INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

ProQuest Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600

University of Alberta

Perspectives on Environmental Exposure to Polychlorinated Biphenyl

By

Susan Mary Fossey

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

in

Medical Sciences – Public Health Sciences

Edmonton, Alberta

Fall 2000

University of Alberta

Library Release Form

Name of Author: Susan Mary Fossey

Title of Thesis: *Perspectives on Environmental Exposure to Polychlorinated Biphenyl*

Degree: *Master of Science*

Year this Degree Granted: *2000*

10951 72 Avenue,
Edmonton, Alberta
T6G 0B1

Date: *September 22, 2000*

University of Alberta

Faculty of Graduate Studies and Research

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled *Perspectives on Environmental Exposure to Polychlorinated Biphenyl* submitted by *Susan Mary Fossey* in partial fulfillment of the requirements for the degree of *Master of Science* in *Medical Sciences – Public Health Sciences.*

Kenneth L. Froese

Steve E. Hrudey

M-A. Armour

Douglas Wilson (Chair)

Date thesis approved by committee 27 Sept 2000

Abstract

The siting of hazardous waste treatment facilities can be a difficult task. Residents near potential sites often express such strongly negative views that placement of such industries in or near a community is virtually impossible. In less common instances, potential communities welcome these industries citing the economic advantages of diversification as a prime-motivating factor. Swan Hills, Alberta is such a community. In the mid 1980's the community successfully competed against other Alberta locations to host the Alberta Special Waste Treatment Centre. Following the development of the plant, a series of technical malfunctions occurred, potentially putting at risk the welcomed neighbor status of the plant. This study, conducted three years after the incidents, was designed to assess understanding in the community and the need for communication about human health risk associated with exposure to polychlorinated biphenyls through an environmentally mediated dietary pathway. It reports on perceptions of health risk among community residents and potential changes in the acceptability of and trust in the plant given the existence of a public health advisory about consumption of wild game and the intense political and media attention the facility has received.

This thesis is dedicated to my parents

Dave and Sheila Fossey

They may not understand what I'm doing but understand why I have to.

Table of Contents

List of Tables

List of Figures

List of Boxes

List of Acronyms

AEP	Alberta Environmental Protection
AhR	Arylhydrocarbon receptor
ASWTC	Alberta Special Waste Treatment Centre
ATSDR	Agency for Toxic Substances and Disease Registry
COPD	Chronic obstructive pulmonary disease
CIHD	Chronic ischemic heart disease
DDE	Dichlorodichlorophenylethylene
DDT	Dichlorodiphenyltrichloroethane
DNA	Deoxynucleic acid
DRE	Dioxin Responsive Element
EPA	Environmental Protection Agency (USA)
EROD	Ethoxyresorufin-0-deethylase
ETS	Environmental tobacco smoke
FVC	Forced vital capacity
FEV_1	Forced expiratory volume in one second
FPS	Fetal PCB syndrome
GLEMEDS	Great Lakes embryo mortality, edema and deformity syndrome
HREB	Health Research Ethics Board
I3C	Indole-3-carbinol
IARC	International Association into Research and Cancer
NIMBY	Not in my back yard
NK cells	Natural Killer cells
PCB	Polychlorinated biphenyl
PCDD	Polychlorinated dibenzo dioxin
PCDE	Polychlorinated diphenylether
PCDF	Polychlorinated dibenzo furan
PCN	Polychlorinated napthalene
PCQ	Polychlorinated quaterphenyl
TCDD	Tetrachlorodibenzodioxin
TCP	Trichlorophenol
TDI	Tolerable daily intake
TEF	Toxic Equivalency Factor
TEQ	Toxic Equivalency Quotient
TSH	Thyroid stimulating hormone
WHO	World Health Organization

Chapter One:

1.1 Introduction

Synthetic chemicals are an integral part of twenty-first century human existence. Many of the products we use every day are a direct result of the explosion of knowledge in the field of chemistry. Products ranging from prescription medicines and cleaning agents to synthetic fabrics and paper are all results of the chemical industry and positively affect our lives and health, increasing life expectancy and decreasing morbidity.

With increased chemical use also comes the potential for undesired exposure to hazardous chemicals. Exposure to hazardous chemicals in food, air or water is a familiar scenario to twenty-first century humans and is often equated in the public eye with adverse, dangerous or debilitating effects. Still other hazardous chemical exposures are seen as neither positive nor negative. What drives our perceptions of hazardous chemical exposure? How do we define risk amidst a myriad of potential exposure scenarios? What constitutes a dangerous level of exposure?

As our knowledge of chemistry has expanded and its products have infiltrated many parts of our lives, the incidents and sources of inadvertent exposure have increased as well. In many cases, people look to episodes involving industrial accidents as the primary source of hazardous chemical exposure. Perhaps this is because industrial accidents are often the most vivid and acutely hazardous reminders of the powerful impact of chemicals on human health. For example, one of the earliest recorded incidents occurred in Oppau, Germany on September 21, 1921. A chemical explosion at a nitrate manufacturing plant killed 561 persons, injured 1500 and destroyed both the plant and homes up to 4 miles away. Other examples are legion. On June 1, 1974 in Flixborough, United Kingdom, a railroad car carrying cyclohexane ruptured and released approximately 400 metric tonnes of flammable chemical. The resulting cloud exploded, setting off a fire over 20 acres of land. Twenty-eight people were killed, 89 injured and over 3000 evacuated from their homes. On April 8, 1979 in Crestview, Florida, 17 railroad cars carrying acetone, anhydrous ammonia, carbolic acid, chlorine and methanol

derailed. The subsequent explosions and fire injured 1000 people and caused the evacuation of 4500 more. In Mississauga, Ontario on November 10, 1972, 21 rail cars carrying caustic soda, chlorine, propane, styrene and toluene derailed. Three of the cars exploded and caught fire injuring 8 emergency workers and prompting the evacuation of 250,000 people.

Even more notorious incidents have occurred. Well known are the incidents in Bhopal, India on December 2-3, 1984 and in Chernobyl, Ukraine between April and May of 1986. In India, more than forty tonnes of methylisocyanate gas were released from a Union Carbide chemical manufacturing plant. The chemical cloud drifted over heavily populated areas killing 2500 people and seriously injuring 10,000. It is estimated that 20,000 people were left partially disabled and over 180,000 were adversely affected by the incident. The explosion at and meltdown of the Chernobyl nuclear reactor led to the release and distribution of radioactive gas over a wide area of the Ukraine and northern Europe. The damage to both the environment and the people inhabiting the area was extensive and the death toll from acute and long-term effects is still unknown (Wier, 1987).

What these incidents all have in common is the presence of hazardous chemicals in uncontrolled releases, human exposure and health consequences. These incidents are however, the exception, not the rule. Incidents involving hazardous chemicals that have caused neither loss of life nor loss of property are much more common but they do not engender the same feelings as major incidents even if exposure levels are comparable. The point in presenting these examples is not simply to emphasize the potential negative effects of hazardous chemicals on human health but to point out that hazardous chemical exposure either by design or by accident has occurred at high levels on numerous occasions.

The study of risk and the perception of risk have expanded enormously in the last two decades. Scholars from a diverse range of fields and occupations have combined to

produce an eclectic mix of opinions and theory that create the field of risk perception. It is a dynamic and ever-evolving topic.

Public reaction to potential facilities within a community is almost universally negative (e.g. Kuhn & Ballard, 1998). The term "NIMBY" or "**n**ot **i**n **m**y **b**ack **y**ard" was coined to describe the overall attitude surrounding hazardous waste siting difficulties. Such was the case in much of Alberta in the 1980's. Public surveys indicated a public awareness of a need to "do something" about hazardous waste so long as it did not include their community. That there are technical risks associated with hazardous facilities is not in question. The question is more, "Why do some communities accept and others reject the siting of hazardous waste facilities?" Many factors are involved and include variability in perception of risk, development of technological stigma and analysis of economic cost-benefit ratios. In Swan Hills, the community chose to accept the risks associated with the ASWTC and voted overwhelmingly in favor of the facility (Sherbaniuk, 1998). In the intervening years, despite an unwavering confidence in the plant among residents, there have been a number of highly publicized environmental contamination incidents and growing technical evidence of long-term environmental contamination.

1.2 Study context

In Alberta, the experience with hazardous chemicals has not been as dramatic as many of the global examples presented despite the presence of numerous industries with reliance on and production of hazardous chemicals. Until the late 1970's, there were no special provisions in the province for the treatment/disposal of hazardous waste. As a result and in an attempt to diversity the provincial economy, the Alberta government set out to site and develop Canada's first and only **stationary** hazardous waste treatment facility. Through a novel process of community participation and site elimination (versus the traditional process of site selection) the Alberta Special Waste Treatment Centre (ASWTC) was located and built fifteen kilometers northeast of the town of Swan Hills in north-central Alberta. The plant has functioned for roughly 10 years amidst considerable controversy, destroying Alberta and Canada's hazardous and refractory chemical waste,

including polychlorinated biphenyls (PCBs) (Figure 1.1 Geographic Location of the ASWTC).

The current study of this community and facility was prompted by a series of events surrounding the ASWTC starting from before its construction and coming to a climax at the end of 1996. This latest phase of controversy peaked on October 16, 1996 with a technical malfunction in an incinerator at the ASWTC. The result was an airborne release of PCBs and polychlorinated dibenzo-p-dioxins (hereafter referred to as dioxins) to the environment. The chemicals drifted southeast over recreational and forested land adjacent to the plant. After an extended internal assessment, the management company of the plant notified Alberta Environmental Protection (AEP) of the incident. AEP contacted Alberta Health who put into effect a health advisory limiting consumption of wild game and fish within a 30-kilometre radius of the plant. Concern from residents regarding personal exposure from this incident and a number of earlier emissions and spills prompted a human health impact survey by Alberta Health (including serum samples in willing residents) in the months following the incident.

1.3 Research Question and purpose

The purpose of this study is to provide a broad perspective on understanding in the community and the need for communication about human health risks associated with exposure to polychlorinated biphenyls and dioxins through an environmentally mediated dietary pathway. The research question was "Is there knowledge or insight available in the scientific literature that will be useful to residents of the community to address any concerns they may have about health risks associated with dietary PCB or dioxin exposure?"

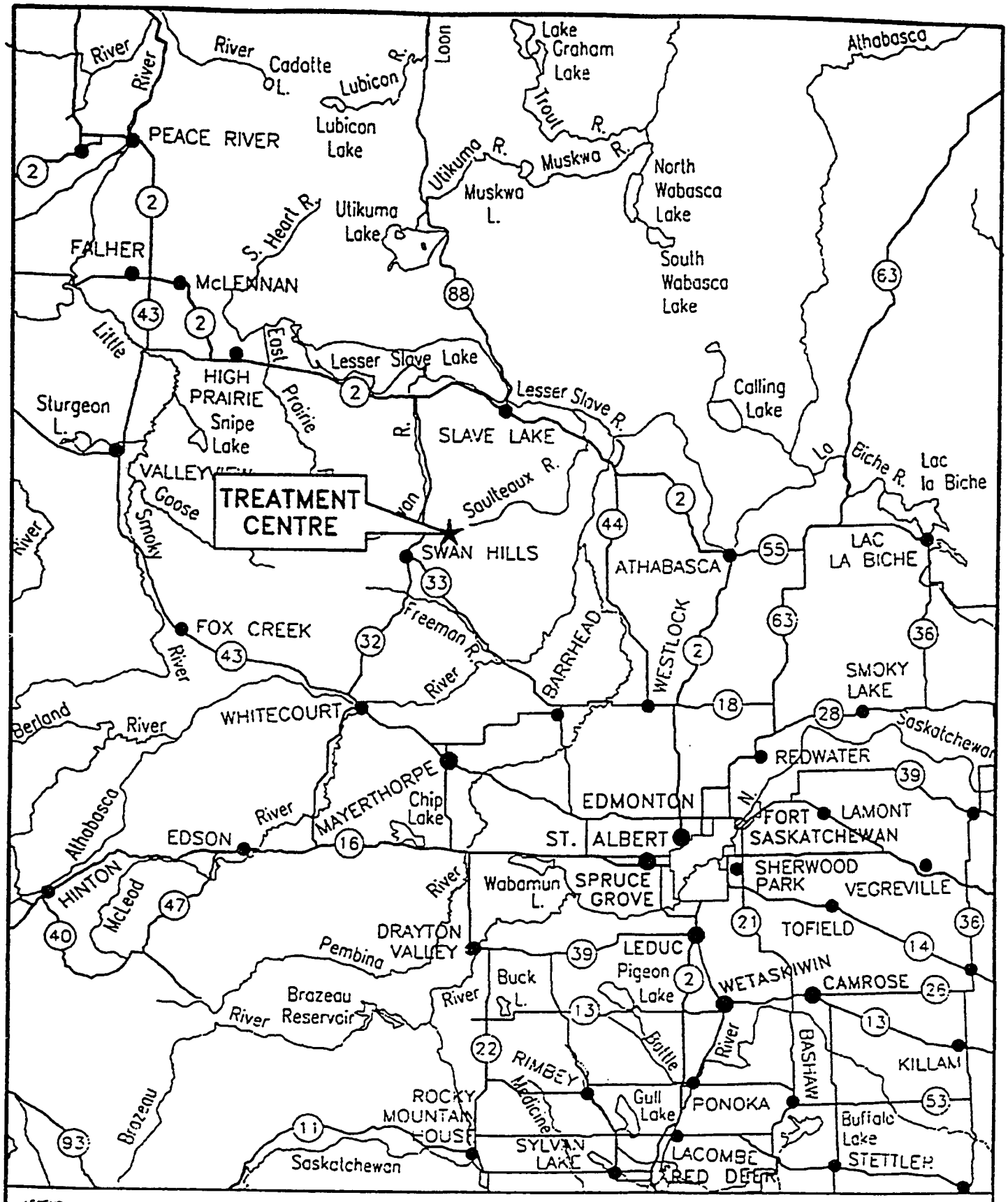


Figure 1.1 **Swan Hills Treatment Centre Location**

This study uses a combined quantitative/qualitative approach and the use of a 35-question survey tool. The majority of survey questions were modified open-ended questions designed to elicit semi-qualitative answers.

1.4 Chapter Summaries

This paper is divided into six chapters (including this introduction). Chapter 2 will review the physical and chemical properties of PCBs, production, uses, environmental fate and current sources. The chemistry of PCBs and dioxins is particularly relevant in understanding these chemicals and their potential to influence human health.

Chapter 3 will describe and attempt to put into context the extensive body of literature surrounding human health effects from PCB and dioxin exposure. Historically there have been a number of incidents that may provide useful insight into the Swan Hills scenario. There are many similarities between the historical incidents and the situation in Swan Hills although the differences are perhaps more important. Chapter 3 will begin with a look at two incidents in Asia that have formed the basis for knowledge of human health effects from PCB exposure. This section will be followed by an examination of incidents involving exposure to dioxin and ending with information on PCB exposure in Canada and the available health data surrounding Swan Hills itself. A cursory examination of the literature from the occupational arena will also be included.

Chapter 4 will look at risk perception and communication and its implications for the situation in and around Swan Hills. The role of science and direct evidence in understanding chemical exposure will be discussed.

Chapter 5 will summarize the design and approach used in this study, including a community profile and history of the events leading up to the initiation of this study. It will also report the results of the survey conducted in the community on perspectives of

environmental chemical exposure and discuss those results in the context of risk and PCBs.

Chapter 6 will conclude the paper with a look at the overall findings and recommendations for future research.

In environmental chemical and contamination scenarios, the amount of information available to persons potentially affected is varied and complex. Information sources can range from social contacts and family members to environmental activist groups and scientific advisors. Whether a risk is perceived more often than not depends on the context of the situation and the personal beliefs of involved persons. The availability of direct evidence and interpretation of scientific uncertainty all influence the degree to which a risk is perceived. In Swan Hills, the presence of the ASWTC is a dynamic and integral part of the community. Hindsight evaluations of the desirability of the plant may be influenced by a number of factors including media, available scientific information and personal experience. As one scholar remarked however, "If anyone understands the health risks associated with PCB exposure, it should be these people."

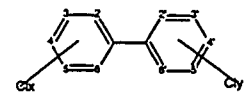
Chapter 2: Physical and Chemical Properties, Production, Uses, Fates and Sources

2.1 Introduction

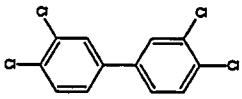
PCB is the term used to describe the entire class of non-polar hydrocarbons with a biphenyl nucleus, substituted with one to ten chlorine atoms. The general chemical formula is $C_{12}H_{10-n}Cl_n$ and the general structure is shown in Figure 2.1. In total, 209 different congeners are possible although few have been found in environmental media. PCBs may be classified in a number of ways such as on degree of chlorination. In such a system, there are 10 groups or homologues. A second PCB classification system divides the class on the basis of position of the chlorine substitutes (i.e., ortho, para, or meta). The position of substitute influences the degree of rotation between the benzene molecules. Isomers can assume planar/coplanar (the two benzene rings in the same plane) or non-planar (the benzene rings at a 90° angle to each other) configurations. The angle of conformation is important because it is a factor in determining some of the physical and toxicological properties of the congener. For example, highly ortho substituted congeners are highly soluble (Erickson, 1997) with the coplanar congeners having a higher degree of toxicity (Brucker-Davis, 1998).

A more discrete division of PCBs is based on binding to the aryl hydrocarbon receptor (AhR). In this classification scheme, there are 6 groups of PCBs: planar/coplanar, mono-ortho coplanar, mono-ortho without the single para chlorine, di-ortho, tri-ortho and tetra-ortho[1]. Coplanar PCBs are those congeners with both para positions, at least two meta positions but no ortho positions occupied by chlorine substituents. Structurally similar to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), these "dioxin-like" congeners strongly bind the AhR conferring on them a high degree of toxicity (Figure 2.2, 2.3). Mono-ortho congeners bind the AhR with moderate affinity and have moderate dioxin-like properties. Both the mono-ortho (no single para chlorine) and the di-ortho congeners are weakly dioxin-like. The di-ortho congeners also have recognized neurotoxic and estrogenic effects that are not AhR mediated. The last two types of PCB congeners are the tri-

[1] Biphenyl itself is co-planar

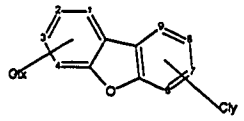


PCB general structure

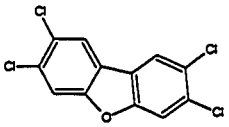


3,3',4,4' - tetrachlorobiphenyl
(PCB 77)

Figure 2.1: General PCB structure and example of planar PCB

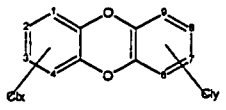


PCDF general structure
C12 H(9-n) 0 Cln

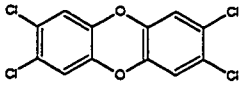


2,3,7,8 -tetrachlorodibenzofuran

Figure 2.2: General furan structure and example of planar furan

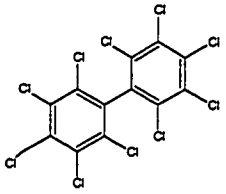


PCDD general structure
C12 H(9-n) Cln

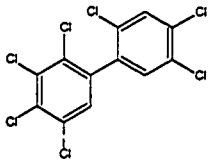


2,3,7,8- tetrachlorodibenzodioxin

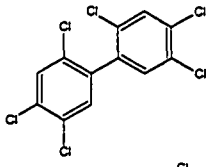
Figure 2.3: General dioxin structure and example of planar dioxin



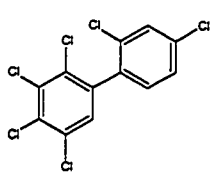
PCB 209:
2,2',3,3',4,4',5,5',6,6'
decachlorobiphenyl



PCB 180: 2,2',3,4,4',5,5'
heptachlorobiphenyl



PCB 153: 2,2',4,4',5,5'
hexachlorobiphenyl



PCB 138: 2,2',3,4,4',5
hexachlorobiphenyl

Figure 2.4: Some PCB congeners found in the Swan Hills Environment

and tetra-ortho congeners. They are not coplanar and do not exhibit affinity for the AhR (ATSDR, 1997).

2.2 Production and Uses

In North America, PCBs were produced from 1929 until 1977 for use primarily in capacitors and transformers. The major producer was the Monsanto Corporation of St. Louis, Missouri and they sold PCBs in a variety of formulations under the trade name Aroclor®. In much of the early literature on PCBs, the terms Aroclor and PCB are used almost synonymously. Aroclors are, however, always mixtures of several PCB congeners. Aroclors are identified by a four digit numbering code with the first two digits indicating the presence of a twelve-carbon parent compound (i.e. biphenyl) and the last two digits indicating the weight percent chlorine. For example, Aroclor 1260, one of the most common mixtures, was a mixture of chlorinated biphenyl molecules with an average of 60% chlorine by mass. In Aroclor 1260, the mixture has congeners generally ranging from those with five chlorine atoms to those with nine. Each lot of a particular mixture could be different and mixtures produced in other nations could have similar although not identical composition.

The commercial use of PCBs exploited their physical and chemical stability and led to the production and use of an estimated 635 million kilograms in the United States alone (93% of world wide production) (ATSDR, 1997). An additional 1.4 million kilograms were imported to the United States from other PCB-producing nations, notably Germany (under the trade name Clophen) and France (Phenoclor). Many other countries also produced PCB-containing products under a variety of names (Table 2.1). Global production estimates closely mirror U.S. production numbers as they were by far the major producers of PCBs. Numbers from the former Soviet Union are somewhat unreliable but are estimated at around 100,000 tonnes. PCBs were not produced in Canada although imports of up to 2450 tonnes per year (1970) did occur.

Table 2.1 Trade Names of PCB products and mixtures containing PCB

Aceclor	Dykanol	PCBs
Apirolio	EEC-18	Phenoclor
Aroclor	Elemex	Polychlorinated Biphenyl
Arubren	Eucarel	Polychlorobiphenyl
Asbestol	Fenchlor	Pydraul
Askarel	Geksol	Pyralene
Bakola 131	Hivar	Pyranol
Biclor	Hydol	Pyroclor
Chlorextol	Inclor	S-42
Chlorinated Biphenyl	Inerteen	Saf-T-Kul
Chlorinol	Kanechlor	Santotherm
Chlorobiphenyl	Kennechlor	Sotother Fr
Clophen	Montar	Santovac 1 and 2
Chlorofen	Monter	Siclonyl
Delor	Nepolin	Solvol
Diaclor	Nitrosovol	Sovol
Dialor	No-Flamol	Sovtol
Disconon	P-53	Therminal FR
Dk	P-296	Trichlorodiphenyl
Duconol	PCB	Turbinol

Source: Erickson, 1997

Commercial production of PCBs ceased in October 1977 in response to studies by Monsanto demonstrating extreme environmental persistence and in response to postulated links to adverse human health effects (Robards, 1990). Imports into the United States for disposal were banned in 1980 due to lasting concern about degraded water quality, persistence in wildlife and risk to public health (Anonymous, 1997). March 18, 1996 saw the United States Environmental Protection Agency (EPA) briefly overturn the import ban to allow for the importation of PCB containing waste (destined for incineration) from Mexico and Canada. This was in spite of highly vocal concerns over carcinogenic incineration breakdown products (specifically 2,3,7,8-TCDD). Public opinion was successful in having the ban reinstated shortly thereafter. In Canada imports of PCB containing waste have been allowed into the country for disposal since late 1999.

PCB use was historically divided into closed, nominally closed and open uses. The term "closed use" was restricted to PCB use in equipment from which release to the environment was controlled and unlikely. For example, PCB use in electrical equipment such as capacitors and transformers was the primary closed use. Nominal use was use from which escape was possible but over which some control existed such as hydraulic

fluids and specialized lubricants. Open uses were those with which control was impractical and included such uses as plasticizers in polyvinyl chloride, non carbon copy paper (Addison, 1986), other surface coatings, paints, inks, adhesives, pesticide extenders, micro encapsulation of dyes, immersion oils for microscopes, catalysts, casting waxes, cutting oils and lubricating oils (Erickson, 1997). PCBs were even used in kiss-proof lipstick (Robards, 1990).

"The overall picture of where Aroclors were used and, by inference, how they have entered the environment is not well characterized" (Erickson, 1997: 39) although the majority of PCBs produced were used in capacitors and transformers (more than 45% and 28% respectively). The last American PCB-containing transformers were produced in 1979 and with an average life expectancy of more than 30 years may still be expected to be in use (ATSDR, 1997). Capacitor life expectancy is somewhat shorter at 10-20 years depending on use and thus some capacitors may still be in use although less likely than transformers.

Open, non-electrical uses of PCBs (and hence inadvertent release of PCBs to the environment) likely account for a greater proportion of existing PCBs due to the lack of initial regulation and control. Specialized destruction of all these small-scale PCB products (as is required for large amounts of PCBs in transformers or capacitors) did/does not occur[2]. For example, non-carbon copy paper, as noted by the U.S. Federal Drug Agency (FDA), may contain 3-5% PCB (Erickson, 1997) or about 110 mg for a three part form. Rastogi (1992) found that seven out of eleven printing inks investigated contained PCB up to 180 ng/g. Concrete joint sealant containing 1-40% PCB was used extensively in (the former West) Germany in the construction of school and office buildings. In Sweden, 80-85% of all small boats and 50-60% of large ships had PCB-laden paint used to protect the metal from ocean water degradation (Jensen, 1972). In grain silo construction, a concrete curing agent containing up to 32.6% PCB after solvent

[2] The reader may notice the ambiguous use of tense throughout this section of the paper. This is intentional. Some uses of and exposures to PCBs still exist but were also historically important. It is difficult at times to separate the two.

evaporation was used; eventually organic acids in the silage eroded the coating, contaminating the grain (Willett et al, 1985).

The only continuing, legal production and use of PCB in North America is by microscopists as mounting media, as components of refractive index liquids and as immersion oils (ATSDR, 1997).

PCBs can also be produced as byproducts of other related chemical processes. The PCBs so produced range from single congeners to complex mixtures. Processes producing PCBs as byproducts include the production of chlorinated benzenes, vinyl chloride, chlorinated solvents, organosilicone drugs and pigments (Erickson, 1997). Because they are not generally part of records kept, estimates of PCB production as byproducts are crude at best (Rastogi, 1992). Such releases can be of significant amount.

2.3 Chemical and Physical Properties

Generally, PCBs are inert. They are acid and alkali resistant. They are not affected by oxidation or reduction. They are relatively insoluble in water with decreasing solubility as degree of chlorination increases. PCBs are soluble in non-polar organic solvents and biological lipids with bioconcentration factors in fish ranging from 10^3 to 10^7. They are slightly volatile and volatility of individual PCB molecules decreases with increasing chlorine content (Table 2.2). PCBs have impressive insulating capacity and are flame retardant with excellent dielectric properties (Brunelle et al., 1985). These properties, combined with being stable and non-flammable contributed to the range of PCB uses.

The physical constants for the commercially prepared mixtures (Aroclors) are comparable although often represented with some uncertainty. As stated above, each commercial mixture varied in congener composition, even from lot to lot. Environmental behavior may be completely different. Tables 2.2 and 2.3 present selected physical properties of PCBs and select commercial PCB mixtures and Table 2.4 presents current detection limits of PCBs, dioxins and other selected environmental chemicals.

Table 2.2: Physical Properties of PCB Homologs

PCB isomer group	Melting point °C	Boiling point °C	Vapor pressure @ 25°C (mmHg)	Water Solubility @ 25°C (mg/L)	Log K_{ow}	BCF in fish	Evap. Rate @ 25° C
Biphenyl	71	256	4.9	9.3	4.3	1000	0.92
MonoCB	25-77.9	285	1.1	4.0	4.7	2500	0.25
DiCB	24.4-149	312	0.24	1.6	5.1	6300	0.065
TriCB	28-87	337	0.054	0.65	5.5	1.6×10^{4}	0.017
TetraCB	47-180	360	0.012	0.26	5.9	4.0×10^{4}	4.2×10^{-3}
PentaCB	76.5-124	381	2.6×10^{-3}	0.099	6.3	1.0×10^{5}	1.0×10^{-3}
HexaCB	77-150	400	5.8×10^{-4}	0.038	6.7	2.5×10^{5}	2.5×10^{-4}
HeptaCB	122.4-149	417	1.3×10^{-4}	0.014	7.1	6.3×10^{5}	6.2×10^{-5}
OctaCB	159-162	432	2.8×10^{-5}	5.5×10^{-3}	7.5	1.6×10^{6}	1.5×10^{-5}
NonaCB	182.8-206	445	6.3×10^{-6}	2.0×10^{-3}	7.9	4.0×10^{6}	3.5×10^{-6}
DecaCB	305.9	456	1.4×10^{-6}	7.6×10^{-4}	8.3	1.0×10^{7}	8.5×10^{-7}

Source: Erickson, 1997

Table 2.3: Nominal Physical Properties of Selected Commercial PCB Mixtures

Property	Aroclor 1254	Aroclor 1260	Aroclor 1262	Aroclor 1268
Average Molecular Weight	328	375.7	389	453
Color	Light yellow	Light yellow	No data	No data
Physical State	Viscous liquid	Sticky resin	No data	No data
Boiling Point, °C	365-390	385-420	390-425	435-450
Solubility				
Water, mg/L	0.012,0.057	0.0027, 0.08	0.053	0.300
Organic Solvent	Very soluble	Very soluble	No data	Soluble
Log K_{ow}	6.5	6.8	No data	No data
Vapor Pressure, mm Hg @ 25° C	7.71×10^{-5}	4.05×10^{-5}	No data	No data
Henry's Law Constant, atm-m^3/mol @ 25° C	2.0×10^{-3}	4.6×10^{-3}	No data	No data
Conversion Factor	1 mg/m^3 = 0.075 ppm	1 mg/m^3 = 0.065 ppm	1 mg/m^3 = 0.061 ppm	1 mg/m^3 = 0.053 ppm

Source: ATSDR, 1997

Table 2.4 Detection Limits for Selected Chemicals

Chemical	Detection Limit[a]
Polychlorinated Biphenyl[1]	
Serum	0.1 to 2.5 ng/mL
Human Milk	3 pg/g (milk); 4 ng/g (fat)
Cow Milk	0.1-0.5 pg/g (congener specific)
Organs	12-33 ng/g (homogenized liver, kidney, brain tissue)
Fish	0.1-3 pg/g
Mammal Blubber	30 ng/g
Air	0.0006 mg/m^3 (50 L sample)
	10 µg/m^3 (4 L sample)
Drinking Water	0.08 –0.24 µg/L
	0.02 – 0.04 pg/L (congener specific)
Waste Water	0.065 – 36 µg/L
Hazardous Waste	60-70 ng/g
Soil/Sediment	80 ng/g
Polychlorinated dibenzodioxins[1]	
Air	0.01 – 5 pg/m^3
Incineration Stack Emissions	1 pg/m^3
Drinking Water	0.5 – 1.1 pg/L
Soil/Sediment	1-3 ng/g
Foods	10 pg/g
Beef fat	0.05 pg/g
Fish	1-50 pg/g
DDT[3]	
Blood	2 ppb
Urine	mg/ml
Human Milk	2 ppb
Adipose Tissue	0.25 ppm
Air	0.49 – 2.60 mg/m^3
Water	0.012 µg/L
Soil	0.012 µg/L
Food	10 –50 ng/g fat
DDE[3]	
Human Milk	2pg/mL
Adipose Tissue	2pg/g
Water	7 ng/L
Soil	300 ng/L
Food	10 – 50 ng/g fat
Mercury	
Air	3 µg/m^3 (elemental)
Water	– 2 ng/L (inorganic)
Drinking Water	~ 1.8 - 45 µg/L
Soil	10 µg/g
Fish	50 ng/g (methylmercury)

I. varies depending on analytical method used

II. 1. ATSDR, 1997; 2. ATSDR, 1998; 3. ATSDR 1994

2.4 Environmental Fate and Current Sources of PCBs in the Environment

PCBs are no longer produced yet the global burden remains high. The complexity of the class (i.e. among the 209 individual congeners) affects both the environmental fate and transport and the toxic properties exhibited by the mixtures. Currently, the presence of PCBs in the environment without specific localized sources is attributed to a complex global recycling system. Volatilization from terrestrial and aquatic surfaces into the atmosphere with subsequent removal from the atmosphere by wet or dry deposition is the primary means of PCB distribution (Erickson, 1997).

The current sources of PCBs for human exposure are many and are intricately linked to the environmental fate of these compounds. Sources include open and uncontrolled use (past and present), legal and illegal disposal practices, and accidental release. All have contributed to the global PCB burden. For the general public however, the main route of exposure to PCBs is through food consumption because PCBs are most readily soluble in fat (lipids). Occupational exposure is relatively rare since the cessation of production in 1977. PCBs did however top the list of accidentally released chemicals in the United States from 1988 to 1992 with 3600 reported accidents[3] (Erickson, 1997). This presumably led to occupational exposure of transportation and emergency personnel. The other 'common' exposure is fire fighters exposed to airborne PCBs during transformer fires. Relative to outdoor concentrations, indoor PCB concentrations during transformer fires can be very high.

Sources of PCB exposure are so numerous that there is not a region on the planet or a person in any region without detectable levels of PCBs in their system. This includes Canada where PCBs were not produced but only used and imported. There are also Department of National Defense sites (former Distant Early Warning line bases) in Canada's far north where decommissioned transformers are buried. The costs associated with transporting these transformers to southern waste disposal sites are prohibitive and as a result, they will likely remain buried and potential PCB sources indefinitely. PCBs

[3] This may be more indicative of strict reporting requirements than abundance of PCBs.

also come to Canada as a result of environmental processes. These will be discussed in the following sections. The majority of known PCBs in Canada are put into storage at the end of their useful lives. The only existing stationary facility in Canada for PCB destruction is in Swan Hills, Alberta.

2.4.1 Water

Water contaminated by PCBs is less an issue than other environmental media due to the chemistry of PCBs. PCBs are extremely lipophilic and partition out of water into organic components such as aquatic organisms and sediment. Water does have readily detectable though very low concentrations of PCB (Table 2.4). As in other media, volatilization is a significant factor in the cycling of PCBs out of water while deposition (either wet or dry) will return PCBs to the system. The Great Lakes are a high profile, heavily studied example of ongoing PCB contamination (Table 2.5).

Table 2.5: Environmental Levels of PCB in Water

Matrix	Location	Concentration (ng/L)
Water	Antarctic, marine	0.03-0.07
	Antarctic, snow	0.3-1.0
	Ocean water	0.3-4200
	North Pacific Ocean	0.2-1.1
	West Pacific Ocean	0.04-0.25
	Deep Ocean	0.03-0.6
	Atlantic	0.3-8
	Mediterranean coast	13
	General, mod. Polluted rivers	<50
	General, high polluted rivers	<500
Rain	Remote	0.1-10
	Marine	0.5-10
	Rural	1-50
	Urban	10-250

Source: Erickson, 1997

Table 2.6: PCB Cycling from the Great Lakes (in percentage)

Water Body	Volatilization	Sedimentation	Outflow to other bodies of Water
Lake Superior	86.6	11.4	2.0
Lake Michigan	68.1	30.6	1.3
Lake Huron	75.3	19.4	5.3
Lake Erie	46.0	45.2	8.8
Lake Ontario	53.4	29.3	17.3

Source: ATSDR, 1997

PCBs in water can also arise as a result of surface run-off, reflecting contaminated soil and deposition from the atmosphere (Loganathan et al, 1997). In the Buffalo River, New York, for example, Loganathan and colleagues found that PCB concentrations in storm sewer discharge were highest from a former industrial site that had previously been remediated for PCBs.

2.4.2 Air

Air is a significant source of PCBs (see Table 2.7) and whether over land or sea, PCB air concentrations can be significant. The vapor pressures of PCBs at 25 °C range from 1.1 mmHg for monochlorinated biphenyl to 7.6×10^{-6} mmHg for the decachlorinated biphenyl (Table 2.2). This range indicates significant potential for volatilization over long periods of exposure. The air (prior to 1980) in rural and marine locations typically had 0.1 to 1 ng/m^3. There were no disposal facilities prior to 1980.

The concentration of PCBs in urban air is generally attributed to the use of electrical equipment and fires although volatilization from waste disposal sites and bodies of water and/or through emissions from hazardous waste incinerators (Atkinson, 1987) can be significant but very localized because there are relatively few hazardous waste incinerators. It is estimated that the air over the continental United States contains approximately 18 tonnes of PCBs; some from volatilization and some from the estimated 842,000 tonnes of PCB incinerated annually (Anonymous, 1997). PCBs persist in the atmosphere and can be transported over long distances until removed from the atmosphere through wet or dry deposition. This mechanism has caused so-called pristine environments such as the Arctic to become contaminated with PCBs (Wania and Mackay, 1996).

Differential volatilization of the lower chlorinated congeners has been observed leaving behind the more highly chlorinated congeners. Half-lives of PCB in the atmosphere depend on the degree of chlorination (see Table 2.8), with the higher chlorinated congeners persisting for months to years. Transformation is slow and may depend on

reaction with hydroxyl radicals. Photodegradation is suspected to be an important tropospheric process in the destruction of PCBs (ATSDR, 1997).

Table 2.7: Representative Environmental Levels of PCB in Air

Matrix	Location	Concentration (ng/m^3)
Ambient Outdoor Air	Antarctic coast	0.06-0.2
	Canadian Arctic (81° N)	0.1-0.3
	Remote	0.02-0.5
	Great Lakes	0.1-5
	Rural	0.1-2
	Urban	0.5-30
	Lake Superior (spring)	0.2
	Lake Superior (fall)	0.065
	Various US Locations	0.02-36
	Marine air	0.05-2.0
	Atlantic Ocean	0.05
	Gulf of Mexico	0.2-0.9
	North Pacific Ocean	0.54
	North Atlantic Ocean	1.84
	West Pacific Ocean	0.06-1.2
	Bermuda	0.4
	Bloomington, IN	0.7-2.5
	Lake Baikal, Siberia	0.009-0.023
	Tokyo, Japan	20
	Matsuyama, Japan	2-5
	Sweden	<0.8-3.9
	Germany	5-10
	US	5
	US, landfills	2-18
	US, electrical substations	1-47
	US, transformer manufacture	17-5,900
	US, spill site	10-10,800
Indoor Air	US, after light ballast burnout	5,860
	Germany	440
	Switzerland	50
	Office bldg, PCB transformers	457
	Office bldg, no transformers	229
	Analytical Lab, UK	5-8
	US, general indoor	39-620
	US, indoor near spill site	<10-190
	NIOSH 10hr TWA	1000
	OSHA PEL, 8hr TWA	500,000

Source: Erickson, 1997

Table 2.8: Half Lives of PCB Homologs in the Atmosphere

Homolog	Tropospheric Half Life (days)	Photodegradation Half Life (days)
MonoCB	3.5-7.6	
DiCB	5.5-11.8	1.4
TriCB	9.7-20.8	0.71
TetraCB	17.3-41.6	0.62
PentaCB	41.6-83.2	

Source: ATSDR. 1997

Prior to 1979 when their production was discontinued, many types of electrical equipment had PCBs incorporated into heat dissipating equipment. The air in closed buildings with electrical equipment and fluorescent lighting (from PCBs in the ballast of fluorescent lights) often has high airborne PCB levels.

As with most other air pollutants, the indoor air concentrations of PCBs are greater than those found in outdoor air. In fact, indoor PCB concentrations are estimated to be roughly an order of magnitude (ten fold) higher than outdoor (Erickson, 1997). The open and nominally closed use products that contribute to indoor PCB concentrations are many. The use of PCB in caulking compounds was commonplace in Germany and their use has led to measurable levels in schools and office buildings. In one study (Fromme et al, 1996), schoolrooms averaged 110 ng/m^3 and childcare centres averaged 48 ng/m^3. Rudiger et al (1985) found that fifteen percent of German schools and three per cent of childcare facilities averaged over 300 ng/m^3, well above the German action level. Five percent of these schools had over 3000 ng/m^3 PCB. In one 21-room school that used a sealing material containing PCB to insulate the pipes of a heating system, 16 rooms had PCB levels between 1000 and 3000 ng/m^3 and five had PCB concentrations above 3000 ng/m^3 (to a maximum of 8000 ng/m^3) (Fromme et al, 1996). Until 1990, a sealing material used in the external joints of residential buildings contributed to indoor air pollution levels of up to 1000 ng/m^3 (Fromme, 1996).

PCBs were/are also used in fluorescent light ballast (Wallace et al, 1996; MacLeod, 1979) and are known to contribute to ambient indoor air concentrations. This and other sources may complicate trace analytical measurements by increasing incidental PCB concentrations. The Newton Research Building on the University of Alberta campus in Edmonton, Alberta has ambient PCB levels from a variety of sources that are high

enough to render trace analysis of environmental PCB samples very challenging (personal communication, Dr. X.C. Le, 1999).

2.4.3 Soil and Sediment

Soil and sediment can be a significant source of PCB contamination depending on the proportion of organic carbon present. The log octanol-water partition co-efficients for PCBs range from 4.7 to 8.3 indicating strong affinity for the organic carbon phase. Researchers estimate that greater than 90% of the global PCB burden is adsorbed to soil (Erickson, 1997). Once adsorbed, PCBs are immobile and persistent. The more highly chlorinated congeners are especially persistent and the half-lives range from months to years (ATSDR, 1997) depending on the degree of chlorination. As with tropospheric half-lives, soil/sediment half lives increase with degree of chlorination and the organic carbon and clay content of the soil or sediment. Leaching from PCB disposal sites is unlikely (and very slow). Bacterial degradation of PCBs has been observed but is a very slow process likely not contributing greatly to overall soil/sediment burden decreases. Table 2.9 lists a variety of locales and their associated PCB soil and sediment levels.

Table 2.9: Various Environmental Levels of PCB in Soil and Sediment

Matrix	Location	Concentration (μg/g)
Soil	Sweden	15
	Great Britain (background)	7.7 ± 4.3
	Great Britain - average 1990's	0.02-0.03
	Lake Baikal, Siberia	0.001-0.09
	Japan – agricultural	<1,000
	Japan – industrial	510,000
	U.S. – industrial	17-17,800
Sediment	Spill site	1,400- 61,000
	16 km downstream	600
	Sweden – atmospheric deposition	8-20
	Sweden – industrial	4-170
	U.S.	20-300
	Japan	<1,000
	Japan – polluted site	Up to 2,700
	Lake Baikal, Siberia	0.00008-0.006
	Great Lakes	Trace – 250

Source: Erickson, 1997

2.4.4 Plants and Animals

PCBs bioaccumulate, bioconcentrate and biomagnify. Bioconcentration is the uptake by plants or animals of a chemical from environmental media; biomagnification is the increase in concentration of that chemical from consumption of contaminated food (plant or animal); and bioaccumulation is bioconcentration plus biomagnification. There are three general mechanisms of PCB bioaccumulation in terrestrial plants; uptake from soil through the roots and translocation to aerial plant parts, uptake of airborne vapors or deposition of atmospheric particulates. As with other physical processes involving PCBs, the degree of congener chlorination influences the process. The lower chlorinated congeners are more likely taken up from the soil and incorporated into plants while the more highly chlorinated congeners are more likely involved in surface deposition (ATSDR, 1997). In fact, studies have found relative PCB concentrations on apple skin were higher than the flesh, illustrating the contribution of atmospheric deposition to PCB concentrations in food (c.f. Lovett et al, 1997).

Bioaccumulation (bioconcentration plus biomagnification) is a complex process and is highly dependent on the chemical properties of the involved chemicals. For a chemical to bioconcentrate, it must first have a high octanol-water partition coefficient. This ensures uptake into the organic phase and hence into the tissue of an organism. Biomagnification also requires chemical stability in water and other compartments of the food chain. In other words, the chemical must not be metabolized by the species in the food chain. Finally, it must possess low toxicity. A chemical that is too toxic that it kills the target organisms early in the food chain cannot biomagnify (Clarkson, 1995). PCBs fulfill all of these requirements and are part of a very small group of chemicals that do so. In addition, a 1998 study suggests that the PCB congeners that do bioaccumulate are in fact more toxic and persist longer in the body[4] than those that remain in the environment (Cogliano, 1998).

[4] This statement is debatable with disagreement among a number of authors.

An interesting example of how PCBs can infiltrate and biomagnify in the food chain was observed in an Indian community being investigated for PCB exposure. This particular community was not near a known source of PCB contamination nor were environmental media (including fish) contaminated but the blood levels of residents were appreciably higher than reference values. In the course of intense investigation, dietary analysis revealed that a local delicacy included loon eggs. Loons are migratory birds who spend their winters in the Chesapeake Bay area of the United States where fish are known to contain high PCB concentrations (Clarkson, 1995) thus consumption of this delicacy led to the observed PCB blood levels.

Biomagnification of PCBs by fish and mammals makes a significant contribution to the PCB intake of human consumers. There have been numerous studies of populations with high levels of fish/wild game consumption (e.g., Fitzgerald et al., 1996) and concomitant exposure to PCBs. Fish in particular have been called the biological integrator because of their propensity to accumulate environmental contaminants. Bioconcentration factors can range up to almost 1,000,000 (ATSDR, 1997) and of all edible game, fish often have the highest PCB concentration with estimates ranging from 0.03 to 190 μg/g in marine fish and 0.1 to 15 μg/g in fresh water fish (Erickson, 1997). Studies on consumers suggest a positive correlation between consumption of fish and PCB body burden. This is not to say that other animals do not have appreciable contaminant levels. Unfortunately, the large land mammals such as moose, deer and caribou (mainly herbivores) and the large marine mammals such as whale and seal (filter feeders or carnivores/piscivores) have been studied only to a limited degree. The smaller mammals that have been extensively studied often have PCB levels high enough to constitute a public health concern if they were in fact consumed by human populations (Langlois & Langis, 1995). Table 2.10 lists PCB concentrations in selected animals and fish. Note that concentrations of PCBs in fat will always be the highest for any organism because of the high K_{ow} of PCBs.

Table 2.10 PCB Concentrations in Selected Animals/Fish

Organism	Concentration Range (µg/g)	Reference
Marine		
Zooplankton	<0.003-1.055	Erickson, 1997
Shellfish	<0.003-7	"
Seals	3-212	"
Whales and dolphin	0.012-147	"
Fish		
Fresh Water	0.1-15	"
Marine	0.03-190	"
Birds		
North America	0.1-14,000	"
Europe	0.5-9,570	"
Eggs	0.1-434	"
Terrestrial Herbivores (ng/g)		
Caribou	11-52 (fat, wet weight)	Thomas & Hamilton, 1988
	9 ng/g (meat)	Kuhlein & Kinloch, 1988
	500 ng/g (fat)	"
White tailed Deer	33-253 (fat, wet weight)	AB Health, 1997
Marine mammal meat[1]	0.080	Chan, 1998
Narwhal		
Blubber	10.0 µg/g	Kuhlein & Kinloch, 1988
Matak	1.0	
Meat	0.1	
Walrus		
Blubber	0.25	Kuhlein & Kinloch, 1988
Meat	0.01	
Marine mammal blubber[1]	1.90	Chan, 1998
Terrestrial mammal meat[2]	0.006	Chan, 1998
Terrestrial mammal organs[2]	0.008	Chan, 1998

1. Includes Beluga whale, narwhal, ringed seal, walrus
2. Includes beaver, buffalo, and caribou. Goat, marten, mink, moose, muskrat, polar bear and rabbit

2.4.5 Food

The most prevalent source of PCBs for humans is through consumption of contaminated food. The bioaccumulating capabilities of PCBs result in the contamination of both terrestrial and aquatic food chains. Aquatic food chains appear to be particularly vulnerable to persistent chemicals and are perceived by many to be much less under human control. PCBs generally biomagnify at a much higher rate in aquatic food chains than in terrestrial food chains and fish generally contain the highest amounts of 'naturally acquired' (versus inadvertent localized contamination) PCBs. Diets high in fish therefore generally engender the highest PCB intake (Baht & Moy, 1997; Gerstenberger et al.,

1996). Several studies have confirmed this, finding a significant positive correlation between fish consumption and serum PCB levels (Laden et al., 1999).

The method of food preparation has a large effect on the ultimate PCB concentration. "In general, contaminant levels drop following extensive food preparation" (Conacher and Mes, 1993:5). Baking, frying, broiling, boiling, smoking and micro waving fish decreases PCB concentrations from 25-68% (Wilson et al., 1998). The decrease in PCB concentration due to cooking appears to be a function of initial chemical mass in the raw fillet (Sherer and Price, 1993) and may also be affected by extent of fat trimming prior to cooking.

Terrestrial food chains may also become contaminated, although contamination is more likely a direct result of human-related activities than environmental processes (Raszyk et al., 1996). There have been a number of incidents where animal feed has been inadvertently contaminated with PCBs, the most recent being a Belgian incident in February 1999 involving several poultry farms. In this incident, chicken feed was contaminated with PCBs closely resembling Aroclor 1260 resulting in PCB concentrations in eggs and chicken meat up to 250 times the tolerance level of 0.2 µg/g fat (Bernard et al, 1999).

Recent research has suggested that PCB exposure through food chain contamination exposes consumers to greater risk than direct PCB exposure. Bioaccumulated congeners tend to be more highly chlorinated. Some authors suggest that bioaccumulated congeners are more highly toxic[5] although strictly speaking, toxicity is not a function of proportion of chlorine but of proportion of coplanar congeners present. The EPA tiered approach to PCB risk assessment is based on this assumption. (See Appendix One).

[5] Strongly dependent on the definition of "toxic". Some authors prefer to differentiate between toxicity and toxic potential thus taking into account potential toxic effects not specifically mediated by interaction with the AhR.

Table 2.11 PCB Concentrations in Selected Foods

Food	Concentration (mg/kg)
Fish	1870
Fish byproducts	1170
Cheese	250
Shell Eggs	550
Milk	2270

Source: Erickson, 1997

Market basket studies are one method of estimating contaminant levels in food. In this type of study, 'typical' foods are purchased, prepared for consumption and combined to make up composite samples that are then analyzed for compounds of interest. Numerous studies have shown that considerable amounts of PCBs are available for everyday consumption world- wide (Schecter et al., 1997; Nendza, 1997; Seward and Jones, 1996; Krokos et al., 1996). Analysis of the 1992-1996 Canadian diet by Newsome et al. (1998) found that fish and butter had the highest concentration of total PCBs while milk and infant foods had the lowest concentrations. In total, dairy and meat contributed the most to a total intake of 5.7 ng/kg/day. A 1997 study by Lovett et al. that examined locally grown fruit and vegetables in Wales and England found that consumption of these items would contribute an additional 3% of the normal dietary intake of PCBs. In other studies, indirect exposure through food packaging materials has been shown to contribute significant PCBs to the diet (Robards, 1990). Generally, aquatic food products from Japan, South Korea, Hong Kong and Taiwan contain significant PCB concentrations (Kannan et al., 1997). The impact of contamination of such products may be greatly increased as the globalization of the food trade increases.

Similar to market basket studies are those that examine specific food items. A 1997 study by Schecter & Li analyzed common American fast foods. As expected, those with the highest fat content (i.e. Pizza Hut pizza) had the highest total PCB content (See Table 2.12).

Table 2.12 Dioxin and PCB in US Fast Food 1995

	Total PCDD/F (pg/g whole wt)	Total Dioxin TEQ (pg/g whole wt)	Dioxin-like PCB (pg/g whole wt)	PCB TEQ (pg/g whole wt)
McDonald's Big Mac® Hamburger	3.81-6.43	0.03-0.28	960	1.27
Pizza Hut's Personal Pan Pizza Supreme®	6.30-9.31	0.03-0.29	1180	1.28
Kentucky Fried Chicken® original recipe 3-piece meal	4.02-6.74	0.01-0.31	1170	1.29
Häagen Daz® Chocolate-chocolate chip Ice Cream	0.58-5.06	0.03-0.49	Unable to determine due to interference	

Schecter and Li. 1997

The Canadian diet has been studied by several researchers (Newsome et al., 1998; Conacher and Mes, 1993). In 1998 Newsome et al. reported that the average daily intake of PCBs was 5.7 ng/kg/day (1992-1996) and 6.4 ng/kg/day (1986-1988) with little change in congener distribution over the study period. The Canadian tolerable daily intake (TDI) is 1 µg/kg/day. The TEQ (based on congeners 105,118,156,157,189, 170,180; see Section 2.5) for the 1992-1996 period was 0.11 pg/kg/day. The World Health Organization TEQ allowable is 10 pg/kg/day. Dairy composites were the greatest contributor to TEQ at 49% of the total, followed by meat at 21%. Freshwater fish contained some of the highest concentrations of PCBs although considerable geographical variation occurred. Total PCB content of fast food was intermediate and similar to that of the meat composite. "Generally, concentrations in composites from Vancouver and Winnipeg were lower than those from (Toronto, Montreal, Halifax and Ottawa)" (Newsome et al., 1998). Most PCB exposure is from the dairy, meat and fish food groups.

A similar study of traditional Arctic food was carried out by Chan (1998). In addition to significant levels of pesticides and metals, traditional foods such as marine mammal blubber exceeded the Health Canada guideline level for edible portion of fish (2 µg/g) (See Table 2.13).

Table 2.13 Health Canada Guidelines/Tolerances for PCBs

Food Item	Value
Meat/Beef/Dairy products (fat basis)	0.2μg/g (200 ppb)
Eggs (whole egg less shell basis)	0.1μg/g (100 ppb)
Poultry (fat basis)	0.5 μg/g (500 ppb)
Fish (edible portion)	2 μg/g (2000 ppt)

Source: AB Health, 1997

2.5 Toxic Equivalency Factors (TEF) and Quotient (TEQ)

As mentioned in the previous sections, PCB as a general term refers to a group of compounds. In order to simplify analytical results and compare across classes of PCBs/PCDD/F, a system of toxic equivalencies was developed. Although not universally accepted, TEFs provide a means to describe the combined toxicity of these mixtures. See Figure 2.4 for an illustrative description.

Recall that some PCBs (and dioxins/furans) are structured such that there is a similarity between the physical layout of the biphenyl molecule and chlorine substituents and 2,3,7,8-TCDD (a.k.a. TCDD, see figure 2.2). It is this similarity in structure and consequent ability to bind with the arylhydrocarbon (Ah) receptor that forms the basis of the comparison. The Ah receptor is capable of binding certain organochlorine molecules on the basis of molecular structure - specifically 2,3,7,8- TCDD, other 2,3,7,8 – substituted PCDD/F, planar/coplanar PCBs, DDT/DDE, polychlorinated diphenylether (PCDE), polychlorinated naphthalene (PCN) and indole – 3-carbinol (I3C). When such molecules enter the body, they are carried by lipid molecules in blood and transported to the liver. In the liver, they bind to the Ah receptor. The activated receptor-ligand complex is translocated to the nucleus of cells. At this point, an interaction with specific sequences on DNA ("dioxin responsive elements" (DRE)) alters gene transcription and results in the production of mRNA and measurable activity of cytochrome P_{450} induction such as ethoxyresorufin-*O*-deethylase (EROD) (Sanderson et al., 1996). This is a distinct and specific interaction with DNA but this mechanism is not an example of DNA damage that is typically caused by DNA-reactive carcinogens or mutagens.

TCDD, designated the "most toxic compound", is assigned an arbitrary 'toxicity equivalency factor' equal to 1. Other structurally similar compounds are assigned lower values by comparison to TCDD. The PCBs with dioxin-like activity are limited to the coplanar, mono-*ortho* and di-*ortho* congeners. The sum of the toxic equivalency factors multiplied by their concentration is the toxic equivalency quotient (TEQ).

There are a number of difficulties associated with the use of TEFs. Among the criticisms of the TEF scheme is the fact that a limited number of congeners have a dioxin-like structure. The assigned values, (including TCDD =1.0) are arbitrary assignments (Table 2.13). In reality, the values may be both species- and response-specific due to observed additive and antagonistic interactions in PCB mixtures. TEF values are based on *in vitro* assays without confirmed human health effects. As well, an extremely wide range of values (>5 orders of magnitude) point to considerable uncertainty and there is growing evidence of PCB action not related to dioxin-like structure or Ah receptor interaction therefore not accurately (or appropriately) represented by the use of TEFs.

Table 2.14: Proposed TEFs for PCBs, Dioxins and Furans

Congener	IUPAC No.	TEF	Relative Potency range	TEF (Ahlborg)	TEF (WHO)
Co-planar PCB					
3,3', 4,4'- TetraCB	77	0.01	0.1-0.000007	0.0027	0.0005
3,4,4', 5- TetraCB	81	-	-	0.0000086	
3,3', 4,4', 5- PentaCB	126	0.1	0.8-0.003	0.40	0.1
3,3', 4,4', 5,5'-HexaCB	169	0.05	1.1-0.006	0.0016	0.01
Mono-ortho co-planar					
2,3,4,4'-TetraCB	60	-	-	0.0000085	
2,3,3', 4,4'-PentaCB	105	0.001	0.001-0.00003	0.0011	0.0001
2,3,4,4',5-PentaCB	114	0.0002	0.0004-0.00005	0.000095	0.0005
2,3',4,4',5-PentaCB	118	0.0001	0.0003-0.000009	0.0000083	0.0001
2',3,4,4',5-PentaCB	123	0.00005	0.0001-0.00001	0.000024	0.0001
2,3,3',4,4',5-HexaCB	156	0.0004	0.001-0.00001	0.000046	0.0005
2,3,3',4,4',5'-HexaCB	157	0.0003	0.0006-0.00006	0.000135	0.0005
2,3',4,4',5,5'-HexaCB	167	-	0.0000055	0.0000072	0.00001
2,3,3',4,4',5,5'-HeptaCB	189	-		0.0000085	0.0001
Di-ortho co-planar					
2,2',3,3',4,4'-HexaCB	128			<0.0000072	

2,2',3,4,',5'-HexaCB	138		<0.0000072	
2,3,3',4,4',6-HexaCB	158		<0.0000072	
2,3,4,4',5,6-HexaCB	166		<0.0000072	
2,2',3,3',4,4',5-HeptaCB	170	0.0001*	0.000016	0.0001
2,2',3,4,4',5,5'-HeptaCB	180	0.00001*		0.00001
All other PCBs		0		
Dioxins*				
2,3,7,8 – TCDD		1.0		
1,2,3,7,8 – PeCDD		0.5		
1,2,3,4,7,8 – HxCDD		0.1		
1,2,3,6,7,8 – HxCDD		0.1		
1,2,3,7,8,9 – HxCDD		0.1		
1,2,3,4,6,7,8 – HpCDD		0.01		
1,2,3,4,6,7,8,9- OcCDD		0.001		
Furans*				
2,3,7,8 – TCDF		0.1		
1,2,3,7,8 – PeCDF		0.05		
2,3,4,7,8 – PeCDF		0.5		
1,2,3,4,7,8 – HxCDF		0.1		
1,2,3,6,7,8 – HxCDF		0.1		
1,2,3,7,8,9 - HxCDF		0.1		
2,3,4,6,7,8 – HxCDF		0.1		
1,2,3,4,6,7,8 HpCDF		0.01		
1,2,3,4,7,8,9 – HpCDF		0.01		
1,2,3,4,6,7,8,9 - OcCDF		0.001		

Source: Data combined from Safe (1990, 1994), Smith et al. (1990), *Ahlborg et al. (1994). WHO (1993)

Figure 2.4: Understanding TEFs

The key to using TEFs is understanding equivalencies. With TEFs, the starting point is 2,3,7,8-TCDD. It is assigned an arbitrary value of 1.0. A comparable example is currency exchange. It too uses equivalencies or relative values and is an easy, illustrative parallel useful in understanding TEFs.

If we arbitrarily choose a reference currency to refer all others to (e.g. American dollars) we can rank all other currencies to it and calculate a relative value (i.e. potency).

Imagine we have a pocket-full of foreign currency. We need to know how much (in reference currency) all of it adds up to. This is comparable to a hypothetical environmental scenario where a number of PCB, PCDD and PCDF congeners are present.

United States: $10
Canada: $10
Japan: 100 Yen
Mexico: 10 Pesos
Germany: 10 Deutsche Marks

France: 10 Francs
Europe: 10 Eurodollars
Britain: 10 Pounds
Cayman Islands: 10 Dollars

2378-TCDD: 50 pg/g
23478-PCDD: 60 pg/g
223445-HxCB: 600 pg/g
3344- TCB: 500 pg/g
334455-HxCB: 800 pg/g

2378-TCDF: 50 pg/g
23478-PCDF: 60 pg/g
2234455-HpCB: 900 pg/g
33445-PeCB: 700 pg/g

Currency

Currency	Exchange rate	Amount	US$ equivalent
USD	1.00	$10	$10
CDN	0.68200	$10	$6.82
Yen	0.009461	100Yen	$0.18
Peso	0.099219	10 pesos	$0.99
Deutsch Mark	0.479801	10 marks	$4.80
Franc	0.142845	10 francs	$1.43
Euro	0.937001	10 dollars	$9.37
Pound	1.497100	10 pounds	$14.97
Cayman Islands	1.219512	10 dollars	$12.20
Total USD equivalents			$60.76

Congeners.

Congener	TEF (WHO)	Conc. (pg/g)	TEQ (pg/g)
2378-TCDD	1.00	50	50
2378-TCDF	0.1	50	5
23478-PCDD	0.5	60	30
23478-PCDF	0.5	60	30
223445-HxCB (PCB 138)	<0.0000072	600	0.0043
2234455-HpCB (PCB 180)	0.00001	900	0.009
3344-TCB (PCB 77)	0.0005	500	0.25
33445-PeCB (PCB 126)	0.1	700	70
334455-HxCB (PCB 169)	0.01	800	8
Total TEQs			190

Currency exchange rates are relative values of difference national currencies and are established by activity in the foreign exchange market. The foreign exchange market is simply transactions between countries for goods, services or hard currency. "Exchange rates ... can move against each other by 10 percent or 20 percent in a year" (Weisweiller, 1990).

There are two common methods for predicting exchange rates:

I. Qualitative method: takes into account factors such as the trade balance or the money supply situation and its likely effect on inflation and employment; currency reserves and changes in their size, overseas assets and overseas debts, the level of investment and the need for modernization or change in industry, the level of and the politically possible changes to exchange control, import duties and taxation, and the world economic situation and its likely impact on domestic production and consumption; short-term flows of capital; government philosophy; electoral prospects; market feelings; interest rates, land prices, and wage rates; social and economic pressures; the age and ability of management in industry and in banking and the efficiency and effect of trade union structures. Once all these factors are taking into account, a guess is made as to the value of the national currency.

II. Econometric Method: associated with charts and models and is largely dependent on computer modeling. An initial decision is made as to which economic indicators to include in the model and what weight to attach to them. The computer can then develop a logical pathway to a value for a national currency.

Both methods require a great deal of speculation and inference and may or may not result in the same estimation between different assessors. That is why different banks or currency exchange businesses will offer the consumer different exchange rates at the same time.

Similarly, TEF values are arrived at by committees who evaluate the available evidence for the determination of the toxic potential of a particular congener in the Ah-receptor mechanism. Often this is done by the EROD assay, using H4IIE rat liver cell lines, but not always. Resultant TEF values are an approximation of the "true" value. In some instances, at least 5 orders of magnitude difference has been reported for the experimentally determined TEF of a particular congener (Safe, 1994). The actual choice of a particular value for TEF depends on risk assessment and risk management issues, safety factor, and political influences. Keep in mind as well that TEFs describe the relative toxic potentials of the listed congeners within the context of a single biochemical mechanism. They may or may not be causally linked to a health outcome/toxic effect. Perhaps the single greatest difference between the currency exchange rate and the TEF is the use of more than one "standard" for TEFs (e.g. WHO, Safe, I-TEF etc.).

Chapter 3: Human Health Effects of PCB Exposure

3.1 Introduction

In environmental situations, PCB contamination is rarely purely PCB. Contaminants such as combustion by-products (e.g. dioxins and furans), pesticides (e.g. DDT and its breakdown products), and metals (e.g. lead and mercury) are commonly found in combination with PCB contamination. Each of these contaminants may have their own health effects that may mask the effects of PCBs, add to them, subtract, work in combination with or mirror those of PCBs. As an additional complication, while spoken of as a homogeneous group, the possible health effects from PCB exposure are widely divergent seeming to depend on a variety of chemical conditions including congener type, mix and concentration. All contaminants have health effects at high enough concentrations. Concern centres around potential health effects at PCB concentrations found under environmental concentrations.

The variety and range of proposed health effects from exposure to PCBs is enormous. PCB exposure has been suspected to be linked to a variety of cancers, liver disease, acute and chronic skin problems, immune deficiency, developmental delay and a host of other problems. The difficulty lies in assigning actual causality. PCBs are a group of 209 different congeners, differentiated by degree of chlorination, structure and toxicity.

Produced commercially from circa 1930 to 1979, the congener composition of commercial PCB mixtures was fairly well defined. In the environment however, the composition of these same mixtures varies widely as a function of environmental processes such as bio-transformation, degradation, and bioconcentration. In environmental media, mixtures have been shown to undergo selective degradation of some congeners and hence preferential concentration of others (bioconcentration). In mammalian systems, humans included, a similar process occurs with lower chlorinated congeners more readily and rapidly metabolized leaving a proportionately higher percentage of higher chlorinated congeners.

The literature surrounding possible human health effects from PCB exposure is large and complex. PCBs were never intended for human consumption. Acute effects are typically assessed on a case-by-case basis under circumstances of high dose, accidental poisoning. Assessment must take into account the variability in individual human physiology, co-contaminants and a multitude of other confounding variables to attempt to assign causality to PCBs. Assessing chronic exposure effects are even more difficult. Since the cessation of production in 1977 the only source of PCBs to the *general* public is low or ultra-low dose environmental exposure (likely food chain exposure) or accidental exposure from recycling or disposal sources. The relevance of acute effects to understanding chronic effects is debatable although it does provide some upper bounds to toxicity. If PCBs were super-toxic then acute exposure should have had extreme effects.

Because it is unethical to expose individuals, groups or populations to potentially toxic substances, researchers have had to rely on epidemiological studies to examine the potential effects of PCBs. Often this means relying on case reports, retrospective or historical studies, or case series involving only those persons most highly exposed or with an identified health deficit. There are numerous difficulties associated with this approach, not the least of which is accurate exposure assessment (i.e. knowing how much exposure to PCBs has occurred). Other considerations include extended latency periods for health effects, variable quality and generally advancing analytical ability, and difficulties associated with studying subtle health effects. As Vyner states in his 1988 book on environmental exposure,

> "...at this late twentieth century date, we know little more about the health effects of exposure to ... invisible industrial contaminants than fourteenth century Europeans knew about the cause and prevention of the Plague" (p.ix).

Such is the case with PCBs.

3.2 Toxicokinetics

The toxicological assessment of PCBs with regard to human effects (versus the toxicology in rodents, for example) is complicated by a number of factors. PCBs as a group, consist of 209 possible congeners, of which approximately 135 have been found to date in environmental media. Human exposure to PCBs is often in conjunction with other chemicals that have potential health effects and exposure can occur by all routes (inhalation, ingestion, and dermal absorption) depending on the circumstances. As well, the typical dose to which the general public is exposed is low or ultra-low making *in vivo and in vitro* assessment technically difficult.

The majority of studies on the absorption, distribution, metabolism and excretion of PCBs *in humans* has come from occupational exposure or accidental exposure through food consumption. These are studies of exposure to commercial mixtures of PCBs. In the case of occupational exposure, the PCBs to which persons are exposed are typically "clean", with little or no contamination by other chemicals. Exposure through food consumption is typically not clean with a variety of collateral contaminating chemicals in addition to PCBs. These scenarios are in contrast to environmental exposures in which the "mixture composition changes over time through partitioning, chemical transformation and preferential bio-accumulation" (Cogliano, 1988) and are accompanied by a variety of other chemicals. PCB mixtures that have bioaccumulated through the food chains are noticeably different from the original commercial mixture as food chains tend to concentrate higher chlorinated congeners resulting in much different (usually higher) TEQ.

Use of laboratory studies of animal response to PCB exposure typically entails the study of single congeners or commercial mixtures. As mentioned, these are chemicals that were never intended for human consumption or widespread exposure. The extreme persistence in environmental media and human tissue was also unexpected. Given these points, controlled study of the health effects was necessary but such studies were ethical only through animal study. There are many difficulties in extrapolating animal data to human

populations. Among the challenges in comparing animal and human data are varied anatomy, physiology[6] and susceptibility of lab animals (i.e. rats, mice, guinea pigs), size differences (which make direct dose comparisons difficult), and the differences in life span (2 years versus 70 years). The use of commercial preparations versus environmentally degraded mixtures, high doses versus low or ultra-low doses, and potential differences in toxicity of PCB congeners and contaminants (e.g. PCDD/F, PCQs, metals) also contribute to the difficulties in extrapolating animal to human data.

3.2.1 Routes of Exposure

There are many potential routes to human PCB exposure; inhalation, dermal and ingestion. Air outside of occupational or industrial situations has typically low PCB concentrations. In the absence of disposal facilities, the air prior to 1980 in rural and marine locations typically had 0.1 to 1 ng/m^3; urban areas could have up to 10 ng/m^3. The concentrations in urban areas are generally attributed to the use of electrical equipment and electrical equipment fires[7]. The air in closed buildings with electrical equipment and fluorescent lighting (due to PCBs in the ballast) often had higher airborne PCB levels. In general however, inhalation is not the major route of exposure in the general public with mean exposures under 1 ng/kg body mass. Inhalation exposure was historically more likely in occupational settings such as capacitor and transformer manufacture and today during improper disposal of PCBs and PCB-containing waste. In instances such as enclosed transformer fires (c.f. the Binghamton State Office Building fire) and the rare lightning strike to outdoor equipment, inhalational PCB exposure may be higher than expected. Dermal exposure to PCBs is rare except in those occupational cases where workers may be exposed during production of equipment or during disposal of used PCBs.

Ingestion is the most common route of exposure to PCBs in the general population. Consumption of contaminated food (especially fish) is the typical source. Other meats

---

[6] Pitot and Dragan (1996) points out a number of assumptions that must be made to extrapolate animal data to humans. These include assuming comparable metabolism, similar metabolic rate, similar response to a given dose, similar mechanism of toxicity, and a biological response dependent only on difference in size.

[7] Prior to 1979 when they were no longer produced, many types of electrical equipment had PCBs incorporated into heat dissipating components.

and dairy products may have significant PCB concentrations. In 1984, the average PCB concentration in fish was 0.39 ppm with market fish usually less than 1 ppm but sport caught fish were much higher. The PCB concentration in other meat products is usually much lower but these products are generally eaten in much greater quantity. Waterways such as the Great Lakes have been highly contaminated with PCBs from long-term industrial waste disposal practices. Suspended organic particles in water adsorb PCBs and then settle to the bottom of the lake or river. The body of water then acts as a long-term sink for the chemical with equilibrium between the water, sediment and suspended organic components eventually attained. Food chain exposure is inevitable due to contamination of the entire food chain from organic particles, invertebrates, bottom feeding fish and vertebrate insects, herbivorous and carnivorous fish and finally piscivorous fish. PCBs have been found to concentrate 10,000 to 1,000,000 fold as they concentrate up a food chain.

A significant amount of PCB may be mobilized from maternal stores and excreted through the breast milk thus exposing the nursing infant to considerable amounts of chemical. "Approximately 30% of the body's PCB burden can be eliminated via lactation resulting in a net increase of the offspring's chemical burden" (Tryphonas, 1998). The daily intake for breast fed babies is 1-2 orders of magnitude higher than for adults (on a body mass basis). Overall, the contribution of six months of breast-feeding to adult body burden TEQ is 12% and 14% respectively in boys and girls (Patandin et al., 1999). In some populations (notably the Quebec Inuit), measured breast milk levels are high enough to exceed government tolerance levels for infant foods although levels of PCB in breast milk are generally declining and are lower than those reported in Europe (Newsome et al., 1995). Interestingly, despite the fact that different PCB mixtures were used in different countries, the same gas chromatographic (GC) pattern is observed in a variety of nations. As with PCBs in general, most breast milk samples have at least detectable levels of PCB. A WHO study (1993) found 10-35 pg TEQ/g milk fat in breast milk in industrialized countries and less than 10 pg TEQ/g milk fat in developing countries.

Breast milk is often used to estimate PCB exposure and body burden because it can be obtained through non-invasive techniques and is a relatively accurate reflection of overall body burden. There are a number of considerations when interpreting breast milk PCB levels including: (1) this measure represents a very limited population group (i.e. women of reproductive age), (2) PCB content is a reflection of milk fat content which can vary widely depending on a number of factors including hind versus fore milk[8], time of day, colostrum versus mature milk, age of mother (positively correlated to PCB concentrations), parity, diet (i.e. fish consumption, dietary fat), period of previous breast feeding (negatively correlated to PCB concentration) and length of lactation and (3) there are a variety of ways of reporting PCB in breast milk (i.e. total PCB, lipid basis, milk fat basis; c.f. Kostyniak et al., (1999)).

Table 3.1: Breast Milk Tolerance Levels

USEPA	0.006 pg TEQ/kg BW
WHO tolerable daily intake	1-4 pg/kg body weight
ADD	1-3 pg/kg BW of dioxin-like compounds considered equivalent in toxicity to 2,3,7,8-tetrachlorodibenzodioxin

The conclusion of most breast feeding studies with regard to PCB contamination is that the effects from transplacental exposure rather than lactational exposure are more likely to be responsible for any potential health deficits observed in infants. Professional judgment is that the nutritional and immunological advantages of breast feeding outweigh potential contamination effects.

3.2.2 Absorption

Absorption of PCBs is dependent on the degree of chlorination of the congener with more highly chlorinated congeners more readily absorbed. For the most part, the extreme lipophilicity of PCBs limits gastrointestinal absorption although they are absorbed quite efficiently "on the coattails" of dietary micelles and through passive diffusion into lipophilic cell membranes in the gastrointestinal tract (ATSDR, 1997). The mechanism of

[8] Hind milk refers to the last milk taken at any one feeding; fore milk is the first milk taken at any feeding

absorption of inhaled PCBs is less clear although indirect evidence through congener pattern identification in body fluids does indicate its existence. Dermal absorption, while not usually a factor in environmental settings, can be significant in occupational settings. The mechanism of dermal absorption is not known (ATSDR, 1997).

3.2.3 Distribution

Ingested PCBs are rapidly cleared from the blood and accumulate in liver and muscle (ATSDR). From there, PCBs are translocated to adipose tissue for storage or distributed within the body according to tissue fat content. Exposure level, age, and duration and recency of exposure also affect distribution. Organs and tissues high in fat will have a higher level of PCBs, especially adipose tissue and breast milk. There are two exceptions. The brain has lower PCB concentrations than would be predicted based on lipid content alone and the liver has higher PCB concentrations. Accumulation in liver and brain may be related more to blood perfusion rates than to actual lipid content, i.e. the brain has lower blood perfusion to lipid ratio while the liver has a higher ratio. Adipose tissues have by far the highest concentrations of PCBs. PCBs are able to cross the placental barrier to some extent (depending on structure and degree of chlorination).

3.2.4 Metabolism

"Information regarding the metabolism of PCBs in humans is limited" (ATSDR, 1997). Metabolism of PCBs is believed to occur by the cytochrome P-450 enzyme system resulting in the formation of polar metabolites that are conjugated with glutathione and glucuronic acid prior to excretion. As with absorption, the degree of and position of chlorination is a factor in the metabolism of PCBs with higher chlorinated congeners less likely to be metabolized and more likely to accumulate[9]. Lower chlorinated congeners are primarily "transformed into hydroxylated derivatives that are ... eliminated in the urine" (ATSDR, 1997).

3.2.5 Elimination

---

[9] Roughly true for homologues, but from Cl_5 to Cl_{10}, their metabolism/bioaccumulation is highly congener specific

Elimination of PCBs from the body is very slow. Estimates of half-lives range from months to years (ATSDR, 1997). The primary routes of excretion are fecal and urinary. Elimination is by first order kinetics with highly chlorinated congeners excreted in the feces and lower chlorinated congeners excreted in the urine (ATSDR, 1997). An important route of PCB excretion is through breast milk. Due to the lipophilic nature of these compounds, large amounts of PCBs may be mobilized and excreted depending on the volume and fat content of the milk.

3.2.6 Factors affecting toxicity

The general toxicity of PCBs is related to their structure. As mentioned earlier, there are 3 general categories of PCB; the co-planar (dioxin-like) PCBs, the non- coplanar, and the mono-ortho congeners. The coplanar PCBs appear to act like their structurally similar counterpart, 2,3,7,8-dibenzo-p-dioxin, binding to a cellular protein receptor (Ah receptor). There is increasing evidence of a second mechanism for PCB metabolism not involving the Ah receptor. For these non-dioxin-like PCBs, the mechanism of action is less clear although "a diverse spectrum of...toxic responses" (Giesy & Kannan, 1998) has been noted.

3.3 Health Effects

3.3.1 The short summary

As alluded to above, PCBs as they are encountered in occupational or environmental settings, are not single compounds. They exist as they were marketed - complex mixtures of congeners. As such, they pose both analytical and toxicological challenges. Each congener is associated with different physical, chemical and toxicological properties. The range of *potential* environmental and human health effects is extensive and has engendered both concern and controversy.

PCB exposure has not been linked to any acutely fatal conditions and the only confirmed human health effect of singular PCB exposure is chloracne, a disfiguring skin condition.

Blood chemistry results are mostly negative. That is, there is no statistically positive association between PCB serum levels and any particular health effect. When a positive association is noted, the effects are generally small with questionable clinical significance. For example, when serum PCB levels are high, the liver enzymes SGOT and GGTP[10] are often slightly elevated, suggesting an association with liver dysfunction (Kreiss et al., 1981). An association between serum PCB levels and increased serum triglycerides has been observed in some studies. This led researchers to investigate an association between PCB exposure and heart disease (e.g. Akagi and Okumura, 1985; Gustavsson and Hogstedt, 1997). Results have been inconclusive.

The liver has been an especially popular target for studies on the health effects of PCB exposure because of its role in detoxifying chemicals. There are reports of hepatomegaly (in the absence of alcohol or drug abuse) associated with PCB exposure but again definitive results are lacking (James et al., 1993).

Reproductive, developmental and immune effects are the latest interests in recent literature. As part of a group of chemicals colloquially known as "hormonally active agents', the co-planar or dioxin-like PCBs have been implicated in a number of endocrine system effects. The majority of work has been done on rodents (limiting the generalizability to human populations) and in linking observations of patterns in wildlife populations to human health effects (c.f. Our Stolen Future, T. Colborn et al., 1997). Nonetheless, animal studies have found a number of effects such as increased spontaneous abortions and decreased ability to conceive in rats. In human populations, there is some data supporting similar difficulties with conception in the Yusho cohort (c.f. Section 3.3.3). Unfortunately, cross-contamination by furans in the Yusho incident make such results difficult to interpret. Occupational studies with elevated blood serum levels have not observed this effect.

Developmental effects related to PCB exposure have also been difficult to define. Many studies have been criticized for conclusions based on neuropsychological tests not

[10] Serum glutamic-oxaloacetic transaminase and gamma glutamyl transpeptidase, respectively.

specifically designed to study environmental exposure effects. Accurate exposure assessment is also a problem as is controlling for the multitude of confounding variables that affect cognitive development. Three large cohorts of children have been studied extensively with varying and sometimes conflicting results. These results will be discussed in section 3.3.3.

Immune system effects from PCB exposure have been studied extensively and linked to the actions of the hormonally active agents. Results are inconclusive at best with suspected associations based on observations of increased susceptibility to bacterial and viral infection, generalized malaise and non-significant increases in T-cell numbers.

3.3.2 Target organ effects:

The focus of this section is human health effects following oral PCB exposure, therefore the health effects reviewed here will not include (for the most part) those due to inhalation or dermal exposure. Table 3.2 summarizes the following review.

1. Liver and Hepatobiliary System

The liver has been the target of much of the overall research effort on human exposure to PCBs. The liver is one of the main target organs of PCB distribution, second only to adipose tissue in overall PCB body burden. The main finding of the majority of studies with respect to liver function is that PCBs are capable of inducing microsomal liver enzymes. The clinical significance of the changes are unclear. Studies of occupationally exposed populations have shown rapid and complete return to normal enzyme levels upon cessation of exposure (Kimbrough, 1995). Persons exposed to heat-degraded PCBs[11] (i.e. PCBs that have been exposed to heat at levels below that intended for incineration) have exhibited elevated mortality from liver disease (not cancer). Statistical significance was reached in Japanese women (Ikeda et al., 1985; Hsieh et al., 1996) and a substantial elevation in overall mortality from chronic liver disease and cirrhosis was noted in Taiwan (Yu et al., 1997). Authors speculated the increased mortality may be a

[11] The term heat-degraded PCBs is used in this context to refer to PCBS that have been exposed to heat at non-incineration levels, for e.g. during the incidents in Taiwan and Japan where PCBs were used as a heat transfer medium.

function of a combination of factors including exposure to PCBs, PCDFs and infectious agents such as the hepatitis B virus. Reports of hepatomegaly (increased liver size) have been unable to link morphological change with dysfunction.

2. Stomach and Gastrointestinal System

Studies of PCB exposed workers have reported a variety of gastrointestinal symptoms ranging from anorexia and weight loss, to nausea, vomiting and abdominal pain. While exposure was mainly through inhalation, PCBs from swallowed expectoration or licked lips may have contributed to the symptoms (Emmett et al, 1988; Fischbein et al, 1979).

3. Skin and Musculoskeletal System

Chloracne is the only confirmed effect of acute and chronic PCB exposure and is characterized by multiple, closed comedones[12] and pale yellow cysts on the skin. Similar in appearance to juvenile *acne vulgaris*, chloracne has been observed as early as 1936 in occupational cohorts exposed to organochlorine products. Although most frequently seen on the cheeks of the face, forehead and neck of exposed persons, comedones and cysts may be found on the shoulders, chest, back, buttocks, genitals and abdomen.

In Asia in the late 1960's and 1970's, dermatologic signs were used almost exclusively to diagnose patients of PCB intoxication. The major dermal symptoms of Yusho were

[12] colloquially known as a "black head"

acneform eruptions (seen in 81.7% of registered patients), enlargement and elevation of the follicular orifice (70.1%), dry skin, hyperkeratotic plaques in the palms and soles, deformation of the nails, changes in hair condition, hyperpigmentation (especially of the corneal limbs, conjunctiva, gingivae, lips, oral mucosa, and nails), dyshydrosis, and ocular signs (such as hypersecretion of the meibomian glands). Table 3.5, section 3.4.1 displays the grading system used by physicians in diagnosing Yusho. Hypertrichosis (excessive growth of hair) and increased skin fragility are also common signs of exposure to heat-degraded PCBs (Higuchi, 1976). The variety of skin effects seen under these circumstances may be more a reflection of the variety of compounds present in contaminated rice oil than PCB effects alone (i.e. PCDD/F, PCQ).

Chloracne has not been associated with low-level environmental exposures (Shields et al. 1992) nor is it common in low-level occupational exposures. Based upon the sum of dioxin-like TEFs, the human serum level of Aroclor 1254 necessary to produce chloracne has been estimated at 6,200 ppb (Brown & Jones, 1981)[13], far above typical environmental exposure levels.

The only other skin and musculoskeletal effect associated with PCB exposure has been reported in men exposed to heat-degraded PCBs. These men reported a higher incidence of herniated vertebral discs and arthritis than controls at a 14-year follow-up (Guo et al., 1999).

4. Cardiovascular system

Neither the heart nor the vascular system appears to be affected by PCB exposure. One report out of the United States found an association between PCB exposure (through fish consumption) and hypertension, specifically increased diastolic blood pressure (Kreiss et al., 1981). The average serum PCB level in this group of people (n=458) was 17.2 ppm and conclusions were based on one blood pressure measurement per subject. There was no control group and subjects were exposed to DDT as well. Attempts to replicate this finding have been unsuccessful.

---

[13] The level of precision cited by the author is not realistic. A more accurate estimate might be 6000 ppb)

In persons exposed to heat-degraded PCBs, Akagi and Okumura (1985) found no association between blood pressure and PCB serum levels 13 years after contamination despite more than half of the subjects having higher than average serum PCB levels. In this study, blood pressure changes were correlated with age, obesity and alcohol intake.

Occupational studies have likewise found no credible association between PCB exposure and cardiovascular disease. Gustavsson and Hogstedt (1997) did find increased mortality from cardiovascular disease among male capacitor workers but did not control for smoking habits, which are a known cause of cardiovascular-related mortality.

5. Nervous system

There is no evidence of a direct neurotoxic effect from PCB exposure. One of the common manifestations of exposure to heat-degraded PCBs was headache and numbness, tingling and pain in the limbs. Extensive study of these persons revealed a typically transient condition indicating an acute/chronic stress response. No sign of central nervous system involvement was found despite abnormal electroencephalogram (EEG) in some (Chia and Chu, 1985). Investigation into causes of numbness, tingling and pain in the limbs found decreased sensory nerve conduction but no effect on motor nerve conduction (Higuchi, 1976). Clinically, most patients did not achieve the diagnostic criteria for true peripheral neuropathy. In aging adults exposed to low levels of PCB through food chain exposure, Schantz et al. (1999) found no effect on hand steadiness or visual motor coordination.

Acute and long-term exposure to PCBs have been reported to cause neurological and unspecific psychological or psychosomatic effects such as headache, dizziness, nausea, depression, sleep and memory disturbances, nervousness, fatigue and impotence (WHO, 1993).

In children, there is concern that exposure to PCBs in utero has long term, permanent effects on neurological development. Central to the investigations is the belief that PCBs

may disrupt the action of thyroid hormone. This will be discussed further in the Developmental Section (3.3.3).

6. Immune system

Many studies have sought to show a causal association between PCB exposure and adverse immune effects. There is in fact a substantial body of evidence in laboratory animals and wildlife demonstrating such an association (Golden et al., 1998). Extrapolating these results to humans has been done with limited success.

Studies of exposure to heat-degraded PCBs report clinical symptoms that could be interpreted as immune deficits. Exposed persons complained of increased susceptibility to bacterial and viral infection with general malaise and weakness. A study by Swain (1991) found that PCB levels in breast milk[14] could be positively correlated with incidence of child-hood infection. These latter authors suggest this could reflect an immune system effect from PCB exposure although further study and follow-up were not done and numerous other confounders exist. Other studies have not found the same association (Rogan et al., 1996).

Studies of immune system cellular components have reported varying results. Exposure to heat-degraded PCBs resulted in a number of immunotoxic effects including changes in cellular and humoral immunity (Tryphonas, 1998) such as slight leukocytosis, monocytosis and decreased levels of IgA and IgM (Higuchi, 1976). One year after exposure, Yu-cheng patients had decreased IgM and IgA but not IgG; decreased percentage of total T-cells, active T-cells and helper T-cells, normal percentage of B-cells and suppresser T-cells (Lu & Wu, 1985). By the next check-up 3 years later, most parameters had returned to normal. Occupational studies of persons exposed to "clean" PCBs found no effect on immune cell types or numbers (Swain, 1991; Tryphonas, 1998).

Studies of environmental exposure to PCBs have reported varying results. Dewailly et al. (1992) found that breast fed Inuit infants in Northern Quebec had lower T-helper/T-

[14] The source of PCBs in breast milk is usually consumption of contaminated food i.e. typically PCBs that have bioaccumulated through the food chain and not typically heat-degraded as was seen in the Yusho/Yu-Cheng incidents.

suppresser cytotoxic cell ratios than bottle-fed infants. The Inuit of Northern Quebec have high body burdens of PCBs due to high consumption of contaminated fish and marine mammals. Clinical significance of such changes were not addressed.

Svensson et al. (1994) examined parameters of immunological competence in Swedish men. They found that high consumption of fatty fish species resulted in lowered proportions and numbers of natural killer (NK) cells[15]. The decline was however statistically non-significant with unknown clinical significance. The study did not reveal any correlation with plasma immunoglobulins or liver enzyme activity.

A study of a cohort of Dutch children examining breast- versus bottle-fed children found that breast fed (and thus more highly PCB exposed) children had increased total T lymphocytes and cytotoxic T cells at 18 months and lower monocyte and granulocyte counts at 3 months of age suggestive of an immune effect (Weisglas-Kuperus et al, 1995). Clinical significance is uncertain.

Some authors suggest that statistical significance alone is not enough to warrant concern and that a four fold change in immune cell numbers would be necessary to be considered clinically meaningful (Tryphonas, 1998). This magnitude of change has not been observed with respect to PCB exposure.

7. Endocrine System

There are many studies examining the endocrine effects of PCB exposure (Ashby et al., 1997; Brucker-Davis, 1998; Golden et al., 1998; Hauser et al., 1998; Maclusky et al., 1998; Sher et al., 1998; Foster, 1998; Porterfield, 1998; Weiss, 1998). This area of study is in fact one of the most controversial of all proposed PCB-related health effects.

The controversy began in the mid 1990's and came to a crest in 1997 with the publication of *Our Stolen Future* by T. Colborn and associates. In the book, Colborn et al. suggest that chemicals in the environment at low or ultra-low doses are disrupting the normal

[15] Natural killer cells are a subset of lymphocytes responsible for combating viral infections (Roitt et al.

functioning of the endocrine system resulting in widespread damage to the reproductive abilities in a multitude of species. Based almost exclusively on observations in wildlife populations, the book goes on to warn of devastating consequences in the human species if exposure to (mostly chlorine containing) chemicals is not immediately discontinued. The theory has garnered enormous interest in both popular and scientific culture despite an almost total lack of human based evidence. The systems about which there are specific concerns are those involving the thyroid hormones and the sex hormone, estrogen.

7.1 The thyroid gland is located in the anterior neck and functions in the normal individual by secreting thyroxine (T_4) in response to stimulation by thyroid stimulating hormone (TSH) from the anterior pituitary gland. T_4 is the precursor to the active form of the hormone, triiodothyronine (T_3) that acts to regulate the rate of cellular respiration and contributes to proper growth and development, particularly during childhood. In utero, thyroid hormones are essential to neurodevelopment and the fetus is exquisitely sensitive to changes in hormone level. Thyroid deficiency or excess during critical developmental periods can lead to permanent neurodevelopmental effects (Porterfield, 1994).

Structurally similar to T_3 and T_4 (Figure 3.1a, 3.1b), coplanar PCBs are thought to compete for binding sites on serum carrier proteins thus decreasing total thyroid hormone concentrations (Hauser et al., 1998) and to compete for binding to thyroid hormone transport proteins in the blood (Porterfield, 1994). Depending on the concentration and

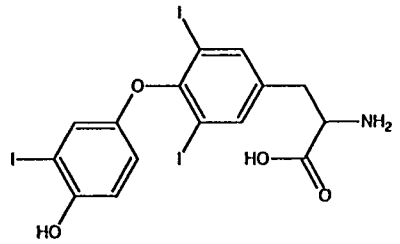


Figure 3.1 (a): Triiodothyronine (T3)

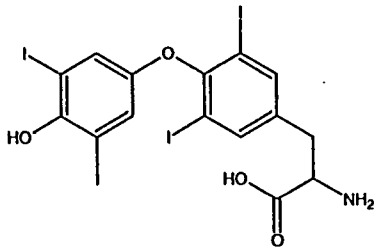


Figure 3.1(b): Tetraiodothyronine (T4)

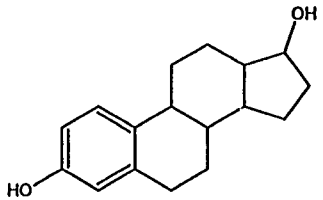


Figure 3.2: Estradiol -17 beta

congener, PCBs could theoretically act as agonists, antagonists, and partial agonists to thyroid hormone (Porterfield and Hendry, 1998). PCBs have been associated with decreased T_4 and T_3 and increased TSH and gland weight in adults. Typically, these findings are not associated with clinical symptoms (Brucker-Davis, 1998; Osius, 1999). Both men and women exposed to heat-degraded PCBs reported higher incidence of goiter (enlargement of the thyroid gland) without accompanying hypo- or hyperthyroidism (Guo et al., 1999)

7.2 Despite early concerns of fatal adrenocortical dysfunction related to heat-degraded PCB exposure, only altered menstrual cycle with the menstrual interval being either prolonged, shortened or irregular (Higuchi, 1976) has been noted with confidence. Other estrogen-related effects that have suspected PCB interactions include breast cancer, endometriosis, decreased sperm quality, cryptorchidism (undescended testicles), decreased penis size, effects on male and female fertility, alterations of sexual behavior. learning disability or delay, testicular cancer, and prostate cancer. Evidence is not conclusive for any of these (Golden et al, 1998).

8. Renal System

There is no evidence of a direct or indirect nephrotoxic effect from PCB exposure.

9. Respiratory System

Inhalation is not the usual route of exposure to PCBs. Respiratory effects following ingestion of PCBs have not been reported (ATSDR, 1997). There are reports from fires involving PCB-containing equipment of choking and difficulty breathing but such effects are difficult to separate from the effects of smoke and heat.

In persons exposed to heat-degraded PCBs, about 40% of patients reported atypical bronchitis-like symptoms and copious expectoration (Nakanishi et al., 1985). In these cases, respiratory symptoms were often complicated by secondary infection, typically *Candida*, *Staphylococcus* and *α-Streptococcus* (Higuchi, 1976).

Reduced forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) have been noted in one occupational study (Emmett et al., 1988). In this study, smoking was not controlled despite its known negative association with pulmonary function. Pulmonary function tests are often judged unreliable as they rely heavily on technician experience and subject effort thus limiting their usefulness for studying effects of exposure to different individuals.

10. Sensory Organs

There is no evidence of an effect from PCB exposure on the senses of taste or smell. In animal studies of congenital hypothyroidism, there are marked hearing deficits reported. This could be a problem in human *in utero* exposure to PCBs but comparable human studies do not exist. Conjunctivitis (inflammation of the conjunctiva of the eye), swelling, and discharge from swollen Meibomian glands of the eyelids were common signs of exposure to heat-degraded PCBs (Higuchi, 1976). Some Yusho victims reported decreased visual acuity (Higuchi, 1976).

11. Reproductive system

In conjunction with the endocrine system, the reproductive system has been one of the most highly studied systems in relation to PCB exposure. Animal studies have suggested some transgenerational effects but human studies of this nature are unknown. There have been reports from neither the Yusho nor the Yu-Cheng cohorts of increased congenital anomalies despite the passage of adequate time for exposed adults and children to reproduce.

There are suggestions of decreased reproductive success in persons exposed to PCBs. Fetal loss has been estimated at 15-20% in women exposed to heat-degraded PCBs (Hsu et al., 1985). In the Yu-cheng cohort in Taiwan, 39 infants showing hyperpigmentation indicative of high level in utero PCB exposure were born in the 15 years following identification of the cohort. Of those, eight died of pneumonia, bronchitis, sepsis and premature and congenital weakness (Hsu et al., 1985). Leoni et al. (1989) found significantly higher serum PCB levels in women hospitalized for miscarriage compared

to controls (women hospitalized with full term pregnancy). In this study, age and alcohol consumption, which are known risk factors for miscarriage, were also positively correlated with incidence of miscarriage thus weakening support for causal association with PCB exposure.

A change in the ratio of male to female births (more females than males) has been reported in some populations, specifically Seveso, Italy (Bertazzi et al., 1998). Exposure in that population was primarily to dioxins and no PCB exposure was involved. A similar change has been suggested but not confirmed for populations exposed to PCBs. Vartiainen et al. (1999) did not find evidence of a sex ratio change in Finland over a 250 year period despite widespread low level PCB contamination throughout the population.

In utero PCB effects have been extensively documented for high-level exposure. Infants exposed in utero to heat-degraded PCBs often display what has come to be known as Fetal PCB Syndrome (FPS). This syndrome is also known as Coca-cola baby syndrome after the dark skin pigmentation noted on birth. Other signs of the syndrome are gingival hyperplasia, exophthalmic edematous eye, dentition at birth, abnormal calcification of the skull, rocker bottom heels, and a high incidence of low birth weight (Yamashita and Hayashi, 1985). These children were also reported to experience developmental delay that continued for years after birth. The variety of effects associated with this syndrome may reflect the variety of contaminants found in the rice oil consumed by the mothers during this incident[16].

There are three main cohort studies that have examined the relationship between PCB exposure and infants. The results of these studies conflict. Some report decreased birth weight (160-190 grams) and head size (0.6 to 0. 7 centimetres) (Jacobsen and Jacobsen,

[16] Some of the other contaminants involved in the Asian incidents included polychlorinated quaterphenyls (PCQs), polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs).

1996; Fein et al., 1984; Higuchi, 1976) while others find no association between birth weight and total PCB concentration, PCB-TEQs or individual PCB congeners (Rogan et al., 1986; Vartiainen et al., 1998).

As Vartiainen points out, there are a number of confounding factors that must be carefully controlled when studying birth weight. First-born infants usually weigh less than second- or third-born infants. The mothers' diet and health status are especially important factors to consider, as are alcohol and tobacco use. Breast-feeding history is another confounder that must be considered as large amounts of PCB may be mobilized during breast feeding thus reducing body burden for subsequent pregnancies and breast feeding. The Jacobsen studies in particular have been severely criticized for methodological problems and inaccuracies in exposure assessment. This topic will be dealt with in more detail in the developmental section (3.3.3).

12. Blood System

There are studies linking PCB exposure with metabolic blood disorders. Specifically there are studies linking PCB exposure with serum hypertriglyceridemia (Okumura et al., 1974; Higuchi, 1974). Hypertriglyceridemia was a hallmark of the Yusho incident, second only to dermatologic criteria in prevalence of diagnosis. Elevated serum cholesterol has been noted in a number of studies but the relationship disappears when corrected for age, sex and body mass (ATSDR, 1997).

The only other blood system related effect that has been reported in association with PCB exposure is in women exposed to heat degraded PCBs. They reported higher incidence of anemia than controls at a 14-year follow-up (Guo et al., 1999).

3.3.3 Non Organ Directed Toxicity

1. Cancer

The International Association for Research on Cancer (IARC) classifies PCBs as probable human carcinogens (limited human evidence, sufficient animal evidence). As mentioned earlier, cancer is the focus of many of the early toxicologic and risk assessments associated with PCBs. Kimbrough (1995) notes that "not all PCB formulations are probable human carcinogens and ... different PCB mixtures do not have the same quantitative potency to cause cancer" (p.152).

Polychlorinated biphenyls are classified as probable human carcinogens based on the weight of evidence from rodent studies. Long-term studies on occupationally exposed persons do not support this classification. In occupational PCB exposure, blood levels of PCBs are often higher than in those persons accidentally exposed, there are fewer dermatologic signs and fewer related health effects. Occupational exposures are more likely to be "clean" PCBs (i.e. without high levels of additional contaminants). Long-term studies of accidentally exposed populations are inconclusive. Many of the difficulties arise from the complexity of the class and determining the effects of individual congeners and in differentiating the effects of PCBs from those of contaminants such as dioxins.

Virtually all animal carcinogenicity studies focus on the effects of PCBs on the liver. The conclusion is that PCBs may act as promoters in cells initiated spontaneously or by other previously administered carcinogens. PCBs may enhance or inhibit carcinogenesis by virtue of their stimulation of liver enzymes that in turn may alter the metabolism of subsequently administered carcinogens. PCBs show little or no genotoxic effect. They are negative in the Ames assay, micronucleus test and the V79 Chinese Hamster assay. PCBs do not produce chromosomal alterations or dominant lethal mutations in rats.

The dioxin-like PCBs are reported to have anti-estrogenic properties thus suggesting a protective effect against estrogen dependent cancers. Other congeners have displayed

very weak to weak estrogenic activity. Adami et al. (1995) found no evidence that PCBs in general and organochlorines specifically cause increased susceptibility to breast or endometrial cancer.

An extensive review of the literature by Ahlborg et al. (1995) also found no evidence to support the hypothesis that environmental levels of PCBs would lead to increased incidence of estrogen-dependent breast cancer.

Two important points must be kept in mind. First, many studies hypothesize that degree of chlorination and placement of the substituents (i.e. the coplanar congeners) are most associated with potential health effects. Second, contamination of PCB mixtures is not taken into account. These two points make environmentally related exposure and risk assessment very complex. There is evidence that PCBs degrade differentially under environmental conditions and during distribution and re-distribution around the globe (c.f. Wania and Mackay, 1996). In addition, accidental release to the environment is often in conjunction with application of heat (i.e. accidental fires, waste incineration processes) and thus the congener and contaminant concentrations may be very unlike the original product.

Studies of carcinogenicity of PCBs in humans have been inconclusive. Most studies do not show a clear association with cancer in any system. This conclusion is supported by a recent study by Kimbrough et al. (1999) which failed to find significant elevations in any cancer in an occupational cohort of more than 7000 people.

2. Developmental effects

Observation of the impact of environmental chemicals was first observed in fish and wildlife, specifically in the Great Lakes basin (c.f. Colborn, T., Our Stolen Future). "The effects are often mild or moderate and the key question of the real consequences in humans in still unresolved" (Brucker-Davis, 1998). One theory related the effect of environmental chemicals on thyroid hormones, which are essential for normal cognitive

development in infants (see section 3.3.2 point 7). "Frank thyroid effects, including altered thyroid hormone concentrations, have been observed only in humans exposed to... PCBs at levels far in excess of background" (Golden et al., 1998).

There have been two major studies examining the effects of transplacental and lactational exposure to PCBs. In the Michigan Maternal and Infant Health Study (Jacobson and Jacobson, 1996, 1997), researchers studied health effects related to consumption of contaminated fish. They found that children with PCB levels at or slightly above background levels had lower birth weight, shorter gestational periods, smaller head circumference and lower IQ at school age.

The second major study took place in North Carolina. Rogan et al. (1994) was unable to replicate the findings of the Michigan group beyond similarities in reflexes and tone at birth. By two years of age, children in this cohort were indistinguishable from control children. Findings were not dose-dependent and were observable only in children from the highest exposure group.

The third major study was an attempt to reconcile the findings of the Michigan and North Carolina groups (Patandin et al., 1999b). Researchers assessed children at 42 months of age for performance on cognitive function tests. Two groups were examined based on whether or not mothers planned to breast feed their infants. Researchers found lower birth weight, decreased postnatal growth, psychomotor development delay, neuro-developmental delay, and alterations in thyroid hormone and immune status in children exposed to background PCB concentrations[17]. They did not find an association between lactational exposure to PCBs or dioxins nor current PCB body burden (in the children) and cognitive ability. These results are comparable to the Michigan studies and with studies of the Yu-Cheng cohorts although some caution must be exercised in comparing exposure data due to differences in analytical methods used. (See Table 3.4 for further discussion).

---

[17] Given that all persons have measurable amounts of PCB in their adipose tissue, all children are exposed in utero to PCBs. This study attempted to compare those children exposed **only** in utero to those also exposed after birth through consumption of PCBs in breast milk.

The debate over the true developmental effects from in utero PCB exposure is less than clear despite the efforts of these three groups of researchers. For example, Seegal (1999) feels the suggestion of neurocognitive deficits in children exposed either in utero or through lactational transfer to PCBs may be due either to contaminants other than PCBs or to complex interactions between PCBs and other neurotoxicants. For example, Eskenazi and Castorina (2000) reviewed the literature on environmental tobacco smoke (ETS) and found evidence to suggest ETS exposure could cause subtle changes in children's neurodevelopment and behaviour. Murata et al. (1999) noted that children exposed in utero to methylmercury via maternal fish consumption were "likely to suffer delays in neurological development".

One of the difficulties with assessment of neurodevelopmental effects is associated with comparing PCB serum levels to PCB milk levels to umbilical cord PCB blood levels. Tilson et al. (1990) believes that 1 mg/kg of lipids in the mother's milk is the level after which neurotoxic effects may be seen in infants; this is equivalent to a level of 5 μg/L of lipids in blood from the umbilical cord. Muckle et al. (1998) believes these figures correspond to 2 mg/kg of PCBs in breast milk given differences in analytical methods from those used by Tilson in the early 1980s.

> "This level of mg/kg of lipids in breastmilk corresponds to a level of 2 μg/kg of lipids in the mother's blood. These concentrations are the equivalent of 5 μg/L in umbilical cord blood, since the average concentration of lipids in blood taken from the umbilical cord is approximately 2.5 g/L (Muckle et al, 1998: S23).

Using these figures, Muckle et al. (1998) showed that a great proportion of Inuit babies born in the Baffin region and the lower and mid north shore of Quebec are highly exposed. The clinical effects of such levels, while potentially worrisome, are unknown at this point.

3. Genotoxic[18] Effects

There is limited evidence of transgenerational effects in laboratory animals due to PCB exposure. Evidence from human studies is negative with little or no evidence from highly exposed populations in Asia despite adequate passage of time for children of exposed parents to have reproduced.

[18] refers to genetic alteration in the form of gene mutations, chromosome aberrations and changes in chromosome number

Table 3.2: Summary of Human Health Effects from PCB Exposure

Target Organ or System	Effect	Reference	Critique
Liver and Hepatobiliary System	Excess mortality in Yusho males and females - significant only in females	Ikeda et al. 1985	The liver is one of the major sites of metabolism in the human body. Evidence of induction of liver enzymes does not necessarily signify dysfunction. PCBs are highly resistant to degradation of any kind and prolonged elevation of liver enzymes may be a simple reflection of ongoing processes. The lack of clinical effect despite elevated levels argues for a no effect stance.
	Elevated mortality from liver disease (not cancer) inYu-Cheng cohort	Hsu et al. 1985 Hsieh et al. 1996	
	Occupational studies have found a relationship between serum PCB and induction of microsomal liver enzymes without significant clinical effect; For example: increased GGTP increased SGOT	Kreiss et al. 1981	
	No clinical evidence for toxic effects on the liver even in highly exposed populations such as transformer workers	James et al. 1993	
	Hepatomegaly		
Stomach and Gastrointestinal System	Anorexia and weight loss Nausea and vomiting Abdominal pain	Emmett et al, 1988 Fischbein, 1979	Reports of effects on this system have been limited to occupational case studies where exposure is primarily through inhalation. GI exposure may have come through swallowed expectorations or licking lips. Subjective complaints have not been linked to physical abnormality.

Skin	Chloracne Hyperpigmentation, Hypertrichosis Increased skin fragility, dyshydrosis Nail deformation Yusho cohort – acneform eruption decreased markedly within 3 to4 years; black comedones within 5-6 years; follicular dots within 4 years; pigmentation within 10 years	Jones and Alden, 1936 Higuchi, 1976 Brown et al., 1991 Shields et al., 1992 Urabe and Asahi, 1985	Chloracne is the only confirmed, uncontested health effect of PCB exposure. There is however no evidence of an effect at low or ultra-low environmental levels. Skin effects other than chloracne are likely not due to PCB as they have not been observed in persons highly exposed to "clean" PCBs.
Cardiovascular System	There is no evidence for a direct cardiotoxic effect from PCB exposure. ± Diastolic hypertension Increased mortality from cardiovascular disease in male capacitor workers (smoking habits not controlled for)	Kreiss et al., 1981 Akagi and Okamura, 1985 Gustavsson and Hogstedt, 1997	Reports of hypertension associated with PCB exposure have not been replicated. The initial study was based on a single measurement and has not been replicated in other studies; In a 13 year post-incident follow-up of Yusho patients, no relationship was found between hypertension and PCB exposure Blood pressure measurements are notoriously unreliable with recognized sources of variability ranging from time of day, diet, age, smoking and examiner ability.
Nervous System	In adults, there is no evidence of a direct neurotoxic effect from PCB exposure. There is limited evidence to support classification of PCBs as neuroteratogens. PCB exposure has been correlated with increased subjective neurological	Higuchi, 1976	Clinical relationships between subjective neurological complaints were not established.

	complaints such as headache, numbness, general fatigues, loss of appetite, sleep and memory disturbances, depression, nervousness, impotence, and nausea and vomiting	Rogan and Gladen, 1992 WHO, 1993	Investigation for peripheral neuropathy often found changes that did not reach the diagnostic criteria for true peripheral neuropathy.
	Abnormal EEG findings in persons exposed to heat degraded PCBs could not be linked to CNS dysfunction.	Chia and Chu, 1985	Combustion of PCBs gives rise to numerous byproducts including dioxins and furans. Typically, deficits resolve quickly without sequelae or clinical effect
	Sensory nerve conduction velocity decreases; motor velocity unaffected		
	No effect on hand steadiness or visual motor coordination in older adult consumers of Great Lakes fish.	Schantz et al., 1999	Exposure among adults often involves multiple chemical exposure making causal attribution difficult.
	Firefighters exposed to byproducts from a PCB fire showed worse results than controls on neurobehavioral tests.	Hauser et al., 1998	
Immune System	Occupational studies where workers were exposed to "clean" PCBs showed no effect on immune cell numbers or types	Swain, 1991 Tryphonas, 1998	To date, definitive evidence of an immune effect from PCB exposure in humans has not been found. Decreased immune cell numbers, while evident, do not appear to pose a risk of clinical illness in the general population. Populations with compromised immune status may be more at risk.
	Wild life studies show advserse immune effects from exposure to complex mixtures including PCBs	Golden et al, 1998	
	Yusho studies revealed fatigue and increased susceptibility to bacterial and viral infections - incidence of respiratory infections were correlated with blood PCB levels	Lu & Wu, 1985	Some authors suggest that statistical significance is not enough to warrant concern; a four fold change is necessary to induce clinical symptoms and impact immune

	Yu-Cheng exposure to heat-degraded PCBs resulted in decreased IgM and IgA but not IgG as well as decreased numbers of T cells (resolved within 3 years). These patients also showed increased susceptibility to infections of the skin and respiratory tract. This suggests a suppressive effect on cellular immunity. Decreased numbers and lower proportions of natural killer cells correlated with weekly fish intake and non-ortho PCBs in Swedish fisheaters In Michigan, breast milk PCB levels were positively correlated with incidence of childhood infections; North Carolina cohort showed no increase in mortality from infections Dutch children had increased numbers of T-cells, decreased monocytes and granulocytes	Svensson et al., 1994 Weisglas-Kuperus et al, 1995	status. That magnitude of change has not been seen with respect to PCB exposure.
Endocrine System	Altered menstrual cycle Decreased sperm quality - evidence overall is unclear with some studies reporting decreases, no change , or increases Effects on the thyroid gland; structural similarity to coplanar PCBs In areas where wildlife have observable thyroid dysfunction associated with environmental chemicals, comparable effects in humans have not been observed. Frank thyroid effects have been seen only in humans exposed to levels far in excess of	Higuchi, 1976 Carlsen et al., 1992 MacLeod and Wong, 1979 Fisch et al. 1996	The etiologies of some disorders thought to be affected by PCBs via an endocrine dysfunction route are unclear at best. For e.g., the cause of endometriosis itself is unclear and human data regarding PCB effect and possible role of endocrine system unclear confounders such as regional differences, age and smoking are not always accounted for

	background (Golden et al., 1998) There are human studies linking decreased T_4 and T_3, increase TSH and goiter to environmental PCBs but these persons are typically asymptomatic Children exposed to a mixture of chemicals including PCBs showed statistically significant positive associations between TSH and PCB 118 and negative associations between FT_3 and PCBs 138, 153, 180, 183 and 187. The greater effect may be the effects on cognitive development (see developmental section)	American Council, 1997 Brucker-Davis, 1998 Emmett et al, 1988 Osius et al., 1999	biological plausibility but evidence lacking phytoestrogens in the diet account for 40 million times more exoestrogen intake than environmental estrogens with no evidence of widespread effect Much of the controversy stems from the work of T. Colborn and associates and their book "Our Stolen Future", which speculates on the effect of environmental chemicals on wildlife and attempts to link observations from animals directly to human effects
Kidneys and Renal System	There is no evidence of a direct or indirect effect.	ATSDR, 1997	
Lungs and Respiratory system	Atypical bronchitis-like symptoms with copious expectoration with secondary bacterial or viral infection reported in the early stage of the Yusho incident; gradual improvement over the first 14 years. Firefighters report shortness of breath after fires involving PCBs Reduced FVC, FEV_1	Higuchi, 1976 Nakanishi et al., 1985 Emmett et al., 1988	Difficult to separate PCB effect from other incineration products and smoke Studies reporting changes to pulmonary function tests generally unreliable given the variability between measurements, examiners, patient effort and smoking status.

Sensory Organs	No evidence for effects to sense of taste or smell Hearing - animal studies of congenital hypothyroid animals show marked hearing deficit - no comparable human studies Yusho – decreased visual acuity, easy fatigability hypersecretion of Meibomian gland, conjunctivitis, swelling of eyelids	Higuchi, 1976	
Reproductive System	Some evidence that embryos and fetuses may be more sensitive to PCB effects because they generally lack the hepatic microsomal enzyme system responsible for detoxification and elimination of PCBs Decreased birth weight (160-190 g) Decreased head circumference at birth Fetal PCB Syndrome (FPS) Dark brown pigmentation of the skin and mucous membranes; gingival hyperplasia; exophthalmic edematous eyes; dentition at birth; abnormal calcification of the skull; rocker bottom heels; high incidence of low birth weight; eyelid swelling and discharge; acne; dystrophic fingernails; pigmented or dystrophic toe nails In the Yusho cohort, 39 babies had been born to intoxicated mothers by 1983. Eight died of pneumonia, bronchitis, sepsis and premature and congenital weakness. The	 Yamashita and Hayashi, 1985 Gladen et al., 1990 Rogan, 1982 Hsu et al., 1985	Enormous amounts of energy have been put into this area. Results are inconclusive at best. The variety of observed effects reflects the variety of contaminants in high level exposure from contaminated rice oil (i.e. PCB, PCDF, PCQ and PCT)

	remainder grew up and gradually recovered height and weight deficits Fetal loss was 15-20% in the Yusho and Yu-Cheng cohorts Reproductive success - case control study of miscarriages versus full term pregnancies showed higher PCB levels in those women having miscarriages. In this study, PCB levels were also positively correlated with age and alcohol consumption. No or moderate association between fish consumption (and hence PCB exposure) and conception delay in men. Dioxin exposure may affect sex ratio of offspring --> excess females Environmental studies of mixed organochlorine exposure does not support a sex ratio change	Leoni et al., 1989 Buck et al., 1999 Courval et al., 1999 Vartiainen et al, 1998	
Blood and Hematopoetic System	Yusho cohort initially showed elevated serum triglycerides, anemia, lymphocytosis, and hypoalbuminemia *distinctive chromatographic pattern which remained unchanged over time Hypertriglyceridemia Anemia in women Elevated serum cholesterol	Akagi and Okamura, 1985 Urabe and Asahi, 1985 ATSDR, 1997 Guo et al, 1999	The association between PCBs and blood lipids is spurious. Increased levels of PCB in blood are more associated with level of exposure, age and diet.

Non Organ Directed Toxicity	Effect	Reference	Critique
Cancer	Breast and Endometrial Cancer - some PCBs have very weak to weak estrogenic activity in animal studies prompting concerns about effects on estrogen dependent cancers in humans. To date, there is no conclusive evidence of a relation to either breast or endometrial cancer.	Ahlborg et al., 1995 Adami et al., 1995 Hunter et al, 1997 Rylander and Hagmar, 1995	The IARC designation of probable human carcinogen is based on rodent studies. Human data from occupational and environmental studies do not support this classification.
	Liver cancer - despite evidence of induction of liver enzymes, there is limited evidence of a link to cancer. The majority of studies examining cancer of the liver, gall bladder and bile ducts show no association. One study of occupationally exposed males showed a slight statistical increase of liver cancer.	Brown, 1987 Loomis et al., 1997	The use of occupational cohorts to study cancer has several difficulties. The most serious of which is the historical nature of most studies in which exposure is assigned on the basis of job title and concomitant exposures cannot be controlled.
	Increased incidence in rectal cancer in women seen in one study but not seen in follow-up of same cohort.	Brown and Jones, 1981	The latency period for cancers of 20-30 years is not uncommon. A study with a 15-year follow-up may miss cases. Few deaths in a study limit statistical power.
	Fatal malignant melanoma; non-significant increase in cancer of the brain and nervous system	Sinks et al., 1992 Loomis et al, 1997	"Findings on specific cancer sites are inconsistent with excess mortality from all cancers combined reported in several studies but excesses of an array of specific cancers reported in one or two studies each" Loomis et al, 1997.
	Non-Hodgkin's lymphoma Positive association (with concomitant pesticide exposure)	Rothman et al., 1997	
	Negative association	Sinks et al., 1992	
	Gastrointestinal cancer in males significantly increased	Bertazzi et al., 1987	

	Lung cancer and hematological neoplasm elevated but not statistically significant Mortality from all cancers significantly below expected in males and comparable to expected in females (occupational cohort n = 7075) No association with PCB exposure	Bertazzi et al., 1987 Kimbrough et al., 1999 Greenland et al., 1994; Gustavsson et al., 1986	Retrospective cohort; significant healthy worker effect observed; 97% of the females had low exposure i.e. clerical positions. Occupational PCB exposure studies with women are few. The Kimbrough study is one of the few to have included a large cohort of women.
Developmental	Yusho and Yu-Cheng children nail abnormalities musculoskeletal changes behavioral problems – **hyperactivity** cognitive deficits (poorer performance on standardized tests) low birth weight in infants whose mothers ate contaminated fish (not correlated to mothers' exposure level) Motor deficit and hypoactive reflexes at birth Deficits in motor skills at one year Reduced scores on memory and cognitive function Poorer autonomic regulation, orientation and habituation on the Brazelton scale There have been 3 major studies examining the neuro-cognitive development of children	Rogan et al., 1988 Chen et al., 1994 Fein et al., 1984 Fein et al, 1984; Rogan et al., 1986 Brucker-Davis, 1998 Schantz, 1996 MacLusky et al., 1998 Lonky et al, 1996	Studies of behavioral changes related to PCB exposure have been severely criticized on a number of fronts. First, if developmental effects at environmental exposure levels were as pronounced as claimed by some authors, there should be widespread evidence of the effects. There is not. Second, environmental studies are notorious for difficulties in determining accurate exposure status. Proxy measures may not accurately reflect true exposure. Third, studies using fish consumption as the source of PCB exposure may not take into account trace contamination by other organochlorines, metals or pesticides, some of which of recognized developmental neurotoxicants. The Jacobsen studies in particular have been severely

	exposed to PCBs in utero. None of the observed effects relate to postnatal exposure. North Carolina – hypotonic and hyporeflexive at birth; initial delays gone by age 2, no difference between exposed and control at 5 years. Observed effects were not dose-dependent and only seen in the most highly exposed groups Michigan – decreased birth weight, more abnormal reflexes, poorer autonomic regulation, limited responsiveness to external stimuli, impaired visual recognition memory at 7 months of age, decreased IQ at school age. Netherlands – decreased birth weight, decreased postnatal growth, psychomotor and developmental delay, thyroid and immune status changes, poorer cognitive functioning in preschool children (no association to lactational exposure or current body burden) . Supported by a German study which found evidence of decreased mental ability in 7 month old infants exposed lactationally to PCBs Inconclusive evidence of an endocrine disrupting effect of PCBs leading to reproductive system changes in offspring	Gladen et al., 1988; Rogan et al., 1994 Jacobsen et al., 1985 Jacobsen and Jacobsen, 1997, 1996 Patadin et al., 1999 Huisman et al., 1995 Winneke et al.,1998 Foster et al., 1998	criticized for a number of study design flaws and faulty conclusions. For example, mothers' exposure was assessed by dietary recall from the previous 6 years; PCB cord blood levels were below the detection limit in over half of their samples yet they chose to average detectable PCB samples from cord blood and maternal serum and milk samples to represent individual child values. Many influences on intellectual development are difficult to control e.g. social, cultural, economic, demographic, genetic, familial or care taking. The use of neuropsychological tests (alone) to assign causality to deficits from specific toxic exposure is inadequate (Bernstein, 1994) Neuropsychological deficits are among the most common birth deficits (Dietrich, 1999) The clinical significance of a small decrease in IQ points is uncertain although may be significant in the borderline child. There is some thought that parents of known exposure may treat offspring differently on the assumption that they are "damaged". Concurrent methylmercury contamination (which is a known

			neurotoxicant) has been shown in rat studies to act synergistically in reducing brain dopamine levels. This could also impact intellectual functioning (Bemis and Seegal, 1999). Behavioral tests that rely on cooperation of the subject make interpretation difficult. In the Michigan studies, failure to cooperate led to exclusion from the study. The differences in results between the Michigan and North Carolina cohorts may be as a result of different congener mixtures thus different exposure scenarios.

Genotoxic	Limited evidence from animal studies of transgenerational effects; possibly affected is reproductive success Negative results in the Ames assay, micronucleus test and V79 Chinese Hamster Assay. In rats, PCBs do not produce chromosome alterations or dominant lethal mutations. In a transgenerational rodent study, F_2 rats had decreased t-helper/inducer lymphocyte subset numbers (although intake was approximately 16 times higher than the average human fish consumer)	Feeley et al. 1998	In the case of the most highly exposed population to date (i.e. Yusho and Yu-Cheng) there are no reports of increased incidence of congenital anomalies in the offspring of exposed persons or their children.

3.4 Historical Incidents

Over the years, there have been a number of human poisonings involving PCBs and dioxins. The first major incident occurred in 1968 in the Nagasaki and Fukuoka Prefectures in Japan. The first indication of problems began at a Fukuoka medical clinic with a three year-old child, her family, and a complex of unusual dermatological symptoms. The strange skin condition crossed all age groups and occupations with the common initial symptoms being increased eye discharge, eyelid swelling, acneform eruptions of the skin, follicular accentuation and increased pigmentation (Higuchi, 1976). Initially, physicians were perplexed. Despite a similarity to industrial chloracne and adolescent *acne vulgaris*, neither the age groups nor the occupations of the patients as a group seemed to lend itself to these diagnoses. Eventually, after extensive epidemiological investigation, the disease was linked to consumption of a specific brand of rice bran oil that had been contaminated with PCBs. The disease was named "Yusho" which translates literally as rice-oil disease and the specific brand of rice oil involved was identified. The source of PCBs was Kanechlor-400, which had leaked from pinpoint holes in a heat transfer conduit[19].

Only eleven years later in May 1979, a strikingly similar incident occurred in Taiwan. Students from the Hui-Ming School for Blindness began to present with a strange skin disease consisting mainly of acneform type lesions and eye discharge. The mystery disease was not limited to students and soon, a full scale outbreak was underway. Unfortunately, the similarity to Yusho was not recognized and epidemiological investigation continued until 5 months later when, after consultation with Tokyo experts, serum PCBs were identified and the source traced to consumption of contaminated rice bran oil. This time, the disease was identified by its geographic location, Yu-Cheng. In much of the literature surrounding these two events, the terms Yusho and Yu-Cheng are used almost synonymously because of the similarity of circumstances and observed health effects.

---

[19] There is considerable evidence to support the conclusion that symptoms associated with Yusho and Yu-Cheng are due in whole or part to PCDF and polychlorinated quaterphenyl (PCQ) contamination of the PCBs and thus the rice oil. If this was the case, direct effects of PCBs might be uncertain.

These two incidents have formed the basis for what is known about human PCB poisoning in particular and organochlorine poisoning in general. Both cohorts have been studied extensively and for upwards of 25 years and the literature surrounding these incidents and the subsequent health effects observed in victims is extensive and complex. The differences between the two incidents are slight and the health outcomes appear to be comparable.

The second incident which may provide insight into health effects concerns in Swan Hills and surrounding area occurred in 1976 in Seveso, Italy. In this case, a chemical manufacturing plant that "produced intermediate compounds for the cosmetics and pharmaceutical industry ... [including] 2,4,5 trichlorophenol (TCP) which is used in the chemical synthesis of herbicides" (Ramondetta and Repossi, 1998) experienced the failure of a safety valve resulting in a twenty minute period during which chemicals were vented directly to the atmosphere. In contrast to the Swan Hills incidents where mixed gases were released, high concentrations of almost pure 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)[20] were released from the Italian plant and nearby residents were evacuated. People who were not evacuated were affected by strict dietary and hygiene guidelines. Extensive studies of both the physical environment and the effects on human health have ensued in the following years with mostly inconclusive results.

A third incident that could be included is the exposure of Vietnamese and American people during the late 1950's to early 1970's through the use of a chemical defoliant known as Agent Orange by the American military inadvertently contaminated with TCDD. Agent Orange, so named because of the colour of the barrels in which it was transported, was a herbicide composed of a one-to-one mixture of 2,4,5 trichlorophenoxy acetic acid and 2,4 dichlorophenoxy acetic acid. Again results from the study of exposed populations have largely been inconclusive but with a number of intriguing associations such as possible associations between soft tissue sarcoma and Hodgkin's disease.

---

[20] TCDD was produced as a result of an extended and uncontrolled exothermic reaction in a 1,2,3,4 tetrachlorobenzene alkaline hydrolysis reaction vessel at 2,4,5 sodium trichlorophenate, an intermediate compound in the preparation of trichlorophenol (Ramondetta and Repossi, 1998).

Unfortunately political issues surrounding the military action itself and concomitant exposure to herbicides confound much of the data surrounding exposure to Agent Orange. Extensive literature on this exposure and health effects is available, generally found under "Operation Ranch Hand", the military code name for the operation but will not be included in this discussion.

With regard to PCBs in Canada, there are two bodies of literature that may provide useful insight into the Swan Hills scenario. First is the literature surrounding PCB (and other chemical) contamination of the Great Lakes basin. Many studies have examined human health in relation to consumption of contaminated fish and game. The similarities however end there in that contamination levels in this region are much higher than those seen in and around Swan Hills and are a result of direct and historically unregulated industrial pollution. Many pollutants are found in conjunction with PCBs, such as metals (e.g. lead and mercury) and pesticides (e.g. chlordane and toxaphene). The second body of literature that may prove useful is that surrounding the health of the Northern Quebec Inuit. The traditional Inuit diet is high in marine mammals and fish and has contributed to high levels of human contamination. There is no identifiable direct source of PCB contamination in this region. Long-range atmospheric and oceanic transport of organochlorine compounds and subsequent bioaccumulation in the Arctic food chain was first suspected in 1974 and confirmed in the subsequent years (Wania & Mackay, 1996). Semi-volatile, chemically stable organic pollutants such as PCBs may experience long-range transport in the global environment. "Warm temperatures favor evaporation from Earth's surface in tropical and subtropical regions. Cool temperatures at higher latitudes favor deposition from the atmosphere onto soil and water" (p.390A).

The last body of literature surrounding PCB exposure comes from the occupational health literature. A number of large retrospective cohort studies have been carried out with this population, mostly in male transformer and capacitor workers. The data centres almost exclusively on exposure to un-used PCBs. This type of exposure may not have the same impact as PCBs from an environmental source due to a variety of confounding factors such as the preponderance of male subjects, exposure to select, "clean" commercial PCB

preparations and a lack of food chain exposure which has been theorized to be different than direct exposure. There are however a number of intriguing parallels with emergency workers exposed during incidents such as transformer fires.

3.4.1 Yusho and Yu-Cheng

The first patient in the Yusho outbreak was a 3-year-old girl who presented on June 7, 1968 to the Kyushu University Dermatology Clinic with an acne-like condition (Higuchi, 1976). From that point on, the number of patients increased steadily, extending throughout the Fukuoka and Nagasaki prefectures (Figure 3.2). After extensive investigation, the disease was linked to consumption of rice oil contaminated with approximately 2000-3000 mg/L PCB. The source of the PCBs was identified as Kanechlor 400 (originally composed of mainly tetrachlorobiphenyl) which had been used as a heat conductor in deodorization of the oil during production and which had leaked into the oil through pinholes in the heating coil. It is estimated that Yusho victims consumed 633 milligrams (mg) PCB, 3.4 mg PCDF and 596 mg PCQ (WHO, 1993).

Figure 3.2: Incidence of Yusho Cases 1968

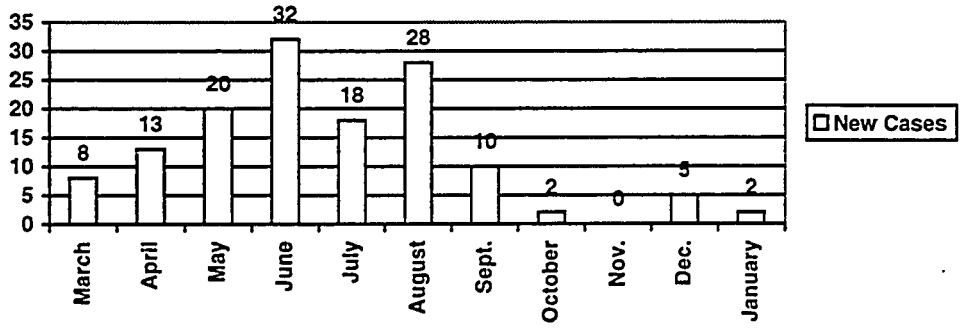


Table 3.3 Rice Oil Contamination

Incident	Mean PCB Concentration	Mean Furan Concentration
Yusho	2000-3000 ppm[1]	3.85 ppm[3]
Yu-Cheng	53-99 ppm[2]	0.14 ppm[3]

1. WHO, 1993 2. Hsu et al., 1985 3. Miyata et al., 1985

Table 3.4: Consumption in Yusho/Yu-Cheng

Incident	PCB	PCDF	PCQ
Yusho	630 mg	3 mg	600 mg
Yu-Cheng	0.7-1.80 mg	4 mg	590 mg

Of the initial complaints, 81.7% complained of skin problems, specifically acneform eruption, 73% of patients complained of eye discharge and 61.3% complained of eye edema (Higuchi, 1976). Eventually the various skin and mucous membrane symptoms became the most prominent of the symptoms until finally, diagnoses were made almost entirely on that basis. (c.f.3.5). Other symptoms included respiratory distress and a bronchitis-like condition that was correlated with serum PCB concentrations (Nakanishi et al., 1985) and was often complicated by viral or bacterial infection, increased incidence of infection, especially of the respiratory tract and skin (Lu & Wu, 1985), hypertension (especially diastolic) which usually resolved within six months of the incident (Akagi & Okamura, 1985), and a number of systemic complaints including fatigue, loss of appetite, headache, nausea and vomiting, numbness and swelling of extremities (Urabe & Asahi, 1985).

As time progressed, "mucocutaneous lesions gradually gave way to systemic disorders" (Urabe & Asahi, 1985). The general syndrome was vague and difficult to define but included complaints of dullness, headache or heavy headedness, indefinite stomachache, numbness or pain in the extremities, swelling and pain of joints and coughing or bronchitis-like symptoms. The bronchitis was different from typical bronchitis in that patients had no crackles but had wheezes with no radiological, physiological or immunological evidence of bronchial asthma or pulmonary emphysema (Nakanishi et al., 1985).

Chloracne associated with PCB intoxication is highly disfiguring and leaves the victim with deep pits and scarring once the acute symptoms recede. In contrast to the more common *acne vulgaris* which is found predominantly in adolescents and is a result of hormonal changes affecting the size of sebaceous glands and amount of sebum production, chloracne is a distinct condition and sign of organochlorine (and PCB or

TCDD) exposure. Unusual pigmentation of the skin is also a common symptom especially along the ala nasi, gingivae and lips of exposed children.

Table 3.5 : Criteria of clinical severity grading of patients with PCB poisoning

Grade	Main Criteria	Reference Criteria
0	Abnormal blood level of PCBs without clinical manifestation	
I	Increased cheese-like discharge from Meibomian gland, pigmentation of nails	Increasing sweating, dry skin, pigmentation of lips, gingiva and mucus membrane of oral cavity
II	Grade I plus comedo	Hair follicle keratosis on the extensor side of extremity joints
III	Grade II plus acneform eruptions, cyst formation of sebaceous gland in genital and evidence of follicular opening in the site of neck and upper chest	Swelling of eye lids, swelling and pain at joints
IV	Enlarged and elevated follicular opening all over the body and extensive distribution of acneform eruption	Swelling of face and limbs, extensive distribution of acne with purulent infection

Source: Goto, M. and Higuchi, K. (1969).

In Taiwan, the rice oil contained a Kanechlor-400/500 mixture at concentrations as high as 65 and 108 μg/ml, respectively (Hsu et al., 1985). There were 1843 cases recorded from December 1978 to November 1980, with 1252 in Taichung County alone. Estimates of PCB intake duration range from 3 to 9 months with estimated average total PCB intake ranging from 0.7 to 1.8 g (Hsu et al., 1985). Victims also consumed an average of 3.8 mg PCDF and 590 mg PCQ. The average latent period (from ingestion to onset of symptoms) was 3 to 4 months (range 1.5 to >6 months). The blood levels of PCBs in 613 patients within the first year of the outbreak ranged from 3 ng/ml to 1200 ng/ml. 82.5% of the affected persons had blood levels between 11 and 150 ng/ml although 169 patients had PCB levels of more than 100 ng/ml.

In the Taiwanese cohort, apart from chloracne, which recedes in severity over the years, the most recent study (Guo et al., 1999) found that despite a slight increase in morbidity, the only significantly increased health effects in this highly exposed group are anemia in women and arthritis and herniated vertebral discs in men.

There are however, a number of confounding factors to be taken into account before coming to any firm conclusions about the health effects associated with PCBs in these two cohorts. The major concern may simply be analytical ability. In 1968 when the first of these incidents occurred, the analytical techniques to detect and monitor PCB concentrations in human matrices were undeveloped. It wasn't until 1973 that the ability to measure PCBs in blood was developed. The majority of studies from that early time period report only total PCB. Today, there is some evidence that not only are individual PCB congeners highly variable in toxicity, some congeners of relatively high concentration are in fact of lower toxicity. In addition, there has been much speculation about the presence and effects of toxic contaminants such as 2,3,7,8-tetrachlorodibenzofuran and polychlorinated quaterphenyls in both rice oil incidents. The general consensus in the scientific community is that PCBs were not likely responsible for the observed health effects outside of chloracne although other organochlorines can also cause chloracne. Also confounding the health assessment is the fact that occupational studies (including those of contemporary Japanese workers) have shown a distinct lack of comparable biological effects despite PCB serum levels orders of magnitude higher than seen in either Yusho or Yu-Cheng (Kimbrough, 1999).

3.4.2 Seveso, Italy (1976)

Another incident that may provide some insight into health effects in Swan Hills and surrounding area occurred in 1976 in Seveso, Italy. The ICMESA chemical plant (Industrie Chimiche Meda Società) located near Seveso, Italy, 15 km north of Milan experienced a major chemical accident on July 10, 1976 which resulted in the release to the atmosphere of approximately 3000 kg of pollutants including trichlorophenol, caustic soda, solvents, other toxic substances and 2,3,7,8-dibenzo-p-dioxin (TCDD) which created a toxic cloud which drifted over nearby communities. The plants major products were intermediate compounds for the cosmetics and pharmaceutical industry including 2,4,5 trichlorophenol used in the chemical synthesis of herbicides (Ramondetta and Repossi, 1998).

Initially, those persons in the path of the cloud reported very few symptoms including nausea, headache and eye irritation. Some children were hospitalized for skin lesions on exposed parts of the body (Bertazzi et al., 1998). By April 1977 (9 months after the incident), 187 cases of overt chloracne were reported. Of these, 164 were children, particularly from zone A in the immediate vicinity of the plant. Estimates of the amount of dioxin in the toxic cloud range from 300 g to 130 kg (Ramondetta & Repossi, 1998) which spread over an area of approximately 1810 hectares. Within 72 hours of the incident there were reports of the deaths of exposed vegetation and small animals. Within 24 hours after that, the first signs of skin inflammation were seen. In addition to extensive environmental contamination, there were numerous persons exposed. Depending on the degree of contamination, the region affected by the release was divided into three zones. The most highly contaminated zone, Zone A, delineated by dioxin soil concentrations higher than 50$\mu g/m^2$ was completely evacuated. Eventually, soil contamination was found to range between 15.5 and 580 $\mu g/m^2$ (Bertazzi et al., 1998). Zone B, with 4613 residents, generally had contamination levels below 5 $\mu g/m^2$, while zone R was generally below 2 $\mu g/m^2$. In total, 36,093 persons were affected by the accident. As with the Yusho and Yu-Cheng populations, the people of Seveso have been intensively studied in the years following the accident.

The advantage of the Seveso incident, as it relates to environmentally mediated chemical exposure, is that it was a relatively **pure** release of TCDD in air that resulted in the contamination of all media, including resident humans and animals. TCDD has a broad toxicity in experimental animals with documented teratogenic, carcinogenic, hormonal, immunologic and sex ratio effects (Landi et al., 1998). Human studies on cancer from the Seveso incident have, however, been inconclusive to date. Other studies of TCDD exposure have been mostly occupational, mostly male, exposed to multiple toxicants and experience chronic versus acute exposure. This limits the generalizability of these studies and hence their usefulness.

In Seveso as in the Yusho and Yu-Cheng incidents, chloracne has been the most definitive sign of organochlorine exposure. Long-term effects appear to be limited to

increased mortality in men secondary to cardiovascular disease, diabetes mellitus and chronic obstructive pulmonary disease. In women, increased mortality secondary to hypertension, chronic obstructive pulmonary disease and diabetes have been noted. Some researchers have suggested that many of the observed health effects are attributable to stress reactions. An interesting finding that may lead to future research is the observation that children born to parents exposed in the Seveso incident are more likely to be female than male. Other studies have not replicated this finding (Vartiainen et al, 1998) albeit in less highly exposed populations. A registry for congenital anomalies set up after the incident (1977-1982) found no evidence of increased congenital anomalies in children born to exposed parents.

The prominence of chloracne, especially among children (n = 193) prompted follow up studies of the Seveso population between the years 1976 and 1983. Researchers found no temporal or trend differences in measures of hepatic function, total cholesterol, serum triglycerides, or motor/sensory nerve conduction. In adults, clinical and electrophysiologic signs of peripheral neuropathy in more than 300 persons evacuated from Zone A were not overtly different from controls. In cases with chloracne (n=153), there was an increased frequency of peripheral nervous system involvement but cases did not meet diagnostic criteria for peripheral neuropathy.

The potential for immune system effects were investigated in a number of studies, the majority of which were inconclusive due to test design limitations and control group difficulties (1976-1979). In 1500 children studied for liver effects, transient changes were noted in 69 children. Hepatic enzymes, which showed initial increases, had returned to normal by 1980 (4 years since the incident).

Long -term studies of the Seveso population have looked mainly at cancer and mortality rates. The mortality rate for death from all causes has not differed in any of the three contaminated zones compared to a control population. There are some interesting differences in non-malignant causes of death. In the most highly exposed region, males have experienced increased mortality from cardiovascular disease, especially chronic

ischemic heart disease (CIHD) and chronic obstructive pulmonary disease (COPD). Women have had increased mortality from chronic rheumatic heart disease and hypertension. Also in women there is a non-significant increase in diabetes mellitus related mortality. Researchers note these increases with caution however because of the small population involved and the relatively few overall deaths (Ramondetta and Repossi, 19xx). In the lesser-exposed region, a modest non-significant increase in overall CIHD incidence has been observed. In women, significant excess mortality for COPD was noted. Diabetes mellitus significantly increased for females. Increased incidence in digestive site cancers was also observed in the lesser-exposed zone (Zone B). There was a 3-fold increase in rectal cancer in males and increases in stomach and liver cancer in females. Leukemia showed a statistically significant increase in males while Hodgkin's disease and myeloma showed a 6-fold increase in relative risk in females.

In the least exposed zone, Zone R, increases in CIHD for both males and females and increased mortality from hypertension for females has been observed (Ramondetta and Respossi, 1998).

Overall, the only conclusive link to TCDD exposure at Seveso is chloracne. Despite strongly suggestive results from a number of studies there are many limitations to interpretation. For example, many of the studies suffer from selective participation, lack of reference data and limited standardization of methods and performance of tests. As in many studies of chemical disasters, the hectic post-accident conditions are not conducive to effective study. Many of the Seveso studies have a restricted number of individual measurements, it has been a relatively short time since exposure (some cancers have latency periods of several decades) and the size of the population in the most contaminated zone was quite small. "The varying population size in different zones had a major influence on the power of the study to detect unusual relative risks. The small number of deaths in some of the subcohorts (especially in zone A and in subcohorts obtained after stratification...) still limits interpretation of the results" (Pesatori et al., 1998, p.129). Exposure in early studies was not based on human serum samples but on assigned zones that were based on soil contamination levels.

Researchers have suggested that the observed excess in cardiovascular mortality may be attributable to factors other than chemical exposure - specifically psychosocial stress and its effects on pre-existing disease. This conclusion is supported by the early post-accident occurrence of some deaths, the advanced age of some of the affected persons and the prevailing chronic type of cardiovascular disease seen among the cases. Some of the other stresses involved are: leaving businesses and homes during the evacuation and fears over the future, including the health of children. This conclusion is despite animal data suggesting a TCDD effect on lipid metabolism and cardiac function and morphology. There is some speculation of an immune effect responsible for the increase in rheumatic heart disease mortality.

The finding of excess mortality due to diabetes mellitus is interesting - suggesting the need for further research. In studies of other dioxin-exposed populations, specifically those of persons exposed to Agent Orange during the Vietnam conflict, results have also been inconclusive. Many of those studies rely on self-reported diagnoses with lack of adequate confounder control in addition to a myriad of politico-legal complications (c.f. Henriksen, G.L. et al., 1997). One interesting aspect of the diabetes question is the observation by some researchers (c.f. Pesatori et al., 1998) of a gender difference. The increases seem to be more pronounced in females even after controlling for confounders such as initial exposure, age, body fat distribution or amount, smoking history and diet (Landi et al., 1998). Researchers hypothesize that there may be some type of hormonal interaction in effect.

Other confounding variables contributing to uncertainty in the assessment of health effects in the Seveso incident are similar to those in the Yusho/Yu-Cheng incidents. For example, analytical methods for detecting/measuring low dioxin levels in blood were undeveloped at the time of the incident. It wasn't until the 1980's that more reliable methods were developed. At that time, retrospective exposure estimates of human exposure were possible. In zone A, the most highly contaminated (as delineated by soil measurements), estimated exposure is 440 pg/ml (n= 177); Zone B, 87 pg/ml (n = 87);

Zone R, 15 pg/ml (n=17). In 1992/93, using a half-life of 7.1 years, zone A exposure was estimated at 330 pg/ml, zone B was 110 pg/ml; Control (unexposed) population was 5 pg/ml.

3.4.3 Canada

In Canada, experience with PCBs has been limited by strict maintenance and handling procedures (Milly and Leiss, 1997). PCBs were never produced in Canada although an estimated 2450 tonnes/year were imported during production years (Anonymous, 1997) to be used in transformers and capacitors that may still be in use (albeit nearing the end of their useful life span). Intense public opposition to incineration facilities has however led to a number of PCB storage sites and incidents involving PCBs across the country. Studies on potential health effects have been limited to long term low level exposure scenarios centred on the Great Lakes and the far north.

The first notable incident involving PCBs and human exposure in Canada occurred on April 13, 1985 near Kenora, Ontario. A truck carrying a flatbed loaded with full electrical transformers leaked, losing about 400 litres of PCB contaminated oil. Initially officials thought only approximately 70 km of asphalt were contaminated but it was eventually found to be close to 220 km. Official reaction to the event was slow (c.f. MacLean's magazine 29 April 1985) contributing to mass speculation and fear.

The second Canadian PCB incident was to leave its mark on Canada's environmental reputation for years to come. On August 23, 1988 a fire at a chemical storage warehouse in St. Basile-le-Grand, Quebec (40 kilometres southeast of Montreal) threatened the security of 1500 barrels of PCB-containing oil. Concern for residents from toxic by-products of the fire led to the evacuation of approximately 3000 residents in a 40-kilometre radius from the fire. "Although only 8% of the PCB-contaminated material actually burned, the St. Basile fire was probably the largest ever uncontained PCB fire worldwide" (Milly & Leiss, 1997: 186). Despite the potential, after 18 days of testing, a panel of experts found that PCB and dioxin residues in buildings near the fire were no higher than uncontaminated control buildings.

One of the greatest difficulties surrounding the St. Basile fire was communication. As is often the case during emergencies, communication took a very secondary role to dealing with the actual incident. In this case, uncertainty over scientific data, "... a dearth of information released by public officials ... and the climate of secrecy that surrounded the expert interpretation of results, created an information vacuum that fueled rampant speculation" (Milly & Leiss, 1997: 186). Into this vacuum stepped the environmental activists from Greenpeace. They had their own experts extol the dangers of PCBs and dioxin exposure providing their own perspectives on a very controversial issue. Despite a number of highly generalized statements, Greenpeace set the stage for prevailing opinion surrounding the entire chemical class that still exists in both media and general culture.

Up to 3 years later, no evidence of long-lasting health effects on residents or emergency workers were found (Milly and Leiss, 1997).

Following the fire in St. Basile, the remaining 3500 tonnes of waste were contracted for disposal at an incinerator in Pontypool, Wales. The freighter was, however, met at the docks by British Greenpeace activists who successfully prevented the ship from docking. Confronted with a returned shipment, the government decided to store the wastes near the town of Baie Comeau on Quebec's lower north shore. When the shipment arrived, more than 2000 of the town's 26,000 residents were waiting. In a nationally televised spectacle, protesters and police clashed and despite an injunction, the freighter was unloaded in the middle of the night. Protects and injunctions continued for two weeks until the PQ government lifted the injunction and allowed transport of the barrels (under police guard) to a storage site. Canada received widespread international criticism over its environmental policy and its lack of responsibility in dealing with this incident.

In August 1990, the federal government enacted legislation outlawing the overseas export of PCBs thus preventing a similar situation from recurring. December 1999 however saw regulations barring the import of foreign waste for treatment lifted thus allowing

importation of toxic waste including PCBs from the United States, Mexico and South America.

3.4.3.1 Great Lakes

The North American Great Lakes contain one-fifth of the earth's total fresh water and have been described as the largest concentration of PCBs on the planet (Johnson et al., 1999). Initially, PCBs and other pollutants (such as lead, mercury, DDT/DDE) were introduced into the water through industrial processes and deliberate dumping of industrial waste. Despite evidence of declining levels of pollutants since the 1970s (Johnson et al., 1999), the lakes have retained very high contaminant levels putting a number of populations potentially at risk for exposure. The nature of the problem (i.e. dumping into the lakes) puts the aquatic food chain at the highest risk although terrestrial animals are also potentially exposed. At the top of both food chains, the potential for human exposure has been a concern.

Traversing the border between Canada and the United States, both the contamination and the solution have international implications. Numerous joint efforts have sought to study and resolve some of the issues surrounding the Great Lakes pollution problems. Among the most visible of the problems surrounding Great Lakes contamination has been observation of reproductive failure among wild life populations. First postulated by T. Colborn and associates, the endocrine disruptor theory, which attributes a wide variety of biological effects to exposure to synthetic chemicals (specifically chlorine-containing), has gained much support and by weight of evidence appears to be a major influence in environmental chemical exposure. This is not to say that human evidence is conclusive. By far, the most compelling evidence in the endocrine disruptor hypothesis comes from lab animal and wild life observations. In Great Lakes wild life populations, adverse reproductive and developmental effects have been identified (Johnson et al., 1999). For example, a syndrome composed of embryo mortality, edema and deformities (GLEMEDS) has been identified in herring gulls, terns and other Great Lakes birds. In herring gulls and Caspian terns exposed after hatching, T-cell mediated immune suppression has been observed. Other health effects seen in wild life populations include

feminization and demasculinization, reproductive failure in mink and otter fed Great Lakes fish (leading to population declines) and enlarged thyroid glands and reproductive failure in a variety of fish species (Johnson et al., 1999).

The potential for multigenerational effects have been investigated by a number of researchers including Feeley and colleagues (1998). They fed two consecutive generations of rats diets high in Great Lakes fish but found only health effects such as induction of hepatic enzymes or immune system changes which could be "described as adaptive responses or of limited biological significance" with minimal effect function that were reversible with cessation of exposure. There were two exceptions. First, in their analysis they found a suggestion of modification of working and reference memory in males of the high dose group possibly related to decreased neurotransmitters in several brain regions. Second, they noted an effect on thymus weights in F_1 animals and an overall effect on T-helper/inducer lymphocyte subsets in F_2 rats fed fish from Lake Huron compared to those fed Lake Ontario fish. Overall, few diet-related effects were found with sporadic statistically significant findings that were inconsistent across sex or generation. Feeley et al. present these findings with some caution.

> Even with the suggestion of neurobehavioral/neurochemical effects...it is important to keep in mind the relative intakes of fish and associated contaminants in the treated rats compared to sport fish consumers in the Great Lakes basin. The estimated intake of fresh fish in the high-dose groups was approximately 60x that of sport fish consumers. Also, the intake of most of the major contaminants...ranged from about 10 to 30 times (mercury, total chlordane) to almost 100 times (total dioxins and furans) the Health Canada tolerable daily intakes (TDI).

In addition, the PCB intake was approximately 80X that of the provisional TDI from Health Canada.

In one of the few studies to use congener specific analysis, Stewart et al. (1999) assessed the pattern and concentration of prenatal PCBs in the umbilical cord blood levels of newborns whose mothers consumed Great Lakes fish. In all blood samples, the concentrations of PCBs were extremely low (approximately 1.0 ng/ml whole blood)[21]. Researchers found no relation between the concentrations of the lower and moderately chlorinated (1-6 chlorine substituents) homologs in serum or breast-milk with fish consumption. Both the proportion and absolute concentration of PCBs with 7-9 chlorine substituents was increased in fish consumers and a significant, positive correlation between fish consumption and breast-milk concentrations of homologs with 7-9 chlorine substituents was found. There was no direct relationship between total PCBs in umbilical cord blood and fish consumption. The authors speculate that "the most heavily chlorinated PCBs are the most valid index of fish-borne PCB exposure measured in cord blood" (Stewart et al., 1999: S95) thus the use of measurements of total PCBs may not adequately characterize PCB exposure[22].

Studies of potential human health effects from PCB exposure have centred on persons exposed to PCBs through consumption of contaminated fish. The Michigan Sport Fisherman's Study (Humphrey, 1976) was the first to demonstrate an association between consumption of fish from the Great Lakes and serum PCBs. In the ensuing years, numerous studies have positively correlated the consumption of Great Lakes fish with PCB serum levels (Hanrahan et al., 1999; Fitzgerald et al., 1999; Falk et al., 1999). The Michigan Maternal and Infant Study, which began in the early 1980s, examined the effects on infants of maternal consumption of Great Lakes fish and hence a number of contaminants. They found adverse developmental health outcomes in infants whose mothers consumed more than twelve kilograms of Great Lakes fish annually. Statistically significant decreases in birth weight (160-190 g), gestational age (average decrease 4.9 days) and head circumference (0.6 cm) compared to a control group were noted.

---

[21] The authors speculate this is a reflection of the low lipid content of cord blood not of maternal body burden.

[22] Most heavily chlorinated should refer to PCBs with 10 chlorine atoms despite the fact that decachlorinated PCBs are rarely seen. Presumably the author is referring to the most heavily chlorinated of the congeners present. This is another example of where terminology has increased confusion and decreased understanding of the effect of PCBs in the environment.

"Neurodevelopmental and behavioral deficits based on tests of visual recognition and memory at 7 months and 4 years of age. [They] reexamined these children at 11 years of age and found that many of the neurobehavioral deficits had persisted, that is, poorer short- and long-term memory an lower IQ scores" (Feeley et al, S6). As mentioned in Section 3.3.3, this series of studies has been severely criticized for flaws in methodology and logic that decreases the weight of conclusions reached.

Health effects other than neurocognitive have been examined as well. Statistics on cancer mortality do not indicate increased incidence among people living in the Great Lakes basin nor is there evidence of adverse human reproductive effects including congenital anomalies (Colborn et al., 1990). Conception delay has been investigated on a number of occasions with little evidence to suggest an association with PCB exposure. Buck et al. (1999) found no association between conception delay and paternal fish consumption while Courval et al. (1999) found a modest association, in males only, between consumption of sport-caught fish and conception delay.

The weight of evidence (human and animal studies) suggest that some human populations (and their health) may be at risk because of greater exposure (i.e. aboriginal people, sport fish consumers and the urban poor) or because of greater sensitivity (i.e. the elderly, pregnant women, fetuses, nursing infants of mothers who consume large amounts of Great Lakes fish, men and women of reproductive age, and immune compromised persons) (Feeley et al., 1998). On the other side of the coin, there is increasing evidence that the nutritional benefit of fish consumption, especially for trace minerals and polyunsaturated fats, outweighs the potential risk associated with fish-borne contaminants (Chan, 1999).

3.4.3.2 Northern Quebec

The Arctic ecosystem has been extensively studied with regard to pollutants throughout the marine and terrestrial ecosystems. Despite a lack of local PCB sources (except abandoned DEW line sites), PCBs are detectable in most media throughout the region. A complex mechanism of long-range transport combined with weak sunlight, excessive ice

cover and low temperatures that reduce the biodegradation processes allow high ambient contaminant levels to persist as compared to southern regions (Pellerin and Grondin, 1998).

For aboriginal peoples in the Arctic, exposure to PCBs is through consumption of traditional or country foods. Fauna of the north are generally long-lived, lipid rich and at the top of relatively simple food webs (Van Oostdam et al., 1999). In much of the population (although there are specific gender and age differences), consumption of country foods makes up a majority of the daily dietary intake (Dewailly et al., 1994). PCB (and other contaminants such as mercury, cadmium, radioactive isotopes, chlordane, and toxaphene) levels in media including human serum and breast milk have been documented in a many of studies with fairly consistent results. For example, mean PCB breast milk levels in 1989 and 1990 were 2.9 μg/g in the milk fat of Inuit women compared with 0.52 μg/g lipid in southern Quebec women (Dewailly et al., 1992).

Serum PCB levels in adult Inuit were 4.1 μg/g lipid versus 0.13 μg/g lipid in southern Quebec adults (Ayotte et al., 1997)[23]. Liver and adipose tissue levels in adult Inuit were found to be well below those which induced severe adverse health effects in lab animals but may be approaching the point where subtle effects may be occurring (Ayotte et al., 1996). Overall, contaminant levels in the far north are from 2 to 8 times higher than in southern populations (Rhainds et al., 1999; Ryan and Dewailly, 1997; Ayotte et al., 1997) even given the variety of analytical techniques and ways of reporting PCB levels (i.e. congener specific analysis, total PCB, as Aroclor 1260). In the case of breast milk, contaminant levels may exceed acceptable daily intake levels for infant foods.

Despite these concerns, very few studies of the health effects from increased contaminant levels have been carried out in this population. In a preliminary study of infant health and

[23] The Great Lakes region (including portions of southern Quebec) have been described by many authors as exceptionally contaminated by numerous chemicals including PCBs. The potential effect on the general population in this region is typically limited to those persons consuming sport caught fish from the lakes. The remainder of the population is exposed to "background" levels of PCBs as in other parts of Canada. The Inuit on the other hand, are relatively dependent on wild game which can be heavily PCB contaminated and hence are exposed to a greater extent than their southern countrymen.

PCB exposure, Muckle et al. (1998) noted an increase in infectious illness thus suggesting an effect on immune ability. The authors note however that the results may also be attributable to decreased immunization success in far north populations. Studies relating the dietary intake of country foods versus market foods and breast-feeding versus bottle-feeding indicate that the health benefits of traditional diets outweigh the potential health deficits of contaminants (Muckle et al, 1998). This is even though daily intake estimates range from 53 – 76 µg/day total PCB (Kuhnlein et al., 1995, Chan, 1998) when the tolerable daily intake guideline from Health Canada is 55 µg/day (Chan, 1998) or 1µg/kg body weight/day. Country foods contribute not only to social and cultural identity but are essential sources of trace minerals such as selenium and long-chain fatty acids that contribute to cardiovascular health (Kuhnlein & Kinloch, 1988; Dewailly et al., 1996). In some communities, the use of country food is not a choice but an economic necessity. The traditional diet is basically healthy despite contaminant levels. Imposed changes based on contaminant levels alone have great potential for negative health impacts (Receveur et al., 1997).

Table 3.6: PCBs in traditional food groups (µg/g wet weight)

Food Group	Arithmetic Mean	Maximum	% > Guideline
Marine mammal meat	0.080	0.390	0 (a)
Marine mammal blubber	1.90	26.6	33 (a)
Terrestrial meat	0.006	0.021	0 (b)
Terrestrial organs	0.010	0.039	0 (b)
Fish	0.052	2.60	<1 (a)
Birds	0.290	8.66	<1 (c)
Plants	0.006	0.030	0

a. 2µg/g for fish
b. 0.2 µg/g for meat
c. 0.5 µg/g for poultry

Chan, 1998

3.4.3.3 Swan Hills, Alberta

In Alberta, problems with hazardous chemicals have not been as dramatic as the United States experience with Love Canal or Times Beach or as devastating as Bhopal or Chernobyl. There are numerous industries with heavy reliance on and production of hazardous chemicals but either through luck or good management, a major incident has

not occurred. There have been many hazardous waste incidents in Alberta but Swan Hills was the first with PCBs.

The only stationary hazardous waste treatment centre in Canada is the ASWTC located northeast of Swan Hills and despite a number of accidents and allegations of fugitive emissions there have been no evidence reported of adverse human health effects in either the local residents or the employees of the plant. Immediately following the 1996 incident, a series of environmental measurements including human serum sampling were carried out. Table 3.7 lists the major findings of a human health impact assessment conducted by Alberta Health.

Table 3.7: Findings of the 1996/7 Alberta Health Human Impact Assessment

Medium	Total PCB µg/kg whole weight	Total PCB µg/kg lipid weight
Fresh Deer		
I. Fat	250	286
II. Muscle	22	890
III. Liver	74	2 400
Freezer Meat		
I. within 20 kilometres of the plant	18	3400
II. outside 20 kilometres of the plant	0.6	60
Fish Muscle		
I. Chrystina Lake	18[a]	Not reported
II. Roche Lake	1	
III. Chip Lake	0.2	
Fish Liver		
I. Chrystina Lake	70	Not reported
II. Roche Lake	8	
III. Chip Lake	6	
Pooled Human Serum Samples	0.2	31

a. different species tested in Chrystina Lake versus Roche and Chip Lakes

Table 3.8 Average Levels of PCBs (µg/kg or ppb) in Human Blood for Selected Nations

Location	Concentration of total PCB µg/kg (lipid weight)	Reference
East Canada	130	Ayotte et al., 1997
Northern Quebec	410	Ayotte et al., 1997
USA	120	Schecter et al., 1994
Germany	740	Wuthe et al., 1996
Sweden	900 – 1300	Svensson et al., 1995
Norway	1300	Johansen et al., 1996
Swan Hills, Alberta	31	AB Health, 1997

Source: AB Health, 1997

Table 3.9: Comparison of Human Serum Levels for Selected Nations

Location	Concentration of total PCBs (whole weight) µg/kg	Reference
Swan Hills, Alberta	0.1	AB Health, 1997
Japan (Yusho)	7	Masuda et al., 1985
Taiwan (Yu-Cheng)	38	Chen et al., 1994
Great Lakes		
I. male sport fish consumers	5	Hanrahan et al., 1999
II. female sport fish consumers	2	
Northern Quebec	26	Ryan et al., 1997

Tables 3.8 and 3.9 compare serum samples from selected locations to Swan Hills and surrounding area. Both tables illustrate how low exposure in Alberta is compared to other more well-studied populations.

Table 3.10 Comparison of Historical Events

	Swan Hills (1996)	**Yusho (1968)**	**Yu-Cheng (1979)**	**Great Lakes (Ongoing)**	**N. Quebec (Ongoing)**
Source	Industrial and fugitive emissions	Accidental contamination of food	Accidental contamination of food	Industrial pollution	Long range environmental Contamination
Main Chemical	PCB waste - PCB and PCDD/F	Heated Kanechlor 400/500 - PCB and PCDD/F/Q	Heated Kanechlor 400/500 - PCB and PCDD/F/Q	PCB, metals, pesticides	PCB, metals, pesticides
Route of exposure	Food chain	Consumption of rice oil	Consumption of rice oil	Food chain especially fish	Food chain especially marine products
Source Levels	Fish muscle: 12 pg/g ww TEQ total White tail deer: 890 ng/g total PCB	Rice oil: 2000 ppm total PCB	Rice oil: 5-200 ppm total PCB	Fish: 3 - 8000 ppm total PCB lipid weight Wild life: 70 ppm (fat basis) Sediment: 12 ppm (wet weight)	Breast milk: Total PCB: 4 mg/kg lipid TEQ total: 180 ng/kg lipid
Serum Levels	Total PCB: 0.14 ng/g whole weight	Total PCB: 6.6 ppb	Total PCB: 99 ppb	Total PCB: 5.4 ppb	
Health Effects	None	Chloracne Anemia in women, arthritis and herniated vertebral discs in men	Chloracne	Neurological disorders	None

1. Lipid adjusted back calculation with half-life of 7.1 years

Chapter 4: Risk Perception

4.1 Introduction

The complex chemistry, abundance of sources and extensive list of potential health concerns related to PCB exposure must compel the thoughtful reader to ask at least one question. Should I be worried about the effect of PCBs on my health? The answer is Yes, No and Maybe. Not a simple answer but entirely true.

Practically speaking, the factors that combine to try to answer this one question are so diverse that the answer is truly yes, no and maybe. Context, personal environmental philosophy, and available direct evidence, are only some of the factors that may influence whether yes, no or maybe is the correct answer for an individual in a given circumstance. **That** is risk perception. All three answers are right (and wrong) and reflect a level of understanding, clarity, knowledge and belief as perceived by an individual or a population.

4.2 Risk Perception

The study of risk perception began in the mid to late 1980's and has had a relatively tumultuous life. It was born of the need to link quantitative risk assessment to real life situations and has strived to bridge a growing gap between expressions of risk that derive from an assessment and the need to explain the relevance and context of those risks to people. *As a concept,* risk perception has evolved into two distinct categories, perceived risk and real risk, setting up *the perception* of a difference between the two.

Early work in the study of the perception of risk was an attempt to understand why some hazards were viewed as more risky than others. The results of these studies established a dichotomy between the expert and the public (Slovic, 1987) that has been difficult to overcome. These studies showed that experts saw what they considered the over-estimation of risks by the public. Risks perceived by the public were not substantiated by

scientific data and were often labeled by experts as emotional (and therefore unreasonable) reactions to risk situations. The public, on the other hand, decried the use of cold, impersonal data to represent the impact of potential hazards on factors important to them but not easily quantified. Experts believed their assessments were based on well-documented evidence (real risk) while the general public based their assessments on speculation and emotion (perceived risk). This chasm between expert and public perception has been blamed for failures in siting industrial facilities such as hazardous waste treatment plants and nuclear-based technologies and chemical use in general (Williams et al, 1999). Truly this dichotomy between real versus perceived risk has not served either group.

One of the most influential studies in the field of risk perception came from Paul Slovic (1987). He noted that despite a decrease in technical risk, advances in health care and a generally rising standard of living, people's perception that they were exposed to greater risk was on the increase. Slovic's efforts to study the apparent difference between technical risk assessments and people's perception of risk led to the development of a taxonomy of hazards using "psychophysical scaling and multivariate analysis techniques to produce quantitative representations or 'cognitive maps' of risk attitudes and perceptions" (Slovic, 1987). This study is typical of early attempts to quantify and define risk and while this technique remains very popular in the risk perception field, its central role has been somewhat overshadowed by the introduction of culturally based theories of risk perception and the influence of context to the study of risk. Nonetheless, it is representative of continuing efforts to quantify subjective issues and to answer the question which initially triggered the study of risk perception – "How safe is safe enough" (Starr, 1969)[24].

Dividing risk into quantitative and qualitative or objective and subjective factions has an historical basis. In the not so distant past, risks, or more accurately hazards to ones

[24] This question, in variety of forms, has been the bane of risk perception from the beginning. In all likelihood it is unanswerable.

health[25], were limited by the physical detection abilities of the five human senses; sight, hearing, smell, taste and touch. If a deviation from normal (and hence safety) was detected, a judgment was made as to the degree of danger that event presented. If the danger was judged great enough, the food was not eaten or the route not taken. Today, a hazard to ones health is more often invisible to the senses and come to one's attention only as a result of someone else's work. In an environmental context, the classic hazard scenario involves ultra-low dose exposure to multiple chemicals, completely invisible and beyond the layperson's range of experience (Lidskog, 1996). If someone doesn't tell us we are exposed, the likelihood of knowing of the exposure based on our own experience is very low. As a result, we have to use information we receive from alternate sources such as scientific research to form our opinions and beliefs. This is often where confusion arises. Contrary to popular belief, science is a tool, a way of learning and a way to view the world around us. It is not the gospel truth, always right or cast in stone.

4.3 Evidence and Inference

There is a distinct difference between what we (and scientists) can accurately classify as direct evidence and what is more accurately classified as indirect evidence, inference and conjecture. Misclassification of evidence may arise when the source of evidence is viewed as authoritative, creating the appearance of direct evidence. In some cases, partial evidence is given the seal of scientific approval just because scientists are involved. This is not direct evidence. Direct evidence encompasses those things one can collect tangible observations about; things about which supposition or assumption are not primarily required for accurate interpretation. For most health risk issues, PCB exposure included, there is surprisingly little direct evidence as was seen in Chapter 3.

There are a number of good sources of evidence for health risk. One of the most common sources of direct evidence is the death certificate. It includes the age, gender and apparent

[25] As the reader may have noticed, new terms have evolved to discuss risk in this field. Science and research have developed their own language quite apart from every day speech, using familiar terms in unfamiliar ways to define concepts within their own subspecialty. This is not purely semantics. Many concepts are new and simply don't have accurate terms which completely and accurately describe them. Take for example, the word risk. The multitude of definitions for that one word is staggering.

cause of death of the individual and requires very little interpretation. A better source of direct evidence is an autopsy which may reveal fairly clear evidence of the cause of death. On the other hand, "indirect evidence and inference is based on epidemiological studies designed to determine risk factors relating to health risks" (Thomas and Hrudey, p.2). As we move from direct to indirect evidence, the level of certainty about that evidence decreases. Box 4.1 illustrates in more detail, the use of evidence in understanding PCB exposure.

Box 4.1: Direct and Indirect Evidence

In Japan in 1968 there was an incident in which people consumed PCB tainted rice oil. Before this incident, human exposure to PCBs was unheard-of. PCBs were never intended for human consumption and exposure had never been an issue. Following the incident, health officials were at a loss for information about the possible health effects from ingestion of PCBs. The available direct evidence was minimal at best. With a limited degree of analytical certainty, researchers at the time were able to measure the amount of PCB in contaminated rice oil. They were able to measure, with limited certainty, the PCB levels in the tissues of presumptive victims of the poisoning. This is direct evidence. We can speculate that consumption of tainted rice oil contributed to increased serum and tissue PCB levels but we cannot say for certain. If we had pre-consumption blood levels, observed the ingestion of a specified amount of contaminated rice oil and were able to measure post-ingestion PCB levels we would have stronger evidence of a link between the rice oil and human tissue PCB levels. Even if researchers were able to do this, they are assuming there were no other sources of PCB contamination in any other food or environmental media (air or water) that our presumptive victim was exposed to. If that same person suffers a health deficit such as liver cancer, we can speculate about possible associations but a broad range of other evidence is needed before even considering whether ingested PCBs were responsible for the cancer.

As shown in Box 4.1, to begin to understand exposure and health effects and to create a logical and cohesive theory, we necessarily use inference and assumption. Assumptions and inferences are standard parts of scientific reasoning based on values and judgments that do not necessarily reflect 'truth' but attempt to extrapolate from the available direct evidence. Scientists are human too. Personal biases and experience shape which assumptions and inferences are used and which are set aside. This facet of the scientific method is perhaps best seen in the final paragraphs of a scientific paper. '*Conclusions*' are not firm and final answers despite the firm and final tone of the word. 'Conclusion' is a misnomer that more accurately could be called "deductions given the available evidence".

Another overlying complication in using and understanding evidence is understanding the difference between causation and the more usual scientific position of a greater or lesser degree of association that usually falls short of the criteria to establish causation (Harrington, 1998). Determining whether a cause-effect relationship exists when a statistical association is found between exposure and disease, and when chance, bias, and confounding are all determined to be unlikely explanations of the findings, requires a judgment from investigators (Hennekens, 1987). "Such a judgment can only be made in the context of all evidence available at that moment and as such must be reevaluated with each new finding" (p. 39). To assist in that judgment, "the WHO published a ... list of eight guidelines for judging causation ... (Beaglehole et al., 1993)

a. Temporal relation
b. Plausibility
c. Consistency
d. Strength
e. Dose-response relationship
f. Reversibility
g. Study design
h. Judging the evidence

These guidelines are ordered in a logical sequence for making judgments on causality. They are not weighted equally, and their relative contribution to a final judgment will vary from one situation to another" (Thomas and Hrudey, 1997).

Another way to examine the health evidence is to classify the evidence as necessary to cause a given health condition, sufficient to effect a given health condition or simply contributory to a given health condition. Necessary means the health condition cannot occur without that cause (i.e. the HIV causes AIDs). Sufficient means that exposure to that cause will guarantee the health condition, a difficult test that is rarely met. In all likelihood, PCBs are simply contributory to observed health effects associated with exposure.

With regard to PCB exposure, despite years of data and countless studies, there is only one health deficit for which there is adequate evidence to support a judgment of cause-effect. That health deficit is chloracne, the disfiguring skin condition associated with high-level organochlorine exposure. All other postulated health deficits have not accumulated the weight of evidence required for a cause-effect judgment nor have they satisfied the available criteria. In all likelihood, PCB exposure is a contributory factor in a variety of health conditions.

4.4 Science as a tool of Risk Perception

> "There is something fascinating about science, one gets such wholesale returns of conjecture out of such a trifling investment of fact" (Mark Twain).

According to Beck (1992), "science is the principal social institution trusted as being competent to make knowledge claims about risk" yet contrary to popular belief however, the aim of scientific study is not to find definitive answers but to stimulate debate and encourage further exploration of topics and ideas. Its dynamic nature is exceeded only by its strong need for thoughtful skepticism. Science is often seen by the general public as possessing the tools to cure the world's basic evils such as air pollution, water pollution and disease and has given humans a belief in the security and manageability of risk. Less positively, science is also seen as a tool used to legitimate risk estimates and foster social acceptance.

The divergence of uses of science highlights one of the controversies associated with it. For all its assets, science cannot provide definitive statements of safety. It is a means not an end and the information it provides can be confusing and contradictory. Science is far from unanimous and "there exist different groups of experts, different ways of seeing things and different judgments" (Lidskog, 1996). This is especially true in new and controversial areas such as environmental risk. Questions of emerging scientific (and public) interest can have a distinct lack of direct evidence and hypotheses surrounding new issues may simply be untested or un-testable given the available technology. Inevitable gaps in direct evidence are filled with inference and assumption based on

individual judgments to create cohesive and logical theories to support understanding a changing world.

Still, for all its limitations, uncertainties and assumptions, science remains the best way we have yet found to pursue knowledge of "the truth" (c.f. C. Sagan, *The Demon Haunted World*, 1995). The scientific method "is an essential balance between ... an openness to new ideas ... and skeptical scrutiny of all ideas, old and new" (p.304). Additionally, not everything produced by science will have equal credibility – the scientific method will not be applied equally between researchers or topics thus a continuum of 'good' to 'less good' research and results will exist. As noted before, scientists are only human and like the general public have unexamined biases and values that may influence their work. Hypothesis testing, peer review and healthy skepticism exist to monitor scientific study and to try to control the effect of personal judgment and bias. Every conclusion must be open to debate and revision because uncertainty is an inevitable part of science. It does not indicate ignorance but is a reflection of the complexity of the interaction of multiple and complex biological systems with a broad spectrum of chemicals and chemical processes. Unfortunately however, even here in the 21st century "we [really] know little more about the health effects of exposure to ... invisible industrial contaminants than fourteenth century Europeans knew about the cause and prevention of the Plague" (Vyner, 1988).

Take PCB exposure for example. Until 1968 there had been no large-scale incidents involving major human exposure to PCBs. As a result, no direct evidence of health effects. The only information scientists had at their disposal once an incident was identified was complementary information on exposure to similar organochlorine compounds and anecdotal reports from occupationally exposed groups. Epidemiological studies were the only option for ethical examination of potential health effects. The conclusions reached by scientists at that time were that PCB ingestion was associated with several health effects including skin disorders, liver failure, cardiovascular abnormalities, nerve conduction deficits and a unique fetal PCB syndrome caused by intrauterine exposure to PCBs. As analytical techniques developed and studies were

repeated and expanded, fewer certainties were associated with human exposure to PCBs. Scientists now feel that contaminants in the rice oil other than PCBs (i.e. PCDFs and PCQs) were *more likely* responsible for observed health effects. The health effects observed in Japan are unlikely due solely to PCB exposure and the contributory effect of PCBs may be minimal at best. Increased data has led to increased uncertainty. Science has been able only to disprove parts of a hypothesis (i.e. that PCBs are responsible for a number of health effects) – not prove it. As with other hypotheses, there are always competing hypotheses for which evidence (direct and indirect) exist. Only prudent judgment will decide which hypothesis is currently most feasible under the current conditions and knowledge.

But humans want certainty. They don't want to be exposed to potentially dangerous chemicals. Scientifically speaking however, environmental chemicals are particularly adverse to definitive answers. In the environment, contaminants are often present at very low levels and in combination with other chemicals. They have not been studied with respect to their health effects and in some cases have barely been identified at all[26]. Analytical techniques have improved to the point that extremely low levels of chemical are detectable but as techniques improve and detection limits fall, the *meaning* of the numbers has become more and more obscure. The gap between the ability to detect a chemical and an understanding of its clinical effect is continually increasing creating more and more uncertainty and less and less understanding of what the presence of those chemicals means. For example, there is a belief among some members of the general public that if a contaminant is detectable, it is a hazard and therefore must be removed. Zero levels of contamination and zero risk are not now nor have they ever been attainable (see Box 4.2). This is one of the greatest fallacies in understanding environmental chemical exposure.

---

[26] Chlorination disinfection byproducts are a particularly good example of this. While the usefulness of chlorination of drinking water is undeniable, the variety of compounds formed by reaction between chlorine and organic components of drinking water is legion, many of which have yet to be identified let alone studied for human health effects.

Box 4.2: **There is no zero!**

Exposure to environmental contaminants cannot be avoided. To talk in terms of zero is futile and only confusing. To illustrate this point, take benzene, a recognized human carcinogen and a typical constituent of gasoline. If you evaporate one teaspoon of benzene into the air, wait for it to mix and disperse equally across the entire planet, how many molecules of benzene from that teaspoon would any typical adult anywhere on earth inhale over the course of a day?

Chemicals become well mixed throughout large portions of the troposphere in a matter of weeks (Hemmond & Fechner, 1994). We will assume that our benzene will evenly disperse across the entire volume of the atmosphere of the entire planet (4×10^{18} m^3)[27], that it will not decompose in a few weeks and that the normal adult inhales about 20 m^3 air/day[28]. Given that the density of benzene is 0.8765 g/mL, the molecular weight is 78.11 g/g-mole and that a teaspoon is 5 mL, there are 0.05611 g-mole of benzene in that one teaspoon. The number of molecules in a g-mole is given by Avogadro's number (6.02×10^{23}) therefore there are 3.38×10^{22} molecules of benzene in that one teaspoon. If this number of molecules were evenly dispersed throughout the lower atmosphere there would be 8,444 molecules in every cubic metre of air in the lower atmosphere. A typical adult breathing at 20 m^3/day anywhere on earth, would breathe in ~170,000 molecules of benzene every day!

Another prominent source of environmental information is the media. The media is not typically known for its completeness or accuracy (Sagan, 1995). Its role is to inform but also to entertain and as someone once said, "the greater sin in news reporting is missing a story, not misrepresenting it". The world of news reporting is big business and as such must present information in such a way as to stimulate readers or viewers to consume their product. Common events are not news nor are those events where few people are involved or effects are minimal or remote. A published or broadcast news item is more likely to be a rare event with catastrophic potential involving a large number of people than an event which affects few people in non-spectacular fashion. Reporting these events however, give the impression that they are commonplace and applicable to a wide audience. Environmental events that have made the news in the recent past include incidents such as the contamination of drinking water in a small Ontario town (and the death of at least six people) (c.f. Walkerton, Ontario, 2000) and the discovery of high PCB levels in dead killer whales on the coast of British Columbia (Washington Post, 1999).

---

[27] McGraw-Hill Encyclopedia of Science and Technology – 8th Edition (1997). McGraw-Hill, Inc.; NY.
[28] Covello & Merkhofer (1993)

Even then, spectacular contamination incidents may not remain in the news for long. Take for example, the fire at the Hub Oil recycling plant in central Alberta, August 9, 1999. In this incident, an oil recycling facility located within a major Canadian city burned to the ground, blanketing much of the city including residential areas with thick black smoke. The fire was news for less than two weeks (Calgary Herald, 1999). The potential (from a technical standpoint) for contamination of the surrounding people and environment was enormous yet sustained public outcry and media interest did not occur. Compare that incident to the ASWTC incident of 1996. A hazardous waste recycling plant experienced a technical failure that resulted in the release of process gases to the environment. After investigation by the company, it was found that approximately 4 kilograms of PCBs were released to the environment (personal communication, G. Latonas, 2000). Within weeks, the media had front-page stories that persisted for months and speculated on the potential impact of the incident to the environment and health (c.f. The Edmonton Journal, 1997). The government commissioned a human health impact assessment and health risk advisories were put in place. Four years later, the Internet Website for the government organization that instituted the advisories lists only the health risk advisory for this region despite numerous other advisories across the province, some of which have been in place for decades.

A further example of media attention to a risk issue is cancer. According to the National Cancer Institute of Canada (2000) there has been a steady decline in cancer related mortality in all age groups under 60 years of age with the most new cases and cancer deaths occurring in Canadians aged 70 years or more. Even cancers that have a suspected interaction with environmental contamination (e.g. breast cancer in women) have not experienced a significant rise in mortality despite growing concerns of increased environmental chemical exposure. Regardless, it is not difficult to find a current story in either print or verbal news about cancer. Given the role of the media, that must indicate that people are interested. To that end, technical risk assessments have, for a number of

years, been driven by the risk of developing cancer[29]. Although research is progressing, there are still many unknowns about the mechanisms of carcinogenesis such as the presence (or absence) of a threshold for carcinogenic transformation remains unanswered. Depending on the model used, different levels of accuracy may be achieved in representing the effect of chemical exposure on the development of cancer. This is of course dependent on the assumptions used to build the model, many of which are untested or un-testable. This uncertainty and lack of definitive answers is difficult. The skepticism with which science must view the world has extended to the general public to the point that science itself is viewed with such disbelief that nothing is believed and leading researchers feel we are on the brink of a new dark age where all of science is distrusted, unused and disbelieved (Sagan, 1995).

Science and the media, despite the best efforts of many talented people, cannot reflect an objective truth. Indeed, many theorists suggest that no 'objective' reality can be defined. Rather, a range of value-laden and person-dependent realities can be articulated. Attempts to present "the truth" rely on inference, assumption, judgment, probability and prediction. Hence the dichotomy in risk perception is not between the lay person and the expert, it is between what we actually know with some degree of certainty and what we would like to, need to or think we know. In most cases, PCBs included, there is a serious mismatch between what we can confidently tell people and what they really want to know. This mismatch arises not because of any industrial or governmental conspiracy or cover-up but because there are practical limits to our ability to know.

4.5 Deductions Given The Available Evidence

To return to the question we posed at the beginning of the chapter, "Should I be concerned about the effects of PCBs on my health?" the answer most reflective of "reality" is maybe. Polychlorinated biphenyls are viewed by much of the general public as the scourge of the planet and as part of a small group of truly global pollutants; there is

[29] Newer assessments take into account the variety of PCB mixtures that were sold (versus a single commercial product) and have resulted in a downgrading of the cancer risk and division of risk into three tiers depending on route of exposure, environmental pathway and persistence in the environment.

some truth to that viewpoint. What cannot be substantiated is the belief in a broad range of confirmed human or environmental health effects.

When PCBs were first developed and exploited for industrial use, they were more effective, efficient and safer than the products they replaced. The resistance to degradation for which they were extolled has now become very much a negative trait as PCBs persist in the environment, bioconcentrate along food chains and are readily stored in adipose tissue. There is however, very little direct evidence of health effects in humans or wildlife/fish (outside of persistence). In the context of environmental contamination, the available direct evidence is also very limited. The multitude of complex biological systems and number of chemicals involved make determining even associations very difficult.

Of the environmental scenarios studied, the most comparable, useful and widely studied scenario is the Great Lakes ecosystem. Enormous efforts have been devoted to studying the ecosystem and the effects that PCBs may have had on the flora and fauna (humans included). Very little if any direct evidence of a causal association between PCBs and human health effects has been found although there is a growing body of speculative literature that attempts to link PCBs (among other organochlorine chemicals) to an expanding list of subtle endocrine effects.

Close to thirty years of study have been conducted on humans exposed to high concentrations of PCBs through dietary exposure. The results are inconclusive. The most recent studies (Guo et al., 1999) link only chloracne, goiter, anemia and joint and spine disease to PCB exposure with any confidence. There have been no significant clusters of any health deficit including cancer. Some trends are intriguing and epidemiological studies have suggested a number of possible subtle health effects such as loss of intelligence quotient (IQ) points, decreased memory, decreased physical growth and depression (Phibbs, 1996) but definitive results remain to be found.

There are few things we can say with certainty about PCBs and human health. They include (but are not limited to):

a. PCBs are not infectious. You cannot 'catch' PCB-disease from someone.
b. There is no evidence of PCB-associated genetic mutation.
c. PCBs cannot cause an adverse effect unless you are exposed to them.
d. At high exposure levels, PCBs may cause a disfiguring skin condition known as chloracne.

The fact remains that there are PCBs in the environment and people are exposed to them through consumption of 'contaminated' food products. Many of the 'consumable' mammals, which are most heavily exposed to PCBs, have not been rigorously studied and the toxicology must be inferred from more mainstream meat products (e.g. using beef guidelines for moose or deer, poultry guidelines for duck or goose). PCBs are not purely benign compounds in the sense that they do not interact with the biological systems of which they become part. Toxicological study has indicated an ability of some PCB congeners to interact with known biological receptors indicating a potential ability to disrupt natural systems. Evidence of a disruptive function is lacking.

What is not lacking are the assumptions and inferences involved in the perception of the risks associated with exposure to PCBs. Understanding risk in today's world is not simple no matter how it is defined. Quantitative methods and statistical analysis together with qualitative methods and social context analysis are integral to defining the meaning of risk but if no one cares, it isn't a problem and logically presents no *meaningful* risk. Qualitative and quantitative analyses are incomplete without the input of the other. Risk is therefore a combination of direct evidence (quantitative) and knowledge (defined here as what we think we know; qualitative) together with the trust that they complement each other and create a semblance of acceptable reality. Truth, even in risk, is what we define it to be.

Chapter 5: Study Context and Design, Results and Discussion

The treatment and disposal of hazardous waste is an important part of environmental management and the only stationary hazardous waste treatment facility in Canada is located in Swan Hills, Alberta. The people of this community were willing to support the siting of the facility within their community despite potential environmental and personal health risks. Personal reasons for accepting the plant near their homes are likely very diverse and may include belief in the economic benefit to the community and a feeling of social responsibility in managing hazardous waste. The plant provides jobs on-site and there is valuable economic spin off for the community. The other major employers in the region are in the oil and gas industry, notorious for its cyclical prosperity. Despite some controversy the plant was, and continues to be a, welcome neighbor in the community.

A variety of hazardous chemicals are routinely handled at the plant; PCBs are simply the most notorious. Community representatives express feeling a sense of social legitimacy in dealing with hazardous waste that has made some of the more negative aspects of hazardous waste treatment more palatable. A feeling persists among some residents of providing a valuable environmental service for the rest of the country. In fact, the ASWTC has successfully destroyed stockpiles of PCBs that have been poorly managed at other locations (c.f. Baie Comeau). In contrast to the media reports, there appears to be very little concern among Swan Hills residents about the possible health effects of PCB exposure. Is this lack of concern based on knowledge or simple resignation to the presence of the plant? Is there in fact, little concern? What types of information do the residents of Swan Hills feel they would like regarding their current situation, if anything?

5.1 Method

Ethics approval from the Health Research Ethics Board (HREB) of the University of Alberta was received prior to beginning this study (Appendix 2). The target population for the survey was Swan Hills residents over the age of 18 years who had resided in the community for two or more years. For ease of interview, English was selected as the

language of choice[30]. The data collection for this study was accomplished through a random digit dialing telephone survey of 50 Swan Hills residents conducted by trained telephone interviewers from the Population Research Laboratory on the campus of the University of Alberta. Due to the random-digit dialing method, all residents of Swan Hills with a telephone had an equal chance of being contacted to participate. An approximately equal gender split was achieved by using the following guidelines:

a. If an adult male answers the phone and is willing to be interviewed, he is the respondent.
b. If an adult female answers the phone and there is an adult male present who is willing to be interviewed, interview the male. If the male is not willing to be interviewed, and the female is willing, interview the female.
c. If an adult female answers the phone and there is no adult male present, choose her as the respondent.

These guidelines are based on previous surveys conducted by the Population Research Lab which have found that 60% of the time, the first household contact is female (personal communication C. Drixler, 2000). A standard script outlining the study was used to inform potential respondents and verbal consent to participate was obtained (Appendix 3). Responses were entered into an SPSS database from which frequency and basic statistics were taken. The Population Research Lab conducted the interviews between May 29, 2000 and June 2, 2000. Twenty-seven men and twenty-three women were surveyed.

Five questions from the survey (questions 3, 19, 20, 22, and 23) were also placed on a general Alberta survey. The Population Research Laboratory of the University of Alberta conducted this survey between January 5, 2000 and February 8, 2000. A total of 1203 persons were surveyed with approximately equal representation from Edmonton, Calgary, and the remainder of the province. As in the Swan Hills survey, the target population was persons 18 years of age or older and living in the designated area. Included in this survey were questions from other University of Alberta researchers on

[30] Post-survey analysis revealed no potential respondent turned away because of a language barrier.

poverty and health, biotechnology, perceptions of hope, health risk advisories and sexual partners.

5.2 Community profile of the Town of Swan Hills according to the 1996 Canada Census
The town of Swan Hills had a population of 2030 persons having experienced a slight decrease in population numbers from the 1990 census. In comparison, the province of Alberta overall experienced a slight increase in overall population going from just over 2.5 million people to more than 2.6 million in 1996. There were slightly more males than females (1085:945) in Swan Hills which is roughly comparable to the situation in Alberta overall (1,348,300 males: 1,348,520 females). The community is young with the average age 25.6 years, more than 10 years less than the Canadian average of 35.8 years and more than 8 years less than the provincial average of 33.7 years. Also indicative of the youth of this community is the percentage of the population under age 14 (32.3%) compared to Canada overall (20.5%) although the province overall is young with 32.8% of the population under 15 years of age. As well, out of 2030 people, only 55 were older than 55 years of age. This is 2.7% of the entire population versus the Canadian value of 20.9% and the Alberta value of 17.4%.

The employment rate and average income in the Town of Swan Hills is higher than the respective national and provincial figures. The employment rate was 96.3% compared to the national average of 89.9% and the provincial average of 92.8%. The average income was more than $5000 more than the Canadian average of $25,196 and more than $4000 higher than the provincial average of $26,138. Of the major employers in Swan Hills, five out of six are oil field related (i.e. Gulf Oil and Gas, Anderson Oil, Shell, Esso and Federated Pipelines). The sixth is Bovar, owner/operator of the ASWTC (c.f. www.ruralnet.ab.ca). They employ around 100 people although the actual number varies depending on the current and anticipated volumes of waste being processed at the plant. The education level in the town is relatively high with 33.6% of the population holding some type of trade or non-university certificate or diploma and 14% having completed a university degree.

5.3 Historical Context of the Siting of the ASWTC

In the late 1970's, the hazardous waste industry in Alberta experienced a number of failures of attempted facility siting. This left the province without an integrated hazardous waste disposal system. As a result, the provincial government decided to attempt a facility siting through a new approach using voluntary community participation and site elimination versus the typically more proscriptive site selection process. Communities across the province were invited to partake in a provincially-funded environmental assessment to determine if their community was suitable for hosting a hazardous waste treatment facility. Many communities took advantage of this opportunity. In some locales, the geography was unsuitable for a hazardous waste facility; in others, there was strong community opposition. In the end, two communities remained suitable and interested - Ryley, Alberta, south east of Edmonton and Swan Hills, north west of Edmonton. One of the final factors in the siting decision was a plebiscite vote in the respective communities. Swan Hills voted 79% in favor of the plant while Ryley voted 77% in favor. Ryley was considered the better site for a number of technical reasons including proximity to waste producers but Swan Hills was ultimately chosen to host the facility for mostly political reasons (Sherbaniuk, 1998). Hosting the Alberta Special Waste Treatment Centre was an economic windfall for the small community, supplying jobs and economic spin off to a town that had lost much of its economic base when the boom-bust cycle of the oil market turned to bust (Figure 5.1).

The plant functioned for roughly 10 years amidst comparatively little controversy. It was responsible for destroying Alberta and Canada's hazardous and refractory chemical waste, including polychlorinated biphenyls (PCBs) On October 16, 1996, a weld failed on ducting carrying PCB vapours to the incinerator allowing the airborne release of PCBs, dioxins and polychlorinated dibenzofurans (a.k.a. furans) (personal communication G. Latonas, 2000). After an internal assessment, the management company of the plant notified Alberta Environmental Protection (AEP) who subsequently contacted Alberta Health. Within a month, Alberta Health issued two health advisories limiting consumption of wild game and fish within a 30-kilometre radius of the plant.

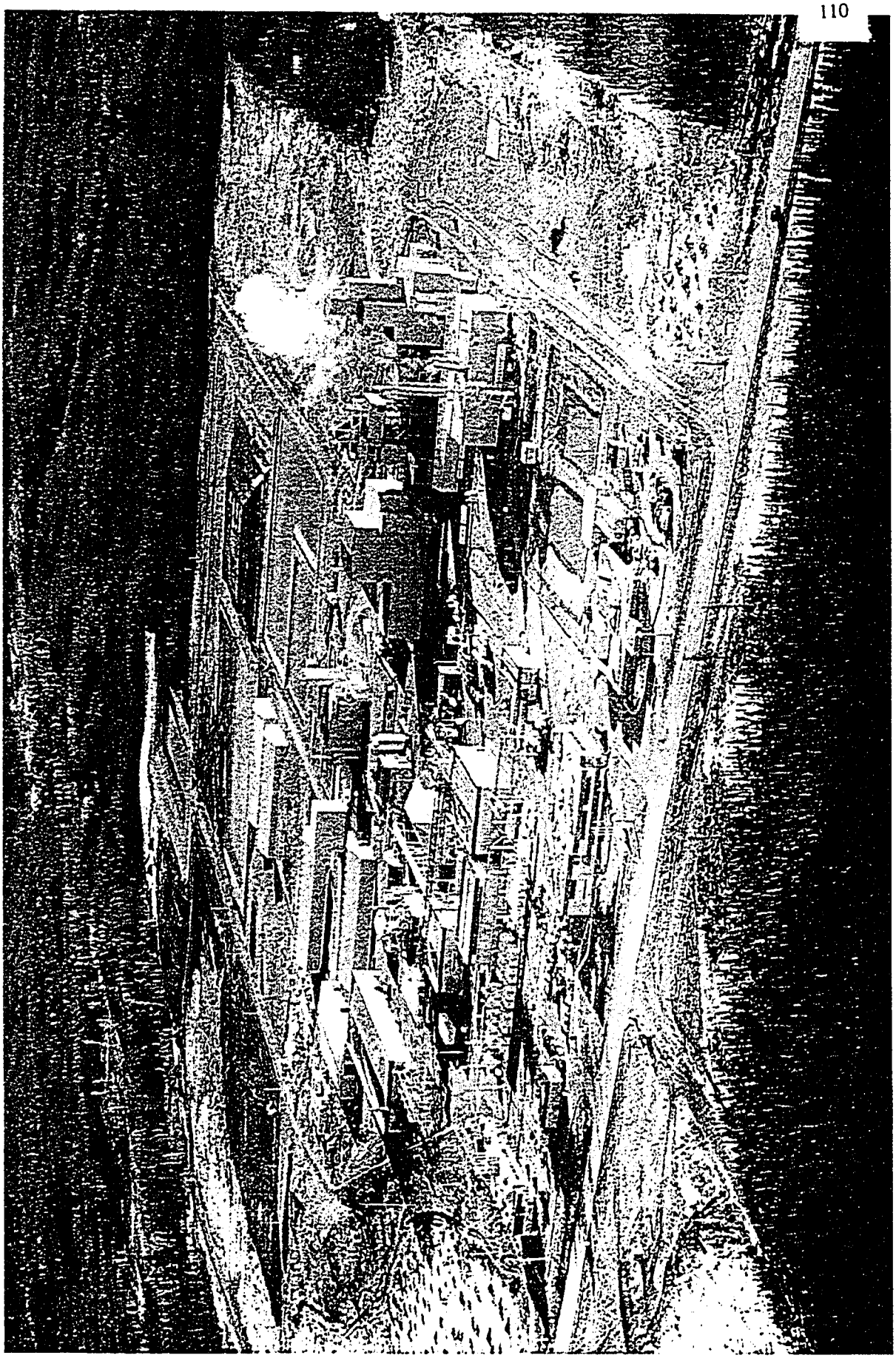


Figure 5.1 Alberta Special Waste Treatment Centre

Further investigation by AEP and non-government scientists revealed that in addition to this accident, there had been a number of smaller incidents at the plant including long-term fugitive emissions. Concerns from community residents about possible personal exposure prompted Alberta Health to conduct a human health impact assessment in Swan Hills and surrounding area. Part one of the assessment consisted of a short questionnaire regarding diet and activity. Part two was a more extensive questionnaire that was supplemented by blood serum samples from willing residents. The findings of the surveys and serum sampling resulted in the modification of the health advisories (i.e. reduced geographic boundaries and special precautions for children and pregnant women) (Appendix 4). Efforts to communicate the results of the survey[31] were limited to one news conference with Dr. John Waters (the provincial medical officer of health), information supplied to the regional medical officers of health, posters and word of mouth communication between residents. Table 5.1 details the chronology of events that led to this study.

The health advisories issued in 1996 remain in effect to this date (July 2000) with no plans for revision or revocation in the foreseeable future. There is limited continuous monitoring of environmental and wildlife parameters in the Swan Hills region. Alberta Health has a wildlife-monitoring program, as does the operating company of the ASWTC and nearby First Nations groups. Results from the Alberta Health monitoring program are supplied to the office of the Provincial Medical officer who is responsible for communicating with residents.

In addition to the Alberta Health Study (1997), a second human monitoring program in the Swan Hills area was conducted by the Medical Services Branch of Health Canada. The federal study was initiated in response to criticism of the Alberta Health study that stemmed from a lack of identifiable First Nations persons in the study and a perceived inadequacy in the assessment of traditional native diet and lifestyle. HPB conducted the study (including serum samples) focusing specifically on First Nations people (50

[31] Results indicated that residents were not unduly exposed nor were blood levels higher than an unexposed control group (AB Health, 1997).

participants in Phase I and 142 participants in Phase II) in the Swan Hills region. The study found that exposure and serum levels (including high consumers of traditional foods) were well below Canadian guidelines for PCBs and dioxins (Health Canada, Medical Services Branch, 1999).

There is no evidence of widespread human contamination or detrimental human health effects in the Swan Hills area from either the Alberta Health Human Impact Assessment or the First Nations study.

Table 5.1: Chronology of events

1972-1979	Alberta government conducts inventories of waste production to determine feasibility of building a waste treatment facility. Results reveal a serious problem. More than 100,000 tonnes/year hazardous waste produced, ~20% dumped directly into the environment.
1979	Kinetics Contaminants (Canada) Ltd. proposes to site a hazardous waste disposal site in Fort Saskatchewan. Public opposition forces abandonment of the plan.
1979	Private attempts to site hazardous waste disposal facility in Two Hills (east central Alberta) fail due to local opposition
1980	Creation of the Hazardous Waste Management Committee to evaluate how to proceed with siting a hazardous waste treatment facility. Committee recommends central Alberta location "in or near" the Edmonton-Red Deer-Calgary corridor.
1980	Environmental Council of Alberta holds hearings across Alberta to determine criteria and concerns with regard to a hazardous disposal site. Creation of Hazardous Waste Task Force composed of government representatives and the Hazardous Waste Team composed of members of the public.
1981-1982	Compilation of short list of candidate sites. Among criteria was voluntary participation of community. A total of 52 communities volunteer for preliminary assessment.
Aug. 1982	Ryley plebiscite - out of eligible voters, 77% vote in favor of hosting the facility.
Sept. 1982	Swan Hills plebiscite. Out of eligible voters, 79% vote in favor of hosting the facility. Veteran, Alberta also votes in favor of the facility.
March 1984	Alberta government announces selection of Swan Hills as the chosen site despite Alberta Environment recommendations for Ryley.
April 1984	Establishment of the Alberta Special Waste Management Corporation (ASWMC)
May 1984	Chem-Security (Ltd.) chosen to build, own and operate the facility. "Stated it was prepared to undertake 100% of the facility's cost if it could be assured of receiving... 40,000 tonnes of waste per year"
Dec. 1984	Chem-Security purchased by Bow Valley Resource Services Ltd. (BVRS) meeting a requirement for Canadian ownership
March 1985	Construction of the plant begins.
Spring 1985	BVRS proposes joint-venture deal based on "deemed equity" i.e. guaranteed return on money borrowed to invest in the waste treatment project.
Dec. 17, 1985	The contract of John Elson, chair of the ASWMC, is terminated by environment minister Fred Bradley because of the committee's refusal to recommend the joint-venture deal proposed by BVRS.
Sept. 1987	Plant officially opens
Feb. 1987	Joint venture deal signed, 60/40 ownership between BVRS and the Crown. "BVRS could borrow all of the money it needed to build the facility and ... write off depreciation as an expense [and] get it back as a reimbursable cost" (Sherbaniuk, p. 30)

1988	Centre complete and processing hazardous waste. Reports of fugitive emissions, complaints from customers about service delays and growing stockpiles begin.
1989	Royal Bank assumes control of BVRS. Renamed Bovar Inc. and purchased later by Trimac Ltd.
May 8, 1992	Natural Resources Conservation Board (NRCB) approves $85 million expansion after extended hearing and strong opposition from aboriginal and environmental groups.
Sept. 1, 1993	Oilfield wastes exempted from classification as hazardous waste.
Feb. 1, 1995	Importation of out-of-province hazardous waste approved by NRCB after lengthy hearings
July 12, 1996	Joint venture agreement ends. Alberta government pays Bovar $140 million. Total provincial subsidy estimated at about $10 per Albertan – about $250 million since 1985 (Sherbaniuk, 1998).
Oct. 16, 1996	**Malfunction of a transformer furnace at the ASWTC results in the airborne release of process gases containing PCBs, dioxins and furans.**
Nov. 8, 1996	Alberta Environment notified of leak and potential environmental impacts.
Dec. 1996	Alberta Health notified of leak and potential human health effects. Bovar and related companies charged with six infractions of the Environmental Protection and Enhancement Act. Potential fines $3 million.
Dec. 13, 1996	First public health advisory issued. Warns the public against eating wild game taken from the Swan Hills area. Advisory based on two animal tests from Alberta Environmental Protection.
Dec. 23, 1996	Two hundred litres of liquid PCB spill at the ASWTC. Not reported to the public (Edmonton Journal, 1997)
Jan. 1997	Canadian federal government allows PCB export to the U.S.
Feb. 1997	Alberta Health begins detailed human impact assessment including serum sampling for PCB and dioxin exposure.
May 15, 1997	Public health advisory revised to limit consumption of wild game taken from within a 30 km radius of the ASWTC to 370 grams per month, avoid eating organ meat or using fat from game harvested within a 30 km radius of the ASWTC, pregnant or breast-feeding women as well as children whould avoid eating meat taken from within a 30 km radius of the treatment centre and to continue to avoid eating fish from within a 30 km radius of the plant. The previous advisory suggested no consumption of fish or wild game from within the 30 km radius for any person.
July 1997	U.S. border closes to PCB import
July 1997	Explosion in the main incinerator at the ASWTC leads to temporary closure. PCBs in air are measured at nine times normal; ground levels are three times normal despite occupational guidelines not exceeded and measured values at the plant back to normal within twenty-four hours.

Sept. 4, 1997	Results of blood testing of Swan Hills and surrounding area residents released. Serum PCB and dioxin levels were found to be lower than other Alberta populations. Advisory remains in effect. Study heavily criticized over lack of aboriginal involvement prompting additional study by Health Canada.
Oct. 30, 1997	Complete restriction on consumption of fish from lakes near Swan Hills down graded to consumption limits. Fish preparation guidelines included in advisory. Ongoing advisories for mercury remain in effect.
Jan. 2, 1998	AEP drops 3 charges and Bovar pleads guilty to 3 violations of the Environmental Protection and Enhancement Act.
Feb. 98	Main incinerator restarted.
April 20, 1998	Chem-Security (Ltd.) guilty plea accepted by the court.
April 1998	Native bands agree to drop all suits against plant in exchange for $100,000 per year to conduct their own environmental monitoring.
May 1998	Forest fires cause the evacuation of the town of Swan Hills
June 1998	Tests on mice and voles show reduced or non-detectable PCB levels as compared to those found immediately following the 1996 incident at the ASWTC
Oct. 23, 1998	$625,000 fine levied against Chem-Security (Ltd.), half of which is earmarked for independent research via a creative sentencing plan. This fine is more than twice the previous record for environmental infractions in Alberta.
Dec. 2, 1998	Swan Hills Natives Health Canada Study finds PCB levels below established guidelines but higher than the general population. Advisories remain in effect.
April 14, 1999	Bovar president lifts company ban on providing media and public access to the plant.
May 15, 1999	Native representative and trapper, Charlie Chalifoux settles with Chem-Security regarding alleged harm to health and livelihood resulting from the 1996 incident.
June, 1999	Draft report of the Lesser Slave Lake Health Study released. Exposure of First Nations people "8x higher" than residents of Swan Hills. Analytical differences cloud comparisons.
Dec. 1999	Importation of international hazardous waste approved by federal government
Dec. 3, 1999	Trust fund for risk communication and assessment research established at the University of Alberta
Mar. - Sept. 00	This study

5.4 The People

There are potentially 5 relevant local stakeholders in this scenario: the management company of the ASWTC (Bovar) and its employees; the other residents of the town of Swan Hills (n~2000); the First Nations people of the region (n~8000); environmental groups; and the health care professionals of the region, including Alberta Health.

Bovar concedes there have been a number of incidents at the ASWTC including emissions, spills and odor incidents. The most serious occurred on October 16, 1996. It is/has been Bovar's position that these incidents have had no significant environmental impact and to refrain from commenting in the media. The medical officer at the plant has a monitoring system in place for the employees of the plant, including yearly physical and blood chemistry examination. He states there have never been any health concerns related to PCBs at the plant nor elevated serum PCB levels (personal communication, Dr. J. Cheng, 1999).

Generally, the town of Swan Hills (i.e. residents and local government) also views the incidents associated with the plant as not harmful to the environment. Residents voted 79% in favor of siting the facility in their community and the plant retains a high approval level within the community (personal communication, Swan Hills Mayor G. Pollock, 2000). The plant provides a source of employment for approximately 100 people and is a world-class facility with state-of-the-art technology. According to the town of Swan Hills Internet website, one of the towns' claims to fame comes from being at the geographic centre of Alberta in a region of virgin boreal and sub-alpine forest. The website describes abundant summer and winter outdoor recreation in a region of diverse flora and fauna not found anywhere else in the province. The town is situated on the third largest oil field in Canada and encroaches on the last habitat of the Great Plains Grizzly. In contrast, the incidents and advisories have been widely reported in the media and have consequently negatively affected the economic base of the region by decreasing tourism, recreational hunting and sport fishing in the area (personal communication, Swan Hills Mayor G. Pollock, 2000).

The third group of stakeholders is the members of the Lesser Slave Lake Regional Council composed of members of the Sucker Creek, Driftpile and Swan River First Nations. Although the closest settlement is roughly 60 kilometres north of the ASWTC, the Lesser Slave Lake Indian Band have expressed their opposition to the ASWTC from its very inception. They are the group most outwardly concerned about adverse effects from the plant. The concerns expressed range from impact on traditional food sources and habitat to lack of economic spin-off from the plant to their communities. Some of the grievances have been addressed through separate monitoring studies conducted by Health Canada and an out-of-court settlement with Bovar. A concurrent study of health risk concerns in the First Nations communities is ongoing as a joint project between Rutgers Centre for Environmental Communication and the University of Alberta.

Some environmental groups, such as the Toxics Watch Society based in Edmonton, are also very vocal in their opposition to the ASWTC. One spokesman has been cited numerous times in the media about the negative effects of the chemicals processed at the ASWTC. Together with the First Nations, a video presentation entitled "Poisoning Paradise: A Native View of the Swan Hills Waste Treatment Centre" (1996) was produced detailing native concerns about the ASWTC. Environmental group personnel provided the 'scientific evidence' to support their statements.

The final local stakeholder group is the health care professionals of the region. The Swan Hills Health Care Centre is a very small facility with only one resident physician and four active treatment beds. The regional health authority (Aspen, No.11) provides health care services to the community through a variety of mobile clinics (personal communication, K. Bouman, Swan Hills Health Care Centre Administrator, 2000). Physicians from the Barrhead region travel to the community on a regular basis. The regional Medical Officer of Health is responsible for the advisories and communication regarding. Alberta Health became involved in health risk communication in response to the incidents involving the ASWTC. Residents at a public meeting criticized Alberta Health for perceptions of short

term involvement and a very low profile in the region despite ongoing concern in the community since the 1996 incident and human exposure study.

5.5 The Survey Tool

The survey used in this study was designed to assess a variety of health risk perception factors such as environmental philosophy and trust in information sources. Accordingly, a variety of question formats were used. Questions on perspectives of quality of life, views specifically directed at the ASWTC, attitude and opinion questions, questions about actual risk and risk avoidance behaviors made up the bulk of the survey. There were a total of 37 questions that took between 25 and 60 minutes to answer. Thirty-three questions were closed ended and four were open-ended (Appendix 5). Questions 30, 32, 33, 34 and 36 were included on the survey for a colleague doing a complementary study of health risk advisories and were not directly analyzed in this study. The main components of the survey are outlined below.

5.5.1 Demographics

Respondents were asked general demographic questions such as age, gender, occupation, education level and duration of residence in the community as well as specific questions designed to elicit preliminary perspective on the ASWTC (i.e., Do you or any member of your family work at the ASWTC? If you had the opportunity to vote today on whether the community should have hosted the ASWTC, how would you vote?)

5.5.2 Risk Perception

Respondents were asked general questions about the perception of risk in everyday life and with respect to the ASWTC. The facility in question has been one of the major employers in the region and has received considerable media attention since before its construction. Rather than alluding to the presence of generic "industrial facilities" as has been done in other studies (c.f. U.S. EPA, 1990), respondents were asked specifically about what they know about this facility.

5.5.3 Sources of Information

Respondents were asked to identify the people or groups of people from whom they receive information about health risk from environmental chemicals. They were also asked to assess the level of trust of and knowledge possessed by each source. They were asked about changes in the level of trust and to identify precipitating factors to changes in level of trust.

5.5.4 Attitudes and Opinions

Questions in this section were modeled after previous studies of risk perception, notably the United States Environmental Protection Agency's (1990) study of public knowledge and perception of chemical risk, Kraus, Malmfors and Slovic's (1992) study of intuitive toxicology and the health risk perception surveys of the Canadian public by Slovic et al. (1992) and Krewski et al. (1995), respectively. Broadly, the questions looked at issues surrounding environmental exposure levels, toxicology, cost-benefit relationship, trust. industry and perception of risk from chemical exposure.

5.5.5 Risk Communication

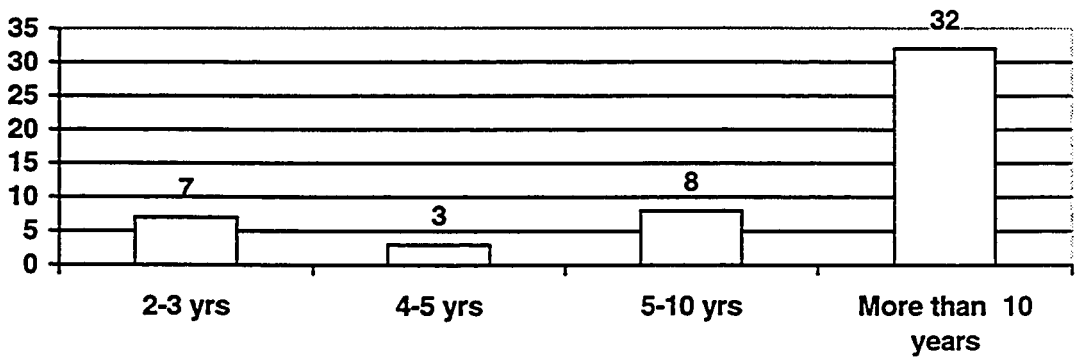
The survey ended with a series of questions about current risk communication efforts in the community. Respondents were also asked to describe their preferences for further risk communication efforts. The survey ended with questions specifically designed to elicit information about health risk (consumption) advisories and the role of the public in establishing and removing such advisories.

5.6 Swan Hills Survey Results and Discussion

5.6.1 Demographics:

Twenty-three women and twenty-seven men were surveyed. Duration of residence in Swan Hills varied according to Figure 5.2 with the majority of respondents (32/50; 64%) residing in Swan Hills for more than 10 years. Men in the sample had generally resided in Swan Hills for a greater duration than the women with twenty-four of twenty-seven men having lived in Swan Hills for more than 5 years compared to only 16/23 of the women.

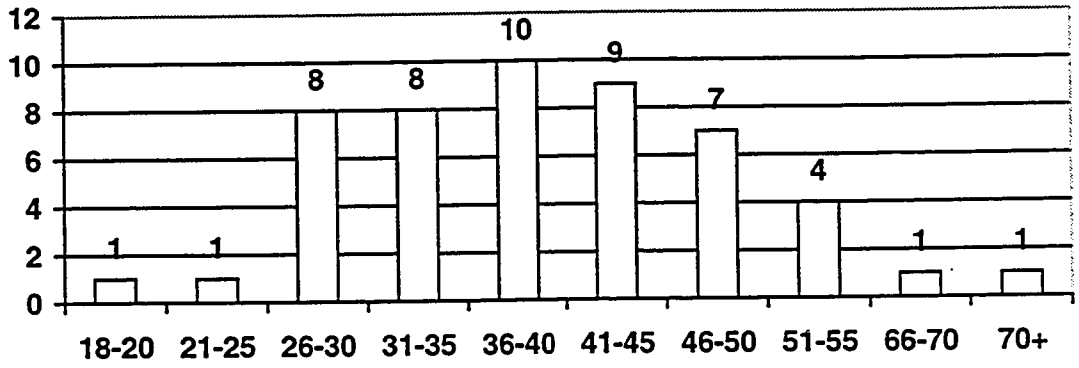
Figure 5.2: Duration of Residence in Swan Hills



All respondents were over 18 years of age with the majority between 26 and 50 years of age (Figure 5.3). The gender split between age groups was roughly equal. Thirty-six of the fifty respondents were married (18 men: 18 women), 5 were not married but were living with someone (2 men: 3 women) and the remainder (9) of respondents was single (7 men: 2 women). Of the fifty respondents, 32 (64%) were living in households with children under the age of 18. The average number of children in those households was 2 with the average age of the children, ~11 years.

The gender split is roughly comparable to Census figures (1996), i.e. 1.14:1 male to female in 1996 and 1.17 to 1 in 2000). Average age of survey respondents was approximately 39.6 years of age which was more comparable to the 1996 Canadian average that the 1996 Swan Hills Census data. Education level was comparable to census data with 36% of respondents holding a trade certificate or university degree (1996 Census, 33.6%).

Figure 5.3: Age of Respondents



The highest level of education achieved by the respondents is illustrated in Figure 5.4. The majority of respondents had completed at least high school with 18 holding trade certificates or university degrees. Of the seven persons who had completed university, six were women (see Table 5.2). Occupation type was divided into 5 groups: resource based industry, service industry, homemaker, construction or other. Twenty-four respondents classified their employment as resource-based, twelve as service industry, seven homemakers, 2 construction workers and 5 others. Twelve respondents or members of their family (24% of the sample) worked at the ASWTC. At the time of the survey, the ASWTC had downsized its workforce to approximately 50 employees thus 12 respondents represented approximately 25% of the ASWTC workforce.

Demographic factors may play an important role in shaping perspectives on risk. Factors such as gender (males perceive fewer and lower risk; childbearing aged females perceive increased risk), presence or absence of children in the household (presence associated with increased perception of risk), education level (increased education associated with increased perception of risk), and length of residency (increased residency associated with increased perception of risk) are often seen as important aspects in the perception of risk.

Figure 5.4: Highest Level of Education Achieved

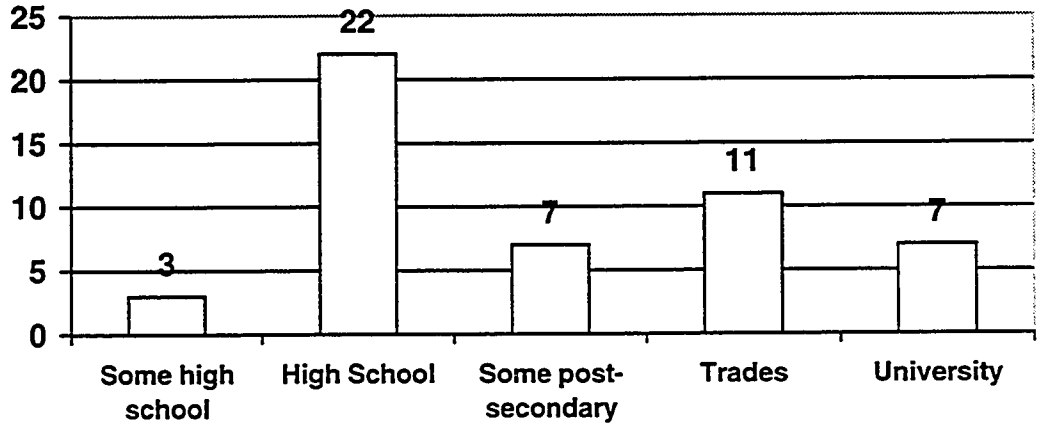


Table 5.2: Cross-tabulation of Gender and Highest Level of Education Achieved

	Highest Level of Education					
	Some High School	Completed High School	Some Post-Secondary	Trades or Non-University	Completed University	Total
Male	2	14	3	7	1	27
Female	1	8	4	4	6	23
Total	3	22	7	11	7	50

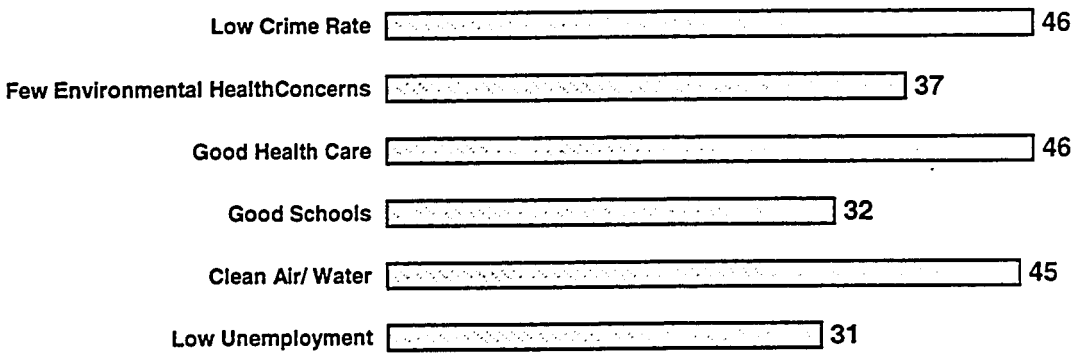
5.7.2 Survey Questions

Question 1 asked respondents to compare the community of Swan Hills to other communities on six scales: unemployment, cleanliness of air and water, schools, health care, environmental health concerns and crime. Overall, respondents believe that the community of Swan Hills has a lower rate of crime and better health care, air and water than other communities ($\geq$ 90% agreement). The most positive response was to the quality of air and water which 45/50 respondents felt was better than other communities. Environmental health concerns, quality of schools and unemployment rates were also judged better overall than other communities although at a somewhat lower frequency. When cross-tabulated with gender, the lowest scores were given by men for the quality of Swan Hills schools where only 13/22 respondents felt Swan Hills has good schools

compared to other communities. The lowest scores given by women in this question were in the subsection that asked them to compare the rate of unemployment in Swan Hills to other communities. Ten women out of 22 respondents disagreed that Swan Hills has a low rate of unemployment compared to other communities. Of those who felt that compared to other communities, Swan Hills does not have a low rate of unemployment, 10/18 had resided in the community for more than 10 years. This question may reflect recent lay-offs at the ASWTC. Overall however, views of residents reflect the positive attitude advertised on the town's Website.

Similar results were seen in other sections of this question. Those who compared Swan Hills poorly to other communities on the basis of clean air and water, good health care, low rate of crime and environmental health concerns, resided in Swan Hills more than 10 years. Note however that of the total number of respondents, 32/50 had lived in Swan Hills for more than 10 years. This may account for the observed trend as previous studies of risk perception have found longer residency periods associated with increased perceptions of risk. Figure 5.5 illustrates the basic results from this question.

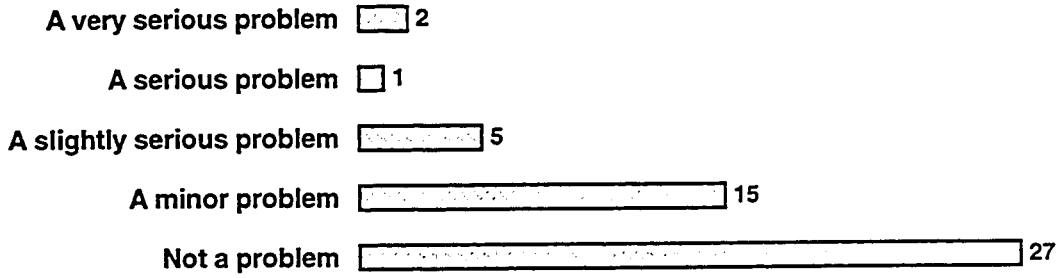
Figure 5.5: Swan Hills compared to other Communities



Question 2 asked respondents to compare health and safety risks and risk to health from chemicals in Swan Hills to those risks in other communities. Most respondents (42/50) felt that chemicals in and around Swan Hills were not a problem or only a minor problem. Five respondents felt chemicals in the community were a slightly serious

problem, one person felt they were a serious problem and 2 people felt they were a very serious problem. Of the people who felt that chemicals in and around Swan Hills were a serious or very serious problem, all three were male. Of those who felt that chemicals were not a problem or a minor problem, 22/27 were males and 20/23 were female. Of those who felt health and safety risks in the community were a serious or very serious problem, all three had resided in Swan Hills greater than 10 years. Figure 5.6 illustrates these responses. Response to this question was not surprising. As mentioned in the demographic description, almost half of the people who responded to this survey are employed in the resource industry. Some jobs in these industries can be dangerous with routine exposure to potentially dangerous chemicals and machinery. Studies of risk perception have shown an association between familiarity with risk and lowered perception of risk. Such may be the case here.

Figure 5.6: Comparison of Health and Safety Risks in Swan Hills to other Communities



Questions 3-8 were designed to assess respondents' knowledge of the ASWTC. Question 3 asked respondents the open-ended question "What do you know about the ASWTC?". Responses were varied with a range of responses from awareness of existence to open positive support. Slightly fewer female respondents were aware of the existence of the ASWTC (17/23 versus 23/27). Of those who knew more about the plant than simply that it exists, the respondent was more often male. Of those respondents with specific information about the plant (e.g. environmental contamination, lawsuits), all resided in Swan Hills greater than 10 years. Interestingly, the only respondent who claimed to know nothing about the plant also had lived in Swan Hills for greater than 10 years.

Comments associated with this question confirmed that a number of persons responding to the survey also worked at the plant. Many respondents feel the plant is providing a vital service to the province and the world at large. Many respondents were well informed about the technology, what is and what is not processed at the plant, expressed confidence in the safety procedures and felt the media had not accurately represented the plant or its contribution to the local and global community. Few comments associated with this question were negative. Negative comments included the one respondent who commented that he/she had

> "worked out there for a while and ...saw a lot of things happening and just being covered up" and one respondent reported, "the Centre has spills that are not reported. They think just because they are under a certain amount in litres that nothing has been harmed".

Respondents were well informed of current events associated with the plant such as the lay off of employees due to decreased waste volume and the proposal to import international waste. The number of comments associated with this question indicated a desire on behalf of respondents to talk about the plant and its effect on the community. Positive comments substantially outweighed negative.

This question was also included on the Alberta Population Survey. Respondents across the province were generally aware of the existence of the plant (784/1203) although at a lower frequency than Swan Hills and area residents (i.e. 65% versus 80%). Responses were as varied as in the Swan Hills survey. Of those respondents on the Alberta Survey who claimed to know nothing about the ASWTC (n = 288), 119 were from Calgary. Of the 288 respondents who claimed to know nothing of the plant 178 were female which was contrary to the Swan Hills survey in which the only person claiming no knowledge of the plant was male. Similar to the Swan Hills survey, respondents who knew more about the plant than that it exists were more likely to be male. Interestingly, of those respondents who claimed the ASWTC processed nuclear waste, only one person was from Edmonton with the remaining 7/8 from either Calgary or the rest of Alberta. The

other most frequent responses to this question were awareness of environmental contamination associated with the plant (261/1203), importation of hazardous waste from other countries (179/1203), and technical malfunctions (88/1203). Overall, respondents from Calgary were less likely to report specific information related to the plant; response frequency was lowest in almost all categories except an awareness that the plant was initially supported through Alberta tax dollars where 16/40 respondents were from Calgary.

Question 4 on the Swan Hills survey, which asked whether respondents believed the ASWTC posed a threat to the environment, found that a majority of respondents (38/50) do not feel the ASWTC poses a threat to the environment. Of those who feel the plant threatens the environment, 8/12 were male. This finding is slightly in contrast to risk perception literature which identifies males as having lower risk perceptions in general. Those who believe the plant poses a threat to the environment had lived in Swan Hills for 2-3 years (n= 2) or for greater than 10 years (n=10).

Confirming the response to Question 4 (i.e. that most respondents do not feel the ASWTC is a threat to the environment) was the response to Questions 5 that asked to what extent respondents were bothered by the presence of the ASWTC in their community. Forty-two out of 50 respondents are not at all bothered by the ASWTC. Of those that were bothered (5 males and three females), the concerns expressed include the danger the plant poses to health in the long run, dust, dirt and smoke in the air, the possibility of a major accident, long term damage to the environment and wild life, and transportation, importation, and communication issues (Question 6). Visible problems such as smoke in the air often initiate concerns over environmental contamination but in this case, invisible threats and concern about the future are more prominent. This is consistent with 21st century environmental threats (c.f Vyner, 1988) that are, more often than not, non-detectable by human senses. Interestingly, of those respondents who had lived in Swan Hills for greater than 10 years, 10/32 believe that the ASWTC poses a threat to the environment yet only 6/32 are bothered by the ASWTC near the community. Figures 5.7-5.9 illustrate the responses to this series of questions.

Figure 5.7: Do you believe the ASWTC poses a threat to the environment?

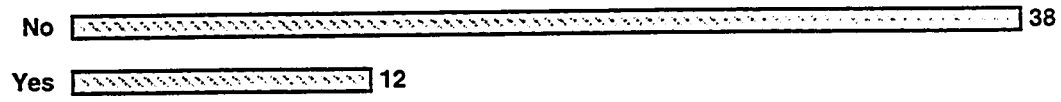
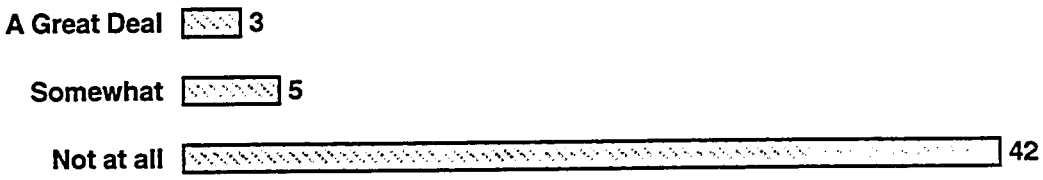


Figure 5.8: To what extent are you bothered by the ASWTC?



Questions 7 and 8 asked respondents to assess whether their perspective on the presence of the ASWTC in the community had changed in the years since the release incident in 1996. Sixty-two percent of respondents (31/50) felt their opinion of the treatment centre had not changed. For those respondents who reported a change in opinion, the most common reason for change was personal experience. A higher proportion of women reported a change in opinion than men (16/23 women versus 15/26 men). Some respondents noted that they have become more informed about the plant and its work either through scientific information or plant tours and have become more confident in its safety. Some respondents reported their opinion has changed due to health concerns (e.g. during pregnancy) or continuing concerns regarding the 1996 accident and subsequent incidents. One person was concerned that recent layoffs at the plant could compromise safety.

Questions 9-16 were designed to assess respondents' sources of information, trust in sources of information and knowledge level of those sources. Question 9 asked if respondents had heard or read anything about health risks from chemicals in the last three months; 38/50 had not, 12/50 had. This is substantially lower than the EPA study (1990) of Public Knowledge and Perception of Chemical Risk from which this question was derived. In that study, a little more than half of respondents had heard or read something

about health risks from chemicals in the last three months. Also different was the education levels of respondents who had heard or read something about chemical health risks. In the EPA study, respondents who had received risk information were more likely to be more educated (58% had at least some college education). In this study, 4 respondents had completed high school only, 4 had some post secondary education and 4 held a university degree or trade certificate.

Of the twelve persons who had heard or read anything about health risks from chemicals in the last three months, 6 were male and 6 were female; 3 respondents either worked at the ASWTC or had a family member who did and 7 out of 12 had lived in the community for more than 10 years.

Question 10 asked specifically what the respondent had heard or read. The types of information ranged from general environmental news in the local paper (e.g. mercury levels in fish) to focused articles written by Bovar to inform town residents of ongoing events and initiatives at the plant. Some respondents reported having read environmental monitoring reports or studies associated with the treatment centre. Some respondents mentioned articles in the Edmonton or Slave Lake newspapers that discussed the proposal by the ASWTC to import international hazardous waste. One respondent reported attending a town meeting sponsored by the plant to inform residents about the importation of international waste. One respondent reported hearing that

> "they are going to take out the treatment center to save our water. Some people from other towns will not come fishing here, they are scared that our fish are contaminated".

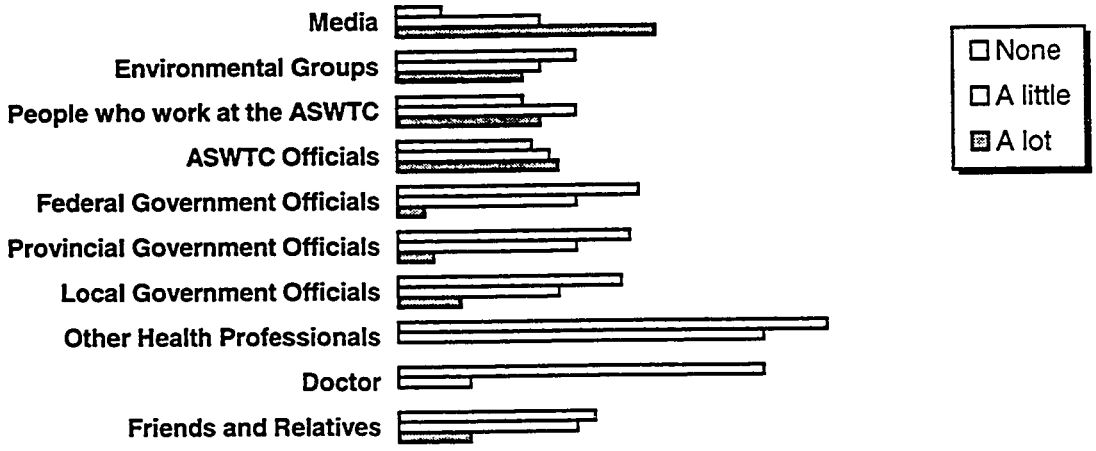
Overall, the most commonly mentioned sources of information were the media (6/50), ASWTC officials (4/50), local government officials (2/50), and environmental groups (1/50)(Question 11). If the question is analyzed by gender, the most commonly mentioned sources of information for males are the media (4/6) or ASWTC officials (3/6). For females, the most commonly cited source of information was also the media (2/6) or friends and relatives (2/6) and the local government (1/6), ASWTC officials

(1/6), or an environmental group (1/6). Respondents who had lived in Swan Hills more than 10 years had more varied sources of information about health risks from chemicals including local government, ASWTC officials, the media and environmental groups. Respondents who had lived in Swan Hills for 4-5 years reported the fewest number of sources of information, reporting receiving information only from ASWTC officials. Overall, the number of sources of information cited by all respondents was low and the foregoing proportions cannot be interpreted with any confidence.

Question 12 asked *how much* information respondents received from sources. Figure 5.7 illustrates the responses. The group from whom respondents received the most information was the media (29/50), followed by ASWTC officials (18) and employees (16), environmental groups (15), friends and relatives (8) and local government (7). The doctor and other health professionals were not mentioned by any respondents as a source from whom they received "a lot" of information. This response supports a previously recognized failure of health professionals to participate in risk communication (Harrington, 1998).

Analysis by gender showed a similar pattern. Men generally got more information from the media (23/27), people who work for the ASWTC (20/27), ASWTC officials (18/27), environmental groups or friends and relatives (15/27), the federal government (13/27) and the provincial and local governments (12/27). Women more frequently reported the media as a source of information (22/23), followed by ASWTC officials (17/23), people who work for the ASWTC (16/23), environmental groups (15/23), friends and relatives or local government (13/23), the provincial government (12/23) and the federal government (10/23). This is very similar to a 1995 survey conducted by Jardine et al. which found that Albertans claimed to receive 70-80% of their information on health risk from the media.

Figure 5.9: Amount of information from Sources

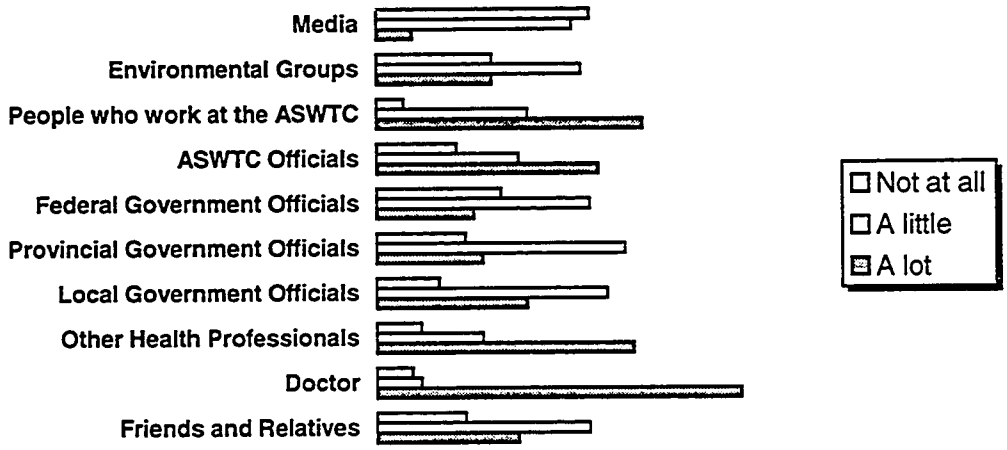


The amount of trust in sources showed a different pattern. Respondents reported having "a lot" of trust most frequently for the doctor (41/50) followed by people who work for the ASWTC (30), other health professionals (29) and ASWTC officials (25). The sources from which the fewest respondents reported having "a lot" of trust in were the media (24/50), the federal government (14) and environmental groups (13) (figure 5.10).

Results by gender were similar. For men, the source of information that received the most frequent assessment of "a lot" was the doctor (21/27), followed by people who work for the ASWTC (16), ASWTC officials and other health professionals (14), local government (11), friends and relatives (10), the provincial and federal governments (8), environmental groups (5), and the media (3). For women, more respondents reported having "a lot" of trust in the doctor (20/23), followed by other health professionals and people who work for the ASWTC (14), ASWTC officials (11), friends and relatives (10), environmental groups (8), local government (6), provincial government (4), federal government (3) and the media (1). The trust in government (at all levels) showed a trend of decreasing trust in the sub group that had resided in Swan Hills for greater than 10 years with more trust at the local level, followed by provincial officials and finally by federal officials.

This is comparable to the EPA study (1990) that also found that health professionals have the highest trust rating. The media on the other hand were given an intermediate rating on trust and chemical industry officials were given the lowest trust rating. In the Swan Hills survey, the media was mentioned least frequently as a trusted source of information but health care professionals were mentioned most frequently.

Figure 5.10: Trust in Sources



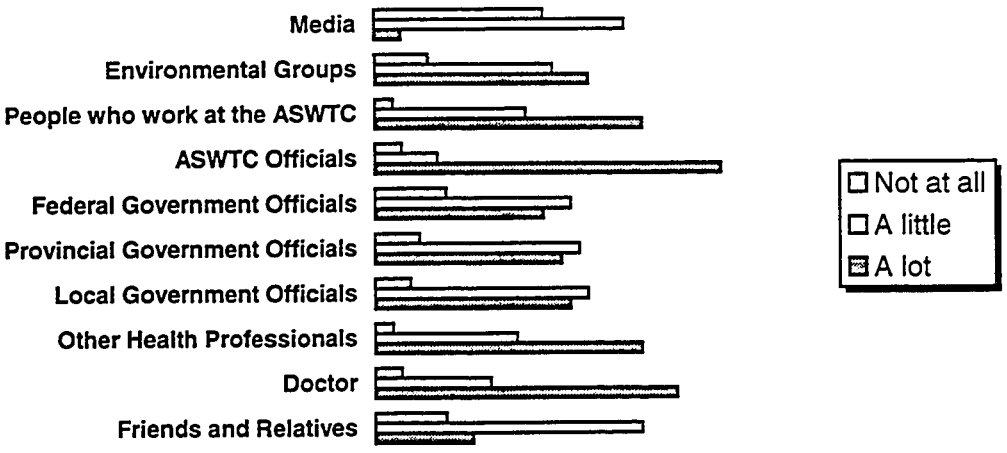
In terms of how knowledgeable sources are about the risks from chemicals to one's health, respondents reported "a lot" most often to least often, are ASWTC officials (39/50), doctor (34), other health professionals and ASWTC employees (30), environmental groups (24), local government officials (22), provincial government officials (21), federal government officials (19), friends and relatives (11), and the media (3). Figure 5.9 is an illustration of this information. The EPA study (1990) also found that chemical industry sources have the highest knowledge rating of all sources. The survey conducted among Swan Hills residents rated health professionals equal to the media in terms of knowledge – both relatively high.

By gender, the ranking of information sources in terms of knowledge is a little different. Men reported ASWTC officials most frequently as possessing "a lot" of knowledge about the risks to health from chemicals (24/27). Sources of information cited second in having

"a lot" of knowledge about risks from chemicals were people who work for the ASWTC and the doctor (17/27), other health professionals (15), the local and provincials governments (12), the federal government (11), environmental groups (10), friends and relatives (5) and the media (2). For women, the most knowledgeable source was the doctor (17/23) followed by other health professionals and ASWTC officials (15), environmental groups (14), people who work for the ASWTC (13), local government (10), provincial government (9), federal government (8), friends and relatives (6), and the media (1).

Analyzed by duration of residence, all groups most frequently assessed the doctor and other health professionals as having "a lot" of knowledge about risks to health from environmental chemicals except those respondents that had lived in Swan Hills for greater than 10 years. This group more frequently reported that ASWTC officials and workers had "a lot" of knowledge about health risks due to chemicals. Interestingly, environmental groups ranked high in sources, trust and level of knowledge about health risks due to environmental chemicals. This is in contrast with comments at a community meeting in Swan Hills where an overall level of distrust in the knowledge level and accuracy of information provided by environmental groups was expressed (Community meeting, Swan Hills, June 2000).

Figure 5.11: Belief in knowledge level of source



Questions 15 and 16 were designed to assess whether trust in information sources has changed in the years since the release incident at the plant and to determine some of the factors involved in that change. Figure 5.10 illustrates the responses to this question. Overall, respondents reported little change in trust of information sources. The level of trust in friends and relatives changed only a little (17/50) or not at all (33/50). For those who reported a change, two major themes emerged from the comments: first, layoffs at the plant have reduced trust in the ability of remaining staff to safely manage the treatment of hazardous waste and second, belief in negative media reports about the plant in friends and relatives of respondents has caused respondents to distrust the information and opinion of those same friends and relatives on issues related to the plant and hazardous chemicals. Many respondents to the survey commented on personal efforts to become more informed about issues related to the plant and report a high level of confidence in that information. Changes in trust of friends and relatives appeared to hinge on the comparison between personal knowledge and friends and relatives beliefs based solely on media reports. Typical comments included,

> "... they don't live in the area and are just going by what they hear in the media"
> " They seem to think whatever the media reports is gospel" , and
> "They are easily affected by the media".

Respondents who report a change in trust of the doctor generally reported an increase in trust. According to respondents, the fact that there is a new doctor in town has increased their trust level as well as the belief that he is in a position where he would have done some research into the environmental issues important to them. The same is generally true for other health professionals. They "are becoming more involved" and also are seen to be in a position where they would have an obligation to be more aware of environmental issues. Trust in other health professionals has not changed at all since the 1996 incident.

The change in trust of local government officials was about equal positive and negative. Two themes emerged from the comments of respondents. First was the belief that local

government officials are trying to communicate more often and more openly with the public. "They are getting better. Not only they are more informed themselves, they also inform people more about what's happening". The second major theme was that the local government was trying to cover up problems at the plant to protect the economic interests of the plant. Some respondents claimed they felt the local government did not care what the plant does so long as it makes money and felt there was pressure on local government to support the plant.

Trust in the provincial government was equivocal. Some respondents felt provincial government interest in the plant had decreased since the plant was sold to private interests and that secrecy and political agendas play a large role in the amount and type of communication they receive from the province. Other respondents felt that the 1996 incident at the plant had prompted better relations with provincial government officials such as the permanent placement of an AEP officer in town dedicated to working with the plant. Respondents reported they feel greater amounts of information from a variety of sources are now available to the provincial government, prompting them to keep residents better informed.

The change in trust of federal government officials was exclusively negative. Respondents felt the lack of support shown by the federal government for initiatives like the treatment centre in dealing with hazardous waste have impacted poorly on relations with the community.

Change in the trust of ASWTC officials was both positive and negative. For some respondents, mixed messages from company officials have left the impression of cover-ups and untruthfulness. Specific instances cited included lack of information about **all** spills and leaks and denial of the possibility of layoffs at the plant until after they occurred have all contributed to feelings of distrust. On the other hand, recent efforts to improve communication with community residents were recognized. Difficulties in communicating technical information to lay people were recognized. As one respondent put it:

> "They are now starting to be more public. They now have meetings to let us be aware of what they are doing out there which includes not just the town people but the whole outlying area which makes them more accountable. There are a lot of knowledgeable people out there so they have to be honest."

Change in trust of ASWTC employees hinged on the recognition of the depth of company loyalty and the need to protect personal employment sources. "They are caught in the middle, they need their job and nobody wants to bad mouth their job as they won't have it anymore". On the positive side, respondents reported getting to know ASWTC employees better and judging their information based more on personal attributes than strictly on an information basis.

> "Some you trust more and some you recognize that they are just as much a rumor monger as the next person".

Change in trust in environmental groups was decidedly negative[32]. Respondents commented that environmental group representatives are misinformed, work mainly to protect their own interests and agendas, fear monger and use the media to portray the situation negatively. This is in contrast to responses to question 14 that asked respondents to comment on knowledge level. In that question, respondents felt that environmental groups possessed "a lot" of knowledge. Perhaps "a lot" of knowledge does not reflect a belief in the accuracy of that same knowledge.

One respondent commented that he/she is

> "Becoming more familiar with their mandates. I trust them less. One of the reasons is that as I read and learn more about the plant, it shows to me how most environment groups are ignorant of the information. They are fear monger

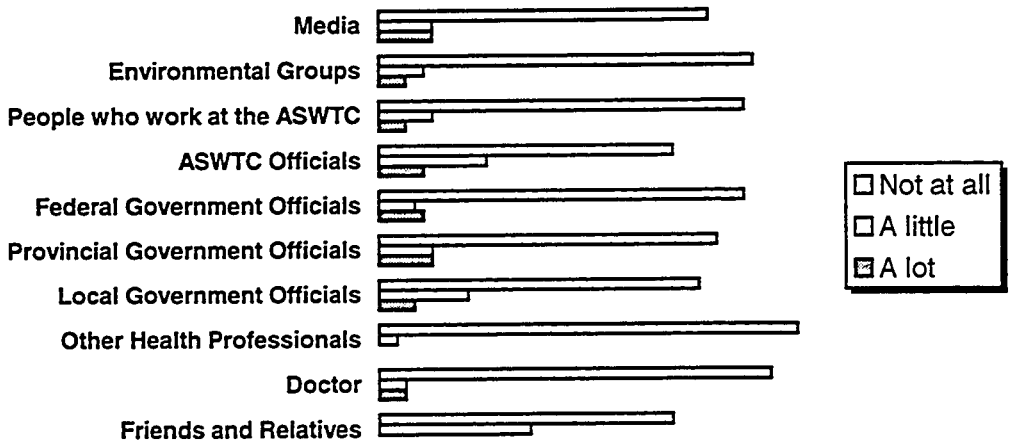
[32] Environmental groups have been vocal opponents to the ASWTC since before its construction. After the 1996 incident, they worked together with First Nations groups to attempt revocation of the plants operating license and therefore closure of the plant.

organizations, most of their people don't have the facts or a lot of knowledge about what they are dealing with".

Reaction to trust in the media was similar. Respondents felt many media reports were biased and inaccurate relying on sensationalization and partial truths.

"The reporters that report on situations at the plant do not understand the consequences to the environment and are not able to put things into perspective. I am very unimpressed and disappointed by the media...they do not get upset with the companies producing the waste."

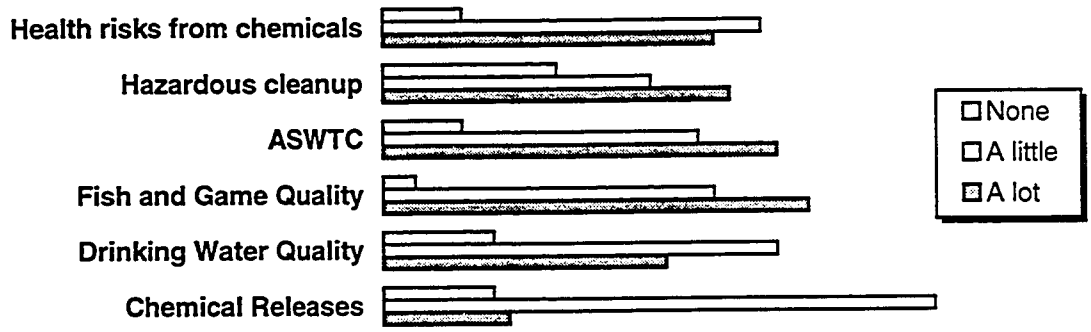
Figure 5.12: Change in the amount of trust



Question 17 asked for respondents' opinion on how much they feel they know about a variety of environmental topics and issues. Respondents reported they knew a lot or little about all the statements presented. More respondents reported knowing "a lot" about the quality of the area's fish and game (27/50), the ASWTC (25/50), activities to clean up releases of hazardous material (22/50) and health risks from chemicals in the area (21/50) than about releases of chemicals into the atmosphere (8/50) and the quality of the area's drinking water (18/50). The only two respondents who disagreed that they were well informed about the quality of the area's fish and game had lived in Swan Hills for greater than 10 years. The statements which respondents reported most frequently not knowing anything about were activities to clean up releases of hazardous material (11/50), releases

of chemicals into the atmosphere (7/50) and the quality of the area's drinking water (7/50). Generally, men felt they were more knowledgeable than women in all categories. In comparison to the EPA study (1990), Swan Hills residents reported a higher knowledge level on all topics. In the EPA study, respondents generally reported they knew a little or nothing about the topics presented. The Swan Hills sample was unaffected by age, industry employed in or education level.

Figure 5.13: General Environmental Knowledge



Question 18 asked for the respondents' opinions on a variety of environmental statements covering understanding of chemical activity, the concept of exposure, toxicological threshold, personal control issues, trust, and attitudes towards chemicals in general.

Question 18a was designed to elicit general views of the safety of chemical and came from the 1990 EPA study. In that study, 51% of respondents disagreed strongly and 70% disagreed overall with the statement "We should assume a chemical is safe until tests prove it to be dangerous". In the Swan Hills survey, respondents also generally disagreed with the statement. Thirty-nine of 49 respondents disagreed (19) or strongly disagreed (20). This suggests that Swan Hills residents are just as likely to be suspicious about the safety of chemicals as respondents to the EPA study.

Question 18b asked for opinions on the statement "Chemicals have improved our health more than they have harmed our health". Fifteen of 49 respondents (31%) agreed or strongly agreed with this statement. Five respondents had no opinion. This question is of course highly dependent on how the word 'chemicals' is defined. Interviewers did not

provide any further explanation but given the context of previous questions on hazardous waste, it is likely respondents would be thinking of hazardous chemicals although this cannot be confirmed. In previous studies (EPA, 1990; Krewski et al., 1995) the percentage of respondents who agreed with this statement were substantially higher (50% and 43% respectively) suggesting that other study respondents may have been less likely to be thinking of chemicals as meaning hazardous chemicals.

Responses to Question 18c which asked for opinions on the statement "Any release of chemicals into the air, water, or soil is unacceptable", support concerns expressed in earlier questions about chemical exposure. Thirty-two of 50 respondents strongly agreed or agreed with this statement. Three respondents had no opinion. Previous studies (EPA, 1990) have shown similar agreement (i.e. 66%) to this statement but contrary to earlier findings, responses from male respondents showed greater agreement. Influences from industry employed in, education and age were unremarkable.

Question 18d asked for opinions on the statement "The only time the public hears about the release of toxic chemicals is when the problem is so big it can't be kept secret any more". Twenty-nine of 49 respondents (59%) agreed or strongly agreed with this statement. Previous studies have shown a higher percentage of agreement (86%) perhaps indicative of the Swan Hills populations' first-hand experience with industrial chemical release.

To obtain views on exposure to chemicals and an understanding of dose-response relationships, Questions 18e and 18f were asked. Question 18e asked for opinions on the statement "It's not how much of a chemical you are exposed to that matters to your health, it's whether you are exposed at all". Comparable with previous studies, thirty-one respondents agreed or strongly agreed with this statement, 17 respondents disagreed or strongly disagreed and two had no opinion. For the majority of chemical risks, dose-response is an important variable, yet only 17/50 respondents to this survey hold an opinion consistent with the role of dose-response in governing health outcomes. As noted by Krewski et al (1995) "the public appears to have an 'all-or-none' view that equated the

mere fact of exposure ... with a high probability of being harmed" (p.237). This is also true of the Swan Hills respondents and is seen again in responses to Question 18f.

Question 18f asked for opinions on the statement "If a person is exposed to a chemical that can cause cancer, then that person is likely to get cancer later in life". Twenty respondents agreed with this statement. Eighteen disagreed or strongly disagreed and 11 respondents had no opinion. Of the respondents who disagreed with this statement, twice as many men as women disagreed (10 versus 5). This question had the highest frequency of no opinion responses perhaps indicating uncertainty or lack of understanding among respondents. The agreement frequency in this question is somewhat lower than in previous studies perhaps indicating a growing understanding of chemical exposure possibly based on personal experience. The opinions expressed in 18e and 18f are also seen in responses to Question 23 in which 22/50 respondents felt they would stop eating a food item if PCBs were found in it (presumably because PCBs are harmful to ones' health).

Question 18g asked for opinions on the statement "As an individual, there are many ways I can reduce my exposure to chemicals in the environment". Thirty-four of 49 respondents agreed or strongly agreed, 14 disagreed or strongly disagreed and one person had no opinion. This response is comparable to other studies that have shown people to feel relatively in control when faced with environmental threats (O'Connor et al., 1998). The response is perhaps reflective of answers to Question 17 which asked respondents to gauge their personal level of knowledge about a number of environmental topics. Swan Hills respondents expressed a high degree of confidence in their knowledge of the topics presented.

Similar to a question posed by Krewski et al. (1995; i.e. Is Canadian society becoming too concerned about small health risks) Question 18h asked for opinions on the statement "There are some chemical risks that are too small to worry about". Twenty-three of 49 respondents agreed or strongly agreed, 25 disagreed or strongly disagreed and one person had no opinion. Again, the definition of 'chemicals' may have influenced responses to

this question. Neil et al. (1994) found that 30% of public respondents to a survey on expert and lay judgments of chemical risk did not agree that a 1 in 10,000,000 lifetime risk of cancer from exposure to a chemical was too small to worry about. These responses have implications for resource allocation in a society increasingly concerned about health care funding. Can society afford to allocate resources to such low-level health risks? Arguments in favour are presumably based on the assumption that someone other than society overall will be spending resources to control the risk.

Question 18i asked for opinions on the statement "Health advisories are a useful way to limit human exposure to toxic chemicals". Forty of 50 respondents agreed or strongly agreed, 7 disagreed or strongly disagreed and three people had no opinion. The respondents who had no opinion resided in Swan Hills for more than 10 years. This answer is reflective of responses in Question 29 where 78% of respondents felt the advisories were necessary, presumably to limit human exposure to the chemicals targeted in the advisories.

Contrary to the views espoused by Rachel Carson in her influential book "Silent Spring" (1962), Swan Hills respondents generally disagreed (28/50) with Question 18j which asked for opinions on the statement "Natural chemicals, as a rule, are not as harmful as manmade chemicals". In other studies the percentage of **agreement** is as high as 56% (Krewski et al., 1995). The Swan Hills responses were more typical of assessments made by toxicologists (Neil et al., 1994).

Question 18k asked for opinions on the statement "There is no safe level of exposure to a cancer causing chemical". Twenty-nine of 50 respondents agreed or strongly agreed with this statement; 16 disagreed or strongly disagreed and two people had no opinion. As with Question 18e, this question assessed respondents understanding of exposure and threshold concepts. Responses are consistent across questions in that an accurate understanding of dose-response is not evident[33].

[33] Assuming one employs a threshold model of carcinogenesis.

Question 18l asked for opinions on the statement "A small amount of risk is OK if it brings economic benefit". Nine respondents agreed or strongly agreed (18%); 37 disagreed or strongly disagreed and three people had no opinion. Previous studies have found 29% of Canadians overall agreed with this statement (Krewski et al., 1995).

Question 18m asked for opinions on the statement "Local businesses are usually very careful with dangerous chemicals". Thirty-five respondents agreed or strongly agreed; 12 disagreed or strongly disagreed and two people had no opinion. This result is contrary to the EPA study (1990) which found 53% of respondents disagreed with this statement. The responses appear to be highly reflective of respondents' experience with toxic chemicals and confidence in the ASWTC.

Question 18n asked for opinions on the statement "I feel I am involved in environmental decisions that may affect my health". Twenty-eight respondents (56%) agreed or strongly agreed, 21 disagreed or strongly disagreed (42%) and one person (2%) had no opinion. As in other studies, the opinions on this question were relatively evenly split.

Question 18o asked for opinions on the statement "Scientists and experts are likely to find ways to significantly reduce the threats from environmental contamination". Forty-three respondents agreed or strongly agreed; four disagreed and three people had no opinion. According to O'Connor (1998), people expect 'experts' to have the knowledge and ability to reduce risks to health. This opinion was also seen in comments about knowledge level. Respondents said they expected the doctor and other health care professionals to be familiar with regional environmental threats and to be aware of potential health risks. This expectation appears to have been based solely on their role in the community as providers of accurate information about health and health risk in any form.

Figure 5.14: Question 18 - parts a,b,c,d

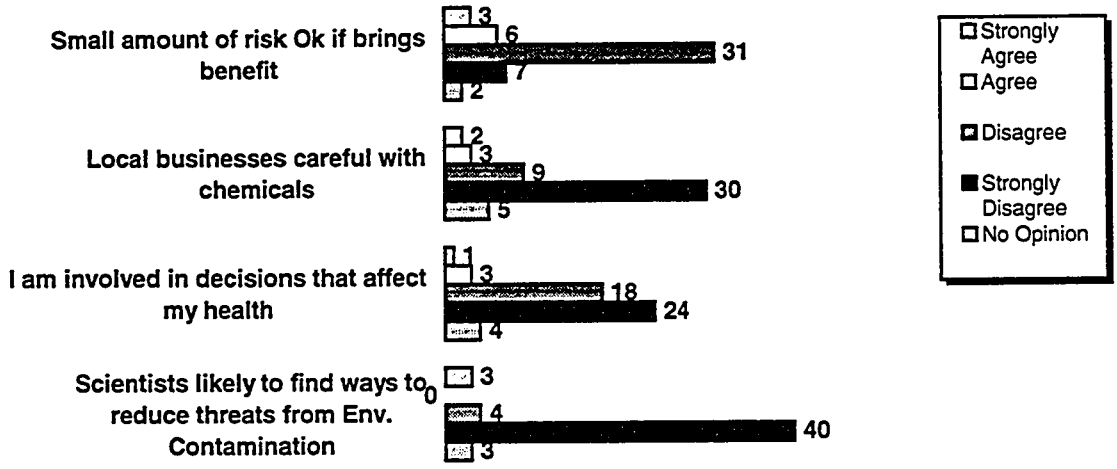


Figure 5.15: Question 18 - parts f, m,n,o

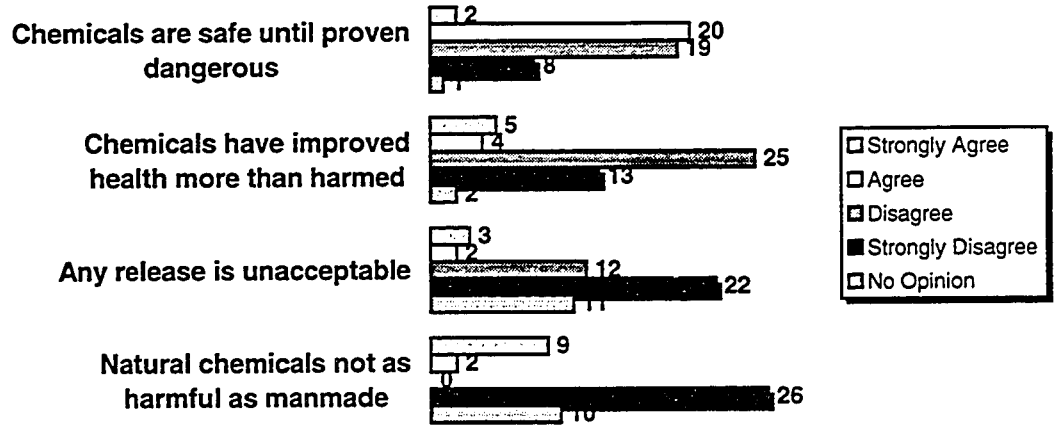
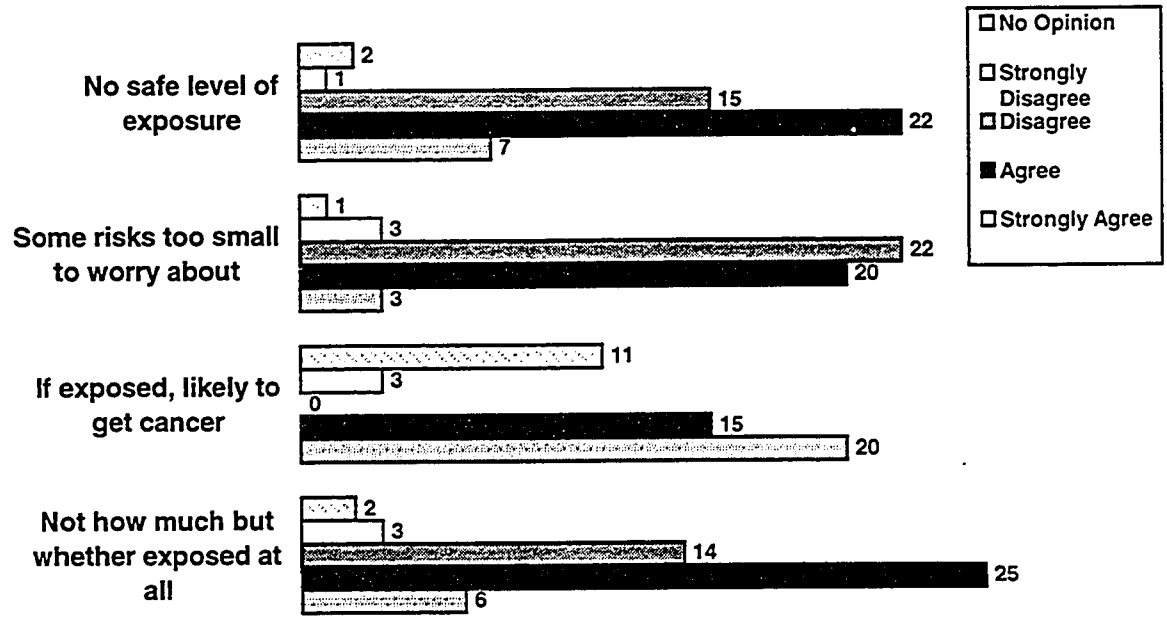


Figure 5.16: Question 18 - parts i,l,g

Figure 5.17: Question 18 - parts e,h,j,k



Questions 19-21 asked respondents to estimate the probability that they have been exposed to toxic chemicals in the past five years, whether that exposure has harmed or will harm their health and whether the probability that living near the ASWTC has

harmed or will harm their health. Questions 19 and 20 were also part of the Alberta Population Survey (1999).

With respect to exposure in the past 5 years, 11 out of 50 Swan Hills respondents (22%) believe the probability is 100% that they have been exposed. Two respondents believe they have not been exposed. In between the two extremes, the distribution of frequencies is roughly bimodal with one peak around 10% and the second around 60%. Respondents to the Alberta survey are not as certain of their exposure although of 1182 respondents to this question, 307 believe the probability of exposure to toxic chemicals is 100% (26%). Eighty-seven respondents believe they have not been exposed (7.4% versus 4% in Swan Hills). Compared to the Swan Hills respondents, respondents from the Alberta survey generally rated the probability of exposure to toxic chemicals lower which under the circumstances seems like a logical conclusion.

The reality is that the probability of exposure in all persons is 100%. As illustrated in Box 4.2, zero exposure is not now nor has it ever been possible. The important question is whether exposure is high enough to cause health effects. It is likely that some respondents equate exposure with harm or a harmful level of exposure and answer accordingly.

Belief in the probability that the estimated toxic exposure has harmed or will harm the respondents' health showed almost a reverse distribution of frequencies. Nine respondents felt there was zero probability that their exposure would result in harm to their health; three respondents felt there was a 100% probability that their exposure would harm their health (18%). The distribution between the two extremes was again roughly bimodal with one peak around 10-20% and the second around 50%. Respondents to the Alberta Population survey (1999) more generally believed that their exposure to toxic chemicals had harmed or will harm their health. Of 1086 respondents to this question, 106 believed that their chemical exposure had or will harm their health. That is almost 10% of respondents compared to only 6% of Swan Hills respondents. Forty-eight respondents did not feel their exposure had or would harm their health. This is 4% of

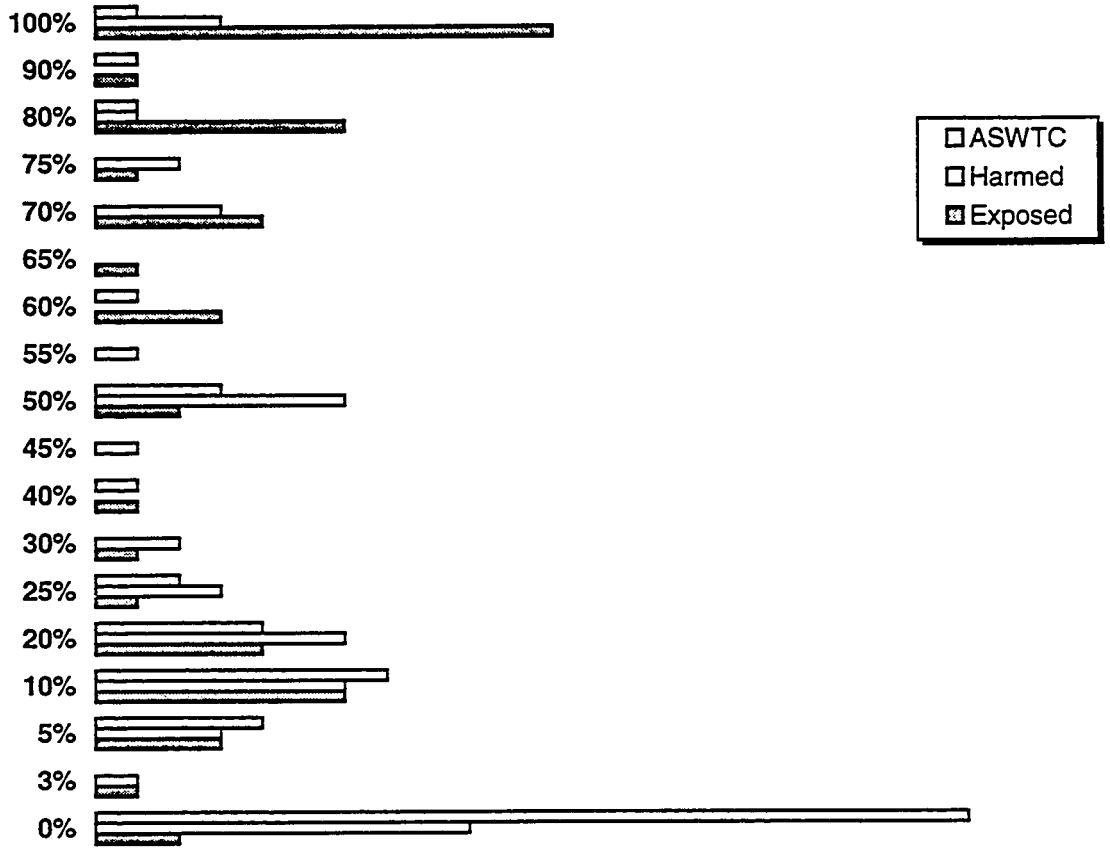
respondents, compared to 18% in Swan Hills. This result may be related to the occupation types in the Swan Hills region. Of the six major employers in the Swan Hills region five are oil field related with associated hazardous chemical use and potential exposure. Respondents to the Alberta Population survey were from a wider variety of occupations.

A survey done in Alberta in 1994 (Jardine et al., 1995) found that "only 15% of Albertans believed they [had] been directly affected by ... environmental risk in the past three years". This value is intermediate to those found in this survey and may simply be a reflection of the amount of variability of responses to this type of question. It does however appear to confirm that few people are concerned about the effect of environmental chemicals on health.

As in many questions in this survey, how words in the question are defined will impact on response. Responses to Question 20 are comparable to opinions expressed in Question 18 and the understanding of dose-response relationships and threshold mechanisms.

When asked whether they believed having the ASWTC proximal to their residence has harmed or will harm their health, a large percentage of respondents felt the probability was zero (21/50). Overall, respondents (93.8%; 45/50) felt the probability of harm due to the ASWTC was $\leq$ 50%. Figure 5.13 illustrates the responses to this series of questions.

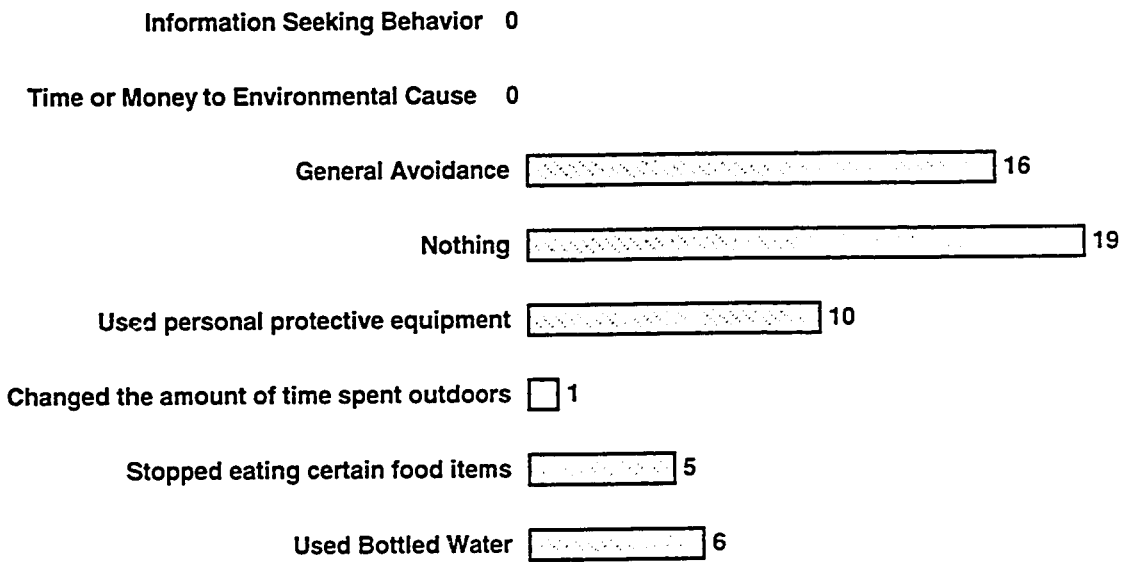
Figure 5.18: Probability of exposure, harm and ASWTC effects



Questions 22, 23 and 24 were designed to elicit information about actual behavior in response to threats to health from environmental chemicals. When asked to describe the actions they have taken in response to potential chemical exposure, the most frequent response was "nothing" (19/50). The second most frequently mentioned behavior included general avoidance of chemicals such as pesticides in the home and following the fish and game advisories for the region (16/50). The third most frequently reported behavior was confined to male respondents. They reported the use of personal protective equipment to avoid chemical exposure (10/50). More women than men reported doing nothing to protect themselves from chemical risks in the environment (12/23 versus 7/27).

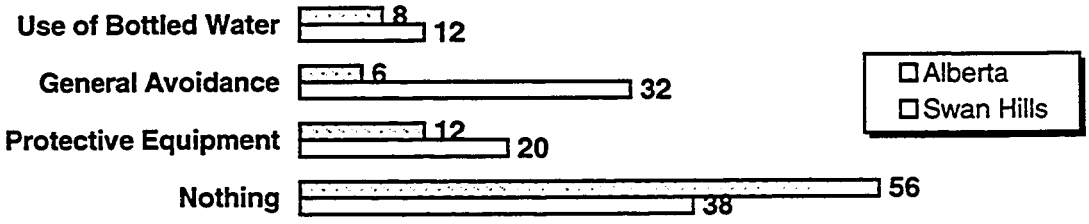
Contrary to previous studies, contributing time or money to an environmental cause was not mentioned in this survey nor was information seeking behaviour.

Figure 5.19: Personal Behavior in Response to Potential Health Threats from Environmental Chemicals (Swan Hills Sample)



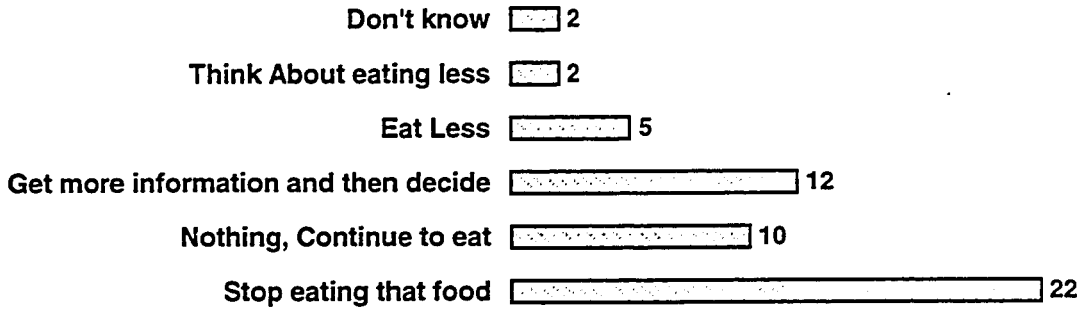
This question was also included on the Alberta Population Survey (1999). As with the Swan Hills survey, the most frequently mentioned activity was nothing (677/1203; 56.2%). Other frequently mentioned behaviors included the use of protective equipment (143/1203), general avoidance of chemicals (73/1203) and the use of bottled water (94/1203). Responses to this question were much more varied than in the Swan Hills survey. Responses include attendance at community meeting and contributing money or time to an environmental cause to quitting smoking and avoiding exposure to tobacco smoke and vehicle exhaust, filtering or boiling drinking water and changing lifestyle activities by using more environmentally friendly chemicals, consuming more organically grown produce and 'natural' dietary supplements, and washing all fruits and vegetables.

Figure 5.20: Comparison of Personal Behaviors between the Swan Hills and the general Alberta surveys (% of respondents)



When asked what they personally would do if they knew PCBs were in a food they ate regularly (Question 23), the most frequent response was that respondents would stop eating that food (22/50; 44%). This is consistent with responses seen in Question 18 equating exposure with harm. The second most common response was that the respondent would get more information such as blood tests or regulatory data and then decide what to do (12/50; 24%). The third most common response was that respondents would do nothing and continue to eat that food (10/50; 20%). Responses to this question are illustrated in figure 5.17.

Figure 5.21: What would you do if PCBs were in your food?



This question was also included in the Alberta Population Survey (1999). As in the Swan Hills survey, the most frequent response was 'stop eating that food' (797/1198; 66%). This is a greater percentage of respondents than in the Swan Hills survey where 44% of respondents felt they would stop eating that food item. Other responses followed a similar

pattern to the Swan Hills survey although the variety of responses was greater. The second most commonly mentioned action was 'do nothing, continue to eat that food item' (57/1198; 4.8%), followed by 'stop eating that food item and encourage others to do the same' and 'get more information and then decide' (38/1198; 3.2%), 'don't know' (37/1198; 3.1%), contact the government or a government agency (30/1198; 2.5%), or contact a doctor (29/1198; 2.4%). Other responses mentioned in the Alberta survey included think about eating less, contact the manufacturer or store where the item was purchased, find an alternate food source, stop eating that food and contact a doctor, lawyer or government agency, and initiate legal or media action. Four respondents felt they wouldn't do anything outside of worry, get sick or get angry.

Question 24 attempted to elicit understanding of a theory in environmental science called *hormesis*. This theory speculates that an agent may be biologically active (i.e. stimulatory) below the toxicity threshold, yielding the so-called U-shaped dose-response curve (versus the more traditional sigmoidal shape) (Figures 5.21 and 5.22). When respondents were told that "Some chemicals, like vitamins or the alcohol in red wine, may prevent some health problems if taken in small amounts. On the other hand, in large amounts, they may cause serious health problems. If you were told that some environmental chemicals, in small amounts, could prevent serious health problems, how would that change your responses to the previous question?", the distribution of behaviors changed in comparison to PCBs in food. The majority of respondents (32/50) reported they would do nothing and continue to eat that food; 8 people reported they would stop eating that food, 5 people said they would get more information, 3 said they eat less, 1 person didn't know what they would do and 1 person said they would think about eating less. Other responses included four people who would get more information and then decide what to do and one person who said he/she would eat more of that food. Of those who felt they would stop eating that food item (8 respondents), seven were male.

Figure 5.22: Traditional Dose-Response Curve

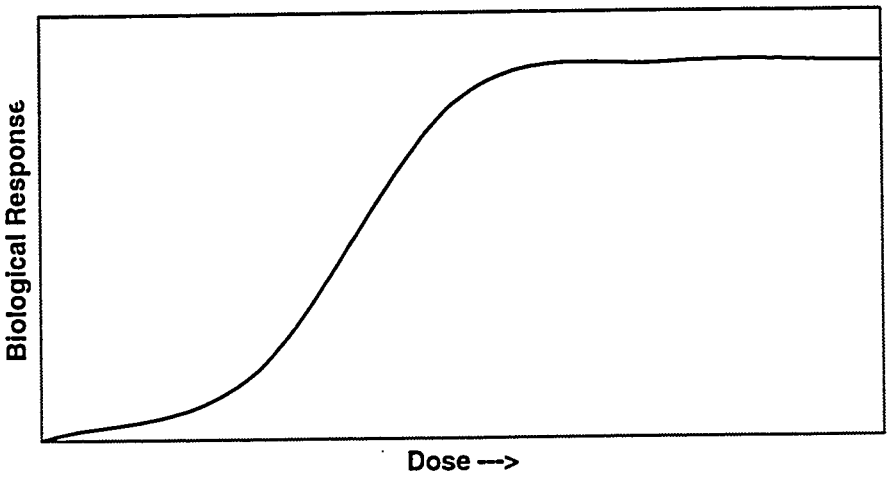


Figure 5.23: U-shaped Dose-Response Curve

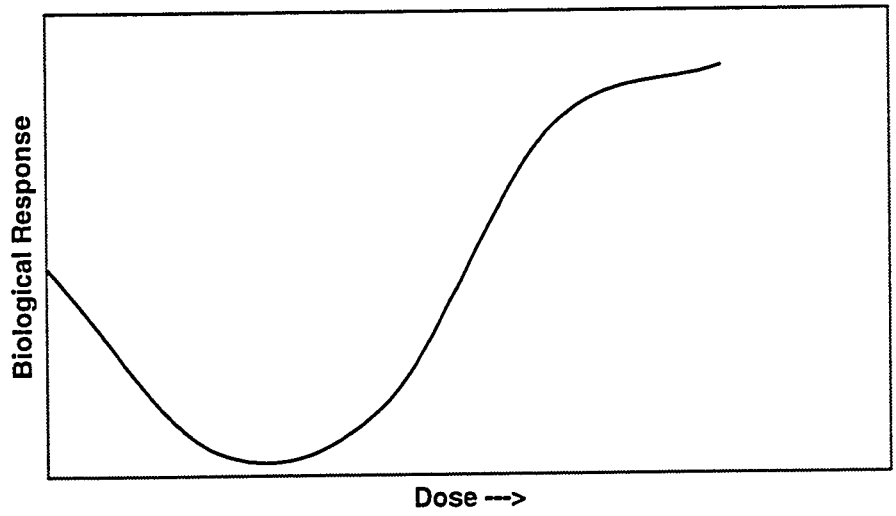
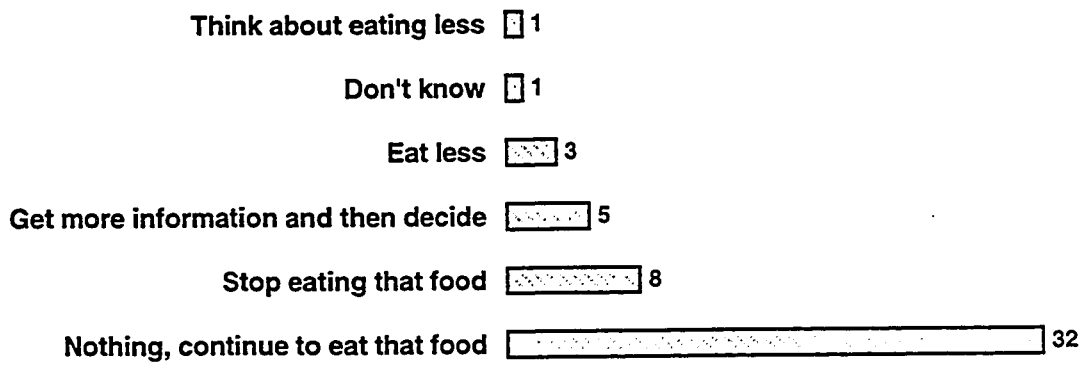


Figure 5.24: If environmental chemicals were in small amounts in a food you ate regularly and could prevent serious health problems, what would you do?



Question 25 asked respondents for their knowledge on another emerging environmental theory that postulates that some chemicals may act like endogenous hormones in the human body. Most respondents (35/50) knew nothing of this theory; 15 respondents had heard of it. One woman was aware of the endocrine disruption aspect of the theory and 2 other people were aware of the potential for "risk in the long run" and that "nothing has been proven yet".

The responses to Questions 24 and 25 are evidence of the difficulty involved in communicating some types of environmental information to the general public. The endocrine disruptor theory for example, has been fairly prominent in the media (c.f. The Financial Post, Oct. 11, 1999) but linking such news stories to personal exposure and health effects has not occurred.

Questions 26-34 asked respondents for their knowledge and opinions about the health and food consumption advisories in effect for the Swan Hills area. Questions 30, 32, 33,and 34 on the survey were not used in this study but were included for a PhD project by another student.

Question 26 asked respondents what they knew about the advisories in effect for the Swan Hills region. Many of the responses were specific information about the species

and chemicals targeted in the advisories and the location and extent of the advisories. Of those who were not aware of the advisories and knew nothing about them, an equal number were male and female (6/27:7/23). Males generally provided more specific information about the advisories than females. Of those respondents who had lived in Swan Hills for more than 10 years, 10/32 knew nothing of the advisories.

Question 27 asked respondents where they had learned about the advisories. The most frequent response was the media (16/50) with an equal number of men and women reporting this source. This supports respondent responses in Question 12 in which the media was mentioned most often as a source of environmental information. The next most frequently reported source of information about the advisory was ASWTC officials and Fish and Game officers (8/50), signs posted at the lakes (7/50), local government (6/50), friends and relatives (5/50), town meetings and provincial government (2/50) and the doctor (1/50). Figure 5.22 illustrates responses to this question.

Females reported finding out about the advisory most often from the media (8), friends and relatives (4), ASWTC officials (3), local government (2) and the doctor (1). Men reported finding out about the advisory most often from the media (8), ASWTC officials (5), local government officials (4), provincial government officials (2) and friends and relatives (1).

Figure 5.25: Advisory knowledge

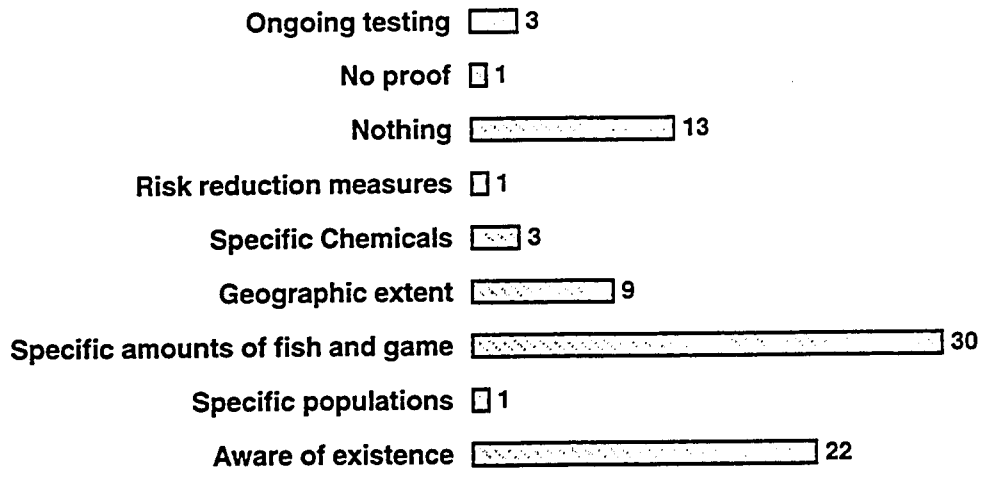
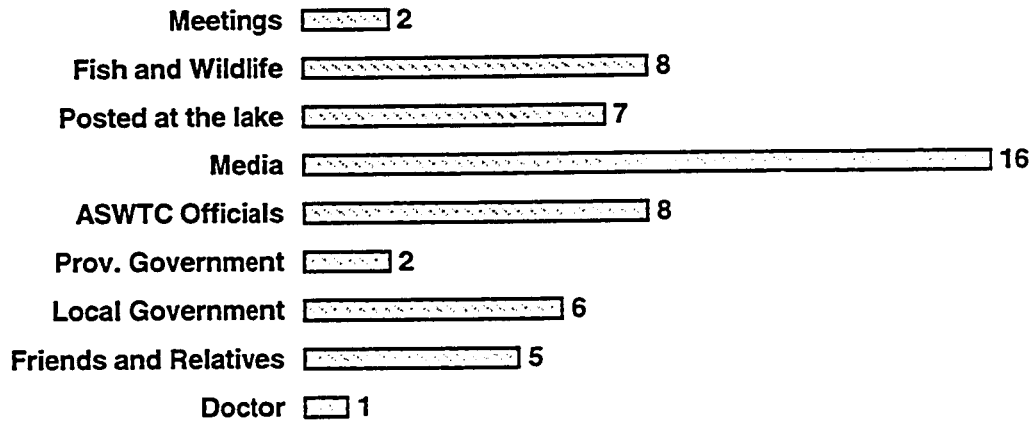


Figure 5.26: Where did you learn of the advisories



Asked whether they follow the advisories or not, 52% (26/50) said yes, 46% (23/50) said no and one person didn't know. Approximately equal numbers of men and women reported following the advisories (14/27 men versus 12/23 women). By duration of residence, 17/32 of those respondents with residence of greater than 10 years follow the advisories; 5/7 with 5-10 years in Swan Hills. The reasons for following the advisories included wanting to protect self and health (13), trusting that the people who instituted the advisories know what is best for the people (4), not eating fish or wild game anyway so following the advisory is not a change in behavior (4), and believing the fish are dangerous due to mercury levels (1). Other reasons included a desire to keep informed about current conditions in the area (3), because they are hunters (1) and depending on what the advisory was (2).

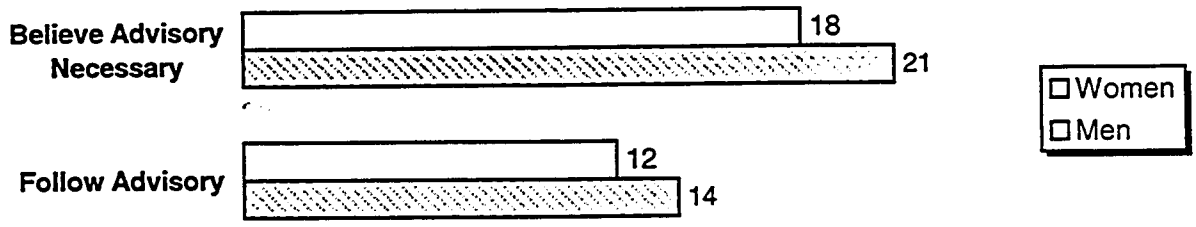
Reasons for not following the advisories included the belief that the advisories are not necessary, right or supported by scientific study (9), have been eating the meat/fish in the region for years without problems (4), didn't know about the advisories (4), don't know why they don't follow the advisories (3), don't eat fish or wild game (1), are not part of the high risk groups named in the advisories (1), and don't trust the information source (i.e. the media) (1).

Asked whether they thought the advisories were necessary, 78% (39/50) said yes, 18% (9) said no and 4% (2 people) didn't know. The results from this question indicate that some people who do not follow the advisories (23/50) may in fact believe they are necessary. Twenty- one out of 26 men thought the advisories were necessary and 18 out of 22 women thought they were necessary. The main reason for why people thought the advisories were necessary was to keep people informed about environmental health risks in their community (17/50). Other reasons for the necessity of the advisories include a need to protect public health and safety (8), to provide information to allow people to make informed choices (4), to avoid lawsuits (1), as a precautionary measure (2), a purely political decision given the source of the contamination (1), to keep people calm (1) and one person felt an advisory was required for reduced fish consumption because the lake in question is down-wind of the plant and the fish are a stationary population, continually exposed to plant emissions.

The reasons for believing that advisories are not necessary include the belief that the scientific and technical information about contaminant levels does not support the advisories (3), lack of trust of the information source (i.e. the media) (2), do not believe there is a problem (2), personal knowledge of person exposed in 1996 incident who has not had any health problems (1), and the belief that there are too many advisories issued and therefore they just ignore them all (1).

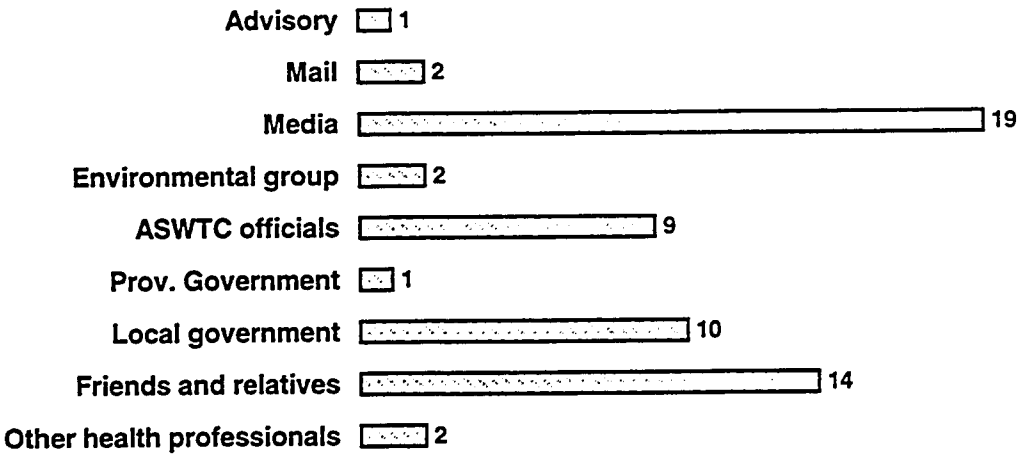
For Swan Hills residents of 2-3 years, 3/7 respondents follow the advisories but 6/7 felt they were necessary. For respondents with 4-5 years in Swan Hills, one person follows the advisories but all 3 felt they were necessary. For respondents with 5-10 years in Swan Hills, 5/7 respondents follow the advisories and 5/7 felt they were necessary. For respondents with greater than 10 years residence in Swan Hills, 17/32 respondents follow the advisories and 25/31 feel they are necessary.

Figure 5.27 Health Advisory Beliefs



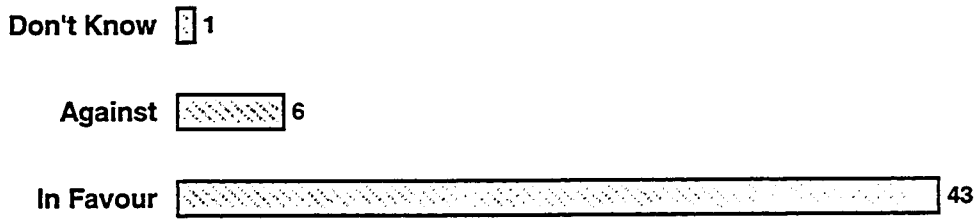
Question 31 asked respondents where they believed they would first find out about risks to human health from a chemical in the community. The majority of respondents stated that the media would be first (19/50) to alert them. Friends and relatives were the second most commonly cited source (14/50) followed by local government officials (10/50) and ASWTC officials (9/50). The results to this question are illustrated in Figure 5.24. The pattern of information sources was similar for males and females except that two males reported they would find out about the advisories from the provincial government or an environmental group whereas two women reported they would hear from a health professional (other than the doctor). These responses support responses from Questions 12 which pointed to the media as a prime source of environmental risk information followed by friends, local government and ASWTC officials and employees.

Figure 5.28: First source of information for a chemical risk



The last question asked respondents how they would vote (i.e. in favour or against), if they could vote today about hosting the ASWTC in their community. Forty-three of fifty respondents would vote in favor (86%), 6 against and 1 person was uncertain. In 1982, during the siting process, the community voted 79% in favor of hosting the plant (Sherbaniuk, 1998). By gender, 24/27 men would vote in favour (89%) and 19/22 women would vote in favor (86%). By duration of residence, of those persons who would vote against hosting the plant, one had resided in Swan Hills for 2-3 years, one for 4-5 years and 4 for greater than 10 years. The plant has retained and expanded its welcome-neighbor status despite a highly publicized incident in 1996, recent layoffs and existing health and consumption advisories.

Figure 5.29: How would you vote today about hosting the ASWTC?



Chapter 6: Conclusion and Directions for Future Research

6.1 Introduction

This thesis is a compilation of the most recent scientific information concerning human health risks associated with PCB exposure through consumption of contaminated food items. It is combined with a case study of a community that volunteered to host a hazardous waste treatment facility that subsequently experienced a release of incineration gases, including a quantity of PCBs. In lieu of a traditional risk perception analysis dividing risk perception along qualitative and quantitative or cultural or psychometric lines, this thesis chose to view risk perception as a means of understanding the world in general and scientific evidence in particular. The basic research question posed at the start of the study was, "Is there knowledge or insight available in the scientific literature that will be useful to residents of the community to address any concerns they may have about health risks associated with dietary PCB exposure?"

The main research objectives of this thesis were:

a) To examine the existing literature on human health effects and PCBs and to determine what health effects, if any, are supported by the literature,
b) To provide a broad perspective on the understanding and communication of human health risks associated with exposure to PCBs through an environmentally mediated dietary pathway, and
c) To explore the understanding of risk and human exposure to PCBs in a community sensitized to PCB exposure.

6.2 Main findings

The main findings of this thesis are summarized below.

1. PCBs are part of a broad class of chemicals known collectively as organochlorines. They are no longer produced commercially but persist in the environment due to their unique chemistry.
2. PCB-related health effects that can be attributed solely to PCB exposure are very limited.

- Chloracne is perhaps the only confirmed health effect of PCB exposure. It has been seen at high exposure levels but has not been associated with exposure at typical environmental levels.
- PCBs are not infectious. You cannot 'catch' PCB-related disease from someone.
- There is no evidence of PCB-associated genetic mutation.
- PCBs, like any substance, cannot cause an adverse effect unless you are significantly exposed to them.

3. Information on developmental or endocrine-related effects due to PCB exposure has a growing body of research but conclusive evidence for either has not been established.
4. There is inconclusive evidence of increased rates of cancer due to PCB exposure. Any conceivable change in cancer rates would be absolutely undetectable in the Swan Hills population of 2000.
5. The ASWTC has retained its welcome-neighbor status in the community of Swan Hills with 86% of respondents willing to vote in favor of hosting the facility (given retrospective choice).
    - Community residents are very positive about the quality of life in the town and compare the town very favorably in comparison to other communities.
    - Community residents do not feel chemical exposure is a problem in the community and are not overall, bothered by the presence of the ASWTC in their community.
    - Residents are well informed of the activities at the ASWTC and environmental issues in general and recognize the efforts of ASWTC officials and employees in keeping them informed.
6. The media is the most common source of information while health care professionals (including physicians) are the least. This distribution occurs in spite of the fact that the media is assessed as having the least knowledge and being the least trustworthy. Health care professionals (including physicians) are seen as very trust-worthy and possessing a lot of knowledge about

environmental health risks. Health care professionals are expected by community residents to have specific information about relevant environmental issues simply as a function of their role in the community. This is not an unreasonable expectation. It requires personal initiative on the part of health care providers to become familiar with the specific environmental health concerns of the community they serve. The scope of topics presented during formal education may not provide complete and up-to-date information and as in any field of study, health care providers must keep informed of changes in pertinent scientific information.

7. Changes in trust in information sources were generally in the positive direction and directed by personal efforts to become more informed or involved.
8. With respect to health and consumption advisories:
    - Swan Hills respondents are generally well informed of the control, extent and duration of health and consumption advisories in the Swan Hills area.
    - Most respondents received their advisory information from the media, ASWTC officials or Fish and Game officers.
    - Only half of respondents follow the advisories although almost 80% feel they are necessary.
9. Information on emerging theories and concepts in environmental chemical risk has not reached this population except in the broadest forms.
10. There is a mismatch between what respondents to the Swan Hills survey believe and what can be supported through the scientific literature.
    - A general lack of understanding of dose-response relationships and basic toxicology in Swan Hills respondents was found. Responses were mirrored in similar questions on a general Alberta survey. If cancer was specifically mentioned as the health effect in question, respondents were less certain of their responses perhaps indicating a growing intuitive understanding of dose-response relationships in response to this one health effect. Respondents generally expressed a

belief in the possibility of zero exposure and most respondents do not believe they have been exposed to hazardous chemicals. For respondents to make informed decisions about their health and the potential effects of exposure to environmental chemicals, residents need accurate information on both of these issues.

- Respondents expressed belief in and desirability of zero exposure levels and have faith in their personal ability to control or reduce chemical exposure. Still, many respondents reported having done "nothing" in response to potential chemical threats to health as was seen in responses to questions 22 (19/50; 38%).

6.3 Directions for future research

The opportunities for research on this topic and with this population are enormous. Five main areas have been identified by this thesis.

1. PCBs are extremely persistent, low level environmental contaminants. As such, potential for human exposure exists, specifically in those populations that consume wild game and fish. A number of theories have been proposed associating human PCB levels with subtle human health effects. Technically difficult, research in this area may not be able to provide definitive answers despite continuing interest.

2. The media was identified as a main source of environmental risk information. A comprehensive analysis of information presented to this population by this route has not been done and could assist other jurisdictions in siting initiatives and those wishing to further study the effect of critical incidents on facility support in host communities.

3. Doctors and other health care professionals hold positions of trust with the expectation of large and accurate knowledge level. They did not however take a primary role in the actual provision of information to study respondents. An analysis of the role of health care professionals (including individuals and government agencies) in terms of their perceived role and actual environmental health risk knowledge would be very informative.

4. Further investigation of community understanding of dose-response relationships is required. Much misunderstanding exists and a need for better understanding in dose-response relationships, cancer causation and exposure levels is required. Efforts in addressing these issues could feasibly be linked to studies suggested above.

6.4 Final thoughts

Overall, health risks from PCBs cannot be predicted with any confidence because the causal evidence for specific adverse outcomes is not clear so the evidence does not support an expectation of measurable health effects occurring in this population. Lack of understanding in this population of basic toxicological concepts is surprising given the duration of the situation with the plant, the extent of media exposure and the claims to knowledge made by survey respondents. Similar misunderstandings have, however, been seen in numerous studies in a variety of populations including university trained toxicologists. This does not negate the need in this population for accurate information about exposure and the potential for health effects related specifically to the situation in Swan Hills and the level of hazardous chemical contamination in the region. Such information could go far in alleviating continuing concerns about hazardous chemical exposure in the community and encourage residents to make better-informed choices about their health and the health of their families in relation to potential environmental health concerns.

Bibliography

Adami, H-O., Lipworth, L, Titus-Ernstoff, L., Hsieh, C-c., Hanberg, A., Ahlborg, U., Baron, J., Trichopoulos, D. (1995). Organochlorine compounds and estrogen-related cancers in women. Cancer Causes and Control, 6, 551-566.

Addison, R.F. (1986). PCBs in perspective. Canadian Chemical News, Feb., 15-17.

Ahlborg, U.G., Becking, G.C., Birnbaum, L.S., Brower, A., Derks, H.J.G.M., Feeley, M., Golor, C., Hanberg, A., Larsen, J.C., Liem, A.K.D., Safe, S.H., Schaltter, C., Waern, F., Younes, M., Yrankeikki, E., (1994). Toxic equivalency factors for dioxin-like PCBs. Chemosphere, 28(6), 1049-1067.

Akagi, K. & Okumura, M. (1985). Association of blood pressure and PCB level in Yusho patients. Environmental Health Perspectives, 59, 37-9.

Alberta Health. (1997). Swan Hills special waste treatment centre human health impact assessment. Author: Edmonton, Alberta.

Anonymous. (1997). Position paper of the American council on science and health: Public health concerns about environmental polychlorinated biphenyls (PCBs). Ecotoxicology and Environmental Safety, 38, 71-84.

Ashby, J., Houthoff, E., Kennedy, S.J., Bars, R., Jekat, F.W., Campbell, P., Van Miller, J., Capanini, F.M., Randall, G.L.P. (1997). The challenge posed by endocrine-disrupting chemicals. Environmental Health Perspectives, 105, 164-169.

Atkinson, R. (1987). Estimation of OH radical reaction rate constants and atmospheric lifetimes for polychlorobiphenyls, dibenzo-p-dioxins and dibenzofurans. Environmental Science and Technology, 21(3), 305-7.

ATSDR (1993). Proceedings of the expert panel workshop to evaluate the public health implication for the treatment and disposal of polychlorinated biphenyls-contaminated waste: Chapter 2 - expert panel report, health effects panel.

Ayotte, P., Carrier, G., Dewailly, E. (1996). Health risk assessment for Inuit newborns exposed to dioxin-like compounds through breast feeding. Chemosphere, 32(3), 531-542.

Ayotte, P., Dewailly, E., Ryan, JJ., Bruneau, S., Lebel, G. (1997). PCBs and dioxin-like compounds in plasma of adult Inuit living in Nunavik (Arctic Quebec). Chemosphere, 34(5-7), 1459-68.

Baht, R.V. & Moy, G.C. (1997). Monitoring and assessment of dietary exposure to chemical contaminants. World Health Statistics Quarterly, 50(1-2), 132-49.

Beaglehole, R. Vonita, R., Kjellstrong, T. (1993). Basic Epidemiology. World Health Organization: Geneva.

Beck, U. (1992). Risk Society: Towards a New Modernity. SAGE: London.

Bernard, A., Hermans, C., Broeckaert, F., De Poorter, G., De Cock, A., Houins, G. (1999). Food contamination by PCBs and dioxins. Nature, 410, 231-232.

Bertazzi, P.A., Riboldi, L., Pesatori, A., Radice, L., Zocchetti, C. (1987). Cancer mortality of capacitor manufacturing workers. American Journal of Industrial Medicine, 11, 165-176.

Bertazzi, PA., Bernucci, I., Brambilla, G., Consonni, D., Pesatori, AC. (1998). The Seveso studies on early and long-term effects of dioxin exposure: A review. Environmental Health Perspectives, 106(Supplement 2), 625-633.

Brown, D.P. & Jones, M. (1981). Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. Archives of Environmental Health, 36(3), 120-129.

Brucker-Davis, F. (1998). Effects of environmental synthetic chemicals on thyroid function. Thyroid, 8(9), 827-856.

Brunelle, D.J., Mendiratta, A.K. & Singleton, D.A. (1985). Reaction/removal of polychlorinated biphenyls from transformer oil: Treatment of contaminated oil with poly(ethylene glycol)/KOH. Environmental Science and Technology, 19(8), 740-9.

Buck, G.M. (1996). Epidemiologic perspective of the developmental neurotoxicity of PCBs in humans. Neurotoxicology and Teratology, 18(3), 239-241.

Bunce, N. (1994). Environmental Chemistry (2nd Edition). Wuerz Publishing: Winnipeg, Canada.

Calabrese, E.J., Baldwin, L.A., Holland, C.D. (1999). Hormesis: A highly generalizable and reproducible phenomenon with important implications for risk assessment. Risk Analysis, 19(2), 261-281.

Carlsen, E., Giwercman,A, Keiding,N., Skakkebaek, N.E. (1992). Evidence for decreasing quality of semen during last 50 years. British Journal of Medicine, 305, 609.

Carson, R. (1962). Silent Spring. Fawcett Publications, Inc: Greenwich, Conn.

Chan, H.M. (1998). A database for environmental contaminants in traditional foods in northern and Arctic Canada: Development and applications. Food Additives and Contaminants, 15(2), 127-134.

Chen, Y.C., Guo, Y.L., Hsu, C.C., Rogan, W.J. (1992). Cognitive development of Yu-Cheng ("oil disease") children prenatally exposed to heat-degraded PCBs. Journal of the American Medical Association, 268(22), 3213-8.

Chen, Y.C., Yu, M.L., Rogan, W.J., Gladen, B.C., Hsu, C.C. (1994). A 6-year follow-up of behavior and activity disorders in the Taiwan Yu-Cheng children. American Journal of Public Health, 84(3), 415-21.

Chia, L.G. & Chu, F.L. (1985). A clinical and electrophysiological study of patients with polychlorinated biphenyl poisoning, Journal of Neurology , Neurosurgery and Psychiatry, 48, 894-901.

Clarkson, TW. (1995). Environmental contaminants in the food chain. American Journal of Clinical Nutrition, 61(3 Supplement), 682S-686S.

Cogliano, V.J. (1998). Assessing the cancer risk from environmental PCBs. Environmental Health Perspectives, 106(6), 317-323.

Colborn, T., Dumanoski, D., Myers, J.P. (1997). Our Stolen Future. Penguin Group: New York, NY.

Colborn, T., Davidson, A., Green, S., Hodge, R.A., Jackson, C.I., Liroff, R.A. (1990). Great Lakes great legacy? The Institute for Research on Public Policy: Ottawa, Ontario.

Conacher, H.B.S. & Mes, J. (1993). Assessment of human exposure to chemical contaminants in foods. Food Additives and Contaminants, 10(1), 5-15.

Courval, J.M., DeHoog, J.V., Stein, A.D., Tay, E.M., He, J., Humphrey, H.E.B., Paneth, N. (1999). Sport-caught fish consumption and conception delay in licensed Michigan anglers. Environmental Research, 80(2 Pt. 2), S183-188.

Daly, H., Darvill, T., Lonky, E., Reihman, J., Sargent, D. (1996). Behavioral effects of prenatal and adult exposure to toxic chemicals found in Lake Ontario fish: two methodological approaches. Toxicology & Industrial Health, 12(3-4), 419-26.

Dewailly, E., Nantel, A., Bruneau, S., Laliberté, C., Ferron, L., Gingras, S. (1992). Breast milk contamination by PCDDs, PCDFs and PCBs in Arctic Québec: A preliminary assessment. Chemosphere, 25(7-10), 1245-1249.

Dewailly, E., Ryan, JJ., Lalierte, C., Bruneau, S., Weber, J-P., Gingras, S., Carrier, G. (1994). Exposure of remote maritime populations to coplanar PCBs. Environmental Health Perspectives, 102(Supplement 1), 205-209.

Dewailly, E., Ayotte, P., Blanchet, C., Grondin, J., Bruneau, S., Holub, B., Carrier, G. (1996). Weighing contaminant risks and nutrient benefits of country food in Nunavik. Arctic Medical Research, 55(Supplement 1), 13-19.

Emmett, E.A., Maroni, M., Jefferys, J., Schmith, J., Levin, B.K., Alvares, A. (1988). Studies of transformer repair workers exposed to PCBs: II - Results of clincial laboratory investigations. American Journal of Industrial Medicine, 14, 47-62.

Eskenazi, B. & Castorina, R. (2000). Association of prenatal maternal or postnatal child environmental tobacco smoke exposure and neurodevelopmental and behavioral problems in children. Environmental Health Perspectives, 107(12), 991-1000.

Erickson, MD. (1997). Analytical chemistry of PCBs(2nd Ed.). New York: Lewis Publishers.

Falk, C., Hanrahan, L., Anderson, H.A., Kanarek, M.S., Draheim, L., Needham, L., Patterson, D., and the Great Lakes Constortium. (1999). Body burden levels of dioxin, furans, and PCBs among frequent consumers of Great Lakes Sport Fish. Environmental Research, 80, S19-S25.

Feeley, M.M., Jordan, S.A., Gilman, A.P. (1998). The Health Canada Great Lakes multigeneration study - summary and regulatory considerations. Regulatory Toxicology and Pharmacology, 27, S90-S98.

Fein, G.G., Jacobson, J.L., Jacobson, S.W., Schwartz, P.M., Dowler, J.K. (1984). Prenatal exposure to polychlorinated biphenyls: Effects on birth size and gestational age. The Journal of Pediatrics, 105, 315-320.

Fisch, H., Feldshuh, J., Goluboff, E.T., Broder, S.J., Olson, J.H., Barad, D.H. (1996). Semen analysis in 1,283 men from the United States over a 25-year period: No decline in semen quality. Fertility and Sterility, 65, 1009.

Fischbein, A. (1985). Liver function tests in workers with occupational exposure to polychlorinated biphenyls (PCBs): Comparison with Yusho and Yucheng. Environmental Health Perspectives, 60, 145-50.

Fitzgerald, E.F., Deres, D.A., Hwang, S-A., Bush, B., Yang, B-Z., Tarbell, A., Jacobs, A. (1999). Local fish consumption and serum PCB concentrations among Mohawk men at Akwesasne. Environmental Research, 80(2 Pt. 2), S97-103.

Fitzgerald, E.F., Brix, K.A., Deres, D.A., Hwang, S-A., Bush, B., Lambert, G., Tarbell, A. (1996). Polychlorinated biphenyl (PCB) and dichlorodiphenyl dichloroethylene (DDE) exposure among native American men from contaminated Great Lakes fish and wildlife. Toxicology and Industrial Health, 12(3/4), 361-368.

Foster, W.G. (1998). Endocrine disruptors and development of the reproductive system in the fetus and children: Is there cause for concern? Canadian Journal of Public Health,89(supplement 1), S37-S41.

Fromme, H., Baldauf, AM, Klautke, O., Piloty, M., Bohrer, L. (1996). [Polychlorinated biphenyls (PCB) in caulking compounds of buildings --- assessment of current status in Berlin and new indoor air sources]. [German]. Gesundheitswesen, 58(12), 666-72.

Giesy, J.P. & Kannan, K. (1998). Dioxin-like and non-dioxin like toxic effects of polychlorinated biphenyls (PCBs): Implications for risk assessment. Critical Reviews in Toxicology, 28(6), 511-569.

Gerstenberger, SL., Tavris, DR., Hansen, LK., Pratt-Shelley, J., Dellinger, JA. (1997). Concentrations of blood and hair mercury and serum PCBs in an Ojibwa population that consumes Great Lakes region fish. Journal of Toxicology - Clinical Toxicology, 35(4), 377-386.

Gladen, B.C., Taylor, J.S., Wu, Y.C., Ragan, N.B., Rogan, W.J., Hsu, C.C. (1990). Dermatological findings in children exposed transplacentally to heat-degraded polychlorinated biphenyls in Taiwan. British Journal of Dermatology, 122(6), 799-808.
Golden, R.J., Noller, K.L., Titus-Ernstoff, L., Kaufman, R.H., Mittendorf, R., Stillman, R., Reese, E.A. (1998). Environmental endocrine modulators and human health: An assessment of the biological evidence. Critical Reviews in Toxicology, 28(2), 109-227.

Goto, M. and Higuchi, K. (1969). The symptomatology of Yusho (chlorobiphenyls poisoning) in dermatology. Fukuoka Acta Med. 60, 409-431.

Greenland, S., Salvan, A., Wegman, D.H., Hallock, M.F., Smith, T.J. (1994). A case-control study of cancer mortality at a transformer-assembly facility. International Archives of Occupational and Environmental Health, 66, 49-54.

Guo, Y.L., Yu, M-L., Hsu, C-C., Rogan, W.J. (1999). Chloracne, goiter, arthritis, and anemia after polychlorinated biphenyl poisoning: 14-year follow-up of the Taiwan Yucheng cohort. Environmental Health Perspectives, 107(9), 715-719.

Gustavsson, P., Hogstedt, C., Rappe, C. (1986). Short-term mortality and cancer incidence in capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). American Journal of Industrial Medicine, 10, 341-344.

Gustavsson, P. & Hogstedt, C. (1997). A cohort study of capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). American Journal of Industrial Medicine, 32, 234-239.

Hanrahan, L.P., Falk, C., Anderson, H.A., Draheim, L., Kanarek, M.S., Olson, J. (1999). Serum PCB and DDE levels of frequent Great Lakes sport fish consumers – a first look. Environmental Research, 80(2 Pt. 2), S26-37.

Harrington, J.M. (1998). Facts, fallacies and fears: The public and the health professionals at odds. Annals of Occupational Hygiene, 42(4), 227-232.

Hauser, P., McMillin, J.M., Bhatara, V.S. (1998). Resistance to thyroid hormone: Implications for neurodevelopmental research on the effects of thyroid hormone disruptors. Toxicology and Industrial Health. 14(1/2), 85-101.

Health Canada (1996).
Hennekens, C. H., Buring J.E. (1987). Epidemiology in Medicine. Little, Brown and Company: Toronto.

Henriksen, G.L., Ketchum, N.S., Michalek, J.E., Swaby, J.A. (1997). Serum dioxin and diabetes mellitus in veterans of Operation Ranch Hand. Epidemiology. 8(3), 252-8.
Higuchi, K. (1976). PCB poisoning and pollution. Academic Press: New York.

Hsieh, S-F., Yen, Y-Y., Hsieh, C-C., Lee, C-H., Ko, Y-C. (1996). A cohort study on mortality and exposure to polychlorinated biphenyls. Archives of Environmental Health. 51(6), 417-424.

Hsu, S-T., Ma, C-I., Hsu, S.K-H., Wu, S-S., Hsu, N.,H-M., Yeh, C-C., Wu, S-B. (1985). Discovery and epidemiology of PCB poisoning in Taiwan: A four-year followup. Environmental Health Perspectives. 59, 5-10.

Hunter, D.J., Hankinson, S.E., Laden, F., Colditz, G.A., Manson, J.E., Willett, W.C., Speizer, F.E., Wolff, M.S. (1997). Plasma organochlorine levels and the risk of breast cancer. The New England Journal of Medicine. 337(18), 1253-1258.

Humphrey, H.E.B., Gardiner, J.C., Pandya, J.R., Sweeney, A.M., Gasior, D.M., McCaffrey, R.J., Schantz, S.L. (2000). PCB congener profile in the serum of humans consuming Great Lakes fish. Environmental Health Perspectives. 108(2), 167-172.

Huisman, M., Koopman-Esseboom, C., Fidler, V., Hadders-Algra, M., van der Paauw, C.G., Tuinstra, L.G. (1995). Perinatal exposure to polychlorinated biphenyls and dioxins and its effect on neonatal neurological development. Early Human Development. 41, 111-27.

Ikeda, M. (1996). Comparison of clinical picture between yusho/yucheng cases and occupational pcb poisoning cases. Chemosphere. 32(3), 559-566.

Jacobson, J.L., Jacobson, S.W. (1996). Evidence for PCBs as neurodevelopmental toxicants in humans. NeuroToxicology.18(2), 415-424.

Jacobson, J.L., Jacobson, S.W. (1996a). Intellectual impairment in children exposed to polychlorinated biphenyls in utero. New England Journal of Medicine. 335(11), 783-789.

Jacobson, J.L., Jacobson, S.W. (1996b). Dose-response in pernatal exposure to polychlorinated biphenyls (PCBs): The Michigan and North Carolina cohort studies. Toxicology and Industrial Health. 14(1,2), 435-445.

James, R.C., Busch, H., Tamburro, C.H., Roberts, S.M., Schell, J.D., Harbison, R.D. (1993). Polychlorinated biphenyl exposure and human disease. Occupational and Environmental Medicine, 136-148.

Jardine, C.J., Krahn, H., Hrudey,S.E. (1995). Health Risk Perception in Alberta. Research report 95-1, Eco-Research Chair in Environmental Risk Management.

Jensen, S. (1972). The PCB story. Ambio, 1(14), 123-31.

Johnson, B.L., Hicks, H.E., De Rosa, C.T. (1999). Key environmental human health issues in the Great Lakes and St. Lawrence River basins. Environmental Research,80, S2-S12.

Kannan, K., Tanabe, S., Giesy, J.P., Tatsukawa, R. (1997). Organochlorine pesticides and polychlorinated biphenyls in foodstuffs from Asian and Oceanic countries. Reviews in Environmental Contamination and Toxicology, 152, 1-55.

Kimbrough, R.D. (1995). Polychlorinated biphenyls (PCBs) and human health: An update. Critical Reviews in Toxicology, 25(2), 133-163.

Kimbrough, R.D., Doemland, M.L., LeVois, M.E. (1999). Mortality in male and female capacitor workers exposed to polychlorinated biphenyls. Journal of Occupational and Environmental Medicine, 41(3), 161-171.

Pitot, H.C. (III) & Dragan, Y.P. (1996). Chemical carcinogenesis. C.D. Klaassen, (Ed.). In Casarett and Doull's Toxicology: The Basic Science of Poisons (5^{th} Edition) (pp.201-267). New York, N.Y.: McGraw-Hill Health Professions Division.

Kostyniak, P.J., Stinson, C., Greizerstein, H.B., Vena, J., Buck, G., Mendola, P. (1999). Relation of Lake Ontario fish consumption, lifetime lactation and parity to breast milk PCB and pesticide concentration. Environmental Researach, 80(2 Pt. 2), S106-174.

Kraus, N., Malmfors, T., Slovic, P. (1992). Intuitive toxicology: Expert and lay judgments of chemical risks. Risk Analysis, 12(2), 215-232.

Kreiss, K. (1985). Studies on populations exposed to polychlorinated biphenyls. Environmental Health Perspectives, 60, 193-199.

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

Krokos, F., Creaser, CS., Wright, C., Startin, JR. (1996). Levels of selected ortho and non ortho polychlorinated biphenyls in UK retail milk. Chemosphere, 32(4), 667-673.

Kinloch, D. & Kuhlein, H. (1988). Assessment of PCBs in Arctic foods and diets. Arctic Medical Research, 47, (Supplement 1), 159-162.

Kuhn, R.G. & Ballard, K. R. (1998). Canadian innovations in siting hazardous waste management facilities. Environmental Management, 22(4), 533-545.

Kuhlein, H.V. & Kinloch, D. (1988). PCBs and nutrients in Baffin Island Inuit foods and diets. Arctic Medical Research, 47(Supplement 1), 155-158.

Laden, F., Neas, L.M., Spiegelman, D., Hankinson, S.E., Willett, W.C., Ireland, K., Wolff, M.S., Hunter, D.J. (1999). Predictors of plasma concentrations of DDE and PCBs in a group of U.S. women. Environmental Health Perspectives, 107(1), 75-81.

Landi, M.T., Consonni, D., Patterson, D.G. Jr., Needham, L.L., Lucier, G., Branbilla, P., Cazzaniga, A., Mocarelli, P., Pesatori, A.C., Bertazzi, P.A., Caporaso, N.E. (1998). 2.3.7.8-tetrachlorodibenzo-p-dioxin plasma levels in Seveso 20 years after the accident. Environmental Health Perspectives, 106(55), 273-277.

Langlois, C. & Langis, R. (1995). Presence of airborne contaminants in the wildlife of Northern Quebec. Science of the Total Environment, 160-61, 391-402.

Leoni, V., Fabiani, L., Marinelli, G., Puccetti, G., (1989). PCB and other organochlorine compounds in blood in women with or without miscarriage: A hypothesis of correlation. Ecotoxicology and Environmental Safety, 17, 1.

Lidskog, R. (1996). In science we trust? On the relation between scientific knowledge, risk consciousness and public trust. Acta Sociologica, 39, 31-56.

Loganathan, B.G., Irvine, K.N., Kannan, K., Pragatheeswaran, V., Sajwan, K.S. (1997). Distribution of selected PCB congeners in the Babcock Street Sewer District: A multimedia approach to identify PCB sources in combined sewer overflows (CSOs) discharging to the Buffalo River, New York. Archives of Environmental Contamination and Toxicology, 33, 130-140.

Loomis, D., Browning, S.R., Schenck, A.P., Gregory, E., Savitz, D.A. (1997). Cancer mortality among electric utility workers exposed to polychlorinated biphenyls. Occupational and Environmental Medicine, 54, 720-728.

Lovett, A.A., Foxall, C.D., Chewe, D. (1997). PCB and PCDD/DF congeners In locally grown fruit and vegetable samples in Wales and England. Chemosphere, 34(5-7), 1421-1436.

Lu, Y-C. & Wu, Y-C. (1985). Clinical findings and immunological abnormalities in Yu-Cheng patients. Environmental Health Perspectives, 59, 17-29.

MacLeod, KE. (1979). Sources of emissions of polychlorinated biphenyls into the ambient atmosphere and indoor air. USEPA. Washington, DC: EPA 600/4-79-0-022.

MacLeod, J. and Wang, Y. (1979). Male fertility potential in terms of semen quality: a review of the last, a study of the present. Fertility and Sterility, 31, 103.

MacLusky, N.J., Brown, T.J., Schantz, S., Seo, B.W., Peterson, R.E. (1998). Hormonal interactions in the effects of halogenated aromatic hydrocarbons on the developing brain. Toxicology and Industrial Health, 14(1/2), 185-208.

Masuda, Y. (1996). Approach to risk assessment of chlorinated dioxins from Yusho PCB poisoning, Chemosphere, 32(4), 583-594.

Milly, P. & Leiss, W. 1997). Mother's milk: Commjunicating the risks of PCBS in Canada and the far north. In D. Powell & W. Leiss (Eds.). Mad Cows and Mother's Milk. (pp.182-209). Montreal, P.Q.: McGill-Queen's University Press.

Miyata, H., Fukushima, S., Kashimoto, T., Kunita, N. (1985). PCBs, PCQs and PCDFs in tissues of Yusho and Yu-Cheng patients. Environmental Health Perspectives, 59, 67-72.

Muckle, G., Dewailley, E., Ayotte, P. (1998). Prenatal exposure of Canadian children to polychlorinated biphenyls and mercury. Canadian Journal of Public Health, 89(Supplement 1), S20-25)

Murata, K., Weihe, P., Araki, S., Budtz-Jorgensen, E., Grandjean, P. (1999). Evoked potentials in Faroese children prenatally exposed to methylmercury. Neurotoxicology & Teratology, 21(4), 471-2.

Nakanishi, Y., Shigematsu, N., Kurita, Y., Matsuga, K., Kanegae, H., Ishimaru, S., Kawazoe, Y. (1985). Respiratory involvement and immune status in Yusho patients. Environmental Health Perspectives, 59, 31-6.

National Cancer Institute of Canada: Canadian Cancer Statistics 2000, Toronto, Canada, 2000.

Neil, N., Malmfors, T., Slovic, P. (1994). Intuitive toxicology: Expert and lay judgments of chemical risks. Toxicologic Pathology, 22(2), 198- 201.

Nendza, M., Herbst, T., Kussatz, C., Geis, A. (197). Potential for secondary poisoning and biomagnification in marine organisms. Chemosphere, 35(2), 1875-1885.

Newsome, WH., Davies, DJ., Sun, WF.(1998). Residues of polychlorinated biphenyls (PCB) in fatty foods of the Canadian diet. Food Additives & Contaminants, 15(1), 19-29.

O'Connor, R.E., Bord, R.J., Fisher, A. (1998). Rating threat mitigators: Faith in experts, governments, and individuals themselves to create a safer world. Risk Analysis, 18(5), 547- 556.

Okumura, M. (1984). Past and current medical states of Yusho patients. American Journal of Industrial Medicine, 5, 13-18.

Osius, N., Karmaus, W., Kruse, H., Witten, J. (1999). Exposure to polychlorinated biphenyls and levels of thyroid hormone in children. Environmental Health Perspectives, 107(10), 843-49.

Patandin, S., Dagnelie, P.C., Mulder, P.G.H., Op de Coul, E., van de Veen, J.E., Weisglas-Kuperus, N., Sauer, P.J.J. (1999a). Dietary exposure to polychlorinated biphenyls and dioxins from infancy until adulthood: A comparison between breast-feeding, toddler, and long-term exposure. Environmental Health Perspectives, 107, (1), 45-51.

Patandin, S., Lanting, C.I., Mulder, P.G.H., Boersma, R., Sauer, P.J.J., Weisglas-Kuperus, N. (1999b). Effects of environmental exposure to polychlorinated biphenyls and dioxins on cognitive abilities in Dutch children at 42 months of age. The Journal of Pediatrics, 134, 33-41.

Pellerin, J. & Grondin, J. (1998). Assessing the state of Arctic ecosystem health: Briding Inuit viewpoints and biological endpoints on fish health. Ecosystem Health, 4(4), 236-247.

Pesatori, A.C., Zocchetti, C., Guercilena, S., Consonni, D., Turrini, D., Bertazzi, P.A. (1998). Dioxin exposure and non-malignant health effects: A mortality study. Occupational and Environmental Medicine, 55, 126-131.

Phibbs, P. (1996). EPA reassessment finds as much as 20 times less cancer risk from PCBs. Environmental Science and Technology, 30(8), 332A-333A.

Porterfield, S. (1994). Vulnerability of the developing brain to thyroid abnormalities: Environmental insults to the thyroid system. Environmental Health Perspectives, 102(Supplement 2), 125-130.

Porterfield, S. , Hendry, LB. (1998). Impact of PCBs on thyroid hormone directed brain development. Toxicology and Industrial Health, 14(1,2), 103-20.

Ramondetta, M. & Repossi, A. (Eds.)(1998). Seveso 20 years after: From dioxin to the Oak Wood. Fondazione Lombardia per l"Ambiente: Milan.

Rastogi, RC. (1992). Investigation of isomer specific polychlorinated biphenyls in printing inks. Bulletin of Environmental Contamination and Toxicology, 48, 567-71.

Raszyk, J., Gajduskova, V., Ulrich, R., Jarosova, A., Sabatova, V., Salava, J., Palac, J. (1996). [Occurrence of chlorinated pesticides and polychlorinated biphenyls in the stalls and outdoor environment in pig-fattening farms]. [Czech]. Veterinarni Medicina, 41(6), 165-71.

Receveur. O., Boulay, M., Kuhlein, HV. (1997). Decreasing traditional food use affects diet quality for adult Dene/Métis in 16 communities of the Cnadian Northwest Territories. Journal of Nutrition, 127, 2179-2186.

Rhainds, M., Levallois, P., Dewailly, E., Ayotte, P. (1999). Lead, mercury, and organochlorine compound levels in cord blood in Quebec, Canada. Archives of Environmental Health, 54(1), 40-8.

Robards, K. (1996). The determination of polychlorinated biphenyl residues: A review with special reference to foods. Food Additives and Contaminants, 7(2), 143-174.

Rogan, W.J. & Gladen, B.C. (1992). Neurotoxicology of PCBs and related compounds. Neurotoxicology, 13(1), 27-35.

Rogan, W.J., Gladen, B.C., McKinney, J.D., Carreras, N., Hardy, P., Thullen, J., Tingelstad, J., Tully, M. (1986). Neonatal effects of transplacental exposure to PCBs and DDE. Journal of Pediatrics, 109(2), 335-41.

Rogan, W.J., Gladen, B.C., McKinney, J.D., Carreras, N., Hardy, P., Thullen, J., Tingelstad, J., Tully, M. (1986). Polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethene (DDE) in human milk: Effects of maternal factors and previous lactation. American Journal of Public Health, 76(2), 172-7.

Rogan, W.J. (1996). Pollutants in breast milk. Archives of Pediatric and Adolescent Medicine, 150(9), 981-90.

Rothman, N., Cantor, K.P., Blair, A., Bush, D., Brock, J.W., Helzlsouer, K., Zahm, S.H., Needham, L.L., Pearson, G.R., Hoover, R.N., Comstock, G.W., Strickland, P.T. (1997). A nested case-control study of non-Hodgkin lymphoma and serum organochlorine results. The Lancet, 350, 240-44.

Rudiger, HW. (1995). [In a 1970-built high school building multiple increased PCB (polychlorinated biphenyl) readings in concentrations of around 3800 micrograms/pro cubic meter were obtained]. [German]. Internist, 36(9), 931-932.

Ryan, J.J., Dewailly, E. (1997). Dioxin-like compounds in fishing people from the lower north shore of the St. Lawrence River, Quebec, Canada. Archives of Environmental Health, 52(4), 309-17.

Rylander, L. & Hagmar, L. (1995). Mortality and cancer incidence among women with a high consumption of fatty fish contaminated with persistent organochlorine compounds. Scandinavian Journal of Work and Environmental Health, 21, 419-26.

Safe, S.H., Gasiewicz, T., Whitlock, J.P. Jr. (1990). Mechanism of Action . in Polychlorinated Cibenzo-p-dioxins and –furans (PCDDs.PCDFs): Sources and Environmental Impact, Epidemiology, Mechanisms of Action, Health Risks. Ed. S. Safe, O Hutzinger, T.A. Hills : Springer Verlag, Berlin.

Sagan, C. (1995). The Demon-Haunted World: Science as a candle in the dark. Random House: New York.

Sanderson, J. T., Aarts, J.M.M.J.G., Brouwer, A., Froese, K.L., Denison, M.S., Giesy, J.P. (1996). Comparison of Ah receptor-mediated luciferase and ethoxyresorufin-O-deethylase induction in H4IIE cells: Implications for their use as bioanalytical tools for the detection of polyhalogenated aromatic hydrocarbons. Technology and Applied Pharmacology, 137, 316-325.

Schantz, S.L., Gardiner, J.C., Gasior, D.M., Sweeney, A.M., Humphrey, H.E.B., McCaffrey, R.J. (1999), Motor function in aging Great Lakes fisheaters. Environmental Research, 80, S46-S56.

Schecter, A., Cramer, P., Boggess, K., Stanley, J., Olson, J.R. (1997). Levels of dioxins, dibenzofurans, PCB and DDE congeners in pooled food samples collected in 1995 at supermarkets across the United States. Chemosphere, 34(5-7), 1437-1447.

Schecter, A. & Li, L. (1997). Dioxins, dibenzofurans, dioxin-like PCBs, and DDE in U.S. fast food, 1995. Chemosphere, 34(5-7), 1449-1457.

Seegal, R.F. (1999). Are PCBs the major neurotoxicant in Great Lakes salmon? Environmental Research, 80(2 Pt. 2), S38-45.

Sher, E.S., Xu, S.M., Adams, P.M., Craft, C.M., Stein, S.A. (1998). The effects of thyroid hormone level and action in developing brain: Are these targets for the actions of polychlorinated biphenyls and dioxins? Toxicology and Industrial Health, 14.(1/2), 121-158.

Seward, A. & Jones, KC. (1996). A survey of PCB congeners in U.K. cows' milk. Chemosphere, 32(12), 2481-2492.

Sherbaniuk, R. (1998). The price of protection. Alberta Views, 1(2), 26-33.

Sherer, R.A. & Price, P.S. (1993). The effect of cooking processes on PCB levels in edible fish tissue. Quality Assurance: Good Practice, Regulation and Law, 2(4), 396-407.

Sinks, T., Steele, G., Smith, A.B., Watkins, K., Shults, R.A. (1992). Mortality among workers exposed to polychlorinated biphenyls. American Journal of Epidemiology, 136(4), 389-98.

Slovic, P. (1987). Perception of Risk. Science, 236, 280-285.

Slovic, P., Flynn, J., Mertz, C.K., Mullican, L, Krewski, D., Bartlett, C. (1992). Health risk perception in Canada. Health and Welfare Canada: Eugene, Oregon.

Starr, C. (1969). Social benefit versus technological risk: What is our society willing to pay for safety? Science, 165, 1232-1238.

Stewart, P., Darvill, T., Lonky, E., Reihman, J., Pagano, J., Bush, B. (1999). Assessment of prenatal exposure to PCBs from maternal consumption of Great Lakes fish: An analysis of PCB pattern and concentration. Environmental Research, 80, S87-S96.

Svensson, B.-G., Hallberg, T., Nilsson, A., Schütz, A., Hagmar, L. (1994). Parameters of immunological competence in subjects with high consumption of fish contaminated with persistent organochlorine compounds. International Archives of Environmental Health, 65, 351-358.

Swain, W.R. (1991). Effects of organochlorine chemicals on the reproductive outcome of humans who consumed contaminated Great Lakes fish: An epidemiologic consideration. Journal of Toxicology and Environmental Health, 33, 587-639.

Thomas, S. & Hrudey, S. (1997). The risk of death in Canada: What we know and how we know it. University of Alberta press: Edmonton, Alberta.

Tryphonas, H. (1998). The impact of PCBs and dioxins on children's health: Immunological considerations. Canadian Journal of Public Health, 89(supplement 1), S49-S52.

United States Environmental Protection Agency (1990). Public knowledge and perceptions of chemical risks in six communities: Analysis of a baseline survey. Author: EPA-230-01-90-074.

Urabe, H. & Asahi, M. (1984). Past and current dermatological status of Yusho patients. American Journal of Industrial Medicine, 5, 5-12.

Van Oostdam, J., Gilman, A., Dewailly, E., Usher, P., Wheatley, B., Kuhlein, HV., Neve, S., Walker, J., Tracy, B., Feeley, M., Jerome, V., Kwavnick, B. (1999). Human health implications of contaminants in Arctic Canada: A review. The Science of the Total Environment, 230, 1-82.

Vartiainen, T., Jaakkola, J.J., Saarikoski, S., Tuomisto, J. (1998). Birth weight and sex of children and the correlation to the body burden of PCDDs/PCDFs and PCBs of the mother. Environmental Health Perspectives, 106(2), 61-66.

Vyner, H. (1988). Invisible trauma: Psychosocial Effects of the Invisible Contaminants. DR. Health: Toronto.

Wania, F. & Mackay, D. (1996). Tracking the distribution of persistent organic pollutants. Environmental Science & Technology, 30(9), 390A-396A.

Wallace, J.C., Basu, I., Hites, R.A. (1996). Sampling and analysis artifacts caused by elevated indoor air polychlorinated biphenyl concentrations. Environmental Science & Technology, 30(9), 2730-2734.

Weisglas-Kuperus, N., Sas, T.C.J., Koopman-Esseboom, C. et al. (1995). Immunologic effects of back-ground prenatal and postnatal exposure to dioxins and polychlorinated biphenyls in Dutch infants. Pediatric Research, 38, 404-410.

Weiss, B. (1998). A risk assessment perspective on the neurobehavioral toxicity of endocrine disruptors. Toxicology and Industrial Health, 14(1,2), 341-359.

Weisweiller, R. (1990). How the Foreign Exchange Market Works. New York Institute of Finance: New York.

Wier, David (1987). The Bhopal Syndrome. Sierra Club Books: San Francisco.

Willett, LB, Liu, TTY, Durst, HI, Cardwell, BD, Renkic, ED. (1985). Quantification and distribution of polychlorinated biphenyls in farm silos. Bulletine of Environmental Contamination and Toxicology,35, 51-60.

Tilson, H.A., Jacobson, J.L., Rogan, W.J. (1990). Polychlorinated biphenyls and the developing nervous system: Cross species comparisons. Neurotoxicology and Teratology, 12, 239-48.

Wilson, ND., Shear, N.M., Paustenbauch, DJ, and Price, PS. (1998). The effect of cooking practises on the concentration of DDT and PCB compounds in the edible tissue of fish. Journal of Exposure Analysis & Environmental Epidemiology, 8(3), 423-440.

Winneke, G., Bucholski, A., Heinzow, B., Krämer, U., Schmidt, B., Walkowiak, J., Wiener, J-A., Steingrüber, H-J. (1998). Developmental neurotoxicity of polychlorinated biphenyls (PCBS): Cognitive and psychomotor functions in 7-month old children. Toxicology Letters, 102-103, 423-428.

World Health Organization. (1993). Polychlorinated biphenyls and terphenyls (2nd Ed.). Author: Finland.

Yamashita, F. & Hayashi, M. (1985). Fetal PCB syndrome: Clinical features, intrauterine growth retardation and possible alteration in calcium metabolism. Environmental Health Perspectives, 59, 41-45.

Yu., M-L., Guo, Y.L., Hsu, C-C., Rogan, W.J. (1997). Increased mortality from chronic liver disease and cirrhosis 13 years after the Taiwan "Yucheng" ("oil disease") incident. American Journal of Industrial Medicine, 31, 172-175.

Appendix One

EPA Tiered Approach to PCB Risk Assessment

- High risk and persistence (upper bound slope, 2 per mg/kg/day); central-estimate slope, 1 per mg/kg/day). The highest slope is used for pathways in which environmental processes tend to increase risk: food chain exposure, sediment or soil ingestion, dust or aerosol inhalation, exposure to dioxinlike, tumor-promoting, or persistent congeners, and early-life exposure (all pathways and mixtures).

- Low risk and persistence (upper bound slope, 0.4 per mg/kg/day; central-estimate slope, 0.3 per mg/kg/day). A lower slope is appropriate for pathways in which environmental processes tend to decrease risk: ingestion of water-soluble congeners and inhalation of evaporated congeners. Dermal exposure is also included because PCBs are incompletely absorbed through the skin; however, if an internal dose has been calculated by applying an absorption factor to reduce the external dose, then the highest slope would be used with the internal dose estimate.

- Lowest risk and persistence (upper bound slope, 0.07 per mg/kg/day; central-estimate slope, 0.04 per mg/kg/day). The lowest slope is used when congener or homolog analyses verify that congeners with more than four chlorines comprise less than one-half percent of total PCBs.

(Source: Cogliano, 1998)

Appendix Two: HREB Approval

Appendix Three

Introduction and Request for Interview

Hello, my name is _____and I'm calling from the Population Research Laboratory at the University of Alberta. Have I dialed XXX-XXXX? Your telephone number was selected at random by computer.

Press "1" to continue.

We would like to speak to a person in your household who is 18 years of age or older. The Lab is helping Dr. Ken Froese and his graduate student Susan Fossey at the University to select residents of Swan Hills and area to participate in a voluntary research study about their views on health risk from environmental chemical exposure.

Optional: We don't always speak to the person who answers the phone. For this study I would like to speak to an adult (male/female) member of the household. Is there an adult (male/female) available? (REPEAT INTRODUCTION)

RECORD SEX OF RESPONDENT:

1 Male
2 Female

What was your age on your last birthday?

If age refused, terminate the interview - Ctrl-End and enter Initial Refusal disposition code.

As you may know, in 1996 the Alberta Special Waste Treatment Centre, located 15 kilometres northeast of Swan Hills, experienced a series of technical problems. This resulted in the release to the environment of polychlorinated biphenyls, which are also called PCBs and dioxins. Almost four years later, some people apparently remain concerned about possible health effects from this event. Health advisories put in place in October 1996 are still in effect with no present plans to change them. In response to these issues, a Provincial Court order has funded the University of Alberta to conduct a follow-up survey of residents in Swan Hills and area.

For this study, the university researchers will be surveying 50 people to assess their views on health risks from environmental chemical exposure. The survey will also ask general questions about residents' views of the events, effects and impact of the 1996 events. We are hoping that the survey will let us know what information area residents would like to know based on what they understand about the incident. It will also identify what scientific information is available to satisfy those information needs. This research will assist in developing better ways to communicate with communities about environmental issues and health. It will also help to identify means to simplify access to environmental

information needed in communities for informed decision-making. The insights gained from this research will be used to develop an understanding of what community residents regard as being safe.

The survey will be conducted over the telephone and could take 20 to 25 minutes. The study is entirely voluntary and I want to assure you that any information you provide will be kept confidential.

The findings of the study will be published in scientific journals. Dr. Froese and his graduate student will provide participants with a copy of the findings if they so desire.

Would you be interested in participating in this survey?

1 Yes (CONTINUE WITH SURVEY)
2 No (TERMINATE INTERVIEW AND CODE AS A REFUSAL)

I would like to interview you and I'm hoping that now is a good time for you. Your opinions are important. Before we start, I would like to assure you that your participation is entirely voluntary and that any information you provide will be used only for the indicated purposes in conformity with the Alberta Freedom of Information and Protection of Privacy Act. If there are any questions that you do not wish to answer, please feel free to point these out to me and I'll go on to the next question. You, of course, have the right to terminate the interview at any time.

If you have any questions about the survey, you may call Dr. Froese at 492-1190 or Donna F: ng, Research Manager, at the Population Research Laboratory at 492-4659, Ext. 228, for further information.

Appendix Four

Wild Game and Fish Public Health Advisory

Wild game

- Limit eating wild game taken from within a 30 km radius of Swan Hills Treatment Centre to 13 ounces per month (370 grams);
- Avoid eating organ meat (liver, kidney) or using fat from wild game harvested within a 30 km radius of the treatment centre
- Pregnant or breast feeding women should avoid eating wild game taken from within a 30 km radius of the treatment centre
- Young children should avoid eating wild game taken from within a 30 km radius of the treatment centre

Fish

- Limit eating fish taken from within a 20 kilometre radius of the Swan Hills Waste Treatment Centre to 6 oz (170 grams) per week or less
- Avoid eating fish organs or eggs taken from lakes within the 20 kilometre radius
- Avoid eating fish from lakes within the 20 kilometre radius if pregnant or breast feeding
- Young children should avoid eating fish taken from within the 20 kilometre radius

Fish Preparation Instructions

- Remove the skin before cooking the fish
- Trim the fat from the fish (belly flap, sides, back and under the skin)
- Broil or bake the fish on a rack so the fats drips away
- Do not use the drippings to prepare any other recipes

The advisory applies to animals and fish taken in the fall of 1996, as well as to those taken in the current year.

Appendix Five

Survey Tool: *Perspectives on Health Risk From Environmental Chemical Exposure*

Demographics

1. Gender
   - ❑ Male
   - ❑ Female

2. Age

❑ 18-20	❑ 36-40	❑ 56-60
❑ 21-25	❑ 41-45	❑ 61-65
❑ 26-30	❑ 46-50	❑ 66-70
❑ 31-35	❑ 51-55	❑ 70+

3. Occupation
   - ❑ resource based industry
   - ❑ agriculture
   - ❑ manufacturing
   - ❑ construction
   - ❑ service industry
   - ❑ homemaker
   - ❑ unemployed
   - ❑ other, please specify _____

4. Do you or any member of your family work at the Alberta Special Waste Treatment Centre?
   - ❑ Yes
   - ❑ No

5. Marital Status
   - ❑ Married or common-law
   - ❑ Single
   - ❑ other, please specify _____

6. Children in the household?
   - ❑ Yes, specify number and ages
     - Child 1 _____
     - Child 2 _____
     - Child 3 _____
   - ❑ No

7. Duration of Residence in Community

❑ 2 to 3 years	❑ 3 to 4 years	❑ 10 or more years
❑ 4 to 5 years	❑ 5 to 10 years	

8. Level of Schooling
   - ❑ some high school
   - ❑ graduated high school
   - ❑ some post secondary education (post secondary not completed)
   - ❑ trades or non-university certificate of diploma
   - ❑ completed university

9. If you had an opportunity to vote today on whether the community of Swan Hills should have hosted the Alberta Special Waste Treatment Centre, how would you vote?

- ❑ in favor
- ❑ against
- ❑ don't know

Questions

1. Compared to other communities in Alberta, do you think Swan Hills has:

	Yes	No	Don't know
a low rate of unemployment			
clean air and water			
good schools			
good health care			
low rate of crime			
few environmental health concerns			

2. Compared to other health and safety risks, such as motor vehicle collisions, violent crime, and heart disease do you think the risks to your health from chemicals produced, stored or processed near your community are:

- ❑ not a problem
- ❑ a minor problem
- ❑ a slightly serious problem
- ❑ a serious problem
- ❑ a very serious problem

3. The Alberta Special Waste Treatment Centre has received substantial media coverage in the last 3 years. What do you know about this facility? (Do not read list, check all mentions)

- ❑ aware of its existence
- ❑ environmental contamination
- ❑ provincial government involvement
- ❑ support through Alberta tax dollars
- ❑ technical malfunctions
- ❑ scientific disagreement
- ❑ lawsuits
- ❑ fines for environmental law infractions

- ❑ nothing
- ❑ other, please specify

4. Do you believe that the ASWTC poses a threat to the safety of the environment, such as a threat to the air, water or soil?

- ❑ Yes
- ❑ No

5. To what extent, if at all, are you personally bothered by the presence of the ASWTC near your community?

- ❑ not at all, skip to question 7
- ❑ somewhat, go to question 6
- ❑ a great deal, go to question 6

6. What in particular bothers you about the ASWTC? (Do not read, record all mentions)

- ❑ an unpleasant smell
- ❑ the danger it poses to health in the long run
- ❑ dust, dirt or smoke in the air
- ❑ the possibility that a major accident could harm or kill people
- ❑ long term damage to the environment
- ❑ decrease in property values
- ❑ negative attention in the media
- ❑ other, please specify _____

7. Has your opinion of the ASWTC changed in the last 3 years?

- ❑ Yes, go to question 8
- ❑ No, go to question 9

8. What made you change your viewpoint? (Do not read list, record all mentions)

- ❑ negative media attention
- ❑ personal experience
- ❑ health concerns
- ❑ other, please specify_____

9. In the past 3 months have you read or heard anything about the health risks of chemicals or hazardous waste in your community?

- ❑ Yes, go to question 10
- ❑ No, skip to question 12

10. What was the information you read or heard?

11. Where did you hear or read this information? (do not read list, record all mentions)

- ❑ doctor
- ❑ other health professional
- ❑ friends or relatives
- ❑ government officials
  - ❑ local
  - ❑ provincial
  - ❑ federal
- ❑ ASWTC officials
- ❑ people you know who work at the ASWTC
- ❑ environmental group, please specify _____
- ❑ media

- ❑ other, please specify _____

12. There are several different sources of information about the risks that chemicals *may* pose to a one's health. Would you say you get a lot, a little, or no information about the health risks of chemicals in your community from:

	A lot	A little	None
friends and relatives			
Doctor			
other health professionals			
local government officials			
provincial government officials			
federal government officials			
ASWTC officials			
people you know who work for the ASWTC			
environmental groups			
the media			

13. How much do you trust each source of information?

	A lot	A little	None
friends and relatives			
Doctor			
other health professionals			
local government officials			
provincial government officials			
federal government officials			
ASWTC officials			
people you know who work for the ASWTC			
environmental groups			
the media			

14. How knowledgeable do you think each source is about the risks of to one's health?

	A lot	A little	None
friends and relatives			
doctor			
other health professionals			
local government officials			
provincial government officials			
federal government officials			
ASWTC officials			
people you know who work for the ASWTC			
environmental groups			
the media			

15. Has the amount of trust in each source changed over the last three years?

friends and relatives	A lot	A little	Not at all
Doctor			
other health professionals			
local government officials			
provincial government officials			
federal government officals			
ASWTC officials			
people you know who work for the ASWTC			
environmental groups			
the media			

16. In the cases where there has been a change in the level of trust, what, if anything caused the change?

17. How much information do you feel you know about the following environmental topics?

	A lot	A little	Not at all
releases of chemicals into the atmosphere			
the quality of your area's drinking water			
the quality of your area's edible fish and game			
The ASWTC			
activities to clean up releases of hazardous material			
health risks from chemicals in your area			

18. Your personal opinions on the next few questions are very important to us. Do you Strongly Disagree, Disagree, Agree, Strongly Agree or hold no opinion on the following statements?

	Strongly agree	Agree	Disagree	Strongly disagree	No opinion
We should assume a chemical is safe until tests prove it to be dangerous					
Chemicals have improved our health more than they have harmed our health					
Any release of chemicals into the air, water, or soil is unacceptable					
The only time the public hears about the release of toxic chemicals is when the problem is so big it can't be kept secret any more					
It's not how much of a chemical you are exposed to that matters to your health, it's whether you are exposed at all					
If a person is exposed to a chemical that can cause cancer, then that person is likely to get cancer later in life					
As an individual, there are many ways I can reduce my exposure to chemicals in the environment					

There are some chemical risks that are too small to worry about					
Health advisories are a useful way to limit human exposure to toxic chemicals					
Natural chemicals, as a rule, are not as harmful as manmade chemicals					
There is no safe level of exposure to a cancer causing chemical					
A small amount of risk is OK if it brings economic benefit					
Local businesses are usually very careful with dangerous chemicals					
I feel I am involved in environmental decisions that may affect my health					
Scientists and experts are likely to find ways to significantly reduce the threats from environmental contamination					

19. From zero to 100, with zero meaning no chance and 100 meaning 100 percent, what do you think is the probability that you have been exposed to toxic chemical in the past 5 years?

Record actual number _____

20. From zero to 100, with zero meaning no chance and 100 meaning 100 percent, what do you think is the probability that your exposure to toxic chemicals has harmed or will harm your health?

Record actual number _____

21. From zero to 100, with zero meaning no chance and 100 meaning 100 percent, what do you think is the probability that living near to the Alberta Special Waste Treatment Centre will harm or has harmed your health?

Record actual number _____

22. There are a number of things that people sometimes do to protect themselves from chemical risks in the environment. Is there anything you personally have done because of potential chemical of risks in the environment? (Do not read list, record all mentions)

- ❑ used bottled water
- ❑ moved or chosen not to live in a certain house
- ❑ gone to the library to find out more about a problem
- ❑ attended a town or community meeting

- ❑ contributed time or money to an environmental cause
- ❑ called or written a government official
- ❑ stopped eating a certain food item
- ❑ stopped drinking a certain beverage
- ❑ changed the amount of time you spend outdoors
- ❑ used personal protective equipment
- ❑ nothing
- ❑ other, please specify _____

23. PCBs and dioxins are common environmental pollutants. Sometimes they are found in small amounts in the foods we eat? If they were found in something **you** ate regularly, what would you do? (do not read list)

- ❑ nothing, continue to eat that food item
- ❑ think about eating less of that food item
- ❑ eat less of that food item
- ❑ stop eating that food item
- ❑ stop eating that food item and try to make sure other did the same
- ❑ I don't know
- ❑ other, please specify _____

24. Some chemicals, like vitamins or the alcohol in red wine, may prevent some health problems if taken in small amounts. On the other hand, in large amounts, they may cause serious health problems. If you were told that some environmental chemicals, in small amounts, could prevent serious health problems, how would that change your responses to the previous question?

- ❑ no change
- ❑ I don't know
- ❑ other, please specify _____

25. A new theory in environmental science is that some chemicals may act in the body like the hormones naturally found there. What, if anything, do you know about this topic? (do not read list, record all mentions)

- ❑ nothing
- ❑ heard of it
- ❑ endocrine disruption
- ❑ other, please specify _____

26. There are health advisories in effect for the Swan Hills region. What, if anything, do you know about them? (do not read list, record all mentions)

- ❑ aware of their existence
- ❑ specific populations to whom the advisories are targeted
- ❑ specific amounts of fish and game recommended
- ❑ geographic extent of advisories
- ❑ specific chemicals targeted by the advisories
- ❑ duration of the advisories
- ❑ risk reduction measures that could be taken

- ❑ nothing
- ❑ other, please specify _____

27. Where did you learn about the advisories?

- ❑ doctor
- ❑ other health professional
- ❑ friends or relatives
- ❑ government officials
  - ❑ local
  - ❑ provincial
  - ❑ federal

- ❑ ASWTC officials
- ❑ people you know who work at the ASWTC
- ❑ environmental group, please specify _____
- ❑ media

- ❑ other, please specify _____

28. Do you follow the advisories?

- ❑ Yes
- ❑ No

Why or why not?

29. Do you think the advisories are necessary?

- ❑ Yes
- ❑ No

Why or why not?

30. What type of information would you **like** to receive about health advisories? (Do not read, record all mentions)

- ❑ Potential health risks from exposure
- ❑ Type and extent of environmental contamination
- ❑ Process for establishing health risk advisories
- ❑ Scientific evidence supporting the advisory
- ❑ Explanatory information
- ❑ what species, sources or types
- ❑ what amount (of water or fish, etc.), or types of exposure to avoid (e.g. licking of PVC blinds)
- ❑ from what areas,
- ❑ what times of the year,
- ❑ frequency of consumption
- ❑ specific populations affected (e.g children, pregnant women, immune-suppressed)
- ❑ Duration of the advisory
- ❑ Risk reduction measures that could be taken (i.e. cooking, cleaning, boiling water, removing blinds from the reach of children)

- ❑ nothing
- ❑ other

31. If there was a risk to human health from a chemical in your community, how do you think you would **first** find out about it? (do not read list, record all mentions)

- ❑ doctor
- ❑ other health professional
- ❑ friends or relatives
- ❑ government officials
  - ❑ local
  - ❑ provincial
  - ❑ federal
- ❑ ASWTC officials
- ❑ people you know who work at the ASWTC
- ❑ environmental group, please specify _____
- ❑ media

- ❑ other, please specify _____

32. How would you **like** to see this information communicated to the public? (do not read list, record all mentions)

- ❑ delivered directly residents
- ❑ posted signs
- ❑ research reports
- ❑ radio
- ❑ newspaper
- ❑ television
- ❑ Internet websites
- ❑ public meeting
- ❑ designated person/telephone number to call
- ❑ other, please specify _____

33. What role, if any, should the public play in setting a health risk advisory or warning? (Do not read, record all mentions)

- ❑ Public meetings
- ❑ Public Advisory Groups
- ❑ Regular consultation (eg. Interviews, focus groups)
- ❑ Opportunity to obtain further information, if desired (e.g. designated person/telephone number to call)
- ❑ Opportunities for input at the beginning of in the process
- ❑ Opportunities for input at specific times during the process
- ❑ Opportunity to comment on advisory before issued

- ❑ other

34. If you or your family were personally at risk, how would you like to be involved in making decisions about setting a health risk advisory or warning? (Do not read, record all mentions)

- ❑ Public meetings
- ❑ Public Advisory Groups
- ❑ Regular consultation (eg. Interviews, focus groups)
- ❑ Opportunity to obtain further information, if desired (e.g. designated person/telephone number to call)
- ❑ Opportunities for input at the beginning of in the process
- ❑ Opportunities for input at specific times during the process
- ❑ Opportunity to comment on advisory before issued

- ❑ other

35. Would you like written results of this survey?

❑ Yes, please provide telephone number and mailing address
❑ No

36. Would you be interested in participating in personal, in-depth interviews or focus groups?

❑ Yes, please provide telephone number and mailing address
❑ No

37. Would you be interested in attending a community presentation on the results of this survey?

❑ Yes, please included telephone number and mailing address
❑ No