UNHEALTHY EFFECTS OF UPSTREAM OIL AND GAS FLARING

(US SIC=1311 and US SIC 1389)

A REPORT PREPARED FOR

SAVE OUR SEAS and SHORES (SOSS)

FOR PRESENTATION BEFORE THE PUBLIC REVIEW COMMISSION
INTO
EFFECTS OF POTENTIAL OIL AND GAS EXPLORATION, DRILLING ACTIVITIES
WITHIN
LICENCES 2364, 2365, 2368

SYDNEY NS

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Summary

In this report we have shown that:

- A large number of hydrocarbons are produced when waste oil-gas and oil-gas-water solutions are flared
- Flaring is inefficient with combustion being most affected by ambient winds and heating value of the fuel. Inefficient burning releases raw fuel
- The historical record shows that the volume of gas flared in Alberta is immense
- The flares examined by Strosher represent operations in the lowest range of volumes. Volumes range upward to over 10 million cubic meters per day.
- Benzene measured by Strosher will be present in enough concentration to pose a measurable risk to inhabitants out to 5 km from the flare, after taking account of the reduced combustion efficiency. Increasing the volume flared by a factor of 10 increases the amount of risk and extends the risk further. Raising the flare to 50 m above ground disperses the contaminants more widely but retains the risk.
- All of the hydrocarbons released by the minimal flare examined by Strosher are present in amounts below their threshold for smell. That is they are present but we have no warning. An ultra-conservative toxicological approach by Jones et.al. is able to estimate potentially safe lower limits. These are several thousand times less than the threshold where the chemical can be smelled.
- In a sour gas flare many reduced sulfur species are formed. Several including hydrogen sulfide and carbon disulfide are potent toxic chemicals. Exposure to H_2S at concentrations below the level it can be smelled is associated with spontaneous abortion. CS_2 is a potent peripheral neurotoxin.
- When sea-water containing chloride is introduced into the flare containing organic matter dioxins form. Chloracne is caused by dioxins. Chloracne-like dermatological conditions are widely found in Western Canada.
- Census Divisions (CD) which contain extensive flaring activity also show elevated median cancer incidence rate ratios greater than 1
- Within the CD's which show median rate ratios > 1 a number of specific cancer sites have elevated median rate ratios >1. These include leukemia, thyroid, pancreas, bone and connective tissue, unknown primary site in both male and female. And invasive cervical in women. Leukemia is caused by benzene. Thyroid is caused by radioactivity and radioactivity accompanies all flaring. Soft tissue sarcomas are caused by dioxins. Unknown primary is recognized as resulting from a multi-focal exposure.
- Traumatic Stress leads to several autoimmune diseases including MS, rheumatoid arthritis, thyroid disease, type II diabetes. A common source is unexpected flaring
- Chronic cyclical chemical exposure induces chemical sensitivity.

Introduction

We have been asked to report on the human health issues associated with living in proximity to oil and gas flaring. We associate flaring in the oil and gas industry with all aspects of exploration, testing, proving and development in the petroleum industry both here in Canada and worldwide. The issues we discuss here are principally associated with industrial activity defined by US SIC = 1311 and SIC = 1389. Flaring in refineries (SIC = 2911) involves a great many of the same components but is not considered here unless noted. Insofar as this does not directly apply to the mandate of this Hearing we ask the Commissioner's indulgence to hear these matters because they will be relevant as soon as any aspect of exploration, testing, proving and development occur.

Our experience and knowledge is the ability to reliably predict how the quality of life will be impacted by many types of industry, and to reliably relate the association between adverse health effects and industrial pollution.

We worked as a scientific contractor and medical geographer for Health Canada from 1987 to 1998. We were involved with the development of WHMIS and the methodology to reveal chemical risk associated with proprietary substances. We developed a National inventory of municipal and industrial waste disposal sites (WDS) and participated on a CCME committee to evaluate and remediate human health risks from exposure to WDS as Secretary.

We developed a GIS-style system to evaluate lifetime chronic exposure from residential exposure to point sources with an end point of cancer. We have published on the subject as well as preparing many reports for the Federal Government. [1] [2] [3] [4] Our focus has

[1] Argo J Downwind risk of all-cancer relative risk about a point source: Single source with reactive and unreactive plumes Env. Sci. Technol. 2000, 34, 4214-4220

[2] Argo J Site-specific *a priori* cancer risk estimator for mixtures and multiple source: Net excess relative odds. Env. Sci. Technol 2000, 34, 4208-4213

[3] Argo J Retrospective exposure assessment with emission inventories: A new approach to an old problem. Environmentrics 1998, 9, 505-518

been on chronic exposure assessment of residents and disease stemming from chronic exposure, specifically cancer. We describe the system as one to evaluate "how an individual's present state of health is affected by their previous residences". Our design succeeded in eliminating the epidemiological confounder, "mobility", and now allows us the unique advantage to identify and evaluate original exposure. We began work with other diseases associated with chronic exposure including chemical sensitivity, and autoimmune diseases including Multiple Sclerosis, and Type II diabetes.

Since about 1995 we have been bringing "science and scientific support" to residents affected by oilfield exploration and development in SIC =1311 in Western Canada. In 1998 we provided the first scientific support as an expert witness for complainants at Hearings before the Alberta EUB.

To prepare for this support role we developed a broad understanding of the adverse impact of chronic exposure from multiple flaring discharges on the health of people who live and work in proximity to the industry. Proximity can be from 0.2 km up to 35+ km. In Alberta in 1996 there were approximately 776,000 persons living in this proximity range and potentially chronically exposed on a daily basis to flaring emissions or about 23% of the population. A smaller proportion of the population appears to be affected in Saskatchewan and western Manitoba where the industry is less developed.

We observe the same adverse health effects in Saskatchewan and Manitoba as in Alberta. We have observed many of the same adverse health effects from off-shore oil and gas flaring in Eastern Brazil and from off-shore development near Los Angeles CA.

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[4] Retrospective Exposure Assessment and Enhanced Cancer Surveillance in Canada ed. Y. Mao and J Argo Special Issue Environmetrics 1998, 9, Sept Oct

OBJECTIVE

It is our objective to convey the knowledge that the development of oil and gas comes with a terrible cost in human health. The cost is not for the workers on-site, though they are affected. It is to women and children, the aged and infirm, the teachers and doctors and pharmacists and priests and First Nations peoples who live away from but in proximity to a flare site. They live at home.

They are unprotected by Labor-code statutes about exposure because they live at home and are exposed where they sleep and eat as wind carries the plume of combustion products from the source to their residence

Oil companies and the oil industry *per se* repeatedly tell us that there is no harm done by their activities, either to humans or livestock. They characterise their actions as responsible and benign, harmless to residents and with risks only to workers immediately affected. We expect Industry to tell this Hearing that no adverse human health effects are known and no adverse ecological effects are expected.

We will rebut their arguments and present flaring as a broad, multi-facetted disturbance of the system of human activities for as much as 30 km from any flare. We will use publicly available peer-reviewed scientific literature to present our rebuttal. [5]

Discussing flaring in terms of single issues is like a painting in greys - lifeless. For a full appreciation of its long-term impact, flaring must be considered on several levels of adverse health effects. These include chronic diseases, viz. cancer, diabetes, heart disease etc.; issues associated with the constant presence of flaring manifested as stress and AI and chemical sensitivity; issues associated with the division of communities into camps, again usually manifested as stress.

[5] San Sebastien M, Armstrong B, Cordoba JA, Stephens C Exposure and cancer incidence near oil fields in the Amazon Basin of Ecuador Occup Environ Med. 2001,58,517-522

FLARING

- A flare has been defined as a toilet up-side-down at the end of a pipe 30 feet in the air with a flame to keep the seat warm.[6]
- A flare is a device for disposing of waste gas and oil and co-produced sea-water very cheaply by burning the organic material in an open, uncontrolled, manner at an elevation above the ground varying from 15 to 75 or more m.. The elevation is to disperse the combustion products and throw them away from the work site. Studies show that working in a refinery with many flares is generally protective for chronic health issues compared to the general population.
- Flares are an unreported source of greenhouse gases. Flaring emissions do not appear in National Pollutant Release Inventory from Environment Canada nor Toxic Release Inventory in the US
- Flares are an unreported source of dioxins. The Secretary of the Ontario chapter of the CCME Dioxin Sub-committee told us verbally that this source of dioxins (flaring) was known and acknowledged by the CCME but this could not be made public. It is illegal under CEPA to release dioxins into the Canadian environment
- Flares operate at **any** location or **any** time waste gas is available for disposal. A flare acts as a security vent to a well and the entire production of a well may be directed to flaring.
- A well operating flare is opposed to well-accepted principles of sustainable development. A poorly operating flare is a travesty against the entire biosphere within up to a 30km radius. It is axiomatic that where a flare is smoking no one should be downwind. It is our judgement as a chemist and well-educated scientist that the monitoring of emissions in Alberta is ineffective and trivial,

[6] Darryl Graff Vulcan AB, Feb 25, 2001 commenting on his experience with Crestar Oil, his MS and my presentation

constrained by a desire to avoid disturbing the dinosaur - the dominant organism with little brain and lots of mass.

- Flaring is ubiquitous when oil and gas operations take place. The economic importance of oil and gas means that industry will expand very rapidly to gain maximum economic impact. In Alberta in 1996 there were 1410 townships where flaring took place. There were in excess of 6500 flares operating. There was in excess of 13,726 thousand cubic meters of gas flared each day in Alberta in 1996. This has risen from about 2,500 thousand cubic meters per day in 1960. [7] There are about 100 - 150 chemicals created in oil/gas flaring.

Cf. fig 1 Petro-Canada Rimbey Gas Plant from about 15 km. Note the stack is about 200 ft high. We could hear the flare roaring at this distance.

CHEMICALS PRODUCED IN FLARING

Strosher carried out on-site chemical characterization of emissions [8] approximately 4-5 m downwind from the sweet gas flare. The chemicals produced in the sweet gas flare are found in Table 1. Amounts in Table 1 are in mg/m3. All the chemicals characterised on-site are included; all the chemicals characterised by thermal absorption and all the chemicals characterised by solvent extraction and present in amounts over 10 mg/m3 are included. Unspeciated chemicals are included in the term "Other HC's".

In Table 1, we have not included the substituents for lack of space, indicating them with the abbreviation "subst".

Ex. 1 Benzene in Strosher: Sweet gas flare ScreenView3 "Good Burn"

[7] Canadian Minerals Yearbooks 1951 to 2001 incl.

[8] Investigations of Flare gas Emissions in Alberta. M Strosher Final report to Environment Canada, Alberta Energy Utilities Board and the Canadian Association of Petroleum Producers 1996

In Table 1, under the first column "On-site Characterisation" Strosher reports finding 116.5 mg benzene/m3 when the gas is flowing at 6 m3/min in a sweet gas flare or 699 mg benzene released per minute. We have used the conditions under which the flare

Table 1

On-Site characterisation of chemical emissions from the sweet gas flare
Strosher Table 33, Table 34, and Table 35 pg. 97,99,100
Liquid Fuel level 45 cm

On site characterization	Mg/m3	Thermal Absorption > 10 mg/m3	mg/m3	Solvent Extraction > 10 mg/m3	mg/m3
Hydrogen	20.0	Pentane	12.8	*Subst* benzene	9.83
CO	15.7	3-penten-1-yne	19.3	Azulene	21.2
CO2	4890	Benzene	144.5	*Subst* benzene	11.47
Carbon	54.2	1,5-hexadiyne	48.2	Naphthalene	99.39
Methane	103.8	Methyl benzene	27.5	2-methyl naphthalene	9.25
Ethylene	29.0	Ethyl benzene	13.7	1-methyl naphthalene	6.18
Acetylene	53.7	Ethynyl benzene	94.8	1,1'-biphenyl	58.7
Ethane	9.9	Ethenyl benzene	82.1	Biphenylene	42.81
C3 HC's	11.7	Benzaldehyde	18.7	1H phenalene	21.01
C4 HC's	6.4	Phenol	26.4	9H fluorene	41.09
Benzene	116.5	Naphthalene	88.7	Phenanthrene	10
Toluene	18.2	1,1'- biphenyl	16.1	Anthracene	42.11
Xylenes	29.8	Biphenylene	19.1	Fluoranthene	51.35
Styrene	75.5	Acenaphthalene	23.2	Pyrene	32.37
Ethynyl benzene	79.6			4-methyl; pyrene	9.1
Naphthalene	77.2			1 methyl pyrene	8.4
Other HC's	128.5	38 Other HC's	132.8	Benzo(ghi)fluoranthene	10.18
CE	65.0 %			Cyclopenta(cd)-pyrene	29.77
				Benz(a)-anthracene	17.33
				48 Other HC's	94.47

The analysis of the sour gas flare is included as Table 2. All amounts are in units of mg/m3.

Table 2

Emissions in the Downwind plume of a Waste gas Flare from a Sour Oilfield battery[9]
Strosher Table 37, 39, 40 pg. 100, 105,106

On-Site characterisation	mg/m3	Thermal Desorption > ~1 mg/m3 n = 36	mg/m3	Solvent extraction > 1 mg/m3 n = 54	mg/m3
Hydrogen	150	Carbon Disulphide	453.3	Hexanoic acid, 2-ethyl	5.04
CO	8	Thiophene	79.2	Naphthalene	77.1
CO2	6870	Benzene	64.3	Benzo[b]thiophene	46.7
Carbon	18.2	Methylbenzene	20.5	Benzoic acid	6.4
Methane	83.1	3-Methyl Thiophene	2.7	Benzo[b]thiophene, 4 methyl	8.9
Ethylene	6	Benzene-ethyl	7.1	Naphthalene, 2-methyl	14.3
Acetylene	36.4	Benzene, 1-3 dimethyl	6.5	Naphthalene, 1-methyl	10.8
Ethane	4.9	Benzene, ethynyl	41.9	Phthallic anhydride	2.3
C3 HC's	5.7	Benzene, ethenyl	34.4	1-1' Biphenyl	78.0
C4 HC's	2.9	Benzene, methoxy	1.4	Naphthalene, 1-ethyl	6.7
Benzene	24.4	2(5H)-thiophene	31.3	Thiophene, 2-phenyl	7.0
Toluene	12.4	2-Thiazolamine	0.9	Thiophene, 3-phenyl	12.0
Xylenes	6.7	Benzaldehyde	12.6	Naphthalene, 2,3 dimethyl	5.4
Styrene	22.7	Benzonitrile	1.3	Biphenylene	13.2
Ethynyl benzene	18.4	Bnezofuran	3.6	Dibenzofuran	7.1
Naphthalene	31.2	Decane	1.2	9H-fluorene	54.2
Other 'HC's	111	Phenol	12.2	Dibenzothiophene	82.2
Efficiency Carbon CP	84%	Ethanone, 1-phenyl	61.9	Phenanthrene	34.1
		Naphthalene	61.5	Sulphur (S8)	157.4
Sulphur Dioxide	6910	Azulene	34.4	Fluoranthene	14.1
Hydrogen Sulphide	126	Benzo[b]thiophene	156.6	Pyrene	83.3
Carbonyl Sulphide	64	Naphthalene, 2-methyl	1.5	Chrysene	2.4
Carbon Disulphide	482	Naphthalene, 1-methyl	0.9	Benzo[a]pyrene	0.5
Other S	210	1,1' Biphenyl	8.0		
Efficiency Sulphur CP	82.4%	Dibenzothiophene	6.6		

Conditions for the Analysis of Sweet and Sour Gas Flares

Parameter	Sweet Gas Plume	Sour gas Plume
Stack Height (m)	12.0	15.0
Wind Speed m/s	1.9	2.0
Flame Length	4.5	1.75
Gas flow rate m3/m	6.0	0.45
H2S in solution gas	1.5	22
Combustion efficiency	64%	84.2%

[9] Strosher M *op.cit.* pg. 103-106

Ex:1 continued

were analysed given below, in the in the very simple EPA model called ScreenView3, distributed by Lakes Environmental in Canada to examine how the benzene is spread out or dispersed.

Screenview 3 selects conditions for a flare and requires for input the rate of release in gm/sec (0.699 / 60 = 0.01165) and the heat released in cal/gm (2.5 E+06) and the height of the flare (12 m). We are estimating a "good burn" with this heat quantity. Poor burning will release less heat. With this input ScreenView3 estimates a profile of the dispersed plume material in concentration (ug/m3) vs. distance. We find that the sweet gas flare has a maximum benzene concentration of 0.06 ug/m3 at about 500 m from the flare and measurable amounts at 5 km.

Fig 3 Profile of Benzene from Strosher Sweet Gas Flare

Is this representative? We think not

Table 3
Volumes Directed to Flare 1996 (volumes in 1000 m3/d)
Sour Gas (contains H2S >2%) Sites only

Gas Plants			**Gas gathering**		
Volume range		No. Sites	Volume Range		No. Sites
Total Sweet Gas Plants		526			
0.1	1	3	1	10	2
1	10	21	10	100	23
10	100	61	100	1000	31
100	1000	124	1000	10000	51
1000	10000	53			15
10000	50000	3			
Total Sour Gas Plants		265	Total Gathering Systems		122
Batteries			**Townships**		
0.1	1	152	0.1	1	29
1	10	736	1	10	87
10	100	1847	10	100	233
100	1000	2113	100	1000	555
1000	10000	388	1000	10000	480
10000	50000	8	10000	50000	26
Total Batteries		5244	Total Townships		1410

The Alberta Energy Utilities Board (EUB) prepared a map of "Volumes Sent to Flaring in 1996". [10]The legend is reproduced in Table 3 and shows that in fact a volume of a few m3/min is quite unrepresentative for all but the smallest sites. The flow in the sweet gas flare of 6 m3/min corresponds to 8640 m3/day assuming continuous operation. Batteries are the most common installation and the volumes sent to flaring in a battery range from 1,000 m3/d to in excess of 10,000,000 m3/d. The 8640 m3/d is in the first decade of the volumes flared.

Ex.2 Benzene in Strosher Sweet Gas Volume increased

Suppose that the volume flared were a factor of 10 greater, that is 86,400 m3/d and the flow rate were 60 m3/min. With this simple change the maximum benzene concentration estimated with ScreenView3 now is 0.6 ug/m3 at about 500 m and the concentration of benzene at 5000 m is approximately 0.25 ug/m3.

We show below that a lifetime risk of 1:1,000,000 for adult leukemia corresponds to an annual average benzene concentration of 0.096 ug/m3.

That is, at the very bottom of the volume of gas flared in Alberta there was almost no risk but increasing the volume flared by a small factor of 10 introduces a measurable risk. The ratio of the estimated maximum benzene concentration to the acceptable exposure has become about 600 / 96 = 6.25 X near the flare at a distance of perhaps 500 m and 250/96 = 2.6 X at a distance of 5 km from the flare. This was done assuming 100% combustion efficiency, a "good burn".

Is this representative? Again, Probably not because combustion efficiency is missing.

Strosher remarks on seeing carbon particles in the flare and reports a reduced combustion efficiency of about 65% in the sweet gas flare.

[10] Alberta Energy Utilities Board Volumes Sent to Flaring map 1996

Wind

Strosher carried out open atmosphere flaring with a natural gas flame containing 22% condensate to examine the effect of cross-winds. He reports (ref 7 pg. 77)

> "one of the major effects created by the cross-winds was the overall reduction in combustion efficiency". Efficiency measurements were lower and more variable indicating a greater variability within the actual flame. "The primary reason for the low combustion efficiency is the hydrocarbons, detected as either unburned condensate (*raw fuel*) or produced hydrocarbons that survived the outer extremities of the flame and occur in the emissions". italics ours.

Combustion efficiency (CE) in a flare is severely affected by wind. Regardless of the regulatory stricture imposed a conventional flare burning waste will never operate at 95+% except in winds of less than about 2 kph. In Alberta these conditions appear with less than 5% probability. Strosher (8) found a CE of 84.2% in the sour gas flare with winds light at 7.2 kph. Winds of 25 kph will cut the CE to below 55-65%. The sweet gas flare operated with a combustion efficiency of about 65% in light winds.

Raw fuel is released in progressively larger amounts when the CE falls. This is due to the basic process of combustion. Combustion takes time, as much as tens or hundreds of seconds. During this time the droplet of fuel can diffuse away from the combustion zone and escape the flame altogether. [11] [12]

Kostiniuk reported at a meeting in Edmonton in 1996 on his work demonstrating experimentally that the major factors that reduce combustion efficiency are wind speed and heating value of the fuel. [13] His work adds the extra parameter of heating value to wind-speed. There is no reason to doubt that low CE has applied to all flaring since the inception of the Industry in 1955.

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[11] Fuels and Combustion Handbook ed A.J. Johnson and G.H. Auth McGraw Hill, 1951
[12] Combustion of Solid Fuels and Waste AJ Tillman 1999
[13] Kostiniuk personal communication 1996

Reduced combustion efficiency must be regarded as the norm in any operation with flaring. Simultaneous with low CE is the release of raw unburned fuel. This will include hydrogen sulfide in perceptible to overwhelming amounts with sour gas flares.

We use 50-year wind roses for Alberta from Environment Canada [14] to gauge the frequency of occurrence of these winds. There are 37 stations situated throughout the province. We calculated the mean and standard deviation for each of eight directions and conducted a stochastic analysis on the data using a Gamma distribution and 65,000 iterations to obtain the percentiles of wind speed from each of eight directions. [15]

The 90% percentile of wind speed is 19.2 kph. A requirement of every flaring license since the inception of the ERCB and EUB is that flares operate with no less than 95% CE. For this we need winds no more than about 5 kph (~1.4 m/s). Our analysis of the historical record shows these conditions occur in Alberta with about a 5% probability. So the flare might be in compliance about 5% of the time. At all other times it is probably not operating in compliance with its operating permit and should be shut down. That never happens - either.

Ex. 3 Benzene in Strosher: Volume unchanged; heat release decreased by factor of ten

We return to ScreenView3 and the original conditions with the release of 0.699 gm/min of benzene but reduce the heat released by a factor of 10 to 2.5 E+05 cal/sec. ScreenView3 now estimates that the peak benzene concentration will be nearly 0.48 ug/m3 (0.48./.096 = 5.0 X acceptable intake) at perhaps 200 m from the flare. The ground level concentration of benzene in the plume is above the acceptable maximum value of 0.096 ug/m3 from the flare out to a distance of about 2,500 m.

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[14] Wind 50 year Wind Averages Atmospheric Environment Services Environment Canada 1986

[15]

Ex.4 Benzene in Strosher: Volume increased by ten; Heat release remains reduced by 10

We return to screenView3 and the slightly elevated emission rate (6.99 gm/min) with the same reduced heat released. The estimated concentration of benzene in the plume is above the acceptable maximum value of 0.096 ug/m3 over the entire range of 5 km from the source and the estimated peak value of benzene is about 4.8 ug/m3 or 50 X the acceptable maximum.

Is this representative? We think so because both volume flared and observed combustion efficiency have been accounted for.

The result is that an elevated risk for cancer may be present in the first 2.5 to 5 km from the flare. Residents in this zone have the potential to be affected by benzene exposure as described below. This concentration of benzene is below the odor threshold (cf. below)

Ex:5 Benzene in Strosher: Flare on a drilling platform 50 m high

We return to ScreenView3 to examine what would be the difference if the flare were now on a drilling platform, such as we find in exploration. We will place a 12 m flare on top of a 50 m drilling platform. Nothing else is changed. Low volume, reduced heat release. This leads to a dramatic change in the shape of the profile.

Fig 4 : Profile of an Elevated Source

Elevating the flare results in broadening the distribution and shifting it outward. With the volume in the lowest volume range reported flared by the EUB, (Table 3), the peak concentration of benzene is about 0.09 ug/m3 at a distance of about 1499 m, Under these conditions the cancer risk is arguable but the effect of wide-spread low level exposure to benzene is pancytopenia (cf. below) and a general morbidity in the population for 5 km is likely.

Ex: 6 Benzene in Strosher: Flare elevated on a drilling platform. Volume and flow increased by factor of 10

We return to ScreenView3 and change only the volume of the flare. The dramatic shape change remains and the peak concentration of benzene now is about 0.85-0.9 ug/m3 at about 1400 m. The concentration of benzene is everywhere over the maximum acceptable value of 0.096 ug/m3. At 5 km the benzene in the plume is about 0.25-0.3 ug/m3.

Increased morbidity and elevated risk of leukemia is everywhere possible for residents within 5 km under these conditions. Those most at risk of long-term health effects are persons under age 30.

A small flare on a drilling platform operating off the coast of Cape Breton will lead to identical conclusions.

ADVERSE HEALTH EFFECTS: PART 1

CHRONIC CHEMICAL EXPOSURE

In the following paragraphs we document observed associations between specific chemicals or chemical types and selected chronic health outcomes. In all the cases we will be considering "*any*" chronic exposure which corresponds to minimal chronic exposure.

1. **Raw Fuel**: Associations between several cancer sites and fuels have been carefully examined by Siemiatycki et. al. [16]They examined cancers and exposure to petroleum fuels, specifically automotive gasoline, aviation gasoline, mineral spirits, kerosene, jet fuel, diesel fuel, heating oil, cutting fluids, hydraulic fluids, lubricating oils, other mineral oils and crude oil. They found statistically significant associations between many sites and minimal (= any) exposure. Statistically significant associations are

[16] Siemiatycki J, Dewar R, Nadon L, Gerin M, Richardson L, Wacholder S, Associations between several sites of cancer and twelve petroleum-derived liquids Scan. J. Work Environ. Health 1987, 13. 493-504

those with the lower confidence interval greater than 1. Statistically significant associations at the 90th percentile were found between the following sites and fuels

Stomach and	Automotive gasoline	OR = 1.5
	Kerosene	OR = 1.7
	Hydraulic Fluids	OR = 1.9
Rectum	Crude Oil	OR = 5.8
	Jet Fuel	OR = 2.1
Lung Oat cell	Heating Oil	OR = 1.7
	Cutting fluids	OR = 1.5
Lung Squamous cell	Kerosene	OR = 1.4
	Diesel Fuel	OR = 1.5
	Lubricating Oils	OR = 1.2
	Crude Oil	OR = 2.8
Lung adenocarcinoma	Kerosene	OR = 1.5
	Other mineral Oils	OR = 1.7
Lung other	Other Mineral Oils	OR = 1.8
Prostate	Mineral Spirits	OR = 1.3
	Diesel Fuel	OR = 1.7
	Heating Oil	OR = 1.4
	Cutting Fluids	OR = 1.2
	Hydraulic Fluids	OR =1.5
	Lubricating Oils	OR = 1.3
	Other Mineral Oils	OR = 1.4
	Crude Oil	OR = 2.3
Bladder	Cutting Fluids	OR = 1.2
Kidney	Aviation Gasoline	OR = 2.6
	Cutting Fluids	OR = 1.2
	Jet Fuel	OR = 2.5

They found many exposure-site scenarios with elevated risk that were not statistically significant. This means that there were not enough cases to distinguish cases from chance. The smaller number of cases is principally due to latency effects. A similar study done later would probably find that there were more cases because the disease has had more time to develop and appear.

Siemiatycki et. al. [17] examined the association of minimal (= any) exposure to fuel exhaust and combustion products and an array of cancer sites. They identified statistically significant associations between the following sites and exhausts or combustion products.

Esophageal	Wood Combustion	OR = 2.2
Stomach	Wood combustion	OR = 1.8
Colon	Diesel exhaust	OR = 1.3
Rectosigmoid	Gasoline Exhaust	OR = 1.2
Rectal	Gasoline Exhaust	OR = 1.2
Pancreatic	Coal Combustion	OR = 2.3
Lung Oat cell	Propane combustion	OR = 1.8
Lung Squamous cell	Propane Exhaust	OR = 1.6
Lung Other	Coal Combustion	OR =1.6
Prostate	Liquid Fuel Comb.	OR = 1.6
	Coal combustion	OR = 1.6
	Diesel Exhaust	OR = 1.2
Bladder	Natural Gas Comb	OR = 1.6
Melanoma	Propane Exhaust	OR = 3.3

Elevated but statistically non-significant associations were found with coke combustion to include the following sites

Esophageal OR =2.1 (.5-9.8), Stomach OR = 1.5 (0.5-4.8), Recto-sigmoid OR = 2.0 (0.6-6.8), Pancreatic OR = 2.2 (0.4-10.5), Non-Hodgkins lymphoma OR = 1.4 (0.3-7.3).

2. Hydrocarbons

Benzene

[17] Siemiatycki J, Gerin M, Stewart Nadon L, Dewar R, Richardson L Associations between several sites of cancer and ten types of exhaust and combustion products Scand. J Work Environ. Health 1988, 14, 79-90

Benzene is a systemic toxicant in humans at *any* concentration and a cause of aplastic anemia (deficient red blood cell production). The major effect of benzene in the body is depression of bone marrow leading to pancytopenia, (a general depression of erythrocytes (red blood cells), leukocytes (white blood cells) and thrombocytes (platelets)). A widespread reduction in erythrocytes in a population will lead to a general increase in morbidity. This has been commented upon, anecdotally, in Alberta in relation to the contrast between relatively high prosperity and relatively high use of the health-care system. Generally a higher socio-economic standard is expected to lead to a reduced use of the health-care system.

Benzene is a known human carcinogen, causing leukemia; it is non-mutagenic. An annual time weighted average concentration (TWA) for a risk of 1 in a million is an annual average concentration of 0.096 μg/m3.

The odor threshold of benzene is 4.5 mg/m3 and the odor is described as sweet. [18] An odor threshold is the concentration when an average person becomes aware of an odor. We draw attention to the quite important observation that an average person will become aware of the presence of benzene at a concentration 4500 / 0.096 = 46,800 x maximum acceptable value for annual exposure of a risk of 1:1,000,000.

An average person can be at risk of leukemia and never be aware, take steps to protect or otherwise act in defense of their health and integrity.

Toluene

Toluene is a chemical with one methyl group in the benzene ring. Toluene is a potent central nervous system toxicant leading to narcosis, incoordination, emotional lability, and subjective symptoms such as headache and fatigue.

[18] Ruth JA, Odor thresholds and irritation levels of several chemical substances. Am. Ind. Hyg. J 1986, 47, A-142 to A-151

Known <u>neurotoxic responses to **acute** toluene</u> exposure include narcosis, hilarity, lassitude, drowsiness, mental productivity decrease, reaction time change, impaired balance, vertigo, disturbed vision, paresthesias.

<u>Neurotoxic responses to **chronic** exposure</u> include emotional lability, bizarre behavior, tremor, unsteadiness, rhythmic limb movements, ataxia, cerebellar ataxia, optic atrophy learning decrease, circling, excitement, delirium confusion, hallucinations, ringing in the ears [19] Emotional lability, that is tearing for no identifiable reason, is symptomatic.

The odor threshold for toluene from coke is 17,550 ug/m3 and the odor threshold for toluene from petroleum is 8,025 ug/m3. An acceptable daily intake of toluene in air using a relative potency and very conservative approach is 120 ng/m3 = 0.12 ug/ m3. [20]

The ratio of the odor threshold of toluene to the ADI from Jones et.al., is 8025 / 0.12 = 66,800 X.

An average person exposed to toluene can experience a wide variety of neurotoxic symptoms without any indication of the cause. We have found that emotional lability is traumatic for many men who regard showing emotion openly as anathema. They often describe it to us as "un-manly but can't help it".

The ultra-conservative scale we have used in this work was formulated by Jones et.al. following a relative potency approach using Benzo[a]pyrene as the primary reference. It accurately predicts some key quantities (benzene risk etc.). It uses a non-threshold approach and in numerous comparisons in the literature proves superior in predictive ability to all but the most sophisticated and complicated procedure. The RASH approach of Jones has been designed as a method for preliminary hazard assessment. In this

[19] Anger WK, and Johnson BL *Chemicals affecting behavior Ch3.* in Neurotoxicity of Industrial and Commercial Chemicals vol 1 CRC Press 1985 page 51 ff

[20] Jones TD, Walsh PJ, Watson AP, Owen BA, Barnthouse LW and Sanders DA Chemical scoring by a Rapid Screening of hazard (RASH) method Risk Analysis 1988, 8, 99-118

work we have used predictions for an air pathway. Predictions for water and occupational pathways were published in the same reference. Our use of the RASH approach has brought us understanding.

Xylene, o-, m-, p-,

Xylenes have two methyl groups substituted into the benzene ring, either adjacent (= ortho), separated by one ring carbon (= meta) or separated by two ring carbon atoms (= para), Xylenes are unequivocal developmental toxins, leading to delayed development, decreased fetal body weights and altered enzyme activities.[21]

There is evidence of behavioral neurotoxicity in individuals occupationally exposed to short term levels of xylene. It appears to cause CNS depression and minor effects in the liver and kidney. In human studies 200 ppm are definitely irritating to the eyes, nose, and throat. [23]

Xylene(s) are *fetotoxic* including delayed development, decreased fetal body weights and altered enzyme activities; they may cause CNS depression in acute exposures resulting in dizziness, staggering, drowsiness and unconsciousness.

The odor threshold of xylene is 348 ug/m3 and the acceptable daily intake from air is 0.120 ug/m3. This means that an average person who becomes aware of the odor of xylenes is exposed to 348 / 0.120 = 2,900 X an acceptable daily intake.

A mother with delayed development or decreased birth weight in her new-born experiences an immense personal trauma.

> *A common comment is "What have I done wrong?"*
>
> *The answer is "Nothing… except live at home".*

Styrene

Styrene (vinyl benzene, ethenyl-benzene) is an irritant of the skin, eyes, and mucous membranes and a CNS depressant. Upper respiratory tract and eye-irritation have been reported at 50 ppm.

Women who worked in the most exposed jobs had offspring with adjusted birth weights 4% less than the offspring of unexposed women. There is a disagreeable odor with eye and nose irritation. This may not be sufficient warning for prolonged exposures. [22] This will contribute to the general eye, nose, throat and mucous membrane irritation and the odor will be found very disagreeable. The odor threshold is 0.2021 mg/m3 and an acceptable daily intake by air has not been established.

Naphthalene

Naphthalene is a hemolytic agent, destroying the membrane of the red blood cells with the liberation of hemoglobin, and an irritant of the eyes that may cause cataracts. Initial symptoms include eye irritation, headache, confusion, excitement, malaise, profuse sweating, nausea, vomiting, abdominal pain, irritation of the bladder. The metabolites are hemolytic, that is the biological damage is secondary to the naphthalene. Headache, nausea and confusion are reported to occur after inhalation of the vapor. Extrapolation from animal data is difficult because of varying results.[23]

The odor threshold of naphthalene is 1.5 mg/m3 and an acceptable daily intake is estimated as 96 ng/m3 (ref.13).

An average exposed person aware of the odor of naphthalene (mothballs) is exposed to 1500 / 96 = 15.6 times a safe limit.

Other substances

Jones et.al. has estimated that an acceptable daily intake (ADI) by air of diesel exhaust particles is 1.2 ng/m3. They estimate an ADI by air of kerosene is 780 ng/m3. They estimate an ADI by air of TCDD is 0.00014 ng/m3. They estimate an ADI by air of Sulfur dioxide from the flaring of sulfur-containing material is 3.8 ng/m3 and an ADI of sulfuric acid is 2.2 ng/m3.

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[21] Calabrese and Kenyon *op.cit.* page 577

[22] *Styrene monomer* in Proctor and Hughes' Chemical Hazards of the Workplace 3rd edition. Ed. Hathaway GJ et.al van Nostrand New York, 1996 pg. 519

Partial combustion products in Table 1 and Table 2 include Pyrene (ADI = 46 ng/m2), Fluroranthene (ADI = 7.5 ng/m3), Acenaphthene (ADI = 0.35 ng/m3), Benzo[a]pyrene (ADI = 0.46 ng/m3), and Chrysene (ADI = 2.1 ng/m3). An acceptable daily intake by air of Nitrogen dioxide (NO_2) a product of any combustion is 0.21 ng/m3 and the ADI for nitric oxide (NO) is 0.22 ng/m3. Oxides of nitrogen are produced in any burning from atmospheric nitrogen.

Sour Gas Flares

Sour or not. Is there any reduced sulfur species in the waste gas-oil? If sour there are some extremely toxic and malodorous sulfur compounds (Table 2) **in addition** to a spectrum of carcinogenic, neurotoxic, fetotoxic and teratogenic hydrocarbons. In Alberta by administrative fiat a well is considered "sweet" when there is less than 2% hydrogen sulfide present. Exposure to this level of hydrogen sulfide is known to be neurotoxic and fetotoxic.

In the downwind plume of sour gas flares sulfur dioxide, hydrogen sulfide and carbon disulfide together with a spectrum of sulfur-containing chemicals are present, in addition to the range of hydrocarbons in Table 2. An annual exposure greater than 4 ug/m 3 hydrogen sulfide is associated with spontaneous abortion in animals and humans. [24] The odor threshold of hydrogen sulfide is about 7 ug/m3, that is less than the critical concentration. An acceptable daily intake of 1.8 ng/m3 has been calculated by Jones et.al.

Hydrogen sulfide induces hypoxia in human and animal. It affects the ability of the cell to process oxygen by interfering with oxygen metabolic processes. Cyanide poisoning induces hypoxia by blocking the process that carries oxygen to the cell. Carbon monoxide acts the same way by forming the very stable carboxy-hemoglobin also blocking the oxygen

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[23] *Naphthalene* in Proctor and Hughes' Chemical Hazards of the Workplace 3rd edition. Ed.Hathaway GJ et.al van Nostrand New York, 1996 pg. 419

[24] Hemminki K, Neimi ML Community study of spontaneous abortion: relation to occupation and air pollution by sulfur dioxide, hydrogen sulfide and carbon disulfide Int Arch Occup Env Health 1982,51,55-

transport process. In the foothills of Alberta where oil and gas are closely mixed with cattle and game ranching and recreation activities the number of spontaneous abortions in the first trimester is distressingly high. [25] Every bovine or elk fetus lost is a tremendous economic loss to the farmer; every child lost is a loss to the nation.

In an open meeting containing ranchers and families and oil company managers (Feb 25, 2001 in High River AB) we were asked if the effect (SA) was also possible in humans. We replied "yes" and were shocked to see that about 10% of women present had the courage to openly answer that they had personally experienced this trauma. The managers remained stone-faced.

3. Dioxins

Dioxin is the abbreviation for large class of persistent, long-lived, and ubiquitous chemicals with the same basic structure; two benzene rings joined through two oxygen atoms. They have up to 8 chlorine atoms substituted in the rings. The ones with 8 chlorines are the octachloro-, those with 6 the hexachloro-, 4 the tetrachloro- etc. and each is called a congener. The tetrachloro- congenors are more toxic and include the most potent cancer promoter, 2,3,7,8-tetrachloro-dibenzodioxin abbreviated TCDD. There are about 145 separate possible dioxins or congenors.

Dioxins form in incinerators wherever chlorine comes in contact with heated organic matter. The octachlordibenzodioxin congeners form in greater amounts than the tetrachloro congenors in the flare.[26]

The chlorine is supplied in a flare by co-produced salt water present in the sour-solution gas, separated from the crude oil in a battery. Exposure to dioxins and furans is well established as the cause of chloracne.[27]

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[25] Wayne and Ila Johnson, Sundre AB, private conversation Dec 13, 01

[26] Gochfeld M, in *Environmental Medicine* ed. Brooks SM, Gochfeld M, Herzstein J, Jackson RJ, Schenker MB, Mosby 1995, pg 603, ref 75

[27] *Casarett and Doull's Toxicology The Basic Science of Poisons* 4th ed. Pergamon 1991, page 478

Chloracne is a dermatological condition distinguished by the distribution of the lesions. The most sensitive areas are the face and behind the ears. The nose is very resistant. In men, the penis and scrotum are frequently affected. As the disease progresses chloracne may occur at sites that are distant from the contact area including the back and buttocks. The lesions are exceptionally itchy with little or no relief available. The condition lasts for months. [28]

Criteria to identify chloracne include, exposure to a chloracnegen (dioxin), onset within 2 weeks to 2 months, predominance of open nodules (comedones) and straw colored cysts, atypically distributed, inflammatory cysts and abscesses on the face behind the ears, on the neck scalp and buttocks, recedes slowly after exposure is reduced.[29] The main causative congenors were the hexachlorinated dibenzodioxins.[30] This work shows that the cumulative dose must exceed a very small threshold amount to display chloracne. Exposed persons who have not exceeded the threshold will not display chloracne - *yet*.

> cf. **Fig 2**. This individual lives about 1.4 miles from a sour gas battery in South-western Manitoba with 35% H2S and co-produced seawater. His residence is downwind with ambient winds. The red rash is on his face, scalp, ears, neck, chest and scrotum and accompanied by comedones (raised red itchy domes). The condition lasted for about 3 months and was unrelieved by any medical treatment. [31]

In workers exposed to TCDD-contaminated products the most frequent cause of sick leave was chloracne, which persisted in 32%. Neurological symptoms frequently

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[28] William F Campbell Tilston MB personal communication, March 2000
[29] Zugerman C *Chloracne, chloracnegens and other forms of environmental acne* in Occupational Skin Disease ed Adams RM 2nd edition WB Saunders 1990 pg 128
[30] Coenraads PJ, Olie K, Tang NJ, *Blood lipid concentrations of dioxins and dibenzofurans causing chloracne* Br. J. Dermatol. 1999:141(4):694-7
[31] Argo J Exposure Assessment for WF Campbell

reported were sleep disturbance 44%, headache 32% and neuralgia (sharp pain) 33%.[32] Workers with chloracne have significantly higher exposure to polychlorinated dioxins, experience mild sensory neuropathy, complain significantly more of sexual impotence, have significantly more frequent clinical signs of sensory neuropathy and significantly more frequent neurophysiological abnormalities. [33]

Chronic exposure to dioxins carries a long-term excess risk of soft tissue sarcoma. This is associated with exposure to *any* polychlorinated dibenzo-dioxin or furan with an odds ratio OR = 5.6 (CI = 1.1 -28). This is a substantial risk. [34] Minimal (= any) exposure can be very dangerous. Jones estimates an ADI for TCDD of 0.00014 ng/m3.

Emission factors for total dioxins can be estimated experimentally by measuring the dioxins formed with several fuels in the presence or absence of chloride. For example newspaper, branches of London plane trees, newspapers mixed with sodium chloride, polyethylene or PVC were combusted and the exhaust gases collected. Total dioxins in the newspapers alone were 0.186 ng/g; total dioxin in the branches of London plane tree was 1.42 ng/g; total dioxin in newspapers impregnated with sodium chloride was 102 ng/g and 101 ng/g from newspapers impregnated with sodium chloride and PVC. More chloride produced more dioxins. [35]

Ex 7: Estimate Dioxin Emission: Strosher Sweet Gas Flare

[32] Neuberger M, Kundio M, Jager R, *Chloracne and morbidity after dioxin exposure (preliminary results)* Toxicol. Letts. 1998:96-97:347-50

[33] Tomke F, Jung B, Besser R, Roder R, Konietzko J, Hopf HC, *Increased risk of sensory neuropathy in workers with chloracne after exposure to 2,3,7,8-polychlorinated dioxins, and furans.* Acta Neurol Scand 1999:100:1-5

[34] Kogevinal M, Kaupinnen T, Winkelmann R, Becher H, Bertazzi PA, Bueno-de Mesquita HB, Coggon D, Green L, Johnson E, Littorin M et.al. *Soft tissue sarcoma and non-Hodgkin's lymphoma in workers exposed to phenoxy herbicides, chlorophenols and dioxins: two nested case-control studies.* Epidemiology 1995:6(4):396-402

[35] Yasuhara A, Katami T, Okuda T, Ohno N, Shibamoto T Formation of dioxins during the combustion of newspapers in the presence of sodium chloride and poly(vinyl chloride) Enviro. Scit Technol. 2001, 35; 1373-1378

We use an emission factor of 100 ng total dioxin /g fuel in the presence of chloride and 0.2 ng/g fuel in the absence of chloride. We estimate the density of the fuel in Strosher by summing the mass of combustion products. The density is approximately 0.0088368 kg/m3. Then in 1 minute the gas flows 6 m3 or ~0.053 kg. or ~ 53 gm. In the presence of chloride this will potentially produce ~100 X 53 = 5300 ng of dioxins. In the absence of chloride we expect approximately 0.2 ng dioxins /g of fuel or about 11 ng total dioxins. The emission rate of dioxins is approximately 5300 ng/min with chloride and approximately 11 ng/min with no chloride.

We return to ScreenView3. With the flare at 12 m elevation, slightly reduced heat release and 5300 ng total dioxins /min a peak concentration of 0.0035 ng dioxins/m3 is expected and is measurable and lower throughout the 5 km radius. This is for the original volume flared of 6 m3/min. Jones estimates an acceptable daily intake of TCDD is 0.00014 ng / m3 by an air path.

Increasing the volume flared by a factor of 10 does not alter the profile of total dioxins in a measurable way. Raising the flare to a drill platform 50 m high produces the same broadened profile with a peak dioxin concentration of .00075 ng/m3 close to 1400 m from the source. At 5 km the peak concentration of total dioxins in the elevated flare under minimal conditions is close to the limit calculated by Jones, 0.00014 ng dioxin.

In summary these estimations from ScreenView3 infer that in the presence of chloride and using volumes on the very lowest range of volumes flared by Industry in Alberta that the peak concentration of total dioxins will be close to 0.0035 ng/m3 or 25 X the limit from Jones. If the flare is elevated on a drill or other platform and the volumes continue small (*a very tenuous assumption*) the peak concentration of total dioxins will be closer to 0.00075 ng/m3 or 5 X the limit from Jones. **Under these conditions we expect chloracne to be widespread to any area with chronic exposure to flaring when co-produced sea water is simultaneously flared.**

4. **Particulates** Strosher refers to carbon in the plume. We include this as one form of particulates. The word "Particulates" as used here refers to both solid and colloidal micron sized particulates that are produced in open, uncontrolled burning. In the presence of sulfur dioxide (SO2) and with any organic material present great amount

of aerosols are preferentially formed initially in the sub-micron size range. These quickly coalesce and form much of the fraction called PM $_{2.5}$. Aerosols are very stable and capable of long range transport for many hundreds of km. The PM $_{2.5}$ particulates include a size range less than 2.5 microns that can be inhaled into the deepest recesses of the lung, the alveoli, and are strongly involved with elevated rates of morbidity and mortality from all-cause, lung cancer, heart disease and respiratory disease. [36] The next largest fraction, called PM_{10} because it includes a size range less than 10 microns that can be inhaled in all but the smallest regions of the lungs, has long been identified with increased morbidity. [37]

Harvard Six Cities Study This study involved adults living in 6 US cities, selected to be representative of the range of particulate air pollution in the U.S. Total suspended particulates (TSP), PM $_{10}$, PM $_{2.5}$ **,** SO4, H^+ , SO2, NO2 and O3 levels were monitored. Mortality risks were most strongly associated with cigarette smoking but after controlling for this and other individual differences in age, sex, cigarette smoking, body mass index, education and occupational exposure relative mortality risks across the cities were strongly associated with difference in pollution levels in the cities. Associations between mortality risk and air pollution were stronger for respirable particles and sulfates measured by PM $_{10}$, PM $_{2.5}$ **,** SO_4, than for H^+ , SO_2, NO_2 and O_3. The association was consistent and nearly linear with no apparent threshold value. Fine particulate pollution (PM $_{10}$, PM $_{2.5}$) was associated with cardio-pulmonary mortality and lung cancer mortality. [38]

In this and other studies it has been shown that statistically significant associations were observed between particulate air pollution and respiratory symptoms. Particulate air

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[36] Epidemiology of Chronic Health Effects: Cross sectional studies Pope A. and Dockery D. ch 6, 7 in Particles in Our Air ed R. Wilson and John Spengler Harvard Univ. Press 1996
[37] The Perils of Particulates American Lung Association, New York 1994
[38] Particles in our air: Concentrations and health effects op. Cit. Pg. 156

pollution was most consistently associated with bronchitic symptoms. The results suggest a 10 ug/m3 increase in PM_{10} is typically associated with a 10 to 25 percent increase in bronchitis or chronic cough. [39]

Ex:8 Particulate Concentration: Sweet Gas Flare

Strosher found 54.2 mg/m3 under the same conditions that produced the benzene discussed earlier. With a gas flow rate of 6 m3/m this is equivalent to 325,2 mg carbon/min. We return to ScreenView3 and the original conditions of a 12 m stack, reduced heat flow and an emission rate of .00542 gm C/ sec. The peak concentration of carbon particulates under these conditions is about 0.23 ug/m3 at about 2-300 m. At 1325 m the concentration is 0.10 ug/m3

Increasing the flow by a factor of 10 raises the peak concentration of carbon particulates to 2.3 ug/m3.

Changing the volume flared by a factor of 10 increases the peak particulate concentration by the same amount. Under these conditions we anticipate that respiratory irritation will be endemic in the areas surrounding a flare. Carbon particles have a very large surface area and are effective absorbers. A carbon particle passing through any unburned fuel or organic matter will absorb the fuel and/or organic matter and stabilize the chemical reactions taking place. When respired the particle will efficiently transfer the absorbed fuel to the tissue

The foregoing example is for a sweet gas flare when the quantity of SO2 formed is low because the sulfur in the gas is quite low. In the presence of any sour gas, flaring will produce, among others, abundant amounts of SO2. In combination with organic materials these form an aerosol, exceptionally fine particles << 1 micron. These are stable and do not disperse in the same way that the carbon particles are expected to behave. Aerosols will be transported distances of several hundreds of km from the

[39] Particles in our air: Concentrations and health effects op. Cit. Pg. 162

source. Cadmium containing aerosols have been traced 300 km from Rouyn-Noranda to Quebec City and from Sudbury to Algonquin Park.

Ex 8: Cancer Incidence Epidemiology in Western Canada

Two atlases of cancer incidence in Canada covering the '80's were produced. Both were products of Canada's Green Plan. They are organized geographically on the census division, abbreviated CD. The CD is a division of the country with fixed and relatively stable boundaries.

The first Atlas covers cancers incident in Western Canada from 1984 to 1988. [40]The cancer rate in each CD is evaluated in comparison to the same site and gender throughout all the region studies giving a rate ratio abbreviated RR. We have analysed the rate ratios for all the cancers reported in each CD, using a procedure called stochastic analysis. This is a statistical procedure that uses the properties of the data to estimate the underlying distribution of the data and from this the percentiles of the distribution. In Table 4 we present the median value of the rate ratio (RR) for each CD calculated for all the cancers. The median value includes 50% of the data points.

Table 5
Median Rate Ratios for Each CD - Alberta
"All-cancer"

CD	Median RR	CD	Median RR
1	1.1597	11	1.1795
2	1.1856	12	0.7327
3	0.8921	13	0.5435
4	0.8115	14	0.5455
5	1.2845	15	0.9048
6	1.2211	16	1.0418
7	0.8211	17	1.1164

[40] Atlas of Cancer Incidence in Western Canada (1984-1988) ed Berkel J, Turner D, Band P, Choi N, Robson D, Whittaker H Alberta Cancer Registry 1992

8	0.7992	18	0.1418
9	0.9154	19	1.1162
10	0.8527		

We compared the CD's with extensive upstream oil and gas flaring activity as depicted on the map produced by the AB EUB with the median rate ratio calculated in the stochastic analysis that appear in Table 4.

Each CD with median RR > 1 contains extensive flaring activities. CD's with less flaring activity have median rate ratios less than 1.

Our earlier comments about hydrocarbons anticipate that Leukemia, Bladder, Bone and Connective Tissue Pancreas and Thyroid have potential association with flaring emissions. Within the set of CD's with a median rate ratio > 1 we examined the median rate ratio of specific cancer sites. We analysed Bladder (M/F), Bone and Connective Tissue (M/F), Breast (F), Cervix Invasive, Leukemia (M/F), Pancreas (M/F), Thyroid (M/F) Prostate, and Unknown Primary (M/F).

Our results are in Table 6

Table 6

Median Rate Ratios and Cancer Sites

CD's With extensive Flaring Activity

Site	Median RR	Site	Median RR
Bladder F	1.066	Leukemia F	1.069
Bladder M	1.111	Leukemia M	1.150
Bone & CT F	1.192	Pancreas F	1.404
Bone & CT M	1.004	Pancreas M	1.619
Breast F	0.701	Thyroid F	1.202
Cervix	1.309	Thyroid M	1.086
O Digestive F	1.192	Unknown Primary F	3.279

Prostate	0.907	Unknown Primary M	2.651

Recall that the rate ratio compares the rate of the cancer site (per 1000,000 pop.) with the rate for the same site in Western Canada. This is for cancer incidence data collected from 1984 - 1988. From Table 6 we can see that in the CD's with extensive flaring certain sites have elevated rate ratios as expected.

In particular we draw attention to the substantial risk of Bone and Connective Tissue F, associated with dioxin exposures: to the substantial risk of Pancreas (M/F) and Thyroid cancers (M/F): to the elevated risk of Leukemia (M/F) from benzene: the high rate ratios of unknown primary site (M/F) are often attributed to multi-focal exposure from exposure to many potential carcinogens. The quite high rate of invasive cervical cancer is unexpected but must be acknowledged. The most likely cancer is an unknown primary, followed by pancreas and thyroid. Pancreatic cancer does not arise from pancreatitis (inflammation of the pancreas). An association with diabetes has long been suspected but is very difficult to study. [41] The most significant cause of thyroid cancers in humans is radiation with many studies reporting an association between radiation exposure and subsequent thyroid malignancies. [42] Radioactive nuclides are common components of the crude oil-gas-water mix that is brought to surface. A portion will be separated in the battery and the remainder separate in the gas plants and sulfur recovery plants. The most common nuclide is from radium. We routinely detect radio-nuclides in soil from around gas plants and batteries.

[41] Human Cancer: Epidemiology and Environmental Causes Pancreas ed Higginson J, Muir CS and Munoz N, Cambridge 1992 pg 317

[42] Human Cancer Epidemiology and Environmental Causes op.cit. Thyroid and other endocrine glands pg 447.

This data very strongly infers an association between the degree of flaring and the type and risk of several cancers. All of these are preventable, by reducing, changing or eliminating flaring.

We draw attention to the reduced median rate ratios found for breast and prostate cancers collected in this survey. We attribute this to latency effects. The oil and gas industry began in Western Canada in about 1955, almost at the beginning of the "baby-boom". A girl child born in 1950 and genetically susceptible to cancer would enter puberty about 1960 - 1962. A 25-year latency from that time would mean she would present with a breast cancer in 1985-1987 and possibly be missed by the survey. A longer latency would have her present later and possibly be included in the Canadian Cancer Incidence Atlas of 1986-1990 [43] or the Enhanced Cancer Surveillance project that collected breast and prostate cancers in 1992-1995 [44]. Breast and prostate cancers rates expanded very rapidly during the '90's [45]. We will present the second Canadian Cancer Incidence Atlas in more detail during our presentation.

ADVERSE HEALTH EFFECTS: PARTS 2

STRESS

5. **Stress:** Stress was broadly defined by Selye in 1950 as the response of an organism to stimulation or change and is characterized by activation of both the autonomic nervous system and the hypothalamus-pituitary-adrenal (HPA) axis. The resulting neuro-chemical changes affect the immune system both directly and indirectly. It has been recognized that there is an association between the CNS and the immune system as it relates to the development of auto-immune disease.

[43] Canadian Cancer Incidence Atlas (1986-1990) ed Le ND, Marrett LD, Robson DL, Semenciew R, Turner D, Walter SD pub by authority of the minister of national Health and Welfare Ottawa 1996

[44] Johnson KC, Mao Y, Argo J, Dubois, S, Enhanced cancer case-control component: Proposal for a collaborative project Health Canada and Canadian Cancer Registries Epidemiology Research Group 1994

Autoimmune disease is a condition in which the immune system "reacts to itself " and includes MS, type II diabetes, rheumatoid arthritis, Graves disease, Hashimoto's disease and lupis. [46] It seems to be associated with chronic stress from chronic exposure after one incident that seems life threatening. In this is shows many properties in common with, and may be related to, PTSD

The onset of **rheumatoid arthritis** usually follows one of two patterns: occurring after a single abrupt life event or after a long-standing series of unpleasant experiences. The former category includes bereavement, separation, divorce, termination of employment or abrupt financial loss. The latter includes long-term discord with a spouse or co-worker, an increase in work pressure or pressures of childcare.

Autoimmune **thyroid disease** includes Graves hyperthyroidism, Hashimoto's hypothyroidism, as well as sub-acute and chronic forms of thryroiditis that may actually represent a continuum of thyroid dysfunction. Stressful life events associated with chemical exposure to crude oils appear to precede the onset of the disease. [47]

Over half of new **insulin dependent diabetes mellitus** (IDDM) cases were found to be psychologically stressed over issues of bereavement and separation. Studies indicate a latency of as much as three years from the stressful events before the onset of disease. Stressful events included problems at work, difficulties in the home pregnancy, accidents and febrile disease. The results were confirmed in three other studies and show that a majority of IDDM patients experience some stressful life event from 1-3 years before the onset.

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[45] Canadian Cancer statistics 1990….2001, National Cancer Institute, Statistics Canada, Health Canada
[46] Autoimmunity by I. Todd and C. Davenport ch. 13 in The Biology of Disease, ed. Johnathon Phillips and Paul Murray pub Blackwell Science 1995
[47] Wayne Johnson, Sundre AB personal communication December, 2001

Multiple Sclerosis patients in general report a stressful life event occurring just prior to the onset of the disease. [48] [49] Many of these reports are somewhat anecdotal, as the date of onset of the disease is difficult to determine as well as the definition of an ideal control population.

The crude rate of MS in Alberta in one area (Black Diamond Health District) exposed to extensive flaring is unofficially estimated to be about 400 new cases annually per 100,000 pop. [50] Compare this to an age-standardized incidence rate of All-Cancer in for all of Alberta of 380 cases per 100,000 women and 451 cases per 100,000 men in 1998. [51]

It is now recognized that the neuro-endocrine and immune systems can be influenced by external stress once it is perceived by the CNS. Chronic stress may cause significant dysfunction of the immune response leading to increased susceptibility to disease. It has been proposed that elevations of neuro-endocrine hormones may be responsible for the immuno-suppression following acute or chronic stress. [52]

CYCLICAL CHEMICAL EXPOSURE & STRESS

6. **Chemical sensitivity:** The influence of xenobiotic chemicals on the human limbic system has been widely investigated. The limbic system is that portion of the brain that controls emotions and is strongly influenced by chemical pollutants. Bell et.al. [53] have established that the answers to five questions are significantly associated with chemical sensitivity in an individual. Sixty percent of 643 persons questioned

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[48] Barbara Graff Vulcan AB, personal communication March 2001
[49] Terri Reuser Turner valley AB, personal communication March 2001
[50] Dr D. Swann MOH for Turner Valley-Black Diamond Health District private communication 2001
[51] Canadian Cancer Statistics 1998 National Cancer Institute of Canada pg 20
[52] The Effects of Stress on Autoimmune Disease, Whiteacre, CC, Cummings SO, and Griffin AC in Handbook of Human Stress and Immunity Glaser R and Kiecolt-Glaser JK ed. Academic San Diego 1994, pg 77-81
[53] Bell IR, Schwartz, GE, Peterson JM, Amend D *Self-reported illness from chemical odors in young adults without chemical syndromes of occupational exposure.* Arch Env. Health 1993:48(1):6-13

reported feeling ill from one or more of the five chemical: 15% identified the smell of at least 4 chemicals as making them ill. Women tended to be more susceptible than men. The syndrome of feeling ill from smells has been defined as cacosmia. The five question screen includes feeling ill from pesticides, from car exhaust, from drying paint, from new carpet, and from perfume.

Irritability, joint and muscle pain, daytime tiredness, constipation, indigestion, headaches, trouble sleeping at night, memory trouble, difficulty concentrating, daytime sleepiness or grogginess and ringing in the ears are all associated with feeling ill from smells. The more of these signs / symptoms that one can answer "yes" to, the greater is the individual's chemical sensitivity.

Mainstream medical practitioners sometimes dismiss complaints of chemical sensitivity as "all in your head". The medical profession is moving, however glacially, to recognize the phenomenon and have developed criteria for chemical sensitivity. The criteria include:

- the disorder is acquired in relation to some documentable exposure; symptoms include more than one organ system;
- symptoms recur and abate in response to predictable stimuli;
- symptoms are elicited by exposures to chemicals of diverse structural classes;
- symptoms are elicited by exposures that are demonstrable though low;
- exposures must be very low and no single widely used test of organ function can explain the symptoms.[54]

The sensitized person will experience adverse effects at chemical concentrations that are very much below anything expected in conventional testing. These concentrations are quite low and generally are dismissed as inconsequential.

EX 9: Chemical Sensitivity Genesis & Development

[54] Harrison R *Multiple chemical sensitivities* in Environmental Medicine ed Brook S et.al. Mosby 1995 pg 369

We showed here that a great variety of hydrocarbons will be formed in a flare and the downwind plume will contain these, particulates, and potentially dioxins. If the gas flared is sour an even larger variety of sulfur containing hydrocarbons will be formed. These are particularly malodorous and noticeable at low level. We have shown that the level at which a selection of hydrocarbons can be sensed is much above an acceptable daily intake prepared in an ultra-conservative approach.

If something is present but cannot be smelled the limbic system will nevertheless react, though the observer will not be initially aware.

The limbic system is part of the basic defense of the organism and reacts to immeasurable small amounts.

Therefore it is clear to us that a heightened state of awareness will result in susceptible individuals reacting defensively, though initially they are not aware of this heightened state.

This condition can be repeated over months or years, and, depending on the amount of material present, the receptor, "you", will hardly be aware that they have been reacting.

With each cyclical repeat the organism reacts to progressively lower amounts of stimulus. This is very classical conditioning.

It has been shown by Bell et.al. that the optimum conditions to induce chemical sensitization are cyclic "waves of stimulus". This corresponds exactly to a cyclical stimulus such as hydrocarbons from a flare carried by changing winds to a receptor.

In the presence of malodorous hydrocarbons or sour gas flaring emissions the level can be below the threshold of smell until a large gust brings the concentration to a level of awareness with predictable consequences. An explosion of rage followed by a nearly instantaneous headache, sore aching muscles and rapid constriction of the bronchus and lungs. The rage is a direct reaction from the limbic system fight-or-flight mechanism. The headache is from a constriction of blood vessels in the brain. The sore, aching muscles take longer to appear because they depend on the circulation to distribute the chemicals systemically. The subsequent let down is when the biochemical state of arousal is reduced toward normal and "you" have time to think again. The chemicals circulating in the brain operate on the aminergic neuro-transmitters with mood altering effects.

CONCLUSION

1: Minimal conditions of flaring volume such as represented here and previously, correspond to flaring less than 10,000 m3/d of waste gas-oil-water mix. The average volume flared in batteries is 607,000,000 m3/day. The volume we have examined (8600 m3/d) corresponds to less than about 0.0014 percent of the average volume sent to flaring in batteries per day. 2: Under these minimal conditions of exposure we show that a measurable risk of cancer is identified.

3: We show that in geographic areas called CD's with extensive flaring there is an elevated rate ratio for cancer.

4: We show that in the CD's with extensive flaring

> Dioxins are formed when chloride is burned in the presence of organic matter. Dioxins exposure poses a recognized risk of >5 for soft tissue sarcomas.
>
> Soft tissue sarcomas are included in the category of bone and connective tissue cancers collected. These cancers show an elevated rate ratio in all those geographic areas with extensive flaring activity.
>
> Radioactivity is a common contaminant of oil and gas wells. It is common in Alberta and can be identified by its auto-radiographic tendency to blacken a film. The most common cause of Thyroid cancers is radioactivity. Thyroid cancers have an elevated median rate ratio in those geographic areas with extensive flaring operations.

The most common cause of pancreatic cancer is diabetes. Chronic exposure to increased levels of dioxins appears to lead to increased risk of Type II diabetes.[55]

Finding one of these would be enough to rebut the arguments of the oil and gas industry that their activities do no harm to anyone. Finding three or more is sufficient to prove our point. Flaring is dangerous to all human and animal life in proximity to the flare.

We close with the observation that

"Rowing harder doesn't help when the boat is headed in the wrong direction"

This is our attempt to change the direction of the boat.

Thank you

James Argo PhD

[55] Steenland K, Calvert G, Ketchum N, Michalek J Dioxin and diabetes milletus: an analysis of the combined NIOSH and Ranch Hand data Occ. Environ. Med. 2001,58, 641-648