

# Critique of Human Health Risk Assessment

concerning

BA Energy Inc - Heartland Upgrader  
EUB Application No. 1347899

March 24, 2005

for

JENNIFER J. KLIMEK  
PROFESSIONAL CORPORATION  
Barrister & Solicitor  
240, 4808 - 87 Street Edmonton

for

Northeast Strathcona County Residents ("NESCR").

by

*Roger Dixon, B.Sc.(Tech,) MPH, P.Eng., CIH  
R. Dixon Associates  
Consultants, Health, Safety and Environment  
60 Prince Edward Street - Ste 2  
Brighton, Ontario, Canada K0K 1H0  
Tel: 613-475-3572 Cell: 416-809-4385  
E-mail rdixonconsult@sympatico.ca*

# CONTENTS

<b>1</b>	<b>INTRODUCTION</b>	<b>4</b>
<b>2</b>	<b>HEALTH IMPACT EVALUATION - CHEMICALS</b>	<b>5</b>
2.1	Sulfur dioxide	8
2.2	Nitrogen dioxide	9
2.3	Carbon monoxide	9
2.4	Particulate Matter	11
2.5	Polyaromatic Hydrocarbons	13
2.6	Dioxins and Furans	13
2.6.1	De Novo synthesis in the Upgrader's stack and flare systems	14
2.6.2	CCME - Assessment of Dioxins and Furans	14
2.7	Ototoxins	15
<b>3</b>	<b>NOISE</b>	<b>15</b>
<b>4</b>	<b>UNCERTAINTY &amp; PROBLEMS WITH MEASURABLE HEALTH EFFECTS</b>	<b>17</b>
4.1	Evaluation of Health Effects	19
4.2	Medical End-points and Measurability	20
4.3	Dose-Response Assessments	21
4.4	Non-Threshold Events	23
4.4.1	Carcinogenic	23
4.4.2	Genotoxic	23
4.4.3	Teratogenic	23
4.4.4	Developmental	24
4.4.5	Endocrine disruption	24
<b>5</b>	<b>PROBLEMS WITH MULTIPLE CHEMICAL EXPOSURES</b>	<b>25</b>
<b>6</b>	<b>RECEPTOR CHARACTERIZATION</b>	<b>28</b>
6.1	Body Burden	29
6.2	Psychological Impact	30
<b>7</b>	<b>REGULATIONS IN THE CONTEXT OF PROTECTING HEALTH</b>	<b>30</b>
7.1	Canada-wide Standards (CWS)	31
<b>8</b>	<b>LIMITATIONS OF THE HUMAN HEALTH RISK ASSESSMENT</b>	<b>33</b>
8.1	Problems in Research Methodologies	33
8.1.1	Animal studies	34
8.1.2	Epidemiological constraints	34
8.1.3	Uncertainty in Statistical Procedures	34
8.1.4	Low-level adverse effects.	35
8.2	Risk Assessment as a Decision-making Tool	35
8.3	Alternative Approaches - Hazard Based	36
8.3.1	United Kingdom	36
8.3.2	Germany	37
8.3.3	The Netherlands	37

8.3.4 Denmark .....	38
8.3.5 Sweden .....	38
8.4 The Precautionary Principle .....	38
<b>A. REFERENCE - CANADA-WIDE STANDARDS FOR PARTICULATE MATTER (PM) AND OZONE; JUNE 5-6, 2000, QUEBEC CITY, CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT (CCME) .....</b>	<b>40</b>
<b>B. REFERENCE - CANADA-WIDE STANDARDS FOR DIOXINS AND FURANS; APRIL 30 - MAY 1, 2001, WINNIPEG, CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT (CCME) .....</b>	<b>40</b>
<b>C. REFERENCE - CONSISTENT TERMINOLOGY FOR THE DEVELOPMENT OF CANADA-WIDE ENVIRONMENTAL STANDARDS .....</b>	<b>40</b>
<b>D. REFERENCE - EVALUATION OF RISK-BASED APPROACHES IN ENVIRONMENTAL GUIDELINE AND STANDARD SETTING - CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT .....</b>	<b>40</b>
<b>E. REFERENCE (ATTACHED) - CONSISTENT TERMINOLOGY FOR THE DEVELOPMENT OF CANADA-WIDE ENVIRONMENTAL STANDARDS (CWES.) .....</b>	<b>41</b>
<b>F. REFERENCE (ATTACHED) - TOXIC IGNORANCE -REPORT BY THE ENVIRONMENTAL DEFENSE FUND - 1997 .....</b>	<b>42</b>
<b>G. REFERENCE (ATTACHED) - USEPA - COMPLEX CHEMICAL MIXTURES .....</b>	<b>45</b>

# 1 INTRODUCTION

In this Response, references to HHRA mean the various documents submitted by BA Energy, Inc., in support of its human health risk assessment. Likewise, EIA refers to BA Energy documents addressing environmental impact, and AIRpt refers to the Additional Information Report: Air, Human Health and Odours, October 2004. References to statements made in the BA Energy documents are preceded by the format "HHRA Section 1.0 - *Section heading*." Bolding within the quoted references is ours - to identify the specific issue of response.

Cross-referencing uses the format [paragraph 1.0, page 1.] In using the expression exposed population, we mean the population of individuals who may be exposed to the emissions from the proposed Upgrader.

Because the very use of a human health risk assessment is considered by some to be a concession that a certain amount of human risk is *acceptable* (however poorly defined,) many communities members are questioning its suitability for defining their environmental health and safety. Some of the more abrasive aspects of the assessments are the frequent statements by risk assessors that certain toxic exposures represent an "acceptable" risk. The question is increasingly asked, - acceptable to whom? To the proponents or to the receptors? A human health risk assessment has to be scientifically plausible in all its stages, in order to be considered seriously by an exposed community, in this case the NESCR.

In reviewing the HHRA, we have focused on problems of uncertainty and knowledge gaps in human toxicology, the problems of multiple chemical exposure and on issues affecting the fourth stage of the HHRA where attempts are made to link environmental exposures to human health effects. Despite the numerous attempts made in the HHRA to explain away the problems using collective biomedical assumptions and "conservative" allowances, the current limitations in human toxicological knowledge have not been acknowledged and allowed for. Rather, such limitations have been discounted, and in some cases trivialized.

As with all human health risk assessments, the BA Energy documents are not an evaluation of health risk in the true human biomedical sense. Rather, the plausibility of the HHRA is restricted to the prediction of community exposures derived from probability-based transport and dispersion modeling. In this manner we draw attention to the distinction between predictions of exposures and predictions of health effects, because these two issues, although obviously related, have different knowledge bases. Regarding health effects, defining a link between environmental exposures to chemicals - including multiple exposures - is compromised by uncertainties predicated by a lack of available research data [paragraph 4, page 17 - Uncertainty and Problems with Measurable Health Effects, and paragraph 5, page 25 - Problems with Multiple Chemical Exposures.]

There are so many unknowns and uncertainties in human metabolic processes (whole body physiology,) that the traditional toxicological approach of dose-response based on one chemical at a time, and the arithmetic treatment of combined effects, is a biomedically inadequate solution

to the problem. In the human health risk assessment sense, models are used to infer answers where facts are unavailable<sup>(1)</sup> and uncertainty is predominant.

Therefore this HHRA, as with other human health risk assessments for process operations (and hazardous waste sites) remains unable to satisfactorily complete the final and critical step, - that of establishing medically meaningful cause-effect linkages between real-world *multiple exposures* to environmental chemicals, and health status.

The “conservative” adjustments applied in the HHRA attempt to compensate statistically for uncertainties in the extrapolation of findings from animal experimentation to humans, and for the almost insurmountable difficulties of defining human receptors in terms of true medical susceptibility and prior body burden [paragraph 6, page 28 - Receptor Characterization.] For toxicokinetic<sup>(2)</sup> and toxicodynamic<sup>(3)</sup> mechanisms addressing *multiple* chemical exposures, the knowledge gaps become substantially greater. This situation is inadequately dealt with in the exposure-prediction model by simply evaluating “permissible” exposures on a chemical-by-chemical basis.

The question of noise as a health stressor should be reviewed from the point of view of perceived loudness and psychological impacts. Further discussion is found at paragraph 3, page 15.

We find the set of BA Energy documents provided for review to be disorganized, with health-impact-related comments on the same issue repeated, often in numerous places and sometimes contradictory. We have attempted to correlate this information as best we can in order to gain an understanding of what is actually being said. However, there may be cases where we have failed to make the necessary cross-connection; in such instances further investigation will be necessary.

We feel that the frequent use of euphemisms in this HHRA is an attempt to discount the implications of certain health impacts. For example we see fatality and death described as “mortality effects.”

## 2 HEALTH IMPACT EVALUATION - CHEMICALS

The traditional paradigm for human health risk assessments invokes a four-step procedure:-

### (a) Hazard identification

To determine whether a given chemical or physical agent could cause an adverse health effect in humans, - at *any* dose. We emphasize the point that chemical hazards are

---

<sup>1</sup> Carrington, Clark D, “An Administrative View of Uncertainty in Public Health,” Franklin Pierce Law Centre, Concord, NH, USA.

<sup>2</sup> Toxicokinetic studies trace the absorption, distribution in the body, metabolism, storage, and excretion of chemicals.

<sup>3</sup> Toxicodynamic studies chart biological responses that are a consequence of the presence of the chemical in the body.

evaluated one at a time.

(b) Dose-response assessment

This task attempts to define the relationship between the dose of an agent, and the *probability* of specific adverse effects on humans, based mostly on laboratory animal experimentation results<sup>(4)</sup>. We discuss below the assumptions and knowledge gaps inherent in the extrapolation of findings from laboratory animal experimentation and human epidemiology to human beings [paragraph 4.3, page 21 - Dose-Response Assessments.]

(c) Exposure assessment

In assessing human exposure and uptake, various routes of dispersion (e.g. through air, water, food) are considered, together with the physiological mode of intake, viz. oral (i.e. eating,) inhalation (respiratory) and dermal (skin contact.) Respiratory exposures can be measured directly with appropriate air-monitoring instrumentation. However, where a source of potentially hazardous airborne contamination is not yet operational, respiratory exposures are predicted at given locations during selected periods of time, based on *projected* contaminant emission rates and resultant air dispersion modeling.

Following exposure, the uptake of some chemicals into the human body can be measured directly by testing body fluids<sup>(5)</sup> (e.g. blood and urine,) and expired air, body wastes, hair and finger nails.

(d) Risk characterization

This step is the most troublesome (and contentious) phase of all human health risk assessments because it requires that the risk assessor make many assumptions in the face of multiple chemical exposures, knowledge deficiencies in human toxicological response, and the biomedical profiling of exposed individuals (receptors.) Further discussion is provided at paragraph 4, page 17 - Uncertainties & Problems with Measurable Health Effects.

AIRpt - page ii - Human Health Risk Assessment (“HHRA”)

*“Health risks associated with operational air emissions are characterized by comparing predicted air concentrations with **regulatory guidelines considered protective** of the most sensitive individuals.”*

In environmental management in Canada there are Regulations, then there are Standards, Guidelines and Objectives. Only Regulations supported by Standards are enforceable.

---

<sup>4</sup> For some environmental toxics (mostly workplace materials,) limited information is available from epidemiological studies carried out on workers, - usually retrospective studies with problematic confounding factors.

<sup>5</sup> Biological monitoring.

Guidelines are optional, and the use of the term “regulatory guidelines” throughout the HHRA diminishes the author’s assertions that human health will be thus protected. However, for the sake of minimizing confusion in referencing, we will accommodate the author’s usage. Reference should be made to the discussion of consistent terminology by Kemper & Associates Inc., which we have included at Reference E, page 41.

Using “*regulatory guidelines considered protective of the most sensitive individuals*” to characterize true human health risk is an unproven procedure, and to describe such regulatory guidelines as being considered protective of the most sensitive individuals is inappropriate.

“If an industrial chemical is allowed by law to be released into the environment, most people assume that it must have been tested and evaluated for its potential risks. Unfortunately, this is simply not true. Keeping chemical hazards under control requires information about what kinds of hazards each chemical poses. If the basic tests to check on a chemical's toxicity haven't been conducted, or if the results aren't publicly available, current laws tend to treat that chemical as if it were perfectly safe<sup>(6)</sup>.”

We discuss the validity of these claims in paragraph 7, page 30 - Regulations in the Context of Protecting Health.

---

#### AIRpt Section 3.2.2.2 - PAHs, VOCs and RSCs

This section contains Table 3-3, “Final List of COPCs for the HHRA.” This is a formidable list of potentially toxic chemicals, and, recognizing the complexities of the petroleum refining process, the total count may well be higher. The response of any human receptor to this combination of chemical assaults is unknown [paragraph 5, page 25.] Nevertheless the HHRA author’s response is:-

*“... ‘Surrogates’ were used for any chemicals that had **little or no toxicity information**. In those instances, reliance was placed on a **toxicological principle** that states that the molecular structure of a chemical has a distinct bearing on its reactivity, biological activity and toxicity. This principle allows the toxicity of a chemical for which **little or no toxicological information exists** to be predicted on the basis of information available on another chemical of similar molecular structure. The second chemical is termed a ‘surrogate’, and the term ‘**read across**’ has been coined to describe the principle. The principle also may be applied to groups of chemicals of similar structure in which toxicity data on individual members of the group may be limited or lacking. In such cases, all members of the group **are assumed to share the same toxic potency** as the most toxic chemical in the group for which toxicity information is known. The following surrogates were used:.....”*

The authors concede that there is a serious lack of reliable toxicological information. Nevertheless, their next step is to introduce a “*toxicological principle*” in order to solve this problem, - based on non-existent biomedical data - by making “predictions” on the basis of

---

<sup>6</sup> “Scorecard,” Environmental Defence Fund

*“information available.”* Regardless, we are informed that, - *“The principle also may be applied to groups of chemicals of similar structure in which toxicity data on individual members of the group may be limited or lacking.”*

It is the information which is not available which concerns us. No knowledge does not mean safe<sup>(7)</sup>. We acknowledge that there is a *limited* amount of research data to support the concept that chemicals of a molecularly similar configuration *may* be predicted to induce similar toxicopharmacokinetic responses in normal healthy human beings; but there is little if any biomedical research to support the theory. True toxic potency is predicted here by assumptions, rather than by biomedical data. This is not acceptable as an adequate procedure for a human health risk assessment of exposed individuals in the community.

If there is clinically supported biomedical evidence that the above rationalizations describe the toxic response of human beings (segregated as being in normal health, male, female, pregnant female, neonatal, developmental, immunocompromised, asthmatic, the elderly, chemically sensitive, etc.) who may be exposed to such a mixture of potentially toxic chemicals, then it is reasonable to ask BA Energy to provide such evidence.

The atmospheric dispersion of the first four substances discussed below was air-modeled separately from the numerous other substances expected to be released by the Upgrader. However, this should not divert attention from the fact that the four substances are components of what could be hundreds of chemicals released from the plant simultaneously. Even though health effects have been evaluated largely on a one-by-one basis, the fact remains that the Upgrader will present nearby residents with a complex multiple-chemical exposure.

## 2.1 SULFUR DIOXIDE

EIA Vol 2 - Executive Summary

Air Quality

*“The Upgrader is projected to increase regional study area SO<sub>2</sub> emissions by 41%”*

It is difficult to rationalize this with the statement that - *“The Upgrader’s contributions to the maximum baseline concentrations is relatively small.”* The use of maximum baseline concentrations instead of averaged or median concentrations as comparison (baseline) numbers may seriously under-report the impact of the proposed Upgrader. Similar criticism can be directed at the NO<sub>x</sub> and PM<sub>2.5</sub> comparisons.

We reject the use of the descriptives *“negligible”* and *“small,”* as subjective, scientifically unsupported, and of no contribution to this risk evaluation.

EIA - Section 6.4.2.2 - Hazard/Toxicity Assessment

The stated “health effects of potential concern” in Table 6-7 are lacking in quoting *respiratory irritation* as the only effect of acute overexposure to NO<sub>2</sub>/SO<sub>2</sub>. Acute (short term) respiratory overexposure to acid gases can cause irreversible tissue damage and permanent respiratory impairment. In older persons, chronic cardiac involvement can follow. Acute SO<sub>2</sub>-related bronchial constriction may also occur in people with asthma or as a hypersensitivity reaction. Clinical studies have found that some asthmatics respond with broncho-constriction to even brief exposure to SO<sub>2</sub>, as low as 0.4 parts per million.

## 2.2 NITROGEN DIOXIDE

Nitrogen dioxide and sulfur dioxide act mainly as irritants, affecting the mucosa of the eyes, nose, throat, and respiratory tract. Extremely high-dose exposure to NO<sub>2</sub> may result in pulmonary edema and diffuse lung injury. Continued exposure to high NO<sub>2</sub> levels can contribute to the development of acute or chronic bronchitis.

The relatively low water solubility of NO<sub>2</sub> results in minimal mucous membrane irritation of the upper airway. The principal site of toxicity is the lower respiratory tract. What is not recognized adequately in the HHRA is that recent studies indicate that prolonged low-level NO<sub>2</sub> exposure may cause increased bronchial reactivity in some asthmatics, decreased lung function in patients with chronic obstructive pulmonary disease, and an increased risk of respiratory infections, especially in young children.

## 2.3 CARBON MONOXIDE

As we discuss later in paragraph 4, page 17, Uncertainty & Problems with Measurable Health Effects, all health risk assessments have difficulty in dealing with the substantial knowledge gaps in human toxicology. For example, we summarize the HHRA’s treatment of carbon monoxide: the table below collates the carbon monoxide data as presented in the HHRA - Section 6.0:-

SUMMARY OF TABULATED EXPOSURE-EFFECTS DATA PRESENTED FOR CARBON MONOXIDE			
Table 6-4	Public Health Effects	Acute over-exposure	Asphyxiation
		Chronic overexposure	N/A = not available
Table 6-5	Acute Exposure Limits	1- hour	15,000 µ/m <sup>3</sup> (13 ppm)
		8 - hrs	6,000 µ/m <sup>3</sup> (5.2 ppm)
		24 - hrs	“No information”
Table 6-6	Chronic Exposure Limits	N/A = Not applicable	
Table 6-15	Acute concentration ratios for carbon monoxide	Footnote says:- “Acute exposure limits were not available for 24 - hour carbon monoxide.”	

Table 6-22	Deals with sulfur dioxide limits	Footnote says:- "A chronic exposure limit was not available for carbon monoxide."
------------	----------------------------------	--

The category "Public Health Effects" is inappropriate; these are more immediately *human health effects due directly to exposure*. In Table 6-4, chronic over-exposure information is N/A meaning "not available;" in Table 6-6 chronic exposure limits are N/A meaning "not applicable." The inference is that this information is not applicable because it is not available. A clearer explanation of the time-period and respiratory concentration (or dose levels) which support the definition of "chronic-exposure" is required, together with its (long-term) human health implications.

In considering multiple-stressor effects, combined exposure to carbon monoxide and methylene chloride produces elevated levels of carboxyhemoglobin, reducing the blood's ability to carry oxygen.

The following study from the International Congress of Toxicology<sup>(8)</sup>, addresses persistent neurological sequelae following carbon monoxide exposure, and is one of many studies of chronic carbon monoxide exposure:-

"CHRONIC CO POISONING - RETROSPECTIVE CHART REVIEW STUDY:

Objective:

Demonstration of substantial persistent neurological sequelae (PNS) in a case series following chronic CO exposure.

Approach:

A series of patients who were chronically exposed to CO were enrolled in the study. Data collected included CO air samples, initial carboxyhemoglobin (COHb) levels, initial treatment rendered, subjective symptoms, and objective findings on neuropsychologic testing (NPT).

# Chronic exposure was defined as lasting more than one week.

# PNS were defined as CNS symptoms and/or abnormalities on NPT persisting for more than 3 months after the CO exposure.

Results:

# 94 patients with CO poisoning were evaluated from Jan., 1995 - Dec., 1997.

# 38 patients had acute exposures and 56 had chronic exposures to CO.

# Of the 56 patients with chronic exposures, 23 developed PNS.

# This included 8 men & 15 women, ages 8 - 58 years of age.

# Initial COHb levels of the 23 were 0.4 - 5.8%.

# Only 1 of the 23 patients was treated with hyperbaric oxygen.

# 17 of the 23 patients had NPT done; 15/17 were abnormal.

---

<sup>8</sup> Bayer, M.J., Orlando, J., McCormick, M.A., Weiner, A., Deckel, A.W. (1998,) Persistent neurological sequelae following chronic exposure to carbon monoxide. Carbon monoxide: The un-noticed poison of the 21st century, - Satellite Meeting, IUTOX VIIIth International Congress of Toxicology, Dijon, France, July 3-4, 1998, Pg. 179.

# PNS included headache, fatigue, irritability, difficulty concentrating, and impaired memory.  
# 15 of the 23 patients were followed for at least 12 months.  
# All 15 were still symptomatic 1 year post-exposure.

Conclusion:

PNS do occur following chronic exposure to CO, even with low apparent blood CO levels.”

---

Since persistent neurological sequelae do occur following chronic exposure to CO, even with low apparent blood CO levels, we reject the credibility of the HHRA’s treatment of, and conclusions regarding, the health effects of chronic exposure to carbon monoxide.

## 2.4 PARTICULATE MATTER

### HHRA Section 6.4.2.2 - Hazard/Toxicity Assessment

#### Page 6-19:

*“The health impacts from exposure to PM are generally small in terms of measurable or relative risk.”*

This is a scientifically unsubstantiated statement. There is considerable documentation addressing the health effects of exposure to particulate matter; secondly, “*measurable*” [see paragraph 4.2, page 20,] and “*relative risk*” are not mutually exclusive terms. The descriptive “*generally small*” is a value judgement and has no meaning in this context. Attempting to compare “*the magnitude of the effect of PM exposure*” with the effects of tobacco smoking is, we suggest redundant, in what is intended to be a health risk assessment of emissions from a proposed Upgrader.

On a contradictory note the authors then state that,

*“.....because exposure to PM is widespread, the public health impact of increased air pollution (and in turn PM) can be significant,”*

and,

#### HHRA Pages 6-20, 21:

*“It should be noted that on the whole, statistical analyses of the epidemiological data do not provide evidence of a threshold level below which no effects would occur (US EPA 2003a, 2003b; HC/EC 1999; Daniels et al. 2000). However, at least one recent single city study did find a threshold for the short-term effects of PM<sub>2.5</sub> at 20–25 µg/m<sup>3</sup> (Smith et al. 2000). The absence of a detectable dose-response threshold, within the range of PM concentrations measured in urban areas, **does not disprove the possibility of a threshold at low levels of exposure that have not been studied (NERAM 1999)**. A threshold for PM may exist within these ranges, but cannot be detected by epidemiological analyses due to the variability in*

*susceptibility across the study population (US EPA 2003b). Within the range of observed concentrations in epidemiological studies, confidence intervals indicate that studies involving lower ambient PM concentrations have greater uncertainty associated with the estimated effects (California EPA 2001.)”*

Although this is a little convoluted in its rationalizations, the authors' summarization of the conclusions from the quoted studies serves usefully to highlight the difficulty, if not the impossibility, of determining attributable risk (to one causal factor - in this case PM<sub>2.5</sub>) in an environment which contains many possible causal factors.

PM<sub>2.5</sub> is of medical interest because of its ability to penetrate into the alveoli of the lung, from where it cannot be expelled. In field monitoring, *particle size selection* is routinely carried out by filtration or centrifugal and impaction techniques. However, unless laboratory-generated for research purposes, anthropogenic air-borne particulate matter does not consist of particles of one specific aerodynamic diameter, such as PM<sub>2.5</sub>. Stack emissions from industrial operations such as the proposed Upgrader will contain not only a discrete cut of PM<sub>2.5</sub>, but a range of particle sizes. Most of the particulate can be expected to consist of chemicals only, with some inert particulate matter (soot) carrying adsorbed chemicals.

The authors are investigating the health effects of PM<sub>2.5</sub> because it is listed as a “criteria compound,” - or more correctly, a criteria substance. PM<sub>2.5</sub> exposure in the context of multiple chemical exposure needs to be considered [paragraph 5, page 25.] A pre-occupation with inhalation exposure to PM<sub>2.5</sub> can divert attention from total inhalable particulate. Consideration of materials capable of deposition and assimilation into the upper respiratory tract, PM<sub>10</sub>, is also important.

#### EIA Vol2 - 6.4.2.2 - Table 6-4 - Potential health effects....

For “acute overexposure” and “chronic overexposure” to PM<sub>2.5</sub>, “increased physician visits, hospitalization and mortality effects.....” are not health effects, - they are the results of health effects. (We are assuming that “mortality effects” means death, rather than irreversible conditions with a terminal prognosis.)

The note under Table 6-4 states that, -

*“The health effects listed have been reported among human subjects following overexposure to the chemicals. The effects occur at varying concentrations and durations of exposure and **are not necessarily expected if the exposure limit is exceeded.**”*

To which we would add with equal justification that the “effects” can occur in many receptors when the exposure is below the exposure limit.

In a contextual report entitled “Canada-wide Standards for PM and Ozone,” Alberta Environment<sup>(9)</sup> has confirmed that PM is a priority substance for assessment under CEPA. The

---

<sup>9</sup> From: “Canada-wide Standards for PM and Ozone,” a Contextual Report by George Murphy, Alberta Environmental Protection, Co-Chair of Workshop, May 2002

draft report released on May 15 for 60-day public comment period, contained the recommendation that PM<sub>2.5</sub> and PM<sub>10</sub> be considered toxic under paragraph 11 ©).

Particulate matter is discussed in more detail in the response by Dr. Rosalie Bertell.

## 2.5 POLYAROMATIC HYDROCARBONS

AIRrpt Section 3.2.2.2 - PAHs, VOCs and RSCs:-

*“With respect to benzo(a)pyrene (B(a)P), the WMM (OMOE 1997) and the IPM were used for the assessment of carcinogenic PAHs (i.e., 1,2-benzanthrene, benzo(a)pyrene, benzo(e)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(b&j)fluoranthene, dibenzo(a,h)anthracene, perylene, phenanthrene, benzo(k)fluoranthene, indeno(1,2,3-cd)pyrene, pyrene, chrysene). The WMM approach is based on the conservative assumption that the potency of the PAH fraction of any environmental mixture is proportional to the B(a)P content of the mixture (OMOE 1997). The WMM uses the concentration of B(a)P together with the toxic potency of the PAH-WMM group, and this group is referred to as B(a)P.”*

This seems to be an overly complicated attempt to explain a series of poorly understood human pharmacokinetic events. To simplify this presentation, the WMM approach should be discarded since it makes too many assumptions, ignoring the (poorly defined) toxic contributions of the remainder of the family of PAHs. If anything, the IPM - using toxic equivalency factors (TEFs) - is preferred. Even so, this exercise has little plausibility in biomedical reality.

Despite the HHRA's rationalizations, the fact remains that a community exposed to polyaromatic hydrocarbons is confronted with a potentially serious health risk. Since many of these compounds are carcinogenic, exposure quantification should be able to provide a starting point for mitigation procedures required to bring exposures down to non-detectable levels. If BA Energy has any clinically confirmed *human* medical cause-effect information to the contrary, then the required target of zero exposure to carcinogens can be left open for further discussion.

## 2.6 DIOXINS AND FURANS

The question of dioxins and furans is not addressed at all in HHRA Section 4.0 Air, Section 6.0 Health, or in the Additional Information Report: Air, Human Health and Odours, even though “dioxin” is contained in the title of seven of the references provided in Section D.8 at the end of the document. Dioxins are listed as developmental toxicants, see paragraph 4.4.4, page 24.

Likewise, chlorine/chloride/chloro is not mentioned in the document except for:

AIRrpt - Section 3.4.1 - AENV air samples

*“The exception was 1,2-dichloroethane, which was above the OAAQC. However, this was only observed during the August 23 to November 29, 2001 sampling period and was not replicated during the later sampling periods.”*

However, chlorine/chloride/chloro is noted in eleven of the references at the end of the document. The HHRA should be re-visited based on the possible presence of chlorine/chloride in the Upgrader process.

### 2.6.1 De Novo synthesis in the Upgrader's stack and flare systems

In addition to being by-products of the pesticide manufacturing industry, dioxins and furans are found in vehicle exhaust emissions and are produced by some industrial combustion processes such as waste incineration - referred to as *de novo* synthesis. This family of highly toxic compounds is frequently characterized by one commonly occurring molecular form (congener,) 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD,) referred to simply as dioxin. The formation of dioxins is combustion-temperature<sup>(10)</sup>-dependent, and requires the presence of chlorine which may be supplied to the combustion mix by chlorinated organics or inorganics such as sodium chloride. TCDD has a very low vapour pressure (low volatility) and a high melting point, - being thermally stable up to about 700°C.

Due to their relative chemical stability and low vapour pressure, the more highly chlorinated dioxins tend to occur in airborne particulate matter, see paragraph 2.4, page 11. There are several literature references to dioxin formation<sup>(11)</sup> in petroleum processing. Since any emissions from the planned BA Energy Upgrader will be additional to existing proximal sources of dioxins<sup>(12)</sup>, this HHRA does not provide sufficient data on dioxin-related health concerns.

### 2.6.2 CCME - Assessment of Dioxins and Furans

In its position paper "Canada-wide Standards for Dioxins and Furans<sup>(13)</sup>," the CCME notes that *"the presence of dioxins and furans in the Canadian environment can be attributed to three principle sources: point source discharges (to water, air and soil), contamination from in situ dioxins and furans, and loadings from the long-range transportation of air pollutants (LRTAP.)"* Referring to emissions targets for industrial boilers, the paper continues, - *"Dioxin and furan emissions will be less than 100 pg/m<sup>3</sup> TEQ for new boilers constructed after the effective date of this standard. Dioxin and furan emissions will be less than 500 pg/m<sup>3</sup> TEQ for all existing boilers by 2006,"* and that *"testing and reporting will be performed using methods and procedures acceptable to the responsible provincial ministry."*

This CWS, which reportedly came into effect on May 1, 2001, addresses primarily the pulp

---

<sup>10</sup> Temperatures and temperature gradients up to final stack discharge are not provided in the BA Energy documents provided with the EIA.

<sup>11</sup> Bear A, Naikwadi KP, Karasek FW; Formation of Polychlorinated Dibenzofurans by Chlorination and de Novo Reactions with FeCl<sub>3</sub> in Petroleum Refining Processes; Environmental Science and Technology, Vol. 27, No. 8, pages 1505-1511, 10 references, 1993

<sup>12</sup> The Shell refinery uses organochlorines to regenerate its catalysts, Dow Chemical reports dioxin & furan emissions to NPRI, and OxyVinyl produces vinyl chloride.)

<sup>13</sup> Canada-wide Standards for Dioxins and Furans; April 30 - May 1, 2001, Winnipeg, Canadian Council of Ministers of the Environment (CCME)

and paper industries and waste incineration operations. The standard does not address oil/gas refinery emissions, certainly not bitumen Upgraders.

However, in a subsequent report March 11, 2002, "PROCEEDINGS - National Multi-Stakeholder Meeting - "Canada -Wide Standards for Dioxins and Furans<sup>(14)</sup>," CCME confirms that it has undertaken the development of Canada-Wide Standards (CWS) for six priority substances: mercury, dioxins and furans, particulate matter (PM), ground level ozone, petroleum hydrocarbons and benzene. On page 5 of the Proceedings, albeit at the end of a footnote, there is finally an acknowledgment that petroleum refineries may create dioxin/furan releases.

We are concerned about the possibility of *de novo* synthesis of dioxins and furans in the discharges from the proposed Upgrader, and would suggest that BA Energy carries out a further examination of its processes and proposed emission controls, to identify any likelihood of dioxin release.

## 2.7 OTOTOXINS

Chemicals with ototoxic properties may potentiate noise-induced hearing loss, in an additive or synergistic manner<sup>(15)</sup>. Ototoxic substances can be absorbed through the skin as well as via the inhalation pathway, e.g. toluene, xylene, n-hexane, organic tin, carbon disulfide, mercury, organic lead, hydrogen cyanide, diesel fuel, kerosene fuel, jet fuel, JP-8 fuel and organophosphate pesticides. Since the exposure thresholds for ototoxic effect are not known, this HHRA should make exposure allowances for the joint impact of noise and chemical exposures.

We discuss in paragraph 5, page 25, the flaws inherent in the HHRA due to inadequate treatment of multiple stressor exposures.

## 3 NOISE

The assessment of noise, and predictive modeling of future noise levels is being considered by others. However from a community health impact standpoint, we note that the EIA and the HHRA fail to adequately consider noise as a health stressor. Rather, the account of noise is a mechanistic description of propagated and attenuated sound pressure levels, with no evaluation of perceived *loudness* and psychological impact.

*"The Upgrader will result in.....long-term continuous localized noise impacts from operational activities. Due to the **localized nature of effects**, the impacts are anticipated to be **not significant**. The predicted comprehensive sound levels from the Upgrader, in addition to sound from existing and future planned projects, are less than the Alberta*

---

<sup>14</sup> "PROCEEDINGS - National Multi-Stakeholder Meeting - "Canada -Wide Standards for Dioxins and Furans," Vancouver BC, March 11, 2002. Prepared by Dovetail Consulting Inc., Vancouver, BC.

<sup>15</sup> U.S. Army Center for Health Promotion and Preventive Medicine, Aberdeen Proving Ground, MD, USA. Advisory 51-002-0903

*Energy Utility Board's permissible sound levels."*

Clearer definitions of - "*localized nature of effects,*" and - "*not significant*" are needed. Again, "*not significant*" appears to be a subjective and unsupported conclusion. Noise is potentially a health effect, and should be evaluated as such.

### EIA Vol 2 - Section 5 - Noise

Noise is unwanted sound<sup>(16)</sup>. Noise creates hearing loss, interferes with communication, and causes psychological stress which in its onset form presents as *annoyance and irritability*.

Noise-effect research has been predominantly based on physiological damage to the human auditory system. In ignoring the psychological impact of noise exposure, this approach is not adequate in terms of evaluating effects on community health and well-being.

#### Section 5.1 - Setting

*"Noise assessment is conducted at the locations of noise-sensitive receptors, which, for the Upgrader, are nearby residences."*

We have no problem with the assertion that "noise-sensitive receptors" are located in nearby residences. However, since the inhabitants of the residences are human beings, the authors should provide a more human-based (and medically substantiated) definition of "*noise sensitive receptors,*" - at the same time perhaps, providing a definition of *non-noise-sensitive receptors*. The requested medical definition of "noise sensitivity" would permit the reader to properly evaluate the implications of the various sound power sources (current and projected,) the factors involved in noise attenuation and the impact on individual residents.

The major problem here is not the accuracy of the sound-pressure readings reported by the authors, or the accuracy of their reporting, - or even the accuracy of the "unknown" noise level predictions from the algorithms used in the *Cadna-A* propagation model, - but the validity of the model input values for future case scenarios. It is difficult to predict sound power generated by future processes and operations.

The human ear is more sensitive to high frequency sound; changing the frequency of a sound changes its relative loudness<sup>(17)</sup> as perceived by the *noise-sensitive receptor*. The health stressor impact of noise is not evaluated by sound pressure levels (SPLs) alone, but also by the evaluation of loudness. Since loudness is the human perception of noise, we suggest that the authors re-visit the question of noise, with psychological stress impact in mind.

The question of resonance should be addressed. Resonance describes the effects of sound pressure peaks combining to produce additive sound pressure levels, and hence amplified

---

<sup>16</sup> American National Standards Institute (ANSI.)

<sup>17</sup> Fundamentals of Industrial Hygiene: National Safety Council 1995: the "Fletcher-Munson contours, pp 205"- adapted from the Handbook of Noise Measurement, 9<sup>th</sup>. ed., GenRad, Inc., 1980.

loudness. As a human health impact, the issue of resonant frequencies from all neighbourhood sound energy sources (all industrial activity, current and projected) should be evaluated.

OBA (octave band analysis) is useful in the identification of noise sources and sound power generation, - and as such its findings can perhaps be transferred to human *effect*, or perceptions of loudness.

Sound energy is propagated as air vibrations, and there is a frequency window at which these air vibrations become physical vibrations, - about 20 Hz<sup>(18)</sup>. An analysis of the propagation of low-frequency (vibrational) energy, and its effects on the community, is suggested.

## 4 UNCERTAINTY & PROBLEMS WITH MEASURABLE HEALTH EFFECTS

The presentation of this HHRA carries the conviction that the authors have a secure knowledge of all possible human medical responses arising from exposure to the Upgrader's emissions, - that they are sufficiently biomedically informed, and always know what health effects to *look for*, and that subsequent diagnoses of affected individuals will be in clinical agreement with the predicted health effects. Implicit in this conviction is the premise that laboratory animals will respond to toxic assault in a manner which will establish the basis for accurate extrapolation to the physiology of human beings.

Particularly disturbing in all human health risk assessments is the inevitable restriction of scientific inference to what we know based on limited data, rather than *what we don't know*, simply because data is unavailable. The available data for responses observed in controlled laboratory test environments, - mostly for single chemicals, has very limited relevance to the more complex relationships, such as multiple chemical exposures, developmental toxicity (teratogenic outcomes) and endocrine disruption (whole-physiology systemic outcomes.)

The problems of identifying, measuring and then evaluating (statistically) true animal responses are considerable, and it should not be assumed that the findings are applicable to humans in the whole-body-system sense. As mentioned, the situation becomes considerably more opaque when multiple chemical exposures are involved. An insight into the practice of inserting default factors into health risk assessment models is provided by Dr. Clark D. Harrington's<sup>(19)</sup> description of default factors:-

“The oldest and most widely used technique for dealing with model uncertainty in the context of regulatory toxicology involves the application of a fixed arbitrary factor whenever an instance of model uncertainty (or in some cases, statistical uncertainty) is encountered. For instance, factors of 10 are often applied when it is necessary to make statements about large populations based on the observations of a few individuals, about humans based on the observations of rodents, or about long-term exposures based on the observations from short-term exposures.

---

<sup>18</sup> Hz = Hertz = cycles per second.

<sup>19</sup> Centre for Food Safety and Applied Nutrition, U.S. Food and Drug Administration; in a paper entitled “An Administrative View of Model Uncertainty in Public Health.”

Judging from appearances, the general rule is to divide by 10 upon each instance of model uncertainty. Because selection of the factor is not dependent on the state of the evidence, it is clear that the application of safety or uncertainty factors is not really a method for evaluating model uncertainty. **Rather, it is a technique for avoiding the issue by not making a prediction.**

Default factors were originally designed for setting regulatory levels. However, the levels are also sometimes represented as factual statements, i.e. thresholds. In this circumstance, the default factor approach may be thought of as a default model (see below), rather than a decision-producing policy. Similarly, default factors are sometimes represented as instruments of a technocratic exercise - **in which case the factors are merely window dressing for a decision that has already been made.** They cannot, however, be considered to be both procedures and products of technocratic judgment as these concepts are antithetical. However, it is possible to mix in some of each to create a hybrid. For instance, a factor of 3 may be used instead of 10 because, on the basis of expert judgment, the degree of model uncertainty is smaller. The process is still semi-procedural to the extent that the factors must follow half-orders of magnitude.” (Bolding is ours.)

Such uncertainty is demonstrated in the HHRA’s discussion of nitrogen dioxide. The authors of have properly reported, regarding a 1992 paper by Magnussen, that:-

EIA - Vol 2, Section B 2.4

*“Magnussen...concluded that asthmatics were more sensitive to NO<sub>2</sub> than healthy subjects, but that their response depended on both the severity of their disease and the study protocol used.”*

This medical finding of thirteen years ago seems to be intuitive today; but the author’s declaration of dependence on *“the study protocol used”* serves as an example to remind us that the published findings of such research are, in the end, subjective and dependent on the researcher’s definition of observed effects, and on the statistical treatment of sample results.

Continuing to address nitrogen dioxide, the HHRA goes on to point out that:-

*“Much variability exists among the NO<sub>2</sub> studies to date, **with no particular study reporting a clearly defined concentration-response relationship (WHO 2000.)”***

This information gap applies to many other chemicals in addition to nitrogen dioxide.

*In Canada*, regulatory authorities rarely carry out their own toxicological investigations, but rely on published information without necessarily reviewing its scientific plausibility in terms of statistical power. We suggest that all the human health effects references used in this HHRA, - directly or via the Government of Alberta, Health Canada and the CCME, - be evaluated for methodology and interpretation and the plausibility of applying animal-based toxicological findings to human beings in a multiple exposure environment.

“Regulators are faced with a wide array of scientific decisions when developing risk assessments for a given chemical: Should they use a "weight of the evidence" approach that considers multiple studies, or base the

assessment on a single "critical" study? What should they choose for the value of the "uncertainty factors" which account for remaining scientific uncertainties and variability within the population? How appropriate is each given human or animal study for the purpose of risk assessment?<sup>(20)</sup>"

## 4.1 EVALUATION OF HEALTH EFFECTS

In this HHRA, the terms acute and chronic with regard to exposures, are frequently used indiscriminately. In the real world, there are high and low exposures for short periods of time, then there are high and low exposures for long periods of time. Then, just to complicate the issue further, there are intermittent exposures of varying times and intensity. The tabulated categorizations of exposures and "health effects" in the HHRA are confusing and difficult to interpret. An example of this problem is illustrated in the earlier section on carbon monoxide, paragraph 2.3, page 9.

HHRA Section 6.7.2 Summary of Project and Cumulative Effects states -

*"Health risks associated with operational air releases were characterized by comparing predicted air concentrations with **regulatory guidelines considered protective of the most sensitive individuals**. Although the predicted air concentrations exceeded health-based guidelines at a few of the discrete receptor locations, the HHRA concluded that **no measurable health effects** would occur as a result of the Upgrader's atmospheric releases of the criteria compounds. This was largely based on the inherent conservatism built into the risk assessment and the apparently negligible differences between the ground-level air concentrations for the Baseline and Application Cases."*

This statement erroneously links "regulatory guidelines" with "no measurable health effects". It then switches from considerations of "regulatory guidelines" to considerations of "health based guidelines" which were "exceeded" at "a few of the discrete receptor locations."

For reasons discussed in various places in this Response, we question the process of substituting regulatory guidelines for actual biomedically safe doses to the individual [paragraph 7, page 30], as we equally question the conclusion, based on statistical prediction, that "no measurable health effects" would occur as a result of the Upgrader's releases of criteria compounds. Having stated that regulatory guidelines are protective of health, the authors then proceed to state that their own predicted exceedances of these regulatory guidelines don't really matter, because of "inherent conservatism" built into the HHRA, and "negligible differences" between Baseline and Application cases.

Inherent conservatism as applied to a human health risk assessment serves to disguise the fact that, dose-response wise, the assessors really don't know how to relate individual person-exposures to medical outcomes. There are a number of reasons why this final step is difficult in a few cases, and impossible in all others.

The lack of definitive knowledge regarding the complexities of individual human responses to

---

<sup>20</sup>

"Rocket Fuel Contamination in California Milk" - Environmental Working Group, June 22, 2004

toxic chemicals exposure is a fundamental weakness of all human health risk assessments. Biomedical knowledge gaps cannot be compensated for by substituting statistically based predictive models. Such models cannot address aspects of human physiology, sensitivities, prior exposures and existing body burdens [see paragraph 6.1, page 29,] multiple chemical exposures [see paragraph 5, page 25,] endocrine disruption [see paragraph 4.4.5, page 25] and other *whole-body* response mechanisms.

The one-by-one evaluation of criteria compounds as health stressors ignores the fact that these are present in a complex mixture of total process emissions. A significant lack of toxicological and biomedical knowledge regarding multiple chemical exposures (as distinct from individual multiple-chemical-sensitivity,) is one of a number of issues which challenge the credibility of the predictions of health outcome attempted by all human health risk assessments. We discuss this in more detail at paragraph 5, page 25.

Regarding the contribution which the criteria compounds make to smog, clear epidemiological findings in Ontario over the last two years have caused concern for the Ontario Medical Association<sup>(21)</sup>:-

“The OMA (Ontario Medical Association) believes that smog is a public health crisis that results in more than 2000 premature deaths and 48,000 emergency department visits in Ontario each year. A significant portion of Ontario's smog pollution is transported into the province on prevailing winds from the U.S. Midwest.”

Is there somewhere a health risk assessment based on emissions and human permissible exposures or RfDs (reference doses) which accurately predicted this breathtaking outcome?

## 4.2 MEDICAL END-POINTS AND MEASURABILITY

### AIRpt - page ii - Human Health Risk Assessment (“HHRA”)

*“Predicted air concentrations do not exceed health-based guidelines at any of the 21 receptor locations. Therefore no **measurable** health effects are predicted as a result of the Upgrader's atmospheric emissions of the identified chemicals of potential concern. This is largely based on the low overall risk estimates and the inherent conservatism built into the risk assessment.”*

In 1995, Fan, Howd and Davies<sup>(22)</sup>, well respected health risk assessors working for the California Department of Environmental Protection, outlined some of the difficulties associated with defining and measuring end-points:-

---

<sup>21</sup> Toronto, March 2, 2005 – Today the Ontario Medical Association (OMA) expressed concern about the effects the U.S. proposed pollution bill will have on Ontario's families and health-care costs. Should be striving to keep citizens on both sides of the border healthy.”  
Ontario's Doctors: U.S. Bill May Worsen Air Quality: Doctors send a strong message to decision-makers south of the border on behalf of Ontario patients.

<sup>22</sup> Fan A, Howd R, Davies B, “Risk Assessment of Environmental Chemicals,” Annual Review of Pharmacology and Toxicology Vol. 35 (1995,) pp. 341-368

- There is no agreement on which tests to use to determine whether someone's immune system has been damaged;
- There is no agreement on which tests should be used to assess damage to the nervous system;
- There is no agreement-and there may never be, on ways to test for genetic damage.

Thresholds of response are those levels at which a response is noted for the particular medical endpoint being investigated, - in terms of measurable harm to the species being studied. In some instances the mechanisms of response exhibit a linear relationship between dose and response, In others, a non-linear, exponential or biphasic dose-response curve is seen. For many toxics, the nature of the dose-response curve remains unknown. Without agreement on test methods, it becomes impossible to design meaningful health-effects studies. Under these circumstances, different risk assessors will select the data in a manner that they believe is supportive of their expectations.

### 4.3 DOSE-RESPONSE ASSESSMENTS

Despite a growing body of literature suggesting the contrary, the chemical/petrochemical industries continue to claim that low dose exposures to hundreds of chemicals simultaneously are safe. These claims however, rely on very limited scientific information on the toxicity of multiple chemical exposures, - and not on a definitive, scientific proof of safety.

Since there are ethical problems in carrying out toxic dose-effect studies on human beings, information on human dose-response is derived:

- Indirectly, by translating the observed effect on laboratory animals into an "equivalent" effect on human beings. This process of extrapolation requires *assumptions* regarding physiologically based pharmacokinetic (PB/PK) /pharmacodynamic (PB/PD) similarities between laboratory animals and human beings, which are becoming increasingly questionable, and
- Directly from exposure-effect data reported for persons in the occupational environment (industrial exposures,) which data are often retrospective in nature, - that is "exposed" and "non-exposed" persons and health-outcomes are identified from past records.

In both cases, an extrapolation is made from the effects observed from the generally high doses (acute) administered to experimental animals or from those high doses experienced in the workplace, to the lesser but more complex exposures experienced by persons in the general environment. In animal experimentation, high doses are usually administered in order to accelerate the metabolic processes which may (or may not) reveal an adverse effect.

Government regulations, exposure limits and standards for contaminants in consumer products, drinking water, food and air are based predominantly on dose/response findings in short-term *high dose* animal studies. For human health effects, this leaves us with a flawed predictive model which assumes that *high dose* animal studies will reveal *all* the toxic impacts on humans - including a latency profile - of the chemical being tested. For long-term (chronic) low dose exposures, we are realizing that this predictive process is highly questionable, particularly for

chemicals with endocrine disruptor potential. In summary, we are left with a *high dose* model with inherent limitations including:

- For carcinogens, the inability to make low dose extrapolations in the study of chronic human exposures to low concentrations;
- Dependence upon complex and poorly understood mechanisms of carcinogenesis;
- Problems in accounting for significant differences in metabolic processes (metabolization, distribution, retention and excretion) between animals and humans;
- The reliance on a *model* or *theory* to estimate the human response to a *hypothetical* dose that may be a thousand times less than the lowest dose applied in animal experiments;
- The possible creation of high-dose effects which may not occur at the doses to which people are exposed.

Dennis J. Paustenbach<sup>(23)</sup>, a recognized authority in environmental toxicology, has said in a paper presented to the 1<sup>st</sup>. International Ecological Risk Assessment Conference, Melbourne, 1995,-

“Although perhaps humbling, most toxicologists agree that they are limited in ability to estimate the risks associated with typical levels of environmental exposure based on results of standard rodent bioassays.”

The Mount Sinai School of Medicine (New York,) and the Environmental Working Group<sup>(24)</sup> have elaborated on the flaws in the high-dose/low-dose model; we quote directly - with acknowledgements - from their text:-

Timing. The timing of a dose can often determine the toxicity of the chemical. Low dose chemical exposures during fetal development or infancy are known to produce more serious toxic effects than similar exposures during adulthood for many chemicals. Lead and mercury are the classic examples, where low dose exposures in utero and during infancy cause permanent brain and nerve damage, while the same doses cause no observable effects in adults. Few high dose studies, with the exception of those required for food use pesticides, target vulnerable periods of development. Most high dose studies include only adult animals. Low dose studies almost always involve in utero exposures.

Genetic vulnerability. Some people are more susceptible to environmental contaminants because of genetic factors. For example, EPA-funded research has documented a 10,000-fold variability in human respiratory response to airborne particles (including allergens and pharmaceuticals) (Hattis, et al. 2001). This variability explains, in part, why we all breathe the same air, but not all of us have asthma attacks. Laboratory animal studies, often conducted with genetically-uniform animals, cannot reveal genetically-induced adverse effects that may occur in a small but significant percentage of a highly diverse human population.

Mechanisms. Chemicals produce a spectrum of health effects that can both vary with dose, and affect the target organ in different ways depending on dose. For instance, some chemicals produce opposite effects at high and low doses - a phenomenon called biphasic dose response. Some produce different effects at high and low doses. Some produce adverse effects at low doses, but not at higher doses. DES, a potent

---

<sup>23</sup> Dr. Paustenbach is Chief Technical Officer of ChemRisk®, a division of McLaren/Hart Environmental Engineering.

<sup>24</sup> Copyright 2005. All Rights Reserved. Environmental Working Group 1436 U St. N.W., Suite 100, Washington, DC 20009.

synthetic estrogen, has been shown to stimulate prostate growth at 0.02, 0.2, and 2 mg/kg-day, but inhibit prostate growth at doses of 100 and 200 mg/kg-day (vom Saal, et al. 1997). Perchlorate, a component of rocket fuel that contaminates drinking water, causes changes in the size of certain parts of the brain at 0.01 - 1 mg/kg-day, but not at 30 mg/kg-day (Argus 1998). Current government testing regimes do not require tests to define different effects of chemicals across a wide range of doses.

There are other problems with the assertion that all low dose exposures are safe, or trivial, simply because they are small. The chief one being that **the toxicity of mixtures is almost never studied**. Current high dose studies, like those required for pesticides used on food, are conducted with purified single chemicals. In the real world, people are exposed to low dose mixtures of several hundred chemicals. Scientists do not understand the toxicity of these mixtures, and with few exceptions are not investigating them.

In the rare cases in which scientists have studied the effects of mixtures, they have found adverse health effects. In two recent studies scientists dosed laboratory animals with a mixture of 16 organochlorine pesticides, lead, and cadmium, each applied at its individual regulatory "safe" dose, and found that the animals developed impaired immune response and altered function of the thyroid, a gland that is critical for brain development (Wade, et al. 2002a, Wade, et al. 2002b)."

## 4.4 NON-THRESHOLD EVENTS

Non-threshold events are medical outcomes for which a conventional dose-response relationship does not apply. This HHRA does not adequately address the following non-threshold events:-

### 4.4.1 Carcinogenic

Any exposure to certain carcinogens may initiate a sequence that results in cancer. Paustenbach goes on to say:-

"In short, many factors including tumor type, species, metabolism, pharmacokinetics, mechanism of action and the epidemiological experience all need to be considered when attempting to predict whether a specific chemical poses a significant hazard to humans at doses to which they might reasonably be exposed.

This helps explain why more than 400 chemicals have been found in animal studies to produce tumors, yet fewer than twenty are known human carcinogens. Even after accounting for the typical epidemiology shortcomings, it is clear that some, if not many, rodent carcinogens do not pose an equivalent cancer hazard in humans. **Although it is plausible that some carcinogens may pose a greater human hazard than that suggested by rodent studies, there are few examples where appropriate animal tests were conducted.**" (Our bolding.)

### 4.4.2 Genotoxic

Any amount of a gene-damaging (genotoxic) substance can cause damage. If such damage occurs in a germ cell, it may be inherited by successive generations.

### 4.4.3 Teratogenic

Any exposure to a reproductive toxic may cause damage; a single exposure may have lifelong effects.

#### 4.4.4 Developmental

Neonatal (birth to six weeks) and onwards: Developmental toxicants are agents that cause adverse effects on the developing child. Effects can include birth defects, low birth weight, biological dysfunctions, or psychological or behavioral deficits that become manifest as the child grows. Maternal exposure to toxic chemicals during pregnancy can disrupt the development or even cause the death of the fetus. Exposure of pregnant women to mercury, lowers birth weight and can cause severe brain damage in children. While developmental toxicity usually results from prenatal exposures to toxicants experienced by the mother, it can also result from paternal exposures. For example, the occupational exposure of men to vinyl chloride has been associated with increased rates of spontaneous abortion in their wives. Early post-natal contact with toxicants can also affect normal development. Exposure to secondhand tobacco smoke, for example, increases an infant's risk of contracting respiratory infections or succumbing to sudden infant death syndrome.

#### 4.4.5 Endocrine disruption

In addressing environmental exposures, hormonal interference - endocrine disruption is a severely understudied problem. At this time, human health risk assessments cannot consider this complex and critical issue, which includes risks presented in the early developmental stage. In a document entitled "Initiatives to Share the Burden of the Testing and Assessment of Endocrine Disrupting Chemicals"<sup>(25)</sup>, 2001, the Organization for Economic Co-operation and Development (OECD,) makes the following observations:-

“One of many issues surrounding the endocrine disruptors concern is the poor understanding of the breadth of the issue. The (Meeting) was aware that a considerable number of experts claim that the globally observed trends in early onsets of puberty/menarche and certain cancers are related to endocrine disruptors.

Furthermore, the reported increase in effects at very low dosages is still a matter of much debate and research. Current test methods are to a large extent only covering receptor-mediated effects and judgements are unclear and certainly not unanimous on issues such as **whether or not hormonal effects without obvious toxic effects should be considered as adverse**. The (Meeting) agreed that a further area of co-operation could be to exchange information on scientific meetings and involve Member countries in the Organization and participation in those meetings and forum discussions. The EC participants mentioned in this respect the plans for a European Workshop in June in Sweden to discuss progress in science, test methodologies, international co-operation and monitoring.”

(Our bolding.)

The phrase - “*whether or not hormonal effects without obvious toxic effects should be considered as adverse*” - is disturbing, but it is a clear recognition of the anticipated difficulties of identifying endocrine disrupting end-points in the absence of conventional toxic responses. Such “discretionary concepts” are common when researchers are confronted with

---

<sup>25</sup> OECD Environment Directorate: Chemicals Committee and the Working Party on Chemicals, Pesticides and Biotechnology, 32nd Joint Meeting 13-15 June 2001. This document describes the outcome of a tripartite exploratory discussion of Japan, the USA and the EC on options for a global strategy on endocrine disruptors assessment and provides recommendations for a practical approach.

unmeasurable medical outcomes. This often leads to biased research findings.

Findings from a U.S. EPA-sponsored Workshop, Environmental Health Perspectives<sup>(26)</sup> confirm a growing concern about the possible endocrine-disrupting properties of environmental chemicals:-

“Evidence has been accumulating which indicates that humans and domestic and wildlife species have suffered adverse health consequences from exposure to environmental chemicals that interact with the endocrine system. To date, these health problems have been identified primarily in domestic or wildlife species with relatively high exposures to organochlorine compounds, including 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (DDT) and its metabolites, polychlorinated biphenyls (PCBs) and dioxins, or to naturally occurring plant estrogens.

It is not known if similar effects are occurring in the general human population, but again there is evidence of adverse effects in populations with relatively high exposures. Several reports of declines in the quality and decreases in the quantity of sperm production in humans over the last four decades and reported increases in incidences of certain cancers (breast, prostate, testicular) that may have an endocrine-related basis have led to speculation about environmental etiologies. However, considerable scientific uncertainty remains regarding the causes of these reported effects. Nevertheless, it is known that the normal functions of all organ systems are regulated by endocrine factors, and small disturbances in endocrine function, especially during certain stages of the life cycle such as development, pregnancy, and lactation, can lead to profound and lasting effects. The critical issue is whether sufficiently high levels of endocrine-disrupting chemicals exist in the ambient environment to exert adverse health effects on the general population.”

## 5 PROBLEMS WITH MULTIPLE CHEMICAL EXPOSURES

The inability to deal with multiple chemical exposures to derive meaningful predictions of human health effects is a profound weakness in this and all human health risk assessments. Since 1996, the Department of Health and Human Services of the U.S. Centres for Disease Control and Prevention has been operating a research programme on multiple chemical exposures<sup>(27)</sup>, known as the National Occupational Research Agenda (NORA.)

Regarding multiple environmental stressors, NORA states that:-

“Mixed exposures may produce acute or chronic effects or a combination of acute and chronic effects, with or without latency. Other exposures in combination with certain stressors may produce increased or unexpected deleterious health effects, or they may combine or interact in the environment to create a new exposure risk. Exposures to mixed stressors can produce health consequences that are additive, synergistic, antagonistic, or can potentiate the response expected from individual component exposures. This is the complex problem that faces environmental scientists and public health officials in setting and carrying out public health policy for the general environment, consumer product and food and drug safety, and the protection of workers. Because the issue of mixed exposures affects all of these areas, it was selected as one of the priority areas of the

---

<sup>26</sup> “Research Needs for the Risk Assessment of Health and Environmental Effects of Endocrine Disruptors,” Environmental Health Perspectives, 1996 - 104(Suppl 4):715-740

<sup>27</sup> Report “Mixed Exposures Research Agenda,” - National Occupational Research Agenda (NORA.) December 2004: NIOSH Publication No. 2005-106.

National Occupational Research Agenda (NORA) to leverage collaborative research efforts for better understanding the complex interactions of mixed exposures.”

This HHRA fails to provide any balanced statements regarding its inability to deal with the lack of information on the potential health effects of mixed exposures. Real-life human exposures such as the consumption of alcohol or tobacco or the use of insect repellents, cosmetics and other consumer chemicals, and individual susceptibility add to the complexity of exposure and the resulting biological responses. New approaches are needed to identify additive, synergistic, antagonistic, or potentiation effects from multiple exposures (sequential or simultaneous,) - at the same time incorporating the body-burden profile of individual human receptors. Consequently it is incumbent upon health risk assessors to point out current deficiencies in toxicological knowledge.

What is lacking is knowledge of whole body physiology and biochemical mechanisms. Additional research is needed to develop better tools for toxicity analysis, exposure-response modeling, and physiologically based pharmacodynamic (PB/PD ) and physiologically based pharmacokinetic (PB/PK) modeling.

Addressing this problem, Paustenbach<sup>(28)</sup> points out that:-

“.....the same generalization applies to developmental and reproductive toxicants. Unfortunately, most human exposure is not to a single chronic toxicant, so the complexities of potentiation, antagonism and synergy must also be considered when the simultaneous uptake of substantial amounts of various toxicants occurs. Clearly, in light of the hundreds of industrial chemicals to which we are daily exposed, the challenge to regulators is to identify toxicants for which exposure should be limited.”

This truism poses a series of problems which the HHRA has not been able to adequately address. We question the practicability of determining the cumulative effect of these stressors in terms of resources and costs.

“Synergism” is not mentioned at all in the AIRpt document, and only once in the EIA Section 6.0 - Health:-

#### EIA Section 6.4.1.4 - Chemicals Acting in Combination

*“Since exposure to chemicals typically does not occur in isolation, some consideration should be given to the potential health risks that may be presented by chemicals acting in combination. The interaction between chemicals can take many forms, all of which are of toxicological interest and some of which may be relevant to the assessment of potential risks. The most common forms of interaction are:*

- *additivity (1 + 1 = 2)*
- *synergism (1 + 1 = 3)*

---

<sup>28</sup>

1st. International Ecological Risk Assessment Conference, Melbourne, 1995. See earlier in this Response.

- antagonism ( $1 + 1 = 0$ )
- potentiation ( $1 + 0 = 2$ )

*In general, toxicological interactions of chemicals are dependent on the chemicals present, their mode of action and their concentrations. Most synergistic interactions can only be demonstrated at high exposures, where clear adverse impacts are observed. Such interactions have not been observed or quantified at the relatively low exposures typical of those associated with most environmental situations (Krewski and Thomas 1992). Additivity is more plausible. It requires that the chemicals act through similar mechanisms and/or affect the same target tissue. For instance, the impacts of irritants will often be added if the chemicals are given in combination. Although antagonism and potentiation are known to occur with some combinations of chemicals in laboratory settings, these interactions have not been observed outside laboratory conditions and are rarely considered in risk assessments.”*

The observation that, “Such interactions have not been observed or quantified at the relatively low exposures typical of those associated with most environmental situations (Krewski and Thomas 1992)” - does not prove that the interactions do not take place. Moreover, “Additivity” is not more *plausible* in determining human health risk, rather it is easier for the health risk assessor; and in truth, - the only other approach they have. In the light of recent findings, we find this account of combined (multiple) exposure to chemicals to be unacceptable.

The HHRA thus fails to accept that synergistic reactions make health-effects predictions difficult if not impossible. An example - relevant to the proposed Upgrader emissions - is the case of benzo(a)pyrene and benzo(a)anthracene. Both are carcinogenic on mouse skin, but their potency increases one thousandfold in the presence of n-dodecane, which by itself is not carcinogenic<sup>(29)</sup>.

Concern with the health effects of multiple chemical exposures was expressed by the USEPA in year 2000. In recognition of the overwhelming lack of knowledge regarding multiple chemical exposures (not to be confused here with the idiosyncratic multiple chemical sensitivity syndrome,) the U.S. Environmental Protection Agency initiated a study to assess the impact of chemical mixtures, focusing on the mechanistic basis for chemical interactions and related health effects. In its preamble to this study the EPA made the following acknowledgment:-

“Historically, the focus of toxicity testing and mechanistic research on environmental chemicals has been on single chemicals. Over the years this approach has been critical in providing information which has led to a better understanding of the interactions of exposure and susceptibility in relation to time and has improved risk assessment. However, it is increasingly recognized that humans are generally not exposed to single chemicals and therefore knowledge of individual chemical toxicity is often inadequate for risk assessment.

Our inability to predict whether agents will act in an additive, synergistic, or antagonistic fashion at concentrations encountered in the environment and the workplace creates real

---

<sup>29</sup> Page T, A Generic View of Toxic Chemicals and Similar Risks, (1978) 7 Ecology L.Q., 207.

problems for human health risk assessment. Many examples exist where the interactions of chemicals with each other or other physical or biological agents affect health to a greater extent than would have been predicted based on toxicity of the individual components.”

The balance of the extract from the EPA statement is attached at Reference G, page 45. Assertions such as this from one of the world’s leading authorities on environmental toxicology, should encourage all community groups to demand a detailed accounting from toxicology consultants who proclaim an “acceptable risk” or a “no risk” status arising from the airborne dispersion of toxic chemicals.

## 6 RECEPTOR CHARACTERIZATION

“Receptor characterization” is a uniquely health-risk-assessment term, and it is appropriate to remember that these hypothetical receptors are human beings exposed to potentially harmful chemicals.

In discussing receptors, AIRpt Section 3.2.3.2 - Receptor Characterization, states:-

*“In HHRAs, hypothetical human receptors are generally selected that exhibit the greatest potential to be adversely affected by the release of airborne contaminants. The rationale for this approach is that if unacceptable risks are not predicted for **highly exposed** and **susceptible** receptors, unacceptable risks would not be expected for **less exposed** human receptors. Human receptors that represent such **highly exposed** scenarios were selected to be conservative.”*

The definitions “exposed” (or “highly exposed”) are quite different to the definition of “susceptible<sup>(30)</sup>.” They are two different considerations, - one relating to the level or concentration or dose of a toxic challenge, and the other entirely dependent on the physiological/biomedical/ sociological profile of the exposed person. This is a puzzling conceptual error. Additionally, the above statement rests on assumptions which are mechanistic and biomedically unsubstantiated [See paragraph 5, page 25, Multiple Chemical Exposures and paragraph 4.4, page 23, Non-Threshold Events.]

In EIA - Vol 2, Section 6.3.2 - Receptor Characterization, a receptor is further defined:-

*“Receptors are considered to have local, year-round residency and are assumed to exhibit **exaggerated lifestyle habits** (e.g., continual residence at the location of predicted ground-level air concentrations) to ensure that exposures are not underestimated. This approach also considers receptor characteristics, such as the most **sensitive gender and age class**.”*

In estimating exposures, we do not understand why “*continual residence at the location of*

---

<sup>30</sup> In fact the medical definition of “susceptible” is:- *Having little resistance to a disease or foreign protein; an individual with little resistance to an infectious disease or who is not known to have become immune to one.* (Taber’s Cyclopedia Medical Dictionary.)

*predicted ground-level air concentrations*” should be treated as an exaggerated life style; rather it would seem like a fairly normal life-style. How this provides consideration of “*the most sensitive gender and age class*” requires clarification. For example, how is the pregnant female in full time residence dealt with, in terms of estimating the health impact of exposures to herself and her fetus?

## EIA - 6.5.2 - Health Studies Associated with Local Industrial Development

*“Shell Canada conducted an HHRA for its Scotford refinery and chemicals plant in Strathcona County and its proposed oil sands upgrader. The air quality analysis showed the greatest air quality changes associated with the upgrader would occur in the immediate vicinity of the Scotford plant, again, within 5 km. The HHRA concluded that members of the public and Shell’s employees were not at risk from acute or chronic inhalation exposures, or from ingestion of locally grown food. These conclusions applied to both operating and plant upset conditions. The following 16 chemicals of concern were investigated: ammonia, benzene, carbon disulfide, carbon monoxide, carbonyl sulphide, cyclohexane, diethanolamine, ethylbenzene, ethylene glycol, hydrogen sulphide, naphthalene, NO<sub>2</sub>, SO<sub>2</sub>, toluene, 1,2,4-trimethylbenzene and xylene.”*

We would like to acquire from Shell whatever human toxicological information it has on the combined effects of these chemicals. Testing only a selection of combinations of 5 chemicals from the total of 16 - would require 243,856 experiments. A reasonable target would be to test individual chemicals, and then in binary combinations selected from the 16. This would require only 256 experiments.

While this health-effects data deficiency would apply equally to BA Energy’s multiple COPC releases, such investigations of multiple-chemical exposures are not immediately practicable. This problem contributes to the HHRA’s failure to predict the true health effects on an exposed population.

## 6.1 BODY BURDEN

References to “sensitive” and “sensitivity” are made throughout the HHRA documents, - in terms of “most sensitive,” “hypersensitive,” “unusually sensitive,” “sensitive populations,” sensitive and significant,” “sensitive human individuals,” “heightened sensitivity,” “varying sensitivity,” - without clarifying the contextual meaning. Again, there is some juxtaposition in the use of “sensitive” and *susceptible*, which adds more confusion. The HHRA fails to take into consideration one of the most significant contributions to human sensitivity to environmental chemicals, - that of existing body burden.

A recent study carried out jointly by the Mount Sinai School of Medicine (New York,) and the Environmental Working Group<sup>(31)</sup>, found 167 chemicals in the blood and urine of nine otherwise healthy adult Americans. None worked with chemicals, and none lived near chemical or petrochemical plants. Collectively, the nine subjects carried:

---

<sup>31</sup> Copyright 2005. All Rights Reserved. Environmental Working Group 1436 U St. N.W., Suite 100, Washington, DC 20009.

- 76 chemicals linked to cancer in humans or animals,
- 94 neurotoxins,
- 86 chemicals which interfere with the hormonal system (potential endocrine disruptors,)
- 79 chemicals associated with birth defects (teratogens) or abnormal development,
- 77 chemicals toxic to the reproductive system (genotoxics), and
- 77 chemicals toxic to the immune system (immunotoxics.)

Blood and urine from the nine volunteers were tested for 210 chemicals that were divided into seven basic groups. Of the chemical groups tested, the most prevalent were those contained in 24 classes of semivolatile and volatile chemicals, with 78 detected. These classes include well-known industrial solvents and gasoline ingredients, such as xylene and ethyl benzene, that are used in a variety of common products like paints, glues, and fire retardants. 48 PCBs were found (collectively) in the nine people tested; lead was found in all 9 participants, and mercury was found in 8.

We contend that existing chemical body burden in individual receptors compromises the HHRA's definition of "sensitive persons." In order to derive a true statement of exposure-health effects, we believe that the HHRA should extend its use of "baselines" beyond air emissions and exposure comparisons, to address also the body-burden-related susceptibility of individual in the exposed population.

## 6.2 PSYCHOLOGICAL IMPACT

Concerns with multiple stressors should include psychological stress. Although psychological stress is well studied, little or no information is available addressing the contribution made by additional factors, including exposure to toxic environmental chemicals. Stress is defined as a state of disharmony or threatened homeostasis. If the homeostasis is disrupted because of physical or psychological stress including social and socioeconomic stress, intricate neural and biochemical events in the brain and in the endocrine and immune systems act jointly to counter the effects of stress and to reestablish homeostasis. If homeostasis is not reset, debilitating illness can result<sup>(32)</sup>.

## 7 REGULATIONS IN THE CONTEXT OF PROTECTING HEALTH

Canadian environmental health legislation, for the most part, reflects a cautious approach to the uncertainties inherent in experimental research: guidelines and objectives are preferred in place of legally enforceable standards. This approach reflects in part, reservations regarding the statistical power attached to the testing of toxic chemicals. Most human health risk studies that fail to detect an effect of a certain substance, do not report the level of statistical power, even though it may be very low for a variety of reasons.

AIRpt - page ii - Human Health Risk Assessment ("HHRA")

---

<sup>32</sup> Ember LR, "Surviving Stress," Chemical Engineering News;" 25<sup>th</sup>. May 1998.

*“Health risks associated with operational air emissions are characterized by comparing predicted air concentrations with **regulatory guidelines considered protective of the most sensitive individuals.**”*

Using regulatory guidelines “*considered protective of the most sensitive individuals*” to characterize health risk is a flawed methodology, and to describe such regulatory guidelines as being considered protective of the most sensitive individuals is inappropriate.

“If an industrial chemical is allowed by law to be released into the environment, most people assume that it must have been tested and evaluated for its potential risks. Unfortunately, this is simply not true. Keeping chemical hazards under control requires information about what kinds of hazards each chemical poses. If the basic tests to check on a chemical's toxicity haven't been conducted, or if the results aren't publicly available, current laws tend to treat that chemical as if it were perfectly safe<sup>(33)</sup>.”

---

## EIA - Vol 2 - Executive Summary

### Human Health

*“Health risks associated with operational air emissions are characterized by comparing predicted air concentrations with **regulatory guidelines considered protective of the most sensitive individuals.**”* {There are numerous references throughout the BA Energy documents to the “most sensitive individuals.” If the reader would accept that we don’t respond to each one, it would be appreciated.}

Regulations, standards and guidelines are as equally associated with what is reasonably achievable under a given set of cost-benefit, engineering feasibility and social circumstances, as they are associated biomedically with health outcomes in an exposed individual. Since we are talking about sensitivity in the medical sense, identification of the “most sensitive individuals” is the professional domain of the environmental health physician, not the engineer, statistician or non-medically qualified health risk assessor.

In the areas of environmental health, toxic emissions control and exposure management, there are Regulations which invoke standards that are legally enforceable, and there are Guidelines which are usually based on realistically achievable goals, set against the back-drop of cost-benefit. Compliance with guidelines remains a voluntary gesture. When discussing “permissible” exposures, the EIA’s authors do not properly differentiate between regulations, standards and guidelines.

## 7.1 CANADA-WIDE STANDARDS (CWS)

In the document “Development of Canada-wide Standards Air, Soil and Water” - What Are CCME's Current Priorities?":-

The CCME adopted four major priorities in 1996/97, which are still in effect today: harmonization, air issues, the management of toxic substances, and the economic dimension of environmental management. The following is a brief summary of recent and current work in the context of the

CCME's priorities where human health is included:-

- A. "The Canada-Wide Environmental Standards Sub-Agreement provides a framework for federal, provincial, and territorial Environment Ministers to work together to address key environmental protection and **health risk reduction** issues that require common standards across the country."
- B. "Canada-Wide Standards (CWSs) can include qualitative or quantitative standards, guidelines, objectives, and criteria for **protecting the environment and reducing the risk to human health**. **CWSs will include a numeric limit (for example, ambient, discharge, or product standard)**, a commitment and timetable for attainment, a list of preliminary actions to attain the standard, and a framework for reporting to the public."
- C. "January - April: Nomination"

CCME governments nominate substances or issues that they believe are a common concern across much of Canada and could be better dealt with by coordinated action. The nomination must be accompanied by a brief report outlining the following information:

- potential impact on human health and the environment;
- level of public priority or concern; and.....

- D. "Ideally, the scientific assessment, which provides the unaltered measure of the risk to environment and **human health**, is examined in the context of technology options which can vary the exposure, and socio-economic concerns which analyze costs and benefits of different risk reduction scenarios."

EIA-Vol 2 - Section 6.4.2.2 - Hazard/Toxicity Assessment (pp 6-17)

The authors quote Pandey and Nathwani, 2003:-

*"The CWS represents a balance between the protection of public health (i.e. avoided mortality) and the costs associated with PM reduction (i.e. social impacts, economic impacts and technical viability.)"*

The definition of public health extends beyond the index of "avoided mortality," which presumably means the prevention of deaths from environmental toxics.

The CCME and the EIA statements above confirm that the finalization of standards for emissions and for ambient air concentrations is only partly focused on human health effects, - the other part being dependent on the feasibility and costs of mitigation. There is no indication that the putative standards (CWSs) or guidelines will incorporate a special consideration for "*the most sensitive individuals*," as stated in the Executive Summary of BA Energy's AIRpt.

The numerous references which the BA Energy documents make to "health-based" limits

(Regulations, Standards, Guidelines) provide the affected (exposed) community with a false sense of safety and security regarding exposure to toxic emissions.

## 8 LIMITATIONS OF THE HUMAN HEALTH RISK ASSESSMENT

The issue of regulatory authorities failing to act because of a “lack of full scientific certainty” is now a global item of debate:-

In its Charter on Industrial Hazards and Human Rights, the Permanent Peoples' Tribunal<sup>(34)</sup> has the following to say regarding hazardous exposure in the workplace:-

Article 23, paragraph 2 - “Right to enforcement of health and safety laws”:-

*All workers have the right to adequate planning control legislation in compliance with the precautionary principle, so that where there are threats of serious or irreversible damage, lack of full scientific certainty shall not be used as a reason to postpone cost-effective measures to prevent hazards and environmental degradation.*

The principle is equally applicable to people whose living environments are threatened by the industrial release of hazardous materials into the environment. We contend that this HHRA should be accompanied by an advisory that the HHRA's findings must be interpreted in the light of a *lack of full scientific certainty*.

By *human health risk assessment* we are describing the attempt to derive a statement about the predicted effects on the health status of individuals in a defined community due to environmental health stressors, mainly toxic chemicals released from nearby and identifiable emission sources. Such emissions may arise from a collection of point sources as is the case in the Alberta Industrial Heartland region. However, the alternative is a technology-based approach with the objective of containing the hazard completely, rather than allowing it to disperse in “acceptable” measured amounts. The accurate identification of hazards is required. As the ultimate solution, some have argued that bans are the only successful means to remove environmental risks. Bans on the chemicals DDT, PCB, lead in gasoline, and asbestos, are examples of success stories.

### 8.1 PROBLEMS IN RESEARCH METHODOLOGIES

Environmental health risk assessments rely predominantly on research findings from experiments carried out on laboratory animals, or on the findings from epidemiological studies in the human population. Increasingly, areas of uncertainty are being identified in the methodologies used in

---

<sup>34</sup> The Permanent People's Tribunal (PPT) Charter on Industrial Hazards and Human Rights is not an official document, but a people's statement, derived directly from the collective experience of those who have been forced to live with the consequences of industrial hazards. The Charter is based on a series of public hearings held on the topic of industrial hazards and human rights. The hearings were held in New Haven (USA, 1991), Bangkok (Thailand, 1991), Bhopal (India, 1992), and London (UK, 1994.)

these studies, which brings into question the validity of current standards applied to environmental exposures.

### 8.1.1 Animal studies

Critics of whole-animal methods claim that species variability invalidates the animal model. In determining health effects, health risk assessments depend heavily on the extrapolation of findings from animal to human, even though, in the context of whole-system toxic response, species variability weakens considerably the plausibility of the permissible exposure limits applied in assessments.

The whole-body response problem applies equally to *in vitro* studies. Although recent advances in *in vitro* techniques may provide an additional source of biomedical knowledge, the relative simplicity of non-whole-animal testing (*in vitro*) has limitations. Cells or tissues in culture cannot predict the effect of a toxic on a living organism with its complex interaction of nervous, endocrine, immune, and hematopoietic systems. *In vitro* systems can predict the cellular and molecular effects of a drug or environmental toxic, but in the final analysis, only a human can exhibit the complex physiological response of the whole organism.

### 8.1.2 Epidemiological constraints

Epidemiological studies may often have low statistical power for a number of reasons. For example sample sizes are usually low, and controlling external (confounding) factors such as exposure to toxics other than the one being studied, is often difficult. Many studies are retrospective, - that is they attempt to evaluate cause-effect based on historic exposure for the study population (often exposed workers,) where such data can be highly unreliable.

The long latency periods characteristic of most carcinogens, present additional restrictions on epidemiologists in their follow-up of outcomes. Studies often require that the cohort be followed for periods of 20 - 30 years, during which time many study individuals may leave for a variety of reasons. The sample size is thus diminished, and hence the statistical power of the study. This reduces the probability of detecting a "true" effect. The confounding effect of concurrent exposures often make it difficult to link observable health outcomes with the suspect carcinogen.

### 8.1.3 Uncertainty in Statistical Procedures

Faced with uncertain information from experimental or observation results, researchers will process their data in a manner which will minimize the probability of *incorrectly concluding that there is an effect (e.g. a predefined medical end-point) when one actually does not in fact exist*. In the interpretation of statistics, this is referred to as a Type I error. This could lead to a "false positive" conclusion, eventually causing the promulgation of exposure regulations which are unnecessary. It is understandable that authorities, perhaps with a vested interest in the commercial application of the chemical, will take great care not to make a Type I error when evaluating their experimental results. However, the greater the constraints put in place to avoid a Type I error, the more opportunity is opened up to make a Type II error, - which is to

conclude that *there is no effect when in fact an effect does exist*.

Clearly, there is room for subjective judgement in such hypothesis-testing, and this can create a significant impact, because the conclusions are translated eventually into permissible human exposures, or permissible environmental concentrations. The potential flaws in this process, and the problems it poses for environmental legislation, are extremely well analyzed by R. Michael M'Gonigle<sup>(35)</sup> *et al* in the Osgoode Hall Law Journal, 1994.

#### 8.1.4 Low-level adverse effects.

Evidence regarding the effects of several classes of chemical compounds at very low levels of exposure is slowly evolving. Data describing the endocrine disrupting ability of certain synthetic compounds has shown that such very low level exposures, during certain periods of development, can cause wide ranging reproductive and developmental effects.

Contrary to the expected pattern, research has shown that some of these effects occur at low levels of exposure and not at high levels. If adverse effects are being observed at very low levels of exposure, there is reason to believe that similar effects may occur at even lower (but still unmeasurable levels of exposure.) Such may be the case with endocrine disruption and immuno-disruption.

## 8.2 RISK ASSESSMENT AS A DECISION-MAKING TOOL

In its broadest sense, risk assessment is a *decision-making* tool that started to be widely applied in the USA during the incumbency of President Carter. At its best, human health risk assessment is an honest attempt to find a rational basis for making control and regulatory decisions, based on an analysis of the available scientific evidence. In theory it is still an attractive ideal - to make rational decisions based on scientific evidence - because in principle this should allow diverse parties (stake-holders) to agree on what needs to be done. However, there appears to be a lack of scientific evidence. Twenty to twenty five years of actual practice reveal serious flaws in the plausibility of the numerous inferences and assumptions that have to be invoked in order to deal with the uncertainties presented by this lack of scientific evidence.

Ideally, the paradigm calls for environmental contamination and human health impact to be viewed through the twin lenses of engineering and traditional toxicology. Traditional toxicology maintains that "the dose makes the poison" - meaning that everything is poisonous at a high enough dose, and that one can prevent poisoning by restricting the dose. Both the engineer (who creates the problem) and the toxicologist (who tries to define its health impact) are forced to lean heavily on concepts of "probability" provided by the statistician. Both seek to develop a numerical formula that will give the desired result time after time; predictability is the "Holy Grail." Unfortunately, that is not the way human physiology works. Clearly, the traditional dose-response model is no longer adequate.

---

<sup>35</sup> M'Gonigle, R Michael, Jamieson, T Lynne, McAllister, Murdoch K, and Peterman, Randall M; "Taking Uncertainty seriously: From Permissive Regulation to Preventive Design in Environmental Decision Making," Osgoode Hall Law Journal, Vol. 32, 1994. Osgoode Hall Law School of York University.

The necessary biomedical and socio-medical data are not available. The traditional toxicological assumptions do not hold up in the face of multiple exposures and the endocrine disrupting, neurotoxic and immuno-disruptive characteristics of toxic doses well below any hitherto derived no observed adverse effect levels (NOAEL.) For many environmental toxics, there is no safe dose.

Many of the problems discussed in this Response regarding the failure of HHRA to adequately address human health effects because of a lack of toxicological knowledge, are also considered in a report by the Environmental Defence Fund, 1997, entitled "Toxic Ignorance." We attach a copy of this report at Reference F, page 42. Since that time, despite considerable additional research effort, we are still lacking critical information on multiple chemical exposures and chronic low-dose scenarios such as endocrine disruption, immunosuppression and neurotoxicity.

### 8.3 ALTERNATIVE APPROACHES - HAZARD BASED

Because of significant gaps in toxicological and human health effects knowledge, the choice between risk-based and hazard-based approaches becomes one of prudence and politics. A hazard based approach is essentially precautionary in nature, and provides the basis for taking preventative measures with respect to substances due to their potential to cause harm to the environment or human health. Risk-based approaches, on the other hand, are fundamentally reactive in nature, and essentially wait for absolute proof of harm to the environment or human health before action can be taken. In this context, it is hardly surprising that economic interests that produce potentially toxic substances prefer the more conservative, risk-based approach to the precautionary hazard-based model.

Because of uncertainties and questionable assumptions, some European countries have rejected the standard health risk assessment model upon which this HHRA is based, while other countries have compromised by adopting modified versions of the protocol.

#### 8.3.1 United Kingdom

In the U.K., risk-assessment methods have not been used to establish an environmental agenda or to regulate specific chemicals. Regulatory agencies do not use risk-assessment models to generate a probability for the risk of cancer from exposure to certain chemicals. U.K. regulators place little reliance on the quantitative assessment of carcinogens because they believe that the statistical models used to extrapolate dose-response effects from animals to humans are not valid.

Papers list several reasons for U.K. skepticism of commonly-used U.S. dose-extrapolation models. First, a linear low-dose model has yet to be validated. Second, the bioassay data used to derive a low-dose model of chemical carcinogenicity are incomplete. Third, the low-dose models are based more on mathematical assumptions than established biochemical mechanisms; consequently, risk estimates vary widely depending on the model used. Finally, models give an unjustified impression of precision, given the approximations and assumptions upon which they are based.

The regulation of chemical carcinogens in the U.K. is based on mechanistic considerations.

For example, if a chemical acts by a genotoxic mechanism, regulators assume, as a matter of prudence, that the compound does not have a threshold.

Chemicals displaying genotoxicity are evaluated using expert judgment and a weight-of-the-evidence approach. In evaluating such compounds, experts consider all available evidence (including human data, animal data, mutagenicity data and structure/activity relationships). If they conclude that the compound should be considered a potential human carcinogen that acts by a genotoxic mechanism, they recommend action to reduce levels to as low as is reasonably practical or to eliminate it entirely.

### 8.3.2 Germany

Under European Community (E.C.) pressure, quantitative risk assessment has recently been adopted. Previously, regulatory authorities did not attempt to quantify the risk from exposure to carcinogens or other toxic substances because the inherent acceptance of a quantitative risk estimate did not comply with principles established by environmental laws. The notion of allowing any degree of risk to humans violates the principle of eliminating any danger to public health, - a basic tenet of German environmental regulations.

Thus, to date risk assessments have infrequently been used in Germany to resolve important environmental issues. As the need for a quantitative form of risk assessment has become increasingly necessary - to facilitate the expansion of European industries and associated chemicals use - regulators have conducted several surveys of the methodologies used by other countries and have selectively adopted some of them. Believing that a case-by-case approach to assessing chemicals leads toward more accurate estimations of risk, the German regulators have advocated using flexible, rather than policy-driven, approaches.

Although German authorities do not widely practice risk assessment, known human carcinogens are strictly regulated. To date, known human carcinogens have been subjected to stringent regulations focusing partially on the best available technology (BAT.) It is also commonplace for decisions concerning the regulation of chemical carcinogens and other hazardous chemicals in Germany to be made by multipartite expert committees on a case-by-case basis.

### 8.3.3 The Netherlands

In the Netherlands regulators use the term quantitative risk assessment (QRA) as synonymous with low-dose modeling conducted by risk assessors in the U.S. The QRA approach to low-dose modeling is used to estimate the probability of risks to human health from carcinogens that have been definitively categorized as genotoxic. The method is currently used by all Dutch agencies, but the model varies somewhat based on knowledge about the mechanism of action of the chemical.

In the Dutch risk-assessment process, scientists initially evaluate a chemical to determine its genotoxicity. When it is impossible to eliminate completely the risk of exposure to a genotoxic carcinogen, the Dutch approach relies upon a very simple linear extrapolation model to determine a dose-response value for human exposure.

#### 8.3.4 Denmark

Regulators in Denmark employ risk-assessment techniques only to a limited extent when determining exposure standards for carcinogens. In cases where new chemicals may be substituted for a suspected carcinogen, an assessment is required to examine whether exposure can be eliminated effectively. Low-dose extrapolation models are used to estimate risk when a non-threshold genotoxic carcinogen cannot be replaced by another chemical.

In controlling toxic substances, authorities in Denmark recognize different carcinogenic mechanisms and use the safety-factor approach for non-genotoxic carcinogens and non-carcinogens. The basic toxicological data used to generate exposure standards are generally the same across the various regulatory agencies, but the manner in which the data are used differs according to the problem being addressed. Danish regulators also use a case-by-case approach when evaluating data for a toxic substance.

#### 8.3.5 Sweden

The Swedish EPA evaluates carcinogens using a case-by-case approach in which each chemical is assessed individually, as opposed to the more generic approach common in U.S. regulatory agencies that follow fairly strict guidelines.

Swedish regulators use a weight-of-the evidence approach to evaluate a chemical's carcinogenic potential. Like most other European countries, genotoxic carcinogens are typically regulated to ensure the lowest possible levels of exposure. Non-genotoxic chemical safe levels exposures are identified from a NOAEL or lowest-observed-adverse-effect level (LOAEL) and a safety factor applied based on the level of uncertainty in the available information.

### 8.4 THE PRECAUTIONARY PRINCIPLE

The Precautionary Principle states that - *"When an activity raises threats of harm to the environment or human health, precautionary measures should be taken even if some cause and effect relationships are not established scientifically."* Defining the hypothetical "most sensitive receptor" and building in arbitrary "conservative" factors to reference doses extrapolated from animal experimentation, are not health-based precautionary measures.

The CCME has problems coming to grips with this idea and its implications for the option of *no exposure*. For example in the Canada-wide Environmental Standards Sub-agreement issued by the CCME (no date given on the document,) the principle is invoked thus:-

***Precautionary Principle:*** *Where there are threats of serious or irreversible environmental damage, lack of full scientific certainty shall not be used as a reason for postponing the development and implementation of standards.*

Full scientific certainty is rarely achievable, - science changes as more knowledge comes to light; particularly in the biomedical sciences. Acceptable standards could be developed, but only in the

context of multiple chemical exposures, a full understanding of pharmacodynamic mechanisms and accurate receptor profiling. Human exposure standards taking one chemical at a time simply do not reflect the modern environmental reality. An alternative to risk analysis based on elusive permissible exposures and doses, is the option of *no exposure*. It is precisely because we lack full scientific certainty that exposure standards become invalid, and *no exposure* becomes the only safe option.

# APPENDICES

- A. REFERENCE - CANADA-WIDE STANDARDS FOR PARTICULATE MATTER (PM) AND OZONE; JUNE 5-6, 2000, QUEBEC CITY, CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT (CCME)
- B. REFERENCE - CANADA-WIDE STANDARDS FOR DIOXINS AND FURANS; APRIL 30 - MAY 1, 2001, WINNIPEG, CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT (CCME)
- C. REFERENCE - CONSISTENT TERMINOLOGY FOR THE DEVELOPMENT OF CANADA-WIDE ENVIRONMENTAL STANDARDS
- D. REFERENCE - EVALUATION OF RISK-BASED APPROACHES IN ENVIRONMENTAL GUIDELINE AND STANDARD SETTING - CANADIAN COUNCIL OF MINISTERS OF THE ENVIRONMENT

E. REFERENCE (ATTACHED) - CONSISTENT TERMINOLOGY FOR THE DEVELOPMENT OF CANADA-WIDE ENVIRONMENTAL STANDARDS (CWES.)

Part 1  
Executive and Policy Summary

J. Bryan Kemper, Dr. Warren Kindzierski, Dr. Connie Gaudet, Dr. Dwayne Moore  
Kemper & Associates Inc.  
Consultants in Environmental Policy and Sciences October 1997  
Proposed Definitions

Canada has tended to use common terminology for the past 15 years within CCME, and practitioners and clients are generally well versed in this terminology. Some exceptions exist and rectifying these would be a significant step forward. Once accepted these changes should be incorporated into the proposed publication of multi-media guidelines by CCME in 1998. In the past the Canadian framework has clearly distinguished between the national science based values (guidelines) and site-specific management values (objectives).

The request by ministers to develop national 'standards' to ensure protection of Canadians and their environment bridges the traditional separation of national guidance in the form of guidelines and provincial development and application in the form of site-specific objectives. This request has the potential benefit of triggering an evaluation and alignment of processes and their products to improve environmental management, increasing public participation at key points, and increasing awareness and understanding among Canadians.

The concept of Canada-wide Environmental Standards is new, and we have attempted to define and coordinate the following terms with the goal of incorporating the scope and intent of the Standards Sub-agreement. Although we considered adapting wholesale changes in nomenclature, the outline below minimizes the number of changes that need to be made, thereby avoiding confusion among jurisdictions and their clients. We propose the following standardized definitions which are an extension of current national usage.

Criteria : the scientific data used to derive ambient chemical specific limits for specific media.

Guideline : a numeric concentration or narrative statement which provides general guidance to support and maintain a designated use. Formerly - Guideline: a numeric concentration or narrative statement recommended to support and maintain a designated use.

Objective : a numeric concentration or narrative statement recommended to support and maintain a designated use at a specific site.

Standard : an objective that is recognized in enforceable environmental control laws of a level of government, or in formal written agreements among jurisdictions. Formerly - Standard: an objective that is recognized in enforceable environmental control laws of a level of government.

Canada-wide Environmental Standard: a (proposed) negotiated quantitative or qualitative standard, in the form of a guideline or objective or commodity or waste limit, for chemicals of national importance, which jurisdictions agree to achieve over a specified time period through their environmental management regimes.

It is necessary to amend the previous definition for guidelines because they have come to mean different things for various media. Because guidelines are the result of the risk assessment process, and do not include risk management, they are not tailored to specific sites. They should, therefore, be considered as general guidance based on scientific data without the addition of site-specific modifying factors, socioeconomic, technical or socio-political considerations. The current protocols for water quality, sediment quality, tissue quality, and the ecosystem portion of air quality produce values that are, by these conventions, general guidance on protective levels which should not be applied without further consideration of specific site conditions. The definition of standards has been modified only slightly to accommodate the intent of the Standards Sub-agreement, which in our interpretation calls for binding adherence to nationally applicable values that incorporate site-specific considerations, and that jurisdictions agree to meet, over time, through written implementation agreements.

F. REFERENCE (ATTACHED) - TOXIC IGNORANCE -REPORT BY THE ENVIRONMENTAL DEFENSE FUND - 1997

The full report (with excellent references) is available from:

Environmental Defense Fund ([www.edf.org](http://www.edf.org)),  
257 Park Avenue South  
New York, NY 10010, USA

In the early 1980s, the National Academy of Sciences' National Research Council completed a four-year study and found that 78% of the chemicals in highest-volume commercial use had not had even "minimal" toxicity testing. Thirteen years later, there has been no significant improvement.

Chemical safety can't be based on faith. It requires facts. Government policy and government regulation have been so ineffective in making progress against the chemical ignorance problem, for so long, that the chemical manufacturing industry itself must now take direct responsibility for solving it. It is high time for the facts to be delivered. Step one toward a solution lies in simple screening tests, which manufacturers of chemicals can easily do. All chemicals in high-volume use in the United States and Canada should long since have been subjected to at least preliminary health-effects screening, with the results publicly available for verification. There is already international consensus on just what needs to be done as a first step. A model definition of what should be included in preliminary screening tests for high-volume chemicals was developed and agreed on in 1990 by the U.S., Canada and the other member nations of the Organization for Economic Cooperation and Development, with extensive participation from the U.S. chemical manufacturing industry.

This report documents that, today, even the most basic toxicity testing results cannot be found in the public record for nearly 75% of the top-volume chemicals in commercial use.

In other words, the public cannot tell whether a large majority of the highest-use chemicals in the United States pose health hazards or not much less how serious the risks might be, or whether those chemicals are actually under control. These include chemicals that we are likely to breathe or drink, that build up in our bodies, that are in consumer products, and that are being released from industrial facilities into our backyards and streets and forests and streams.

In the early 1980s, the National Academy of Sciences' National Research Council completed a four-year study and found that **78% of the chemicals in highest-volume commercial use had not had even "minimal" toxicity testing.** Thirteen years later, there has been no significant improvement.

Chemical safety can't be based on faith. It requires facts. Government policy and government regulation have been so ineffective in making progress against the chemical ignorance problem, for so long, that the chemical manufacturing industry itself must now take direct responsibility for solving it. It is high time for the facts to be delivered.

Step one toward a solution lies in simple screening tests, which manufacturers of chemicals can easily do. All chemicals in high-volume use in the United States should long since have been subjected to at least preliminary health-effects screening, with the results publicly available for verification. There is already international consensus on just what needs to be done as a first step. A model definition of what should be included in preliminary screening tests for high-volume chemicals was developed and agreed on in 1990 by the U.S. and the other member nations of the Organization for Economic Cooperation and Development, with extensive participation from the U.S. chemical manufacturing industry. All that is missing is the industry's commitment to act, without waiting any longer.

Analysis of the extent of health-hazard information on chemicals is rare. In 1980, the National Academy of Sciences' National Research Council began an extensive study to determine what need there was for additional toxicity testing. It concluded in 1984 that 78% of the chemicals in U.S. commerce with production volume of greater than one million pounds per year lacked even "minimal toxicity information." This report is the first public attempt to update the 1984 findings on the extent of toxicity testing for chemicals in U.S. commerce.

The chemicals addressed in this report do not include all, or even most, of the approximately 75,000 chemicals that the U.S. Environmental Protection Agency lists as being made in the U.S. in 1996. This report covers only those chemicals that are produced in or imported into the U.S. in amounts greater than 1 million pounds per year (high-production-volume chemicals), as documented by the U.S. Environmental Protection Agency. Because EPA's list does not include certain categories of chemicals, such as food additives, drugs, and pesticides, this study excludes those materials.

This report uses the same approach as the 1984 National Research Council report, analyzing the availability of hazard identification data (i.e., toxicity testing results) by examining chemicals in a randomly selected representative sample and then extrapolating the sample results to all high-production-volume chemicals.

As in the 1984 report, the results from the sample are extrapolated to all 3,000 high-production-volume chemicals. This approach almost certainly overstates the degree of knowledge about hazard information for this larger group of chemicals, as explained above, and thus understates the actual degree of ignorance.

In measuring whether a chemical qualifies as having hazard identification data available, this report takes the internationally accepted definition of a minimum screening information data set that was created by the Organization for Economic Cooperation and Development (OECD) Chemicals Program in 1990. It focuses only on the portion of the definition that covers screening for human health effects ("Toxicological Data"). If enough data to meet this portion of the OECD minimum screening requirements were found to be available for a particular chemical, it was assumed that an informed preliminary judgment about that chemical's potential human health hazards could be made.

There is international consensus that this data set represents the minimum amount of data required for a preliminary assessment of human health hazard of a chemical. However, it is important to note that the minimum screening information data set generally does not include enough data to conduct a comprehensive health risk assessment. It is only a starting point, and it is no substitute for the risk assessment that is called for under most major toxic chemical control laws.

The analysis in this report uses only information from publicly available sources. For some chemicals there is undoubtedly private information as well: for example, tests on specific chemicals that major manufacturers have performed, or paid for, which to date have not been made available to the public. A specific example is discussed below at the end of this chapter. However, a report like this has no way to evaluate private data. More importantly, for purposes of assuring the public about the safety of specific chemicals, non-public data are of no real value. To rely on them is to ask the public to take chemical safety on faith the exact opposite of the intent of modern toxic chemical control laws passed by Congress since 1970.

The actual facts are particularly hard to establish for chemicals with no hazard identification data because, almost inevitably, such chemicals are not tracked or monitored. Proving whether people are being exposed to such chemicals or not is therefore extremely difficult. **Nearly three quarters (71%) of the sampled high-priority chemicals do not meet the minimum data requirements for health hazard screening** set by the Organization for Economic Cooperation and Development Chemicals Program.

Thus, for the group of chemicals with the highest volume use in the United States, there is no basis for assurance that their use does not pose health risks to the American people, whether that assurance is offered by industry or by government.

Lack of meaningful assurance is not the same as proof of harm, of course. It is only proof of ignorance. But ignorance means that any conclusion about safety is unfounded. A system that relies on ignorance has no basis for inviting public confidence that chemical risks are under control even from the chemicals being sold and used in the largest amounts. For approximately 75% of those chemicals, minimum critical information is lacking. **Of the potential health effects ("endpoints") that would be**

**covered by minimum screening tests, a majority of chemicals in the high-priority sample have been tested for only two: genetic toxicity (i.e., ability to cause mutations) and developmental toxicity (e.g., ability to cause birth defects).**

Reproductive toxicity tests have not been conducted on 53% of high-priority chemicals. Carcinogenicity tests have not been conducted on 63% of high-priority chemicals. Neurotoxicity tests have not been conducted on 67%. Immunotoxicity tests have not been conducted on 86%. **Endpoints of particular concern for evaluating impacts on children (such as postnatal performance and developmental neurotoxicity) have not been assessed for more than 90% of high-priority chemicals.**

Exposure to these high-priority chemicals can occur from various sources, including from use of consumer products, from indoor or outdoor air, and in the workplace. In the workplace, use of chemicals can result in regular occupational exposures to production workers. Workplace use may also lead to ongoing exposures to the general public if these chemicals are released to the environment or are included in consumer products. To assess the safety of chemical use in such contexts, it is important to have data from chronic toxicity tests; i.e., tests investigating the effect of exposure to the chemical over substantial periods of time. More than half of the sampled high-priority chemicals have not been tested for any form of chronic toxicity. For acute toxicity, by contrast, testing is much more likely to have occurred: over 90% of the sampled chemicals have been tested for some form of acute toxicity (usually death).

Most toxicity testing has not focused on the route of exposure that is most relevant for assessing human health risks. Both for the general public and for workers, the predominant route of exposure to many compounds is likely to involve breathing contaminated air (inhalation exposure). Yet more than two-thirds of high-priority chemicals have not been subjected to chronic inhalation tests that evaluate long-term air exposures to a toxicant.

These results, for high-priority chemicals as a whole, are dismayingly meager. But an observer might raise the possibility that, despite their priority for regulators and their high volume of commercial use, the chemicals under study might not be representative of those actually out in the environment. Perhaps, for example, chemicals we are most likely to be exposed to outdoors have been tested, even if other high-volume chemicals have not. To test this possibility, EDF looked only at the chemicals in its sample that are reported on the national Toxics Release Inventory as being released by industry into the environment, a total of 47 chemicals.

Even of the sampled chemicals that are known to be released into the environment, 51% do not meet minimum screening requirements for health hazard identification. This result is particularly striking, since to be included on the Toxics Release Inventory a chemical must already have been found to be "toxic" on the basis of some evidence of harm. This finding illustrates an important point: that even with chemicals for which one health hazard may have been found, we are likely not to have even a preliminary idea whether other health hazards are also presented.

For the portion of the sampled chemicals for which we have especially strong reasons to anticipate human exposure, the results are similar. The U.S. EPA has established criteria for assessing the exposure potential of chemicals based on bioaccumulation and persistence; i.e., whether they are likely to build up in our bodies, and whether they are likely to last for a long time in the environment. Looking only at sampled chemicals with "high" and "medium" exposure potential, a total of 42 chemicals, 57% do not meet minimum screening requirements for health hazard identification. This finding means that chemicals with special likelihood of exposure have not been tested to any significantly greater degree than other chemicals. Just because regulators can identify chemicals with special likelihood of exposure does not mean that better testing for their potential health effects has yet occurred, or that the results of any such testing are publicly obtainable.

ENVIRONMENTAL DEFENCE FUND - END

## G. REFERENCE (ATTACHED) - USEPA - COMPLEX CHEMICAL MIXTURES

*Quoted from:-*

NATIONAL CENTER FOR ENVIRONMENTAL RESEARCH  
U.S. Environmental Protection Agency  
National Institute for Occupational Safety and Health  
National Institute of Environmental Health Sciences

FY 2000 Science to Achieve Results (STAR) Program  
Opening Date: April 10, 2000 Closing Date: July 10, 2000

### COMPLEX CHEMICAL MIXTURES

The magnitude of the problem is immense. In our daily living, mixtures of chemicals are ubiquitous in the air we breathe, the food we eat, and the water we drink. There are over 80,000 existing chemicals on the Toxic Substances Control Act inventory. Each year an additional 2,000 chemicals are added. Humans are exposed to thousands of agents in various combinations every day in the home, the ambient environment, and the workplace. Many of those chemicals are members of chemical classes that have similar or intersecting modes of action. Furthermore, chemicals with different modes of action may still affect the same organ or tissue and thus interact with each other.

The task of testing these chemicals is formidable. Chemical mixtures vary significantly in their composition; some contain two or three chemicals of a similar class, whereas more complex mixtures can be composed of hundreds of chemicals representing a variety of organic and inorganic classes of chemicals, with varying degrees of toxicity and different modes of action. Changes in the toxicokinetics and/or toxicodynamics of one chemical by another may alter the resultant toxicity from predicted values. Though changes in toxicity have been described for simple binary mixtures, unraveling the effects of complex mixtures has not been achieved.

Increasingly, regulatory agencies such as EPA face the formidable task of assessing risk to mixtures of chemicals when information is available on only the component chemicals in isolation. The task is to move beyond the simplistic assessment of single chemicals to consideration of aggregate and cumulative risk, as required by the Food Quality Protection Act of 1996. In addition, standards for limits on air and water pollutants must also consider the impact of mixtures of chemicals.

EPA - END