BRITISH COLUMBIA HYDRO AND POWER AUTHORITY

HAT CREEK PROJECT

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Prepared for
British Columbia Hydro and Power Authority

Air quality and climatic effects of the proposed Hat Creek project

Appendix G
Epidemiology

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APPENDIX G

G1.0 INTRODUCTION

The proposed Hat Creek Project of British Columbia Hydro & Power Authority (B.C. Hydro) would be a major new thermal power facility in British Columbia, consisting of a (nominal) 2000 Mw power plant, a coal mine, and associated facilities. The project's potential environmental effects have been investigated in a series of detailed environmental studies. While many potential effects of the proposed power plant, mine, and associated facilities have been evaluated, none is more important than the potential for direct human health effects. Public health considerations have, therefore, been addressed specifically and separately in this Appendix. The primary purpose of these studies is to provide a basis for recommendation of ambient air quality guidelines that are consistent with human safety and well being. The material presented herein is intended to satisfy the criteria specified in Appendix D3 of the Terms of Reference for Detailed Environmental Studies.

A major consideration in terms of potential health effects concerns material emitted directly to the atmosphere due to mining activities and power plant operation. Most of the solids, liquids, and gases ejected from these sources will be harmless material from a health perspective; examples include air (in excess of combustion requirements) and carbon dioxide from the generating units, water vapor from cooling towers, and nontoxic dust from the mine. However, some substances emitted during the operation of the project warrant careful evaluation in terms of possible adverse effects on human populations exposed to them. Two categories of potentially toxic contaminants are identified and discussed in this Appendix.

Five common atmospheric contaminants of coal-fired power plants and surface mines are considered in terms of available health data and recommended ambient guidelines. These include sulfur dioxide (SO_2),

total suspended particulates (TSP), nitrogen oxides (NO_x), carbon monoxide (CO), and photochemical oxidants (most notably O_3). Predicted incremental effects of Hat Creek Project emissions on local and regional ambient levels of these substances are discussed at length in the report to which this study is an Appendix.

The second group of contaminants studied includes fourteen selected trace elements, suspended sulfates and nitrates, polycyclic organic matter, and nitrosamines. Each of these has been studied in terms of existing regulations, projected emissions and health risks.

B.C. Hydro has engaged Western Research & Development Ltd. and Greenfield, Attaway and Tyler, Inc. (Flow Resources) to evaluate health effects of both sets of contaminants with regard to the Hat Creek Project. Their work, in conjunction with the air quality studies of Environmental Research & Technology, Inc. has been documented in a two-volume report: "Public Health Considerations Relative to the Hat Creek Project, Volume I, Ambient Air Quality Criteria; Volume II, Trace Contaminants." The remainder of this Appendix consists of these reports.

HAT CREEK ENVIRONMENTAL STUDIES

EPIDEMIOLOGY
VOLUME I
MAJOR CONTAMINANTS

Prepared for:

British Columbia Hydro and Power Authority

Prepared by:

Western Research & Development
and
Flow Resources Corporation

April 1978

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1.0 INTRODUCTION

The purpose of this report is to recommend ambient air quality achievement guidelines for the proposed Hat Creek Project. British Columbia Hydro & Power Authority engaged Western Research & Development and Flow Resources Corporation to assist in establishing these guidelines as part of the Hat Creek Environmental Studies. These achievement guidelines are intended to provide a basis for evaluating potential impacts to public health as well as for determining the need and degree of control required for the release of potential contaminants. The report consists of two volumes.

Volume I

Volume I considers five commonly occurring contaminants: sulphur dioxide, suspended particulate matter, nitrogen oxides, carbon monoxide and oxidants. This volume of the report is intended to:

- provide a concise review of the relevant ambient air quality regulations of the provinces of British Columbia, Alberta, Ontario, the states of Washington, Idaho and Montana and the Canadian and United States federal governments.

- provide an overview of public health data relevant to these contaminants and recommend public health based guidelines.

- compare existing ambient air quality objectives of British Columbia to these recommended public health based guidelines.

- provide to B.C. Hydro with ambient air quality achievement guidelines for the region surrounding the Hat Creek Thermal Project.

The selection of jurisdictions to be included in the review was based in part on the geographic location of the proposed plant, and on the historical development of air pollution control legislation. In Canada the three provinces that have lead in the development of air quality legislation are British Columbia, Alberta and Ontario. Thus, a comparison that includes these provinces was considered appropriate.

Inclusion of the Canadian federal guidelines for ambient air quality recognizes the Federal Government's effort in the development of uniform objectives across Canada.

The inclusion of United States federal and state jurisdictions recognizes the prominent position of the United States Environmental Protection Agency in establishing ambient air quality objectives and the geographic locations of the three states.

A data base is presented to establish public health (based) guidelines for each contaminant and to discuss the data evaluation process. It would be repetitious to quote and discuss the hundreds of epidemiological studies relating to the subject. Instead, this report attempts to highlight the more relevant epidemiological findings, and, based on the review of the applicable literature and up to date medical evidence, provide a rationale for ambient air quality achievement guidelines for the proposed Hat Creek Project.

After establishing the relationship of British Columbia's ambient air quality objectives to those of the other jurisdictions, and relevant public health based guidelines for each contaminant, a direct comparison of the two is made. Based on this comparison air quality achievement guidelines are recommended for B.C. Hydro.

Volume II

Other potential contaminants, including trace elements and selected decay products of the above mentioned contaminants, such as sulphates and nitrates, are the subject matter of Volume II. For these contaminants a public health risk assessment is made based on predicted ground level concentrations and existing health data.

2.0 RECOMMENDATIONS

Existing ambient air quality objectives and relevant health data have been reviewed for the purposes of recommending public health based ambient air quality achievement guidelines for the B.C. Hydro Hat Creek Project. This review was conducted for sulphur dioxide, suspended particulate matter, nitrogen oxides, carbon monoxide and oxidants. The recommended guidelines are:

Contaminant	Units	1 hr	8 hr	24 hr	Annual
Sulphur dioxide	ug/m^3	n.r.*	n.r.	300-400	90-100
Suspended particulate	ug/m^3	n.r.	n.r.	150-300	60-100
Nitrogen oxides as NO_2	ug/m^3	n.r.	n.r.	n.r.	100-200
Carbon monoxide	mg/m^3	40-60	15-20	n.r.	n.r.
Oxidants as O_3	ug/m^3	150-300	n.r.	n.r.	n.r.

* no recommendation

Guidelines have not been recommended for certain time periods for several contaminants, even though there exists ambient air quality objectives for these conditions. The reasons for not recommending guidelines in every case either derive from a lack of health data relative to the contaminant for the exposure duration, or the problem with extrapolation of data from other exposure conditions to these time periods.

3.0 REVIEW OF EXISTING AMBIENT AIR QUALITY CRITERIA

Current ambient air quality criteria are, for the most part, the result of legislation passed during the past ten years. Prior to 1967 most air quality control regulations were contained in the legislation of departments such as public health. In the mid sixties the general concern for air quality resulted in the establishment of environmental protection departments and the creation of comprehensive environmental protection legislation.

A comparison of the Canadian federal and provincial agencies and their regulations to those of the USA federal and state agencies and their regulations shows a distinct difference in philosophy and responsibility.

In Canada the Provinces have the direct responsibility of controlling air pollution. Their role can be summarized as follows:

- the enactment of appropriate provincial legislation;
- the setting of control standards and the enforcement of control measures;
- the establishment of programs and priorities within the framework of agreed national air quality objectives.

The Canadian Federal Government takes a protective management approach to environmental control. The primary objectives of their program are:

- promotion of a uniform approach across Canada;
- provision of the mechanisms and institutions needed to ensure that all measures to control air pollution can be taken and for direct action by the Federal Government when this is necessary.
- establishment of a leadership role for the Federal Government through various activities and incentives in dealing with the Provincial Governments.

In addition the Federal Government has the responsibility of legislating air pollution control for the Yukon, Northwest Territories and Crown controlled lands such as the national parks.

The consequences of having the primary responsiblity of enacting air quality control legislation rest with the provinces has been twofold; considerable disparity exists in the regulations enacted by each of them and some provinces have lagged behind others in establishing regulations.

In the United States the federal government is responsible for air quality management and through the Air Quality Act (1967) and the Clean Air Act and ammendments (1970-) national primary and secondary ambient air quality standards have been established. Each state has been required to develop compliance programs that must be approved by the Environmental Protection Agency (EPA). In these programs the establishment of time schedules for achievement of specified goals are perhaps as important as the goals themselves. The individual states may adopt directly the EPA regulations or may, if they wish, proceed to develop more stringent regulations based on their special conditions or requirements. If an acceptable compliance program is not developed then the EPA can impose one on the state. Thus, a strong federal Agency provides leadership, technical direction, and the legal power to effectively control regional as well as national air quality.

3.1 Jurisdictions reviewed

Ambient air quality criteria for the provinces of British Columbia, Alberta and Ontario, the Canadian Federal Government, the United States Federal Government and three states, Washington, Idaho and Montana (all bordering on British Columbia) were reviewed.

In 1975 British Columbia published "Pollution Control Objectives for Food Processing, Agriculturally Orientated, and Other Miscellaneous Industries" (1) to meet the requirements of the Pollution Control Act, 1967. The objectives for ambient air quality and emission rates are listed under three levels: Level A, Level B and Level C. Quoting directly from the Objectives, Section 1.4.

> "All new or proposed discharges should meet Level A objectives. All existing discharges should meet Level C either immediately or within the shortest possible time technically feasible to do so. Existing discharges should be upgraded to interim Level B and ultimately to Level A by a staged program of improvement."

By complying with a stated level for maximum emission rates it is anticipated that the comparable level of ambient air quality will be achieved. For example if Level B emissions rates are achieved then Level B ambient air quality objectives would be met.

Canadian Federal "maximum desirable" and "maximum acceptable" ambient air quality guidelines were published in the Canada Gazette in March 1974 (2). The published guidelines reflect the policy of the Federal Government (3) to establish air quality guidelines for three categories. These categories are referred to as "desirable", "acceptable", and "tolerable", indicating successively higher concentration levels. To date "maximum tolerable" guidelines have been proposed but not published.* Federal Government emission guidelines for thermal power plants are currently being developed. (4)

Air pollution control in Alberta, prior to 1971, was carried out under the Public Health Act. In 1971 Alberta passed the Clean Air Act. This act provides the basis for control of maximum permissible ambient concentrations and mass emission rates from individual sources. Although only particulate emission rate regulations have been specified in the 1975 Clean Air Regulations (5) the permit system used by Alberta allows for the control of the mass emission rate of each contaminant from any source.

Air quality control in Ontario was placed under the direct control of the provincial government when the Air Pollution Control Act was passed in 1967. Previously control had been delegated to local municipalities under the Municipal Act as well as through earlier versions of the Air Pollution Control Act. Current regulations are those contained in the

Environmental Protection Act (1971) as ammended up to February 1976 (6). Regulations outlined in this Act control emission rates based on specified calculation methods and ambient air quality criteria for each contaminant. Criteria exists for some 23 contaminants including those reviewed in this report.

In the United States national primary and secondary ambient air quality guidelines were published in the Federal Register (7). In addition, specific legislation has been adopted to control the contaminant emission rates with respect to the total heat input to the facility. The states of Washington, Idaho and Montana have similar federally approved compliance programs (references 8, 9 and 10 respectively).

Terminology used by the various jurisdications makes it somewhat difficult to select an appropriate single term to properly refer to the standards, guidelines, objectives or regulation. For the jurisdictions reviewed the following terminology is used.

•	Objectives	- British Columbia	: three levels
•	Guidelines	- Canadian Federal	: three levels (only two published)
•	Standards	- Alberta	: one level
•	Criteria	- Ontario	: one level
•	Standards	- USA Federal and State	: two levels
•	Regulations	- orders issued by an executive authority of a government and having the force of law.	

* Note:

- On February 8th, 1978 "Maximum tolerable" guidelines were passed by the Federal Government. These values were not included in the report.
- The new guidelines do not affect our recommendations.

Table 3.1

AMBIENT AIR QUALITY STANDARDS OR CRITERIA

Contaminant	Averaging Time	Units	Ontario	Alberta	Canada Federal[a] 1	Canada Federal[a] 2	B.C. Level A	B.C. Level B	B.C. Level C	USA Federal	Washington	Idaho	Montana
Sulphur dioxide	Arn. Arith	ug/m^3	55	30	30	60	25	50	80	80 primary	50	80 primary	50
	24 hr.	ug/m^3	275	150	150	300	160	260	360	365 primary	260	365 primary	260
	1 hr.	ug/m^3	690	450	450	900	450	900	900		1050[b]		650
				525 (1/2hr)						1300 secondary (3 hr ave.)	650[b]	1300 secondary (3 hr ave.)	
Suspended Particulate	Arn. geo.	ug/m^3	60	60	60	70	60	70	75	75 primary	60	75 primary	75
	24 hr.	ug/m^3	120	100		120	150	200	260	260	150	260	200
										60 secondary		60 secondary	
										150		150	
Nitrogen oxides as NO_2	Arn. arith	ug/m^3		60	60	100				100[c]	100		
	24 hr.	ug/m^3	200	200		200							
	1 hr.	ug/m^3	400	400		400							
Carbon monoxide	8 hrs.	mg/m^3	15.7	5	6	15	5.5	11	14.3	10[c]	10	10	
	1 hr.	mg/m^3	36.2	15	15	35	14.3	28	35	40[c]	40	40	
Oxidants as O_3	Arn. arith				20	30							
	24 hr.	ug/m^3		30	30	50							
	hr.	ug/m^3	165	100	100	160				160[c]	160	160	
Reference			6	5	2	2	1	1	1	7	8	9	10

a. Canada Federal Level 1 - maximum desirable level and Level 2 - maximum acceptable level
b. 1 hr average may occur twice per 7 days.
c. primary and secondary standard.
d. maximum total for 30 days

3.2 Review of ambient air quality criteria

Ambient air quality standards or criteria for sulphur dioxide, nitrogen dioxide, carbon monoxide, oxidants and suspended particulate are summarized in Table 3.1. References for the source of information for each jurisdiction are indicated. All units have been standardized to metric units.

3.2.1 Sulphur dioxide

The ambient concentration of sulphur dioxide is regulated by the application of criteria that control the average concentration for three averaging periods. In Canada these are 1 hour, 24 hours, and annual time periods. In the USA a similar approach is taken but for Idaho and the Federal Government a 3 hour average is used instead of the 1 hour average. Figure 3.1 graphically presents all existing criteria for the 1 hour, 24 hour and annual time periods.

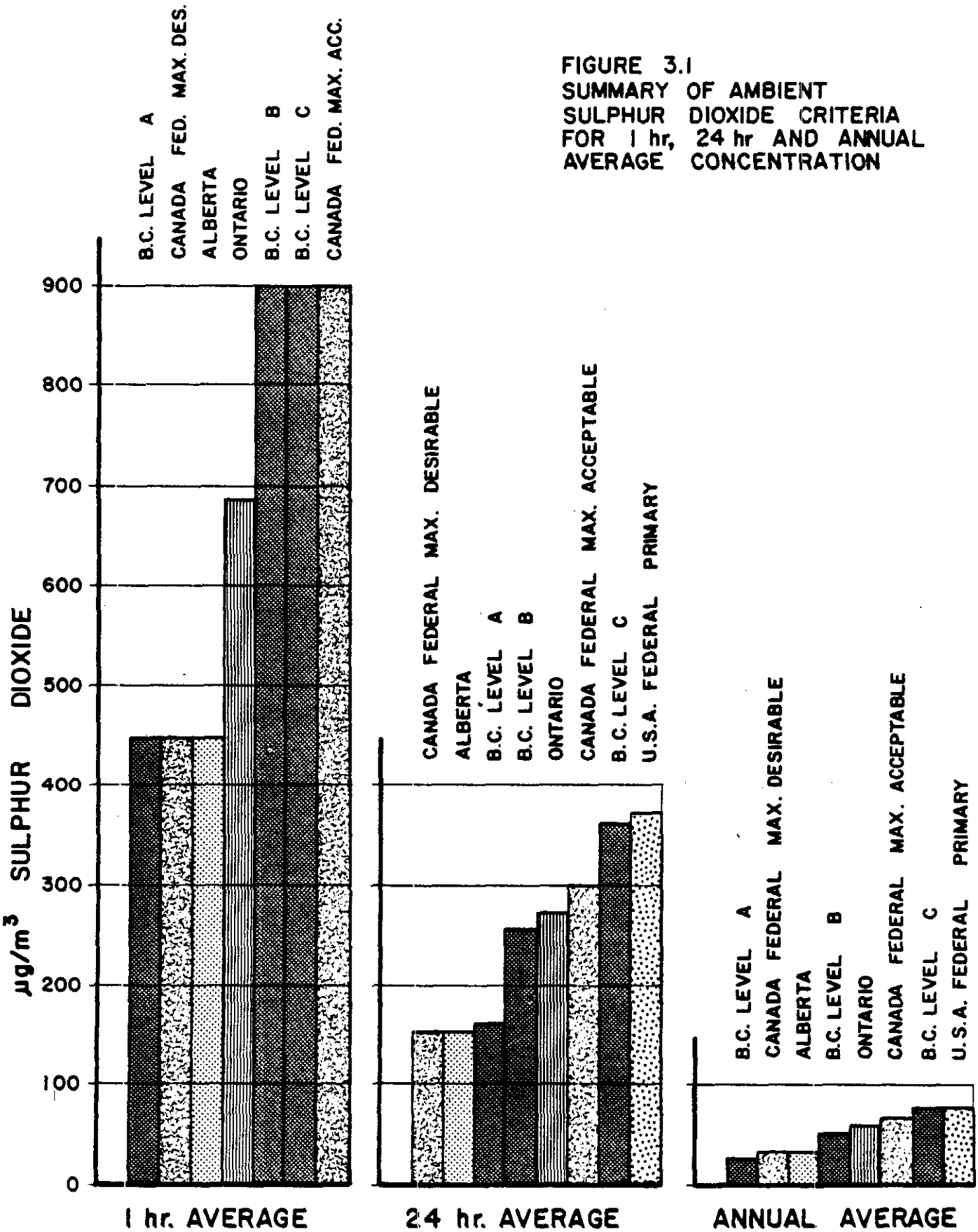
- 1 hour average

British Columbia Level A, the Canadian Federal "maximum desirable" level and the Alberta standard are the most stringent of all jurisdications. Conversely, British Columbia Level B and Level C, and the Canadian Federal "maximum acceptable" requirements are the least stringent. The Ontario 1 hour average requirement is midway between British Columbia Level A and Level B. As mentioned earlier USA Federal standard for 1 hour does not exist.

- 24 hour average

For this time period British Columbia Level A, the Canadian Federal "maximum desirable" level and the Alberta standards are again the most stringent. British Columbia Level C and the USA Federal primary standard are nearly identical and the highest of those compared. Intermediate between these two extremes are, in ascending order, British Columbia Level B, the Ontario criteria and the Canadian Federal "maximum acceptable" level.

FIGURE 3.1
SUMMARY OF AMBIENT
SULPHUR DIOXIDE CRITERIA
FOR 1 hr, 24 hr AND ANNUAL
AVERAGE CONCENTRATION



- Annual average

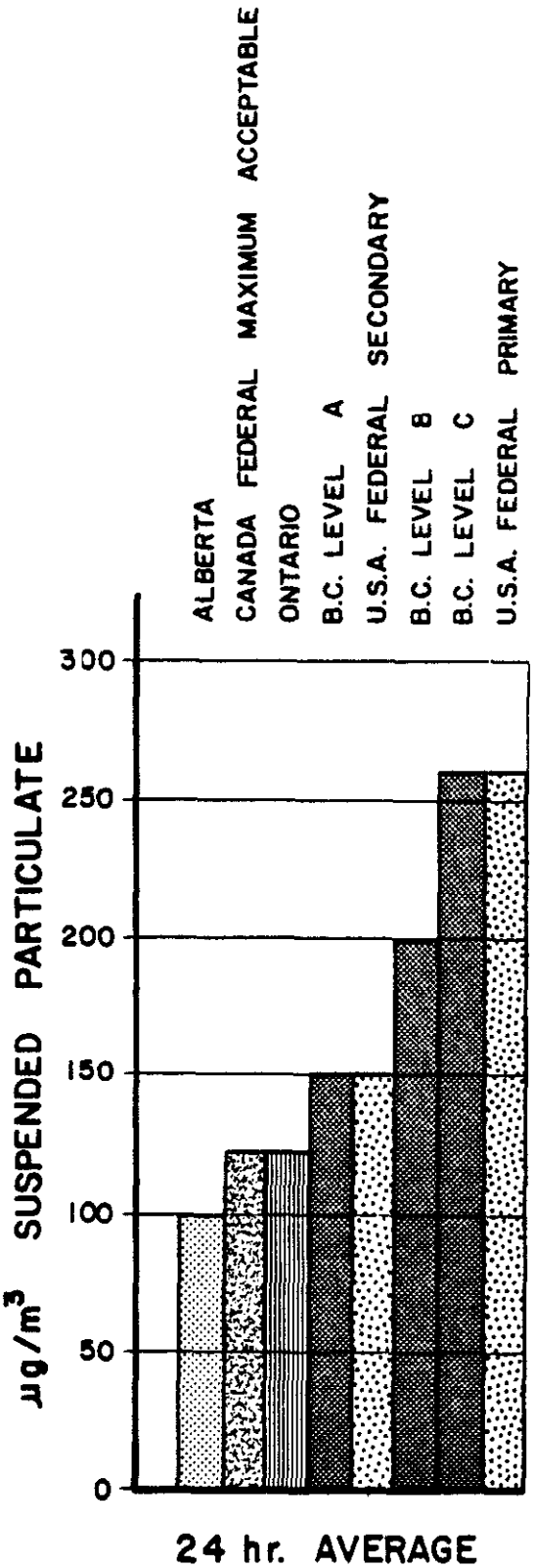
With only one exception, the ranking of the jurisdications by increasing concentration specified for the annual average criteria is the same as the 24 hour average. The exception is British Columbia Level A which is slightly more stringent than the Canadian Federal "maximum desirable" level.

- Other

The USA Federal secondary standard, a 3 hour average of 1300 ug/m^3 is considerably less stringent than any Canadian federal or provincial criteria.

Alberta also specifies a 1/2 hour average concentration standard of 525 ug/m^3 that is used for process design and control purposes. Thus this is perhaps the most significant sulphur dioxide standard for industry in Alberta. Ontario has a comparable emission control criteria for the calculated point of impingement concentrations.

From Table 3.1 the most stringent sulphur dioxide criteria specified by any of the three states review are those of Washington and Montana. They are comparable to British Columbia Level B.



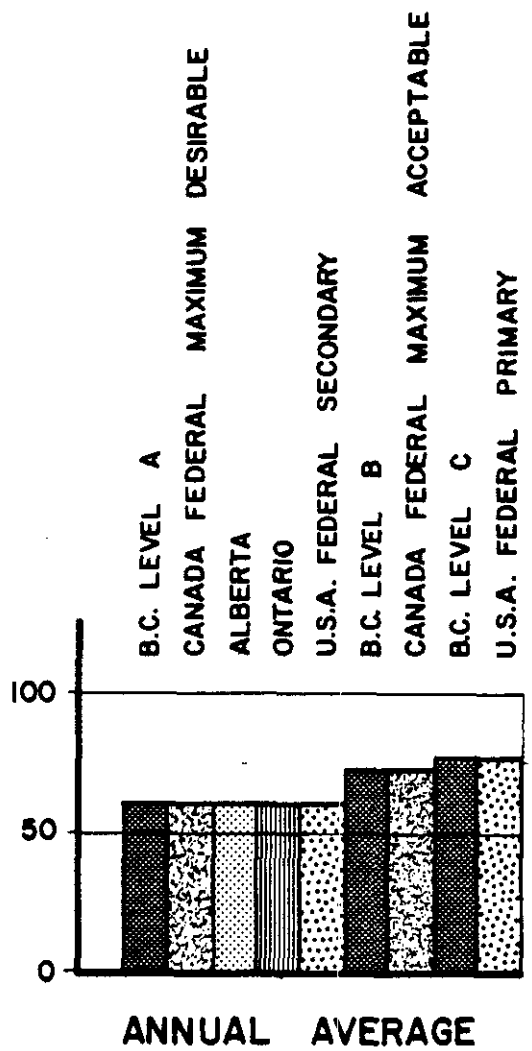


FIGURE 3.2
SUMMARY OF AMBIENT SUSPENDED PARTICULATE CRITERIA FOR 24 hr. AND ANNUAL AVERAGE CONCENTRATIONS

3.2.2 Suspended particulate

Criteria for suspended particulate have been established for 24 hour and annual time periods as presented in Figure 3.2.

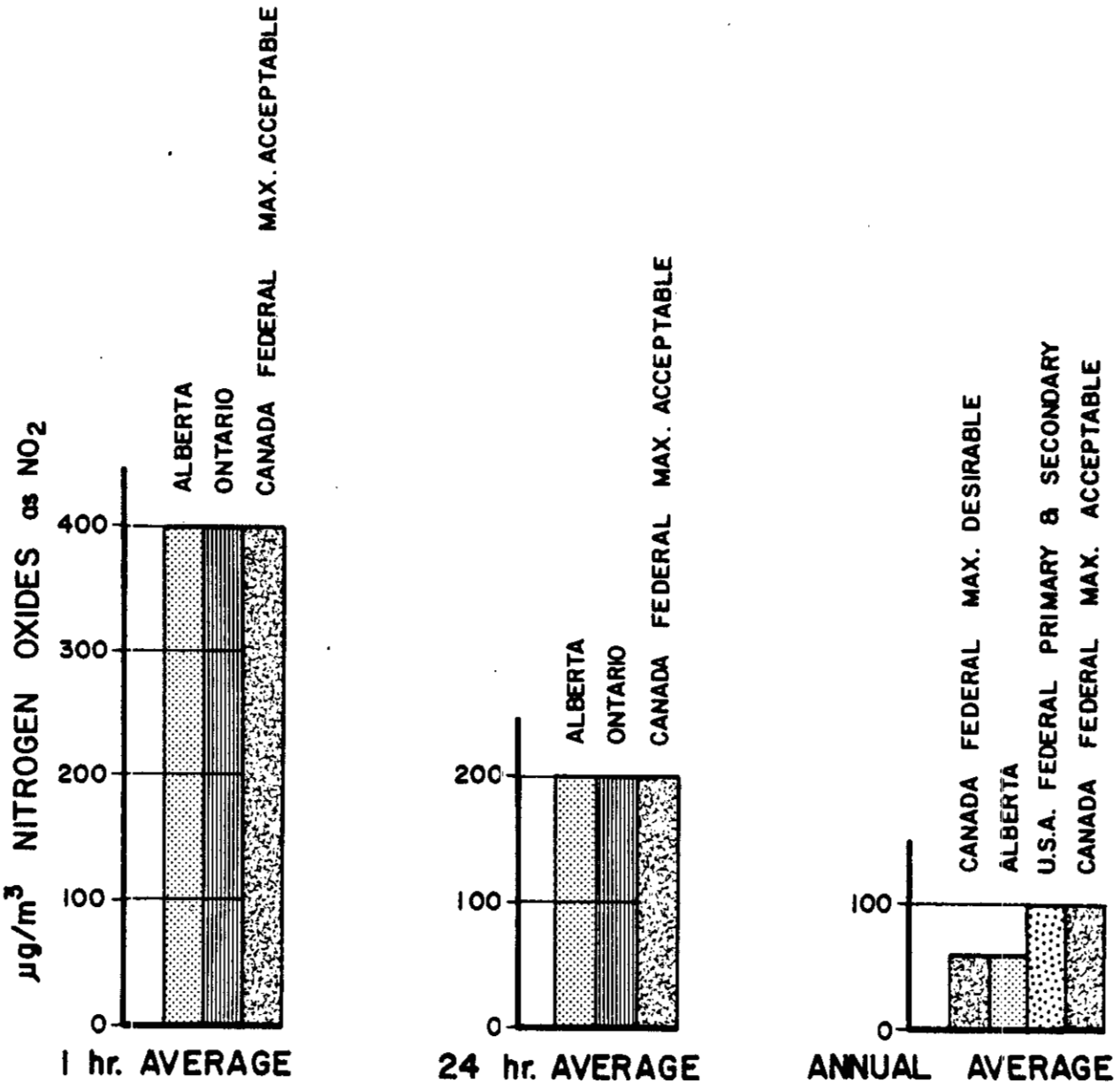
- 24 hour average

Of the jurisdications reviewed Alberta has established the lowest 24 hour average standard. British Columbia Level A and the USA Federal secondary standard are approximately equal and somewhat lower than British Columbia Level C and the USA Federal primary standard.

- Annual average

British Columbia Level A is identical to the Canadian Federal "maximum desirable" level and the Alberta, Ontario and USA Federal secondary standards. British Columbia Level B and the Canadian Federal "maximum acceptable" level are equal and slightly lower than B.C. Level C and the USA Federal primary standard.

FIGURE 3.3
SUMMARY OF AMBIENT NITROGEN OXIDES CRITERIA FOR 1 hr, 24 hr AND ANNUAL AVERAGE CONCENTRATION.



3.2.3 Nitrogen oxides

In Canada the control of nitrogen oxides is based on controlling the maximum ambient concentration for several averaging periods. In the USA only one averaging period, the annual average, is used. British Columbia does not specify ambient air quality objectives for nitrogen oxides but does specify nitrogen oxide emission rate objectives for thermal power plants. Existing ambient air quality criteria are summarized in Figure 3.3.

- 1 hour and 24 hour averages

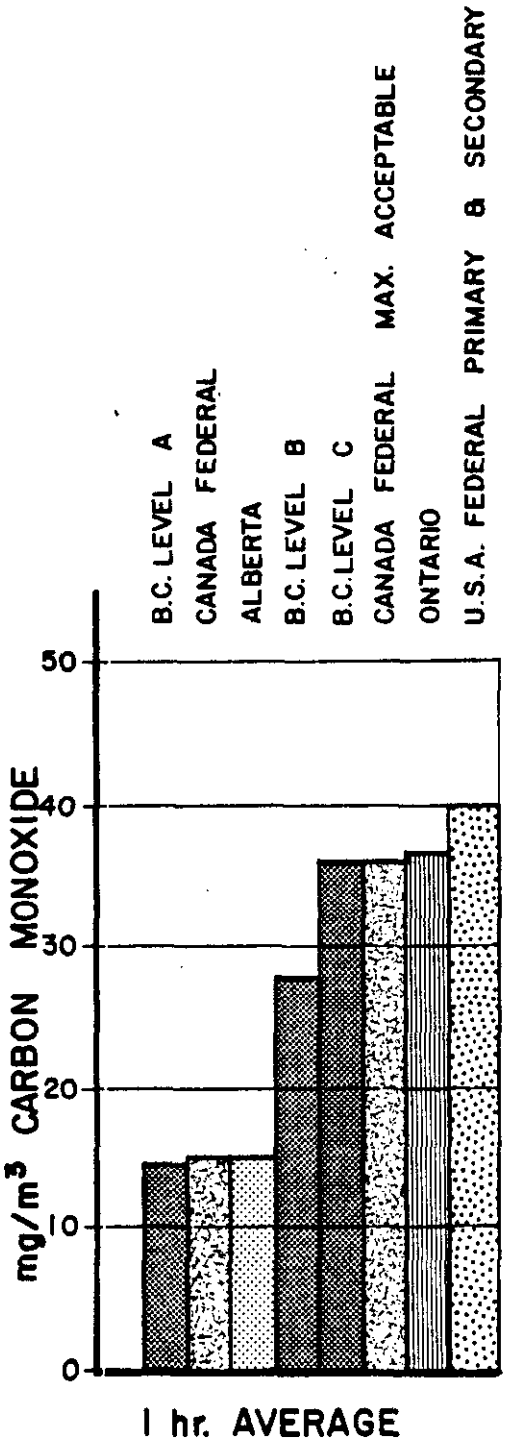
Alberta, Ontario and Canadian Federal "maximum acceptable" criteria for nitrogen oxides are 400 ug/m^3 and 200 ug/m^3 for the 1 hour and 24 hour average time periods respectively. No other jurisdications have standards for these time periods.

- Annual average

For the annual average, Canadian Federal "maximum desirable" and Alberta criteria are 60 ug/m^3. The USA Federal primary and secondary standards and the Canadian Federal "maximum acceptable" level are all 100 ug/m^3.

- Other

Of the three states reviewed only Washinton has a nitrogen oxide standard and it is equal to the USA Federal standard.



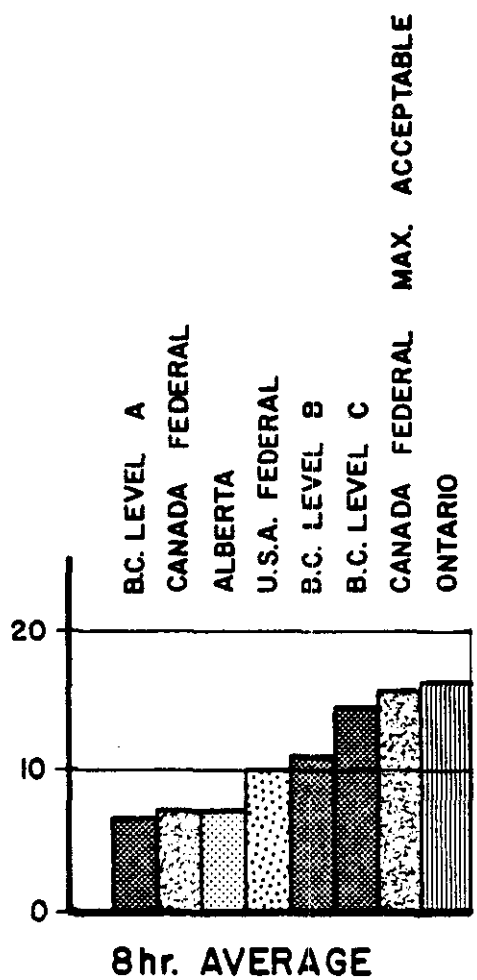


FIGURE 3.4
SUMMARY OF AMBIENT CARBON MONOXIDE CRITERIA FOR 1 hr AND 8 hr AVERAGE CONCENTRATIONS

3.2.4 Carbon monoxide

For all jurisdictions reviewed 1 hour average and 8 hour average carbon monoxide criteria have been established. Figure 3.4 graphically presents criteria currently in effect.

- 1 hour average

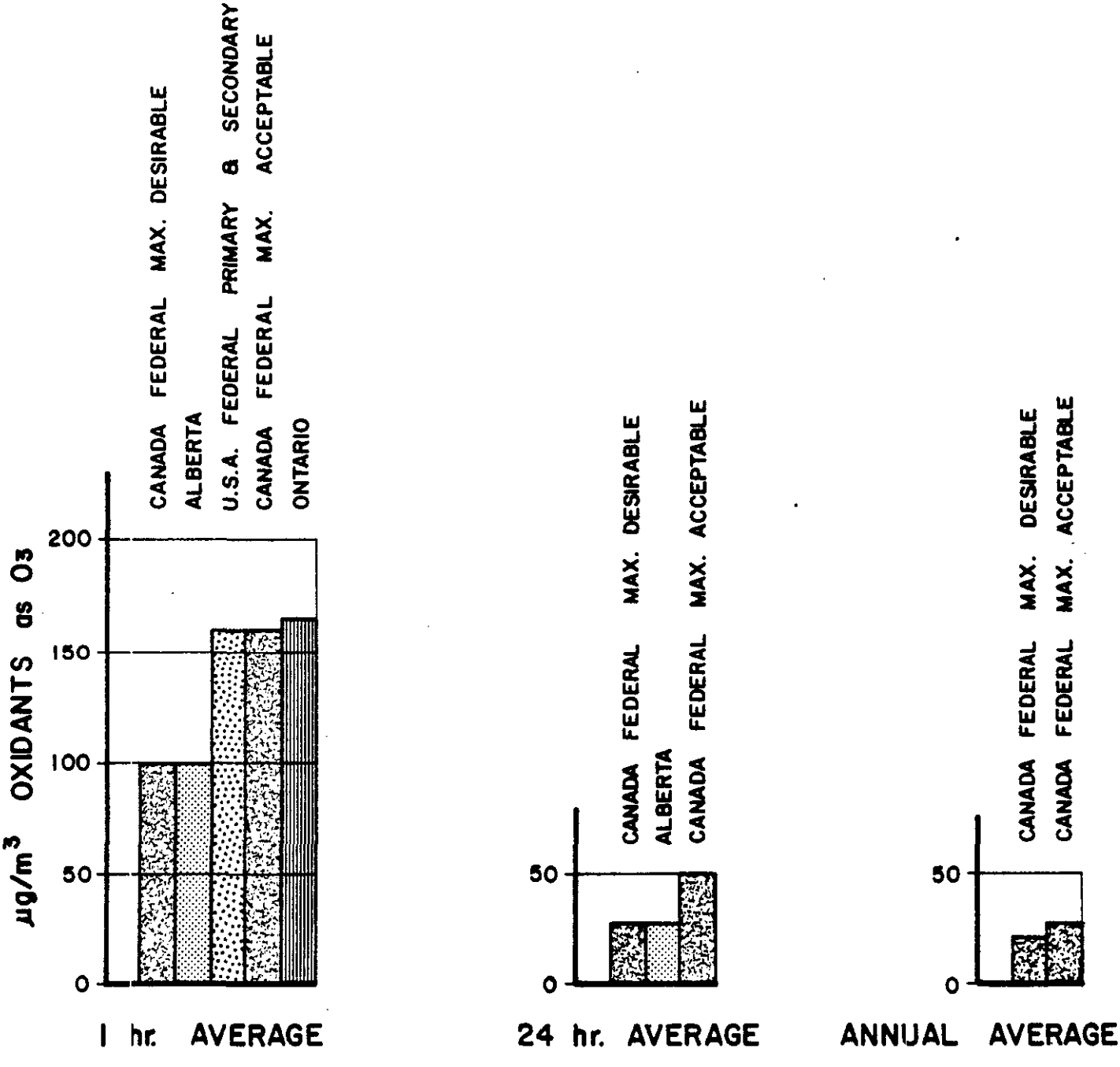
The lowest criteria for the 1 hour average concentration are British Columbia Level A, Canadian Federal "maximum desirable" level and the Alberta standard. The highest criteria are British Columbia Level C, the Canadian Federal "maximum acceptable" level, the Ontario criteria and the USA Federal primary and secondary standards. Intermediate between the high and the low is British Columbia Level B.

- 8 hour average

British Columbia Level A, the Alberta standard and the Canadian Federal "maximum desirable" level require the lowest ambient concentrations of carbon monoxide. Slightly higher maximum concentrations are set for British Columbia Level B and the USA Federal primary and secondary standards.

The highest maximum concentration criteria are British Columbia Level C, the Canadian Federal "maximum acceptable" level and the Ontario criteria.

FIGURE 3.5
SUMMARY OF AMBIENT
OXIDANT CRITERIA
FOR 1 hr, 24 hr AND ANNUAL
AVERAGE CONCENTRATIONS



3.2.5 Oxidants

Maximum ambient concentration criteria for oxidants have been established for three averaging periods by the Canadian Federal government, for two periods by Alberta and for only the 1 hour average period by Ontario and the USA. British Columbia has not specified objectives for ambient oxidant concentrations. The various criteria have been summarized in Figure 3.5.

- 1 hour average

Two concentration levels have been set as the objectives of the various jurisdiction. The lowest of the two is the Canadian Federal "maximum desirable" level and the Alberta standard. The USA Federal primary and secondary standard, the Canadian Federal "maximum acceptable" level and Ontario criteria are the highest.

- 24 hour average

Only Alberta and the Canadian Federal government have set 24 hour average criteria. The Alberta standard and the Canadian Federal "maximum desirable" level are equal and somewhat less than the Canadian Federal "maximum acceptable" level.

- Annual average

Only the Canadian Federal government has established annual average guidelines.

Table 3.2

MAXIMUM ALLOWABLE EMISSIONS RATE CRITERIA

	Ontario	Alberta	Canada Federal	B.C. Level A	B.C. Level B	B.C. Level C	Units	U.S.A. Federal	Washington	Idaho	Montana
Particulate	Max. 1/2 hr. ave. concentration at point of impingement 100 ug/m^3	229 mg/m^3 0.1 gr/scf .2 lb/1000 lb (50% E.A.)	See Below	229 0.1 5 0.45	343 0.15 7 .63	564 0.25 13 1.17	mg/m^3(a) gr/scf(a) lb/ton(a) g/10^6 cal(b)	90 0.04 2.0 0.18(a)	0.1	0.22	0.22
Sulphur Dixoide	Max. 1/2 hr. ave. concentration at point of impingement 830 ug/m^3 plus additional restrictions (see comment)	Mass emission rate controlled through permit system.	See Below	798 20 1.8	1596 30 2.7	2660 60 5.4	mg/m^3(a) lb/ton(a) g/10^5 cal(b)	975 24 2.2(a)	2660	20	20
Nitrogen Oxides as NO_2	Max. 1/2 hr. ave. concentration at point of impingement 500 ug/m^3	Mass emission rate controlled through permit system.	See Below	1146 27 2.4	1910 45 4.0	2292 54 4.8	mg/m^3(a) lb/ton(a) g/10^6 cal(b)	600 14 1.26(a)			
Comment	Calculated ground level concentration 525 ug/m^3 (rural), 390 ug/m^3 if within 30 miles of a town of 40,000 or 150 ug/m^3 if in an industrial area with other sources.		Guidelines are currently in final development stage and expected in 1977							For sources greater than 2.5 x 10^{13} cal/hr	Same as Idaho

(a) specified emission rate, others are calculated.
(b) basis 5550 calories/gram of coal.

3.3 Review of emission rate controls

Maximum allowable emission standards are tabulated in Table 3.2 for the same provinces, states and federal agencies as listed in Table 3.1. The contaminants for which emission standards exist include particulate, sulphur dioxide and oxides of nitrogen.

British Columbia pollution control objectives include emission concentration levels and mass emission rates per unit of coal burned. In order to compare the British Columbia objectives with those of other jurisdictions emission rates were converted to grams per million calories of heat input (based on 5550 cal/gm (10,000 BTU/lb) of coal burned).

The Canadian Federal emission guidelines for thermal power plants are still being developed. Environment Canada guidelines will probably specify the allowable emissions as a mass emission per unit of coal burned or per unit of heat input (4).

Alberta Environment set maximum emission concentration standards for particulates and regulates mass emission rates of particulate, sulphur dioxide and nitrogen oxides emissions based on an evaluation of the process in question. Based on this evaluation, a maximum mass emission rate is established for the process.

Ontario controls emissions based on point-of-impingement concentrations for the three pollutants noted. These are calculated based on the Holland plume rise formula and the Pasquill dispersion model. Thermal power plants must meet both the general criteria noted for each contaminant in Table 3.2 plus the more stringent criteria noted for sulphur dioxide under the comments section of Table 3.2.

USA Federal emission regulations are those promulgated in 1971, revised to October 1975. The criteria reviewed in Table 3.2 are for new installations.

3.3.1 Particulate matter

Particulate emission concentration objectives of British Columbia Level A are comparble to the Alberta emission concentration standards but both are higher than the USA Federal standards. Similarly the emission rate based on coal burned or heat input is higher for British Columbia Level A than for the USA Federal standard.

The Washington standard for the maximum emission concentration is the same as British Columbia Level A and the Alberta standard. Idaho and Montana have emission rate standards only slightly higher than the USA Federal standard and about half of the British Columbia Level A objective.

Ontario's criteria is based on a point of impingement concentration and cannot be directly compared to the other criteria.

3.3.2 Sulphur dioxide

The British Columbia Level A objectives are slightly more stringent than the USA Federal standard and Levels B and C are increasingly less stringent.

Alberta and Ontario criteria does not readily compare to those of other jurisdictions. Comparisons would have to be made using specific cases.

The Washington maximum emission concentrations for sulphur dioxide is the same as British Columbia Level C. Idaho and Montana control the emission rate at a level approximately equal to British Columbia Level A.

3.3.3 Nitrogen oxides

British Columbia Level A is less stringent than the USA Federal primary standards and of course Levels B and C are increasingly less stringent.

Again Alberta and Ontario control the emissions in such a way that direct general comparisons cannot be made.

Washington, Idaho and Montana do not have emission controls for nitrogen oxides.

3.4 Discussion

British Columbia has developed a three tiered system of objectives for ambient air quality control of sulphur dioxide, carbon monoxide and suspended particulate that spans the criteria of all other jurisdictions that were reviewed. Thus it is difficult to conclude that British Columbia has the most stringent, the least stringent or average objectives. Rather one concludes that British Columbia legislation provides flexibility in the control of contaminants and the potential for very strict control.

4.0 AMBIENT AIR QUALITY GUIDELINES BASED ON PUBLIC HEALTH CONCERNS

Historically, most regulatory bodies, in attempting to set guidelines to forestall adverse human health effects of air contaminants, have assumed that there exists a threshold concentration for each contaminant below which deleterious health effects are not observed. Such a threshold is desirable as a regulatory tool as it allows for the establishment of a unique standard with consequent ease of enforcement. However, the application of this threshold concept suffers from a number of serious drawbacks.

First, continuing research reduces the uniqueness of any previously established threshold in that a specific threshold is subject to substantial change as more sophisticated means are used to detect subtle differences in health status relative to any given health indicator. A further refinement in measurement techniques and methods of ascertaining individual exposure to a given contaminant are expected to impact significantly upon any established threshold.

Individual contaminant thresholds are predicated on the assumption that a unique effect attributable to that specific contaminant can be identified and the effect is truly independent of other contaminants. This is not possible using existing statistical analytical procedures. Therefore, most derived thresholds are plagued with a worrisome uncertainty that, to a greater or lesser extent, observations may be attributable to a factor or contaminant that was not measured or well quantified.

Historically, attempts to identify the threshold of effect to establish defensible guidelines for specific contaminants have been very frustrating. This does not necessarily mean that clearly defined effects are not visible and attributable to specific pollutants at high concentrations, but, rather, as one moves to the lower concentrations the ability to clearly establish the cause-effect relationship becomes extremely tenuous.

It is for these reasons that it is far more useful to address a range of contaminant concentrations which may be protective of public health rather than select a single value which is often derived by subjective weighting of data. In the following sections data relative to the adverse health effects of specific contaminants will be examined in the range context, as will the development of recommended guidelines.

4.1 Data sources

The development of public health guidelines has made use of two basic types of studies. Epidemiological studies, involving free living populations and conducted in a natural setting are the most reliable. The second type involves clinical and animal toxicology experiments where selected individuals or animals are studied under artificially controlled laboratory conditions.

4.1.1 Epidemiologic studies

The primary research vehicle for establishing a human adverse health effects response range with regard to any contaminant, especially for a 24-hour averaging interval, has been epidemiologic studies. These are essentially studies of free living populations which may be representative of a more general population subject to contaminant exposure. On a number of occasions the currently available epidemiologic studies relevant to these five contaminants do not yield unequivocal conclusions. The primary reason is that at lower concentrations the apparent health effects are identical for most of these contaminants and usually they appear in concert in the ambient atmosphere. In addition, the concentration of each component can vary simultaneously thus making it difficult to associate a health effect with contaminants in such a manner as to confidently suggest that the data supports a cause-effect relationship.

It is recognized that one rarely, if ever, has an adequate data base that can be stratified to reveal a significant cause-effect relationship for any single contaminant. It is also recognized that there exists the possibility of interaction between the various contaminants present or the subject contaminant(s) and naturally occurring factors such as temperature, humidity and salt nuclei. Indeed, the natural phenomena may itself be the primary cause of the observed health effect symptom. Before considering a specific contaminant exposure it must be demonstrated that reasonable effort has been made to isolate the impact of the contaminant and the factors which currently interact with it.

Epidemiology studies can be divided into two types. Studies of mortality excesses that occur in response to exposure to air contaminants, and morbidity studies which assess changes in disease patterns or exacerbations of existing disease in relation to changes in the concentration of ambient contaminants.

4.1.2 Clinical and animal toxicologic experiments

Cortrolled experiments on man, or on animals, have been attempted in orcer to overcome the aforementioned problems associated with epidemiological studies of free living populations. However, these studies are also deficient in many ways. In controlled human clinical experiments good medical practice dictates that healthy adults should be used rather than a more vulnerable cross section of the population as heart patients. Of necessity, therefore, the dosages which are administered are low and the administration is restricted to relatively short periods of time very much unlike the real world. Ultimately replication expenses usually preclude examination of the interaction of multiple contaminants or multiple stressors such as temperature or humidity.

Studies of these types are valuable but it is difficult to design them in a manner to reflect adequately ambient or real world exposure conditions. For these reasons it is concluded that the clinical experiments, in general, are not useful in establishing guidelines for many contaminants. However, in some cases they are the only data that are available with any degree of credibility and therefore they will be cited when appropriate.

These human clinical exposure studies are extremely inappropriate for the development of guidelines which involve long-term exposure because they are of short-term duration and the results cannot be directly extrapolated to 24-hour or annual exposure scenarios.

For animal toxicologic studies the situation is different. These studies, although costly, at least make it possible to examine a wide spectrum of contaminants over differing time durations and concentrations and in conjunction with other stressors. These studies suffer from the deficiency that, at present, adequate animal models do not exist that relate animal exposure directly to human exposure. As a result, animal toxicologic studies have been valuable for identifying harmful pollutants, but they have not been useful in developing guidelines to prevent adverse health effects.

4.2 Health based guidelines for contaminants

For each contaminant adverse health effect data have been reviewed with the intent of identifying concentration ranges for which an adverse health risk exists.

4.2.1 Sulphur dioxide guidelines

This section examines the technical data base for establishing health guidelines for sulphur dioxide with primary emphasis on human exposure information relative to both mortality and morbidity. These data are summarized in Table 4.1. Health data exists only to establish 24 hour and annual average guidelines.

TABLE 4.1

RECENT SCIENTIFIC EVIDENCE RELATING TO THE ADVERSE HEALTH EFFECTS OF SULFUR DIOXIDE

Adverse Health effect	Concentration $\mu g/m^3$	Averaging time	References
1. Increased mortality	300-500	24-hour	11, 12,23,26,29,31 32,33,34
2. Aggravation of symptoms in elderly	365	24-hour	15,18,23
3. Acute irritation symptoms	340	24-hour	15,18,20,21
4. Aggravation of asthma	180-250	24-hour	11,12,13,14,18,23
5. Increased hospital admissions with respiratory illness	300-500	24-hour	11,23
6. Increased frequency and severity of respiratory illness	130	annual mean	17,18,23,25,27,28,30
7. Increased specific mortality	180	annual mean	23
8. Decreased lung function in children	200	annual mean	11,18,22,23
9. Increased acute lower respiratory disease in families	90-100	annual mean	11,18,23
10. Increased prevalence of chronic bronchitis	95	annual mean	11,16,18,19,23,28 29,35

- Morbidity Data - 24 hour

Review of the available epidemiological data relative to the adverse health effects of sulphur dioxide for 24 hour exposures indicates a range of response between 120 and 500 ug/m^3 with the strongest evidence suggesting a range approaching 300-400 ug/m^3.

While more recent results from studies indicate that deleterious health effects occurred from sulphur dioxide at concentrations below 300 ug/m^3, it was the best judgement of the researchers conducting the studies that this effect was due to sulphates, which are alleged to arise from the interaction of sulphur dioxide and particulate matter in the atmosphere, and not sulphur dioxide alone (18).

The effect of sulphates will be discussed in Volume II of this report.

- Mortality Data - 24 hour

There exists epidemiological data which are alleged to relate excess mortality to increases in ambient sulphur dioxide concentrations. However, examination of these data reveals a consistent simultaneous presence of high smoke or particulate concentrations of 500 ug/m^3 or higher, during mortality episodes. This is compared to lower particulate concentrations, e.g. 300 ug/m^3 smoke, when describing morbidity. The observance of such differences has led researchers to ascribe these effects to the joint interaction of particulates and sulphur dioxide. This has led, in turn, to the current hypothesis that sulphates are the responsible agent in certain health effects (18).

It should be pointed out with regard to the use of mortality as an indicator of pollution health effects, that responsible groups have concluded it is an insensitive parameter for all but the most extreme deviations in pollutant levels (11). They further conclude that no single cause of death has been identified as being exclusively attributable

to the effects of air pollution, as opposed to any of the multitude of other stresses of urban life. Thus, we conclude that mortality data deriving from epidemiological studies can neither support nor preclude a 24 hour sulphur dioxide guideline in the range of 300 ug/m^3 to 400 ug/m^3 or below, because these data are confounded by pollutants other than sulphur dioxide.

The range of 300-400 ug/m^3 is supportable on the basis of available data.

- Morbidity Data - Annual

Health information gathered thus far indicates that no significant increase in morbidity results from long-term exposures to sulphur dioxide at concentrations below 90 ug/m^3 annual average. In fact, with respect to several key parameters, such as increased prevalance of chronic bronchitis in adults and increased acute lower respiratory disease in children, adverse effects are only observed at concentrations in the range of 90 ug/m^3 to 200 ug/m^3 annual average.

- Mortality Data - Annual

The data that attempts to link excess mortality to increased sulphur dioxide levels are, as stated previously, very equivocal. Most studies have focused on increased mortality during episodic conditions of severe inversion which are not relatable to long-term exposures. Further, all study results have been obscured by other subtle effects, effects which are not contaminant-specific enough to factor out sulphur dioxide or even natural phenomena.

The range of 90-100 ug/m^3 therefore is supportable on the basis of available data.

4.2.2 Suspended particulate matter guidelines

The aim of this section is to examine the technical basis of health guidelines for suspended particulate with primary emphasis on human exposure information relative to mortality and morbidity. These data are summarized in Table 4.2.

- Morbidity Data - 24 hour

Review of the available epidemiological data relative to the adverse health effects of suspended particulate matter indicates a range of response between 75 and 375 ug/m^3 with the strongest evidence suggesting a range of 150-300 ug/m^3. While more recent results from studies indicate that deleterious health effects occurred at particulate matter concentrations below this range, in the best judgement of the researchers conducting the studies these adverse health effects were due to sulphates rather than particulate matter per se (18).

- Mortality Data - 24 hour

There exist epidemiological data which have attempted to relate increases in mortality to increases in ambient particulate concentration. These data suggest an adverse effect range between 200 and 750 ug/m^3 and as with sulphur oxides the mortality range overlaps the morbidity threshold.

As was stated with regard to the question of sulphur oxides excess mortality is not a very sensitive parameter. Thus it is concluded that these data are of little help in establishing a 24 hour suspended particulate matter guideline.

The range of 150-300 ug/m^3 is supportable on the basis of available data. However, the question of whether specific particulate-borne contaminants are producing adverse health responses below these concentrations is an issue yet to be resolved.

TABLE 4.2

RECENT SCIENTIFIC EVIDENCE RELATING TO THE ADVERSE HEALTH EFFECTS OF TOTAL SUSPENDED PARTICULATE MATTER

	Adverse Health Effect	Concentration $\mu g/m^3$	Averaging Time	References
1.	Increased mortality	750 or a rise of 200	24-48 hours	24,34,35
2.	Increased infant mortality and cancer deaths	>200	3 days	23,33
3.	Increased upper respiratory infection and cardiac morbidity	375	24 hours	24,31,32
4.	Excess bronchitis mortality	200	24 hours	24,29
5.	Acute worsening of symptoms in bronchitis patients	300	daily	24,35
6.	Increased cough, chest discomfort and restricted activity	100-269	24 hours	11,18,21
7.	Aggravation of cardiorespiratory symptoms in healthy persons, and in elderly patients with heart and lung disease; increased asthma attacks in people with asthma	80-100	24 hours	11,15,18
8.	Aggravation of cardiorespiratory disease symptoms in elderly patients with heart or chronic lung disease	76-260	24 hours	11,15,18
9.	Increased mortality from chronic respiratory disease and all causes	100	2 years	24,26

TABLE 4.2 continued

RECENT SCIENTIFIC EVIDENCE RELATING TO THE ADVERSE HEALTH EFFECTS OF TOTAL SUSPENDED PARTICULATE MATTER

Adverse Health effect	Concentration $\mu g/m^3$	Averaging time	References
10. Increased chronic respiratory disease prevalence in adults; increased upper and lower respiratory tract disease and diminished pulmonary function in children	100-200	annual	24,27,28
11. Decreased pulmonary function in schoolchildren	110	annual	11,18,22
12. Increased frequency and severity of acute lower respiratory disease in schoolchildren	100	annual	11,18,20
13. Increased chronic respiratory disease symptom prevalence in adults	100	annual	18,19

- Morbidity Data - Annual

Information relative to adverse health effects of long-term exposures to particulate indicates increases in morbidity result from long-term exposure to particulate matter in the concentration range of 60-220 ug/m^3 annual average.

- Mortality Data - Annual

As with the 24 hour suspended particulate matter guideline, these mortality data allow no defensible guideline range to be established.

The range of 60-100 ug/m^3 annual average is supportable on the basis of available data, however it must be stated once again that a strong possibility exists that specific particulate-borne contaminants may be producing effects below this range.

4.2.3 Nitrogen oxides guidelines

The aim of this section is to examine the technical basis of health guidelines for nitrogen oxides (as NO_2) with primary emphasis on human exposure information relative to mortality and morbidity. These data are summarized in Table 4.3.

- Morbidity Data Short-Term Averages (1-2 hours)

There exists only very limited data on response of humans to short-term nitrogen oxide exposure and these data suggest an adverse response range of 2000-3000 ug/m^3.

- Mortality Data Short-Term Averages (1-2 hours)

There exist no short-term studies of human mortality in response to nitrogen oxide.

TABLE 4.3

RECENT SCIENTIFIC EVIDENCE RELATING TO THE ADVERSE HEALTH EFFECTS OF NITROGEN OXIDES (as NO_2)

Adverse Health Effect	Concentration $\mu g/m^3$	Averaging Time	References
1. Susceptibility to acute respiratory infection	2800	2 hours	12,48,49
2. Diminished lung function	3800	1 hour	12,43,50
3. Structural changes in lungs of experimental animals. Changes include changes in lung chemitries, damage to cilia.	564-940 940 750	6 months 1 month 7 days	12,39,44,45
4. Diminished lung function of humans experimentally exposed to NO_2	3000	10-15 min.	48,50
5. Increased prevalence of chronic respiratory disease in humans possibly attributable to NO_2	100-540	1 + year	12,38,40,41
6. Association of hypertensive heart disease mortality with NO_2 exposure	unknown	unknown	33
7. Increased susceptibility to acute respiratory infection in families	150-560	1 year	48,49
8. Increased frequency of lower respiratory disease in children	150-450	1 year	46
9. Increased mortality of animals exposed to NO_2 and challenged with infectious aerosols.	940	5 hrs/day x 3 months	12,53
10. Structural and biochemical changes in lungs of experimental animals. Changes include alteration in structure of lung collagen, rupture of mast cells, peroxidation of lung lipids, decreased cilia on bronchiolar epithelium	940 940	4 hrs/day x 5 days 6 hrs/day x 3 months 1 year	12,51,52,53 54,55,56,57

Therefore, based on the limited morbidity data a concentration range of 2000-3000 ug/m^3 would be a reasonable short-term guideline. However, because these data are so scanty it would be better to postpone establishment of a short-term nitrogen oxide guideline until more data are available. The same would be true of attempts to establish 24-hour guidelines as well.

- Morbidity Data - Annual

There exists only limited data relative to the response of humans to long-term nitrogen oxide exposures and these data suggest that effects are adverse in the range of 100-600 ug/m^3 annual average.

- Mortality Data - Annual

Only animal exposure data are available on excesses in mortality attributed to nitrogen oxide exposures. However, as previously discussed (Section 4.1.2), it is considered that these data have direct input into human health guidelines.

Therefore based on the available data a concentration range of 100-200 ug/m^3 would be a reasonable annual guideline.

TABLE 4.4

RECENT SCIENTIFIC EVIDENCE RELATIVE TO THE ADVERSE HEALTH EFFECTS OF CARBON MONOXIDE

Adverse Health Effect	Concentration mg/m^3	Averaging Time	References
1. Interference with mental activity	150-250 15- 50	1 hour 8 hours	65,66,68,70
2. Impaired physical performance in normal adults	180-360 30- 70	1 hour 8 hours	64,66,69
3. Increased risk of atherosclerosis	40- 50	8 hours	60
4. Impaired fetal development	40-100	8 hours	62
5. Altered tolerance to common drugs	60-160	8 hours	63
6. Impaired exercise tolerance in persons with heart disease	60-140	1 hour	58,59,61
7. Myocardial hypoxia in patients with cardiac and pulmonary disease	40- 50	8 hours	66,67
8. Decrease in visual threshold	27- 34	8 hours	66,71

4.2.4 Carbon monoxide guidelines

The aim of this section is to examine the technical basis of health guidelines for carbon monoxide with primary emphasis on human exposure information. In contrast to other contaminants thus far discussed, the data relative to the adverse effects of carbon monoxide are largely deduced from human clinical exposure experiments or epidemiology studies of short duration. These data are summarized in Table 4.4.

- Morbidity - 1 hour

Review of the available data relative to adverse effects of carbon monoxide on humans at one hour averaging times indicates adverse effects are observed at concentration ranges from 40-360 mg/m^3.

- Mortality - 1 hour

Insufficient data are available to use them as rational inputs into guidelines.

A carbon monoxide health guideline in the range of 40-60 mg/m^3, one hour average, is supportable based on the health data. However, because of its diverse physiological effects and acute tendency to alter cardiopulmonary functions the lower value of the range is preferred.

- Morbidity - 8 hour

In this case adverse response occurs over an 8 hour carbon monoxide concentration range of 15-50 mg/m^3. It is noted that the most important physiological changes occur at the lower end of the range.

- Mortality Data - 8 hour

Insufficient data are available.

A health guideline in the range of 15-20 mg/m^3 for the 8 hour average is supportable based on available data. As with the one hour exposure guideline the lower end of the range is preferred.

4.2.5 Oxidant guidelines

The aim of this section is to examine the technical basis of health guidelines for oxidants reported as ozone. In this case, only morbidity data summarized in Table 4.5 are available. Similarly it is noted that few, if any, studies have been conducted on humans during long-term exposure to oxidants, thus at this time, there exists insufficient data to establish health guidelines for the 24 hour or annual averaging time.

- Morbidity Data Short-term (1/2 to 2 hour)

Review of this relatively more complete data base indicates adverse response by humans to oxidants occurs at concentrations in the range of 100-1500 ug/m^3 over a 1/2 to 2 hour period. The most reliable data both from the standpoint of completeness and physiological significance indicate this range is too broach and a short-term oxidant guideline in the range of 150-300 ug/m^3 is supportable on the basis of these health data. As with carbon monoxide, oxidant effects very acute responses in particularly susceptible population subgroups and an attempt should be made to adhere to the lower values in the range.

TABLE 4.5

RECENT SCIENTIFIC EVIDENCE RELATED TO ADVERSE HEALTH EFFECTS OF PHOTOCHEMICAL OXIDANTS (as O_3)

Adverse Health Effect	Concentration $\mu g/m^3$	Averaging Time	References
1. Irritation of Respiratory Symptoms in Student Nurses -Threshold for Cough and Chest Discomfort	500-600	Peak daily hourly average	89
2. Irritation and Respiratory Tract in otherwise healthy adults			
(a) Substernal soreness, chest tightness and in several subjects shortness of breath and wheezing while performing intermittent exercise.	740 (ozone) and 1500 (ozone)	2 hours	72
(b) Burning and tightness of the chest when exercising.	1000 (ozone)	2 hours	78
(c) Substernal soreness and cough and in some instances pharynigitis and dypsnea while performing light exercise.	1500 (ozone)	2 hours	80
3. Increased eye irritation			
(a) Threshold for eye irritation in student nurses	260-340	Peak daily hourly average	79
4. Decreased cardiopulmonary reserve in healthy subjects			
(a) Impaired pulmonary function in healthy male subjects performing intermittent light exercise.	740 (ozone)	2 hours	77

TABLE 4.5 continued

Adverse Health Effect	Concentration ($\mu g/m^3$)	Averaging Time	References
(b) Synergism between ozone and SO_2- marked decrease in pulmonary function in healthy subjects performing intermittent light exercise.	740 (ozone) 1000 (SO_2)	2 hours	75
(c) Increased airway resistance in subjects performing intermittent light exercise.	100 (ozone)	2 hours changes 4-6 hours greater changes	78
(d) Decreased pulmonary function while performing intermittent light exercise.	1500 (ozone)	2 hours	80
Risk of Mutagenesis			
(a) Chromosomal aberrations in circulating lymphocytes of Chinese hamsters	400-600 (ozone)	5 hours	82,83
5. Increased susceptibility for acute respiratory disease.			
(a) Stability of alveolar macrophages reduced in rabbits - these are cells which protect the lung against infection	196 (ozone)	2-1/2 hours	81
6. Aggravation of Asthma	500	Peak hourly exposure	84
7. Aggravation of Chronic Lung Disease			
(a) Impaired pulmonary function among subjects with bronchitis or emphysema	100-450	Uncertain but likely a peak reading, perhaps hourly	90
(b) Improved pulmonary function among emphysematous patients when removed from air pollution on smoggy days.	390-1370 (390-1040) (ozone)	Uncertain	96

TABLE 4.5 continued

Adverse Health Effect	Concentration $\mu g/m^3$	Averaging Time	References
8. Irritation of respiratory tract in otherwise healthy adults.			
(a) Impaired pulmonary function	980 (ozone)	3 hrs/day 6 days/week for 12 weeks	93
(b) Impaired pulmonary function and respiratory tract irritation	1180-1570 (ozone)	2 hours	92
(c) Increased airway resistance. Respiratory irritation.	2000 (ozone)	1 hour	85
9. Aggravation of certain anemias			
(a) Increased sphering of red blood cells in mice, rats, rabbits and man (reflects accelerated aging of red blood cells)	400-500 (ozone)	1/2 - 1 hour	91
10. Impaired fetal development or survival			
(a) Increased neonatal mortality among offspring of exposed mice	200-400 (ozone)	7 hours/day for 3 weeks	91
11. Structural changes in cell nuclei of myocardial muscle fibers in experimental animals	400 (ozone)	5 hrs/day for 3 weeks	91
12. Decreased visual activity in man	400-1000 (ozone	3 hours single or twice repeated exposure	79
13. Increased eye irritation			
(a) among female telephone employees	200 and above	Uncertain	98,100
(b) residents of Los Angeles	100-880	Instantaneous peaks	99

TABLE 4.5 continued

Adverse Health Effect	Concentration µg/m³	Averaging Time	References
(c) Student nurses	200-880	Maximal daily levels (uncertain duration)	89
14. Increased susceptibility for acute respiratory disease			
(a) Susceptibility to bacterial streptococcal infection in mice	160 and above (ozone)	3 hours	86,87
(b) Susceptibility to Klebsiella pneumonia increased in mice and hamsters	1700 or 2600-8800 7600-8200 (ozone)	4 hours/day 5 days/week for 2 weeks, 3 hours 3 hours	86,87
(c) Impaired phagocytic ability of pulmonary alveolar macrophage in rabbits	1340-8000 (ozone)	3 hours	86,87
15. Decreased cardiopulmonary reserve in healthy standards			
(a) Impaired athletic performance of cross-country track runners in Los Angeles	60-590	1 hour	88
(b) Threshold for impaired athletic performance of cross-country runners	240	1 hour	73
(c) Impaired pulmonary function in adults	980 (ozone)	3 hours/day 6 days/week for 12 weeks	93
(d) Impaired pulmonary function	1180-1570 (ozone)	2 hours	92
(e) Increased airway resistance	2000 (ozone)	1 hour	85

4.3 Discussion

The public health guideline ranges set forth in the foregoing sections are based on an objective assessment of the adequacy of each health data base relative to each critical contaminant. These data are derived from a multitude of diffuse sources of differing quality and quantity and the guideline ranges are best judgement concentration ranges, which at a minimum, must be adhered to in order to protect public health.

5.0 COMPARISON OF AMBIENT AIR QUALITY CRITERIA TO RECOMMENDED PUBLIC HEALTH GUIDELINES

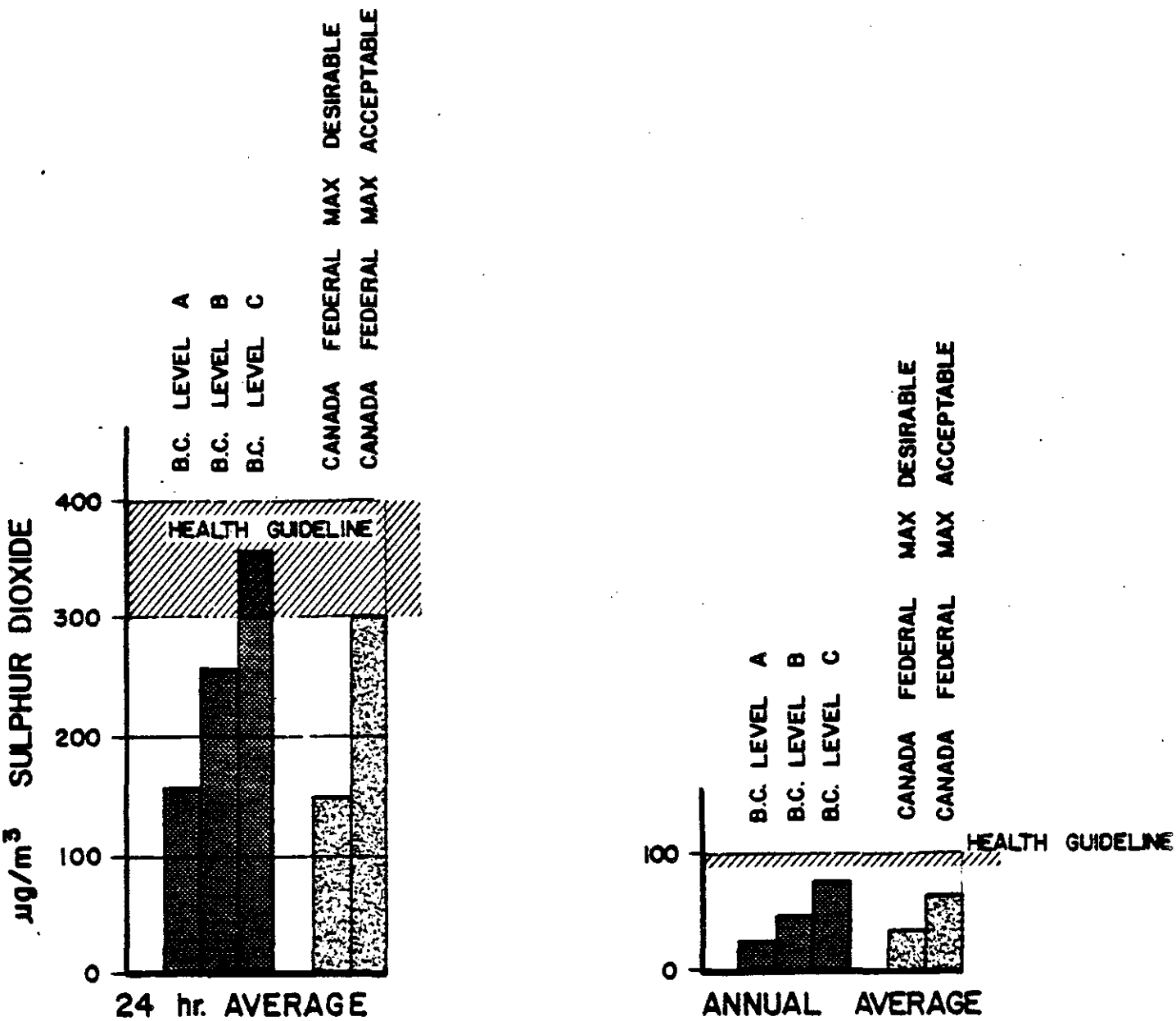
Based on a review of existing health data, the sections that follow set forth recommended guidelines for the Hat Creek Project and compare them to existing British Columbia and Canadian Federal criteria for ambient air quality. Implicit in these recommendations is the judgement that adherence to these concentrations will ensure adequate protection of public health.

It should be emphasized that these judgements are derived soley from a consideration of the health consequences of exposure to these contaminants, and the fact remains that in the establishment of ambient air quality objectives other factors may be considered. Consideration of these other factors is beyond the scope of this report, however, their importance cannot be overlooked.

5.1 Sulphur dioxide

The recommended ambient air quality achievement guidelines for the Hat Creek Project are 300-400 ug/m^3 for the 24 hour average and 90-100 ug/m^3 for the annual average. No 1 hour average guideline is recommended. The comparison of the recommended health guidelines to the British Columbia ambient air quality objectives and Canadian Federal guidelines is illustrated in Figure 5.1. Compliance with British Columbia Level C and the Canadian Federal "maximum acceptable" guidelines is sufficient based on public health concerns.

FIGURE 5.1
SULPHUR DIOXIDE AMBIENT AIR QUALITY CRITERIA AND RECOMMENDED HEALTH GUIDELINES



5.2 Suspended particulate matter

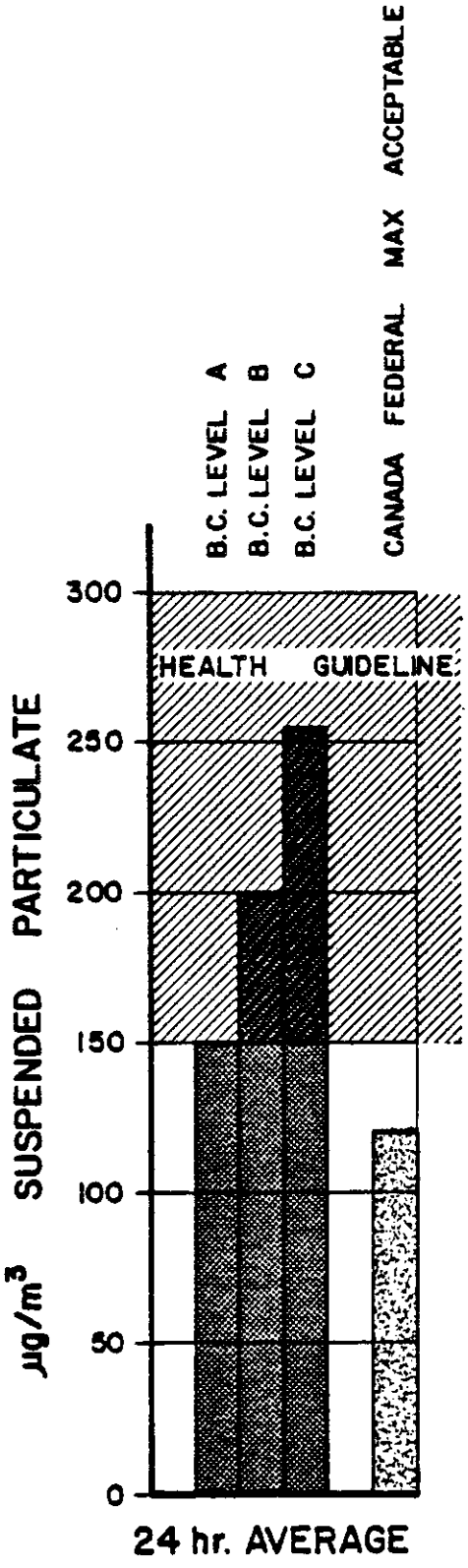
Based on health data a 24 hour guideline of 150-300 ug/m^3 and an annual guideline of 60-100 ug/m^3 are recommended to protect public health. A great deal of uncertainty exists regarding the potential toxicity of related particulate borne contaminants and an effort to achieve values in the lower end of the concentration range is desirable. The comparison of the health recommended guidelines to the British Columbia ambient air quality objectives and the Canadian Federal guidelines is illustrated in Figure 5.2. Compliance with British Columbia Level C is sufficient to protect public health.

5.3 Nitrogen oxides (as NO_2)

Currently, health data suggests a short-term (1-2 hour) range of 2000-3000 ug/m^3 is appropriate to protect public health. However, since these data are so limited, a guideline is not recommended.

The most appropriate nitrogen oxides guideline relates to long-term annual exposure. In this regard, achievement of nitrogen oxide concentrations of 100-200 ug/m^3 annual average is recommended to protect public health. Clearly, as for all contaminants, efforts should be directed toward the lower limit of the concentration range but the health benefits of achieving ambient concentrations below this range cannot be quantified. The comparison of the recommended health guideline to the Canadian Federal guidelines is illustrated in Figure 5.3. Compliance with the Canadian Federal "maximum acceptable" guideline is sufficient to protect public health.

FIGURE 5.2
SUSPENDED PARTICULATE MATTER AMBIENT AIR QUALITY CRITERIA AND RECOMMENDED HEALTH GUIDELINE



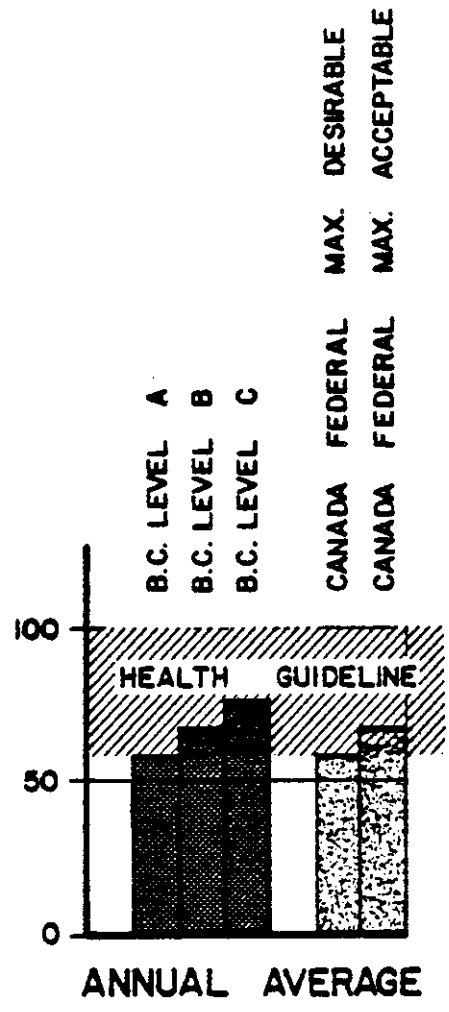
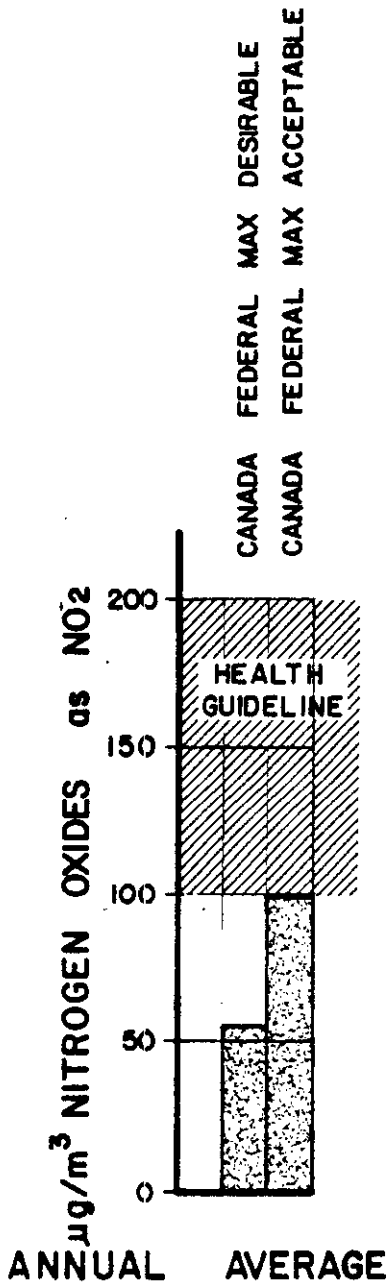


FIGURE 5.3
NITROGEN OXIDES
AMBIENT AIR QUALITY CRITERIA AND RECOMMENDED HEALTH GUIDELINE



5.4 Carbon monoxide

A 1 hour carbon monoxide guideline range of 40-60 mg/m^3 and 8 hour guideline of 15-20 mg/m^3 are recommended. This contaminant has many diffuse physiological effects in particularly susceptible population subgroups and all efforts should be made to achieve concentrations in the lower end of the range. The comparison of the recommended health guidelines to the British Columbia ambient air quality objectives and the Canadian Federal guidelines is illustrated in Figure 5.4. Compliance with British Columbia Level C or the Canadian Federal "maximum acceptable" guideline is more than sufficient to protect public health.

5.5 Oxidants (as O_3)

Based on the review of existing health data the recommended guideline for oxidants is the concentration range of 150-300 ug/m^3 for short-term (1/2-2 hour) exposure. Because of its reactive potential, every effort should be expended to maintain the lowest practicable concentration in this range.

Insufficient data exist to quantify the adverse effects of oxidant exposures for the 24 hour and annual time durations and it is premature to recommend guidelines for these two averaging periods. The comparison of the recommended health guideline to the Canadian Federal guidelines is illustrated in Figure 5.5. Compliance with the Canadian Federal "maximum acceptable" guideline is sufficient to protect public health.

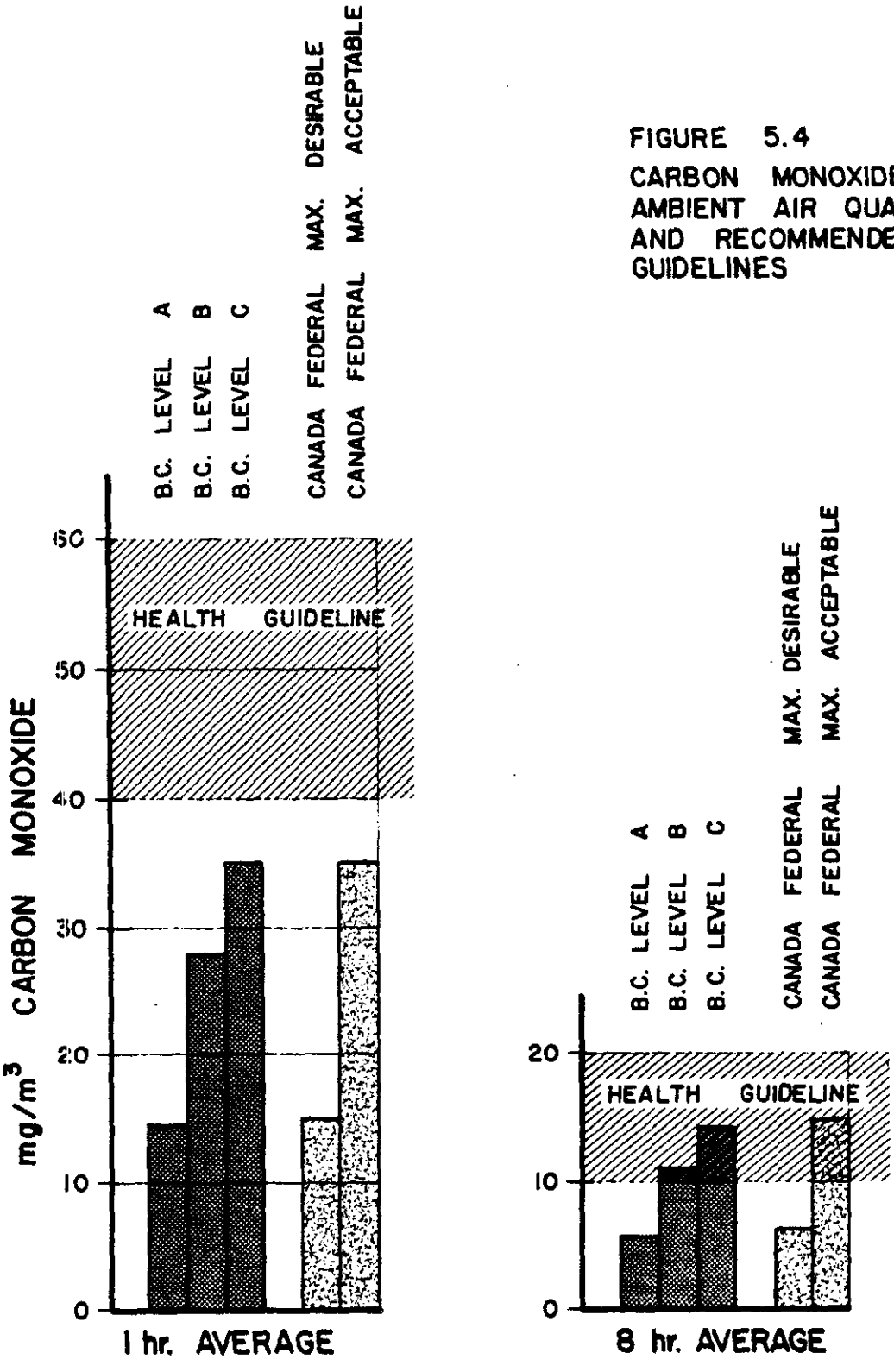


FIGURE 5.4
CARBON MONOXIDE
AMBIENT AIR QUALITY CRITERIA
AND RECOMMENDED HEALTH
GUIDELINES

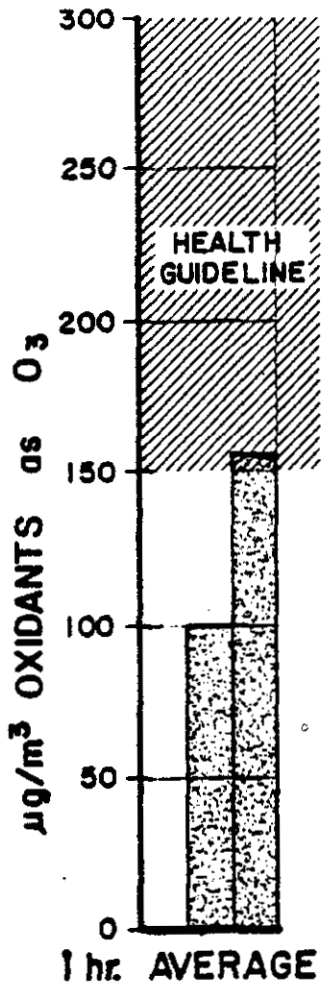


FIGURE 5.5
OXIDANTS
AMBIENT AIR QUALITY CRITERIA AND RECOMMENDED HEALTH GUIDELINE

5.6 Discussion

In order to establish unequivocal ambient air quality achievement guidelines based only on public health considerations requires that an adequate data base exist. In theory the dose response information should be complete with respect to exposure durations and exposure concentrations for each contaminant or combination of contaminants. Clearly this situation does not exist and the existing data base has many deficiencies.

The health data base was found to be lacking with respect to sulphur dioxide exposures for 1 hour duration, nitrogen oxides exposure for 1 hour and 24 hour durations and oxidant exposures for 24 hour and annual exposures. For these cases guidelines could not be recommended. Specifically the following deficiencies were noted:

- Sulphur dioxide

In the case of this contaminant, no studies have been conducted, at concentration ranges more common to the ambient air objectives for the 1 hour averaging time, which would enable a quantification of effects dose response. The limited short-term sulphur dioxide exposure data which exist derive primarily from occupational exposure studies and in these cases concentrations were an order of a magnitude greater than those set forth in existing 1 hour objectives. These data cannot appropriately be used to establish a defensible ambient air quality objective for 1 hour exposures to sulphur dioxide.

- Nitrogen oxides

At present insufficient data exist upon which to support the recommendation of 1 hour or 24 hour guidelines. This condition arises largely because community studies have had, as their primary focus, long-term effects. As with sulphur dioxide, the data derived from the occupational exposure studies are not directly extrapolatable inasmuch as they would invariably dictate guideline concentrations which would be unrealistically high.

- Oxidants

The situation relative to oxidants is the antithesis of what is observed with nitrogen oxides, namely the fact that the primary concern has been for short-term effects with little concern for long-term oxidant exposures. As such, community studies have been directed as assessing the adverse health effects induced by peak oxidant exposures during 1 to 4 hour intervals and these studies form the bulk of existing health knowledge relative to this contaminant. As a result, it is not possible at this time to establish guidelines for either 24 hour or annual averages. This concern with short-term exposures to the exclusion of other time periods derives from the fact that historically episodes of high ambient concentrations of oxidants have always occurred over short time periods.

Clearly all of the standards or objectives of the jurisdictions reviewed are more than adequate to protect public health. Of the contaminants studied, standards for suspended particulate are in close agreement with the recommended guideline range, indicating that the standards set for suspended particulate are based primarily on human health considerations. Standards for the other contaminants are somewhat more stringent than the recommended health based guidelines. This situation is most likely the result of other factors, such as contaminant effects on vegetation, being considered in establishing these standards.

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HAT CREEK ENVIRONMENTAL STUDIES

EPIDEMIOLOGY
VOLUME II
TRACE CONTAMINANTS

Prepared for:

British Columbia Hydro and Power Authority

Prepared by:

Western Research & Development
and
Flow Resources Corporation

April 1978

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1.0 INTRODUCTION

The purpose of this report is to assess the public health risk associated with the atmospheric emission of contaminants from the proposed Hat Creek Project. British Columbia Hydro & Power Authority engaged Western Research & Development and Flow Resources Corporation to conduct this assessment and to assist in establishing ambient air quality achievement guidelines as part of the Hat Creek Environmental Studies. This report is the second of the two volume dissertation on this topic.

In Volume 1 the following five commonly occurring contaminants were discussed; sulphur dioxide, suspended particulate matter, nitrogen oxides, carbon monoxide and oxidants. Ambient air quality regulations were reviewed and existing public health data were assessed. Recommendations for ambient air quality achievement guidelines were made to British Columbia Hydro & Power Authority regarding the Hat Creek Project.

In this second volume trace contaminant emissions are discussed and the potential impact of fourteen selected trace elements is examined. In addition to trace elements the report examines the question of suspended sulphates and nitrates, polycyclic organic matter and nitrosamines. The trace elements considered in detail are:

arsenic	manganese
beryllium	mercury
cadmium	nickel
chromium	selenium
copper	uranium
flourine	vanadium
lead	zinc

Existing regulations in various jurisdictions are reviewed and relevant health data for each contaminant are assessed. The projected emissions for the proposed Hat Creek thermal power plant, expressed in terms of maximum ground level concentrations are considered in order to determine the potential public health risk. Accepted health data are also factors used in this assessment.

2.0 SUMMARY

- In view of the present knowledge regarding the effects of trace elements on public health, no adverse health risk is foreseen from the proposed Hat Creek thermal power plant.

- For gaseous fluorine the predicted maximum 24-hour average ground level concentration is approximately one half of the most stringent ambient air quality objective.

- For the remaining trace elements, a comparison of the predicted maximum 24-hour average ground level concentration with selected North American ambient air quality objectives has shown that acceptable ambient levels are at least ten times higher than the levels predicted from the proposed Hat Creek power plant.

- Epidemiological studies relating morbidity to either suspended sulphate or nitrate exposure are considered insufficient for use in promulgating ambient air quality objectives. Therefore it is not possible at this time to assess conclusively the impact of sulphates and nitrates on human health. Three states in the USA have set ambient air quality standards for sulphates but these are considered by the study team to be overly protective.

- The potential impact due to polycyclic organic matter emissions is even more difficult to assess. Several members of this class of atmospheric contaminants, including benz-α-pyrene and 3-methylcholanthrene, have been implicated as mutagens and carcinogens. However, the necessary epidemiological studies have not been conducted and it remains to be shown that these compounds, as encountered in the atmospheric environment, contribute to the incidence of cancer in humans.

- There is no evidence to suggest that nitrosamines are produced by the combustion of fossil fuels. The formation of nitrosamines, via chemical reactions occurring in the atmosphere, requires sources of both nitrogen oxides and organic amines. No potential sources of amines were identified in the region of the Hat Creek Project and therefore nitrosamines are not expected to appear as either primary or secondary contaminants.

- Neither specific emission nor ambient air quality control for trace contaminants is recommended for the Hat Creek Project. This recommendation is based primarily on the analysis conducted with respect to trace elements. However, it also takes into consideration four other trace contaminants that are of concern, and are currently under study, in the USA and Canada.

3.0 ASSESSMENT METHODOLOGY

The health risk assessment of the trace contaminants identified in this report is based on reported relevant health data, published ambient air quality objectives and predicted maximum ground level concentrations for each contaminant.

3.1 Health data base

The data base examined in this study includes reported human and animal toxicological experiments, occupational health studies and community epidemiological studies. For the most part, human exposure studies have not been conducted at low contaminant concentrations. Thus, a heavy reliance is placed on animal toxicology studies and data collected during acute and subacute exposure episodes related to occupational scenarios.

The majority of the relevant health data regarding atmospheric trace contaminants, and in particular polycyclic organic matter and trace elements, are found in reports of occupational health studies. Data on the health of workers exposed to various contaminants usually does not contain precise contaminant concentration or length-of-exposure information. Also, the synergistic and antagonistic effects of multiple exposures are usually not taken into account in these studies. The majority of occupational health information describes subacute and acute exposures. In many cases, few qualified studies have been done which define the levels at which chronic exposure to air pollutants causes adverse health effects.

Knowledge of the effect of nitrosamines, nitrates and sulphates on health is derived primarily from animal toxicology experiments. It is widely accepted that due to various physiological differences, results from animal experiments cannot be directly applied to humans, but can only provide baseline information on the toxicity of a given compound. More useful data on the toxicology of trace contaminants is derived from controlled human toxicologic and epidemiologic studies. Unfortunately, few such studies have been conducted at this time of this publication.

While scientific information regarding the effect of air contaminants on health is not extensive for the majority of contaminants, research is continuing. The data base is also growing due to improved atmospheric monitoring techniques and more effective experimental strategies. In recent years, certain contaminants such as beryllium, cadmium, lead and mercury have been the subject of intensive scientific studies and more complete health data have been developed. The health data which are judged to be most reliable and relevant, are used to make the public health risk assessment.

3.2 Review of existing legislation pertaining to trace contaminants

Existing ambient air quality objectives for trace contaminants, as established by the provinces of British Columbia, Alberta, Ontario, the Canadian Federal Government and the United States Federal Government, were reviewed. In the discussion of sulphates, standards currently in force in California, Montana and Pennsylvania were also included.

The development of specific regulations for trace contaminants in the ambient air, rather than in the workplace atmosphere, has only occurred in the last few years. This is a direct result of inadequate monitoring of contaminant concentrations and the lack of specific epidemiological studies. Thus it is not surprising to find that the provinces of British Columbia, Alberta and the Canadian Federal Government do not have ambient air quality objectives for many of the trace contaminants under study. In Canada, only the Province of Ontario has ambient air quality regulations for trace elements. At this time no limits have been established for sulphates, nitrates, nitrosamines and polycyclic organic matter.

The Province of British Columbia has published pollution control objectives for trace contaminants relating to some industrial operations but not specifically to coal mining nor to thermal power plants. Pollution Control Objectives for the Food Processing, Agriculturally Orientated, and Other Miscellaneous Industries (1), and for the Mining, Mine-milling and Smelting Industries (2), have been published in 1975 and 1974 respectively. These are included in this study for comparative purposes.

The United States Federal Government has established relatively few standards for trace elements. Of the elements under investigation, ambient air quality standards have been set for only three; beryllium, mercury, and uranium.

Recently the U.S. EPA sponsored a study to make recommendations for presumed safe ambient air quality levels for selected trace elements. The study team included a panel of six occupational health experts and panel members independently reviewed health data dealing with the trace elements and they recommended safe ambient air quality levels. These recommended values were averaged to yield a final estimate of a safe 24 hour ambient air quality level. Their report (3) was published in 1973.

The ambient air quality objectives set by the provinces of British Columbia (1,2) and Ontario (4), the U.S. Federal Government (5,6) and the EPA Sponsored Panel are summarized in Table 3-1.

The current controversy over sulphates exemplifies the problems associated with assessing epidemiological data and subsequently establishing ambient air quality standards. A discussion of sulphate standards in California, Montana, and Pennsylvania is presented in section 4.16.

It is important to understand the reason for the lack of ambient air quality standards and the difficulty in establishing realistic criteria. It is significant that little work has been done to define the levels at which chronic exposure to air pollutants causes adverse health effects. Most dose-response data are from acute and subacute exposures in the work place and this data has been used to establish occupational standards. However, occupational standards are established for forty hours a week of exposure for healthy workers while ambient standards must deal with continuous exposure to all segments of the population.

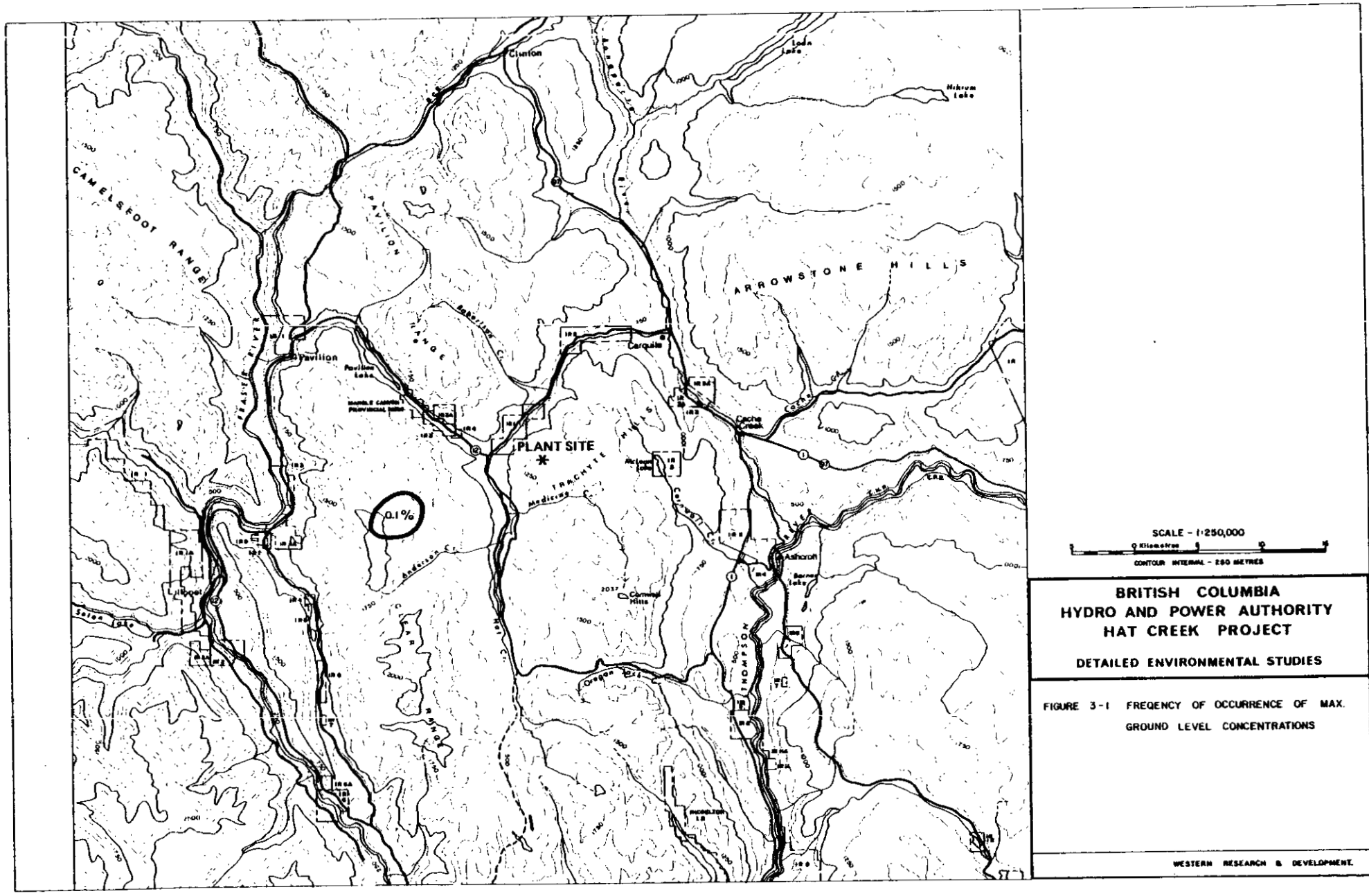
Statistical methods are available to extrapolate comparable ambient concentrations for continuous exposure from occupational standards (7), but many errors are inherent to such a procedure. Chronic exposure to low levels of some trace elements may exhibit cumulative effects, which are not allowed for in occupational standards based on intermittent exposures.

Table 3-1

AMBIENT AIR QUALITY STANDARDS FOR TRACE ELEMENTS

ELEMENT	Province of B.C. Pollution Control Objectives (1, 2) 24-hr Average (except as noted) $\mu g/m^3$ Level A	B	C	Province of Ontario Ambient Air Quality Criteria 24-hr Average (4) (except as noted) $\mu g/m^3$	United States Ambient Standards (5, 6) $\mu g/m^3$	U.S. EPA Sponsored Panel Safe 24-hr Ambient Air Quality Levels (3) $\mu g/m^3$
Arsenic	1.0	1.0	1.0	25		5.9
Beryllium					0.01 (30 day average)	0.005
Cadmium	0.1	0.1	0.3	2.0		1.2
Chromium						4.6
Copper						10.0
Fluoride	gaseous 7 day average			Expressed as HF		47
				Apr. 15 to Oct. 15 0.86 for 24 hrs (gaseous)		
	1.0	1.0	1.7	1.72 for 24 hrs (gaseous plus particulate)		
				Oct. 16 to Apr. 14 1.38 for 24 hrs (gaseous plus particulate		47
Lead	4.0	4.0	6.0	5.0		4.7
Manganese						11.4
Mercury	1.0	1.0	1.0	2.0	1.0 (24 hour average)	0.8
Nickel				2.0		3.7
Selenium						5.4
Uranium					0.4 (1 x 10^{-13} μcuries/ml)*	
Vanadium				2.0		6.8
Zinc	5.0	5.0	8.0			34

* Conversions based on the specific activity of naturally occurring uranium being 6.77 x 10^{-1} μcuries/gm (6).



BRITISH COLUMBIA HYDRO AND POWER AUTHORITY
HAT CREEK PROJECT

DETAILED ENVIRONMENTAL STUDIES

FIGURE 3-1 FREQENCY OF OCCURRENCE OF MAX. GROUND LEVEL CONCENTRATIONS

WESTERN RESEARCH & DEVELOPMENT.

Intermittent exposures allow for pulmonary clearance time on weekends and nights, whereas ambient levels involve constant exposure. An important difference is that occupational standards are based on the working population, which is known to be healthier than the general population. Such standards do not take into account especially susceptible individuals in the community.

As further consideration the contaminants which exhibit carcinogenic potential are being given special consideration when extrapolating standards from occupational to ambient conditions. Reliance on this method of creating standards demands questionable assumptions regarding the effects of the above variables.

3.3 Predicted emissions and ambient concentrations

Hat Creek coal reserves were originally cored and analyzed for trace contaminants for the Preliminary Environmental Impact Study of the Proposed Hat Creek Development. This study was conducted by B.C. Research in 1975 and it pointed to the existence of certain trace elements that warranted further study. Subsequent studies conducted at the Canadian Combustion Research Laboratory produced fly ash samples. These were analyzed for trace elements by Environmental Research & Technology Inc. Based on these analyses, Environmental Research & Technology Inc. (8, 9) predicted the emission rates for twenty-nine elements from the Hat Creek project. From this list of twenty-nine elements, fourteen trace contaminants were identified for study in this report. The other fifteen elements were not considered to be of concern due to either their low emission rates or the absence of known adverse health effects.

Suspended sulphates and nitrates, which are potential oxidation products of sulphur dioxide and nitrogen oxides respectively, are also included. The potential health effects of these have been the subject of considerable debate in recent years.

Two other trace contaminants that are more recently considered to be of potential concern are polycyclic organic matter and nitrosamines. Both are included in the health risk assessment.

Air quality modeling of stack effluents was performed by Environmental Research & Technology, Inc. Using a steady-state diffusion model which has been calibrated to the atmosphere of the Hat Creek Valley, potential effects on the local air quality of the Hat Creek Valley were assessed. (96)

Table 3-2 contains the project trace contaminant emission rates and projected maximum 24-hour ground level concentrations for a 2,000 MW capacity thermal power station. For the purpose of these calculations the effluent was assumed to be controlled with a 99.7 percent efficient particulate control device and then discharged to the atmosphere through a 365 m stack.

Calculations conducted by Environmental Research and Technology Inc. (9) (96) show that ambient concentrations equal to approximately 90 percent of the predicted 24-hour maximum concentrations would occur less than 0.1 percent of the time. This is less than one 24 hour period per year. In addition, these peaks would occur in a relatively remote region northwest of the proposed plant as shown in Figure 3-1. At all other locations concentrations would be lower than those shown in Table 3-2.

Table 3-2

PROJECTED EMISSION RATES AND GROUND LEVEL CONCENTRATIONS

Element	Symbol	Emission rate[1] Kg/Day Particulate	Gaseous	Maximum Predicted 24-hr Average concentration[2] $\mu g/m^3$
Arsenic	As	7.13	11.9	0.024
Beryllium	Be	0.55	0.11	0.00083
Cadmium	Cd	0.195	0.00	0.00024
Chromium	Cr	2.29	0.00	0.0029
Copper	Cu	0.094	0.00	0.00012
Fluorine	F	25.7	265.0	0.364
Lead	Pb	2.59	4.95	0.0095
Manganese	Mn	4.4	0.00	0.0055
Mercury	Hg	2.28	3.67	0.0075
Nickel	Ni	3.14	0.00	0.0039
Selenium	Se	0.0337	0.132	0.00021
Uranium (3)	U	0.91	0.00	0.0011
Vanadium	V	0.12	0.00	0.00015
Zinc	Zn	3.0	0.00	0.0038
Sulphate	$SO_4^=$	No emission		0.60 maximum 12 hour ambient concentration (summer)
Nitrate	NO_3^-	No emission		
Polycyclic Organic Matter	POM	No emission		
Nitrosamines	NNA	No emission		

(1) Calculated from test burn sample analysis and coal consumption of 42,630 metric tons per day. (96)

(2) From stack height modeling for 365 metre stack, uncontrolled emissions, Ref. 96

(3) Calculated based on Ref. 97.

4.0 TRACE CONTAMINANT ASSESSMENT

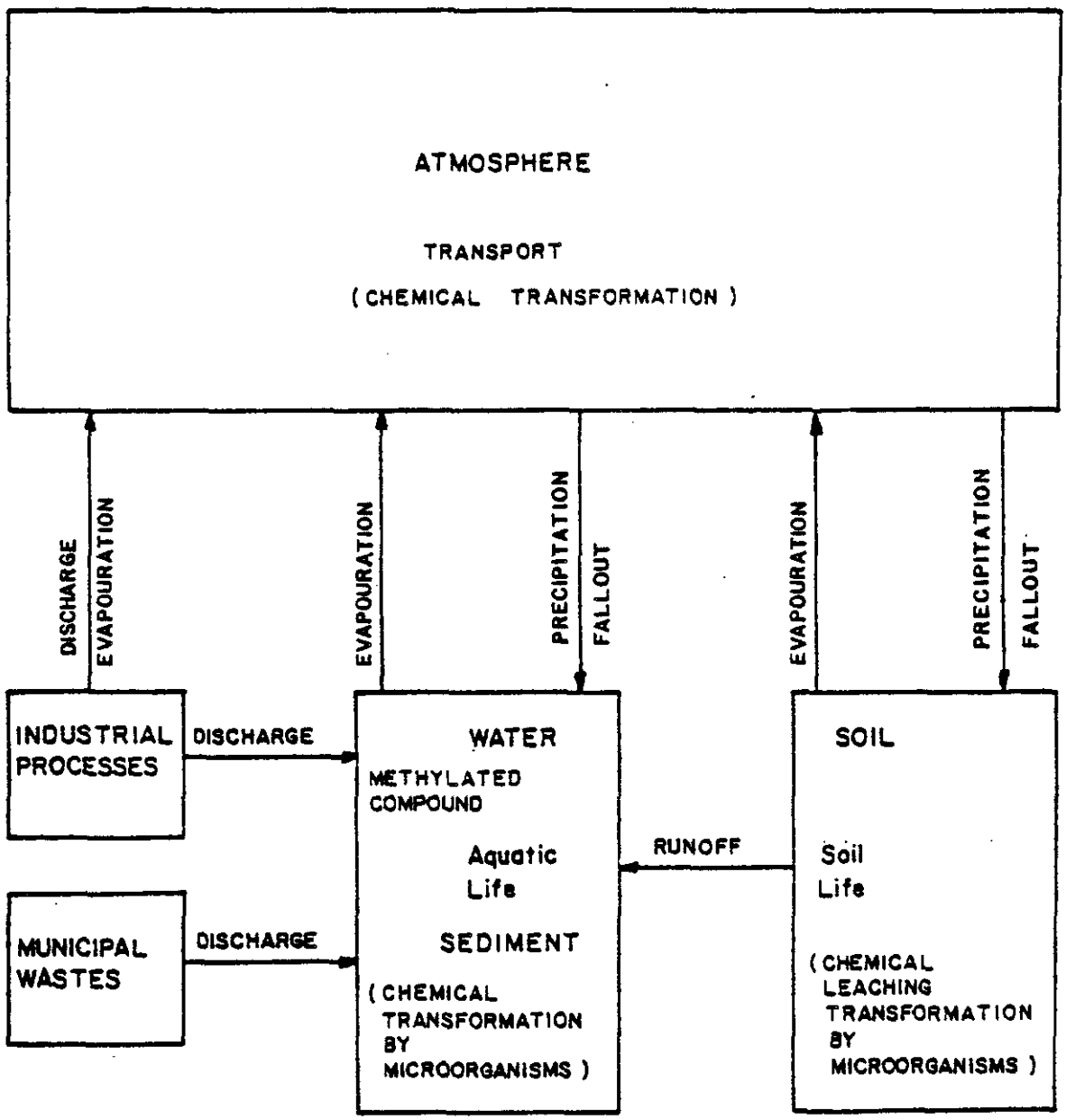
In the following assessment, the potential chemical and physical forms of each contaminant as produced during the combustion of coal are identified and the appropriate toxicity data are reviewed. The toxicity data are selected from a wide range of sources that are considered appropriate to the evaluation of exposure to low level ambient concentrations of each contaminant. The predicted maximum ground level concentration of each contaminant is compared to existing ambient standards or to estimated safe levels derived from the existing data base. From this data a health risk assessment is made.

4.1 Arsenic (As)

Arsenic occurs naturally in small concentrations throughout the environment. It is known to cycle in the environment, to become methylated in aquatic environments and it may enter the aquatic food chain as shown in Figure 4-1. Arsenic exists in trace amounts in coal and is one component of the fly ash produced during coal combustion. Smaller particles of fly ash tend to have higher concentrations of arsenic than do the coarser particles. This factor would indicate that the potential for arsenic to penetrate into the human respiratory tract is high and that it may penetrate comparatively deeper (10). Coal combustion also releases arsenic trioxide in a vapour state (11).

- Toxicity

Different arsenic compounds exhibit different toxicities. Arsenic trioxide is one of the more biologically potent arsenic compounds while elemental arsenic is relatively nontoxic when compared with salts of arsenic. However, the exact compounds of arsenic in the particulate have not been characterized (11).



Source: Ref 12

FIGURE 4-1 CYCLING OF SOME TRACE ELEMENTS IN THE ENVIRONMENT.

Arsenic is toxic to humans at acute and subacute concentrations. All forms of arsenic accumulate in the body in the liver, muscles, hair, nails and skin and are retained at these sites. Chronic exposure to ambient arsenic is characterized in severe cases by nasal septum ulceration, darkening of the skin, keratosis of the palms and soles, malaise and fatigue. Gastrointestinal effects and peripheral neuropathy characteristically occur. Children appear to have an increased sensitivity to arsenic toxicity, and accumulate relatively more arsenic in their bodies than adults (11).

Arsenic has been implicated as a carcinogen by many occupational and epidemiological studies. It is cited as causing cancer of the lungs, lymphatic system, scrotum, liver and skin. However, several human epidemiological studies have produced contrary evidence regarding some of these sites. Generally, after chronic exposure, arsenic is accepted as producing a delayed appearance of skin cancer in both workers and populations (11).

Animal toxicology experiments fail to substantiate the carcinogenic effects of arsenic. However, experiments show that arsenic causes chromosomal mutation in cell cultures in the laboratory (11). Arsenic has also been found to be teratogenic in animal toxicology experiments (13, 14, 15). Selenium has been suggested as protecting against this teratogenicity.

- Evaluation of health risk

To protect the public health, the Environmental Protection Act of 1971 of the Government of Ontario established as a standard a 24-hour average concentration of 25 $\mu g/m^3$ for arsenic (4). A panel of six public health experts have derived a safe 24-hour ambient standard of 5.9 $\mu g/m^3$(3). British Columbia has objectives for arsenic in the context of arsenic production that allow ambient levels up to 1.0 $\mu g/m^3$.

The maximum predicted 24-hour concentration of arsenic to be expected from the Hat Creek Project power plant is 0.024 $\mu g/m^3$ (96). No risk to the public health is foreseen relative to atmospheric arsenic emitted from the Hat Creek Project power plant.

4.2 Beryllium (Be)

Beryllium is not widely distributed in nature. However, it is one of the trace elements in coal. The chemical species of beryllium released through coal combustion are not known, although beryllium oxide and elemental beryllium are suspected. These two forms of beryllium are both insoluble and are comparatively less toxic than the more dangerous, soluble forms of beryllium (11). Beryllium which has been inhaled presents a much greater health risk than beryllium which has been ingested.

- Toxicity

Many animal toxicology studies of beryllium have been undertaken because beryllium has demonstrated marked adverse occupational health effects. However, the response of various animal species does not coincide with that of humans (16). Acute beryllium disease similar to that of man has been produced in animals. By contrast, chronic responses in animals similar to those of humans have been produced by only a few researchers. Beryllium compounds have caused cancer of the lungs and bones in animals although this is not typical of humans (17). Dermatitis has been produced in animals through beryllium dust exposure, and this symptom does occur in man.

Exposure of workers to air heavily concentrated with beryllium causes delayed acute pneumonitis (lung inflammation). Less serious acute exposures (100 to 400 $\mu g/m^3$) are characterized by the insidious onset of

non-productive cough, substernal pain, fatigue and weight loss, usually with complete recovery within one month of termination of exposure (18). However, sensitization to further beryllium exposure is known. Chronic beryllium exposure is characterized chiefly by a granulomatous lung disease. Latent periods of up to 20 years may occur before the onset of this pulmonary cancer. Skin lesions, either from acute or chronic exposures, are also highly characteristic of beryllium exposure. Other organs of the body may develop granulomatous lesions, including the heart (5). Cancer of the liver, bile duct and gall bladder have all been cited (11). The carcinogenicity of beryllium has been confirmed through animal experimentation.

The major effects of beryllium exposure may be due to a hypersensitivity (allergic) response in some individuals coupled with the naturally slow excretion of beryllium from the body. Even after discontinuation of exposure to beryllium, damage may progress due to a delayed immune response (15). This allergic phenomena explains the fact that only a small percentage of any exposed population will contract "berylliosis" or chronic beryllium disease.

Pulmonary cancer and other symptoms of chronic beryllium disease have been said to occur from over one year's occupational exposure to 25 $\mu g/m^3$ beryllium (19). It does not occur with ambient occupational concentrations of less than 2 $\mu g/m^3$ for an 8-hour day (17).

- Evaluation of health risk

Beryllium is one of three air pollutants considered in 1973 by the U.S. Environmental Protection Agency to be a major health hazard.

The U.S. EPA stringently controls those industries which it considers to have potentially harmful levels of beryllium in their atmospheric effluent (5). The U.S. EPA does not consider coal-fired power plants to be major sources of ambient beryllium (5). The United States National Emission Standard for Hazardous Air Pollutants limits ambient beryllium to 0.01 $\mu g/m^3$ averaged over 24 hours (5). A study by six public health experts recommended 0.005 $\mu g/m^3$ as a 24-hour safe ambient standard (3). The predicted 24-hour maximum ground level concentration, due to the Hat Creek Project, is 0.00083 $\mu g/m^3$ (96). No risk to the public health is foreseen from this potentially hazardous element.

4.3 Cadmium (Cd)

Cadmium occurs in some metal bearing ores throughout the environment. It is especially prevalent in zinc bearing ores. It enters the environment as airborne particulate matter from zinc smelters and metal reprocessing plants (20). Ambient cadmium is also emitted through coal combustion. It is deposited and retained in the surrounding soil in the same proportional amount as is found in the coal burned. This element does not appear to be methylated in the environment (11). Cadmium can enter waterways as tailings from zinc mining operations (21). Cadmium accumulates in plants that are directly exposed to cadmium dusts or cadmium contaminated irrigation water and thus enters the food chain. Once absorbed into animal bodies, it has a very long biological half-life, estimated at from 10 to 25 years (11). There is no known biological usefulness for cadmium.

- Toxicity

Cadmium occurs in the smallest particles in fly ash. Thus, it is readily respirable and can easily enter the lung, where it is absorbed and

transported throught the body. Cadmium-toxicity is manifested primarily as bone, kidney or pulmonary disease. Long-term ingestion can lead to abnormal bone development, osteoporosis and multiple fractures. Chronic occupational exposure induces pulmonary dysfunction and emphysema (15). Proteninuria, indicative of kidney damage, is one of the first evidences of excessive cadmium exposure. Acute high dose inhalation of cadmium can cause pulmonary edema, severe irreversible lung damage and death.

Carcinogenic and teratogenic potentials have been shown in animal experiments. Cadmium has also been shown to inhibit the immune response in animals (11). Experiments with animals, using unnaturally large dosages, have produced hypertension (22). However, the relationship of cadmium levels with hypertension in man remains unclear (23).

Other trace elements emitted through coal combustion such as selenium, zinc, iron, lead and manganes are known to affect the toxicity of cadmium either synergistically or antagonistically. However, these interactions are not well understood. They are usually studied in the laboratory using isolated situations. Therefore, extrapolation of these effects to the environment is difficult.

- Evaluation of health risk

The Government of Ontario has set a 24-hour ambient air quality standard for cadmium of 2.0 $\mu g/m^3$ (4). A panel of six public health experts has estimated a 24-hour safe ambient air quality for cadmium of 1.2 $\mu g/m^3$ (3). In the context of mining, mine-milling and smelting, British Columbia has established ambient air quality objectives of 0.1, 0.1 and 0.3 $\mu g/m^3$ for Levels A, B and C respectively.

The predicted 24-hour maximum ground level concentration of cadmium due to the Hat Creek Project is 0.00024 $\mu g/m^3$ (96). This level should cause no risk to human health.

4.4 Chromium (Cr)

Chromium occurs naturally in the environment in air, water, soil, plants and animals. Chromium in the ambient air comes from natural sources, such as forest fires, and from industrial sources, particularly the metallurgy industry and the burning of coal contributes to the concentration of chromium in the air, particularly in urban areas. In emissions from coal combustion, chromium is concentrated in the fine particle portion of the fly ash. Thus, chromium is readily respirable (10). The concentration of chromium in the air is higher in urban areas than in nonurban areas, ranging from 0.01 to 0.03 $\mu g/m^3$ in urban areas to an average value of less than 0.01 $\mu g/m^3$ in nonurban areas (24). Chromium is known to accumulate in the soil surrounding an emission source in concentrations proportional to the amounts emitted.

Although chromium exists in oxidation states of +2, +3 and +6, only the trivalent (Cr^{+3}) and hexavalent (Cr^{+6}) forms are of environmental significance. Most authors report no data on the valence state of chromium in emissions from any source. However, one study reports that most airborne chromium is in the hexavalent state. Unless the hexavalent form is first dissolved in rain or precipitation, it is slowly reduced to the trivalent form by the actions of sunlight and air (25).

- Toxicity

No harmful effects of chromium have been reported at ambient concentrations (24). The major route of entry of chromium to the body is through ingestion. However, adverse health effects of chromium have been reported from inhalation of chromium in occupational scenarios. Most of the information concerning the toxicity of inhaled chromium comes from such occupationa exposures (15).

The toxicity of chromium is a direct function of the valence state. Hexavalent chromium (Cr^{+6}) displays the most marked toxicity. The trivalent form (Cr^{+3}) is an essential element and plays an important role in lipid and glucose metabolism. Hexavalent chromium is a strong oxidizing agent and is able to penetrate biological membranes. It is irritating to the respiratory system and the skin. Hexavalent chromium may cause dermatitis, penetrating ulcers of the hands and forearms, perforation of the nasal septum and inflammation of the larynx and liver. Hemmorhages of the gastro-intestinal tract have been reported after ingestion (26).

Epidemiological studies suggest a high incidence of lung cancer in workers associated with the manufacture of chromium chemicals. Animal studies confirm this carcinogenic effect. Long-term exposure to low levels of chromium may also cause tumors. It has been observed that chromium concentrations in the lungs of the average man increases with age (24).

- Assessment of health risk

In Canada no agencies have suggested safe ambient levels for chromium. However a panel of six public health experts has suggested a safe ambient air quality standard for chromium of 4.6 $\mu g/m^3$, averaged over 24 hours (3). The predicted 24-hour maximum ground level concentration for chromium due to the Hat Creek Project is 0.0029 $\mu g/m^3$ (96). No risk to the public health is predicted at this level.

4.5 Copper (Cu)

Copper is widely distributed in nature. Due to its physical properties, copper is used extensively by industry. The major industrial sources of atmospheric copper are copper smelters and refineries, iron and steel production plants and coal combustion facilities (27). Coal usually contains relatively little copper, although coals from different areas

vary greatly in their content (28). However, through the process of coal combustion, copper is concentrated in the fly ash more than in the slag. This is attributed to copper's volatilization during coal combustion followed by condensation or adsorption on the surfaces of particles in the fly ash. It has been suggested that the possible solid copper compounds thus emitted are elemental copper and copper (II) oxide (CuO) (10).

- Toxicity

Copper is an essential element for man as well as many plants and animals. In man, copper is present in many oxidative enzymes, and is necessary to the formation of hemoglobin and erythrocytes. However, man appears to have a good homeostatic mechanism regulating copper levels in the body under normal circumstances. Copper does not accumulate in human tissues with age, although serum copper levels do increase. Therefore, some scientists postulate that increased serum copper levels may accelerate arteriosclerosis. This theory, however, has not been confirmed (15).

Acute intoxication with copper is usually evidenced through ingestion of copper salts. Inhalation toxicity, although relatively uncommon, is manifested mainly through occupational exposures to high concentrations of copper dusts and fumes. "Metal fume fever" is the major illness caused by these exposures, although some authors question whether copper is the causative agent of this fever. The major symptoms of metal fume fever are respiratory and dermatologic (29). In recent years, a new illness involving copper sulfate inhalation by vineyard sprayers has been noted. It is characterized by granulomatous, copper-containing lesions in the liver and lung. These lesions in the lung can progress despite cessation of exposure (30, 31). In agreement, coppersmiths display an increased incidence of lung cancer. However, this latter fact has not generally been accepted as proof of copper's carcinogenicity

(15). Varying results have been observed in animal toxicologic studies regarding tumor production by copper (32). Copper does not produce teratogenic effects in experimental animals.

- Assessment of health risk

Copper is not generally considered to be an extremely toxic element in the concentrations normally encountered. The predicted 24-hour maximum ground level concentration of copper due to the Hat Creek Project is 0.00012 $\mu g/m^3$ (96). A group of public health experts has concluded that an ambient concentration of 10 $\mu g/m^3$ averaged over twenty-four hours is not hazardous to health (3). No adverse health effects are predicted for the Hat Creek Project.

4.6 Fluorine (F)

The element fluorine is widely distributed throughout the earth's crust. It is usually chemically bound as fluoride. Coal combustion is generally considered as one of the major industrial sources of atmospheric fluorides. Fluoride is considered by some authors to be one hundred percent volatilized during coal combustion and possibly partially trapped by calcium in the fly ash particles (12). Another source suggests that only fifty percent of the fluoride in coal is emitted as gaseous hydrogen fluoride (HF), silicon tetrafluoride (SiF_4), and solid particles of inorganic compounds (33). Atmospheric fluoride is accumulated in vegetation exposed to large concentrations of fluoride and has caused extensive damage to crops and to domestic animals fed these crops (34). Fluoride, however, is not solely encountered in the atmosphere. It also exists in water, soil, plants and animals.

- Toxicity

Fluorine is not one of those elements essential for normal physiological functioning in the body, although it is one component of animal bones. Bone is the major accumulation site for fluorides. Fluoride is considered

to be beneficial to humans in some instances. It prevents dental caries, osteoporosis in women and aortic calcification in men when present in optimal amounts. Most human exposure to fluoride is through food and water. Inhalation is a less efficient and less common exposure route (35).

Acute exposure to hydrogen fluoride gas causes burns on the skin and intense and sometimes fatal lung irritation (36). Chronic exposure to extremely high concentrations of atmospheric fluoride is known from occupational scenarios to cause crippling fluorosis (debilitating bone disease). Osteosclerosis (absolute reduction in bone mass) is a much more common adverse health effect caused by occupational fluoride exposure. Accumulation of approximately 6000 ppm fluoride in bone is associated with osteosclerosis. It is generally thought that concentrations of 2.5 $\mu g/m^3$ fluoride in occupational exposures will not cause osteosclerosis in workers (37). Under normal conditions, inhaled fluoride constitutes a very small portion of the body's total fluoride intake. However, in a few instances, community health effects have been cited in people living near fluoride-emitting industry. Symptoms of hematological changes and general health effects were described. These symptoms are not unique to fluoride exposure, nor are they common in occupational fluoride exposure (33).

- Assessment of health risk

As with some other elements, atmospheric standard for fluorides are difficult to set. The amounts taken into the body by ingestion and inhalation are variable and difficult to estimate. British Columbia objectives are 1.0, 1.0 and 1.7 $\mu g/m^3$ for Levels A, B, and C respectively for mining, mine-milling and smelting industries (2). The Government of Ontario ambient air quality criteria for atmospheric fluorides are 1.72 $\mu g/m^3$ per 24 hours of total gaseous and particulate fluoride between April 15

and October 15 and 3.44 $\mu g/m^3$ between October 16 and April 14. Gaseous fluorides are controlled below 0.86 $\mu g/m^3$ for 24 hours between April 15 and October 15 (4). These regulations are promulgated to reduce ambient fluoride during the growing season since fluoride can demonstrate acute damage to vegetation.

The predicted 24-hour maximum ground level concentration for particulate fluoride due to the Hat Creek Project is 0.032 $\mu g/m^3$ (96). This concentration should not present a public health risk. The predicted 24-hour maximum ground-level concentration of gaseous fluorine is 0.332 $\mu g/m^3$. This is approximately half of the maximum allowed by Ontario. No adverse health impact is predicted due to gaseous fluoride.

4.7 Lead (Pb)

Lead occurs naturally as several insoluble minerals, extractable by mining and smelting. Because of its low melting point, lead has been worked by man from prehistoric times. Since the industrial revolution, and especially in the 20th century, the ambient amount of lead in the environment has increased dramatically (15, 38). Currently, the major sources of lead in the atmosphere are lead smelters and automobile exhaust emissions due to tetraethyl lead being used as a gasoline additive. Ambient lead is an important atmospheric pollutant since it can act to facilitate other atmospheric reactions.

Atmospheric lead emitted through coal combustion is most likely to exist as solid inorganic compounds in the fly ash particulate. These have been shown to occur in the smallest fly ash particles which are of respirable size and may penetrate into the lungs (10).

- Toxicity

Lead has long been known as a poison. Currently, sources of ingested lead are primarily old lead paint, moonshine whiskey, and leaded eating and cooking utensils. Atmospheric lead pollution can still be of significance near lead smelters (39, 40). Busy streets in urban areas can have a

significant airborne lead level from automobile exhaust. Areas of rural land near smelters can become uninhabitable for cattle (39). Lead tends to sediment out in water systems and thus its threat may be minimized. Inorganic lead compounds are probably not methylated in these sediments (12). Lead is not generally concentrated in the food chain although the human body accumulates lead. The half-life of lead in the human body is about two months.

Lead has long been associated with central and peripheral nervous system disease, renal disease, anemia and effects on hemoglobin formation (41). The effects of lead on the central nervous system of children are devastating. Because the toxicology of lead is medically well known, current research is tending toward elucidation of subtle factors of lead poisoning. The accepted "safe" values for the lead content of blood and urine are currently being challenged (42). The threshold values for chronic lead poisoning are being studied, especially considering the hypersensitivity of children to chronic low-level lead exposure (39, 40).

Some earlier animal studies show possible carcinogenic effects of lead (15). Human studies have not borne this out so far. Teratogenic effects are known in animals (43, 44, 45) and chromosome aberrations caused by lead have been detected through laboratory experiments. Lead burdens in experimental animals, similar to burdens experienced by man, have been shown to cause decreased resistance to infection and decreased life span (12).

- Assessment of health risk

For the mining, mine-milling and smelting industries, British Columbia has ambient air quality objectives of 4, 4 and 6 $\mu g/m^3$ for Levels A, B, and C respectively (1, 2). The Government of Ontario maintains a 24-hour ambient air quality standard for lead of 5.0 $\mu g/m^3$ and a geometric mean for 30 days of 2.0 $\mu g/m^3$ (4).

A panel of six public health experts suggests a 24-hour ambient standard for lead of 4.7 $\mu g/m^3$ to adequately protect the public health (3). The predicted 24-hour maximum ground level concentration of lead for the Hat Creek Project is 0.0095 $\mu g/m^3$ (96). This value is well below suggested standards and no risk to the public health is predicted.

4.8 Manganese (Mn)

The manganese emitted through coal combustion is generally not considered as being hazardous to health (46, 47). Manganese emitted through coal combustion is most probably in the form of solid inorganic oxide compounds in the fly ash particulate (48). Manganese can exist in eight different oxidation states; toxicity is relative to the type of manganese ion present and its oxidation state. Manganese compounds are known to act as catalysts in the atmosphere, possibly oxidizing other airborne pollutants (3).

- Toxicity

Of the trace elements, manganese is one of the least toxic to mammals (47). Because of its importance as an activator of different enzymes, manganese is one of the elements essential to man's diet in trace amounts. Man seems to have a good homeostatic mechanism to maintain the appropriate level of manganese in the human body. Pathology due to deficiency or excess is uncommon (35).

Inhalation of manganese dusts and fumes is not a normal exposure route but cases of manganic pneumonia and chronic manganese poisoning have resulted from these occupational exposures. Acute poisoning by manganese is actually rare (49). Manganic pneumonia is caused by exposure to high concentrations of dust and characterized by sudden onset of pneumonia (usually affecting only one lung). Antibiotics are usually ineffective in treating this condition. A much more common form of chronic manganese

poisoning involves the central nervous system. Exposure to manganese dust in high concentrations for a few months or more may cause symptoms which are possibly partially irreversible and usually not fatal. Symptoms include sleepiness, muscular twitching, leg cramps, increased tendon reflexes, spastic gait, emotional irregularity, and a fixed mask-like facial expression. There is some similarity to symptoms of Parkinsons' disease. Cirrhosis of the liver is also frequently observed in chronic manganese exposure. Some studies have shown manganese to have a delayed mutagenic effect in vitro (50).

Recent studies have found increasing incidences of pneumonia and respiratory problems in communities near industries emitting large amounts of manganese dust. Similarly, industrial exposure to manganese causes increased bronchitis, possibly due to an adverse effect of manganese on the immune system (12). However, the data resulting from epidemiological and occupational experiments is always suspect due to the deficiencies of the experimental methods. It is difficult to measure the extent of exposure after the disease has occurred. Generally, the exposure dose and length of exposure are not precise, thus producing unreliable dose-response data. An additional complication is the fact that exposure could include ingestion, inhalation and skin absorption.

- Assessment to health risk

It has been shown that manganese normally occurs in urban air in the United States at 0.10 $\mu g/m^3$ and is thought to cause no adverse health effects at this level. This value may be increased by 0.05 $\mu g/m^3$ with the use of a new fuel additive containing manganese to replace the present lead additive (51).

A panel of six public health experts have set a 24-hour standard for ambient manganese at 11.4 $\mu g/m^3$ (3). The predicted 24-hour maximum ground level concentration of manganese due to the Hat Creek Project is 0.0055 $\mu g/m^3$ (96). This value is far below any level at which adverse human health effects may result.

4.9 Mercury (Hg)

Mercury is somewhat unique among elements due to its characteristics of ubiquity, volatility and biotransformation. This latter quality accounts for the cycling of mercury between air, land, water, plants and animals. Mercury is one constituent of coal and is released through coal combustion mainly in its elemental form. Elemental mercury is very toxic when inhaled in the vapour state but is not toxic when ingested. Emissions of elemental mercury that deposit in the surrounding soil tend to disperse because of its volatility (12).

Elemental mercury in the soil cycles in the environment and is methylated in soil, stagnant water, sediments, or even in mammalian intestines, and enters the food chain (52, 53) as shown in Figure 1. Once altered, methylmercury is highly bioavailable, occurring in high concentrations in some fish. Methylmercury is the most toxic mercury compound and is most probably not directly emitted through combustion of coal. No data exist at this time on the rate of cycling or biotransformation. Therefore, it is difficult to assess the impact on the biosphere from atmospheric inorganic mercury effluents.

- Toxicity

Mercury poisoning was extensively documented before the field of occupational medicine came into being, due to human exposures to large concentrations of mercury in several occupational fields. Mercury vapor is efficiently absorbed by man; 75-85% being absorbed from 50 to 350 $\mu g/m^3$ and more at lower concentrations (12). The effects of inhalation of acute doses of elemental mercury are most commonly manifested in the respiratory tract by pneumonitis, bronchitis, chest pains, dyspnea (shortness of breath), and coughing. Acute poisoning is cited as being caused by mercury levels of 1.2 to 8.5 $\mu g/m^3$ (15). Chronic exposure to lower doses, characteristically named "mercurialism", produces the insidious onset of long-term symptoms caused by the accumulation and retention of mercury in the brain, testes and thyroid. These long-term

symptoms have been shown to be only partially reversible. Classic symptoms of "mercurialism" include gingivitis (gum inflammation), stomatitis (inflammation of the oral mucosae), erethism (abnormal physiological and psychologic disturbances) and tremor. General disturbance of the gastro-intestinal system is frequently seen. Renal toxicity is also a classical result of chronic mercury poisoning (54, 55). Humans are exposed to mercury in food, water and air. Thus, with multiple exposure routes and the easy accumulation of mercury in the body it is difficult to cite the actual amounts of mercury which cause chronic toxicity.

Both inorganic mercury and the organic mercury secondarily formed in the environment demonstrate teratogenic properties in experimental animals. Organic mercury crosses the placenta more easily, making it more dangerous to the fetus (54). Animal experiments also suggest that mercury causes decreased reproductive performance. Cell culture experiments have shown that both organic and inorganic mercury cause chromosomal mutation although the former of these has been refuted (12).

- Assessment of health risk

The United States Environmental Protection Agency has promulgated a National Emission Standard for Hazardous Air Pollutants for mercury at 1.0 $\mu g/m^3$ for 24 hours (5). The Government of Ontario maintains a 24-hour standard of 2.0 $\mu g/m^3$ (4). British Columbia has ambient air quality objectives for mercury of 1.0 $\mu g/m^3$ for Levels A, B and C (2). A panel of six public health experts has derived a safe ambient standard for mercury of 0.8 $\mu g/m^3$ for a 24-hour period (3). For the Hat Creek Project the predicted 24-hour maximum ground level concentration for particulate mercury is 0.0075 $\mu g/m^3$ (96). This level should pose no threat to public health. Similarly the gaseous portion of the mercury emissions should pose no threat to public health.

4.10 Nickel (Ni)

Nickel occurs in the urban air in appreciable amounts and ranges from less than 0.01 to 0.16 $\mu g/m^3$ in the atmosphere. The major sources of this contamination are exhaust from cars and trucks, fuel oil combustion for space heating, and coal and oil combustion for power generation (56). Large amounts of nickel do not naturally occur in water although large amounts do accumulate in many plants grown for food (15).

During the combustion of coal, hot carbon monoxide is passed over the finely divided nickel in the fly ash and gaseous nickel carbonyl is formed (56). However, nickel carbonyl is only stable in the presence of carbon monoxide. In the atmosphere, nickel carbonyl is readily transformed to nickel oxide in dry air and nickel carbonate in moist air (57). Nickel oxide and nickel carbonate are thus the most probable chemical forms of nickel present in the fly ash particulate. They are deposited in the surrounding soil proportionately to the amount of nickel in the coal burned. Nickel is concentrated in the smallest particle of fly ash. Thus, those particles containing nickel are more likely to be inhaled, and to reach the lungs (10). The concentration of nickel in the lungs increases with age (15).

- Toxicity

Nickel is potentially one of the trace elements essential to human health. However, occupational exposures to large amounts of nickel have proven that it can be both toxic and carcinogenic. The effects on health, caused by exposure to nickel, are dependent on the chemical form of nickel. Nickel carbonyl, the element directly emitted through coal combustion is by far the most toxic nickel compound. Acute poisoning by nickel carbonyl causes a delayed onset of fever, respiratory problems, leukocytosis and cyanosis and possibly resulting in death in four to eleven days (58). Chronic occupational exposures of nickel carbonyl are cited as causing cancer of the lungs, sinuses, and nose. This carcinogenic potential has been confirmed by animal experimentation (58, 59).

The toxicity of inorganic nickel compounds and elemental nickel has not been as extensively characterized as that of nickel carbonyl. Animal toxicological experiments have demonstrated that all compounds of nickel are potentially carcinogenic (58, 59). Respiratory changes have been noted in animals inhaling inorganic nickel compounds (57). Dermatitis has been ascribed to skin contact with nickel, in some sensitive humans (3).

- Assessment of health risk

Due to adverse health effects caused by exposure to nickel, the Government of Ontario maintains a 24-hour ambient standard of 2.0 $\mu g/m^3$ (4). The U.S. Environmental Protection Agency-sponsored panel of experts has suggested a 24-hour ambient standard for nickel of 3.7 $\mu g/m^3$ (3). The predicted 24-hour maximum ground level concentration of nickel due to the Hat Creek Project is 0.0039 $\mu g/m^3$ (96). No adverse health conditions are predicted

4.11 Selenium (Se)

Selenium is widely distributed in the environment in a variety of rare minerals and in coal. Coal combustion is the principal source of selenium contamination in the environment (60). The most likely chemical forms of selenium emitted through coal combustion are elemental selenium and selenium dioxide. Selenium dioxide can be emitted as a vapor although the majority occurs in particulate form. The selenium concentration in airborne fly ash particulate resulting from coal combustion has been shown to increase with decreasing particle size. Therefore, the selenium in the particulate is readily respirable and may penetrate deeply into the lungs (10). Airborne selenium is deposited in the soil, where it concentrates in certain plants and is consequently ingested by animals (61). It has also been suggested, on the basis of selenium's chemistry, that selenium is capable of being methylated in the environment as shown in Figure 1 (12).

- Toxicity

Selenium is a non-metallic trace element essential to human life and helps to maintain body tissue elasticity (61). Selenium can exhibit toxic effects when ingested, inhaled, or absorbed in unnaturally large amounts. Most reports on the adverse effects to human health due to inhalation of selenium describe industrial exposure to dusts, fumes, and vapours of selenium and its compounds.

Elemental selenium is relatively non-toxic although it has been implicated as causing irritation to the mucous membranes, catarrh, nosebleed, loss of sense of smell and dermatitis. Acute exposure is known to cause severe headache, difficult breathing, intense mucous membrane irritation and central nervouse system effects (62). Similar health problems occur with chronic ingestion of selenium. Selenium ingestion has also been implicated as a cause of tooth decay. Generally, health problems caused by selenium seem to concentrate on the epidermal (skin, hair, nail) and gastrointestinal systems. The length of exposure and concentrations causing any of the above pathology due to elemental selenium and selenium dioxide are not generally known (15).

In acute exposures to selenium dioxide, symptoms of severe dermatitis, burning of the eyes, lacrimation and conjunctival congestion, "garlic breath", dizziness, and lassitude occur. Chronic intoxication with selenium dioxide in occupational scenarios has caused gastrointestinal disorders, nervousness, liver and spleen damage, anemia, mucosal irritation and lumbar pain.

Selenium has been suggested as being teratogenic in one occupational study on humans (12). It has been implicated as both a carcinogen and an anticarcinogen in animal toxicological studies (63,64). Occupational studies of humans exposed to selenium have demostrated no cancer mortality (65). Epidemiological studies have shown a relationship between high selenium concentrations in the soil or air and lower incidence of cancer (66, 67). These studies, however, do not provide sufficiently convincing results (60). A recent review of carcinogenic data on selenium states, "It must be concluded, therefore, that inorganic selenium is unlikely to be carcinogenic as such and that all previous claims to the contrary are either undounded or controversial" (64).

- Assessment of health risk

The predicted 24-hour maximum ground level concentration of selenium due to the Hat Creek Project is 0.00021 $\mu g/m^3$ (96). A panel of public health experts doing a study for the U.S. Environmental Protection Agency has set a safe ambient 24-hour ambient standard for selenium at 5.4 $\mu g/m^3$ (3). No risk to public health is predicted. Similarly potential gaseous emissions of selenium present no risk to public health.

4.12 Uranium (U)

Uranium (U) is one constituent of coal released in the fly ash particulate through coal combustion. Uranium is a radioactive element occurring in nature in three isotopes: U^{238}, U^{235}, and U^{234}. Naturally occurring uranium contains 99.28% U^{238}, 0.71% U^{235}, and 0.005% U^{234}. Uranium 238, by far the most common of these isotopes, has a very long half-life of 4.49×10^9 years (68, 69). As with all radioactive elements, disintegration of uranium occurs continuously, producing many disintegration products. These disintegration products have unique toxicities and half-lives. Examination of each would be far too extensive for this paper.

- Toxicity

The most common isotope of uranium, U^{238}, emits alpha particles of a very low energy level. Although these alpha particles possibly cause dermal effects (70), they cannot penetrate human skin. The less common isotopes, U^{234} and U^{235}, constitute a radiation hazard if internally deposited through inhalation or ingestion (71). Insoluble salts of uranium are retained in the lungs for long periods of time. However, studies of animals and workers exposed to dusts of insoluble uranium salts for long periods of time have demonstrated no toxicity (15, 71). By contrast, some cases have proven that there is a long-term carcinogenic hazard (70).

Adverse health conditions related to uranium usually are a result of the highly toxic nature of uranium and its salts, rather than its radioactivity (70). Generally, uranium causes pathology in the kidneys, lungs, liver, cardiovascular, nervous and hematologic systems. Changes in protein and carbohydrate metabolism are also described. Chronic exposures have also been cited as inhibiting reproductive activity and affecting uterine development in experimental animals (70). However, toxicity is relative to the solubility of the uranium compound; more soluble compounds being more toxic. Soluble uranium compounds can cause acute illness and renal damage is the primary pathological effect. Prolonged exposure to insoluble uranium compounds results in chronic disease. These insoluble compounds are accumulated in tissues and organs and are retained there for long periods of time.

- Assessment of health risk

Uranium is a highly dangerous element relative to human health due to its toxicity and radioactivity. The United States Nuclear Regulatory Commission has promulgated a standard for ambient uranium released from licensed facilities in the United States. This standard of 1×10^{-13} µcurie/ml (or 1×10^{-7} µcurie/m^3) regulates radioactivity. For the Hat Creek Project the predicted 24-hour maximum ground level concentration of uranium emitted through coal combustion is 0.0011 µ/gm^3 (97). Based on a specific radioactivity of naturally occurring uranium of 6.77×10^{-1} µcurie/gm (6), this uranium emission is equal to about 7.5×10^{-16} µcurie/ml. This level is far below the U.S. standard and no adverse health risk is predicted.

4.13 Vanadium (V)

Vanadium is found in numerous ores throughout the world. It is emitted as a contaminant from industries involved in vanadium production. The burning of coal contributes a substantial amount of vanadium to the

environment. The burning of high sulphur residual oils constitutes the largest source of vanadium contamination (72). In the atmospheric environment, airborne vanadium may contribute to the formation of secondary contaminants.

- Toxicity

Vanadium occurs in low concentrations in the natural environment. However, in areas of the country where large amounts of residual oils are burned the concentrations of vanadium are high. The vanadium concentration is normally low in water. Vanadium appears to be concentrated in certain marine animals and in certain plants, especially legumes. Vanadium is poorly absorbed in mammals when ingested, but more easily absorbed when inhaled. Insoluble forms of vanadium at concentrations over 50 $\mu g/m^3$ are thought to accumulate in the human lung and cause pulmonary irritation (12). Otherwise, body turnover for vanadium is probably rather rapid. Vanadium appears to be a necessary trace element in some birds, but this is not yet proven for man (15).

Airborne vanadium emitted through coal combustion probably occurs in solid forms in the fly ash. The toxicity of vanadium oxides, which are the most likely compounds in fly ash, is proportional to their valence. The pentoxide is the most toxic. Although comparatively little research has been done on the toxicity of vanadium, some authors consider it to be a significant health hazard because of its submicron aerosol form.

At levels occurring in industrial accidents, vanadium acts primarily as a moderate to severe mucous membrane irritant, affecting the eyes and the entire respiratory tract. There do not appear to be significant sequelae from exposures. Occasionally an almost asthmatic-like condition results from exposure. Epidemiological studies of atmospheric vanadium in urban areas suggest a correlation of ambient vanadium with mortality from bronchitis and pneumonia, especially in males (12).

In animal experiments using high concentration exposures of vanadium, multi-system toxicity and death occur. Compared to industrial or ambient exposures these levels are extremely high. Animal experiments using more reasonable exposure levels demonstrate an adverse effect on pulmonary defense.

Some epidemiologic evidence exists linking vanadium to lung cancer and heart disease, but this is by no means proven (73). In fact, animal experimentation supports the fact that vanadium is not carcinogenic (12).

- Assessment of health risk

The toxicology of vanadium is not extremely well known and thus agreement has not been reached as to its importance as a contaminant. The Government of Ontario has established a 24-hour standard for ambient vanadium at 2.0 $\mu g/m^3$ (4). A panel of public health experts in the United States suggests a standard of 6.8 $\mu g/m^3$ over 24 hours (3). Relative to these standards, the Hat Creek Project represents no risk to the public health through vanadium emission since the predicted 24-hour maximum ground level concentration of vanadium is 0.00015 $\mu g/m^3$ (96).

4.14 Zinc (Zn)

Zinc is found in several ores. It is a component of iron ore and coal. Zinc as an atmospheric contaminant comes primarily from zinc refining as well as primary production of other metals (74). Zinc chloride, a well known health hazard, is not likely to be emitted through coal combustion. Zinc oxide is used in vulcanizing, photocopying, and paint production (75). It is through occupational uses of zinc that the majority of adverse health conditions occur.

Zinc is most probably emitted as solid compounds in fly ash particulate. Zinc is normally deposited in the soil surrounding coal combustion facilities in relation to the amount of zinc in the coal burned. Zinc is found in all waterways and is generally not considered to be methylated in sediments. Zinc is concentrated in food chains and is present in all living organisms. It is an essential element to plants and animals and is a normal enzyme co-factor in the human body (76). The average dietary zinc intake is 12.6 mg/day/person (15). The average adult body burden of zinc is 2.3 gm.

- Toxicity

Illness from zinc occurs primarily as zinc "fume fever". This illness occurs with occupational exposure to fresh zinc oxide fume, usually in concentrations greater than 15 mg/m^3. This tends to be a short-lived illness with complete recovery, even after multiple exposures. Zinc chloride ($ZnCl_2$) is more caustic, and more irritating. Symptoms of zinc chloride exposure can include dermatitis, and changes in mucous membranes. The usual symptoms of excess zinc ingestion are gastrointestinal in nature (15). Reported gastrointestinal effects of ZnO_2 in occupational inhalation, however, are unproven.

Large doses of both inhaled and ingested zinc caused death or illness in experimental animals. The concentration levels used, however, are much higher than workplace levels or any ingested amount normally available in the diet.

- Assessment of health risk

Relative to the other trace elements discussed in this document, zinc is less toxic. A panel of health experts suggests that ambient zinc should

be maintained below 34 $\mu g/m^3$ averaged over 24 hours to protect the public health (3). British Columbia (1, 2) has ambient air quality objectives for zinc of 5, 5 and 8 $\mu g/m^3$ for Levels A, B and C respectively. The maximum ground level concentration predicted value for the Hat Creek Project is 0.0038 $\mu g/m^3$ (96). No adverse health risk is predicted.

4.15 Polycyclic Organic Matter (POM)

Polycyclic organic matter (POM) is a large group of ring-structured aromatic hydrocarbons which may have substituted groups attached to one or more rings. POM occurs either as a vapor or, more commonly, as a condensed particulate. Particulate polycyclic organic matter (PPOM) can be subdivided into nine groups of aromatic compounds including pesticides, arene groups, and polycyclic (polynuclear) aromatic hydrocarbons (PAH) (77). Relative to the public health consideration of the proposed Hat Creek power plant, PAH may be an important group. The composition of airborne PAH includes, among other hydrocarbons, benz(a)anthracene and benzo-α-pyrene (BaP). Many of the constituents, though not all, have been suspected or classed as organic carcinogens (78).

Atmospheric POM is a combustion product of processes involving carbon and hydrogen. Consequently POM is formed both naturally in the environment and anthropogenically. The major technologic emission sources include the burning of fossil fuels (coal, oil and gas), refuse burning, motor vehicle exhaust and industrial processes (coke production, roofing operations and the aluminum and steel industry). The amount or composition of POM formed varies widely and is dependent on the emission source and its efficiency. Less efficient combustion sources emit higher POM concentrations (78, 79, 80).

The formation of POM and PAH takes place in a pyrolyzing rather than an oxidizing environment. The processes are highly complex and extremely variable. The pyrolysis mechanism involves radical chain propagation by

converting simpler organic structures into more complex molecules at temperatures between 500 and 900°C (80, 81). POM is thought to be emitted from the flame as a vapour. Upon cooling, the vapour usually condenses into small particle condensates or into already existing airborne particles. PPOM is generally concentrated in particles less than 5 μm in diameter (77, 80). Thus, atmospheric transport and distribution of particulate POM is dependent on such physical characteristics as particle size, surface area, shape and density. Airborne particles are removed from the atmosphere by gravitational forces or precipitation. The residence time in dry air is dependent on the particle size and varies from 0.4 to 40 days. Chemical reactions involving oxidants or sulphur dioxide may shorten the half-life of POM drastically. POM is highly reactive and oxidation reactions with other atmospheric contaminants are fairly simple. The daughter compounds formed are relatively more stable (77, 81).

Particulate POM in the ambient air is collected on High Volume Samplers as part of the total suspended particulate (TSP). PPOM is designated as the benzene-soluble fraction of the sample or is represented as the sample's concentration of BaP.* The three indices for POM measurement; TSP, the benzene-soluble fraction of TSP, and BaP, vary independently of each other in the atmosphere. They also vary geographically, spatially and temporally with the concentrations generally higher in urban areas during the winter months. In urban atmospheres, the TSP values range from 100 to 200 $\mu g/m^3$ with 8-14% of that value consisting of the benzene-soluble fraction. BaP composes approximately 0.01 to 0.02% of the benzene solubles (77). Thus typical BaP concentrations are in the 0.08 to 0.46 $\mu g/m^3$ range.

*Although the measurements of BaP are fairly precise, the reliability of BaP as an accurate index of the total concentration of POM in ambient atmosphere is questionable.

- Toxicity

Of the many adverse human health conditions associated with exposure to polycyclic aromatic hydrocarbons, the most significant is carcinogenic activity. For the last two centuries, occupational studies have provided the largest body of evidence on human cancer production. Workers in the fossil fuel-related industries have been shown to have an increased incidence of both skin and lung tumors (79, 80, 82, 83, 84). The cause-effect or dose-response relationships for these tumors have been difficult to ascertain. The compounding factors include the complex personal histories of many workers, the lengthy latency period between initial exposure and tumor appearance, and the variable high exposure levels encountered in the occupational setting. Recently, animal toxicological experiments have pin-pointed polycyclic organic matter as the carcinogenic agent contained in the derivatives of fossil fuels. However, the majority of these studies are limited to the topical application of POM resulting only in the induction of skin cancer. Toxicologic studies involving inhalation exposures have revealed little valuable information (80, 82).

At ambient atmospheric concentration levels, polycyclic aromatic hydrocarbons have not been proven to cause human tumors (78, 80). In epidemiologic studies, an association has been shown between air contamination and human mortality rates from lung cancer. Many such studies have implicated POM or BaP as contributing factors in increasing cancer mortalities in urban areas. However, dose-response relationships for specific causative factors for human lung cancer have not been identified or quantified (77, 81, 85).

Other diseases have been reported after occupational exposures to PPOM. These include chronic bronchitis, nonallergic dermatitis, allergic contact dermatitis, cutaneous photosensitization and pilosebaceous

reactions (80). No teratogenic or mutagenic effects have been attributed to POM exposure (77).

- Assessment of health risk

In the occupational scenario, a Threshold Limit Value (TLV) of 0.2 $\mu g/m^3$ has been assigned to the benzene soluble fraction of Coal Tar Pitch Volatiles (CTPV) by the U.S. Occupational Safety and Health Administration (86). Although CTPV is not synonymous with POM, benzo-α-pyrene and similar compounds have been isolated from CTPV and identified as carcinogens. In 1974, the Federal Register defined CTPV to include polycyclic hydrocarbons which volatilize from the distillation residues of coal, petroleum, wood and other organic matter (77).

An association between ambient levels of POM and adverse health effects has not been scientifically validated and no regulations for POM at ambient concentration levels have been promulgated. Therefore no health risk assessment is made for the Hat Creek Project with respect to POM.

4.16 Suspended Sulphates ($SO_4^=$)

Sulphates are a secondary contaminants generated by the chemical conversion of sulphur dioxide. This conversion probably occurs through several different chemical processes. A discussion on the atmospheric chemistry of suspended sulphates is beyond the scope of this report. However, it is important to point out that the subject of conversion of sulphur dioxide to sulphate is under intense investigation by federal agencies and academic institutions in the United States. Ultimately, the control strategy by which levels of atmospheric sulphates will be reduced is highly dependent upon a very precise knowledge of the atmospheric chemistry which leads to the production of these sulphates.

Atmospheric suspended sulphates are contained exclusively in the suspended particulate fraction of the atmosphere (i.e. the nongaseous fraction of air contaminants). The current method for monitoring atmospheric concentrations of suspended sulphates utilizes the high volume filter as a sampling device and a spectrophotometric analytical technique for measuring sulphates extracted from the high volume filter. The precision and accuracy with which atmospheric levels of suspended sulphate can be measured is highly dependent upon the sampling process as well as the chemical process by which water soluble sulphates are analyzed. The high volume filter, even though it has been used for twenty-five years for collecting total suspended particulate samples from the atmospheric environment, still leaves a great deal to be desired as a sampling device (87). The U.S. EPA is currently trying to develop dichotomous samplers for the purpose of replacing the existing high volume samplers used in epidemiological studies (87).

The dichotomous sampler will enable the scientist to study the respirable suspended particulate fraction rather than the total suspended particulate fraction. The dose of particulates received by an individual is not a function of the total suspended particulate matter. Instead it is a function of the respirable suspended particulate fraction. Thus, better experimental correlations of particulate exposure with public health should be observed when the respirable fraction is known.

A serious deficiency of the existing data base for suspended sulphates is the fact that sulphates have been measured in total suspended particulates and not in the respirable suspended particulate fraction which is actually inspired by humans. In addition, it is generally agreed that the method by which sulphates are extracted from the high volume filter can produce artifacts which lead to errors in estimating the atmospheric concentration of suspended sulphates.

- Toxicity

The biological activity of suspended sulphates has been the subject of investigation for about five years (88). A large number of animal studies has been conducted for the purpose of delineating the toxicology of numerous sulphate species (89). These studies have revealed that sulphuric acid (H_2SO_4) is the most toxic of all the sulphates being studied. The biological activity of the various inorganic sulphate species that has been tested in animal models presents an extensive and variable data base. It should be pointed out that the toxicity of the various suspended sulphates is a function of particle size, relative humidity and temperature. The subject of the biological activity of suspended sulphates is highly controversial and cannot at this time be definitively characterized. Studies are still being conducted into the nature of the interaction of suspended sulphates with elements of the mammalian respiratory system.

At present, the only epidemiological studies that have been conducted on the exacerbation of human health by atmospheric suspended sulphates have been conducted by the United States Environmental Protection Agency under their Community Health and Environmental Surveillance System program (CHESS) (88). The scientists who designed and executed the CHESS program have attempted to relate suspended sulphate concentrations in several different communities with the exacerbation of asthma and chronic bronchitis. The results of these studies have been exhaustively analyzed by several different scientific bodies as well as the EPA investigators themselves. It was the initial position of the CHESS investigators that exacerbation of human health could be detected at suspended sulphate concentrations ranging between 8 and 16 $\mu g/m^3$. The results of these studies were summarized in the report from CHESS (88). It was the publication of this report from CHESS that ignited controversy over the toxicology of suspended sulphates. This controversy is unresolved at this point.

- Assessment of health risk

The controversy surrounding suspended sulphates has resulted in the adoption of an ambient air quality standard (AAQS) for sulphates by only three states in the U.S.A. and the adoption of no objectives in Canada. Those AAQS adopted are summarized below.

State	Standard	Averaging Time
California	25 $\mu g/m^3$	24 hours
Montana	4 $\mu g/m^3$	Max. Annual Average
	12 $\mu g/m^3$	Not to be exceeded over 1% of the time
Pennsylvania	10 $\mu g/m^3$	30 day Average
	30 $\mu g/m^3$	24-hour Average

The wide disparity in the standards adopted by these states is indicative of the confusion which exists concerning the hazards to human health associated with suspended atmospheric sulphates. It is the opinion of the authors of this report, based upon an analysis of the existing data base, that these ambient air quality standards are overly stringent and that human health would be adequately protected at a level of 50-100 $\mu g/m^3$ averaged over 24 hours.

The current status of the suspended sulphates issue may be summarized as follows:

(1) United States Environmental Protection Agency has taken the position that there does not exist a sufficient data base for the derivation of a primary ambient air quality standard.

(2) A program should be initiated (which in fact the EPA has already embarked upon) for the purpose of delineating the bio-

logical activity of suspended sulphates. These studies are to include animal toxicology experiments, human clinical exposures and epidemiological studies.

(3) The State of California has decided that atmospheric sulphate concentrations need to be controlled and has promulgated a standard of 25 µg per cubic meter over 24 hours. In view of the deficiencies already revealed in the current sulphate data base it is difficult to see how the promulgation of this standard can be defended.

(4) The EPA has concluded that a data base sufficient to permit the derivation of ambient air quality standards would not be available for five to eight years. At least two of these years would have to be devoted to the development of improved monitoring methodology for the epidemiological studies.

With these comments in mind and giving due consideration to the existing standards the predicted maximum 12 hour average ambient concentration of 0.6 µg/m^3 for the Hat Creek Project should present no risk to public health.

4.17 Suspended Nitrates (NO_3^-)

Suspended nitrates are secondary atmospheric contaminants being generated through chemical reactions that occur in the atmosphere. Even though a precise understanding of the processes through which atmospheric suspended nitrates are generated is not currently available, it is obvious that nitric oxide (NO) is the primary precursor to this class of atmospheric contaminant. Nitric oxide is a by-product of the combustion of fossil fuels and is a major contaminant species found in automobile exhaust.

A photochemical process involving hydrocarbons and nitric oxide results in the production of ozone (O_3) and nitrogen dioxide (NO_2) (90). It has been speculated that nitrogen dioxide interacts with hydrocarbons and ozone to produce a hazardous organic nitrate known as peroxyacyl nitrate (PAN). This highly reactive molecule is unstable and probably decomposes in such a manner as to produce inorganic nitrate.

The inorganic nitrate species are found in the respirable fraction of the suspended particulate fraction of atmospheric contaminants.

To this date a definitive chemical analysis of respirable suspended particulates has not been undertaken. Thus it is not known what dose of nitrate would be delivered to a human population through the respirable suspended particulate fraction. However, an attempt has been made to calculate the NO_3^- dose inhaled by an adult living in an urban setting in which the suspended nitrate concentration is 3-5 $\mu g/m^3$. It was estimated that the daily dose of NO_3^- would be approximately 10 μg. This is less than 1% of the total body burden of NO_3^- received by ingestion.

The current method of monitoring suspended nitrate is by the use of high volume filtering devices (91). The high volume filter collects the total suspended particulate on glass fiber filters from a specified volume of air. A segment of this filter is removed and after being carefully weighed it is analyzed for nitrate. The precision with which atmospheric concentrations of suspended nitrates is measured is dependent both upon sampling precision and precision of the chemical analysis. Even though the high volume filter has been used for sampling atmospheric particulates for approximately twenty-five years it still possesses many deficiencies which to a certain extent compromise the existing data base with regard to suspended nitrates (87). It has also been demonstrated that certain metal ions can produce a positive error in the chemical analysis of nitrates. This presents another source of error in the existing data base for atmospheric nitrates (91).

- Toxicity

The biological effects of nitrate salts have been studied in animals primarily with regard to the oral ingestion of these salts (92). It has been demonstrated that the ingestion of large quantities of nitrates can generate relatively high levels of methemoglobin in circulating blood. Methemoglobin is an inactive form of hemoglobin and consequently is unavailable for oxygen transport. In addition, it has been demonstrated that high concentrations of nitrate salts in the diet can produce kidney damage. Unfortunately, no studies have been conducted into the toxicology of inhaled nitrate salts. This is probably due to the complexity of producing a nitrate salt aerosol possessing a mass median diameter of 3.0 microns or less, which is the size of respirable particles.

The United States Environmental Protection Agency, through its Community Health and Environmental Surveillance System (CHESS) studies, has attempted to relate adverse human health response to suspended atmospheric nitrate levels in several large cities (93). These studies have produced evidence which suggests that suspended nitrate levels of 2-7 $\mu g/m^3$ will produce an elevated asthma attack rate. The investigators however caution that insufficient knowledge concerning the chemical and physical nature of the suspended particulates limits the interpretation of their observation.

- Assessment of health risk

The current data base with respect to human health response to the inhalation of suspended inorganic nitrate is inadequate. The few epidemiological studies that have attempted to delineate the biological activity of inhaled inorganic nitrates, have suffered from the use of inadequate atmospheric monitoring methodology and from the lack of suitable parameters for estimating the impact of inhaled nitrates on human health. The U.S. EPA is currently moving vigorously to correct these deficiencies. Studies have been initiated for the purpose of defining the inorganic

nitrate species contained in the respirable suspended particulate fraction in urban atmospheres. At this time there does not exist a sufficient data base which would permit the derivation of ambient air quality standards. Indeed it is entirely likely that such a data base will not come into existence for another five to ten years.

However, the current data base does not suggest that suspended inorganic nitrates present a significant health hazard to humans.

4.18 Nitrosamines (NNA)

During the late 1960s it was revealed that nitrosamines are produced by the combustion of tobacco and occur in significant levels in cigarette smoke. Only within the past five years have nitrosamines been observed in ambient atmospheres around certain urban centers (94). Studies conducted in 1973 and 1974 utilizing highly sensitive gas chromotographic techniques coupled with electron capture detectors revealed that nitrosamines exist in the ambient atmosphere in proximity to certain chemical plants in the northeastern United States. Studies conducted in the Baltimore and Philadelphia areas have measured nitrosamines concentrations in the parts per billion range in the atmospheric environment in certain areas of those cities. Most disturbing is the fact that it has not yet been determined whether the nitrosamines that are being observed are primary emissions from chemical plants or whether they are being formed by chemical reactions occurring in the atmosphere. This latter fact is extremely important to the development of a control strategy. It has not yet been demonstrated that nitrosamines are primary emissions from the combustion of fossil fuels. However, should it be demonstrated that nitrosamines can be produced by the interaction of nitrogen oxide (NO_x) with other atmospheric contaminants it would mean that all NO_x emissions would have to be more closely controlled.

The emergence of this high health risk molecule as a potential atmospheric contaminant will probably result in the establishment of routine monitoring programs at other urban centers in the United States. Even though nitrosamines have been observed in concentrations no higher than 10 parts per billion at any location, the nature of their biological activity (carcinogenicity) requires that they be carefully monitored. At present the only means of monitoring nitrosamines is through the use of gas chromatographs equipped with electron capture detectors. No other technique is capable of achieving the sensitivity and specificity required to monitor this class of compounds.

- Toxicity

The biological activity of the nitrosamines has been studied extensively in experimental animals (95). So far, every nitrosamine studied has been proven to be carcinogenic in at least one animal species and every animal species studied has proven to be sensitive to at least one nitrosamine. These facts alone are sufficient in establishing the nitrosamine family as being one of the most hazardous in our environment.

As to be expected, there have been no human clinical studies conducted on the biological activity of the nitrosamines. Consequently, we can only extrapolate from existing animal studies to the human population. While this form of extrapolation is undesirable for most toxic substances it is absolutely essential in the case of carcinogens.

At the present time the United States Environmental Protection Agency, the National Cancer Institute and the National Institute of Environmental Health Sciences are considering epidemiological type studies which would include the atmospheric monitoring of nitrosamine concentrations for the purpose of determining to what extent nitrosamines may be responsible for generating cancer in urban populations.

- Assessment of health risk

While it is certain that the nitrosamines are some of the most dangerous chemicals that could be injected into our environment it is equally clear that the existing levels of nitrosamines in the atmospheric environment are extremely low. In most cases they are probably too low for detection and measurement. The current data base regarding the effects of atmospheric nitrosamines on human health is non-existent. The major question to be considered is whether or not the various nitrogen containing species emitted by the combustion of fossil fuels can lead to the production of nitrosamines as secondary pollutants. Until careful studies have been conducted for the purpose of answering this question and relating this potential hazard to exacerbation of human health, no effective ambient air quality standards can be promulgated.

In summary, coal-fired power plants are not recognized as primary sources of nitrosamines and in addition no source of amines exists in the region of the proposed Hat Creek power plant. Therefore, nitrosamine formation in the atmosphere due to potential mixing and reaction between the emission plumes is not expected. No public health risk is anticipated.

5.0 DISCUSSION

Selected atmospheric contaminants, which would be emitted through coal combustion at the Hat Creek Project and are potentially hazardous to human health, have been discussed in this report. The salient points of the toxicology of each substance have been reviewed relative to the most recent data.

The Province of British Columbia and the Canadian Federal Government have not established specific ambient air quality objectives for coal-fired thermal generation plants. However, Pollution Control Objectives for the Mining, Mine-milling and Smelting Industries and for Food Processing, Agriculturally Oriented and Other Miscellaneous Industries of British Columbia, have been included for comparative purposes. In addition, criteria established by the Province of Ontario, and air quality standards or recommended safe levels developed by the USA have been used in making the public health risk assessments.

Throughout the risk assessment, predicted maximum 24-hour concentrations have been compared to 24-hour objectives or regulations. The 24-hour maximums are predicted to occur less than once per year and over a very small area. For all other times, the contaminant concentrations will be less than the maximums. The annual average, taking into consideration all meteorological conditions, would be considerably lower than the 24-hour values.

Estimated ground level concentrations of each trace element were compared with selected ambient air quality objectives. It is concluded that relative to these ambient objectives and the projected maximum 24-hour average ground level concentrations, the fourteen trace elements reviewed present no risk to human health. Only for gaseous fluorine do predicted levels approach levels approximately one half the lowest governmental objective.

Of the other atmospheric pollutants discussed; polycyclic organic matter, suspended nitrates, nitrosamines and suspended sulphates, either their behaviour in the atmosphere is not fully understood or the monitoring techniques employed are inadequate. Additionally, the data base on the health effects of these atmospheric contaminants is sparse. As a result, the adverse health risk of polycyclic organic matter, suspended nitrates and suspended sulphates cannot be established with reasonable certainty. Since nitrosamine formation is not expected, this contaminant does not appear to present a public health concern.

As demonstrated in this report, the predicted levels of trace contaminant emissions at the Hat Creek Project power plant are acceptable within the limits of the objectives presented and existing epidemiological and toxicological data. However, the lack of substantial data to develop the current objectives and the absence of data on the interactions of trace contaminants both in the atmosphere and in the human body, indicates a need for caution in assessing the overall health impact of coal-fired plants on human health.

6.0 GLOSSARY*

acute effects - acute effects occur or develop quickly after a single exposure to a substance.

acute exposure - an acute exposure may occur either as a rapid single exposure or as more prolonged exposures, occurring over periods of hours.

aerosol - a gaseous state containing suspended particles of fine solids or liquids.

allergic reaction - a hypersensitive state caused by exposure to a specific substance, reexposure causing an altered capacity to react.

alpha particles - a positively charged particle composed of two protons and two neutrons, equal to the helium atom nucleus.

ambient air - the atmosphere surrounding the earth.

ambient air quality criteria - the data which support standards regulating substances in the ambient air.

ambient air quality standard - a concentration, upheld by some organization, which suggests the level of a certain substance in the air which should not be exceeded for reasons of health or welfare.

antagonism - opposition or contrariety between two or more similar things.

anthropogenic - of, or relating to the evolution of man.

anticarcinogen - a substance which counteracts the effects of a carcinogen.

aromatic - referring to a particular type of chemical characteristic associated with unsaturated cyclic organic molecules.

BaP - benzo-α-pyrene.

bioavailability - the extent to which a substance becomes available to various tissues after being presented to a test subject.

biological half-life - the time required for a given body concentration of a compound to be reduced by 50% due to elimination or metabolism or both.

biotransformation - the biochemical conversion of a compound to another form.

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*Definitions have been adapted from references 5, 94, 95 and 96.

carcinogen - a cancer-producing substance.

cardiovascular - pertaining to the blood vessels and the heart.

catarrh - mucous membrane inflammation, producing a free discharge, especially pertaining to the mucous membranes of the head and throat.

cause-effect relationship - a relationship between that which produces an effect and the effect produced.

chromosome - the DNA-containing body in the cell nucleus of all living matter which is responsible for the determination and transmission of hereditary characteristics.

chronic effects - chronic exposure occurs when exposure to a substance is frequent, daily or weekly.

conjunctiva - the membrane lining the eyelids.

cumulative effects - cumulative effects are those which increase by successive additions.

cutaneous - of, or pertaining to the skin.

daughter compounds - compounds formed by the spontaneous disintegration of unstable atomic nuclei.

dermatitis - an inflammation of the skin.

dose-response - the correlative relationship between the characteristics of exposure and the spectrum of resultant effects.

dyspnea - difficulty in breathing.

edema - an excessive accumulation of fluid in the body tissues; swelling.

enzyme - a protein which can catalyse or accelerate changes in a substance for which it is often specific.

enzyme co-factor - an element or compound with which an enzyme must unite in order to function.

epidemiology - the study of those factors influencing the frequency and distribution of disease in the human community.

epidermis - the outermose protective, nonvascular layer of the skin.

erethism - abnormal physiological and psychological disturbances.

erythrocytes - red blood cells.

fluorosis - a mottled discoloration of tooth enamel caused by the ingestion of excess fluorine during tooth development.

flyash - airborne particles of unburnable ash, especially as relating to air pollution.

fume - a gas with very small particles (0.2 - 1.0μ in diameter) of a solid or liquid suspended in it.

gingivitis - inflammation of the gums.

glucose (dextrose) - a sugar which is the principle source of energy for living organisms.

granulomatous - composed of granulomas, tumor-like masses or nodules of granulation tissue; usually due to chronic inflammation or invasion by non-living foreign bodies.

ground level concentration - the concentration of a specific substance in the ambient air at ground level.

hematologic - pertaining to the blood or blood-forming tissues.

hemoglobin - the oxygen-carrying, iron-containing protein in human red blood cells.

homeostasis - a tendency toward physiological equilibrium in an organism.

hypersensitive - abnormally heightened sensitivity.

hypertension - persistently high arterial blood pressure.

immune response - a physiological response mechanism which creates resistance to toxic or pathogenic substances.

ion - an atom or group of atoms which carry a positive or negative electric charge.

isotope - a chemical element having the same number of nuclear protons as another but having a different atomic mass.

keratosis - a horny growth on the skin, like a wart or callous.

lacrimation - secretion and discharge of tears by the eyes.

latency period - a period of seeming inactivity, as between initial stimulation and response.

legume - a plant of the family Leguminosae; including peas, beans, clover, alfalfa, etc.

leukocytosis - an increase of leukocytes, the white blood cells in the blood, due in many instances to inflammation, fever, hemorrhage, etc.

lipids - a group of organic substances, soluble in fat solvents, which act in the body as a fuel source and are important in cell structure.

lumbar - referring to the part of the body between the thorax and pelvis.

malaise - general feeling of bodily discomfort.

meteorology - the study of the atmosphere, especially as relating to the weather and weather conditions.

methemoglobin - a compound formed by the oxidation of the iron in hemoglobin, resulting in defective hemoglobin.

methylation - the addition of a methyl group (CH_3^-) to an element or compound.

morbidity - the state or quality of being diseased.

mucous membrane - the membrane which lines all bodily channels which communicate with the air, such as the respiratory tract and the alimentary tract, and which secretes mucous.

mutagen - an agent which causes changes in the genes or chromosomes of an organism.

nasal septum - the partition separating the two nasal cavities vertically in the center of the nose.

neuropathy - the state of disease or abnormality of the nervous system.

osteoporosis - an abnormal condition in which the bones decrease in density and weight, seen most commonly in the elderly.

osteosclerosis - an abnormal condition in which the bones harden or increase in density.

oxidation - combination of a substance with oxygen or a reaction in which the atoms in an element lose electrons and thus increase in valence state.

PAH - polycyclic aromatic hydrocarbons.

particulate - very small separate particles in the atmosphere which are collected on a high volume filter.

pathology - the medical study of disease, and especially the structural and functional changes in tissues caused by disease.

photochemical - chemical interaction of energy from the sun and chemical systems.

photosensitization - a heightened sensitivity of the skin to the sun.

pilosebaceous - relating to the hair follicles and sebaceous glands of the skin.

plume dispersion - a phenomenon in which the atmospheric effluent, or combustion products, emitted from a tall stack, are transported various directions or distances according to the weather.

pneumonitis - an inflammation of the lungs.

POM - polycyclic organic matter.

PPOM - particulate polycyclic organic matter.

proteinuria - a condition of protein in the urine, most commonly caused by kidney damage.

pyrolyzation - decomposition of organic substances due to increases in temperature.

radio-toxicity - toxic effects derived from radioactive properties of a substance.

renal - pertaining to the kidney.

respirable - capable of being breathed, fit to be breathed.

sequelae - a pathological condition following and caused by disease.

stomatitis - inflammation of the mucous tissue of the mouth.

subacute effects - effects which are identical to acute effects although less intense.

submicron - less than a micron (μ), or less than a length of one-millionth of a meter (10^{-9}m).

substernal - beneath the sternum, a bone which occurs as a plate over the middle of the anterior of the thorax.

synergism - a relationship of acting together so that the combined effects of two agents are greater than the sum of their individual effects.

teratogen - a substance causing physical defects in the developing embryo.

toxicology - the study of poisons, their nature, effects and detection and the treatment of poisoning.

trace contaminant - a substance occurring in very small, close to immeasurable amounts.

trace element - an element which occurs only in very small quantities.

TSP - total suspended particulates.

ulceration - the formation or development of a local defect or excavation on the surface of an organ or tissue, produced by a sloughing of inflamed tissue.

valence state - a chemical term referring to the extent to which an atom has undergone oxidation (loss of electrons) or reduction (gain of electrons).

vapour - particles of liquid suspended in gas.

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