FINAL REPORT

CANADIAN PUBLIC HEALTH ASSOCIATION

TASK FORCE ON ARSENIC

Yellowknife, Northwest Territories

Final Report

Canadian Public Health Association
Task Force on Arsenic

Yellowknife, Northwest Territories
1977
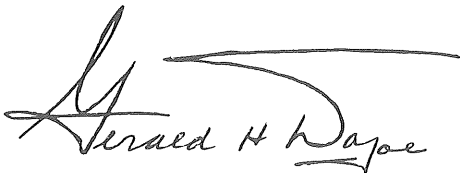
Foreword

This study of possible arsenic poisoning in Yellowknife, Northwest Territories, was undertaken by the Canadian Public Health Association at the request of the Department of National Health and Welfare. For the purposes of this report, the term "arsenic poisoning" has been defined to include all short-term and long-term ill effects of arsenic exposure.

This report outlines the activities undertaken by the CPHA Task Force in examining the issue "whether or not there is a serious health hazard to the community of Yellowknife as a result of possible arsenic poisoning", and includes the findings and recommendations of the Task Force.

To ensure representation and participation of all those concerned with this issue, submissions were invited and public meetings were held in Yellowknife, Northwest Territories and in Ottawa, Ontario. During the course of the study the Task Force had the benefit of consultation and input from many organizations and individuals. The Association is grateful for the consultation provided to the Task Force by Dr. Hector Blejer, Director, Department of Occupational Health, City of Hope, National Medical Centre, Duarte, California, U.S.A. and all those who supported and participated in this study. The cooperation and support of the general population of Yellowknife is sincerely acknowledged.

Gerald H. Dafoe
Executive Director
Canadian Public Health Association

December, 1977

View of Yellowknife

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Summary

Present arsenic contamination of Yellowknife results from gross emissions from gold mining and smelting operations which began in 1938. These emissions resulted in the accumulation of arsenic in the soil and the pollution of water draining from the area. While the control measures which were taken have considerably reduced the rates of emission, arsenic is still entering the environment. In consequence, a continuing potential hazard exists to the health of local residents and workers.

The Task Force attempted to evaluate this hazard from three approaches:

1. Arsenic in the Yellowknife environment.
2. Occupational exposure to arsenic in Yellowknife.
3. Arsenic and the health of the people of Yellowknife.

Considerable environmental data are available on Yellowknife. Unfortunately the data available on the health factors are very limited. Epidemiological studies have been sporadic. Such health data that do exist indicate the existence of some human exposure, but do not identify adverse health effects outside of the mine mill setting.

The Task Force's opinion, based on the best available information, is that:

1. The present arsenic input into the Yellowknife environment can be decreased by 1979 to about 20 percent of its present level. Further smaller reductions can be achieved by 1981. This will reduce the hazard from snow and soil contamination.

2. Only two surveys have been made of airborne arsenic levels in the mills. While the available data indicate that exposure is not particularly high, the Task Force recognizes that these data are not necessarily representative of present year-round conditions, nor of conditions which existed in the past. No clear-cut evidence of acute or chronic arsenic poisoning was found in past health studies, but localized effects such as skin rashes and upper respiratory irritation were noted. There are insufficient available records to provide evidence whether past occupational exposure has caused a change in the normal incidence of cancer in the employee population. Present arsenic levels in hair strongly suggest that the workers are still absorbing the arsenic.

3. Outside of the mine mill no immediate health effects can be found which are due to the present environmental levels of arsenic in Yellowknife. But a

number of continuing, long-term monitoring programs have been recommended to ensure that the arsenic hazard continues to decrease and that possible long-term effects are immediately identified.

4. A lung cancer death rate above the national average is noted for all the Northwest Territories and Whitehorse in the Yukon. Much of this increase is attributable to the high incidence in native groups and is unrelated to arsenic exposure.

   A cancer registry for the Northwest Territories has been recommended. This unit will be able to facilitate the investigation of the possible existence of arsenic associated cancers.

5. A community and environmental health service directly responsible to the people of the Northwest Territories has been recommended to carry out adequate health surveillance and preventive medicine.

Recommendations

The Task Force recommends:

1. THAT a continuing atmospheric monitoring program be conducted by the Government of the Northwest Territories, in cooperation with the Environmental Protection Service.

2. THAT ambient air monitoring techniques be based on the most up-to-date advice available through close consultation with the Environmental Protection Service.

3. THAT all vegetables and berries grown in the Yellowknife area be completely and efficiently washed prior to human consumption.

4. THAT routine monitoring of arsenic in drinking water based on the requirements of the *Canadian Drinking Water Standards and Objectives* should be continued indefinitely, as a normal public health monitoring program.

5. THAT the use of the Giant Yellowknife water supply as a source of potable water for plant employees be discontinued as soon as possible. The municipal system should be extended to provide employees with water for drinking and washing purposes.

6. THAT every effort be made to ensure that melted snow is not used as a potable water source in the Yellowknife area, and
   a) THAT every member of the community at risk be routinely advised and reminded of the hazard of using melted snow for drinking and cooking purposes, and
   b) THAT adequate quantities of potable water be made available to residents of unserviced communities. While the provision of water supply is a function of local government, the Task Force considers that both Giant Yellowknife and Cominco Mines have a responsibility to financially support such a program.

7. THAT any future use of lakes in the Yellowknife area as possible water supply sources should be approached with caution. In any such case, arsenic monitoring should be conducted over a period of several months to ensure the acceptability of the water for human consumption.

8. THAT Giant Yellowknife take immediate steps to reduce arsenic air emissions by commencing application of the best available technology even before this application becomes mandatory.

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9. THAT Giant Yellowknife obtain appropriate stack testing equipment as soon as possible and that baseline stack monitoring be conducted.

10. THAT the results of the current pilot project on water pollution abatement technology for Giant Yellowknife be made public and that the Northwest Territories Water Board apply effluent treatment requirements similar to those specified in the case of Cominco.

11. THAT routine monitoring of liquid effluent quality be conducted both at the outlet of the decant structure and at the outlet of Baker Creek, under the direction of the Northwest Territories Water Board.

12. THAT close attention be directed to the prevention of seepage from tailings ponds in future. The use of backup catch basins is a useful safety mechanism, and

    THAT routine surveillance and maintenance programs be conducted to ensure the adequacy of tailings storage.

13. THAT no further use of dry tailings for construction or fill be permitted and that access to tailings areas be completely restricted.

14. THAT the underground bulk method of arsenic storage continue to be used during the operation of the Giant Smelter.

15. THAT the Department of the Environment be kept informed of all new plans for underground storage of arsenic, notwithstanding that the Task Force recognizes that development of future underground storage areas is subject to assessment and approval by the Mining Inspection Branch.

16. THAT, at such time as the Giant Yellowknife operation is discontinued, the company take all necessary steps to seal off all points of entry of surface water or runoff into the mine.

17. THAT provision be made for monitoring water levels within the mines following closure, and that the Department of Indian and Northern Affairs (Mining Inspection Branch) be responsible for routinely monitoring variation in ground water level in the mines.

18. THAT the Giant Yellowknife Mines take appropriate steps to ensure that drainage from open pit workings cannot enter the arsenic storage area, and THAT through the Department of Indian and Northern Affairs, the Department of the Environment be kept advised of these activities.

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19. THAT the containment and reclamation of the arsenic storage areas at the Con Mine be completed prior to March 1st, 1980, either by provision of satisfactory containment structures at the site or by removal of the arsenic to underground storage, based on requirements comparable to those applied at Giant Yellowknife.

20. THAT air sampling for arsenic be carried out at the Giant Yellowknife Mine and Cominco Con Mine every 3 months for at least one year to obtain information as to possible seasonal variation, and data be obtained as to particle size distribution of the airborne dusts. This information would be of value in assessing the respiratory exposure; and

    THAT, since air sampling at the Con Mill is usually conducted by company personnel, on at least one survey a duplicate sampling be conducted by the Department of National Health and Welfare.

21. THAT an airborne concentration of 30 micrograms of arsenic per cubic metre of air be adopted as an 8-hour time-weighted average exposure level for inorganic arsenic dusts.

22. THAT the results of air sampling surveys be posted for the information of the employees.

23. THAT medical examinations, both preplacement and annual, of all employees occupationally exposed to arsenic, include:

    a) work history

    b) medical history

    c) a 14" x 17" chest x-ray

    d) a careful examination of the skin

    e) palpation of superficial lymph nodes

    f) complete blood count, including differential count

    g) hair and urine sampling for arsenic content

    h) electromyographic sensory and motor testing of at least two nerves.

24. THAT urine sampling be collected for arsenic determination at 3-monthly intervals for at least one year. For workmen in whom a level exceeding 150 micrograms per litre is found, a repeat sample should be taken within the next two weeks.

25. THAT, since arsenic may act as a cocarcinogen to increase the risk of lung cancer in persons who smoke, such persons who are occupationally exposed to arsenic be especially advised to stop smoking.

26. THAT sputum cytology examinations be conducted at 6-month intervals on workers who first entered arsenic exposure more than 10 years ago and who have attained the age of 40 years. Smoking histories should be recorded at the same time.

27. THAT each company appoint a physician to carry out the program of annual and periodic medical examinations and to provide medical surveillance.

28. THAT the results of each worker's urine and hair arsenic determinations and sputum cytology examinations be given to him, and be made available to his private physician on request.

29. THAT the work history, all medical records, and the chest x-rays for the last five years prior to termination of employment be maintained by the employer for each employee for at least 30 years.

30. THAT fresh coveralls and gloves be provided daily to workmen exposed to arsenic.

31. THAT, when it is necessary for a workman to enter areas of high exposure, such as a baghouse or electrostatic precipitator, he be provided with a self-contained breathing apparatus with positive air pressure at the face-piece, or its equivalent.

32. THAT the health services of the Northwest Territories be organized in the same manner as a Provincial public health jurisdiction. This organization to have two major components, community health services and environmental health services, and
THAT ongoing health monitoring be conducted and reported by this organization.

33. THAT a strong environmental health program be established for carrying out health studies, for providing health interpretation and consultation, for establishing occupational and community environmental guidelines and standards based on human health and for reviewing the adequacy of occupational and community environmental monitoring for health purposes.

34. THAT a legislative base be established by the Territorial Government for the development of occupational and community environmental health criteria, guidelines and standards.

35. THAT the Environmental Health component of the Northwest Territories health services organization have the primary jurisdiction for the protection of the health of workers and community residents from local environmental health hazards.

36. THAT the Environmental Health component have the power in law, inter alia, for the Director of the component or his agent to:

    a) enter any place of work at any reasonable time of day or night to carry out such inspections and investigations as the Director shall deem necessary;

    b) bring with him during such inspections and investigations any persons who may be necessary to assist him;

    c) take such samples of air, water, dusts and chemicals used in the workplace, or make such other measurements of environmental exposure as are necessary to assess the hazard to health;

    d) require the submission of information pertaining to the chemical constitution of any materials manufactured or used in the workplace which may be hazardous to health;

    e) require the pre-employment and periodic medical examination of workmen and the taking and submission of biological samples;

    f) require the keeping of records of all medical examinations of workmen and of the results of biological tests;

    g) require that all workmen be informed of the results of their medical examinations and biological tests;

    h) require the maintenance of records of morbidity and mortality of workmen and pensioners for 30 years or more for the purpose of conducting long-term epidemiological investigations;

    i) specify the information which shall be collected on medical examination forms, morbidity records and mortality records;

    j) determine such changes as are necessary to correct or control any hazard to health, and issue mandatory orders and/or recommendations for such correction or control;

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k) require the submission of plans prior to construction or alteration of any factory, mining or commercial establishment which entails the use of hazardous chemicals or minerals or which may result in the release of such materials to the workplace atmosphere or their emission to the ambient air or to water;

l) require the reporting of cases of industrial disease or of cases of cancer or of other disease conditions which may be suspected of being caused or aggravated by occupational exposure;

m) require the immediate closure or cessation of operations in any industrial, mining or commercial establishment which is, or may be, an acute hazard to health;

n) require control of noise levels of health significance at work or in the community;

o) regulate the installation and maintenance of x-ray equipment and other potential sources of ionizing radiation not specifically covered by provisions of Federal legislation;

p) require the examination as to competence and the licensing of persons applying pesticides as a business.

37. THAT in addition to carrying out the responsibilities inherent in the legislation (such as the periodic inspection of workplaces, the taking of samples and arranging for their examination, and the ordering of correction of hazardous conditions), the Environmental Health component shall be responsible for:

a) reporting on the findings and recommendations of all workplace investigations and/or sampling to the industries, labour unions, and other governmental agencies concerned;

b) reporting the findings of all community inspections or investigations and/or sampling to the individuals concerned, to other governmental agencies, interested community groups and the general public;

c) arranging for the periodic review of occupational morbidity and mortality records and the records of medical examinations and reporting the findings of such reviews;

d) arranging for the periodic review of hospital and school morbidity records and reporting on the findings of such reviews.

38. THAT Territorial public health engineering and inspection programs, such as those for the setting of hygienic standards, licensing and inspection of

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restaurants, food-handling establishments, dairies and pasteurization plants, public laundering facilities, etc., and for the approval or treatment of drinking water sources, for the approval of public and private sewage disposal and installations and for sanitary land-fill sites, etc., be the responsibility of the Environmental Health component.

39. THAT the Environmental Health component be headed by a health professional with training and experience in the field of environmental health or occupational health, and that the Director of the Environmental Health Component be directly responsible to the chief medical officer of health for the Territories. *doubtful.*

40. THAT supportive professional and/or technical expertise be obtained as needed from Health and Welfare Canada or from an adjacent province until such time as there is jurisdiction for the appointment of fulltime staff to the Environmental Health component.

41. THAT such penalties as may be deemed appropriate be established in law for failure to comply with an order issued by the Director of the Environmental Health component, unless such order is appealed to the Environmental Review Board (see below) and the appeal is upheld or varied.

42. THAT an Environmental Review Board be established in law to hear appeals from the decisions of the Director of the Environmental Health Component or from decisions of other agencies which are empowered to regulate the emission of contaminants to air, water or soil in the Territories. The Environmental Review Board should be empowered to hold public hearings, to set the time and place for such hearings, and to set the conditions under which the hearing shall be held including the requirement for the submission of evidence under oath or by affidavit. The Environmental Review Board shall confirm, deny, or alter the decision of the Director of the Environmental Health component or that of other enforcement agencies whose decision is being appealed. Appeal from a decision of the Environmental Review Board shall be to a court of law.

43. THAT the Environmental Review Board shall be composed of 7 members, appointed by the Territorial Government. 5 members of the Board shall be permanent members for the term of tenure and shall include one member from industry, one member from labour, one member from the native population, and two other members at large. Two members of the Board should be appointed pro tem for their expertise in the subject of the

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Replacement by Advisory Committee.

hearing. The Director of the Environmental Health component should not be a member of the Board. The Chairman of the Environmental Review Board shall report to the Commissioner of the Northwest Territories, and his report shall be made public and tabled before the Territorial Council.

44. THAT the Community Health component of the Northwest Territories health services organization be responsible for the provision of all community health services in the Territories including:

a) community nursing services;

b) school health services;

c) preventive health services;

d) monitoring of the health status of the population.

45. THAT the ongoing monitoring of the human population in Yellowknife be conducted by the Community Health component for the detection of subclinical or preclinical effects of the arsenic in the environmental reservoir. The continuous programs should include:

a) Recording and comparing the age-sex-specific hospital admissions in the major hospitals in the region.

b) Recording and comparing the visits to nursing stations by age, sex, and cause.

c) Recording and comparing school absenteeism with periodic studies to determine the proportion of absences due to illness and the nature of the illness.

d) Recording and investigating infectious disease epidemics with special note being made of differences in attack rates and case-fatality rates.

The periodic monitoring programs should include:

e) hair sampling of the general child population;

f) serial testing of hair from children known to have arsenic in their hair to determine if levels vary due, inter alia, to systemic arsenic uptake of a seasonal or variable nature;

g) testing of hair and urine from anyone admitted to hospital for any cause;

h) comparative review of hospital admissions by cause for Yellowknife and other centres in the Territories;

i) electromyographic studies of nerve conduction times;

j) sweeptest audiometry tests on school children;

k) such other community monitoring procedures as may from time to time seem indicated.

46. THAT the Northwest Territories Health Service Organization establish a Cancer Registry, as presently exists in all provinces except Ontario. This could be carried out in cooperation with an adjacent province.

Introduction

The Issue

On January 15th, 1977, a Press Conference was held in Toronto, Ontario by the National Indian Brotherhood (NIB) and the United Steelworkers of America (USW). At that time, the NIB and USW issued a statement declaring that there was a most serious arsenic problem in the Yellowknife area. The Brotherhood and the Steelworkers stated that certain specific groups in the Yellowknife area, especially miners and certain Indian people, had come into contact with high levels of arsenic. The response from the Department of National Health and Welfare was that the best available data to date suggested there was not a significant health hazard to Yellowknife residents as a result of possible arsenic poisoning. Despite a number of studies and discussions on the arsenic situation in Yellowknife, there continued to be differences of opinion over the significance of the data and the interpretation of the data.

The issue therefore, in its simplest terms, could be stated as follows:

Does there exist in Yellowknife, Northwest Territories a serious health hazard to the population of that community as a result of possible arsenic poisoning?

In an effort to resolve the issue, the Department of National Health and Welfare decided to establish an independent, impartial Task Force to study the question of possible arsenic poisoning in Yellowknife. The study and the selection of the Task Force members was to be the responsibility of the Canadian Public Health Association (CPHA).

On January 18, 1977, the Minister of National Health and Welfare, the Honourable Marc Lalonde, requested CPHA to select three distinguished scientists and to conduct an independent study of the issue. The Minister further advised CPHA of the terms of reference for the Task Force.

Terms of Reference for CPHA Task Force

The terms of reference for the Task Force were as follows:

(a) to review all available data relevant to arsenic in Yellowknife, and to receive submissions from interested individuals and groups;

(b) to advise on any additional data required and to ensure steps are taken to obtain such data;

(c) to specify areas requiring further scientific research;

(d) to assess the present monitoring program and to recommend improvements;

(e) to recommend any remedial action required;

(f) to provide a public report of their findings.

Under the terms of reference, the members of the Task Force were to approach the study in two phases. The first phase was to involve a review of all available relevant data, the submission and hearing of briefs as required by the Task Force from interested individuals and groups, and the submission of an initial report within three months of the beginning of the study.

Selection of the Task Force

Members of the CPHA Council were advised on January 20th of Mr. Lalonde's request. Consultation was carried out with a number of professionals knowledgeable about occupational health and, in particular, arsenic poisoning. In consultation, the CPHA Executive Committee determined the Task Force members must meet certain criteria. The combined expertise was to include:

(a) Medical background — expertise in research in trace metals;
(b) Epidemiological background — expertise in industrial health;
(c) Engineering background — expertise in environmental health;

The following criteria were also considered:

(d) Impartiality
(e) Availability
(f) Not a member of the Federal Public Service.

Individuals were identified who might meet the criteria for selection to the Task Force. After preliminary screening, the individuals were contacted to determine whether or not they wished to be considered as members of the CPHA Task Force on Arsenic. All individuals contacted by CPHA were fully informed of the terms of reference for the study, as outlined by the Department of National Health and Welfare. The final selection of the three-member Task Force was made by the CPHA Executive Committee. The members selected were:

> Dr. C.J.G. Mackenzie,
> Head,
> Department of Health Care and Epidemiology,
> University of British Columbia,
> Vancouver, British Columbia.
>
> C.E. Tupper, P. Eng.,
> Administrator,
> Environmental Health,
> Department of Health,
> Halifax, Nova Scotia.
>
> Dr. R.B. Sutherland,
> Consultant in Occupational Health,
> Nobel, Ontario.

The Minister of National Health and Welfare was informed of the selection of the Task Force members by CPHA. The Task Force members were contacted and advised of the Executive Board's decision and requested to meet at CPHA Offices in Ottawa as soon as possible. It was agreed that the first meeting of the Task Force would take place early in February, 1977.

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Methodology

Task Force Meetings

The first meeting of the Task Force was held at CPHA National Offices in Ottawa on February 16th and 17th. Dr. Cortlandt J.G. Mackenzie was appointed Chairman. It was agreed at this initial meeting that the investigation would be open; all individuals and organizations concerned with the issue would be invited to make submissions and meet with the Task Force. Public meetings would be held in Yellowknife and Ottawa. All information received by the Task Force relevant to the study would be available for public review at the Association's National Offices and at the office of the Northwest Territories Branch of CPHA in Yellowknife.

The Task Force would meet as a group as often as necessary and would meet on an individual basis with individuals and organizations as required. All meetings and information distribution would be coordinated through the CPHA National Offices.

Request for Information

The Task Force requested individuals and agencies, both government and private, to provide any information that would be of assistance to them in the study. This request was made by direct correspondence as well as through public notices in Canadian national newspapers and local newspapers in Yellowknife and Hay River, Northwest Territories. These notices invited the submission of briefs and informed the public that the Task Force would be holding public meetings in Yellowknife, on March 14-15, 1977, and in Ottawa, on March 28-29, 1977. To date forty-nine written submissions have been received at CPHA Offices, as well as approximately one hundred items of correspondence relating to the arsenic issue in Yellowknife.

Public Meetings

The purpose of the public meetings was to identify the issues and range of opinion on the issue and to offer an opportunity for public discussion. Both oral and written submissions were received at the public meetings. Presentations were received from representatives of government, unions, associations, agencies and private individuals in both Yellowknife and Ottawa. The presentations were taped and transcripts of the proceedings are available for review by the public at CPHA Offices in Ottawa and Yellowknife. Interpretation services were provided at the meetings, in English, French and the Dogrib languages.

Public Information Services

Concern was expressed to the Task Force members that the Yellowknife residents were not always kept as fully informed as desirable concerning the arsenic issue in the Northwest Territories. In an attempt to better accommodate

this concern, the Task Force, through the media, stressed the importance of public involvement at the public meetings and the wish of the Task Force members to receive and review submissions as well as to meet with agencies and individuals at their request.

In addition to the foregoing efforts by the Task Force to inform and involve the public about the study, arrangements were made for audio-visual documentation to be carried out at the public meetings in Ottawa and to be made available immediately thereafter to interested groups and individuals in Yellowknife. This provided up-to-date information of what transpired at the public meetings in Ottawa.

The Task Force returned to Yellowknife on May 16 and 17, 1977, for the purpose of discussing with those concerned the approach to be taken by the Task Force in the Interim Report.

The Interim Report

The Interim Report documented the activities carried out in Phase I of the study. The Task Force isolated certain areas of concern and made 27 recommendations reflecting these concerns. The Interim Report was released as a public document in May 1977.

In Phase II of the study, the Task Force continued their scientific research as well as assessing the effectiveness of current monitoring programs in Yellowknife. Consultation continued with individuals and groups concerned with the Yellowknife issue. The Task Force requested that additional samples of water, soil and fish be collected from specified areas in the Yellowknife vicinity. The results of these samples are noted in Appendices A and B and addressed in Chapters II & IV.

In keeping with the mandate to advise the Minister of the need for any additional data and further scientific research, the Task Force recommended that an electromyography program be implemented in Yellowknife. Following consultation with the Department of National Health and Welfare, an agreement was reached for the CPHA to initiate an EMG program. The EMG program began in Yellowknife in early November. This program is referred to in Chapter IV of this report and the protocol is outlined in Appendix C.

CANADA

Scale in miles

Miles 100 50 0 100 200 300 400 500 Miles
Kilometres 100 0 100 200 300 400 500 600 700 800 Kilometers
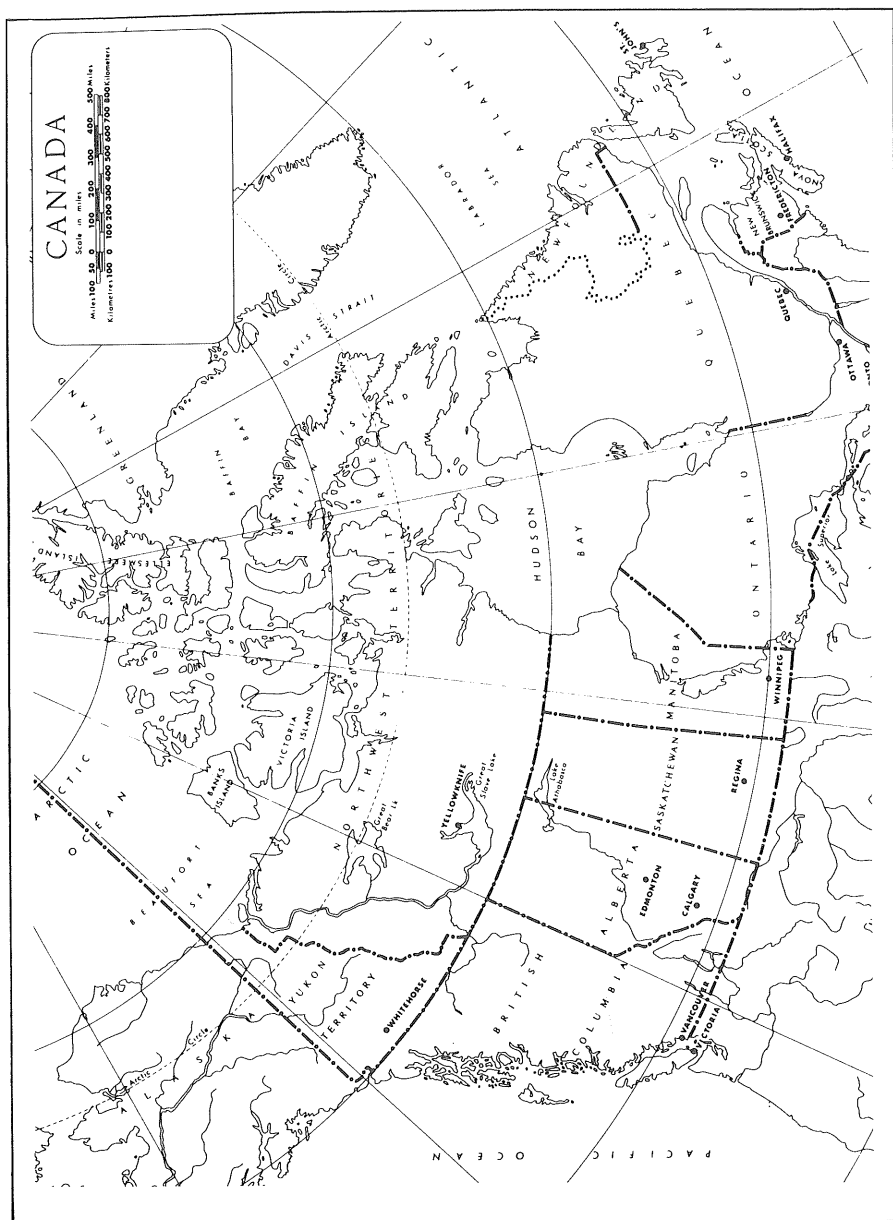
Courtesy Energy, Mines and Resources Canada

Chapter I.   Background to Yellowknife

Climate and Population

Yellowknife is situated on the rugged terrain of the Pre-Cambrian shield on the shores of Great Slave Lake, Northwest Territories. At 62°N latitude, Yellowknife experiences an extreme climate with only a few hours of daylight in the winter, and with summer days with more than 20 hours of sunlight. The lifestyles of these northern residents vary accordingly. The population of the Northwest Territories consists of Indian, Eskimo, and other groups totalling 39,869 (100%). In 1976 there were 7,812 Indians representing 20% of the population, 14,723 Eskimos (37%), while the remaining 17,334 made up 43% of the population. This last group includes all but the two primary Native People and includes the Metis, but is largely composed of white immigrants from the south. While the percentage group distribution has remained the same, the total number of people in the Northwest Territories has increased approximately 50% in the last ten years. The population of Yellowknife specifically has risen from 3,700 in 1966 to roughly 10,000 today. From 1967 it has been the thriving capital of the Northwest Territories.

Gold Mining Operations

Gold was discovered in Yellowknife in 1936. One year later a small town was established on a peninsula and adjoining island which jut into the northwest end of Yellowknife Bay. An enlarged townsite was developed in 1948, but many people remained in the old town on the peninsula and on Latham Island. The town is situated between the plants of two gold-producing companies: Giant Yellowknife Gold Mines Limited, 3½ miles to the north, and the Consolidated Mining and Smelting Company of Canada (the Con Mine), a subsidiary of Cominco Limited, 1½ miles to the south.

Arsenic is widely distributed in small amounts through the waters and soil of the world. Traces are also present in most foods, particularly seafood. Arsenic is principally found in the ores of copper, lead, zinc, gold, and silver. The smelting of these ores can result in the release of arsenic into the atmosphere chiefly as arsenic trioxide. Arsenic occurs naturally in the gold ore bodies of the Yellowknife area mainly as arsenopyrites.

As discussed further in this report, full recovery of the gold requires extraction of the ore in the milling process. This process sometimes changes the arsenopyrite into more toxic forms. The fumes produced by the roasting process contain arsenic trioxide (As_2O_3) and sulphur dioxide (SO_2). These materials, if emitted into the environment, present a potential health hazard.

Giant Yellowknife Mines

The Giant Yellowknife Mines gold mine has been in production since 1948 and employs about 360 people. It is situated 3.5 miles north of Yellowknife (see

Figure 1.
Giant Mine Site, Yellowknife Bay Area

Figure 1.
Giant Mine Site, Yellowknife Bay Area
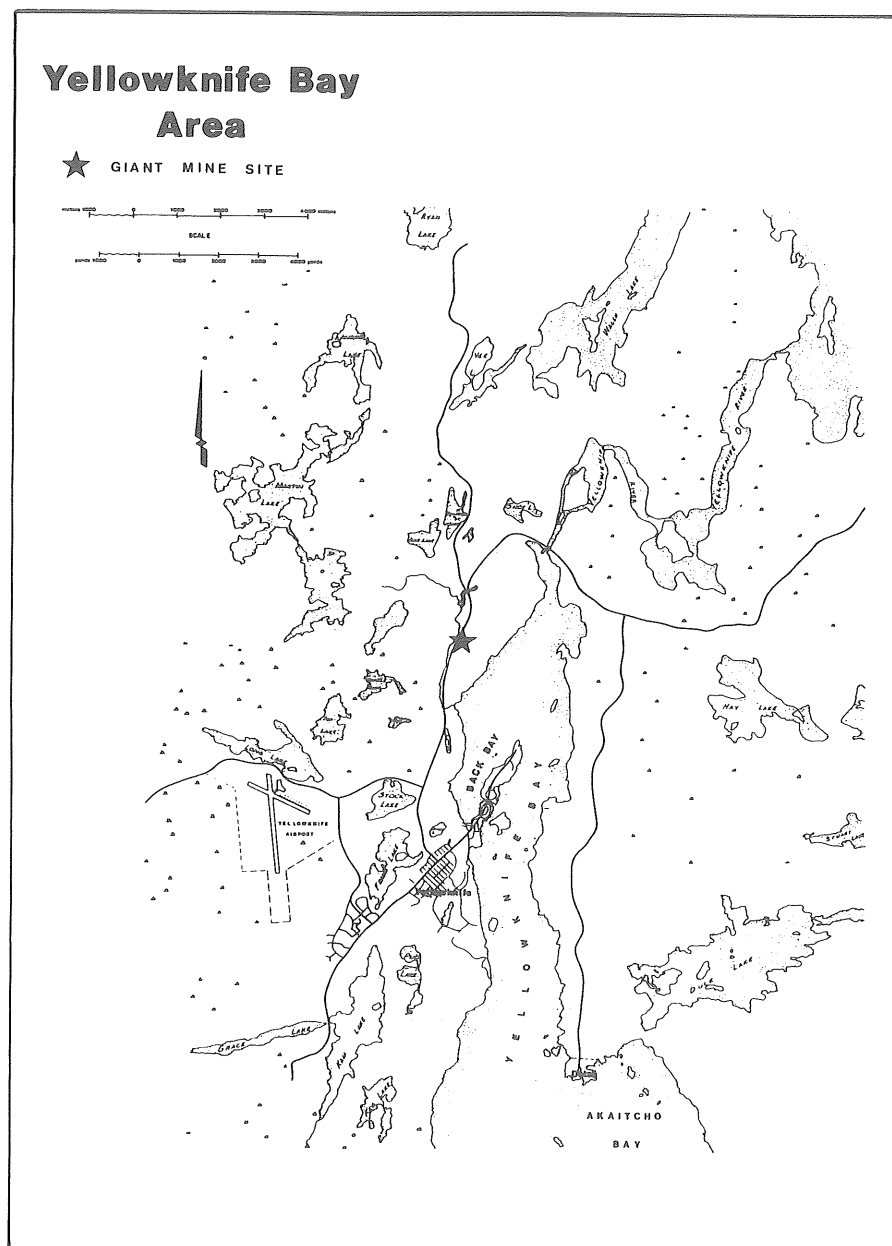
Figure 2.
Disposal System, Giant Yellowknife Mines, Yellowknife Bay Area

The arrows indicate the intended direction of flow of effluent through the tailings ponds; into Baker Creek, and into Yellowknife Bay. Dykes are shown as dark, solid lines separating the three tailings ponds, and are also located at the southwest and northeast corner of the first pond.

1 Mile
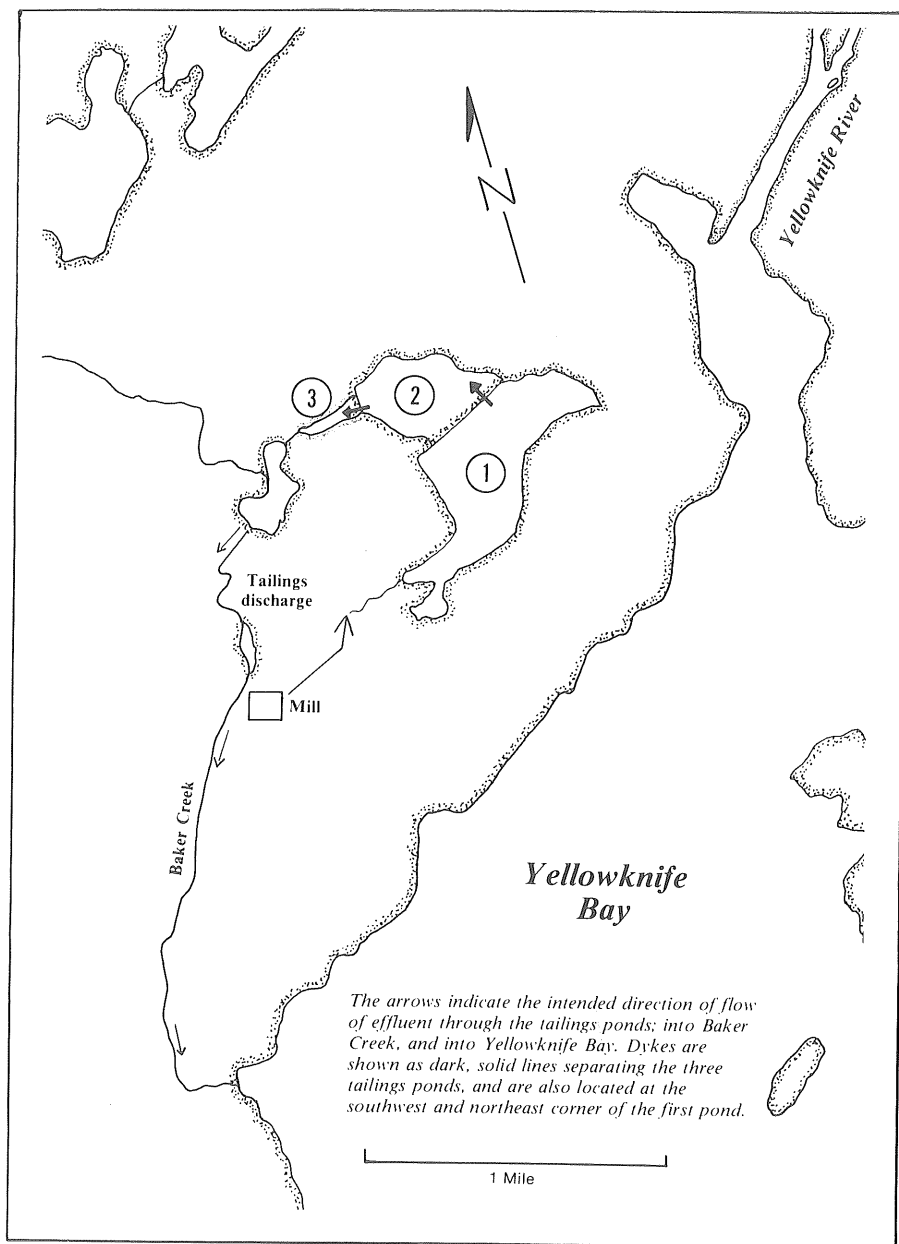
Figure 1) and mines both underground and open pit areas at the rate of 975 tons per day. The yield from mining results in 0.32 oz. of gold per ton (1).

The main underground shaft goes down to about 2,300 feet, and most of the mining is done by cut and fill stoping methods. Gold ore occurs in schist containing 30% quartz with various amounts of calcium and iron carbonates, together with sulphides and other sulphur compounds. The bulk of the sulphides are composed of arsenopyrites and pyrites along with stibnite and sulphantimonides of lead, iron, and copper (2).

At the Giant Yellowknife mine the gold is bound so intimately with sulphides that it must be roasted before being subjected to the cyanide process. The Giant ore is hoisted to the surface and is reduced to smaller particles by jaw and cone crushers, and more finely pulverized by two ball mills (wet) with classifiers. The flotation process is then used to separate and remove most of the waste rock and thereby obtain a more concentrated mixture which contains the gold along with most sulphide minerals. This concentrated mix is roasted at 900°F to remove arsenic, sulphur, and antimony, and the resulting product is a porous calcine. Gold is dissolved from the calcine by adding sodium cyanide and lime in the presence of oxygen, and the gold is subsequently precipitated from solution and finally refined in a gold bullion furnace.

In conjunction with the above ore-roasting process there is a treatment system for the roaster gases and dusts which are emitted. The first stage of this system is to remove dust from the emissions and collect them in an electrostatic precipitator. This dust is treated by a cyanide process and activated carbon to recover the fraction of gold it contains. The gas which leaves the electrostatic precipitator is cooled to 210°F, condensing arsenic and antimony oxides that are then filtered out in a bag type collector. The arsenic trioxide dust collected by the dust collection system is permanently stored in sealed stopes or underground storage vaults. Gases leaving the bag collector are disposed of at an elevation of 150 feet into the atmosphere (2). The waste washings go to the tailings pond.

The tailings pond at Giant Yellowknife Mines drains into Baker Creek which, after passing a series of dams, flows into Yellowknife Bay (see Figure 2). The dust collection system has substantially reduced the arsenic emissions to the atmosphere. This was not the case prior to the introduction of this system in 1958.

The Con Mine

The Con Mine site has been in production since 1938 and the present operation employs about 250 people. It is located 1.5 miles south of Yellowknife (see Figure 3) and is limited to underground mining of 404 tons per day yielding 0.50 oz. of gold per ton (1). Ore bodies down to about 2,300 feet depth were first mined, and then a shaft was extended to 4,900 feet where the bulk of the ore is currently being mined.

Figure 3.
Con Mine Site, Yellowknife Bay Area
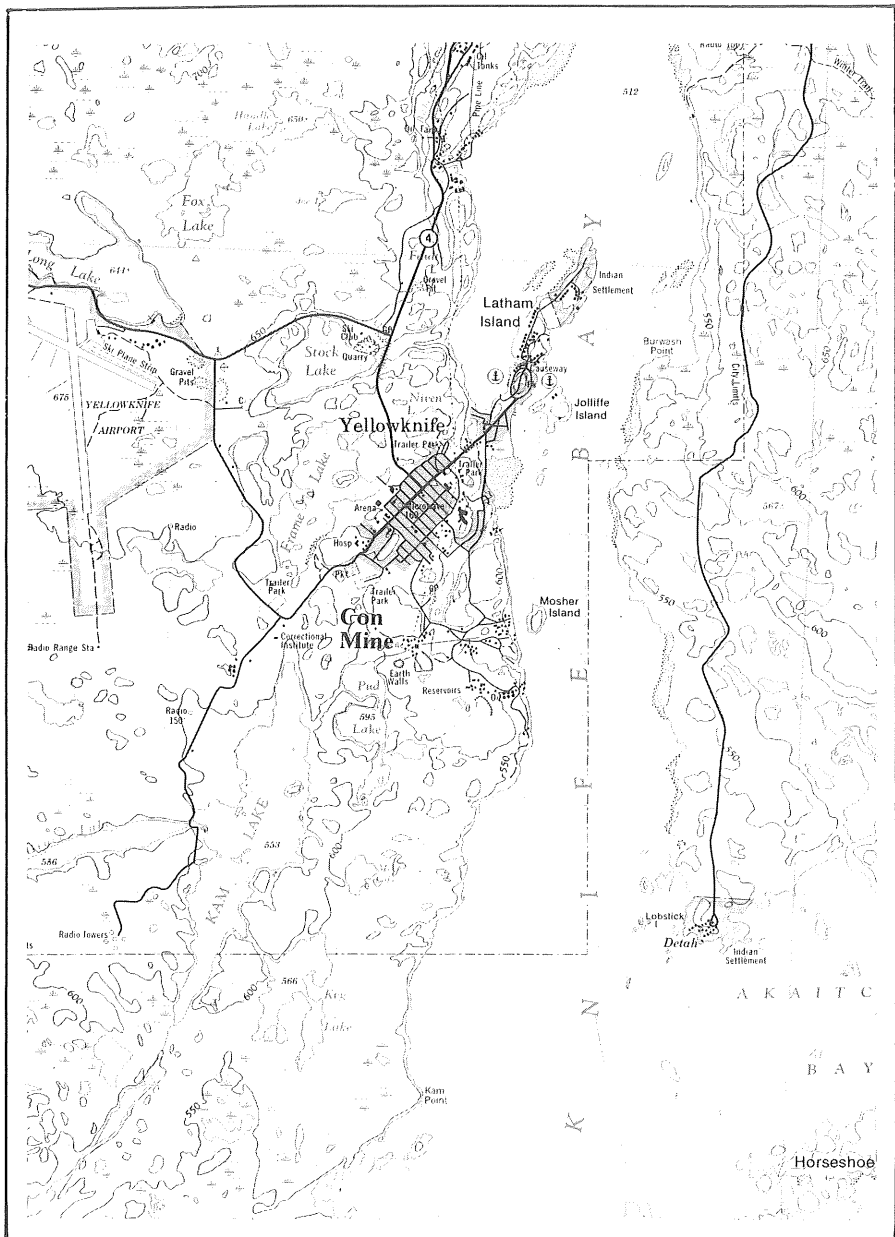
After extracting the ore by drilling and blasting it is hoisted to the surface, crushed and then wet ground in a ball mill with cyanide solution. The slurry is agitated in large tanks to dissolve the gold, which permits the solids to be filtered out and sent to waste as tailings. The solution is put through a precipitation unit under vacuum to remove all oxygen, and at this stage zinc dust is added to precipitate the gold. The precipitate is then filtered from the solution and after adding flux, refined in a gold bullion furnace (2).

Roasting the ore began at Con Mine in 1941, and was interrupted during the war years and restarted in 1948. Con Mine installed a wet scrubber system in 1949 to minimise the dispersion of arsenic into the air environment. The resulting arsenic-containing slurry was, and still is held in tailings ponds near the Con Mine (see Figure 3). Con Mine stopped roasting the ore in 1970, and now removes the arsenic by means described above, storing it in tailings ponds. Tailings from the Con Mine are contained in Pud Lake, which also flows towards Yellowknife Bay via Meg Lake, Keg Lake and swampland (see Figure 3).

Investigations were undertaken by government agencies as early as 1949 to evaluate the potential hazard to the community and determine whether preventive measures were necessary. Over the years there has been much concern about and subsequently many studies relating to the arsenic situation in Yellowknife (2-6). Some of these were initiated by Federal and Territorial Government departments, and there was also a joint study by the United Steel Workers of America and the National Indian Brotherhood more recently.

While early data indicate much heavier emissions of arsenic into the environment of Yellowknife, recent data show that these earlier emissions have been reduced (3, 6, 7). Elaboration of these earlier studies are included in the following chapters.

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II. Arsenic in the Yellowknife Environment

Distribution

Arsenic is ubiquitous in nature. It ranks twentieth in abundance among the elements in the earth's crust. Arsenopyrite is by far the most common mineral form. In addition to the natural presence of arsenic in the environment, there are a variety of man-made sources. The production of gold requires separation of all associated minerals including arsenic. This process of separation can lead to the introduction of arsenic into the surrounding environment, by atmospheric emissions, liquid effluents, and solid wastes, and, in the case of Yellowknife, constitutes the major man-made source.

(a) *Air Quality*

Arsenic levels in ambient air are usually low, from less than 0.01 micrograms to 0.2 micrograms per cubic metre (1 microgram = 1 millionth of a gram). The average annual concentration at 133 sampling stations in the United States was 0.02 micrograms per cubic metre (8). A study in Toronto found arsenic concentrations ranging from 0.003 to 0.336 micrograms per cubic metre in the vicinity of two secondary lead smelters, while control areas in that city had arsenic concentrations ranging from 0.007 to 0.051 micrograms per cubic metre (9). Non-urban areas have been found to have a maximum average concentration of 0.02 micrograms per cubic metre, with most values less than 0.01 micrograms per cubic metre. Large cities generally have a higher arsenic concentration in the air than do small cities because of fuel combustion for electricity and heating. An ambient arsenic concentration of 0.03 micrograms per cubic metre was calculated on the basis of the amount of coal burned in New York City. This agrees well with the observed air concentrations for that city (10). Average annual concentrations as high as 0.75 micrograms per cubic metre have been recorded in 1964, in El Paso, Texas, 0.73 micrograms per cubic metre in 1974, in Welland, Ontario, and 0.5 micrograms per cubic metre in Anaconda, Montana, in 1961-62 (10).

The Environmental Protection Service (EPS), Environment Canada, conducted an ambient air quality study in the Yellowknife area from 1973 to 1975 (11). Annual geometric mean arsenic concentrations in ambient air during that period (as measured by the hi-vol method) were 0.08 micrograms per cubic metre in 1973, 0.09 micrograms per cubic metre in 1974, and 0.06 micrograms per cubic metre in 1975. Maximum levels recorded in any 24-hour period were 0.95 micrograms per cubic metre in 1973, 1.34 micrograms per cubic metre in 1974, and 3.91 micrograms per cubic metre in 1975. The individual 24-hour maxima were all recorded close to the Giant Yellowknife smelter (11).

Additional conclusions of the EPS study with respect to ambient air quality included: (a) the annual geometric means for total suspended particulate matter during the period did not exceed the Maximum Desirable National Air Quality Objective (see appendix D), although the 24-hour total suspended particulate

levels for the period exceeded the Maximum Acceptable National Air Quality Objective approximately 10% of the time; (b) with respect to sulphur dioxide concentrations in ambient air, the annual arithmetic mean did not exceed the Maximum Desirable National Air Quality Objective, although the hourly ambient concentrations occasionally exceeded the Maximum Desirable Level, but only rarely exceeded the Maximum Acceptable National Air Quality Objective; (c) the average arsenic deposition rate over many square miles covered in the study was found to be 10 pounds per square mile per month (Table I); (d) the average deposition rate for total particulate matter, for the area, was found to be 11 tons per square mile per month.

TABLE I
Mean Arsenic Deposition Rate by Station
June — October 1975 (Lbs/Sq Mile/Month)

Station	Arithmetic Mean	Geometric Mean
D 1	2.61	2.44
D 2	8.12	7.01
D 3	4.77	4.26
D 4	3.02	2.69
D 5	7.50	6.55
D 6	3.65	2.09
D 7	3.72	3.32
D 8	4.39	4.19
D 9	2.19	1.79
D10	3.13	1.43
D11	7.12	6.42
D12	4.30	3.77
D13	2.50	1.52
D14	27.32	25.0
D15	31.94	25.0
D16	37.44	29.6
D17	9.42	8.22
D18	7.62	6.27
D19	4.42	4.28
D20	3.34	3.16
D21	33.88	8.87
D22	2.87	2.79
Overall Mean	9.79	4.83

Source: (11)

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TABLE II
Description of Dustfall Stations

Station Identification	Type of Field Mounting Used	Height Above Ground Level of Container Opening (ft)	General Comments on Site Location
D 1	Stand	13	On Detah elementary-school roof.
D 2	Pole	6	100 ft. west of Con Mine ducking area, on rocky out-crop.
D 3	Stand	14	On mobile trailer home in Northland Trailer Park, 50 feet from NT High-volume sampler.
D 4	Stand	17	On hospital roof.
D 5	Stand	22	On Hudson Bay Store roof, 50 feet from NAPS High-volume sampler.
D 6	Pole	6	300 feet east of paved roadway near Niven Lake.
D 7	Pole	10	In Back Bay area, 10 feet from shoreline on rock out-crop.
D 8	Stand	30	On airport terminal building, 50 feet from AIR High-volume sampler
D 9	Pole	7	300 feet east of MOT radio tower.
D10	Stand	4	2,000 feet north off gravel roadway, 10 miles west of Giant Mine, serves as background station.
D11	Stand	15	On mobile trailer home in housing quarters of Giant Mine employees, 50 feet from GT High-volume sampler.
D12	Pole	11	25 feet east of shoreline on the tip of Latham Island.
D13	Stand	4	300 feet west of gravel road to Detah on rock mound near clearing.

TABLE II (continued)
Description of Dustfall Stations

Station Identification	Type of Field Mounting Used	Height Above Ground Level of Container Opening (ft)	General Comments on Site Location
D14	Pole	6	1,000 feet east of Giant's open pit mining operations on leeward side of hill, 150 feet from paved roadway.
D15	Pole	6	⅓ mile directly west of Giant Mine stack on rocky ridge, ½ mile north of open pit mining operations, 300 feet from GW High-volume sampler.
D16	Pole	6	300 feet off gravel roadway intersection, ½ mile north of Giant.
D17	Stand	4	300 feet off gravel roadway on rock mound clearing, 1 mile north of Giant.
D18	Stand	4	300 feet south of road and elevated 100 feet above lake level.
D19	Stand	4	300 feet from paved road north of Stack Lake on rock mount 50 feet above road level.
D20	Stand	4	1,000 feet south of gravel roadway, 10 miles east of Giant Mine, serves as second background station.
D21	Stand	4	Adjacent to fenced-in As_2O_3 tailings pond and between Negus tailings pile and Con Mine operations, on rocky mound.
D22	Pole	4	On sparsely vegetated ridge halfway between Con Mine operations and new city housing subdivision.

Source: (11)

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Up to the present time, no atmospheric standard for arsenic has been established in Canada or the United States. However, a maximum 24-hour atmospheric concentration of arsenic has been recommended in the USSR and Czechoslavakia at 3 micrograms per cubic metre. The Province of British Columbia has a preferred standard of 1 microgram per cubic metre, 24-hour average.

It is the opinion of the Task Force that with respect to arsenic the quality of ambient air in the Yellowknife area is acceptable at the present time. It should be noted however that individual 24-hour arsenic concentrations measured in the vicinity of the Giant Yellowknife Smelter have been high, on occasion, and that the trend seems to be towards an increase.

Although ambient air quality in earlier years was not measured, it seems certain that present ambient air quality is greatly improved over the air quality during the earlier years of smelter operation. We have noted above that the EPS study determined an average arsenic deposition in the area of approximately 10 pounds per square mile per month (Tables I & II), a finding which has relevance to soil, snow, and plant concentrations of arsenic as discussed later in this report. This figure also has significance in terms of ambient air quality, since arsenic is brought into the atmosphere by combustion and normally exists as an oxide. Removal of arsenic from the air takes place by settling or rainfall, and as a result, arsenic concentrations do not build up in the air. It has been reported that during the mid-1950's, the average arsenic deposition rate was approximately 106 pounds per square mile per month (6).

The Task Force considers it essential that a continuing atmosphere monitoring program be conducted by the Government of the Northwest Territories, in cooperation with the Environmental Protection Service. A continuing program is needed to: (a) provide a continuing assurance that ambient air quality remains within safe acceptable levels; (b) provide continuing information on air-quality variations which occur; and (c) provide verification of the accuracy and validity of earlier ambient survey results.

This community air-monitoring program should be based upon strategic distribution of monitoring stations, sufficient to allow complete community coverage. Guidance and assistance in this respect can be obtained from professional staff of the Environmental Protection Service. Routine monitoring for arsenic, sulphur dioxide, suspended particulates, and dustfall should be included. It is recognized by the Task Force that there are difficulties in conducting ambient air monitoring for arsenic. Electrostatic precipitators and impingers will not provide adequate samples in a reasonable sampling period because of the low concentrations in ambient air of arsenic compounds to be measured. The traditional and most common sampling method, at present, is high-volume filtration and collection, on membrane or glass-fibre filters. The accuracy of this method is compromised by the physical properties of arsenic

compounds found in ambient conditions (5). Particulate arsenic compounds, such as arsenic trioxide, are appreciably volatile. Therefore losses may be suspected during and after collection. Based upon the equilibrium established between solid particulates on the filter, and vapour in the passing air, the significance of this loss will depend upon the pollution level, increasing with decreasing levels, and may be significant at the very low levels encountered in the community (5). This problem of ambient sampling is not considered to be a major constraint with respect to the significance of ambient air-quality data collected in Yellowknife in recent years, since even large variations in the accuracy of measured results would still provide acceptable levels. It has also been noted that collection by the high-volume sampling method may only be efficient at low temperatures (5), possibly a positive consideration in the Yellowknife case. It is recommended that ambient-air monitoring techniques for arsenic be based upon the most up-to-date advice available through close consultation with the Environmental Protection Service.

(b) *Soil and Vegetation*

The natural arsenic content in virgin soils varies from 0.1 to 40 ppm with an average of about 5-6 ppm. Arsenic levels in plants not treated with arsenical spray or exposed to arsenic fall-out seldom exceed 0.5 ppm fresh weight (5 ppm dry weight) (10).

Studies conducted in the Yellowknife area have consistently indicated (12) considerable contamination by arsenic compounds in the soil and vegetation of the area. In addition, there is minor contamination by antimony and trace contamination by other heavy metals. Soils in the city of Yellowknife contain a highly variable concentration of arsenic ranging from 1 to 600 ppm. In the vicinity of the mines, levels of more than 4,000 ppm have been reported (8). Also arsenic concentrations up to 10,000 ppm were measured in lichen, and up to 100 ppm in black spruce and willow leaves (13). Background levels of arsenic in soil approximately 80 kilometres from Yellowknife have been found to be approximately 25 ppm (14). Road-dust samples contain from 20-200 ppm (14).

Vegetables in the area seem to contain far less arsenic than the soil in which they grow (this is consistent with results of studies conducted in other areas) and arsenic levels in garden vegetables in Yellowknife do not appear to indicate heavy contamination. Vegetables do not have a significant arsenic content even when grown in soils containing high concentrations of applied arsenic trioxide (10). In addition, there appears to be little chance that animals would be poisoned by consuming plants containing arsenic residues taken up from contaminated soils, because plant injury occurs before toxic concentrations could build up. Nevertheless, surface contamination of plants due to heavy arsenic fall-out could present a potential hazard to ruminants (10).

It is important to note the wide variability in the relationships among soil-arsenic content, plant-arsenic content, injury symptoms, and phytotoxicity reported by different investigators. Work carried out in Kentville, Nova Scotia, in 1960, using pea and bean plants, showed that while soil plots contained arsenic at 126-157 ppm, most of the arsenic in the plant was found in the vines (2.1 ppm) and pods (0.88 ppm), with only small amounts in seeds (0.18 ppm) (15). Results of analysis of fruits and vegetables collected in August 1971 in Yellowknife by Health and Welfare Canada showed clearly that vegetables and fruits grown in a contaminated area had arsenic chiefly on their surface, and that customary cleaning procedures would reduce the arsenic content to normal levels (5). At the present time, considerable contamination of soil by arsenic compounds is a product of many years of heavy deposition, augmented by a continuing current rate of deposition of approximately 10 pounds per square mile per month. It is essential therefore that all vegetables and berries grown in the Yellowknife area be completely and efficiently washed before being eaten. Maximum allowable arsenic limits in food, as designated by the Health Protection Branch, Department of National Health and Welfare, are 2.0 ppm arsenic for fresh fruits, and 1.0 ppm arsenic for fresh vegetables.

A study carried out in September 1975 (16) categorized vegetation zones, based upon the effects of air pollution, as follows:

Zone 1. Within ½ mile of the Giant Mine, especially to the west and northwest. Most plants have been killed and eroded away. Only some of the small trees and shrubs are still standing, and these show damage due to sulphur dioxide.

Zone 2. Between ½-2½ miles to the west and north and ½-1 mile to the east and south. Most lichens have been killed, but the rest of the vegetation appears fairly normal.

Zone 3. Between 2½-12 miles west, 2½-6 miles north, 1-7 miles south, and 1-2 miles east. There is mild damage to some of the vegetation.

Zone 4. Beyond the outer limits of Zone 3, there is no evidence of damage by pollution.

In summary, the above study indicated that the pollution-sensitive lichens show symptoms of air-pollution injury as far as 12 miles from Yellowknife, but the trees and shrubs seem unaffected beyond half a mile of the pollution source. Very close to the Giant Mine, many of the trees and shrubs have been damaged, but the symptoms seem to be due to sulphur dioxide. None of the injury could be related to arsenic sensitivity (16).

A submission to the Task Force by the Environmental Protection Service noted that arsenic concentration (in soil and vegetation) decreases as the inverse square of distance from the Giant Yellowknife Mine Limited roasting facility (17). Another study (13) on arsenic concentrations in soil, lichen and AO Horizon soil* determined that the contribution to arsenic concentrations in soil

*AO Horizon soil is soil to a depth of 2 cm.

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Figure 4.
Arsenic Concentration (Soil Lichen and AO Horizon) along Transect A-A,
Yellowknife, Northwest Territories
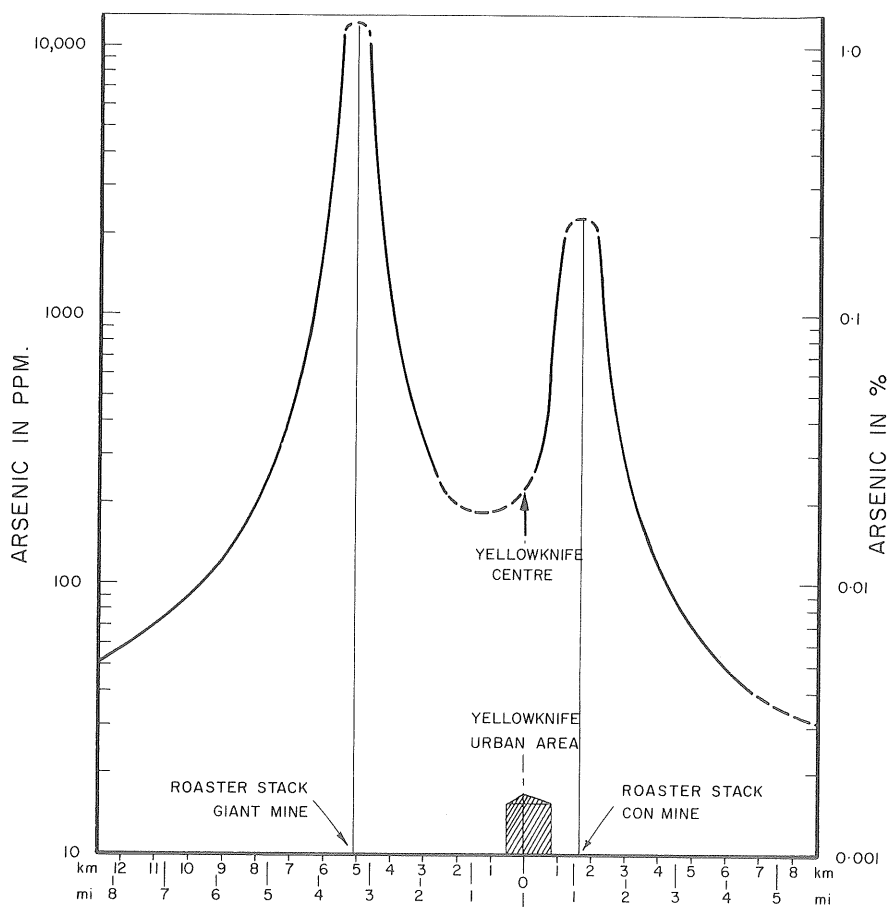
Source: (13)

PROFILE (A−A)

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Figure 5.
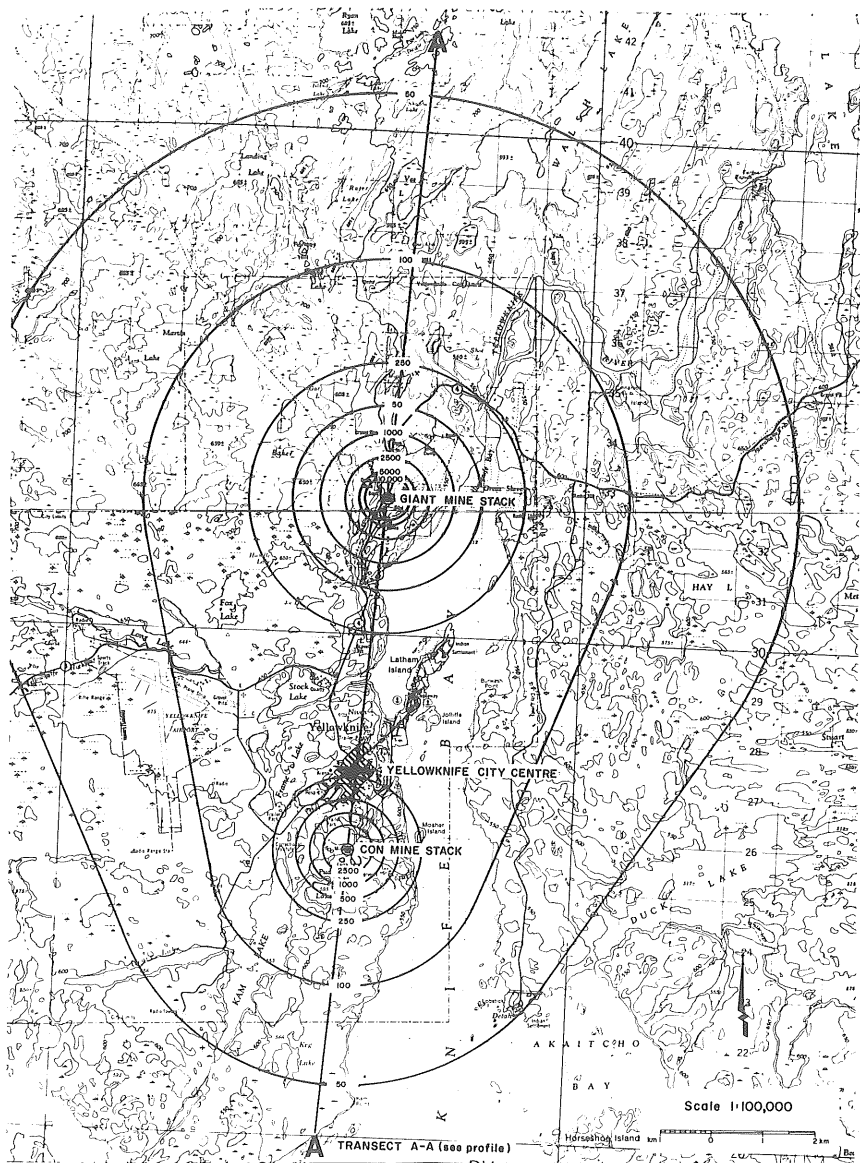
Source: (13)

and lichen from Cominco Mines was 1/3 that due to the Giant Mine, rather than the 1/6 expected by comparing airborne emissions. It was concluded that mobilization of arsenic from the arsenic ponds at Con Mine was probable. The arsenic concentrations identified in that study are shown in Figure 4 and, by use of isocons, in Figure 5. During the same study, samples were also analysed for chromium, manganese, tellurium, vanadium, beryllium, tin, copper, zinc, nickel, aluminium, iron, potassium, sodium, calcium, magnesium, phosphorus, strontium, titanium, gold, silver, and silicon. However, these elements were found to be present at levels not significantly higher than the normal values to be expected. Levels of uranium, boron, and molybdenum were below the level at which they could be detected (13).

(c) *Snow*

We pointed out earlier that arsenic deposition rates in the Yellowknife area have been determined to be approximately 10 pounds per square mile per month. This figure has been confirmed by surveys of snow quality in and around Yellowknife (17). Since snow remains on the ground throughout the entire winter season, arsenic levels would be expected to build up as a result of continuing deposition. Since the greatest significance of snow, in terms of human exposure, would be as a source of drinking water, arsenic levels can be compared to the maximum permissible level of 0.05 milligrams per litre or ppm specified in the Canadian Drinking Water Standard for arsenic.

A snow survey conducted in 1975 (17) found that 96% of all scoop snow samples exceeded the Canadian standard. In the case of core samples of snow, 85% of the samples exceeded the maximum standard. Average concentrations for each varied between 0.17 and 0.52 milligrams per litre. In addition, snow melt usually contains undissolved particles which have been shown to contain very high concentrations of arsenic which would increase the risk.

Exceedingly high levels of arsenic would be expected to build up in the snow in the vicinity of the Giant Smelter, since deposition rates as high as 564 pounds per square mile per month have been measured inside the Giant property (Figure 6). This situation has been confirmed by measurements recorded during several different surveys (8) ranging as high as 8.6 - 11.4 ppm.

The Task Force concludes that the use of snow as a source of drinking water could constitute a serious health hazard.

(d) *Potable Water Supplies*
(i) Yellowknife Municipal Water Supply

The Canadian Drinking Water Standards and Objectives, 1968 (18) specify that the maximum permissible limit for arsenic in drinking water is 0.05 milligrams per litre, and that the acceptable limit is 0.01 milligrams per litre. Before December 1969 the source of water supply for the town of Yellowknife

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Figure 6.
Arsenic Isopleths for Snow Survey, 1975, Yellowknife, Northwest Territories
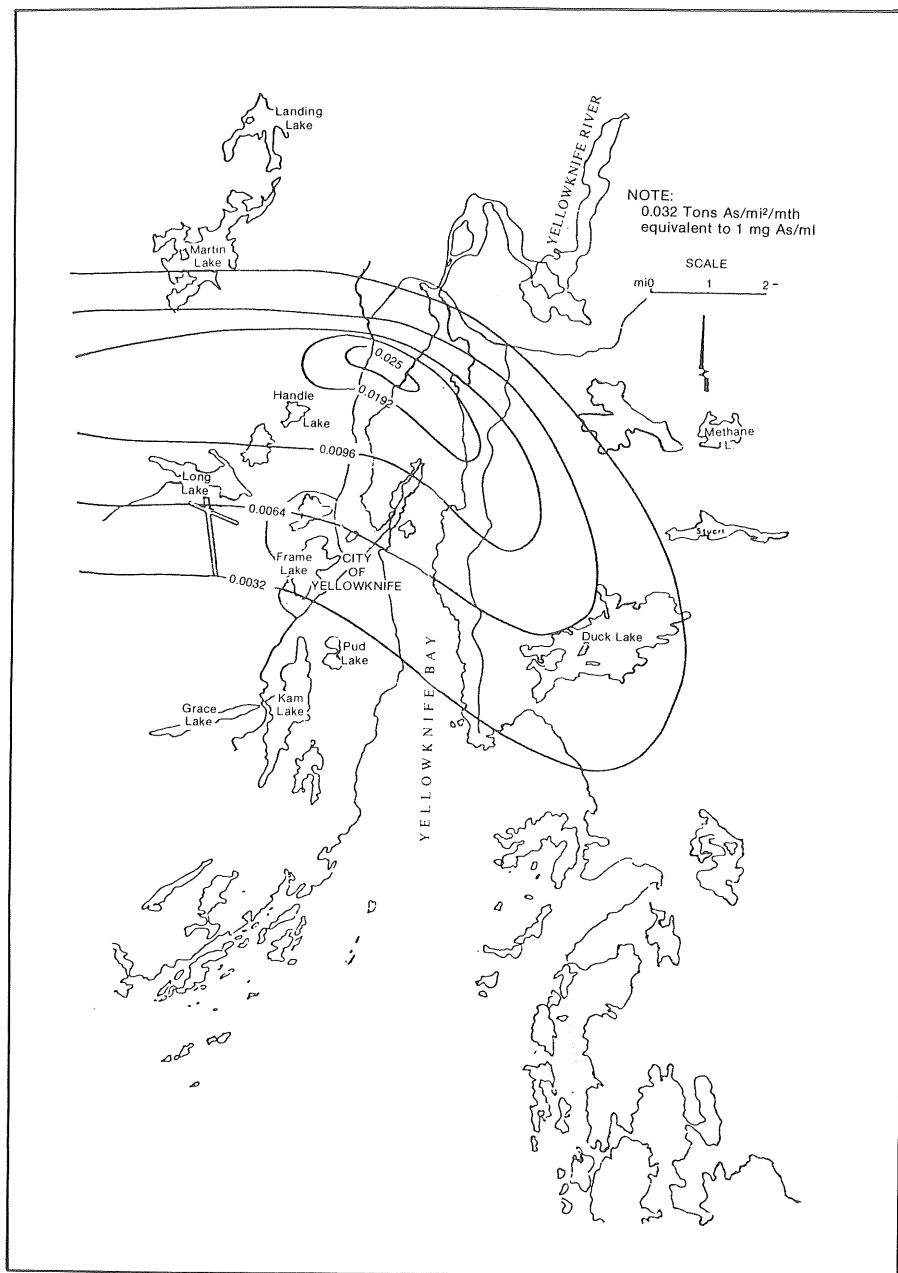
was the west side of Yellowknife Bay on Great Slave Lake. Available data during the period 1951-1969 indicate that the average concentration of arsenic in drinking water for the period (0.048 mg/l) was very close to the maximum permissible level, based upon the *Canadian Drinking Water Standards* (6). However, there was a sizable variation in arsenic concentration by month, the highest concentrations being recorded during the spring run-off period of May, June, and July of each year. The average concentration of arsenic in drinking water for the month of June for the period 1951-69 was 0.107 ppm. In summary, the water supply was within acceptable limits during that period less than 16% of the time, and for almost 70% of the time the level of contamination was between 0.01 ppm and 0.05 ppm. Approximately 15% of the time the water supply is estimated to have been above the maximum permissible level of 0.05 ppm (6).

The present source of water supply for the town of Yellowknife was put into service in December 1969. This source of supply is the Yellowknife River north of its confluence with Yellowknife Bay. The existing system also supplies Cominco and the Giant Yellowknife Staff House area. Arsenic concentrations are monitored routinely and have been consistently found to be barely detectable. Short-term introduction of water from Yellowknife Bay into the municipal system, for fire-fighting purposes, would not be sufficient to constitute a health hazard. Routine monitoring for arsenic in drinking water based on the requirements of the *Canadian Drinking Water Standards and Objectives* should, of course, be continued indefinitely, as a normal public health monitoring program.

(ii) Giant Yellowknife Water Supply

This water supply is primarily a source for process water for Giant; however, a small portion of the water from this source has been used, up until this year, for drinking and washing purposes, by employees at the mine and smelter. The water supply source is from Back Bay, not far from the Baker Creek outlet. Water is chlorinated prior to use and is monitored routinely for arsenic and coliform bacteria.

Back Bay is the depository for sewage effluent from the town of Yellowknife and for the Baker Creek discharge from the Giant tailings ponds. In addition, it has been suggested that domestic sewage from the Giant plant has overflowed into Baker Creek during power failures. The only treatment provided for this supply is basic chlorination. Monitoring for other heavy metals likely to be present in Back Bay has not been conducted nor has any total chemical quality assessment been made, as required by the *Canadian Drinking Water Standards*. Studies (19) have shown that heavy metals and arsenic have spread well into the Bay from Baker Creek. Sediments contain an average of 1,320 ppm arsenic at a distance of 500 yards from Baker Creek and range up to 2,400 ppm. The levels remain high in Back Bay and an average concentration of 130 ppm has been

recorded at a distance of 3 miles from the mine. Bottom organisms have been killed in a large part of the Bay, with a zone of influence extending about 1.5 miles into Back Bay and 0.7 miles out from the mine. The same study (19) has shown that metal levels in the water in Back Bay are unusually high with arsenic sometimes exceeding 1 ppm at 0.5 miles from the mine.

Monitoring of this water supply by Giant Yellowknife mines has shown that average monthly arsenic concentrations have consistently exceeded the maximum acceptable level, with an average concentration for 1976 of 0.03 ppm. Individual monthly maximums have been as high as 0.229 during 1976 and 0.32 during 1975. Although bacteriological monitoring results have consistently indicated the absence of coliforms, there is serious cause for concern. Arsenic concentrations have been variable and approaching maximum permissible levels. The potential hazards associated with the source itself would dictate the rejection of this water supply for purposes of human consumption.

While it is understood that action has already begun in this respect, the Task Force recommends that the use of the Giant Yellowknife water supply as a source of potable water for plant employees be discontinued as soon as possible. The municipal system should be extended to provide employees with water for drinking and washing purposes.

(iii) Cominco Water Supply

Available data on water quality for the Cominco water supply from 1951 to 1969 indicate water quality very similar to that of the town of Yellowknife during the same period (6). The Con Mine is currently supplied by the town of Yellowknife water supply system, and as a result, water quality in terms of arsenic concentration is well within acceptable limits.

(iv) Snow Melt

As we point out above, the use of snow as a source of drinking water could constitute a serious health hazard. It is important, therefore, that every effort be made to ensure that melted snow is not used as a potable water source in the Yellowknife area.

It is probable that some members of the Indian communities in Latham Island and Detah continue to make use of snow as a potable water source, in spite of the fact that water is routinely made available to the community by tank-truck. In order to deal with this potential problem steps should be taken to: (a) ensure that every member of each community at risk is routinely advised and reminded of the hazard of using melted snow for drinking and cooking purposes and (b) ensure that adequate quantities of potable water are made available to the residents of unserviced communities. The provision of public water supplies is a function of local government. The Task Force considers that both Giant Yellowknife and Cominco have a responsibility financially to support such a program.

(e) *Surface Waters*

Arsenic levels in natural surface waters are usually low, ranging from less than 1 ppb to less than 100 ppb†. Nearly 80% of the U.S. waters contain less than 10 ppb. Much higher concentrations ranging upwards from 1 ppm are not infrequently encountered in hot springs and groundwaters in areas of thermal activity, and in well waters and streams draining areas of industrial activity (10). The natural levels of dissolved arsenic in Canadian rivers measured during 1968-74 appear to be low. In most cases, arsenic was undetected by the methods employed which had a sensitivity which varied from 5-13 micrograms per litre. Lakes Superior, Ontario, Huron, and Erie have been found to have levels ranging from 0.25 to approximately 1 microgram per litre (5).

There is a wide variation in arsenic levels in lakes and other surface waters in the Yellowknife area. Most are well within the limits of the *Canadian Drinking Water Standards*. However, some lakes (such as Long Lake, Fault Lake, and Range Lake) have measured levels of arsenic several times the maximum acceptable level recommended for drinking purposes. Measurements taken in February 1975 indicated a concentration of 1.29 ppm in Range Lake, 0.27 in Fault Lake, and 0.135 in Long Lake (8). The arsenic in these cases seems to come from natural sources.

Arsenic levels in Yellowknife Bay are relatively low and generally well within standards (less than 0.005 - 0.02 ppm). Kam Lake has measured levels of arsenic in the 2-3 ppm range (8), apparently due to seepage from the Cominco tailings ponds. Surface water and sea waters are believed to be self-cleaning with respect to arsenic, the element being removed from solution and deposited in the sediments. The latter invariably contain higher concentrations of arsenic than do the waters above. However it is clear that any future use of lakes in the Yellowknife area as possible water-supply sources should be approached with caution. In any such case, arsenic monitoring should be conducted over a period of several months to ensure the acceptability of the water for human consumption.

(f) *Fish and Shellfish*

It has been suggested by some that fish might be an important source of arsenic exposure, since certain species could be subject to exposure to high levels of arsenic in lake water. The allowable limit for arsenic in fish protein, as established by the Department of National Health and Welfare, is 3.5 ppm.

Arsenic in edible parts of fish and fishery products in Canada has been found to vary from an average of 0.25-40.2 ppm for sea-water species and from 0.01-0.62 ppm for freshwater species. It is chiefly the fish living near or at the bottom of the sea (ground fish) which tend to accumulate high amounts of arsenic (flounder, sole, skate, lobster, shrimp). No significant accumulation was

†Parts per billion or micrograms per litre.

observed in samples taken in fresh waters (5). Marine animals seem to possess the potential to deal with arsenic, which is moderately toxic in the inorganic state, by converting it to an organic derivative that is biologically and chemically stable, and probably non-toxic (20). Work published by the Fisheries Research Board of Canada indicates that concentrations of arsenic are not magnified in food chains, at least as a general phenomenon.

It is likely that the physical transport of arsenic in the dissolved state is unrelated to its transport in the solid state, so that particulate arsenic-containing material would be expected to remain within a relatively restricted area around an outfall, while dissolved arsenic should remain in solution until thoroughly dispersed. In this respect, it is quite unlike mercury, for example, the water sediment interactions of which are highly complex (20). The evidence is that toxic levels of inorganic arsenic are not present in marine foodstuffs, even in those containing very high levels of total arsenic (21).

Available data on arsenic concentrations in a variety of fish types sampled in 1973 in the Yellowknife area indicated very low levels of arsenic, well within acceptable limits (8). The highest concentrations were found in fish taken from Kam Lake (2-3 ppm), while most samples indicated concentrations well below 1 ppm wet weight. Further assessment of arsenic levels in the Yellowknife area was carried out during the summer of 1977, as recommended in the Interim Task Force Report. The results of this sampling program confirm that earlier results were representative: the readings were very low and well within acceptable limits (Appendix A).

Sources of Arsenic

The bulk of arsenic contamination in the Yellowknife area is due to considerable emissions from both Giant Yellowknife and Cominco in the past. But all of the more recent environmental studies dealing with air quality, arsenic deposition, snow, soil, and vegetation pin-point precisely and unequivocally the Giant Smelter as the major source of current environmental arsenic contamination in the area. In addition, Cominco arsenic ponds constitute a source of possible fugitive emissions at present, as well as an additional serious hazard for the future. Liquid effluents from both Giant Yellowknife and Cominco are also sources of environmental contamination in a more localized sense.

(a) *Atmospheric Emissions: Giant Yellowknife*

Giant Yellowknife Mines Limited has operated a gold mine and smelter in the Yellowknife area since 1948. In January 1949 an Allis Chalmers "Edwards" type hearth roaster was installed and began operation. The dusts from this roaster contained very high arsenic levels and as a result, a cold electrostatic precipitator was installed in October 1951 to remove the dusts from the exhaust gases. Since that time, the collected dust has been stored in special stopes underground. A

second roaster was installed, followed by the construction in 1953 of a 9' diameter, 150' high stack. A hot electrostatic precipitator was installed in 1955 to reduce the load on the cold collector. However this change was only partially successful and a Dracco baghouse gas filter was installed in November 1958 to replace the cold electrostatic precipitator. At the same time, a new larger fluo-solids roaster was installed to replace the two previously operated roasters. In 1962 the cold electrostatic precipitator was converted to a hot mode of operation to supplement the existing hot collector. Historically, the most significant step in pollution control was the installation in 1958 of the baghouse gas filter (22). It is estimated that during the early years of operation, 16,000 pounds of arsenic trioxide per day were released directly into the atmosphere from the Giant stack (6). During the period 1954-1958, approximately 7,000 pounds of arsenic per day on average, were emitted from the Giant Stack (11).

After the installation of the baghouse in 1958, emission rates dropped significantly, with an average of 400 pounds of arsenic per day being emitted between 1958 and 1970 (11). Raised emission rates have been experienced since that time, with a high of 1,930 pounds per day experienced in 1971 and close to 900 pounds per day in both 1972 and 1973 (Table III). It is clear that the improved emission rates experienced during the 1960's have not been maintained during the 1970's.

Available data on emission rates are not consistent, varying from an average emission rate calculated by the Environmental Protection Service at 167 pounds per day (23), to rates of the order of 500 pounds per day quoted by the company. These differences may well be due to variations in measurement techniques, or to production variations during the test period. In any case, it is clear that further reductions in emission rates are required and can be achieved.

At present, a Task Force established by Environment Canada, and with the Federal and Provincial Governments and the Gold Processing Industry represented, is reviewing the technical aspects of gold-roasting operations with respect to "best available control technology", and the emission limits achievable by the use of such control technology. The work of the Gold Roasting Task Force should be completed this fall, after which formal regulations will be developed under the Clean Air Act (14). It is anticipated that these formal regulations will probably be announced by the summer of 1978 with a specified effective date during 1979. Emission limits have not yet been set, but will be specified in the regulations.

Best available control technology for roasters treating high arsenic sulphide concentrates consists of hot-gas cleaning, followed by the addition of outside cooling air to condense the arsenic, and capture of the condensed fume in cold baghouses. The baghouse operating temperature is a critical factor influencing the efficiency of arsenic collection, as the concentration of gaseous arsenic trioxide increases dramatically with increased temperature. The application of

TABLE III
Stack Emission Summary Giant Mine

Year	Arsenic Emissions (lbs/day)	No. of Data Submissions	Dust Recovery Efficiency	References
1949	(16,000)	–	–	c
1950	(16,000)	–	–	c
1951	(16,000)	–	–	c
1952	–	–	–	–
1953	–	–	–	–
1954	12,000	–	41.8	b
1955	6,400	4	66.5	b
1956	6,000	11	54.6	b
1957	6,500	10	61.6	b
1958	(3,300)	–	–	c
1959	115	7	99.6	b
1960	165	9	99.4	b
1961	(330)	–	–	c
1962	(330)	–	–	c
1963	(330)	–	–	c
1964	690*	4	98.2*	a
1965	–	–	–	–
1966	535*	3	98.3*	a
1967	285*	4	99.0*	a
1968	500*	2	98.8*	a
1969	660*	3	97.7*	a
1970	485	3	98.3	a
1971	1,930	8	93.1	a
1972	875	4	96.5	a
1973	890	5	95.1	a
1974	485	5	98.1	a

Source: (11)
*Corrected from previous data
() Estimated
References: a. Company emission reports.
b. Company emission data as given to Health and Welfare Canada.
c. Estimated values from company production data.

this cold baghouse technology to the Giant Yellowknife case could be expected to reduce emissions to approximately 25-30 pounds per day of arsenic. This, of course, would be a significant improvement over the emission rates currently being experienced at the Giant smelter.

The application of new emission standards and abatement regulations of the type outlined above could be attained by a number of mechanisms: (a) voluntarily; (b) by use of the "Ordinance to Provide for the Protection of the Environment of the Northwest Territories"; and (c) by use of regulations under the Clean Air Act (Federal Department of the Environment).

The Task Force recommends that Giant Yellowknife take immediate steps to commence application of "best available technology" requirements, in advance of mandatory application of such requirements.

Frequency and quality of technique of stack monitoring currently being conducted by Giant Yellowknife is inadequate. The Department of Environment is developing a reference stack sampling method for arsenic emissions. This reference method was to be in draft form by the fall of 1977 (14). It is understood that representatives of Giant Yellowknife are on the committee preparing the draft reference method, and will therefore be aware of the necessary elements of such a procedure. Periodic stack testing will be a necessary requirement of these future emission standards. We recognize that practical limitations exist at Giant with respect to the conducting of this stack sampling regularly. In particular, winter conditions present major impediments. It is therefore important that (a) stack-sampling frequency during summer conditions be sufficient to provide an accurate indication of actual emission rates throughout the year, and (b) operational parameters be correlated with stack emission rates so that reasonable estimates of winter emissions can be obtained, as a basis for future monitoring.

We recommend that Giant Yellowknife take steps to obtain appropriate stack-testing equipment as soon as possible and that baseline stack monitoring be conducted.

(b) *Atmospheric Emissions: Cominco*

The Con mill began operation in July 1938, and by late 1940 the arsenic content of the ore had risen to the point where roasting was essential to separate the gold. The roasting process was started in April 1942, but was interrupted from August 1943 until July 1946 due to wartime restrictions. The roaster resumed production in July 1948 and operated until November 1970. In August 1949 a Doyle impingement-type scrubber was installed in the roaster section to recover the arsenic before it was discharged to the atmosphere. The efficiency of this unit was approximately 93%, with removed material being mixed with water and being pumped to storage ponds (24).

Since the roasting operation was closed in 1970, a cyanidation process has been used to recover the gold from the ore. This process involves wet fine grinding of the crushed ore, the addition of fresh lime and sodium cyanide to dissolve the gold from the ore, and the use of thickeners, agitators, and filters to separate the gold-bearing solutions from the ground waste powder. The gold-

Figure 7.
Arsenic Data Summary, Final Liquid Effluent, Northwest Territories Mines

58
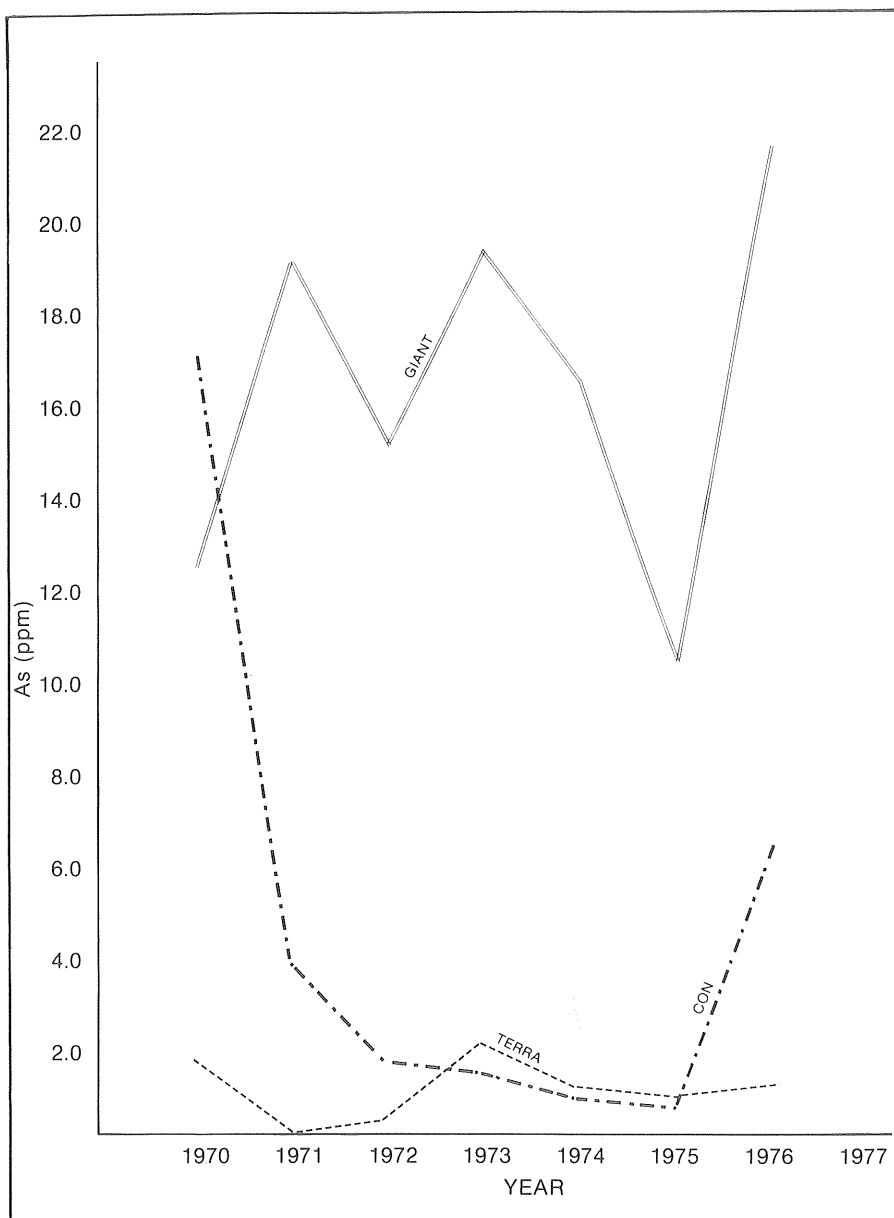
bearing solution is then treated under vacuum with lead acetate and zinc dust to recover the gold in a filter. The precipitate is refined by melting with fluxes and casting impure molten gold into bullion bars (24). As a result of this process, there have been no stack emissions from the Con operation since 1970. However it should be noted that particulate emissions can still occur from operations of this type. Possible particulate emission sources include mine ventilation air, ore transport, crushing, screening, fine ore conveying, and gold bullion casting. In addition, fugitive dust from dry tailings and arsenic storage ponds can present a problem, as we note below.

A summary of stack emissions for the Con mine during the period 1950-1970 is shown in Table IV. Emission rates during that period seem to have been reasonably consistent, varying between approximately 300 and 500 pounds per day.

(c) *Liquid Effluents: Giant Yellowknife*

Liquid effluents from Giant Yellowknife are currently pumped to a tailings impoundment area, Bow Lake, from where by use of a decantation system effluent discharges to Baker Creek, and subsequently to Back Bay. Before discharge to the impoundment area, arsenic-bearing effluents are agitated with lime to precipitate soluble arsenic out of the effluent stream. In spite of this process, arsenic concentrations in effluent entering Baker Creek have been measured at levels up to 31.0 ppm.

Improvement in both the treatment and control of liquid effluents at Giant Yellowknife is required. It was stated in submissions to the Task Force that approximately 80% of the arsenic in the mill effluent is currently being removed by the addition of lime (22). In spite of this, lake-bottom sediments near Yellowknife and within 500 metres of the local mines contain from 400 to 1,300 ppm arsenic (19,25). A summary of arsenic concentrations in final effluent from Giant Yellowknife for the period 1970 to 1976 is shown in Figure 7.

In addition to the problem of effluent quality, incidents of seepage from the tailings pond into adjacent lakes have been experienced in recent years. Three major seepages occured in 1974 (26). It was determined at that time that the concentrations of contaminants in the seepage were of the same order of magnitude as those measured during 1972 and 1973 at the outfall of the third tailings pond to Baker Creek, and generally exceeded concentrations of arsenic, copper, and cyanide in the effluent decanted to Baker Creek. Excursions of this type are unacceptable. It is essential that close attention be directed to the prevention of seepage from tailings ponds in future. The use of back-up catch-basins is a useful safety valve. However it is essential that routine surveillance and maintenance programs be conducted to ensure their adequacy.

Effluent discharges to Baker Creek over the years have resulted in a high level of contamination in Back Bay. Sediments contain an average of 1,320 ppm

TABLE IV.
Stack Emission Summary Con Mine

Year	Arsenic Emissions (lbs/day)	Dust Recovery Efficiency	Reference
1949	—	—	—
1950	(200)	(95)	c
1951	(200)	(95)	c
1952	(200)	(95)	c
1953	—	—	—
1954	395	97.8	b
1955	420	98.3	b
1956	410	97.9	b
1957	400	—	b
1958	385	—	b
1959	435	97.8	b
1960	585	97.3	b
1961	(440)	—	c
1962	(440)	—	c
1963	(440)	—	c
1964	295	—	b
1965	370	95.1	b
1966	310	—	b
1967	340	85.2	b
1968	335	—	b
1969	430	—	b
1970	550	86.2	b
1971	Roaster ceased 10/11/70		

References: a. Company emission reports.
b. Company emission data as given to Health and Welfare Canada.
c. Estimated values from company production data.
Source: (11).

arsenic at a distance of 500 yards from Baker Creek, and range up to 2,400 ppm (19). The levels remain high in Back Bay and an average concentration of 130 ppm was recorded at a distance of 3 miles from the mine (19). Metal levels in the water of Back Bay are high, with arsenic sometimes exceeding 1 ppm at 0.5 miles from the mine, with the zone of influence of contaminated sediments extending about 1.5 miles into Back Bay and 0.7 miles from the mine (Figure 8) (19).

The Federal Department of the Environment has not yet established liquid effluent standards for gold-mining operations. The Department is however

Figure 8.
Zone of Influence, Contaminated Sediments, Back Bay, Northwest Territories

= Zone of influence

Site of earlier
tailings disposal

Baker
Creek

Giant
Mine

D

A

B

Back
Bay

City of
Yellowknife

Yellowknife
Bay

0.5 Miles

61
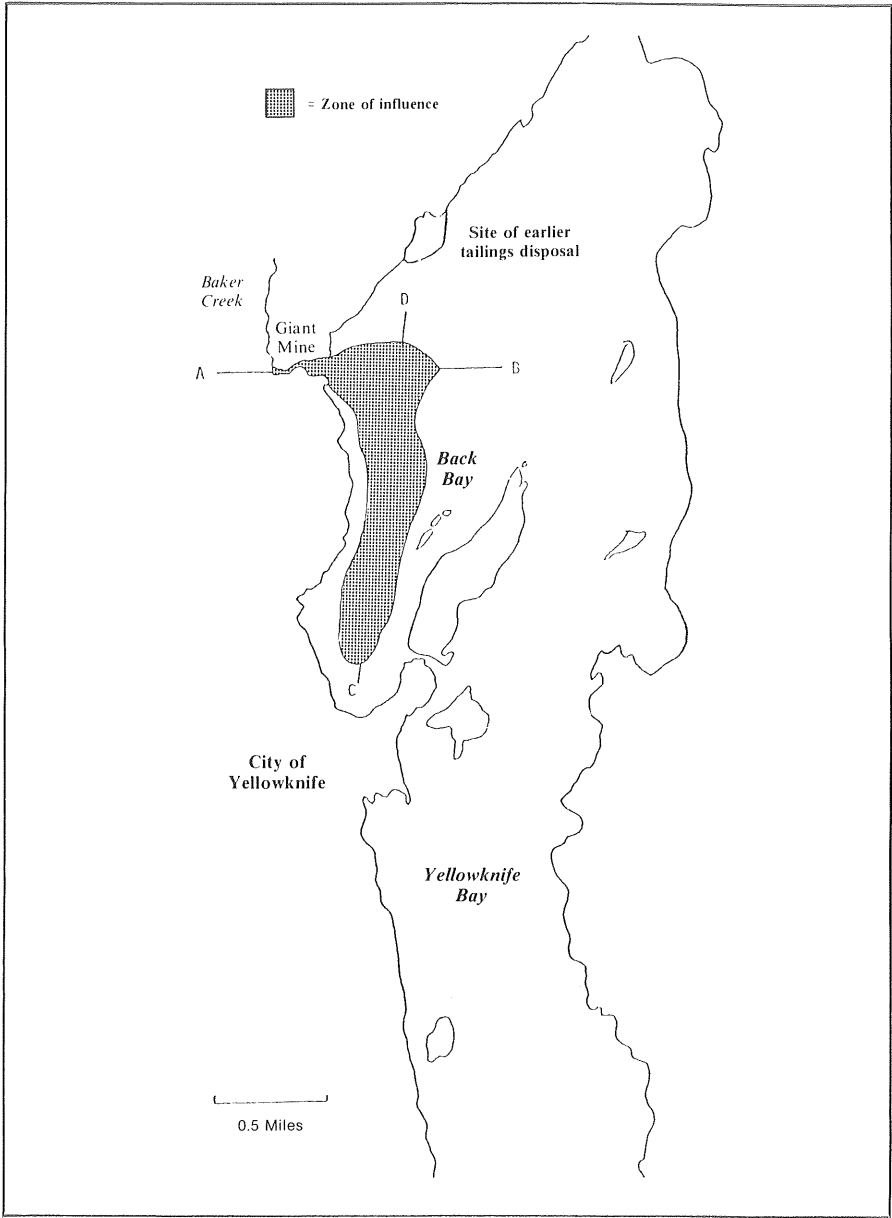
currently working closely with Giant Yellowknife on a research project on pollution abatement technology for arsenic effluents from gold-mining facilities (24). We understand the report on this pilot project, which involves the use of both a chlorine and a ferric sulphate process, will be completed during the fall of 1977. We recommend that the results of this study be made public and that the Northwest Territories Water Board apply effluent treatment requirements similar to those specified in the case of Cominco.

It is recommended that routine monitoring of liquid effluent quality be conducted both at the outlet of the decant structure and at the outlet of Baker Creek, under the direction of the Northwest Territories Water Board.

(d) Liquid Effluents: Cominco

Liquid effluent from the Con mill is deposited into a separate tailings pond, Pud Lake, and the decanted effluent flows by gravity into a series of small lakes and ponds that drain into Great Slave Lake approximately 5 miles south of the mill. Effluent flowing from the tailings pond is stopped during the winter and most of the summer months to allow for more oxidation and the breakdown of cyanide to take place. During the periods of low flow, the effluent is diverted down a ditch constructed from the control structure to Meg Lake to prevent seepage into Kam Lake (24). However seepage into Kam Lake has been experienced in the past. Excursions of this type are unacceptable. It is essential that close attention be directed to the prevention of seepage from tailings ponds in future. It is essential that routine surveillance and maintenance programs be also conducted to ensure that future seepage excursions are prevented.

A summary of final effluent quality from the Con mill for the period 1970 to 1976 is shown in Figure 7. The Northwest Territories Water Board has directed Cominco to implement appropriate effluent treatment provisions by March 1, 1980. The water licence issued by the Board, pursuant to the Northern Inland Waters Act and Regulations (Department of Indian and Northern Affairs), was issued on March 1, 1977, and expires on February 29, 1980. It specifies effluent quality requirements for arsenic, cyanide, and other heavy metals, as discharged through the decant structure at Pud Lake. The maximum monthly average concentration of arsenic has been specified at 0.50 milligrams per litre, to be implemented by March 1, 1980. Steps are currently being taken by Cominco to meet this requirement.

The Task Force supports the requirements for arsenic abatement and monitoring at Con Mine as specified by the Northwest Territories Water Board.

(e) Dry Tailings

Drying of the surface of the tailings ponds, and dust movement due to wind action, can cause arsenic mobilization around the area away from the mines.

Air quality in the Yellowknife area can be affected by contributing sources of this type, and monitoring levels may reflect such contributions.

It has been reported that tailings have been used on a variety of construction projects in the area and are a source of fill. This would contribute to a further distribution of arsenic throughout the Yellowknife environment. Hence we recommend that no further use of tailings for construction or fill be permitted and that access to tailings areas be completely restricted.

The Task Force has noted that one of the requirements of the Cominco water licence issued by the Northwest Territories Water Board specifies "the complete restoration and reclamation or abandonment of existing tailings areas". This requirement is supported by the Task Force.

(f) *Arsenic Storage: Giant Yellowknife*

Arsenic removed during the smelter operation has been stored in underground vaults since 1951. The requirements for underground storage, as specified by the Mining Inspection Branch of the Department of Indian and Northern Affairs include: (a) All openings to be securely sealed to prevent escape of arsenic-bearing dust. All drifts, cross-cuts and raises to be bulk-headed in specified locations; (b) All changes or modifications to be subject to the prior approval of the Department of Indian and Northern Affairs and the Department of National Health and Welfare; (c) Stopes to be dry prior to storage and to be kept dry; (d) Permafrost to be evident in the arsenic storage areas before arsenic storage commences, and these areas to be kept frozen; (e) All air discharged from the storage areas to be filtered through the existing bag filter system prior to discharge to the atmosphere; and (f) Regular inspection of bulkheads and storage operation by the Department of Indian and Northern Affairs.

While arsenic is stored within the permafrost, in areas of competent rock, storage areas are also sealed and secured with concrete bulkheads, constructed to withstand hydrostatic pressure (27). Two types of bulkheads are used:

1. A reinforced concrete slab designed with a compressive strength of concrete greater than 2,000 psi, and with steel reinforcement and concrete thickness designed for bending movement and punching shear. All bulkheads of this type are at least 3' thick.
2. A massive plug of concrete of a compressive strength in excess of 2,000 psi, and designed on the basis of punching shear. In all cases of this type, the thickness of the bulkhead is at least the minimum opening dimension of the stope.

Bulkheads are keyed into rockwalls 24 inches and anchored to walls using pins set in an additional 18-24 inches. Before arsenic storage begins the arsenic chamber is pressure-tested to ensure there are no air leaks (27).

The Task Force has reviewed the design criteria and inspected the arsenic storage areas and is convinced that the methods used and the conditions under

which the arsenic is stored should be sufficient to contain the waste arsenic and prevent any future entry of this waste material into the environment. A review of current practices for arsenic disposal throughout North America clearly demonstrates that the method used by Giant Yellowknife is the safest and most acceptable approach to the problem (10). We recommend that the underground vault method of arsenic storage continue to be used during the operation of the Giant smelter.

While it is recognized that development of future underground storage areas is subject to the assessment and approval of the Mining Inspection Branch, we also recommend that the Department of the Environment be kept informed of all new plans for underground storage of arsenic.

Concern has been expressed with respect to the possibility of flooding of mine workings following closure of Giant Yellowknife at some time in the future. We recommend that, at such time as the Giant operation is discontinued, the company take all necessary steps to seal off all points of entry of surface water and runoff into the mine. In addition, we recommend that provision be made for monitoring water levels within the mine following closure, and that the Department of Indian and Northern Affairs (Mining Inspection Branch) be responsible for routinely monitoring variation in groundwater level in the mine.

Currently, Giant Yellowknife Mines Limited is developing open pit operations in the vicinity of the underground arsenic disposal areas. Current separation distances would appear to be adequate. However, we recommend that the company take appropriate steps to ensure that drainage from open pit workings cannot enter the arsenic storage area and that, through the Department of Indian and Northern Affairs, the Department of the Environment be kept advised of these activities.

(g) *Arsenic Storage: Cominco*

Arsenic removed during Cominco's roasting operation, until this ended in 1970, was placed in two large surface storage ponds on the Cominco property. It is estimated that approximately 50,000 tons of arsenic are contained within the two storage ponds.

A requirement of the recent licence issued by the Northwest Territories Water Board specifies complete containment and reclamation by 1980 of all arsenic oxide storage areas located on the Cominco property. We support the directive of the Northwest Territories Water Board and stress the need to ensure complete containment of the arsenic in question, either by provision of satisfactory containment structures at the site, or by removal of the arsenic to underground storage, based upon requirements comparable to those applied at Giant Yellowknife.

III. Occupational Exposure to Arsenic in Yellowknife

Health Effects of Arsenic

The potential for human exposure to arsenic, both at work and in the community, has existed at Yellowknife since 1938 when gold mining first began. A number of studies to assess the possible effects of this exposure on workmen and residents have been made, and are reviewed in Chapter IV of this report. No special announcement was made to the public at the time the earlier studies were completed. This fact has been responsible, at least in part, for the assumption by some segments of the media and the public that information was deliberately withheld in an attempt to minimize the seriousness of the problem.

The Task Force has found that there was a failure to communicate the findings of the earlier studies to concerned groups and individual members of the public. The reasons behind this lack of communication have been the subject of speculation by members of the media, including charges of cover-up by the federal government and bureaucratic incompetence. The controversy has been further fuelled by an apparent disparity in the data reported in, and the conclusions drawn from, the government studies and those resulting from a study done jointly by the United Steelworkers of America and the National Indian Brotherhood. It is also possible, of course, that another factor in the early failure to communicate the findings of the government studies to the public was the desire not to alarm the public if, in fact, no threat to community health was definitely shown to exist. It should be remembered that the data collected in the early studies (1951 and 1965) could only be interpreted in the light of the published literature existing at the time of those studies.

There has been a considerable increase in the information published on arsenic since 1965. Much of this information is pertinent to the intelligent interpretation of the Yellowknife data, and it is in the context of this most recent information that the problem of present and future exposure to arsenic at Yellowknife must be assessed.

The task is neither easy nor straightforward. The literature is extensive. On the other hand, there still exist many gaps in our knowledge. Arsenic in the environment exists in a number of forms or "species", with variation in toxicity between different species. The effects of chronic exposure may not become apparent for many years. There is a dearth of information on dose-response relationships in humans and animals for various species of arsenic, particularly where chronic exposure is involved. The presence of concomitant exposure to other chemicals, as is the case in the majority of reports on human exposure to arsenic, often precludes the possibility of drawing definite conclusions. In particular, the relationship between dose or dose-rate and possible carcinogenic, mutagenic and teratogenic effects is the subject of deep debate among scientists.

The Task Force has considered it a primary responsibility to review the literature on the health effects of arsenic, particularly those reports which deal with its effects on humans who have sustained exposure to environmental

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sources, either at work or in the community. Brief reference is made to studies on experimental animals, but no attempt is made to deal with this part of the literature in detail. Where possible, throughout this report we have attempted to relate the responses described in the various studies to the levels of exposure sustained by the human or animal subjects, in the hope that this will assist in putting the past and present exposure to arsenic at Yellowknife into reasonable perspective.

Much of the environmental data, particularly those describing arsenic levels in air, water, soil and fish, are reviewed in Chapter II. However, in order to more readily relate such data to health effects, we have intentionally repeated some of this information in this chapter.

A number of comprehensive reviews of the literature dealing with the biological and toxicological effects of arsenic have been published in recent years, namely:

- A review paper by B.L. Vallee, D.D. Ulmer and W.E.C. Wacker in 1960 (28).

- A section on arsenic in the textbook *Toxicity of Industrial Metals* by E. Browning in 1961 (29).

- A monograph edited by W.D. Buchanan in 1962 (30).

- A review paper by D.V. Frost in 1967 (31).

- The proceedings of a symposium edited by B.W. Carnow in 1976 (32).

- A criteria document published by the U.S. National Institute for Occupational Safety and Health (NIOSH) in 1975 (33).

- A monograph on the medical and biological effects of environmental arsenic published by the U.S. National Academy of Sciences (NAS) in 1977 (10).

- The proceedings of an international conference on arsenic, B.A. Fowler, Chairman, published in *Environmental Health Perspectives* in August, 1977 (34).

Attention is also drawn to a Report of the Task Force on Arsenic in Ambient Air, prepared by the Environmental Health Directorate, Health and Welfare Canada (P. Toft, Chairman) in July 1975 (5). This review contains references to a number of European papers not listed in the otherwise more comprehensive publications by NIOSH and NAS.

Environmental Exposure

The toxic effects of arsenic compounds have been known for many centuries, as evidenced by their use as poisons from very early times. Prior to the discovery of the sulphonamides and antibiotics, arsenical compounds were also used for

several centuries in the treatment of disease, e.g., potassium arsenite in Fowler's solution (7.6 mg arsenic per ml administered in doses of 0.1 to 0.5 ml thrice daily) and organic arsenicals such as the arsphenamines and arsenoxides used in the treatment of syphilis and trypanosomiasis. While the use of arsenic in medicine has declined greatly since the 1930's, it is still used therapeutically to some extent. It is also used as dietary supplement (in the form of arsanilic acid, carbarsone and 3-nitro-4-hydroxyphenylarsonic acid) for disease control and for improving weight gain in cattle.

(a) *Community Exposure*

Arsenic is ubiquitous in nature. Extensive information on "background" levels in the environment is given in the National Academy of Sciences monograph (10). As mentioned previously, a summary of the levels commonly found in air, soil, water, fish and vegetation is included in Chapter II of this report, together with a consideration of the arsenic levels found at Yellowknife.

Aside from exposure to arsenic which may be encountered at work, the exposure sustained by community residents is determined largely by the concentrations of arsenic which are present in the air, water and food of the area, and the quantities of each which are utilized in daily living. Other factors influencing the significance of the exposure are the species of arsenic (whether trivalent or pentavalent, whether organic or inorganic) and their solubility in body fluids. The presence of concomitant exposure to other chemicals may also be of importance.

Arsenic in the ambient air exists primarily as particulates of arsenic trioxide and arsenates. It is not known which is the most prevalent form of arsenic found in water. In sea water the inorganic arsenic found appears to be mainly in the form of arsenite. In aerated fresh waters the arsenic is mostly in the form of arsenate, while that in anaerobic reservoirs is present as arsenite (Clement and Faust, 35). Well waters in Argentina have been reported to contain up to 3 ppm of arsenic as trioxide.

Arsenic in soil is of significance as a source of dust, which may be inhaled if the particles are of respirable size, and as a potential source of water contamination. Contaminated soil may also present a source of intake for small children at play, through hand-to-mouth transmission. While contaminated soil has been considered a major source of lead exposure for children, there appears to have been little, if any, assessment of the significance of this route for children in communities where arsenic contamination of the soil has occurred.

Arsenic is present in low concentrations in most foods. Land animals and plants do not accumulate arsenic. On the contrary, they discriminate against it (Bowen, 36). Land plants not treated with arsenical insecticides or sprays or not exposed to arsenic fall-out seldom contain more than 0.5 ppm arsenic on a fresh weight basis (5 ppm dry weight). In plants grown in high arsenic soils, the up-

take of arsenic by the above-ground portions of the plant is low, with most arsenic accumulating in the roots. Onions grown in soil containing 223 ppm arsenic were found to have a maximum concentration of 2.25 ppm arsenic in the foliage (McLean et al, 37). Washed leafy vegetables contaminated by fall-out may have levels of more than 100 ppm dry weight (about 6 ppm wet weight, on the basis that the leaves contained 95% moisture (Griffin, 38). The levels in domestic animals are usually less than 0.3 ppm (fresh weight). Fresh water fish usually contain less than 1 ppm, but levels of up to 3 ppm are found (10), (Sandi, see ref. 5).

Marine plants and animals, on the other hand, tend to concentrate arsenic in their tissues, often to relatively high levels. Concentrations of from 4 to 109 ppm (dry weight) have been found in algae and seaweed, and up to 10 ppm in sea fish. Mollusks and crustaceans may contain up to 50 or 75 ppm arsenic or more (10).

(b) *Occupational Exposure*

During the smelting of arsenical ores, the arsenic sublimes, then condenses as impure arsenic trioxide, As_2O_3 (arsenious oxide, "white arsenic"). This oxide, and the pentoxide, As_2O_5, form the basis for many arsenical pesticides, insecticides, fungicides, herbicides and defoliants. The most important industrial exposures occur in the recovery of copper, lead, zinc, gold and tin from the ores or from concentrates, as well as in the manufacture, packaging and application of arsenical pesticides, etc. Arsenic is also used in glass manufacture and in the production of alloys. A small amount is used in the manufacture of organic arsenical compounds for therapeutic purposes and as a food additive for cattle.

Occupational exposure to arsenic may also occur through the inhalation of the gas arsine, AsH_3. This very toxic gas is formed by the action of an acid on arsenic-containing metal or when hydrogen is evolved, as by hydrolysis, in the presence of arsenic.

Intake and Absorption

Arsenic may enter the body by ingestion, inhalation, or by absorption through the skin.

There have been no controlled balance studies in humans on the absorption of arsenic compounds into the body and their subsequent excretion, as have been done for lead. There has been considerable research using animals, but much of this has involved the use of rats, a species in which the metabolism of arsenic is quite different from that of other species. The rat concentrates up to 80% or 90% of administered arsenic in the haemoglobin of the red blood cells, from which it is released only when the cells rupture and disintegrate (10).

(a) *Digestive System*

In persons not occupationally exposed to arsenic, the total daily intake by

ingestion appears to be low, though the data reported show fairly wide variation. Schroeder and Balassa (39) in the United States found a daily intake of 400 μg in a sample of an institutional diet in 1966. More recently, Jelinek and Corneliussen (40) reported that the average daily intake of arsenic in the U.S. has dropped from about 130 μg in 1968 to about 20 μg in 1974. The daily dietary intake in Great Britain has been estimated at about 100 μg (Hamilton et al, quoted in ref. 10). Sandi (see ref. 5) estimates the average intake of arsenic from Canadian diets to be less than 100 μg per day. On the other hand, a Joint FAO-WHO expert committee in 1967 estimated that most normal daily diets probably supply 1.5 to 2.0 mg arsenic per day, and recommended that the maximum acceptable load of arsenic be tentatively placed at 0.05 mg/kg of body weight per day (i.e., 3.5 mg per day for a 70 kg man (41). From recent epidemiological studies, it appears that this figure is open to question.

Though the data regarding daily arsenic intake in food show considerable variation, it would appear fairly reasonable to assume an intake of about 100 μg arsenic per day in food.

The average daily intake of water is of the order of 1.5 litres per day. If the water contains 10 μg arsenic per litre, the daily intake of arsenic from this source would be 15 μg. Arsenic ingested in water is thus, under ordinary circumstances, a relatively small fraction of the total dietary intake.

Absorption of arsenic from the digestive tract is dependent upon a number of factors, including the chemical form of the compound, its purity, its valence, its solubility and its fineness or coarseness. Harrison et al (42), using rats, found that the toxic dose of arsenic trioxide in the dry state was ten times that in solution. Schwartze (quoted in ref. 10) also found that a solution of arsenic trioxide was more toxic than the dry form and that the toxicity of solid arsenic trioxide varied markedly depending upon the physical coarseness of the preparations administered.

Most of the arsenic in marine fish and in shrimp and other crustaceans has been shown to be in an organic form which is more readily absorbed from the intestines and excreted by humans than are the inorganic forms of arsenic.

It does not appear possible, in our present state of knowledge, to state rates of absorption from the human digestive tract for different forms of arsenic.

(b) *Respiratory System*

The daily intake of arsenic in air would appear to be negligible. The volume of air breathed by a man doing light work is estimated to be 23 m³ per day (43, page 51). Assuming an average concentration of 0.02 μg/m³ in the ambient air, this would result in a daily intake by air of about 0.5 μg.

A minor contribution to arsenic intake occurs in cigarette smoking. During the 1940s and 1950s British and American cigarettes contained approximately 20 to 50 μg arsenic/g (or per cigarette) as the result of the use of arsenical sprays

(Buechley, 44). Daff and Kennaway (45) in Great Britain found concentrations mainly in the 40 to 60 $\mu g/g$ range, and estimated that from 7% to 18% was volatilized during smoking. Assuming an average of 40 μg per cigarette, and that 12% is volatilized, the quantity of arsenic inhaled would be about 5 μg per cigarette, or 100 μg per pack of 20 cigarettes. The amount retained in the lungs would be less than this. Holland et al (46) found that, among 8 human volunteers, the "uptake" of radioactive arsenic which had been added to cigarettes was from 2.2% to 8.6% of the total added. From the text, the word "uptake" is used to mean deposited in the respiratory tract. If an average deposition of 5% is assumed, the smoking of 20 cigarettes containing 40 $\mu g/$cigarette would result in the deposition of 40 μg arsenic.

Following the discontinuance of arsenical sprays on American tobacco crops in the early 1950's, the arsenic content in cured leaf tobacco has been reported to have fallen to around 1 to 3 ppm ($\mu g/g$) (10). The contribution made by cigarette smoking to daily arsenic intake would thus appear to have diminished greatly in the past 20 years.

In view of the very small amount of arsenic which is inhaled daily in the ambient air (0.5 $\mu g/$day estimated), a detailed consideration of the factors influencing absorption from the respiratory tract might appear unnecessary. The subject becomes important, however, when one realizes that men at work may be exposed to concentrations of airborne arsenic dusts measuring hundreds of $\mu g/m^3$ for 8 hours a day.

With respect to the absorption of inhaled arsenic it is necessary to have some knowledge of the particle size distribution of the aerosol, and its specific gravity, as well as its concentration in the air, the amount of air breathed daily, the depth of respiration, etc.

Particles larger than 10 or 12 μ diam. are completely retained in the nose, or in the upper bronchi if the subject is breathing through the mouth. Particles between 3 μ and 10 μ are practically all deposited and retained in the bronchi and larger bronchioles. Particles less than 3 μ diam. may reach the smaller bronchioles and the alveoli, where maximal deposition (some 60% to 80%) occurs for particles of from 0.8 μ to 1.6 μ diam. For particles in the range of 0.2 to 0.3 μ, only about 20% are deposited, the remainder being breathed out during exhalation. Below 0.2 μ diam. deposition in the upper bronchi and trachea again predominates. Particles of ion size are completely deposited on the walls of the trachea by the effect of Brownian movement and diffusion (Green and Lane, 47; Davies, 48; Pavia and Thomson, 49). A graph of aerosol deposition curves for the three major regions of the respiratory tract, prepared by the International Commission for Radiological protection (ICRP), is shown in (50, 51).

Insoluble particles which are deposited in the nose and pharynx and in the tracheo-bronchial tree above the level of the finer (respiratory) bronchioles are removed in the mucus by the action of cilia which sweep the mucus toward the

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pharynx where it is coughed up and swallowed or spat out. In a study of the clearance of radioactive iron particles from the human lung in 1955, Albert and Arnett (52) noted that the clearance pattern appeared to have two phases, the first phase lasting 2 to 4 hours, the second about 30 hours. In two of their subjects they showed that as the radioactivity over the chest fell, it increased over the abdominal region, supporting the hypothesis that dust brought up in the mucus from the tracheo-bronchial region was, in fact, swallowed. The ICRP lung model (50) gives a biological half-time for the clearance of particles from the naso-pharynx of 4 minutes, and from the tracheo-bronchial region of 10 minutes. In the pulmonary compartment, from the respiratory bronchioles to the alveoli, clearance is of two types. Some particles are carried upwards by dust cells to the ciliated region; the biological half-time for these particles is approximately 24 hours. Other particles penetrate the alveolar wall and enter the tissues; the half-time for these particles may vary from a few days to several years. For arsenic trioxide (and probably other arsenic compounds of low solubility) the clearance half-time from the pulmonary compartment, as reported by the Task Group on Lung Dynamics of the ICRP, is 16 days (50).

Holland et al (53) studied the clearance of arsenic from the human respiratory tract and reported that, in 8 terminal lung cancer patients who inhaled smoke from cigarette tagged with arsenic-74, and in 3 others who received it in the form of an aerosol, the radioactive arsenic disappeared very rapidly from the chest during the first few days, so that only 20% to 30% of the original intake remained by the 4th day. Thereafter the rate of clearance tapered off slowly.

There is a need for specific information respecting the clearance of various arsenic compounds from the lung if one is to estimate with any degree of accuracy the dose which is absorbed into the body as the result of intake via the respiratory tract. Part of the inhaled arsenic, particularly the particles above 3 μ to 5 μ in diameter will be cleared from the naso-pharynx and the tracheo-bronchial region and will be swallowed. Particles retained in the alveoli will be dissolved and enter the blood stream, or will be carried by dust cells through the alveolar walls where they will be more slowly dissolved and enter the blood. The particles which have been swallowed will, in the more soluble compounds, be absorbed via the gastro-intestinal tract. Particles of low solubility and larger size may not be retained in the gastro-intestinal tract long enough to be absorbed.

(c) *Percutaneous*

Absorption of arsenic through the skin has been proven in rats bathed in mineral water containing various concentrations of radio-active arsenic, As[76] (Danilova et al, quoted by Bogoroch in ref. 5). Percutaneous absorption has also been demonstrated in men whose skin was wetted while applying cacodylic acid and monosodium methanearsonate spray (Tarrant el al, 54).

Metabolism, Distribution and Excretion

Though widely distributed in all living tissues, arsenic is not considered to be an essential element for human health. As mentioned previously, it is used as a food additive to promote growth and weight gain in cattle. While dietary deficiency of arsenic has not been recognized in animals on natural diets, Nielson et al (quoted in ref. 10) have recently described deficiency signs in male rats receiving a diet containing less than 30 ppb of arsenic. Deficiency signs have also been reported in goats and minipigs fed a semi-synthetic diet containing less than 50 ppb arsenic (Anke et al, quoted in ref. 10). No deficiency signs developed in control animals receiving the same diet supplemented with 350 ppb of arsenic.

Arsenic is an antagonist of selenium; when added to drinking water in proportions of 5 to 10 ppm, it has been found to protect agricultural animals from selenium toxicity to which they are exposed through forage in seleniferous areas (28). Arsenic has been found to stimulate the excretion of selenium in the bile, and selenium has been shown to exert a similar stimulation of biliary excretion of arsenic (10). Arsenic may also be antagonistic to iodine.

Arsenic which is absorbed through the gastro-intestinal tract passes through the liver before reaching the general circulation. Once in the blood stream, trivalent arsenic is rapidly distributed to all tissues, including the hair and nails; the largest proportion goes to the muscles. Most of the arsenic is lodged in the protein fraction of the tissues, with a small amount in the acid soluble fraction and only a trace in the lipoid fraction. In humans, arsenic does not pass into the spinal fluid. While small amounts are present in cows' milk (0.03 to 0.04 ppm), experiments have shown that neither inorganic nor organic forms of arsenic readily pass the blood-mammary barrier.

Reeves (in ref. 32) lists the following concentrations of arsenic as "completely normal": for blood, 0.1 to 0.5 ppm; for hair, 0.5 to 2 ppm, and for nails, 0.5 to 5.5 ppm. Increased levels in the hair and nails remain for months after exposure has ceased and urinary excretion has returned to normal. In persons with no known occupational or medicinal exposure to arsenic, Roberts et al (9) found average concentrations of 0.68 ppm in hair from rural residents and 0.75 ppm in hair from urban residents, compared with an average of 1.9 ppm in the hair of 122 persons living near two secondary lead smelters in Toronto. The maximum level reported in the rural and urban control groups was 2.1 ppm. A paper by Colucci et al (55) suggests a correlation between the arsenic content of hair and residence in cities ranked in five grades according to degree of arsenic exposure. He found that the mean arsenic content of the hair ranged from 0.4 ppm in the city with the lowest exposure to 10.6 ppm in the city with the greatest exposure. It would be necessary to review the studies quoted by Colucci to determine the routes of arsenic intake on which his findings were based. Of 565 residents of Yellowknife, 49 (8.7%) were reported in 1975 to have hair levels in excess of 5 ppm. In the same study, 74 of 135 mine and mill workers (54.8%) had levels exceeding 5 ppm; in 6

workers the level exceeded 100 ppm (8). To what extent such high levels result from systemic absorption of arsenic and to what extent they reflect contamination by sweat and direct adsorption is not clear. Washing in mild detergent will remove much of the arsenic when it is due to external contamination (Dubois, quoted in ref. 10) whereas Lander et al (also quoted in ref. 10) state that it cannot be removed by the most meticulous washing. Further controlled studies are obviously needed to assess the value of hair levels as an indicator of arsenic absorption.

Pentavalent arsenic is excreted faster than trivalent arsenic (30, 39). Excretion in man of arsenic in the trivalent form appears to be relatively slow. Hunter et al in 1942, reported that excretion of subcutaneously injected radioactive arsenic (as potassium arsenite) was mainly via the kidneys, and was essentially complete after 6 days (quoted in ref. 10). In the report by Holland et al (53) in which lung cancer patients inhaled cigarette smoke or an aerosol tagged with arsenic-74, approximately 28% of the absorbed arsenic was excreted in the urine in the first 24 hours. By the end of ten days, urinary and fecal excretion of the radioactive arsenic was approaching zero.

Mealey et al (quoted in ref. 10) administered arsenic-74 intravenously as a trivalent sodium salt and found that the pattern of excretion was compatible with a 3-compartment model of storage having clearance rates of 25% per hour, 2.5% per hour, and 0.3% per hour. At first the arsenic was excreted mainly in the trivalent form but the proportion excreted as pentavalent arsenic rose steadily until the fourth day when it remained constant at 75%.

Recent research using cows and dogs has shown that more than 50% of both trivalent and pentavalent inorganic arsenic salts were methylated and excreted in the form of less toxic methylarsonates within 7 days of discontinuing oral administration (Lasko and Peoples, 56). It appears that most of the urinary arsenic excreted by man is also methylated. Braman and Foreback (quoted in ref. 10) found approximately 74% of the arsenic excreted by four Florida residents was methylated. Whether this resulted from the ingestion of sea-food or as the result of detoxification of inorganic arsenic was not determined. It is known that ingestion of shrimps and other sea-foods may greatly elevate the level of arsenic in urine (more than tenfold) over a period of 20 hours; after 48 hours values approach normal excretion levels (33).

More recently, Smith et al have reported that most of the arsenic excreted by copper smelter workers exposed to trivalent inorganic arsenic dusts was methylated, the majority of it being in the form of dimethylarsinic acid (114).

In persons not exposed to arsenic occupationally or receiving it thera- peutically, urinary concentrations are reported to be about 0.1 mg/1 (33). Reeves (in ref. 32) gives the normal range as 0.01 to 0.3 ppm (or mg/1). It is probable that urinary arsenic levels do not often exceed 0.2 ppm in the absence

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of increased arsenic intake, such as by ingestion of shellfish or other sea-food or occupational exposure.

Urinary arsenic levels are considered a useful group index of recent absorption in persons occupationally exposed. Nelson (in ref. 32, p. 225) found a significant correlation between urinary arsenic levels and airborne arsenic concentrations, namely:

Y (airborne arsenic in $\mu g/m^3$) = 0.309 X (urinary arsenic in $\mu g/l$)

The urinary arsenic values were corrected for specific gravity of 1.018.

Arsenic is also excreted in the sweat, and is lost from the body in the hair and nails and in desquamation of the skin. Appreciable amounts may be exhaled; in 1944 Satterlee et al reported an average of 110 μg arsenic/m^3 in air exhaled by a group of smokers of both sexes (quoted by Satterlee in ref. 57). Whether this route of excretion would be as important today, when tobacco contains less arsenic, would have to be verified.

Toxicity

When absorbed into the body, trivalent arsenic reacts with the sulphydryl groups (-SH) of proteins and enzymes. Enzymes known to be inhibited by arsenic include glutamic oxaloacetic transaminase, pyruvate oxidase, monamine oxidase, choline oxidase, glucose oxidase and urease (Reeves, in ref. 32). It is to its interference in such a wide spectrum of enzymes that Reeves attributes arsenic's effects on many organ systems.

Of the trivalent inorganic arsenic compounds, the soluble forms such as sodium arsenite are much more toxic than those of low solubility such as arsenic trioxide. Trivalent compounds are in general several times more toxic than pentavalent forms such as arsenates (10). Pentavalent arsenic does not appear to react directly with the active sites of enzymes, and the mechanism by which it produces its toxic effects is not clearly understood.

In general, aliphatic arsenicals such as cacodylic acid and the sodium salts of methylarsonic acid are less toxic than the inorganic arsenicals. Cacodylic acid was at one time prescribed as a tonic for humans; the dose was 30 mg/day (10, p. 116). The methylarsonates and the dimethylarsinates are only one two-hundredth as toxic as sodium arsenite.

Aromatic arsenicals, such as arsanilic acid and the phenylarsonic compounds are also usually less toxic than the inorganic forms. They appear to be relatively poorly absorbed from the intestine; the part that is absorbed is excreted unchanged (in chickens, rats, rabbits and swine).

Elemental arsenic is not considered to be toxic.

In addition to the differences in toxicity associated with chemical formulation, valency, solubility, etc., a number of workers have demonstrated wide variations in toxicity between animal species. Rats, in particular, are more resistant, probably because they store arsenic in their haemoglobin. Even within

one species, the mouse, different strains vary in their ability to tolerate arsenic trioxide (Harrison et al, 42). A summary of information on the toxic and no-effect doses of a number of arsenic compounds in several animal species and a few domestic fowl is presented in Table 5-2, in ref. (10). Toxicity data on several arsenic compounds for fresh-water and marine fish and shellfish are given in Table 5-6, ref. (10).

The National Academy of Sciences (10) summarizes its review of the toxicity of arsenic by stating "Because so many factors influence the toxicity of arsenic, there is little point in attempting to state its toxicity in terms of milligrams per kilogram of body weight. The lethal oral dose for most species, however, appears to be 1 to 25 mg/kg of body weight as sodium arsenite, and three to ten times that range as arsenic trioxide".

The fatal oral dose of arsenic trioxide for man is stated by Vallee (28) to be between 70 and 180 mg (1 to 2.5 mg/kg body weight). Harrison et al (58) state that as little as 30 mg has been fatal, but no reference is given.

Morbid Effects

The known or suspected harmful effects on health resulting from arsenic intake in humans are:

(a) Acute arsenic poisoning
(b) Subacute/chronic arsenic poisoning
(c) Arsine poisoning
(d) External irritant effects
(e) Sensitization
(f) Suppression of immunity
(g) Teratogenesis and mutagenesis
(h) Carcinogenesis

Most of the references in the medical literature refer only to acute and chronic arsenic poisoning (e.g., Vallee, 28; Browning, 29; Harrison, 58) and no attempt is made to distinguish an intermediate type which might be classified as subacute. On the other hand, the National Academy of Sciences (10) reports much of the literature dealing with non-acute arsenic poisoning under the heading subacute, and confines its description of chronic arsenic poisoning largely to cases of skin cancer, hyperkeratosis and hyperpigmentation occurring in persons exposed to arsenic.

In our review of the literature, aside from acute arsenic poisoning which is readily distinguished as a separate entity because of the acute and severe gastro-intestinal symptoms, we consider that the terms subacute and chronic describe a variety of cases resulting from chronic exposure. The term subacute describes cases in which there is a broader variety of symptoms, the symptoms develop earlier and are of greater severity and the outcome may be more severe. Cases showing fewer and less specific symptoms, and which come on more slowly

would be classified as chronic. While cancer may be a feature of chronic arsenic poisoning, we have deferred discussion of this to a separate section.

(a) *Acute Arsenic Poisoning*

Acute arsenic poisoning is almost always the result of ingestion (as contrasted with inhalation or skin absorption) of rodenticides, pesticides, herbicides or other preparations containing arsenic. Ingestion may have occurred accidentally, or by suicidal or homicidal intent. Acute systemic poisoning as the result of inhalation of arsenic trioxide or other arsenic-bearing dusts is very seldom encountered in industry, even when the exposure is heavy.

Signs and symptoms of acute arsenic poisoning usually develop within 1 hour of ingestion and reflect the severe damage to the gastro-intestinal and cardio-vascular systems; they include nausea, vomiting, diarrhoea, burning of the mouth and throat and severe abdominal pains. The vomitus often contains blood. Circulatory collapse with cyanosis and shock may develop, followed by death within a few hours. During the acute phase, urinary arsenic excretion may be ten times normal or more (28). There is usually some excretion of protein in the urine, and liver function tests are abnormal.

Recovery from acute poisoning may be followed in 1 to 4 weeks by exfoliative dermatitis and peripheral neuropathy. About 5 weeks after exposure (either acute or chronic) white transverse lines (Mees' lines) may appear in the finger nails.

(b) *Subacute/Chronic Arsenic Poisoning*

Ingestion would appear to be the commonest route of intake in subacute poisoning. Chronic arsenic poisoning is also usually the result of ingestion, but may occur from inhalation in industries where men are exposed to high concentrations of airborne arsenic and not protected with respirators. Even in industry, exposure by ingestion may be of more importance in causing systemic poisoning than is exposure to very dusty atmospheres. Cases of chronic systemic arsenic poisoning in an Ontario silver refinery handling arsenical residues in the late 1920's were found to have occurred only in employees ingesting soup made with arsenic-contaminated well water (Sutherland, 59).

The symptoms of subacute arsenic poisoning include gastro-intestinal disturbances, nausea, alternating diarrhoea and constipation, loss of weight, anorexia, and in some cases respiratory symptoms (rhinitis, laryngitis and bronchitis) appearing several weeks after the beginning of exposure. The later developments in cases of subacute poisoning are similar to the signs and symptoms of chronic poisoning, namely the insidious onset of neuralgic pains and changes in sensation affecting the limbs (from loss of sensation to "pins and needles"), muscle tenderness, diminished to absent reflexes, weakness of the extensor muscles of the wrists, fingers and toes, and paralysis of the extremities. There may be personality changes along with headache. Transverse white lines

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(Mees' lines) may appear on the finger nails. Increased salivation, chronic hoarseness, cough and perforation of the nasal septum may also be present. Leukopaenia (decrease in the white blood cells) has been noted in several studies, and toxic hepatitis and optic nerve toxicity have also been reported.

Particularly characteristic of chronic arsenic poisoning are the changes which may appear in the skin. These consist of a branny, non-itching desquamation with scattered areas of deeper pigmentation affecting the neck, armpits, trunk, and around the nipples and old scars. Late manifestations include the development of thickened palms and soles and hyperkeratotic or wart-like lesions, as well as cancers of the skin.

Most of the foregoing signs and symptoms were described by Reynolds in 1901 (quoted in ref. 10) when he reported on more than 500 (of a total of some 2,000) patients in Great Britain who were poisoned by drinking beer made with sugar contaminated by arsenic. Ingestion of alcohol may well have played a role in these cases.

Other outbreaks of subacute or chronic poisoning have generally presented similar symptoms, but sometimes with added or different features.

Mizuta et al (quoted in ref. 10) reported in 1956 on 220 patients who had consumed soy sauce contaminated by arsenic (probably calcium arsenate) over a 2- to 3-week period. Daily consumption of arsenic was estimated at approximately 3 mg. In this group, gastro-intestinal symptoms, headaches and fever were prominent, 85% had facial oedema, 20% had neuropathy, and less than 10% developed skin rashes, desquamation and pigmentation. In the majority of the cases there was liver enlargement but few significant changes in liver function tests or in liver pathology (5 biopsies). There were no significant clinical abnormalities of the heart but 16 of 20 patients examined by electrocardiography showed abnormalities. The neuropathies, in some cases, developed one or two weeks after arsenic ingestion was terminated. Hair samples taken near the root contained from 3.8 to 13 ppm arsenic, compared with 0 to 1.5 ppm near the ends. It should be noted that the estimated daily intake of arsenic in this episode, approximately 3 mg, is essentially the same as that considered safe by the Joint FAO-WHO Expert Committee in 1967 (41).

Peripheral vascular changes, including Raynaud's phenomenon, acro-cyanosis, ischaemia of the tongue, hemiplegia, mesenteric thrombosis and myocardial ischaemia were a prominent feature in children who consumed water containing 0.8 ppm arsenic for some years in Antofagasta, Chile, during the early 1960's (Borgono et al, 60). Abnormal skin pigmentation and hyperkera-toses were also reported. Cough, bronchopneumonia and bronchiectasis were more common among the exposed children than in a control group.

In 1968, Tseng et al (quoted in refs. 10 & 61) reported on more than 40,000 persons in a number of villages in Taiwan who were consuming well waters ranging from 0.017 to 1.1 ppm arsenic. Of this large group, 18.4% had hyper-

pigmentation, 7.1% had keratotic lesions, 1% had skin cancer, and 0.9% had "black foot" disease, apparently the result of arterial spasm in the legs and leading to gangrene. The conditions found increased in frequency with increasing arsenic level in the well waters.

Silver and Wainman (62) describe a typical case of chronic arsenic poisoning in a man who ingested 8.8 mg of potassium arsenite daily for 28 months. Gastrointestinal symptoms appreared about the 13th month. Redness and puffiness about the eyes and hyperkeratoses developed around the 18th month, and neurological changes after about 2 years.

In February 1976 a diagnosis of chronic arsenic poisoning was made in a Nova Scotia patient whose well water was found to contain approximately 5 mg/l of arsenic (Grantham and Jones, 63). Further well sampling showed that 29 of the 200 wells in the community had arsenic levels exceeding 0.05 mg/l. A clinical study, including electromyography, was undertaken and a preliminary report presented before the Royal College of Physicians and Surgeons of Canada in January 1977 by Dr. J.T. Hindmarsh. Hindmarsh reported that of 110 residents on well waters with more than 0.05 mg/l arsenic, 91 had hair levels greater than 1 ppm. No hair levels exceeding 1 ppm were found in 21 control patients whose well water arsenic levels were below 0.05 mg/l. There were abnormal electromyographic findings in some of the patients whose hair arsenic levels exceeded 1 ppm, but none in the control group. Grantham's report indicates that the frequency of mild clinical signs and symptoms compatible with chronic arsenic intoxication, and of hair arsenic levels greater than 1 ppm, increased with higher arsenic concentrations in the well waters.

Another episode of subacute and chronic arsenic poisoning was reported from Japan in 1955 (reports by Eiji, Nagai et al, and Yamashita et al, quoted in ref. 10). More than 12,100 babies who had consumed milk formula contaminated with arsenic over a 4-month period were affected. There were 130 fatalities. The main symptoms were anorexia, nausea, vomiting and diarrhoea, fever, skin rashes and/or hyperpigmentation, and swelling of the abdomen. Enlargement of the liver was a prominent feature. Peripheral neuritis was not observed, even by electromyelography. Laboratory findings included anaemia, leukopaenia, abnormal electrocardiograms and lines of increased density at the growing ends of the long bones (similar to the "lead line"). On follow-up 15 years later, the children have shown reduced growth, a 15% incidence of hyperkeratosis and an increased incidence of mental retardation, epilepsy and other findings suggesting brain damage.

It will be noted that in the above-mentioned studies the route of arsenic intake was by ingestion. There are other similar reports in the literature. The number of studies in which subacute or chronic arsenic poisoning has resulted from inhalation are relatively few, if one leaves aside those in which the main finding was skin cancer and malignancies of other sites.

In 1966 clinical examinations were carried out on 369 male residents of Yellowknife, Northwest Territories (de Villiers et al, 6). The group included some individuals who were, or had been exposed at work to high levels of airborne arsenic. The major findings included a fairly high number of skin lesions (scaly dermatitis, eczema, rashes around the naso-labial folds thought to be due to arsenic, and one basal cell carcinoma), a large number of neurological findings including loss of sensation and weakness, and a high frequency of electrocardiographic abnormalities. Among men exposed to arsenic at work, skin complaints were most closely correlated with duration of employment in the mill. Urinary arsenic excretion tended to be higher among mill employees than among town residents, but all were within the normal range (less than 150 μg/l).

In 1948, Perry et al (64) reported that all of 31 chemical workers handling inorganic arsenic compounds had hyperpigmentation, and nearly one-third of them had warts. A survey of airborne arsenic levels showed mean concentrations in the plant ranging from 78 μg/m^3 to 1,034 μg/m^3. Urinary arsenic levels in the chemical workers averaged 243 μg/l compared to 92 μg/l in an unexposed control group. Average levels of arsenic in hair ranged from 13 ppm in the control group to 108 ppm in the chemical workers. The urinary excretion levels reported in this study appear to be relatively low, and the hair levels high (particularly in the control group) when compared with the values reported in other studies (e.g., Pinto and McGill 65). The study by Perry et al is also of interest in view of the cancer findings reported by Hill and Faning (see section on Carcinogenesis).

An investigation by Watrous and McCaughey (66) of visits to the plant medical department by men employed in the manufacture of organic arsenicals found only a significant increase in the number of complaints of hyperkeratosis. The airborne arsenic levels to which the men were exposed ranged from 5 to 456 μg arsenic/m^3.

In addition to the studies previously mentioned in which cardiovascular effects and changes in the electrocardiogram were noted, ECG abnormalities in men exposed to arsenic have been reported by Zettel, Butzengeiger, Barry and Henderson, and Glazener et al (quoted in ref. 33). The ECG changes have apparently been reversible following termination of exposure.

Cirrhosis of the liver has been reported by Franklin et al following long ingestion of Fowler's solution, and by Butzengeiger and by Roth in German vineyard workers (quoted in ref. 33). In the latter group consumption of alcohol may have been a contributing factor. Lee and Fraumeni (67) found an increase in mortality from cirrhosis of the liver in men employed in an American smelter, but did not find the increase related to length of exposure. Liver damage has been shown in animals after ingestion of sodium arsenite and sodium arsenate (Byron et al) and after inhalation of arsenic trioxide (Rozenshtein) (both quoted

in ref. 33). The relationship between cirrhosis of the liver and environmental or occupational exposure is as yet not clear.

With regard to the significance of electromyographic changes in assessing chronic arsenic exposure, Dr. Hindmarsh's report is awaited with interest. His preliminary findings appear to parallel those reported by Seppalainen et al (68) in men occupationally exposed to low concentrations of airborne lead. Results are also awaited on a nerve conduction study on workmen at the Tacoma smelter. At present, more research is needed to determine the value of EMG as a method of detecting early biological response to low levels of arsenic exposure.

A recent study by Bencko and Symon (69) using control subjects describes hearing loss in a group of 56 Czechoslovakian children living near a power plant burning high arsenic coal. Both air conduction and bone conduction were affected. Milham (70), on the other hand, failed to find any hearing loss on pure tone screening in a group of 566 school children living near a smelter in Tacoma. Six of the children with high urinary arsenic excretion ($>$ 200 μg/litre) were tested by pure tone threshold audiometry. No hearing loss was detected. The possibility of hearing loss associated with arsenic exposure should receive further investigation.

In Milham's study school attendance records were also examined. There was no increase in absenteeism at the school near the smelter when compared with that at 6 other Tacoma schools. No effect on blood haemoglobin levels was found in 33 children attending the school near the smelter.

(c) *Arsine Poisoning*

The gas arsine (arsenic hydride) is the most toxic form of arsenic. As previously indicated, it may be produced whenever nascent hydrogen (from acid or water) comes in contact with arsenic. Exposure is always accidental, and nearly always industrial. As such, it is not discussed here in detail.

Its biological action is quite different from that of other arsenic compounds. When inhaled, it enters the blood stream where it acts as a potent haemolytic agent, causing rupture of the red blood cells and resultant anaemia and jaundice. Haemoglobin is excreted in the urine. Less acute exposures cause nephritis, hepatitis and myocarditis, and neuritis has been reported in one group of 14 men exposed to very low concentrations of arsine for some months (Bulmer et al, 71).

Inhalation of 3 to 10 ppm of arsine for several hours may produce symptoms; 250 ppm may be lethal in 30 minutes. Depending upon the degree of exposure, in the acute cases symptoms may develop from within a few minutes to 24 hours, and death from anuria may occur within a few days.

(d) *External Irritant Effects*

Solid arsenic compounds, such as the trioxide, are caustic to the skin and mucous membranes and are a common cause of dermatitis in exposed workmen.

The dermatitis usually begins as an erythema associated with burning and itching, affecting warm moist areas such as the neck, armpits, cubital fossae, scrotum, wrists and hands. The rash may disappear or be followed by papular and vesicular eruptions. Healing is often accompanied by a fine desquamation leaving areas of pigmentation.

Irritant effects on the mucous membranes may result in conjunctivitis, rhinitis, pharyngitis and chronic laryngitis. Chronic cough is a frequent symptom. Perforation of the nasal septum is not uncommon in men who have had long exposure.

(e) *Sensitization*

Acquired sensitivity to arsenic was described in a lengthy report by Holmqvist in 1951 (quoted in ref. 10). During a 2-year investigation of smelter employees he found that 80% of arsenic workers were sensitive, by patch testing, to weak solutions of sodium arsenate and arsenic pentoxide which caused reactions in only 35% of non-exposed employees and 30% of new employees.

(f) *Suppression of Immunity*

The possibility that arsenic compounds might suppress immunity in humans was probably first suspected following the observation of a relatively high incidence of herpes simplex (cold sores) and herpes zoster (shingles) infections in patients with subacute arsenic poisoning. Reynolds (quoted in ref. 10) noted 21 cases of herpes zoster in his group of more than 500 patients who had ingested contaminated beer. In the Antofagasta outbreak, herpes of the lip was common, as were recurrent respiratory infections. It is of interest that a similar higher-than normal incidence of herpes infections has been noted in kidney transplant patients whose immunity has been deliberately suppressed by steroids to avoid rejection of the transplant. The lowered white blood cell counts reported in some of the subacute arsenic poisoning episodes also suggest interference with the immune response system, again analogous to the effects of steroids.

Gainer and Pry (quoted in ref. 10) have found that exposure of mice to large doses of arsenic lowered their ability to resist several kinds of viral infections. While laboratory studies of the effect of arsenic on immunity in humans do not appear to have been undertaken as yet, the subject is of more than passing interest because of arsenic's association with cancer (of the skin and other sites) and the possible role of immunity in protecting the body against it.

(g) *Teratogenesis and Mutagenesis*

A few reports on the teratogenic effects of arsenic in animals have appeared in the last 12 years. Ferm and his associates (72, 73) reported a very high incidence of dead, resorbed and malformed embryos in hamsters given a single intravenous dose of sodium arsenate. The malformations involved the brain, rib and genitourinary regions. Administration of the arsenic on the eighth day of

gestation was critical. The doses ranged from 15 to 20 mg/kg, and the incidence of defects in the embryos varied with the dose. It was also shown that the incidence of malformations and resorptions could be decreased by the administration of sodium selenite along with the sodium arsenate.

Similar results have been reported by Hood and Bishop (74) working with mice. Injections of 45 mg/kg of sodium arsenate, given intraperitoneally, were most effective when given on the 9th day of gestation, with up to 60% of the embryos dead, resorbed or malformed. Comparable results were obtained using sodium arsenite in doses of 10 mg/kg. The teratogenic effects of sodium arsenate could be prevented by early intraperitoneal injections of British anti-lewisite (BAL).

Beaudoin (quoted in ref. 10) has shown teratogenic effects with sodium arsenate injected intraperitoneally in rats at dosages of 20 to 50 mg/kg.

Schroeder and Mitchener (75) found that 5 ppm of sodium arsenite in the drinking water, from weaning to death, was not toxic to rats and only slightly so to mice. When the exposure was continued over three generations of mice, there was an increase in the ratio of male to female births, compared to the ratio in a control group. While not strictly a teratogenic study, the findings are of considerable interest because contaminated water supplies, such as well waters, have occasionally been found to contain concentrations of arsenic comparable with that used in this investigation.

Teratogenic effects have not been reported in humans. It should be noted that the dosages used in the above experiments (other than that of Schroeder and Mitchener) were very large. The results do suggest caution when administering arsenical preparations to pregnant women, in employing women in occupations where they would be exposed to arsenic, and perhaps in allowing them unlimited consumption of high-arsenic foods such as shrimp and other shellfish.

There are few reports on the mutagenic effects of arsenic. However, chromosomal breaks have been observed in human leukocyte cultures exposed to sodium arsenate in vitro, and in cultures obtained after long exposure to arsenical compounds in vivo (Petres et al, quoted in ref. 10).

More recently, Beckman et al (76) have reported an increased frequency of chromosome aberrations in smelter workers, though due to their simultaneous exposure to other agents as well as arsenic, the effect could not be attributed to arsenic with certainty.

(h) *Carcinogenesis*

In the group of chemical agents known or suspected of being associated with an increased risk of cancer, arsenic occupies a rather unique position in that the evidence is based almost entirely upon epidemiological studies and clinical observations of its occurrence in man. To date there has been an almost complete failure to produce cancer in experimental animals regardless of the

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species of animal, the chemical or physical form in which the arsenic was administered, the route of administration or the dose used.

The epidemiological evidence that arsenic may act as a carcinogen or cocarcinogen is reviewed in some detail because of the contention by some scientists that there is not a threshold dose below which no cancers will be produced. In other words, it is their contention that any exposure above zero will cause an increase in cancer cases proportionate to the dose.

Arsenic was at one time thought to be responsible for the high incidence of lung cancer in miners in Joachimstal and Schneeberg, in Czechoslovakia and south-eastern Germany. It is now generally accepted that these cancers were caused by radioactivity acquired through the inhalation of radon daughters.

Arsenic was also implicated in the development of lung and sinus cancers in a nickel refinery in Wales during the 1920's (Morgan, 77). Arsenic was present in sulphuric acid used in the plant prior to 1922 or 1923, after which time arsenic-free sulphuric acid was used. The increased respiratory cancer risk was confined to men hired prior to 1925. Men hired after 1925 have had a normal incidence of sinus and lung cancer to date, suggesting that elimination of the arsenic may have been largely responsible for eliminating the increased cancer risk. However, among the pre-1925 employees, many of the men who developed sinus or lung cancer were never employed in that part of the refinery where the sulphuric acid was used. It is also noteworthy that sinus and lung cancers were subsequently reported in the nickel industry in Ontario at two refineries where nickel sulphide matte was calcined or sintered (Sutherland, 78, 79) and in Sweden in a similar operation (Pedersen et al, 80). In none of the last three reports was arsenic exposure likely to have been high, though small amounts were present in the ore and would have been sublimed during the sintering and calcining operations if not already eliminated by earlier treatment in blast furnaces or multi-hearth roasters.

Aside from the cancer experiences at Joachimstal and Schneeberg and in the nickel industry, there have been a considerable number of reports of skin cancer and lung cancer, in particular, in persons exposed to arsenic via medical therapy, occupationally, and by ingestion of contaminated food or water and, more recently, by reason of residing in certain industrialized areas.

Reviews of most of this literature have already been published. The International Agency for Research on Cancer, in volume 2 of its series of monographs on *The Evaluation of Carcinogenic Risk of Chemicals to Man*, has prepared a brief but excellent summary of both experimental studies and clinical and epidemiological reports on human experience to the end of 1971, and including a few reports from 1972 (81). A more detailed review of the literature dealing with arsenic and human cancers is contained in the NIOSH document listed in ref. 33. This review includes reports up to 1974, and one or two from

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1975. The most detailed and most recent survey of the literature is that given in the National Academy of Sciences monograph *Arsenic* (10).

The occurrence of skin cancers in persons who have ingested arsenic compounds for medicinal purposes has been known for many decades. Usually other skin changes characteristically associated with ingested arsenic, such as hyperpigmentation and keratoses, were present (Neubauer, 82). Not infrequently the skin cancers were multifocal, and in some cases primary cancers of other organs (most frequently the lung and liver) were also present (Somers and McManus, 83; Minkowitz, 84; Robson and Jelliffe, 85; Regelson et al, 86). The cancers tend to be late in developing, the latent period from first exposure to diagnosis of the cancer being 20 to 30 years or longer. Some evidence of a dose relationship between the medicinal dose of arsenic and the risk of skin cancer has been reported. In Neubauer's review, the average medicinal intake of arsenic was 28 g, though cases were reported following ingestion of as little as 180 mg and 700 mg arsenic two or three decades earlier.

Skin cancer has also been reported in one study following ingestion of arsenic contaminated drinking water. Tseng et al (61, 87) found that the incidence of skin cancer in one district in Taiwan increased sharply as the arsenic content of the drinking water rose above 0.3 mg/l.

According to the IARC Monograph (81), no excess of cancers of other sites has been reported in areas where the water has contained high levels of arsenic. One case of haemangioendothelioma of the liver was reported by Rennke et al (quoted in ref. 81) in an area of Chile where the drinking water content of arsenic was high.

With regard to occupational exposure to arsenic, precancerous and cancerous skin lesions have been reported in vineyard workers in Germany and France, and in potato farmers applying arsenical insecticides for many years. The vineyard workers were exposed to arsenic by inhalation of lead arsenate dust and ingestion of contaminated wine. Among 47 cases of vintners showing signs of chronic arsenic poisoning, such as hyperkeratoses and hyperpigmentation, who were autopsied, 30 deaths were attributed to cancer including 18 lung cancers, 6 haemangiosarcomas of the liver, 5 cancers of the oesophagus, and one cancer of the bile duct. In 10 men there were multiple tumours of the skin and internal organs. Cirrhosis of the liver was reported in 23 of the 47 autopsies (Roth, quoted in ref. 33). The German vintners were estimated by Roth to have ingested an average of 53.6 g arsenic over a 12-year period. Roth also reported a higher proportion of lung cancer deaths in the Moselle region generally, as compared with that in urban and non-vineyard areas.

Hill and Faning (88) found a significant increase in the proportion of deaths due to cancers of the skin and lungs in men engaged in the manufacture of arsenical sheep-dip. They were exposed to airborne arsenic levels ranging from

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several hundred $\mu g/m^3$ to more than 1,000 $\mu g/m^3$. Unexposed workers at the factory did not show a similar excess of cancer deaths.

Two studies of mortality in men employed in the smelting and refining of arsenic-bearing ores and concentrates failed to find increased risks of cancer (Snegireff and Lombard, 89; Pinto and Bennett, 90). Both of these studies have been criticized on the basis that the follow-up of pensioned and/or retired employees was incomplete. A recent mortality survey by Milham and Strong (91) of the workers from the plant studied by Pinto and Bennett revealed 40 deaths from lung cancer compared with 18 deaths expected on the basis of U.S. age-specific death rates. The excess of observed deaths was statistically significant.

In a recent follow-up study of 527 retired workers from the same plant, Pinto et al (92) reported no excess of lymphatic or haemopoietic cancers among 324 deceased workers, but they did find an excess of deaths due to cancer of the respiratory system. Using urinary arsenic excretion and duration of employment as an index of exposure, they found an increasing incidence of lung cancer mortality with increase in exposure. No increase was found in 99 men who had less than 25 years of exposure that produced less than 200 μg of arsenic per litre of urine. In men with more than 25 years of exposure that produced 350 μg or more per litre, there was an eight-fold increase in lung cancer. The authors were of the opinion that certain of their data supported the existence of a safe threshold for airborne arsenic trioxide exposure.

In addition to the experience at the foregoing plant, several other investigations have shown a higher-than-normal mortality from lung cancer in smelter and refinery workers exposed to arsenic as well as other contaminants.

In 1956 Sutherland (59) reported a significant increase in lung cancer in men exposed to dust and fumes containing arsenic and other elements produced in the roasting of high-arsenic residues.

In 1959 Rockstroh (93) reported 45 cases of cancer of the bronchus over an 11-year period in a group of 111 men working in a plant which smelted arsenical ores and concentrates for their nickel and cobalt content. Two men developed cancers of the skin. In 39 workers there were perforations of the nasal septum. Almost all workers had hyperkeratoses of the hands and feet. Melanosis was observed in only 3 cases. Among workmen not employed in the production department, only one developed a bronchial carcinoma.

In 1969 Lee and Fraumeni (67) examined the mortality experience of more than 8,000 men employed as smelter workers during the period 1938-1963. Exposure to arsenic trioxide and to sulphur dioxide was classified into high, medium and low categories. For all smelter workers, mortality from lung cancer was three times higher than expected on the basis of the age-specific death rates for the state. In men employed for 15 years or more in high arsenic exposure, lung cancer mortality was 8 times higher than expected. The risk of lung cancer

also increased in proportion to the degree of exposure to sulphur dioxide. While this study supports the contention that arsenic is carcinogenic, exposure to sulphur dioxide and other contaminants may have played a contributory role.

In 1974 Kuratsune et al (94) reported that 11 out of 19 men dying of lung cancer in a Japanese town had worked at a copper smelter. Of 19 men dying of diseases other than cancer of the lung, bladder and skin, only 3 had been employed at the smelter. In 1976 a cohort study of plant employees was reported by Tokudome and Kuratsune (95). Their study covered 2,675 men employed during the period 1949-1971. Among 839 copper smelters mortality was increased 12-fold for lung cancer and 3-fold for cancer of the colon. A dose-response relationship was demonstrated between mortality from lung cancer and the degree of exposure, reaching 25 times expected in smeltermen most heavily exposed to arsenic and in workmen who had spent more than 15 years in sintering and blast furnace operations prior to 1949. No data as to airborne concentrations were presented. The latent period from first exposure to development of lung cancer averaged 37.6 years and was not related to the level of exposure.

Ishinishi et al (96) recently reported the results of instilling suspensions of pure arsenic trioxide, flue dust from the Japanese copper smelter referred to above, and copper ore into the lungs of rats. No squamous cell carcinomas were produced, though one adenocarinoma was found. Squamous cell carcinomas were produced when the dusts were administered with benzopyrene, in greater frequency than when benzopyrene was administered alone. The results suggest that solid arsenic compounds act as cocarcinogens.

Two unpublished studies by Rencher and Carter, in which the mortality experience of active and retired employees of a Utah copper company during the period 1959-1969 was investigated, are reviewed in ref. 33. Smelter workers had a higher proportion of deaths due to lung cancer (7.0%) than did mine or concentrator employees (2.2%). Both smoking and non-smoking smelter workers had a higher relative frequency of lung cancer than did their counterparts who worked in the mine or concentrator. Hourly airborne arsenic levels in the reverberatory furnace area of the smelter averaged 22 $\mu g/m^3$ during recent years. No data were available for the years prior to 1959 but it was estimated that arsenic concentrations within the plant were at least 3 times higher in the earlier years.

A third unpublished study on the same company employees, by Milby and Hine, is also quoted in ref. 33. This study failed to show an excess of lung cancer. The NIOSH reviewers, however, considered this study to contain several weaknesses from the epidemiological point of view.

In only one locality has the mining of ores containing arsenopyrite been reported to entail an increased risk of respiratory cancer. A 3-fold increase in Rhodesian gold miners, based upon proportionate mortality, was reported by

Osburn in 1957 (quoted in ref. 81). In a subsequent report in 1969 he noted that many of the miners had palmar hyperkeratoses suggesting chronic arsenicism.

Because occupational exposure among smelter workers is usually a mixture of contaminants, several studies from the chemical industry are of considerable interest because the exposure to arsenic compounds was more specific.

In an unpublished report in 1974, Baetjer et al (quoted in ref. 33) found a significant increase in lung cancer (16-fold) and lymphatic cancer (50-fold) in retirees from a plant manufacturing arsenical insecticides. Ferguson (97) stated that arsenic exposure levels were probably 5 mg/m³ or higher in that part of the plant where arsenic acid had been prepared from arsenic trioxide and nitric acid prior to 1952, and about 1 mg/m³ in the insecticide manufacturing area.

Probably the most important single study is that by Ott et al (98) published in 1974. The authors reported that the proportion of deaths due to lung cancer among 173 decedents who had been exposed primarily to lead and calcium arsenates was three times higher than that in 1,809 decedents who were not exposed. Cancer of the lymphatic and haemopoietic system (excluding leukaemia) was also significantly increased in the exposed group (2.5-fold). The authors also carried out a cohort study of 603 chemical workers with at least one month of exposure to arsenic. The period covered was from 1940-1973 inclusive. The results confirmed the findings of the proportionate mortality study.

Of particular interest was the authors' attempt to determine whether a dose-response relationship between lung cancer and exposure to arsenic could be demonstrated. Dust levels in 1943 had ranged from 0.18 mg to 19.0 mg arsenic per cubic metre of air in the packaging area. In 1952 breathing zone samples near the drum dryer had ranged from 1.7 mg to 40.8 mg/m³. Ott and his colleagues established four job categories and estimated 8-hour time-weighted average exposures to arsenic for each category, namely 5 mg/m³; 3 mg/m³; 1 mg/m³; and 0.1 mg/m³. Cumulative doses for each man were calculated by summing the products of the number of months worked at each exposure level times the 8-hour TWA concentration. A working month was taken as 21 days, and it was assumed that a man breathed 4 cubic metres of air over 8 hours. The cumulative doses were converted to log normal doses and tabulated against the ratios of observed to expected lung cancer deaths. A dose-response relationship was found. No excess of lung cancer occurred in men whose cumulative dose of arsenic was 42 mg. A 2-fold to 3-fold increase was observed in 5 groups who had cumulative doses ranging from 127 mg to 1.5 g arsenic. The excess lung cancers in these five separate groups were: 1, 2.6, 1.6, 1.3 and 1. In none of these groups do we find the increase statistically significant when tested individually by Poisson probability. When the five groups are taken together, the excess of lung cancers (7.5 cancers in all) is quite significant (P = 0.006). There was a four-fold increase in men who had a cumulative dose of 3.5 g (an excess of 2.2 cases), a 6.3-fold increase in those with a cumulative dose of 6.5 g (an excess of 4.2 cases), and

a seven-fold increase in those with a cumulative dose of 29.7 g (an excess of 4.2 cases). All increases in the last three groups were statistically significant.

Blejer and Wagner (99) have used the cumulative arsenic dosages, as estimated by Ott et al, to calculate what the daily 8-hour time-weighted average arsenic concentrations would have been if the total dosages had been accumulated over a 40-year working lifetime. Their calculations suggest that respiratory cancer mortality is increased 2-fold over that expected for men exposed to 3 $\mu g/m^3$ for 40 years. These calculations have been used to support the NIOSH recommendation of 2 $\mu g/m^3$ for men occupationally exposed to airborne arsenic. Their argument is debatable on a number of points.

Firstly, the estimate by Ott et al that a working man breathes 4 cubic metres of air per 8 hours is low. The "standard man" doing light work is considered to breathe 9.6 cubic metres per 8 hours (ref. 43, page 51). Thus the cumulative exposures estimated in the study by Ott et al should be increased by a factor of approximately 2.5, or more in men doing moderately heavy work.

Secondly, of the 28 respiratory cancer deaths reported by Ott et al, 10.5 might normally have been expected. The excess, totalling 17.5 cases, might be considered due to arsenic exposure. It is of considerable scientific interest to attempt to see whether these excess cases demonstrate an increase in risk with increase in cumulative dose, as Ott and his colleagues did, using nine exposure groups. One might question the wisdom, however, of trying to justify what is essentially a zero threshold standard for occupational exposure by the use of such limited data, particularly when the justification amounts to only 17.5 cases spread over eight exposure groups.

Re-arrangement of the data presented by Ott et al shows that if they had combined the three groups with the least cumulative exposure, i.e. all men with up to a log normal dose of 5.53 (252 mg) there would have been 7 deaths from lung cancer versus 4.16 deaths expected. The probability of an excess of this magnitude is not statistically significant (P = 0.24). Only if the group with the next highest exposure is included, i.e. those with a log normal dose of 6.04 (419 mg) would the excess of lung cancers (10 observed versus 5.52 expected) have reached the 5 percent level of significance. If one corrects the cumulative exposure by a factor of 2.5 for the amount of air breathed daily, a cumulative dose of approximately 1 gram would have been reached before the excess of lung cancers became statistically significant.

Figure 1 in the paper by Ott et al graphs the ratio of observed to expected deaths against log dose. The resultant curve suggests that the increase in lung cancer deaths is proportional to log dose, i.e. a logarithmic curve. If this is so, then there probably is a threshold dose below which excess lung cancer deaths would not occur.

Thirdly, Blejer and Wagner make the basic premise that a cumulative dose acquired in small daily amounts over 40 years is carcinogenically equivalent to

the same cumulative dose acquired in much larger daily amounts over a few weeks, months or years with no further intake during the following years. This premise would apply only if there were no threshold dose for cancer induction. It ignores the possibility of a dose-*rate* effect. It also ignores the body's ability to detoxify arsenic in small doses, and makes no allowance for its ability to protect itself from cancer by immunological responses.

The elimination of the sinus and lung cancer hazard in the nickel industry in Wales about 1925 provides a practical example of the existence of a safe threshold for an industrial carcinogen. The reduction of furnace fumes, and the protection afforded workmen through the wearing of Martindale face masks, did not in all probability reduce inhalation exposure below the hundreds of micrograms per cubic metre of air level, yet the measures taken have proven completely effective.

Another example, though less convincing because the follow-up period is only about 25 years, is the experience in the chromate industry. According to Ferguson (ref. 32, p. 326) reduction of exposure to insoluble chromate dusts to a time-weighted average of 50 μg/m^3 has enabled the industry to "lick the chrome problem or certainly control it 95 per cent". The future cancer experience in this industry will be watched with real interest.

The evidence against the no-threshold dose for carcinogens has recently been discussed by Dr. H.E. Stokinger, Chief of the Toxicology Branch of NIOSH (100). Of 22 chemicals suspected of presenting a carcinogenic risk for industrial workers, threshold limit values have been established for 14, including arsenic trioxide. To protect against cancer, the TLV's for concurrent exposure to arsenic trioxide, antimony trioxide and sulphur dioxide were set at 50 μg/m^3 for arsenic trioxide, 50 μg/m^3 for antimony trioxide and 5 ppm for sulphur dioxide by the American Conference of Governmental Industrial Hygienists in 1976.

One other occupational study is reviewed in ref. 33. In 1973 Nelson et al (101), using proportionate mortality, found no evidence of increased mortality from cancer in 1,231 men who had worked as orchard sprayers in the 1930's. Air concentrations of arsenic during the spraying had averaged 140 μg/m^3. NIOSH re-examined the data in this report, using other data sources (ref. 33, pp. 51-53) and concluded that there was a significant increase in male lung cancer mortality in one county in which the majority of the orchardists resided, but no increase in female lung cancer deaths. In two other counties male and female mortality from lung cancer were not increased.

Three studies of cancer mortality in populations not occupationally exposed to arsenic have recently been reported. Blot and Fraumeni (102) found that lung cancer deaths were significantly higher among males and females in 36 counties where arsenic-containing ores of copper, lead or zinc were smelted than in the rest of the United States. The average increase was 17% for males and 15% for females. Mortality from lung cancer was not increased in 35 counties where

other non-ferrous ores were processed. The authors concluded that occupational exposure, differences in smoking habits, urbanization and other socioeconomic factors could not have accounted for the general increase in lung cancer mortality in the lead-copper-zinc industry counties, and that the most likely explanation was air pollution from industrial sources of inorganic arsenic. No data were available as to the levels of arsenic in the ambient air of the counties studied.

Nelson (103) has criticized this study on the grounds that no distinction was made between smelters and refineries. The latter have little or no arsenic emission. Secondly, Nelson states that most of the arsenic-bearing ores go to the copper smelters, rather than to the lead and zinc smelters. Of 13 counties having copper smelters, 9 had lower lung cancer mortality rates, and only 4 had higher rates, than the U.S. national average.

Matanoski et al (104) studied cancer mortality in four census tracts in the immediate vicinity of a chemical plant in Baltimore which produced arsenic insecticides, and compared the mortality rates with those in 23 other census tracts matched for five variables such as age, sex, ethnic origin and socioeconomic characteristics.

A 4-fold increase in lung cancer in males was found in the census tract in which the plant was located. In two other of the neighbouring tracts an increase in lung cancer deaths was noted, but the excesses were not statistically significant. No excess of lung cancer mortality for females was found.

The authors reported that examination of the plant records failed to account for the excess of male lung cancers. They felt that environmental exposure to arsenic may have been a factor, and that possibly the synergistic action of cigarette smoking would explain the differences found in male and female lung cancer mortality. Again, no data as to arsenic levels in ambient air were given in the report.

Pershagen et al (105) examined mortality over a 14-year period in an area around a smelter in Sweden handling high-arsenic ores. No increase in male lung cancer mortality was found when cases who had worked at the smelter were excluded. There was a statistically significant increase in lung cancer among men who had been occupationally exposed. Female deaths from lung cancer appeared to be increased and further investigation is being undertaken on this aspect of their findings.

Newman et al (106) have reported on the histological characteristics of lung cancers found in a group of copper smelter workers and in residents in a city adjacent to copper mines in Montana. They found an excess of poorly differentiated epidermoid cancers, and suggested that this cell type may be characteristic of exposure to arsenical dusts. In contrast, uranium miners have been found to show an excess of small cell undifferentiated cancers ("oat" cell).

In summary, from the clinical observations and the epidemiological evidence, there appears to be no doubt that exposure to inorganic arsenic compounds in relatively large amounts can cause an increase in cancers of several organs in man, particularly the skin and the respiratory system. From a few studies in the pesticide manufacturing industry it appears that concurrent exposure to other industrial contaminants is not required to produce the carcinogenic response. There is suggestive evidence that exposure to airborne arsenic may be a factor in the increased mortality from lung cancer which has been found in several studies in persons living in the neighbourhood of industrial operations in which inorganic arsenic was emitted to the atmosphere.

Occupational Exposure Standard

One of the most difficult tasks faced by the Task Force was to determine a "safe" concentration of arsenic in air for occupational exposure. The Task Force considers that the most critical risk is that of developing cancer, particularly of the lungs.

Any attempt to determine an exposure level sufficiently low to prevent this disease, or at least to reduce the risk to a very low level, requires a knowledge of the biological processes involved in the initiation of a cancer in the body and those that control its growth and spread.

Animal or human response to varying doses of a carcinogen is primarily measured in two ways. One is the incidence or number of cases which develop at each dose level over a life-time. It is of interest to note that the tumour yield is markedly increased when a fixed dose of carcinogen is administered over a period of time in divided doses, as compared with administering it as a single dose (Kotin, 107). The second way of measuring dose-response is to determine the time-to-occurrence, i.e. the time from first exposure to the carcinogen to the development of the cancer or until death of the animal. Much of our knowledge of dose-response relationships in humans is limited to the first kind of measurement. The time-to-occurrence method would have practical application if it could be shown that by limiting the dose to a certain level the development of cancer could be delayed to an age beyond normal life-time expectation.

The question of whether threshold levels exist for carcinogens, i.e. dose levels below which no cancers will develop, has been the subject of much debate among scientists. The existence of a dose-response relationship over certain dose ranges has been clearly established for many carcinogens in many animal experiments. (Animal experiments with arsenic, however, have failed to show any direct carcinogenic effect, with the possible exception of that of increasing the response to other carcinogenic agents, i.e. a possible cocarcinogenic effect (Ishinishi, 96)). Few published experiments are capable of detecting tumour yields of less than 10 per cent, or possibly 5 per cent (Shubik and Clayson, 108). To show whether even lower doses produce cancer would require testing on much larger

groups of animals — the lower the dose tested, the larger the group necessary. At some point it would become impossible, even with extremely large groups of animals, to determine by experiment that the dose was or was not carcinogenic.

In view of the inability to prove a safe threshold, it has been considered by some scientists that carcinogens must be regarded as effective (if only minutely so) to infinitely small doses, that in effect there is no threshold. The question then becomes one of completely eliminating all exposure or of defining a level of acceptable risk, i.e. a small additional risk of acquiring cancer as the result of exposure above that which is considered a normal or "background" risk of the disease. This introduces the question of "Acceptable to whom — government, labour, industry?" One level of risk suggested as acceptable is 1 case in 100,000 men. For lung cancer, such a risk would be statistically immeasurable against the prevailing male death rates. It should also be noted that in a group of 100 workmen living for 50 years after first exposure, i.e. a total of 5,000 man-years, the chances of that one additional lung cancer occurring in this group would be only 1 in 20. The risk to each individual man would, of course, be 1 in 100,000.

To look at the other side of the question, other scientists believe that safe thresholds for carcinogens do exist. Their opinion is based on the fact that cancer initiation and development in the body takes place in a series of stages or steps, and that failure of the cancer process at one or other stage can forestall its subsequent development. To quote Kotin (107):

"Nevertheless, for carcinogenic organic and inorganic chemicals, metals, nonionizing and ionizing radiation, and specifically for vinyl chloride and asbestos, dose-response data and no-effect levels have been found. To deny the existence of dose response would erroneously place chemical carcino-genesis outside the universe of pharmacological principles that govern enzyme induction, feedback, repair mechanisms, primary and alternate metabolic pathways, metabolite excretion, and so on — clearly an insupportable concept".

As mentioned previously, Dr. H.E. Stokinger of NIOSH argues for the existence of a threshold for carcinogens (100). Friedman is quoted by Magee (109) to the effect that "the existence of a threshold for a biologically active substance is a biological reality and not a subject of probabilistic speculation". Shubik and Clayson (108) point out that the concept of zero tolerance for car-cinogens arose before the ability of a cell to repair lesions induced in its own genetic material was recognized.

There is, then, as yet no final proof as to which concept is correct, or if indeed some other concept will prove to offer a better understanding of carcinogenic risk at very low dose levels.

Governments charged with protecting the health of workers are similarly divided in their approach to the problem. The U.S.S.R. accepts the concept that

there is a threshold for all types of harmful action, including carcinogenic and mutagenic effects (110). In the United States, on the other hand, NIOSH has opted for a "non-detectable" level for all inorganic solid arsenic compounds and has recommended 2 micrograms per cubic metre of air as the lowest practical measurable concentration. The U.S. Occupational Safety and Health Administration states that "the exposure level must be as low as feasible" and proposes an action level of 2 micrograms per cubic metre determined on an 8-hour time weighted average basis, and 10 micrograms per cubic metre for a 15 minute period as a ceiling limit (111). It is almost three years since OSHA presented these proposed exposure standards; as yet they have not been formally adopted.

The Task Force is primarily concerned that the health of workmen be protected and considers that the concentration of arsenic trioxide in air to which workmen are exposed should be maintained at a level which ensures that the occupational risk of lung cancer is negligible. Between the concepts of threshold or no-threshold, we feel that the balance of research to the present tends to favour the threshold concept.

With regard to a level of airborne exposure which entails negligible risk, we consider 30 micrograms per cubic metre of air as an 8-hour time weighted average to be acceptable. This value is derived after a consideration of ambient air standards. Neither Canada nor the United States has as yet adopted air quality standards for community air. The standard in the U.S.S.R. and in Czechoslovakia is 3 micrograms per cubic metre maximum over 24 hours. The preferred standard in British Columbia is 1 microgram per cubic metre 24-hour average. Ambient air quality standards are customarily set some 5 to 10 times lower than those for occupational exposure to protect residents in the community who may be unduly susceptible, such as the young, the aged and the sick. It is a matter of simple arithmetic to show that occupational exposure to 30 micrograms per cubic metre of air for 8 hours a day for 250 working days a year for 35 years would increase the lifetime intake of arsenic less than five-fold over that of an individual living in a community with an ambient air level of 1 microgram per cubic metre. With an ambient air level of 2 micrograms per cubic metre, the worker's intake would be less than three times that of the community resident. Adherence to the 30 micrograms per cubic metre value which we recommend for occupational exposure would thus provide a reasonably low increment of arsenic intake over that acquired from ambient air, and would reduce the risk of occupational lung cancer to an immeasurably low level.

Early Detection of Lung Cancer

Two procedures are presently being employed or are being explored for the early detection of lung cancer. The first of these is sputum cytology — the

microscopic examination of cells from the lung or trachea shed in the sputum. This procedure has proven of value in the surveillance of men known to have a high risk of lung cancer, such as uranium miners, sintermen and calcinermen in the nickel industry (McEwan, 112) and coke oven employees. In practice, the procedure presents problems, including those of organizing the program, obtaining the sputum samples, arranging for their prompt examination and communicating the results to the worker. The Task Force also recognizes that an increased risk of lung cancer has not yet been shown to exist for the mill workers at Yellowknife. Nevertheless, the potential for carcinogenic injury exists, particularly in older workmen who have had long exposure. We therefore recommend that sputum cytology examinations be carried out at 6-month intervals on men aged 40 years or more who have had 10 years or more of exposure to arsenic.

A decision as to the possible value of extending sputum cytology examinations to the general public at Yellowknife should be made on the basis of the results obtained in the occupational surveillance program.

The second procedure (really a group of procedures) for the early detection of cancer is the examination of the blood or urine for the presence of abnormal antigens and enzymes, such as CEA (carcino-embryonic antigen), AFP (alpha-fetoprotein) and acid phosphatase. These procedures have been found of value in monitoring the progress of patients being treated for cancer. Their value in the early detection of lung cancer in persons with a high risk are as yet open to question, since the tests are not very specific for this form of malignancy. The Task Force considers these procedures require further research before their value for the surveillance of high risk groups is known. As stated by Maugh in a recent issue of *Science*, "It may not yet be possible to recognize the presence of a tumour and identify its site by means of a single blood test, but it now seems much more likely that such a day will eventually arrive" (113).

Exposure to Arsenic in Yellowknife

Most of the information and data pertaining to occupational exposure to arsenic and the examination of employees was made available to the Task Force prior to its completion of the Interim Report. This information is repeated here, amended as necessary, for the sake of completeness. Such new information as has been obtained since publication of the Interim Report is introduced at appropriate locations in this chapter.

The Task Force made tours of inspection of the Giant Yellowknife mine and mill and of the Con mine and mill to view conditions of work and the underground arsenic storage vaults at Giant. Data pertaining to the exposure of employees to arsenic were received from both companies. These consisted of reports of surveys of airborne arsenic concentrations in the mills and of levels of arsenic excreted in the urine by employees. In addition, some data on levels of

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arsenic in employees' hair were available from surveys conducted on residents in the Yellowknife area by the Medical Services Branch of the Department of National Health and Welfare and by the United Steelworkers of America.

No surveys of airborne arsenic at underground workings have been reported, since the arsenic in the ore occurs as arsenopyrite and is insoluble[1]. The main occupational exposure to arsenic occurred in the mills.

During the 1965-66 investigation of the health status of inhabitants in Yellowknife made by A.J. de Villiers and P.M. Baker of the Occupational Health Division, Department of National Health and Welfare, urinary arsenic levels were determined for 53 mill workers and for 308 non-mill workers. No information was provided as to the numbers of mill workers or non-mill workers from each company.

The average urinary arsenic level for the mill workers was 0.0203 ppm (20.3 micrograms per litre of urine), whereas for the non-mill workers the average was 0.0110 ppm (11.0 micrograms per litre). Only one mill worker showed a level higher than 100 micrograms per litre; none of the non-mill workers exceeded this level. While the urinary arsenic levels tended to be higher for the mill workers than for the non-mill workers, excretion levels were within the normal range. For persons with no known exposure to arsenic, levels from 100 to 300 micrograms per litre have been reported in the literature as the upper limit. Rarely however were levels above 100 micrograms per litre found in the absence of occupational exposure or seafood ingestion.

No data were given on airborne arsenic levels in either mill in this report.

Giant Mill

Air samples were taken by the Occupational Health Unit, Medical Services Branch, Department of National Health and Welfare, in July, 1975, at 39 locations throughout the mill. All values reported were below 15 micrograms per cubic metre of air, well below the concentration of 50 micrograms (the standard proposed by the American Conference of Governmental Industrial Hygienists (ACGIH) for the protection of workers from the possible carcinogenic effects of concurrent exposure to arsenic trioxide, antimony trioxide and sulphur dioxides). A few spot samples for sulphur dioxide showed levels of less than 1 ppm, well below the ACGIH recommended level of 5 ppm.

The Giant mill was re-surveyed in July, 1976, at 81 locations. The highest airborne arsenic concentration reported was 47.8 micrograms per cubic metre, the next highest 35.6 micrograms per cubic metre. Antimony determinations were made at 29 locations, the highest value recorded being 4.1 micrograms per cubic metre, well below the ACGIH recommended standard of 50 micrograms per cubic metre. All sulphur dioxide concentrations were less than 2 ppm.

[1]The absorption of arsenic from arsenopyrite in the lungs or digestive tract has never been investigated.

Measurements of urinary arsenic excretion were carried out at Giant Yellowknife by the Department of National Health and Welfare on 16 employees in March-April, 1976, and on 24 employees in October-November, 1976. Most of those employees worked in the mill, around the roasters, the electrostatic precipitator and the baghouse; a number of supervisory staff and maintenance crew were included.

In the first survey, analyses were done by a private firm in Ottawa. Four of the 16 were reported to have urinary arsenic levels exceeding 100 micrograms per litre. Three of them were rechecked during the following month by Health and Welfare Canada and showed levels of 2.1 micrograms per litre or less. There remains some question as to the accuracy of these analyses.

In the second survey of 24 men all urinary arsenic levels were below 75 micrograms per litre.

A third series of 38 urine samples was collected in May 1977 and analysed by the Occupational Health Laboratory, Health and Welfare Canada, Ottawa. The highest urinary arsenic concentration was 51 micrograms per litre.

The urinary arsenic levels mentioned (with the exception of 2 or 3 in the first survey, the accuracy of which is in some doubt), are all within the normal range.

The urinary excretion data, in general, support the results of the air sampling surveys, that recent exposure to arsenical dusts prior to the urinary sampling was not severe. However, it should be noted that both air sampling surveys were done in July (1975 and 1976) when dust conditions in the mill may well have been better than average because of open windows, etc. It should also be noted that hair samples taken in May 1977 show higher levels of arsenic than normal (see below).

Cominco Con Mill

During July, 1975, three samples were taken by the Occupational Health Division, Department of National Health and Welfare, for determination of airborne arsenic at the Con mill. The levels found were quite low, the highest being 2.5 micrograms per cubic metre.

In July, 1975, air sampling was also carried out by the staff of Cominco. This report showed average arsenic concentrations as follows:
   a) Refinery, 3 locations, average 32 micrograms per cubic metre.
   b) Crushing plant (not operating), 3 locations, average less than 10 micrograms per cubic metre.
   c) Crushing plant (operating), six locations, average 45 micrograms per cubic metre.
   d) Mill, 12 locations, average 8 micrograms per cubic metre. More recent data are not available.

A survey of urinary arsenic levels was carried out in February 1972 by the staff of Cominco. There were 41 samples submitted. The average concentration for 7

mill workers was 186 micrograms per litre; for 4 shift bosses 88 micrograms per litre, and for 7 mechanical and trades personnel and for 13 staff 50 micrograms per litre. In all, 8 men had levels of 100 micrograms per litre; in three of these the levels ranged from 200 to 300 micrograms per litre.

Another survey of arsenic levels in urine was conducted in August, 1975, by Cominco. The 55 workmen covered in this survey included 12 mill workers, 14 mechanical and trades, 20 underground workers and 9 miscellaneous and office employees. Only 3 of the 55 had urinary arsenic levels exceeding 100 micrograms per litre; two of these were mill workers and one was an underground worker.

The most recent survey of urinary arsenic levels was done during February and March 1977. There were 213 samples submitted; the analytical work was performed by an independent laboratory in Vancouver. Of the 25 mill employees, four had levels between 100 micrograms per litre and 200 micrograms per litre, and one had a level of 235 micrograms per litre. Of the 34 mechanical and trades employees, 11 miscellaneous surface workers, and 20 office staff, none had levels above 50 micrograms per litre. Only two of 114 underground employees had levels exceeding 50 micrograms per litre, and these two had values between 60 and 80 micrograms per litre.

To summarize these data, for men employed at locations other than the mill all values of urinary excretion have been well within the normal range. The levels reported for the mill workers tend to be higher than those for the other workers, and in each survey a small number were at the upper limit of the normal range or just above it.

Though the Task Force has as yet only the July 1975 data on arsenic levels in air in the Con mill, the most recent urinary arsenic levels suggest that exposure to arsenic during March-April 1977 has probably not been more than moderately high in the mill, and low elsewhere. One approximation of the relationship between urinary arsenic levels and airborne concentrations has been given by K. Nelson of the American Smelting and Refining Company, who found that Y (airborne arsenic in micrograms per cubic metre) = 0.309 X (urinary arsenic in micrograms per litre). Thus, a urinary level of 180 micrograms per litre would suggest exposure to an airborne arsenic concentration of about 60 micrograms per cubic metre.

Further data on past airborne arsenic surveys were requested from the company. Unfortunately, the data available did not include samples taken within the mill, so that further information to assist in assessing the exposure in the mill is lacking.

Arsenic Levels in Hair

Arsenic absorbed into the body, whether by inhalation or by ingestion in water or food, is stored in the hair and nails where it may remain for many months after termination of exposure. Hair has also been shown to adsorb

arsenic on its surface from external contamination. Reports in the literature indicate a distinct difference of opinion between investigators as to whether external contamination can be completely removed by washing prior to carrying out the arsenic analyses. Professor R.E. Jervis, of the University of Toronto, who did arsenic measurements on the hair of a number of Yellowknife workmen and residents, has had long experience in this field. It is his opinion that nearly all external arsenic contamination can be removed by careful washing. His opinion is supported by an independent report by R.A. Smith of Edmonton who has succeeded in visually demonstrating the arsenic distributed across a transverse section of hair obtained from four Yellowknife residents.

Arsenic in hair has been used, in previous investigations, as an index of past occupational exposure. In persons not exposed to arsenic, the concentrations of arsenic in hair samples seldom exceed 1 part per million, though values up to 2 or 3 ppm have been reported. Higher values have also been found in persons eating shellfish and other sea foods which may contain relatively high amounts of arsenic.

In men occupationally exposed to arsenic, hair levels may range from less than 1 ppm to several hundred ppm or even higher. Levels of several hundred ppm are usual in persons with chronic arsenic poisoning, but such high levels may occur in men exposed at work without any obvious signs of chronic poisoning being found.

Two surveys of arsenic levels in hair of Yellowknife residents have recently been made. The first collection of hair samples was made in February of 1975 by the Medical Services Branch of the Department of National Health and Welfare. The 703 persons tested included workmen from mining companies, native people and non-native residents. The data supplied to the Task Force showed that 135 mine and mill workers were tested. Of these, nearly one-third had hair levels exceeding 10 ppm compared with 3.4% in the other residents. While a detailed table showing place of employment was not presented, the survey was reported to have shown a highly significant correlation of high levels of arsenic in hair samples and employment by the Giant Yellowknife: "the majority of persons with levels exceeding 10 ppm and all those above 50 ppm worked in the mill". The brief summary provided to the Task Force thus indicates absorption of arsenic by the Giant Yellowknife mill workers; a more detailed review of the data collected may provide further information of value to the Task Force in its assessment of the seriousness of this occupational exposure.

As part of this same study, clinical examinations were conducted in June 1975 on 50 persons who had hair levels above 10 ppm in the February survey, and on 8 others, 6 of whom were Giant Yellowknife employees. The total number of mill and mine workers was 49.

The examining team included two internists and a general practitioner. Dr. Otto Schaefer of Medical Services Branch, Department of National Health

and Welfare, reported that "one is impressed by the absence of pathological findings typically to be found in chronic systemic arsenic poisoning". However, 17 employees gave a history of having had an arsenic rash at one time or another; all were mill employees with exposure around the roasters, the electrostatic precipitator or the baghouse. Two mill employees had rashes compatible with the effects of long arsenic exposure. While 18 men had abnormalities in one or more liver function tests and four had enlarged livers, in none could other signs of chronic arsenic toxicity be found. Five men were found to have high arsenic levels both in hair and urine and to have a history and/or clinical or laboratory findings suspicious of mild chronic systemic effects of arsenic. Four of them worked around the roasters or electrostatic precipitator.

Six of the 57 persons included in the clinical study had hair levels exceeding 100 ppm (the mean for these was 203 ppm). The majority, if not all, of these six were occupationally exposed to arsenic.

In the second survey of arsenic levels in hair, samples were collected by the United Steelworkers of America in September 1976. At the same time, hair samples were collected from Indian children living in Yellowknife, on Latham Island, and in Whitehorse. The analyses were carried out by Professor R.E. Jervis of the University of Toronto. Hair specimens from 20 Giant Yellowknife employees (including 16 mill workers) had an average concentration of 72 ppm; in two the level ranged between 100 and 199 ppm, and in three it ranged from 200 to 278 ppm. Among 12 steelworkers at Whitehorse, with no exposure to arsenic, there were no hair levels exceeding 1 ppm arsenic.

The survey of hair samples taken by the Steelworkers more than a year after the survey by the Department of National Health and Welfare thus confirms the findings of that survey. While some of the raised arsenic levels found may have been due to external contamination, it would appear that most, if not all, of the increase has been due to increased intake of arsenic through occupational exposure. Review of the literature does not indicate sufficiently close correlation between occupational exposure and the arsenic content of hair to enable one to assess the severity of the exposure from the hair levels reported, other than in very general terms.

In May 1977 hair samples were collected from 38 employees of the Giant Yellowknife mine. The average arsenic concentration in the unwashed hair was 34.4 ppm. Two values exceeded 200 ppm (212 and 275 ppm). One of these was a roaster operator, the other worked in the baghouse. All other values were 90 ppm or less. Sixty percent of the 38 unwashed samples had levels of 10 ppm or more.

Sufficient hair was available on 32 specimens to carry out analyses after washing the hair. The average arsenic concentration was 26.3 ppm (compared with 30.1 ppm on the same 32 samples unwashed). The highest value in the

unwashed hair (275 ppm) dropped to 180 ppm after washing. There was insufficient hair to test the second highest sample after washing.

It is of interest to note that of the 32 samples in which before- and after-washing analyses were made, the washed hair values were lower in only 17, higher in 10, and the same as in the unwashed samples in 5.

The results leave considerable confusion as to the efficacy of the washing procedure. Since some of the values increased after washing, they also cast some doubt on the accuracy of the laboratory analyses. There appears to be no doubt, however, that as a group the arsenic hair levels in these workmen are considerably higher than normal. These same 38 men had urinary arsenic excretion values within the normal range. One must conclude that either the hair samples were all contaminated by adsorbed dust which could not be removed by the washing procedure used, or that the hair levels reflect past absorption of arsenic during the preceding winter months. This in turn lends support to the opinion of the Task Force that air sampling in the mills at Yellowknife should be done seasonally to determine if exposure levels are higher during the winter. We also consider that more research is needed into the question of adsorbed versus systematically deposited arsenic in hair.

As indicated, in addition to a history of skin rashes, a number of clinical and/or laboratory findings suspicious of mild chronic arsenic intoxication were reported by Dr. Schaefer in the examination of the mill workers. However, the signs and symptoms found were not those typical of chronic poisoning, such as neuritis, hyperkeratosis, hyperpigmentation and anaemia, and the abnormal findings could have been due to other causes such as alcohol, viral diseases, etc. The study by de Villiers and Baker in 1966 also reported a high frequency of skin rashes probably due to skin contact with arsenic. In addition, they found a high incidence of respiratory diseases and of electrocardiographic abnormalities among men in the community, but they were not able to show that these were specifically related to arsenic exposure.

While signs and symptoms characteristic of chronic arsenic poisoning have not, as yet, been reported among the mill workers, contact skin rashes commonly occur. Any further clinical assessment of the effects of long term occupational exposure to arsenic should include a control group matched for age, ethnic origin, and residence in Yellowknife. A second control group from a centre other than Yellowknife would also be desirable. To be worthwhile, the study would have to concentrate on rather subtle effects, such as electrocardiographic or electromyographic abnormalities, and should attempt to show whether the frequency of such changes are dose-related.

Consideration has been given to a review of the lung cancer and skin cancer experience of older mill employees or others exposed to arsenic dust or fumes. In other studies, workmen exposed for many years to arsenic in the form of roaster fumes and dust, and in the manufacture and spraying of arsenical insecticides,

have been found to have a higher-than-normal risk of skin cancer and of lung cancer, usually developing 20 or more years after first exposure. In most instances, the concentrations of airborne arsenic were high, and in many of the exposures other contaminants were present such as antimony trioxide, sulphur dioxide, etc. The increased risk of skin cancer and lung cancer in such situations is now generally accepted. Though there has been practically no success in demonstrating experimentally that arsenic, even in large doses, will cause cancer in animals, in one preliminary study recently reported from Japan arsenic trioxide did increase the effect of one well-known carcinogen, benzopyrene, in producing lung cancer in rats, i.e. it acted as a cocarcinogen.

An attempt has been made to determine what company records were available for conducting an epidemiological study of employees and former employees who had had exposure to arsenic in the mills. Because of the long latent period (more than 20 years) between first exposure and the development of cases of lung cancer, it was hoped that records might be available going back to the 1940's or 1950's.

At Giant Yellowknife mine, all personnel files and pay records prior to 1969 had been destroyed by water when a basement flooded. The company had information on only 16 men with arsenic exposure who had left the company. While it appears, therefore, that an epidemiological study at this mill is not possible at present, the Task Force considers that an effort should be made to determine whether the 16 former employees are alive or have developed lung or skin cancer.

At Con mine, all records of employees who have left were transferred to the very large central files at the Cominco plant at Trail, B.C., August, 1977. Employee numbers indicate at which mine or smelter a man was first employed by the Company. They are not changed if he moves to another mine or smelter within the Company. Thus an employee with a Con Mine number might have had most of his services at Kimberly or a man with a Trail number could have had substantial exposure at Con. An individual review of each of the approximately 100,000 personnel records would be necessary to identify the 200 or so men who have been employed at the Con Mill at Yellowknife since 1938. The index to these files is maintained at Vancouver. It was the opinion of the Task Force that an inordinate amount of effort and expense would be involved in attempting to identify former Con mill workers in the more than 100,000 personnel records held by the Company, and then to attempt to trace these former employees to see if any had died of lung cancer.

The Task Force is agreed that for the future evaluation of the lung cancer hazard the companies should maintain employment records showing arsenic exposure for the existence of their present operations. When these cease, the records should be maintained by the parent company in an accessible form for the next 30 years or more.

Table V
Cancer Deaths in Yellowknife and Whitehorse, 1964-73

Municipality/Cancer	Male		Female	
	Obs	Exp	Obs	Exp
Yellowknife				
Lung Cancer				
1964-68	4	2.4	0	0.2
1969-73	6	3.3	2	0.5
1964-73	10	5.7	2	0.7
Other Cancer				
1964-68	5	8.3	6	4.8
1969-73	8	8.8	11	6.9
1964-73	13	17.1	17	11.7
Whitehorse				
Lung Cancer				
1964-68	6	3.0	3	0.3*
1969-73	9	6.3	1	0.9
1964-73	15	9.3	4	1.2
Other Cancer				
1964-68	14	10.0	6	6.9
1969-73	17	17.4	10	13.8
1964-73	31	27.4	16	20.7

* $P < 0.05$ based on assumption that observed value is a Poisson variable

Obs = observed deaths among residents of municipalities including those deaths registered in the provinces.
Source: Health Division, Statistics Canada.

Exp = expected number of deaths based on 1966 and 1971 populations by age and sex and the 1965-67 and 1970-72 average annual age-, sex-, cause-specific Canadian mortality rates.

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The Task Force has also looked at the possibility of a long-term follow-up study of former residents of the city of Yellowknife. Again, it does not appear practical since the necessary information (names, birthdates, social insurance numbers, etc.) are not available. Statistics Canada and Dr. D. Wigle of the Laboratory Centre for Disease Control, Health and Welfare Canada, have provided information showing an increased mortality from lung cancer in both males and females in Yellowknife in each of the two time periods, 1964-1968 and 1969-1973. However, similar increases were found in the residents of Whitehorse in the Yukon, where there is no known environmental contamination by arsenic (see Table V). It should also be noted that a larger proportion of the lung cancer deaths in Yellowknife occurred in the native population than was the case in Whitehorse (Table VI). The high incidence of lung cancer among the Inuit, particularly the women, quite unrelated to arsenic exposure, has previously been reported by Dr. Otto Schaefer et al in the *Canadian Medical Association Journal* of 21 June 1975. It is apparent that much of the increase in mortality from lung cancer in the Northwest Territories (and hence in Yellowknife) is attributable to the high incidence in the native groups.

Table VI
Cancer Deaths By Ethnic Group, 1964-1973

	Cancer		
Place/Ethnic Group	Lung	Other	
Yellowknife			
Indian, Eskimo, Métis	5	4	
			p = 0.06
Other	6	23	
Whitehorse			
Indian, Eskimo, Métis	1	5	
			p = 0.42
Other	18	40	

Notes: (1) Probabilities are based on Fisher's exact test.

(2) Does not include 7 deaths for which ethnic group was not recorded on the death certificate.

Cancer Section
Bureau of Epidemiology   January 26, 1977

IV. Arsenic and the Health of the People of Yellowknife

Discussion of Previous Studies

There have been four medical surveys of the people of Yellowknife. Three have been conducted by the Department of National Health and Welfare and one by the United Steelworkers of America and the National Indian Brotherhood.

In 1951 the first reported medical survey was carried out in association with an environmental survey. Two hundred and thirty school children were given a physical examination and urine arsenic determinations were done on a sub-sample (6). The urine survey confirmed the absorption of small amounts of arsenic. The physical examinations evidently revealed nothing unusual. As part of this study hospital admissions for a five-year period from 1948 to 1952 were reviewed. High admission rates for diseases of the skin and cellular tissues and diseases of the respiratory system were noted but these findings evidently were not compared with those of other northern areas. In the 1950's most northern settlements had high rates of skin and respiratory infections.

A quarter of a century after the completion of the 1951 survey it can be concluded that no actual arsenic poisoning was found but there was evidence of some arsenic absorption in at least some children.

The second medical study was undertaken about 15 years after the first (1965). This study was considerably more extensive. Morbidity and mortality data were reviewed in considerable detail. A complete clinical examination was carried out on three hundred and sixty-nine male residents of Yellowknife. Three hundred and sixty-one urine samples were taken (53 from millworkers and 308 from non millworkers). With the exception of one millworker all were considered to be within the normal range of 0.003 to 0.150 mg/litre (6).

This study, reported by A.J. de Villiers and P.M. Baker, was extensive and well carried out. Comparisons were made between Yellowknife mortality and morbidity findings and corresponding rates from other parts of Canada. No attempt was made to acquire a matched control sample in another northern town.

The summary given by the authors of this extensive study is as follows: — "Evidence of an association between arsenic exposure and a high prevalence of skin lesions among individuals occupationally exposed to contact with arsenical dusts was found. Ingestion of arsenic appeared to play only a minor role, if any.

There is high incidence of acute respiratory disease (in males) in the Yellowknife community together with a high prevalence of chronic non-specific respiratory disease. It is possible that the irritant action of inhaled arsenical dusts may have had a minor contributing role to play in the aetiology of these conditions but this could be of less importance than other environmental factors, for example, the harsh climate in association with other insults on the respiratory system such as smoking.

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The high incidence of deaths and out-patient visits due to accidents, poisonings and violence and the high incidence of hospital admissions for mental, and particularly psycho-neurotic disorders appear to reflect the severity of non-specific stress factors inherent in the transplantation of a sizeable European population to a new environment characterized by isolation, severe climatic conditions and lack of accustomed facilities.

Abnormal electrocardiographic changes and certain other neurological findings were found more frequently than would have been expected. The significance of this occurrence cannot be explained at this time".

This report was the first to demonstrate a clear difference between the people employed in the mine mills and the rest of the population. It is also of possible significance that the report noted that "abnormal electrocardiographic changes and certain other neurological findings were found more frequently than would have been expected". There are a number of possible explanations for these observations in the male population of Yellowknife. The Task Force considers that this particular observation should be followed up in detail using a newer clinical tool — electromyography.

The third survey in Yellowknife was conducted in 1975. This was not planned as an epidemiological survey but consisted of a study of hair samples. Seven hundred residents volunteered to have their hair tested. The study was primarily directed towards three groups in the community: mine-millworkers, native people and "long-term residents" (4).

63 people from this group (9% of the total 700) had more than 10 ppm arsenic in their hair. 57 of this group were given an extensive and thorough clinical examination by Dr. O. Schaefer and two associates. Some clinical findings were noted but these were not considered as signs of arsenic damage. The Task Force considers that Dr. Schaefer and his group did an excellent clinical assessment of this group. As mentioned, this program was not intended as an epidemiological study but was intended to identify individuals who might be in poor health due to arsenic exposure.

Table VII, adapted from the Technical Data Summary, Arsenic in the Yellowknife Environment (8), gives the results of this survey:

Table VII
Arsenic in Human Unwashed Hair — Yellowknife NWT 1975

	0 — 4 ppm		5 — 10 ppm		over 10 ppm		Total	
	#	%	#	%	#	%	#	%
Mine Mill Workers	61	45.2	30	22.2	44	32.6	135	100.0
Other Residents	516	91.3	30	5.3	19	3.4	565	100.0
Total persons tested	577	82.4	60	8.6	63	9.0	700	100.0

Here again the difference between the mine-millworkers and the general population is demonstrated.

The hair arsenic levels, taken from the above source, for 57 of the 63 people classed as over 10 ppm is given in the following table:

Table VIII
Washed Hair with 10 ppm Arsenic or More when Unwashed

Arsenic in hair (ppm)	Number of persons #	%	Arsenic in hair mean value, ppm
<10	10	17.6	7.1
10 — 49	34	59.6	21.2
50 — 99	7	12.3	66.7
>100	6	10.5	203.0
Totals	57	100.0	43.5

Unfortunately this table does not distinguish between the three groups included in the study but evidence provided to the Task Force from subsequent studies would support the premise that this group contained a disproportionate number of mine-millworkers and native people.

The fourth survey of arsenic in the Yellowknife population made available to the Task Force was completed in January 1977. This was the first study from the area to use a control group from another comparable area. This study was initiated by the National Indian Brotherhood and the United Steelworkers of America (116). The analytical work on the samples of hair was done by Dr. R.E. Jervis, Professor, Nuclear and Radiochemistry, Department of Chemical Engineering and Applied Chemistry, at the University of Toronto.

This study was confined to two groups in the population, native people and members of the United Steelworkers. Previous studies had defined mine-millworkers as being at greatest risk of arsenic exposure.

The study group was small but significant and important. Fourteen Indian children and twelve Steelworkers in Whitehorse were the control group. The test group included 47 native people and 20 mine-mill-workers from Yellowknife. There is no indication of randomization in choosing the sample. The group of 47 native people from Yellowknife is the largest sample available to the Task Force and represents about 5% of the native population in the area. All 47 are identified as children. The sample thus represents about 11% of the native child population of the Yellowknife area.

The overall results of the study are given in the following table supplied to the Task Force by Dr. Jervis:

Table IX
Arsenic Hair Levels
Mine-Mill Workers and Native Children, Yellowknife and Whitehorse, January 1977

	Yellowknife (median concentration, ppm Arsenic)	Whitehorse (controls) (median concentration, ppm Arsenic)
Mill and Mine workers	32.5	0.38
Indian children	3.0	0.23

The highest value in any of the controls was 0.66 ppm (in Steelworkers). The highest value in any of the Yellowknife mine-millworkers was 278.0 ppm; the lowest were 1.8 ppm (an ex-worker) and 4.5 (presumably an active worker in the mill).

The highest value in any of the Yellowknife native children was 28 ppm and two children were below 1 ppm.

The complete results of this survey from Yellowknife are given in Table X.

Table Xa.
Arsenic in Human Head Hair Controls, Source of Sample, Whitehorse
Date: December, 1976

Sample No.	PPM As wet wt.	Sample No.	PPM As wet wt.
1	0.37	14	<0.38
2	<0.21	15	<0.61
3	<0.45	16	<0.37
4	<0.16	17	0.51
5	<0.56	18	0.39
6	<0.25	19	0.53
7	0.14	20	0.19
8	0.22	21	<0.65
9	0.24	22	<0.37
10	0.30	23	<0.35
11	0.24	24	0.52
12	0.49	25	<0.66
13	<0.17	26	0.30

R.E. Jervis, B. Tiefenbach, University of Toronto.

CONTROL GROUPS
Sample No.   1 — 14   Indian children from Whitehorse

15 — 26   Steel workers from Whitehorse

TABLE Xb.

Arsenic in Human Head Hair Exposed

Source of Sample: Yellowknife   **Date: December, 1976**

Sample No.	PPM As wet wt.	Sample No.	PPM As wet wt.
27	9.0	61	5.0
28	200.0	62	13.5
29	6.7	63	5.3
30	20.0	64	3.0
31	88.0	65	1.5
32 (ex-worker)	1.8	66	1.5
33	39.0	67	7.0
34	4.5	68	6.0
35	102.0	69	3.0
36	168.0	70	10.3
37	9.0	71	3.0
38	99.0	72	8.3
39	278.0	73	2.0
40	6.5	74	6.0
41	26.0	75	4.3
42	89.0	76	2.5
43	11.0	77	0.95
44	67.0	78	5.8
45	203.0	79	4.0
46	16.0	80	3.7
47	28.0	81	3.2
48	3.0	82	0.8
49	11.0	83	7.0
50	3.0	84	2.0
51	5.3	85	0.65
52	3.8	86	1.0
53	4.0	87	6.2
54	1.7	88	2.5
55	1.5	89	1.7
56	2.2	90	1.5
57	1.5	91	13.0
58	7.2	92	1.7
59	3.0	93	0.8
60	12.2		

R.E. Jervis, B. Tiefenbach, University of Toronto.

  Sample No.   27 — 46   Smelter workers from Yellowknife
                47 — 93   Indian children from Yellowknife

   The foregoing is a review of the four major surveys that preceded the CPHA Task Force on Arsenic. The most detailed and complete was done by de Villiers and his associates in 1965. The hair studies are recent and, taken together, give an indication of the extent of arsenic exposure in the population in Yellowknife.
   From the data now available it is evident that workers in and near the ore roasting process are at risk of exposure to arsenic compounds. Much of this report by the Task Force is devoted to this problem and its amelioration.
   The other group that is obviously exposed is Indian children. The observed hair levels in Indian children are lower than in industrially-exposed individuals. However, the levels are considered sufficiently high to require further investigation and continuing surveillance.
   The National Indian Brotherhood categorized 18 children by residence.

TABLE XI.
Arsenic Hair-Levels in Native Children by Residence, January 1977

Sample No.	Detah (ppm)		Latham island (ppm)
1)	6.0	8)	6.2
2)	0.9	9)	19.2
3)	1.8	10)	2.9
4)	3.0	11)	22.5
5)	3.8	12)	3.7
6)	3.6	13)	1.3
7)	4.8	14)	5.6
		15)	0.45
		16)	1.6
		17)	8.4
		18)	2.4

Note: Sample numbers are not connected with those in the previous Table.

   It is not surprising that arsenic levels in hair tend to be higher in children living on Latham Island. The unexpected feature of this table is that children from Detah had elevated levels of hair arsenic. The settlement of Detah is the inhabited area furthest from the present source of arsenic emission. Only one of the children from Detah had a level low enough to be interpreted as not elevated. One other hair level might be interpreted as within the limits of error of the test. The assumption that two of these samples were not elevated would be justified if there was no significant arsenic reservoir known. The Task Force has made the assumption that all the children from Detah show evidence of contact with arsenic. As a result of this information the Task Force recommended additional

environmental monitoring of the Detah environment and of fish from Yellowknife Bay. The results of this monitoring confirm that earlier results were representative: the readings were very low and well within acceptable limits (Appendices A & B).

As noted in the Interim Report, boys tend to have higher hair arsenic levels than do girls. Children tend to have higher levels than adults. This same phenomenon has been noted in other areas with increased environmental levels of either arsenic or lead.

Individuals with hair arsenic levels in excess of 5 ppm. in the general population of Yellowknife have not been reported as having significant arsenic urine levels. This pattern in the non-industrially exposed population might be explained in various ways. One obvious explanation would be that very few urine samples have been tested for arsenic. A second possibility is that arsenic exposure is intermittent and since excretion of arsenic in the urine is quite rapid elevated arsenic in hair can exist in the absence of urine arsenic if the exposure has stopped. A third explanation has been suggested by some observers. In children, and particularly boys, hair arsenic could be acquired by direct contact with the arsenic-containing environment. Dust, earth and airborne fall-out could contaminate the hair and scalp directly and, with time, the arsenic compounds could become bonded to the hair. This is a comforting theory which cannot be proved or disproved at this time. It is prudent to reject this theory until all possibility of hair contamination by arsenic by a systemic route is ruled out.

To determine if the elevated arsenic levels in hair are acquired externally or systemically and continuously or intermittently the Task Force has asked the Northern Research Unit, Medical Services Branch, to obtain hair from up to five children with known elevated hair arsenic (levels in excess of 15 ppm would be desirable). If a small sample of complete hairs, from their roots to their tips, is obtained it will be possible to do serial determinations along the full length. If the arsenic is evenly distributed along the shaft of the hair the exposure is probably continuous and could be systemic or external or both. If the arsenic is irregularly distributed along the hair the exposure is probably intermittent and systemic. Systemic exposure results in a distribution of arsenic across the shaft of the hair. This distribution of hair arsenic can be checked by x-ray fluorescence in a methodology demonstrated by R.A. Smith at the University of Alberta (117).

Little information is available on the non-native children of Yellowknife. The 1951 study concentrated on schoolchildren but since then the emphasis has shifted, with reason, to millworkers and native people.

Current Situation

The present situation in the Yellowknife area is as follows:

The public water supply is arsenic-free. Some of the local lakes contain a relatively large amount of arsenic, but these lakes are either tailings ponds and

inaccessible to the public, contaminated by sewage, or at a level where they would be undesirable as a regular potable water source but of no danger when used for water contact sports during the short summer season. Most people, but not all, get their water from the safe municipal water system.

Arsenic in the air and suspended particles are at acceptable levels. The "dust" season is relatively short.

Fish, except from one particular lake, are satisfactory for food.

Vegetables are satisfactory if washed before use. Berries, available for a short period, are safe if washed.

Soil is high in arsenic but normally not ingested except on unwashed vegetables or possibly directly by children.

Snow does contain arsenic and could be dangerous if used as a source of drinking water.

A review of this list would suggest that there are few possibilities of acquiring arsenic outside of the work-place in Yellowknife. For the great majority of the people in the area this seems to be the case. They evidently have about the same arsenic intake as people in other industrial towns in Canada.

For perhaps 10% of the population this is not so. Indian people and particularly Indian children are acquiring an increased arsenic load. Some non-native people may also be acquiring increased arsenic. Some workers have increased arsenic levels at least some of the time.

Improvement can be brought about in this situation. The Task Force has made a number of recommendations:

1) To ensure that everyone has an arsenic-free water supply.
2) To markedly decrease the emissions and effluents of arsenic compounds from the mine mill setting.
3) To carry out ongoing environmental and health monitoring to detect any changes in the situation.

The pattern in Yellowknife has been to have a survey and a review of the situation every ten to fifteen years. Each of these events seems to have produced an improvement in one or more aspects of the pollution problem. It is surprising that at the time of the excellent review by de Villiers and his associates ongoing programs were not put into effect. These programs should have taken the form of regular public health and industrial hygiene practices. Industrial practices have been modified and improved but the provision of public health and industrial medical monitoring and practice have lagged.

Medical surveys without exception have remarked on the prevalence of skin and respiratory infections. No ongoing follow-up of these facts is evident.

Arsenic compounds have been shown to have an anti-immune effect. The resulting lowering of resistance has been associated with increased skin disease and higher respiratory infection rates. No serious effort seems to have been made

to compare morbidity rates in Yellowknife with other comparable northern communities.

There is no question that, under certain conditions, arsenic compounds are causally related to cancer. There has been little evident effort to follow trends over time in Yellowknife and other areas. Some valuable observations on cancer in northern peoples have been made by Dr. Schaefer in the course of his work; none of these relates to Yellowknife (115).

The small number of deaths (and even smaller number of cancer deaths) and mobility of the population in Yellowknife make meaningful studies difficult. There are few existing health or population records available. Prospective cancer studies have never been initiated.

Dr. de Villiers made some very pertinent observations on electrocardiographic changes in some cases. These observations have not been followed up.

The Task Force deplores the previous pattern of intermittent surveys followed by periods of relative inactivity. Future action must take the form of continuous surveillance and corrective action when and if necessary.

The evident absence of clinical arsenic toxicity in Yellowknife except among some industrial workers is encouraging. Ongoing monitoring of the human population is needed to determine if there are any subclinical or preclinical effects from the arsenic in the environmental reservoir.

The Task Force recommends that the health services of the Northwest Territories be organized in the same manner as provincial public health jurisdictions. The organization to have two major components: community health services and environmental health services. Ongoing Health monitoring could be conducted and reported by such an organization.

Among some of the manifestations that could be considered as signs of arsenic toxicity are gastrointestinal symptoms and skin pigmentation, as well as more non-specific symptoms such as bronchopulmonary disease, chronic coryza, lip herpes, chronic cough, some cardiovascular manifestations with increases in Raynaud's syndrome, acrocyanosis, and blood-pressure changes. None of these manifestations has been reported from Yellowknife.

In high arsenic dosages over long periods neurological changes are described, usually as a late manifestation. Paresthesiae and weakness were the usual changes noted (10). A recent report from Czechoslovakia suggests an increased frequency of minor hearing loss may be associated with environmental arsenic contamination (69).

Most studies on clinical toxicity from arsenic indicate that such signs and symptoms usually occur only when hair arsenic levels are about 20 ppm or higher. Recent studies indicate that it is possible to detect slowing of the speed of nerve impulses when the hair arsenic levels are below 20 ppm and perhaps at levels as low as 1 ppm (118). Since there is considerable variation in the normal

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speed of nerve impulses between individuals, changes of this type can only be assessed in population studies.

All these observations can be used to monitor the possible effect of low doses of arsenic on the population of Yellowknife. At the present time there are no coherent morbidity studies in the area or in the Northwest Territories. No serious attempt has been made to determine if the people of Yellowknife have the same or different morbidity experience than people in other parts of the Territories or in the rest of Canada.

Records have been kept of births, stillbirths, and deaths. In a young and frequently transient population these data have not been as clear indicators of the health of the population as they might be in an older, larger, and more stable population.

Regular morbidity monitoring has long been a routine part of public health practice. Such simple observations as absenteeism from school can often be used effectively to gain an insight into the morbidity of children in the community.

The fact that arsenic in reasonably high doses taken by mouth can lower the immune response to infections, and produce detectable vascular and neurological changes makes population monitoring over time a useful procedure.

The question whether there is any real difference in the health of the general population of Yellowknife and of other communities such as Fort Smith, Hay River, or Whitehorse should be investigated. It is probable that it would take a minimum of five years to answer this question. Populations in all these areas are relatively small. Common epidemics, such as influenza, the common cold, or chicken-pox move slowly through sparsely-populated regions. All territorial vital and morbidity indices show great variation from year to year. This is a statistical phenomenon of small numbers.

Organizational Requirements for Health Monitoring

In order to make possible proper health monitoring, the health services of the Territory should be brought under one medical health officer, responsible to the Territorial Government. This is almost the case at the present time. Yellowknife and Fort Smith are exceptions to the rule. Each has its own medical health officer. In the case of Yellowknife the activities of the city health services in health monitoring have been largely neglected. The present health officer of the Northwest Territories is responsible to the Federal Government.

The Municipal Councils of Yellowknife and Fort Smith should contract to have the territorial health officer as the municipal health officer. Assistant health officers, under the general coordination of the territorial health officer, can be appointed on a part-time basis where and when required. In the vast majority of areas in Canada (and elsewhere) it has been found more satisfactory to have medical health officers on a full-time basis and not employed in the private practice of medicine. Except for a few rare and notable exceptions, part-time

medical health officers who also must engage in private practice cannot, or do not, give the type of service the public should receive.

The population of the Northwest Territories is smaller than that in most health unit jurisdictions in the rest of Canada. The vast distances between the populated centres and the unique problems affecting health compensate for the small population.

The Task Force recommends that all the Territory, including Yellowknife and Fort Smith, be administered by a single health organization reporting to the Territorial government. Within the same overall organization there should be an environmental health agency for the entire Territory. All public health inspectors, occupational health workers, public health nurses and similar health workers should be included in this organization.

Such an organization does not infringe on the rights of local municipal councils or school boards or administrations. Within their jurisdictions the overall "Health Organization" will serve their needs and at the same time bring a continuity of basic services to the whole region.

Without such an overall administrative arrangement the type of ongoing day-to-day health monitoring (both personal and environmental) with comparison between regions and populations is difficult to carry out. Without an overall administrative jurisdiction measures to correct threats to health as soon as they are detected cannot be effective. Long-term trends in morbidity and mortality patterns will not be detected if an ongoing program is not provided.

The public health arrangement recommended by the Task Force should not be set up as a full "provincial" health department with all specialties permanently staffed. The numerous consultants and specialists required by the average health unit and provided by a provincial health service can, in the case of the Northwest Territories, be obtained for example from the Department of National Health and Welfare through its various branches and agencies or from an adjacent province.

With the organization for morbidity monitoring in place the Task Force recommends that a continuous program be set up. It should include the following elements:

1) Recording and comparing the age-sex-specific hospital admissions in the major hospitals in the region.
2) Recording and comparing the visits to nursing stations by age, sex, and cause.
3) Recording and comparing school absenteeism with periodic studies to determine the proportion of absences due to illness and the nature of the illness.
4) Recording and investigating infectious disease epidemics with special note being made of differences in attack rates and case-fatality rates.

All of these procedures are normal public health practice and many are

currently the practice in the Territory. More extensive ongoing study of morbidity data is necessary.

Monitoring practices specific to Yellowknife and control communities should begin at once.

Additional hair monitoring coupled with urine studies have begun in Yellowknife. These should be repeated at regular intervals in future years to check that arsenic levels remain low or decline. The cause of any increase or continuously high levels must be determined and immediately corrected.

Immediate and short term monitoring

(a) Additional hair sampling in the general child population. As noted previously, children in other areas of above-normal environmental arsenic or lead show a greater tendency to have elevated levels of the material in their tissues. This phenomenon has only been demonstrated in Yellowknife in native children. This same tendency may exist in the general child population.

(b) Serial testing of hair from children with known hair arsenic levels to determine if there is variation indicating systemic arsenic uptake of a seasonal or variable nature.

(c) Testing of hair and urine from anyone (more particularly children) admitted to hospital for any cause. This should be continued periodically in future years. We do not recommend that tests for arsenic should be "routine" for every person falling ill in Yellowknife, but an awareness that the presence of arsenic might be an incidental factor in some patients and a causal factor of the illness in a few should be an integral part of medical practice in Yellowknife for some time into the future.

(d) Review of hospital admissions by cause in Fort Smith and Hay River for comparison with comparable data from Yellowknife. It is anticipated that the populations and lifestyles of the three communities are sufficiently similar to minimize many of the variables in morbidity.

(e) Electromyographic studies of nerve conduction times. This is a safe, essentially painless procedure. It may prove of some assistance in establishing the extent of arsenic morbidity in industrial exposures. Its greatest value may be as a very sensitive tool in measuring any differences between the people of Yellowknife and other areas in the Territories. Unfortunately arsenic is only one of many factors that may alter nerve conduction time. Heavy metals, nutritional differences, metabolic diseases, injuries, and heavy alcohol consumption are among the factors that can slow nerve conduction time. If however there is a significant variation in nerve conduction between two populations having similar lifestyles, many of the variables can be minimized and arsenic may emerge as the chief factor. Electromyographic studies are being carried out in Yellowknife and Hay

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River. The program is discussed in detail in Appendix C. Monitoring arsenic levels should include electromyographic studies done as part of the medical examination on some people being admitted to the Yellowknife Hospital. Electromyography on an ongoing basis may prove useful in individual health programs.

Other Possible Monitoring Programs:

a) Minor vascular changes in individuals are very difficult to detect. Considerable normal variation exists in blood-pressure levels and heart rate. Raynaud's syndrome is not unusual in the general population and, in most cases, is more of a curiosity than a disability. It is possible to compare populations but the results might be equivocal and the monitoring would not be as sensitive as the nerve conduction studies.

   Raynaud's syndrome manifests itself in white numb fingers or fingertips after exposure to cold. It is not the same as frostbite but to the uninitiated observer the two conditions might appear similar. A simple monitoring program could be conducted on children exposed to the usual winter temperatures of the Northwest Territories in the normal course of their travel to school, or at play. The rate of Raynaud's syndrome in children of specific ages arriving at school on an average winter day could be compared between Yellowknife, Hay River, and Fort Smith. It is quite possible that the syndrome would not be observed in any of these places. A preliminary set of observations on Yellowknife children would confirm the presence or absence of this condition in school children.

   It is possible to test for Raynaud's syndrome under controlled conditions. Such a survey would only be justified if "naturally-occurring" Raynaud's syndromes are prevalent in Yellowknife and more prevalent there than in other areas.

   It seems extremely unlikely that the arsenic levels already observed in Yellowknife would be high enough to produce this condition in greater numbers than normally observed.

b) The cytological examination of sputum in people over 40 years of age or after 15 years of residence in Yellowknife has been suggested. It is a means of detecting early or precancerous changes in the respiratory tract. Laboratories are available in Canada to carry out this clinical test. If the procedure proves of value in the occupational surveillance program, consideration can be given to extending it to high-risk groups (e.g. smokers) in the general population.

c) Sweep test audiometry should be conducted at intervals throughout the school experience. Any hearing loss detected should be correlated with the arsenic status of the child.

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The long-term effects of arsenic should be looked for in ongoing monitoring in the same individuals. It is for this reason that the Task Force is recommending periodic examination, electromyography, and laboratory examinations of people in exposed work settings. Similar, but less intense, programs must be available to the general population.

Appendices

Appendix A

Arsenic Levels in the Edible Muscle of Freshwater Fish§

Species	Lake	Area	Year	Samples	Mean ppm Arsenic	Range in ppm Arsenic	Mean Wt. Fish (kg)
Whitefish	Great Slave	Yellowknife Bay	1972	—	0.31[1]	—	—
			1974	28	<0.40	<0.40-<0.40	0.90
			1975	55	0.22	<0.15- 0.30	0.86
			1976	14	0.20	0.02 0.61	0.94
			1977	4	0.36	<0.20- 0.77	1.26
N. Pike	Great Slave	Yellowknife Bay	1972	—	0.44[1]	—	—
			1976	5	0.24	0.09- 0.47	1.87
			1977	19	0.26	0.14- 0.67	1.31
White Sucker	Great Slave	Yellowknife Bay	1974	1	<0.40	—	1.28
			1976	7	0.16	<0.10- 0.28	1.05
			1977	1	0.82	—	1.24
Maria	Great Slave	Yellowknife Bay	1975	1	0.32		1.41
Round Whitefish	Great Slave	Yellowknife Bay	1977	14	0.14	0.07- 0.23	0.51
Inconnu	Great Slave	Yellowknife Bay	1977	1	0.82		1.24
Whitefish	Great Slave	Detah Village	1975	6	0.19	<0.15- 0.30	0.78
			1976	9	0.04	0.02- 0.10	1.01

CONTROL AREAS

Whitefish	Great Slave	Wool Bay	1975	24	0.19	0.04- 0.32	0.81
			1976	6	0.40	0.28- 0.64	0.94
	Athabasca R.		1975	133	0.05^2	—	0.74
N. Pike	Split, Manitoba		1976	9	0.24	0.17- 0.27	4.00
	Athabasca R.		1975	126	0.03^2	—	1.14

§Fisheries and Environment Canada, November 16, 1977

[1]Source: (119)
[2]Source: (120)

NOTE: In comparing mean levels of arsenic artificial differences between years may be caused because of inconsistencies in the lower quantitative detection limits among different years and sample batches.

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The actual final results are not expected to exceed the values indicated below but may in fact be less than those presented:

TABLE XIII

Arsenic Levels in Edible Muscle of Freshwater Fish
Preliminary Data, 1977

Species	Area	Sample	Arsenic Content PPM
Whitefish	Detah	2	<.10
Whitefish	Detah	1	.15
Whitefish	Detah	1	.11
Whitefish	Back Bay	4	<.10
White Sucker	Back Bay	1	<.10
Maria	Back Bay	1	.14
Northern Pike	Back Bay	1	.16
Whitefish	East Shore — Latham Island	4	<.10
White Sucker	East Shore — Latham Island	1	.11
Northern Pike	East Shore — Latham Island	1	<.10

Appendix B
Arsenic in Soil and Water Supplies from Latham Island, Detah and Vicinity

TABLE XIV
Department of Indian Affairs & Northern Development
Water Resources — Northwest Territories

RESULTS OF LABORATORY ANALYSIS
Arsenic in Water Samples Collected at Various Locations
in Yellowknife and Vicinity — September 1977

Sample Number	Location	Total Arsenic (As) mg/1
1*	North end of Latham Island	0.03
2	Latham Island at Rainbow Valley	0.02
3	Latham Island at Causeway	0.04
4	West side of Detah in Yellowknife Bay	<0.01
5	South East side of Detah, off Main Wharf	<0.01
6	South side of Detah	<0.01
7	Lake along Detah road, 2 miles south of Radio Tower	0.02
8	Yellowknife River at bridge	<0.01
9	Mouth of Baker Creek	19.8

*Sampling stations No. 1 to 9 are identified on the attached map of Yellowknife and vicinity (Figure 9).

Figure 9.
Sampling Stations, Yellowknife, Northwest Territories
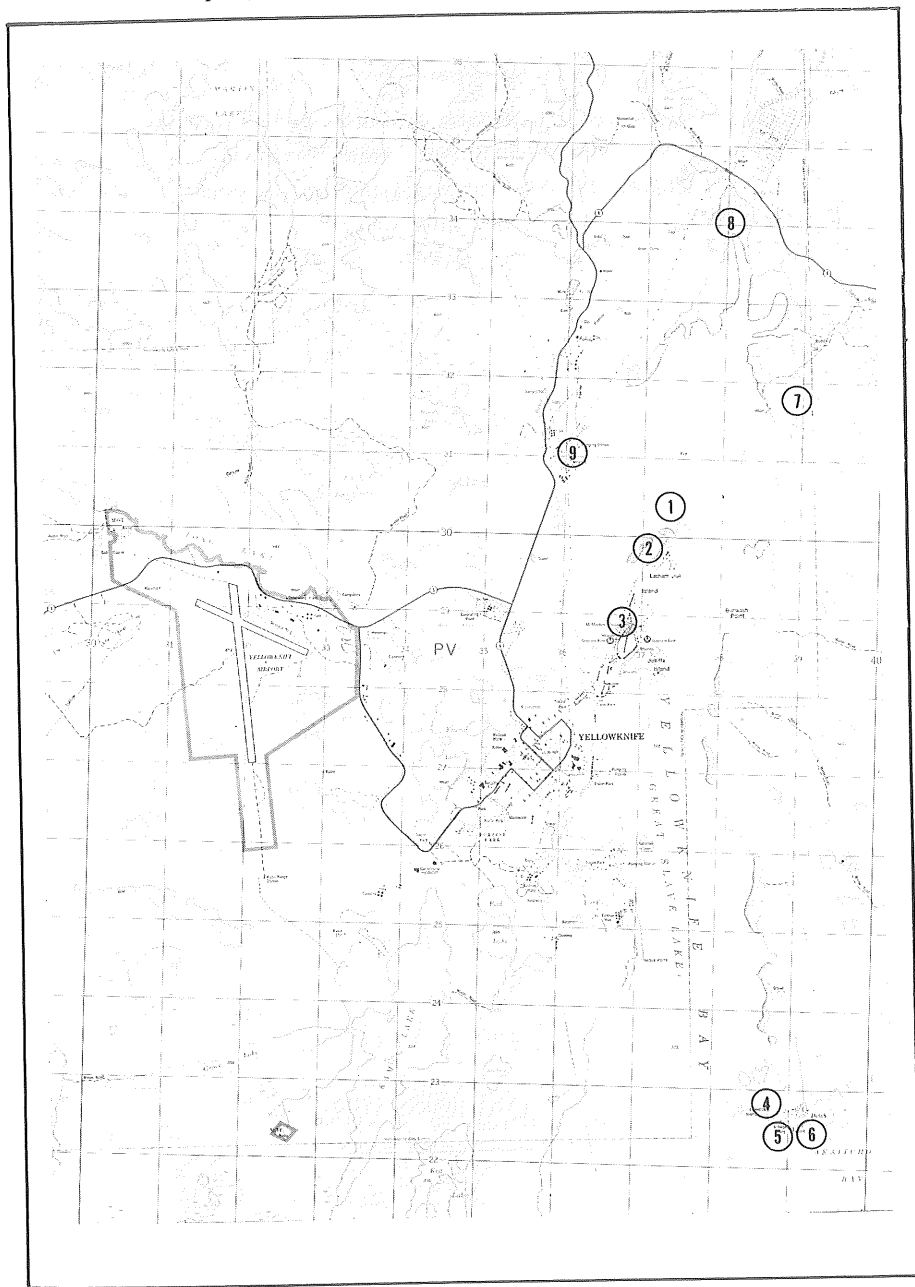
Figure 10.
Sampling Stations, Latham Island, Northwest Territories
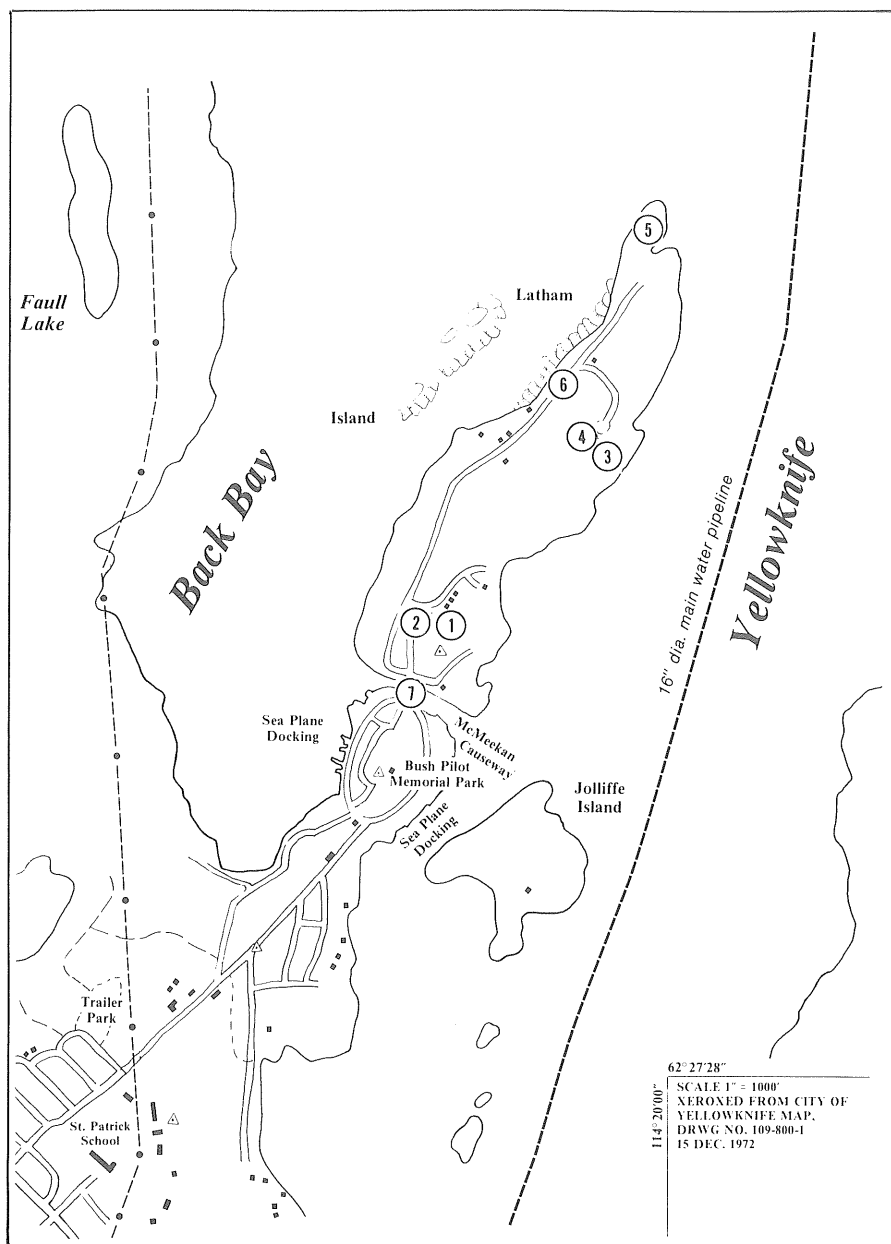
TABLE XV

Department of Indian Affairs & Northern Development
Water Resources — Northwest Territories
RESULTS OF LABORATORY ANALYSIS
Arsenic in Soil Samples Collected at Various Public Places on Latham Island
— September 1977

Sample Number	Location	Arsenic (As) (mg/kg air dry wt. soil)	
		Soluble	Total
1T*	On playground, near swing	< 1	1
1M*	,, ,,	< 1	1
1B*	,, ,,	2	61
MEAN*	,, ,,	1	21
2T	On playground, in front of permanent building	3	60
2M	,, ,,	< 1	49
2B	,, ,,	< 1	14
MEAN	,, ,,	2	41
3T	In community school yard, at back of school	3	57
3M	,, ,,	9	57
3B	,, ,,	4	20
MEAN	,, ,,	5	45
4T	In community school yard, in front of school	3	49
4M	,, ,,	< 1	30
4B	,, ,,	< 1	28
MEAN	,, ,,	2	36
5T	At north end of Latham Island	31	524
5M	,, ,,	18	196
MEAN	,, ,,	16	240
6T	At Bus stop, in front of Rainbow Valley	2	43
6M	,, ,,	< 1	10
6B	,, ,,	< 1	23
MEAN	,, ,,	1	25
7T	At causeway, on road side	< 1	170
7M	,, ,,	5	233
7B	,, ,,	< 1	36
MEAN	,, ,,	2	146

*Sampling stations No. 1 to 7 are identified on the attached map of Latham Island (Figure 10).   Other notes as Table XVI, p. 129

TABLE XVI

Department of Indian Affairs & Northern Development
Water Resources — Northwest Territories
RESULTS OF LABORATORY ANALYSIS
Arsenic in Soil Samples Collected at Various Public Places in Detah
— September 1977

Sample Number	Location	Arsenic (As) mg/kg air dry wt. soil Soluble	Total
8T*	In front of community church	3	29
8M*	"	<1	10
8B*	"	<1	9
MEAN	"	2	16
9T	Beside Wharf	4	34
9M	"	4	30
MEAN	"	4	32
10T	In community school yard, at back of school	3	75
10M	"	1	87
10B	"	1	80
MEAN	"	2	81
11T	In hockey rink	<1	8
11M	"	<1	7
11B	"	<1	8
MEAN	"	1	8

*Sampling stations No. 8 to 11 are identified on the attached map of Detah (Figure 11).

T, M and B represent the top, middle and bottom sections respectively of a soil profile 6 inches deep, where T = 0 to 2", M = 2 to 4", B = 4 to 6".

The mean value represents the mean of the 6" profile. For purpose of calculation the <1 values are taken as equal to 1.
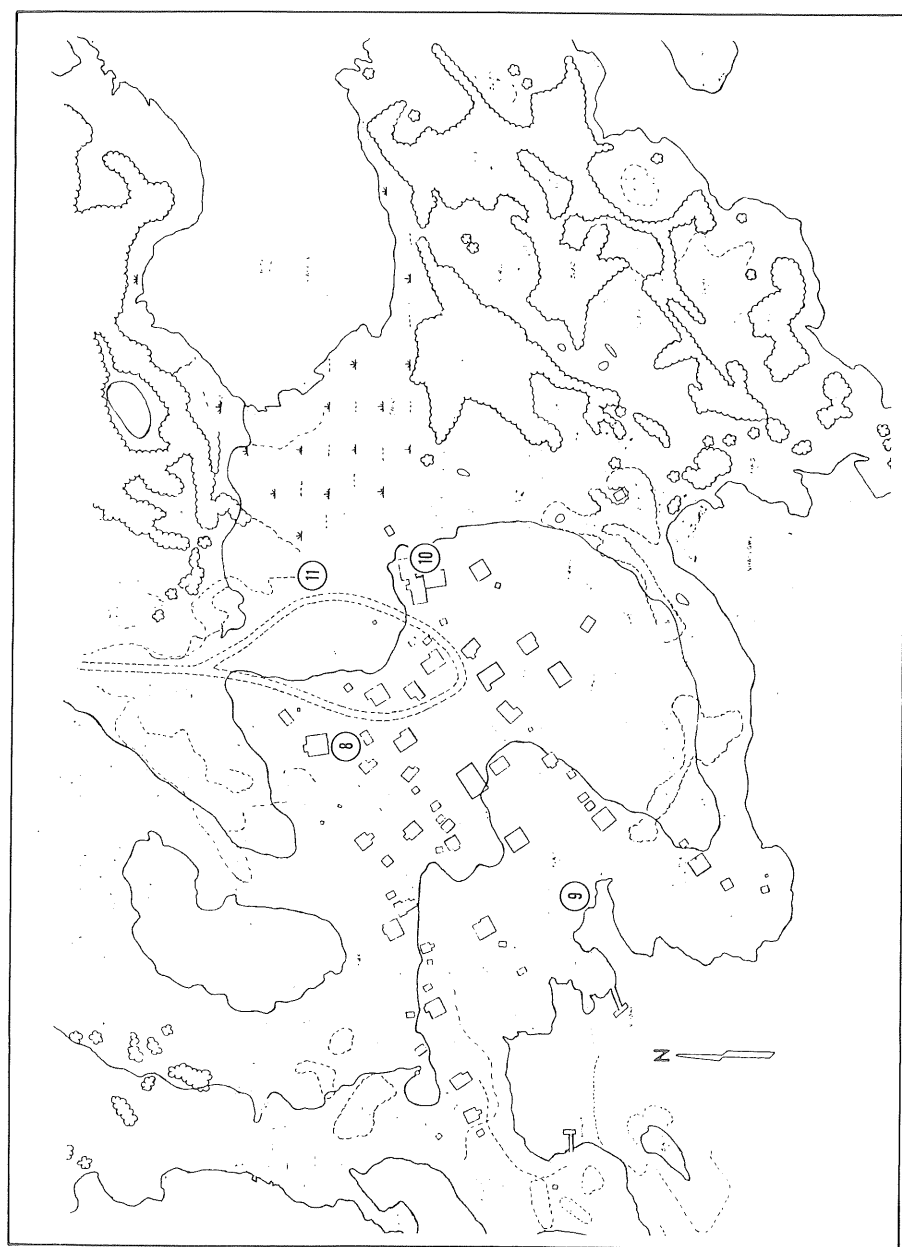
Figure 11.
Sampling Stations, Detah, Northwest Territories

Appendix C

Electromyography Program

The electromyograph can be used to measure the speed of an impulse moving along a nerve. The procedure can be carried out in children and adults. It is not dangerous or painful but may be perceived as slightly unpleasant by some.

The speed of nerve impulses varies in normal individuals. Nerve-impulse speed is slowed or disrupted in certain medical conditions. Injury to the nerve, frostbite, alcohol poisoning, mercury poisoning and a number of organic diseases can cause a slowing of nerve impulses.

Recent reports by Dr. J.T. Hindmarsh and his associates in Nova Scotia indicate that slowing occurs in nerve conduction in individuals exposed to arsenic who do not show clinical signs of arsenic poisoning. This work would indicate that alterations in nerve conduction speed may occur when hair arsenic levels exceed 1 ppm but are below 10 or 20 ppm (118).

Unfortunately an individual with a low normal conduction time might have the same reading as an individual with slight slowing due to arsenic or any other toxic or disease exposure.

The Task Force is aware that the variations in normal coupled with the numerous and prevalent toxic or disease states that can impair nerve conduction times make the electromyograph a difficult screening tool. The Task Force is nevertheless impressed with the ability of the method to detect changes due to arsenic present in low levels when exposed groups are compared with control groups. For this reason a survey program has been started in Yellowknife using electromyography (EMG).

This program has three objectives:
1. To determine if there are any minimal differences between the population of Yellowknife and other towns in the Territories that can be attributed to arsenic.
2. To put into the hands of medical workers in Yellowknife a diagnostic tool that may help them in detecting early damage due to arsenic in cases where there is known exposure and possibly clinical signs. Myography can become one part of an overall assessment.
3. To establish a baseline for serial electromyographic studies in future years for the same individual.

To determine if there are any minimal differences between the people of Yellowknife and other areas in the Territories, the Task Force has put into effect a preliminary study in Yellowknife and in Hay River.

It is technically possible to conduct about 30 EMG determinations in a normal working day. When a number of technical and social delays are deducted from this it may prove practical to carry out 15 tests per day. Thus in a normal working week 75 to 100 tests may be completed.

The Task Force has arranged a three-month program. This will involve at least five or six weeks of operation in Yellowknife and two or three weeks in Hay River. In this period it is hoped to test approximately a 10% sample of the people in Yellowknife with the following rough distribution:

Preschool children   6%
School children   47%
Adult population   47%

This sample is biased and weighted approximately 2:1 in favour of children and very young adults. A sample representing 10% of the population of this makeup would involve about 425 people. In addition all native people requesting the test should have it done. We hope this would involve at least 100 of the 941 native people in the area. A high proportion of children and young adults would be desirable. Due to the unexplained increased arsenic uptake in native children the Task Force considers that this service is particularly important for the native community.

Hay River residents and those from Yellowknife who have been there for less than three months (including visitors) will be taken as controls. The Hay River sample should consist of between 150 and 200 individuals with the same age distribution as the Yellowknife group. The male-female distribution in both samples should be nearly equal or, failing this, similar. As in Yellowknife any native people who present themselves for the test will have it done. We hope that at least 36 native people, half of them children and young adults, will present themselves in Hay River.

We assume that the health problems and lifestyles of Yellowknife and Hay River are sufficiently similar for the only important difference to be the presence of arsenic in the Yellowknife environment. There seems to be little difference in the age distribution of the native people in the two areas. There are proportionally more non-native infants and preschool children in Hay River than Yellowknife. The Yellowknife average income probably exceeds that of Hay River. Important differences in lifestyle or disease burdens may become evident during the program and will have to be taken into account. At the present time it seems likely that if any significant differences are detected between the two communities arsenic will be the most likely factor.

The program will be simple, confidential, safe, and painless. Individuals are being asked to answer a few simple health questions. A small hair sample and a urine sample will be taken for arsenic determination and the electromyographic studies will be done. Electrical contacts on the skin pick up the electrical impulses in the nerves. It is analogous, from the patient's point of view, to an electrocardiographic test but the contacts are placed on the arm or leg, and not the chest. Information on individuals will be grouped for statistical analysis. The actual readings will be available to the individual and to his physician but will otherwise be kept in the strictest confidence.

Table XVIIa
Sample Size, Native Population, EMG Survey, Yellowknife, Northwest Territories

| | Total | | 10% Sample | | Weighted 10% Sample | | Practical Sample |
	No.	%	No.	%	No.	%	
0 - 1 year	25	2.7	—	—	—	—	—
Preschool	101	10.7	10	11	20	19	All native people of a
School	312	33.2	31	34	60	57	sufficient age to test
Adult	503	53.4	50	55	25	24	
	941	100.0	91	100	105	100	

Table XVIIb
Sample Size, Non-Native Population, EMG Survey, Yellowknife, Northwest Territories

| | Total | | 10% Sample | | Weighted 10% Sample | | Practical Sample | |
	No.	%	No.	%	No.	%	No.	%
0 - 1 year	183	2.3	—	—	—	—	—	—
Preschool	186	2.4	18	2.4	25	3.4	25	6
School	2,101	26.9	210	27.6	200	27.6	200	47
Adult	5,337	68.4	533	70.0	500*	69.0	200*	47
	7,807	100.0	761	100.0	725	100.0	425	100

*Not to exceed 50% smelter or mineworkers and their families.

Controls:
 1. Any person who has not been in Yellowknife longer than 3 months and any visitors to Yellowknife with stays under 3 months to be considered as controls. Hay River to be a control community.

Table XVIIc
Control Sample, Native Population, Hay River

| | Total | | 10% Sample | | Practical Sample |
	No.	%	No.	%	No.
0 - 1 year	8	2.2	—	—	All native people of
Preschool	33	9.3	4	11	sufficient age for test
School	115	32.3	12	33	who present themselves.
Adult	200	56.2	20	56	10% sample should be
	356	100.0	36	100	minimum.

Table XVIId
Control Sample, Non-Native Population, Hay River

| | Total | | 10% Sample | | Practical Sample | |
	No.	%	No.	%	No.	%
0 - 1 year	74	2.0	—	—	—	—
Preschool	406	11.0	40	11	20	12
School	726	20.0	75	20.5	50-60	29
Adult	2,420	67.0	250	68.5	100-120	59
	3,620	100.0	365	100.0	170-200	100

Table XVIII
Population Figures — N.W.T.

Fort Smith

	Native	Non Native
0 - 1 year	4	47
Preschool	36	192
School	132	647
Adult	268	1,221
	440	2,107

Hay River

	Native	Non Native
0 - 1 year	8	74
Preschool	33	406
School	115	729
Adult	200	2,420
	356	3,620

Yellowknife

	Native	Other
0 - 1 year	25	183
Preschool	101	186
School	312	2,101
Adult	503	5,337
	941	7,807

Appendix D
Clean Air Act

Registration
SOR/74-325   21 May, 1974

CLEAN AIR ACT

Ambient Air Quality Objectives
P.C. 1974-1153   14 May, 1974

His Excellency the Governor General in Council, on the recommendation of the Minister of the Environment, pursuant to subsection 4(2) of the Clean Air Act, is pleased hereby to prescribe as national ambient air quality objectives, the annexed ambient air quality objectives for air contaminants formulated by the Minister of the Environment on the 13th day of May, 1974.

AMBIENT AIR QUALITY OBJECTIVES FOR AIR CONTAMINANTS

Short title

1. These Objectives may be cited as the *Ambient Air Quality Objectives.*

Formulation of Ambient Air Quality Objectives

2. The ambient air quality objective with respect to an air contaminant set out in column I of an item of Schedule I is the range of quality of the ambient air in relation to that contaminant set out in column III of that item where the air contaminant is in a concentration set out in column II of that item.

3. For the purpose of section 2, an air contaminant set out in column I of an item of Schedule II shall be measured
   (a) by the method set out in column II of that item; or
   (b) by a method that will consistently give a measurement from which the measurement that would be determined by the method prescribed in paragraph (a) can be calculated.

4. For the purpose of section 2, the concentration of an air contaminant shall be measured and corrected to a reference temperature of 25 degrees Centigrade and to a reference pressure of 760 millimetres of mercury.

SCHEDULE I

Column I	Column II	Column III
Air Contaminants	Concentrations	Range of Quality
1. Sulphur dioxide	*(a)* 0 to 30 micrograms per cubic metre annual arithmetic mean *(b)* 0 to 150 micrograms per cubic metre average concentration over a 24 hour period *(c)* 0 to 450 micrograms per cubic metre average concentration over a one hour period	Desirable
2. Sulphur dioxide	*(a)* 30 to 60 micrograms per cubic metre annual arithmetic mean *(b)* 150 to 300 micrograms per cubic metre average concentration over a 24 hour period *(c)* 450 to 900 micrograms per cubic metre average concentration over a one hour period	Acceptable
3. Suspended particulate matter	0 to 60 micrograms per cubic metre annual geometric mean	Desirable
4. Suspended particulate matter	*(a)* 60 to 70 micrograms per cubic metre annual geometric mean *(b)* 0 to 120 micrograms per cubic metre average concentration over a 24 hour period	Acceptable

SCHEDULE I

Column I	Column II	Column III
Air Contaminants	Concentrations	Range of Quality

5. Carbon monixide
 (a) 0 to 6 milligrams per cubic metre average concentration over an 8 hour period
 (b) 0 to 15 milligrams per cubic metre average concentration over a one hour period
} Desirable

6. Carbon monoxide
 (a) 6 to 15 milligrams per cubic metre average concentration over an 8 hour period
 (b) 15 to 35 milligrams per cubic metre average concentration over a one hour period
} Acceptable

7. Oxidants (ozone)
 (a) 0 to 30 micrograms per cubic metre average concentration over a 24 hour period
 (b) 0 to 100 micrograms per cubic metre average concentration over a one hour period
} Desirable

8. Oxidants (ozone)
 (a) 0 to 30 micrograms per cubic metre annual arithmetic mean
 (b) 30 to 50 micrograms per cubic metre average concentration over a 24 hour period
 (c) 100 to 160 micrograms per cubic metre average concentration over a one hour period
} Acceptable

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SCHEDULE II

Column I	Column II
1. Sulphur dioxide	West-Gaeke Method (Pararosaniline Method) Report No. EPS 1-AP-72-4
2. Suspended particulate matter	High Volume Method Report No. EPS-1-AP-73-2
3. Carbon monoxide	Non-dispersive Infra-red Spectrometry Method Report No. EPS-1-AP-73-1
4. Oxidants (ozone)	Chemiluminescent Method Report No. EPS 1-AP-73-7

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