	>5	**21.3**	36.0	24.7	14.6	3.4
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	<1	**28.6**	54.8	7.1	0.0	9.5
	1-5	**34.4**	50.8	8.2	0.0	6.6
	>5	**56.0**	28.6	8.8	0.0	6.6
6. Fruits and vegetables contain natural substances that can cause cancer.	<1	2.4	19.0	**40.5**	**9.5**	**28.6**
	1-5	6.6	18.0	**44.3**	**13.1**	**18.0**
	>5	6.8	12.5	**50.0**	**17.0**	13.6
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	<1	0.0	14.3	**47.6**	**21.4**	**16.7**
	1-5	1.6	18.0	**37.7**	**18.0**	**24.6**
	>5	3.5	7.0	**38.4**	**40.7**	10.5
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	<1	**7.1**	**21.4**	45.2	0.0	**14.3**
	1-5	**1.6**	**23.0**	55.7	9.8	9.8
	>5	**7.8**	**38.9**	40.0	7.8	5.6
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	<1	**21.4**	**38.1**	14.3	0.0	**26.2**
	1-5	**36.7**	**40.0**	16.7	0.0	6.7
	>5	**32.2**	**53.3**	12.2	0.0	2.2
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.	<1	**7.1**	**28.6**	31.0	0.0	**33.3**
	1-5	**26.2**	**32.8**	36.1	0.0	4.9
	>5	**22.5**	**42.7**	29.2	3.4	2.2

[a] Cell entries are percentages

[b] 0 to 1 year experience with environmental epidemiology (n = 42)

[c] 1 to 5 years experience with environmental epidemiology (n = 62)

[d] More than 5 years experience with environmental epidemiology (n = 91)

On other statements, respondents with less experience were also less likely to (Table 20):

- agree that fruits and vegetables contain natural substances that can cause cancer (Statement 6),
- agree that lifestyle factors pose a much greater risk for cancer than chemicals in the environment (Statement 7),
- disagree that the degree of exposure is usually the largest element of uncertainty in any health risk assessment (Statement 16),
- disagree that a statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis (Statement 17), and
- disagree that statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study (Statement 18).

Table 20 also indicates that respondents with less than 5 years experience in environmental epidemiology were far more likely than those with more than 5 years experience to respond "don't know" to these statements. This was particularly evident for responses to statements on statistical inference in environmental health risk assessment (Statements 17 and 18) from those with less than one year experience. Also note that these views, while more common among more experienced (> 5 years) respondents, were still a majority view among those with lesser experience (0 to 5 years).

Interpreting the results for the environmental engineering professors is more difficult as there was a large discrepancy in subgroup sizes. Of the 413 respondents, 40 (9.7%) indicated between 1 and 5 years experience with environmental engineering and only 1 respondent indicated less than 1 year experience. A large majority (372 respondents - 90.1%) indicated more than 5 years experience with environmental engineering. Table 21 presents selected statements for which years of experience showed large differences in response among environmental engineering professors.

The majority of environmental engineering professors were in disagreement with statements on dose-response and exposure relationships (Statement 1, 2 and 5). However, responses also indicate that respondents with more than 5 years experience were much more likely to "strongly disagree" to these statements than those with less than 5 years experience. Of these statements, Statement 5 showed the greatest difference in disagreement percentage; those with less experience were much less likely to disagree that if a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day (Table 21). However, a much greater percentage of these respondents answered "don't know" to this statement.

Responses to Statement 6 also indicate that while many respondents in all experience categories answered "don't know", environmental engineering professors with 1 to 5 years experience were less likely to agree that fruits and vegetables contain natural substances that can cause cancer. Furthermore, these respondents were much more likely to agree that the land, air and water around us are, in general, more contaminated now than ever before (Statement 9).

Table 21. Experience Differences Among Environmental Engineering Professors to Selected Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
1. A chemical is either safe or dangerous. There is really no in between.	1-5[b]	**52.5**[a]	40.0	5.0	0.0	2.5
	>5[c]	**71.8**	25.0	1.9	1.1	0.3
2. There is no safe level of exposure to a cancer-causing agent.	1-5	**10.0**	55.0	17.5	5.0	12.5
	>5	**27.8**	46.9	11.9	1.9	11.6
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	1-5	**25.0**	**45.0**	10.0	0.0	**20.0**
	>5	**36.7**	**49.3**	4.0	0.3	9.7
6. Fruits and vegetables contain natural substances that can cause cancer.	1-5	5.0	10.0	**37.5**	**12.5**	**35.0**
	>5	2.2	8.1	**49.5**	**15.4**	**24.9**
9. The land, air and water around us are, in general, more contaminated now than ever before.	1-5	0.0	30.0	**50.0**	**15.1**	5.0
	>5	11.3	37.9	**34.9**	**10.4**	5.5
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.	1-5	**2.6**	**46.2**	35.9	0.0	**15.4**
	>5	**14.3**	**50.3**	26.2	0.5	8.6
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.	1-5	**5.0**	**37.5**	12.5	2.5	**42.5**
	>5	**16.3**	**40.8**	26.4	2.5	14.0
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	1-5	**5.0**	**22.5**	37.5	0.0	**35.0**
	>5	**8.1**	**38.6**	29.7	5.7	17.8
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	1-5	**10.0**	**22.5**	27.5	2.5	**37.5**
	>5	**15.0**	**42.3**	21.6	1.9	19.1
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.	1-5	**2.5**	**22.5**	27.5	2.5	**45.0**
	>5	**7.9**	**34.7**	25.2	1.9	30.4

[a] Cell entries are percentages

[b] 1 to 5 years experience with environmental engineering (n = 40)

[c] More than 5 years experience with environmental engineering (n = 372)

Table 21 also indicates that respondents with less experience were much less inclined to:

- disagree that scientific experts are able to make accurate estimates of health risks from chemicals in the environment (Statement 13),
- disagree that a lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen (Statement 15),
- disagree that the degree of exposure is usually the largest element of uncertainty in any health risk assessment (Statement 16),
- disagree that a statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis (Statement 17), and
- disagree that statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study (Statement 18).

Note the very high percentage of respondents with 1 to 5 years experience who responded "don't know" to these statements (Table 21). The differences in response to these statements suggest that experience with environmental engineering may be an important factor for recognizing the limitations and uncertainties associated with environmental health risk assessment.

Age Effects

For the environmental epidemiology sample, differences in response because of age were rather small. Of the 196 respondents, 103 (52.6%) were between ages 25 and 44; another 81 (41.3%) were between 45 and 64 and only 9 were either younger than 25 or greater than 65. The statements with the largest age differences are shown in Table 22.

Table 22. Age Differences Among Environmental Epidemiologists to Selected Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
2. There is no safe level of exposure to a cancer-causing agent.	25-44[b]	**15.7**[a]	**36.3**	24.5	12.7	10.8
	45-64[c]	**16.5**	**49.4**	22.8	7.6	3.8
6. Fruits and vegetables contain natural substances that can cause cancer.	25-44	5.9	13.9	**42.6**	**14.9**	**22.8**
	45-64	6.3	16.3	**52.5**	**15.0**	10.0
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	25-44	**13.7**	**31.4**	26.5	1.0	27.5
	45-64	**13.8**	**46.3**	16.3	0.0	23.8
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.	25-44	**8.8**	**44.1**	36.3	0.0	10.8
	45-64	**16.5**	**46.8**	32.9	1.3	2.5
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.	25-44	**10.0**	**33.0**	28.0	3.0	26.0
	45-64	**16.5**	**38.0**	17.7	0.0	27.8

[a] Cell entries are percentages
[b] Environmental epidemiologists aged 25 to 44 (n = 103)
[c] Environmental epidemiologists aged 45 to 64 (n = 81)

The largest difference between respondents aged 25 to 44 and 45 to 64 was found for Statement 2. Although the majority in both age categories were in disagreement with this statement, respondents aged 25 to 44 were much less likely to disagree that there is no safe level of exposure to a cancer-causing agent (52.0% disagreement versus 65.9% for respondents aged 45 to 64 -Table 22).

For the statements concerning the validity of animal testing for predicting human health effects (Statements 3 and 4), age difference did not appear to have much influence on patterns of response. Statement 10 however, indicated difference in percent disagreement with respondents aged 45 to 64 much more likely to disagree that an untested prescription drug that has been widely used for 20 years is safer than a new drug that has been tested and approved under current regulatory guidelines (Table 22).

On other statements, although differences were not large, respondents aged 25 to 44 were less inclined to (Table 22):

- agree that fruits and vegetables contain natural substances that can cause cancer (Statement 6),
- disagree that scientific experts are able to make accurate estimates of health risks from chemicals in the environment (Statement 13), and
- disagree that a lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen (Statement 15).

Again, though these views were more common among respondents aged 45 to 64, they were still held by the majority of respondents aged 25 to 44. Other statements showed little differences in response among age groups.

Of the 413 environmental engineering professors, 199 (48.2%) respondents were aged 25 to 44 and 189 (45.8%) were aged 45 to 64. There were no respondents aged less than 25 and only 24 respondents were older than 65 years. No consistent

trends in response were found for these age categories. All statements had little differences in response between age categories 25 to 44 and 45 to 64. Some large differences in response were found however for those respondents older than 65 years (Statements 3, 4, 5, 14, 17 and 18). However, these differences are more difficult to interpret as the number of respondents in this category was small (only 24 respondents).

Affiliation Effects

The previous study by Kraus et al. (1992) examined the effects of affiliation for toxicologists working for industry, academia and government. It was found that although these subgroups responded similarly on many questions, toxicologists working for industry saw chemicals as more benign than do their counterparts in academia and government. Furthermore, industrial toxicologists were somewhat more confident in the general validity of animal tests. However, when these tests produced evidence of cancer in animals, the industrial toxicologists became much less confident than academic or government scientists in the ability to extrapolate effects to humans (Kraus et al. 1992). Similar findings were also observed in the study of Canadian toxicologists by Slovic et al. (1995). However, the affiliation bias only appeared once the study produced evidence of cancer in animals.

Because almost all members of the Association of Environmental Engineering Professors were affiliated with an academic institution, affiliation effects were only examined for the group of environmental epidemiologists. Of the 196 respondents from this group, 47 (24.0%) indicated affiliation with a government office; 105 (53.6%) indicated affiliation with an academic institution. Other affiliations for this group included private consulting, industry and public interest organizations. However, because the number of respondents in these last categories was small (only between 6 and 13 respondents), examination of affiliation bias was only done for those affiliated with an academic institution or government agency. Table 23 presents the statements for which affiliation effects were the largest.

Table 23. Affiliation Effects Among Environmental Epidemiologists to Selected Statements

		Strongly Disagree	Disagree	Agree	Strongly Agree	Don't Know
3. The health effects that a laboratory animal experiences from a chemical are a reliable predictor of the human health effects of the chemical.	G[b]	15. 6[a]	46.7	28.9	2.2	6.7
	U[c]	14.7	53.9	26.5	0.0	4.9
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	G	6.4	46.8	**36.2**	**2.1**	8.5
	U	12.4	52.4	**23.8**	**1.9**	9.5
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	G	**14.9**	**51.1**	17.0	0.0	17.0
	U	**11.7**	**38.8**	19.4	0.0	**30.1**
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	G	8.5	36.2	**38.3**	**8.5**	8.5
	U	2.9	27.2	**50.5**	**7.8**	11.7
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	G	**27.7**	**46.8**	17.0	0.0	8.5
	U	**35.3**	**48.0**	13.7	0.0	2.9
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study.	G	**19.6**	**34.8**	34.8	4.3	6.5
	U	**22.3**	**42.7**	29.1	0.0	5.8

[a] Cell entries are percentages
[b] Environmental epidemiologists affiliated with a government office (n = 47)
[c] Environmental epidemiologists affiliated with an academic institution (n = 105)

With regard to the validity of animal testing for predicting human health effects, some affiliation bias among environmental epidemiologists was observed. Although there was not much difference in response between the two subgroups to Statement 3, when the study produced evidence of cancer (Statement 4), environmental epidemiologists in government had more confidence than those in academia in the

ability to extrapolate to humans (38.3% agreement versus 25.7% for academia). Note however that this was a minority view overall. Also, respondents affiliated with a government office were more inclined to change their opinions and percent agreement increased slightly from 31.1% to 38.3% (Table 23).

The majority of respondents in both subgroups were in disagreement with Statement 10. However, respondents affiliated with a government office were much more likely to disagree that an untested prescription drug that has been widely used for 20 years is safer than a new drug that has been tested and approved under current regulatory guidelines (66.0% disagreement versus 50.5% for academia). However, a much greater percentage of respondents affiliated with an academic institution responded "don't know" to this statement. Statement 16 also indicted some difference in response. For this statement, respondents from a government office were less likely to agree that the degree of exposure is usually the largest element of uncertainty in any health risk assessment (58.3% agreement versus 46.8% for government employees).

On statements pertaining to statistical inference (Statements 17 and 18), although the differences are not large, respondents affiliated with a government office were consistently less inclined to (Table 23):

- disagree that a statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis (74.5% disagreement versus 83.8% for academia), and
- disagree that statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiological study (54.4% disagreement versus 65.0% for academia).

CONCLUSIONS

Environmental health risk assessment has developed as a means of examining health risks due to environmental hazards so that they may be better avoided, reduced or otherwise managed. Aside from the numerous difficulties and limitations inherent in the scientific analyses of risks, there is also great difficulty with communicating risk information in a form that is both useful and helpful for gaining perspective on the risks we face and the decisions that must be made.

Although much progress has been made in improving risk communication by understanding the differing risk judgments between the lay public and the scientific community, there is increasing awareness that discrepancies in risk judgments between experts themselves also creates difficulties for communicating risk effectively. Because disagreements often result in conflicting and contradictory messages, divergence of opinion among scientists may be a significant cause of the dissatisfaction and distrust with risk assessment.

In this study, we attempted to reveal some perspectives concerning risk judgments among members of various environmental disciplines by conducting a survey to determine the extent to which these members share similar beliefs and conceptual frameworks concerning several basic assumptions and concepts in environmental health risk assessment. Because risk assessment is inherently multidisciplinary, we chose to survey two major disciplines whose members contribute knowledge to the risk assessment process and to the applications of risk assessment findings to environmental management. The first group was comprised of the participants at an international conference on environmental epidemiology and the second group was comprised of environmental engineering professors. In addition, previous published findings from surveys of toxicologists in both the United States and

Canada provide a comparison for some of our analysis because several of the statements in the questionnaire were replicated from earlier surveys on these groups.

The questionnaire consisted of 20 statements as well as a general demographic section detailing the respondent's personal and educational background, affiliation, and experience. The statements were developed from several underlying assumptions and concepts in environmental health risk assessment. These statements addressed many different issues which encompass environmental health risk assessment such as:

- conceptions of toxicity (dose-response relationships and exposure) and carcinogenic risk assessment,
- trust in animal studies,
- causal inference in epidemiological studies,
- statistical inference in environmental health risk assessment,
- uncertainty and confidence in health risk analyses, and
- objectivity and values in scientific analyses.

Other statements were also included to elicit the respondents' perceptions of various risks in the environment and to determine their attitudes towards natural and synthetic chemicals, and contaminants in the environment.

The findings from this survey indicate that divergent interpretations do exist among respondents for several of the statements provided. Although no sharp distinctions were found between the disciplines themselves (i.e. different disciplinary perspectives), differences in opinion were often apparent within each group. While the qualitative responses were not always useful for explaining differences within and between the groups, they did provide an indication of issues in which a certain level of misunderstanding among some respondents is apparent.

One area in which there was a strong divergence in opinion for all groups was the value of animal studies for predicting human health effects. Although toxicologists

showed the most favorable attitudes toward such studies, all three groups displayed little confidence in the reliability and validity of animal testing for assessing human harm. However, when animal studies found positive evidence of carcinogenicity, many environmental engineering professors became more confident while many toxicologists became less confident that such tests were reliable indicators for cancer in humans.

These responses as well as others on dose-response and exposure relationships indicate that the mention of cancer provokes a different response pattern. While this is difficult to interpret, it suggests that a better appreciation of the limitations and additional uncertainties of animal cancer testing as well as the mechanisms of carcinogenesis itself may be needed.

Causal inference and statistical inference in environmental health risk assessment were other issues which seemed to offer opportunities for increased awareness. Although divergence in opinion was sometimes not great for these statements, the responses suggest that some respondents do not fully appreciate the role of epidemiological studies in establishing causation. Furthermore, distinguishing statistical inference from causal inference also appeared to present difficulties for some respondents, particularly within the group of environmental engineering professors.

The responses to statements evaluating uncertainty and confidence in health risk analyses also reflect the potential need for improved understanding of the substantial uncertainties and limitations in environmental health risk assessment among the members of the various disciplinary groups. Both the environmental engineering professors and the environmental epidemiology groups were divided in their opinions regarding the accuracy of expert risk estimates (Statement 13) and whether a lifetime cancer risk as low as one chance in a million can be known (Statement 15). These results suggest that some respondents may have unrealistic expectations of what risk

assessment can accomplish and that an awareness of the uncertainties and limitations in risk estimation, particularly cancer risk estimation, may be lacking.

Analysis of consistency in individual response also supports these findings. Inconsistent individual responses were found on several statements suggesting that there are likely misunderstandings on some of these issues. Large inconsistencies were found for the environmental epidemiologists and environmental engineering professors particularly for statements concerning cancer versus any other adverse effect, validity of animal studies, and confidence in risk estimates.

Analysis of demographic information such as gender, experience, age or affiliation also provided further insight into the responses given to these statements. Within the sample of environmental epidemiologists and environmental engineering professors, differences in opinion between men and women were not great. Except for statements on dose-response and exposure relationships where women were much less likely to "strongly disagree" than men, there were no consistent patterns found between male and female respondents. However, the differences in responses for several statements between those respondents with less than 5 years experience and those with more than 5 years experience in both groups suggest that experience may be an important factor in recognizing dose-response relationships as well as the limitations and uncertainties associated with environmental health risk assessment.

For the environmental epidemiologists and environmental engineering professors, difference in response because of age were rather small. However, results suggest that some affiliation bias among environmental epidemiologists was observed. For this group, environmental epidemiologists affiliated with a government office had more confidence than those in academia in the ability to extrapolate animal results to humans and also were much more likely to disagree that an untested prescription drug

that has been widely used for 20 years is safer than a new drug that has been tested and approved under current regulatory guidelines. These respondents were also less likely to agree that the degree of exposure is usually the largest element of uncertainty in any health risk assessment.

In light of the apparent difficulties in evaluating and communicating risk, the findings from this study provide a possible basis for which risk judgments can be more informed. Because many environmental scientists and engineers become involved in studying risk issues and/or conveying risk information to others, they should be capable of providing properly qualified and educated opinions concerning the assumptions and concepts in environmental health risk assessment. Furthermore, because many of the issues presented offer no hard evidence to rely on, it is necessary that these experts evaluate their own knowledge and understanding of these concepts and be fully aware of the strengths and limitations of the methods used for risk assessment.

REFERENCES

Ames, B.N and L. Swirsky Gold. 1990a. Too many rodent carcinogens: Mitogenesis increases mutagenesis. *Science*. 249, 970-971.

Ames, B.N and L. Swirsky Gold. 1990b. Chemical carcinogenesis: Too many rodent carcinogens. *Proceedings of the National Academy of Sciences, USA*. 87, 7772-7776

Ames, B.N and L. Swirsky Gold. 1993. Environmental pollution and cancer: Some misconceptions. In: K.R. Foster, D.E. Bernstein and P.W. Huber (Eds.). *Phantom Risk: Scientific Inference and the Law*. Massachusetts: MIT Press. Chapter 7.

Ames, B.N., Profet, M. and L. Swirsky Gold. 1990a. Dietary pesticides (99.99% all natural). *Proceedings of the National Academy of Sciences, USA*. 87, 7777-7781.

Ames, B.N., Profet, M. and L. Swirsky Gold. 1990b. Nature's chemicals and synthetic chemicals: Comparative toxicology. *Proceedings of the National Academy of Sciences, USA*. 87, 7782-7786.

Ames, B. N., Swirsky Gold, L. and W.C. Willett. 1995. The causes and prevention of cancer. *Proceedings of the National Academy of Sciences, USA*. 92, 5258-5265.

ATSDR. 1990. Agency for Toxic Substances and Disease Registry.

Beaglehole, R., Bonita, R. and T. Kjellstrom. 1993. *Basic Epidemiology*. Geneva: World Health Organization.

Brzuzy, L.P. and R.A. Hites. 1996. Global mass balance for polychlorinated dibenzo-p-dioxins and dibenzofurans. *Environmental Science and Technology*. 30, 1797-1804.

Calabrese, E.J. 1988. Animal extrapolation and the challenge of human interindividual variation. In: C.C. Travis (Ed.) *Carcinogenic Risk Assessment*. New York: Plenum Press. pp. 115-122.

Carson, R. 1962. *Silent Spring*. New York: Houghton Mifflin.

Cohn, V. 1989. *News and Numbers: A Guide to Reporting Statistical Claims and Controversies in Health and Other Fields*. Ames: Iowa State University Press.

Davis, D.L. 1985. The shotgun wedding of science and law: risk assessment and judicial review. *Columbia Journal of Environmental Law*. 10, 67.

Doll, R. and R. Peto. 1981. The causes of cancer: Quantitative estimates of avoidable risks of cancer in the United States today. *Journal of the National Cancer Institute*. 66, 1193-1308.

Epstein, S.S. 1979. *The Politics of Cancer*. Revised and expanded edition. New York: Anchor Press.

Finkel, A. 1989. Is risk assessment really too conservative?: Revising the revisionists. *Columbia Journal of Environmental Law*. 14, 427-467

Finkel, A. 1990. *Confronting Uncertainty in Risk Management*. Washington D.C.: Center for Risk Management Resources for the Future.

Fisher, A. 1991. Risk communication challenges. *Risk Analysis*. 11, 173-179.

Foster, K.R., Bernstein, D.E. and P.W. Huber. 1993. A scientific perspective. *Phantom Risk - Scientific Inference and the Law*. Cambridge, Mass.: MIT Press. Chapter 1.

Freedman, D.A. and H. Zeisel. 1988. From mouse-to-man: The quantitative assessment of cancer risks. *Statistical Science*. 3, 6

Freudenburg, W.R. 1988. Perceived risk, real risk: Social science and the art of probabilistic risk assessment. *Science*. 242, 44-49.

Gallo, M.A. and J. Doull. 1991. History and scope of toxicology. In: M.O Amdur, J. Doull and C.D. Klaassen (Eds.) *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 4th Edition. New York: Pergamon Press. Chapter 1.

Gots, R.E. 1993. *Toxic Risks: Science, Regulation, and Perception*. Boca Raton: Lewis Publishers.

Government of Canada. 1991a. *The State of Canada's Environment*. Ottawa, Canada.

Government of Canada. 1991b. *Toxic Chemicals in the Great Lakes and Associated Effects*. Synopsis. Toronto: Environment Canada; Burlington, Ontario: Department of Fisheries and Oceans; and Ottawa: Health and Welfare Canada.

Graham, J.D. and L. Rhomberg. 1996. How risks are identified and assessed. In: H. Kunreuther and P. Slovic (Eds.) Challenges in Risk Assessment and Risk Management. *The Annals of the American Academy of Political and Social Science*. 545, 15-24.

Grandjean, P. and J.B. Neilson. 1996. Laegers og laegestuderendes opfattelse af miljorisici. *Ugeskr Laeger*. 158, 5291-5295.

Harrison, K. and G. Hoberg. 1994. *Risk, Science, and Politics: Regulating toxic substances in Canada and the United States*. Montreal and Kingston: McGill-Queen's University Press.

Hart, R.W. and L. Fishbein. 1985. Interspecies extrapolation of drug and genetic toxicity data. In: D.B. Clayson, D. Krewski and I. Munro (Eds.) *Toxicological Risk Assessment*. Volume I. Boca Raton, Florida: CRC Press, Inc. Chapter 1.

Higginson, J. 1968. Distribution of different patterns of cancer *Israel Journal of Medical Sciences*. 4, 460.

Hill, A.B. 1965. The environment and disease: Association or causation? *Proceedings of the Royal Society of Medicine*. 58, 295-300.

Hrudey, S.E. 1995. Major concerns with quantitative cancer risk assessment for environmental risk management: North American experience. *Transactions of the Institution of Chemical Engineers*. 73(B), S24-S27.

Hrudey, S. E. 1996. A critical review of current issues in risk assessment and risk management. *Technical Risk Issues in Business and Regulatory Decision-Making.* D.D.B. Belluz, Institute for Risk Research: 3-17.

Hrudey, S.E. and D. Krewski. 1995. Is there a safe level of exposure to a carcinogen? *Environmental Science and Technology.* 29(8), 370A-375A.

Hrudey, S.E. and T. Lambert. 1994. Mythology cannot expose myths in risk communication. *Journal of Environmental Health.* 57 (2), 19-20.

Hrudey, S.E. and W. Chen. 1996. Course notes for Civil Engineering 626: Environmental Health Risk Assessment, University of Alberta.

Jardine, C.G. and S.E. Hrudey. 1997. Mixed messages in risk communication. *Risk Analysis.* In Press.

Kaplan, S. and B.J. Garrick. 1981. On the quantitative definition of risk. *Risk Analysis.* 1, 11-27

Klaassen, C.D. (Ed.). 1996. *Casarett and Doull's Toxicology: The Basic Science of Poisons.* 5th Edition. New York: McGraw-Hill.

Klaassen, C.D. and W.C. Kershaw. 1994. Environmental epidemiology for chemists. In: W.M. Draper (Ed.) *Environmental Epidemiology.* Washington, D.C.: American Chemical Society. 39-51.

Koshland Jr., D.E. 1994. Molecule of the year: The DNA repair enzyme. *Science.* 266, 1925.

Kraus, N., Malmfors, T. and P. Slovic. 1992. Intuitive toxicology: expert and lay judgments of chemical risks. *Risk Analysis.* 12, 215-232.

Krewski, D., Slovic, P., Bartlett, S., Flynn, J. and C.K. Mertz. 1995. Health risk perception in Canada II: Worldviews, attitudes and opinions. *Human and Ecological Risk Assessment.* 1(3), 231-248.

Maldonado, G. 1994. Interpreting epidemiological studies. In: W.M. Draper (Ed.) *Environmental Epidemiology.* Washington, D.C.: American Chemical Society. 29-38.

Mandl, L. and J. Lathrop. 1983. "LEG risk assessments: Experts disagree." In H. Kunreuther (Ed.) *Risk Analysis and Decision Processes: The Siting of Liquefied Energy Gas Facilities in Four Countries.* Berlin: Springer-Verlag.

Marx, J. 1990. Animal carcinogen testing challenged. *Science.* 250, 743-745.

Maynard, R.L., Cameron, K.M., Fielder, R., McDonald, A. and A. Wadge. 1995. Setting air quality standards for carcinogens: an alternative to mathematical quantitative risk assessment - discussion paper. *Human & Experimental Toxicology.* 14, 175-186.

National Academy of Sciences. 1983. *Risk Assessment in the Federal Government: Managing the Process.* Washington D.C.: National Academy Press.

National Cancer Institute of Canada. 1996. *Canadian Cancer Statistics 1996*, Toronto, Canada.

NRC (National Research Council). 1989a. *Improving Risk Communictaion.* Washington, D.C.: National Academy Press.

NRC (National Research Council). 1989b. *Diet and Health.* National Academy of Sciences.

NRC (National Research Council). 1994. *Science and Judgment in Risk Assessment..* Washington, D.C.: National Academy Press.

NRC (National Research Council). 1996. *Understanding Risk.: Informing Decisions in a Democratic Society.* Washington, D.C.: National Academy Press.

Noble, D.G. 1990. Contaminants in Canadian Seabirds. SOE Report No. 90-2. Ottawa: Environment Canada, Canadian Wildlife Service and State of the Environment Reporting.

Otway, H. 1992. Public wisdom, expert fallibility: Toward a contextual theory of risk. In: S. Krimsky and D. Golding (Eds.). *Social Theories of Risk.* Westport: Praeger Publishers.

Pidgeon, N., Hood, C., Jones, D., Turner, B. and R. Gibson. 1992. Risk Perception. In: The Royal Society (Eds.) *Risk: Analysis, Perception and Management.* pp. 89-134.

Rothman, K.J. 1988. Inferring causal connection - Habit, faith or logic? In: K.J. Rothman (Ed.) *Causal Inference.* Chestnut Hill, Mass.: Epidemiology Resources. 3-12.

Rodricks, J.V. 1992. *Calculated Risks: The Toxicity and Human Health Risks of Chemicals in Our Environment.* Cambridge, U.K.: Cambridge University Press.

SGACSH (The Surgeon General's Advisory Committee on Smoking and Health). 1964. Rockville, MD: U.S. Public Health Service.

Slovic, P. 1997. Trust, Emotion, Sex, Politics, and Science: Surveying the risk-assessment battlefield. In M. Bazerman, D. Messick, A. Tenbrunsel, and K. Wade-Benzoni (Eds.). *Psychological Perspectives to Environment and Ethics in Management.* San Francisco: Jossey-Bass.

Slovic, P., Malmfors, T., Krewski, D., Mertz, C.K., Neil, N. and S. Bartlett. 1995. Intuitive toxicology. II. Expert and lay judgments of chemical risks in Canada. *Risk Analysis.* 15, 661-675.

Sowers, M. 1994. Design methods for occupational and environmental epidemiology. In: W.M. Draper (Ed.) *Environmental Epidemiology.* Washington, D.C.: American Chemical Society. 21-27.

Spivey, G.H. 1994. The epidemiological method. In: W.M. Draper (Ed.) *Environmental Epidemiology*. Washington, D.C.: American Chemical Society. 9-19.

Stehbens, W.E. 1992. Causality in medical science with particular reference to heart disease and atherosclerosis. *Perspectives in Biology and Medicine*. 36, 97-119.

Susser, M. 1991. What is a cause and how do we know one? A grammar for pragmatic epidemiology. *American Journal of Epidemiology*. 7, 635-648.

Taubes, G. 1995. Epidemiology faces its limits. *Science*. 269, 164-169.

U.S. EPA (United States Environmental Protection Agency). 1986. Guidelines for carcinogenic risk assessment. *Federal Register*. 51(185): 33992-34003.

Walter, S.D. 1995. Methods of reporting statistical results from medical research studies. *American Journal of Epidemiology*. 141, 896-906.

Whittemore, A.S. 1983. Facts and values in risk analysis for environmental toxicants. *Risk Analysis*. 3, 23-34.

WHO (World Health Organization). 1964. *Prevention of Cancer*. Technical Report Series 276. Geneva: WHO.

Zaridze, D. and R. Peto (Eds.). 1986. Tobacco: A Major International Health Hazard. IARC Scientific Publications No. 74. Lyon: International Agency for Research on Cancer.

APPENDIX A

Questionnaire for Environmental Epidemiologists

Demographic Information:

Please mark the boxes which apply to you; when required please PRINT or write legibly.

AGE: ☐ ≤ 24 ☐ 25 - 34 ☐ 35 - 44 ☐ 45 - 54 ☐ 55 - 64 ☐ ≥ 65

GENDER: ☐ Male ☐ Female

COUNTRY OR REGION OF RESIDENCE: _____

PLEASE INDICATE ALL COMPLETED DEGREES/DIPLOMAS, SPECIALIZATION(S), UNIVERSITY/INSTITUTION(S) AND COUNTRY, AND THE YEAR IN WHICH THE DEGREE/DIPLOMA WAS OBTAINED.

Degree/ Diploma	Major/ Specialization(s)	University/Institution, Country	Year Obtained
e.g. B.Sc.	*Biology*	*University of Alberta, Canada*	*1983*

WHO IS YOUR PRIMARY EMPLOYER?

- ☐ Government Office/Ministry
- ☐ Consulting (private sector, nongovernment)
- ☐ Public Interest and/or Non-profit
- ☐ Academic Institution
- ☐ Industry (private sector)
- ☐ Other (please specify):_____

WHAT IS (ARE) YOUR CURRENT POSITION(S) / TITLE(S)?

Please state in full (without identifying the organization):

_____

_____

_____

PLEASE INDICATE YOUR EXPERIENCE WITH ENVIRONMENTAL EPIDEMIOLOGY:

☐ none* ☐ Limited (< 1 year) ☐ Significant (1 - 5 years) ☐ Extensive (> 5 years)

* We are still interested in your responses to the following questions.

Questionnaire:

Please mark the box that most closely reflects your view concerning the following statements. These are given as absolute statements to provoke a response; your response should indicate your level of agreement with these statements.

For the purposes of this study, the term 'chemical' refers to all chemical elements and compounds, including pesticides, food additives, industrial chemicals, household cleaning agents, prescription and non-prescription drugs, etc.

You are encouraged to include any comments and/or an elaboration of the reasons for your response to each question.

	Scale of Agreement				
	Strongly Disagree	**Disagree**	**Don't Know**	**Agree**	**Strongly Agree**
1. A chemical is either safe or dangerous. There is really no in between.	☐	☐	☐	☐	☐
Comments:					
2. There is no safe level of exposure to a cancer-causing agent.	☐	☐	☐	☐	☐
Comments:					
3. The health effects that a laboratory animal experiences from a chemical is a reliable predictor of the human health effects of the chemical.	☐	☐	☐	☐	☐
Comments:					
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	☐	☐	☐	☐	☐
Comments:					
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	☐	☐	☐	☐	☐
Comments:					
6. Fruits and vegetables contain natural substances that can cause cancer.	☐	☐	☐	☐	☐
Comments:					
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	☐	☐	☐	☐	☐
Comments:					
8 Natural chemicals are not as harmful as man-made chemicals.	☐	☐	☐	☐	☐
Comments:					
9 The land, air and water around us are, in general, more contaminated now than ever before.	☐	☐	☐	☐	☐
Comments					

	Scale of Agreement				
	Strongly Disagree	Disagree	Don't Know	Agree	Strongly Agree
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	☐	☐	☐	☐	☐
Comments:					
11. Environmental epidemiology is an applied science (i.e., not a basic science).	☐	☐	☐	☐	☐
Comments:					
12. Applied sciences are rarely value-free or value-neutral.	☐	☐	☐	☐	☐
Comments:					
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.	☐	☐	☐	☐	☐
Comments:					
14. A single epidemiology study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.	☐	☐	☐	☐	☐
Comments:					
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.	☐	☐	☐	☐	☐
Comments:					
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	☐	☐	☐	☐	☐
Comments:					
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	☐	☐	☐	☐	☐
Comments:					
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiology study.	☐	☐	☐	☐	☐
Comments:					
19. Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.	☐	☐	☐	☐	☐
Comments					

20.a) Where do you obtain <u>most</u> of your information about health risks from chemicals and other health hazards (list in order of your usage in the space provided below)?

b) In general, how <u>reliable</u> do you think the information from each source is?

	Reliability		
Information Sources	Very Reliable	Somewhat Reliable	Not Very Reliable
1._____	☐	☐	☐
2._____	☐	☐	☐
3._____	☐	☐	☐
4._____	☐	☐	☐
5._____	☐	☐	☐

ADDITIONAL COMMENTS (please write legibly):

Thank you for taking the time to assist in this study.
Please return this questionnaire in the postage paid, pre-addressed envelope provided.
If you would like to receive a copy of our research report and/or participate in a follow-up survey, please return the attached reply card separately.

APPENDIX B

Questionnaire for Environmental Engineering Professors

Demographic Information:

Please mark the boxes which apply to you; when required please PRINT or write legibly.

AGE: ☐ ≤ 24 ☐ 25 - 34 ☐ 35 - 44 ☐ 45 - 54 ☐ 55 - 64 ☐ ≥ 65

GENDER: ☐ Male ☐ Female

COUNTRY OR REGION OF RESIDENCE: _____

PLEASE INDICATE ALL COMPLETED DEGREES/DIPLOMAS, SPECIALIZATION(S), UNIVERSITY/INSTITUTION(S) AND COUNTRY, AND THE YEAR IN WHICH THE DEGREE/DIPLOMA WAS OBTAINED.

Degree/ Diploma	Major/ Specialization(s)	University/Institution, Country	Year Obtained
e.g. B.Sc.	*Biology*	*University of Alberta, Canada*	*1983*

WHO IS YOUR PRIMARY EMPLOYER?

- ☐ Government Office/Ministry
- ☐ Consulting (private sector, nongovernment)
- ☐ Public Interest and/or Non-profit
- ☐ Academic Institution
- ☐ Industry (private sector)
- ☐ Other (please specify):_____

WHAT IS (ARE) YOUR CURRENT POSITION(S) / TITLE(S)?

Please state in full (without identifying the organization):

_____

_____

_____

PLEASE INDICATE YOUR EXPERIENCE WITH ENVIRONMENTAL ENGINEERING:

☐ none* ☐ Limited (< 1 year) ☐ Significant (1 - 5 years) ☐ Extensive (> 5 years)

* We are still interested in your responses to the following questions.

Questionnaire:

Please mark the box that most closely reflects your view concerning the following statements. These are given as absolute statements to provoke a response; your response should indicate your level of agreement with these statements.

For the purposes of this study, the term 'chemical' refers to all chemical elements and compounds, including pesticides, food additives, industrial chemicals, household cleaning agents, prescription and non-prescription drugs, etc.

You are encouraged to include any comments and/or an elaboration of the reasons for your response to each question.

	Scale of Agreement				
	Strongly Disagree	**Disagree**	**Don't Know**	**Agree**	**Strongly Agree**
1. A chemical is either safe or dangerous. There is really no in between.	☐	☐	☐	☐	☐
Comments:					
2. There is no safe level of exposure to a cancer-causing agent.	☐	☐	☐	☐	☐
Comments:					
3. The health effects that a laboratory animal experiences from a chemical is a reliable predictor of the human health effects of the chemical.	☐	☐	☐	☐	☐
Comments:					
4. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	☐	☐	☐	☐	☐
Comments:					
5. If a person is exposed to a chemical that can cause cancer then that person will probably get cancer some day.	☐	☐	☐	☐	☐
Comments:					
6. Fruits and vegetables contain natural substances that can cause cancer.	☐	☐	☐	☐	☐
Comments:					
7. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	☐	☐	☐	☐	☐
Comments:					
8. Natural chemicals are not as harmful as man-made chemicals.	☐	☐	☐	☐	☐
Comments:					
9. The land, air and water around us are, in general, more contaminated now than ever before.	☐	☐	☐	☐	☐
Comments:					

	Scale of Agreement				
	Strongly Disagree	Disagree	Don't Know	Agree	Strongly Agree
10. A prescription drug that has not been formally tested but has been widely used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present regulatory guidelines.	☐	☐	☐	☐	☐
Comments:					
11. Environmental science is an applied science (i.e., not a basic science).	☐	☐	☐	☐	☐
Comments:					
12. Applied sciences are rarely value-free or value-neutral.	☐	☐	☐	☐	☐
Comments:					
13. Scientific experts are able to make accurate estimates of health risks from chemicals in the environment.	☐	☐	☐	☐	☐
Comments:					
14. A single epidemiology study can be sufficient to establish that a contaminant in the environment causes a specific human health effect.	☐	☐	☐	☐	☐
Comments:					
15. A lifetime cancer risk as low as one chance in a million can be known for a given level of exposure to a carcinogen.	☐	☐	☐	☐	☐
Comments:					
16. The degree of exposure to an environmental contaminant is usually the largest element of uncertainty in any health risk assessment.	☐	☐	☐	☐	☐
Comments:					
17. A statistically significant association (at the 1% level) between an environmental contaminant and a health effect can confirm a causal hypothesis.	☐	☐	☐	☐	☐
Comments:					
18. Statistical confidence intervals accurately represent most of the uncertainty associated with the findings from a rigorous epidemiology study.	☐	☐	☐	☐	☐
Comments:					
19. Residents of a small community (30,000 people) observed that malformed children had been born there during each of the past few years. The town is in a region where agricultural pesticides have been used during the past decade. It is very likely that these pesticides were the cause of the malformations.	☐	☐	☐	☐	☐
Comments:					

20.a) Where do you obtain most of your information about health risks from chemicals and other health hazards (list in order of your usage in the space provided below)?

b) In general, how reliable do you think the information from each source is?

	Reliability		
Information Sources	Very Reliable	Somewhat Reliable	Not Very Reliable
1._____	☐	☐	☐
2._____	☐	☐	☐
3._____	☐	☐	☐
4._____	☐	☐	☐
5._____	☐	☐	☐

ADDITIONAL COMMENTS (please write legibly):

Thank you for taking the time to assist in this study.
Please return this questionnaire in the postage paid, pre-addressed envelope provided.
If you would like to receive a copy of our research report and/or participate in a follow-up survey, please return the enclosed reply card separately.

APPENDIX C

Request for Ethical Review and Approval

June 25, 1996

Our file #2061

Dr. S. Hrudey
Department of Public Health Sciences
13-102 CSB

Dear Dr. Hrudey:

Re: Expert judgements of environmental risk

Thank you for submitting the above study to the Research Ethics Board. Dr. Morrish has approved the study on behalf of the REB and your approval form is enclosed. This is an interesting proposal and we will be interested to hear the results. I hope the conference goes well and that you get a good response to the questionnaire.

Next year, a few weeks prior to the expiration of your approval, a Progress Report will be sent to you for completion. If there have been no major changes in the protocol, your approval will be renewed for another year. All protocols may be subject to re-evaluation after three years.

It is a policy of the Faculty of Medicine that signed copies of the consent form must be retained, and be available on request. They should be kept for the duration of the project and for a full calendar year following its completion.

Yours sincerely,

Judith R. Abbott (Ms.)
Administrative Assistant (Research)

cc: Dr. D.W. Morrish, Chair, Research Ethics Board

/ja
enc.

RESEARCH ETHICS BOARD

ETHICS APPROVAL FORM

Date: June, 1996

Name(s) of Principal Investigator(s): Dr. S. Hrudey

Department: Department of Public Health Sciences

Title: Expert judgements of environmental risk

The Research Ethics Board has reviewed the protocol involved in this project which has been found to be acceptable within the limitations of human experimentation. The REB has also reviewed and approved the patient information materials and consent form.

Specific Comments:

Signed - Chairman of Research Ethics Board

for the Faculty of Medicine
University of Alberta

This approval is valid for one year.

Issue #2061

REQUEST FOR ETHICAL REVIEW

1. Investigator(s)*:	2. U of A Department:	3. Phone No.:
Dr. S. Hrudey	**Public Health Sciences**	**492-6807**

4. Institution(s) at which the research will be carried out:

U of A Hospital ( ) Cross Cancer ( ) Royal Alex ( ) U of A (**X**)
Other (specify):

5. Title of Project:

Expert Judgments of Environmental Risk

6. Purpose and objectives of project - <u>please state your hypothesis</u>.

Different assumptions, conceptions and values are known to influence views of chemical risks. In this study, participants at the 8th Annual Conference of the International Society for Environmental Epidemiology as well as members of the Association of Environmental Engineering Professors will be surveyed regarding various aspects of environmental risk management, exposure and safety. The purpose of this study is to determine the attitudes, beliefs and perceptions of chemical risks of these expert disciplines. The survey results will explore the similarities and differences within the expert community and will serve as a means to compare the concerns and perceptions of chemical risks with those from other disciplines and previous surveys of the lay public (Jardine et al 1995).

Signature of Principal Investigator*

_____ Date: Jan 10/96

I have read this application, believe that the benefits of the proposed research outweigh the risks to patients or normal subjects, and support the implementation of this project.

Signature of U of A Department Chair

_____ Date: Jan 13/96

*Please read #5 of Notes on Page 7 of this document

7. Which of the following best describe(s) the type of investigation proposed? Check more than one, if appropriate.

( ) clinical trial
( ) multicentre trial
( ) pilot study
( ) drug study
( ) sequel to previously approved project
( ) first application in humans
(**X**) other (specify):
Questionnaire Survey

8. If a drug study, which of the following best describes it? Check more than one, if appropriate.
Not Applicable

( ) drug being using for currently approved application
( ) approved drug but being used in a non-approved application
( ) experimental, non-approved drug

9. Will funding be sought from

( ) a granting agency; if so, please name the agency(ies):
( ) a drug company; if so, please specify:

The research is funded by the Tri-Council Secretariat (MRC, NSERC, SSHERC) support to the Eco-Research Chair in Environmental Risk Management.

The notion of faculty members entering into contractual agreements or receiving consultation fees from the private sector is not only acceptable, it is encouraged by both the University and the provincial governments. There may be occasions, however, when conflicts arise between an investigator's university responsibilities and his/her arrangements with the private sector. It is important from an ethical standpoint that the Research Ethics Board Chair be aware of the nature of any such arrangements, in order to ensure that there are no conflicts which could be perceived to have the potential to adversely affect subjects enrolled in research projects. If there is any doubt as to the possibility of there being a conflict of interest the onus is on the investigator to discuss the situation with the Committee Chair.

10. Are any of the investigators involved in this study receiving any direct personal remuneration or other personal or family financial benefits (either direct or indirect) for taking part in this investigation?

( ) yes (**X**) no

"Other financial benefits" may include contractual agreements, stock or share holdings or future options with the sponsoring company, computing equipment, travel benefits, etc. If the answer is 'yes', please append a letter detailing these activities to the Research Ethics Board Chair. This information will not be circulated to the full committee without prior consultation with the investigator(s) concerned.

Finders Fees: The Research Ethics board considers the payment of any fee directly to an individual for soliciting the enrollment of subjects into a clinical trial to be unacceptable and such payments will not be allowed. When a private sector company provides fees to the investigator which are based on the numbers of subjects enrolled in a study then it is acceptable for the investigator to use these funds to set up a research trust account with the approval of the department chair (e.g. research or educational fund, as appropriate). Such remuneration must not be tied to the enrollment of any individual(s) into the study.

11. Summarize briefly the research which has led up to this trial. Include information on relevant animal studies, human trials (to date), etc.

It appears that no studies have been conducted to examine the attitudes, beliefs and perceptions of the expert groups being surveyed. Previous related studies include surveys given to toxicologists and the lay public about basic toxicological concepts, assumptions and interpretations to determine discrepancies between expert and lay views of chemical risks (Kraus et al 1992; Slovic et al 1995; Krewski 1995; Jardine et al 1995).

DESCRIPTION OF POPULATION - For ethical purposes, your numbers must be sufficient for the study to be considered statistically valid.

12. How many subjects will be enrolled? **Approximately 1000**

    How many normal subjects? **Not Applicable**

13. Inclusion criteria for patients/subjects:

Participants in the survey questionnaire will include the registrants at the 8th Annual Conference of the International Society for Environmental Epidemiology, August 17 - 21, 1996 at Edmonton, Alberta and the members of the Association of Environmental Engineering Professors.

14. Exclusion criteria for patients/subjects:

Not Applicable

15. How are the subjects being recruited? (If initial contact is by letter or if a recruitment notice is to be posted, attach a copy.)

Subjects will be contacted by letter. A sample contact letter is attached and will be included at the beginning of the questionnaire.

METHODOLOGY AND PROCEDURES

16. Summary of methodology and procedures. Include details of any specific manipulations: type, quantity and route of administration of drugs or radiation; operations; tests; use of medical devices that are prototypic or altered from those in clinical use; interviews or questionnaires; methods of data evaluation. (If insufficient space, please append additional pages.)

Questionnaire will be included in the registration package for the 8th Annual Conference of the International Society for Environmental Epidemiology, August 17 - 21, 1996 at Edmonton, Alberta. As well, a mail survey will be conducted to include members of the Association of Environmental Engineering Professors in the analysis.

Questionnaires are to be completed by the subjects and returned. Responses will be extracted from questionnaires and analyzed. Follow-up studies may be conducted if deemed valuable.

17. What procedures or treatments in this project are dictated by the protocol and which ones are ADDITIONAL to those required for standard patient care?

Not Applicable

IF THE PROCEDURES DESCRIBED ABOVE ARE LIMITED TO ANY OF THE FOLLOWING PLEASE CHECK THE APPROPRIATE BOX. IF THIS IS THE CASE, ONLY THE ORIGINAL PROTOCOL AND ONE COPY NEED BE SUBMITTED.

( ) withdrawal of blood or collection of urine
(**X**) **examination** of medical records and/or recorded data/**questionnaire only**
( ) use of specimens acquired non-invasively or of materials normally discarded
( ) the project is a modification of a previously approved clinical protocol (include title and date of approval)

* The decision as to whether or not submissions falling into one of these categories must go before the full Board rests with the Chair. N.B.: All studies involving paediatric patients go before the full Board.

18. What are the benefits of the proposed research? Include possible benefits to the patient/normal subject, and to medical knowledge in general.

This research will provide useful insights into the attitudes and perceptions of environmental epidemiologists and environmental engineering professors. It will also serve as a means to compare concerns and perceptions of chemical risks with those from other disciplines and areas such as toxicology and the lay public. These insights will be used to develop recommendations for development of consensus principles for education and research purposes in the field of environmental risk.

19. What adverse effects may result from the experimental treatment? What discomfort or incapacity are the subjects likely to endure as a result of the experimental procedures? Include risks, discomfort, incapacity and any reported side-effects of the procedure or drug.

No adverse effects are expected. Investigation consists of a written response to a questionnaire by volunteer subjects.

20. Who will have the code of a blinded study? Under what conditions will the code be broken, and what provisions are made for this?

Not Applicable

21. (a) If monetary compensation is to be offered the subjects, provide details of amounts.

Not Applicable

(b) Indicate if payment is to compensate for
(i) expenses incurred
(ii) lost wages
(iii) other (specify):

Not Applicable

22. How much time will a patient/normal subject have to dedicate to the project beyond that needed for a standard treatment?

Completion of survey questionnaire will require approximately 15-20 minutes.

23. Does this study include procedures which involve the administration of radioisotopes or additional radiation?

No

If 'yes', please append the appropriate Radioisotope and Radiation Project Approval. Ethics approval may be withheld if this is not done.

24. (a) What provisions are made for maintaining the confidentiality of data and patients' identities?

1. All information will be treated in confidence.
2. Subjects' names will not be recorded on the questionnaire.
3. For the mail out survey, identification numbers on questionnaires will be used only for the purpose of follow-up contact. However, anonymity of those responding will be ensured by completely blinding the investigator from the identification numbers. This will be achieved by using a third party to collect, record and remove the numbers prior to the investigator examining the questionnaires.

(b) Name any agencies outside the university (if any) who will have access to any data which identify individuals

Not Applicable

INFORMED CONSENT (See "Guidelines for Informed Consent" attached).
A copy of the proposed Information Sheet/Consent Form must be attached to this form for submission to the Research Ethics Board.

25. (a) Will the group of subjects have any problems giving informed consent on their own behalf? Consider physical or mental condition, age, language, and other barriers.

No problems with informed consent are expected.

(b) If the subjects are not competent to give fully informed consent, who will consent on their behalf?

Not Applicable

Intuitive Toxicology: Expert and Lay Judgments of Chemical Risks

Risk Analysis, Vol. 12, No. 2, 1992

Nancy Kraus,[1] Torbjörn Malmfors,[2] and Paul Slovic[1]

Received March 1, 1991; revised August 26, 1991

Human beings have always been intuitive toxicologists, relying on their senses of sight, taste, and smell to detect harmful or unsafe food, water, and air. As we have come to recognize that our senses are not adequate to assess the dangers inherent in exposure to a chemical substance, we have created the sciences of toxicology and risk assessment to perform this function. Yet despite this great effort to overcome the limitations of intuitive toxicology, it has become evident that even our best scientific methods still depend heavily on extrapolations and judgments in order to infer human health risks from animal data. Many observers have acknowledged the inherent subjectivity in the assessment of chemical risks and have indicated a need to examine the subjective or intuitive elements of expert and lay risk judgments. We have begun such an examination by surveying members of the Society of Toxicology and the lay public about basic toxicological concepts, assumptions, and interpretations. Our results demonstrate large differences between toxicologists and laypeople, as well as differences between toxicologists working in industry, academia, and government. In addition, we find that toxicologists are sharply divided in their opinions about the ability to predict a chemical's effect on human health on the basis of animal studies. We argue that these results place the problems of risk communication in a new light. Although the survey identifies misconceptions that experts should clarify for the public, it also suggests that controversies over chemical risks may be fueled as much by limitations of the science of risk assessment and disagreements among experts as by public misconceptions.

KEY WORDS: Intuitive toxicology; risk perception; risk assessment; chemical risks; expert judgment.

Intuitive Toxicology. II. Expert and Lay Judgments of Chemical Risks in Canada

Risk Analysis, Vol. 12, No. 2, 1992

Paul Slovic,[1,5] Torbjörn Malmfors,[2] Daniel Krewski,[3] C. K. Mertz,[1] Nancy Neil,[4] and Sheryl Bartlett[3]

Received December 6, 1994; revised July 27, 1995

This study is a replication and extension in Canada of a previous study in the United States in which toxicologists and members of the public were surveyed to determine their attitudes, beliefs, and perceptions regarding risks from chemicals. This study of "intuitive vs. scientific toxicology" was motivated by the premise that different assumptions, conceptions, and values underlie much of the discrepancy between expert and lay views of chemical risks. The results showed that Canadian toxicologists had far lower perceptions of risk and more favorable attitudes toward chemicals than did the Canadian public. The public's attitudes were quite negative and showed the same lack of dose–response sensitivity found in the earlier U.S. study. Both the public and the toxicologists lacked confidence in the value of animal studies for predicting human health risks. However, the public had great confidence in the validity of animal studies that found evidence of carcinogenicity, whereas such evidence was not considered highly predictive of human health risk by many toxicologists. Technical judgments of toxicologists were found to be associated with factors such as affiliation, gender, and worldviews. Implications of these data for risk communication are briefly discussed.

KEY WORDS: Intuitive toxicology; risk perception; chemical risks; expert judgment; risk communication

Human and Ecological Risk Assessment: Vol. 1, No. 3, pp. 231-248, 1995

Health Risk Perception in Canada II: Worldviews, Attitudes and Opinions

Daniel Krewski[1,2], Paul Slovic[3], Sheryl Bartlett[1],
James Flynn[3] and C.K. Mertz[3].

[1]*Health Canada, Environmental Health Centre, Ottawa, Canada*

[2]*Carleton University, Ottawa, Canada*

[3]*Decision Research, Eugene, OR*

ABSTRACT

In this article, we present the results of a national survey of 1500 Canadians on their attitudes and opinions about health risks. Ratings of perceived risk, sources of information on health risks and responsibility for risk management were also investigated, with findings reported separately. A high degree of concern about health risks was associated with industrial pollution and chemical products (with the exception of medicines), with almost complete agreement that the land, air and water are more contaminated than ever. In addition, there was widespread belief that a risk-free environment was an achievable goal, and an unwillingness to accept some health risks to improve the economy. Lifestyle factors such as diet, exercise, and tobacco smoking were perceived to be important modifiers of health risk. On the other hand, many respondents endorsed the idea that they had little control over the risks to their health.

Health Risk Perception in Alberta

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Research Report 95-1

ABSTRACT

A survey designed to assess different aspects of health risk perception among the residents of Alberta was conducted in 1994 under the auspices of the Eco-Research Chair in Environmental Risk Management. The survey was done in conjunction with the 1994 Alberta Survey - the eighth annual provincial survey administered by the Population Research Laboratory of the Department of Sociology, University of Alberta.

The questions posed in the survey were divided into six categories, designed to elicit information on "attitudes, opinions and worldviews", "individual exposure to environmental health risks", "risk perception", "sources of information", "reliability of information" and "status of health risks". These questions were based in part on a previous Canadian health risk perception survey designed and conducted in 1992 by Decision Research and Goldfarb Consultants, under the supervision of the Department of National Health and Welfare.

The Alberta public reported a relatively high degree of perceived health risk for many of the 22 hazards presented in the survey. The highest perceived health risk to the Alberta public as a whole was cigarette smoking, followed closely by stress. AIDS and suntanning were the third and fourth most highly rated perceived risks. This ranking indicates that the Alberta public are very aware of the health risks associated with lifestyle choices, as opposed to technology or pollution hazards. The high ratings of "chemical pollution", "ozone depletion", "nuclear waste" and "PCBs and dioxin" appear to reflect the influence of the media on the attitudes and opinions of the respondents, particularly when compared with the low prevalence of environmental health problems Albertans were able to identify for themselves. However, the two hazards which might be of specific concern to Albertans ("sour gas wells" and "hazardous waste transport") were ranked as only moderate health risks, perhaps because concern on these issues was very localized.

The respondents in the 1994 Alberta Survey generally perceived the hazards evaluated to be of lower risk than did the respondents in the 1992 Canada Survey. Although the six hazards rated to be of highest health risk were the same for the 1994 Alberta Survey, the 1992 Canada Survey and the 1992 Alberta only responses, the ranking of these risks was different.

Gender, age and education had a sizable effect on risk perception. Women were more likely than men to rate a hazard as a 'high risk' in almost all cases. In addition, women ranked "stress" rather than "cigarette smoking" as the highest risk factor. In general, older persons were more likely to rate a health risk as high, although the younger respondents displayed slightly higher perceived risks for technological or pollution hazards such as "chemical pollution", "nuclear waste", "ozone depletion" and "waste incinerators". With the exception of "indoor air quality" and "bottled water", the likelihood of rating a risk as 'high' decreased with the level of education.

When asked about their major sources of health risk information, without prompting, Albertans claimed to receive 70 to 80% of their information from the media (magazines, newspapers, TV and radio), with "other people", "books" and "work" ranking as secondary sources. "Doctors", "government organizations" and "environmental organizations" all ranked fairly low as a source of health risk information. However, when prompted with possible information sources in the 1992 Canada Survey, respondents professed more reliance on these other sources of information, perhaps indicating the induced survey bias of prompted responses. Given that the media was stated to be the primary source of information, it is disconcerting that the majority of respondents in the 1994 Alberta Survey believed that the information they received about health risks from chemicals and other hazards was "somewhat reliable" or "very reliable".

Sample Contact Letter

Dear Colleague:

Due to the conflicting and often contradictory nature of the assessment of environmental risks, there is a need to examine the subjective elements which influence views of chemical risks. You are invited to participate in environmental risk research currently being conducted in this area on behalf of the University of Alberta *Eco-Research* Chair in Environmental Risk Management. The *Eco-Research* Chair is devoted to exploring the intellectual basis for environmental risk management through an interdisciplinary approach.

"Expert Judgments of Environmental Risk" is research which focuses on exploring the similar and differing assumptions, conceptions and values which influence views of chemical risks within the community of environmental professionals. The purpose of this study is to determine and compare the attitudes, beliefs and perceptions of various scientific disciplines within the environmental field. This study is a partial replication and an extension of previous work which examined the intuitive elements of risk judgments by expert toxicologists and the lay public. We intend that this research will seek useful insights into the differences which exist between and within various environmental disciplines.

As a participant in the environmental field you may assist in this research by completing this survey questionnaire. Please note that all information which you provide in this questionnaire is strictly confidential and precautions have been taken to ensure that individual responses cannot be identified. If you are interested in receiving the results of this study and/or participating in a follow-up study please complete the address card and submit it separately from the questionnaire in accordance with the attached instructions.

Should you require additional information please do not hesitate to contact me at:

address: 13-103 Clinical Sciences Building
University of Alberta
Edmonton, Canada
T6G 2G3
phone: (403) 492-6408
fax: (403) 492-0364

Thank-you for your assistance in this research.

Steve E. Hrudey, Ph.D P.Eng.
Professor of Environmental Health
Eco-Research Chair in Environmental Risk Management

Eco-Research Chair in Environmental Risk Management
Environmental Health Program, Department of Public Health Services
University of Alberta, Edmonton, Alberta

Questionnaire

Please answer survey questionnaire by checking the box next to the most appropriate answer, or by writing your answer in the space provided.

1. AGE: _____ 2. GENDER: ☐ male ☐ female

3. COUNTRY OF RESIDENCE: _____

4. Please indicate all completed degrees/diplomas. Provide your specialization, the name of the university/institution and the country in which it is located, and the calendar year in which the degree/diploma was obtained.

Degree/ Diploma	Major	University	Country	Year
☐ Ph.D.				
☐ Dr.P.H				
☐ M.D				
☐ M.Sc.				
☐ M.P.H				
☐ B.Sc.				
☐ B.A.				
☐ other:				

5. Who is your primary employer?

☐ Government Office/Ministry
☐ Private Consulting
☐ Public Interest and/or Non-profit
☐ Publicly Funded Academic Institution
☐ Privately Funded Academic Institution
☐ Industry
☐ other:_____

6. What is (are) your current position(s) / title(s)?

Please state in full:_____

	Strongly Disagree	Disagree	Agree	Strongly Agree	Don't know/ No opinion
7. The way that an animal reacts to a chemical is a reliable predictor of how a human would react to it.	☐	☐	☐	☐	☐
8. If a scientific study produces evidence that a chemical causes cancer in animals, then we can be reasonably sure the chemical will cause cancer in humans.	☐	☐	☐	☐	☐
9. A prescription drug that hasn't been formally tested but has been used for 20 years is safer than a new prescription drug that has been tested and approved for use under the present guidelines.	☐	☐	☐	☐	☐
10. Government has no right to regulate people's personal risk-taking activities such as smoking, mountain climbing, etc..	☐	☐	☐	☐	☐
11. Epidemiological evidence is adequately used in regulation.	☐	☐	☐	☐	☐
12. If you are exposed to a carcinogen, then you are likely to get cancer.	☐	☐	☐	☐	☐
13. Chemicals are either safe or dangerous. There is really no in between.	☐	☐	☐	☐	☐
14. There is no safe level of exposure to a cancer-causing agent.	☐	☐	☐	☐	☐
15. If even a tiny amount of a substance that can cause cancer were found in my tap water, I wouldn't drink it.	☐	☐	☐	☐	☐
16. Fruits and vegetables contain natural substances that can cause cancer.	☐	☐	☐	☐	☐
17. The risk of getting cancer from lifestyle factors such as smoking and diet is much greater than the risk of cancer from chemicals in the environment.	☐	☐	☐	☐	☐
18. Natural chemicals are not as harmful as man-made chemicals.	☐	☐	☐	☐	☐

19. The land, air and water around us are, in general, more contaminated now than ever before.	☐	☐	☐	☐	☐
20. Experts are able to make accurate estimates of health risks from chemicals in the environment.	☐	☐	☐	☐	☐

21.a) Where do you obtain <u>most</u> of your information about health risks from chemicals and other health hazards (list in order of reliance in the space provided below)?

b) In general, how <u>reliable</u> do you think the information from each source is?

	Reliability		
Information Sources	Very Reliable	Somewhat Reliable	Not Very Reliable
1._____	☐	☐	☐
2._____	☐	☐	☐
3._____	☐	☐	☐

NOTE: **Following pre-testing of this material, some of the questions may be modified or further questions may be added.**