IPCS INTERNATIONAL PROGRAMME ON CHEMICAL SAFETY

Health and Safety Guide No. 62

NICKEL, NICKEL CARBONYL, AND SOME NICKEL COMPOUNDS HEALTH AND SAFETY GUIDE

UNITED NATIONS
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WORLD HEALTH ORGANIZATION, GENEVA 1991

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NICKEL, NICKEL CARBONYL, AND SOME NICKEL COMPOUNDS HEALTH AND SAFETY GUIDE

This is a companion volume to
Environmental Health Criteria 108: Nickel

Published by the World Health Organization for the International Programme on Chemical Safety
(a collaborative programme of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization)

WORLD HEALTH ORGANIZATION, GENEVA 1991

This report contains the collective views of an international group of experts and does not necessarily represent the decisions or the stated policy of the United Nations Environment Programme, the International Labour Organisation, or the World Health Organization

WHO Library Cataloguing in Publication Data

Nickel, nickel carbonyl, and some nickel compounds : health and safety guide.

(Health and safety guide ; no. 62)

1.Nickel - adverse effects 2.Nickel - standards 3.Environmental exposure 4.Environmental pollutants I.Series

ISBN 92 4 151062 5 (NLM Classification: QV 290)
ISSN 0259-7268

Computer typesetting by HEADS. Oxford OX7 2NY, England
Printed by Wissenschaftliche Verlagsgesellschaft mbH · D-7000 Stuttgart 10

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INTRODUCTION

The Environmental Health Criteria (EHC) documents produced by the International Programme on Chemical Safety include an assessment of the effects on the environment and on human health of exposure to a chemical or combination of chemicals, or physical or biological agents. They also provide guidelines for setting exposure limits.

The purpose of a Health and Safety Guide is to facilitate the application of these guidelines in national chemical safety programmes. The first three sections of a Health and Safety Guide highlight the relevant technical information in the corresponding EHC. Section 4 includes advice on preventive and protective measures and emergency action; health workers should be thoroughly familiar with the medical information to ensure that they can act efficiently in an emergency. Within the Guide is a Summary of Chemical Safety Information which should be readily available, and should be clearly explained, to all who could come into contact with the chemical. The section on regulatory information has been extracted from the legal file of the International Register of Potentially Toxic Chemicals (IRPTC) and from other United Nations sources.

The target readership includes occupational health services, those in ministries, governmental agencies, industry, and trade unions who are involved in the safe use of chemicals and the avoidance of environmental health hazards, and those wanting more information on this topic. An attempt has been made to use only terms that will be familiar to the intended user. However, sections 1 and 2 inevitably contain some technical terms. A bibliography has been included for readers who require further background information.

Revision of the information in this Guide will take place in due course, and the eventual aim is to use standardized terminology. Comments on any difficulties encountered in using the Guide would be very helpful and should be addressed to:

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World Health Organization
1211 Geneva 27
Switzerland

THE INFORMATION IN THIS GUIDE SHOULD BE CONSIDERED AS A STARTING POINT TO A COMPREHENSIVE HEALTH AND SAFETY PROGRAMME

1. PRODUCT IDENTITY AND USES

1.1 Identity

Common name:	Nickel
Chemical symbol:	Ni
CAS registry number:	7440-02-0

1.2 Physical and Chemical Properties

1.2.1 *Nickel*

Nickel is a naturally occurring, shiny, light-coloured metal with high electrical and thermal conductivities. It is resistant to corrosion by air, water, and alkalis, but reacts with dilute oxidizing agents.

1.2.2 *Some nickel salts*

Nickel carbonate hydroxide tetrahydrate ($2NiCO_3.3Ni(OH)_2.4H_2O$)

The composition of basic nickel carbonate varies. The most common forms range from $2NiCO_33Ni(OH)_2.XH_2O$ to $NiCO_3Ni(OH)_3.XH_20$. Nickel carbonate hydroxide is insoluble in water, but soluble in ammonia and in dilute acids.

Nickel carbonyl ($Ni(CO)_4$)

Nickel carbonyl is a colourless, volatile liquid that is formed when nickel powder is treated with carbon monoxide at about 50 °C. It is insoluble in water, but soluble in most organic solvents.

Nickel chloride ($NiCl_2$) and nickel chloride hexahydrate ($NiCl_2.6H_2O$)

Nickel chloride and nickel chloride hexahydrate are soluble in both water and ethanol.

Nickel hydroxide ($Ni(OH)_2$)

Nickel hydroxide is insoluble in water, but soluble in acids. It forms complexes with ammonia.

Nickel nitrate ($Ni(NO_3)_2$)

Nickel nitrate is readily soluble in both water and alcohol.

Nickel oxide (NiO)

The name "nickel oxide" covers several nickel-oxygen compounds that differ in stoichiometry, and chemical and physical properties.

Nickel oxide (NiO) exists in two forms. Black nickel oxide is chemically reactive and forms simple salts in the presence of acids. Green nickel oxide is an inert and refractory material. Nickel oxide is insoluble in water. The solubility in acids and other properties depend on the method of preparation of the nickel oxide.

Nickel sulfate ($NiSO_4$)

Nickel sulfate exists as a hexahydrate, initially in the α-form, which changes into the β-form at 53.3 °C. Nickel sulfate is soluble in water, ethanol, and methanol.

Nickel sulfide (NiS)

Nickel sulfide is insoluble in water.

Nickel subsulfide (Ni_3S_2)

At high temperatures, nickel subsulfide exists in a bronze-yellow form (β-Ni_3S_2). At lower temperatures, it changes to a green β-form, which is stable at normal temperature. α-Nickel subsulfide occurs naturally as the grey mineral heazlewoodite. Nickel subsulfide is insoluble in water.

1.3 Analytical Methods

The most commonly used analytical methods for biological and environmental samples are atomic absorption spectroscopy and voltammetry. In order to obtain reliable results, especially in the ultratrace range, specific procedures have to be followed to minimize the risk of contamination during sample collection, storage, processing, and analysis. Depending on sample pretreatment, extraction, and enrichment procedures, detection limits of 1–100 ng/litre can be achieved in biological samples and water.

For water, electrothermal atomic absorption spectroscopy (EAAS) has a detection limit of 10 ng nickel/litre. Voltammetry is more sensitive and the use of differential pulse voltammetry (DPV) can achieve a detection limit of 1 ng/litre.

DPV can also be used for the determination of the nickel contents of foodstuffs.

Flame atomic absorption spectroscopy (FAAS) is a commonly used method for measuring nickel concentrations in air. Inductively coupled plasma atomic emission spectroscopy (ICP-AES) can also be used for the analysis of air samples.

Atomic absorption spectroscopy is a sensitive method for nickel determination in blood, serum, urine, other biological samples, and soil.

Electron microscopy and X-ray microanalysis can be used for the determination of nickel levels in dust particles, such as grinding dust and welding fumes.

1.4 Uses

Nickel is mined from sulfide or oxide ores (laterites). Production of nickel metal is performed by pyro- and hydrometallurgical methods. Final refining can be achieved by electrolytic techniques or by passing carbon monoxide gas over nickel powder to form nickel carbonyl, which is decomposed to yield pure nickel.

A major use of nickel is as an alloying element for steel and cast iron, yielding alloys and steels with increased strength and resistance to

corrosion and temperature. Nickel compounds are used in nickel-cadmium batteries, in electronic and computer equipment, and as constituents of pigments in the glass and ceramics industries. Other important applications include the use of nickel sulfate and nickel chloride in electroplating, and of nickel compounds as catalysts in the manufacture of organic chemicals, petroleum refining, and edible oil hardening. Nickel carbonyl is used in nickel refining.

2. SUMMARY AND EVALUATION

2.1 Sources of Nickel

Nickel is a ubiquitous trace metal and occurs in soils, water, air, and in the biosphere. The average content of the earth's crust is about 0.008%. Farm soils contain between 3 and 1000 mg nickel/kg. Levels in natural waters have been found to range from 2 to 10 μg/litre (fresh water) and from 0.2 to 0.7 μg/litre (marine). Atmospheric nickel concentrations in remote areas range from < 0.1 to 3 ng/m^3.

Nickel ore deposits are accumulations of nickel sulfide minerals (mostly pentlandite) and laterites. Nickel is extracted from the mined ore by pyro- and hydro-metallurgical refining processes. Global mining production of nickel was approximately 67 million kg in 1985.

Most of the nickel is used for the production of stainless steel and other nickel alloys with high corrosion and temperature resistance. Nickel alloys and nickel plating are used in vehicles, processing machinery, armaments, tools, electrical equipment, household appliances, and coinage. Nickel compounds are also used as catalysts, pigments, and in batteries. The primary sources of nickel emissions into the ambient air are the combustion of coal and oil for heat or power generation, the incineration of waste and sewage sludge, nickel mining and primary production, steel manufacture, electroplating, and miscellaneous sources, such as cement manufacturing. In polluted air, the predominant nickel compounds appear to be nickel sulfate, oxides, and sulfides, and, to a lesser extent, metallic nickel.

Nickel from various industrial processes and other sources finally reaches waste water. Residues from waste-water treatment are disposed of by deep well injection, ocean dumping, land treatment, and incineration. Effluents from waste-water treatment plants have been reported to contain up to 0.2 mg nickel/litre.

2.2 Behaviour in the Environment

Nickel is introduced into the environment from both natural and man-made sources and is circulated throughout all environmental

compartments by means of chemical and physical processes, as well as by being biologically transported by living organisms.

Atmospheric nickel is considered to exist mainly in the form of particulate aerosols containing different concentrations of nickel, depending on the source. The highest nickel concentrations in ambient air are usually found in the smallest particles. Nickel carbonyl is unstable in air and decomposes to form nickel oxide.

The transport and distribution of nickel particles to, or between, different environmental compartments is strongly influenced by particle size and meteorological conditions. Particle size distribution is primarily a function of the emitting sources. In general, particles from man-made sources are smaller than natural dust particles.

Nickel is introduced into the hydrosphere by removal from the atmosphere, by surface runoff, by discharge of industrial and municipal waste, and also following natural erosion of soils and rocks. In rivers, nickel is mainly transported in the form of a precipitated coating on particles, and in association with organic matter; in lakes, it is transported in the ionic form, predominantly in association with organic matter. Nickel may also be absorbed on clay particles and via uptake by biota. Absorption processes can be reversed leading to the release of nickel from the sediment. Part of the nickel is transported via rivers and streams to the ocean. Riverine suspended particulate input is estimated to be 135×10^7 kg/year.

Depending on the soil type, nickel may exhibit a high mobility within the soil profile, finally reaching ground water and, thus, rivers and lakes. Acid rain has a pronounced tendency to mobilize nickel from the soil. Terrestrial plants take up nickel from soil, primarily via the roots. The amount of nickel uptake from soil depends on various geochemical and physical parameters including the type of soil, soil pH, humidity, the organic matter content of the soil, and the concentration of extractable nickel. The best known example of nickel accumulation is the increased nickel levels, in excess of 1 mg/kg dry weight, found in a number of plant species ("hyperaccumulators") growing on relatively infertile serpentine soils. Nickel levels exceeding 50 mg/kg dry weight are toxic for most plants. Accumulation and toxic effects have been observed in vegetables grown on sewage sludge-treated soils and in vegetation near nickel-emitting sources. High concentration factors have been observed in aquatic plants.

Laboratory studies showed that nickel had little capacity for accumulation in all the fish studied. In uncontaminated waters, the concentrations reported in whole fish, on a wet-weight basis, ranged from 0.02 to 2 mg/kg. These values could be up to 10 times higher in fish from contaminated waters. However, there is no evidence for the biomagnification of nickel in the food chain.

In wildlife, nickel is found in many organs and tissues, due to dietary uptake by herbivorous animals and their carnivorous predators.

2.3 Human Exposure

Typical atmospheric nickel levels for human exposure range from about 5 to 35 ng/m^3 at rural and urban sites, leading to a nickel uptake via inhalation of 0.1–0.7 μg/day. Drinking-water generally contains less than 10 μg nickel/litre, but, occasionally, nickel may be released from the plumbing fittings resulting in concentrations of up to 500 μg nickel/litre.

Nickel concentrations in food are usually below 0.5 mg/kg fresh weight. Cocoa, soybeans, some dried legumes, various nuts, and oatmeal contain high concentrations of nickel. Daily intake of nickel from food varies widely, according to different dietary habits, and can range from 100 to 800 μg/day; the mean dietary nickel intake in most countries is 100–300 μg/day. Release of nickel from kitchen utensils may contribute significantly to oral intake. Pulmonary intake of 2–23 μg nickel/day can result from smoking 40 cigarettes a day.

Daily skin contact with nickel-plated objects or nickel-containing alloys (e.g., jewellery, coins, clips) is an important factor in the induction and maintenance of contact hypersensitivity.

Iatrogenic exposure to nickel results from implants and prostheses made from nickel-containing alloys, from intravenous or dialysis fluids, and from radiographic contrast media. An estimated average intravenous nickel uptake from dialysis fluids is 100 μg per treatment.

In the working environment, airborne nickel concentrations can vary from a few μg/m^3 to, occasionally, a few mg/m^3, depending on the process involved and the nickel content of the material being handled.

Globally, millions of workers are exposed to nickel-containing dusts and fumes during welding, plating, grinding, mining, nickel refining, and in steel plants, foundries, and other metal industries.

Dermal exposure to nickel may occur in a wide range of jobs, either by direct exposure to dissolved nickel, e.g., in the refining, electroplating, and electroforming industries, or by handling nickel-containing tools. Wet cleaning work may involve exposure to nickel, because of the nickel that becomes dissolved in the washing water.

2.4 Metabolism

Nickel can be absorbed by humans and animals via inhalation, ingestion, or percutaneously. Respiratory absorption with secondary gastrointestinal absorption of nickel (insoluble and soluble) is the major route of entry during occupational exposure. A significant quantity of inhaled material is swallowed following mucociliary clearance from the respiratory tract. Thus, poor personal hygiene and work practices can contribute to gastrointestinal exposure. Percutaneous absorption is negligible, quantitatively, but is important in the pathogenesis of contact hypersensitivity. Absorption is related to the solubility of the compound, following the general relationships nickel carbonyl > soluble nickel compounds > insoluble nickel compounds. Nickel carbonyl is the most rapidly and completely absorbed nickel compound in both animals and man. Studies in which nickel was administered via inhalation are limited. Studies on hamsters and rats, exposed to insoluble nickel oxide, showed poor absorption with retention of much of the material in the lung after several weeks. In contrast, absorption of soluble nickel chloride or amorphous nickel sulfide was rapid. Nickel is transported in the blood principally bound to albumin.

Gastrointestinal absorption of nickel is variable and depends on the composition of the diet. In a recent study on human volunteers, absorption of nickel was 27% from water versus less than 1% from food. All body secretions are potential routes of excretion including urine, bile, sweat, tears, milk, and mucociliary fluid. Non-absorbed nickel is eliminated in the faeces.

Transplacental transfer has been demonstrated in rodents.

Following parenteral administration of nickel salts, the highest nickel accumulation occurs in the kidney, endocrine glands, lung, and liver: high

concentrations are also observed in the brain following administration of nickel carbonyl. Data on nickel excretion suggest a two-compartment model. Nickel concentrations in the serum and urine of healthy non-occupationally exposed adults are 0.2 μg/litre (range: < 0.05–1.1 μg/litre) and 2 μg/litre (range: 0.5–6.0 μg/litre) equivalent to 2 μg/g creatinine (range: 0.4–6.0 μg/g creatinine), respectively. Increased concentrations of nickel are noted in both of these fluids following occupational exposure. The body burden of nickel in a non-exposed, 70 kg adult is 0.5 mg.

2.5 Effects on Organisms in the Environment

In microorganisms, growth was generally inhibited at nickel concentrations in the medium of 1–5 mg/litre in the case of actinomycetes, yeast, and marine and non-marine eubacteria, and at concentrations of 5–1000 mg/litre in the case of filamentous fungi. In algae, no growth was observed at approximately 0.05–5 mg nickel/litre. Abiotic factors, such as the pH, hardness, temperature, and salinity of the medium, and the presence of organic and inorganic particles, influence the toxicity of nickel.

Nickel toxicity in aquatic invertebrates varies considerably, according to species and abiotic factors. A 96-h LC_{50} of 0.5 mg nickel/litre was found for *Daphnia* spp. while, in molluscs, 96-h LC_{50} values of around 0.2 mg/litre were found for two freshwater snail species and of 1100 mg/litre for a bivalve.

In fish, the 96-h LC_{50} values generally fall within the range 4–20 mg nickel/litre, but they can be higher in some species. Long-term studies on fish and fish development demonstrated some effects on rainbow trout in soft water at levels as low as 0.05 mg nickel/litre. In terrestrial plants, nickel levels exceeding 50 mg/kg dry weight are usually toxic. Copper was found to act toxicologically in a synergistic way, whereas calcium reduced the toxicity of nickel. Data on the effects of nickel on terrestrial animals are limited. Earthworms seem to be relatively insensitive to nickel, when the medium is rich in microorganisms and organic matter, thus making nickel less available to earthworms. Nickel has not been considered as a wide-scale global contaminant; however, ecological changes, such as decreases in the number and diversity of species, have been observed near nickel-emitting sources. Microecosystem studies have shown that the addition of nickel to soil disturbs the nitrogen cycle.

2.6 Effects on Experimental Animals and *In vitro* Test Systems

Nickel is essential for the catalytic activity of some plant and bacterial enzymes. Slow weight gain, anaemia, and decreased viability of offspring have been described in some animal species, after dietary deprivation of nickel.

The most acutely toxic nickel compound is nickel carbonyl, the lung being the target organ; pulmonary oedema may occur within 4 h following exposure. The acute toxicity of other nickel species is low.

Though contact allergy to nickel is very common in humans, experimental sensitization in animals is only successful under special conditions. Long-term inhalation exposure to metallic nickel, nickel oxide, or nickel subsulfide caused mucosal damage and an inflammatory reaction in the respiratory tract in rats, mice, and guinea-pigs. Epithelial hyperplasia was observed in rats after inhalation exposure to nickel chloride or nickel oxide aerosols.

High-level, long-term exposure to nickel oxide led to gradually progressive pneumoconiosis in rats. Inflammatory reaction, sometimes accompanied by slight fibrosis, was observed in rabbits after high-level exposure to nickel-graphite dust. Pulmonary fibrosis was seen in rats exposed to nickel subsulfide.

Nickel salts induced a rapid transistory hyperglycaemia in rats, rabbits, and chickens, after parenteral administration. These changes may be associated with effects on alpha and beta cells in the islets of Langerhans. Nickel also decreased the release of prolactin. Nickel chloride, given orally or by inhalation, has been reported to decrease iodine uptake by the thyroid.

Nickel salts, given intravenously, decreased blood flow in the coronary arteries in the dog; high concentrations of nickel decreased the contractility of the dog myocardium *in vitro*.

Nickel chloride affects the T-cell system and suppresses the activity of natural killer cells. Parenteral administration of nickel chloride and nickel subsulfide has been reported to cause intrauterine mortality and decreased weight gain in rats and mice. Inhalation exposure to nickel carbonyl caused

fetal death and decreased weight gain, and was terato- genic in rats and hamsters. No information on maternal toxicity was given in these studies. Nickel carbonyl has been reported to cause dominant lethal mutations in rats.

Several inorganic nickel compounds were tested for mutagenicity in various test systems. Nickel compounds were generally inactive in bacterial mutagenesis assays, except where fluctuation tests were used. Mutations were observed in several cultured mammalian cell types. Nickel compounds inhibited DNA synthesis in a wide variety of organisms. In addition, nickel compounds induced chromosomal aberrations and sister chromatid exchange (SCE) in both mammalian and human cultured cells. Chromosomal aberrations, but not SCE (except in one study on electrolysis workers), were observed in humans occupationally exposed to either insoluble or soluble nickel compounds. Nickel induced cell transformation *in vitro*.

Nickel subsulfide induced benign and malignant pulmonary tumours in rats in an inhalation study. A few pulmonary tumours were seen in rats in a series of inhalation studies with nickel carbonyl. There was no significant increase in lung tumours in rats in an adequate inhalation study with metallic nickel. Inhalation exposure to black nickel oxide did not induce lung tumours in Syrian golden hamsters (a species resistant to lung carcinogenesis). Adequate carcinogenicity studies using inhalation exposure were not available for other nickel compounds. However, nickel subsulfide, metallic nickel powder, and an unspecified nickel oxide induced benign and malignant lung tumours in rats after repeated intratracheal instillations.

Nickel carbonyl, nickelocene, and a large number of slightly soluble or nonsoluble nickel compounds, including nickel subsulfide, carbonate, chromate, hydroxide, sulfides, selenides, arsenides, telluride, antimonide, various unidentified oxide preparations, two nickel-copper oxides, metallic nickel, and various nickel alloys, induced local mesenchymal tumours in a variety of experimental animals after intramuscular, subcutaneous, intraperitoneal, intrapleural, intraocular, intraosseous, intrarenal, intra-articular, intratesticular, or intra-adipose administration. No local carcinogenic response was seen in single-dose studies with some nickel alloys, colloidal nickel hydroxide, or with two specimens of nickel oxide, especially prepared for carcinogenicity testing by calcining at 735 °C or 1045 °C.

Nickel sulfate and nickel acetate, but not nickel chloride, induced tumours of the peritoneal cavity in rats after repeated intraperitoneal administration.

Metallic nickel and a very large number of nickel compounds have been tested for carcinogenicity by the parenteral route of administration; with few exceptions, they caused local tumours.

Only nickel subsulfide has been shown convincingly to cause cancer after inhalation exposure. However, the number of adequate inhalation studies is very small.

In studies using repeated intratracheal instillation, nickel powder, nickel oxide, and nickel subsulfide caused pulmonary tumours.

When three different soluble nickel salts, which had not induced local tumours in earlier studies, were tested using repeated intraperitoneal administration, two of the salts elicited a carcinogenic response.

The International Agency for Research on Cancer concluded, in 1989, that there was:

a) *sufficient evidence* in experimental animals for the carcinogenicity of metallic nickel, nickel monoxides, nickel hydroxides, and crystalline nickel sulfides;

b) *limited evidence* in experimental animals for the carcinogenicity of nickel alloys, nickelocene, nickel carbonyl, nickel salts, nickel arsenides, nickel antimonide, nickel selenides, and nickel telluride;

c) *inadequate evidence* in experimental animals for the carcinogenicity of nickel trioxide, amorphous nickel sulfide, and nickel titanate.

2.7 Effects on Human Beings

In terms of human health effects, nickel carbonyl is the most acutely toxic nickel compound. The effects of acute nickel carbonyl poisoning include frontal headache, vertigo, nausea, vomiting, insomnia, and irritability, followed by pulmonary symptoms similar to those of a viral pneumonia. Pathological pulmonary lesions include haemorrhage, oedema, and

cellular derangement. Liver, kidneys, adrenal glands, spleen, and brain are also affected. Cases of nickel poisoning have also been reported in patients dialysed with nickel-contaminated dialysate and in electroplaters who accidentally ingested water contaminated with nickel sulfate and nickel chloride.

Chronic effects, such as rhinitis, sinusitis, nasal septal perforations, and asthma, have been reported in nickel refinery and nickel plating workers. Some authors have reported pulmonary changes with fibrosis in workers inhaling nickel dust. In addition, nasal dysplasia has been reported in nickel refinery workers. Nickel contact hypersensitivity has been documented extensively in both the general population and in a number of occupations, including those in which workers were exposed to soluble nickel compounds. In several countries, it has been reported that 10% of the female population and 1% of the male population are sensitive to nickel; 40–50% of these have vesicular hand eczema, which, in some cases, may be very severe and lead to loss of working ability. Oral nickel intake may aggravate vesicular hand eczema and possibly eczema arising on other parts of the body where there has been no skin contact with nickel.

Prostheses or other surgical implants made from nickel-containing alloys have been reported to cause nickel sensitization or to aggravate existing dermatitis.

Nephrotoxic effects, such as renal oedema with hyperaemia and parenchymatous degeneration, have been reported in cases of accidental industrial exposure to nickel carbonyl. Transient nephrotoxic effects have been recorded after accidental ingestion of nickel salts.

Very high lung and nasal cancer risks have occurred in nickel refinery workers employed in the high-temperature roasting of sulfide ores, involving substantial exposure to nickel subsulfide, nickel oxide, and perhaps nickel sulfate. Similar risks have been reported in processes involving exposure to soluble nickel (electrolysis, copper sulfate extraction, hydrometallurgy), often combined with some nickel oxide exposure, but with low nickel subsulfide exposure. The risk to miners and other refinery workers has been much lower. Cancer rates have generally been close to normal in workers in stainless steel welding and in the nickel-using industries, except where exposure to chromium has been involved, particularly electroplating. However, the risk of lung cancer may have been slightly increased in nickel/cadmium battery workers exposed

to high levels of both nickel and cadmium. Excesses of various cancers, other than lung and nasal cancers (e.g.,renal, gastric, or prostatic cancers), have occasionally been reported in nickel workers, but none has been found consistently.

There is evidence of a cancer risk in workers who had been exposed to soluble nickel concentrations of the order of 1–2 mg/m^3, both in electrolysis and in the preparation of soluble salts. These workers were also exposed to other nickel compounds, but often at lower levels than in other high-risk processes. In the absence of historical exposure measurements, it is impossible to draw unequivocal conclusions, but the evidence that soluble nickel is carcinogenic is certainly strong. Refinery dust sometimes contains a substantial proportion of nickel sulfate, in addition to nickel subsulfide. This raises the possibility that the very high cancer risk observed in workers employed in the high-temperature oxidation of nickel subsulfide may partly be due to soluble nickel.

In refinery areas where cancer risks were high, exposure to nickel subsulfide almost always occurred together with exposure to the oxide and perhaps the sulfate (see above). Therefore, it is difficult to demonstrate that nickel subsulfide is carcinogenic on the basis of epidemiological data alone, though this seems likely.

Nickel oxide was present in almost all circumstances in which cancer risks were elevated, together with one or more other forms of nickel (nickel subsulfide, soluble nickel, metallic nickel). As for nickel subsulfide, it is difficult either to demonstrate or to disprove its suspected carcinogenicity from epidemiological data alone.

No increased cancer risk has been demonstrated in workers exposed exclusively to metallic nickel. The combined data on nickel alloy workers and gaseous diffusion workers, who were exposed to average concentrations of the order of 0.5 mg nickel/m^3, show no excess risk, though the total number of lung cancers in these cohorts is too small to exclude a small increase in risk at this level.

The International Agency for Research on Cancer concluded, in 1989, that nickel compounds are carcinogenic to humans (Group 1) and metallic nickel is possibly carcinogenic to humans (Group 2B).

3. CONCLUSIONS AND RECOMMENDATIONS

3.1 Conclusions

(*a*) *Exposure*

Nickel is an ubiquitous element and has been detected in different media in all parts of the biosphere.

Nickel is introduced into the environment from both natural and man-made sources and is circulated throughout all environmental compartments by means of chemical and physical processes, as well as by the biological transport mechanisms of living organisms.

Acid rain may leach nickel as well as other metals from plants and soil.

Atmospheric nickel is considered to exist mainly in the form of particulate aerosols.

Nickel is introduced into the hydrosphere by removal from the atmosphere, by surface run-off, by the discharge of industrial and municipal wastes, and also following natural erosion of soils and rocks.

A major source of nickel in the environment is the combustion of fossil fuels, particularly coal.

Uncontrolled emissions and disposal of wastes may impact the environment and have adverse effects.

CONCLUSIONS AND RECOMMENDATIONS

The chemical and physical forms of nickel and its salts strongly influence their bioavailability and toxicity.

Nickel from soil and water is absorbed and metabolized by plants and microorganisms and these small quantities of nickel are widely present in all foods and water.

Some foods, such as pulses and cocoa products, contain relatively high amounts of nickel, but these quantities have not been correlated with adverse health effects.

(*b*) *Human health effects*

Nickel is normally present in human tissues and, under conditions of high exposure, these levels may increase significantly.

The general population is exposed to nickel via the diet and objects containing nickel, especially jewellery and coins.

Occupational exposure to nickel is important.

Inhalation is an important route of exposure to nickel and its salts with regard to health risks. The gastrointestinal route is of lesser importance.

Nickel absorption from the gastrointestinal tract is poor, though, in an empty stomach, nickel in drinking-water is absorbed to a greater extent. This may be a risk for sensitized persons.

CONCLUSIONS AND RECOMMENDATIONS

Smoking tobacco may contribute to nickel intake, but there is no agreement on the chemical nature of nickel or on its health significance in tobacco smoke.

Target organs are the respiratory system, especially the nasal cavities and sinuses, and the immune system.

The percutaneous absorption of nickel is minor, but important in sensitization.

Nickel and its salts are potent skin sensitizers and possible respiratory sensitizers in man. Nickel dermatitis is a common result of nickel exposure, especially in women.

Primary skin and eye irritation reactions to high concentrations of soluble nickel salts have also been reported.

Acute nickel toxicity is a minor risk, except in the case of nickel carbonyl.

There is no convincing evidence that nickel salts produce point mutations in bacterial systems. However, some nickel salts are clastogenic *in vitro*, producing chromosome aberrations (transformation), and sister chromatid exchanges, in mammalian cells.

Evidence for a carcinogenic risk from oral nickel exposure is lacking, but the possibility that nickel acts as a promoter has been raised.

There is evidence of a carcinogenic risk in association with the inhalation of nickel metal dusts and some nickel compounds.

Only very high concentrations of nickel induce teratogenic or genotoxic effects.

The effects of nickel on the immune system are not clear.

(*c*) *Environmental effects*

Nickel is accumulated by plants. Growth retardation has been reported in some species exposed to high nickel concentrations.

There is no evidence that nickel undergoes biotransformation, though it does undergo complexation.

Nickel has been shown to be essential for the nutrition of many microorganisms, a variety of plants, and for some vertebrate animals.

3.2 Recommendations

The use of nickel in consumer products that may release nickel in contact with skin should be regulated. The specification and testing requirements should be standardized.

Priority should be given to improving industrial hygiene in occupations where exposure to high levels of soluble nickel compounds may occur.

4. HUMAN HEALTH HAZARDS, PREVENTION AND PROTECTION, EMERGENCY ACTION

4.1 Main Human Hazards, Prevention and Protection, First Aid

The human health hazards associated with exposure to nickel and nickel compounds, preventive and protective measures, and first aid are listed in the Summary of Chemical Safety Information in section 6.

4.1.1 *Advice to physicians*

In cases of suspected poisoning by inhalation, attention should be paid to the lungs and upper respiratory tract for irritant effects. Acute poisoning can be associated with heart failure. Admit to hospital as soon as possible. Obtain detailed advice on diagnosis and treatment from the nearest Poisons Information Centre.

Nickel carbonyl is the only nickel compound that causes acute poisoning by inhalation. If breathing stops, apply artificial respiration and administer oxygen. Measurement of urinary nickel will assist in assessing the severity of poisoning. Inhaled steroids will help to prevent lung damage and oedema. When poisoning is the result of ingestion, gastric lavage can be performed, providing precautions are taken to prevent accidental aspiration into the respiratory tract.

4.1.2 *Health surveillance advice*

Workers occupationally exposed to nickel and its compounds should undergo periodic health checks, with emphasis on the condition of the skin, lungs, and upper respiratory tract. Cases of dermatitis should be patch tested (usually with 0.5% nickel sulfate) by a qualified dermatologist; in case of positive results they should be given alternative employment, where available. Since nickel exposure can interfere with immune defence mechanisms, careful attention should be given to persistent infective diseases.

The physician should be aware of the carcinogenic potential of nickel and nickel compounds.

4.2 Explosion and Fire Hazards

4.2.1 *Explosion hazards*

The vapour of nickel carbonyl is heavier than air. It can react violently with atmospheric oxygen, with risk of explosion at about 60 °C.

4.2.2 *Fire hazards*

With the exception of nickel carbonyl, most nickel compounds of commercial interest do not normally constitute a fire hazard. Liquid nickel carbonyl is extremely flammable and autoignition is possible at its boiling point (42.2 °C). Nickel carbonyl vapour can ignite spontaneously at room temperature.

4.2.3 *Prevention*

For nickel carbonyl, use closed systems, suitable ventilation, non-sparking tools, explosion-protected electrical equipment and lighting. Do not use nickel carbonyl near sources of ignition. Do not use compressed air for filling, discharging, or handling nickel carbonyl. In case of fire, keep containers with nickel carbonyl cool by spraying with water. Fire fighters should use self-contained breathing apparatus.

4.2.4 *Fire extinguishing agents*

Suitable agents include: carbon dioxide, powder, or water.

4.3 Storage

Nickel compounds should be stored in tightly closed and correctly labelled containers. In the case of nickel carbonyl, these should be kept in a cool, ventilated area away from heat and oxidizing agents, such as nitric acid and chlorine.

4.4 Transport

In case of accident during the road transport of nickel carbonyl, stop the engine. Remove all sources of ignition. Evacuate the danger area. Keep

bystanders at a distance, put warning signs on the road, and keep upwind. Notify the police and fire brigade immediately. If self-contained breathing apparatus is available, use it. In case of spillage or fire, follow the advice given in sections 4.7 and 4.4, respectively. In case of poisoning, follow the advice in the Summary of Chemical Safety Information (section 6).

4.5 Spillage and Disposal

4.5.1 *Spillage*

In case of spillage of nickel carbonyl, remove all ignition sources and evacuate the danger area. Wear full protective clothing and a self-contained breathing apparatus. Collect and put leaking liquid in sealable containers. Cover smaller quantities of spilled liquid with water and slowly add nitric acid to convert nickel carbonyl into nickel nitrate. In case of larger spillage, absorb the spilled liquid in a absorbent and remove in a sealable container to a safe place. Do not allow nickel carbonyl to runoff into sewers and ditches. To avoid water contamination, do not allow spilled nickel compounds (especially soluble nickel salts) to run into soil and ground water.

4.5.2 *Disposal*

Large quantities of nickel carbonyl should be collected and atomized in a suitable combustion chamber equipped with an efficient effluent gas-cleaning device. Other nickel compounds should also be treated as hazardous wastes.

5. HAZARDS FOR THE ENVIRONMENT AND THEIR PREVENTION

Nickel circulates throughout all environmental compartments (air, soil, and water), and can be accumulated by microorganisms and higher aquatic and terrestrial organisms. Nickel is toxic for many organisms, but problems arise only when it is present in high concentrations as a result of man-made contamination.

Contamination of soil, water, and air can be minimized by proper methods of storage, transport, handling, and waste disposal. In case of spillage, apply methods recommended in section 4.7.1.

6. SUMMARY OF CHEMICAL SAFETY INFORMATION

This summary should be easily available to all health workers concerned with, and users of, nickel and nickel compounds. It should be displayed at, or near, entrances to areas where there is potential exposure to nickel and nickel compounds, and on processing equipment and containers. The summary should be translated into the appropriate language(s). All persons potentially exposed to the chemical should also have the instructions in the summary clearly explained.

Space is available for insertion of the National Occupational Exposure Limit, the address and telephone number of the National Poison Control Centre, and for local trade names.

SUMMARY OF CHEMICAL SAFETY INFORMATION

NICKEL CARBONYL

$Ni(C0)_4$,: CAS Registry No. 13463-39-3

PHYSICAL PROPERTIES		OTHER CHARACTERISTICS
Melting point (°C)	-19.3	Nickel carbonyl is a colourless liquid that autoignites at its boiling point (42.2 °C); itsl vapour can ignite spontaneously at room temperature.
Boiling point (°C)	43	
Water solubility	insoluble	
Specific density (g/cm^3) (25 °C)	1.318	
Relative vapour density (25 °C)	1.2983	
Vapour pressure (kPa) (20 °C)	4.28	
Flash-point (°C)	-20	

HAZARDS/SYMPTOMS	PREVENTION AND PROTECTION	FIRST AID
SKIN: nickel carbonyl may enter body through skin	Wear effective, impervious clothing, gloves and boots; change clothing daily, maintain high standard of personal hygiene	Wash skin immediately with plenty of water; remove contaminated clothing

EYES: nickel carbonyl may enter body through mucous membranes	Wear safety goggles or face-shield	Rinse eyes with plenty of water for at least 15 minutes; obtain medical advice
INHALATION: fatigue, nausea, vomiting, headache, dyspnoea; after a latency period of 12–36 h: chest pain, difficulty in breathing, coughing, elevated temperature, lung oedema, cyanosis, death in severe cases	Use closed systems with automatic devices and alarm systems; apply exhaust ventilation; wear self-contained breathing apparatus for non-routine operations	Fresh air, rest; keep victim warm, conscious victim may inhale steroid spray; if breathing has stopped, apply artificial respiration; obtain medical attention and hospital admission urgently
INGESTION: effects on gastro-intestinal and respiratory system; in severe cases: death	Do not drink, eat, or smoke when working with nickel carbonyl	Give plenty of water to drink; administer activated charcoal if available; obtain medical attention and hospital admission
REPEATED EXPOSURE		
INHALATION: excitement, sleeplessness, headache, dizziness, weakness, poor memory, tightness in chest, polyidrosis, loss of hair, sexual frigidity, increased risk of nasal and lung cancer	Avoid exposure	

SUMMARY OF CHEMICAL SAFETY INFORMATION (*continued*)

SPILLAGE	STORAGE	FIRE AND EXPLOSION
Remove ignition sources; evacuate area; collect leaking liquid in sealable containers	Store in tightly closed containers; store containers with nickel carbonyl in a cool, ventilated area away from oxidizers	Nickel carbonyl is extremely flammable; no open fires, no sparks; extinguish with carbon-dioxide, powder, fluorocarbons, or water; nickel carbonyl vapour can react violently with air

WASTE DISPOSAL
Atomization in a combustion chamber with appropriate effluent gas-cleaning device

SUMMARY OF CHEMICAL SAFETY INFORMATION

NICKEL, NICKEL ALLOYS AND NICKEL COMPOUNDS

Ni: CAS Registry No. 7440-02-0

PHYSICAL PROPERTIES (OF ELEMENTAL NICKEL)		OTHER CHARACTERISTICS
Melting point (°C)	1555	Nickel is a naturally occurring, lustrous, light-coloured metal, very resistant to corrosion by air, water, and non-oxidizing acids; most nickel compounds do not constitute a fire or explosion hazard
Boiling point (°C)	2837	
Water solubility	insoluble	
Specific density (g/cm^3) (25 °C)	8.90	
Relative vapour density (25 °C)	-	
Vapour pressure (kPa) (20 °C)	-	
Flash-point (°C)	-	

HAZARDS/SYMPTOMS	PREVENTION AND PROTECTION	FIRST AID
(Nickel is a possible human carcinogen: nickel compounds are carcinogenic)		
SKIN: irritation, dermatitis (eczema)	Wear effective, impervious clothing, gloves and boots; change clothing daily; maintain high standard of personal hygiene	Wash skin immediately with plenty of water; remove contaminated clothing; obtain medical advice

SUMMARY OF CHEMICAL SAFETY INFORMATION (*continued*)

HAZARDS/SYMPTOMS	PREVENTION AND PROTECTION	FIRST AID
EYES: irritation by dust and aerosols	Wear safety goggles or face shield	Rinse eyes with plenty of water for at least 15 minutes; obtain medical advice
INHALATION (powders, dusts, aerosols): irritation of respiratory tract; carcinogenicity	Use closed system with automatic monitoring devices and alarm systems; apply exhaust ventilation; use self-contained breathing apparatus for non-routine operations	Remove victim to fresh air; keep victim warm and quiet; obtain medical advice
INGESTION (solids, solutions): vomiting, diarrhoea, tremor, respiratory problems, death; and in case of nickel salts solutions: nausea, headache, giddiness, lassitude	Do not drink, eat, or smoke when working with nickel compounds	Keep victim warm and quiet; give plenty of water to drink; administer activated charcoal, if available; obtain medical attention and hospital admission
REPEATED EXPOSURE		
SKIN: Dermatitis (eczema)	Wear clean impervious clothing, gloves, and boots; change clothing daily and maintain high standard of personal hygiene	

INHALATION: Chronic irritation of upper respiratory tract, loss of sense of smell, bronchial asthma, pulmonary fibrosis, pneumoconiosis; increased risk of nasal and lung cancer	Avoid exposure or keep exposure as low as possible	
SPILLAGE	STORAGE	FIRE AND EXPLOSION
Remove larger spillages of dusts or solutions containing nickel by special vacuum cleaners or with water	Store in tightly closed containers	Not flammable
WASTE DISPOSAL		
Recycle, if possible; otherwise use hazardous waste disposal site; highly toxic nickel salts, e.g., arsenide, antimonide, selenide, should be encapsulated before disposal		

7. CURRENT REGULATIONS, GUIDELINES, AND STANDARDS

The information given in this section has been extracted from the International Register of Potentially Toxic Chemicals (IRPTC) legal file and other United Nations sources. Its intention is to give the reader a representative, but not an exhaustive, overview of current regulations, guidelines, and standards. When no effective date appears in the IRPTC legal file, the year of the reference from which the data are taken is indicated by (r).

The reader should be aware that regulatory decisions about chemicals, taken in a certain country, can only be fully understood in the framework of the legislation of that country. Furthermore, the regulations and guidelines of all countries are subject to change and should always be verified with the appropriate regulatory authorities before application.

7.1 Previous Evaluations by International Bodies

No information available.

7.2 Exposure Limit Values

Some exposure limit values are given in the table on pages 38–44.

7.3 Specific Restrictions

In the Federal Republic of Germany, the concentration of respirable dusts and aerosols of nickel metal, nickel sulfide and sulfidic ores, nickel oxide, nickel carbonate, and nickel carbonyl in air emissions, may not exceed 1 mg nickel/m^3 at a mass flow (= mass of emitted compound related to time) of 5 g/h or more. In the European Economic Community (EEC), nickel emissions in waste gas, resulting from the combustion of waste oils, may not exceed 1.0 mg/m^3 (plants with thermal input of 3 MW or more). The USSR has set daily, average, maximum allowable concentrations in ambient air of 0.0002 mg/m^3 for water-soluble nickel salts and 0.001 mg/m^3 for metallic nickel.

The EEC, Sweden, and the USA regulate nickel waste. In the EEC, member states must limit the introduction of nickel and its compounds into

ground and marine waters by controlling direct and indirect discharges. Nickel in titanium dioxide waste discharged on to soil can result in the migration of nickel to surface and ground waters. The use of sludge as an agricultural fertilizer is prohibited, if the concentration of nickel exceeds 400 mg/kg (dry matter) and the limit for the amount of nickel that can be added annually to agricultural land is 3 kg/ha per year (10-year average). Sweden requires reporting of the composition of nickel wastes, and authorization for transport, handling, and export. The United Kingdom treats waste consisting of nickel and nickel compounds as "special waste", with specified disposal procedures. In the USA, nickel and its compounds are classified as toxic pollutants and permits are required for discharges into water.

7.4 Labelling, Packaging, and Transport

The United Nations classifies dry nickel catalyst as:

Hazard Class 4.2 – Substance liable to spontaneous combustion;

Packing Group 1 – Very dangerous substance.

Czechoslovakia classifies nickel in nickel ore processing and nickel production and refining as a carcinogenic substance; requirements are listed for handling, labelling, packing, storing, and transport.

7.5 Waste Disposal

The International Registry of Potentially Toxic Chemicals advises "Recycling; Precipitation, Solidification, Landfill. Sort, classify and put in a box properly labelled. Salvage profitably for reuse by local shop or sell as scrap metal. Nickel antimonide, nickel arsenide, nickel selenide – encapsulation followed by disposal in a chemical waste landfill. However, nickel from various industrial wastes may also be recovered and recycled. Insoluble nickel compounds may be landfilled. Soluble nickel compounds should be treated to precipitate an insoluble nickel compound, solidified and landfilled".

CURRENT REGULATIONS, GUIDELINES, AND STANDARDS

EXPOSURE LIMIT VALUES

Medium	Specification	Country/ organization	Exposure limit description[a]	Value	Effective date
AIR	Occupational	Australia	Threshold limit value (TLV)		1985 (r)
			- Time-weighted average (TWA) Metallic nickel	1.0 mg/m^3	
			- Time-weighted average (TWA) Soluble nickel compounds	0.1 mg/m^3 (as Ni)	
			- Short-term exposure limit (STEL) Soluble nickel compounds	0.3 mg/m^3 (as Ni)	
		Belgium	Threshold limit value (TLV)		1989 (r)
			- Time-weighted average (TWA) (metal)	1 mg/m^3	
			- Time-weighted average (TWA) (soluble compounds)	0.1 mg/m^3 (as Ni)	
		Bulgaria	Maximum permissible concentration (MPC)	0.5 mg/m^3	1985 (r)
		Canada	Threshold limit value (TLV)		1980
			- Time-weighted average (TWA)	1 mg/m^3	
			- Time-weighted average (TWA) (soluble compounds as Ni)	0.1 mg/m^3	

		Czechoslovakia	Maximum allowable concentration (MAC) - Time-weighted average (TWA) - Ceiling value (CLV) (calculated as Ni) (applies to nickel and its compounds)	 0.05 mg/m^3 0.25 mg/m^3	1985
AIR	Occupational	Germany, Federal Republic of	No maximum allowable concentration (MAK) value established (applies to dusts/aerosols from nickel metal, nickel sulfide and sulfidic ores, nickel oxide and nickel carbonate arising in production and processing)	carcinogenic working material	1989 (r)
			Technical reference concentration (TRK) (nickel and compounds except nickel carbonyl) - Time-weighted average (TWA)	 0.5 mg/m^3 (as Ni) respirable dusts and fumers, except dusts from nickel alloys (calculated as nickel in total inhalable dust) 0.05 mg/m^3 (as Ni) respirable droplets (calculated as Ni sensitization in entire respirable portion)	
		Japan	Maximum allowable concentration (MAC) - Time-weighted average (TWA) working material)	 1 mg/m^3 (carcinogenic)	1988 (r)

CURRENT REGULATIONS, GUIDELINES, AND STANDARDS

EXPOSURE LIMIT VALUES

Medium	Specification	Country/ organization	Exposure limit description[a]	Value	Effective date
AIR	Occupational	Netherlands	Maximum limit (MXL)		1987 (r)
			- Time-weighted average (TWA), metal	1 mg/m^3	
			- Time-weighted average (TWA), (water soluble nickel compounds)	0.1 mg/m^3 (as Ni)	
		Romania	Maximum permissible concentration (MPC)	Nickel salts as hydroaerosols	1985 (r)
			- Time-weighted average (TWA) (as Ni)	0.5 mg/m^3	
			- Ceiling value (CLV) (as Ni)	1.5 mg/m^3	
		Sweden	Threshold limit value (TLV)		
			- Time-weighted average (TWA), 1 day metallic nickel	0.5 mg/m^3	1988
			- Time-weighted average (TWA), 1 day (soluble nickel compounds, nickel oxide and nickel carbonate)	0.1 mg/m^3 (as Ni) carcinogenic sensitization	
		Switzerland	Maximum work-site concentration (MAK)		1985 (r)
			- Time-weighted average (TWA) metal dust, sulfide, oxide and carbonate	0.5 mg/m^3 (as Ni)	

Switzerland	- Time-weighted average (TWA) dust of water soluble nickel compounds	0.05 mg/m^3 (as Ni) sensitizer carcinogenic
United Kingdom	Recommended limit (RECL)	1987 (r)
	- Time-weighted average (TWA) elemental nickel	1 mg/m^3
	- Time-weighted average (TWA) soluble nickel compounds insoluble nickel compounds	0.1 mg/m^3 (as Ni) 1.0 mg/m^3 (as Ni)
	- Short-term exposure limit (STEL), (10-min time-weighted average) soluble nickel compounds insoluble nickel compounds	3 mg/m^3 (as Ni) 0.3 mg/m^3 (as Ni)
USA (ACGIH)	Threshold limit value (TLV)	1987
	- Time-weighted average (TWA), metal	1 mg/m^3 (as Ni)
	- Time-weighted average (TWA), soluble compounds	0.1 mg/m^3 (as Ni)
USA (OSHA)	Permissible exposure limit (PEL)	1987 (r)
	- Time-weighted average (TWA), metal and soluble compounds	1 mg/m^3 (as Ni)
USSR	Maximum allowable concentration (MAC)	1989
	- Ceiling value (CLV), metal, oxides, sulfide and mixtures of these compounds	0.05 mg/m^3 (as Ni)
	- Ceiling value (CLV), nickel salts, aerosols	0.005 mg/m^3 (as Ni)

CURRENT REGULATIONS, GUIDELINES, AND STANDARDS

EXPOSURE LIMIT VALUES

Medium	Specification	Country/ organization	Exposure limit description[a]	Value	Effective date
AIR	Ambient	USSR	Maximum allowable concentration (MAC) Metallic nickel (daily average) Water soluble nickel salts, (daily average)	 0.001 mg/m^3 (as Ni) 0.0002 mg/m^3 (as Ni)	1984
WATER	Surface	Czechoslovakia	Maximum allowable concentration (MAC) Nickel and its compounds	 0.1 mg/litre	1975
		USSR	Maximum allowable concentration (MAC) Nickel and its inorganic compounds - Surface water for fishing Nickel and its compounds	 0.1 mg/litre (as Ni) 0.01 mg/litre (as Ni)	1983 1982 (r)
WATER	Drinking	European Economic Community	Maximum allowable concentration (MAC)	0.05 mg/litre	1982
		United Kingdom	Maximum residue limit (MRL)	0.05 mg/litre	1985

Water	Drinking	USA (EPA)	10-day health advisory (HA) - child 10-day health advisory (HA) - adult Acceptable daily intake (ADI)	1.0 mg/litre 3.5 mg/litre 0.35 mg/litre	1985
SOIL	Agricultural	European Economic Community	Maximum limit (MXL) dry matter in soil sample with pH = 6	30-75 mg/kg	1989 (r)
SOIL	General	USSR	Maximum allowable concentration (MAC) mobile forms of nickel extractable by ammonium acetate buffer solution pH = 4.6	4 mg/kg	1985
SEWAGE SLUDGE	Agriculture	European Economic Community	Maximum limit (MXL) dry matter Annual limit value (10 year average)	300-400 mg/kg 3 kg/ha/yr	1989
FOOD		CMEA	Maximum permissible concentration (MPC) Nickel and its compounds (as Ni) - Milk products - Meat products - Fish products (as Ni) - Cereals - Vegetables, fruits - Other products - Beverages	 0.1 mg/kg 0.5 mg/kg 0.5 mg/kg 3.0 mg/kg 0.5 mg/kg 0.2-8.0 mg/kg 0.3 mg/kg	1983

CURRENT REGULATIONS, GUIDELINES, AND STANDARDS

EXPOSURE LIMIT VALUES

Medium	Specification	Country/ organization	Exposure limit description[a]	Value	Effective date
		Czechoslovakia	Maximum permissible concentration (MPC) Nickel and its compounds (due to production, packing, transport and storage of food products)		1986
			- Specified food products	0.1-8.0 mg/kg	
			- Beverages, general	0.3 mg/kg	
		USSR	Maximum permissible concentration (MPC) Nickel and its compounds (as Ni)		1981
			- Fish products	0.5 mg/kg	
			- Meat products	0.5 mg/kg	
			- Milk products (as Ni)	0.1 mg/kg	
			- Cereals	0.5 mg/kg	
			- Vegetables, fruits	0.5 mg/kg	
			- Beverages	0.3 mg/kg	

[a] TWA = time-weighted average over one working day (usually 8 h).

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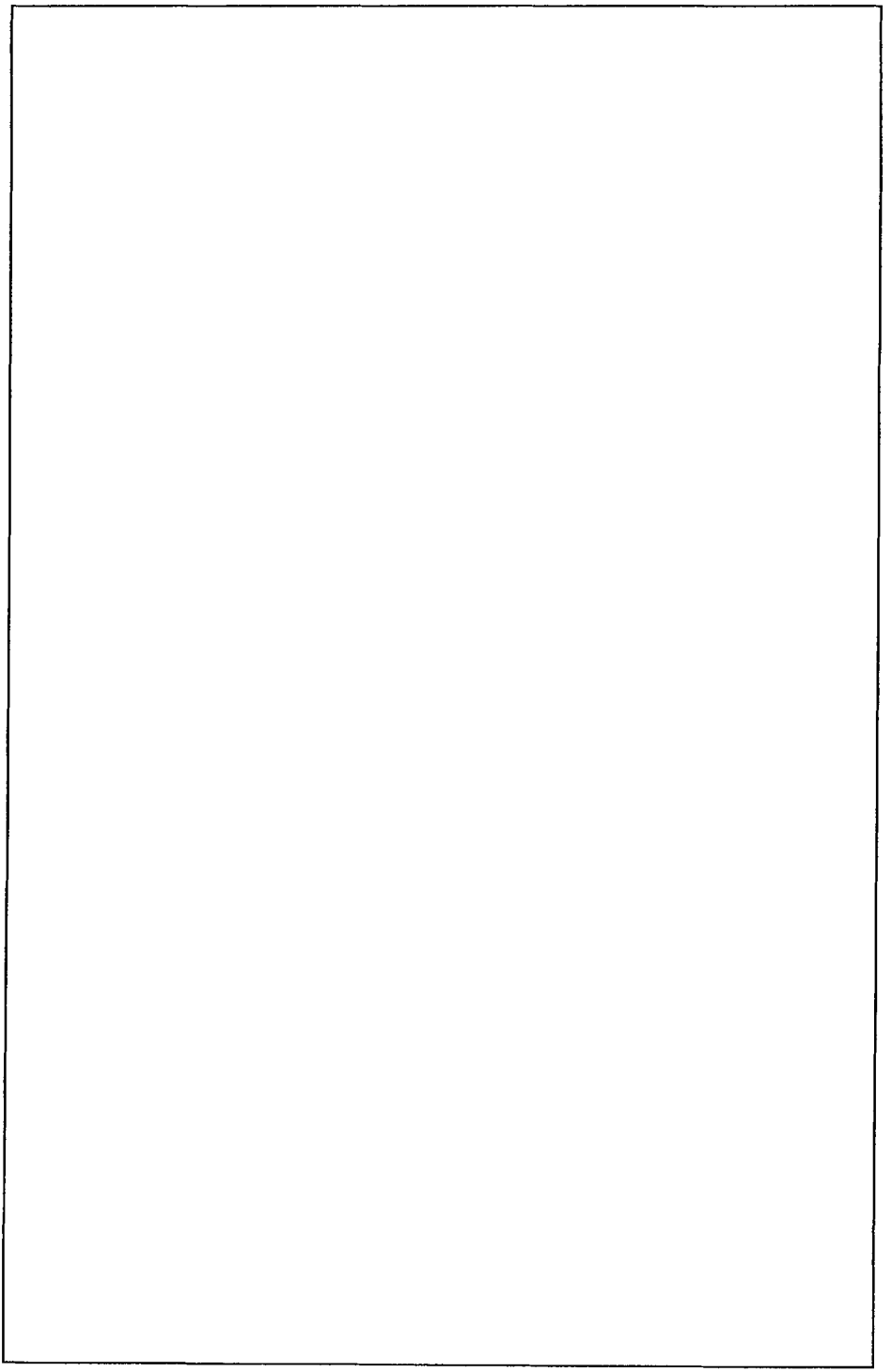

Price: Sw. fr. 5.–
Price in developing countries: Sw. fr. 3.50

ISBN 92 4 151062 5