# REVIEWS

Dent. Med. Probl. 2010, **47**, 4, 465–471
ISSN 1644-387X



Joanna Antoszewska, Agnieszka Sieja, Michał Sarul

# Heavy Metals: Lead, Cadmium and Nickel Polluting the Environment Versus Danger of Orthodontic Patients – Review of the Literature

## Skażenie środowiska metalami ciężkimi: ołowiem, kadmem i niklem zagrożeniem dla pacjenta leczonego ortodontycznie – przegląd piśmiennictwa

Department of Dentofacial Orthopedics and Orthodontics, Wroclaw Medical University, Poland

### Abstract

Plumbism and cadmium poisoning are a combination of a symptom complex responsible for metal-intoxications. The express of allergy during treatment with fixed appliances usually results from nickel influence. Symptoms of nickel allergy, visible on oral mucosa, follow nickel-release during orthodontic treatment. Furthermore, additional factors, such as diet, nicotine, and individual sensibility may intensively promote contribution of heavy metals in the etiology of serious illness. Cadmium and lead intoxication symptoms are widely discussed in the literature. Cadmium and lead also influence the prevalence of caries and periodontal diseases. The aim of the study was to present the literature review concerning either oral environment (accumulated cadmium and lead reservoirs influencing health status of oral cavity) or expression of hypersensitivity to nickel as the factors potentially endangering orthodontic patients treated with fixed appliances. Heavy metals such as cadmium, lead and nickel take part in chemical reactions simultaneously damaging functions of the organism. Subsequently, designation of accumulation of cadmium and lead in the oral cavity as well as observation symptoms of hypersensitivity to nickel may play a significant role in marking the level of environment contamination by heavy metals. Although individuals particularly endangered are those working with alloys and battery production, permanently increasing interest towards treatment of malocclusion makes orthodontic patients jeopardized as well, especially these surrounded by environment polluted with either lead or cadmium (**Dent. Med. Probl. 2010, 47, 4, 465–471**).

**Key words:** environmental contamination, cadmium, lead, nickel, contact allergy.

### Streszczenie

Ołowica i kadmica to złożone zespoły objawów toksycznego oddziaływania ołowiu i kadmu. Alergia pojawiająca się podczas leczenia ortodontycznego jest zwykle rezultatem oddziaływania niklu. Objawy alergii na nikiel mogą występować na błonie śluzowej jamy ustnej u pacjentów leczonych aparatami stałymi. Dodatkowe czynniki, takie jak: dieta, palenie tytoniu i indywidualna wrażliwość, mogą ponadto wzmacniać rolę metali ciężkich w etologii poważnych chorób ogólnych. Toksyczne objawy kadmu i ołowiu są szeroko omawiane w literaturze. Uważa się, że zarówno kadm, jak i ołów mają wpływ na rozwój próchnicy i choroby przyzębia. Celem pracy jest przegląd literatury dotyczącej stanu jamy ustnej, rezerwuaru dla kadmu i ołowiu mających wpływ na zdrowie jamy ustnej, oraz nadwrażliwości na nikiel jako czynników, na które potencjalnie są narażeni pacjenci leczeni aparatami stałymi. Metale ciężkie, takie jak: kadm, ołów lub nikiel, biorą udział w reakcjach chemicznych stymulujących uszkodzenie organizmu. Oznaczanie akumulacji kadmu i ołowiu w jamie ustnej oraz obserwowanie objawów nadwrażliwości na nikiel może odgrywać ważną rolę w określaniu poziomu kontaminacji środowiska metalami ciężkimi. Chociaż bezpośrednie narażenie występuje u osób pracujących przy produkcji baterii, to jednak stałe zwiększanie zainteresowania leczeniem wad zgryzu sprawia, iż pacjenci leczeni ortodontycznie są także narażeni, szczególnie ci żyjący w otoczeniu zanieczyszczonym ołowiem i kadmem (**Dent. Med. Probl. 2010, 47, 4, 465–471**).

**Słowa kluczowe:** zanieczyszczenie środowiska, kadm, ołów, nikiel, alergia kontaktowa.

Among heavy metals: Cu, Cr, Cd, Fe, Zn, Pb, Sn, Hg, Mn, Ni may be listed, divided into microelements (cooper, zinc, nickel and chromium) and trace elements toxic in minimal concentration (cadmium, mercury and lead). However, accumulation of the latter ones in different parts of the human body negatively influences living organisms. Either lead or cadmium intoxications, together with cigarette smoke, have the spectrum of symptoms widely described in the literature and are considered as a very strong carcinogen. Exposure to heavy metals occurs in different environments: human humors transfer cancerogenic elements and this arises questions:

1) may open space of the oral cavity also constitute the reservoir of toxins, thus making teeth the reliable indicators of overexposure to negative environmental influence?

2) do levels of common carcinogens influence the development of particular pathologic syndromes in the oral cavity?

Release of toxins possibly accumulated in the oral cavity requires major attention, especially in orthodontic patients subjected either to eventual environmental pollution or to hypersensivity to nickel: strong allergen released from elements of fixed appliances [1–6].

Literature review concerning either oral environment as accumulated cadmium and lead reservoirs influencing health status of oral cavity, or expression of hypersensivity to nickel as the factors potentially endangering orthodontic patients treated with fixed appliances.

## Environmental Factors Influencing Accumulation of Heavy Metals in Humans

Tracing environmental pollution, Początek et al. measured the concentrations of various ions: calcium, magnesium, fluorine, phosphorus in the form of phosphates, potassium, sodium, iron, zinc, copper, cadmium, and lead in specimens of body fluids: blood, urine and saliva of employees working in three different industries: Institute of Rolling Stock Repairs (Zakłady Naprawcze Taboru Kolejowego), Philips Lighting Poland and Metalplast. The levels of ions were also evaluated in 100 teeth extracted in the employees. Applying measured spectrophotometrical measurement (ASA), fluorine with an ion-selective electrode, and phosphates with a colorimetric method, authors didn't find any statistical differences in concentration of cadmium and lead neither in teeth nor in humor fluids of the examined individuals [7].

Galikeeva et al. [8] analysed dental status and heavy metal concentration in organisms of oil drillers and driller assistants. Authors noted high prevalence of periodontal disorders among the examinees, as well as the excessive amount of metal ions, such as cadmium and nickel in the peripheral blood vessels and saliva.

On the other hand, Ursinyova and Masanova [9] analyzed level of lead, cadmium and mercury in breast milk of 158 healthy, lactating women from Slovak Republic. The examinees neither lived in polluted regions, nor were occupationally exposed to influence of heavy metals. Their levels were evaluated in relation to selected parameters, such as: mother's age, number of mother's filled teeth, newborn's gender and birth weight, as well as smoking habits in the family. Authors found mercury and lead levels exceeding the normal values only in one and two breast milk samples respectively. Nonetheless, breast milk samples of either active/passive female smokers' as well as their amalgam tooth-fillings significantly increased levels of lead and mercury respectively.

Considering cigarette smoke as the source of cadmium and lead, Malara et al. [10] examined different teeth as potential reservoirs of the cancerogenic elements. In consecutive research, either deciduous or wisdom teeth were evaluated in passive and active smokers respectively; control groups comprised teeth of non-smoking individuals. Concentration of cadmium, copper, iron, manganese, lead, zinc, calcium and magnesium were analyzed in 386 extracted deciduous teeth collected from the young passive smokers (205) and control individuals (181). In this study, atomic absorption spectrometer with flame atomization allowed detection of significantly higher levels of cadmium, copper, lead and zinc in teeth of children exposed to cigarette smoke. The achieved result was in accordance with the previous one, obtained after measurement of ratios of cadmium/calcium and lead/calcium concentration in deciduous teeth collected from 13-year-old children, either exposed (37 individuals) or unexposed (34 ones) to cigarette smoke. Passive smokers' teeth contained significantly higher ratios of cadmium/calcium and lead/calcium concentration [11]. Cigarette smoke exposure possibly influencing cadmium and lead levels in wisdom teeth was also investigated. Material consisted of 127 retained wisdom teeth (65 from smokers and 62 from non-smokers). Cadmium and zinc contents determined by means of atomic absorption spectrometry turned out to be higher in smokers' teeth comparing with non-smokers' ones [12].

Bayo et al. searched for correlation of lead and cadmium contents in deciduous teeth of children from Cartagena, with some environmental and physiological factors: parental social-economic status, home antiquities and zone of residence, child's habits, age and sex, as well as tooth-related factors (presence of caries, type, weight and location). Home antiquities, nail biting habit and tooth location significantly correlated with increased lead level, whereas zone of residence displayed significant ineraction with increased cadmium accumulation in teeth. Multiple analyses revealed tooth-type as the only variable significantly connected with increase of both heavy metal levels – the highest in incisors and the lowest in molars [13].

Similarly aimed research was conducted by Tvinnereim et al. [14] who studied influence of various factors upon concentration of lead, cadmium, zinc, and mercury ions in primary teeth. Material comprised powder from more than 1754 teeth without fillings: 1200 samples analyzed for presence of lead, zinc and cadmium and 554 analyzed for presence of mercury. Samples were divided into sub-groups according to the tooth-position in dental arch, developed carries and degree of root resorbtion. Authors found significantly higher lead, mercury and zinc concentrations in carious teeth comparing with non-carious ones; teeth with the roots still present contained higher lead and zinc concentrations, if compared with the rootless ones. The achieved findings allowed conclusion: metal concentrations in primary teeth were affected either by presence of caries and roots or by tooth-type: front or posterior one.

In turn, Alomary et al. [15] analyzed factors influencing the concentration of cadmium and lead in permanent teeth. Research material comprised 268 permanent teeth of individuals living in four different cities of Jordan. Using atomic absorption spectrometry, authors proved that concentration of lead and cadmium in permanent dentition was affected by: presence of amalgam fillings, smoking habit and place of living.

## Toxicity of Accumulated Heavy Metals Versus Tooth Supporting Structures

Issa et al. [16] assessed the variable concentrations of several metal salts on human oligodendrocyte MO3.13 and human gingival fibroblasts HGF. Cytotoxicity was measured as mitochondrial dehydrogenase activity assessed by MTT assay (colorimetric assay with use of 3-(4,5-Dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide). Authors proved that cadmium and mercury produced the highest cytotoxic effects on MO3.13 cells and gingival fibroblasts HGF respectively. Such results enabled to consider both metal ions as potential risk factors jeopardizing periodontal support. Alike Arora et al. [17] who examined the relationship between environmental cadmium exposure and periodontal disease in USA adults. Authors analyzed data of 11412 individuals, gained from archieves of the third National Health and Nutrition Examination Survey (NHANES III). Statistical analyses showed that a three-fold increase in creatinine corrected urinary cadmium concentrations was associated with 54% greater odds of prevalent periodontal disease. Arora et al. [18] also examined correlation between high cadmium urinary concentration and dental carries in group of 5201 children, 6–12 years of age. Of these children, 2315 and 2886 had dental caries and fillings recorded in deciduous and permanent teeth respectively. Cadmium concentration in urine was quantified by Zeeman effect graphite furnace atomic absorption spectrophotometry; exposure to cigarette smoke was also assessed. Statystical analysis revealed, that high urine concentration correlated with higher prevalence of carries in deciduous dentition and was particularly apparent in group of children not exposed to cigarette smoke. Authors presumed that this effect might be associated with high concentration of harmful substances in cigarette smoke, possibly also caries--promoting. However, no correlation was found between cadmium concentration in urine and caries prevalence in permanent dentition.

Saraiva et al. [19] studied an influence of exposition on lead ions on the frequency of periodontitis in population of adult citizens of United States of America. Third National Health and Nutrition Examination Survey supported database for retrospective analysis of 2500 men and 2399 women, 20–56-year-olds, who received complete periodontal examination. It turned out that blood lead level higher than > 7 µg/dL was positively and statistically associated with periodontitis in both sexes. Dye et al. [20] analysing data obtained from the archives of NHANES III, assessed the influence of high blood lead levels and alveolar bone loss attributed to periodontal disease. For analysis, data of 10333 individuals aged 20–69 years, were collected. Study showed that dental furcations were the best periodontal bone loss indicators for lead levels.

## Nickel – Toxicity and Immunization of Oral Environment in Orthodontic Patients

Nickel, in low concentration, is an essential element for human body: its deficiency makes liver use less oxygen and increase accumulation of fats. Nickel activates some enzymes, increases hormonal activity and plays significant role in lipid metabolism. Almost all nickel present in blood is situated in serum, however lungs, liver, kidneys and intestines are the reservoirs of highest element concentration. Nickel levels in either blood plasma or urine are good indicators of environmental and work related exposure to the element [21–26]. Severe nickel poisoning is very rare. The most toxic component is nickel carbonyl entering organism through respiratory tracks and partially through skin, thus acutely irritates oral mucosa and air passages; liver and central nervous system damage may also develop [21]. In severe inhalatory poisonings, dizziness, headaches, nausea, vomiting, the feeling of "heavy chest", cough and shallow breath may appear. The symptoms can lead to the development of toxic pulmonary oedema, as well as malignant neoplasm of nasal sinuses and lungs [27]. The mechanism of carcinogenic activity still remains unclear. It is said that nickel disturbs DNA synthesis and causes inhibition of its repair. Metal ion activity of nickel results in catalysis of molecular oxygen conversion. Similarly, other chemical compounds which stimulate phagocytes to produce free oxide radicals cause damages to DNA structure and also protein and lipids. These processes may lead to carcinogenic alterations. Nonetheless, evidence based carcinogenic activity of nickel oxide, crystal sulphides, nickel hydroxide, and metallic nickel in either animals or humans require further research.

Properties of nickel make it miscellaneously applicable in chemical industry, food industry, production of steel, alloys and surgical instruments. Emission of nickel to the atmosphere in the process of burning coal and liquid fuel is a very significant source of environment contamination. Polluted air, water and food result in occurrence of hypersensivity with predominant contact allergy. According to Gall-Coombs, it is the type IV allergy described as cellular immunity or delayed hypersensitivity [28]. It is triggered by sensitized lymphocytes T, at the lack of circulating antibodies. Once allergic host has encountered antigen repetitively, the population of proper lymphocytes T becomes larger. Within 24–48 hours immune response: local inflammatory reaction takes place [29]. One can distinguish three groups of contact allergens: a) very rare allergens present in cosmetics, plastic, work tools, b) new allergens, to which hypersensitivity is still increasing, present in a considerable group of patients: golden jewellery, gold based prosthetic crowns and corticosteroids, c) known allergens which cause skin lesions in patients suffering from eczema. Chromium compounds, reported as the strongest allergens in Poland in 1975–1979, and nickel that has been considered the most powerful sensitizer, belong to the last group [30].

Iatrogenic exposure to nickel may be caused by: a) implants containing nickel (heart valves, joints, dentures), b) fluids used intravenously that can be contaminated with nickel, c) alloy components used in dentistry. Utilization of nickel is mandatory for fabrication current orthodontic materials: shape-memory feature makes nickel containing archwires the only ones suitable for biologically compatible levelling. Furthermore, typical steel utilized in production of orthodontic brackets and bands contains 8–12% of nickel [26]. Kerosuo et al. [31] have shown high content of nickel in nickel-titanium brackets, reaching level of 45% of the alloy. However, nickel released to the oral environment may result in either electrogalvanic processes or allergic response.

Alloys subjected to saliva together with metal ions submerged in this electrolyte create a galvanic cell [32, 33]. Stimulation of tissues with electric impulses accompanied by poisoning or allergic effect of metals cumulated in organism result in an ailment of elctrogalvanic nature [34]. Symptoms of ailment appear locally in the form of: periodontitis, inflammation of oral mucosa, glossitis, xerostomia, susceptibility to ulceration of oral cavity, leucoplakia, allergic reactions and secondary caries [35, 36]. Tongue or oral mucosa burning, metallic taste in mouth or neuralgia may also occur. Base metal ions released in the process of corrosion may result in systemic symptoms such as: irritation of oesophagus, disruption of circulatory, headache-incidents, dizziness, impairment of concentration.

Nickel and its salt cause irritation of conjunctivae and nasal septum ulceration, itchy eczema mainly on hands and forearms, so called nickel pruritus. Symptoms of asthma and pneumoconiosis are also observed. It is considered that 5–13% of cases with skin eczema were caused by nickel or its components. Hand alveolar eczema determines 40–50% of allergic cases [21]. Diagnosis of allergic contact eczema requires 48–72-hour-patch testing and bases on the occurrence of exudative papules and vesicles, chronic and recurrent course of disease, pruritus, regression without leaving any visible trace or scarring. Characteristic feature of nickel allergy is the occurrence of skin changes in places remote from the areas of direct contact: general haematogenous papules rash, in accordance with nickel pruritus [37].

Analysis of evidence based hypersensitivity to nickel also showed the prevalence of incidents in individuals who had family-history of atopy [38]. Grochowski [39] studied the group of randomly chosen patients from Allergologic Clinic of the Medical University of Warsaw with diagnosed nickel allergy and skin lesions. Colour of mucosa as well as moisture, smoothness, rash-presence, condition of gums and dental defects were examined. Basing on this studies, it has been established that the appearance of skin lesions occurred always within 20 to 40 years of age, in the places of direct contact with the allergen. None of the examined patients reported the ailments of oral mucosa. Only two patients with severe skin hypersensitivity to nickel displayed the redness of palate-oral mucosa in the area of contact with nickel, as well as intensification of skin lesions and the appearance of new ones in the region of previous eczema. Such symptoms would be possibly subsequent to reaction of immune system resulting from nickel-release in the oral cavity. Long-term observations revealed neither hypersensitive reaction of oral mucosa nor intensified symptoms of skin allergy. In some cases weaker allergid was noted thus allowing assumption nickel tolerance might develop. According to Spiechowicz, there are four mechanisms or factors explaining no reaction of the oral mucosa [acc. 32]: glycoproteins of saliva constitute diffusive barrier, difference in permeability of skin and mucosa, different mechanisms of hypersensitivity of skin and mucosa, different placement and function of Langerhans cells.

Allergy to nickel ions concerns from 5.8% up to 30% of patients treated orthodontically. Long term exposure of mucosa and skin to orthodontic appliances, may cause cheilitis, and tongue hypersensitivity [40]. There are reports on contact stomatitis in patients allergic to nickel, who were treated with extraoral tractions [32]. There are reports in the literature proving golden earrings and rings consecutively to be the most allergic items among jewellery; there is also an evidence that the symptoms of allergy intensify in summer [30]. Kerosuo et al. [41] proved that 35% of individuals wearing earrings prior to orthodontic treatment displayed greater sensitivity to nickel in comparison with no-jewellery users. This study comprised 700 patients, from 14 to 18 years of age; 478 individuals passed orthodontic treatment. The reasearch revealed predominant hypersensitivity occurring in 30% of females in comparison to low hypersensitivity evident in 3% of males. Incidents of allergy occurring during orthodontic treatment appeared in 31% of individuals wearing earrings prior to orthodontic treatment, and in 2% of patients using no-jewellery. The authors suggested that the orthodontic treatment proceeding the incidence of hypersensitivity related to wearing earrings, might intensify the immunologic response.

There results of orthodontic patients' saliva examination regarding nickel are divisive.

Kocadereli et al., Singh et al. [acc. 42] and Ağaoğlu et al. [24] examined samples of saliva obtained from patients in different time spans. Comparison of patients under orthodontic treatment with untreated ones displayed different results. According to Singh et al. [acc. 42] statistically significant quantity of the element in saliva was noted just within the first months after bonding of appliances, whereas Kocadereli et al. [25], and Ağaoğlu et al. [24] reported no difference in the level of nickel ions, established in orthodontic patients after several months of treatment and in untreated individuals. Apparently different levels of nickel ions associated with lack of allergic syndromes concerning oral mucosa not only indirectly confirm thesis suggested by Spiechowicz [acc. 32], but also prove that nickel concentration necessary to cause pathological symptoms on the oral mucosa is greater than the concentration responsible for skin lesion. Nonetheless, since question, whether a small dose of nickel released during orthodontic treatment permanently impairs function of cells, still remains unanswered, treatment with nickel-free appliances should be the approach of choice in patients with hypersensitivity to the discussed element.

Developing industry, changeability of conditions of social life as well as development of technologies increase susceptibility to adverse conditions of environment. Heavy metals such as cadmium, lead and nickel, after entering organism through alimentary system, respiratory tract or skin, take part in chemical reactions simultaneously damaging functions of the organism. To mark the level of contamination of environment by heavy metals, a significant role is ascribed to bioindicators such as teeth and saliva. Cited results of studies showed that individuals inhabiting regions exposed to excessive emission of the elements from industrial sources displayed accumulation of cadmium and lead in the oral cavity, thus allowing obvious conclusion: teeth – due to the constancy of their chemical composition – may be used for comparison the effects of long term exposure to various levels of local environment contamination by heavy metals.

Frequently occurring hypersensitivity to environment becomes an equally significant problem.

Nickel is regarded to be the main contact allergen responsible for dermatitis in Western Europe. Although individuals particularly endangered are those working with alloys and battery production, permanently increasing interest towards treatment of malocclusion makes orthodontic patients jeopardized as well. Evidence based literature indicates that this problem is especially intensified in patients with very high skin hypersensitivity to nickel, therefore elements of orthodontic appliances should be manufactured from materials less prone to electrogalvanic processes, thus resulting in higher resistance to corrosion and obvious reduction of toxic metals release.

Despite evidence proving reduced occurrence of allergy in oral cavity, resulting from defensive mechanism, expression of allergic potential of nickel still remains hazardous, hence nickel-free appliances should be mandatory for treatment of malocclusion in patients with incidents of skin allergy.

## References


[1] Koizumi S., Yamada H.J.: DNA Microarray analysis of altered gene expression in cadmium-exposed human cells. Occup. Health 2003, 45, 331–334.

[2] Arora M., Weuve J., Schwartz J., Wright R.O.: Association of environmental cadmium exposure with periodontal disease in US adults. Environ. Health Perspect. 2009, 117, 739–744.

[3] Menke A., Muntner P., Silbergeld E.K., Platz E.A., Guallar E.: Cadmium levels in urine and mortality among U.S. Adults. Environ. Health Perspect. 2009, 117, 190–196.

[4] Eisinger J.: Lead and wine – Eberhard Gockel and the Colica Pictonum. Med. Hist. 1982, 26, 279–302.

[5] Needleman H.L., Schell A., Bellinger D., Leviton A., Allred E.N.: The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. N. Engl. J. Med. 1990, 322, 83–88.

[6] Bellinger C.: Very low lead exposures and children's neurodevelopment. Curr. Opin. Pediatr. 2008, 20, 172–177.

[7] Początek M., Machoy Z., Gutowska I., Chlubek D.: A comparative analysis of occupational risk in industry employees based on concentrations of some elements in teeth and body fluids. Ann. Acad. Med. Stetin. 2004, 50, 107–114.

[8] Galikeeva A.Sh., Astakhova M.R., Larionova T.K., Iakhina M.R.: Peculiarities of generalized periodontitis formation in oil extraction workers. Med. Tr. Prom. Ekol. 2008, 5, 42–45.

[9] Masanova V., Ursinyova M.: Cadmium, lead and mercury in human milk from Slovakia. Food Addit. Contam. 2005, 22, 579–589.

[10] Malara P., Kwapuliński J., Malara B., Drugacz J.: Effect of passive smoking on the level of selected metals in deciduous teeth. Przegl. Lek. 2006, 63, 1002–1006 [in Polish].

[11] Malara P., Kwapuliński J., Drugacz J.: Lead and cadmium occurrence in deciduous teeth of children exposed to cigarette smoke in apartments. Przegl. Lek. 2004, 61, 1122–1125 [in Polish].

[12] Malara P., Kwapuliński J., Drugacz J., Malara B.: Effect of cigarette smoking on coexistence of cadmium and zinc in retained wisdom teeth. Przegl. Lek. 2005, 62, 1072–1074 [in Polish].

[13] Bayo J., Moreno-Grau S., Martinez M.J., Moreno J., Angosto J.M., Guillen Perez J., Garcia M.L., Moreno-Clavel J.: Environmental and physiological factors affecting lead and cadmium levels in deciduous teeth. Arch. Environ. Contam. Toxicol. 2001, 41, 247– 254.

[14] Tvinnereim H.M., Eide R., Riise T.: Heavy metals in human primary teeth: some factors influencing the metal concentrations. Sci. Total Environ. 2000, 255, 21–27.

[15] Alomary A., Al-Momani I.F., Massadeh A.M.: Lead and cadmium in human teeth from Jordan by atomic absorption spectrometry: Some factors influencing their concentrations. Sci. Total. Environ. 2006, 369, 69–75.

[16] Issa Y., Brunton P., Waters C.M., Watts D.C.: Cytotoxicity of metal ions to human oligodendroglial cells and human gingival fibroblasts assessed by mitochondrial dehydrogenase activity. Dent. Mater. 2008, 24, 281–287.

[17] Arora M., Weuve J., Schwartz J., Wright R.O.: Association of environmental cadmium exposure with periodontal disease in US adults. Environ. Health Perspect. 2009, 117, 739–744.

[18] Arora M., Weuve J., Schwartz J., Wright R.O.: Association of environmental cadmium exposure with pediatric dental caries. Environ. Health Perspect. 2008, 116, 821–825.

[19] Saraiva M.C.P., Taichman R.S., Braun T., Nriagu J., Eklund S.A., Burt B.A.: Lead exposure and periodontitis in US adults. J. Periodontal. Res. 2007, 42, 45–52.

[20] Dye B.A., Hirsch R., Brody D.J.: The relationship between blood lead levels and periodontal bone loss in the United States, 1988–1994. Environ. Health Perspect. 2002, 110, 997–1002.

[21] Chmielnicka J.: Toksyczność metali i półmetali (metaloidów). W: Toksykologia współczesna. Red.: W. Seńczuk, Wydawnictwo Lekarskie PZWL, Warszawa 1990, 360–446.

[22] Menezes L.M., Quintão C.A., Bolognese A.M.: Urinary excertin levels of nickel in orthodontic patients. Am. J. Orthod. Dentofac. Orthop. 2007, 131, 635–638.

[23] Park H.Y., Shearer T.R.: *In vitro* release of nickel and chromium from simulated orthodontic appliances. Am. J. Orthod. 1983, 84, 156–159.

[24] Ağaoğlu G., Arun T., Izgi B., Yarat A.: Nickel and chromium levels in the saliva and serum of patients with fixed orthodontic appliances. Angle Orthod. 2001, 71, 375–379.

[25] Kocadereli L., Ataç P.A., Kale P.S., Ozer D.: Salivary nickel and chromium in patients with fixed orthodontic appliances. Angle Orthod. 2000, 70, 431–434.
[26] Huang T.H., Yen C.C., Kao C.T.: Comparison of ion release from new and recycled orthodontic brackets. Am. J. Orthod. Dentofac. Orthop. 2001, 120, 68–75.
[27] Kłopotowski J.: Metale i metaloidy. W: Toksykologia kliniczna. Red.: T. Bogdanik, Wydawnictwo Lekarskie PZWL, Warszawa 1988, 411–439.
[28] Schuster G., Reichle R., Rane I., Bauer R., Schopf P.M.: Allergies induced by orthodontic alloys: Incidence and impact on treatment. J. Orofac. Orthop. 2004, 65, 48–59 [in German].
[29] Braun-Falco O., Plewig G., Wolff H.H., Burgdorf W.H.C.: Reakcje na leki. W: Dermatologia. Red.: O. Braun-Falco, G. Plewig, H.H. Wolff, W.H.C. Burgdorf, Wydawnictwo Czelej, Lublin 2002, 383–406.
[30] Rudzki E., Rebandel P., Parapura K.: The most frequent and novel contact allergens. Przegl. Derm. 2000, 2, 103–109 [in Polish].
[31] Kerosuo H., Moe G., Hensten-Pettersen A.: Salivary nickel and chromium in subjects with different types of fixed orthodontic appliances. Am. J. Orthod. Dentofac. Orthop. 1997, 111, 595–598.
[32] Szeląg J.: Allergy to nickel – case report. Dent. Med. Probl. 2002, 39, 309–312 [in Polish].
[33] Gryziecki H., Kaska M.: Leczenie protetyczne w przypadkach elektrogalwanicznego zapalenia błony śluzowej jamy ustnej i kątów ust. Protet. Stomat. 1976, 26, 113–116.
[34] Jamroszczyk K., Lipski M., Wędrychowska-Szul C.B., Gałęska M.: Corrosion of nikel-titanium alloy used in orthodontics – potential causes and clinical sequelae. Magazyn Stomat. 2008, 18, 3, 26–29 [in Polish].
[35] Łuszczuk M.: Electrochemical phenomena in the oral cavity. Magazyn Stomat. 2003, 13, 11, 36–38 [in Polish].
[36] Masalski J.: Electrochemical behaviour of selected biomaterials in artificial saliva. Protet. Stomat. 2000, 50, 110–115 [in Polish].
[37] Jabłońska S., Chorzelski T.: Choroby alergiczne skóry. W: Choroby skóry. Red.: S. Jabłońska, T. Chorzelski, PZWL, Warszawa 1997, 158–197.
[38] Rudzki E.: Alergeny (odcinek 31) – nikiel (uzupełnienie). Med. Prakt. 1997, 9, 111–113.
[39] Grochowski P.: Zastosowanie stopów zawierających nikiel w leczeniu protetycznym pacjentów uczulonych na metal. Część I. Protet. Stomat. 1992, 42, 206–209.
[40] Szarmach I.J, Buczko P.: Nickel in orthodontics – review of the literature. Magazyn Stomat. 2008, 18, 10, 39–47 [in Polish].
[41] Kerosuo H., Kulla A., Kerosuo E., Kanerva L., Hensten-Pettersen A.: Nickel allergy in adolescents in relation to orthodontic treatment and piercing of ears. Am. J. Orthod. Dentofac. Orthop. 1996, 109, 148–154.
[42] Grochowski P.: Zastosowanie stopów zawierających nikiel w leczeniu protetycznym pacjentów uczulonych na metal. Część II. Protet. Stomat. 1992, 42, 210–215.

## Address for correspondence:

Agnieszka Sieja
Department of Dentofacial Orthopedics and Orthodontics
Wroclaw Medical University
Krakowska 26
50-425 Wrocław
Poland
Tel. +48 71 784 02 99
E-mail: ws-3@stom.am.wroc.pl

Received: 30.08.2010
Revised: 12.10.2010
Accepted: 3.11.2010

Praca wpłynęła do Redakcji: 30.08.2010 r.
Po recenzji: 12.10.2010 r.
Zaakceptowano do druku: 3.11.2010 r.