



Environmentally Attributable Cancers in Washington State, US: Applying Economic Cost Estimates and the Precautionary Principle

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R. Dietrich Hauge
275 W. Roy St. #311,
Seattle, WA 98119, United States
deehauge@hotmail.com

Thesis Advisor: Göran Ewald PhD.
Lund Institute of Technology/
Chemical Engineering
Goran.Ewald@chemeng.lth.se
046-222-38-78

Local Supervisor: Kate Davies MA, DPhil.
Antioch University-Center for Creative Change
2326 Sixth Ave., Seattle, WA 98121-181
kdavies@antiochsea.edu
01-202-268-4908

Abstract

BACKGROUND: The carcinogenic effects of chemicals in the environment are a growing concern across the world. Significant research has established the connections between toxic chemicals and a multitude of cancer variations (CHE, 2004). Subsequently, to protect the health of its citizens and the environment, the State of Washington (located in the Pacific Northwest region of the United States) is currently attempting to be the first state to implement policies eliminating the production and circulation of persistent bioaccumulative toxic substances (PBTs). These actions are based on the tenets of the precautionary principle. However, stakeholder disputes regarding the costs accrued by precautionary measures and the potential exclusion of certain chemicals has threatened the overall effectiveness of the proposal. **OBJECTIVES:** The objectives of this study are threefold: one, to highlight the available literature concerning incidence, trends, and environmental linkages associated with cancer in Washington State (WA); two, to estimate the economic costs of environmentally attributable childhood cancers in WA; and three, to discuss the application of the precautionary principle in WA, specific to chemical regulation and its contribution to sustainable development. **METHODS:** This study conducts a literature review to strengthen the association between toxics and cancer; a comparative analysis of cancer with environmental origins between Washington State and the United States' mean; and economic cost estimations of environmentally attributable pediatric cancers in Washington State. The concept of the environmentally attributable fraction (EAF), the percentage of cancer directly caused by environmental factors, was applied to total direct and indirect costs. **RESULTS:** WA has especially high incidence of cancers with environmental origin and is above the national average in 9 of 10 cancer categories. Most significantly, WA has the nation's highest incidence of female breast cancer and melanoma of the skin. Pediatric cancers are also above the United States' average. This study has estimated that pediatric cancers alone cost the state of Washington \$222.9 million in 2004. Assuming a conservative 5% EAF, the cost of pediatric cancer due to toxic substances in the environment is \$11.2 million per year, with a possible cost range of \$4.5 to \$200.6 million. **CONCLUSIONS:** There is enough evidence to suggest that anthropogenic chemicals are significant contributors to the elevated rates of cancer in WA. Estimating the costs of these cancers will demonstrate to policy makers that investing in precautionary measures, rather than continual treatment, is intelligent fiscal policy. A population with high rates of life-threatening diseases is not sustainable by any measure. Therefore, to promote sustainable development, this study supports the integration of the precautionary principle into Washington State chemical regulation.

Keywords: cancer, precautionary principle, persistent, bioaccumulative, toxic substances, chemical regulation, economic cost assessment, body burden, Washington State.

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Acronyms & Abbreviations

ACS	American Cancer Society
CHE	The Collaborative on Health and the Environment
CLD	Causal Loop Diagram
CNS	Central Nervous System
CPI	Consumer Price Index
DDT	Dichloro Diphenyl Trichloroethane
DES	Diethylstilbestrol
DNA	Deoxyribonucleic Acid
DOE	Washington State Department of Ecology
DOH	Washington State Department of Health
EAF	Environmentally Attributable Fraction
EU	European Union
IR	Ionizing Radiation
NHL	Non-Hodgkin’s Lymphoma
PAHs	Polycyclic Aromatic Hydrocarbons
PBDEs	Polybrominated Diphenyl Ethers
PBTs	Persistent Bioaccumulative Toxic Substances
PCBs	Polychlorinated Biphenyls
PP	Precautionary Principle
SEER	Surveillance, Epidemiology and End Results Program
US	United States
USD	United States Dollar
US EPA	United States Environmental Protection Agency
UV	Ultraviolet
WA	Washington State
WSCR	Washington State Cancer Registry
WHO	World Health Organization

1. Introduction

There are currently over 85,000 anthropogenic chemicals circulating in the world's environment (City of San Francisco, 2003). Many of these chemicals have been found in the breast milk of mothers, the tissues of children, and measured in the most remote corners of the Earth (Thornton, McCally, & Houlihan, 2002). Science has established that every single day our bodies absorb and store toxic chemicals; this phenomenon is appropriately called the 'body burden' (Northwest Environment Watch, 2004). Some of these compounds have been determined to cause a variety of cancers and other adverse health outcomes. There is a great deal of evidence linking the increased production of chemicals to the increasing rates of breast cancer, Non-Hodgkins lymphoma, as well as childhood leukemia and brain cancer (National Cancer Institute, 2003; Solomon, Ogunseitan, & Kirsch, 2000). However, to what degree these chemicals are contributing to health problems such as cancer is under debate, as is the best policies to protect our communities. Furthermore, the economic burden of cancer has not been fully calculated or internalized. It is now believed that approximately 75% of all cancer cases in the US can be attributed to some form of environmental exposure (American Cancer Society, 2004). These cancers are entirely preventable. This study proposes that preventive measures, such as the precautionary principle (PP), are needed to sufficiently safeguard the health of our people and the environment from the effects of toxics through improved chemical regulation. The definition of the PP originates from the 1992 Rio Declaration, Principle 15: "In order to protect the environment, the precautionary approach shall be widely applied by States according to their capabilities. Where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation" (United Nations, 1992). The proper mixture of common sense, scientific awareness, and preventative action will promote a sustainable future. This study will focus its attention on environmentally attributable cancers in Washington State (WA) of the United States, the current attempts made to advance the implementation of the precautionary principle, the interaction between chemicals in the environment and cancer, and the economic costs resulting from these cancers.

In January 2004, the Seattle Precautionary Principle Working Group submitted a discussion-paper titled, *A Policy Framework for Adopting the Precautionary Principle, to the City of Seattle and King County*. The objective of this document was to incorporate the precautionary principle into the language of the 2004 city and county comprehensive plans. The Seattle and King County governing officials and policy makers are currently considering this proposal. When discussing the economic costs of childhood disease attributable to environmental quality, the authors had stated, "Researchers and policymakers do not know the overall costs of childhood illness in Washington State" (Gilbert, Diver, & Miller, 2004: 9). Their argument had to be associative using cost-estimates from other states. In response to this lack information, one fundamental goal of this analysis is to determine the economic costs of pediatric cancer. A cost assessment will bolster the argument of the Precautionary Principle Working Group to advocate for precautionary measures and promote the political ambition necessary to do so.

Currently, Washington State is the only state in the union with a comprehensive strategy for managing persistent bioaccumulative toxic substances (PBTs). Many of these chemicals are known carcinogens. In 1998, the Washington State Department of Ecology (DOE) announced a statewide

strategy to end all releases of PBTs by 2010, prohibit future releases, and clean up previously polluted locations. The statewide action plans, mandating the reduction of toxic chemicals in the environment, are based on the ideological foundations of the precautionary principle. In December 2000, the *Proposed Strategy to Continually Reduce Persistent, Bioaccumulative Toxins in Washington State*, was published. This document identified nine specific PBTs, and established the need and framework to remove these chemicals from circulation in WA. (The working list of PBTs has now grown to twenty-four). As a result, two action plans to eliminate PBTs have subsequently been released. First, in 2002, under the auspices of the Washington State Legislature, the DOE developed the *Mercury Chemical Action Plan*. The proposed measures of this plan went into effect in January 2003. The purpose of the document is to identify anthropogenic sources and describe strategies to eliminate mercury (Peele, 2003). Second, in October 2004, the *Draft PBDE Chemical Action Plan* was released to promote the voluntary phase-out of two forms of flame-retardants called polybrominated diphenyl ethers (PBDEs) (Peele, 2004). However, stakeholder disputes exist regarding the implementation of such precautionary measures. Industry is concerned that heavy regulations will put WA businesses at a competitive disadvantage in the national and world market, and that costs may be too high to invest in cleaner practices (Gallagher, 2001: 37-54). There is also current disputes regarding the application of the precautionary principle, which chemicals should be included in the PBT action plans, and if there is enough scientific evidence to warrant concern. This study will discuss the validity of stakeholder opposition and aspires to strengthen the rebuttals of PP advocacy based on economic cost assessment and public health arguments. (Gallagher, 2001)

In 1775, the first cause and effect relationship between cancer and toxic substances in the environment was established. A British surgeon, named Percival Pott, published his observations on the increased incidence of scrotal cancer in men working as chimney sweeps. Pott attributed the rise in cancer to the soot that the chimney sweeps were in daily contact (Rodricks, 1992: 108-144). Unbeknownst to Pott, the carcinogenic effect of soot can be attributed to polycyclic aromatic hydrocarbons (PAHs). These are the same PAHs that are currently under review for inclusion on the Washington State's PBT reduction list. It may bewilder health advocates that it has taken over 200 years to consider banning this substance in Washington. Furthermore, our long running knowledge of the carcinogenic properties of certain chemicals is not isolated to PAHs. Polychlorinated biphenyls (PCBs) are also currently being considered for the PBT working list and have been linked with causing chloroachne (a painful disfiguring of the skin) since 1899. Due to three deaths of factory workers from PCB exposure in the 1930s, Monsanto, the main US producer of PCBs, became aware of its carcinogenic properties. PCB production continued until the 2001 decree of the Stockholm Convention for Persistent Organic Pollutants. Benzene is another example of a compound with early indication to cause cancer. In 1897, increased observations were reported of aplastic anaemia amongst young Swedish women working in a bicycle tire manufacturing plant. That same year, hemorrhaging had been observed in men in a dry-cleaning business in France. Despite clinical observations linking benzene to adverse health outcomes, a century later, benzene is still produced and contained in everyday products such as automobile petrol. In 1987, a ten-year delay in the United States in setting a benzene regulation under 1 part per million can be held responsible for an additional 198 adult leukemia and 77 multiple myeloma deaths of workers acutely exposed to benzene. (European Environmental Agency, 2001)

By considering the long latency period between when science first suspects compounds as harmful and when any action or regulation takes place, leads us to an imperative question: When is there enough evidence enough to act? Sir Austin Bradford Hill, Professor Emeritus of Medical Statistics at the University of London, had addressed this issue when he said, “All scientific work is incomplete- whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time” (Tickner, 2003: 7). Washington State is in a position where it may protect its citizens from the harms of PBTs. However, progress is slow and encounters resistance from competing stakeholders. The Washington State PBT advisory committee has heard industry’s objection that the precautionary principle is not based on sound science, and arguments are made against the inclusion of certain chemicals on the PBT reduction list due to inconclusive causal evidence (Gallagher, 2001). Opponents of the PP must heed the early warning signs, as government and industry did not in the cases of PAHs, PCBs, and benzene. The precautionary principle states that if early an indication of plausible harm exists, and scientific uncertainty persists, it is our obligation to implement preventative actions. Inconclusive science must not be a reason to delay justifiable actions. Had the precautionary principle been implemented in cases of the past, thousands of people and their families would have been saved from the detriments of cancer and other adverse outcomes.

1.1. Four principle problems to address

I. Current policy paradigm is too reactionary, focuses not enough on precaution

Policy makers use the methodology of risk assessment to make public health decisions. Risk assessment is based on the evaluation of the potential harmful effects of a defined toxic to the environment or human health using toxicological and epidemiological data (Rodricks, 1992). There are several fundamental problems when using the risk assessment method. First, determining what is an acceptable risk is hugely debatable and unclear. For carcinogens, the acceptable risk usually falls from one in 100,000 to one in 1,000,000 chance of cancer being caused by a particular agent over a lifetime. Second, the process lacks moral and ethical considerations. It regards citizens as statistics, not human beings. What may be considered an acceptable risk to a policy-maker may not be acceptable to the individual. Thirdly, it places the burden of proof on the effected, not the polluter. Those exposed to chemicals will commonly lack the resources or know-how to combat the polluting industries. Finally, it can be very difficult to prove the etiology of cancer due to the variety of potential exposures, a long latency period before signs and symptoms manifest, the windows of exposure may also be short and hard to pinpoint, and exposures may be lifelong at low or virtually undetectable levels. A fundamental philosophical difference exists between the risk assessment and precautionary approaches. When risk assessment asks how much risk is tolerable, the precautionary approach asks how much risk can be avoided? (Davies, 2004)

II. Insufficient understanding of the linkages between cancer and environment

An astonishing, 99% of chemicals (by volume) on the United States market have never been subject to any form of toxicological or epidemiological testing (Tickner, 2003: 104). Therefore, it is obvious that scientific understanding of the potential harms to the public by these chemicals is entirely lacking. For example, there is currently little information regarding the consequences of pesticide application in

agriculture and gardens. There is only a modest understanding of the relationship between toxic chemicals and the rising rates of childhood leukemias and brain cancer. Factors such as antagonistic and synergetic effects of chemicals are poorly understood, and associations concerning the timing of exposure need much more attention (Colborn, Dumanoski, & Peterson-Myers, 1997). Research that aims to establish the causal linkages between chemicals and health outcomes is of the utmost importance; because protecting the physical wellbeing of our citizens begins with improving the understanding how and where the threats manifest.

III. High rates of cancer in Washington State

It is safe to say that cancer is a major health problem in Washington State. In fact, one out of every two males, and one of every three females in Washington will be diagnosed with cancer at some point in their lifetime (American Cancer Society, 2004). The Center for Disease Control (2004) reported in 2000, that 23,600 new cases of cancer were reported in WA. Exceeded only by heart disease, cancer is the second overall leading cause of death in the state, and it is the leading cause of death for those ages 45-75 (DOH, 2004a). The Washington State Cancer Registry reported that female breast cancer has the highest rate of new diagnoses, followed by prostate cancer in males, lung cancer, and melanoma (DOH, 2001b). Specific rates, trends, and a national comparison of cancer in Washington will be detailed later in this analysis.

IV. Lack of information detailing the economic costs of cancer

In 2002, the American Cancer Society calculated that 11,100 deaths in WA were caused by cancer, costing the state an estimated \$3.4 billion USD (Trust for America's Health, 2004). The categories included in this estimate include: direct medical costs, cost of lost productivity due to illness, and cost of lost productivity due to death. However, this study does not consider environmentally attributable factors into their equation. Landrigan et al., (2002) conducted the only national study that incorporates environmentally attributable factors. They concluded that the total costs of pediatric cancer in the US to \$332 million, with a range from \$132-663 million. The only state specific cost estimates derive from Ackerman and Massey (2002). Implementing a similar methodology as the Landrigan study, they determined that the cost of pediatric cancer in Massachusetts ranges from \$9.1 to \$163.8 million per year. To date, no economic cost assessment of cancer or pediatric cancers caused by environmental factors has been conducted in WA. Economic cost estimates can benefit policy makers when making choices regarding public health. The discourse surrounding public health issues involves constant debate between industry and environmental advocacy. Monetary estimates can help bridge the divide between competing stakeholders and allow policy makers to make responsible governance and fiscal decisions.

1.2. Connection to sustainable development

This project was designed to be holistic in nature and pragmatic in application. This study addresses the topic of public health in a holistic approach, because one sphere of sustainability cannot be addressed without affecting the system as a whole. Sustainable development is broadly defined as development that meets the need of the present without compromising the ability of future generations to meet their needs (World Commission on Environment and Development, 1987). These needs certainly consist of healthy air and drinking water, but also includes economic security, job

availability, health care, and social services to name a few. Therefore, this study incorporates all three pillars of sustainability (environmental, social, and economic) and attempts to strengthen the interconnections between overlapping issues. Advocating the precautionary principle will, by design, advocate sustainable development. For example, by improving our understanding of how environmental toxics contribute to cancer, efforts can be made to minimize these compounds in the environment. Such measures can benefit both the citizens of Washington and its natural surroundings. Additionally, by conducting a cost-assessment of cancer, the connections between economic and social sustainability are made clearer. Understanding the linkages and commonalities, between what is often competing interests, can foster collaboration and ultimately make major strides toward achieving sustainability.

2. Objectives, Scope & Methodology

Objective 1. Describe the historical application of the precautionary principle, and discuss its relevance in the regulation of chemicals in Washington State

This paper will illustrate the need for the precautionary principle to be implemented in WA's comprehensive policy and highlight its strategy to continually reduce persistent bioaccumulative toxic substances. The history of precautionary measures in the United States and internationally will be included to demonstrate prior successes, indicating a methodical change in paradigm; departing from the reactionary policies of the past toward more preventative approaches of the future. This study will also discuss how precautionary measures can promote sustainable development. Current literature will be reviewed including stakeholder arguments and critiques. To maintain a limited scope of this study, discussion of precautionary principle critiques must remain general to be universally applicable. The cost-benefit ratio of incorporating the precautionary principle will vary case by case and cannot be discussed individually. Examples of primary data sources for precautionary principle discussion derive from publications of the European Environmental Agency, Washington State Departments of Health and Ecology, the Seattle Precautionary Principle Working Group, as well as various online resources.

Objective 2. Highlight the available literature concerning incidence, trends, and environmental linkages associated with cancer in Washington State

When possible, a comparison will be made with national statistics, which will determine the need for preventative cancer policies in Washington State. Theorizing why WA has certain rates of cancer is not the focus of this analysis. Emphasis will be placed on summarizing the available existing research, linking certain cancers to environmental toxics. Any temporal trends will be noted, such as increasing or decreasing rates of incidence. For the sake of consistency and comparison, incidence will be highlighted (opposed to mortality or morbidity). Incidence is defined as the rate or occurrence of new cases of a given disease in a particular year (DOH, 2004a). Environmental associations will only focus on cancer variations with the highest incidence rates. To maintain a precise scope of this analysis, certain cancers must be omitted due to lack of existing information and/or low incidence rates.

Cancer sites with strong environmental linkages omitted from analysis include:

- Angiosarcoma
- Bone Cancer / Ewings Sarcoma
- Mesothelioma
- Myelodysplastic syndrome
- Nasal Cancer
- Pancreatic Cancer
- Salivary Gland Cancer
- Skin Cancer (Non-Melanoma)
- Soft Tissue Sarcoma
- Scrotal Cancer

The primary sources for data retrieval come from environmental monitoring studies, risk assessments, epidemiological studies, health surveillance studies, published studies as well as online literature from reputable sources. Occupational studies were used sparingly, only in supporting environmental toxic and health risk associations. Data sources providing ambient population exposures were emphasized. More specifically, Washington State cancer statistics were primarily derived from the WA Department of Health, WA Department of Ecology, and WA Cancer Registry. Table 7.2 (found on p. 27) was compiled using the strength of evidence associations between environmental factors and diseases compiled by scientists for the Collaborative on Health and the Environment (CHE). Washington specific incidence data was obtained by the Washington State Cancer Registry's (WSCR) publications, *Cancer by Site* and *Cancer by County*. The WSCR is a nationally renowned cancer tracking system and WA is considered a leading state in cancer prevention. The Trust for America's Health (2004), a non-profit group that assesses state cancer tracking systems, awarded the WSCR the highest grade possible, an 'A.' National incidence data was derived from the Surveillance, Epidemiology, and End Results program of the National Cancer Institute. The CHE (2004) database was used as a reference template, which categorizes health outcomes to environmental toxics and rates the strength of evidence: strong, moderate, limited or conflicting. To be considered 'strong evidence,' the causal relationship between toxic and cancer must be well established. The medical community must accept it as truth, and the connection must be described in major textbooks or documented in major prospective or retrospective cohort studies. Cancer carcinogens have to be specifically classified as a Group One by the International Agency for Research on Cancer. Toxics considered as 'good evidence,' must have associations described in epidemiological studies (cross sectional, case series, or case-control studies). The cohort in such studies may be small; conclusions could not be drawn across the population. Some human observations, in conjunction with strong animal carcinogen data, can also be grounds for a compound to be considered good evidence. 'Limited or conflicting' evidence is not scientifically conclusive and will not be considered in this analysis. Cancer outcomes will only be illustrated if they have strong associations with environmental toxics.

Limitations in the database exist in scope and depth by the current level of scientific understanding between environmental chemicals and carcinogenesis. The toxic chemicals listed do not comprise a finalized list. The dynamic nature of research will undoubtedly lead to additions and omissions from this list. Furthermore, the cancer associations do not consider variations in genetic

susceptibility, age, or body size. The database does not reference the intensity, route, timing, or duration of exposure.

Washington specific statistical information is limited because it simplifies variations across the state. It does not include county specific discrepancies, nor localized cancer cells. Incidence rates are examined according to age, but do not consider racial or socio-demographic differences. Moreover, due to low frequencies and protection of patient's rights, certain data sets are not available. For example, when searching for pediatric Non-Hodgkin's lymphoma incidence rates, the statistic is not reported or calculated because the observation rate is less than five cases per year (WSCR, 2001).

Objective 3. Estimate the economic costs of environmentally attributable childhood cancers in Washington State

This section will be dedicated to estimating the economic costs of pediatric cancers. Two preexisting publications had previously assigned dollar values to environmental cancers in the United States: a nation-wide study by Landrigan et al. (2002), *Pollutants and Disease in American Children*; and a Massachusetts-specific assessment by Ackerman and Massey (2003) titled, *Costs of preventable childhood illness: The price we pay for pollution*. Therefore, deriving cost estimates for environmentally attributable cancers will be limited to pediatric cancers due to this study's reliance on these previous assessments. Calculating the costs of all cancers caused by toxic substances in the environment is considered outside the scope of this analysis.

For comparative purposes and to maintain consistency with the Landrigan et al. and Ackerman and Massey studies, incidence rates were chosen to classify the sample population in the economic cost assessments. Incidence rates provide the costs accrued over the entire lifetime of an individual. It should be noted that advances in medical science have decreased the rates of mortality in several cancer variations, simultaneous with increasing incidence rates (American Cancer Society, Inc., 2004). Focusing on mortality could then paint an overly optimistic picture of cancer in Washington State. The most current available incidence data from the DOH is derived from the year 2001. Assumptions were made that 2004 incidence rates will be equivalent to 2001; however, the actual 2004 incidence rates may prove to be higher.

The categories included in the cost-assessment include: treatment costs, lost school and parental work time, special education, home and institutional care, costs of related illnesses in adulthood, loss of projected future earnings, and costs of suffering and death. To approximate the costs occurring in the future, the concept of discounting was utilized. The discount rate applied to future costs in this study equates to an annual rate of 3% (Landrigan et al., 2002: 724). However, there are aspects involved in cancer that cannot be assigned monetary values. This study recognizes the non-quantifiable aspects of cancer such as pain and suffering which can never truly be represented by cost estimations.

To establish the linkages between cancer incidence and environmental quality, the concept of the environmentally attributable fraction (EAF) was incorporated. The EAF is the percentage of cancer directly caused by environmental factors. We used the EAF implemented in the Landrigan et al. study,

which was determined by a panel of experts on pediatric cancer. The panel had concluded that genetic predisposition could only be attributed to 10-20% of pediatric cancer etiology; therefore the high-end estimates for EAF are 80-90%. There is no current scientific consensus regarding the actual degree the environment plays on the formation of childhood cancers. Therefore, the panel agreed on estimates of at least 5-10% and no more than 80-90%. Various estimates of cost could be taken from the calculated total cost figure, contingent on the environmentally attributable fraction. Landrigan et al. used the conservative EAF approximations of 2, 5, and 10%. The EAFs utilized in this study are: 2, 5, and 10%, to provide conservative estimates, as well as 80 and 90% to present the potential range of costs.

All monetary values in this study are based on the United States Dollar. To provide accurate and current economic estimates, the cost-category of ‘care and treatment cost per child,’ was converted from 1998 to 2004 dollar values and rounded to the nearest 10,000 dollars. (Refer to Table 8.4). Larger quantities are rounded to the nearest 100,000 dollars. Dollar values are adjusted using the US Department of Labor’s Consumer Price Index (2004) (CPI) inflation calculator, which uses the averages of the CPI over the 1998 to 2004 timeframe. This gives us current cost estimates. Once the ‘care and treatment cost’ category has been converted into current dollars, calculating the ‘total cost’ is simply product of the ‘number of children diagnosed’ and ‘care and treatment costs.’ The value for ‘total cost’ is then multiplied by various EAFs to provide the full-range of costs from childhood cancers caused by environmentally attributable factors. The general formula is as follows:

$$\text{Costs} = \text{Disease rate} \times \text{EAF} \times \text{Population size} \times \text{Cost per case}$$

To estimate the costs of environmentally attributable cancer specific to Washington State, we must integrate incidence rates with the assumptions and framework of the Ackerman and Massey and Landrigan et al. studies. Pediatric incidence rates in WA are taken from 24 total categories, representing all verifiable cancer variations in the year 2001. Of the total number of cases, leukemia and brain/CNS cancers comprise well over half the incidence. For this analysis the age range considered pediatric is 0-19 years. Incidence rates result from the average annual observations divided by the average annual population. For clarity, average annual observations were used in the calculations. The number of cases for all sites combined in WA for 2001 includes: 85 cases for the age group of 0-4, 41 cases for ages 5-9, 70 cases for ages 10-14, and 112 cases for ages 15-19 (DOH, 2004). The number of pediatric cancer cases in 2001 totaled 308.

It is important to note that calculating the costs of pediatric cancer in Washington State has a great deal of limitations. First, the estimates contain soft-variables such as loss of projected future earnings and costs of death. In no way can these variables ever be precisely calculated, however, economists use techniques such as “willingness to pay” and “contingent valuation” to derive best estimates. Factoring the loss of lifetime earnings due to cranial irradiation caused loss of IQ is speculative at best. Nonetheless, these factors were included to derive the total costs in the most accurate assessment possible.

3. Hypothesis

To determine the appropriate precautionary measures needed to improve public health, we must clarify our understanding of the health problems caused by environmental toxics. A problem must first be identified before it can be remedied. Keeping the ultimate ambition of promoting sustainable development in mind, this study hypothesizes that:

1. Rates of cancer are elevated in Washington State; this is demonstrated through a comparison with the national mean. Research is highlighted that associates cancer formation to toxic chemicals in the environment. Therefore, by drawing parallels between chemicals and cancer, strong arguments can be made to advocate for policies that improve environmental health.
2. Estimating the economic costs of environmentally attributable childhood cancers in Washington State will give precautionary principle advocates the pragmatic evidence needed to counter resistance from industry and push through policy changes.

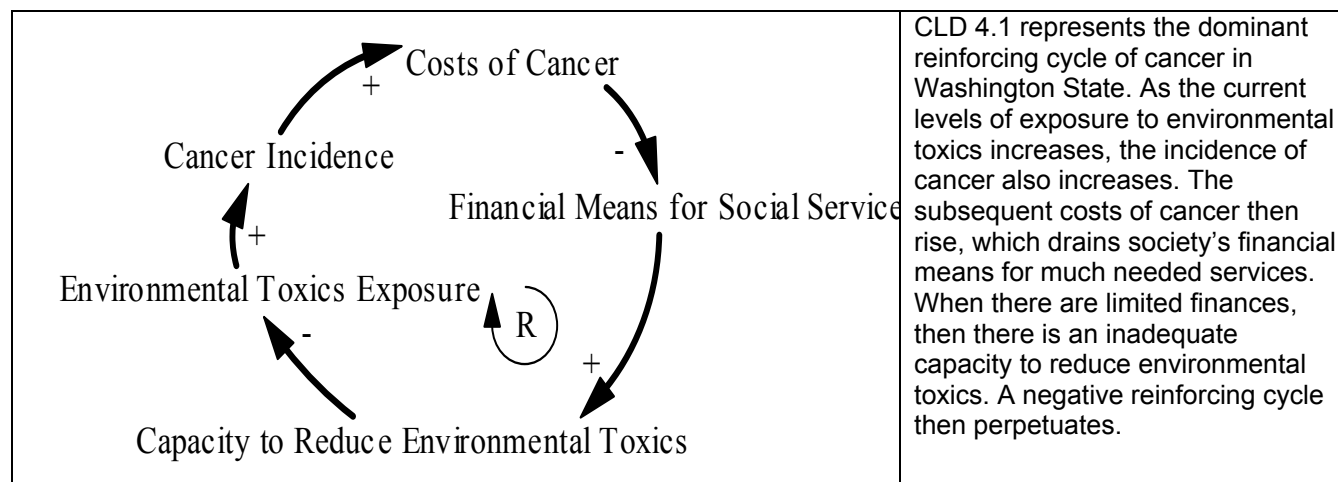
4. Analytical / Theoretical Framework

The concept of holism and systemic thinking are driving forces in the framework of this analysis. By acknowledging that wholes are greater than the sum of their parts, then no single issue can be viewed in isolation. This analysis uses this systemic approach when addressing the problem of cancer in Washington State. By stressing the connections between economy, policy, environment, and public health, innovative solutions can be incorporated. This analysis attempts to bridge the gap between various stakeholders, promote scientific understanding and precaution in policy that internalizes the full costs of cancer.

4.1. The current policy paradigm

The present high rates of cancer in Washington State represent a reinforcing cycle that negatively affects society on all levels of sustainability. This behavior is represented in the causal loop diagram (CLD) 4.1.

CLD 4.1. The current policy paradigm

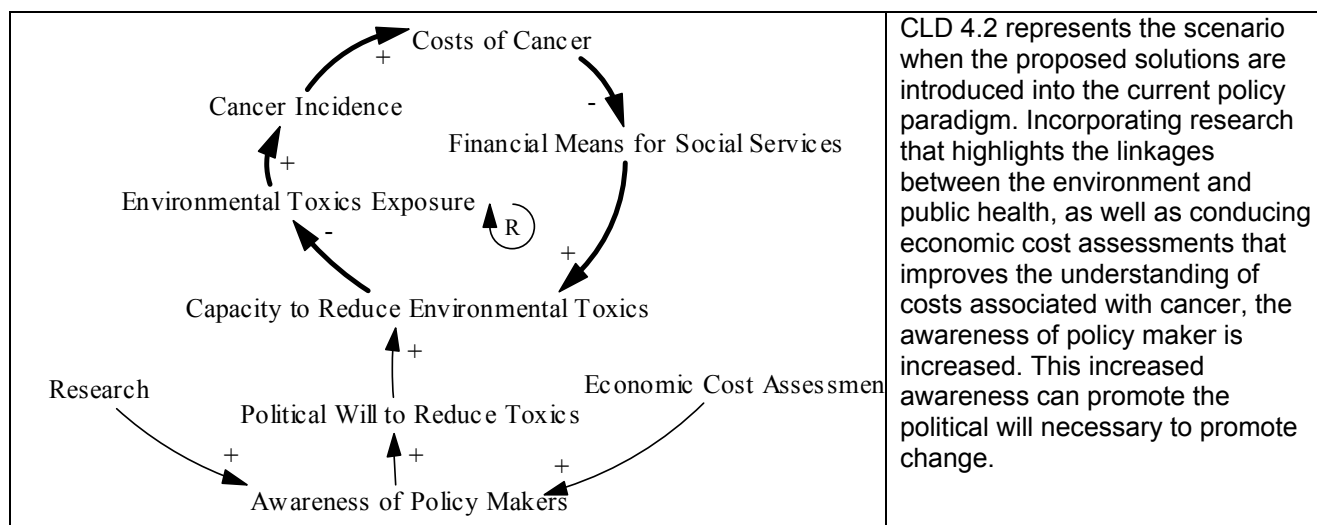


When the current levels of toxics circulate in the environment, detriments to the public's health result in the form of cancer. These effects place a huge economic burden on society. The economic costs compound as incidence rates increase. The more money allocated to the treatment of cancer, fewer resources can be invested into social services and prevention. The cost of cancer also includes the loss of productivity in the work force for the cancer-inflicted. The financial means for overall social services is then diminished. This reduced financial means translates to a very tight budget for policy makers. The capacity to reduce environmental toxics is lessened and the cycle of financial constraints, environmental pollution, and high rates of cancer persist.

4.2. Promoting an alternative approach

This analysis suggests several methods to increase the awareness of policy makers, which will then help break the reinforcing cycle described by CLD 4.1. The definition for the 'capacity to reduce environmental toxics' must be broadened from simply the financial means, but also the political will. Therefore, solutions can originate by increasing the political will of policy makers to reduce environmental toxics. It is acknowledged that political will has a multitude of variables, but such aspects are considered outside the parameters of this study. The solutions proposed in this analysis are twofold. First, increase the awareness of policy makers through research that better establishes the connections between the environment and public health. And second, perform economic cancer cost-assessments to communicate, in monetary terms, the actual costs of cancer. Increasing awareness will increase precautionary measures in the policy framework. Such measures will encourage a positive reinforcing cycle, whereby reducing the amount of environmental toxics exposures, cancer incidence may decline and finances will be less strained. (See CLD 4.2).

CLD 4.2. Alternative policy paradigm



6. Literature Review

This section is a review of the application of the precautionary principle in a variety of contexts. The first objective of this study is to reveal the historical development of the PP. We will begin by establishing a common language and clarify certain fundamental components of this analysis, then move to methods that Washington State is incorporating the PP into toxic chemical regulation.

6.1. Defining key concepts

Enhancing environmental health is a fundamental component toward achieving sustainability and is a crucial premise of this study. Advocating environmental health will improve public health by protecting the natural environment. The definition of environmental health derives from the World Health Organization (2004), “Environmental health comprises those aspects of human health and disease that are determined by factors in the environment. It also refers to the theory and practice of assessing and controlling factors in the environment that can potentially affect health.” Lifestyle choices such as smoking, UV exposure, and obesity are also aspects of environmental health. Health effects include: “both the direct pathological effects of chemicals, radiation and some biological agents, and the effects (often indirect) on health and wellbeing of the broad physical, psychological, social and aesthetic environment which includes housing, urban development, land use and transport” (WHO, 2004).

A second concept that needs defining is environmental contaminants. A broad definition includes chemical substances from human origin in environmental media; this includes air, food, water, soil, the home, and the community (Landrigan et al., 2002: 721). The environmental factors important to this analysis are toxics, such as chemicals, pesticides, and herbicides with persistent, bioaccumulative, and carcinogenic properties. Throughout the rest of this study the terms environmental contaminants, environmental toxics, and toxic chemicals will be used synonymously.

The primary environmental contaminants of focus in this analysis are persistent bioaccumulative toxic chemicals. The Washington State Department of Energy defines PBTs as chemicals and/or pollutants, which persist in the environment without breaking down; accumulate in the environment and build up in the tissues of humans, fish and animals; and are toxic to living organisms including humans (Gallagher, 2000: 5). PBTs can also be transported great distances via wind, water, or in organic tissues and move readily through the different mediums. This term differs from persistent organic pollutants (POPs), due to its inclusion of inorganic compounds such as mercury and lead. The term ‘toxin,’ was utilized in the original Washington State PBT action plans. By definition, ‘toxin’ refers to poisonous substances produced by living cells capable of causing disease. This study regards the word ‘toxin’ as misleading; therefore, throughout this study, ‘toxin’ will be replaced by the term ‘toxic’ (a chemical substance that can cause injury or death).

The first official definition of the precautionary principle originates from the 1992 Rio Declaration, Principle 15 (United Nations, 1992). Later, the 1998 Wingspread Statement on the precautionary principle broadened the Rio Declaration definition by declaring, "When an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically. In this context the proponent of an activity, rather than the public, should bear the burden of proof. The process of applying the precautionary principle must be open, informed and democratic and must include potentially affected parties. It must also involve an examination of the full range of alternatives, including no action" (Ashford et al., 1998). Although various definitions of the precautionary principle exist, all contain three fundamental features: 1) If plausible reason for harm exists, 2) and scientific uncertainty persists, 3) then there is an obligation for precautionary action.

The following features are gaining consensus regarding action plans and are included in the recent San Francisco principle ordinance and the proposed policy framework for the City of Seattle and King County:

- **Right to Know:** Includes shifting the responsibility for providing evidence of causation to proponents of an activity.
- **Alternative Assessment:** To consider all actions which will impact human health and environmental quality. This includes the prospect of no action.
- **Full Cost Accounting:** Considers the full range of costs over short-term and long-term timelines.
- **Participatory Decision Processes:** Incorporate stakeholder participation and democratic decision-making. Decisions must be transparent and based on the best available information.

6.2. The history of the precautionary principle

Though the official definition of the precautionary principle was written in 1992, policies embracing precautionary measures have an extensive history. Successful examples of precautionary application in international, national, and local policy frameworks, present the PP as a pragmatic and contemporary method for environmental health protection, not as an alternative paradigm. The birth of the precautionary principle can be traced back to the German Clean Air Act of 1974. In response to the degradation of the Black Forest, preventative measures were officially implemented despite the existence of scientific uncertainty. The causal agent of forest damage was unknown (acid rain), however, pollution was suspected. Actions were taken that ultimately improved the health of the Black Forest and subsequently the German people (Schettler, Barrett, & Raffensperger, 2002). The PP is directly translated from the German Vorsorgeprinzip: Vorsorge translates to forecaring, and prizip, which means principle. Since its inception in 1974, the PP has slowly generated support internationally. Similar protective policies have been long established in Sweden and other Nordic countries (European Commission, 2001a).

Recent embracement of the precautionary principle is found in the European Union's new approach toward chemical regulation. In 2001, responding to widespread concern regarding the effects of chemicals on human health and the environment, the European Commission had proposed a new regulatory policy called REACH (Registration, Evaluation, and Authorization for Chemicals). This new system is based on the precautionary principle and endorses the ultimate goal of sustainable development. Registration of chemicals exceeding 1 ton will be required by 2012, production exceeding 100 tons by 2008, and 1000 tons by 2005 (Rogers, 2003). The manufacturers will be required to conduct full risk assessments on all chemicals on the market. Industrial usage of chemicals that are carcinogenic, mutagenic, or toxic to the reproduction system, as well as persistent organic pollutants, will necessitate special authorization by regulatory authorities (European Commission, 2001a). The European Commission hopes the REACH program and the PP will protect and benefit human health and the environment. Diverging views exist from industry, trade representatives, and environmental advocates; nonetheless, all stakeholders agree that sustainable development and safe use of chemicals should be regarded as primary objectives of the new European chemical policy (European Commission, 2001b). Table 6.1 provides examples where the international community directly integrated the precautionary principle into official frameworks.

Table 6.1. International examples of the precautionary policies

Issue & Time-frame	Precautionary policies
Ozone Protection, 1987	Montreal Protocol on Substances that Deplete the Ozone Layer- ban of Chlorofluorocarbons
Environmental Health, 1992	UN Rio Declaration on Environment and Development (Agenda 21)
Food Safety, 2000	The Cartagena Protocol on Biosafety- protects citizens on potential hazards of genetically modified organisms
Environmental Health, 2001	The Stockholm Convention on Persistent Organic Pollutants
Environmental Health, 2001	European Union's Registration, Evaluation, and Authorization for Chemicals

Source: Adapted from (European Environmental Agency, 2001; Gilbert et al., 2004).

The PP has been formally part of the American environmental discourse since the Wingspread Statement of 1998. Though not described explicitly as the precautionary principle, the concepts contained in the PP have been interwoven in US policy for many years. Examples of precautionary prevention and connected issues in the United States are listed in Table 6.2.

Table 6.2. Examples of the precautionary policies in the United States

Issue & Time-frame	Precautionary prevention
Food Safety, 1957-96	The Delaney Clause in the Food, Drug, and Cosmetics Act- banned animal carcinogens from the human food supply
Food Safety, 1970s	(BSE) Banned scrapie-infected sheep and goat meat in the animal and human food chain, contributed to avoiding Mad Cow Disease in the US
Chemicals in Water Supply, 1972	The Clean Water Act- established limits and goals to protect water purity and the health of the citizens
Ozone Protection, 1977	(CFCs) Outright Chlorofluorocarbons ban in aerosols, years prior to EU
Public Health, 1972-79	(DES) A ban on the use of DES as a growth promoter in beef, nearly 10 years before the EU ban
Environmental Health, 1990	Pollution Prevention Act- Following the Exxon-Valdez oil spill. Goals to prevent future catastrophes
Public Health, 1996	(Pesticides) Food Quality Protection Act; phase-out of several organophosphate pesticides, and requires pesticides to be proven safe for children usage

Source: Adapted from (European Environmental Agency, 2001; Gilbert et al., 2004).

The most progressive American example of precautionary principle implementation has taken place in the City of San Francisco, California. In March 2003, the City and County of San Francisco had become the first and only municipality to adopt the precautionary principle into its official environmental policy framework. Chapter One of the Environment Code now reads, “All officers, boards, commissions, and departments of the City shall implement the precautionary principle in conducting the City and County’s affairs” (City of San Francisco, 2003: 1). This was a monumental achievement that required the collaboration of the government of San Francisco, the Breast Cancer Fund, the Science and Environmental Health Network, as well as many other stakeholders. The pioneering effort of the City of San Francisco has promoted advocacy for the incorporation of the PP into the environmental policies of other cities. A collaborative effort from ten state and non-governmental organizations has put Seattle and King County on the threshold to become the second municipality to adopt the precautionary principle in the US. Policy decisions will be released by the end of December 2004. The proposed language in the 20 Year Comprehensive Plan Toward a Sustainable Seattle would then read: “Every resident of Seattle/King County has an equal right to a healthy and safe environment. This requires that our air, water, earth and food be of a sufficiently high standard that individuals and communities can live healthy, fulfilling and dignified lives. The duty to enhance, protect and preserve Seattle’s/King County’s environment rests on the shoulders of government, residents, resident groups and businesses alike” (Gilbert et al., 2004: 20). The precautionary principle is a departure from reactionary environmental policies of the past, to modern policies that embrace prevention.

7. Application / Empirical Analysis

This section addresses the second objective of this study; to summarize the available literature concerning incidence, trends, and environmental linkages associated with cancer in WA. The main focus will be on establishing the connections between the environment and public health. The difficulties of proving the linkages between toxic chemicals and cancer will be discussed, levels of unavoidable ambient exposures will be established, as well as addressing the mechanism of cancer formation and specific vulnerabilities in children. The analysis will then focus on levels of specific cancers in WA and highlight research that strengthens the association between environmental quality and cancer incidence.

7.1. Difficulties exist in proving causation

Linking cancer with environmental factors is rarely, if ever, definitive. There are a variety of explanations for why it is difficult to determine the causality of environmentally attributable cancers. First, much scientific knowledge of carcinogenic chemicals is derived from animal studies. Though medical science has a strong grasp on animal physiology and responses to chemicals, risk-assessments should always be taken with caution. Humans may simply respond to chemical exposures differently than test animals. Second, variations in vulnerability to environmental exposures exist amongst individuals. Variations of physiologic and genetic susceptibilities will ultimately determine why one person can smoke a pack of cigarettes a day without repercussion, versus why a young child develops leukemia. Dose doesn't always equal response. Each individual's body responds to foreign chemicals differently, and therefore thresholds of carcinogenesis, due to environmental exposures, are also different. Thirdly, the timing of exposure is very important in cancer formation. Windows of exposure allow vulnerabilities to the formation of cancers; such periods exist from fetal development throughout childhood. Fourth, long latency periods exist from exposure to the time symptoms appear. Cancer often takes decades to develop, and pinpointing the moment of exposure can be highly speculative. Finally, chemical exposures to carcinogens may be cumulative and multi-factorial. Low-level exposure may stem from a variety of sources and can accumulate and aggregate in our bodies over time. This occurs throughout life and usually goes unnoticed. People are continually being exposed to chemicals in much of the food they eat, the air they breathe, and in the water they bathe and drink (Landrigan, et al., 2002).

7.2 Cancer and the environment

Despite existing difficulties in proving the linkage between environmental toxics and cancer etiology, we know that genetic factors can only be attributed to cause 10-20% of cases of chronic disease (Gilbert et al., 2004: 8). Subsequently, this leaves the cause of 80-90% of disease up to speculation. In industrialized counties, breast cancer has increased 1-2% per year for several decades, Non-Hodgkins lymphoma has increased 3-4% per year throughout the world, and cancers of the central nervous system has increased by 50-100% (Solomon et al., 2000). Many medical experts believe that chemicals circulating in the ambient environment greatly contribute to the increasing incidence of cancers, as well as non-cancerous ailments like attention deficit disorder, asthma, Parkinson's Disease, declining fertility rates, and birth defects. Research has established over 200 health outcomes linked to chemical exposures (CHE, 2004). However, the concentration, duration, and timing of exposures determine the degree of risk to the individual. Certain linkages have been well established; yet, achieving scientific

certainty may never occur. Lifestyle factors affecting the susceptibility of cancer such as UV exposure, diet, exercise, smoking, nutrition, and alcohol consumption have received a great deal of attention. We know that even low levels of exposure can increase your risk for cancer, such as inhalation of second-hand smoke. Conversely, our understanding of the overall affect of ambient chemical exposures on the health of the populous is quite meager and needs further research. Studies have determined the risk of cancer formation, by combining smoking with asbestos and arsenic exposures, is much greater than their individual effects (ACS, 2004). However, the synergetic effects of other chemical combinations are poorly understood and need further investigation.

Despite varying degrees to which chemicals can cause cancer, certain carcinogenesis mechanisms have been well documented. In *Pesticides and Human Health*, Solomon, Ogunseitan and Kirsch (2000: 21) state that the current understanding of cancer formation favors that even a small dose of a genotoxic agent can convert a normal cell cancerous, and the process that carcinogenic compounds attack cells in the body is known by three basic mechanisms:

- Genotoxic effects: A chemical agent that damages cellular DNA, resulting in mutations or cancer.
- Promotion: Stimulation of the progress or growth of a tumor following initiation. This process includes endocrine effects that may stimulate otherwise inactive but hormonally sensitive cells to carcinogenesis.
- Immunotoxic effects: Disturbing the body's normal cancer surveillance mechanisms.

7.3. The body burden of chemicals

Some chemicals, with persistent bioaccumulative properties, known as PBTs, can accumulate in our bodies and their levels of concentration will increase with time. Many of these chemicals are identified carcinogens. Biomonitoring studies have concluded that all US citizens have a "body burden," which means that potentially toxic chemicals are stored in our blood, fat, semen, urine, breath, and breast milk (Northwest Environment Watch, 2004). This is not surprising to many scientists considering over 15,000 chemicals are circulating the environment that are produced in quantities greater than one million pounds annually. These chemicals have been measured in our drinking water, air, and food supply, and contained in many consumer products. As a result, the adipose tissue of the average American contains 700 contaminants. This includes 190 synthetic pesticides called organochlorines (Thornton et al., 2002). A fundamental relationship exists between the body burden and the precautionary principle. The necessary lesson to be learned by the body burden is that chemicals are indeed accruing in our bodies, but their actual effects are poorly understood. Does the daily accumulation of chemicals circulating in our ambient environment cause cancer? Scientific uncertainty remains regarding the actual effects of these bioaccumulative compounds, but that should not be grounds for inaction. (Thornton et al., 2002)

One example of a body burden chemical is an organochlorine pesticide called dichloro diphenyl trichloroethane, commonly referred to as DDT. This compound is a well-known carcinogenic

pesticide. Other chemicals confirmed to be carcinogenic include: benzene, asbestos, vinyl chloride, arsenic, and aflatoxin. Due to reliable animal studies, other chemicals with strong evidence to cause cancer include: chloroform, formaldehyde, polychlorinated biphenyls (PCBs) and polycyclic aromatic hydrocarbons (PAHs) (ACS, 2004). A pilot study, conducted by Thornton, McCally, and Houlihan (2002), measured 150 chemicals in the blood and urine of nine US citizens with no abnormal exposures. They concluded that the body burden of phthalate plasticizers, dioxins, furans, PCBs, metals, and pesticides is not isolated to acute exposure but occurs in all citizens. This in an important point worth reiteration: every person is exposed to potentially toxic chemicals, regardless of location, occupation, or lifestyle.

Washington State is certainly not exempt from the body burden phenomenon. In fact, a recent breast milk study conducted by the Northwest Environmental Watch (2004) determined that Pacific Northwest residents have 20 to 40 times the concentration of polybrominated diphenyl ethers (PBDEs) than levels measured in Europe and Japan. This is not surprising, since the United States is the largest producer and consumer of PBDEs in the world. PBDEs are a class of brominated flame-retardants applied to consumer products such as furniture, textiles, plastics, foams and electronics. PBDE variations have been linked to developmental and sexual development, and thyroid dysfunction in test animals (Peele, 2004: 20). Because PBDE is structurally similar to the know carcinogen group of PCBs, it is considered a suspected carcinogen. Further research is required to indicate its degree of carcinogenicity to the human population. As Table 7.1 demonstrates, the Washington median concentration of PBDEs was 50 parts per billion, a stark contrast to Japan's 1.3 and Sweden's 2.1 parts per billion.

Table 7.1. Comparison of breast milk PBDE levels

	United States (2002-03)	Washington (2003)	Texas (2002)	Canada (2001-02)	Sweden (2001)	Japan (2000)
Median PBDE level (parts per billion)	58	50	34	25	2.1	1.3

Source: Table compiled from Northwest Environmental Watch (2004) data

A prime example of a country using precaution in its policy regulating PBDEs is found in Sweden. The success of its measures is evident by the low levels of PBDEs found in the breast milk of Swedish mothers. Following initial indications of the potential adverse effects of this compound, Sweden implemented precautionary measures to phase-out usage of PBDEs prior to the bans imposed by the EU. In May 2004, Sweden commissioned the national chemicals inspectorate to consider a ban on all brominated flame-retardants (DOE, 2004). Companies such as IKEA and Volvo found safer alternatives and the PBDE breast milk concentrations began to decline (Gruvenius & Norén, 2001). The current low levels in Sweden are testimony to the success of an alternative and precautionary approach and should be used as a template in the US and other countries.

7.4. Cancer and children

Children are a subpopulation that could benefit from precautionary approaches manifested in better chemical regulation. The US Environmental Protection Agency (1998) states that 8,000 children under the age of 15 in the US are diagnosed with cancer each year and is the fourth leading cause of death. These children are highly susceptible and vulnerable to toxic chemicals in the environment. The bodies of infants and children are rapidly changing, their bodies are growing, organs developing, and immunities are forming to combat disease. More specifically, Gordon (2004: 14) has identified five reasons why children are more susceptible to cancer:

1. Cell division occurs more often, which increases the opportunity of genetic mutations in the DNA. Less time is available to repair the miscodings of DNA cells.
2. DNA repair enzymes do not exist in embryonic cells, such as in the brain.
3. The immune system of children is not yet fully functional.
4. Periods of sensitivity exist because “hormonal systems operate at different levels during different life stages.”
5. A linkage exists between developmental abnormalities and predisposition to cancer.

It is also known that children eat, drink, and breathe more per unit body weight than adults, and therefore, their exposures to ambient carcinogens is higher. This includes highly carcinogenic PAHs found in automobile exhaust, and the multitude of chemicals inhaled in second-hand smoke. In conjunction with the previously mentioned biological susceptibilities and higher physical intake, dermal and oral exposure routes are disproportionately higher due to childhood teething and playing on the floor and outside. (Gordon, 2004: 11-26)

Some linkages between childhood cancer and the environment have been well established. For example, the synthetic estrogen diethylstilbestrol (DES), given to mothers to promote healthy pregnancies, has been proven to cause rare vaginal cancers in their daughters (Colborn et al., 1997). Once the causal relationship was established, the usage of DES was halted. There is strong evidence linking childhood leukemia and brain cancer with ionizing radiation exposure (Solomon, 2003a; National Cancer Institute, 1999). However, other than exposure to ionizing radiation and genetic susceptibility in children, information is scattered regarding factors contributing to cancer formation. Research exists linking a variety of exposures to pediatric malignancies. Such associations include parental occupational exposures to chemicals and solvents such as benzene, pesticides, motor vehicle exhaust, and electromagnetic fields (US Environmental Protection Agency, 2004). Note that clear and definitive linkages between environmental toxics childhood cancer is very rare, and that research and data is always limited.

In 2002, the US Environmental Protection Agency funded Dr. Richard Fenske’s research to better establish the degree that the chemical body burden in the average child in Washington State is the direct result of chemical pesticide exposure. (The majority of pesticide exposure research has been conducted with at-risk groups such as agricultural workers). Specifically, the urine of Seattle area children was analyzed for the presence of dialkylphosphate metabolites. The study determined that 92% of the children tested were positive for at least one metabolite of an organophosphate pesticide (Fenske, 2002). This study determined that the primary route of exposure was via pesticide application

in their home gardens. Only one child lacked the presence of pesticides in their urine; their parents used organic pesticides in the garden.

The conclusions of Dr. Fenske's research are applicable nationally. In 2004, the Pesticide Action Network North America (PANNA), in cooperation with partner groups in twenty cities, conducted a nationwide assessment of pesticides in blood and urine. Analyzing samples from 9,282 people nationwide, pesticides were found in 100% of the samples, with an average person having 13 of 23 pesticides under consideration (Schafer et al., 2004).

7.5. Overview of cancer in Washington State

With the knowledge that carcinogenic chemicals are being stored and accumulating in our bodies, it can be reasonably suspected that their contribution is significant to the overall rates of cancer. According to the Washington State Department of Health (2004b), cancer causes a quarter of all deaths statewide. Cancer is second leading overall reason for death, attributable to 10,656 people in 2000. From 1995 to 2000, the general trend indicates a 14% increase where breast, prostate, lung, colorectal, and melanoma of the skin comprise 62% total reported cancers. While the news that increasing incidence levels is disheartening, from 1980 to 2001, the overall age-adjusted mortality trends are decreasing. This is true of all cancers with the only exception of female lung cancer. In 2000, the five leading causes of cancer mortality were: lung and bronchus (3,100 deaths), colorectal (911), female breast (747), pancreatic (591), and prostate (574).

Of the five most common types of cancer in Washington State, three have strong linkages with environmental toxic chemicals: breast, lung and melanoma of the skin (CHE, 2004):

1. **Female breast cancer:** 5,577 new cases (reported in 2001)
2. Prostate cancer: 4,433 new cases
3. **Lung cancer:** 3,864 new cases
4. Colorectal: 2,937 new cases
5. **Melanoma of the skin:** 2,184 new cases

7.6. Cancer and environmental linkages

Due to high incidence nationally and in WA, research linking cancer of the breast, lung, melanoma of the skin, bladder, and Non-Hodgkins lymphoma will be discussed. This study does not attempt to provide an exhaustive discussion of studies with linking cancers with environmental factors. (For a comprehensive list of cancers and associations with environmental toxics, see Appendix Table 12.2). Greater detail will be given to childhood leukemia and CNS cancer due to their inclusion in the economic cost assessment, issues of environmental equity, and the apparent high vulnerability of this subpopulation.

I. Breast cancer

We begin by describing the most widespread cancer in Washington State, breast cancer. Breast cancer is the most common cancer for females in the world. The incidence of breast cancer in industrialized

countries has increased 1-2% per year, with a steep increase in the 1980's and a relative stabilization in the 1990s (Solomon, 2003b). This trend is also apparent in Washington State. Assuming rates remain the same, a female child born today in WA has a 1 in 8 (or 12.5%) chance of being diagnosed with breast cancer at some point in her lifetime (DOH, 2001b). Only a small fraction of carcinogenesis is attributable to genetics, however, the contribution of environmental factors is not fully understood. The best-established association is lifelong exposure to estrogen, amplified by early menarche, never having given birth, and late menopause. Ionizing radiation, alcohol, and exposure to synthetic estrogens are also known to contribute to breast cancer formation. Some research supports diet, electromagnetic radiation, pesticides, tobacco smoke, and unusual light cycles having possible causal associations. Overall, environmental agents are attributable for causing approximately 70% of breast cancer. (DeBruin & Josephy, 2002)

II. Lung cancer

The Washington State Department of Health (2001b) reports smoking as the cause of an estimated 85% of lung cancer deaths, and the rate for males is 1.5 times that of females. The WA rates are slightly lower than the United States average. Nationally, smoking causes 90% of all lung cancer and one third of all cancers (Solomon et al., 2000). WA mortality trends indicate a 3.2% decline in the male population since 1993; unfortunately, women's mortality rates have increased 1.5% per year since 1990 (DOH, 2001b). Basing conclusions on epidemiologic studies, Gottshall (2002) states that 15% of lung cancers in men and 5% in women are caused by occupational exposures to chemicals such as arsenic, asbestos, chloromethyl ethers, chromium, polycyclic hydrocarbons, and radon. (See Table 7.2 for extensive list). One study determined that an occupation as a painter would increase your chances of lung cancer by 40% (Lynge, Anttila, & Hemminki, 1997).

III. Melanoma of the skin

From 1992-2001, the incidence of melanoma of the skin has shown a slight increase in WA. Exposure to ultraviolet radiation is the only strong association to melanoma of the skin. Some research exists linking pesticides and other chemical to occupational exposures such as farming; however, data is inconclusive. UV radiation most likely has greater influence on cancer formation than dermal, inhaled or oral pesticide exposures. (Spiewak, 2001)

IV. Bladder cancer

There is no temporal increase or decrease of bladder cancer incidence in WA. Notably, rates are four times higher in the male population than females (DOH, 2001b). Cancer of the bladder has been linked to a variety of toxic substances, although research is extremely limited. A German literature review concluded that there is a strong causal relationship between bladder cancer and aromatic amines. By comparing two European studies with a Chinese study, they determined there are differences in susceptibilities between racial populations (Igolka, Prior, Blaszkewicz, & Bolt, 2002). A Nebraska study strengthens the associations between chemical exposure and bladder cancer as well as diesel exhaust, chlorination by-products and arsenic in drinking water (Cohen, Shirai, & Steineck, 2000). McDuffie (1994) stated that increased incidence of bladder cancer in female farm workers can be attributed to pesticide exposure.

V. Non-Hodgkins lymphoma

There has been a 3-4% increase per year of Non-Hodgkins lymphoma (NHL) worldwide, with some research indicating a 4-8% yearly rise (Solomon et al., 2000). Trends in Washington State show a similar escalation in incidence. Currently, dioxins are the only toxic having strong associations with NHL formation. This was confirmed by a 20-year retrospective mortality study in Seveso, Italy, where the rates of NHL to those heavily exposed to dioxin nearly quadrupled (Bertazzi et al., 2001). Some research exists linking pesticide exposure to NHL carcinogenesis (Hardell, Eriksson, & Nordstrom, 2002). There is evidence of increased risk when exposed to trichloroethylene (Lyng, Anttila, & Hemminki, 1997). For complete list of chemicals associated the etiology of Non-Hodgkins lymphoma, refer to Table 7.2.

VI. Childhood leukemias

According to the Collaborative on Health and the Environment (2004), there is enough research to conclude the following etiologic associations:

- Strong association: benzene, ionizing radiation
- Good association: pesticides, metal dusts, chlorinated solvents, carbon tetrachloride, and trichloroethylene (TCE)
- Limited or conflicting data: electromagnetic fields, hazardous air pollutants/vehicle exhaust, insecticides (Chlordane, Dichlorvos, Propoxur), radon

Incidence

Leukemia has the highest incidence rates of cancer for the pediatric population in the State of Washington. The WSCR reported in 2001 that 107 new cases of Leukemia were identified. This represents 35% of childhood cancers for citizens under the age of nineteen. The incidence rate of 4.2 per 100,000 is slightly higher than national incidence rates. Comparing the percentage of leukemia according to age against all childhood cancers, it comprises 42% for 0-4, 46% for 4-9, 41% for 10-14, and then decreases to 21% for the 15-19 age groups. The total incidence rates are particularly higher in male population than female. Leukemia accounts for 62% of cancer incidence in males ages 0-4 and over 50% for ages 5-9 and 10-14. The counties of Adams (22), Grant (19), and Jefferson (18) have the highest incidence rates in Washington State (1999-2001). The counties of Franklin (6), Asotin (9), and Benton (9) have the lowest incidence rates in Washington State (WSDH, 2001a). Countywide variations exist; however, conclusions cannot be made regarding elevated or depleted incidence rates due to location. (WSDH, 2001b)

Trends

The national incidence of childhood leukemia has shown an increase over the last two decades, with a 0.9% increase yearly from 1977 to 1995 (National Cancer Institute, 1999). The trend is most prevalent in the Acute Lymphoid Leukemia variation of the cancer. National trends are reflected by Washington State Cancer Registry data (WSDH, 2001b).

Ionizing radiation

Medical research has established an increased risk (1.5 times) of leukemia formation from in utero x-ray exposure (National Cancer Institute, 1999). The association has been understood for a many years and has contributed to reduced usage of prenatal x-rays and better precautionary measures.

Pesticides

A study conducted in the Northern California Childhood by, Ma et al., (2002), concluded that there is a strong association between indoor pesticides and childhood leukemia and that risk levels increase with increasing exposures. This research highlights that households, which use professional indoor pesticide services, have double the odds of their children developing leukemia. It also supports the associations between cancer development and in utero exposures.

Additional exposure linkages

A literature review by Colt & Blair (1998) of forty-eight published studies supports the association between parental exposure to environmental toxics and childhood leukemia. Their research helps strengthen the relationship between benzene exposure and cancer. Colt and Blair conclude that the strongest connection between childhood leukemia and environment is paternal exposure to solvents, paints, and employment in motor vehicle-related occupations.

VII. Childhood Brain and CNS Cancer

According to the Collaborative on Health and the Environment (2004), there is enough research to conclude the following etiologic associations:

- Strong association: ionizing radiation
- Good association: organic solvents, pesticides (DDVP, Lindane)
- Limited or conflicting data: aromatic amines, chlorophenates, dyes, electromagnetic fields, N-nitrosoamines, pesticides: Carbaryl, Diazinon, and phenoxyacetic herbicides

Incidence

Pediatric malignancies of the brain (or brain cancer), is the second leading cause of death for children under twenty in Washington State. Brain cancer is attributed to 25% of the diagnosed childhood cancer. It has also been confirmed that boys have higher rates of incidence than girls. The rate of brain cancer in the 0-19 age group in Washington State is 3.1 per 100,000. (DOH, 2004a)

Trends

There is a gradual and steady increase in the incidence of brain cancer in children in the United States. Smith, Freidlin and Simon (1998) reported a 35% increase from 1973 to 1994, which translates to increases of 1.8% per year. In the US and Europe, rates of adult cancers of the brain show a similar increase in trends, up to 40% during the last 20 years (Solomon, 2003a). Additional estimates of incidence increase have been as high as 50-100%, with at-risk occupations including farming, orchard work, pesticide application, and golf-course superintendence (Solomon et al., 2000). Only a tiny fraction of the rise in incidence can be attributed to the increasing sophistication of detection, such as magnetic resonance imaging (Gordon, 2004: 11). Therefore, understanding environmental linkages may help shed some light on the rise of childhood brain cancer rates.

Ionizing radiation

Ionizing radiation, such as radon and x-rays, is the best-established environmental contributor to the formation of childhood brain cancer (Doll & Wakeford, 1997; Solomon, 2003a). Doll and Wakeford (1997) stated that there may be no threshold in which ionizing radiation has no effect on fetal development and exposures are particularly influential in carcinogenesis during the last trimester. There is also a link between parental occupational exposure and cancer formation of their offspring. It should be noted that radiation is attributed to a tiny fraction of all pediatric brain cancer cases.

Pesticides

In 1998, the National Cancer Institute conducted a large-scale literature review of case-control studies and case reports, which strengthened the associations between pesticide exposure and malignancies of the brain. This study had concluded that childhood exposures to pesticides are attributed to a variety of sources including, homes, schools, and other buildings; outdoors exposures from lawns and gardens; ingested exposure from food and contaminated drinking water; and exposure through inhalation from agricultural application drift, overspray, or off-gassing (Zahm & Ward, 1998). Their research had also strengthened the association between cancer and gestation and even preconception exposures. Five other studies exist, supporting the conclusions of Zahm and Ward, which resulted in a slight positive correlation between pesticides and childhood brain cancer (Solomon, 2003a).

A population-based control study was conducted in Los Angeles with 224 subjects and 218 controls. The purpose of this analysis was to investigate the risk of household pesticide use from pregnancy to diagnosis. The results of this study concluded a 70% increase in brain cancer formation in homes that used flea and tick pesticides. Increased cancer incidence was most notable in infants. The study only linked sprays/foggers with elevated risk levels. There was also an increase in cancer rates among pets in the same homes. (Pogoda & Preston-Martin, 1997)

A 1994 case-control study had specifically sited the pesticide, Lindane, with a strong association with brain cancer formation. This research also concluded a significant association with use of flea collars containing dichlorovos (DDVP) on pets, citing that usage results in five times a greater likelihood to develop brain cancer. The herbicide, diazinon, used in home gardens was also determined to be carcinogenic. (Davis et al., 1994)

Additional exposure linkages

There is strong evidence that associates pediatric cancers of the central nervous system to parental exposures to paints (Colt & Blair, 1998). The connection between chemical solvent, parental occupational exposures, and pediatric brain cancer was strengthened by studies in California and Washington State (McKean-Cawdin et al., 1998). Parents of 540 cases were interviewed from 1984-1991. Strong associations were found with childhood cancer rates with parents who worked in the chemical industry or fathers who worked as electrical workers.

There is some research that indicates an inverse correlation between glioma formations and those previously exposed to viruses such as chicken pox or allergies and incidence rates (Mensch et al, 2002). This implies that the immune system may play a role in protecting the body from brain tumors.

Table 7.2 is an abbreviated list of 15 cancers with strong or good evidence linking environmental toxics to carcinogenesis. Chemicals listed in bold are considered persistent and bioaccumulative and are included on the Washington State's PBT working list. The cancer types are listed from highest incidence to the lowest.

Table 7.2. List of cancers linked to chemical exposures and strength of evidence

Cancer Type	Toxics- Strength of Evidence	
	Strong	Good
Breast	Estrogens/DES, Ethanol, Ionizing radiation	Aromatic amines (B-naphthylamine and benzidine), Ethylene oxide, PAHs , Tobacco smoke
Lung	Aluminum, Arsenic (including arsenical pesticides); Asbestos, Attapulgit, Benzo(a)pyrene (PAH's) , Beryllium, Cadmium , Chloromethyl ethers, Chromium, Coal tars, Diesel engine exhaust, Ionizing radiation, Mineral oils, Mustard gas, Nickel, Radon, Silica, Soots, Tobacco smoke, Uranium	Acid aerosols, Acrylonitrile, Aromatic amines, Chlorophenols, Coal dust, Copper, Dimethyl sulfate, Formaldehyde, Solvents, Nitrosamines (NNK); PAHs (Benz(a)anthracene, Benzo(a)pyrene , Dibenz(a,h) anthracene
Melanoma	UV radiation	
Bladder	Aromatic amines (4-Aminobiphenyl, Auramine, B-Naphthalamine, Benzidine, MOCA), Benzidine-derived dyes, Chlordimeform (and its metabolite 4-COT), Coal tar, Nitrobiphenyl, Tobacco smoke, trihalomethanes (disinfection byproducts)	Arsenic, Benzo(a)pyrene (PAH's) , Chlornaphazine, Chlorphenol, Ionizing radiation, Methylene dianiline, Organic solvents, o-Toluidines
Non-Hodgkin's Lymphoma	Dioxins (TCDD)	Aromatic amines, Benzene, 1,3 Butadiene, Chlorophenols, Creosote, Ionizing radiation; Organic solvents: carbon disulfide, carbon tetrachloride, trichloroethylene (TCE), tetrachloroethylene (PCE); PCBs , Pesticides: Carbamates (Carbaryl), Dicamba, Fungicides (Captan), Organophosphates (dichlorovos, malathion), DDT , Phenoxyacetic acid herbicides (2,4-D, MCPA, mecoprop)
Adult-onset Leukemias	Benzene, Ethylene oxide, Ionizing radiation	Arsenic, Aromatic amines, 1,3-Butadiene, Carbon disulfide, Dioxins/TCDD , Chlorinated solvents [carbon tetrachloride, 1,2-dichloroethane]; Pesticides (Alachlor, DDT , Phenoxyacetic herbicides); Tobacco smoke
Kidney	Tobacco smoke	Arsenic, Asbestos, Benzene, Coal tar/soot/pitch/asphalt/creosote, Copper sulfate, PAHs , Pentachlorophenol, Pesticides, Trichloroethylene (TCE)
Oral	Tobacco	Ethanol, Nitrosamines (NNN and NNK)
Thyroid	Ionizing radiation	Ethylene thiourea (ETU)
Cervical	DES, Tobacco smoke	Organic solvents
Brain - adult	Ionizing radiation	Chromium, Methylene chloride
Esophageal	Ethanol, Tobacco smoke	Nitrosamines; Organic solvents [Tetrachloroethylene (PCE)]; PAHs , Silica
Multiple Myeloma	Benzene, Ionizing radiation	Chlorinated dioxins/TCDD ; Pesticides (arsenical pesticides, phenoxyacetic herbicides)
Laryngeal	Ethanol, PAHs , Sulfuric acid, Tobacco smoke	Diethyl sulfate, Leather dust, Mustard gas, Nickel, Wood dust
Liver	Aflatoxic B1, Androgens, Ethanol, Hydrocarbons	Arsenical pesticides, Dimethylnitrosamine, PCBs , Thorium dioxide (Thorostat), Trichloroethylene (TCE), Vinyl chloride

Source: Adapted from the Collaborative on Health and the Environment Database, <http://www.protectingourhealth.org/>

8. Results

8.1. Comparing adult cancers statistics

Table 8.1 illustrates a comparison of cancer incidences for Washington State to the national averages. The list represents the top fifteen cancer variations in WA with strong linkages to environmental toxics. The results of the comparison indicate that WA has higher levels of cancer in 11 of the top 12 categories than the national average. Of the nine categories with the highest incidence rates, Washington State has higher levels in all. Only cervical cancer (ranked 10th overall) and liver cancer (ranked 15th overall) have rates lower than the national average. Several counties can claim the highest incidence rates in two categories: Pend Oreille (melanoma and bladder), Mason (lung and liver), and Adams (cervical and adult-onset leukemia). When comparing cancer by county there is no definitive evidence that cancer is more prevalent in one part of the state over the next.

Table 8.1. Incidences of adult cancers in Washington State and United States

Cancer Type (2001)	Incidence in Washington*	Incidence in United States	Comparison	County with Highest Incidence
1 Breast cancer	181.7	163.7	Higher	San Juan
2 Lung cancer	70.1	59.0	Higher	Mason
3 Melanoma	37.6	27.1	Higher	Pend Oreille
4 Bladder cancer	23.7	20.3	Higher	Pend Oreille
5 Lymphoma - non-Hodgkin's	21.1	18.5	Higher	Walla Walla
6 Adult-onset Leukemias	13.4	11.5	Higher	Adams
7 Renal (kidney) cancer	13.3	11.7	Higher	Wahkiakum
8 Oral cancer	11.8	10.7	Higher	Skamania
9 Thyroid cancer	8.5	7.4	Higher	Lincoln
10 Cervical cancer	7.1	8.7	Lower	Adams
11 Brain cancer - adult	6.6	5.7	Higher	Kittitas
12 Esophageal cancer	5.8	4.5	Higher	Asotin
13 Multiple Myeloma	5.5	5.4	Similar	Okanpgam
14 Laryngeal cancer	4.1	4.0	Similar	Pacific
15 Hepatocellular cancer (Liver cancer)	4.1	4.9	Lower	Mason

*Data compiled from the Washington State Department of Health. (2001). *Cancer by Site*. Olympia, WA: Office of Wellness and Prevention and office of Epidemiology. Age-adjusted per 100,000 (adjusted to US standard population).

Table 8.2 shows a comparison of childhood leukemia and brain cancer with national rates. These two cancer types are attributable for 61% of the pediatric cancer incidence in Washington State (WSDH, 2004a). Childhood brain cancer incidence is higher (3.1 per 100,000) than the national average (2.7 per 100,000). Childhood leukemia (4.2 per 100,000) is also higher in WA than national incidence (4.1 per 100,000); however, the rate is only slightly higher (0.1 per 100,000). For both brain cancer and leukemia, males have considerably higher incidence levels.

Table 8.2. Incidence of pediatric leukemia and brain cancer in WA and US

	WA Incidence 0-19 years (2001)	National Incidence 0-19 years*	Comparison
Brain Cancer	3.1	2.7	Higher
Leukemia	4.2	4.1	Similar

*1990-1995 Statistics taken from National Cancer Institute SEER data age-adjusted per 100,000, incidence rates for specific ICD-O codes, age <20, all races, both sexes, www.cancer.gov

Table 8.3 presents Washington State’s national ranking of the five cancers with the highest incidence rates that have environmental associations. Washington females have the uppermost incidence rates in the United States for cancer of the breast. (Refer to table 8.1 for WA and national incidence rates). WA females also have the peak rates of melanoma of the skin, while the males are also quite high, with a national ranking of 5th overall (statistical average of 1993-1997 incidence rates). Washington ranks very high in Non-Hodgkin’s lymphoma, where the males rank 4th and females are 6th in the nation. Males have elevated rates of bladder cancer, 8th nationally, while women are near the US mean, at 24th. Finally, females have the 9th highest rates for cancer of the lung. Males are below the national average at 30th overall.

Table 8.3. Washington’s national ranking of the top five adult cancers with environmental associations

Cancer Type	Breast Cancer	Lung	Melanoma	Bladder	Non-Hodgkin’s Lymphoma
National Ranking*	1st Female	9 th Female 30 th Male	1st Female 5 th Male	24 th Female 8 th Male	6 th Female 4 th Male

Source: Adapted from American Cancer Society, Inc. (2004). Cancer facts and figures 2004

* Ranking of 50 US States; 1st is highest incidence, 50th is lowest

8.2. Cost Estimates of Cancer in Washington State

Landrigan et al. had calculated the average annual charges per child in 1998 USD to be \$35,000 for physician services, \$189,600 for inpatient services, \$20,400 for out patient services, and \$263,200 for laboratory services. Survivors of childhood cancers have increased odds of developing cancer in the future. Landrigan et al. estimates the costs of the secondary cancer to be the same and then uses a 3% discount rate to adjust for future costs. An additional \$74,000 is added to the total accounting for lost parental wages based on five days taken off per seven days their child is in the hospital. A 2.8-point decline in IQ can be attributed to whole brain irradiation (Siber et al., 1993). Factoring loss of future income attributable to IQ loss, an estimated at \$60,500 is added to the total per child. The total cost per case for pediatric cancer is determined to be \$623,000.

The total cost per case was converted from 1998 to 2004 dollar amounts. The current dollar value for care and treatment cost per child is \$724,000. Factoring the sample population of 308 children diagnosed with cancer, the best estimates of 2004 total cost of pediatric cancer in WA is estimated at \$222.9 million. Table 8.4 utilizes the Landrigan et al. estimates then divides the results into two categories: including and excluding loss of projected income. This framework was also applied in Ackerman and Massey (2003) Massachusetts specific study. After factoring the EAF into the total cost, we then get the range of costs in WA of cancers with environmental origin; (\$4.5 million to \$200.6 million) including projected loss of income and (\$3.7 million to \$164 million) excluding the losses. To provide consistency with the Landrigan et al. study, 5% EAF estimates will be regarded as most accurate. Therefore, the most reliable cost estimates of pediatric cancers with environmental origin in Washington State are \$11.2 million or \$9.1 million excluding loss of projected income.

Table 8.4. Estimated costs of cancer in Washington State

	Number of Children Diagnosed (2001)	Care and treatment cost per child (2004\$)	Total Costs (2004\$) in millions	EAF	Cost of EAF in millions
Including loss of projected income	308	\$724,000	\$222.9	2%	\$ 4.5
				5%	\$ 11.2
				10%	\$ 22.3
				80%	\$ 178.4
				90%	\$ 200.6
Excluding loss of projected income*	308	\$592,000	\$182.3	2%	\$ 3.7
				5%	\$ 9.1
				10%	\$ 18.2
				80%	\$ 145.8
				90%	\$ 164.0

* Does not include corresponding loss of lifetime earnings due to cranial irradiation caused loss of IQ, lost parental wages, costs of treating a second primary cancer.

9. Discussion / Analysis

Research: Better understanding of connections between environment and public health increases the awareness of policy makers

This study attempts to demonstrate the need for the precautionary principle by means of a public health argument. We began by presenting an overview of cancer in Washington State; this established the levels of mortality, incidence, and trends. However, such statistics have a diminished relevance unless a comparison is made. When comparing to national cancer incidences, we found that WA has exceptionally high rates of environmentally attributable cancers. We examined fifteen adult cancer variations, and eleven categories are higher than the national average. Most notably, the nine cancers with the highest incidence are above the US average; and in two categories, WA has the nation's most elevated rates. Next, a supplemental comparison was made for childhood cancers. This study recognizes the need for an isolated focus of pediatric cancers on grounds of environmental equity and the high susceptibility of this group. Like adults, Washington children have rates above the US average. Furthermore, childhood leukemia and brain cancer incidence rates are rising yearly.

Efforts were then made to strengthen the associations between environmental toxic substances and the formation of cancer. A large portion of the analysis was focused on evidences for environmental linkages, especially pediatric brain cancer and leukemia. The critical point of the section is to demonstrate the vast array of chemicals that contribute to carcinogenesis of various types. The persistent bioaccumulative toxic substances have been listed. These dangerous compounds are currently receiving a lot of attention in Washington State policy discourse. However, chemicals without persistent bioaccumulative properties were included in this analysis. This study advocates for further research, to better understanding of connections between environment and public health; this includes all chemicals, not just PBTs. Researching specific compounds is crucial in establishing the existence of plausible reason for harm to human health and environment, however, it is more important if this research is applied to policies that end current levels of exposure and prevent future exposures.

Economic cost assessment: Better understanding of cost increases the awareness of policy makers

A crucial objective of this analysis, as indicated in Section 2, is to establish economic costs of pediatric disease attributable to environmental toxic substances in Washington State. By doing such, our understanding of the economic burden of toxic chemicals will become clearer. We estimate that costs of pediatric cancers with environmental origin to be \$11.2 million, with a plausible range from \$4.5 million to \$200.6 million per year, depending on which environmentally attributable fraction is utilized. The EAF definition derives from the Landrigan et al., (2002) study; the percentage of a particular disease category that would be eliminated if environmental risk factors were reduced to their lowest possible levels. When the contributions of environmental toxic substances are documented in the overall costs of health care in Washington State, then funds can be appropriated to stop current and preventing future exposures.

Critics have expressed apprehension on grounds that the PP will be economically troublesome. Such feedback was received from industry when the DOE released its strategy to reduce PBTs in

Washington State. One comment cited a 500-600% increase in cost of products with zero impacts and concluded that environmental compliance is too costly for industry (Gallagher, 2001: 44). Ackerman and Massey (2002) have refuted this notion claiming that precautionary policies can stimulate technological innovation and actual costs are almost never as high as industrial estimates. They cite an example from the Bureau of Labor Statistics, which indicates that only 0.1% of total costs are attributable to environmental restrictions. Furthermore, imposing environmental regulations have proved to be incentive for technological innovation by forcing companies to become more efficient in their processes. Policy makers are naturally cautious because they must juggle the wants of various stakeholders such as industry, government, farmers, the private sector, and environmental advocates. Yet, proponents say that it will encourage superior technologies, which will ultimately prove to be cheaper and better for society. Countries who have implemented precautionary measures have not witnessed a slump in their economy, but an increase in exportation of environmentally friendly technologies (Environmental Health Alliance, 2004).

Some opponents of the precautionary principle state that such measures will cost jobs and hurt the economy (Gallagher, 2001: 44). In response, Ackerman and Massey (2002) claim that workers will actually profit by precautionary actions. More jobs are created by environmental protection than previously existed. Local jobs in trades such as building, installation, operating, inspecting pollution control systems as well as jobs created at recycling plants will invigorate, not impede the workforce. Innovative and sustainable technologies will prove to be more efficient and replace industries that are incompliant with natural laws. Concepts of industrial symbiosis, localization, and green technologies will provide jobs and endorse economic growth while adhering to the sustainable foundations of the precautionary principle.

Another critique states that the PP will make Washington industries less competitive in the national and world market, fearing that the high costs of compliance will force companies to move to less regulated countries. Contrary to this belief, research has indicated that environmental protection laws do not hamper the competitiveness of compliant companies. The actual costs of regulations are only 2-3% of total revenue, a small fraction compared to relocation costs. Factors such as wage reduction and markets have proven to have a greater influence on business relocation than environmental regulation (Ackerman and Massey, 2002: 4).

Increasing awareness of policy makers will increase the political will to reduce toxins

In recognition of the deleterious and carcinogenic effects of PBTs, the Washington State Governor's Offices appropriated \$1,216,000 from the State Toxics Control Account for the 2001-2003 biennium. During this timeframe, the *Mercury Chemical Action Plan* was produced. Following the recommendations of the *Proposed Strategy to Continually Reduce Persistent Bioaccumulative Toxins in Washington State*, additional funding has been ordered. On January 28, 2004, Governor Locke signed the Executive order 04-01, which allocated \$50,000 in emergency funding to begin the rule development process. The Washington State Legislature, (2004) excerpt from House Bill 2459, Section 301, then allocated \$166,000 of the general fund for the fiscal year 2000 to the rulemaking and development of chemical action plans for PBTs. Half of this money was directed for the *Draft PBDE Chemical Action Plan*, which was released for public review on October 20, 2004. The other half of

this endowment was to develop the criteria for which chemicals will be on the PBT list, develop the specific list, and establish the criteria for selecting chemicals for chemical action plans. Currently, \$159,000 of state toxics control account is provided for the mercury chemical action plan's implementation: \$84,000 to develop a memorandum of understanding with WA hospitals and auto recyclers for proper removal and disposal of mercury containing products, \$75,000 is directed for ongoing fluorescent lamp recycling. The total amount of money invested in 2004 for developing and implementing action plans to reduce PBTs in Washington States equaled \$375,000. (Washington State Legislature, 2004)

This study has estimated that pediatric cancers alone cost the state of Washington \$222.9 million per annum. Assuming a conservative 5% of these cancers are attributable to environmental factors, the cost of pediatric cancer due to environmental contamination is \$11.2 million per year. The care and treatment costs for just a single child has been determined to be \$724,000. If the reduction of carcinogens from the PBT action plans prevents even one child from developing cancer, the return on the investment would nearly be doubled. In 2001, 308 children were diagnosed with cancer. Assuming the cancer rates in 2004 are equivalent to 2001, the \$375,000 allocation from the state funds equates to \$1,218 per child diagnosed with cancer in WA. When considering the investment per child, return is nearly 600 times. Preventing children from exposures to carcinogenic chemicals will also benefit the adult population. This study is limited in that it does not factor the costs of environmentally attributable adult cancers, asthma, or lead poisoning. Had we done so, the return on investments into precaution would be even greater. This study regards investing in precaution as a win-win scenario. Certainly, the implementation costs of the PBDE and Mercury action plans will be much higher than the costs to develop. The return on the investment will be less. However, the reasoning of the argument of this study remains resolute. When policy makers understand the economic costs of cancers caused by anthropogenic carcinogens in the environment, the political motivation to remedy the situation will amplify.

Increasing the capacity to reduce environmental toxins

The definition used in this analysis for the 'capacity to reduce environmental toxins' is the 'political will' plus the 'financial means for social services.' (See CLD 4.2). The political will has been supportive in Washington State; this is demonstrated by the coordinated actions of the Department of Health, Department of Ecology, and the State Legislature to produce action plans that minimize the circulation of PBTs. This study has stressed the importance of estimating the economic costs for pediatric cancers. By doing so, it has been demonstrated that reducing persistent bioaccumulative toxic substances in Washington has high return on the initial investment. Understanding that treatment costs far exceed the costs of prevention supports the argument to invest further in preventative policy measures; reducing the cancer incidence will reduce the direct cost per case of treatment. Therefore, the financial means for further social services will not be stressed. This represents a positive reinforcing cycle that promotes economic, human, and environmental sustainability.

This topic was chosen to address several ethical and human rights issues. First, society is morally obligated to protect its citizens, especially its children. It is known that fetuses and small children are highly vulnerable to toxics in the environment. The US EPA (2004b) acknowledges that

metabolic, detoxification, and removal processes in children are unable to cope with many pollutants in which they are exposed. Children are considered an at-risk population; yet, they have little or no influence on the degree of their exposure. This group must be protected. Numerous advocates for the precautionary principle have called for improved childhood cancer registries (Tickner, 2003: 371-383). Washington's cancer registry is a leading system in the United States. Unfortunately, across the nation childhood cancer registries are inconsistent; drastic variations exist state-by-state (Trust for America's Health, 2004). Improved tracking systems on a nationwide level can gather information on geographical distribution of cancer, promote epidemiological studies, and better indicate a need for allocation of state and national funds. Such an approach will foster better protective measures for this vulnerable subpopulation.

Second, environmental health is an environmental justice issue. Environmental justice is defined as the fair treatment and meaningful involvement of all people regardless of race, color, national origin, or income with respect to the development, implementation, and enforcement of environmental laws (US EPA, 2004c). Nationally, and in Washington State, a disproportionate amount of environmental toxicants and ecological hazardous sites are located in poor and impoverished areas (Faber & Krieg, 2002). In 1995, the Washington State Department of Ecology conducted a study to determine the proportional distribution of 900 industrial facilities in relation to communities of color and low-income. These facilities include: contaminated sites, hazardous waste treatment, storage and disposal facilities, waste-water and air releasers, solid waste landfills, incinerators, and toxic release inventory reporters. The study concluded that a higher percentage of industries are located in minority and poor neighborhoods. Subsequently, people who live in these neighborhoods may tolerate a disproportionate burden of health effects, such as cancer, associated with industrial emissions. Estimates from the US EPA (1998) articulate that 12 million people, including 4 million children, live within one mile of a hazardous waste sites. Policy makers have a moral obligation to protect such populations. The current approach of policy makers and much of the health community is to remedy problems once they have manifested. Such a paradigm allows the perpetuation of social detriments. The significance of the PP is that it finds ways to prevent hardship, prevent elevating cancer incidence, and prevent social injustices. The precautionary principle aims to identify and address the needs of the highly susceptible subpopulations.

This study has stressed the efforts of Washington State to incorporate the precautionary principle and supports the opportunity to continually reduce persistent bioaccumulative toxic substances in the environment. However, certain questions remain regarding the actual success of such proposals: to what degree will vulnerable subpopulations actually be protected from toxic chemicals once the PBT action plans are effective? How long until effects subside? When can we see results? If WA adopts the precautionary principle but other states do not, will results be observed? The answers to these questions are rife with uncertainty. This study does not attempt to hypothesize such answers. Washington State's PBT list contains twenty-four compounds; six are already banned in the United States. When the remaining eighteen toxic compounds are discontinued, it will take many years, and great sums of money to reduce these chemicals from circulation. Because of the inherent qualities of persistent chemicals it is difficult to estimate the length of time until current levels subside, if ever. Nonetheless, we must understand that all scientific work is incomplete. Uncertainties will always exist when establishing the causal relationship between toxic exposure and health outcome, such as cancer

formation. Therefore, by comparison, proposed solutions will contain measures of uncertainty as well. With increased interdisciplinary research focusing on the effects and transport of PBTs, doubt will slowly diminish. Nonetheless, we cannot postpone appropriate responses to protect our environment and citizens due to temporal uncertainties or varying degrees of success by precautionary measures. We must recognize that high rates of cancer in Washington State may be the result of a tradition of unabated chemical usage. We must correct the policy failures of the past in order to promote a healthy and sustainable future.

10. Conclusions

The essential premise of this study is to discuss and advocate the implementation of the precautionary principle in the regulation of chemicals in Washington State. In review, the three main components of the precautionary principle state: if plausible reason for harm exists, and scientific uncertainty persists, then we have an obligation for precautionary action. To better establish a plausible reason for harm caused by chemicals, this study demonstrated that Washington State has exceptionally high rates of cancer of environmental origin when compared to national averages. Most significantly, WA has the highest incidence of female breast cancer and melanoma. Washington's incidence of adult cancer, likely to be caused by toxic substances in the environment, is higher than the national average in 9 of 10 categories. Incidences of childhood leukemia and brain cancer are also above the national average. Incidence rates for breast cancer, melanoma, Non-Hodgkin's lymphoma, childhood leukemia and brain cancer are increasing in both Washington State and the United States. Research linking toxic chemicals to carcinogenesis was highlighted and consolidated in a list. Establishing the associations between toxics and cancer bolsters the argument that anthropogenic chemicals have contributed to the elevated and increasing rates of cancer in WA.

The results of this study can enhance the State's efforts to reduce persistent bioaccumulative toxic substances in the environment. Many of these are well-known carcinogens. Washington State has made bold strides in the protection of its citizens and natural environment. However, stakeholder conflict persists regarding aspects such as costs to the economy, and inclusion of certain chemicals due to the existence of scientific uncertainty. Subsequently, we conducted cost estimates to determine the full costs of pediatric cancers with environmentally attributable origins. This study concluded that these cancers cost the state approximately \$11.2 million, with a possible range of \$4.5 to \$200.6 million per year. In 2004, the Washington State legislature has invested \$375,000 into PBT chemical prevention action plans. By comparing the economic costs of pediatric cancers of environmental origin to the costs directed to precautionary measures, the investment return would nearly double the investment costs if a single case of childhood cancer is avoided. This information can be used to strengthen the environmental argument during the current debate before the PBT advisory committee. This study concludes that investing in precaution is a win-win scenario for the environment, public health, and the economy.

This project was designed to be holistic in its analysis and pragmatic in application. In this sense, cancer is regarded as a symptom to a greater problem. A population with rising rates of life-threatening disease is not sustainable by any measure. Advocating the precautionary principle will, by design, promote sustainable development. This study attempts to bring synthesis to the three spheres of

sustainability, by promoting a solution that has a common and mutually beneficial objective. The economic cost estimates in this study have shown that precautionary measures are smart fiscal policy. Establishing the linkages between cancer and toxic chemicals demonstrates that improving environmental quality will benefit social wellbeing.

Sustainability is not achievable by a single city, state, or country. It is a goal that must be shared across national and geographic boundaries. This study supports the incorporation of the PP into international treaties, such as the Stockholm Convention on Persistent Organic Pollutants, and national agendas, such as the EU's REACH program to regulate chemicals. This study also acknowledges that steps toward a sustainable future will come incrementally. Therefore, the actions of the Seattle Precautionary Principle Working Group and the Washington State PBT chemical action plans are imperative to the promotion of sustainable development. We suggest that further estimates are needed to better establish the economic costs of all environmentally attributable diseases in WA. Estimating the full costs of health outcomes such as lead poisoning, neurobehavioral disorders, asthma, and adult cancers will strengthen the argument for improved environmental and public health protection in the state. Research in the fields of toxicology and epidemiology must continue to promote our understanding of the relationship between toxic substances and the environment. Future research and proper funding for protection of the environment and public health should be considered high on the Washington State, United States, and international priority list. Scientific knowledge bridges the interactions between the complex systems of economy and ecology. Yet, the understanding between these spheres will always be imprecise. The essential role of science is to improve the connections between chemicals and health outcomes, and assess alternatives that encourage the most sustainable alternatives. The precautionary principle aids policy-makers by evaluating how to avoid potential hazards entirely, opposed to how to minimize the damage. The precautionary principle will promote the compulsory change in ideology and actions needed to bring forth the ultimate goal of sustainability.

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12. Appendix

The following is a list of various management strategies of persistent bioaccumulative toxic substances that incorporates the values and actions of the precautionary principle. All strategies are based on actions taken in the absence of complete scientific certainty; prioritizing and managing PBTs with the greatest risk to the environment and human health; establishing management goals; attempting to virtually eliminate all of anthropogenic sources; while using best available scientific understanding and technology.

- Ontario's Candidate List of Substances for Bans and Phase-Outs
- The Canadian Government's ARET (Accelerated Reduction/Elimination of Toxics) Program
- The Canadian Government's Toxic Substances Management Policy
- The Sound Management of Chemicals Initiative of North American Commission for Environmental Cooperation
- The Great Lakes Binational Toxics Strategy
- The Environmental Protection Agency's Multimedia Strategy for Priority, Persistent, Bioaccumulative, and Toxic Pollutants and Waste Minimization program
- The UN Convention on Persistent Organic Pollutants

Washington State's PBT working list

Below is the working list of compounds currently under review in the State of Washington, by the PBT Rule Advisory Committee. The role of this committee is to determine the specific what chemicals will be on the persistent bioaccumulative toxins list for chemical action plans, this list will be complete by December 31, 2004. The PBTs listed in bold are considered known or suspected human carcinogens; listed by the University of California (2004). Due to industry objections several compounds may be omitted from the final list.

Table 12.1. Washington State's PBT working list

Metals	Combustion by-products	Pesticides	Banned Pesticides	Banned Organic Chemicals	Organic Chemicals
Cadmium	Dioxins / Furans	Dicofol	Aldrin / Dieldrin	PCBs	Pentabromo diphenyl ether (PBDE)
Lead	PAHs	Endosulfan	Chlordane		Hexachlorobenzene
Mercury		Lindane	DDT/DDD/DDE		Hexachlorobutadiene
		Methoxychlor	Heptachlor Epoxide		
		Pendimethalin	Toxaphene		
		Pentachlorobenzene / Pentachloronitrobenzene			
		Trifluralin			
		1,2,4,5-tetrachlorobenzene			

The exact wording of the *Proposed Strategy to Continually Reduce Persistent, Bioaccumulative Toxins in Washington State* to incorporate the precautionary principle into the policy framework is stated below (Gallagher, 2000: 16):

Precautionary Principle

The precautionary principle is designed to promote a “better safe than sorry” approach for dealing with hazardous substances and technologies. The principle originated in Europe in the early 1970s and appears in more than a dozen international treaties. For example, the 1992 Rio Declaration on the Environment and Development states: “When there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason for postponing cost-effective measures to prevent environmental degradation.”

The precautionary principle is one of 10 principles that the Department of Ecology identified to help guide the development and implementation of the PBT strategy. The department received numerous comments that both support and oppose including this principle in the revised strategy. Ecology has carefully reviewed those comments and continues to believe it should be included as one of the strategy’s guiding principles. However, based on that review, Ecology believes there are several issues associated with the practical application of the principle that require clarification.

Role of Scientific Information: Measures to address environmental problems must have a sound scientific and policy basis. A rigorous scientific review will be conducted when identifying what substances should be included on the list of PBTs. However, Ecology believes that respect for the limits of our scientific knowledge means that the inability to develop a precise risk assessment value should not be used as a reason to postpone measures to prevent threats of serious, cumulative, and/or irreversible environmental damage.

New and Existing Sources of PBTs: Once a substance has been identified as a PBT, a full range of response options (e.g. control, prevention, use reduction, phase-out) need to be identified and evaluated. Consistent with many current environmental laws, applying the precautionary principle creates a preference for using safer alternatives. However, that presumption can be overcome by considering the technical, economic, and social circumstances surrounding the specific activity. □
Cleaning-up Historical Releases of PBTs: Once a PBT has been released into the environment, cleanup measures must consider the environmental threats posed by these contaminants as well as threats posed by the cleanup measures themselves. Consequently, efforts to clean up historical releases will continue to be guided by risk assessment/risk management concepts.

Consistency with Current Laws and Regulations: Most state and federal laws are based upon precautionary/preventative approaches to environmental problems. Consequently, applying the precautionary principle is consistent with current laws. This was acknowledged by the U.S. delegation to the recently concluded (December 2000) United Nations Treaty Negotiations on Persistent Organic Pollutants (POPs).

Table 12.2. Comprehensive list of cancers with environmental linkages

Cancer	Toxins - by strength of evidence		
	Strong	Good	Limited or Conflicting
1. Adult-onset Leukemias	Benzene+, Ethylene oxide+, Ionizing radiation+	Arsenic, Aromatic amines, 1,3-Butadiene, Carbon disulfide, Dioxins/TCDD, Chlorinated solvents [carbon tetrachloride, 1,2-dichloro-ethane]; Pesticides (Alachlor, DDT, Phenoxycetic herbicides); Tobacco smoke	Asbestos, Electromagnetic fields, Chromium, PAHs; Pesticides: atrazine, carbamates, captan, organochlorines (chlordane, dieldrin, hexachloro-cyclohexanes [^]), organophosphates (crotoxyphos, dichlorvos), and pyrethins; Styrene, Tetrachloroethylene (PCE) [^] , Trichloroethylene (TCE)
2. Childhood Leukemias	Benzene+, Ionizing radiation+	Pesticides, Metal dusts; Chlorinated solvents: carbon tetrachloride, and trichloroethylene (TCE)	Electromagnetic fields, Hazardous air pollutants/Vehicle exhaust, Insecticides (chlordane, dichlorvos, propoxur), Radon
3. Multiple myeloma	Benzene, Ionizing radiation	Chlorinated dioxins/TCDD; Pesticides (arsenical pesticides, phenoxyacetic herbicides)	Asbestos, DDT, Fungicides, Heavy metals, Organic solvents [trichloroethane, trichloroethylene (TCE)], Petroleum products
4. Adrenal			Acrylamide [^] , Methyl isocyanate
5. Angiosarcoma (hepatic)	Arsenic+, Vinyl chloride+	Anabolic steroids, Copper, Thorium dioxide (Thorostat)	Chlordimeform, Methylhydrazine, Nitrosamines, Urethane
6. Bladder	Aromatic amines (4-Aminobiphenyl+, Auramine, B-Naphthalamine+, Benzidine+, MOCA), Benzidine-derived dyes, Chlordimeform (and its metabolite 4-COT), Coal tar+, Nitrobiphenyl, Tobacco smoke, trihalomethanes (disinfection byproducts)	Arsenic, Benzo(a)pyrene (PAH's)#, Chlornaphazine, Chlorphenol, Ionizing radiation, Methylene dianiline, Organic solvents, o-Toluidines	Antimony, Asbestos, Chromium, Dichloropropene, Lead, Nitrosamines, Tetrachloroethylene (PCE), Pesticides (organochlorines), Saccharin, TCDD
7. Bone / Ewings sarcoma	Radium+	Pesticides	Benzenes, Beryllium, PAHs, PCBs, Vinyl chloride
8. Brain - adult	Ionizing radiation	Chromium, Methylene chloride	Acrylonitrile, Cadmium, Electromagnetic fields, Ethylene oxide, Iron, Lead, , Oil mists, Organic mercurials; Organic solvents: benzene, carbon tetrachloride, tetrachloroethylene (PCE), trichloroethylene (TCE), toluene, and xylene; Pentachlorophenol (PCP), Pesticides (including atrazine, 2,4-D, and hexachlorobenzene), Radiofrequency fields, Vinyl chloride
9. Brain - childhood	Ionizing radiation	Organic solvents, Pesticides (DDVP, Lindane)	Aromatic amines, Chlorophenates, Dyes, Electromagnetic fields, N-Nitrosoamines, Pesticides: Carbaryl, Diazinon, and Phenoxyacetic herbicides
10. Breast cancer	Estrogens/DES, Ethanol, Ionizing radiation	Aromatic amines (B-naphthylamine and benzidine), Ethylene oxide, PAHs, Tobacco smoke	Acrylamide [^] , 1,3-butadiene, Dioxins/TCDD, Electromagnetic fields, Hydrazine [^] ; Organic solvents (Benzene, 1,2-dibromoethane, 1,1-dichloroethane, 1,2-dichloroethane, 1,2-dichloropropane, methylene chloride, tetrachloroethylene (PCE), trichloroethylene (TCE) and 1,2,3-trichloropropane); Organochlorine pesticides (DDT/DDE, chlordane, benzene hexachloride, dieldrin, mirex and aldrin); PCBs, Phenoxyacetic herbicides, PhIP, Styrene, Triazine herbicides, Vinyl chloride
11. Carcinoid			Lead, Organic solvents
12. Cervical	DES, Tobacco smoke	Organic solvents	Pesticides, Tetrachloroethylene (PCE). Trichloroethylene (TCE)
13. Cholangio-carcinoma		Thorium dioxide (Thorostat)	
14. Colo-rectal		Acrylonitrile#, Alachlor, Aromatic amines, Ionizing radiation, Organic solvents, trihalomethanes (disinfection byproducts)	Asbestos, Chlorophenols, Nitrosamines, Organochlorine pesticides (aldrin/dieldrin, DDT/DDE), Phenoxyacetic herbicides (2,4-D, 2,4,5-T), PAHs, PhIP, TCDD, Toluene, Xylene

15. Gallbladder		Thorium dioxide (Thorostat)	Organochlorine pesticides (benzene hexachloride, DDT)
16. Esophageal cancer	Ethanol, Tobacco smoke	Nitrosamines; Organic solvents [Tetrachloroethylene (PCE)#]; PAHs, Silica	Chromium, Pesticides
17. Renal (kidney)	Tobacco smoke	Arsenic, Asbestos, Benzene, Coal tar/soot/pitch/asphalt/creosote, Copper sulfate, PAHs, Pentachlorophenol, Pesticides, Trichloroethylene (TCE)	Benzidine, Cadmium, Chlorothalonil, Chromium, Dibromochloropropane (DBCP), p-Dichlorobenzene [^] , Gasoline [^] , Inorganic Lead [^] , Mineral/cutting/ lubricating Oils, MTBE?, Mustard gas, Nickel, Organic solvents (carbon tetrachloride, chloroform [^] , tetrachloroethylene), Potassium bromate, Vinyl chloride
18. Laryngeal	Ethanol, PAHs, Sulfuric acid ⁺ , Tobacco smoke	Diethyl sulfate#, Leather dust, Mustard gas, Nickel, Wood dust	Acetaldehyde [^] , Asbestos, Formaldehyde, Pesticides, Petroleum products, Vinyl chloride
19. Hepato-cellular (Liver)	Aflatoxin B1 ⁺ , Androgens, Ethanol, Hydrocarbons	Arsenical pesticides, Dimethylnitrosamine, PCBs#, Thorium dioxide (Thorostat), Trichloroethylene (TCE)#, Vinyl chloride	Arsenic, Benzene, p-Dichlorobenzene [^] , Dichloropropene, 1,4-Dioxane [^] , Hydrazine [^] , Nitrosamines, PBBs [^] ; Pesticides: chlordimeform, dibromochloropropane (DBCP), ETU, herbicides (acifluorfen, amitrole, fumeqyclox, lactofen), Organochlorines (aldrin, chlordane, DDT/DDE, endrin, heptachlor, hexachlorocyclohexanes [^] , toxaphene), Nitrofen, Oxadiazon, and Phenoxyacetic herbicides; Solvents: carbon tetrachloride [^] , chloroform [^] , formalin, methylene chloride [^] , and tetrachloroethylene (PCE) [^] ; TCDD [^] ,
20. Lung	Aluminum, Arsenic ⁺ (including arsenical pesticides); Asbestos ⁺ , Attapulgit, Benzo(a)pyrene (PAH's)#, Beryllium ⁺ , Cadmium ⁺ , Chloromethyl ethers ⁺ , Chromium ⁺ @, Coal tars ⁺ , Diesel engine exhaust, Ionizing radiation, Mineral oils ⁺ , Mustard gas ⁺ , Nickel ⁺ , Radon ⁺ , Silica ⁺ , Soots ⁺ , Tobacco smoke, Uranium	Acid aerosols, Acrylonitrile#, Aromatic amines, Chlorophenols, Coal dust, Copper, Dimethyl sulfate#, Formaldehyde, Solvents, Nitrosamines (NNK); PAHs (Benz(a)anthracene#, Benzo(a)pyrene#, Dibenz(a,h)anthracene#)	Antimony trioxide [^] , Benzene, Bromoform, Ceramic fibers [^] , Cobalt [^] , Dibromochloropropane (DBCP) [^] , Dichloropropene, Fluoride, Glasswool [^] , Hydrazine [^] , Hydrogen chloride [^] , Lead [^] (inorganic), Methylene chloride [^] , Nitrobenzene, Nitrosomorpholine, Organochlorine pesticides (chlordane, DDT [^]), Paraquat, Phenoxyacetic herbicides, Rockwool [^] , Slagwool [^] , Styrene [^] , Talc, TCDD [^] , Tetrachloro-ethylene (PCE), Trichloroethylene (TCE), Urethane, Vinyl chloride
21. Lymphoma - non-Hodgkin's	Dioxins (TCDD ⁺)	Aromatic amines, Benzene, 1,3 Butadiene#, Chlorophenols, Creosote, Ionizing radiation; Organic solvents: carbon disulfide, carbon tetrachloride, trichloroethylene (TCE)#, tetrachloroethylene (PCE)#; PCBs, Pesticides: Carbamates (Carbaryl), Dicamba, Fungicides (Captan), Organophosphates (dichlorovos, malathion), DDT [^] , Phenoxyacetic acid herbicides (2,4-D, MCPA, mecoprop)	Asbestos, MTBE; Pesticides: alachlor, atrazine, glyphosate, organochlorine pesticides (aldrin/dieldrin, chlordane, heptachlor, lindane, toxaphene); UV radiation, Vehicle exhaust
22. Hodgkin's Disease (lymphoma)		Chlorophenols, Phenoxyacetic acid herbicides, TCDD/Dioxins	Creosote, Ethylene oxide, Organic solvents, Pesticides: organochlorines (aldrin, DDT, lindane), Trichloroethylene (TCE)
23. Mesothelioma	Asbestos ⁺ , Erionite	Ionizing radiation, Zeolite	Beryllium, Ceramic fibers, Ethylene oxide, Nickel, Silica, Talc
24. Mycosis fungoides (cutaneous T-cell lymphoma)		Organophosphate pesticides	Metals, Oils/Petrochemicals, Organic solvents, Pesticides (glyphosate)

25. Nasopharyngeal/Sino-Nasal	Chromium, Formaldehyde#, Tobacco Smoke, Leather dust, Nickel+, Wood dust	Diisopropyl sulfate, Isopropyl oils, PAHs	Acetaldehyde^, Benzene, Chlorophenols, Dioxane^, Hexamethyl-phosphoramide, Hydrazine^, Mustard gas, Nitrosamines, Pentachlorophenol (PCP); Pesticides: Alachlor, Organochlorines (Acetochlor); Dibromochloropropane (DBCP)^; Ethylene dibromide (EDB), Phenoxyacetic herbicides (2,4,5-T, MCPA); Propylene oxide, Radium
26. Oral	Tobacco	Ethanol, Nitrosamines (NNN and NNK)	PAHs
27. Ovarian		Ionizing radiation	Aromatic amines (dyes), Asbestos, Diesel exhaust, Organic solvents, Pesticides (Triazene herbicides), Talc
28. Pancreatic	Tobacco smoke	DDT/DDE, Ethylan, Fungicides, Herbicides, Ionizing radiation, Nitrophenol, Organic solvents, PAHs, PCBs, Pentachlorophenol	Acrylonitrile, Cadmium, Chlorohydrin, Chromium, Ethylene oxide, Nitrosamines (NNK), Nitrofen, Silica; Solvents: carbon tetrachloride, formaldehyde, methylene chloride, styrene, tetrachloroethylene (PCE), trichloroethylene (TCE); Vinyl chloride
29. Prostate		Acrylonitrile#, Aromatic amines, Cadmium, Organic solvents, PAHs	Androgens/Estrogens, Bisphenol A, Chlorophenols, Chromium, Nickel; Pesticides [dibromochloropropane (DBCP), DDT, methyl bromide, phenoxy acetic herbicides]; PhIP (2-amino-1-methyl-6-phenylimidazol[4,5-b]pyridine), Trichloroethylene
30. Retinoblastoma			Pesticides
31. Salivary gland	Ionizing radiation		
32. Soft tissue sarcoma	Dioxins (TCDD+)	Chlorophenols^, DDT, Phenoxyacetic acid herbicides^ (2,4-D, 2,4,5-T, MCPA)	Cadmium, Captotol, Chromium, Cobalt, Hexachlorobenzene, Iron, Nickel; Pesticides: amitrole, organochlorines (chlordane, lindane); Titanium
33. Scrotal	Coal tar+, Shale oils+, PAHs	Creosotes#,	
34. Melanoma	UV radiation		Asbestos, Carbon tetrachloride, Formaldehyde, PAHs, Pesticides
35. Skin (non-melanoma)	Arsenic+, Coal tar+, Ionizing radiation+, Mineral oils+, Shale oils+, UV radiation+	Aromatic amines, Creosotes#, Ethylene oxide, Mineral oils; PAHs: anthracene, benzo(a)pyrene#, dibenzanthracene, dimethyl benzanthracene, methycolanthrene; Pesticides (arsenicals)	Acrylamide^, Vinyl chloride
36. Stomach		Asbestos, Aromatic amines, Chromium, Coal dust, Dioxins/TCDD, Ethylene oxide, Ionizing radiation, Nickel, Nitrates, Organic solvents, Phenoxyacetic herbicides	Butadiene, Lead; Pesticides: amitrole, dibromochloropropane (DBCP)^, dichlorvos, and ethylene dibromide (EDB); Toluene, Xylene
37. Testicular		DES/Estrogens, Pesticides	Cadmium, Chlorophenols, Dimethyl-formamide^, Electromagnetic fields, Ethylene glycol ethers, MTBE?; Pesticides: dibromochloropropane (DBCP), hexachlorobenzene, methyl bromide, organochlorines (chlordanes), organophosphates phenoxy herbicides (2,4-D, MCPA); PCBs, Trichloroethylene (TCE), Zinc
38. Thyroid	Ionizing radiation	Ethylene thiourea^ (ETU)	Acrylamide^, Chlorophenols, Nitrosamines, PBDEs; Pesticides: amitrole, ethylene bis-dithio-carbamate fungicides (EBDCs-maneb, mancozeb and zineb), hexachlorobenzene, phenoxyacetic herbicides; TCDD
39. Uterine			Estrogens, Ethylene oxide, Pesticides (DDE, Dieldrin)
40. Vaginal	DES		
41. Wilm's Tumor			Aromatic amines, Lead, Pesticides

Source: Adapted from the Collaborative on Health and the Environment Database, <http://www.protectingourhealth.org/>