

MEMORANDUM
GOVERNMENT OF CANADA



NOTE DE SERVICE
GOUVERNEMENT DU CANADA

FROM Dr. O. Schaefer
DE Northern Medical Research Unit.

TO A/Regional Director
A Northern Region.

SUBJECT Yellowknife Arsenic Survey
SUJET

SECURITY - CLASSIFICATION - DE SÉCURITÉ

OUR FILE — N/RÉFÉRENCE

YOUR FILE — V/RÉFÉRENCE

DATE

4 Nov. 1971

My reply to your memo dated September 13 was delayed by the fact that I spent only a few days in my office between two trips to the Arctic and one to a post-graduate course in September and October.

I restudied this quite lengthy (119 pages) report. The report goes into great detail and contains a great wealth of data concerning the general aspect of prevalence of clinical conditions in different population groups arranged by age, sex, time of residence and occupational exposure to arsenic (mill workers).

The following impressed me as the essential highlights:

1. Arsenic contamination of the atmosphere:

Between 1948 -1953 inclusive a daily emission of arsenic trioxide by stack-effluents of approximately 11 tons (22,000 lbs). Actual Roaster stack tests were only begun in 1954 and these revealed an emission of approximately 7,250 lbs of elemental arsenic per day between 1954-1958 inclusive from both Giant & Consolidated mine stacks. New extracting equipment and changes of procedures introduced a Giant gold mine between 1957-1959 reduced drastically its atmospheric emissions to less than 5% of previous averaged while no remarkable change was noticeable in emissions from the Consolidated mine stack, but Consolidated had installed a scrubbing device in 1949 and since emitted considerably lower amounts than Giant until 1959.

The result of improvements implemented at Giant from 1957-1959 was a reduction of the total atmospheric emission in the Yellowknife area from approximately 7,250 lbs. daily during the 1954-1958 period to approximately 695 lbs. per day in 1959-1969.

The total amount of arsenic emitted into the air between 1954-69 is estimated at 8000 tons (to which should be added approximately 7000 tons during the early period of mill operations 1948-1953 for which no regular testing data are available, for an estimated total of approximately 15,000 tons atmospheric arsenic release up to 1969 in the Yellowknife area - bracket is my own addition).

2. Fall-out and contamination of vegetation:

Fall-out measurements varied much due to weather conditions and seasons and were not systematic enough to allow exact calculations. But a general trend of lessening fall-out is recognizable from 1960 on and the average fall-out on the town-site of Yellowknife for the period of measurements (1951-1963) was estimated at 2 lbs. of elemental arsenic⁽¹⁾ per acre per year. No significant amounts of arsenic could be found in locally grown vegetables.

3. Arsenic contamination of water supply:

The water supply of townsite and Consolidated Mine were similar, that of the Giant Mine slightly higher in arsenic content.

There was in contrast to atmospheric emission and fall-out no definite trend noticeable when comparing corresponding monthly and annual averages in the 1950's and 1960's. The considerably higher arsenic concentration in the months with springtime run-off (May, June and July) indeed was at least as marked in the 1960's as in the 1950's (and one wonders how that fits with the above described improvements in atmospheric emission noticeable since 1959 - or could there be a leakage ^{at} run-off time from the arsenic effluents discharged by Consolidated Mine into tailings, ponds and a natural depression? - remark in bracket is my own query).

Arsenic contamination of the town water supply during 1951-69 was within acceptable limits (= less than 0.01 ppm) less than 16% of time and approximately 15 % of the time above permissible limits (0.05 ppm). The arsenical contamination of Frame Lake, likely representative of many smaller and relatively stagnant (fed by spring run-off) water bodies, was more than 10 times that of the town water supply. This may have significance for the local wildlife and directly or indirectly also for some of the native Indian population.

(In regard to my personal - above expressed - idea, that the lack of trend in water contamination over the years despite diminished atmospheric emission and fall-out, in particular the persistence of large seasonal increases during spring run-off it may be of interest to note the exceptional high value on June 20, 1966

namely 2.92 ppm arsenic, which, if correct, would indeed favor the suspicion of a sudden influx at run-off time from sludge-discharge sites - I am wondering if this question should be followed up by examination of location etc. of these sludge-drainage sites?)

4. Health survey results and possible relation to arsenic toxicity:

The greatest part of the report (I presume that previous communications from the Occupational Health Division have dealt mainly with the aspect of environmental contamination which is discussed with relative brevity only in this report) deals with the results of a very detailed clinical health survey and discusses the results in regard to possible relation to arsenic toxicity.

A relatively high prevalence of

- a) skin - lesions
- b) chronic non-specific respiratory disease
- c) neurological findings
- d) codable electrographic changes (in particular RBBB) were found

A statistically significant relation to occupationally increased arsenic exposure could only be demonstrated for afflictions of the skin, which were markedly increased in mill workers vs. other residents of comparable age, sex and duration of residence and were explained by local irritation ^{of} arsenical dust particles.

Relatively high prevalence of chronic non-specific respiratory disease evidenced by clinical examination, spirometry and chest films appeared more related to time of residence in Yellowknife area than duration of mill (arsenic dust) exposure. Dr. De Villiers is inclined to attribute the increase in skin lesions directly to exposure to elemental arsenic (dust) while more general or non-specific factors are suggested in his opinion such as cold climate and local stress conditions for increased incidence of non-specific respiratory, neurological and electrocardiographic pathology.

Local stress factors resulting from transplantation into a strange and isolated environment and heterogenic ethnic backgrounds as well as a mobile and age and sex wise not well balanced population structure (together with high rates of alcohol, nicotine and coffee consumption? - my remark) may also be causal factors in the extraordinary high rates of deaths due to accidents, violence, suicide, poisoning, etc. and explain higher than normal rates of psychoneurotic conditions and "stress" diseases such as gastric and duodenal ulceration, dyspepsia and thyroid dysfunction. A possible locally irritating role for the respiratory tract is attributed to arsenic dust likely aggravated by smoking and harsh climate, as suggested in Dr. De Villiers opinion by the higher, incidence of non-specific respiratory disease in mill workers, with

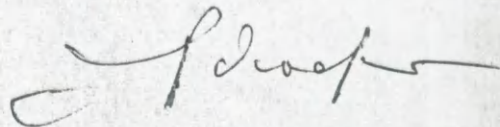
more than 10 y. exposure vs. those of less than 10 y. exposure. A somewhat higher incidence in mill workers was also found for neurological disorders, but the authors did not attribute this to any arsenic related factor.

I personally am wondering, however, if the significantly higher urinary arsenic excretion of mill workers (mean 0.0203 ppm vs. 0.0110 for non-mill workers), but both groups were found in the suggested normal range) which reflects naturally higher total arsenic absorption rates from gut and skin may have something to do with their higher rate of neurological abnormalities - even if that did not reach in the small numbers available statistically significant degrees.

I presume that the report is not yet finalized as correlations of symptoms with physical findings and pulmonary function tests (and EKG findings - my remark) have not yet been made and might be interesting.

I might also add that a correlation of EKG findings particularly those with RBBB with pulmonary function, diameter of pulmonary artery and clinical chest findings would be of interest in view of my findings in elderly Eskimo hunters. Dr. De Villiers remarks on the high frequency of codable EKG pathology in particular RBBB, but stresses that the significance is obscure at this time.

Our own findings comparing Eskimo hunters and non-hunters would suggest that cold weather exposure particularly if extreme physical exertion in cold weather forces panting respiration allowing little protective warming of the air has indeed a traumatic effect on the upper and lower respiratory tract leading to obstructive bronchitis and increased pulmonary artery pressure with signs of widening of the proximal parts of the pulmonary artery and "cut-off" effects in the periphery and right ventricular overloading with RBBB in later years. I shall confer on occasion with Dr. De Villiers on this aspect.



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