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Science Advisory Board of the Northwest Territories

METALLIC CONTAMINANTS OF SIGNIFICANCE

ract Report
No. 6

Metallic Contaminants of Significance to Northwest Territories Residents

by
R.D.P. Eaton

Statistical Appendix
by
W.A. Fuller

Prepared for the
Science Advisory Board
of the Northwest Territories
September, 1982

Metallic Contaminants of Significance to
Northwest Territories Residents

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Foreword

The S.A.B. has considered a number of topics to be of sufficient importance to the people of the Territories to warrant the compilation of "state of the art" reports. As an addition to this series the Board asked Dr. Derek Eaton to prepare a paper on heavy metals in the N.W.T. environment.

As the report itself indicates, this subject is one which has generated considerable public interest and controversy. In the course of his years as a medical scientist with Medical Services Branch, Health & Welfare Canada, Dr. Eaton has been directly involved in the investigations concerning the four heavy metals he writes about in this paper, giving a very readable and personal account.

Dr. Eaton's account of arsenic centres on the Yellowknife gold mining and smelting operations. With modern methods there is now little release of arsenic to the atmosphere but in certain workplaces the risk of toxic exposure mandates special precautions and regular monitoring.

Cadmium and lead are each dealt with rather briefly. Cadmium seems also to be confined to mining operations and is considered a small and localized risk since smelting is not involved in the lead-zinc mining operations in the N.W.T. where cadmium could pose a problem. Regarding lead, although mine workers are at some relatively small risk of excessive lead intake, the use of leaded gasoline makes lead a more ubiquitous hazard from inhalation of lead from automobile exhaust fumes. This affects the lead burden of the general population and to a greater extent those who work with gasoline operated vehicles in poorly ventilated spaces. However, those risks seem small compared to the hazard to those — mostly school age children — who become addicted to "gas-sniffing". In such cases the hydrocarbon in addition to the lead may have toxic effects.

The major part of Eaton's paper relates to mercury, the distribution of which is widespread in nature. The neurotoxic effect of the ingestion of mercury in food (Minamata disease) is due to the effects of methylmercury. High levels of mercury have been found in seals in the Arctic and the Inuit have at times been given controversial advice against eating seals, particularly seal liver. The fact that there have been no unequivocal human cases of methylmercury poisoning reported from eating seal meat or liver does not alleviate the concern since methylmercury accumulation in the body may be slow and silent but neurological damage, when it does occur, is likely to be irreversible. On the other hand Eaton has demonstrated in his own laboratory that feeding cats with methylmercury in seal liver is not as toxic as feeding them the equivalent doses added to other food. This may be due to a detoxification function (demethylation) particularly well developed in seal liver or it may be due to the protective effect of selenium which is also present in seal liver.

Although Eaton reports that no stigmata of methylmercury intoxication have been found on clinical examination of Inuit, the reports from Ontario and Quebec are not so definitive. Of particular note in this regard is the study conducted in Quebec by the McGill Methylmercury Study Group under the joint sponsorship of the Government of Canada, Government of Quebec and the Donner Canadian Foundation, which was published in 1980. The McGill group, in what appears to have been a careful epidemiological study, found some weak associations between neurological signs and methylmercury exposure.

This study clearly demonstrates the difficulties in identifying early toxic effects and of apportioning the cause between methylmercury and other confounding variables including such things as tobacco, alcohol and poor nutrition. Seal and fish are important to the nutritional health of native peoples and are an important part of their culture. The evidence from the McGill study is not sufficiently clear-cut to justify limiting the consumption of seal or fish. It certainly emphasizes the need for continuing careful observation.

Also the fact that the situation keeps changing due to local industrialization, global changes such as acid rain, cultural changes, or refined methodology strongly indicates the need for further study and regular monitoring concerning heavy metals in the northern ecosystem. It is worth emphasizing that it is important to know the distribution of natural concentrations of heavy metals in the soils and sediments if the industrial sources of contamination are to be identified.

We are grateful to Derek Eaton for providing this review which will be a useful benchmark for future studies.

J.A. Hildes, MD

November, 1982

Executive Summary

This paper discusses the occurrence of four metallic contaminants, cadmium, lead, arsenic and mercury, and their significance to residents of the NWT. The discussion is based on a review of available literature, as well as the results of some original research.

Arsenic levels are only of concern in those areas where smelting and roasting of gold-bearing ore result in the production of arsenic trioxide. In the Yellowknife area, although such emissions were once quite large, recovery techniques have reduced emissions by at least a factor of 1000 with the result that concerns are now confined to those involved with the recovery process itself. Recent studies have identified procedures which industry may use to reduce this residual hazard.

Literature relevant to the NWT dealing with the occurrence of mercury in freshwater and marine environments, as well as in the food chain, is reviewed. The high levels of mercury found in seals and in some freshwater fish are noted. Although no cases of methylmercury poisoning in man have been recorded in the N.W.T., elevated levels of mercury have been identified in residents of the Mackenzie valley, at Cambridge Bay and in communities on Baffin Island. These are attributed to a reliance on dietary sources of protein which may contain high levels of mercury, such as occasional catches of freshwater fish, seal meat, and seal liver, and the meat and muktuk of the beluga whale.

It is noted that the mercury toxicity of seal meat and seal liver is determined by the methylmercury fraction of their total mercury content. Results of original research which further support the hypothesis that adequate levels of selenium in the diet can result in the inhibition of methylmercury toxicity are discussed. It is suggested that if this is the case, then mercury toxicity should not be expected from marine fish since such fish have a high selenium content.

Cadmium, which is apparently non-toxic in its natural state, can be acutely toxic as a soluble salt or elemental vapour. Hence, its toxic effect is usually confined to smelter workers. In the NWT, industrial sources of cadmium are confined to the lead/zinc mines, but since none of these include smelters, the probability of cadmium poisoning is very low.

Although lead is a rather widespread problem in the south as a result of lead-based paint, smelters, secondary industries and gasoline derived sources, in the north only lead mines constitute a major potential hazard. In the NWT, mines operating in heavy permafrost must use a dry drilling technique though the potential problems of respirable dust and silica appear to have been avoided by a vacuum-extraction ventilation process. Even so, regular surveillance of blood and urinary lead in the workers at Nanisivik has demonstrated some increase in lead levels. Probably this is attributable to ingestion of particulate lead as a result of sub-optimal hygiene rather than to inhalation. Efforts are being made to promote cleanliness in the work environment, as well as to reduce dust exposure to a minimum.

Elevated blood lead levels were noted in two other groups in Arctic Bay, hunters and municipal employees. This may result from members of both groups performing maintenance and repair tasks on internal combustion engines in poorly ventilated workshops. However, the possibility of other sources cannot be ruled out.

In an appendix to the report, Dr. W. A. Fuller applied further statistical tests to the data. His analysis of arsenic levels amongst residents of the Yellowknife area indicates that amongst non-natives there are more males than females with high hair arsenic levels. This is presumably related to employment. However, when native and non-native people in the age group from 0-19 are compared, the proportion of natives with elevated arsenic levels is greater. Although the cause for this difference is probably environmental, it has not been identified. It should be noted however, that all levels found were lower than those associated with clinical toxicity.

Finally, Fuller's analysis of blood lead levels in Arctic Bay suggest elevated levels among Inuit mine employees. However, he emphasizes that no cause and effect relationship has been established.

ATANGUYAN NAITUK TITIGAKHIMAYAN

HAMNA TITIGAK UKAUTIKAKTUK HITAMANIK HAVINGNIK TOKONAKAKTUNIK HAPKUNINGA CADMIUM, LEAD, ARSENIC, MERCURY LU, KANUGITYUTAILU NUNATIAMIUTANUT. UKAKHUNILU NAONAIYAINAGIALGIT MAKPIGAKUT ILITOKHOIYAUTIKULU.

ARSENIC KAKPAKTUN NUNAN KUUNIK HAVAKANGAMIK ILAKAGLUNI ARSENIC TRIOXIDE MIK. YALUNAIMI ARSENIC KAGALUAKHUNI ANGIYUMIK, PIYAKTAUYUK KIHIMI TALVATUAK UNGUVAKHIMAYAN. ILITOKHOIYUN ATUKTAKHANIK PIIYAUTIKHANIK HAVAKVIN NAONAIYAIVAKTUN.

MAKPIGAN NUNATIAMI IHUAKTUN IHIVGIUKTAULIKTUN KANUGITYUTAINIK MERCURY M IMANGMIUTANIT NUNAMIUTANULU NIGIYAUVAKTUNUT. MERCURY KANGNIK NATTINGNI IKALUNGNILU NALVAKTAUVAKTUK. TUKUNAKANGNIK MERCURYMIT NALVAKTAUNGITKALUAKHUNI INUNGNI, MERCURY KANGNIMIK NALVAKPAKTUN MACKENZIE VALLEY MI., IKALUKTUTIAMILU KIKIKTALUNGMILU. HAPKOA NIGIVAGAINIT PIKAKTUN MERCURY KAGANGAMIK IKALUNGNI NATTINGNILU TINGUINILU NIKALU MAKTAILU KILALUGAM.

MERCURY KANGNIA NATTIUM TINGUATALU NAONAIYAKTAUVAKTUK METHYLMERCURY KANGNIANIT TAMAN. NALVAKTAINIT KANGIKHINAHUAKTAMINGNIT NAUNAIKTAN UNA SELENIUM KAGUMI NIGIYAUYUK METHYLMERCURY KAGUNAITUK. IMAITKUMI MERCURY KAGUNAITUT IKALUIT, IKALUIT SELENIUM GIAKUMIK.

CADMIUM TAUK, TOKONANGUNGITKALUAKHUNI HAVAKTAUHIMAITKUMI, TOKONANGUYOK TAGIUTUN AUKTUKTAGUMI. TALVUNA HAVAKTUN OYAGALIKIYONI TOKONAKAKHUNGUYUN. NUNATIAMI CADMIUM KAKTUN LEAD LU ZINC LU OYAGAKHIOLTIN, KIHIMI HAPKOA ILAKANGINAMIK AVITIGIYINIK OYAKANIK CADMIUMIK TOKONAMIK PIKALLUNGITUN.

LEAD KANGNIK NUNANI AHINILLUAK PIYAUNAHUGIYAUVAKTUK, TALVALUNIT HAVIGUYAKHIUKTUNI. NUNATIAMI OYAGAKHIUKTUN KIHIMI IMA IKUTAKTUN KIHIMI IKUTAIT OKHOKTIKTAOYUITUN. KIHIMI, HILUKHUTIKUT PIYAKTAOVAKTUN HIVOGANAKTUMIK. IHIVGIONGNIK AUNGINIKLU KUNGINIKLU HAVAKTUN NANISIVINGMI NAONAIKTAUYOK MIKAK LEAD KANGNIAN INUIT. KIHIMI HAMNA OAKHIMAINIKOT PIVAKTOK.

PINAHUAKTAIN KIKHINGNIK HAVAKVINGNI UVVALU HAVAKVIN HALOMAKHIMALOGIN.

AUNGITIGUN LEAD KANGNIKTAUK NALVAKTAUVAKTUK MALGUNGNI IKPIAKYUNGMIUTANI HAPKONANI ANGUNAHUAKTUNILU AKHALUTITUKTUNILU. IMAITUNGNAKHIUT HAPKOA HAVAKATANGNIMIK INGNIKUTINIK IGLUNI ANGMAKUKITUNI. KIHIMI ALLAKUT LEAD KANGNIMIK PIKAGUNAKHIUK.

TITIGAKHIMAYAMINGNI ILALIKPAGA TAKTI W.A. FULLER ILITOKHOIYOTINIK. NALVAKHIMAYAIT ARSENIC KAKTUN NAONAIKHUGIN YALUNAIMIUTAN KABLUNAN AMIGAITKIAN ANGUTIN ANGNANIT NUYAIT ARSENIC KAKTUN. HAMNA HAVAKVINIT TAIMAILINAGUNAKHIYOK. KIHIMI KABLUNALU KABLUNANGUNGITULU OKIOLGIT O MIT 19 MUT ATYIKIKTAUNAHUAGANGAMIK KABLUNANGUNGITUN ARSENIC KAKPALATKIANGUVAKTUN. ARSENIC KANGNIK HUNAVLAUNGMIT PIYAUVAGUNGNAKHIUK KIHIMI NALUYUN. KANGIKHIMAYOKHAT KIHIMI ARSENIC KANGNIK HIVUGANAITUK TAYA.

KINGULLIKUN, FULLERM IHIVGIOKTAIN AUNGIN IKPIAKYONGMIOTAN NAONAIKPAKTON ARSENIC KAKTUN OYAGAKHIOKTUNI HAVAKTUN. KIHIMI KANUK ARSENIC KANGNIK NAONAITAIKHIMAITA.

ᐊᐅᓛᑦᓯᔩᑦ ᑲᑎᒪᔨᕋᓚᒋᓐᓄᑦ ᓇᐃᓴᒋᐊᖅᑕᐅᓯᒪᔪᑦ ᐅᓂᒃᑳᑦ

ᑖᒃᑯᐊ ᐸᐃᑉᐹᑦ ᐅᖃᐅᓯᖃᖅᐳᑦ ᓯᕗᓂᕈᑕᐅᖃᑦᑕᕐᒪᕐᓖᑦ ᑕᒪᒃᑯᐊ ᓯᑕᒪᐃᑦ ᓴᐱᕐᔭᒃᓴᔮᑦ ᒪᐅᕐᒪᑐᐃᓐᓈᖅᓯᒪᓕᕐᓚᑦ, ᑕᒪᒃᑯᐊ ᓯᑎᐊᑦ ᑐᕐᑯᓐᓇᕈᓐᓇᖅᑐᑦ (CADMIUM), ᓴᐱᕐᔭᒃᓴᖅ (LEAD), ᑐᕐᑯᓐᓇᖅᑐᖅ (ARSENIC), ᐊᒻᒪ ᒎᑦᑎᐅᓂᖅᑕᐅᔪᖅ (MERCURY) ᑕᒪᒃᑯᐊᒍ ᖃᓄᖅ ᑐᕐᕋᓂᖃᕐᒪᕐᓖᑦ ᓄᓇᓯᐊᕐᒥᐅᑕᓄᑦ. ᑖᒃᑯᐊ ᐅᖃᐅᓯᕆᔭᐅᓯᒪᔪᑦ ᐱᔭᐅᓯᒪᓚᐅᑎᒃ ᑕᒪᒃᑯᐊ ᖃᐅᔨᓴᕐᑎᐱᓃᑦ ᐅᖃᓕᒫᓚᐃᑦ ᐅᖃᓕᒫᖅᑕᐅᕙᒃᐸᑎᒃ ᐊᒻᒪ ᖃᐅᔨᓴᑦᑕᓇᕐᑐᑎᐱᓂᕐᓂᕐᓛᖅᓯᒪᔪᑦ.

ARSENIC ᑐᕐᑯᓐᓇᖅᑐᖅ ᖃᓄᖅ ᐊᕐᒥᑎᒥᓂᕐᓗᓄᑦ ᐃᓯᓛᒍᑐᑕᐅᒥᐊᕐᑲᖅᐳᖅ ᑭᓯᐊᓂ ᑕᒪᒃᑯᓇᓂ ᓄᓇᓂ ᑰᒍᕐᓂᒃ (GOLD) ᐅᔭᕋᕐᓂᐊᕐᕕᐅᕙᓂ, ᑰᒍᓂᒃ ᓴᒍᒻᒪᖅᓴᐃᔪᑎᐅᕙᒃᑐᑦ ᑕᐃᒪᓐᓇ ᓴᖅᑭᔮᖅᑎᑦᓯᓂᕈᓐᓇᕐᓚᑦ ᑐᕐᑯᓐᓇᖅᑐᒥᒃ (ARSENIC TRIOXIDE-ᕐᒍᓂᖅᑕᐅᕙᒥᒃ). ᑖᓂ ᔪᒐᓇᐃᒥ, ᐱᑕᖃᐅᒐᐊᓚᐅᖅᓯᒪᓪᓗᒐᐊᖅᑯᓂ ᑐᕐᑯᓐᓇᖅᑐᒥᒃ, ᑕᐃᒪ ᒥᑭᓪᓕᒥᐊᕐᕋᕙᒃᓯᒪᓕᖅᐳᖅ ᐆᒃᑐᑎᒍᑦ 1000-ᒍᑲᖅᓴᖅ ᑭᓯᐊᓂ ᐃᓯᓛᒍᖃᒐᐊᓕᖅᐳᑦ ᖃᐅᔨᓴᖅᑏᑦ ᐱᑕᖃᒐᐊᕐᒥᐸᓪᓕᐊᓕᕐᓂᐊᓄᑦ. ᓛᓐᓇᕈᓛᓚᐅᖅᑐᖅ ᖃᐅᔨᓴᕈᑕᐅᓯᒪᕙᓂ ᖃᐅᔨᕙᖃᖅᓯᒪᓕᖅᐳᖅ ᑕᒪᒃᑯᐊ ᐱᓕᕆᕝᕕᐊᕙᑦ ᐊᑐᕈᓐᓇᖁᔭᖅᑖᕐᒥᓐᓂᒃ ᑐᕐᑯᓐᓇᖅᑐᖃᐊᖅᑐᖃᕐᑯᓇᒍ.

ᐅᖃᓕᒫᓚᐃᑦ ᓄᓇᓯᐊᒍᑦ ᑐᕋᕐᓚᕙ ᐱᕙᑎᖃᖅᐳᑎᒃ ᖃᓄᖅ ᒎᑦᑎ ᓴᖅᑭᐸᐸᓕᐊᕙᕐᓚᕐᓖᑦ ᑕᓯᒥ ᐊᒻᒪ ᖃᓄᐃᓕᑎᕐᓯᒍᓐᓇᕐᓚᕐᓖᑦ ᐃᒪᐅᑉ ᐊᕙᒍᒥᐅᑕᓇᔭᕐᒥᓐᓄᑦ ᐊᒻᒪ ᐃᒪᕐᒥᐅᑕᐃᑦ ᓂᕿᕆᕙᑦᑎᕐᒥᓐᓄᑦ ᖃᓄᐃᒍᑕᐅᕙᕐᓚᕐᓖᑦ ᖃᐅᔨᓴᖅᑕᓚᐅᖅᐳᖅ. ᑕᒪᒃᑯᐊ ᓇᓯᕗᒍ ᑕᓯᒥᒍ ᐃᖃᓗᐃᑦ ᐃᓚᕐᒥᑦ ᒎᑦᑎᖃᒍᐊᕐᓂᕐᒥᑦ ᑖᕙᓂ ᐅᖃᐅᓯᕆᔭᐅᓯᒪᔫᑦ. (METHYLMERCURY-ᒧᑦ) ᑐᕐᑯᓐᓇᖅᑐᒥᒃ ᐱᔭᕙᑎᖃᖅᐳᑎᒃ ᑐᕐᑯᔭᕐᑲᕐᓂᐊᓄᑦ ᑎᑎᕋᖅᓯᒪᔭᖅᑕᖃᕐᒥᒃᑲᒐᐊᖅᑎᓪᓗᒍ, ᐊᕐᒥᔭᒻᒪᓇᐅᓂᕐᓗᓄᑦ, ᐱᑕᖃᖅᑐᒻᒪᓇᐅᓂᕐᓗᓄᑦᑑᓐᓃᑦ ᒎᑦᑎ ᖃᐅᔨᔭᐅᓯᒪᓕᖅᐳᖅ ᑖᓂ ᒪᑲᓯᕙᓕᐅᓂᖅᑕᐅᔭᒥ, ᐃᖃᓗᒃᑑᑎᐊᒥᒍ, ᐊᒻᒪ ᓄᓇᓕᕐᓂ ᐃᓚᕐᒥᓂ ᖃᑭᖅᑳᒍᕐᒥ. ᑕᓐᓇ ᖃᐅᔨᔭᐅᓯᒪᔫᖅ ᓂᕿᖃᒐᐊᖅᐸᕐᓂᕐᒥᓐᓄᑦ ᐆᒪᔭᕐᓂᒃ ᑕᒪᒃᑯᓂᕐᓗ ᒎᑦᑎᖃᒐᐊᖅᑐᓂᒃ, ᐱᕙᕐᓚᑦ ᐃᓄᐃᑦ ᐃᖅᑭᓚᒐᒍ ᑕᓯᒥᐅᑕᓂᒃ ᐃᖃᓗᕐᓂᒃ, ᓇᑦᓯᕐᓂᓪᓗ ᓇᑦᑐᕋᓗ ᑎᕐᑐᕐᒥᓐᓂᒃ, ᓂᕿᑐᐃᓐᓇᕐᓂᓪᓗ ᐊᒻᒪ ᒪᒃᑖᖅᑐᖃᑦᑕᕐᓂᕐᒥᓐᓄᑦ ᕿᓇᓗᕐᓂᑦ.

ᐅᖃᖅᓯᒪᔭᖅᑕᖃᖅᐳᖅ ᒎᑦᑎᐅᑉ ᑐᕐᑯᓐᓇᕐᓂᑎᒥᔭᕐᓗ ᓇᑦᓯᑦ ᓂᕿᕐᒥᓂ ᐊᒻᒪ ᑎᕐᑐᕐᒥᓂ ᖃᐅᔨᔭᐅᒍᓐᓇᖅᐳᖅ ᖃᐅᔨᓴᖅᑕᐅᒐᓂ ᒎᑦᑎᐅᑉ ᑐᕐᑯᓐᓇᕐᓂᓴᕐᓗ ᒎᑦᑎ ᓇᑦᓯᕐᑎᑦᑐᓕᓚᖅ ᖃᐅᔨᓴᖅᑕᐅᒐᓂ. ᓯᕗᓪᓕᖅᑰᕐᒥ ᖃᐅᔨᓴᕈᑕᐅᓚᐅᖅᑐᑦ ᖃᐅᔨᓐᓇᕈᑕᐅᓚᐅᖅᐳᑦ ᐅᖃᐅᓯᖅᓯᒪᕐᓚᑦ ᑕᒪᒃᑯᐊ ᓴᐱᔭᐃᑦ ᓈᒻᒪᓗᐊᕐᓂᕐᒥᓐᓄᑦ ᓂᕈᔭᐅᕙᒃᑐᓂᑦ ᑐᕐᑯᓐᓇᕐᓂᕐᒥᑦ ᓯᕿᐅᑕᐅᒐᕐᒥᕐᒥᓐᓄᑦ ᐅᖃᐅᓯᕆᔭᐅᓯᒪᕗᑦ. ᐅᖃᖅᓯᒪᕗᖅ ᑕᒪᓐᓇ ᑕᐃᒪᐃᓕᕐᓗᒃᐸᑦ, ᒎᑦᑎᐅᑉ ᑐᕐᑯᓐᓇᕐᓂᐊ ᐱᑕᖃᑎᐊᕐᑲᖅᑰᔨᕐᒥᓂᐊᓄᑦ ᐃᖃᓗᕐᓂ ᑕᒪᒃᑯᐊ ᐃᖃᓗᐃᑦ ᓴᐱᕐᔭᒃᓴᖃᖅᓴᐅᑎᒥᓂᕐᒥᓄᑦ ᑎᒥᒧᑦ ᐱᐅᔭᓂᒃ.

ᓯᑎᐊᖅ ᐅᔭᕋᒃᓴᖅ ᑐᕐᑯᓐᓂᕈᓐᓇᖅᑐᖅ (CADMIUM-ᕐᒍᓂᖅᑕᐅᔭᖅ) ᑐᕐᑯᓐᓇᕐᒥᒃᑲᒐᐊᖅᑯᓂ ᐆᓪᓚᖅᑎᑕᐅᓯᒪᕐᒥᑐᐊᕈᓂ, ᑐᕐᑯᓐᓇᖅᓯᒍᓐᓇᓪᓚᑎᒃᐳᖅ ᑕᓇᐅᕐᒍᔮᔭᓕᕈᓂ ᐅᑉᐹᓐᓃᑦ ᐳᔭᕐᒍᑐᐊᕈᓂ. ᑕᐃᒪ ᑕᒪᒃᑯᐊ ᐅᔭᕋᕐᓂᐊᕐᕕᕐᒥ ᐅᔭᕋᕐᓃᑦᑐᓂᒃ ᐊᑭᑐᔭᒃᓴᓂᒃ ᐊᐅᒃᓱᑦ ᑕᐃᒪᓐᓇᐃᑦᑐᒥᒃ ᑎᒥᒥᕐᓂ ᐱᑕᖃᓪᓚᑎᒃᐸᒃᐳᑦ. ᓄᓇᓯᐊᒥ, ᑕᒪᒃᑯᐊ ᐱᓕᕆᕝᕕᕐᓂ ᐱᓕᕆᕙᒃᑐᑦ CADMIUM-ᒥᒃ ᐱᑕᖃᐅᖅᐳᑦ ᑕᒪᒃᑯᐊ ᐅᔭᕋᕐᓂᐊᖅᑐᑦ LEADZINC-ᓂᒡᒍ, ᑭᓯᐊᓂ ᓄᓇᓯᐊᒥ ᐅᔭᕋᕐᓂᐊᖅᑕᒥᓂᒃ ᐊᐅᒃᑰᐸᕐᒥᓪᓚᑦ ᑐᕐᑯᓐᓇᖅᓴᖅᑐᖅ ᐱᑕᖃᓯᐊᕐᒥᓪᓚᓗᓴᖅᐳᖅ.

ᑕᒪᒃᑯᐊ LEAD-ᕐᒍᓂᖅᑕᐅᔪᑦ ᐅᒍᓇᐊᓇᒍᐊᖃᓯᒥᔭᐅᕗᖅ ᖃᑦᑐᓈᑦ ᓄᓇᕐᓚᓐᓂᓐᓇᖅ ᐱᑕᖃᕐᕕᓪᓚᐊᖄᒍᕐᓂ ᓴᐱᕐᔭᒃᓴᓂᒃ ᐊᐅᒃᑰᕐᕕᕐᓂ, ᐊᒻᒪ ᐱᑕᖃᖅᓯᖃᒍᐊᕐᒍᓂ ᐅᔭᕋᕐᓂᐊᕐᕕᕐᒥ ᐅᔭᕋᕐᓂᐊᕐᕕᐅᔭᒥ LEAD-ᕐᒍᓂᖅᑕᐅᔭᒥᒃ. ᓄᓇᓯᐊᒥ,

ᐅᔭᕋᒃᓂᐊᕐᕖᑦ ᐱᓕᕆᐊᕐᑲᖕᒍᑦ ᕐᑯᐊᒃᑐᐃᖓᖕᒍᒥ ᓄᓇᒥ ᐸᓂᖕᒍᒥᒃ ᐃᒌᑕᕐᑲᕐᓗᑎᒃ ᐃᒌᑕᕆᐊᕐᑲᖕᐸᒃᓚᑕ. ᑭᓯᐊᓂᑦᑕᐅᖕ, ᐳᕙᖕ ᐊᓂᖕᓴᖕᑕᐅᔪᓇᕋᔭᖕᒍᖕ ᐊᒻᒪᓗ ᐃᒌᑕᕐᓂᕐᔪᑦ ᐳᕙᖕ ᓯᕙᓂᓗᐊᕈᑕᐅᓴᕋᓯᒪᔭᐅᒃᒥᓚᖕ ᑐᖕᓗᐊᒃᔪᑦ ᐊᓂᐊᒃᒥᓇᐅᔭᕐᕕᕐᑲᕐᓂᒃᒥᓈᑦ ᐳᕙᕐᔪᑦ ᐊᓂᐊᕐᕕᐅᕙᒥᒃ. ᕐᑲᐅᔨᓴᕐᕚᒥᕙᒃᓗᑕ LEAD-ᑕᕐᑲᕐᒪᒃᒫᑦ ᐱᓕᕆᔾᔪᑦ ᐊᐅᒃᓚᓐᓂ ᕐᑯᐃᒃᒥᓐᓂᓗ ᓇᓂᓯᕝᕕᒃᒥ ᐱᓕᕆᕙᑦ ᐱᑕᕐᑲᖕᐸᓪᓕᐊᒻᒪᕆᒃᓯᒪᔪᖕ. ᑕᒪᓐᓇ ᑕᐃᒪᐃᓕᐊᕐᑭᐳᑰᖕ ᐱᓕᕆᔾᔪᑦ ᐅᔭᕋᒃᓂᐊᕐᕕᒃᒥ ᐊᒻᒪᒃᒥᒃᓂᒃ ᐃᕐᒥᑦᓯᐊᒃᒥᓗᐊᕐᑲᑦᑕᕐᓂᒃᒥᓐᓄᑦ, ᐊᓂᖕᓴᕐᑲᑦᑕᕐᓂᒃᒥᓐᓄᑦ ᑕᐃᒪᐃᑦᑐᒥᒃ ᐱᕙᕆᑎᕐᑲᓗᐊᒃᒥᒃᑳᖕᓗᑎᒃ. ᑕᐃᒪᓕ ᓴᓗᒻᒪᖕᓴᕐᑯᔪᐅᓪᓗᑎᒃ, ᐃᕐᒥᑦᓯᐊᕐᑲᑦᑕᕐᑯᔪᐅᓪᓗᑎᒃ ᐱᓕᕆᔾᔪᑦ ᐅᕐᑲᐅᑎᔪᐅᕙᓕᖕᐳᑦ ᐊᒻᒪᓗ ᐳᕙᕐᓰᑎᑕᐅᓗᐊᖕᑕᐃᓕᒪᑎᑕᐅᒪᓯᒃᓗᑎᒃ.

ᐊᒻᒪᓗ ᐃᒃᐱᐊᕐᕙᒃᒥ ᐊᐅᒃᒥ LEAD-ᑕᕐᑲᖕᒍᕐᑲᓚᐅᑎᓚᐳᖕ ᐃᓄᒃᓂ, ᑖᒃᑯᐊ ᐊᒃᑐᓇᓯᐊᖕᕖᒃᓗ ᓄᓇᓕᑎᕈᓂᓗ ᐱᓕᕆᕈᑦ. ᑕᐃᒃᑯᐊ ᓯᕐᑯᒥᓯᒪᕙᓂᒃ ᐋᖕᑭᒃᓯᐃᑎᑕᐅᕐᑲᑦᑕᕐᓂᒃᒥᓐᓄᑦ ᓄᓇᓯᐅᑎᕐᑲᕐᕕᒃᒥ ᑐᖕᓗᐊᕐᑲᑦᓯᐊᒃᒥᑦᑐᒥ ᓴᓇᕐᑲᑦᑕᕐᓂᒃᒥᓐᓄᑦ ᑕᐃᒪᐃᓐᓇᓯᒥᔭᐅᓚᐅᖕᓗᑎᒃ. ᑭᓯᐊᓂᑦᑕᐅᖕ, ᑐᖕᓗᐊᕐᑲᒃᒥᓗᐊᖕᒍᒥ ᓴᓇᕐᑲᑦᑕᕐᓂᒃᒥᓐᓄᑦ ᑕᐃᒪᓐᓇᐃᑦᑐᖕᑖᓕᐊᕐᑭᓂᒃᒥᓐᓄᑦ ᐃᓯᒪᒥᔭᐅᕙᑐᐊᒃᑐᓚᐅᒃᒥᕐᐸᑦ ᐊᓯᐊᒍᑦᑕᐅᖕ ᑕᐃᒪᓐᓇᐃᑦᑐᕐᑲᓕᐊᕐᑭᓂᒃᒥᓐᓄᑦ ᕐᑲᐅᔨᓴᖕᑕᐅᒍᒪᓚᐅᖕᐸᑦ.

ᑕᐃᒪ ᔫᒃᑖᑉ ᑖᒃᑐ ᐳᔫᑉ ᐅᓂᒃᑳᐱᓂᒃᒥᑦᑕ ᐃᓚᒥᐊᕈᑎᒃᒥᓐᓂ ᐅᕐᑲᖕᓯᒪᕙᖕᑕᕐᑲᖕᐸᖕ ᕐᑲᐅᔨᓴᖕᓇᐊᕈᑎᕐᑲᒃᑲᓐᓂᓚᐅᕐᓂᐊᓄᑦ ᑕᒪᑐᒪ ᒥᖕᓴᓄᑦ. ᕐᑲᐅᔨᓴᕈᑎᐱᓂᒃᒥᓐᓂ ᔪᓗᓇᐃᒥᐅᓂᒃ ᑐᕐᑯᓇᖕᑐᕐᑲᕐᓂᒃᒥᓐᓄᑦ ᓄᔭᒃᒥᓐᓂ ARSENIC-ᒃᑐᓂᕋᖕᑕᐅᕙᒥᒃ, ᕐᑲᓪᓗᓈᑦ ᕐᑲᐅᔨᓴᖕᑕᐅᕙᑦ ᐊᒃᑐᑎᒃᒥᑦ ᑐᕐᑯᓇᖕᑐᖕᓴᕐᑲᕐᓂᖕᕙᒃᑐᓚᐅᖕᐸᑦ ᐊᕐᓇᓂᓂᑦ. ᑕᒪᓐᓇ ᐱᔭᕙᑎᕐᑲᕋᓯᒥᔭᐅᓪᓗᓂ ᓴᓇᐳᕐᑲᕐᓂᖕᓴᐅᒃᓚᑕ ᐊᒃᑐᑎᓂᒃ ᐅᔭᕋᒃᓂᐊᕐᕕᒃᒥ. ᑭᓯᐊᓂᑦᑕᐅᖕ, ᑕᒪᒃᑯᐊ ᕐᑲᓪᓗᓈᒃᑐᒃᒥᑦᑐᓪᓗ ᕐᑲᓪᓗᓈᓪᓗ ᐅᑭᐅᓖᑦ 0-ᒥᑦ 19-ᒧᑦ ᐆᒃᑑᑎᒥᔭᐅᓕᕐᓚᑕ, ᑕᒪᒃᑯᐊ ᕐᑲᓪᓗᓈᒃᑐᒃᒥᑦᑐᖕ ᑐᕐᑯᓇᖕᑐᕐᑲᕐᓂᖕᕙᒃᑐᓚᐅᖕᐸᑦ. ᑕᐃᒪ ᓄᓇᐅᑉ ᐊᕙᓗᒥᐅᑕᒃᒥᓂᑦ ᐱᕙᕆᑎᕐᑲᖕᓗᑎᒃ ᑕᐃᒪᓐᓇᐃᓇᓯᒥᔭᐅᖕ, ᑭᓯᐊᓂ ᓯᓚ ᑕᒪᓐᓇ ᕐᑲᐅᔭᐅᓚᑎᓚᐅᕋᓂ. ᕐᑲᐅᔮᒪᔭᐅᔭᑎᐊᕐᑲᖕᐸᖕ ᑕᒪᒃᑯᐊ ᑐᕐᑯᓇᖕᑐᖕ ᒥᑭᓂᖕᓴᐅᓚᐅᕐᓂᒃᒥᓐᓄᑦ ᑕᒪᒃᑯᓇᓂ ᕐᑲᐅᔨᓴᖕᑕᐅᓗᐊᖕᐸᒃᓂᖕᓴᐅᕙᓂᑦ.

ᐊᒻᒪᓗ, ᑖᑦᓯᒪ ᔫᒃᑖᑉ ᕐᑲᐅᔨᓴᕈᑎᐱᓂᒃᒥᑦ ᐊᐅᒃᒥ LEAD ᐱᑕᕐᑲᕐᓂᐊᓄᑦ ᐃᒃᐱᐊᖕᕙᒃᒥ ᐱᑕᕐᑲᓗᐊᕐᓂᖕᓴᐅᓂᐊᓄᑦ ᑕᐃᒪᓐᓇᐃᑦᑐᖕ ᐃᓄᒃᓂ ᐅᔭᕋᒃᓂᐊᕐᕕᒃᒥ ᐱᓕᕆᔪᕙᓂ. ᐅᕐᑲᓚᐅᖕᓗᓂᓗ ᓯᓇᒧᑦ ᐱᕙᕆᑎᕐᑲᖕᓗᓂ ᑕᐃᒪᓐᓇᐃᒃᒪᒃᒫᑦ ᕐᑲᐅᔭᐅᓯᒪᒃᒥᓐᓂᐊᓄᑦ ᓯᓚ.

Introduction

Mercury, lead, arsenic and cadmium were selected from amongst the wider spectrum of metallic contaminants of the environment because they have caused, or have been suspected of causing, a problem to inhabitants of the Northwest Territories. As industrialization proceeds other problems may arise or may be perceived as potential hazards, especially if the proximity of high grade ores to local sources of fossil fuels leads to the development of on-site refining and smelting.

Of the four metals discussed lead and arsenic are of local importance only as a result of mining activities; cadmium was at one time thought to be of local importance to the human population though it is wide-spread in the marine fauna; and mercury has been shown to be widely distributed in the Arctic environment and has shown up in a variety of species at quite astonishingly high levels but with no toxic impact detected.

No attempt is made to cover the literature on the toxicology of these metals outside the context of the current situation in the N.W.T., and even the historical situation in the N.W.T. is largely ignored because it has been both complex and controversial.

Nor is more than passing reference made to contaminant levels in species other than man except where they are perceived as relevant to the human situation. There is a large literature available on such levels, mostly in publications of the Departments of Environment and of Fisheries and Oceans, and in departmental reports on specific situations.

Finally no attempt is made to review material that has already been published or has appeared in government reports except where such material is essential to current understanding.

Arsenic

Arsenic levels in man are commonly measured in tissues such as hair and fingernails, which give an indication of long term accumulation. In general, in the absence of extrinsic deposition, hair levels greater than 5 parts per million (ppm) are considered to indicate a possible hazard to health. Arsenic may also be measured in urine. Urine levels are indicative of acute, or short-term exposure rather than chronic exposure. In general, urine levels greater than 100 parts per billion (ppb) represent a health hazard, but levels 20 times this may follow ingestion of pentavalent arsenic in seafood such as crab, lobster, shrimp, etc.

Arsenic is widely found in a combined form in igneous rock throughout the world. In the N.W.T. it is most frequently associated with gold-bearing ores in which there is often a major content of arseno-pyrite, an insoluble mineral that contributes little if any arsenic to the food chain under normal circumstances.

Arsenic becomes a health problem when native arseno-pyrite is changed to arsenic trioxide during ore roasting and smelting. Arsenic trioxide (As_2O_3) volatilizes and is readily dispersed in the atmosphere unless meticulous care is directed towards its recovery.

During the early years of gold extraction from mines in the Yellowknife area huge quantities of arsenic were released to the atmosphere because no effort was made to trap arsenic trioxide and dispose of it in a safe fashion. Devilliers & Baker (1973) estimated that up to 11 tons of arsenic trioxide per day were liberated into the atmosphere through the stacks in the late 1940s. Much of this fell back to earth in the immediate neighbourhood of the producing mines.

Subsequent to that time several processes have been used, with steadily increasing efficiency, to entrap and retain arsenic trioxide, so that today estimated daily losses to the environment are less than 10 kg (20 lbs.) per day, an amount of little health significance to residents of the area.

Certain problems have, however, been produced by the entrapment procedures because the arsenic produced was concentrated and thus caused local "hot-spots" of intense arsenic pollution. For example, wet scrubbers used in the Con Mine resulted in a concentrated, arsenic-containing slurry that was disposed of into Arsenic "Ponds" on Con Mine property. The "Ponds" remain to this day, although no arsenic has been fed into them since the cessation of the roasting process at Con Mine about 10 years ago. They probably contain about 29,000 tonnes (32,000 tons) of dry arsenic trioxide (Soniassy and Brown 1980).

In the Giant Mine, where ore is still roasted, particulate arsenic-containing material is extracted in electrostatic precipitators, and condensed arsenic trioxide is collected from the cooled exhaust gases in a large bag-house from which the arsenic is led away either for purification reprocessing or to storage in sealed underground stopes. This collection procedure, though it virtually eliminates the exterior hazard, results in a work environment in which a small number of employees find themselves in a highly contaminated area where they must wear protective clothing, use a controlled breathing apparatus, and practice rigorous personal hygiene to avoid excessive arsenic intake. Despite all these measures, the bag-house workers constantly have urine arsenic levels that approach or exceed recommended maxima. Hair arsenic in these workers, and in some others in less exposed situations, is usually very high, probably as a result of external contamination of the hair shaft by airborne arsenic-containing dust.

In 1966 Medical Services carried out an intensive survey of the inhabitants of the City of Yellowknife.

The survey was a carefully designed and elaborate investigation into all aspects of possible arsenic effects in both city residents and mine workers. The findings (Devilliers & Baker 1973) were substantially negative with the only definitive evidence of arsenic toxicity being dermatoses of sweaty areas in some individuals working in areas of high exposure to arsenic. No long term effects of arsenic toxicity were detected either in the gold mine workers or in the citizenry.

In 1975, following allegations made on the CBC radio program "As it Happens" the Yellowknife arsenic question was reopened and a preliminary survey of arsenic levels in hair was carried out on a sample of 705 residents of the town. The sample contained mine and mill workers, native people, and long term residents. The results (Tables 1 to 4) demonstrate that the general population of Yellowknife was not abnormally exposed to arsenic (Eaton 1975). Adult males not employed at the gold mines and adult females wherever employed showed no abnormally high arsenic levels. High levels were found in some employees at Giant Mine who were associated with the bag house, the cottrell or the roasters, and in some laboratory workers, pipefitters, etc. whose duties took them into high dust areas of the mill. Underground workers in general showed levels that were not elevated or only moderately higher than the average. In addition some children and youths, particularly males, from both Indian and white families, had a modest elevation.

The high levels in a few children were probably a result of external contamination. The same may be true for many of the high levels in mill employees. However, there is a distinct possibility of a toxic hazard for bag-house and roaster workers and possibly also for those in the laboratory.

In a second phase of this study all individuals showing hair levels in excess of 10 ppm were invited to undergo a thorough medical examination and the majority availed themselves of the opportunity. This part of the study showed that the children and laboratory workers who had high hair levels had low urine levels thus confirming that their high hair levels resulted from external contamination rather than ingestion or inhalation.

As a result of this inactivity the news media once again became involved, this time at the behest of the unions, who saw that they had a problem, and the Indian population, who believed that they had a problem but had not. Rhetoric flowed in print and on the air waves to the pronounced embarrassment of the Federal Ministry of Health. To attempt to resolve the dilemma, and to temper the heat, the Canadian Public Health Association (CPHA) was commissioned by the then Minister of Health to undertake a review of the entire Yellowknife arsenic story and to make recommendations for the future. An independent three-man task force, which was appointed early in 1977, gathered evidence and held hearings in Yellowknife and Ottawa and published a final report in December, 1977 (CPHA 1977). Although the report revealed nothing that was not already known it provided a set of guidelines that enabled a number of departments of government to work with each other, and with the mines and unions, to develop a code of conduct within the industry that might be expected to reduce the residual hazard.

TABLE 1

FREQUENCY OF OCCURRENCE OF VARIOUS HAIR ARSENIC LEVELS IN 350 NON-NATIVE MALES BY AGE GROUP. YELLOWKNIFE, FEBRUARY, 1976.

PPM Arsenic	Age Group 0-4	5-9	10-	15-	20-	30-	40-	50-	60-	70+	Unknown	Total
0.0-0.9	1	1	3	12	14	11	5	4	1	2	1	55
1.0-4.9	2	13	24	18	35	24	34	19	11	2	4	186
5.0-9.9	–	3	4	2	8	9	11	6	1	1	2	47
10.0+	–	–	–	4	14	15	14	6	4	1	4	62
Total	3	17	31	36	71	59	64	35	17	6	11	350

Although bag-house workers had elevated urine levels indicating excessive arsenic intake, the levels were lower than those usually associated with clinical toxicity, and no chronic arsenicism was demonstrated.

Although these investigations identified a work hazard, jurisdictional problems seemed to make it impossible at the time to take effective action. Responsibility for health and safety in mines and associated above-ground buildings was vested in the Department of Indian and Northern Affairs (DINA) under the N.W.T. Mining Safety Ordinance. Although Medical Services perceived the hazard and actually increased the inspectorate to allow for coverage of such industrial situations, they had no legislative authority to insist on control measures. Though both Giant and Con Mine had been co-operative in the course of the investigation and were taking steps on their own to rectify deficiencies which they perceived there was no mechanism for supervision by Medical Services.

The task force also mounted an electromyographic survey of residents of Yellowknife and, for comparison purposes, of Hay River which has a similar population mix but no arsenic. Though hampered by indifferent support by the groups allegedly most interested they found no evidence of arsenically-induced nerve conduction deficit in the City of Yellowknife (CPHA 1978).

Another spin-off from the Task Force report was an investigation of the arsenic content, and hence the safety as food, of vegetables grown in the Yellowknife area. As might be expected no level of arsenic was observed that would render

TABLE 2

FREQUENCY OF OCCURRENCE OF VARIOUS HAIR ARSENIC LEVELS IN 292 NON-NATIVE FEMALES BY AGE GROUP. YELLOWKNIFE, FEBRUARY, 1975.

PPM Arsenic	Age Group 0-4	5-9	10-	15-	20-	30-	40-	50-	60-	70+	Unknown	Total
0.0-0.9	—	1	19	10	37	46	39	29	6	3	6	196
1.0-4.9	2	17	21	7	9	8	13	5	5	1	3	91
5.0-9.9	—	1	1	—	1	1	—	—	—	—	—	4
10.0+	—	—	—	—	1	—	—	—	—	—	—	1
Total	2	19	41	17	48	55	52	34	11	4	9	292

such vegetables unfit for consumption. This was a notable improvement from findings 20 or more years previously when leafy vegetables, as well as berry fruits had been classed as unfit for consumption as a result of arsenic contamination of surfaces, and was a direct reflection of the massive reduction in airborne emissions that had occurred (Soniassy 1978).

Finally, in a study that is still incompletely analyzed, the Canadian Association of Smelter and Allied Workers (CASAW), with the collaboration of Giant Mine and the three full-time bag-house workers launched a 90-day study of arsenic excretion as a reflection of work activities, protective devices employed, and diet. One of the most striking findings was a huge increment in arsenic excretion following ingestion of marine crustaceans such as shrimp or lobster. These animals show tissue arsenic levels of 50-60 ppm or more, which however, is in an organic form, is not toxic to man, and is rapidly excreted in the urine. However, such findings vitiate biological monitoring of arsenic-exposed workers unless diet is controlled, or at least taken fully into account (Braid 1978).

Data generated by this study are being reviewed by the statistical team of the Canadian Centre for Occupational Safety and Health. Their findings are awaited with keen anticipation.

TABLE 3

FREQUENCY OF OCCURRENCE OF VARIOUS HAIR ARSENIC LEVELS IN 24 NATIVE MALES BY AGE GROUP. YELLOWKNIFE, FEBRUARY, 1975.

PPM Arsenic	Age Group 0-4	5-9	10-	15-	20-	30-	40-	50-	60-	70+	Total
0.0-0.9	—	—	—	—	1	1	—	—	—	2	4
1.0-4.9	—	—	1	1	4	2	—	1	2	1	12
5.0-9.9	—	—	1	1	2	1	—	—	—	1	6
10.0+	—	1	1	—	—	—	—	—	—	—	2
Total	—	1	3	2	7	4	—	1	2	4	24

TABLE 4

FREQUENCY OF OCCURRENCE OF VARIOUS HAIR ARSENIC LEVELS IN 37 NATIVE FEMALES BY AGE GROUP, YELLOWKNIFE, FEBRUARY, 1975.

PPM Arsenic	Age Group										
	0-4	5-9	10-	15-	20-	30-	40-	50-	60-	70+	Total
0.0-0.9	—	—	1	3	9	2	—	—	—	—	15*
1.0-4.9	—	1	1	2	3	1	3	1	3	—	15
5.0-9.9	—	1	2	2	—	--	1	1	—	—	7
10.0+	—	—	—	—	—	—	—	—	—	—	—
Total	—	2	4	7	12	3	4	2	3	—	37

* Includes 3 female Inuit.

Mercury

Mercury, like arsenic, tends to accumulate in metabolically inactive tissues such as hair and it can be detected in body fluids such as blood. Acceptable hair levels are generally less than 7 ppm, and acceptable blood levels are less than 20 ppb. A conversion factor is sometimes applied to relate hair levels to blood levels, but the conversion must be accepted with caution.

Mercury occurs in mineralized areas of the earth's crust either as the free metal or as a sulphide in the form of cinnabar. Elemental mercury is found as dissolved vapour in fresh and salt water and in rain (Landner & Larsson 1972). Mercury vapour is liberated to the atmosphere from volcanoes, by natural out-gassing from free mercury in the earth's crust, and in like manner, but at an accelerated rate, from perturbations of the earth's crust due to mining activities (Charlebois & Rivers 1977). It has been shown (Jonasson 1973) that mercury collects in the snow overlying mineralized deposits at a concentration which may reach fifty times that over non-mineralized areas. Eaton (1979) suggested that snow, acting as a distillation trap, may account for localized variations in concentration of mercury in surface waters and thus be the ultimate basis for observed higher levels of tissue mercury in predatory fish living in isolated lakes on the Canadian Shield. Rivers coursing through mercury-bearing rock carry dissolved mercury to the sea (Nelson et al., 1977).

Further additions to the mercury burden of the biota occur as a direct result of the introduction of mercury compounds to surface waters in the form of run-off of fungicides used agriculturally, and as wastes from industrial operations using organic or inorganic mercury compounds. Major epidemics of neurologic disease occurred in Japan in the communities of Niigata and Minamata due to organic mercury in industrial waste products.

The paper industry has been responsible for greatly enhanced levels of organic mercury in freshwater fish and sediments in several locations in southern Canada (Wheatley, 1979). In the N.W.T. increases in mercury levels of fish and other biota have been found locally associated with run-off from gold mine tailings dumps in which waste mercury from the amalgam process of gold extraction had been discharged (Moore et al. 1978).

MERCURY IN FRESH WATER

Mercury levels in the rivers of Western Canada, including the Continental N.W.T., were compiled by Niemanis (1976). Though figures for the N.W.T. are relatively scanty they are all acceptably low. Oddly, levels at all points sampled were higher in 1973 than more recently by a factor of about 5. Since there had been no changes in industrial activity that could have led to such a reduction, it seems necessary to propose that a change in sampling method, laboratory methods, or reading of results must be held responsible for the reported differences.

MERCURY IN FRESH WATER SEDIMENTS

Nelson et al. (1977) have studied the carriage of mercury ores in Alaska in rivers and have shown a rapid return to background levels by dilution downstream, and rapid settling of the high density ore particles. Such a rapid settling is postulated also for mercury in sediments as a result of metallic pollution from amalgamation sites. Attention is drawn to the difference between this pattern and that of simultaneously bacterially-polluted European rivers.

MERCURY IN SEA WATER

It has been suggested that a possible source for the observed high levels of mercury in marine mammals in the Arctic is industrially released pollutants coming from the Lena and Ob' Rivers draining Siberia. The Eastern half of Siberia drains into the Western Arctic and natural clockwise rotation of the waters westward (from the Canadian viewpoint) of the Lomonosov Ridge has been postulated to carry such pollutants from the Russian industrial regions to the Canadian side. However, Weiss et al. (1974) found that mercury levels in the Beaufort Sea off the Alaskan North Slope were substantially lower than those in the Atlantic and Pacific oceans by a factor of 3 to 10. Johnson and Wong (1976) also demonstrated that mercury levels in the Beaufort Sea were low and did not vary much in samples taken above, below and at the pycnocline. They also found that mercury levels in the upper sediments were only about 10% of those in samples taken from the Atlantic and Pacific Oceans (31-109 ng/g dry weight compared with 400-965 ng/g). These findings are surprising in view of the observed levels in seals and polar bears (see below).

Johnson and Wong (1976) also reported mercury levels in ciscoes and plankton. Cisco levels were in the same range as found in other non-piscivorous fish in similar habitats (35-75 ng/g). Plankton levels, however, average 0.93 μ g/g, which is about 30 times the amount reported by Weiss et al. (1974) elsewhere in the Beaufort Sea. Johnson and Wong expressed doubts about the validity of this part of their work.

MERCURY IN THE FOOD CHAIN

Mercury reaches the biota either as elemental mercury in the form of dissolved vapour or as an organic compound. Inorganic mercury sulphide, which is found as the mineral cinnabar, is insoluble in water and non-reactive. However, it is soluble in acids such as occur in acid rain.

Elemental mercury plays little part in the food chain. Before being taken up by higher organisms mercury, in solution or in sediments, is methylated by bacterial action (Jenson & Jernelov 1969). As with all biological conversions this reaction is reversible and demethylation may also occur when methylmercury is present in excess.

Biologically produced methylmercury may be ingested, along with the producing bacteria, by crustaceans, and thus enter the food chain near the bottom end. It may also enter the food chain higher up by direct absorption through the gill membranes of fish. Such absorption leads to a baseline methylmercury level in all freshwater and sea fish that is probably between 25 and 75 ppb.

Above this base level further concentration occurs in piscivorous fish such as lake trout, northern pike and pickerel, which may accumulate levels of organic mercury up to 2 to 3 ppm by simple biological accumulation over time. Such levels may occur in the absence of all man-made pollution and are found in the oceans in fish such as shark, marlin, and tuna, and in freshwater fish in highly mineralized areas, most noticeably on the Canadian Shield. Thus levels of 1 to 3 ppm have been identified in widely divergent sites from Victoria Island through the Central Keewatin to Arctic Quebec (Sherbin 1979).

Organic mercury levels greater than 3.0 ppm in fish appear to be associated always with man-made releases of inorganic or organic mercury to the aquatic environment. Thus the well documented occurrences at Minamata and Niigata in Japan and in the Wabigoon-English River system downstream from Dryden, Ontario, are to be attributed to the release of both metallic and organic mercury, whereas high levels in Gaique Lake in Mackenzie District, N.W.T. result from inorganic mercury deposits in the tailings from gold extraction activities. In both the latter areas individual fish have been taken with mercury levels higher than 10.0 ppm.

Berglund et al. (1971) put the acceptable level for mercury in food fish at < 0.5 ppm. If their criterion is accepted, there are potential hazards for heavy fish eaters in Canada in areas that have not been contaminated industrially as well as in those that have. To date no unequivocal evidence of mercury toxicity in man due to fish consumption in the absence of industrial pollution has been demonstrated (Taylor, 1976).

Work on mercury levels in Canadian seals has been reported in two papers by Smith & Armstrong (1975, 1978) and by Sergeant & Armstrong (1972). All have demonstrated remarkably high mercury levels in seal liver — up to 420 ppm in the liver of one adult bearded seal taken near Holman but averaging 27 ppm in a large sample (90+) of ringed seals. Smith and Armstrong (1978) also compared mercury levels in seals taken from a variety of locations across the Canadian Arctic and concluded that there was no significant difference in mercury levels among the various locations. However, they paid little attention to the age of the seals in their samples when they made their calculations.

Eaton et al. (1978) pointed out that, based on the data of Smith and Armstrong (1978) the rate at which liver mercury increased during the life of the seal was about twice as fast in seals from Amundsen Gulf as in seals in Barrow Strait and three times as fast as in Hudson Strait, which suggests a richer source of dietary mercury in the western than in the eastern arctic.

Eaton et al. (1980) found a similar level of mercury (27 ppm) in the livers of a sample of 119 ringed seals from the Holman area. They also pointed out that about 97% of the liver mercury is inorganic and less than 1 ppm is in the form of alkyl-mercury. Seal meat contains much less mercury than seal liver (up to about 2 ppm) though a greater proportion of it is organic. The mean alkylmercury level in ringed seals is thus less than 1 ppm in all tissues.

Mercury also occurs in polar bears. Once a polar bear becomes entirely independent of its mother, the hair mercury level provides an index to the relative, though not the absolute mercury content of its diet. Mercury levels are low in suckling cubs.

Westoo (pers. comm.) assayed mercury levels in hair from five bears killed or found dead along a north-south gradient in the eastern Canadian Arctic. Her figures suggest that mercury levels increase with increasing latitude.

Eaton et al. (1982) assayed hair from 109 live bears taken from Amundsen Gulf, the region of Cornwallis Island, the north side of Hudson Strait and Hudson Bay (Table 5). Excluding sucklings, the mean mercury level in bears from Amundsen Gulf was approximately ten times the mean level for Hudson Strait. Those from Cornwallis Island had intermediate levels.

Lentfer (1976) found that levels of mercury in the livers of polar bears taken in the Point Barrow area were seven times as high as those in livers of bears from western Alaska

bordering on the Chuckchee Sea. The difference was not attributable to a difference in age of the bears in the sample.

Thus, from three studies on two species at the top of the food chain, seals and polar bears, we have evidence to suggest a much greater intake of mercury in the area of the Beaufort Sea than elsewhere in the Arctic. In discussing this perceived difference Lentfer (1976) quoted W.A. Galster who suggested a submarine geologic source of mercury in the Beaufort Sea bottom. This is an attractive argument but difficult to bring into line with the very low findings of mercury in the water and sediments of the Beaufort (Johnson & Wong 1976, Weiss et al. 1974) already described.

According to Stirling & Archibald (1977) there are differences in the feeding habits of bears in Hudson Strait and bears of the Amundsen Gulf area. These could conceivably be responsible for differences in acquired mercury. Similarly, Burns, cited by Lentfer, observed feeding differences between Beaufort and Chukchee bears. However, these could not account for the observed differences in the rate of accumulation of mercury by seals, and the correspondence which exists between the mercury levels in seals and in bears seems too strong to be easily dismissed as an unrelated coincidence.

MERCURY IN MAN

Prior to the discovery of high mercury levels in marine mammals in the Hudson Bay area no attention had been paid to the possibility of high mercury residues in man. Because of the unexplained discovery of what have subsequently been shown to be quite modest total mercury levels in the livers and meat of beluga whales, coupled with failure to differentiate between total mercury and methyl mercury, a number of steps were taken by various Federal Agencies. First, the Inuit of Hudson Bay area were advised to curtail their consumption of whale and seal products. (Fortunately they took little notice of this advice). Second, the Muktuk Cannery at Whale Cove was closed because it was turning out a product containing more than 0.5 ppm of mercury. Third, the British Columbia consulting firm, Environmental Research Consultants Ltd. (E.R.C.), was contracted to carry out an investigation into mercury levels in selected communities across the N.W.T.

This survey showed unexpectedly high levels of mercury (though not dangerously so even on the basis of then current knowledge) in several settlements. The evidence thus gathered was one of the arguments in favour of a complete survey of all N.W.T. settlements, which was started in 1976.

These investigations into mercury in man were part of a general survey of such levels across Canada and were triggered, and coloured, by the discovery of mercury in fish in the Wabigoon-English River system of Northwestern Ontario. This was in a way unfortunate because it produced a sense of alarm and concern in the entire country which, in retrospect, was inappropriate. However, without that concern it is probable that the investigations would not have been made and that the situation would still remain unclear.

The survey in the N.W.T. showed two significant dietary sources of mercury. Among Indian residents of the Mackenzie Valley there were a few isolated individuals showing levels above 30 ppm in hair. These were traced to heavy dependence on freshwater fish as a source of protein. In each case the mercury showed up as a time-limited spike and possibly reflected the results of a single catch. In no case has it proved possible to pinpoint a population of high mercury content fish to account for the observed levels.

A level of mercury in hair samples, which suggests a short term blood level of up to 180 ppb, and which appears to be derived from fish, has been measured in an Inuit family resident in Cambridge Bay. The family depended strongly on lake trout and char and used little seal. The head of the family had earlier made his living as guide at a fishing camp on a lake identified only as "Char Lake" some 60 miles north of the settlement. Lake trout from Char Lake contained less than 1 ppm of mercury, but trout from Ferguson Lake, which is closer than Char Lake to Cambridge Bay have shown levels of up to 2.8 ppm.

Hair levels in coastal-dwelling Inuit from communities on the northeast coast of Baffin Island have been rather more intensively studied than those elsewhere, and in those communities it appears that seal tissues are the most likely source of ingested mercury. Older Inuit preferentially eat seal liver, and seals are eaten year around, which would account for the observed lack of seasonality in mercury deposition. Mean blood levels of mercury in Inuit residents of Arctic Bay approximate 20 ppb, with a range of 5-65 ppb.

One final, somewhat anomalous group, was identified first by E.R.C. in Tuktoyaktuk and was subsequently confirmed by Health & Welfare Canada. The source of this mercury has been extremely difficult to identify with certainty, though from an epidemiological standpoint it seems that the beluga whale is the most likely source. The high levels are found only in Inuit residents of Tuktoyaktuk, while in Inuvik the mean and highest Inuit levels exceed either Indian or White levels. The Indian and the Inuit diet, among those subsisting to a great extent on native protein sources, differ only in respect of whale meat and muktuk which are eaten by the Inuit but not by the Indians.

In the rest of the N.W.T. mercury levels have proven to be generally higher than observed southern norms, but not of a level to generate excessive concern. They appear to reflect the degree of dependence on seal meat and seal liver. For example, in Sachs Harbour where there are high mercury levels in seal and polar bears high human hair levels might be expected, but there is virtually no use of seal in the diet, and hair levels are uniformly low.

In the communities strung along the west shore of Hudson Bay, where caribou forms the major protein source, mercury levels are also low. Only some members of the island community of Sanikiluaq showed some increased levels, the source of which has not as yet been identified, but may well be seal.

There is an anomaly in the communities along Hudson Strait, those on the North side having relatively low mercury levels, whilst among those on the Quebec side there are a significant number of high levels. This situation is under investigation by Quebec Region, Health & Welfare Canada. There must be a dietary difference to account for it, but it is yet to be identified. Perhaps there is a greater use of landlocked salmonid fish on the Quebec side of the Strait.

TABLE 5

TOTAL MERCURY (PPM) IN POLAR BEAR HAIR SAMPLES BY GEOGRAPHIC AREA OF CAPTURE, 1977-1980

Location	Date	(n) Sample Size	Mean	S.D.	Range
Amundsen Gulf	1977	5	18.54	14.52	9.3-44.3
Cornwallis Island	1980	7	7.85	4.49	3.6-17.3
North Baffin Island	1980	27	6.93	1.80	4.5-11.8
Cornwallis Island	1977	7	6.59	1.72	4.5- 9.2
Clyde River	1980	9	4.92	0.63	4.0- 5.8
South Baffin Island	1977	13	3.53	0.77	2.7- 5.7
Southern shore of Hudson Bay	1980	41	2.54	1.00	1.1- 6.3

EXPERIMENTAL WORK

Because of the apparent discrepancies between clinical observations (zero) and predictions of mercury toxicity as a result of high mercury levels in the meat and liver of seals and other sea mammals, the Northern Medical Research Unit undertook to conduct studies of the toxic potential for laboratory cats of a diet consisting largely of liver of ringed seals. This preliminary work (Eaton et al. 1980) showed first that the total mercury content of seal liver bore no relation to the rate of accumulation of mercury in cat tissues, and second, that only the low methylmercury fraction contributed to the body stores, the remainder apparently being excreted. A diet composed of 68% seal liver with a mean mercury content of 27 ppm produced no clinical or histological evidence of mercury toxicity and raised tissue levels to only about 10% of levels known to be associated with frank toxicity. This intake (100g/day for a 3.5 kg cat) is roughly equivalent to 2 kg (4.4 lbs.) of liver daily for a 70 kg man, patently an unthinkable level for human diet.

As a corollary to this work, which involved the use of 119 seal livers, selenium levels were also measured in every sample. These assays showed that mercury in seal liver was matched on an equimolecular basis by selenium as previously reported by Koeman et al. (1975) and Smith & Armstrong (1978). The presence of selenium appeared to be related to a detoxification and storage mechanism for methyl mercury.

Such a concept has been widely held (Ganther et al. 1972) and selenium has been shown to be protective against inorganic mercury compounds as well (Paricek et al. 1966); in fact the presence of excess selenium in sea-derived sources of mercury may protect against mercury toxicity from this source (Taylor 1976, Krehl 1972).

It appeared that no attempt had been made to achieve the inhibition of methylmercury toxicity using measured quantities of a selenium source sufficient to provide molecular equivalents of selenium (Ganther for example only added tuna fish to a diet containing methylmercury chloride (MMC). Since the tuna was known to be rich in selenium he did not apparently consider the possibility that selenium was already bound to mercury and therefore incapable of further protective action. Yet some protective effect, albeit modest, was obtained indicating residual activity.

A further experiment was designed in which cats were fed a diet containing sodium selenite in quantities measured to balance MMC given by capsule. Control cats were given MMC alone or sodium selenite alone. The dosage of MMC was that worked out by Charbonneau et al. (1974) to cause

acute toxicity and death between 60 and 90 days. The control group on MMC all reached a terminal disease state in less than 75 days. Controls on sodium selenite alone showed no toxic signs whatever and no histological changes at autopsy. Of six cats given both MMC and sodium selenite, four were alive and physiologically normal after 120 days when the experiment was terminated. Histology revealed only minor lesions at the neuronal level which might have been attributable to MMC. Of the other two, one developed typical signs of toxicity at 60 days. It was then learned that this animal had been refusing the part of its diet (seal meat) that was used as a carrier for the sodium selenite, and that no attempt had been made to administer the selenite by another route.

The sixth cat was terminated after 119 days, at which time it was showing some signs of MMC toxicity, but had additionally just delivered three kittens.

This experiment provides considerable support for the views of Krehl (1972) and Taylor (1976) that toxicity should not be expected from mercury-containing marine fish since such fish invariably have a high content of selenium usually far in excess, on a molecular basis, of the mercury content. In this context it is rather ironic that early in the investigation of the Minamata episode, selenium was tentatively suggested as the toxicant responsible since selenium compounds were being released by industrial concerns into Minamata Bay.

Cadmium

Cadmium constitutes about 1.1 X 10^{-6}% of the earth's crust. As such it is about as common as silver (and five times as common as mercury). In nature it is found as an insoluble ore, mostly as a sulfide, in metalliferous ore bodies, especially in association with zinc and to a lesser extent with copper and lead.

In its natural state it appears to be non-toxic, but can be acutely toxic as a soluble salt or as an elemental vapour. Thus cadmium toxicity is most commonly seen in smelter workers where inhalation of cadmium fumes may give rise to an acute pneumonitis, or in individuals who have ingested solutions containing cadmium salts. The commonest source of ingested cadmium is vessels with a cadmium glaze from which the cadmium has been released by a low pH drink.

In the N.W.T. there are currently two industrial sources of cadmium — the lead/zinc mines at Strathcona Sound and at Pine Point. Since neither of these operations includes a smelter the probability of cadmium poisoning is very low.

However, during the development period of the Nanisivik Mine at Strathcona Sound the possiblity of cadmium toxicity was taken seriously, sparked particularly by the discovery of surprisingly high levels of cadmium in the tissues of marine mammals.

Hatfield and Williams (1976) quoted levels as high as 40 ppm in a seal liver and up to 118 ppm in narwhal liver, though they commented that both of these levels were exceptional. Assays done on seal livers obtained from the Holman Island area showed levels between 3 and 7 ppm. It is evident that seals do accumulate cadmium in the liver in the form of a metal-protein complex known as metallathionein. Thionein apparently acts as an inhibitor of toxicity (Cherian et al. 1976).

The discovery of high cadmium levels in marine mammals was a major argument against the proposal to dispose of mine tailings by direct gravity dumping into the deep waters of Strathcona Sound. An alternative land disposal system was developed that uses a small freshwater lake as a catchment basin for the settleable solids, which contain a proportion of cadmium ore. Subsequent analysis of the supernatant and run-off from this catchment area has shown cadmium levels at or below the level of detectability (S. Metikosh pers. comm.) which argues strongly against any hazard from the operation on this account. This assay finding is to be compared with a single reported level of 140 ppb in a small stream draining the ore body prior to commencement of mine operation (Hatfield & Williams 1976). This finding was unique, however, and samples from larger creeks and lakes showed cadmium levels ranging between 0.4 and 2.20 ppb which should be compared with recommended acceptable levels of 10 ppb for drinking water (Saskatchewan and Alberta water quality criteria).

One result of the revelations of the Hatfield & Williams report was a decision to perform baseline studies of blood cadmium levels in residents of Arctic Bay, the closest Inuit community to the Strathcona mine, which at the time (April, 1976), had still not gone into production. Though blood cadmium levels are not considered a particularly valuable measurement for clinical purposes, they are about the only tissue sample possible using acceptable, minimally invasive, procedures.

This was an ill-fated endeavour which it is perhaps instructive to document.

Two hundred blood samples were taken by Medical Services staff into lead-free glass vacutainers and submitted for assay of mercury, arsenic, lead and cadmium. Results were received promptly for the first three. Cadmium assay was deferred pending development of equipment and methodology, the specimens being held in storage meantime.

Approximately 6 months later the assays for cadmium were completed, yielding a range of results from low to extremely high. Because there is little experience with, or need for, cadmium assay of blood, normal levels are not well defined, but for the purpose of this exercise were taken to be below 2 μg/100 ml. Some readings were found up to 40 μg/100 ml.

When careful and thorough assessment failed to reveal any consistent pattern to these results, (which had been repeatedly checked by a variety of techniques) an investigation was launched into the collection methods to try to find an extraneous source for the cadmium. It was found that the rubber stoppers of the collecting tubes used contained 0.7% cadmium sulfide as a colourant. Cadmium had obviously leached from the stopper to the contained blood during storage.

Examinations of bloods collected subsequently and stored in plastic revealed universally normal levels.

This very brief statement of what was in fact a rather prolonged and extremely worrisome investigation serves to demonstrate the fallibility of all testing programmes, especially when they are in a new field. Though every attempt was made to reach a clear understanding as soon as possible some of the preliminary data did reach the news media and were there elaborated into a scare story which was at one time unsettling for the native people, and the cause of a time- and money-wasting medical examination of some perfectly normal young Inuit.

Given the low solubility of cadmium ore it is extremely unlikely that cadmium will prove a significant biological hazard in the N.W.T. unless a smelting industry is developed. The presence of large stores of fossil fuels in the North perhaps makes this not totally improbable though there are no known current moves in that direction.

Lead

Although lead is encountered in the industrial South in a great variety of circumstances and trades, in the North lead appears to be a potential problem only in the lead mines at Pine Point, Nanisivik, and Little Cornwallis Island. The classical source of occupational lead poisoning which results from burning off old lead-based paint is rendered unlikely by the dual consideration of the newness of the North, and the extreme danger of fire, which makes blow torch removal of old paint extremely improbable.

Leaded gasolines are a possible source of alkyl leads for addicted children, and possibly for amateur fixers of internal combustion engines who operate in ill-ventilated overheated rooms. Other possibilities for the acquisition of excessive lead burdens in the North include the pouring of shot for hand-loaded shells and the ingestion of home-distilled spirits from soldered distillation apparatus.

for much of the year may give opportunities for alkyl lead inhalation and particulate lead ingestion which would be much greater than would occur, for example, in automotive workshops in the south, where adequate ventilation and washing facilities are more readily available.

Clearly, among the Inuit of Arctic Bay in 1976, more males than females had elevated levels of blood lead (Table 7)[2]. That difference makes a dietary source improbable. However, the possibility of alternative sources that have not been identified cannot be discounted. In 1976 too, the mean blood lead levels among a subsample of males (Table 6) who were employed on construction and maintenance, and who commuted daily to the mine were lower than those of the male population in general (Table 7). Thus, at that time, neither the mine nor environmental sources in general were

TABLE 6

FREQUENCY OF OCCURRENCE OF LOW & HIGH BLOOD LEAD LEVELS AMONG MALE HEADS OF HOUSEHOLDS IN ARCTIC BAY IN 1976 AS A FUNCTION OF OCCUPATION.

Blood Lead	Place of Work or Occupation					
μg/100ml	Strathcona Mine	Panarctic Oil	Hunter	Retired Welfare	Municipal Employee	Total
25 or less	15	3	3	4	19	44
26 or more	1	1	4	1	4	11
Total	16	4	7	5	23	55

It perhaps seems strange to even consider lead as a possible toxin in the north but the fact is that mean lead levels taken on the general population of Arctic Bay prior to the operations of Nanisivik Mine were surprisingly high in males, quite as high as would be expected in residents of a busy southern city (Table 6).

Although sample sizes are small, there were significantly more hunters than mine-workers with blood lead greater than 25 μg/100 ml[1]. A second occupational group that appears to be exposed to some hazard from lead is municipal employees, whose duties include operation of water and garbage trucks.

The only obvious factor common to these two groups is prolonged association with petrol-driven internal combustion engines. Though this may not be thought to be a usual source of lead accumulation, the fact that much work is done on these engines in ill-ventilated and cramped quarters

thought to be a significant factor in explaining the observed elevated blood lead levels.

Since the opening of the mine regular surveillance of blood lead and urinary lead levels has been maintained, especially of the permanent Inuit employees. A modest increase in lead levels has been noted and the mine authorities have been urged to promote cleanliness in the work environment and especially amongst workers prior to ingestion of food. Up till the present no individual has been found whose blood lead has risen to a level considered hazardous.

Regular air sampling surveys have been carried out by the Occupational Health Unit of Medical Services Branch. Because the mine is operated in a heavy permafrost area the rock drilling for explosives placement is done by a dry process in which the resultant dust is collected by a vacuum extractor.

In general this has resulted in acceptable levels for respirable dust and silica content though lead levels have on occasion been found to exceed threshold limit values (T.L.V.). However, such T.L.V.s are specified for continuous exposure, 8 hours per day, 5 days per week, 50 weeks per

1 P = 0.035, Fisher's Exact Test (Ed).

2 For a more detailed analysis, see the statistical appendix.

TABLE 7

FREQUENCY OF OCCURRENCE OF VARIOUS BLOOD LEAD LEVELS (μg/100 ml) AT ARCTIC BAY AND NANISIVIK

	< 25		25-40		> 40		N
	no.	%	no.	%	no.	%	
Arctic Bay							
Inuit Females 1976	97	97	3	3	-	-	100
Inuit Males 1976	76	76	23	23	1	1	100
Inuit Males 15-65 1976	56	71	22	28	1	1	79
Nanisivik							
Male Employees 1976	48	51	40	43	6	6	94
Inuit Male Employees 1980	14	54	7	28	5	10	26
White Male Employees 1980	40	83	4	8	4	8	48
All Male Employees 1980	54	73	11	15	9	12	74

year. Such continuous exposure does not take place in the Nanisivik Mine and the intensity of intermittent exposure, even though a level of 10 times T.L.V. has been shown at one sampling site on one occasion, is not considered as excessive.

The increased blood lead levels observed are thought to be the result of ingestion of particulate lead, caused by less than adequate hand-washing, rather than a result of inhalation, but despite this belief every effort should be made to reduce dust exposure to the minimum.

At the Pine Point Mine no comparable dust hazard at the drilling face exists because wet drilling is used. Neither biological monitoring nor ambient air monitoring has been carried out because of lack of indication.

It is possible that at the future Arvik Mine, with its much richer ore, even more rigorous standards will need to be maintained, but at the moment this is only a matter of conjecture. Surveillance on a regular basis will be necessary.

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Statistical Appendix

Dr. Eaton's report deals with samples drawn from various populations (males and females from Yellowknife, hunters and miners from Nanisivik/Arctic Bay, etc.). No two samples drawn from the same population are likely to give identical results. But, if all subunits of a population contain the same proportion of individuals with elevated arsenic levels, for example, then we would expect only slight differences in the observed proportions between our samples. Those differences we would ascribe to chance. If we drew a handful of beans from a jar that contained 80% white beans and 20% red beans, and found that our sample handful contained 18% or 22% red beans we would not be too surprised. But if we got 40% red, we would be surprised and we would suspect that someone had switched jars on us or added more red beans — in other words, that we were not sampling the same population.

Statistical tests are quantitative devices for telling us how likely it is that the differences we observe between samples are simply due to chance. If the probability of obtaining the observed difference is small, we suspect that our samples did not come from a homogeneous population characterized by a fixed proportion of the feature we are studying (red beans, high arsenic, etc.). By convention our suspicions are aroused if the probability is less than one chance in twenty, which is less than 5% probability or, in mathematical shorthand, $P < 0.05$. We are pretty sure to be dealing with separate populations if the probability is less than 1% ($P < 0.01$), and very sure if the odds are less than one chance in a thousand ($P < 0.001$).

The tests used in this Appendix are fully described in Sokal and Rohlf, 1969. Biometry. W.H. Freeman Co., San Fransisco.

To check for differences in the proportion of elevated hair arsenic levels in Yellowknife (Tables 1 to 4) it is necessary to forego some detail and consolidate and combine some of the data. In Appendix Table 1, the number of age-groups has been reduced to three: 0-19 (pre-workforce), 20-49 (peak working years), 50-69 (late working years). Those over 70 and of unknown age have been omitted. Also, instead of four levels of hair arsenic, only two are used - less than 5 parts per million (< 5ppm), which is equivalent to the white beans in the jar; and more than 5 parts per million (> 5ppm), which is equivalent to the red beans.

The proportions of individuals having high and low hair arsenic levels in the samples were compared by means of Fisher's Exact Test. The results of the comparisons for different subgroups of the Yellowknife population for each of three age groups are shown in Appendix Table 2.

Interpretation of the information in Appendix Table 2 is as follows:

1) For all age groups, there were unequivocally more males than females with high hair arsenics among the <u>non-native</u> component.

APPENDIX TABLE 1

CONSOLIDATION AND SIMPLIFICATION OF TEXT TABLES 1 TO 4 — HAIR ARSENIC LEVELS IN YELLOWKNIFE.

Sample	Arsenic ppm	Age group 0-19	20-49	50-69	Total
Non-native males	<5	74	123	35	232
	>5	13	71	17	101
Non-native females	<5	77	152	45	274
	>5	2	3	0	5
Native males	<5	2	8	3	13
	>5	4	3	0	7
Native females	<5	8	18	4	30
	>5	5	1	1	7
All non-natives	<5	151	275	80	506
	>5	15	74	17	106
All natives	<5	10	26	7	43
	>5	9	4	1	14

2) In the age-group 0-19, more native males and females had high hair arsenic than did non-native males and females. For males and females combined there are only 2 chances in 10,000 that the observed difference is due to sampling error.

3) For all age-groups combined, there were more native females with high hair arsenics than non-native females.

The interpretation of these results is as follows:

1) The proportion of individuals with low (< 25) intermediate (26-40) and high (> 40) blood levels among Inuit males was not the same as that among Inuit females at Arctic Bay.

2) The proportion of individuals with low, intermediate and high blood levels among Inuit male employees in 1980 was not the same as that among Inuit males aged 15-65 in 1978.

3) The proportion of individuals with low, intermediate and high blood levels among male employees in 1980 was not the same as that among male employees in 1978.

4) The proportion of individuals with low, intermediate and high blood levels among Inuit male employees was not the same as that among white male employees in 1980. This is the least certain of the four conclusions (odds about 40 to 1).

APPENDIX TABLE 2

RESULTS OF COMPARISONS BETWEEN VARIOUS POPULATION SUBGROUPS AND AGE GROUPS, OF THE DATA IN APPENDIX TABLE 1 USING FISHER'S EXACT TEST.

	Age Group			
Comparison	0-19	20-49	50-69	Total
Non-native males Non-native females	P = 0.009	P << 0.001*	P << 0.001	P << 0.001
Non-native males Native males	P = 0.019	ns +	ns	ns
Native males Native females	ns	ns	ns	ns
Non-native females Native females	P = 0.001	ns	ns	P < 0.001
All non-natives All natives	P < 0.001	ns	ns	ns

* Read as "probability that there is no difference between the observed proportions is much less than 1 chance in 1000". We therefore conclude that there is a real difference.

\+ ns means "not significant". By convention, ns means that the odds are greater than 1 in 20.

The causes for most of these differences are not obviously related to employment in the mines. Presumably, not many of the under 19 group and not many females, are employed in the mines. For males aged 20-49 (and perhaps for older males as well) the difference between non-native and native is likely to be related to employment. For the others we must look to an environmental cause.

With respect to text Table 7, I first compared various rows with other rows by means of a G-test for independence. This test enables one to compare two or more subgroups for more than two levels of lead. The results for 2 row x 3 column tests are given in Appendix Table 3.

The foregoing analysis does not enable us to say whether the difference is primarily due to the number of individuals with intermediate levels or high levels. To help sort this out, data in text Table 6 can be reorganized, first by combining columns 2 and 3 (Appendix Table 4), and second, by combining colums 1 and 2 (Appendix Table 5). We now have only two columns, and can look for significant differences in the proportions of individuals having any elevation of blood level above 25 μg/100 ml. Fisher's Exact Test was once again used for the analysis.

Three of the four differences revealed by the G-test are again significant. More Inuit males than Inuit females had elevated blood levels; more male employees had elevated blood levels in 1978 than in 1980; and more Inuit than white employees had elevated blood levels in 1980. The difference between Inuit males 15-65 in 1976 and Inuit employees in 1980 was not significant by Fisher's test.

APPENDIX TABLE 3

RESULTS OF 2 × 3 G-TESTS FOR INDEPENDENCE OF DATA IN TEXT TABLE 6.

Comparison	G	P
Inuit females vs Inuit males	20.857	<0.001
Inuit males (15-65) vs Inuit male employees 1980	10.02	≈ 0.005
Male employees 1978 vs male employees 1980	16.089	<0.001
Inuit employees vs white employees 1980	7.353	≈ 0.025

APPENDIX TABLE 4

CONSOLIDATION OF TEXT TABLE 6 TO COMBINE FREQUENCIES OF INTERMEDIATE AND HIGH BLOOD LEAD LEVELS, AND SIGNIFICANT DIFFERENCES REVEALED BY FISHER'S EXACT TEST.

Sample	Blood level (μg/100 ml) <25	>25	Total	%>25
Arctic Bay 1976				
Inuit Females	97	3	100	3
All Inuit males	76	24	100	24
Inuit males 15-65	56	23	79	29
Nanisivik				
Male employees 1978	48	46	94	49
Inuit male employees 1980	14	12	26	46
White male employees 1980	40	8	48	17
All male employees 1980	54	20	74	27

Significant differences	
Comparison	**P**
Inuit males vs Inuit females	$\ll 0.001$
Male employees 1978 vs 1980	0.002
Inuit vs white male employees 1980	0.015

APPENDIX TABLE 5

CONSOLIDATION OF TEXT TABLE 6 TO COMBINE FREQUENCIES OF LOW AND INTERMEDIATE BLOOD LEAD LEVELS

Sample	Blood level (μg/100 ml) <40	>40	Total	% > 40
Arctic Bay 1976				
Inuit Females	100	0	100	0
All Inuit males	99	1	100	1
Inuit males 15-65	78	1	79	1
Nanisivik				
Male employees 1978	88	6	94	6
Inuit male employees 1980	21	5	26	19
White male employees 1980	44	4	48	8
All male employees 1980	65	9	74	12
Significant differences				
Comparison		P		
Inuit males 15-65 vs Inuit employees 1980		0.007		

Appendix Table 5 separates out only those having high blood levels (> 40 μg/100 ml). Only one comparison yielded a significant result, namely, that between Inuit males 15-65 in 1976 and Inuit male employees in 1980. Since this comparison for the data in Appendix Table 4 did not indicate a difference in the proportion of individuals with at least some elevation of blood level, the present finding is to some extent surprising, though not contradictory. It may have significant health implications, and therefore, should not be ignored.

One other comment about Appendix Table 5 is worthy of note. The raw data show an increased proportion of all male employees with high blood levels between 1978 and 1980 (12% vs 6%) even though the proportion with some elevation declined from 49% to 27% (Appendix Table 4). Although the increase from 6% to 12% is not statistically significant it suggests that a small number of employees may be exposed to high risk.

Conclusion

All that this kind of statistical analysis can do is estimate the odds against obtaining the observed differences by chance if the samples were drawn from two populations with equal incidences of elevated levels of arsenic or lead. If chance is virtually eliminated (e.g. odds of 1 in 1000), then we should look for a real cause.

It should also be noted that an association between mine employment and elevated levels of a heavy metal does not prove cause and effect. Many other factors are uncontrolled, such as, exposure to leaded fuels in poorly ventilated buildings; place of living with respect to location of stack and direction of prevailing wind; diet; length of period of continuous employment; nature of the employment, and so forth. However, the association does raise legitimate suspicion and reinforces Dr. Eaton's suggestion that additional surveillance and continuous effort to reduce exposure is required.

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