Draft Report

VALUING THE BENEFITS OF THE NEW SUBSTANCES NOTIFICATION REGULATIONS

Prepared for:

Environmental Protection Service
Environment Canada

Prepared by:

Lara B. Sheer, Anne P. Downey, Mark D. Ewen,
Timothy B. Petersen (Project Manager) and Brian G. Morrison (Project Director)
Industrial Economics, Incorporated
2067 Massachusetts Avenue
Cambridge, MA 02140

Project Steering Committee:

Elaine DuWors
National Wildlife Research Centre
Environmental Conservation Service
Environment Canada

Jim Frehs
Water & Habitat Conservation Branch
Environmental Conservation Service
Environment Canada

R.A. (Sandy) Matheson
Commercial Chemicals Branch
Environmental Protection Service
Environment Canada

Danny Guay
Regulatory and Economic Assessment Branch
Environmental Protection Service
Environment Canada

Jacqueline Sitwell
Bureau of Chemical Hazards
Environmental Health Directorate
Health Canada

Scientific Authority:

Paul De Civita
Regulatory and Economic Assessment Branch
Environmental Protection Service
Environment Canada
(819) 997-7061

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Author: STEWARTT@epndce.nci.nih.gov at internet
Date: 11/22/96 9:32 AM
Priority: Normal
TO: robert venezia at CMAHQ
Subject: RE:

---------- Message Contents ----------

Dear Mr. Venezia:
I think something got lost in the message. NCI is not following up a cohort of vinyl chloride workers, nor do I know of anyone who has, other that those who have published in the occupational hygiene literature. I'm not sure what I said to Dr. Lewis (I did talk with him), but I may have mentioned that at one time I had wanted to get measurement data on vinyl chloride workers to do a methodologic study; I was not successful. I'm sorry I cannot provide you with more data. Trish Stewart

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INTRODUCTION

The New Substances Notification Regulations ("the NSNR" or "the Regulations") were developed to ensure "that no new substances will be manufactured in or imported into Canada on a commercial scale before an assessment has been carried out to determine the risks posed to the environment and human health by the substances (Canada Gazette 1994)." To characterize the potential benefits of the Regulations, this report analyzes the benefits that would have resulted had the Regulations been implemented in time to control or prohibit the use of three substances currently or previously manufactured in or imported into Canada: vinyl chloride (VC), dichloromethane (DCM), and polychlorinated biphenyls (PCBs). This approach is analogous to evaluating the human health and environmental damages incurred due to the release of these substances into the Canadian environment, assuming that these damages would not have resulted had the Regulations been in place at the time the substances were introduced. This type of approach is often referred to as an "avoided damages" approach.

This executive summary describes the process Environment Canada (EC) and Health Canada (HC) employed to select the substances analyzed. It then outlines the benefits typology used in this report, summarizes the methods used to estimate annual and total benefits, and presents a summary of the results for each substance. It also cites the major limitations of the analysis and provides an overview of the structure of the remaining chapters.

SELECTION OF SUBSTANCES ANALYZED

In the course of developing the NSNR, EC and HC committed to a mult-stakeholder review of regulatory impacts, including an analysis of the Regulations' environmental and health benefits. This analysis is to be submitted to the New Substances Notification Impact Working Group (NSNIWG), which is comprised of representatives from EC, HC, Industry Canada, and the Industry Coordinating Group for the Canadian Environmental Protection Act (CEPA). The analysis is to be completed by September 1997, approximately three years after the rules' promulgation.

Ideally, the impact analysis would focus on benefits attributable to regulation of new substances under the NSNR. The data required to conduct such an analysis, however, are currently unavailable, and unlikely to become available by 1997. Therefore, EC and HC chose to characterize the Regulations' potential benefits through case studies of three CEPA toxic substances that in all

likelihood would have been prohibited or controlled had they been evaluated through the new substance notification program.

EC and HC selected the three substances analyzed in this report using a screening approach to narrow the list of CEPA toxic substances (substances subjected to regulatory control under CEPA and identified as toxic under the Priority Substances Program). First, to ensure a focus on commercially manufactured or imported products, substances that generally would be classified into one of the following categories were eliminated from consideration:

- Effluents and wastes. Effluents and wastes are not notifiable under the Regulations, and therefore are not affected by them.
- By-products of manufacturing and processing operations. Manufacturing and processing by-products are also not directly notifiable under the regulations.
- Elements and naturally-occurring compounds. Elements and naturally-occurring compounds were eliminated from consideration due to the inherent difficulty in distinguishing anthropogenic impacts from natural impacts.

The remaining substances were reviewed to determine:

- the completeness of data on human health and ecological effects;
- their suitability as surrogates for the NSNR analysis (i.e., solely or primarily anthropogenic in origin); and
- whether they had been determined to be CEPA toxic by both EC and HC.

The result of this effort was the selection of VC, DCM, and PCBs as the most appropriate candidates for analysis. These substances are anthropogenic in origin and have been determined to be CEPA toxic by the two Departments. In addition, the data available on the impacts of these substances is relatively substantial, facilitating the development of a benefits assessment.

OVERVIEW OF METHODS

We evaluate the benefits of the NSNR using an approach resource economists commonly refer to as the damage function approach.[1] As generally applied, this approach includes the following steps:

[1] The term damages refers to negative environmental or human health effects. The benefits of a particular policy (e.g., a pollution control regulation) are the reduction in damages that result from the policy's implementation.

(1) Estimating the extent to which a regulation would change the quantity of a pollutant or toxic substance released to the environment;

(2) Employing dispersion modeling techniques to characterize changes in the concentration of pollutants in environmental media;

(3) Applying concentration- or dose-response functions to estimate changes in physical impacts (e.g., human health impacts or impacts on species abundance) attributable to pollution;

(4) Valuing the change in physical impacts, thereby creating an economic benefits estimate; and

(5) Aggegating benefits estimates across all relevant categories and time periods.

The broad scope of this analysis and the limited time and resources available for the effort have necessitated some simplification of this approach. For example, we assume that control or prohibition of a substance under the NSNR would eliminate all associated health or environmental impacts; as a result, the benefits of the Regulations are simply the absolute value of the damages attributed to unregulated manufacture and/or use of each substance. In addition, we have not undertaken any original pollutant dispersion modeling, choosing to rely instead on previously developed information to characterize concentrations of VC, DCM, and PCBs in the environment. Nonetheless, the approach employed here is consistent with the underlying theory of scientific injury assessment and environmental impact valuation, and provides a reasonable characterization of the benefits of three hypothetical applications of the NSNR.

The following discussion notes key methodological issues in our implementation of the damage function approach to analyze the benefits of controlling VC, DCM, and PCBs. As described below, implementation of the methodology includes the following major elements:

- A preliminary evaluation of each substance to identify major benefit categories and to determine if sufficient data are available to quantify key benefits;
- Use of this information to develop a detailed evaluation of the effect of each substance on human health and/or the environment; and
- Valuation of human health and ecological effects, including the incorporation of simulations to address uncertainty in the underlying estimates.

Identification and Screening of Benefits

A number of different classification approaches have been developed to structure assessments of the benefits of environmental protection. This report employs a benefits classification framework that Environment Canada itself recommends (EC 1996). The Environment

Canada framework, which is similar to many of the classification schemes in the published literature, is summarized in Exhibit 1-1.

Exhibit 1-1

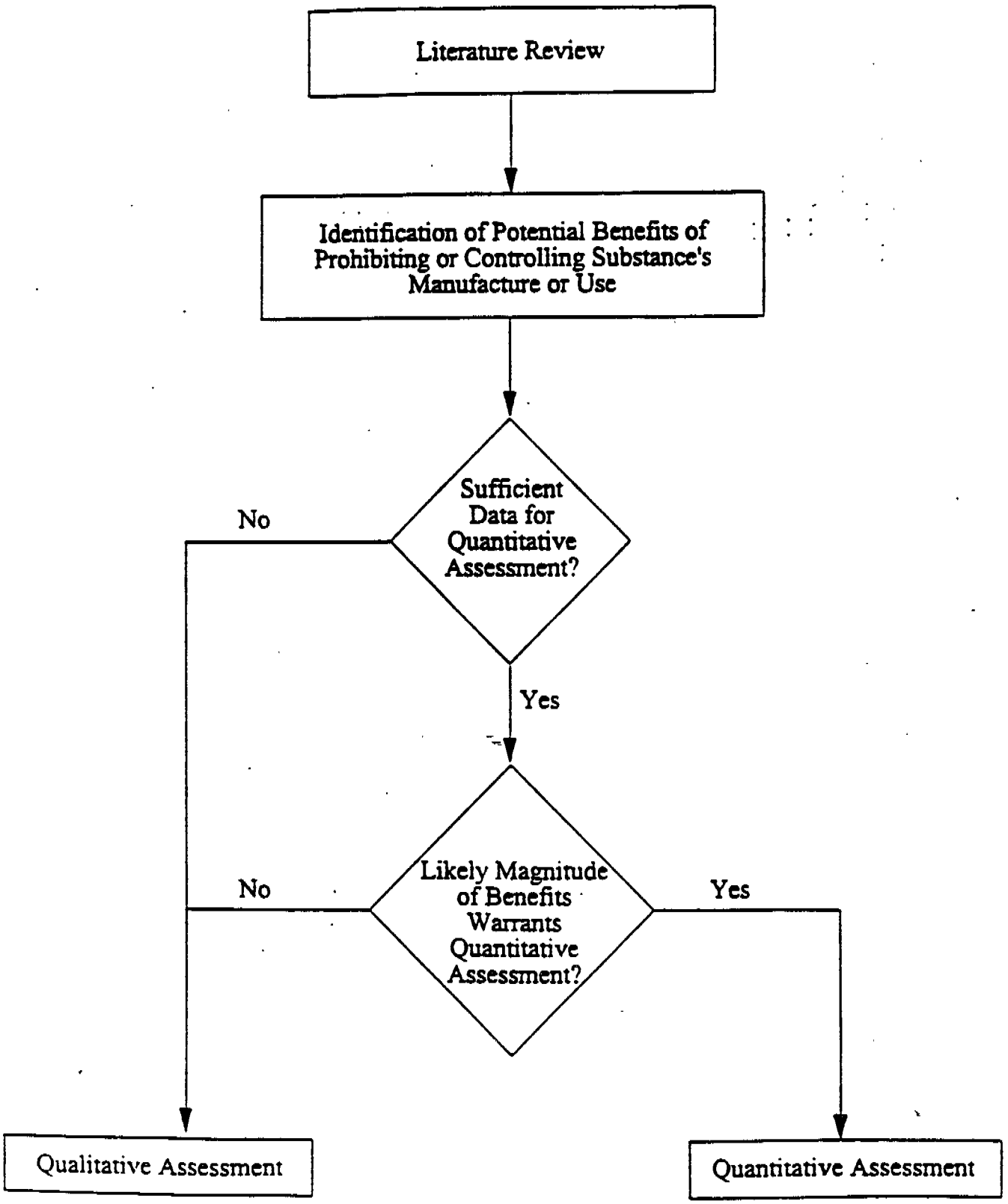
EC's FRAMEWORK FOR CLASSIFYING BENEFITS

Category	Explanation/Examples
Human Health	Benefits resulting from decreased mortality and/or morbidity.
Extractive Uses	Benefits resulting from the increased use of natural resources that are used directly for commercial, recreational, or subsistence needs (e.g., increased recreational fishing opportunities).
Non-Extractive Uses	Benefits resulting from the increased use of environmental services that are not consumed (e.g., activities such as hiking and canoeing).
Ecological Functions	Benefits resulting from a wide range of ecological services that are dependent on a healthy ecosystem (e.g., benefits that humans receive from the ability of soil to filter impurities from groundwater).
Passive Uses	Benefits resulting from the knowledge of the continued existence of natural resources and the desire to leave natural resources to future generations.
Built Environment	Benefits resulting from reduction in materials damage and soiling.
Optional Uses	Benefits resulting from potential future uses (in the above categories), including insurance against unknown future events.

As the preceding exhibit shows, the benefits of environmental regulations can take a variety of forms. Exhibit 1-2 illustrates the process we have employed to identify the potential benefits f controlling VC, DCM, and PCBs, and to determine which benefits categories to subject to quantitative analysis. As the exhibit indicates, we first conducted an extensive literature review to identify potential benefits. We then screened the available data for each benefits category to determine whether it was sufficient to conduct a quantitative benefits assessment; if so, we judged whether the benefits were likely to be great enough to warrant devoting resources to a quantitative analysis. Benefits categories that passed this screen were evaluated quantitatively; all others were qualitatively assessed.

Exhibit 1-2

BENEFITS SCREENING PROCESS



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Exhibits 1-3 through 1-5 give the results of the screening assessment, listing the potential benefits associated with regulation of the three substances of concern and indicating which categories of benefits have been quantitatively or qualitatively evaluated. As the exhibit indicates, the focus of the VC and DCM analyses is on human health benefits, while the PCB analysis addresses a range of environmental benefits, including extractive, non-extractive, and passive uses.[2]

Exhibit 1-3

POTENTIAL BENEFITS OF NSNR CONTROLS: VINYL CHLORIDE

Benefit Category	Impacts of Concern	Form of Analysis
Reduced Mortality	Studies link VC exposure to angiosarcoma of the liver and cancer of the brain, lung, and digestive tract.	Quantitative
Reduced Morbidity	VC can cause hepatic, respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, renal, dermal, ocular, and other adverse effects.	Quantitative
Ecological Benefits	Byproducts of VC production can adhere to plankton and bioaccumulate in the marine environment.	Qualitative

Exhibit 1-4

POTENTIAL BENEFITS OF NSNR CONTROLS: DICHLOROMETHANE

Benefit Category	Impacts of Concern	Form of Analysis
Reduced Mortality	Chronic exposure to DCM has been linked to liver and lung cancer.	Quantitative
Reduced Morbidity	Chronic exposure to DCM has been linked to Central Nervous System effects such as headaches, dizziness, nausea, and memory loss; effects on liver and kidney function; and benign mammary gland tumors.	Quantitative
Ecological Benefits	Exposure to DCM may adversely affect some aquatic organisms, such as freshwater nematodes.	Qualitative

[2] At the instruction of HC, the analysis of PCBs excluded consideration of human health risks. HC considers available data on such risks inconclusive, and therefore insufficient for an analysis of this type.

Exhibit 1-5

POTENTIAL BENEFITS OF NSNR CONTROLS: PCBs

Benefit Category	Impacts of Concern	Form of Analysis
Extractive Use		
Recreational Fishing	Diminished abundance and diversity of fish species due to reproductive abnormalities lowers quality of the angling experience. Fish consumption advisories (based on human health concerns) reduce the value of angling experience and affect the choice of fishing location.	Quantitative
Waterfowl Hunting	High tissue concentration levels and potential waterfowl consumption advisories diminish enjoyment of the hunting experience and affect the choice of hunting locations.	Quantitative
Trapping	Decline in mink populations due to reproductive abnormalities limits pelt harvest and diminishes trapping experience.	Quantitative
Commercial Fishing	Diminished abundance and diversity of fish species due to reproductive abnormalities decreases catch. Fish consumption advisories reduce volume of saleable fish.	Qualitative
Non-Extractive Use		
Bird Viewing	Reductions in bird populations due to reproductive abnormalities and the presence of physical deformities diminishes the enjoyment of birdwatching.	Quantitative
Other Recreation	Concern over health risks diminishes the enjoyment of swimming, boating, or similar activities and affects the choice of location.	Qualitative
Passive Use	Contaminated environment decreases the value individuals derive from a clean natural resource, above and beyond its direct use.	Quantitative
Human Health	Increased risk of both mortality (cancer) and morbidity (e.g., adverse reproductive effects).	Qualitative

Developing Annual Benefits Estimates

We develop quantitative estimates of annual benefits for each substance by estimating annual human health and ecological effects resulting from exposures to the substance, and by valuing those effects using a "benefits transfer" approach. Further, we explicitly incorporate uncertainty into our analysis by using a statistical technique known as Monte Carlo simulation. A summary of the methods employed to estimate annual benefits is provided below.

Estimating Human Health and Ecological Effects

While the particulars of each analysis vary to some extent, we estimate human health and ecological effects by collecting information on:

- the sources and releases of each substance;
- the pathways from these sources to human health and ecological receptors;
- the concentrations of each substance to which human and ecological receptors are exposed;
- the estimated dose that humans or biota receive; and
- dose-response relationships.

This information is used to quantify likely human health effects and impairment of ecological r natural resource services. For instance, we use health risk assessment methodologies to develop estimates of the number of cancer cases that would be avoided by controlling VC and DCM.[3] For PCBs, we assess the effect of the contaminant on the services provided by natural resources, such as the number of recreational fishing trips affected by PCB contamination in fish.

Estimating these effects requires careful consideration of the timing of harm to human health and the environment, as well as consideration of the effects of other regulatory measures that may mitigate the risks a substance might otherwise pose. For instance, damages that VC emissions cause in Canada today are likely to be quite small, since (1) VC emissions are currently regulated under CEPA and (2) pre-CEPA emissions do not cause lingering problems due to the transient nature of VC in the environment. Thus, to evaluate the benefits of controlling VC as if it were a new substance, we employ 1970s estimates of average atmospheric concentrations of the pollutant in the vicinity of facilities that used or manufactured it; these estimates, developed prior to the regulation of the substance, allow us to estimate the adverse health impacts that would occur in the absense of controls. For PCBs and DCM we use more current information, as each substance continues to cause harm in Canada.[4]

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[3] For those readers unfamiliar with health risk assessment methods and terms, we have included a brief primer on these topics in Appendix 1A, which is attached to this chapter.

[4] While PCBs have been banned from new uses in Canada since 1980, previous discharges of PCBs continue to cause present day problems due to the persistence of the substance in the environment. DCM is not currently regulated under CEPA, so current releases provide a reasonable basis for assessing the benefits associated with regulating this substance.

Valuing Human Health and Ecological Effects

We value human health and ecological changes using the benefits transfer approach. Benefits transfer involves the application of value estimates, functions, data and/or models developed in one context to address a similar resource valuation question in another context. Benefits transfer in its simplest form involves the application of existing "per unit" values derived at one site (or in one study) to estimate the benefits attributable to an environmental improvement at another site. Such transfers may include adjustments to account for differences in the characteristics of the activity evaluated in the existing study versus the characteristics of the activity for the "site" (in this case "country") under analysis.

Although this technique has been used for many years to evaluate environmental benefits and to assess natural resource damages, it continues to generate some controversy in the environmental and natural resource economics community. This controversy focuses on the applicability of value estimates developed for a particular site in one context to the same or similar site in another context. Determining whether an existing study is appropriate for benefits transfer requires consideration of the overall quality of that study and the degree to which the case examined by the study is similar to the case of interest (typically referred to as the "policy" case). Factors f concern include:

(1) Commodity Characteristics

- The commodity (environmental goods and services or human health effects) valued in the policy case and study case should be the same or similar.
- The magnitude of change in the commodity between the policy and study case should be similar.
- The availability of substitute sources of environmental goods and services should be similar.
- The assignment of property rights should be the same (e.g., willingness to pay versus willingness to accept).
- The temporal perspective (i.e., when a commodity is provided and when payment is made) should be the same.

(2) Population Characteristics

- The characteristics of the populations of interest in the study and policy cases (e.g., age, income, proximity to the site or source of risk and degree of environmental concern) should be the same or similar.
- Any valuation function employed should include socioeconomic variables, to allow the transfer to account for differences in population characteristics between the study and policy cases.

- The population's familiarity with the environmental problem of concern should be similar in the study case and policy case (Boyle 1992; Desvouges 1992; Unsworth 1993).

Detailed information on the Canadian and U.S. studies relied upon in this report is provided in Chapters 2, 3 and 4.

To make the values collected from each study comparable, we use the following approach to adjust for inflation and for the difference in value of the U.S. and Canadian dollars:

- We adjust dollar values presented in past Canadian studies by applying the Canadian Consumer Price Index (CPI) to inflate figures to 1995 Canadian dollars.

- We adjust dollar values presented in past U.S. studies by first translating the U.S. dollar figures to Canadian dollars using the Purchasing Power Parity (PPP) index, and by then applying the Canadian CPI to inflate figures to 1995 Canadian dollars.

This approach was recommended to us by the Scientific Authority in consultation with Statistics Canada (De Civita 1996).

Incorporating Uncertainty

In the course of conducting this effort, we were faced with data uncertainties at several stages. For instance, our ability to estimate the cancer cases resulting from human exposures to VC and DCM is limited by a number of factors, including uncertainty regarding the size of the exposed population and uncertainty as to the actual responses of human receptors. Similarly, there is uncertainty associated with the economic unit values identified for particular benefits categories. To generate annual and total benefits estimates deterministically would imply a higher level of certainty than actually exists.

As a result, we explicitly recognize uncertainty associated with key independent variables through the use of a statistical procedure commonly known as Monte Carlo simulation. This technique provides information on the variable of ultimate interest (the dollar value of benefits) that is the function of input variables (e.g., unit economic values) that are probability distributions. The output of this process is a probability distribution reflecting the magnitude and likelihood of various benefit figures.[5] For instance, in Chapter 2 we provide benefits estimates associated with avoiding VC-induced cancer cases by reporting not only the mean value, but also the 10th and 90th percentile estimates. Although the selection of these figures as our low- and high-end estimates is arbitrary,

[5] We use the computer software program @Risk to conduct our uncertainty analysis. The program offers two sampling techniques: Monte Carlo sampling and Latin Hypercube sampling. We employed Monte Carlo sampling for this analysis.

their use should provide a more plausible characterization of the likely range of benefits than would use of the simulation's extreme upper and lower bounds.

Developing Total Benefits Estimates

Since the benefits of the NSNR will be realized over more than one year, we also estimate the total benefits of regulating each substance. The total benefits estimates are the present value of the stream of annual benefits discounted over a thirty year time period. We selected a thirty year time period as a common and reasonable "long" time horizon (e.g., 30 years is commonly used in estimating total operation and maintenance costs at hazardous waste sites). We acknowledge that the selection of 30 years in calculating total benefits for this project is somewhat arbitrary; in reality, the benefits from the NSNR could be longer or shorter than 30 years.[6]

The selection of a discount rate for this type of calculation has been the subject of much debate in the economics profession. Treasury Board, Canada recommends the use of five, 10 and 15 percent real discount rates in conducting analyses of this sort. We have selected lower figures (two, four and six percent), however, as the majority of environmental economists view five, 10 and 15 percent as too high for this type of analysis. The range of discount rates we employ is consistent with the findings of A. Myrick Freeman, a noted resource economist who recently reviewed the literature on this topic and recommends using a discount rate of 2 to 3 percent (Freeman 1993).

RESULTS

The following discussion summarizes the benefits of controlling or prohibiting the manufacture and/or use of vinyl chloride, dichloromethane and polychlorinated biphenyls in Canada. Detailed information supporting these benefits estimates is provided in the body of the report.

Vinyl Chloride (VC)

Vinyl chloride is a chemical compound that has been used in Canada's industrial and commercial sectors for over 50 years. The primary use of VC is in the production of polyvinyl chloride (PVC), which is used to manufacture plastic and vinyl products such as electrical wire, food packaging materials, pipe, and other construction products. VC can cause significant adverse

[6] Determining the period of time over which the benefits of the NSNR will accrue depends upon how the NSNR are viewed in relation to other public health and environmental regulations. For example, in the absence of the NSNR regulations, releases of hazardous substances into the environment will be regulated, but in all likelihood only after human health and environmental problems are discovered. Therefore, the benefits of the NSNR accrue between the introduction of the harmful (new) substance and when it would be regulated under the Canadian Environmental Protection Act. The extent of the damages caused in the interim is largely dependent on the nature of the substance. For instance, unregulated PCB discharges of only a few years can cause 50 or more years of damages, as the substance is persistent in the environment. In contrast, VC is transient in the environment, limiting the time period over which damages are likely to occur.

human health effects if production and use of the chemical are not regulated. In the early 1970s, unregulated indoor air concentrations at VC and polyvinyl chloride (PVC) production facilities were extremely high, causing a variety of health problems in plant workers. Further, releases to the ambient air posed significant health risks to those living near VC and PVC production facilities. As a result, the economic benefits of regulating the manufacture and use of VC are significant. As shown in Exhibit 1-6, the annual benefits associated with avoiding worker and general population cancer cases and occupational morbidity are estimated to range from $15.2 million to $106.2 million, with a mean estimate of $53.0 million; total benefits over 30 years, using a four percent discount rate and the mean annual benefits figure, are estimated to be $916.4 million. Additional avoided morbidity benefits may also exist, although we have been unable to quantify these benefits.

Dichloromethane (DCM)

Dichloromethane is a clear, colorless liquid that is primarily used as a solvent in cleaning or stripping operations (e.g., as a metal parts degreaser), as a solvent and propellant in aerosol products, and as a blowing agent in the production of urethane foams. The benefits of controlling or prohibiting DCM use in Canada are significant. As summarized in Exhibit 1-7, the annual benefits associated with avoiding worker and general population mortality are estimated to range from $12.8 million to $58.2 million, with a mean estimate of $32.6 million; total benefits over 30 years, using a four percent discount rate and the mean annual benefits figure, are estimated to be $563.7 million. Additional morbidity or ecological benefits may also exist, though we were not able to quantify such benefits in this report.

Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are a family of synthetic chemical compounds used extensively as a dielectric fluid in electrical equipment from the 1940s to the 1970s. PCBs can cause significant harm to the environment and human health if production, use, and disposal of the substance are not properly regulated. The results of our analysis indicate that there are significant economic benefits related to the regulation of substances like PCBs through the NSNR. We summarize the range of annual benefits and total benefits in Exhibit 1-8. For the extractive use benefit categories that we were able to monetize, we estimate annual benefits of $8.3 million to $57.6 million, with a mean estimate of $30.3 million; the present value of these benefits over 30 years, using a four percent discount rate and the mean annual benefits figure, is $524.0 million. Our estimate of annual non-extractive use benefits ranges from $5.7 million to $37.4 million, with a mean of $19.1 million; the present value of these benefits, using the four percent discount rate and mean annual benefits figure, equals $330.3 million. In addition, our estimate of annual passive use benefits ranges from $21.6 million to $86.4 million, with a mean of $54.0 million; using the four percent discount rate and mean annual benefits figure for this category yields a present value benefits estimate of $933.8 million. As illustrated by these totals, the direct (extractive and non-extractive) use and passive use categories each account for roughly 50 percent of the total estimated benefits of controlling PCBs.

Exhibit 1-6

SUMMARY OF ANNUAL AND TOTAL BENEFITS: VINYL CHLORIDE
($1995, millions)

Category	Annual Benefits			Present Value of Total Benefits – 30 years		
	Low	Mean	High	Low @ 6%	Mean @ 4%	High @ 2%
Mortality						
General Population	$10.3	$39.1	$80.0	$141.8	$676.1	$1,791.7
Occupational	$4.2	$12.1	$22.9	$57.8	$209.2	$512.9
Morbidity						
General Population	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Occupational	$0.7	$1.8	$3.3	$9.6	$31.1	$73.9
Ecological	Small	Small	Small	Small	Small	Small
Total	> $15.2	> $53.0	> $106.2	> $209.2	> $916.4	> $2,378.5

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Exhibit 1-7

SUMMARY OF ANNUAL AND TOTAL BENEFITS: DICHLOROMETHANE
($1995, millions)

Category	Annual Benefits			Present Value of Total Benefits – 30 years		
	Low	Mean	High	Low @ 6%	Mean @ 4%	High @ 2%
Mortality						
General Population	$6.5	$16.7	$30.0	$89.5	$288.8	$671.9
Occupational	$6.3	$15.9	$28.2	$86.7	$274.9	$631.6
Morbidity						
General Population	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Occupational	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Ecological	Small	Small	Small	Small	Small	Small
Total	> $12.8	> $32.6	> $58.2	> $176.2	> $563.7	> $1,303.5

Exhibit 1-8

SUMMARY OF ANNUAL AND TOTAL BENEFITS: PCBs
($1995, millions)

Category	Annual Benefits			Present Value of Total Benefits -- 30 years		
	Low	Mean	High	Low @ 6%	Mean @ 4%	High @ 2%
Extractive Use						
Recreational Fishing	$7.3	$26.9	$47.3	$100.5	$465.2	$1,059.4
Waterfowl Hunting	$0.6	$1.3	$2.4	$8.2	$22.5	$53.8
Trapping	$0.4	$2.1	$7.9	$5.5	$36.3	$176.9
Commercial Fishing	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Subtotal	$8.3	$30.3	$57.6	$114.2	$524.0	$1,290.1
Non-Extractive Use						
Bird Viewing	$5.7	$19.1	$37.4	$78.5	$330.3	$837.6
Other Recreation	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Passive Use	$21.6	$54.0	$86.4	$297.3	$933.8	$1,935.1
Human Health	Not Estimated	Not Estimated	Not Estimated	Not Estimated	Not Estimated	Not Estimated
Total	> $35.6	> $103.4	> $181.4	> $490.0	> $1,788.0	> $4,062.8

CMA 114651

Limitations

The analyses described above are subject to certain limitations. A summary of these limitations for each substance follows.

- **Vinyl Chloride (VC).** To estimate the health effects associated with VC, we make a series of assumptions that introduce uncertainty into the analysis. For instance, our analysis of the incidence of VC-induced cancer in the general population is based on standard assumptions regarding the location and mobility of the affected population, which may over- or under-state total cancer cases. Further, our assessment of occupational health effects relies heavily on incidence data collected in the U.S.; we do not know the extent to which plant conditions may differ between U.S. and Canadian facilities. Finally, we have been unable to monetize certain health effects in this assessment (e.g., general population health effects other than cancer).

- **Dichloromethane (DCM).** Data limitations affect our ability to estimate occupational exposures for DCM, as it is difficult to estimate how many Canadian workers are chronically exposed to significant levels of dichloromethane. While we extrapolate information from the U.S. to estimate occupational effects, it is difficult to assess how accurately this method approximates the exposed occupational population in Canada. Further, all the quantified benefits for DCM are based on chronic exposures. Since DCM is known to cause serious health problems when individuals receive acute doses, our health benefits are likely to be somewhat underestimated.

- **Polychlorinated Biphenyls (PCBs).** The most significant limitation of our analysis of PCBs is the lack of quantitative estimates for all of the possible benefits resulting from a Canadian environment free of PCBs. For instance, we were unable to estimate or monetize the benefits for commercial fishing and certain recreational activities (e.g., boating and swimming), and at HC's instruction did not attempt to quantify the human health benefits of PCB control. In addition, numerous factors simultaneously affect the quality of the aquatic environment, including toxic chemicals other than PCBs (e.g., mercury, dioxins), pesticides, and municipal wastewater discharges. As a result, it is difficult to isolate the environmental impacts attributable to PCBs. Further, the long-lived nature of PCBs, along with nearly 20 years of PCB regulation, make it difficult to establish a base year from which to estimate annual benefits.

ORGANIZATION OF REPORT

The remainder of this report provides detailed information on the benefits of controlling or prohibiting the use and/or manufacture of vinyl chloride (Chapter 2), dichloromethane (Chapter 3) and polychlorinated biphenyls (Chapter 4). Each chapter contains:

Background information on the substance, including a description of the compound, information on its use and/or manufacture in Canada, and information on the releases of the substance into the environment;

- A description of human and ecological exposures to the substance;
- An overview of the benefits of controlling releases of the substance, using the benefits typology presented above; and
- A detailed description of the annual and total benefits that result from controlling these substances.

Each chapter also presents the specific limitations associated with these analyses, including key data gaps.

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Appendix 1A

SUPPLEMENTAL INFORMATION ON HEALTH RISK ASSESSMENT

SUPPLEMENTAL INFORMATION ON HEALTH RISK ASSESSMENT

This appendix provides background information on health risk assessment terms and methods that are used in Chapters 2 and 3. This appendix is divided into two sections. The first addresses carcinogenic health effects; the second addresses noncarcinogenic health effects. Since the health risk assessment relies on data and techniques developed in large part by the U.S. Environmental Protection Agency (EPA), the appendix focuses on U.S. EPA terms and methods.

CARCINOGENIC EFFECTS[7]

Toxicological assessment of potentially carcinogenic substances often includes a rating of the "weight of evidence" concerning carcinogenicity. For example, the U.S. EPA assigns evidence from human and animal studies to one of five categories: (1) sufficient evidence, (2) limited evidence, (3) inadequate evidence, (4) no data concerning carcinogenicity, and (5) no evidence of carcinogenicity. U.S. EPA's classification scheme is presented in Exhibit 1A-1.

Exhibit 1A-1

U.S. EPA CLASSIFICATION SCHEME FOR CARCINOGENS

Group	Description
A	Human carcinogen; sufficient human evidence.
B1	Probable human carcinogen; limited human evidence.
B2	Probable human carcinogen; sufficient animal evidence, inadequate or no human evidence, or no human data.
C	Possible human carcinogen; limited animal evidence, inadequate or no human evidence, or no human data.
D	Not classifiable as to human carcinogenicity; inadequate human and animal evidence or no data available.
E	Evidence of noncarcinogenicity for humans.

Source: U.S. EPA, *Guidelines for Carcinogen Risk Assessment*, 51 FR 33992-34054, 1986

In addition to the qualitative rating assigned to potentially carcinogenic substances, U.S. EPA develops substance-specific cancer slope factors (CSFs) for potential carcinogens. A CSF gives the increase in lifetime cancer risk for an individual continuously exposed to a substance, from birth to

[7] For more detailed information on EPA's method for estimating cancer risks, see U.S. EPA, *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual*, December 1989.

death, per unit increase in the substance's concentration.[8] CSFs provide estimates of risks associated with low-dose exposure to suspected or known carcinogens. They are estimated from data on high dose human exposures and/or high dose laboratory exposure to animals. The CSF for an agent with an "A" rating will be based -- at least in part -- on human data, while one with a "B2" rating will be based entirely on animal data.

Generating a Cancer Slope Factor

In deriving a CSF, U.S. EPA evaluates available toxicological information about a chemical and selects an appropriate data set. In choosing appropriate data sets, human data of high quality are preferred to animal data. If animal data are used, the species that responds most similarly to humans is preferable. If there is no clear choice among species for which data exist, data for the most sensitive species is given the greatest emphasis. Occasionally, when no single study is judged to be most appropriate, U.S. EPA may derive a CSF based on the geometric mean of slope factors from several studies that collectively support the estimate.

Concept of Nonthreshold Effects

Carcinogens are assumed to have no threshold, i.e., there is no level of exposure that does not pose some probability, however small, of generating a carcinogenic response. Therefore, the development of a CSF usually involves extrapolating from the high doses administered to experimental animals to the lower exposure levels expected for human exposure in the environment. Because the extrapolation of the dose-response is to a range where data are not available, the actual CSFs are uncertain. Hence a large margin of safety is built into the slope factors. Generally, U.S. EPA takes a *conservative* approach in estimating a CSF by calculating the slope factor as the highest value that can be justified with 95 percent confidence. Thus, given the data, it is unlikely that the CSF is greater than that reported.

Assessing Carcinogenic Risks

A substance-specific CSF is multiplied by the substance-specific daily dose to estimate the incremental probability of a person developing cancer over a lifetime (70 years) as a result of exposure to the potential carcinogen. A cancer risk of 1×10^{-6} implies a probability of one in a million of developing cancer.

[8] U.S. EPA, *Integrated Risk Information System*, 1995.

NONCARCINOGENIC EFFECTS[9]

Unlike carcinogenic effects, noncarcinogenic effects are assumed to occur only at exposures above a threshold concentration below which protective mechanisms and function reserve capacities prevent reactions to exposure. Therefore, a range of exposures exists from zero to some finite value that can be tolerated by the individual without the manifestation of an adverse effect. The reference dose (RfD) is an estimate of a daily exposure level for humans, including sensitive subgroups, that is not likely to cause adverse effects during a lifetime of exposure. To develop an RfD, it is necessary to identify the upper bound of a tolerance range for the most sensitive populations.

Derivation of a Reference Dose

In developing an RfD for a substance, U.S. EPA examines all available toxicity studies. If adequate human data are available, this information is used as the basis of the RfD. Otherwise, animal study data are used. When choosing the appropriate human or animal study to use in deriving the RfD, U.S. EPA must make judgements regarding the relevance and quality of the available experimental studies. In addition, U.S. EPA must identify the effect characterized by the "lowest observed adverse effect" (LOAEL), which is referred to as the critical toxic effect, and serves as the basis for the RfD.

After the study and critical toxic effect have been selected, U.S. EPA identifies the exposure level that represents the highest exposure level tested in the study at which the critical effect was not observed. This highest "no observed adverse effect level" (NOAEL) is used to develop the RfD.

Applying Uncertainty Factors

The RfD is derived from the NOAEL by consistent application of the following uncertainty factors (UF):

- A factor of 10 is used to account for variation in biological response to exposure among the general population and is intended to protect sensitive subgroups, such as the elderly, from adverse effects.
- A factor of 10 is used when extrapolating from animals to humans to account for interspecies variability.
- A factor of 10 is used when a NOAEL derived from a subchronic rather than a chronic study is used as the basis for a chronic RfD.
- A factor of 10 is used when a LOAEL rather than a NOAEL is used as the basis for an RfD.

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[9] For more detailed information on EPA's method for estimating noncancer risks, see U.S. EPA, *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual*, December 1989.

In addition to the UFs listed above, a modifying factor (MF) ranging from greater than zero to 10 is used to reflect a qualitative professional assessment of additional uncertainties in the critical study and in the entire toxicity database for the substance not specifically addressed by the preceding UFs. The default value for the MF is 1.0.

Calculation of the Reference Dose

To calculate the RfD, the appropriate NOAEL (or LOAEL if a suitable NOAEL is not available) is divided by the product of all of the applicable uncertainty factors and the modifying factor. The calculation is as follows:

$$RfD = NOAEL \text{ or } LOAEL/(UF_1 \times UF_2 \ldots \times MF)$$

RfDs are expressed in units of mg/kg-day.

Assessing the Potential for Noncarcinogenic Effects

The Rfd assumes that there is an exposure threshold below which adverse effects will not occur. If the exposure level (dose) exceeds this threshold, adverse effects could potentially occur. This potential for adverse systemic effects is characterized by the hazard quotient (HQ). The HQ is calculated according to the following equation:

$$HQ = Dose/Rfd$$

The greater the value of the HQ above one, the greater the potential for adverse effects. However, the HQ is not a statistical probability; a ratio of 0.001 does not mean that there is a one in one thousand chance of the effect occurring. Furthermore, the potential for adverse effects does not increase linearly as the RfD is approached or exceeded.

OVERVIEW OF CHAPTER

Vinyl chloride (VC), a commercially important chemical compound used in the plastics industry, can cause significant adverse human health effects if production and use of the chemical are not regulated. In this chapter, we characterize and value the health effects that could have been avoided if the New Substances Notification Regulations (NSNR) had been promulgated at the time VC was introduced into Canada. As described below, the New Substances Notification Regulations would have produced significant economic benefits by reducing individual and worker exposures to VC.

Summary of Benefits

In the early 1970s. unregulated indoor air concentrations at VC and polyvinyl chloride (PVC) production facilities were extremely high, causing a variety of health problems in plant workers. Further, releases to the ambient air posed significant health risks to residents living proximate to VC and PVC production facilities. As a result, the economic benefits of regulating the manufacture and use of VC are significant. As shown in Exhibit 2-1, the mean annual benefits associated with avoiding worker and general population cancer cases and occupational morbidity are estimated to be $53.0 million dollars per year; total benefits over 30 years are estimated to be $916.4 million, using a four percent discount rate. Further, additional avoided morbidity benefits may exist, although we have been unable to quantify these benefits for purposes of this report.

Limitations

To estimate the health effects associated with VC, we make a series of assumptions that introduce uncertainty into the analysis. For instance, our analysis of the incidence of VC-induced cancer in the general population is based on standard assumptions regarding the location and mobility of the affected population, which may over- or under-state total cancer cases. Further, our assessment of occupational health effects relies heavily on incidence data collected in the U.S.; we

do not know the extent to which plant conditions may differ between U.S. and Canadian facilities.[1] Finally, we have been unable to monetize certain health effects in this assessment (e.g., general population health effects other than cancer). Specific data limitations are discussed in later sections.

Exhibit 2-1

SUMMARY OF ANNUAL AND TOTAL BENEFITS: CONTROLLING VC MANUFACTURE AND USE IN CANADA

Category	Benefits Estimates ($1995, millions)	
	Annual Benefits	Present Value of Total Benefits (30 years, 4%)
Mortality		
General Population	$39.1	$676.1
Occupational	$12.1	$209.2
Morbidity		
General Population	Unknown	Unknown
Occupational	$1.8	$31.1
Ecological	Small	Small
Total	>$53.0	>$916.4

Organization of Chapter

This chapter is organized into six sections. The first section provides a general description of the VC and PVC industries, including information on VC releases into the environment. We then summarize available data on human health and ecological exposures to VC in uncontrolled settings. The third section describes the health effects that commonly result from VC exposures, covering both carcinogenic and non-carcinogenic effects. The fourth and most lengthy section of this chapter provides a detailed description of the methods and assumptions used to estimate the incidence of cancer and other health effects in workers and the general population. The final two sections present annual and total benefits estimates, respectively, based on the incidence levels developed in the fourth section.

[1] As discussed later in this chapter, we have developed a range of benefits estimates to reflect the uncertainties present in the benefits categories we quantify.

BACKGROUND INFORMATION ON VINYL CHLORIDE

Description of the Compound

VC is a chemical compound that has been used in Canada's industrial and commercial sectors for over 50 years. It is a synthetic chemical with no known natural sources. A colorless petrochemical gas, VC has a mild, sweet odor, is slightly soluble in water, and is highly soluble in fats and organic solvents. In addition, VC has a low boiling point, a high vapor pressure, and is a known human carcinogen (U.S. EPA 1994b; VC in Drinking Water 1992).

The primary use of VC is in the production of PVC. PVC is used to manufacture plastic and vinyl products such as electrical wire, insulation and cables, industrial and household equipment, medical supplies, food packaging materials, piping, and other building and construction products. PVC is also used as a raw material in the paper, glass, rubber, and automotive industries, and in the production of 1,1,1 trichloromethane (U.S. EPA 1994b; VC in Drinking Water 1992). In addition, VC was once used as a refrigerant in cooling equipment and as an aerosol propellant in some cosmetics, drugs, pesticides, and other consumer products (Doniger 1978); these uses were discontinued when VC's carcinogenic properties became known.

Information on Manufacture in Canada

Unlike PCBs and dichloromethane, VC and PVC are manufactured in Canada. The nation's major production facilities -- including two VC plants and four PVC plants -- are located in Alberta, Ontario, and Quebec. The locations of these plants are presented in Exhibit 2-2. Expansion of these facilities over the past 20 years has increased domestic production of both VC and PVC. These facilities, however, are highly mechanized, requiring a relatively small number of workers. Fabrication plants, which manufacture end-use consumer products using PVC resins, are more labor intensive (Doniger 1978).

Exhibit 2-2

LOCATIONS OF VC AND PVC MANUFACTURING PLANTS

Type of Plant	Region
VC and PVC	Fort Saskatchewan, Alberta
VC and PVC	Sarnia, Ontario
PVC	Niagara Falls, Ontario
PVC	Shawinigan, Quebec

Source: EC 1986

Two primary processes are used to produce VC. One involves the pyrolysis of ethylene dichloride (EDC): ethylene is chlorinated to EDC, which is then thermally dehydrochlorinated, producing VC and HCl (EC 1986). The other process involves the hydrochlorination of acetylene. In the presence of a catalyst, a vapor-phase reaction occurs between acetylene and hydrogen chloride, producing VC (Lowenheim 1975).

To produce PVC, the monomer VC is polymerized. The concentration of VC entrapped in PVC depends upon the production process; it can range from 0.1 to 8,000 ppm (U.S. EPA 1975). In Canada, concentrations of VC monomer in PVC were reduced drastically between 1973 and 1975, and range from 1 to 10 ppm (VC in Drinking Water 1992).

Releases to the Environment

Emissions and effluents from VC and PVC processing plants are the primary sources of VC in the work place and in the environment. There are, however, other pathways by which VC can enter the environment, including accidental spills and the outgas of VC from plastic products. Other release routes include the disposal of VC wastes in landfills; the biodegradation of trichlorethylene, tetrachloroethylene and 1,1,1-trichloromethane; the evaporation of chemical wastes; and tobacco smoke (ATSDR 1993).

Once VC enters the environment, it can affect a variety of environmental media. Because VC is highly volatile, it vaporizes easily. Therefore, most VC releases end up in the atmosphere. There is potential for the contamination of other environmental media, however, such as water and soil. The following subsections provide a detailed description of atmospheric, water, and soil contamination from VC releases, as well as potential release routes.

Atmospheric Contamination

The primary sources of VC in the atmosphere are VC and PVC plant emissions. PVC manufacturing facilities account for approximately 85 percent of VC emissions, while VC facilities account for approximately ten percent.[2] Data on total VC emissions to the atmosphere in the early 1970s from uncontrolled plants are limited; it was estimated, however, that a total of 4,500 tons of VC were emitted to the ambient air in 1973. After imposing controls on VC emissions from VC and PVC plants, emissions were reduced by about 95 percent (EC 1986).

VC emissions from VC and PVC plants can be categorized by source: regulated point sources, incidental releases, and fugitive emissions. Regulated point sources include equipment such as the reactor, which may release VC when it is opened for cleaning, maintenance and inspection; the VC condenser, which discharges VC to the atmosphere after treatment by air strippers or other pollution control devices; equipment downstream from the stripper; and process vents. Emissions from incidental releases generally come from emergency venting due to the malfunction or breakdown of equipment. Fugitive emissions originate from sources other than the vent, stack, flue,

[2] The remaining five percent comes from the plastics fabrication industry and other minor sources.

or other openings that are specifically designed for the purpose of emitting VC. Fugitive emissions can be caused by leaking valves, flanges and seals, open-ended lines, drains, vessel opening losses, building ventilators, and open ponds. Individual occurrences of fugitive emissions may be small, although total fugitive VC releases can be large when emissions from all possible sources are summed (EC 1986).

Transportation accidents are another source of VC releases to the atmosphere, as not all VC is polymerized into PVC at its initial point of production. VC is typically shipped to PVC factories under pressure as a liquefied gas. In the U.S., for example, about 95 percent of VC is shipp d in rail tank cars; the rest is shipped in tank trucks, barges, and tank vessels. An accidental leak, puncture or explosion of these containers can release the liquefied gas, which is then likely to evaporate into the atmosphere and pose a potential health risk to transportation workers, nearby residents, travellers, and safety officers (Doniger 1978).

Other potential sources of VC in the atmosphere include the outgas of VC from new plastic products and the volatilization of VC products in municipal and hazardous waste landfills. In addition, VC is released in the indoor air of VC and PVC plants. As a result, VC and PVC workers without proper protection can be exposed to potentially significant concentrations of the compound.

Water Contamination

VC releases into the environment can also affect surface water, groundwater, and drinking water.

- Surface Water. The most likely source of surface water contamination is wastewater from VC and PVC manufacturers. In Canada, the effluent from the production of PVC is discharged into a variety of waterways, including a waterway in Quebec and the Welland River and St. Clair River in Ontario (EC 1988). VC reaching surface water via contaminated effluent is likely to migrate rapidly, on the order of hours or days, to the air (ATSDR 1993). The presence of materials such as fumates, surfactants, and particulates, however, may extend the residence time of VC in water (U.S. EPA 1980).

- Groundwater. VC can leach into groundwater as a result of spills, leachate from landfills and hazardous waste sites, and effluents from the plastics industry (ATSDR 1993). In addition, the biodegradation of trichloroethylene and perchloroethylene in groundwater can form VC. For a variety of reasons, including resistance to microbial degradation, VC can remain in groundwater from months to years (VC in Drinking Water 1992).

- Drinking Water. Small amounts of VC may be present in public water supplies as a result of industrial wastewater discharges (U.S. EPA 1980) or as a result of groundwater contamination (see above). The occurrence of VC in potable water, however, is primarily associated with the conveyance of water via PVC pipes manufactured with incompletely polymerized VC monomer. These extremely small amounts of entrapped VC monomer can leach from PVC pipes (VC in Drinking Water 1992). Since the level of

residual VC that is not molecularly bound in PVC pipe is extremely low, significant exposure to VC via this pathway is considered unlikely (Coiner 1996).

Soil Contamination

The primary sources of VC releases to soil are spills, effluent discharges, and leachate from disposed VC materials. VC is likely to volatilize rapidly from dry soil surfaces, however, due to its high vapor pressure. VC placed ten centimeters deep in dry soil, for example, is expected to have an effective half-life of about 12 hours. In addition, VC has a low soil organic carbon adsorption coefficient, and is highly mobile in soil, suggesting a strong potential to migrate from soil to groundwater (ATSDR 1993).

HUMAN AND ECOLOGICAL EXPOSURES TO VINYL CHLORIDE

This section describes the concentrations of VC to which humans and biota were likely t be exposed in the early 1970s. The focus of this section (and the remainder of the analysis) is on human health, as ecological impacts are a relatively minor concern for this compound. The following subsections describe the most common pathways for human exposure to VC, and the concentrations at which VC was found in different subpopulations. We also provide a brief summary of potential ecological effects of VC exposure.

As discussed in the first chapter of this report, we focus on emissions from uncontrolled VC sources to estimate human exposures in this analysis. This approach follows logically from the ultimate goal of this project, to evaluate the benefits of controlling or prohibiting new substances. As a result, much of the information we present in this section is based upon data from the 1970s, when VC emissions were largely uncontrolled.

Human Exposures

VC and PVC workers and individuals living in close proximity to VC and PVC production facilities were exposed to the greatest concentrations of VC. Exhibit 2-3 summarizes the typical concentrations to which various subpopulations were exposed in uncontrolled conditions.

Exhibit 2-3

TYPICAL EXPOSURE TO VINYL CHLORIDE VIA INHALATION IN THE EARLY 1970s

Subpopulation	Concentration
General population	0 ppb
Individuals living in close proximity to VC and PVC plants	0 to 814 ppb
Average VC and PVC workers	1 to 100 ppm
Maximally exposed VC and PVC workers	250 to 300 ppm

VC concentrations in ambient air were generally fairly low. Background levels of VC are often expressed in parts per billion (ppb), whereas indoor air concentrations of VC in VC and PVC plants are expressed in parts per million (ppm). The average concentration of VC to which populations living in the vicinity of emissions sources were exposed ranged as high as 814 ppb (ATSDR 1993). For individuals not living in the vicinity of point sources, exposure to VC via inhalation was expected to be essentially zero. Inhalation is also the primary route for occupational exposure. Workers responsible for cleaning reactor vessels, which are the chambers in which VC is converted to PVC in polymerization plants, were frequently exposed to airborne concentrations on the order of 250 to 300 ppm (Amdur 1991).

In addition to inhalation, humans can be exposed to VC via ingestion. As discussed above, VC can be found in drinking water at very low concentrations due either to the contamination of underground sources of drinking water or to the migration of VC from PVC pipes. In addition, VC can migrate from packaging material containing VC into food, since the monomer is soluble in alcohols and mineral oil. The presence of VC in drinking water or food, however, was relatively uncommon, and the average daily intake of VC through diet was essentially zero (ATSDR 1993). The only potentially significant exposure to VC via ingestion was found in PVC production plants, where workers could have been exposed to VC through the ingestion of PVC dust containing entrapped VC monomer (U.S. EPA 1975).

Ecological Exposures

Data on the ecological effects of VC contamination are limited. It is unlikely, however, that VC releases to the environment posed a threat of significant ecological damage, due to the quick volatilization of VC into the atmosphere. The potential for biomagnification in the food chain does not appear to be important because of the high volatility of VC and the fact that it is readily metabolized by higher-trophic-level organisms (ATSDR 1993).

One of the by-products of VC production may cause ecological damage if discharged into aquatic environments, as this by-product (EDC tar) can adhere to substances such as plankton. In addition, marine animals rapidly accumulate EDC tar from contaminated sea water. Since these tars have a relatively short half-life, ecological effects from exposure were likely to be much less severe than from exposure to other types of chlorinated hydrocarbons (U.S. EPA 1975).

THE BENEFITS OF CONTROLLING VINYL CHLORIDE RELEASES: OVERVIEW

This section provides an overview of the benefits associated with the prohibition or control of VC in Canada. The primary benefits of controlling VC emissions are reductions in mortality and morbidity due to VC exposures in the general population and among VC and PVC workers. Once VC is inhaled or ingested, it can cause a variety of adverse health effects. In this section, we summarize the considerable amount of information available on the health problems related to VC exposure.

Mortality from Cancer

In the early 1970s, the cancer-causing capacity of VC was recognized. The available epidemiological studies tend to focus on the incidence of angiosarcoma of the liver, a rare form of cancer, because of the ability to draw a causal relationship between exposure to VC and the development of tumors. Exposure to VC by inhalation is also suspected to increase the risk of cancer of the brain, lung, and digestive tract (U.S. EPA 1994b).

The types of cancer associated with VC are often fatal. Studies demonstrate, for example, a significant association between exposure to PVC dust and the risk of lung cancer mortality (Wagoner 1983). The average life expectancy (post-diagnosis) for an individual who has contracted cancer via exposure to VC is estimated to be 0.4 to 8.9 years (Sax 1981). Due to the nature of these VC related cancers, we value the cancer cases estimated from VC exposures (later in this chapter) as fatalities.

Morbidity

In addition to cancer, VC can cause a variety of other deleterious health effects. For instance, exposure to VC can cause hepatic, respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, renal, and dermal/ocular problems. Specific examples of common VC-related problems are listed in Exhibit 2-4. A number of studies also suggest that exposure to VC may affect sexual function and hormonal levels in humans and animals (ATSDR 1993). In addition, some studies suggest a statistically significant increase in chromosomal aberrations in humans exposed to VC, and a variety of studies show that members of communities with nearby VC

polymerization facilities have greater incidences f some forms of developmental toxicity.[3] For example, some of these studies show an excess of fetal loss when fathers have been exposed to VC. These studies and others, however, fail to demonstrate a statistically significant correlation between developmental toxicity and either parental occupation or proximity to the facility (ATSDR 1993).

Exhibit 2-4

NONCARCINOGENIC HEALTH EFFECTS FROM VINYL CHLORIDE EXPOSURE

Type of Exposure	Health Effects
Acute exposure to high levels of VC	Dizziness, headaches, giddiness, light-headedness, nausea, poor memory, tingling sensations, weight loss, chronic bronchitis
Chronic exposure to lower levels of VC	Liver damage, Raynaud's syndrome, circulatory disturbances in extremities, thrombocytopenia, dermatitis, scleroderma-like skin changes, lytic lesions of the terminal phalanges in hands and feet, pseudo clubbing of fingers, thyroid insufficiency, damage to the spleen and lungs, functional disturbances of the central nervous system

Source: ATSDR 1993

ESTIMATING HUMAN HEALTH EFFECTS

The following discussion provides specific information on the health-related benefits of controlling VC in Canada. In particular, we describe the detailed methodologies used to estimate general population and occupational cancer cases, as well as morbidity effects in workers. The information developed in this section (e.g., estimated annual cancer cases) serves as the basis for the benefits estimates developed in the following section.

Cancer Cases Resulting from Exposure to Vinyl Chloride

General Population

In 1975, the U.S. Environmental Protection Agency conducted a health risk assessment for community exposure to VC, in support of regulating air emissions around VC and PVC plants (Kuzmack 1975). To estimate potential exposures, EPA divided the area surrounding each plant into four bands ranging up to five miles from the plant. Through standard diffusion modeling of over 40 plants, the annual average ambient VC concentrations in each band were calculated. Detailed information for each individual plant was weighted to reflect differences in the type of plant, size of plant, and meteorological and topographic conditions at the plant site. The results were then used to estimate average VC exposures for individuals living in the vicinity of a "model" VC or PVC plant.

[3] Developmental toxicity is the occurrence of adverse effects that may result from exposure to a chemical prior to conception, during prenatal development, or postnatally to the time of sexual maturation.

Estimating Annual Cases

We use a similar approach to estimate potential VC exposure for the population living close to VC and PVC plants. First, we estimate the size of the affected population. Because the VC and PVC industry is based in only four Canadian cities, the size of this subpopulation is relatively small. Exhibit 2-5 presents estimates of the population that resides within specified distances of Canada's two VC and four PVC plants. We developed these estimates using information on the total size and density of the population in each of the cities of interest. A detailed description of how these estimates were developed is provided in Appendix 2A.

Exhibit 2-5

ESTIMATED POPULATION LIVING WITHIN A FIVE-MILE RADIUS OF VC OR PVC PLANTS

Distance from Plant	VC Plants	PVC Plants
Band 1 (0-0.5 mi)	1,444	2,876
Band 2 (0.5-1 mi)	4,331	8,629
Band 3 (1-3 mi)	38,142	61,213
Band 4 (3-5 mi)	42,540	107,589

To estimate the concentrations of VC to which individuals within each band might be exposed, we use the ambient air concentrations provided in the U.S. EPA study, which are presented in Exhibit 2-6. These figures represent average annual VC concentrations in ambient air near an uncontrolled VC and PVC plant under typical meteorological conditions.[4] As shown in this exhibit, VC concentrations are higher around PVC than VC plants. Further, as one would expect, concentrations drop substantially with distance from the facility.

Exhibit 2-7 summarizes our high-end estimates of the number of cancer cases associated with each type of facility. As shown by the rows labelled "VC Concentration" and "Dose", we estimate the annual dose of VC to which the average person in each band could be subjected based on the estimated annual average concentration of VC to which the individual is exposed, adjusted for average adult inhalation rates and body weight.[5] We calculate the individual lifetime cancer risk by multiplying the dose by the cancer slope factor for VC exposure via inhalation.[6] Finally, we

[4] The ambient air concentration estimates presented in the U.S. EPA study are a result of two independent studies, the results of which differed by less than 25 percent. The data presented in the EPA assessment included variations in meteorological conditions from location to location (Kuzmack 1975).

[5] The average adult inhalation rate is assumed to be 20 m^3/day, and the average adult body weight is 70 kg (U.S. EPA 1989).

[6] The inhalation cancer slope factor for vinyl chloride is 0.3 $(mg/kg\text{-}day)^{-1}$ (U.S. EPA 1994a).

estimate the number of annual cancer cases by multiplying the population by the cancer risk and dividing by the average lifespan (70 years). The rows labelled "Annual Cancer Cases" in Exhibit 2-7 provide a summary of our best high-end estimates of the number of cancer cases likely to be prevented under the New Substances Notification Regulations for those individuals living near a VC or PVC plant.[7] As shown at the bottom of the exhibit, we estimate that 1.7 cancer cases would occur around VC facilities and 9.5 cases around PVC facilities, for a total of 11.2 avoided annual cancer cases.

Exhibit 2-6

AVERAGE ANNUAL VINYL CHLORIDE CONCENTRATIONS IN AMBIENT AIR

	Vinyl Chloride Concentration (ppb)	
Distance from Plant	VC Plant	PVC Plant
Band 1 (0-0.5 mi)	113	323
Band 2 (0.5-1 mi)	20	57
Band 3 (1-3 mi)	5	15
Band 4 (3-5 mi)	2	6

Source: Kuzmack 1975

An on-going U.S. EPA review of VC's cancer slope factor suggests that the number of cancer cases estimated above may be high. The new slope factor will incorporate pharmacokinetics, which will reduce the slope factor and thus the individual lifetime cancer risk (Guth 1996).[8] To reflect this information, we have calculated low-end estimates by dividing our high-end estimates by a factor of 10. As a result, we value the general population mortality benefits in the next section by using annual case estimates that range from 1.1 to 11.2 annual cancer cases.

[7] A detailed example of how these estimates are developed is provided in Appendix 2B.

[8] Pharmacokinetics represents the movement of a substance within the body, which is affected by the uptake, distribution, elimination and transformation of the compound within the body. Thus, a pharmacokinetic model develops an estimated internal dose of the contaminant that is a more accurate measure of exposure than the applied dose.

Exhibit 2-7

GENERAL POPULATION CANCER CASES: HIGH-END ESTIMATE

		VC Plant	PVC Plant
Band 1 (0-0.5 mi)	Population	1,444	2,876
	VC Concentration	0.294 mg/m^3	0.840 mg/m^3
	Dose	0.084 (mg/kg-day)	0.240 (mg/kg-day)
	Cancer Risk	$2.5X10^{-2}$	$7.2X10^{-2}$
	Annual Cancer Cases	0.5	3.0
Band 2 (0.5-1 mi)	Population	4,331	8,629
	VC Concentration	0.052 mg/m^3	0.148 mg/m^3
	Dose	0.015 (mg/kg-day)	0.042 (mg/kg-day)
	Cancer Risk	$4.5X10^{-3}$	$1.3X10^{-2}$
	Annual Cancer Cases	0.3	1.6
Band 3 (1-3 mi)	Population	38,143	61,213
	VC Concentration	0.014 mg/m^3	0.039 mg/m^3
	Dose	0.004 (mg/kg-day)	0.011 (mg/kg-day)
	Cancer Risk	$1.2X10^{-3}$	$3.3X10^{-3}$
	Annual Cancer Cases	0.6	2.9
Band 4 (3-5 mi)	Population	42,540	107,589
	VC Concentration	0.005 mg/m^3	0.015 mg/m^3
	Dose	0.001 (mg/kg-day)	0.004 (mg/kg-day)
	Cancer Risk	$4.5X10^{-4}$	$1.3X10^{-3}$
	Annual Cancer Cases	0.3	2.0
TOTAL ANNUAL CANCER CASES		1.7	9.5

Limitations

The methodology described above requires a series of assumptions that may introduce uncertainty into the results of this analysis. For instance, we assume that the population is evenly distributed throughout the region, and that the VC or PVC plant is located in the center of the region. Further, our approach assumes that the potentially affected population breathes the ambient air within their respective band 24 hours a day, seven days a week, 365 days a year. Our methodology does not consider the travel patterns and daily mobility of each population, and thus the variation in levels of VC exposure. These simplifying assumptions may lead to an over- or under-estimate of cancer cases among the subpopulation surrounding VC and PVC plants.

Occupational

Before much attention was given to the monitoring of VC levels in the work place, indoor air concentrations exceeded what health authorities now consider to be acceptable exposure levels.[9] In the absense of regulations and subsequent controls on VC-related activities, it is possible that VC and PVC workers could be subject to potentially significant cancer risks.[10]

Estimating Annual Cases

To estimate the annual cancer cases expected within the population of VC and PVC workers under unregulated conditions, we must first estimate the size of the affected occupational population. We do so using data from U.S. VC and PVC facilities. In the early 1970s, 940 production workers were engaged in VC production at 17 U.S. plants, for an average of 55 workers per plant. At PVC facilities, approximately 5,600 workers were engaged in production at 40 facilities, for an average of 140 workers per plant (U.S. EPA 1975). In Exhibit 2-8, we use these average figures, adjusted upward and downward by 50 percent to account for uncertainty, to project the average number of Canadian workers potentially exposed to high concentrations of VC.[11,12] As shown in this exhibit, we estimate that between 336 and 1,006 Canadian production workers could be exposed, in the absence of occupational and environmental safety standards, to high concentrations of VC.

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[9] As of 1978, high levels of VC in the workplace were believed to be responsible for angiosarcoma of the liver in at least 68 workers worldwide (Doniger 1978).

[10] For the purpose of this analysis we ignore the potential mitigation of health risks by occupational health and safety requirements, which may only be applied after a problem is discovered.

[11] For instance, the "low" estimate of VC workers per plant is simply 55*0.5, or 28 workers.

[12] The adjustments for uncertainty reflect the fact that we do not have adequate information on labor intensity and plant size across U.S. and Canadian facilities.

Exhibit 2-8

ESTIMATED VC AND PVC WORKER POPULATION IN CANADA

	VC	PVC	Total
Workers per plant	28 - 83	70 - 210	—
Total, all plants	56 - 166	280 - 840	336 - 1,006

The remaining data we use to estimate annual cancer cases among VC and PVC workers is derived from published epidemiologic studies of health effects among workers who were exposed to high concentrations of VC before 1974. We take this approach for two reasons. First, although these studies do not include specific estimates of worker exposure levels, they do provide the most direct indication of the health risks associated with working in these plants. Second, the available cancer slope factor for VC is applicable to exposures up to only several hundred $\mu g/m^3$ (approximately 0.1 ppm). It has been well documented that in the U.S. before 1974, exposure to VC concentrations of 250 to 300 ppm was common for many job categories in VC and PVC industries (Doniger 1978).[13] In short, the expected ambient concentration of VC in the air of uncontrolled VC and PVC plants significantly exceeds the upperbound concentration for which the cancer slope factor is appropriate.

Based on a review of four different studies, the U.S. EPA estimated that the incidence rate of liver angiosarcomas among highly exposed VC and PVC workers (i.e., working in areas with VC concentrations of roughly 200 to 500 ppm), adjusted for the average latency period, is approximately 3.1×10^{-3} cases per worker-year of exposure (Kuzmack 1975). Assuming an average career length of 20 years for these workers, their individual lifetime cancer risk is estimated to be 6.2×10^{-2}. In other words, approximately six percent of all highly exposed VC and PVC workers would be expected to contract liver angiosarcoma during the course of their lifetime.

If we assume that all of the VC and PVC workers in Canada were exposed to high levels f VC in the early 1970s and would have continued to be subject to these exposures in the absence f the Regulations, the incidence rate of cancer among VC plant workers would range from 3.4 to 10.0 cases and among PVC plant workers would range from 16.8 to 50.4 cases, for a total ranging from 20.2 to 60.4 cases. An incidence rate of 20.2 to 60.4 cancer cases is equivalent to approximately one to three total cancer cases per work year for a work force of the size presented in Exhibit 2-8.[14]

[redacted]

[13] It w[redacted]not uncommon for peak exposures to reach up to 4,000 ppm for short amounts of time. Workers in other occupations, before the control of VC levels in the work place, are likely to have been exposed to lower and less sustained concentrations.

[14] One to three cancer cases per work year is high for a relatively small work force. Therefore, it is unlikely that this situation would continue unabated year after year once the problem was discovered. The benefits associated with even a few years of improved control, however, are significant.

Limitations

In estimating occupational effects, we use incidence information from U.S. workers. To the extent that Canadian and U.S. worker exposures are different, our estimates could be under or over-estimates. Further, we do not take into account the incidence of other types of cancer in the above assessment (i.e., cancers other than angiosarcoma of the liver), as we only have qualitative information on the incidence of other types of cancer. To the extent these cancers would occur, the estimates presented above could be under-estimates.

Noncarcinogenic Effects Resulting from Exposure to Vinyl Chloride

General Population: Point Source Exposures

Because uncontrolled VC and PVC plants emit high concentrations of VC to the ambient air, individuals living within a five mile radius of these plants could experience noncarcinogenic health effects related to VC emissions. We determined the likelihood of risk of noncarcinogenic effects by calculating a hazard quotient for potentially exposed populations. The hazard quotient is the ratio of the expected dose to a reference dose (RfD) or reference concentration (RfC); a hazard quotient greater than one is an indication that some noncarcinogenic effects may occur.[15] Generally, an RfD or RfC is used to determine the level below which there are no observed adverse impacts in humans due to daily exposure to a particular toxic substance. The U.S. Environmental Protection Agency, however, has not established an RfD or RfC for VC (ATSDR 1993). Instead, a minimal risk level (MRL) for VC of 0.002 ppm has been derived by ATSDR, based on an animal study.[16]

Exhibit 2-9 presents the hazard quotients for each band surrounding a VC or PVC plant. As indicated in this exhibit, the hazard quotients for individuals living within each band equals or exceeds one in all cases. This information suggests that there is a considerable likelihood that some noncarcinogenic effects could occur in the general population due to VC exposures. We note, however, that U.S. EPA is in the process of developing an RfC for VC. Although this value has not been released as of yet, it is likely to be higher than the MRL established by ATSDR (Guth 1996), which would produce lower hazard quotients. As a result, we believe that the figures presented in Exhibit 2-9 should be employed with caution.

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[15] See Appendix 1A for more information on this topic.

[16] The results of a study conducted by Bi, et al. (1985) indicate that exposing rats to 10 ppm of VC for six hours a day, six days a week, for six months of the year, results in increased liver weight. The lowest observed adverse effect level (LOAEL) is 10 ppm. ATSDR used the results of this study to establish the MRL of 0.002 ppm for humans, using a series of adjustment factors. The adjustment factors account for (1) the difference between intermittent and continuous dosing, (2) the uncertainty surrounding the difference between a LOAEL and a NOAEL, (3) the uncertainty surrounding the extrapolation from animal data to humans, and (4) the uncertainty surrounding human variability. The MRL is meant to be used for screening purposes only. The MRL is not used for regulatory action.

Exhibit 2-9

GENERAL POPULATION NONCARCINOGENIC EFFECTS

		VC Plant	PVC Plant
Band 1 (0-0.5 mi)	Dose in (mg/kg-day)	0.084	0.240
	Hazard Quotient	57	162
Band 2 (0.5-1 mi)	Dose in (mg/kg-day)	0.015	0.042
	Hazard Quotient	10	29
Band 3 (1-3 mi)	Dose in (mg/kg-day)	0.004	0.011
	Hazard Quotient	4	8
Band 4 (3-5 mi)	Dose in (mg/kg-day)	0.001	0.005
	Hazard Quotient	1	3

Note:
MRL = 0.002 ppm = 0.001 (mg/kg-day)

General Population: Other Exposures

The general population as a whole may face health risks from VC exposure due to sources of release other than emissions from VC or PVC plants, such as transportation accidents, drinking water contamination, or direct use and disposal of VC under unregulated conditions. In the following subsections, we briefly describe these other sources of VC release and discuss their potential contributions to adverse health effects in the general population. The discussion focuses on noncarcinogenic health effects, since the incremental cancer risks associated with these sources of release are likely to be dwarfed by the cancer risks associated with emissions from VC and PVC production facilities.

Transportation Accidents

VC produced in Canada is shipped by pipeline to neighboring PVC plants and by railroad to other plants in Canada, the U.S. and Mexico. VC was also once shipped in tank trucks, but this practice was discontinued in 1975 due to the unacceptable risk of highway accidents (EC 1986). Shipment of VC by rail reduces the risk of accidental release, but does not totally eliminate it. In 1980, for example, a train derailment in MacGregor, Manitoba resulted in a spill of 49.7 tons of VC; accidental releases of VC from tank cars have also occurred in the U.S. Nonetheless, the safety record for VC transport in recent years has been good, suggesting that the risk of noncarcinogenic health effects in the general population resulting from transportation accidents is likely to be small.

Drinking Water

In the absence of regulatory controls, the general population might be exposed to elevated concentrations of VC in drinking water, due primarily to the leaching of VC from PVC pipes. The available data, however, are insufficient to allow us to estimate the extent to which such exposures might occur. For example, the leaching of VC from PVC pipes depends on several factors, including dwell time, which is the period of time water stands in the pipes; leaching rate, which is determined by the amount and distribution of VC in the pipe and in the water; the equilibrium partitioning of VC between the pipe and the water; and the diffusion of leachate out of the pipe and into the air. Few data exist on pipe/water partitioning coefficients or plastic pipe diffusion coefficients, making the prediction of VC concentrations in water difficult (Podoll 1986).

Despite these limitations, the available data suggest that the risks associated with VC in drinking water are likely to be several orders of magnitude below the risks associated with inhalation of VC production emissions. For example, a drinking water study conducted by the U.S. Environmental Protection Agency examined VC concentrations in the drinking water of five water distribution systems using PVC pipes. The highest VC concentration was 0.54 ppb, which came from the most recently installed and longest pipe system. Traces of VC (0.012 to 0.023 ppb) were found in two other systems nine years after installation (VC in Drinking Water 1992). These concentrations are so low that the risks of adverse health effects appears minimal.

Other VC Uses and Releases

In the absence of regulation, VC might be used in a variety of consumer products. The associated adverse health effects would depend on the type and extent of use, manufacturing processes, and subsequent disposal practices. Since the range of potential practices is highly uncertain, we have not attempted to analyze the health risks they might present.

Occupational

As discussed earlier in this chapter, VC is known to cause a number of noncarcinogenic health effects in workers exposed to high concentrations of the substance. Information on the incidence of various effects, based on research reported by U.S. EPA in the 1970s, is summarized in Exhibit 2-10. As shown in this exhibit, the incidence rates for a number of these health effects are quite high, particularly for exposed workers. While they do not present detailed exposure data for these workers, these studies do provide a clear indication of the range of potential noncarcinogenic effects for workers exposed to VC. To provide an indication of the quantitative benefits of reducing or eliminating these health effects, we assume that 10 percent of all highly exposed workers would suffer from one or more noncarcinogenic effects that would impair their daily activities. Based on a worker population of between 336 and 1,000 (see Exhibit 2-8), we estimate that between 34 and 101 workers would be thus affected if VC emissions were uncontrolled.

Exhibit 2-10

INCIDENCE RATE OF OCCUPATIONAL NONCARCINOGENIC EFFECTS

Study	Health Effect	Incidence
Literature Review of 12,724 PVC Workers	Acroostaolysis[1]	0.9%
	Raynaud's Syndrome Symptoms[2]	0.8%
	Skin Lesions	0.3%
	Liver Disturbances[3]	0.6%
Clinical Observations of 50 Highly Exposed PVC Workers	Raynaud's Syndrome Symptoms[2]	16%
	Liver Disturbances[3]	76%
Liver Function Screening Programs for 1,183 PVC Workers	Liver Function Abnormalities[4]	4.8%

Source: Kuzmack 1975

Notes:

1. Gradual erosion of bone at fingertips
2. Cold hands and feet
3. Changes in liver function suggestive of cellular liver damage
4. Abnormalities considered serious enough by plant physicians to justify moving workers to areas where there is no exposure to hepatic toxins

VALUING HUMAN HEALTH EFFECTS: ANNUAL VALUES

In this section, we describe the process of valuing the health effects analyzed above. As indicated in Exhibit 2-11, we estimate the mean annual benefits associated with avoided morbidity and mortality to be $53.0 million. Below we discuss our approach and calculations.

Exhibit 2-11

SUMMARY OF BENEFITS ESTIMATES

Category	Mean Annual Benefits (1995$, millions)
Mortality	
General Population	$39.1
Occupational	$12.1
Morbidity	
General Population	Unknown
Occupational	$1.8
Ecological	Small
Total	>$53.0

Carcinogenic Effects: Mortality

For purposes of this analysis, we assume that all cancers attributable to VC exposure would be fatal. This assumption is based on the particularly lethal nature of angiosarcoma of the liver, the type of cancer most often contracted from VC exposures.[17] To value these effects, we utilize recent research into the statistical "value of life" conducted by IEc for the U.S. EPA's Office of Air and Radiation. As part of this effort, IEc evaluated three types of value of life estimates: wage-risk studies, which estimate the additional compensation individuals demand in the labor market for taking riskier jobs; contingent valuation studies, in which individuals are asked to state their willingness-to-pay to avoid additional increments of statistical mortality risk; and consumer behavior studies, which examine situations other than the labor market that involve risk-dollar tradeoffs. Based on this research and analysis, we have developed a lognormal distribution of the value of a statistical life, using a mean value of $6.5 million and a standard deviation of $4.4 million ($1995). Appendix 2C presents a list of the studies used to develop this estimate, which draws largely from a survey article authored by one of the field's leading researchers (Viscusi 1992).[18]

In Exhibit 2-12, we summarize the benefits associated with avoided cancer cases from VC exposure in the general population surrounding VC and PVC plants. As shown in this exhibit, the majority of the benefits come from reducing VC exposures to the general population living close to PVC plants. This is not surprising, considering that, under uncontrolled conditions, ambient concentrations of VC around these plants are estimated to be more than twice those around VC plants. Note that the values presented in this table are the tenth percentile, mean, and 90th percentile values from a 1,000 iteration Monte Carlo analysis. We generated the distribution using the lognormal distribution described above to characterize the statistical value of life, coupled with a uniform distribution to represent the estimated number of annual cancer cases, which range from 1.1 to 11.2 per year.[19]

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[17] We note that other researchers have valued carcinogenic health effects using a weighted average of estimates of the "value of life" (for mortality) and "cost of illness" (for non-fatal cases) (CCME 1995). While this approach may be appropriate for forms of cancer that are potentially treatable, we believe that using a weighted average that includes "cost of illness" estimates in our analysis would result in an underestimate of the benefits of avoiding a generally fatal form of cancer.

[18] The studies cited in Appendix 2C include analyses of compensating wage differentials for fatal injury risk in several countries, including Canada; the values are reported in the source document in American dollars, and have been converted to Canadian dollars using the method outlined in Chapter 1. We note, however, that the list of studies does not include four wage-risk studies for Canadian labor markets (Vodden et al. 1994, Martinello and Meng 1992, Meng and Smith 1990, and Meng 1989). We have not reviewed these four studies in detail, but have reviewed a summary of the studies contained in a report prepared for the Canadian Council of Ministers of the Environment (CCME 1995). The value of statistical life estimates reported for these four studies are comparable to those presented in Appendix 2C, with means ranging from $4.7 million to $7.6 million. The mean of the distribution of values upon which we rely ($6.5 million) would be unchanged if these studies were added to those cited in the appendix.

[19] A uniform distribution in this context means that there is an equal chance that any value between 1.1 and 11.2 is the true number of cases.

Exhibit 2-12

ANNUAL BENEFITS ASSOCIATED WITH AVOIDED DEATHS FROM CANCER IN THE GENERAL POPULATION ($1995, millions)

Type of Plant	Low Estimate (10th Percentile)	Mean	High Estimate (90th Percentile)
VC	$1.5	$6.0	$12.0
PVC	$8.8	$33.1	$68.0
Totals	$10.3	$39.1	$80.0

In Exhibit 2-13, we summarize the benefits associated with avoided cancer cases from occupational exposures to VC. We used the lognormal distribution to represent the statistical value of life, as described above, and a uniform distribution to represent the likely number of annual cancer cases, which we estimate to range from one to three per year. As shown in this exhibit, the majority of the benefits estimated come from avoided cancer cases among the PVC worker population, which is substantially larger than the population of workers in VC plants.

Exhibit 2-13

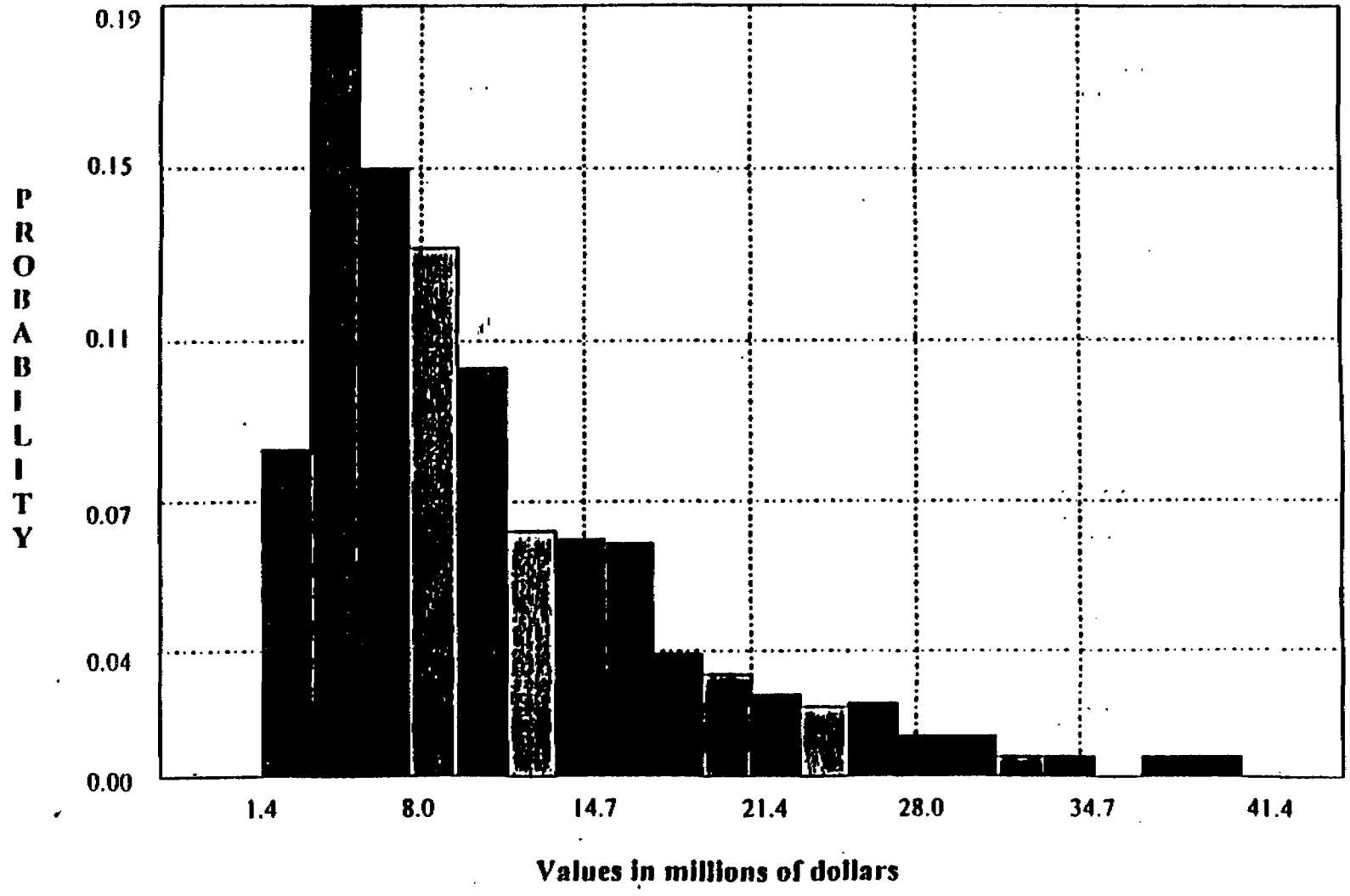
ANNUAL BENEFITS ASSOCIATED WITH AVOIDED DEATHS FROM CANCER IN THE OCCUPATIONAL POPULATION ($1995, millions)

Type of Plant	Low Estimate (10th Percentile)	Mean	High Estimate (90th Percentile)
VC	$0.8	$2.1	$3.9
PVC	$3.4	$10.0	$19.0
Totals	$4.2	$12.1	$22.9

To provide a sense of the uncertainty associated with the values presented in this table, Exhibit 2-14 presents a distribution of potential benefits associated with occupational exposures from PVC plants. As shown in this graphic, total benefits estimates could potentially range from $1.4 million to $41.4 million, based on this particular Monte Carlo run of 1,000 iterations.

Exhibit 2-14

DISTRIBUTION OF ANNUAL BENEFITS FROM AVOIDED PVC WORKER CANCER CASES



CMA 114680

Noncarcinogenic Effects: Morbidity

As discussed above, there are a number of noncarcinogenic effects that may result from acute and/or chronic exposures to high concentrations of VC. The benefits associated with reducing morbidity can include the following.

- Avoidance of direct medical costs, including the expenditures to combat a particular illness, such as doctor's fees, hospitalization costs, and pharmaceutical purchases.
- Indirect costs, including lost wages and leisure time due to illness.
- Pain and suffering, which encompasses the physical and psychological discomfort associated with morbidity effects (Unsworth 1993).

Several estimation techniques have been developed to assign economic benefit estimates to reductions in air pollution related morbidity effects, although these techniques generally address only a portion of total morbidity benefits.

Much of the morbidity literature associated with air pollutants focuses on respiratory effects (e.g., asthma and bronchitis symptoms caused by air pollutants). While VC is known to cause some respiratory problems (e.g., chronic bronchitis), more commonly cited noncarcinogenic effects are of the type cited in the previous section, such as Raynaud's Syndrome and acroostaolysis. As a result, we value morbidity effects using information collected in the CCME (1995) study on restricted activity days (RADs). RADs are a measure of illness defined as a day on which illness prevents an individual from engaging in some or all of his or her usual activities, including days spent in bed and days with minor activity restrictions due to illness. As part of valuing morbidity in the CCME study, the authors analyzed a number of studies estimating RADs. Based on this review, they provide a central estimate of $70 (1994$) per RAD, with a low estimate of $35 per RAD and a high estimate of $105 per RAD. For purposes of our analysis, we have adjusted these figures to annual values by multiplying by 365 days, and have adjusted the figures to 1995 dollars.[20] As a result, we value morbidity effects assuming the annual benefits of avoiding restrictions on activity range from $13,000 to $39,800 per year, with a mean value of $26,180.

In Exhibit 2-15, we summarize the benefits of avoided morbidity from occupational exposures. To develop these estimates we use a discrete probability distribution to represent the estimated annual RAD values, and a uniform distribution for the number of cases, which are estimated to range from 34 to 101 per year. As shown in this exhibit, we estimate total benefits to range from $0.7 million to $3.3 million per year, with a mean value of $1.8 million, based on a Monte Carlo run of 1,000 iterations.

[20] Implicit in this approach to developing an annual benefits estimate is the assumption that the activity of affected VC and PVC workers is restricted 365 days a year. Many of the health effects of interest (e.g., acroostaolysis) could have such persistent effects; however, to the extent these health effects would not impair activity year-round, the methodology lends an upward bias to the benefits estimate.

Exhibit 2-15

ANNUAL BENEFITS ASSOCIATED WITH AVOIDED MORBIDITY IN VC AND PVC WORKERS
(in millions)

Low Estimate (10th Percentile)	Mean	High Estimate (90th Percentile)
$0.7	$1.8	$3.3

VALUING HUMAN HEALTH EFFECTS: TOTAL VALUES

The results of the previous section indicate that the annual benefits of controlling VC are considerable. In addition, these benefits presumably would continue to accrue for a number of years, since, in the absence of controls, health effects would continue unabated. In Exhibit 2-16, we summarize our estimates of annual benefits and the present value of total benefits attributable to an environment free of VC. As illustrated in this exhibit, we estimate that annual benefits (for quantifiable benefits categories) range from $15.2 million to $106.2 million, with a mean annual benefits estimate of $53.0 million. Further, the present value of benefits ranges from $0.2 billion to $2.4 billion over a 30 year period, depending upon the annual benefits estimate (low, mean, or high) and discount rate employed.[21]

[21] See the discussion on discounting in Chapter 1.

Exhibit 2-16

SUMMARY OF ANNUAL AND TOTAL BENEFITS: VINYL CHLORIDE
($1995, millions)

Category	Annual Benefits			Present Value of Total Benefits – 30 years		
	Low	Mean	High	Low @ 6%	Mean @ 4%	High @ 2%
Mortality						
General Population	$10.3	$39.1	$80.0	$141.8	$676.1	$1,791.7
Occupational	$4.2	$12.1	$22.9	$57.8	$209.2	$512.9
Morbidity						
General Population	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Occupational	$0.7	$1.8	$3.3	$9.6	$31.1	$73.9
Ecological	Small	Small	Small	Small	Small	Small
Total	> $15.2	> $53.0	> $106.2	> $209.2	> $916.4	> $2,378.5

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Appendix 2A

GENERAL POPULATION EXPOSED TO VC EMISSIONS FROM POINT SOURCES

Overview

Applying ambient air concentration estimates developed in the 1975 EPA study requires that we estimate the number of individuals living proximate to VC and PVC facilities. Specifically, we need to estimate the number of individuals living within four bands located around each facility. The bands have the following distances:

- Band 1: 0 to 0.5 miles.
- Band 2: 0.5 to 1 miles.
- Band 3: 1 to 3 miles.
- Band 4: 3 to 5 miles.

The purpose of this appendix is to describe the methodology we employ to estimate the size of the affected population within each band. We use 1991 Canadian census data on the total population and size of the four Canadian cities in which VC and PVC plants are located.

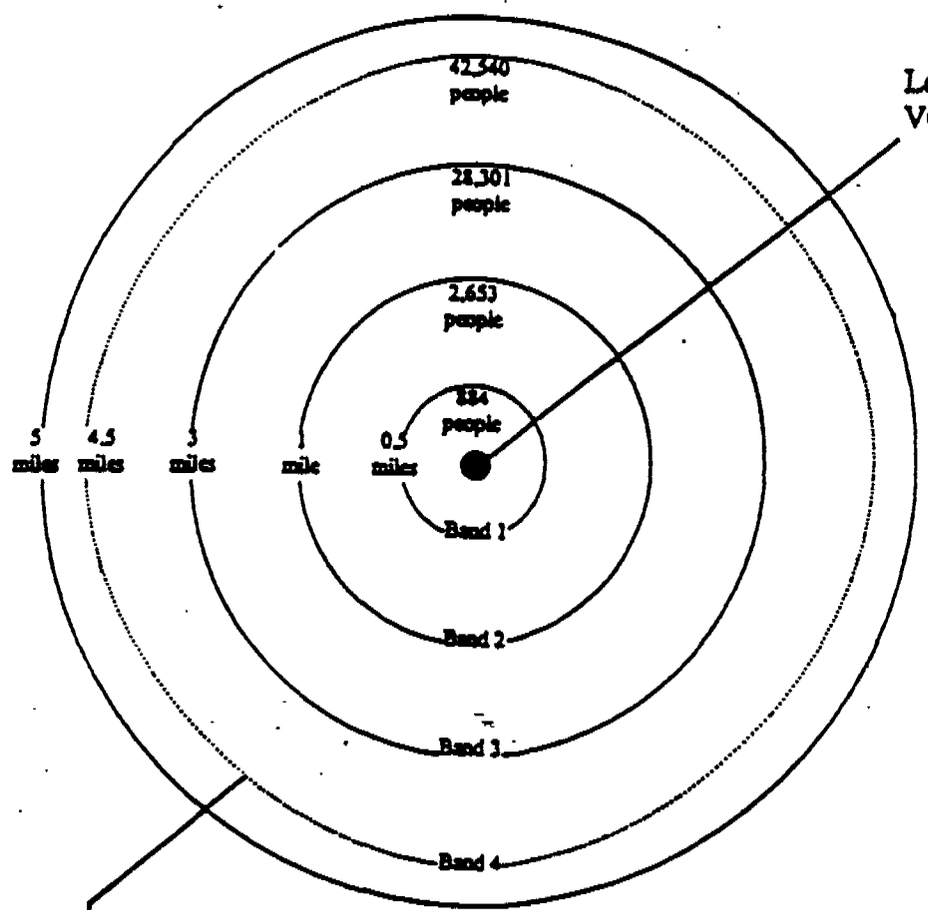
Method

We develop the estimates of the total affected populations (presented in Exhibit 2-5 of the body of the chapter) by summing affected populations across each of the four cities with VC or PVC manufacturing facilities. We employ the following method to estimate the affected population within each city.

- Step 1. Based on the area of each city, determine if the city has a radius of less than 5 miles (assuming the city is "round"). If so, determine the number of bands covered by the city (see Exhibit 2A-1 for an illustration for Sarnia, Ontario).
- Step 2. Calculate the area of each band within the border of each city.
- Step 3. Assuming a uniform distribution of population within each city, apply population density information for each city to estimate the number of individuals within each band.

Exhibit 2A-1

EXAMPLE OF GENERAL POPULATION SURROUNDING A VC OR PVC PLANT IN SARNIA, ONTARIO



Implicit in this approach are the following assumptions: (1) the VC or PVC plant is located in the center of each city, and (2) the population of the areas immediately outside of each city is zero.[22]

Results

Exhibit 2A-2 summarizes our results by city and population band.

Exhibit 2A-2

ESTIMATED POPULATION BY CITY AND BAND

City	Band				
	1	2	3	4	Total
Sarnia, Ontario	884	2,653	28,301	42,540	74,378
Niagara Falls, Ontario	246	739	7,885	65,050	73,920
Shawinigan, Quebec	1,186	3,559	15,186	0	19,932
Ft. Saskatchewan, Alberta	559	1,678	9,842	0	12,079

[22] While this second assumption is an over-simplification, it does not have a large impact on our results.

Appendix 2B

ESTIMATING GENERAL POPULATION CANCER CASES: DETAILED EXAMPLE

Overview

The purpose of this appendix is to provide a detailed example of the calculations perf rmed to estimate the total annual cancer cases in the general population living near VC and PVC plants in the absence of controls on VC emissions. The following example estimates the annual number of cases for the 1,444 individuals estimated to live in Band 1 around VC plants.

Example

- **Step 1: Calculate the Ambient Air Concentration of Vinyl Chloride.** From the U.S. EPA study, we apply an ambient air concentration of 113 ppb to Band 1 (0 to 0.5 miles from a VC facility). We converted this concentration from ppb to mg/m^3:

  - (113 ppb) * (1 ppm/1000 ppb) = 0.113 ppm
  - (0.113 ppm) * (2.6 mg/m^3 / 1 ppm) = 0.294 mg/m^3

- **Step 2: Calculate the Dose.** To estimate the dose to which each resident is potentially exposed, we multiplied the ambient air concentration of VC by the inhalation rate and the inverse of the average weight of an adult:

  - (0.294 mg/m^3) * (20 m^3/day) * (1/70 kg) = 0.084 mg/kg-day

- **Step 3: Calculate the Cancer Risk.** To estimate the individual lifetime risk of cancer, we multiplied the dose by the cancer slope factor for VC:

  - (0.084 mg/kg-day) * $(0.3\ mg/kg\text{-}day)^{-1}$ = 2.5×10^{-2}

- **Step 4: Calculate the Annual Cancer Cases.** To estimate the annual number of cancer cases in a given population, we multiplied the cancer risk by the population and divided by the average adult lifespan:

  - (2.5×10^{-2}) * (1,444 people) / (70 years) = 0.52 people/year

Appendix 2C

ESTIMATES USED TO GENERATE PROPOSED DISTRIBUTION OF VALUE OF LIFE

Author and Year	Value of Life - Best Estimate ($1995, millions)	Full Reference
Kneisner and Leeth (1991) (US)	0.8	T.J. Kneisner and J.D. Leeth. 1991. "Compensating Wage Differentials for Fatal Injury Risk in Australia, Japan, and the United States," *Journal of Risk and Uncertainty* 4(1):75-90.
Smith and Gilbert (1984)	1.0	V.K. Smith and C. Gilbert. 1984. "The Implicit Risks to Life: A Comparative Analysis," *Economics Letters*, 16: 393-399.
Dillingham (1985)	1.2	A. Dillingham. 1985. "The Influence of Risk Variable Definition on Value of Life Estimates," *Economic Inquiry* 24: 277-294.
Butler (1983)	1.5	R.J. Butler. 1983. "Wage and Injury Rate Responses to Shifting Levels of Workers' Compensation," In John D. Worrall, ed. *Safety and the Work Force*, Ithaca: Cornell University, ILR Press.
Miller and Guria (1991)	1.6	T. Miller and J. Guria. 1991. "The Value of Statistical Life in New Zealand," Report to the New Zealand Ministry of Transport, Land Transport Division.
Moore and Viscusi (1988a)	3.4	M.J. Moore and W.K. Viscusi. 1988. "Doubling the Estimated Value of Life: Results Using New Occupational Fatality Data," *Journal of Policy Analysis and Management* 7(3): 476-490.
Viscusi, Magat, and Huber (1991b)	3.7	W.K. Viscusi, W.A. Magat, and J. Huber. 1991. "Pricing Environmental Health Risks: Survey Assessments of Risk-Risk and Risk-Dollar Tradeoffs," *Journal of Environmental Economics and Management* 201:32-57.
Gegax et al. (1985) *	4.5	No full reference available
Marin and Psacharopoulos (1982)	3.8	A. Marin and G. Psacharopoulos. 1982. "The Reward for Risk in the Labor Market: Evidence from the United Kingdom and a Reconciliation with Other Studies," *Journal of Political Economy* 90(4): 827-853.
Kneisner and Leeth (1991) (Australia)	4.5	T.J. Kneisner and J.D. Leeth. 1991. "Compensating Wage Differentials for Fatal Injury Risk in Australia, Japan, and the United States," *Journal of Risk and Uncertainty* 4(1):75-90.
Gerking, de Haan, and Schulze (1988)	4.6	S. Gerking, M. de Haan, and W. Schulze. 1988. "The Marginal Value of Job Safety: A Contingent Valuation Study," *Journal of Risk and Uncertainty* 1(2): 185-200.
Cousineau, Lacroix, and Girard (1988)	4.9	J. Cousineau, R. Lacroix, and A. Girard. 1988. "Occupational Hazard and Wage Compensating Differentials," University of Montreal Working Paper.
Jones-Lee (1989)	5.2	M.W. Jones-Lee. 1989. *The Economics of Safety and Physical Risk*, Oxford: Basil Blackwell.
Dillingham (1985)	5.3	A. Dillingham. 1985. "The Influence of Risk Variable Definition on Value of Life Estimates," *Economic Inquiry* 24: 277-294

Appendix 2C (continued)

ESTIMATES USED TO GENERATE PROPOSED DISTRIBUTION OF VALUE OF LIFE

Author and Year	Value of Life - Best Estimate ($1995, millions)	Full Reference
Viscusi (78, 79)	5.6	W.K. Viscusi. 1978. "Labor Market Valuations of Life and Limb: Empirical Estimates and Policy Implications," *Public Policy* 26(3): 359-386. ---------- 1978. "Wealth Effects and Earnings Premiums for Job Hazards," *Review of Economics and Statistics* 60(3): 408-416. ---------- 1979. *Employment Hazards: An Investigation of Market Performance*, Cambridge: Harvard University Press. ---------- 1979. "Job Hazards and Worker Quit Rates: An Analysis of Adaptive Worker Behavior," *International Economic Review* 20(1): 29-58. ---------- 1979. "The Impact of Occupational Safety and Health Regulation," *Bell Journal of Economics* 10(1): 117-140.
R.S. Smith (1976)	6.3	R.S. Smith. 1976. *The Occupational Safety and Health Act: Its Goals and Achievements*, Washington: American Enterprise Institute.
V.K. Smith (1976) *	6.4	No full reference available
Olson (1981)	7.1	C.A. Olson. 1981. "An Analysis of Wage Differential Received by Workers on Dangerous Jobs," *Journal of Human Resources* 16: 167-185.
Viscusi (1981)	8.9	W.K. Viscusi. 1981. "Occupational Safety and Health Regulation: Its Impact and Policy Alternatives," J. Crecine, ed., *Research in Public Policy Analysis and Management*, vol. 2, Greenwich, Conn.: JAI Press, pp. 281-299.
R.S. Smith (1974)	9.8	R.S. Smith. 1974. "The Feasibility of an "Injury Tax" Approach to Occupational Safety," *Law and Contemporary Problems* 38(4): 730-744.
Moore and Viscusi (1988a)	9.9	M.J. Moore and W.K. Viscusi. 1988. "Doubling the Estimated Value of Life: Results Using New Occupational Fatality Data," *Journal of Policy Analysis and Management* 7(3): 476-490.
Kneisner and Leeth (1991) (Japan)	10.4	T.J. Kneisner and J.D. Leeth. 1991. "Compensating Wage Differentials for Fatal Injury Risk in Australia, Japan, and the United States," *Journal of Risk and Uncertainty* 4(1):75-90.
Herzog and Schlottman (1987)	12.4	H.W. Herzog, Jr. and A.M. Schlottman. 1987. "Valuing Risk in the Workplace: Market Price, Willingness to Pay, and the Optimal Provision of Safety." University of Tennessee Working Paper.
Leigh and Folson (1984)	13.2	J.P. Leigh and R.N. Folson. 1984. "Estimates of the Value of Accident Avoidance at the Job Depend on Concavity of the Equalizing Differences Curve," *The Quarterly Review of Economics and Business* 24(1): 56-66.
Leigh (1987)	14.2	J.P. Leigh. 1987. "Gender, Firm Size, Industry and Estimates of the Value-of-Life," *Journal of Health Economics* 6: 255-273.
Garen (1988)	18.4	J. Garen. 1988. "Compensating Wage Differentials and the Endogeneity of Job Riskiness," *The Review of Economics and Statistics* 70(1): 9-16.

Source: W.K. Viscusi. 1992. *Fatal Tradeoffs: Public and Private Responsibilities for Risk*. New York: Oxford University Press.
* Added after consultation with Viscusi.