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Skeena Region

PAH: Ambient Air Quality Monitoring in British Columbia

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PAH EMISSIONS

Production

- incomplete combustion of high-molecular-weight hydrocarbon species;
- favoured by an oxygen-deficient flame, temperatures in the range of 650-900deg.C, and fuels which are not highly oxidised;
- due to their low vapour pressures, most PAHs will immediately condense and form a thin film (adsorb) onto soot particles or form as very small particles themselves.

Sources

- natural
- anthropogenic
- aluminum manufacturing
- catalytic cracking
- fuels

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SINKS

- Removal of PAHs from the atmosphere is facilitated through photo-oxidisation. PAHs react rapidly with NO_x, HNO_3, SO_2, SO_3, and H_2SO_4 (the latter reactions form sulphinic and sulphonic acids).
- These processes tend to yield carcinogens of increased potency.
- An obvious method to prevent exposure is to reduce emissions or the possibility of exposure.

HEALTH EFFECTS

- Deteriorating PAH air quality will cause increasing adverse effects on human health. (Fellin and Davis, 1991).
- PAH are readily absorbed; detectable levels can be observed in most internal organs from minutes to hours after administration. They have a slight preference for adipose tissue.
- Acute toxicity appears to be low for humans (no recorded cases), however, subchronic and chronic toxicity exists. Some PAHs are known immunosuppressants. Animal studies have shown B[a]P to be teratogenic, embryotoxic, and mutagenic. (No data exist on the developmental or reproductive toxicity of PAH to humans).
- Epidemiological investigations into lorry drivers, wharfpersons, jamb cleaners, tar workers, and gas and coke over workers have revealed an elevated risk of lung cancer (Sollenberg, 1983). Exposure to PAH (especially B[a]P) increases one's risk of developing cancers.
- Inherent in human-based assessments are a mix of PAHs. Therefore, exposure to any single PAH compound in occupational or environmental situations is unlikely (US EPA, 1984) and epidemiological study of groups exposed to a single PAH is infeasible (Bureau of Toxic Substance Assessment, 1989).
- Little is known at present concerning the potential interactions of PAH compounds themselves. Interaction between individual PAH may result in additive, synergistic, or antagonistic effects (Bureau of Toxic Substance Assessment, 1989).
- It seems likely that the potential for synergism is far outweighed by the difference in carcinogenic potency between B[a]P and other PAHs (US EPA, 1984).

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AIR QUALITY MONITORING

Ambient Concentrations and Apportionment

- Previous studies which found a strong linear correlation between B[a]P and total PAH now viewed as controversial since the B[a]P:PAH ratio is site and source-dependent. Auto emissions, for example, are low in B[a]P whereas emissions from burning of refuse are high in B[a]P.

Relative Potency and the Dose Additivity Assumption

- Total exposure units equivalent to B[a]P in a mixture to which an individual is exposed is calculated by taking the sum of the products of the relative potencies and the exposure levels for each PAH. This determines the joint response to a group of carcinogenic PAH:

$$T_{PAH} = \sum_{j=1}^{n} R_j y_j + x$$

where:

TPAH = Total PAH exposure;

n = the total number of indicator PAHs exclusive of B[a]P;

yj = the exposure to the jth indicator PAH;

x = exposure to B[a]P;

Rj = Rj relative potency of the jth indicator PAH compared to B[a]P (see Table 1).

Table 1: Relative potency of various PAHs. The Bureau of Toxic Substance Assessment (BTSA; 1989) assessment is adapted from Deutsch-Wenzel *et al.* (1983); the ICF-CA (ICF-CA; 1988) data are based on 11 experimental studies which support the consistency of the relative potency number and/or those studies considered the "most reliable".

Compound	Relative Potency		BTSA 95%
	ICF - CA	BTSA	Confidence Intervals
Anthanthrene	0.320	0.19	0.02 - 1.32
Benz [a] anthracene	0.145		
Benzo [a] pyrene	1.00	1.00	0.02 - 1.32
Benzo [b] fluoranthene	0.140	0.11	0.04 - 0.74
Benzo [ghi] perylene	0.022	0.01	0.00 - 0.06
Benzo [j] fluoranthene	0.061	0.03	0.005 - 0.24
Benzo [k] fluoranthene	0.066	0.03	0.005 - 0.24
Benzo[e] pyrene	0.004	0.003	0.00 - 0.02
Crysene	0.0044		
Cyclopentadieno [cd] pyrene	0.023		
Dibenz [ah] anthracene	1.11		
Indeno [1,2,3-cd] pyrene	0.232	0.08	0.01 - 0.57
Pyrene	0.081		

ICF-Clement Associates (ICF-CA). 1988. *Comparative Potency Approach for Estimating the Cancer Risk Associated with Exposure to Mixtures of Polycyclic Aromatic Hydrocarbons.* Interim Final Report. Fairfax, USA.

In the case of mixtures of carcinogenic PAHs, dose additivity has been postulated to be a reasonable assumption because, as a group, PAH have similar biological mechanisms of action: they appear to be metabolised to similar reactive derivatives that interact with DNA and produce histologically similar tumours at the site of introduction (IARC, 1983 in ICF-CA, 1988).

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Air Quality Objectives

- Although we have the relative equivalency estimates for B[a]P and the lifetime cancer risk assessments, few ambient standards have been set for the general public regarding exposure to PAH.

Table 2. Polycyclic Aromatic Hydrocarbons (PAH) Standards (Benzo (a) Pyrene)

ASIL denotes acceptable source impact levels.

Jurisdiction	Averaging Time	Concentration (ng m-3)
Michigan, USA		0.3a
Ontario	30 minutes	3.3b
Connecticut, USA	8 hours	100c
Ontario	24 hours	1.1d 0.3e
USSR (now defunct)	24 hours	1.0a

Kansas, USA	1 year	0.303c
North Carolina, USA	1 year	33.0c
Ontario	1 year	0.22f 0.30g
Vermont, USA c	1 year	0.3
Pennsylvania, USA	1 year	0.7a
Philadelphia, USA c	1 year	0.7
Washington, USA	10-6 risk ASIL annual average	0.6

NOTE: most long-term averages are in the 0.3 - 0.7 ng m-3

a. St. Germain (1991)
b. Single source, point of impact (Sterling, 1990). Air Resources Branch (1991) indicates this is an interim standard and is part of Regulation 308 of the *Environmental Protection Act*.
c. Numbers supplied in an unreferenced table by Barrid Manna, Environmental Safety Program, Waste Management Branch, Victoria and by St. Germain (1991).
d. Single source; Regulation 296 of the *Environmental Protection Act*; Air Resources Branch (1991).
e. All sources, Sterling (1990); could not be confirmed by Marg Tepina, Air Resources Branch, Ontario MoE.
f. Single source; Regulation 296 of the *Environmental Protection Act*; Air Resources Branch (1991).
g. All sources; Air Resources Branch (1991).

Human Health Risk Screening Model

- It is not possible, at present, to perform a quantitative cancer risk assessment for PAH as a class.
- Sufficient data are available for B[a]P from which cancer risk estimates may be derived.
- B[a]P-equivalent exposure units can be substituted into a dose response model for B[a]P to obtain the cancer risk associated with exposure to the PAH mixture.
- Total cancer risk from exposure to a mixture of PAHs should be based on measurements of the concentrations of its carcinogenic components and relative estimates of their potency.
- Carcinogenic potency factor (CPF) of 0.453 (mg kg-1 day-1)-1 has been estimated using rodent tumour response data following exposure to B[a]P (ICF-CA, 1988).
- The carcinogenic risk equation is (from Office of Emergency and Remedial Response, 1986) 1:

$$RISK = CDI * CPF$$

where:

RISK = Carcinogenic (10-5 used for probable human carcinogens such as B[a]P;

CDI = Chronic daily intake (projected human intake of a chemical averaged over a long time period, up to 70 years, and expressed as mg kg-1 day-1);

CPF = Carcinogenic potency factor (mg kg-1 day-1)-1.

therefore:

$$CDI = \frac{RISK}{CPF}$$

Future Initiatives

- Re-initiate Smithers PAH monitoring program if resources ever become available again.
- One year, four station program with meteorological support in Kitimat.

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