

Passive smoking as an environmental health risk factor

Małgorzata H. J. Sikorska-Jaroszyńska¹, Maria Mielnik-Błaszczak¹, Dorota Krawczyk¹, Alicja Nasiłowska-Barud², Jerzy Błaszczak³

¹ Chair and Department of Paedodontics, Medical University, Lublin, Poland

² Department of Clinical Psychology, Medical University, Lublin, Poland

³ Department of Dental Prosthetics, Medical University, Lublin, Poland

Sikorska-Jaroszyńska MHJ, Mielnik-Błaszczak M, Krawczyk D, Nasiłowska-Barud A, Błaszczak J. Passive smoking as an environmental health risk factor. *Ann Agric Environ Med.* 2012; 19(3): 547-550.

Abstract

Initially, tobacco was considered as a decorative plant and only later began to be treated as a herb with special therapeutic properties. With time, it was found that tobacco had strong insecticidal and addictive properties. There also occurred reports about the negative influence of tobacco on human health. The World Health Organization (WHO) classifies smoking as a chronic, progressive disease which is also 'contagious'. It is also considered to be a neurobiotic addiction. Nicotine addiction does not cause changes in the behaviour or functioning of a smoker; however, it adversely affects his or her general health status and the health status of people within their environment. Passive smoking (so-called ETS – Environmental Tobacco Smoke), which means accompanying smokers negatively influences the health of passive smokers. Environmental tobacco smoke, on the one hand, is the result of spontaneous cigarette burning and, on the other hand, the result of the side-stream of cigarette smoke, as well as the smoke exhaled by active smokers. Health personnel should clearly and convincingly present the data concerning the adverse results of smoking, as well as the dangers of exposure to environmental tobacco smoke, thereby making their patients aware that breaking their addiction will not only be beneficial for their own health, but will also protect non-smokers in their environment from the adverse effects of exposure to environmental tobacco smoke.

Key words

Passive smoking, Environmental Tobacco Smoke, Second Hand Smoking

INTRODUCTION AND OBJECTIVE

Tobacco appeared 6,000 BC and from the very beginning it accompanied humanity like 'tea and food'. Tobacco is a crop known to the ancient civilizations of Central and South America, as well as the Middle East and China. The plant was treated by the ancestors of modern Indians not only as God's gift, but also as 'holy smoke' that gave people the possibility of reconciliation and eternal peace. It reached Europe as late as the 15th century, and the first person who brought the tobacco seeds to Spain in October 1492 was a participant of the second trip by Christopher Columbus, a monk called Romano Paro. Tobacco seeds reached Poland almost 100 years later, in 1590 [1, 2, 3, 4].

It should be stressed that initially tobacco was considered as a decorative plant and only later began to be treated as a herb with special therapeutic properties. In 1571, Nicolas Manardes wrote a dissertation concerning the incredible properties of tobacco, which allowed for the treatment of many diseases. In his work, he stated that tobacco can be used in the treatment of 36 diseases, including mental illnesses, fever, headache, frost bite, ulcers, wounds, oedemas, and toothache, as well as dermatological and venereal diseases and poisoning. Over the centuries, the way of tobacco usage changed. In the beginning, it was chewed, drunk in the form of brew, eaten, and sniffed as snuff. It was used as

eye drops, enema, analgesic, antiseptic, anti-inflammatory, diuretic medicine, and an antitoxin. Later on, there occurred cigarettes, cigars and pipes. Nowadays, tobacco is a plant that is most frequently used for smoking after being previously dried and fermented [1, 3, 4].

With time, it was found that tobacco has strong insecticidal and addictive properties. There also occurred reports about the negative influence of tobacco on human health [1, 5].

At the beginning of the 17th century in England, the first attempt to reduce tobacco consumption was undertaken by imposing tax on tobacco. In the middle of the 18th century in Germany, Frederick the Great issued an edict against tobacco smoking. Actually, the entire 18th century was the period in which many countries, including Russia and the Vatican State, introduced a ban on tobacco smoking. However, it should be stressed that these prohibitions were rather connected with the requirements of faith and morality, and not due to concern about the negative influence of tobacco on human health. Despite the discovery of the harmful properties of the plant, it became more and more popular. Wars and epidemics facilitated further popularization of tobacco smoking. During World War II, in the United States, it was even decided that tobacco plantations should become areas of special protection. In the 1940s and 1950s, there appeared reports that cigarette smoking may be involved in the development of lung cancer, and that the tar included in cigarettes adversely affected human health and could participate in the occurrence and development of neoplasms. However, it was in the 1960s when more evidence for the adverse influence of tobacco on human health was found and presented [1, 3, 4, 5].

Address for correspondence: Małgorzata H. J. Sikorska-Jaroszyńska, Wieniawska 10/4, 20-071 Lublin, Poland.
E-mail: MHJSJ@interia.pl

Received: 28 February 2012; accepted: 17 August 2012



State of knowledge. Nowadays, smoking is considered to be one of the unhealthiest of human behaviours and the most hazardous of addictions. The World Health Organization classifies smoking as a chronic, progressive disease which is also 'contagious'. It is also considered to be a neurobiotic addiction. According to the definition issued by the WHO, a smoker is somebody who smokes at least one cigarette a day for at least 6 months [6]. The number of cigarettes smoked within one's lifetime and exposure to cigarette smoke is determined using a 'pack/year' index. One 'pack/year' equals 10 cigarettes a day for the period of one year [7]. In Poland, cigarette consumption is 3,650 cigarettes a person a year, and the average number of smoked cigarettes is about 20 a day [8, 9]. According to the WHO data, more than 4.9 million people die every year due to diseases resulting from cigarette smoking and using tobacco based products, as well as passive smoking [10, 11].

Nicotine addiction does not cause changes in the behaviour or functioning of a smoker; however, it adversely affects his or her general health status and the health status of people within their environment. Passive smoking (so called ETS – Environmental Tobacco Smoke), which means accompanying smokers negatively influences the health of passive smokers. Environmental tobacco smoke, on the one hand, is the result of spontaneous cigarette burning, and on the other hand, the result of side-stream of cigarette smoke, as well as the smoke exhaled by active smokers. It should also be noted that smokers who are in the company of active smokers are also considered to be passive smokers. It can be said that in case of these people the negative results of smoking are cumulated [12]. Oberg et al. observed that, worldwide, 40% of children, 33% of male non-smokers, and 35% of female non-smokers were exposed to second-hand smoke in 2004 [13]. At the same time, according to the WHO, it is not possible to declare any level of exposure to the environmental tobacco smoke to be safe. In places where smoking is forbidden, the concentration of nicotine in the air is lower than in places where people are allowed to smoke. However, even in places where smoking is forbidden, the level of nicotine in the air never equals 0. Recently, apart from second hand smoking, another term, third-hand smoking, is being used to describe a situation in which non-smokers move into apartments previously occupied by smokers, or buy a car from a smoker [14].

Smoking, as well as exposure to environmental tobacco smoke, in pregnancy adversely affects the development of the foetus. Children are exposed to ETS *in utero* through the umbilical cord bond. It should be stressed that the placenta does not constitute a barrier against tobacco smoke. Study results show that the ETS can be a factor in such complications as premature birth, lower birth weight (33-335g depending on the author), more frequent birth (by about 22%) of children with birth weight below 2,500 g, occurrence of birth defects, and smaller head circumference. The influence of environmental tobacco smoke probably contributes to foetuses being too small for their gestational age; however, the ETS was not found to contribute to the delay of intrauterine development. It has been confirmed that children born to mothers exposed to environmental tobacco smoke attain lower APGAR scale results. It has also been established that women infants exposed to second-hand tobacco smoking during pregnancy had their BMI index at the age of 2 and 3 years higher than their contemporaries, whose mothers were not passive smokers. At the same time, on the basis of many

years of observation of children exposed to environmental tobacco smoke *in utero*, it was found that passive smoking affected the functioning of the immunological system, probably by adversely influencing neurodevelopment; the occurrence of respiratory system diseases is also significantly higher after birth. It is supposed that the ETS affecting the foetus may facilitate the incidence of allergic disorders. However, it should be stressed that, to date, the results of epidemiological studies have not unequivocally confirmed whether a mother's active smoking during pregnancy causes allergy in her child [15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25].

Exposure to environmental tobacco smoke, both in foetal life and after birth, contributes to the occurrence of diseases of the lower and higher airways, asthma, wheeze, lung lesions detectable in computer tomography, as well as lung function impairment [26, 27, 28, 29, 30, 31, 32]. In the case of passive smokers, the risk of the occurrence of chronic obstructive pulmonary disease is significantly higher. By 2020, this disease is probably going to be the top third cause of mortality [17, 33].

Recently, it has been suggested that the ETS contributes to the occurrence diabetes mellitus type 2 [34], multiple sclerosis [35], increases the possibility of the occurrence of pain in the abdominal cavity, joints, back and head [36]. In daughters of smoking mothers who smoked during pregnancy, or who were exposed to the ETS, the first period occurs later [37]. The results of other studies showed that there is a small-to-modest association between *in utero* exposure to tobacco smoke and reduced fertility [38].

Passive smoking increases the risk of occurrence of cardiovascular diseases. The ETS contributes to endothelial function impairment and increases the oxidative stress [39]. As demonstrated, significant exposure to the ETS is associated with the presence of an increased level of C – reactive protein (CRP) which, in turn, explain the relationship between second- hand smoking (SHS) and death due to cardiovascular reasons [13, 40].

There are also reports that exposure to environmental tobacco smoke influences the development of hard and soft oral tissues. Passive smoking contributes to alteration of the composition of the oral bacterial flora. The number of *Streptococcus mutans* and *lactobacilli* increases [41, 42]. It was found that in case of *in utero* exposure, as well as after birth, there was increased risk of early caries and deciduous teeth decay. It is worth noting that smoking by the father is less harmful than that by the mother [43, 44]. It was also demonstrated that smoking by household members, as well as second-hand exposure outside one's house, may contribute to the development of permanent teeth caries in adolescents [42, 45, 46, 47]. In the case of smokers, there is an increased risk of tooth loss, while it decreases in people who give up smoking. However, it is not clear whether the ETS affects tooth loss in non-smokers and, according to Arora et al., this requires further studies and confirmation [48].

Animal studies revealed the occurrence of cytoplasm and acinar cells nuclei involution, inflammatory infiltration, type I collagen accumulation in the stroma, and the increase of interacinar space filled with extracellular matrix which, in effect, leads to salivary glands function disorder [49]. It has also been observed that in passive smokers, the level of sIgA is decreased, amylase activity is increased, and the level of sialic acid in the whole stimulated saliva is also increased [50]. The results of other, 10-months-long studies, of children aged



3.5-5 years exposed to the ETS, showed that the levels of IgA and IgG in saliva were increased [51]. In other studies, it was observed that passive smoking contributes to the decrease of salivary pH, the rate of the secretion of saliva and its buffering abilities [50]. In case of children exposed to the ETS, the level of cotinine is increased and accompanied by lowered CAL (Clinical Attachment Level) [52]. In the case of passive smokers, the inflammatory response from the periodontal tissues is increased, and the risk of progression of periodontal diseases is also higher [53]. It was also found that environmental tobacco smoke impairs the final differentiation of keratinocytes [54], and contributes to increased gingival pigmentation, evaluated on the basis of the gingival pigmental index and digital oral photographs [55].

As already stated, cigarette smoking contributes to the occurrence of neoplasms. Reports have also appeared that the ETS is directly associated with lung cancer [56], and may contribute to higher risk of breast cancer in younger, primarily premenopausal women [57], as well as to the occurrence of pancreatic cancer [58]. However, it should be stressed that studies performed recently did not confirm the influence of passive smoking on the general risk of incidence of neoplasms, with pancreatic cancer being the only exception [59].

In the 1980s and 1990s, due to the results of experimental, clinical and epidemiological studies revealing the harmfulness of tobacco, there appeared new laws limiting smoking in public places, especially at work. The Framework Convention on Tobacco Control (FCTC) is the first international treaty concerning public health. It was issued after four years of negotiations among the WHO members. It was signed in 2004 by 192 countries, and its provisions were officially introduced in February 2005. The provisions of FCTC mainly concern the tobacco industry, the necessity for the labeling of tobacco products packaging, introduction of new taxes on tobacco products, and illegal trade and protection from exposure to tobacco smoke. In 2005, the Oral Health Network of Tobacco Use Prevention and Cessation was established [60]. In 2008, a work group was formed in the European Parliament aimed at the introduction of a complete ban on the tobacco trade in the European Union by 2025. Guidelines included in the Healthy People 2010 Programme are meant to limit cigarette smoking. According to these guidelines, cigarette smoking frequency should drop to 12%. According to current Polish law, persons below 18 years of age are not allowed to purchase cigarettes. It should be stressed that cigarette vending machines and sales of single cigarettes are also illegal. It is also forbidden to advertise cigarettes on TV, radio, in the press, cinema, and on billboards. There is also a complete ban on smoking in hospitals, schools, universities, institutions, offices, theatres and cinemas, and a partial ban in restaurants and bars. There are warnings about the health hazards of smoking printed on cigarette packets. As a result of this campaign, a decrease of the incidence of lung cancer has been observed in young and middle-aged people [11, 61].

Nowadays, 12 types of smokers are distinguished, including 'childish smokers'. These are people smoking 'in hiding', in whom smoking evokes a sense of security due to memories from childhood. Smoking reminds them of one or both parents smoking at home and helps them come to grips with the passage of time. It can also be stated that such people feel well and safe in an environment with a relatively high concentration of cigarette smoke. Parents who smoke

and live in a house with high concentration level of cigarette smoke often make children start smoking at a very early age, thereby adopting negative, unhealthy behaviour. Despite the constant flow of information about the harmfulness of smoking from the mass media (newspapers, radio, television, the internet), doctors and dentists still have a very important role to play in anti-nicotine campaigns, convincing people about the negative influence of tobacco smoke on the state of their health. In the course of control visits, health personnel should clearly and convincingly present the data concerning the adverse results of smoking, as well as the dangers of exposure to environmental tobacco smoke [1, 11, 61, 62], thus, make their patients aware that breaking their addiction will not only be beneficial for their own health, but will also protect non-smokers in their environment from the adverse effects of exposure to environmental tobacco smoke.

REFERENCES

1. Grzybowski A. Historia zainteresowań tytoniem i nikotynizmem do połowy XX wieku w Europie. (The history of Tobacco consumption and nicotine addiction in Europe until the middle of the XXth century.) *Przegl Lek.* 2005; 62(10): 1211-1214.
2. Borio G. http://www.tobacco.org/History/Tobacco_History.html
3. Musk AW, De Klerk NH. History of tobacco and health. *Respirology* 2003;8: 286-290.
4. Randall VR. Tobacco, Health and the Law. <http://academic.udayton.edu/health/syllabi/tobacco>
5. Brusilo J. Palenie tytoniu – antropologiczne ujęcie ludzkich uzależnień. (Tobacco smoking – anthropological context of human dependence.) *Przegl Lek.* 2005; 62(10): 947-953.
6. Przewoźniak K, Zatoński W. In: Palenie tytoniu w dorosłej populacji Polski w latach 1974-1995. Zatoński W, Przewoźniak K (Eds.). Palenie tytoniu w Polsce: postawy, następstwa zdrowotne i profilaktyka. (Tobacco smoking in adult population in Poland in 1974-1995. Zatoński W, Przewoźniak K (Eds.). Tobacco smoking in Poland: Essentials, health consequences and prophylactics). M. Skłodowska-Curie Memorial Centre and Institute of Oncology Warszawa; 1999, 129-163.
7. Prignot J. Quantification and chemical markers of tobacco – exposure. *Eur J Respir Dis.* 1987; 70(1): 1-7.
8. Zatoński W, Przewoźniak K. Palenie tytoniu w Polsce. (Tobacco smoking in Poland.) Warszawa 1999.
9. Zatoński W. Rozwój sytuacji zdrowotnej w Polsce. (Development of the health situation in Poland.) Warszawa 2001
10. De Beyer J, Lovelace C, Yürekli A. Poverty and tobacco. *Tob Control.* 2001; 10: 210-211.
11. World Health Organization. The World Health Report: Reducing Risk, Promoting Healthy Life. Geneva, World Health Organization 2002.
12. Sobczak A, Wardas W, Zielińska-Danch W, Szołtysek-Boldys I. Biomarkery narażenia na dym tytoniowy. (Biomarkers of Tobacco smoke). *Przegl Lek.* 2005; 62(10): 1192-1199.
13. Oberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. *Lancet* 2011; 377(9760): 139-146.
14. Matt GE. Residual tobacco smoke in used cars: futile efforts and persistent pollutants. *Nicotine Tob Res.* 2010; 12(10): 1029-1036.
15. Wdowiak A, Wiktor H, Wdowiak L. Maternal passive smoking during pregnancy and Neonatal health. *Ann Agric Environ Med.* 2009; 16: 309-312.
16. Braun JM, Daniels JL, Poole C, Olshan AF, Hornung R, Bernert JT, et al. Prenatal environmental tobacco smoke exposure and early childhood body mass index. *Paediatr Perinat Epidemiol.* 2010; 24(6): 524-534.
17. Tanaka K, Miyake Y. Association between prenatal and postnatal tobacco smoke exposure and allergies in young children. *J Asthma.* 2011; 48(5): 458-463.
18. Król M, Florek E, Kornacka MK, Bokinić R, Piekoszewski W. Stan kliniczny noworodka a stopień narażenia na dym tytoniowy w życiu płodowym. (Clinical condition of the newborn versus tobacco smoke exposure during fetal life). *Przegl Lek.* 2009; 66(10): 548-553.

19. Savitz DA, Murnane P. Behavioral influences on preterm birth: a review. *Epidemiology*. 2010; 21(3): 291-299.
20. Salmasi G, Grady R, Jones J, McDonald SD. Environmental tobacco smoke exposure and perinatal outcomes: a systematic review and meta-analysis. *Acta Obstet Gynecol Scand*. 2010; 89(4): 423-441.
21. Leonardi-Bee J, Smyth A, Britton J, Coleman T. Environmental tobacco smoke and fetal health: systematic review and meta-analysis. *Arch Dis Child Fetal Neonatal Ed*. 2008; 93(5): F351-F361.
22. Cheraghi M, Salvi S. Environmental tobacco smoke (ETS) and respiratory health in children. *Eur J Pediatr*. 2009; 168(8): 897-905.
23. Newman RB, Momirova V, Dombrowski MP, Schatz M, Wise R, Landon M, et al. The effect of active and passive household cigarette smoke exposure on pregnant women with asthma. *Chest*. 2010; 137(3): 601-608.
24. Polańska K, Hanke W, Sobala W, Ligocka D. Wpływ ekspozycji na środowiskowy dym tytoniowy w okresie prenatalnym na parametry urodzeniowe. (Impact of prenatal environmental tobacco smoke exposure on birth parameters). *Przegl Lek*. 2010; 67(10): 835-837.
25. Polańska K, Hanke W, Sobala W, Brzeźnicki S, Ligocka D. Narażenie palących kobiet ciężarnych na wielopierścieniowe węglowodory aromatyczne. (Exposure of smoking pregnant women to polycyclic aromatic hydrocarbons). *Med Pr*. 2009; 60(2): 103-108.
26. Wang L, Pinkerton KE. Detrimental effects of tobacco smoke exposure during development on postnatal lung function and asthma. *Birth Defects Res C Embryo Today*. 2008; 84(1): 54-60.
27. Butz AM, Halterman JS, Bellin M, Tsoukleris M, Donithan M, Kub J, et al. Factors associated with second-hand smoke exposure in young inner-city children with asthma. *J Asthma*. 2011; 48(5): 449-457.
28. Burgess JA, Matheson MC, Gurrin LC, Byrnes GB, Adams KS, Wharton CL, et al. Factors influencing asthma remission: a longitudinal study from childhood to middle age. *Thorax*. 2011; 66(6): 508-513.
29. Constant C, Sampaio I, Negreiro F, Aguiar P, Silva A, Salgueiro M, et al. Environmental tobacco smoke (ETS) exposure and respiratory morbidity in school age children. *Rev Port Pneumol*. 2011; 17(1): 20-26.
30. Kasznia-Kocot J, Kowalska M, Górny RL, Niesler A, Wypych-Ślusarska A. Environmental risk factors for respiratory symptoms and childhood asthma. *Ann Agric Environ Med*. 2010; 17(2): 221-229.
31. Thomson NC. The role of environmental tobacco smoke in the origins and progression of asthma. *Curr Allergy Asthma Rep*. 2007; 7(4): 303-309.
32. Lovasi GS, Diez Roux AV, Hoffman EA, Kawut SM, Jacobs DR Jr, Barr RG. Association of environmental tobacco smoke exposure in childhood with early emphysema in adulthood among nonsmokers: the MESA-lung study. *Am J Epidemiol*. 2010; 171(1): 54-62.
33. Burgess JA, Matheson MC, Gurrin LC, Byrnes GB, Adams KS, Wharton CL, et al. Factors influencing asthma remission: a longitudinal study from childhood to middle age. *Thorax*. 2011; 66(6): 508-513.
34. Ko KP, Min H, Ahn Y, Park SJ, Kim CS, Park JK, et al. A prospective study investigating the association between environmental tobacco smoke exposure and the incidence of type 2 diabetes in never smokers. *Ann Epidemiol*. 2011; 21(1): 42-47.
35. Hedström AK, Bäärnhiel M, Olsson T, Alfredsson L. Exposure to environmental tobacco smoke is associated with increased risk for multiple sclerosis. *Mult Scler*. 2011; 17(7): 788-793.
36. Pisinger C, Aadahl M, Toft U, Birke H, Zytphen-Adeler J, Jørgensen T. The association between active and passive smoking and frequent pain in a general population. *Eur J Pain*. 2011; 15(1): 77-83.
37. Ferris JS, Flom JD, Tehranifar P, Mayne ST, Terry MB. Prenatal and childhood environmental tobacco smoke exposure and age at menarche. *Paediatr Perinat Epidemiol*. 2010; 24(6): 515-523.
38. Ye X, Skjaerven R, Basso O, Baird DD, Eggesbo M, Uicab LA, et al. In utero exposure to tobacco smoke and subsequent reduced fertility in females. *Hum Reprod*. 2010; 25(11): 2901-2906.
39. Al-Dissi AN, Weber LP. Resveratrol preserves cardiac function, but does not prevent endothelial dysfunction or pulmonary inflammation after environmental tobacco smoke exposure. *Food Chem Toxicol*. 2011; 49(7): 1584-1591.
40. Hamer M, Stamatakis E, Kivimaki M, Lowe GD, Batty GD. Objectively measured secondhand smoke exposure and risk of cardiovascular disease: what is the mediating role of inflammatory and hemostatic factors? *J Am Coll Cardiol*. 2010; 56(1): 18-23.
41. Brook I. The impact of smoking on oral and nasopharyngeal bacterial flora. *J Dent Res*. 2011; 90(6): 704-710.
42. Avşar A, Darka O, Topaloğlu B, Bek Y. Association of passive smoking with caries and related salivary biomarkers in young children. *Arch Oral Biol*. 2008; 53(10): 969-974.
43. Hanioka T, Nakamura E, Ojima M, Tanaka K, Aoyama H. Dental caries in 3-year-old children and smoking status of parents. *Paediatr Perinat Epidemiol*. 2008; 22(6): 546-550.
44. Tanaka K, Miyake Y, Sasaki S. The effect of maternal smoking during pregnancy and postnatal household smoking on dental caries in young children. *J Pediatr*. 2009; 155(3): 410-415.
45. Ayo-Yusuf OA, Reddy PS, van Wyk PJ, van den Borne BW. Household smoking as a risk indicator for caries in adolescents' permanent teeth. *J Adolesc Health*. 2007; 41(3): 309-311.
46. Arora M, Weuve J, Schwartz J, Wright RO. Association of environmental cadmium exposure with pediatric dental caries. *Environ Health Perspect*. 2008; 116(6): 821-825.
47. Vellappally S, Fiala Z, Smejkalová J, Jacob V, Shriharsha P. Influence of tobacco use in dental caries development. *Cent Eur J Public Health*. 2007; 15(3): 116-121.
48. Arora M, Schwarz E, Sivaneswaran S, Banks E. Cigarette smoking and tooth loss in a cohort of older Australians: the 45 and up study. *J Am Dent Assoc*. 2010; 141(10): 1242-1249.
49. Ferragut JM, da Cunha MR, Carvalho CA, Isayama RN, Caldeira EJ. Epithelial-stromal interactions in salivary glands of rats exposed to chronic passive smoking. *Arch Oral Biol*. 2011; 56(6): 580-587.
50. Avşar A, Darka O, Bodrumlu EH, Bek Y. Evaluation of the relationship between passive smoking and salivary electrolytes, protein, secretory IgA, sialic acid and amylase in young children. *Arch Oral Biol*. 2009; 54(5): 457-463.
51. Ewing P, Otczyk DC, Occhipinti S, Kyd JM, Gleeson M, Cripps AW. Developmental profiles of mucosal immunity in pre-school children. *Clin Dev Immunol*. 2010; 2010: 196785.
52. Erdemir EO, Sönmez IS, Oba AA, Bergstrom J, Caglayan O. Periodontal health in children exposed to passive smoking. *J Clin Periodontol*. 2010; 37(2): 160-164.
53. Nishida N, Yamamoto Y, Tanaka M, Kataoka K, Kuboniwa M, Nakayama K, et al. Association between involuntary smoking and salivary markers related to periodontitis: a 2-year longitudinal study. *J Periodontol*. 2008; 79(12): 2233-2220.
54. Donetti E, Gualerzi A, Bedoni M, Volpari T, Sciarabba M, Tartaglia G, et al. Desmoglein 3 and keratin 10 expressions are reduced by chronic exposure to cigarette smoke in human keratinised oral mucosa explants. *Arch Oral Biol*. 2010; 55(10): 815-823.
55. Sridharan S, Ganiger K, Satyanarayana A, Rahul A, Shetty S. Effect of environmental tobacco smoke from smoker parents on gingival pigmentation in children and young adults: a cross-sectional study. *J Periodontol*. 2011; 82(7): 956-962.
56. Lodovici M, Bigagli E. Biomarkers of induced active and passive smoking damage. *Int J Environ Res Public Health*. 2009; 6(3): 874-888.
57. Miller MD, Marty MA, Broadwin R, Johnson KC, Salmon AG, Winder B, et al. The association between exposure to environmental tobacco smoke and breast cancer: a review by the California Environmental Protection Agency. *Prev Med*. 2007; 44(2): 93-106.
58. Vrieling A, Bueno-de-Mesquita HB, Boshuizen HC, Michaud DS, Severinsen MT, Overvad K, et al. Cigarette smoking, environmental tobacco smoke exposure and pancreatic cancer risk in the European Prospective Investigation into Cancer and Nutrition. *Int J Cancer*. 2010; 126(10): 2394-2403.
59. Chuang SC, Gallo V, Michaud D, Overvad K, Tjønneland A, Clavel-Chapelon F, et al. Exposure to environmental tobacco smoke in childhood and incidence of cancer in adulthood in never smokers in the European Prospective Investigation into Cancer and Nutrition. *Cancer Causes Control* 2011; 22(3): 487-494.
60. Ramseier CA, Mattheos N, Needleman I, Watt R, Wickholm S. Consensus report: first European workshop on tobacco use prevention and cessation for oral health professionals. *Oral Health Preventive Dentistry* 2004; 6(1): 7-18.
61. Zatoński WA. Improvement of health through tobacco control in Poland-evidence for action in Europe, (in) WHO European Ministerial Conference for a tobacco-free Europe. Reports of the conference. *World Health Organization* 2002, 25-26.
62. Nasry HA, Preshaw PM, Stacey F, Heasman L, Swan M, Heasman PA. Smoking cessation advice for patients with chronic periodontitis. *Br Dent J*. 2006; 200(5): 272-275.

