IN VITRO RELEASE OF NICKEL AND CHROME FROM STAINLESS STEEL ORTHODONTIC APPLIANCES

Hatsumi Y. Park, D.M.D.

This paper submitted in partial fulfillment of the
requirements for a Certificate in Orthodontics
University of Oregon School of Dentistry

June 1981

ACKNOWLEDGMENTS

I would like to express my sincere appreciation to the following:

Dr. Douglas Buck for his assistance, editorial guidance and support;

Dr. Tom Shearer for his guidance and the use of his laboratory facilities;

Dr. David Phillips and Dan Gray for statistical advice;

Dr. David Mahler for technical advice;

Kathy Jenson and Diane Kieckhafer for typing the manuscript; and

the men in my life, Dick, Paul and Matthew, for their support and understanding.

TABLE OF CONTENTS

INTRODUCTION

The austenitic stainless steels were first exploited in Germany between 1912 and 1914 by Krupp steel works which based development on the research of E. Maurer and B Strauss. These steels contain approximately 18% chrome and 8% nickel and are often referred to as 18-8 stainless steel. Stainless steels are given type numbers by the American Iron and Steel Institute (AISI) by which they are frequently called in practice. The types of 18-8 austenitic stainless steels, their compositions, and properties are well documented (Uhlig 1971). Types 302 and 304 are most commonly used for orthodontic bands and wires. Type 316L is more commonly used in orthopedic implant devices.

Nickel is the most common cause of allergic contact dermatitis and produces more allergic reactions than all other metals combined. Second in frequency is chrome (Moffa et al 1977). Incidence for the general population in North America is 5.8% nickel positive, of these 9% are women and 0.9% are men (North American Contact Dermatitis Group 1973). Incidence among the general population in Finland was 4.5% nickel positive of which 8% were female and 0.8% were male (Peltonen 1979).

Of clinical patients studied, the North American Contact Dermatitis Group (1973) found 11% were nickel positive, chrome was second with an 8% incidence. Other incidence studies of clinic patients vary between 10.8% to 6.4% nickel positive, and 6.6% to 5.7% chrome positive (Fregert and Rorsman 1966, Fregert 1969, Hammershoy 1980). Sexual difference

between nickel and chrome reactivity were noted in most studies. There is generally a higher incidence of nickel hypersensitivity in women while men generally have a higher incidence of hypersensitivity to chrome. Fregert (1969) also observed a striking geographic difference in the European clinic population.

Principal non-occupational sources of contact with nickel are jewelry, especially pierced earrings (Zapffe 1955,Gaul 1967, Watt and Baumann 1968, North American Contact Dermatitis Group 1973, Samitz and Katz 1975, Prytowsky et al 1979). Type 18-8 stainless steel cooking and eating utensils have been shown to release nickel under varying conditions, such as pH, temperature, and type of food by several groups (Brun 1979, Stoewsand 1979, Moller 1980). Kaaber and Vein (1977) believed the ingestion nickel may be of greater significance in the persistence of dermatitis than external contact. Dietary intake accounts for much of the estimated 500 ug daily intake (Louria et al 1972, Venugapal and Luckey 1978). Corrosion of orthopedic implant has been an occasional source of nickel dermatitis. Dental prosthesis of non-precious alloys has been reported as possible sources of nickel and chromate hypersensitivity (Cohen 1955, Bickley 1970, Fisher 1974).

Allergy to chrome from wearing stainless steel dental prosthesis in more than 160 patients has also been reported (Ovrulskii 1976). Also non-occupational exposure to chrome occurs with household detergents and bleach (Samitz 1956,Nater 1963, Feuerman 1971). Chrome is the most common occupational contact allergen; various industrial sources are cement, chrome-plating, leather tanning,etc. (Nater 1963, Cronin 1971, North American Contact Dermatitis Group 1973, Polak et al 1973,Thorman et al 1979). Release of chrome from stainless cooking utensils has been

shown to occur by Brun (1979), Stoewsand (1979), and Moller (1980), but has been disputed by Fregert et al (1969). Dietary chromate ingestion is believed by Kaaber and Vein (1977) to be responsible for chronic chromate dermatitis. Controversy exists concerning the role of trivalent and hexavalent chrome in hypersensitivity (Samitz and Gross 1961, Nater 1963, Mali et al 1963, 1964, Cronin 1971, Polak et al 1973, Burrows 1978, Liden and Lundberg 1979). Most authors feel that hexavalent chromate is the more active form.

Injury to the skin from mechanical, physical, or chemical agents followed by intimate contact with sensitizing allergens favors the development of allergic eczematous dermatitis. There is, therefore, the possibility that the gingivitis in orthodontic patients may predispose an individual to becoming hypersensitized to corrosion products released from stainless steel, or if previously hypersensitized to nickel or chrome to elicit an allergic response.

The purpose of this investigation was to measure the in vitro release of nickel and chrome from stainless steel orthodontic appliances.

REVIEW OF THE LITERATURE

The susceptibility of stainless steel implants to corrosion in situ has been known to metallurgical engineers since the mid-1950's (Zapffe 1955). Two of the earliest reports in the medical literature occurred in 1959 (Cohen and Hammond, Scales).

There are several reports in the literature of reactions to nickel released from implanted stainless steel orthopedic prosthesis (Foussereau and Laugier 1966, Brendlinger and Tarsitano 1970, Barranco and Solomon 1972, Symeonides et al 1973, Pegum 1974, Tilsley and Rotstein 1980). Patch tests for nickel were positive and removal of the prosthesis resulted in complete involution of the eczemas, implying that nickel released from the metallic devices produced the allergic reaction in nickel sensitive patients.

The phenomena of nickel dermatitis following placement of an implant does not occur in all patients. Fisher (1972) rejects the possibility of such reactions on grounds that nickel is so firmly bound in the alloy that body fluids and perspiration cannot "leach" out the nickel and make it available to produce an allergic reaction. As evidence, he noted the dimethylglyoximine spot test for nickel does not give positive results with stainless steel. He found no evidence to contraindicate the use of stainless steel wires in nickel and chrome sensitive patients.

Brettle (1970) acknowledged the corrosion of stainless steel implants and felt they may cause more severe allergic and/or toxic reactions with minimal corrosion because of the presence of nickel and chrome.

The incidence of corrosion of stainless steel implants has been studied by several authors and reports vary. Macroscopic analysis of multicomponent implants removed for various reasons by Scales (1959) revealed 51% for 18-8 stainless steel, 24% for 18-8 molybdenum stabilized stainless steel, 52% for 18-8 titanium stainless steel. Later Scales (1961) reported a 30% incidence of corrosion for both 18-8 stainless steel and 18-8 molybdenum stabilized stainless steel implants removed for various reasons. Colangelo and Greene (1969) reported corrosion in randomly excised implants was 56% with 10% for single component and 91% for multi-component devices. There was a high incidence of short term corrosion with an increase in severity with long term implants. Generally, incidence as well as severity tended to increase with increasing time.

Corrosion of implants has been attributed to use of dissimilar metals, metallic transfer from surgical tools to implant device, differential concentration of electrolyte over the implant (e.g. O_2), and destruction of the protective oxide layer by abrasion (Brettle 1970, Williams and Meachim 1974). Manufacturing defects such as a deficiency of molybdenum, a carbon content greater than 0.03%, the use of sensitized steel, variations in the degree of cold working within an implant, porosity,and fine cracks have also been responsible for corrosion (Colangelo and Greene 1969, Cahoon and Paxton 1970,Williams and Meachim 1974).

Pitting, crevice, galvanic, and stress corrosion of stainless steel implants have been reported in clinical and laboratory studies (Emneus 1960,1961, Brettle 1970, Williams and Meachim 1974). Qualitative in vitro corrosion studies in 0.9% sodium chloride with potassium ferricyanide and gelatin indicator by Emneus (1961) of stainless steel orthopedic screws and wires revealed release of iron, nickel, and cobalt.

Assay for chrome was not included in this study. There was an increase in corrosion with cold working of Type 303 and 316. Emneus has derived a galvanic series for different metals and alloys in saline.

Chemical examination of parenchymal tissue adjacent to stainless steel implants has demonstrated the presence of corrosion products in animals (Ferguson et al 1960,1962) and humans (Mears 1966,1975). Mears concluded if there is ionization of one element from an alloy there tends to be ionization of all elements.

Controversial evidence regarding the release of nickel from stainless steel in various physiologic solutions exists in the literature. Fink and Smatko (1948) and Uhlig(1971) found no difference between the destructive effect of physiologic saline, ordinary serum, and serum acidified to pH 5.0-5.5. Clarke and Hickman (1953) measured anodic potentials in serum in an attempt to predict behavior of orthopedic implants. Generally the inert metals all had high potentials while those which corrode had negative values. Samitz and Katz (1975) demonstrated the solubilization of nickel from AISI 302 and 303 stainless steel orthopedic prosthesis by the action of sweat, blood, and physiologic saline after one week at room temperature. Most of the samples values for nickel were above the eliciting safety limit (ESL) proposed by Malten and Spruit (1969).They defined ESL as the nickel concentration which does not result in a contact allergic reaction in even a highly sensitized person. The authors believed this value to be in the range of 1 uM nickel/liter which is approximately 0.06 PPM nickel. Brown and Merritt (1980) compared the effects of saline alone with that of saline with various concentrations of calf serum. There was an increased release of nickel with increasing concentrations of serum which led him to conclude the mechanism and rates of corrosion of stainless

steel are modified by the presence of serum proteins that interfere with passivation.

Histologic study has revealed the presence of iron (Scales et al 1961, Winter 1974), and large and small unidentified granules suggesting an insoluble corrosion product (Williams and Meachim 1974, Winter 1974). Winter (1974) notes that the existence of soluble corrosion products should not be overlooked.

There has been much speculation on the effects of corrosion on cells. Wright and Axon (1956) observed increased corrosion rates of stainless steel screws and resultant, non-osteoclastic, dissolution of "dead" bone by passing a current between the two screws invested in bone in either saline solution or plasma. Toxicity of heavy metals used for implants may be associated with inhibition of enzymes, prevention of diffusion through cell membranes, and the breakdown of lysosomes with the release of enzymes. It is possible that such lytic processes may occur around orthopedic metallic implants and may contribute to bone resorption (Mears 1966). Three possible cellular effects of corrosion have been proposed by Mears (1966): 1) metallic dissolution products may affect cell metabolism, 2) corrosion may be accompanied by changes in the chemical environment of the cell via the production of hydrogen, hydroxyl ions, or the evolution of hydrogen, oxygen or chlorine gas, or 3) corrosion currents may affect cell behavior. Mear's observation on the effect of direct current on cell cultures have included induction of osteogenesis, alignment of randomly oriented collagen fibers, and the transformation of red cell precursor cells into fully differentiated red cells. The possible deleterious or useful changes in cell behavior induced by corrosion current, however, remains unknown.

Soft tissue effects of oral galvanism was at one time a prominent

concern in dentistry. With increased use of non-precious dental alloys there has been a renewal of interest (Smith 1978). Electrochemical changes of dental materials have been suspected of causing increased or decreased salivation, metallic taste, burning or tingling sensations in the mouth, and mucous irritation by some authors (Lain et al 1940, Soremark 1979, Caputo 1980, Gjerdet 1980). Other studies have shown no evidence of serious pathologic conditions as a direct result of electric current (Hyams 1933, Schriever and Diamond 1952, Inovay and Banoczy 1961, Bergman et al 1978) between various dental materials and stainless steel. However, some authors (Lain et al 1940, Schoonover and Souder 1941, Schriever and Diamond 1952, Bergman et al 1978) were concerned about possible hypersensitivity or toxic reactions to metallic ions, or corrosion products released by galvanism. A search of the literature did not reveal any studies on the incidence of contact stomatitis resulting from exposure to corrosion products released from stainless steel orthodontic appliances.

The lower incidence of contact stomatitis than contact dermatitis has been attributed to the brief period of actual contact, the diluting, washing and possible buffering and neutralizing action of saliva, and extensive vascularization which aids in rapid dispersion and absorption of allergens (Kreshover 1958, Fisher 1973).

Characteristic lesions of contact stomatitis vary from barely visible, mild erythema to a firey red color with or without edema, lingual papilla may disappear. Symptoms may include loss of taste, numbness, burning sensation , and soreness of the involved area. Itching is not a frequent symptom (Bickley 1970, Fisher 1973). Allergic stomatitis is often accompanied by chelitis (Fisher 1973).

There are a few case reports of allergic reactions to corrosion

products from stainless steel dental prosthesis (Adomnicai et al 1969, Ovrulskii 1976). Histologic sections (Adomnicai et al 1969) of gingiva in contact with stainless steel prosthesis exhibited the presence of chronic infiltrates and erosion of the epithelium. Gozhaya (1969) determined the content of copper, iron, and manganese in human saliva under "normal" conditions and in the presence of a stainless steel dental prosthesis. There was an increase in the above metals. Increases in nickel and chrome occurred but values were not given.

Two cases of complications (osteomyelitis, stomatitis, edema) following mandibular fixation with stainless steel sutures have been reported (Shriever and Diamond 1976, Roed-Petersen et al 1979). In both, the patients were skin test positive for nickel and there was a rapid resolution upon removal of the wires. Schriever and Diamond (1976) concluded clinically significant allergic responses to stainless steel are rare and the use of stainless steel sutures is not contraindicated. Simpson and Carter (1966) reported a case in which stainless steel sutures were used for mandibular fixation. After 5 months in situ there was no metallurgical evidence of corrosion, and histologic section revealed the presence of normal healing. Cutright (1973) compared 55-Nitinol with stainless steel sutures. After 9 weeks subcutaneous implantation in rats, he concluded that histologically both were acceptable for use in deep tissues.

Few reports exist in the dental literature concerning the results of stainless steel in contact with other dental metals and alloys. Stainless steel in contact with amalgam in vivo exhibited currents approximately 3 times higher than other combinations of alloys tested i.e., amalgam to gold, amalgam to chromium-cobalt alloy indicating a higher corrosion rate (Gjerdet and Brune 1977). Rogers (1977) used an accelerated corrosion

test solution of ferric sulfate and hydrofluoric acid to show crevice corrosion occurred at stainless steel-silver solder joints. Root fracture due to corrosion of stainless steel pins with amalgam post restorations have been observed by several authors (Angmar-Mansson et al 1969, Peterson 1971, Silness et al 1979). Analysis of the corrosion products by Silness et al (1979) demonstrated the presence of chrome and iron as well as minor amounts of nickel. Angmar-Mansson et al (1969) assay of corrosion products demonstrated tin and chrome as the predominant metals; nickel was not present in these deposits. His varying results led him to conclude some of the corrosion products are water soluble. Futhermore, there is the possibility that released corrosion products have been deposited either on the surface of the alloys and/or on adjacent materials (Arvidson and Wroblewski 1978). Insoluble corrosion products had penetrated and discolored the dentin in both Angmar-Mansson et al (1969) and Silness et al (1979) studies. The process has been attributed to corrosion resulting from the presence of metals with very different potentials in an electrolyte. Active and passive areas on a stainless steel pin will result in pitting corrosion when exposed to an electrolyte (Angmar-Mansson et al 1969). Both Angmar-Mansson et al (1969) and Emneus (1960) have observed active 18-8 stainless steel has a corrosion resistance equal to ordinary iron while passive 18-8 stainless steel has a corrosion resistance almost of noble metals.

Contrary to the above reports, Simonsen et al (1973) in a long term clinical study, found no radiographic evidence that stainless steel pins with amalgam posts results in corrosion.

A review of the orthodontic literature revealed Hyams (1933) studied the effects of galvanic currents generated by dissimilar alloys in orthodontic appliances (gold-palladium alloy, stainless steel, brass);

he concluded there was no correlation between the intensity of current and oral lesions. Rickles (1972) noted the nickel in stainless steel orthodontic bands may contribute to gingivitis in some patients despite excellent oral hygiene and well fitted bands. Zachrisson (1972) has shown histologic evidence of chronic inflammation during active orthodontic treatment characterized by presence of chronic inflammatory cells. Predominant types were plasma cells, lymphocytes, and mast cells. He did not attempt to assay the tissue for presence of corrosion products. Mueller et al (1979) have shown macroscopic surface changes occur when as-received orthodontic stainless steel (Type 302,316, Elgiloy) wires are immersed in Ringer's solution. They felt there would be a further decrease in corrosion resistance for stainless steel appliances requiring soldered connections. Recently Ceen and Gwinnett (1980) studied the source of a greenish-black stain on enamel and bonding resin discovered while debonding a patient. Analysis of the resin revealed the discoloration was due to the presence of chrome. There was no evidence of nickel contamination.

MATERIALS AND METHODS

PART I: Static Immersion: Determination of Cumulative Soluble Nickel and Chrome

A simulated orthodontic appliance was constructed for half a mandibular arch consisting of similar sized first and second molar bands, first and second premolar bands,* cuspid, lateral incisor and central incisor bonding brackets** (Fig. 1). A lingual button*** was welded to the second molar band, all other bands and bonds were used in the as-received condition from general clinic supply. Bands were filled with clear cold cure orthodontic acrylic. The mesh backs of the bonded brackets were filled with orthodontic bonding resin.+ Bands and bonds were held securely in place to a 70 mm long 0.48 x 0.64 mm rectangular arch wire++ with a 2 mm L-bend at one end by elastomeric units.+++ Controls consisted of equivalent pieces of clear cold cure acrylic, bonding resin, and elastomeric units (Fig. 2). The 10 experimental and 10 controls were statically immersed in a screw cap Nalgene® bottle containing 100 ml of 15 mEq Cl^-/liter of a sodium chloride solution

*Unitek, Twin Torque bands, size 38½ molar, size 24 premolar (bracket AISI 303 Se, bands AISI 304)
**Unitek, standard edgewise bracket, Dyna-Bond pads (bracket AISI 303 Se, backing and mesh pad AISI 304)
***ORMCO, lingual button flat #610-0001
+ORMCO, Endur Adhesive
++Unitek, Permachrome standard (AISI 304)
+++Unitek, A-lastik, medium gray Al

(approximately 0.05% sodium chloride). Two crops of toluene were added to all the bottles as a perservative. The bottles were then placed in a 37° C water bath.

Soluble nickel and chrome were determined by atomic absroption spectrophotometry* (Fig3) at days 3,6,9, and 12. The sensitivity of the instrument for both nickel and chrome is 0.06 ug/ml (PPM). Standard operating conditions for nickel were as follows: wave length 232.0,band pass 0.15, scale 5X, integration 1, high voltage 1000, no curve correction, deuterium lamp correction for interfering ions. Standard operating conditions for chrome were: wave length 357.9, band pass 0.5, scale 5X, integration 1, high voltage 530, no curve correction.

The assay required 1 ml for nickel and 1 ml for chrome. Two ml was saved for possible use at a later date. This 4 ml was replaced with fresh sodium chloride solution.

Standard solutions were prepared by diluting stock solution of 1000 PPM nickel and 1000 PPM chromium standard** with the sodium chloride solution. The sodium chloride solution served as the blank. Standard curves were determined before and after each experimental assay.

The condition of the appliance and solution was determined by macroscopic inspection at days 3,6,9, and 12. Corrosion was determined by the appearance of rust on the appliance and evaluated as slight (+), moderate (++), or severe (+++). The presence of a precipitate was judged as slight (+),moderate (++), or severe (+++). Changes in the color of the solution were noted as either clear (-) or rusty (+). The pH of the sodium chloride solution was determined*** at the beginning and at

*Instrumentation Laboratory Inc., Atomic Absorption Spectrophotometer #IL 150
**Scientific Products, #S7385-24 (Chromium, #S7385-28 (nickel)
***ElectroMark Analyzer #4403

the end of day 12 for controls and experimental samples.

No attempt was made to identify the chemical composition of the soluble nickel or chrome.

PART II:Static Immersion: Determination of Cumulative Precipitated Nickel and Chrome

At the end of day 12 the rust-colored precipitates in the experimental samples from Part I were centrifuged, washed three times in 10 ml aliquots of the sodium chloride solution, then dried overnight at 85°C. The weights of the dried precipitate was not accurately determined. The dried precipitate was solubilized in 5 ml cold concentrated nitric acid overnight. Each sample was diluted to 20 ml with the sodium chloride solution. Blanks and standards were prepared using equivalent dilutions of concentrated nitric acid with the sodium chloride solution. Nickel and chrome in the solubilized precipitate were analyzed by atomic absorption spectrophotometry as in Part I. No attempt was made to remove the rust-colored residue from the appliance for assay of nickel and chrome. The molecular composition of the precipitate was not determined.

PART III: Shaking Immersion: Determination of Soluble Nickel and Chrome

A simulated orthodontic appliance was constructed as in Part I. The 10 experimental samples were placed in capped Nalgene® bottles and immersed in 100 ml of 15 mEq Cl^-/liter of sodium chloride solution. The bottles were placed in a shaking water bath at 37°C. Soluble nickel and chrome were determined as in Part I at day 1; a 2 ml aliquot was saved. This volume was replaced with fresh sodium chloride solution. At 3 day intervals, i.e. days 3,6,9,and 12, soluble nickel and chrome were determined and an aliquot saved, the remaining solution was discarded and replaced

by 100 ml of fresh sodium chloride solution. The bottles were then returned to the shaking water bath.

The condition of the appliance and solution was determined by macroscopic inspection at days 1,3,6,9, and 12 as in Part I. The pH of the sodium chloride solution was determined at the beginning of the experiment and at the end of day 12.

RESULTS

PART I: Static Immersion: Determination of Cumulative Soluble Nickel and Chrome

At the end of 12 days there was a significant ($P<.001$) release of soluble and precipitated nickel (Table 1). Twelve day total cumulative mean release was 125 µg, or a mean daily release of 10µg/simulated mandibular quadrant. The mean cumulative soluble fraction of nickel at day 3 was 53± 49 µg/sample. Soluble nickel increased to a mean value of 112±82 µg/sample at day 6. The levels of nickel plateaued at this time (Fig.4). At day 9 and 12 the mean values were 106±54 µg/sample, and 121±69 µg/sample respectively. Sample number 1 exhibited very high levels of soluble nickel at all time periods, samples number 4,5,and 8 also reached high levels (Fig.5).

The twelve day cumulative release of soluble chrome was significant ($P < .05$); precipitated chrome was also significant ($P < .001$)(Table 1). The mean cumulative soluble fraction of chrome at days 3,6,9, and 12 were 4±7,28±18,27±17,and 40±28 µg/sample respectively. The levels of soluble chrome also plateaued at day 6 (Fig. 4). There was considerable variation in the pattern of release of soluble chrome(Fig.6).

Macroscopic examination of the appliance revealed corrosion occurred at weld sites of bands and once started became progressively severe. Severe corrosion was generally confined to one area of a simulated

appliance. There was no macroscopic evidence of corrosion of bonded brackets. Initial signs of corrosion were noted in most samples at day 6. A rust colored precipitate occurred at about day 3 and became progressively darker with time.

Beginning pH was 6.2 and the mean value at day 12 was 6.4. There was no change in pH in the control samples, nor were there any macroscopic changes in the acrylic, bonding resin, elastomeric units or the color of the solution.

PART II: Static Immersion: Determination of Precipitated Nickel and Chrome

At the end of 12 days there was a significant ($P < .001$) release of precipitated nickel (Table I). Mean twelve day total released was 4 ± 2 ug/sample. The mean 12 day chrome present in the precipitate was also significant ($P < .001$) with a mean of 72 ± 40 ug/sample.

Release of soluble nickel is three times greater than chrome with static immersion; while release of precipitated chrome is 18 times that of precipitated nickel (Table I), i.e. nickel is released primarily in a soluble form, while chrome is released in a precipitate.

PART III: Shaking Immersion: Determination of Soluble Nickel and Chrome

With 12 days of shaking immersion there was also a significant ($P<.001$) total release of soluble nickel. Precipitated nickel could not be determined because most of the sodium chloride solution was discarded. Since most of the samples had non-detectable levels of chrome, statistical analyses were determined for assumed minimum; i.e. assumed non-detectable levels were actually zero, and for assumed maximum, i.e. sensitivity of the instrument is 0.06 PPM which is equivalent to 6 ug/sample/interval,

so that 6 µg maximum could be present and not detected. The data (Table I) indicates significant ($P < .05$ and $P < .001$) release of soluble chrome for assumed minimum and maximum values respectively.

At day 1, there was a mean soluble nickel concentration of 18±12 µg/sample (Fig.7). There was no detectable chrome in any of the samples at this time (Fig.9). After the first 3-day interval mean soluble nickel was 23±18 µg/sample. Chrome levels were again not detectable. At the end of the second 3-day interval, the sixth day of the experiment, mean soluble nickel was 42±24 µg/sample. Three samples had 10 µg of soluble chrome, the rest of the samples still had non-detectable levels. At the end of the third 3-day interval, ninth day of the experiment, mean soluble nickel concentration was 38±19 µg/sample. Four samples had detectable levels of soluble chrome. On the twelth day of the experiment, fourth 3-day interval, the mean concentration of soluble nickel was the same as that of the previous interval, 38±22 µg/sample. Chrome was detectable at the same levels in the same samples as on the third 3-day interval. The additive means for soluble nickel at each experimental interval were 18,41,83, 121, and 159 µg/sample. Assumed minimum additive mean values for soluble chrome were 0,0,3,8,and 13 µg/sample (Fig.10); while the assumed maximum additive values were 6,12,15,20, and 25 µg/sample. As in Part I, there was a variation in the pattern and amounts of soluble nickel released (Fig.8). The pattern of soluble chromium release was not as variable as in Part I (Fig.9).

There were no apparent macroscopic changes present on the appliance at day 1. Corrosion was evaluated as in Part I and appeared at about day 3, and as in Part I, once started became progressively worse, especially in one area of a simulated appliance. There was no macroscopic evidence

of corrosion on any of the bonded brackets. A ring of rust colored residue formed along the surface of the sodium chloride solution at about the first 3-day interval and became progressively denser. The solution did not change color until about the third 3-day interval. The presence of a precipitate was also noted at this time.

The mean pH at the end of the twelth day was 6.4.

There is no statistical difference in soluble nickel release between static and shaking immersion (Table II). Nor is there a statistical difference for soluble chrome when assumed maximum values are used. Using assumed minimum values there is a significant ($P < .05$) difference.

DISCUSSION

Under the conditions of the present study, nickel is primarily released as a soluble product, while chrome is released primarily as a non-soluble product with corrosion of the simulated appliance. The data indicates that dissolution of both nickel and chrome occurs with soluble nickel being released at 3 to 4 times that of chrome. In contrast, 18 times greater concentration of chrome is present in the precipitate.

This pattern of chrome and nickel release from stainless steel is similar to the findings of earlier studies. The investigations of Angmar-Mansson et al (1969) indicates corrosion of stainless steel endodontic pins results in deposits which contain chromium. Silness et al (1979) has also demonstrated the presence of chrome in deposits from corrosion of stainless steel endodontic pins. While Silness et al (1979) found minor amounts of nickel in the corrosion product, Angmar-Mansson et al (1969) did not find any. Ceen and Gwinnett (1980) found chrome present in the discolored bonding resin following debonding, but no detectable nickel.

The "stainless" property of a stainless steel is due to the presencce of a closely adherent, passive chromium oxide layer which forms on the surface after exposure and reaction with oxygen. Olefjord (1980) has shown that the reaction products formed on the surface of chrome-nickel-molybdenum alloyed stainless steels consists mainly of chromium oxide and chromium hydroxide; nickel is not present in the passive film. The chromium oxide film is 300-400 Angstroms thick (Emneus 1960).

Sensitization is the loss of this passivating layer and can result from a number of ways. Physically, surface abrasion as well as dynamic contact of two surfaces can remove the passivating layer. The passive film is destroyed by the slightest mechanical trauma so that the presence of oxygen is necessary for stainless steel to remain corrosion resistant (Angmar-Mansson 1969). Sensitization can also occur from heating.

When passive alloys such as the stainless steels corrode, the corrosion develops where passivity has been destroyed. The loss of the passive layer causes these areas to become anodic and the remaining passive layer of the same material becomes cathodic. The reactions which occur at the anode and cathode during corrosion are:

ANODIC REACTION:

$M \longrightarrow M^{+n} + ne^-$ Metal dissolution

CATHODIC REACTION: (Two possible reactions)

(A) $2H^+ + 2e^- \longrightarrow H_2$ Hydrogen evolution

$O_2 + 4H^+ + 4e^- \longrightarrow H_2O$ Oxygen reduction

(B) $O_2 + 2H_2O + 4e^- \longrightarrow 4OH^-$ Oxygen reduction

$M^{+3} + e^- \longrightarrow M^{+2}$ Metal ion reduction

$M^+ + e^- \longrightarrow M$ Metal deposition

The present data indicates corrosion of 18-8 stainless steel results in release of soluble nickel and the deposition of chrome in a precipitate. There was no apparent release of gas during either Part I or Part III so the reaction occurring at the cathodic sites are probably due to reaction (B).

Various forms of corrosion of stainless steel are:

1. intergranular, due to heating 18-8 stainless steel between 400-900° C
2. pitting, due to the attack of halide ions; especially Cl^-
3. concentration or crevice, due to difference in oxygen concentration

4. stress, due to excessive cold working, and

5. galvanic, due to the presence of dissimilar metals and alloys in an electrolyte.

Sensitization due to heating 18-8 stainless steel between 400-900° C results in intergranular corrosion. The stainless steel becomes more susceptible to intergranular attack. The most widely accepted explanation for this form of sensitization is the chromium depletion theory which attributes the increased susceptibility to the formation of chromium carbide along grain boundaries with the accompanying depletion of chromium from the adjacent austenitic matrix. The carbon content of stainless steel may be of significance in intergranular corrosion; tests indicate AISI 304 L is relatively immune to low temperature weld sensitization (Povich and Rao 1978).

Pitting corrosion is a localized attack occurring most frequently in media containing chloride. In this form of attack,there is a surface cavity having a depth:surface diameter ratio of about one. Many factors affect pitting corrosion. In general, pitting corrosion is adversely affected by increased oxygen content and chloride content of a solution (Emneus 1960,1961,Hoar and Jacob 1967, Angar-Mansson 1969,Colangelo and Greene 1969, Uhlig 1971). Stabilized austenitic steel is more resistant to pitting corrosion (Emneus 1960, Brettle 1970). The effects of pH and temperature on pitting corrosion within physiologic limits in 1.0% sodium chloride are not pronounced (Uhlig 1971). In a preliminary study, a used complete maxillary appliance was placed in distilled water overnight at 37°C. The same patient's complete mandibular appliance was placed in 0.09% sodium chloride solution overnight at 37°C. There was approximately 20 ug soluble nickel released in water overnight vs.

approximately 70 µg in the 0.09% sodium chloride, indicating the probable occurence of pitting corrosion.

Uhlig (1971) observed that chlorides promote more pitting than fluorides. The effects of zinc phosphate and fluoride containing cements on corrosion of orthodontic appliances remains to be studied.

Crevice corrosion is an intense local attack which occurs in shielded areas on a metallic surface. Stainless steel and other active-passive metals are especially susceptible to this form of corrosion. The breakdown of passive film can occur spontaneously by abrasion between metal-metal or by the relative motion of metal-metal contact areas. With restricted oxygen diffusion to the bare metal surface, re-passivation cannot occur and the crevice remains in an active condition and corrodes at a rapid rate while the remaining surface is maintained passive. The clinical significance of crevice attack lies in its intensity, which may give rise to large amounts of corrosion products being released. Crevice corrosion may be of significance in the orthodontic situation, since the conditions mentioned above do occur. Since differential oxygenation will result in the more oxygenated portion becoming cathodic (Wright and Axon 1956) the presence of dental plaque could be of significance in crevice corrosion. The conditions of this study did not allow evaluation of crevice corrosion.

Cold working occurs both during manufacturing and clinical use of orthodontic appliances, but the effect this may have on corrosion of 18-8 stainless steel has not been reported in the dental literature. Mechanical distortion of stainless steel results in the distorted portion becoming anodic with respect to the undistorted portion so that an alloy behaves electrochemically as if two different alloys were present. The theoretical explanation is the change from austenitic, non-magnetic face-centered

cubic to martensitic, magnetic, body centered cubic and consequent greater ease with which atoms from an irregular, distorted lattice arrangement can dissolve than can those from a regular, undistorted lattice arrangement (Wright and Axon 1956). Williams and Meachim (1974) correlated the amount of transformation from austenitic to martensitic stainless steel with the nickel content and concluded that more than 10% nickel is needed for complete austenitic stability and a minimum of 11% nickel would be more desirable. Angar-Mansson et al (1969) study indicates that austenite with a nickel content of greater than 14% would be required for stability. Sutow (1980) demonstrated increased corrosion resistance by electropolished specimens of AISI 316L stainless steels. He attributes this improved corrosion resistance to the removal of a cold worked surface layer, as well as the decreased surface roughness. For all types of cold working there is a decrease in resistance to pitting corrosion in a buffered physiologic solution of AISI 316L and 304L stainless steels (Cigada et al 1977). The loss of austenitic property was the mechanism believed responsible for loss of corrosion resistance in several other studies (Cohen and Hammond 1959,Simpson and Carter 1966, Angar-Mansson et al 1969). It is doubtful that cold working contributed to the macroscopic corrosion observed in the present study since none of the arch wires and bands exhibited gross corrosion. The possible occurence of microscopic corrosion remains to be studied.

Galvanic corrosion occurs when metals and alloys with different potentials are in contact and surrounded by an electrolyte with the consequent formation of an electric current. In galvanic corrosion the less noble metal is attacked, while the more noble one with a higher potential remains intact. The risk of galvanic corrosion on contact

between two different metals and alloys increases with the difference in potential. This form of corrosion also varies with the properties of the electrolyte.

Galvanic corrosion of dissimilar metals has been widely described in the surgical literature. Levine and Staehle (1977) do not recommend the use of dissimilar alloys in orthopedic implants. However, Mears (1975) experiments led him to conclude that corrosion of passive metals is provoked by the presence of a crevice on a susceptible alloy and not by the presence of dissimilar passive alloys.

Lain et al (1940) measured electrogalvanism in vivo and in vitro between dissimilar metals used in dentistry. Inovay and Banoczy (1961) measured the in vivo potential differences between various metals. Although higher values were found between stainless steel and amalgam, there were also high values between similar metals (stainless steel to stainless steel, amalgam to amalgam). He attributed these findings to the lack of homogeneity in an alloy, variability in surface finishing and polishing. Rogers (1977) used an accelerated corrosion testing solution of ferric sulfate and hydrofluoric acid and found corrosion occurred at stainless steel to silver solder joints with preferential dissolution of the stainless steel. Gjerdet and Brune (1977) found the currents generated between stainless steel and amalgam was three times higher than other combinations of metals.

Gjerdet (1980) discussed the possibility that the presence of dissimilar metals placed in contact in the oral cavity may cause galvanic currents and release of ions from the metals.

The present study did not incorporate any metals or alloys other than 18-8 stainless steels. However, the above studies indicate there

would be greater corrosion with greater release of nickel and chrome in the presence of siler solder, amalgam, or gold. Whether galvanic corrosion occurred under these experimental remains to be investigated.

La Que (1965) and Gjerdet (1980) have both pointed out that metallurgical tests for corrosion are valuable, but no test or group of tests gives results which corresponds to the performance of materials in service. This is especially true when the complexity of the oral environment is considered. Variable in the oral cavity include changes in temperature, quantity and quality of saliva, amount of bacterial plaque, pH, physical and chemical properties of foods and liquids, intake of drugs, and general and local health conditions. All of which may influence corrosion of metals and alloys placed in the mouth. Saliva is a non-static fluid, changing in pH and conductivity throughout the day and varies from patient to patient, the "protective" properties of saliva will also vary (Solomon et al 1938, Fusayama et al 1963, von Fraunjofer and Staheli 1972). Corrosion occurs at a faster rate in saline than saliva (Solomon et al 1938, Fusayama et al 1963) but the exact mechanism of this "protective" effect remains unknown. Because of the great variability of saliva several different formulas are available (Fusayama et al 1963, Tani and Zucchi 1967, Ardvidson and Johansson 1977). Meyer and Nally (1975) compared various artificial salivas, Ringer's solution, various chloride, thiocyanate, and phosphate solutions with stimulated saliva. Fusayama's formula with "slight modifications" was found to best correlate with results obtained with natural saliva. The authors failed to state what modifications were made. In the present study, it was decided to use a 15 mEq Cl^-/liter solution of sodium chloride as the electrolyte rather than any of the

artificial salivas or natural stimulated saliva. This concentration of chloride is the average chloride concentration of saliva (Guyton 1981).

Recently Gettleman (1980) attempted to correlate in vivo with in vitro corrosion current between dissimilar metals and alloys. In vitro experiments were conducted in Tani and Zucchi's (1967) artificial saliva, 37°C, pH 7.0. His results, however,were inconclusive as certain technical problems remained to be solved.

There are no reports of the possible corrosive action of dental plaque on orthodontic stainless steels. Thorpe (1978) reported the corrosion of AISI 321 stainless steel beneath deposits of "microbial slime," the mechanism of corrosion was thought to be deposit or crevice corrosion plus the presence of sulfide produced by sulfate reducing bacteria. He observed that AISI 316 stainless steel appears to be resistant to corrosion from such "microbial slime."

The pH was measured in this study, but no statistical tests were performed since previous experiments indicate the in vitro behavior of AISI 316 at 37°C in Ringer's solution is independent of both pH (5.9 to 7.4) and pO_2 (0.7 to 19 mm Hg) within physiologic conditions (Sutow et al 1976).

Comparison of surface topography of the bands and brackets before and after immersion by any microscopic techniques was not included in this study. There was a generalized loss of reflectivity of the samples but this criteria was not used in macroscopic evaluation of corrosion. Further research will be required to determine if nickel and/or chrome are released from these "etched" areas of 18-8 stainless steel orthodontic appliances.

Soremark (1979) raised the concern that corrosion products can

sooner or later in one way or another, be harmful to the tissues of the oral cavity and/or distant parts of the body. Since (1) orthodontic patients usually remain in active treatment between 18-24 months, (2) the daily dietary intake of nickel (300-600 µg) and chrome (60-100 µg)is greater than that released under these experimental conditions, and (3) both metals are readily excreted, nickel in feces and chrome via the kidney (Venugapal and Luckey 1978), the long term effects are probably not of clinical significance. The short term effect of hypersensitivity to nickel and/or chrome released from 18-8 stainless steel remains to be studied.

SUMMARY & CONCLUSIONS

Simulated Orthodontic appliances were constructed and statically immersed in 15 mEq/liter of a sodium chloride solution at 37°C. At 3 day intervals the sodium chloride solution was measured for the presence of soluble nickel and chrome by atomic absorption spectrophotometry. The 12 day accumulated precipitate was solublized and assayed for nickel and chrome. Both soluble nickel and chrome were released from the appliance in statistically significant quantities with three times more soluble nickel released than soluble chrome by the twelth day. Nickel and chrome were also found in statistically significant amounts in the precipitate. Eighteen times more chrome than nickel was found in this fraction. The controls were negative for soluble nickel and chrome; there was no precipitate formation.

When simulated orthodontic appliances were placed in 15 mEq/liter of sodium chloride solution and placed in a shaking water bath at 37°C with complete changes in the chloride solution at 3 day intervals, there was also a statistically significant release of total soluble nickel and chrome. Again more soluble nickel than soluble chrome was released. Once soluble nickel and chrome were detected in the chloride solution the release progressed in a linear fashion.

There was no statistical difference in soluble nickel released during static or shaking immersion, nor was there a statistical difference in assumed maximum chromium released.

Corrosion was easily noticeable on macroscopic examination as a rust colored residue on the weld sites of the simulated appliance and once initiated becomes progressively worse for static and shaking immersion. A rust colored precipitate occurred in all experimental samples by the twelth day with either static or shaking immersion.

Under the experimental conditions, nickel and chrome are released in statistically significant quantities. However, the amounts of nickel and chrome released are within the daily dietary intake of both metals. The mean daily release of nickel and chrome per simulated half mandibular arch was 10 µg and 9 µg respectively.

Further research is required to determine if 1) selenium is released from the brackets, 2) the presence of silver solder, amalgam, and/or gold will accelerate the release of nickel and/or chrome to clinically significant levels, and 3) the molecular composition of the released nickel and chrome, and whether it is of clinical significance in sensitizing patients or causing a hypersensitivity reaction in patients who have a prior history of hypersensitivity to nickel and/or chrome.

BIBLIOGRAPHY

1. Adomnicai, M.C. et al. "Clinical, Histopathological and Histochemical Aspects of Gingival Mucosa in Contact with Stainless Steel," Stomatologia,16:213,1969.

2. Angmar-Mansson, B. et al. "Root Fractures Due to Corrosion. I. Metallurgical Aspects,"Odontol. Revy , 20:245,1969.

3. Arvidson, K. and Johansson, E.G. "Galvanic Series of Some Dental Alloys," Scand. J. Dent. Res., 85:485,1977.

4. Arvidson, K. and Wroblewski, R. "Migration of Metallic Ions from Screwposts into Dentin and Surrounding Tissues," Scand. J.Dent. Res., 86:200,1978.

5. Barranco V.P. and Solomon, H. "Eczematous Dermatitis from Nickel," J. Am. Med. Assoc., 220:1244,1972.

6. Bergman, M. et al. "Potential and Polarization Measurements In Vivo of Oral Galvanism," Scand. J. Dent. Res., 86:135, 1978.

7. Bickley, H.C. "A Concept of Allergy with Reference to Oral Disease," J. Periodont., 41:302,1970.

8. Brendlinger, D.L. and Tarsitano, J.J. "Generalized Dermatitis Due to Sensitivity to Chrome Cobalt Removable Partial Denture," J. Am. Dent. Assoc., 81:392,1970.

9. Brettle, J. " A Survey of the Literature on Metallic Surgical Implants," Injury, 2:26,1970.

10. Brown, S.A. and Merritt, K. " Electorchemical Corrosion in Saline and Serum," J. Biomed. Mater. Res., 14:173,1980.

11. Brun, R. "Nickel in Food: The Role of Stainless Steel Utensils," Contact Dermatitis, 5:43,1979.

12. Burrows, D. "Chromium and the Skin," Brit. J. Dermat., 99:587,1978.

13. Cahoon, J.R. and Paxton, H.W. "A Metallurgical Survey of Current Orthopedic Implants," J. Biomed. Mater. Res., 4:223,1970.

14. Caputo, A.A. " Biological Implications of Dental Materials," Dent. Clin. N. Am., 24:331,1980.

15. Ceen, R.F. and Gwinnett, A.J. "Indelible Iatrogenic Staining of Enamel following Debonding: A Case Report," J. Clin. Orthod.,14:713,1980.

16. Cigada, A. et al. "Influence of Cold Plastic Deformation on Critical Pitting Potential of AISI 316L and 304L Steels in an Artificial Physiologic Solution Simulating the Aggressiveness of the Human Body," J. Biomed. Mater. Res.,11:503,1977.

17. Cohen, E.L. "Aspects of Allergy," Brit . Dent. J., 99:185,1955.

18. Cohen, J. and Hammond, G. "Corrosion in a Device for Fracture Fixation," J. Bone Joint Surg., 41A:524,1959.

19. Clarke, E.G.C. and Hickman,J. "An Investigation into the Correlation between the Electrical Potentials of Metals and Their Behavior in Biological Fluids," J. Bone Joint Surg., 35:467,1953.

20. Colangelo, V.J. and Greene, N.D. "Corrosion and Fracture of Type 316 SMO Orthopedic Implants," J. Biomed. Mater. Res., 3:247,1969.

21. Cronin,E. "Chromate Dermatitis in Men," Brit. J. Dermat.,85:95,1971.

22. Cutright, D. E. et al. "Tissue Reaction to Nitinol Wire Alloy," Oral Surg.,Oral Med., Oral Path.,35:578,1973.

23. Emneus, H. "Comparison between Biological Assays and the Ferroxyl Test for Studying Corrosion of Experimental Metal Implants," Acta Orthopaed. Scand., 44a:1,1960.

24. Emneus, H. "Experimental Investigation of Corrosion of Stainless Steels Used in Bone Surgery," Acta Orthopaed. Scand., 44b:1,1961.

25. Ferguson, A.B. et al. "The Ionization of Metal Implants in Living Tissues," J. Bone Joint Surg., 42A:77,1960.

26. Ferguson, A.B. et al. " Characteristics of Trace Ions Released from Embeded Metal Implants in the Rabbit," J. Bone Joint Surg.,44A:323,1962.

27. Feuerman, E. "Chromate as the Cause of Contact Dermatitis in Housewives," Dermatologica, 143:292, 1971.

28. Fink, C.G. and Smatko, J.S. "Bone Fixation and the Corrosion Resistance of Stainless Steel to Fluids of the Human Body, "J. Electrochem. Soc., 94:271,1948.

29. Fisher, A.A. "Safety of Stainless Steel in Nickel Sensitivity," J. Am. Med. Assoc.,221:1279, 1972.

30. Fisher, A.A. Contact Dermatitis, 2nd Ed., Lea and Febiger, Philadelphia, 1973.

31. Fisher, A.A. " Allergic Reactions Due to Metals used in Dentistry," Cutis, 14:797,1974.

32. Foussereau, J. and Laugier, P. "Allergic Eczemas from Metallic Foreign Bodies," Trans. St. Johns' Hosp. Dermat. Soc.,52:220,1966.

33. Fregert, A. and Rorsman,H. "Allergy to Chromium, Nickel and Cobalt," Acta. Dermatoven.,46:144,1966.

34. Fregert, S. et al. "Epidemiology of Contact Dermatitis," Trans. St. Johns' Hosp. Dermat. Soc.,55:17,1969.

35. Fusayama, T. et al. "Corrosion of Gold and Amalgam Placed in Contact with Each Other," J. Dent. Res., 42:1183,1963.

36. Gaul, L.E., "Development of Allergic Nickel Dermatitis from Earrings," J. Am. Med. Assoc., 200:186,1967.

37. Gettleman L. et al. "Measurement of In Vivo Corrosion Rates in Baboons, and Correlation with In Vitro Tests," J. Dent. Res.,59:689, 1980.

38. Gjerdet, N.R. and Brune, D. "Measurement of Currents between Dissimilar Alloys in the Oral Cavity," Scand. J. Dent. Res., 85:500,1977.

39. Gjerdet, N.R. "Galvanic Cells Including Cobalt-Chromium Alloys," Acta Odont. Scand., 38:273,1980.

40. Gozhaya, L.D. "Trace Elements in the Saliva in the Use of Prosthesis from Stainless Steel," Stomatologia, 48:63,1969.

41. Guyton, A.C. Textbook of Medical Physiology, 6th Ed.,W.B. Saunders Co., Philadelphia,1981.

42. Hammershoy, O. "Standard Patch Test Results in 3225 Consecutive Danish Patients from 1973-1977," Contact Dermatitis, 6:263,1980.

43. Hoar, T.P. and Jacob, W.R. "Breakdown of Passivity of Stainless Steel by Halide Ions," Nature,216:1299,1967.

44. Hyams. B.L. "The Electrogalvanic Compatibility of Orthodontic Materials," Int. J. Orthod. Dent. Child., 19:883, 1933.

45. Inovay, J. and Banoczy,J. "The Role of Electrical Potential Differences in the Etiology of Chronic Diseases of the Oral Mucosa," J. Dent. Res., 40:884, 1961.

46. Kaaber, K and Vein, N.K. "The Significance of Chromate Ingestion in Patients Allergic to Chromate," Acta. Dermatoven.,57,321,1977.

47. Kreshover, S.J. "Oral Manifestations of Allergy and Their Treatment," Dent. Clin. N. Am., p. 231,1958.

48. Lain, E.S. et al. "Problem of Electrogalvanism in the Oral Cavity Caused by Dissimilar Dental Metals, " J. Am. Dent. Assoc.,27:1765, 1940.

49. La Que, F.L. Corrosion and Resistance of Metals and Alloys, 2nd Ed., Rheinhold Publishing Co, New York, 1965.

50. Levine D.L. and Staehle, R.W. "Crevice Corrosion in Orthopedic Implant Metals," J. Biomed. Mater. Res., 11:553,1977.

51. Liden, S. and Lundberg,E. "Penetration of Chromium in Intact Human Skin In Vivo," J. Invest. Dermatol.,72:42,1979.

52. Louria, D.B. et al. "The Human Toxicity of Certain Trace Elements," Ann. Intern. Med.,76:307,1977.

53. Mali, J.W. et al. "Some Aspects of the Behavior of Chromium Compounds in the Skin," J. Invest. Dermatol.,41:111,1963.

54. Mali, J.W. et al. "Quantitative Aspects of Chromium Sensitization," Acta Dermatoven.,44:44,1964.

55. Malten, K.E. and Spruit, D. "The Relative Importance of Various Environmental Exposures to Nickel in Causing Contact Hypersensitivity," Arch Dermatoven (Stockholm), 49:14,1969.

56. Mears, D.C. "Electron Probe Microanalysis of Tissue and Cells from Implant Areas," J. Bone Joint Surg., 45B:567,1966.

57. Mears, D.C. "The Use of Dissimilar Metals in Surgery," J. Biomed. Mater. Res. Symp. 6:133,1975.

58. Meyer, J.M. and Nally, J.N. "Influence of Artificial Salivas on the Corrosion of Dental Alloys," J. Dent. Res.,54:678,1975.

59. Moffa, J.P. et al. "Allergic Response to Nickel Containing Dental Alloys, " J. Dent. Res.,56B: B78, Abstract no. 107, 1977.

60. Moller, H. "Nickel Dermatitis," Contact Dermatitis, 6:19,1980.

61. Mueller, H.J. et al. "Corrosion by External Polarization by Soldered Orthodontic Wires in Cleanser Solutions," Am. J. Orthod.,76:555,1979.

62. Nater, J.P. "Possible Causes of Chromate Eczema," Dermatologica , 126:160,1963.

63. North American Contact Dermatitis Group,1972. "Epidemiology of Contact Dermatitis in North America," Arch. Dermat.,108:537,1973.

64. Olefjord,I. "Passive State of Stainless Steels," Mater. Sci. Eng. 42:161,1980.

65. Ovrulskii, G.O. "Allergy to Chrome from Wearing Steel Dental Prostheses," Stomatologica,55:60,1976.

66. Pegum, J.S. "Nickel Allergy," Lancet, 1:674,1974.

67. Peltonen, L. "Nickel Sensitivity in the General Population," Contact Dermatitis, 5:27,1979.

68. Peterson, K.B. "Longitudinal Root Fracture due to Corrosion of an Endodontic Post," J. Cand. Dent. Assoc., 2:66,1971.

69. Polak, L. et al. "Studies on Contact Hypersensitivity to Chromium Compounds," Prog. Allergy, 17:145,1973.

70. Povich, M.J. and Rao, P. "Low Temperature Sensitization of Welded Type 304 Stainless Steel," Corrosion,34:269,1978.

71. Prystowsky, S.D. et al. "Allergic Contact Hypersensitivity to Nickel, Neomycin, Ethlylenediamine, and Benzocaine: Relationship between Age, Sex, History of Exposure, and Reactivity to Standard Patch Tests and Use Tests in a General Population," Arch.Dermatol., 115:959,1979.

72. Rickles, N.H. "Allergy in Surface Lesions of the Oral Mucosa," Oral Surg., Oral Med., Oral Path., 33:744,1972.

73. Roed-Petersen, B. et al. "Nickel Allergy and Osteomyelitis in a Patient with Metal Osteosynthesis of a Jaw Fracture," Contact Dermatitis, 5:108,1979.

74. Rogers, O.W. "A Study in the Control of Crevice Corrosion of Silver Soldered Stainless Steel Joints," Brit. Dent. J.,143:397,1977.

75. Samitz, M.H. "Studies of Chromate Dermatitis," AMA Arch. Ind. Health, 14:269,1956.

76. Samitz, M.H. and Gross, S. "Effects of Hexavalent and Trivalent Chromium Compounds on the Skin," Arch. Dermat. ,84:94,1961.

77. Samitz, M.H. and Katz, S.A. "Nickel Dermatitis Hazards from Prosthesis. In Vivo and In Vitro Solubilization Studies," Brit. J. Dermat., 92:287,1975.

78. Scales, J.T. et al. "Corrosion of Orthopaedic Implants: Screws, Plates, and Femoral Nail Plates," J. Bone Joint Surg., 41B:810,1959.

79. Scales, J.T. et al. "Corrosion of Orthopaedic Implants," Brit. Med. J., 2:478,1961.

80. Schoonover, I.C. and Souder, W. "Corrosion of Dental Alloys," J. Am. Dent. Assoc., 48:1278,1941.

81. Schriever, W. and Diamond, L.E. "Electromotive Forces and Electric Currents Caused by Metallic Dental Fillings," J. Dent. Res.,31:205,1952.

82. Schriever, W.R. et al. "Allergic Response to Stainless Steel Wire," Oral Surg.,42:578,1976.

83. Silness, J. et al. "Distribution of Corrosion Products in Teeth Restored with Metal Crowns Retained by Stainless Steel Posts," Acta. Odont. Scand.,37:317,1979.

84. Simonsen, T.L. et al. "A Follow-up Study of Post-or Pin Retained Amalgam Filings with Special Reference to Possible Corrosion," Scand. J. Dent. Res.,81:415,1973.

85. Simpson, W. and Carter, J.M. "Metallurgical and Histological Investigations Following the Interosseous Wiring of a Fractured Mandible," Dent. Practit., 17:120,1966.

86. Smith, D.C. "A Review of the Potential Hazards of Nickel and its Alloys in Dentistry," Unpublished review, Johnson and Johnson Dental Products Co., New Jersey, 1978.

87. Solomon, H. et al. "Salivary Influence on Galvanism," Dent. Items Interest,60:1047,1938.

88. Soremark, R. "Some Biological Effects Caused by Prosthetic Materials," Swed. Dent. J., 3:1,1979.

89. Stoewsand, G.S. "Chromium and Nickel in Acidic Foods and By-Products Containing Stainless Steel During Processing," Bull. Envirn. Contam. Toxicol., 21:600, 1979.

90. Sutow, E.J. et al. "An In Vitro Investigation of the Anodic Polarization and Capacitance Behavior of 316L Stainless Steel," J. Biomed. Mater. Res., 10:671,1976.

91. Sutow, E.J. "The Influence of Electropolishing on the Corrosion Resistance of 316L Stainless Steel," J. Biomed. Mater. Res., 14:587,1980.

92. Symeonides, P.P. et al. "An Allergic Reaction after Internal Fixation of a Fracture Using Vitallium Plate," J. Allergy Clin. Immunol. 51:251,1973.

93. Tani, G. and Zucchi,F. "Electrochemical Evaluation of the Corrosion Resistance of Commonly Used Metals in Dental Prosthesis," Minerv. Stomatol.,16:710,1967.

94. Thorman, J. et al. "Persistence of Contact Allergy to Chromium," Contact Dermatitis, 5:261,1979.

95. Thrope, P.H. "Bacterial Corrosion of Stainless Steel Caused by Water Recycling," Austral. Corros. Assoc. 18th Conf.,Parkville, Victoria, Aust.,1978.

96. Tilsley, D. and Rotstein, H. "Sensitivity Caused by Internal Exposure to Nickel, Chrome and Cobalt," Contact Dermatitis, 6:175,1980.

97. Uhlig, H.H. Corrosion and Corrosion Control, John Wiley and Sons, Inc., New York, 1971.

98. Venugapal, B. and Luckey, T.D. Metal Toxicity in Mammals·2. Chemical Toxicity of Metals and Metalloids, Plenum Press, New York, 1978.

99. von Fraunhofer, J.A. and Staheli, P.J. " Gold Amalgam Galvanic Cells. The Measurement of Corrosion Currents," Brit. Dent. J. ,132:357,1972.

100. Watt, T.L. and Baumann, R.R. "Nickel Earlobe Dermatitis," Arch. Dermat., 98:155,1968.

101. Williams, D.F. and Meachim,G. "A Combined Metallurgical and Histological Study of Tissue-Prostheses Reactions in Orthopaedic Patients," Annual Internat. Biomater. Symp. No. 5 (Part I):1,1974.

102. Winter, G.D. "Tissue Reaction to Metallic Wear and Corrosion Products in Human Patients," J. Biomed. Mater. Res. Symp. No. 5 (Part 1):11, 1974.

103. Wright, J.K. and Axon, H.J. "Electrolysis and Stainless Steel in Bone," J. Bone Joint Surg., 38B:745,1956.

104. Zachrisson, B.U. "Gingival Condition Associated with Orthodontic Treatment II. Histologic Findings," Angle Orthod., 42:352,1972.

105. Zapffe, C.A. " Human Body Fluids Affect Stainless Steel," Metal Prog., 68:95,1955.

TABLE I

Cumulative Amounts of Nickel and Chrome Released from Orthodontic Appliance After 12 Days in Sodium Chloride Solution

Group	Nickel	Chrome
	ug Soluble Ni and Cr	
Control	0	0
Static Immersion	121 ± 69*	40 ± 28
Shaking Immersion	154 ± 87	13 ± 17
	ug Precipitated Ni and Cr	
Control	0	0
Static Immersion	4 ± 2	72 ± 40

*mean ± S.D. (N = 10), all means significantly different from controls at $P < 0.05$

Table II

Static Immersion Vs. Shaking Immersion, Cumulative 12 Day Soluble Nickel and Chrome

	Static Immersion,N=10	Shaking Immersion,N=10	
	$\bar{X}$	$\bar{X}$	t,df=9
ug cumulative soluble Nickel	121	154	0.93^{NS}
ug cumulative soluble chrome	40	13 assumed minimum	2.61^{**}
ug cumulative soluble chrome	40	36 assumed maximum	0.46^{NS}

NS=Not Significant
**P < .05

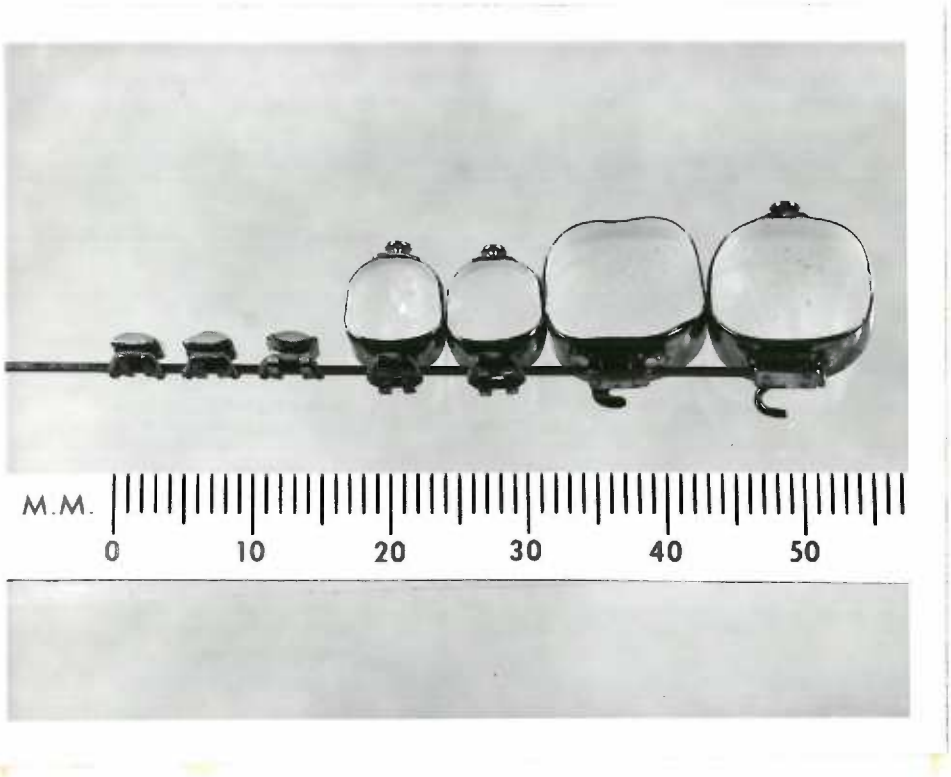

Fig. 1: Simulated Half-Mandibular Arch

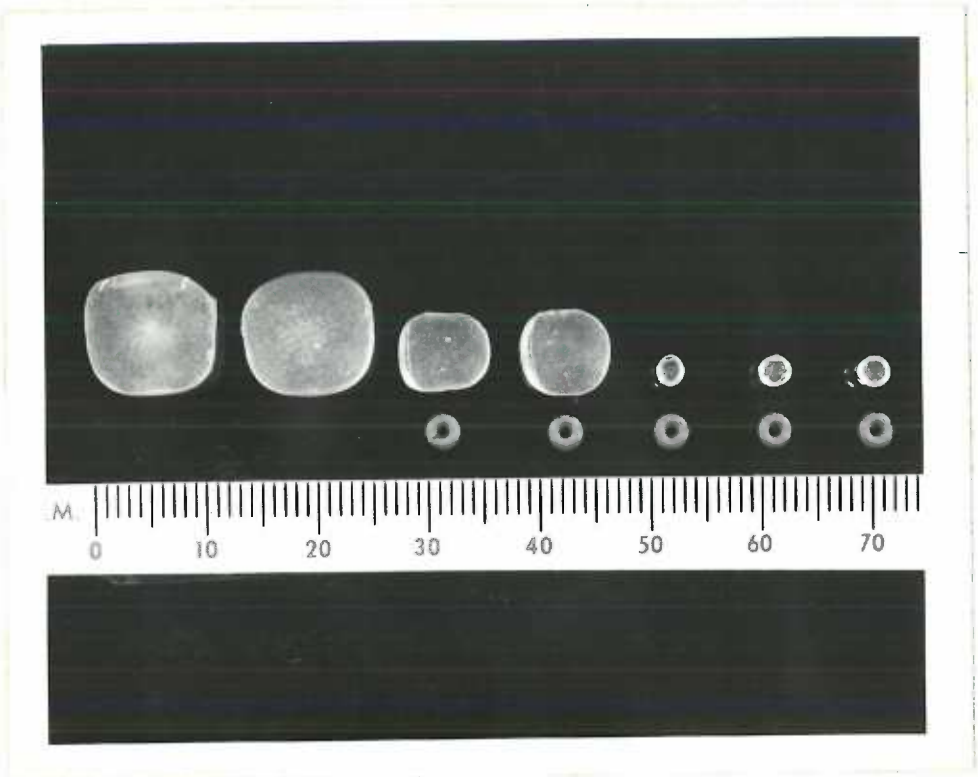

Fig. 2: Control

Fig. 3: Atomic Absorption Spectrophotometer

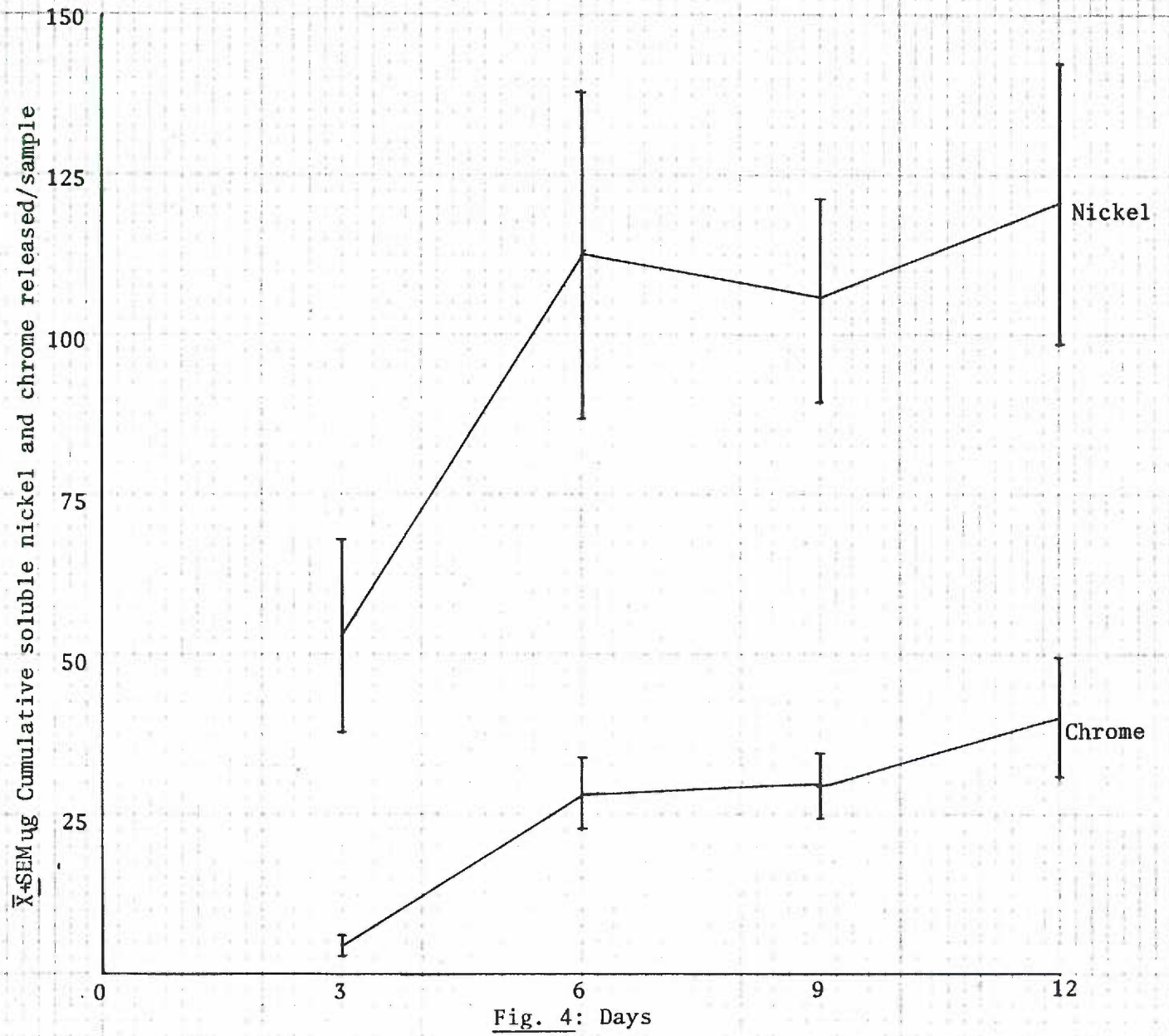

Fig. 4: Cumulative Release of Soluble Nickel and Chrome with Static Immersion Vs. Time

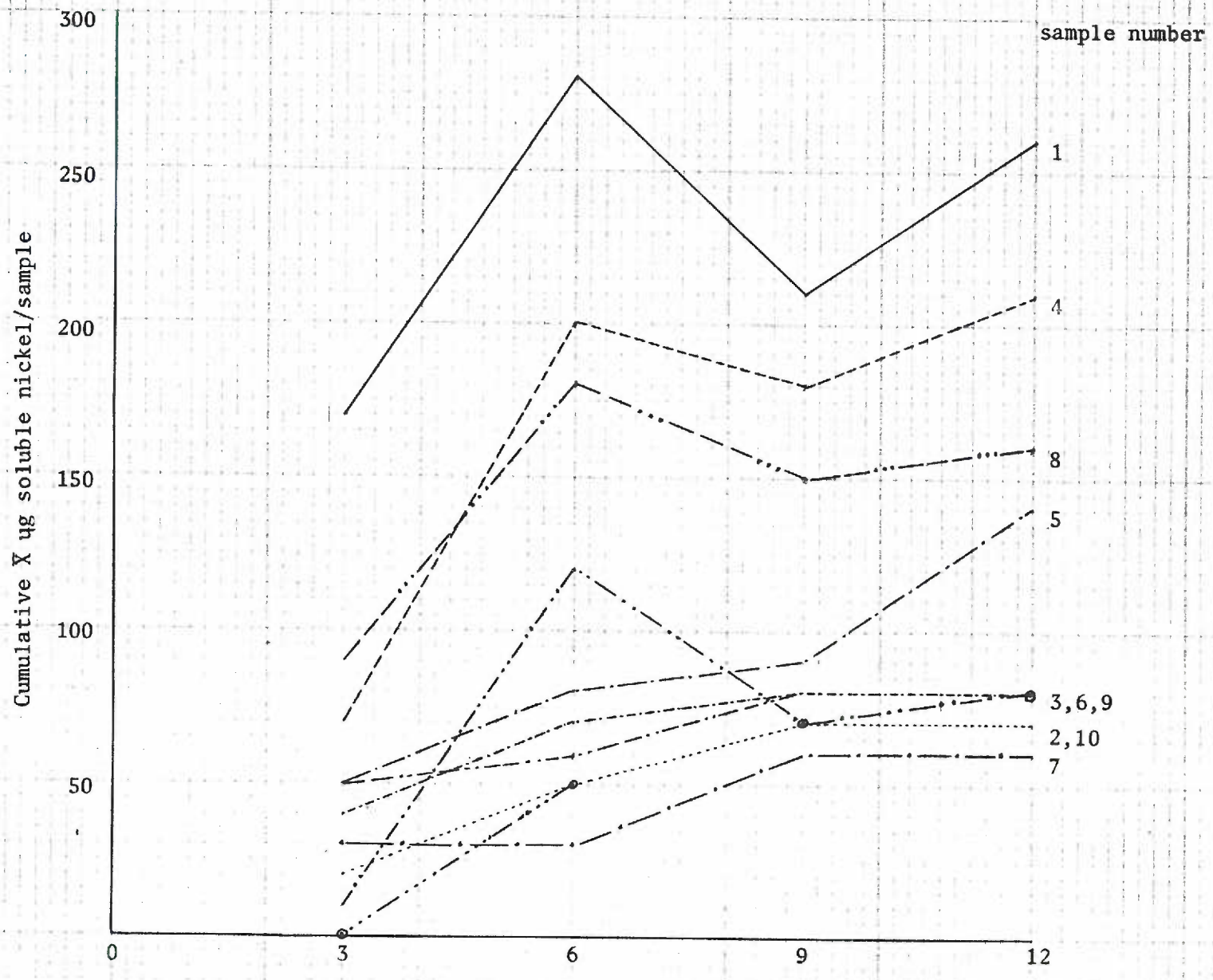

Fig.5 Days

Release of Soluble Nickel with Static Immersion Vs. Time

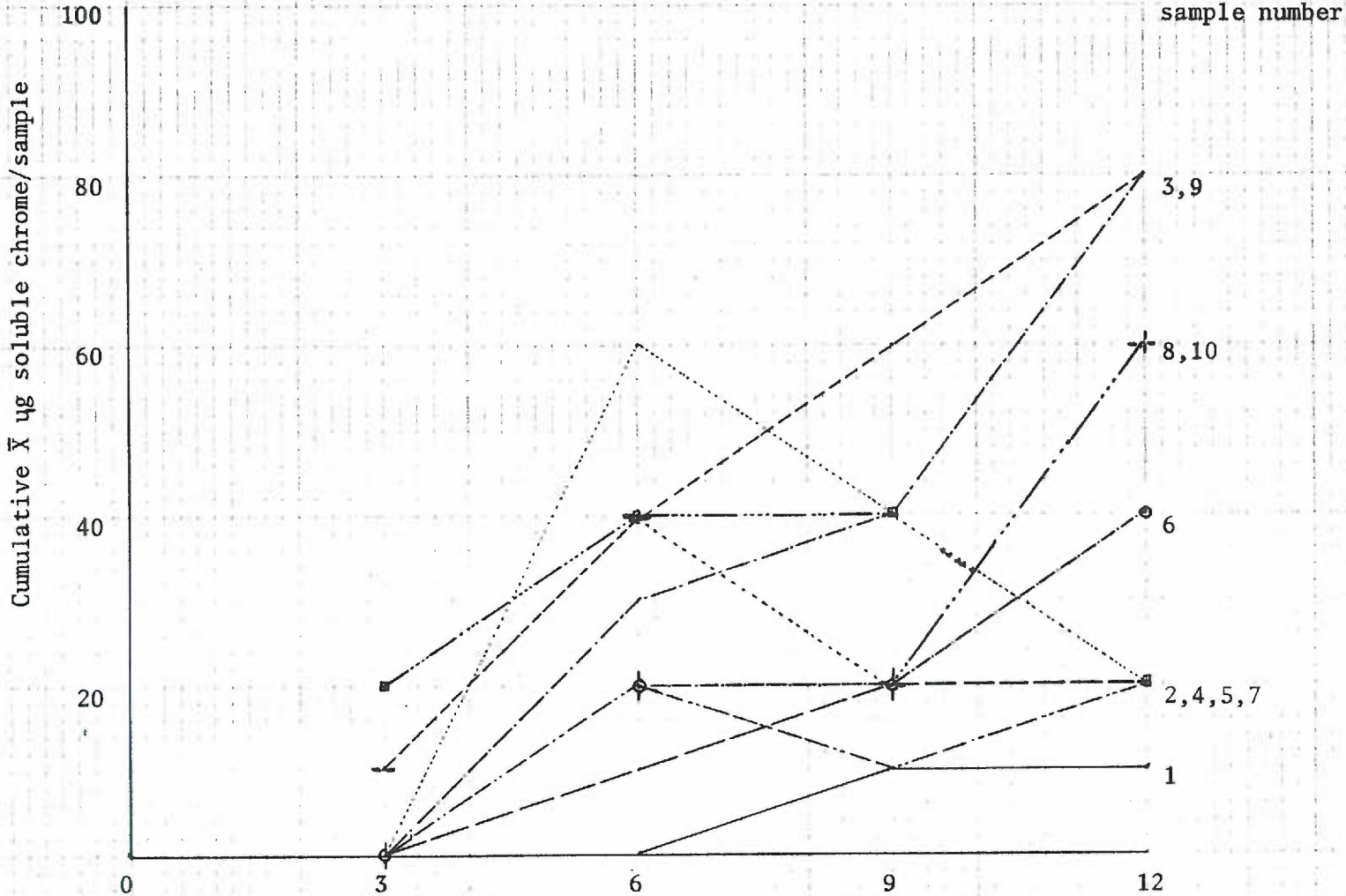

Fig.6: Days

Release of Soluble Chrome with Static Immersion Vs. Time

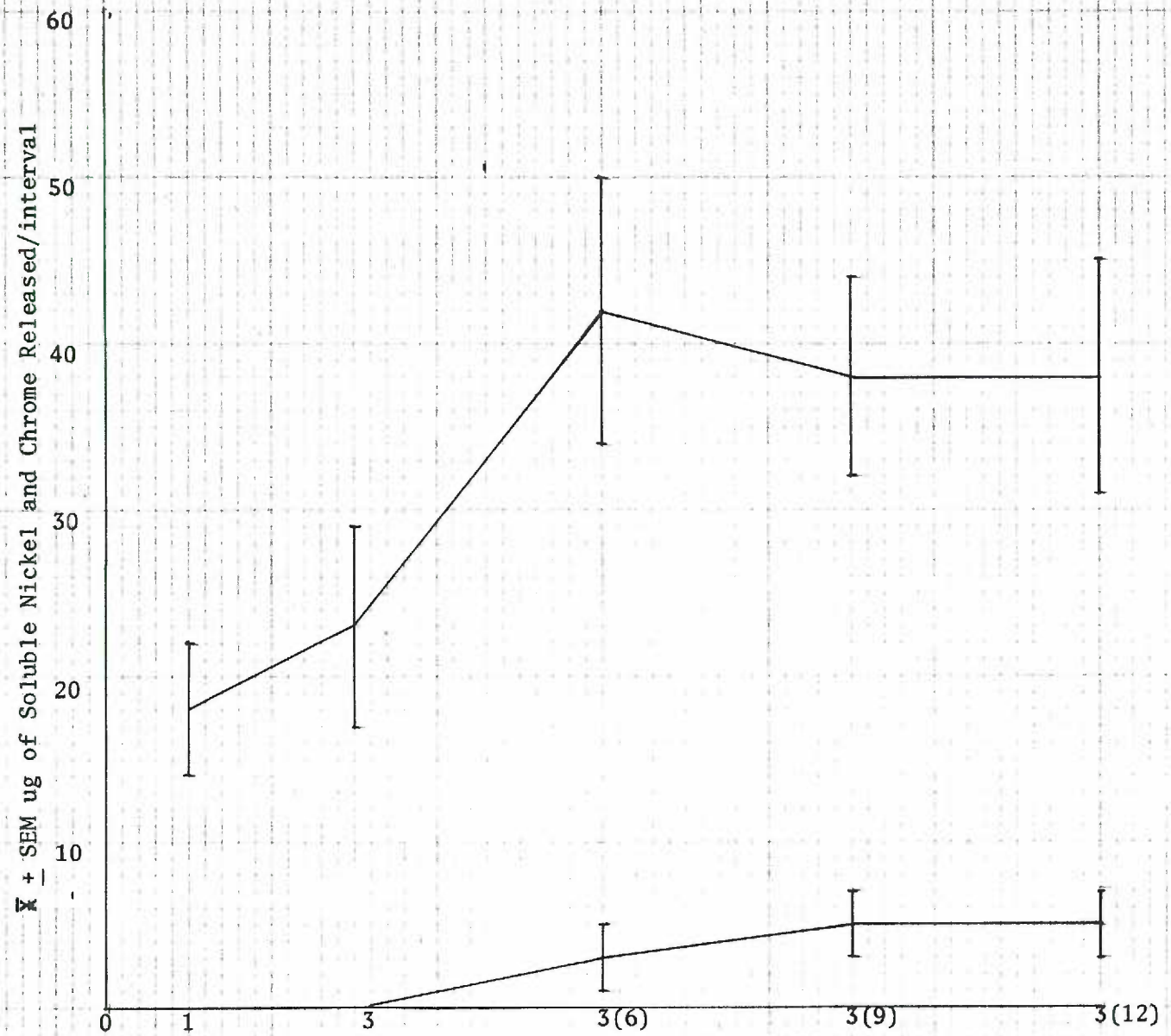

Fig.7: Release of Soluble Nickel and Chrome with Shaking Immersion Vs. Time

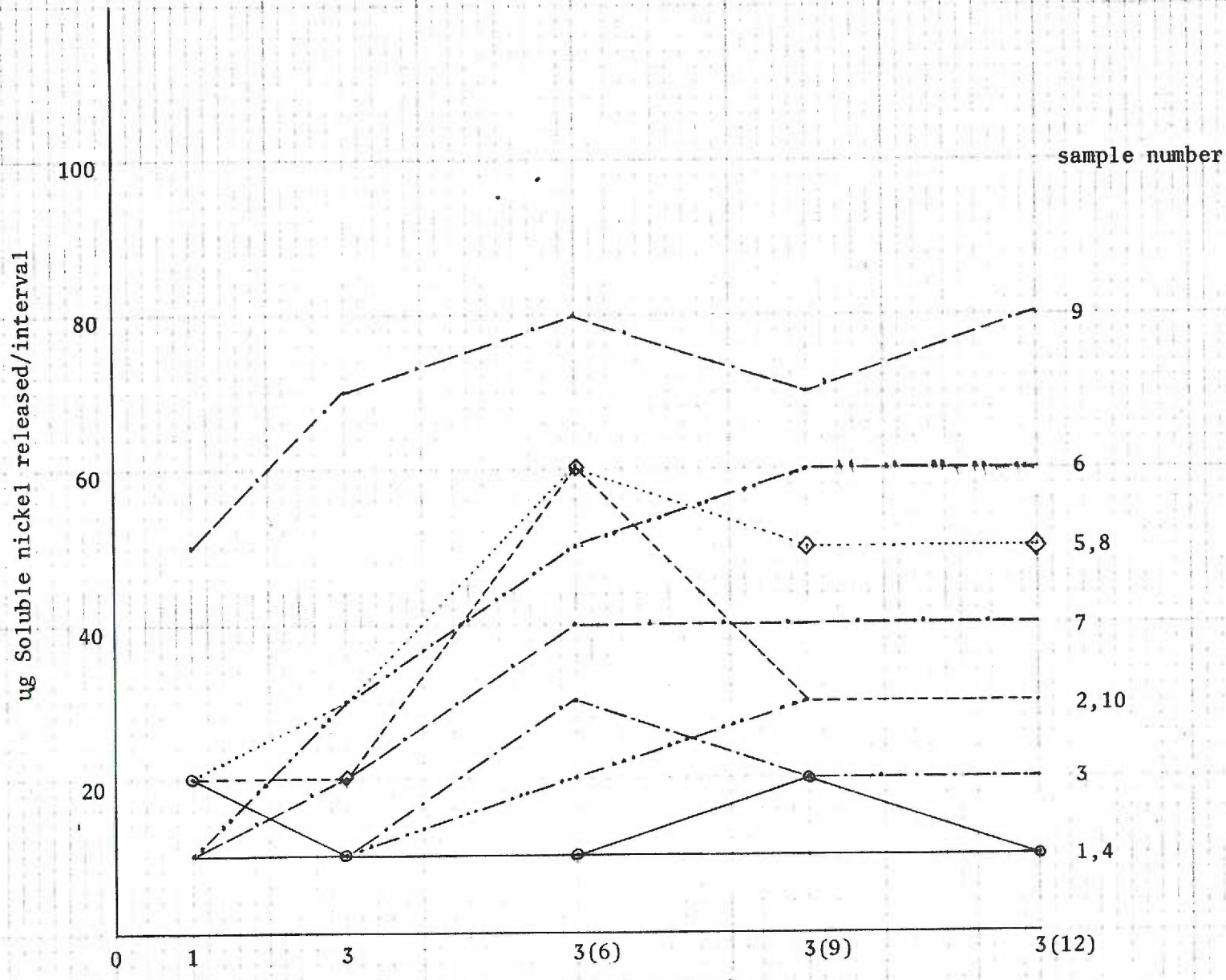

Fig.8: Soluble Nickel Released with Shaking Immersion Vs. Time

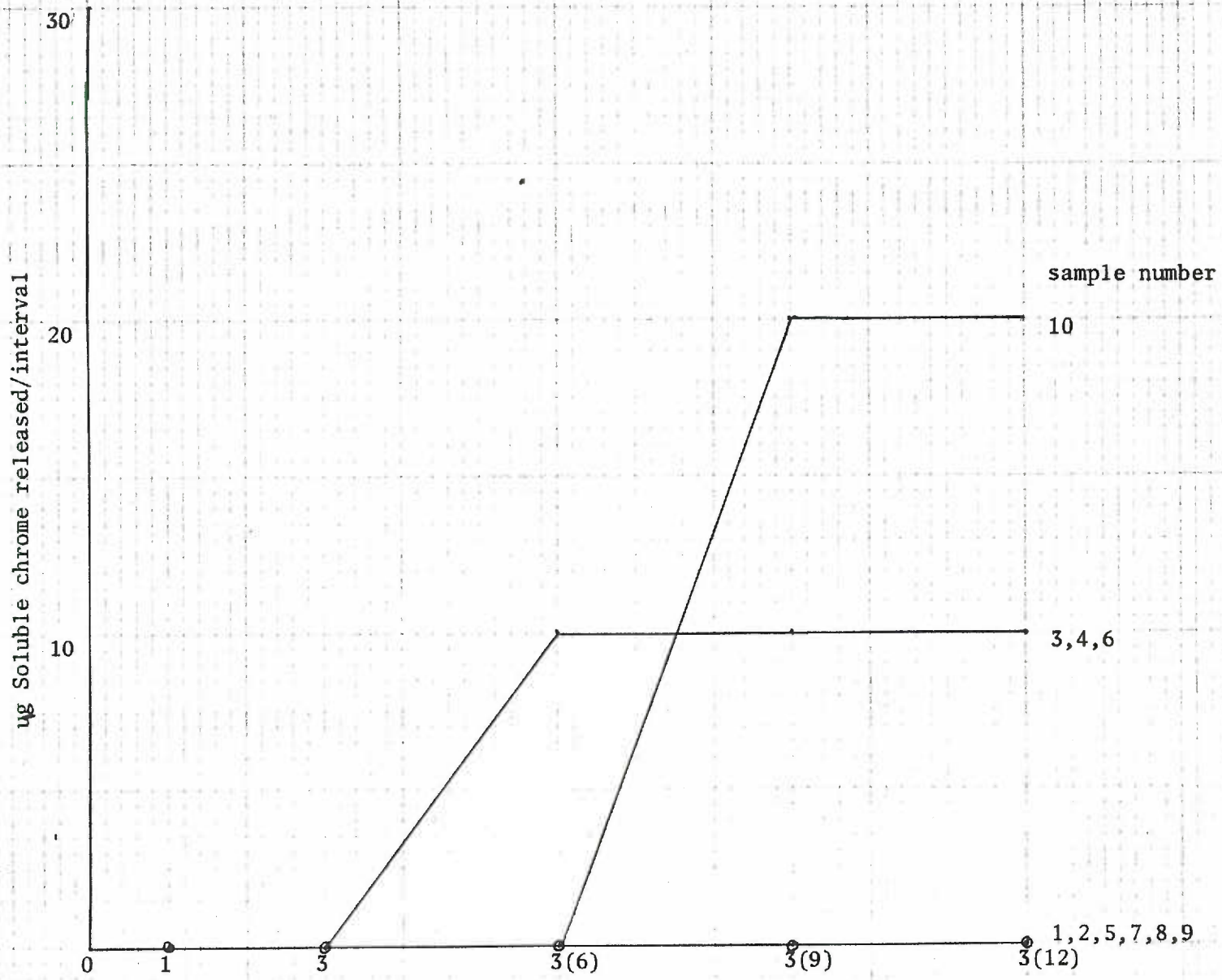

Fig. 9: Daily interval (total days)

Soluble Chrome Released with Shaking Immersion Vs. Time

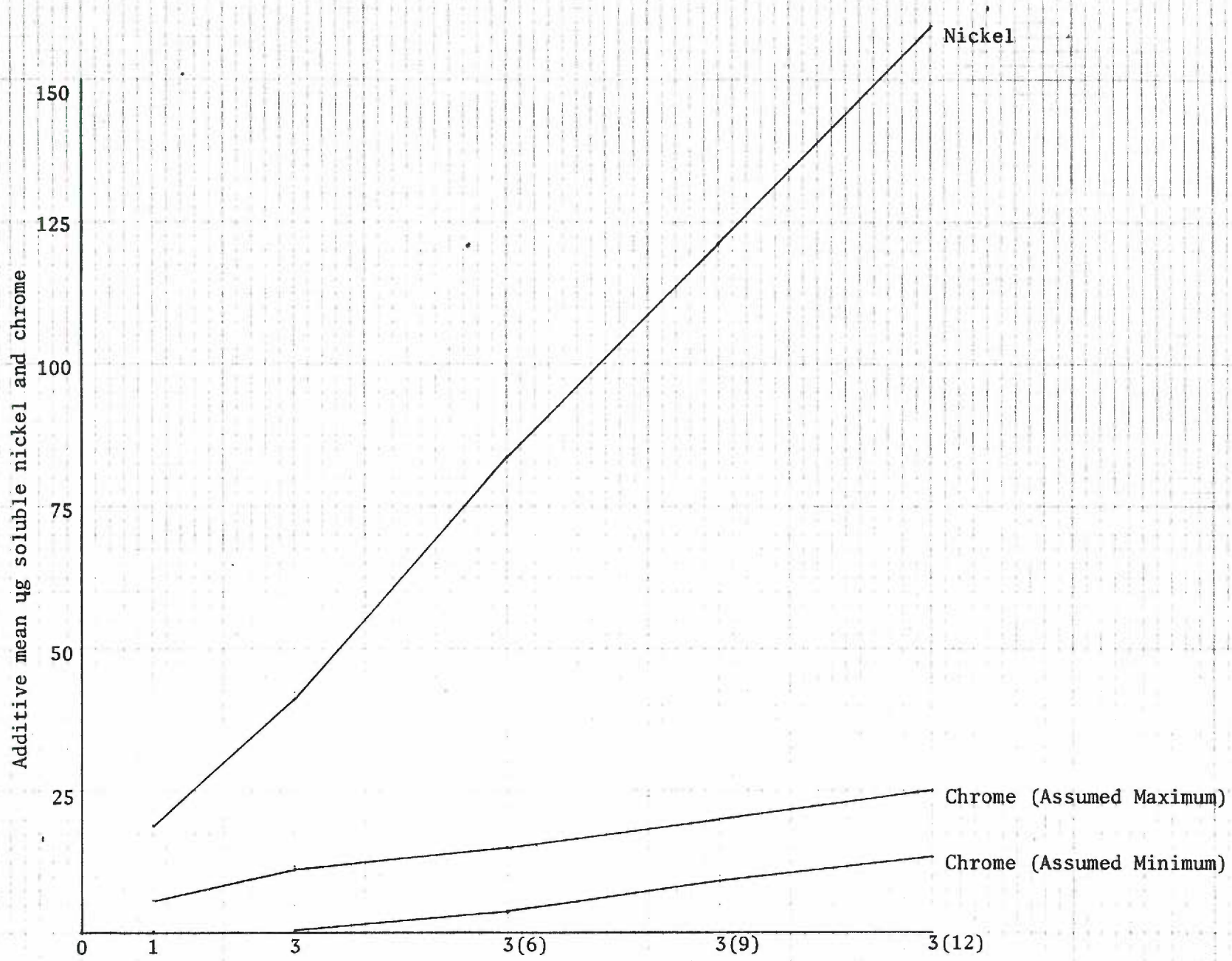

Fig.10: Additive Soluble Nickel and Chrome Released with Shaking Immersion Vs. Time