



Health and Welfare
Canada

Santé et Bien-être social
Canada

Health Protection
Branch

Direction générale de la
protection de la santé

Environmental Health Centre
Tunney's Pasture
Ottawa, Ontario
K1A 0L2

RECEIVED
DEPT. OF HEALTH

AUG 10 1994

ENVIRONMENTAL
HEALTH

JUL 29 1994

Mr. J.A. MacKinnon
Head, Environmental Health
Medical Directorate
Northwest Territories Health
Government of The Northwest Territories
Yellowknife, N.W.T.
X1A 2L9

Dear Mr. MacKinnon:

Thank you for your letter dated July 6, 1994, with the enclosed reports from Mr. James Sparling in which you requested an appraisal of the potential human health risks associated with the sulphur dioxide and arsenic emissions measured in Yellowknife.

The reports indicate that the one hour maximum levels of sulphur dioxide consistently exceed the one hour desirable limit of $450 \mu\text{g}/\text{m}^3$, with a number of isolated incidents of levels exceeding the one hour maximum acceptable level of $900 \mu\text{g}/\text{m}^3$ (as established under the Canadian Environmental Protection Act). While the levels are somewhat increased over those reported in the study examined for Dr. Gilchrist in 1993, we do not feel that an imminent health hazard exists from the sulphur dioxide levels recorded in Yellowknife. The levels detected which exceeded the one hour maximum acceptable levels remain in the range at which mild, reversible, respiratory effects would be seen in sensitive individuals, particularly asthmatics. We would caution that the analytical data provided were for a single monitor in Yellowknife and do not necessarily represent personal exposure values.

With regard to the arsenic levels, as noted in our previous correspondence, there is no current federal air quality guideline for arsenic. Arsenic has been declared toxic under Section 11(C) of the Canadian Environmental Protection Act (CEPA) as it has been demonstrated to be a known human carcinogen. However, as part of this assessment, an evaluation was carried out on the epidemiologic studies conducted on populations residing near point sources of arsenic emissions. The conclusions was that "Due to limitations of the database, it is not currently possible to draw any firm conclusions on the effects of arsenic emissions on the risk of cancer for residents in the vicinity of industrial sources." I have included a copy of the relevant section and tables from the CEPA Priority Substances Supporting Documentation for Arsenic and Its Compounds.

Canada

I would note, however, that the CEPA Assessment Report also recommended a high priority for the development of control options, based on the determination that arsenic is a known human carcinogen, and for that reason alone every effort should be made to reduce potential exposure. I have enclosed a copy of the CEPA report and the risk assessment guideline document. Specific control options, including development of guidelines or objectives, are currently at the initial stages of development under the CEPA Strategic Options Process.

To help put the potential risk into context, it is possible to consider the annual levels reported in Yellowknife in relation to the cancer risks reported in occupational setting. As reported in the CEPA Assessment Report, based on three epidemiology studies conducted on occupationally exposed workers, a range of respiratory cancer potency values ($TD_{0.05}$) of 7.83 to 50.5 $\mu\text{g}/\text{m}^3$ were derived. These values represent the concentration which was calculated to induce a 5% increase in the incidence of respiratory cancer in the study populations. In comparing the $TD_{0.05}$ to the lowest arsenic level reported recent years (1991 level of 0.006 $\mu\text{g}/\text{m}^3$), the results represents 0.00077 to 0.00012 of the $TD_{0.05}$. The 1993 level would, of course, represent an increase in this range (0.0019 to 0.0003 of the $TD_{0.05}$), corresponding directly to the higher arsenic levels. The 1994 levels have not been analyzed as they do not represent a full year of data, although, to date, they appear to be lower than those reported in 1993. One must recognize, however, that the Yellowknife monitoring data were derived from a single site and do not directly equate to human exposure. Additionally, the levels to which the industrial workers were exposed far exceed those report in Yellowknife.

The risk should also be viewed with respect to levels reported in other areas across Canada. While the mean level in Yellowknife in 1993 was 15 times higher than the mean value of 0.001 $\mu\text{g}/\text{m}^3$ reported in several industrial cities across Canada, the levels are certainly within the range of 0.0086 to 0.22 $\mu\text{g}/\text{m}^3$ reported in communities near base metal smelters (see enclosed copy of Section 6.3.1 for the CEPA Priority Substances Supporting Documentation for Arsenic and Its Compounds).

I trust you will find the above information useful in your consideration of the issues arising from the arsenic and sulphur dioxide monitoring data from Yellowknife.

Yours sincerely,

Roy Hickman

J. R. Hickman
Director General
Environmental Health Directorate

Enclosures

Studies have shown that inorganic and organic arsenic compounds are generally not excreted into milk of cows (Vreman et al. 1986; Hesse et al. 1990), although a slight elevation, to 0.022 and 0.019 mg As/kg, occurred after five days in milk from cows fed 3.2 mg As/kg body weight as arsanilic acid or 3-nitro-4-hydroxyphenylarsonic acid, respectively (Calvert, 1973). Milk arsenic levels returned to background concentrations of 0.013 and 0.004 mg As/kg after a seven-day withdrawal period.

6.3 Concentrations in the Environment

Arsenic levels in air, water, sediment, soil and biota were reviewed. Except in the case of biota for which Canadian data are lacking, only a limited amount of non-Canadian data are discussed in this Section. Data for air, water, sediment and soil from more severely contaminated Canadian sites, and some non-Canadian locations, are summarized in Tables 6.3 and 6.5. Other data for Canadian air, drinking water, sediment and biota are presented in Table 6.4, 6.6, 6.7 and 6.8, respectively.

As indicated in Section 5.3.2, releases of arsenic by the metal production industry in Canada have been reduced significantly in recent years. Concentrations of arsenic in air, water and possibly biota close of point sources such as mines or smelters, would be expected to exhibit similar temporal variation. In the descriptions that follow, therefore, the year of sampling of Canadian air, water and biota has been specified wherever possible. Although concentrations in soils and sediments can also change in response to changes in arsenic release rate, (see for example, Sutherland, 1989), contaminated soils and sediments typically remain in place for many years. Time of sampling is, therefore, less important for these materials.

6.3.1 Air

Measurements in the 1970s by Walsh et al. (1979a) suggested that average arsenic concentrations in air over land were about $0.001 \mu\text{g}/\text{m}^3$. Levels over oceans were found to be much lower (typically $< 0.0002 \mu\text{g}/\text{m}^3$). Most of this arsenic was associated with fine ($< 1 \mu\text{m}$ diameter) suspended particulates.

Airborne arsenic is generally associated with particulate matter, except in the immediate vicinity of smelters which emit arsenic vapours (proportion of non-particulate arsenic present in emissions not specified) (U.S. Environmental Protection Agency, 1984). Arsenic levels were recently measured in inhalable ($< 10 \mu\text{m}$ diameter) particulates, collected in the commercial districts of 11 major metropolitan centres across Canada, over a four year period (Table 6.4). Concentrations in this size fraction ranged from $< 0.0005 \mu\text{g}/\text{m}^3$ up to $0.017 \mu\text{g}/\text{m}^3$ (24-hour averages) between January 1985 and April 1990, with the highest value occurring at a Montreal station. Mean concentrations were $\leq 0.002 \mu\text{g As}/\text{m}^3$ at all sites (Dann, 1990). Concentrations during 1983 and 1984 ranged from < 0.003 to $< 0.013 \mu\text{g}/\text{m}^3$ (Environment Canada, 1984). Levels in Windsor ranged between 0.001 and $0.004 \mu\text{g}/\text{m}^3$ in 1987-1988 (Environment Canada, 1988c). Arsenic in $< 10 \mu\text{m}$ diameter air particulates taken at a near-by rural site on Walpole Island, Ontario, between 1985 and 1990 reached as high as $0.003 \mu\text{g}/\text{m}^3$, but the majority of samples from this station contained $< 0.0005 \mu\text{g As}/\text{m}^3$ (Dann, 1990; Environment Canada, 1989b; Environment Canada, 1990). NRCC (1978) cited data indicating that in rural areas in Canada, total arsenic levels ranged up to about $0.005 \mu\text{g}/\text{m}^3$.

Somewhat higher arsenic levels have been measured in suspended particulates in ambient air of communities affected by base metal smelters (Table 6.3). However, due to the paucity of information provided on methods of sampling and analysis for these surveys, it is not possible to assess the comparative validity of the concentrations reported at these sites. Average annual arsenic levels in "hi-vol" samples of Rouyn-Noranda air ranged from 0.004 to $0.032 \mu\text{g}/\text{m}^3$ between 1982 and 1991. Average annual levels of arsenic in suspended particulates from 1987 to 1991 within 0.8, 1.7 and 2.5 km of the copper smelter ranged from 8.6 to 18.2, 6.9 to 10.7 and 3.6 to $6.3 \text{ ng}/\text{m}^3$,

NHW/DOE, April 1993

stratified for age and cumulative arsenic exposure, the odds ratios increased with amount smoked (1.0, 6.9 and 8.0 in 0, 1 to 10 and ≥ 10 g of tobacco per day). The exposure-response relationship was stronger between arsenic exposure and lung cancer mortality among nonsmokers and medium smokers than among the heavy smokers. Similar to the results obtained in the cohort study, average intensity of exposure had more influence on the risk of lung cancer death than did duration of exposure; inclusion of exposure to sulphur dioxide did not affect the odds ratios for exposure to arsenic.

Enterline et al. (1987b) have investigated the effects of exposure to sulphur dioxide and cigarette smoking on the relationship between airborne arsenic and respiratory cancer in workers at eight copper smelters (neither the Tacoma nor the Anaconda smelter was included). The mortality status of 6,078 male workers who had been employed for at least three years between 1946 and 1976 was determined. Follow-up was 98.4%; death certificates were available for 94.1% of those known to be dead. Only in one smelter with arsenic levels much higher than the others, was the mortality due to lung cancer significantly greater than national, state or local rates; this excess remained after adjustment for cigarette smoking and sulphur dioxide exposure. Exposure-response relationships between lung cancer and cumulative exposure to arsenic as well as peak sulphur dioxide exposure were noted when internal controls were used. Only smoking and exposure to arsenic were significant factors associated with increased respiratory cancer when data on smoking and exposure to both arsenic and sulphur dioxide were considered in a nested case-control study.

Cancers at sites other than the lung may be associated with occupational exposure to arsenic, though these relationships have not been investigated as extensively. For example, based on a review of available epidemiological data including several of the studies addressed here, Gibb and Chen (1989) concluded that excess risk of cancer of the gastrointestinal system, including the stomach, colon and liver and the urinary system is associated with occupational exposure to arsenic.

10.4.2 Populations Residing in the Vicinity of Arsenic Emitting Industries

Epidemiological studies on populations residing in the vicinity of arsenic emitting industries, such as smelters, have been reviewed by the U.S. Environmental Protection Agency (U.S. EPA, 1984). The results of these studies and two more recent investigations, are presented in Table 10.5. An excess of lung cancer incidence or mortality was reported in a few ecological correlational studies and one proportionate mortality study; these results cannot be weighted heavily, however, owing to the lack of examination of exposure of individuals in such investigations; moreover, with the exception of the studies by Matanoski et al. (1976, 1981) and Cordier et al. (1983) possible confounding by smoking or occupation was not taken into account. However, no association was found between cancer rates and proximity to the source of arsenic emissions in three more inherently sensitive case-control studies (Frost et al. 1987; Rom et al. 1982; Lyon et al. 1977). Due to limitations of the database, it is currently not possible to draw any firm conclusions on the effects of arsenic emissions on the risk of cancer for residents in the vicinity of industrial sources.

10.4.3 Populations Exposed to Arsenic in the General Environment

The carcinogenicity of ingested arsenic has been investigated in several epidemiological studies in environmentally exposed populations. Studies reviewed by the U.S. Environmental Protection Agency (1988), which involve populations exposed to drinking water contaminated with arsenic, have been presented in Table 10.6. In the majority of these studies, conducted in Taiwan, Central and South America, U.S.A. and England, ingestion of arsenic has been associated with an increased prevalence of skin cancer or mortality due to skin cancer and in some cases, possibly also cancer of internal organs. In several case reports and descriptions of case series, cancer following ingestion of arsenic medicinals has been observed. Although such studies are considered relevant to assessment

NHW/DOE, April 1993

Table 10.5 Epidemiological studies of cancer rates in populations residing near point sources of arsenic emission (modified from U.S. EPA, 1984, to incorporate additional recent studies).

Study Population	Reference	Type of Study	Results	Highlights/Deficiencies
Residents living near a smelter in El Paso, Texas.	Rom et al. 1982	Case-Control	No association was found between lung cancer and distance from the plant.	Effects of migration, smoking and occupation were not considered.
Residents of Deer Lodge and Silver Bow Counties, Montana.	Newman et al. 1976	Ecological correlation	There was an increase found in the incidence of lung cancer among men. In one of the cities there was also an increase in lung cancer among women.	No adjustment was made for cancer cases which may have been occupational.
All counties in the United States with smelters.	Blot and Fraumeni, 1975	Ecological correlation	Average lung cancer mortality rates were significantly elevated for both males and females in 36 counties with smelters processing copper, lead or zinc ores.	
Residents near a smelter in Utah.	Lyon et al. 1977	Case-control	No association between cancer and distance from the smelter was found.	Lymphoma cases, which may have been associated with arsenic exposure, were used as controls. Effects of smoking, migration and occupation were not considered.
Residents near Ronnekar-verken smelter in northern Sweden.	Pershagen et al. 1977	Ecological correlation	A significantly higher mortality rate for lung cancer was noted for men in the exposed area. The increase was no longer significant when occupational cases were excluded, however.	When excluding occupational cases of lung cancer from the study population, lung cancer cases for a comparable occupation group were not excluded from the comparison population.

Table 10.5 cont.'d

Women residing near a copper smelter in Washington.	Frost et al. 1987	Ecological correlation Case-control	Ecological correlation: There was no increase in mortality due to lung cancer compared to expected rates in any exposure group classified by distance from smelter. Case-control: Distance from smelter was similar in cases and controls, as was mean years of residency. "Exposure index" was slightly (not significantly, $p=0.07$) higher in cases than controls, when adjusted for 20 year latency period.	No data on smoking habits of residents were available.
Residents near smelter towns in Arizona.	Arizona Department of Health Services. 1990	Ecological correlation	1.5 fold elevation in lung cancer mortality rates in residents over 60 years of age, compared to nonresidents.	No data on smoking habits or duration of residency of subjects was reported. Occupation was not considered.