

Assessment of the Potential for Health Problems Associated with the Export of Sulfidic Nickel Concentrate Through the Port of Esperance

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1. Definitions of Abbreviations ands Key Terms Used in This Review

There are some international organizations which enjoy unique authority in relation to chemistry and human health and which will be referred to in this review. Normally they are referred to by the abbreviations given below:

ACGIH - American Conference of Government Industrial Hygienists

ATSDR - Agency for Toxic Substances and Disease Registry

IARC - International Agency for Research on Cancer

IUPAC - International Union of Pure and Applied Chemistry

NAS - National Academy of Sciences (U.S.)

USEPA - United States Environmental Protection Agency

WHO - World Health Organization

In assessing a situation that involves exposure of a population to a potentially toxic substance, it is important to understand some key terms that describe the situation and the general approach to assessment of exposure and the route from exposure to effect. The definitions below are taken from the IUPAC "Glossary of Terms Used in Toxicology, 2nd

Edition” (Duffus, Nordberg, and Templeton, 2007) which is widely used as the most authoritative glossary of toxicology terms available. It should also be noted that the IUPAC preferred spelling, “sulfide”, will be used in this document except where quoting verbatim from another document where the older usage, “sulphide”, prevailed.

bioaccessibility - Potential for a substance to come in contact with a living organism and then interact with it. This may lead to absorption.

Note: A substance trapped inside an insoluble particle is not bio-accessible although substances on the surface of the same particle are accessible and may also be bio-available. Bio-accessibility, like bio-availability, is a function of both chemical speciation and biological properties. Even surface-bound substances may not be accessible to organisms which require the substances to be in solution.

bioavailability - Extent of absorption of a substance by a living organism compared to a standard system.

biomonitoring - Continuous or repeated measurement of any naturally occurring or synthetic chemical, including potentially toxic substances or their metabolites or biochemical effects in tissues, secreta, excreta, expired air or any combination of these in order to evaluate occupational or environmental exposure and health risk by comparison with appropriate reference values based on knowledge of the probable relationship between ambient exposure and resultant adverse health effects.

epidemiology - study of the distribution and determinants of health-related states or events in specified populations and the application of this study to control of health problems.

Note: Epidemiology uses statistical analysis to establish significant association between disease and hypothetical causes of disease. This, in itself, does not prove cause and effect. Association does not prove causation (other evidence must be considered, especially the existence of a plausible mechanism relating the proposed causal agent to the effect). Further, high precision of the data should not be mistaken for validity (nonrandom errors can occur).

exposure -

1. Concentration, amount or intensity of a particular physical or chemical agent or environmental

agent that reaches the target population, organism, organ, tissue or cell, usually expressed in numerical terms of concentration, duration, and frequency (for chemical agents and micro-organisms) or intensity (for physical agents).

2. Process by which a substance becomes available for absorption by the target population, organism, organ, tissue or cell, by any route.

sorption - Noncommittal term used instead of adsorption or absorption when it is difficult to discriminate experimentally between these two processes.

speciation (in chemistry) - Distribution of an element amongst defined chemical species in a system.

species (in chemistry) - Specific form of an element defined as to isotopic composition, electronic or oxidation state, and (or) complex or molecular structure.

2. General Introduction

Recently, various regulatory authorities have been re-examining the carcinogenic hazards and risks associated with occupational exposures to nickel and certain nickel compounds (Cal OSHA, 2005; EURA, 2005a, b). IARC, which is an international quasi-regulatory body which provides influential classifications of carcinogenicity, will be reexamining the carcinogenic hazards of metals and other substances (including nickel compounds) in the near future (IARC, 2008). Studies of primary nickel workers involved in the processing of nickel from sulfidic ores (the so-called “high risk” cohorts) will probably be the focus of the deliberations of these bodies, as these are the studies where excess respiratory cancer risks have been observed (Roberts *et al.*, 1989; ICNCM, 1990; Easton *et al.*, 1992; Andersen *et al.*, 1996; Grimsrud *et al.*, 2002; 2003). Outwith the nickel refineries, there is no epidemiological evidence to associate exposure to any nickel compound, and certainly not nickel metal or alloys, to any increased incidence of cancer. This suggests that the excess rates of respiratory cancer in refineries may be a product of a mixed exposure that occurs only in the context of nickel refining.

Even in the context of other industrial activities in which exposure to nickel occurs, there is no report of any excess in respiratory carcinogenic risks associated with inhalation exposures to

nickel, although these activities employ the vast majority of nickel industry workers. Many of these industries are pursued in countries with good cancer registries and so any excess should have been detected. Studies of workers involved in the smelting and refining of lateritic ores (which do not contain nickel sulfide minerals) and studies of workers in nickel-using industries, including plating, nickel alloy and stainless steel production, stainless steel fabrication, certain welding operations, and barrier manufacturing in the enrichment of uranium, have consistently shown no occupationally related respiratory cancer risks (Sivulka *et al.* 2007). Thus, the human evidence for nickel-related carcinogenicity is confined to those industries involved in the refining of nickel from sulfide-containing ores. That being the case, we should not extrapolate risks associated with refinery exposure to a mixture of nickel compounds produced by the refining process to assessment of environmental health risks associated with exposure to particulates from nickel concentrate during its transport from the originating mine to a harbour and subsequent loading onto vessels for export.

3. General Toxicological Considerations

3.1 Route and Source of Exposure

A substance may be toxic by one route of exposure, and relatively harmless by another. Nickel is a good example. Inhalation of aerosols containing soluble nickel salts has been associated with respiratory cancers, while ingestion of nickel compounds, in any form, is not considered to carry any carcinogenic risk (IARC, 1990). Thus, it is important to clarify the source of the nickel exposure, the exact form (its chemical speciation) in which the nickel occurs, and the consequent route of human intake (inhalation, ingestion, dermal absorption). In the Esperance environment, direct exposure is probably restricted to inhalation or ingestion of particles.

Exposure is a critical concept in toxicology, and not as straightforward as it seems at first. It can be defined as the process by which a substance becomes available for absorption by the target population or organism. Before we can consider exposure, we must the nature of the environmental source and the precise mechanism by which exposure occurs.

3.2 Bioavailability

Most elements are absorbed by living organisms from aqueous solution, whether it is from fresh water, soil water, aerosols, or dietary intake to the gut (Apostoli *et al.*, 2006). A substance is bio-accessible if it is possible for it to come into contact with a living organism, which may then absorb it. For example, any substance trapped inside an insoluble particle will not be bio-accessible, although substances on the surface of the same particle will be accessible and may also be bioavailable. However, even surface-bound substances may not be accessible to organisms that can only incorporate the substance if it is in solution. Thus, bioaccessibility is a function of both chemical speciation and biological properties. In some cases, bioaccessibility will be the limiting factor determining uptake, and this is particularly true of metallic elements in soils, sediments, and other particulate matter to which humans may be exposed.

Substances are biologically available if they can be taken up by living cells and organisms and can interact with target molecules. Substances that are not bioavailable may still cause physical damage or may alter the availability of other substances. The bioavailability of an element depends upon its chemical speciation. Elements occur in the soil, for instance, in either the solid phase or in an aqueous soil solution. In the solid phase, ions can be bound to organic and inorganic soil components in various ways, including ion exchange and surface complexation, or they can exist in minerals or be co-precipitated with other minerals in the soil. In the soil solution, the elements can exist either as free ions or as complexes with organic groups, such as amino, carboxyl, and phenolic groups, or inorganic groups, such as carbonate, chloride, hydroxide, nitrate, and sulfate. Ions in solution are generally bioaccessible, and ions in the solid phase of the soil may become accessible if environmental conditions change. A number of factors determine bioavailability, e.g.,

- a) Elemental species are often transported into cells by specialized protein carriers (Simkiss and Taylor, 1995) or by organic ligands, such as chelating agents or ionophores. Some may diffuse passively through the phospholipid bilayer of the cell membrane (Gutknecht, 1981).
- b) The physical form is important in addition to chemical speciation. For example, inhalation of an insoluble species as a dust or aerosol will localize the effect to the lung.

c) The inorganic complexation of many ionic species in water, including nickel(II), is often negligible, and thus the ions are mainly in the form of uncomplexed hydrated ions. However, hydrated ions may still be of low bio-availability because of their size. Some ions react with the water molecules in a process called hydrolysis that may leave the ion attached to hydroxide and with a different net charge from the free ion.

3.3 Speciation

The terms “species” and “speciation” are now well established and it is recognized that identifying the occurrence of an element in different forms is essential to our understanding the environmental and occupational toxicity of that element. A number of definitions of chemical speciation can be found in the literature. In the past, the term “speciation” has been used to refer to “reaction specificity” (rarely); in geochemistry and environmental chemistry, to changes taking place during natural cycles of an element (species transformation); to the analytical activity of measuring the distribution of an element among species in a sample (speciation analysis); and to the distribution itself of an element among different species in a sample (species distribution).

Several recent discussions of speciation outline its importance in understanding toxicity (Apostoli *et al.*, 2006; Templeton *et al.*, 2000; Templeton, 2003; Yokel, Lasley and Dorman, 2006; Cornelis *et al.*, 2005; Imran and Aboul-Enein, 2006). In addition, the European Virtual Institute for Speciation Analysis (EVISA) maintains a website at [HYPERLINK "http://www.speciation.net/index.html" http://www.speciation.net/index.html](http://www.speciation.net/index.html) that includes data bases of speciation analyses and toxicity studies for individual metals as well as ongoing dialogue within the speciation community and occurrences of the incorporation of speciation data in legislation on a continent-by-continent basis.

3.4 Acute and chronic toxicity

Acute and chronic exposures to harmful substances typically have different patterns of effects. In toxicology acute studies generally refer to single exposure and a study duration of less than two weeks, and acute effects usually occur within the first 24 hours, or up to two weeks

following a single exposure. Chronic refers to mammalian studies lasting considerably more than 90 days or to studies occupying a large part of the lifetime of an organism. A chronic effect is one that develops slowly and (or) has a long lasting course, or the term may be applied to an effect that develops rapidly but is long lasting. In the context of environmental exposures in Esperance, we are concerned about chronic effects on humans. Thus, many of the toxicological studies that have been carried out with animals exposed for a short time to high exposure levels, often well above anything people will experience, are of little relevance.

Chronic toxicity testing is expensive and labour-intensive, and a particular problem arises when the exposure is low or the effect develops a very long time after exposure. It becomes difficult to attribute a cause to the delayed effect, and to test substances for such effects. Cancer is an example. In humans, cancer may take up to 40 years to develop after exposure to a carcinogen. Our normal test rodents have life spans of about 2 years or less. To cause malignant tumours within such a short time, very large doses of test compounds must be applied, and these may overwhelm defence mechanisms that work well within the normal human exposure ranges.

3.5 Environmental Exposure and its Relationship to Occupational Exposure

It should be clear from the above that risks from exposures to inorganic elements such as nickel are highly dependent on the chemical species, route, and duration of exposure. Thus, we need to consider these factors carefully when assessing a possible environmental risk for a human population.

Guidelines are set differently for occupational and general environmental exposure according to relevant conditions for workers and the general public. An exposure that increased an annual risk by 1 in a million for a specialized occupation that employed 100 people would not be expected to produce an adverse effect in our lifetime. The same exposure applied to millions of Australians might still be of concern. Much of the toxicology that we base opinions on comes from occupational studies. Data on environmentally acceptable exposures are hard to find, and while there is a large body of literature on occupational exposures to nickel species, the focus here is on general environmental health on which there is much less information.

4. Nickel Toxicology

4.1 Environmental Toxicology of Nickel

We are exposed to nickel through air, water, and diet. Dietary intake of nickel is usually about 100 µg/day (Grandjean, Nielsen, Andersen, 1989). Nickel is not generally believed to be essential for higher organisms, but it is a cofactor in several enzymes of plants and microorganisms, and vegetarian diets may be higher in nickel content; diets rich in nuts and soy products may lead to intakes of nearly 1 mg/day (Sunderman, 1986). Urban atmospheres in the U.S. have been reported to contain nickel at about 25 ng/m³ and urban dwellers inhale 0.2 - 1.0 µg nickel/day (Templeton, 1996; Sunderman, 1988). Values of about 150 ng/m³ have been recorded in polluted areas, particularly where fossil fuels are burned. Nickel in cigarette smoke increases intake by as much as 4 µg/pack in smokers. Other non-occupational exposures arise from handling metal objects such as jewelry and coins, and from implantation of medical prostheses made from nickel-containing alloys. These exposures are primarily of interest for immunosensitization (see below). It is noteworthy that nickel alloy implants have never been associated with any specific toxic effects.

Nickel inhaled from fumes and dusts, including that from occupational exposure to welding fumes, is mainly deposited as particulates in the nasal sinuses, upper airways, and lungs, depending on the aerodynamic size distribution of the particles. Aerosolized nickel compounds encountered in some occupations are absorbed and result in immediate increases in blood nickel levels (Templeton, 1996). Transdermal absorption of nickel is low, but binding of nickel as a hapten to ligands in the skin can result in antigen presentation and contact dermatitis (Templeton, 2004; Grandjean, Nielsen, Andersen, 1989). Oral absorption of nickel with food is low, but increases to about 30% when soluble nickel salts are given in water to fasting persons (Sunderman *et al.*, 1989; Templeton, Xu, Stuhne-Sekalec, 1994).

The acute toxicity of ingested soluble nickel is low (Sunderman *et al.*, 1988; Webster *et al.*, 1980); accidental ingestion of gram quantities has been tolerated with minimal side effects. The main concern is for the carcinogenic potential of chronic exposure. Certain nickel compounds are potent carcinogens in animals and there is now sufficient evidence from epidemiological studies

for carcinogenicity in humans. The Doll Committee (Doll, 1990) concluded that increased risks of respiratory cancers are primarily related to exposures to less soluble (essentially insoluble in water in the absence of acid) nickel oxides and sulfides at concentrations more than 10 mg/ m³ and to soluble nickel compounds at concentrations above 1 mg/m³. However, it is important to note that the epidemiological studies relate to exposures that were poorly chemically characterised at the time they occurred. In particular, the main carcinogenic components of the exposure may have been nickel subsulfide, nickel suboxide and (or) nickel arsenide generated as a result of the refining process.

IARC reviewed the Doll and other data (IARC, 1990) and concluded that nickel compounds are carcinogenic to humans (Group 1), whereas metallic nickel is possibly carcinogenic to humans (Group 2B). There is no significant evidence linking purely environmental nickel exposure, in any form, to cancer. The major occupational studies (summarized from IARC) were conducted at INCO Ontario, MONDO/INCO South Wales, and Falconbridge Norway. Among the Ontario workers no nasal cancer was observed among sintering workers with more than five years of exposure to sulfidic nickel, but the standard mortality ratio (SMR) for lung cancer was 492. In another group of sinter workers with additional exposure to nickel oxides and soluble nickel, this value rose to 789 and nasal cancers were also observed (SMR ~ 13,000). Comparable results were observed in leaching and calcining workers with similar exposures, whereas among electrolysis workers with lower exposures to all classes of nickel compounds no excess cancers at any site were found.

In Wales, exposures of five or more years duration to nickel arsenides, oxides, sulfides, salts, or metal in a variety of occupations associated with nickel production were associated with SMRs of 300-1200 for lung cancer and 1,000-78,000 for nasal cancer. In the Norwegian study, electrolysis workers with low exposure to nickel salts, sulfidic nickel (and probably arsenical derivatives) had an increased SMR for lung cancer compared to workers in other regions of the plant who had not been significantly exposed to nickel salts (SMR = 476 vs. 254).

It is important to stress that these positive cancer statistics are based on inhalation only, in occupational settings where exposures were far above those encountered in the general atmosphere, whatever the level of local soil contamination, and involved mixed exposures that

could not be fully characterised at the time. It should also be emphasised that the proven associations with human lung and nasal cancers are for mixed exposures involving nickel sulfates, nickel arsenides, nickel oxides and sulfides met with in the nickel refining industry, along with exposure to other undefined substances as well. Production of human tumours by other nickel species or at other sites than the lung and nose has never been confirmed.

4.2 Speciation and toxicokinetics (physiological handling of different nickel species)

Soluble nickel salts dissociate and release Ni^{2+} ions in aqueous media. Oral nickel absorption is greater with more soluble compounds, for example, 34% for nickel(II) nitrate (NiNO_3), 11% for NiSO_4 , 10% for NiCl_2 , 0.5% for nickel subsulfide (Ni_3S_2), 0.09% for metallic nickel, 0.04% for black nickel oxide, and 0.01% for green nickel oxide. Absorption is reduced by binding to amino acids such as cysteine and histidine. Oral absorption of nickel sulfate given in drinking water to fasting human subjects was $(27 \pm 17) \%$ (mean \pm s.d.) but was only $0.7 \pm 0.4\%$ when the nickel sulfate was given in food (Sunderman *et al.*, 1989). The water solubility of nickel compounds correlates well with the rate of absorption of Ni^{2+} into the blood from nickel-containing particles that reach the alveolar level in the lung. When soluble nickel chloride was deposited in the lungs of rats by intratracheal instillation, 75% per cent was absorbed within 3-4 days, whereas 80% of deposited nickel oxide (NiO) aerosol still remained in hamster lungs after 10 days. The half-lives of nickel in rat lungs following inhalation of nickel(II) sulfate (NiSO_4), nickel(II) subsulfide (Ni_3S_2), and nickel(II) oxide (NiO) were about 30 h, 4–6 days, and 120 days, respectively. The depth of penetration into the skin of nickel from nickel(II) salts is a function of the counterion (acetate > nitrate > sulfate > chloride). Using excised human skin under occlusion, penetration of Ni^{2+} from nickel chloride through the skin was about 0.23% of the applied dose after 6 days and 40-50 times quicker than from nickel sulfate. Without occlusion, the permeation of nickel chloride was reduced by more than 90%, and no absorption was detectable using nickel sulfate.

4.3 Immunosenitization

Occupational exposures to nickel are widespread and occur in mining and refining of

nickel ores, and production of alloys. Electroplating with nickel, stainless steel welding, and use of nickel alloys in the electronics industry are also important sources of exposure. Further exposures occur in nickel-cadmium battery manufacture, the chemical, pigment, and ceramics industries, and manufacture of nickel-based catalysts for industrial hydrogenation reactions. Some nickel platers, refiners, and welders have developed occupational asthma, chronic bronchitis, and pneumoconiosis. While nickel may be a causative agent in these cases, IARC (1990) did not consider that the evidence was adequate to reach such a conclusion.

Non-occupational exposures arise from handling metallic objects such as jewelry and coins. A high rate of nickel allergy is associated with ear piercing and subsequent wearing of nickel alloy jewelry (Schubert et al., 1987). Interestingly, comparing the lower incidence of nickel allergy in those who wore nickel-releasing dental braces before ear piercing, with the higher incidence in those who wore them only after or not at all, provides evidence for the development of tolerance to nickel (van Hoogstraaten, 1991; Kerosuo, 1996). Dietary intake of nickel is at least 100 µg/day (Grandjean, Nielsen, Andersen, 1989), and diets naturally high in nickel can exacerbate dermatitis in nickel-sensitized individuals (Nielsen et al., 1990). Dermal exposure to nickel is primarily related to contact dermatitis that begins as an erythematous lesion, usually on the hands and forearms, eventually becoming eczematous (Sunderman, 1992). Asthma, conjunctivitis, and local inflammation associated with prosthetic devices have also been reported (Sunderman, 1992). Sensitivity can also be manifest as asthma, and rare instances of anaphylaxis have been reported following parenteral injections of nickel-contaminated medications (Sunderman, 1992). Much is known about the mechanisms of nickel sensitization, including specific metal-protein interactions, activation of subsets of T cells, and activation of various cytokines (Templeton, 2004; Klein, Schwenk, Templeton, 2006). Cross-reactivity is an important issue in nickel allergy, as nickel exposure often occurs in the context of multiple metal exposures, and positive patch tests to other metals (e.g., cobalt and chromium) are common.

4.4 Biomonitoring

Absorbed nickel is cleared rapidly in the urine. Thus, serum and urine nickel concentrations are correlated after exposure to soluble nickel compounds and both are indicative

of recent exposure (Templeton, 1996). Nickel does not accumulate in the body with common levels of non-occupational exposure. However, insoluble and particulate forms can accumulate in the upper airways and the lungs, and can serve as an internal source of exposure at a much later time. Therefore, whereas changes in nickel concentration in serum and urine are good indicators of exposure to bio-available nickel over the preceding 1-2 days, increases in the nickel content of these fluids will also reflect long-term exposure from these insoluble sources, and ongoing, long-term monitoring is implemented in occupational health.

The nickel concentration in urine is a good indicator of current exposure to bioavailable nickel, and is useful for end-of-shift monitoring in the occupational setting (Sunderman et al., 1988; Angerer, Heinrich-Ramm, Lehnert, 1989). Spot urines are collected at the beginning and end of the workday. Nickel in serum or plasma correlates with urinary levels in acute absorption, and is more reliable for long-term monitoring of insoluble nickel compounds (Sunderman et al., 1988; Angerer, Heinrich-Ramm, Lehnert, 1989; Angerer, Lehnert, 1990; Lauwerys, Hoet, 1993). Biopsies of the nasal mucosa yield information on the long-term inhalation of insoluble nickel compounds (Torjussen, Andersen, 1979). This invasive procedure is not feasible for screening the general population.

5. Assessment of the Current Situation at Esperance, Western Australia (WA)

The following assessment is based on the information I have received from the Department of Health, Environmental Health Directorate, Government of Western Australia.

On 30 September 2008 the Chief Executive Officer (CEO) of the Esperance Port Authority (EsPA) advised nickel exporters of the following:

“The Board RESOLVES that, in order to comply with its statutory obligations, and to allow three months grace to facilitate the modal shift from solid bulk to bags and (or) containers the Esperance Port Authority will not be in a position to and will no longer accept bulk nickel concentrate from 1st January 2009.”

This ban on the export of bulk sulfidic nickel concentrate by the EsPA has created problems for the sustainability of WA's nickel industry. In the documents I have received, the

concentrate is often referred to as nickel sulfide concentrate. I think that this may give the impression that it is largely nickel sulfide (NiS). This cannot be true and the precise chemistry of the concentrate is probably complex. Thus, to avoid confusion, I prefer to use the term “sulfidic nickel concentrate”. In particular, I wish to avoid confusion between nickel sulfide, which has not been identified as a carcinogen, and nickel subsulfide, which has. I doubt if much, or any, nickel subsulfide is present in the concentrate since nickel subsulfide appears to be a product of the refining process.

The decision given above followed from the discovery that the infrastructure used for bulk sulfidic nickel concentrate was also used to load Magellan Metals lead carbonate during the period April 2005 to January 2007 and that this procedure had resulted in significant lead contamination of the town, many bird deaths, and elevated blood lead levels in the community and particularly in children. It was decided that the existing infrastructure is inadequate, and unsuitable for the export of bulk commodities while, at the same time, meeting reasonable environmental and health guidelines. However, sulfidic nickel concentrate is very different in physicochemical and toxicological properties from lead carbonate and therefore in its potential to harm the local environment and the human population. For example, nickel ions in water are much less toxic than lead ions (see above).

According to the information available from the nickel producers, sulfidic nickel concentrate is produced at numerous sites in WA. It contains about 12% nickel and is produced by a flotation process using several chemical forms of xanthate to separate the nickel-rich material from the crushed rock bearing ore. The concentrate is in the form of a grey black powder. This concentrate is exported to smelters in several countries including China, Canada and Finland. It is logistically not possible to use purpose built containers for the transport of sulfidic nickel concentrates and then to return these containers to WA. Thus, containerisation is not a practical option.

Clearly, the lead carbonate contamination of the surrounding environment shows that there are serious deficiencies in the existing infrastructure at the Esperance Port. Since the discovery of the lead pollution, improvements have been made in the infrastructure, including installation of a new cleaning device on part of the conveyor system, a shroud between the wharf and the

ship to minimise spillage into the harbour, improvements in the rail transport of nickel concentrates, and a truck wash system. It is accepted that all components of the existing infrastructure are outdated, inadequate and need to be replaced. Air quality monitoring and visual observations have shown that particulates from nickel concentrates continue to be emitted from the facilities during loading operations.

Following an Environmental Protection Notice issued by the DEC in October 2007, the EsPA has upgraded its environmental emission monitoring program in and around the Esperance port. The monitoring program was originally undertaken on a three monthly basis but is now required on a monthly basis. Monitoring is carried out by Sinclair Knight Mertz consulting. Following the recommendations of the Parliamentary Inquiry Report, environmental monitoring reports of each ship loading operation are posted on the EsPA website.

The Department of Health (DOH) has recently advised the DEC of the following requirements for nickel sulfide emissions in Esperance:

- A target of $0.14 \mu\text{g}/\text{m}^3$ to be determined on a 24 hour basis.
- An annual target for the nickel concentration in the air, when averaged over 365 days is not to exceed $0.003 \mu\text{g}/\text{m}^3$ ($3 \text{ ng}/\text{m}^3$).

Comment on the above requirements - JHD

The chosen target level would undoubtedly protect against potential carcinogenic long term exposures to nickel subsulfide in air but is irrelevant since it is highly unlikely that there is any nickel subsulfide in the particulates released from the nickel concentrate. Even if there were, measurement of total nickel would still be irrelevant since it gives no clue as to the nickel speciation in the particulates that might be inhaled. Strictly, only nickel subsulfide should be monitored but this would probably be a waste of resources since it seems to be mainly a product of the nickel refining process. It should also be noted that other regulators have recently set much higher targets for normal ambient air. In Europe, the target for nickel in ambient air is currently not to exceed $20 \text{ ng} / \text{m}^3$ for the total content in the PM10 fraction averaged over a calendar year (European Union, 2005). This equates by simple analogy to a target of $0.92 \mu\text{g}/\text{m}^3$ to be determined on a 24 hour basis. Even this figure is probably much below a level of exposure that could cause any harm.

The DOH values are apparently based on World Health Organisation (WHO) and US National Toxicology Program (NTP) studies and recommendations. However, it should be remembered that the WHO and US NTP values were chosen to protect industrial populations subjected to exposure from nickel refinery dust, an exposure situation that is quite different from exposures to dusts that might be caused by fugitive releases from transport of nickel concentrate or from those found in the natural environment. Quoting from the WHO Air Quality Guidelines, “On the basis of the most recent information of exposure and risk estimated **in industrial populations** (my emphasis - JHD), an incremental risk of 3.8×10^{-4} can be given for a concentration of nickel in air of $1 \mu\text{g}/\text{m}^3$. The concentrations corresponding to an excess lifetime risk of 1:10 000, 1:100 000 and 1: 1 000 000 are about 250, 25 and $2.5 \text{ ng}/\text{m}^3$, respectively”. These calculations are not relevant to the exposures likely to occur at Esperance.

The low risk of exposure to carcinogenic nickel species in an environment uncontaminated by the refinery processes is clearly shown by the epidemiological evidence cited in Section 4.1 above. Epidemiological studies have so far failed to demonstrate carcinogenicity unassociated with nickel refining and the conclusion to be drawn is that some of the nickel species generated during the refining process, either individually or together, are carcinogenic. Among the main species generated by the refining process are crystalline nickel subsulfide and high temperature (sintered) green nickel oxide. These compounds have been thoroughly tested for carcinogenicity by the U.S. National Toxicology Program (1996) in animal tests. For crystalline nickel subsulfide there are good animal data indicating carcinogenicity. For the sintered green nickel oxide, there is some evidence but it is at best indicative and not conclusive.

Sometimes there is loose reference in the literature to carcinogenic sulfidic nickel or oxidic nickel but the evidence from animal studies refers strictly to the crystalline subsulfide and the green oxide, and the carcinogenicity of the oxide is still uncertain. Here, the DOH is implicitly assuming (though the assumption is unstated) that any exposure of the public in Esperance will be to nickel subsulfide. In fact, any potentially harmful exposure will be to inhalable particles from sulfidic nickel concentrate in which most, if not all the sulfide will be in some other sulfidic form, probably not a simple nickel sulfide but a more complex chemical form, much of which may not be bioavailable. Consider that the sulfidic nickel ores are principally associated with the

iron mineral pyrrhotite (Fe_7S_8), the copper mineral chalcopyrite (CuFeS_2) and pentlandite, $(\text{Ni.Fe})_9\text{S}_8$. Compare the chemical structures for these minerals with that for the carcinogenic crystalline nickel subsulfide, Ni_3S_2 , and the difference is clear. In the recent EU Draft Risk Assessment (2005) it is clearly stated “It should be noted that the “sulphidic” species to which miners are exposed is pentlandite which is different from the nickel subsulphide and sulphides found in refining”. Another nickel sulfide, beta-nickel sulfide, NiS , which is much closer chemically to nickel subsulfide has never to my knowledge been identified as a carcinogen of any kind.

6. Conclusion

The possible health problems associated with the export of sulfidic nickel concentrate through the port of Esperance have been exaggerated as a result of the historic and persistent error of ignoring or describing inadequately the chemical speciation of nickel exposures. This has meant that for many years nickel exposure has been regulated mainly on the basis of total nickel per unit volume of environmental medium, i.e. soil, water or air. Gradually, it has become appreciated that the toxicity of metallic nickel is not the same as that of nickel salts (even nickel salts may differ in toxicity), and that the toxicity of other nickel compounds may vary considerably. In particular, carcinogenicity, our main concern, has been conclusively demonstrated for only one nickel compound, nickel subsulfide, with inconclusive evidence for another, sintered nickel oxide, and is restricted to respiratory tract cancers. This focuses our attention on the inhalation route of exposure and on these particular compounds and their presence in the atmosphere as inhalable particulates as risk factors. It is almost certain that the compounds of concern will not be present in such particulates in Esperance.

It further appears that solubility in water is an important factor in assessing the carcinogenicity of these compounds. In other words, compounds that are relatively water-insoluble and can remain in the respiratory system for a long time are the most likely to be carcinogenic. Thus, sparingly soluble crystalline nickel subsulfide is the one fully confirmed human carcinogen. This, in turn, suggests that the hypothesis that the mechanism of

carcinogenesis of nickel compounds involves only free hydrated nickel ions is incorrect since nickel subsulfide will release nickel ions slowly and it is unlikely that at low concentrations they can outcompete hydrated magnesium ions to enter cells. If nickel ions cannot enter cells, they will probably be removed from the body before they can have any effect. Thus, the old idea that all forms of nickel can release nickel ions and thus cause cancer becomes debatable. At the very least, there must be a threshold for any toxic effect of free ions and this is likely to be high because of the protective effect of magnesium ions always present in the body in significant amounts.

7. Summary

1. There is no reason to suppose that anyone in the general population of Esperance will develop any form of cancer as a result of inhaling nickel-containing particles released during the bulk loading of nickel concentrate at Esperance harbour. No other form of toxicity is likely to occur following exposure by other routes. Even the small proportion of the potentially exposed population which may be hypersensitive to nickel and show a skin reaction following prolonged skin contact is unlikely to be affected by temporary contact of particulate with the skin, itself an unlikely occurrence outwith the working environment. Even workers in the port should not have any health problems if they observe normal care and take appropriate action to avoid inhaling dust particles. They should wear appropriate dust masks and, if necessary, remove any contaminating dust by showering before changing into their normal clothes to leave the workplace.

2. Although adverse health effects resulting from exposure to nickel and (or) its compounds are unlikely, inhaled dust from the concentrate can still act in quantity as a nuisance dust and damage the lungs by nonspecific inflammatory mechanisms following chronic exposure to high concentrations. Inhalable dust should therefore be kept to a minimum and further local engineering steps taken to achieve this. However, this should not need not to go as far as complete containerisation in order to reach acceptable concentrations of nuisance dust in the air,

ensuring that no adverse effects will occur.

3. As far as I know, no classifications of any kind have yet been made under the REACH regulations and so it would be premature for me to comment on, for example, the consequences of a blanket REACH classification of nickel products as class 1 carcinogens. This suggestion seems to me most unlikely bearing in mind the irresistible scientific logic of the requirement to consider chemical speciation and to classify nickel and its compounds individually as described above. Even IARC is planning to reconsider its carcinogenicity classifications in the light of speciation considerations (IARC, 2008). An interesting paper about speciation of exposures and cancer in the US nickel alloy industry will shortly be published in “Regulatory Toxicology and Pharmacology” (Sivulka and Seilkop, 2009). No increased cancer rates could be detected at exposures to various defined nickel species.

4. It has been suggested that there is a need for a legally enforceable standard in regard to exposure to nickel sulfide in the circumstances pertaining in Esperance. Since the exposure to nickel concentrate dust is unlikely to involve nickel sulfide in a simple form, this no longer seems relevant. In principle, guidelines are to be preferred if necessary. In the situation affecting Esperance, guidelines relating to inhalable particulates and their health effects would be the most appropriate, not those relating to nickel, ignoring its chemical speciation.

Guidelines to protect people are set with regard only to the relationship of exposure to health effect. That they are not legally enforceable is generally not a problem as most legislation for human safety incorporates the concept of a “Duty of Care”. Thus, any action taken by industry that causes an excess chemical exposure according to the health based guidelines which may lead to disease in the affected population can be prosecuted for failing in its duty of care to the people at risk. Legislation incorporating the concept of “Duty of Care” may use different wording for this concept.

Legally binding standards have to be enforceable. Unenforceable standards bring the law into disrepute. They must also not be used as a trade barrier. Thus, establishing such standards requires involvement of stakeholders who have other concerns than simply health. Enforcement

agencies must be able to monitor the suggested standards using validated analytical techniques so that analytical results are beyond reasonable doubt. At international level, trade agreements may have to be modified. Important industries may have to close down with resultant unemployment. In the U.K., standards for occupational exposure to benzene and lead are higher than a toxicologist would like because too many people would be put out of work if relevant industries were closed because they were unable to reach the ideal standard. This does not necessarily mean that the workforce suffered any illness as a result of higher exposures than preferred because, in setting health based guidelines, toxicologists always err very far on the side of safety. From the preceding, it will be seen that health based guidelines will always be more protective of a population at risk than legally enforceable standards, if the guidelines and standards are set properly.

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