EVALUATION OF WORKER EXPOSURES TO NICKEL DURING THE PLATING, MACHINING, AND FACRICATION OF NICKEL PLATED METAL.

By
Jeff Keyes

A Research Paper
Submitted in Partial Fulfillment of the
Requirements for the Master of Science Degree
With a Major in

Risk Control Management

Approved: 3 Semester Credits

_____

Investigation Advisor

The Graduate College
University of Wisconsin-Stout

May 10, 2001

The Graduate College
University of Wisconsin - Stout
Menomonie, Wisconsin 54751

Abstract

(Writer) (Last Name)	(First)	(Middle)
Keyes	Jeffrey	Jon

Evaluation of Worker Exposures to Nickel During the Plating,
Machining, and Fabricating of Nickel Plated Metal
(Title)

(Major)	(Research Advisor)	(Month/Year)	(Pages)
Risk Control Management	Dr. Gene Ruenger	07/01	47

APA
(Name of Style Used in this Study)

The goal of this study was to assist a company involved in nickel plating, machining, and fabricating of nickel plated metal, in evaluating and reducing its employee exposure to nickel concentrations in the affected work areas. Monitoring was conducted to determine nickel concentrations experienced by operators in the plating, grinding, and laser welding areas. Operators in the laser welding area seemed to experience the greatest adverse effects of nickel

exposure; therefore, follow-up sampling was performed in this area. Sampling results revealed one sample of exposure levels in excess of the current American Conference of Governmental Industrial Hygienists (ACGIH) published Threshold Limit Value (TLV) of 1mg/m^3. Nickel, however, is on the 1996-1997 "Notice of Intended Changes" list. The new proposed limit for nickel is 0.05 mg/m^3. At this level seven of the samples would exceed the proposed TLV. Recommendations to achieve acceptable levels of the contaminant in the work areas were based on the sampling results and a detailed analysis of the literature reviewed.

TABLE OF CONTENTS

Contents continued Page

Contents continued .....Page

LIST OF TABLES

LIST OF FIGURES

1.0 Introduction

It is estimated that approximately two percent of the work force in the nickel producing and using industries may be exposed to airborne nickel at concentrations near 1 mg/m^3 (Snow and Costa, 1992). The National Institute of Occupational Safety and Health (NIOSH) estimates that 250,000 workers in the United States may be occupationally exposed to nickel and its compounds. Nickel is not just limited to occupational exposures. The general population can be exposed through the air that we breathe, in our diet through food and water, and from various consumer products such as metallic cooking utensils. An average person's diet would yield a nickel intake range of 100-800 *u*g/day. Smokers can expect an additional intake of 2 to 23 *u*g/day.

Occupational exposure to nickel can be evaluated by air sampling or by biological monitoring. Air sampling involves collecting the contaminant metal on a mixed cellulose easter filter. Biological monitoring includes measuring the nickel concentrations in urine and in fingernail tissue.

A review of toxicology data and epidemiology studies for nickel has prompted the American Conference of Governmental Industrial Hygienists (ACGIH) to place nickel on their 1995 - 1996 "Notice of Intended Changes" list. The proposed change will reduce the 8-hour Threshold Limit Value (TLV)

from 1.0 mg/m^3 for insoluble nickel material and 0.1 mg/m^3 for soluble nickel compounds to 0.05 mg/m^3 for all forms of nickel. In addition to the TLV change, nickel will also carry the "A1 Confirmed Human Carcinogen" designation. This change should help companies that use nickel materials recognize the health hazards associated with nickel.

1.1 Purpose of the Study

The purpose of this study is to evaluate worker exposure to nickel while performing nickel plating, and machining and fabrication of nickel-plated metal.

1.2 Objectives of the Study

1. To measure air concentrations of nickel in the breathing zones of workers in the plating, grinding, and welding work areas.
2. To assess employee exposure data and to select necessary personal protective equipment and appropriate engineering controls.

1.3 Limitations of the Study

1. Information presented in this paper.
2. Changes in the processes occurred during the study.
3. This study evaluated one facility. Conclusions derived from this study may not be applicable. The confidentiality requirements of the company limited the process to other processes utilizing nickel.
4. Exposure concentrations were calculated using air-sampling data performed during portions of the scheduled work period.
5. Sampling was performed during a limited number of days. Day to day variations of air concentrations of nickel compounds would be expected to have a lognormal distribution.

2.0 Review of Literature

2.1 Introduction

Nickel is a ubiquitous trace metal, occurring naturally in soil, water, and air. Nickel is mined from sulfide or oxide ores and refined using pyro- and hydrometallurgical methods (World Health Organization, 1991).

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 sulfate and nickel chloride is used in electroplating and as catalysts in the manufacture of chemicals and petroleum. In 1987, approximately 39% of the nickel was used in stainless and alloy steel production, while 22% was used in electroplating processes (US Department of Health and Human Services, 1994). Other important applications include the use of nickel compounds in batteries, electronic and computer equipment, and as constituents of pigments in the glass and ceramics industries (World Health Organization, 1991).

2.2 Health Effects

Human health hazards from exposures to nickel and its compounds fall into three major categories: (a) allergies, (b) rhinitis and sinusitis, and (c) cancers of the nasal cavities, lungs, and oral cavities (Sunderman, 1988). The amount of nickel released from metal objects varies with atmospheric conditions such as temperature, humidity, pressure, and the presence of acidic substances (Vein, 1994). Nickel can enter the body via inhalation, oral ingestion, or percutaneously, with the absorptivity related to the solubility of the compound.

Nickel is essential to maintain health in animals. Dogs fed a diet containing zero amounts of nickel metal experienced a suppressed weight gain and lower food consumption than control animals (Ottolenghi, 1974). Although a lack of nickel has not been found to affect the health of humans, nutritionists believe a small amount of nickel is probably essential for humans (Wheeler, 1995).

2.2.1 Allergic Reactions

The most common adverse health effect of nickel in humans is an allergic reaction to nickel. People can become sensitive to nickel when jewelry or other items containing nickel are in direct contact with the skin. Wearing pierced earrings containing nickel may cause a sensitized person to have a reaction (Widstrom, 1985). Once a person is sensitized to nickel, further contact with the metal will produce a reaction. The most common reaction is a skin rash at the site of contact. In some sensitized people, dermatitis may develop at a site away from the site of contact (Wheeler, 1995). Hand eczema is fairly common among people sensitized to nickel.

In rare instances, exposure to nickel can also induce asthma attacks (Sunderman, 1984). Sensitive people have reactions when nickel is in contact with the skin, additionally some sensitized individuals react when they eat nickel in food or water, or breathe dust containing nickel. More women are sensitive to

nickel than men (Wheeler, 1995). This difference between men and women is thought to be a result of greater exposure of women to nickel through jewelry and other metal items.

2.2.2 Inhalation Exposure

2.2.2.1 Respiratory Effects

The respiratory system is the primary target of nickel toxicity following inhalation exposure. Inhalation of nickel particles has been shown to adversely affect the lungs and respiratory tract in the forms of cancer, reduced lung function and respiratory diseases by acting as a casual agent of various forms of cancer and respiratory diseases and by limiting lung function.

2.2.2.1.1 Respiratory Effects in Test Animals

Benson and Dunnick (1989) evaluated the respiratory effects of nickel exposure. The researchers exposed rats and mice for 6 hours/day, 5 days/week for 16 days. Chronic active inflammation in the lungs, fibrosis, macrophage hyperplasia, and increased lung weight required exposures to >0.05 mg nickel/m^3 for nickel sulfate, > 0.11 mg/m^3 for nickel subsulfide and > 3.9 mg/m^3 for nickel oxide. Muscular atrophy of the nose was also reported following exposure to nickel sulfate and nickel subsulfide but not nickel oxide. Rats appeared to be

more sensitive to the respiratory effects of nickel than mice. The toxicity appeared to depend on the solubility of the compounds more than the lung burden. The compounds have the following toxicity ranking nickel sulfate> nickel subsulfide> nickel oxide (Wheeler, 1995). The toxicity data is based on a NOAEL (no observed adverse effect level) of 0.02 mg/ nickel/m^3 for rats exposed to nickel sulfate for 13 weeks (Dunnick, 1989).

Ottolenhi (1974) reported an increase in pneumonitis, atelectasis, bronchitis, and emphysema in rats exposed to nickel subsulfide at 0.7 mg nickel/ m^3 for 78 weeks. The exposures were followed by a 30-week observation period.

Pneumoconiosis was observed in all exposed hamsters following lifetime exposure to 42 mg nickel/m^3 of nickel oxide alone or in combination with cigarette smoke (Wehner, 1986). The pneumoconiosis increased in severity as a function of exposure time and age. Emphysema was observed in the animals that died before developing pneumoconiosis.

2.2.2.1.2 Respiratory Effects in Humans

An increased incidence in deaths from respiratory disease was found in workers chronically exposed to > 0.04 mg nickel /m^3, usually as nickel oxide or metallic nickel (Cornell and Landis, 1984). The respiratory effects in the workers included chronic bronchitis, emphysema, and reduced vital capacity. The workers were also exposed to a variety of other metals including uranium, iron, lead, and

chromium; so it cannot be concluded that nickel was the sole causative agent. Other studies of workers exposed to nickel aerosols performed by Cox (1984), Crege (1984), and Redmond (1984) did not show an increase in the incidence of deaths from respiratory disease.

Donovich (1984) and Novey (1983) have documented asthma induced by occupational exposure to nickel. Shirakawa (1990) reported that a worker who had apparently developed cutaneous sensitization from inhalation of nickel sulfate had also developed asthma. Asthma can result from either primary irritation or from an allergic response (Novey, 1983).

Pneumoconiosis has been reported among workers exposed to nickel dust, but exposure to known fibrogenic substances could not be excluded (Cox, 1984). Other reported respiratory effects resulting from exposure to nickel aerosols include nasal irritation, damage to the nasal mucosa, perforation of the nasal septum, and loss of smell (Vieboer, 1992).

2.2.2.2 Body Weight Effects

Exposure to nickel aerosol concentrations greater than 0.4 mg/m^3 of nickel has been associated with decreased body weight in animals. A decrease in body weight (20-30%) was observed in rats intermittently exposed (6 hours/day, 5 days/week) to nickel subsulfide at 0.7 mg nickel/m^3 for 78 weeks (Ottolenghi, 1974)

2.2.2.3 Reproductive Effects

Testicular degeneration was observed in rats and mice exposed to nickel sulfate (greater than 1.6 mg nickel/m^3) 6 hours/day for 12 days over a 16 day period (Benson, 1988). The author indicated that the testicular lesions were probably the result of emaciation rather than a direct effect of nickel.

2.2.2.4 Cancer

2.2.2.4.1 Animal Studies

Ottolanghi (1974) concluded that chronic (6 hours/day, 5 days/week 78 weeks) exposure to nickel subsulfide resulted in an increase in lung tumors in rats exposed to 0.7 mg nickel/m^3. Short term exposure up to 6.3 mg nickel oxide/m^3 for 6 hours/day, 5 days/week for 1 month resulted in no significant increase in lung cancer up to 20 months after exposure (Horie, 1985).

2.2.2.4.2 Epidemiology Studies

Epidemiology studies of workers exposed to nickel have demonstrated a correlation between nickel exposure and lung and nasal cancer (Doll, 1977 and Chovi, 1981). Wheeler (1995), however, reported that all studies of nickel exposed workers are confounded by exposure to relatively high concentrations of other metals, including suspected carcinogens such as chromium.

One study of 1,916 refinery workers reported an observed to an expected death ratio of 7:1 for lung cancer and 40:1 for nasal cancer (Wheeler 1995). Higher concentrations of nickel can be found in the nasal mucosa of active and retired workers than in unexposed controls (Torjussen and Anderson 1979). The nickel was cleared from the nasal mucosa with an estimated half-life of 3.5 years.

The latency period for the lung cancers appears to be shorter than for nasal cancer. In a cohort study of 2,247 refinery workers, an excess of lung cancer was found by 3-14 years after first employment, while an increase in nasal cancer was not found until 15-24 years after first employment (Magnus 1982). In a reanalysis of the described studies by the International Committee on Nickel Carcinogenesis in Man (1990), significant increases were reported for lung and nasal cancers for workers exposed to soluble nickel compounds at concentrations >1 mg/m^3 and insoluble nickel compounds at concentrations >10 mg nickel/m^3.

The World Health Organization (1991) reported no association between inhalation of metallic nickel and lung and nasal cancer risks. They also indicated that no substantial evidence was obtained to suggest that occupational exposure to nickel or any of its compounds was likely to produce cancers other than in the lung or nose. The International Committee also reported a similar conclusion on Nickel Carcinogenesis in Man in 1990.

2.2.2.5 Death

Benson and Dunnick (1988) found that intermittent inhalation to 1.6 mg nickel/m^3 as nickel subsulfide for 12 days resulted in death for all the mice, while only 2 of 10 rats exposed to 6.7 mg/m^3 died. Neither species died following exposure to 23.6-mg nickel/m^3 as nickel oxide.

A significant decrease in mean survival time was reported by Takenakas (1985) in a study of rats exposed to nickel oxide for 23 hours/day for life at concentrations of 0.06 mg nickel/m^3 . The inhalation exposure data is summarized in table 2.1.

Table 2.1 Levels of Significant Exposure to Nickel - Inhalation

Species	Duration	Level	Result	Form
Rat[1]	12 d 5/d wk 6hr/d	6.7 mg/m^3	2/10 died	sulfate
Mouse[1]	12 d 5d/wk 6hr/d	1.6 mg/m^3	10/10 died	sulfate
Rat & mouse[1]	12 d 5d/wk 6hr/d	23.6 mg/m^3	0/20 died	oxide

Rats[2]	23hr/d life	.06 mg/m^3	all died ave 87.7 wks	oxide
Rat[2]	23hr/d life	0 mg/m^3	all died ave 125.2 wks	oxide

1. Dunnick and Benson, 1988
2. Takenaka, 1985

The average survival time for animals exposed to 0 and 0.06 mg/m3 of nickel oxide was 125.2 and 87.7 respectfully.

2.2.3 Oral Exposure

People who are not sensitive to nickel must eat very large amounts of nickel to suffer adverse health effects (Wheeler, 1995).

2.2.3.1 Respiratory Effects

Pneuomonitis was observed in rats treated for 91 days by gavage with 8.6 mg nickel/kg/day of nickel chloride (American Biogenics Corporation, 1988). Emphysema, bronchiolectasis, and cholesterol granulomas were observed in dogs exposed to 62.5 mg nickel/kg/day of nickel sulfate in the diet for 2 years, but not in rats exposed at up to 188 mg/kg/day for 2 years (Ambrose, 1976).

2.2.3.2 Gastrointestinal Effects

Symptoms of gastrointestinal distress were reported by workers who drank water from a water fountain contaminated with nickel sulfate and nickel chloride (Sunderman, 1988). Of the 35 workers exposed, 20 reported symptoms and 10 were hospitalized. The workers who reported symptoms were exposed to an estimated dose of 7.1 - 35.7 mg/nickel/kg. The symptoms included nausea (15 workers), abdominal cramps (14 workers), diarrhea (4 workers), and vomiting (3 workers).

Sunderman (1993) reported that workers who accidentally drank light green water containing 250 ppm of nickel (metal) from a contaminated drinking fountain had stomach aches and suffered adverse effects to the blood (increased red blood cells) and kidneys (increased protein in urine).

Ambrose (1976) reported no gastrointestinal effects in rats that received dietary supplements of nickel sulfate for two years at the level of 188 mg nickel/kg/day. He also noted that during the first three days of a 2-year study the dogs vomited following treatment with nickel sulfamate in the diet at 62.5 mg nickel/kg/day.

2.2.3.3 Dermal Effects

Several studies indicate that a single oral dose of nickel given as nickel sulfate can result in a flare up in the dermatitis in nickel sensitive individuals (Burrows, 1981; Moller, 1975; Cronin, 1980; Kaaber, 1978; Veien, 1987). The lowest single dose resulting in arrhythmia on the body, worsening of hand eczema, and a flare-up at the patch test site, was 0.009 mg nickel/kg/day (Cronin 1980).

Intermediate-duration studies suggest that longer-term oral exposure can be tolerated by some nickel sensitive individuals and may even serve to desensitize some individuals. Jordan and King (1979) found that only 1 of 10 nickel-sensitive women exhibited a dermal flare-up to a patch-test challenge of nickel sulfide after being given nickel sulfate at 0.007 mg/kg/day for two weeks.

Nickel sulfate has also been administered orally as a treatment for eczema. After one month, clinical improvement in hand eczema was observed in eight women who were given increasing daily doses of nickel (0.01 - 0.03 mg/kg/day) as nickel sulfate for 178 days (Santucci 1994). Continued treatment resulted in the healing of all dermal lesions except those on the hands.

An oral exposure treatment before the sensitizing exposure may also help prevent nickel sensitization in some individuals. A study examining the relationship between ear piercing and orthodontic treatment found that nickel sensitivity was reduced when orthodontic treatment preceded ear piercing (van Hoogstraten 1991). The investigators hypothesized that the oral nickel exposure

that occurred during orthodontic treatment helped prevent the sensitization that occurred following ear piercing with earrings containing nickel. Orthodontic treatment after ear piercing did not effect the risk of nickel sensitization. Further evidence that oral exposure to nickel before a sensitizing exposure can prevent hypersensitivity is provided by the observation that nickel sensitivity in mice could be consistently produced only when metal frames to cover the cages and metal water nipples that released nickel were replaced with glass covers and nipples free of nickel (van Hoogstraten, 1991).

2.2.3.4 Reproductive Effects

Nickel salts have been demonstrated to have reproductive effects in animals. An increase in spermatozoa abnormalities was observed in mice treated orally with a single dose of nickel sulfate (28 mg nickel/kg) or nickel chloride (43 mg nickel/kg) (Sobti and Gill, 1989). Ambrose (1976) found a dose - related increase in the number of stillborn pups in a study in which rats were fed nickel chloride in the diet at 0, 22.5, 45, or 90 mg/kg/day. The number of offspring weaned also decreased with increasing doses of nickel. An increase in the number of spontaneous abortions was observed in mice treated on gestation days 2-17 with nickel chloride in the drinking water at 160 mg/kg/day (Berman and Rehnberg, 1983).

2.2.3.5 Cancer

Ingested nickel has not been demonstrated to increase the frequency of cancer. Lifetime drinking water studies using rats and mice found nickel acetate (0.6 mg/kg/day rats; 0.95 mg/kg/day mice) to be non-carcinogenic (Schroeder, 1974).

2.2.3.6 Death

One human death following oral exposure to nickel was reported (Dalrup, 1983). Nickel sulfate crystals (rough estimate of 570 mg nickel/kg) were accidentally ingested by a 2 year old child. Four hours after ingestion, cardiac arrest occurred, and the child died 8 hours after exposure.

Single-dose oral lethality studies indicate that soluble nickel compounds are more toxic than insoluble nickel compounds, table 2.2.

Table 2.2 Single-dose Oral Lethality

Compound	LD_{50} (mg nickel/kg)	Solubility
Nickel sulfate (Mastromatteo, 1986)	39 (rat)	soluble
Nickel acetate (Haro, 1968)	116 (rat) 136 (mouse)	soluble
Nickel oxide (Mastromatteo, 1986)	>3,930 (rat)	insoluble

In a study in which rats were administered nickel chloride in the drinking water, the death of female rats from pregnancy complications at the time of delivery suggests that females are more susceptible to nickel toxicity during parturition (RTI, 1988).

2.2.4 Dermal Exposure

2.2.4.1 Respiratory Effects

McConnell (1973) reported on a possible link between skin contact and asthmatic responses. A patient, who was diagnosed with nickel-related asthma, exhibited severe respiratory distress when challenged with nickel sulfate in scratch and intradermal tests.

2.2.4.2 Dermal Effects

An allergy to nickel is the most frequent contact allergy in women (Sunderman, 1984). The author also stated that the sensitizing reaction may often be exposure to nickel in consumer products including jewelry, rather than occupational exposures.

Widstrom (1985) reported an association between ear piercing and nickel sensitivity. In a study of 960 girls ages 8, 11, 15, the prevalence of nickel allergy

was 9% for girls with pierced ears compared to 1% for girls without pierced ears. Girls with more than one hole in each ear were more likely to be sensitive to nickel than girls with only one hole in each ear (19% vs. 9%).

Once an individual is sensitized, even minimal contact with nickel may cause a reaction. Cronin (1980) reported that the lowest single dose resulting in dermatitis at the patch test site was 0.009 mg nickel sulfate/kg/day. Sensitivity to nickel appears to remain for many years. Fourteen out of 14 people who tested positive for nickel sensitivity using nickel sulfate also tested positive 10 years later (Keczkes 1982).

Patch test studies in sensitive individuals using nickel sulfate have shown a dose-response relationship between the amount of nickel and the severity of the test response (Marks 1990). In the study of 12 individuals, a nickel concentration of 316 ppm in petrolatum resulted in dermatitis, while a concentration of 100 ppm did not produce adverse effects. Allenby (1993) concluded that 0.5 ppm was the minimal amount of nickel needed to cause an allergic reaction.

Adults with hand eczema have a higher incidence of allergy to nickel (Allenby, 1993). It is estimated that approximately 40% of nickel allergic women are also affected by hand eczema (Linden, 1994). This suggests those persons who experience eczema on the hands may also be prone to allergic responses from nickel.

A common allergic reaction is called “nickel itch”. This reaction is dermatitis resulting from sensitization to nickel. The first symptom is usually pruritis (severe itching), which occurs up to 7 days before skin eruption occurs (Sunderman, 1989). The study also states that the primary skin eruption is erythematous or follicular; and it may be followed by superficial discrete ulcers, which discharge and become crusted or by eczema. The eruptions may spread to areas related to the activity of the primary site, such as the elbow flexure, eyelids, or sides of the neck and face. In chronic stages pigmented or depigmented plaques may be formed.

2.2.4.3 Cancer

No studies were located regarding cancer in humans or animals after dermal exposure to nickel.

2.3 Toxicokinetics

Following inhalation exposure, about 20-35% of nickel deposited in the lungs of humans is absorbed into the blood stream. Absorption from the respiratory tract is dependent on the solubility of the nickel compound, with higher urinary nickel observed in workers exposed to soluble nickel compounds than in those exposed to insoluble nickel compounds (Wheeler, 1995). Following oral exposure, about 27% of nickel metal given to humans in drinking water is

absorbed, while only about 1% was absorbed when nickel metal was given with food (Linden, 1994). Nickel applied directly on the skin can be absorbed into the skin where it may remain rather than entering the blood stream. Inhaled nickel tends to accumulate in the lungs. Nickel has been shown to cross the placenta, and nickel can accumulate in milk resulting in exposure to offspring (Wheeler, 1995).

2.3.1 Absorption

2.3.1.1 Inhalation Exposure

Inhaled nickel particles are deposited in the upper and lower respiratory tract. Particle size greatly affects where the particles are deposited. Gordon and Amdur (1991) reported that large particles (5-30 um) were deposited in the nasopharyngeal area, small particles (1-5 um) in the trachea and bronchiolar region, and minute particles (<1 um) in the alveolar region of lungs.

In humans, about 20-35% of the inhaled nickel that is retained in the lungs is absorbed into the blood (Sunderman, 1991). The rest is swallowed, expectorated, or remains imbedded in the respiratory tract. Nickel can be excreted in the urine with higher concentrations found in workers exposed to soluble nickel compounds as compared to those exposed to insoluble nickel compounds (Angerer and Lehnert, 1990). Torjussen (1979) indicates that soluble compounds were more readily absorbed from the respiratory tract.

2.3.1.2 Oral Exposure

Sunderman (1989) reported that 40 times more nickel was absorbed from the gastrointestinal tract when nickel sulfate was given to humans in the drinking water than when it was given in food. In a study performed by Christens and Lagenson (1981), only 4.3% of the given dose was absorbed in subjects who also were consuming food.

2.3.1.3 Dermal Exposure

Nickel has been shown to penetrate human skin. When a radioactive dose of nickel sulfate was applied to occluded skin, 55-77% was absorbed within 24 hours, with most being absorbed in the first few hours (Norgaard, 1955). It was not determined if the nickel was absorbed into the deep layers of skin or into the blood stream. Nickel absorption did not differ in nickel-sensitive individuals. In a study using excised human skin, only 0.23% of an applied dose of nickel chloride permeated skin after 144 hours when skin was not occluded, while 3.5 % permeated occluded skin (Fullerton, 1986).

2.4 Exposure Limits

The Occupational Safety and Health Administration (OSHA) permissible exposure limit for nickel and nickel compounds in the workplace is an 8-hour time-weighted average of 1 mg/m^3. Recently the Chemical Substance Threshold

Limit Value, TLV, committee of the American Conference of Governmental Industrial Hygienists, (ACGIH), reviewed toxicology data and epidemiology studies for nickel. The ACGIH determined that the current TLV of 1 milligram per cubic meter for nickel may not provide sufficient protection.

The TLV committee concluded that most forms of nickel have the ability to cause nasal and respiratory cancers in humans. Based on the committees recommendation, the ACGIH placed nickel on their "Notice of Intended Changes" list. The proposed change reduces the 8-hour nickel TLV to 0.05 mg/m^3 and adds the "A1 Confirmed Human Carcinogen" designation.

2.5 Monitoring of Nickel in Air

Two types of sampling can be done to detect nickel concentrations in the air; Personal samples, or Area samples.

A personal sample is often used because it provides the best information about the actual exposure received by the worker. OSHA requires personal samples when evaluating exposures to substances with Permissible Exposure Limits (PEL). The personal sample is collected by locating the sampling device within one foot of the workers breathing zone (nose and mouth). Usually the sampling device is attached to the workers collar or lapel.

Area samples are collected to provide information about contaminant concentrations within a given area or to provide exposure information about the

"maximum exposed worker". For workers who do not interact directly with the contaminant source, area samples can provide some information about background exposure levels. Because most workers move through many exposure zones within a typical day, area samples cannot be used to predict actual worker exposures without detailed time-motion studies. Area samples are an effective tool for evaluating engineering controls or supply supporting data for personal samples when the number of samples is very small.

2.6 Selection of Employees

The sampling done for personal exposures will be either "Maximum Risk Employee" or random selection. The "Maximum Risk Employee" sampling strategy is used to determine which employees have the highest potential for exposure. It is a useful strategy for the initial assessment process and usually the strategy employed for compliance sampling. If the "maximum risk" employee's exposure is below the exposure limit, then it is assumed that the exposures of other employees working in the same area or process will be acceptable. The sampling of the "maximum risk" employee can provide useful information about numerous processes for the lowest expenditure of resources. The disadvantage is that exposures to employees who are not sampled are unknown.

Random selection is another method in which exposed employees are selected at random. Focusing on the same employees builds biases into the

exposure evaluation based on work habits and other factors unique to the individual. The facility will also employ temporary or contract workers who may perform tasks differently from employees regularly assigned to a work area and this difference may affect their exposures.

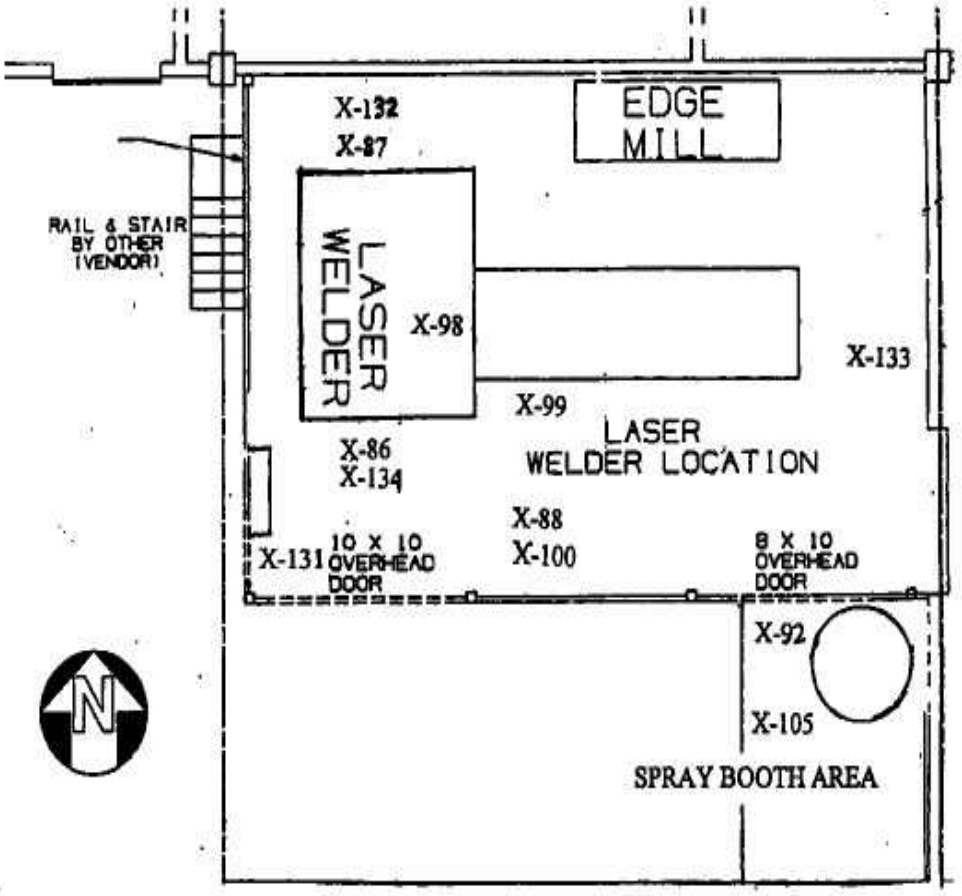
3.0 Methodology

3.1 Subject Selection

The test group consisted of 15 workers who were exposed to airborne nickel during normal job operations at a nickel plating and machining facility. A summary of the area and personal samples is presented in Table 3.1. Identity numbers in figures 3.1 to 3.3 indicates sampling positions in the facility.

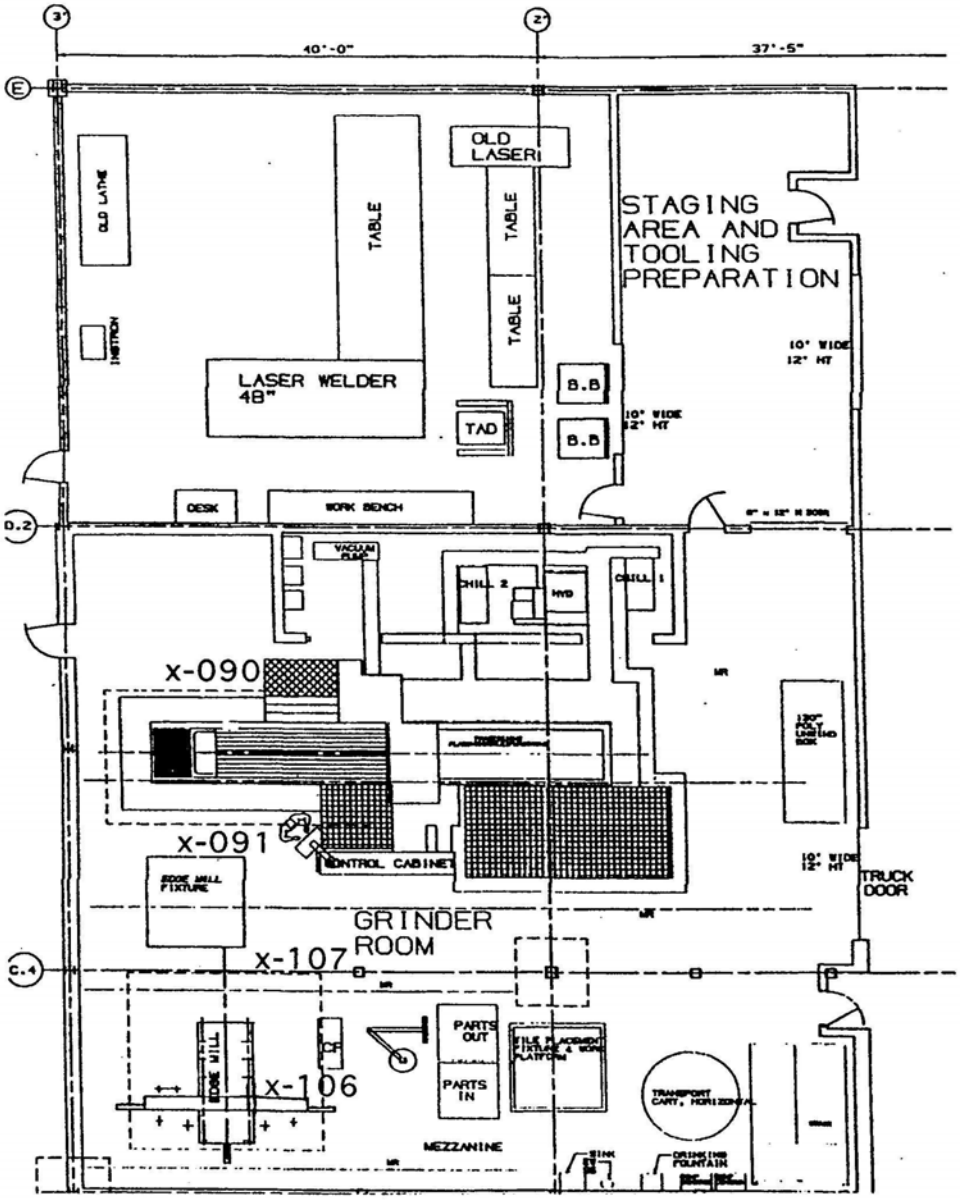
Table 3.1. Air Sampling August 8, 1995 through November 14, 1995

ID No.	Type of Sample	Description of Activities During Sampling
95 - 086	Area	Laser Welding - South end of Welder
95 - 087	Area	Laser Welding - North end of Welder
95 - 088	Personal	Operator - Laser Welding
95 - 090	Area	Surface Grinding - North side of Unit
95 - 091	Area	Surface Grinding - South end of Unit
95 - 092	Personal	Operator - Using Hand Grinder
95 - 094	Personal	Operator - Using Hand Grinder
95 - 098	Area	Laser Welding - Above Welder
95 - 099	Area	Laser Welding - Lower Control Panel
95 - 100	Personal	Operator - Laser Welding
95 - 104	Personal	Operator - Adding Nickel to Tanks
95 - 105	Personal	Pro.Engineer - Using Hand Grinder
95 - 106	Area	Grinder - Edge Milling
95 - 107	Personal	Grinding Operator - Edge Milling
95 - 161	Area	Above Plating Tank
95 - 162	Area	Above Plating Tank



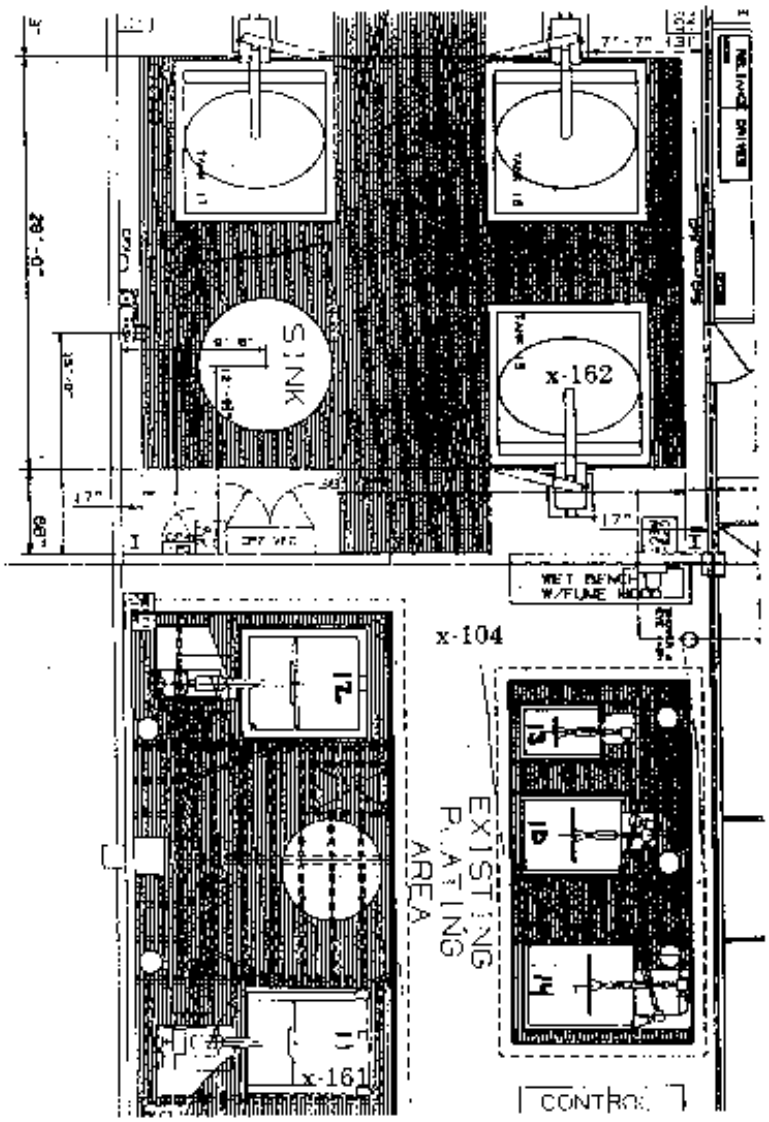
Facility Map and Sampling Locations

Figure 3.1



Facility Map and Sampling Locations

Figure 3.2



Facility Map and Sampling Locations

Figure 3.3

Three exposure groups were identified within the facility and a randomly selected individual from each group was monitored in addition to the area measurements. This strategy was based upon the concept that the area measurements would identify the maximum possible exposure while the personal samples would measure actual operator exposures. The exposure areas and the manufacturing tasks in each group are presented in Table 3.2.

Table 3.2. Exposure Groups Identified for August 8, 1995 through November 11, 1995 Monitoring

Exposure Group	Area	Tasks
#01	Plating Area	Plating, Edge Grinding, Shearing and Mixing
#02	Welding Area	Welding and Plate Prep.
#03	Grinding	Surface Grinding, Edge Milling and Plate Prep.

3.2 Devices and Analysis

The sampling was conducted according to OSHA Method 121 (SKC Inc., 1996). One blank filter cassette was included with every set of samples. Samples were stored and shipped at room temperature. Samples were analyzed following individual sampling experiments. During the experiments, the facilities air temperature and humidity were recorded. The data is presented in Appendix A-1.

An American Industrial Hygiene Association, accredited laboratory, performed all sample analysis. The samples were analyzed using the Inductively Coupled Plasma method specified by NIOSH Method 7300 and the OSHA Method #121.

Eight-hour time-weighted average (TWA) nickel concentrations were calculated for all samples. As sampling times varied for the tasks, air concentrations were estimated for unsampled times. Employees were assumed to have no exposures to nickel during times away from the work area. Air concentrations during unsampled work times were assumed to be the same as those measured. The nickel air sampling results are presented in Table 4.1.

3.2.1 Sample Media

Air samples were collected using 37mm filters in clear styrene acrylonitrile cassettes. The matched weight cassettes contain two filters that allow for gravimetric determinations. The first filter collects the contaminant while the bottom filter serves as the control. The cassettes are pre-weighed and marked with a serial number. The cassette identification number, sample date, area identification, and start times were recorded on the field data sheets prior to sampling. At the conclusion of the sampling the end sampling time was noted on the field forms.

3.2.2 Sampling Pump Calibration

All pumps were calibrated with a precision rotometer (secondary standard) prior to each sample collection, with the collection media inline, in the sampling train. Pumps were post-shift calibrated with the collection media in line, using the same calibration equipment. The pre-sampling and post-sampling flow rates were recorded on the field data sheets.

The rotometer is calibrated annually by the facility's Metrology department.

3.3 Follow-up Exposure Monitoring

Four follow-up samples were taken in the laser welding area on November 7th, 1995. The welder operator had experienced the greatest adverse health effects presumably coming from over-exposure to nickel. This testing was performed to examine the change in nickel fume concentrations resulting from the installation of additional local exhaust ventilation near the laser light beam.

Table 3.3. Air Sampling November 7, 1995

ID No.	Type of Sample	Description
95 - 131	Area	Laser Welding - On Desk
95 - 132	Area	Laser Welding - On Left Side
95 - 133	Area	Laser Welding - On Far Wall

95 - 134 Area Laser Welding - On Right Side

4.0 Results and Discussion

4.1 Air Concentrations of Nickel – Laser welding

Sampling was performed during the day shift (6:00 am - 6:00 pm) August 8, 1995 to November 11,1995. Laser welding only occurs during the day shift. The laser welder wears a dual cartridge respirator along with laser safety glasses, and vinyl gloves. Air concentrations of nickel ranged from 0.16 mg/m^3 to 0.81 mg/m^3 with a mean concentration of 0.5233 mg/m^3.

Based on the results from the lab, none of the samples exceed the current exposure limit. However, all the samples exceed the proposed limit of 0.05 mg/m^3.

4.1.1 Air Concentrations of Nickel – Grinding and Milling

Sampling was performed during the day shift only. The grinding and milling operations operate 24 hours a day. The operators performing these tasks wear safety eyewear. Air concentrations of nickel ranged from <0.004 mg/m^3 to 5.6 mg/m^3 with a mean concentration of 0.8097 mg/m^3 . One sample taken in the grinding area exceeds the current OSHA PEL of 1 mg/m^3. In comparison, one sample exceeds the ACGIH proposed limit of 0.05 mg/m^3.

4.1.2 Air Concentrations of Nickel – Plating

Three air samples were taken for the plating process. Only one sample yielded a detectable limit as noted by the analysis lab. A concentration of 0.007 mg/m^3 was detected while an operator added nickel pellet to the plating baths. Operators performing this task wear a neoprene-blended glove and an additional smock.

4.1.2 Follow-up Exposure Monitoring, November 7, 1995 – Laser Welding

Follow-up exposure monitoring conducted with the same sampling media as the previous samples revealed a range of 0.005 - 0.23 mg/m^3 with a mean concentration of .066 mg/m^3. None of the samples exceeded the current exposure limit, and only one sample exceeded the proposed limit of 0.05 mg/m^3.

4.2 Threshold Limit Value Adjustment

The operator is the laser welding area currently works a varied work schedule. Depending on the workload, the laser welder operator could work up to a 12-hour day. Therefore, a TLV adjustment will be identified to account for an increased time of uptake and a decreased time for elimination of the contaminant. According to the formulas in Chemical Properties Handbook (Yaws, 1993) the OSHA adjustment for a 12 hour workday would be a daily limit of 0.667 mg/m^3. Using the same reference, but the Breif and Scala model, the 12-hour daily limit falls to 0.5mg/m^3. If the employee

chose to work a 10-hour day then the exposure limits would be 0.8 mg/m^3 and 0.7 mg/m^3 respectively.

4.3 Potential Health Effects

4.3.1 Estimation of Risk from Inhalation Exposure

Inhalation of nickel particles has been shown to adversely affect the lungs and respiratory tract in the forms of; reduced lung function, respiratory diseases, and cancer. At exposures greater than 0.04 mg/m^3 to nickel oxide and or metallic nickel, research has shown increased incidences of respiratory diseases (Cornell and Landis, 1984). Thirty-five percent of the collected samples exceeded this level. All but one of these samples was collected in the laser welding area prior to follow-up monitoring. The other remaining sample occurred in the edge milling process. With out proper personal protective equipment, employees in the welding and milling operations are at risk of respiratory disease.

Exposures to low levels of nickel, 0.009 mg/m^3, have resulted in asthmatic responses in sensitized individuals. Responses can come from primary contact or from an allergic response. Any sensitized individuals in the work areas are at risk of an asthmatic reaction due to inhalation exposures.

4.3.2 Estimation of Risk from Dermal Exposure

Direct contact with nickel can cause an allergic reaction in sensitized individuals. Adults with known conditions of hand eczema have a higher

incidence of nickel allergy. Once individuals are sensitized even a small amount of nickel can initiate a response. It is estimated, based on the sampling results, that any sensitized individual with-in the work area would be subject to allergic responses from dermal exposures.

4.3.3 Estimation of Risk from Oral Exposure

There is only one reported death from ingestion of nickel. Wheeler (1995) reported that non-sensitive people must ingest very large amounts of nickel to suffer any adverse health effects. Oral exposure risks at this facility should only be a problem for sensitized individuals. Other oral contact symptoms would be estimated to be minimal due to the physical size of the nickel material.

4.3.4 Estimation of Risk of Cancer

Respiratory cancers are primarily related to exposures to soluble nickel compounds at >1 mg nickel / m^3 and to exposure to less soluble compounds at >10 mg nickel/m^3 (International Committee on Nickel Carcinogenisis in Man, (1990). Of the samples taken at this facility only one, 95-106, was above the 1mg/m^3. This result was associated the edge mill process. Therefore, unless the proper personal protective equipment or engineering controls are applied operators performing this task are at risk of cancer.

5.0 Conclusions

Air-borne concentrations were measured in three locations with personal and area sampling at a nickel using facility August 8, 1995 through November 14, 1995. Evaluation of exposure data indicates that this facility needs to employ engineering and administrative controls to reduce the workplace exposures.

The one activity that had an exposure which exceeded the ACGIH exposure limit of 1 mg/m^3 was in the grinding area where edge milling was performed. This activity, along with seven additional samples, had exposures that exceeded the proposed new limit for nickel, 0.05 mg/m^3. All of the exceeded samples came either from the welding or grinding areas.

5.1 Recommendations/Action Required

Based on the contaminate sampled, the company should engage in the following activities to provide for the well fare of exposed employees and also to comply with the new exposure limit.

5.1.1 Air Monitoring

A. Initial sampling should also include any clean up operations done at this facility performed by the operators or the maintenance staff temporarily assigned to the area. These employees may be exposed to unusually high concentrations of nickel during clean-up or other non-routine tasks.

B. Periodic monitoring for nickel should be performed in areas or on specific individuals known to be a or above the action level to establish a progressive basis of supportive data for discontinuance of monitoring.

C. A procedure of mechanism should be employed whereby any new process changes (new equipment, ventilation changes, etc...) where known

exposures of nickel would exist, be accompanied by a thorough set of sampling to accumulate additional exposure data. The data results need to be shared with affected personal and then filed appropriately.

5.1.2 Hazard Communication

A. Employees participating in the evaluations of nickel on August 8, 1995 through November 14, 1995 should be informed of the results of these tests within 15 days from the receipt of this report, according to CFR 1910.1048 (d)(6) Employee Notification of Monitoring Tests. This can be done conveniently in crew meetings, safety meetings and/or in writing, or what ever bests suits current practice at this facility.

B. Other employees who did not participate in the sampling, and whose job requirements involve the handling of nickel, should also be similarly informed.

C. Develop a process or procedure enabling new or transferred employees whose job tasks involve nickel handling to be informed about the nickel evaluations along with their usual hazard communication information.

5.1.3 Engineering Controls

This facility needs to explore engineering controls to reduce the affected employees' exposure to nickel. On the laser welder additional local exhaust

ventilation is needed as close to the source as possible. This will reduce the amount of air-borne contaminant as well as the nickel oxide fumes that settle on the surrounding equipment. These settling particles pose a threat to operators in the form of; an increased risk of dermal contact with nickel.

5.1.4 Personal Protective Equipment

Various forms of personal protective equipment (PPE) must be worn when performing certain job functions in the affected work areas. The correct PPE was selected for the individual work areas by the use of using vendor consultants and from the hazard assessments performed for the different job tasks. The appropriate PPE is presented in Table 5.1.

Table 5.1 Personal Protective Equipment for Job Tasks Involving Nickel

Laser Welding:

1. Respirator suited for welding such as the 3M 9920 when welding or assisting in the laser welding area.
2. Laser eye protection.
3. Vinyl gloves.
4. A work uniform that is changed daily.

Surface Grinder / Edge Milling

1. Neoprene gloves when handling coolant scrap.

2. Leather gloves when handling parts with burred edges.

Plating Area

1. Neoprene or Neoprene/Latex when working in wet areas.
2. Kevlar (cut-proof) inner gloves when handling part.
3. Leather gloves when shearing of handling parts.
4. A #9920 respirator when hand grinding parts.
5. Neoprene gloves when adding plating solution to tanks.
6. Neoprene gloves and an apron when cleaning Anode baskets.

Bibliography

Allenby, C.F., (1994). The Effect of Repeated Open Exposure to Low Levels of Nickel on Compromised Hand Skin of Nickel Allergic Subjects. Contact Dermatitis, 30:135-138.

Ambrose, A.M., (1976). Long Term Toxicological Assessment of Nickel in Rats and Dogs. Journal of Food Science and Technology 13:181-187.

American Biogenics Corporation, (1988). Ninety Day Gavage Study in Albino Rats Using Nickel. Research Triangle Institute and American Biogenics Corporation.

Angerer, J., Lehnert, G., (1990). Occupational Chronic Exposure to Metals. International Archives of Occupational Environmental Health 62:7-10.

Berman, E., Rehnberg, B., (1983). Fetotoxic Effects of Nickel in Drinking Water in Mice. EPA 600:1-83.

Benson, J.M., (1988). Comparative Inhalation Toxicity of Nickel Sulfate to F344/N Rats and B6C3F1 Mice Exposed for Twelve Days. Fundamentals of Applied Toxicology, 10:164-178.

Burrows, D., (1981). Nickel, Hands, and Hip Prosthesis. British Journal of Dermatology, 105:437-444.

Chovil, A., (1981). Respiratory Cancer in a Cohort of Nickel Sinter Plant Workers. British Journal of Industrial Medicine, 38:327-333.

Christiansen, O.B., Lagesson, V., (1981). Nickel Concentrations of Blood and Urine After Oral Administration. Annual Clinical Laboratory Science, 11:119-125.

Cornell, R.G., Landis, J.R., (1984). Mortality Patterns Among Nickel/Chromium Alloy Foundry Workers. International Agency for Research on Cancer, 53:87-53.

Cox, J.E., (1981). Mortality of Nickel Workers. British Journal of Industrial Medicine, 38:235-239.

Crege, J., (1984). Allergic Contact Dermatitis From Nickel with Unusual Localization. Contact Dermatitis, 27:330-331.

Cronin, E., (1980). Oral Challenge in Nickel-Sensitive Women with Hand Eczema. Academic Press, p.149-152.

Daldrup, T., (1983). Nickel Sulfate Intoxication. Toxicology, 25:289-292.

Doll, R., (1970). Cancers of the Nasal Sinuses in Nickel Workers. British Journal of Industrial Medicine, 34:102-105.
Dolovich, J., (1984). Occupational Asthma from Nickel Sensitivity. British Journal of Industrial Medicine, 41:51-55.

Dunnick, J.K., (1989). Lung Toxicity After 13-week Inhalation Exposure. Fundamentals of Applied Toxicology, 12:584-594.

Fullerton, A., (1986). Permeation of Nickel Salts Through Human Skin *in vitro.* Contact Dermatitis, 15:173-177.

Gordon,T., Amdur, M.O., (1991). Responses of the Respiratory System to Toxic Agents. Casarett and Doull's Toxicology. 4th ed. p.383-406.

Haro, R.T., (1968). Studies on the Acute Toxicity of Nickelocene. Science of the Total Environment, 54:29-30.

Horie, A., (1985). Electron Microscopy of Pulmonary Lesions Including Carcinoma, Induced by Inhalation Exposure of Rats to Nickel-oxide Carcinoma, Induced by Inhalation Exposure of Rats to Nickel Oxide Aerosol. Proceedings of the 3rd International Congress on Nickel Metabolism and Toxicology, p.41-44.

IARC, (1990). Monographs on the Evaluation of Carcinogenic Risks to Humans. World Health Organization, 49:257-445.

Jordan, W.P., King, S.E., (1979). Nickel Feeding in Nickel-sensitive Patients with Hand Eczema. Journal of American Academic Dermatology, 1:506-508.

Kaaber, K., (1978). Low Nickel Diet in the Treatment of Patients with Chronic Nickel Dermatitis. British Journal of Dermatology, 98:197-201.

Keczkes, K., (1982). The Persistence of Allergic Contact Sensitivity. British Journal of Dermatology, 107:461-465.

Linden, C., (1994). Occupational Contact Dermatitis Due to Nickel Allergy. Science of the Total Environment, 148:283-285.

Magnus, K., (1982). Cancer of Respiratory Organs Among Workers at a Nickel Refinery in Norway. International Journal of Cancer, 30:681-685.

Marks, R., Eun, H.C., (1990). Dose-response Relationships for Topically Applied Antigens. British Journal of Dermatology, 122;491-499.

Mastromatteo, E., (1986). Nickel. American Industrial Hygiene Association Journal, 47:589-601.

McConnell, L.H., (1973). Asthma Caused by Nickel Sensitivity. Annual Internal Medicine, 78:888-890.

Moller, H., (1984). Attempts to Induce Contact Allergy to Nickel in the Mouse. Contact Dermatitis, 10:65-68.

Nielson, F.H., (1982). Possible Future Implications of Nickel in Human Nutrition. Clinical and Biochemical Nutritional Aspects of Trace Elements, p.379-404.

Nielson, F.H., Sandstead, H.H., (1974). Is Nickel Essential for Man. Annual Journal of Clinical Nutrition, 27:515-520.

Norgaard, O., (1955). Investigation with Radioactive Ni-57 into the Resorption of Nickel Through the skin in Normal and in Nickel-hypersensitive Persons. Dermatology Venereol, 35:111-117.

Novey, H.S., (1983). Asthma and IgE Antibodies Induced by Chromium and Nickel Salts. Journal of Allergy and Clinical Immunology, 72:407-412.

Ottolenghi, A.D., (1974). Inhalation of Nickel Sulfate in Pulmonary Carcinogenesis of Rats. Journal of National Cancer Institute, 54:1165-1172.

Redmond, C.K., (1984). Site-specific Cancer Mortality Among Workers Involved in the Production of High Nickel Alloys. International Agency for Research on Cancer, p.73-86.

RTI, (1988). Fertility Study of Nickel Chloride. Report to Office of Solid Waste Management, U.S. Environmental Protection Agency by Research Triangle Institute.

Santucci, B., (1994). Serum and Urine Concentrations in Nickel Sensitive Patients After Prolonged Oral Administration. Contact Dermatitis, 30:97-101.

Schroeder, H.A.,(1974). Life-term Effects of Nickel in Rats. Journal of Nutrition, 104:239-243.

Shirakawa,T.,(1990). Hard Metal Asthma. Thorax, 45:267-271.

Snow, E.T., Costa, M., (1992). Nickel Toxicity and Carcinogenesis. Environmental and Occupational Medicine, 2:807-813.

Sobti, R.C., Gill, R.K., (1989). Abnormalities of Spermatozoa Caused by Heavy Metal Nickel. Cytologia, 54:249-254.

Sunderman, F.W. Jr. (1989). Carcinogenicity of Metal Alloys in Orthopedic Prostheses. Fundamentals of Applied Toxicology, 13:205-216.

Sunderman, FEW. Jr. (1988). Acute Nickel Toxicology in Electroplating Workers Who Accidentally Ingested a Solution of Nickel Sulfate and Nickel Chloride. American Journal of Industrial Medicine, 14:257-266.

Sunderman, FEW. Jr. (1991). Nickel. NCH Verlagsgesellschaft, p.1101-1126.

Sunderman, F.W. Jr. (1993). Biological Monitoring of Nickel in Humans. Scandinavian Journal of Work Environmental Health, 19:34-38.

Sunderman, F.W. Jr. (1984). Mechanisms of Nickel Carcinogenesis. Scandinavian Journal of Work Environmental Health, 15:1-15.

Takenaka, S., (1985). Alveolar Proteinosis Induced in Rats by Long-term Inhalation of Nickel Oxide. Proceedings of the 3rd International Congress on Nickel Metabolism and Toxicology, p.89-92.

Torjussen, W., (1979). Nickel Concentrations in Nasal Mucosa, Plasma, and Urine in Active and Retired Nickel Workers. Annual Clinical Laboratory Science, 9:289-298.

Torjussen, W., Anderson, I., (1985). Occupational Nasal Cancer Caused by Nickel and Nickel Compounds. Rhinology, 23:101-105.

van Hoogstraten, I.M.W., (1991). Effects of Oral Exposure to Nickel or Chromium on Cutaneous Sensitization. Current Problem Dermatology, 20:237-241.

Veien, N.K., (1987). Oral Challenge with Nickel and Cobalt in Patients with Positive Patch Tests to Nickel an/or Cobalt. Dermotology Venereol, 67:321-325.

Vieboer, E., (1992). Occupational Exposures to Nickel. Advances in Environmental Science and Technology, 25:37-48.

Wehner, A.P., (1986). Health and Environmental Effects of Aerosols. Journal of Aerosol Science, 17:305-315.

Widstrom, L., (1985). Ear-piercing-- A Cause of Nickel Allergy in Schoolgirls? Contact Dermatitis, 13:289-293.

Wheeler, M.,(1995). Toxicological Profile for Nickel. U.S. Department of Health and Human Services, p.1-198.

APPENDIX A-1
Raw Sampling Data

Sample No.	Type	Location	Date	Time	Flow Rate	Volume Collected	Result
95-86	Area	Laser welding	8-8-95	12:28 - 15:30	3.0 l/min.	546 liters	0.16 mg/m^3
95-87	Area	Laser welding	8-8-95	12:28 - 15:30	3.0 l/min.	546 liters	0.81 mg/m^3
95-88	Personal	Laser welding	8-8-95	12:28 - !5:30	3.0 l/min.	546 liters	0.48 mg/m^3
95-90	Area	Grinding	8-18-95	8:42 - 15:52	3.0 l/min.	1290 liters	0.007 mg/m^3
95-91	Area	Grinding	8-18-95	8:42 - 15:45	3.0 l/min.	1290 liters	0.002 mg/m3
95-92	Personal	Plating	8-18-95	13:24 - 13:38	3.0 l/min.	42 liters	0.02 mg/m^3
95-94	Personal	Plating	8-18-95	13:38 - 13:52	3.0 l/min.	42 liters	0.007 mg/m^3
95-98	Area	Laser welding	9-8-95	08:30 - 10:38	3.0 l/min.	384 liters	0.56 mg/m^3
95-99	Area	Laser welding	9-8-95	08:30 - 10:38	3.0 l/min.	384 liters	0.5 mg/m^3
95-100	Personal	Laser welding	9-8-95	08:30 - 10:38	3.0 l/min.	384 liters	0.63 mg/m^3
95-104	Personal	Plating	10-4-95	09:40 - 10:08	3.0 l/min.	84 liters	0.007 mg/m^3
95-105	Personal	Plating	10-4-95	11:10 - 11:40	3.0 l/min.	90 liters	0.028 mg/m^3
95-106	Area	Grinding	10-4-95	11:17 - 11:34	3.0 l/min.	51 liters	5.6 mg/m^3
95-107	Personal	Grinding	10-4-95	11:17 - 11:34	3.0 l/min.	51 liters	<0.004 mg/m^3
95- 161	Area	Plating	11-14-95	08:38 - 12:57	3.0 l/min.	777 liters	<0.0002 mg/m^3
95-162	Area	Plating	11-14-95	08:34 - 13:00	3.0 l/min.	798 liters	<0.0002 mg/m^3
95-131	Area	Laser welding	11-7-95	08:56 - 11:27	3.0 l/min.	453 liters	0.008 mg/m^3
95-132	Area	Laser welding	11-7-95	09:05 - 11:25	3.0 l/min.	420 liters	0.23 mg/m^3
95-133	Area	Laser welding	11-7-95	09:09 - 11:23	3.0 l/min.	402 liters	0.02 mg/m^3
95-134	Area	Laser welding	11-7-95	09:06 - 11:23	3.0 l/min.	411 liters	0.005 mg/m^3

APPENDIX A-2

Calibration Chart