# the world's leading publisher of Open Access books Built by scientists, for scientists

4,800

Open access books available

122,000

International authors and editors

135M

Downloads

154

**TOP 1%** 

Our authors are among the

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us? Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.

For more information visit www.intechopen.com



# **Smoking Habit and Nicotine Effects**

Brankica Juranić, Štefica Mikšić, Željko Rakošec and Suzana Vuletić

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.77390

### **Abstract**

Smoking is a gained habit with which one starts experimenting at the age of 10, and it usually becomes part of the habit at the age of 20. It is the combination of narcotic addiction and deep-seated smoking habits. Nicotine is the main cause of smoking addiction, and the custom of preparation for smoking itself and smell of cigarettes create addictive behavior among smokers. Today, nicotine is socially most widely accepted legal drug in the world, and its uptake into the organism through the respiratory tract is 10 times stronger than heroin. Addiction and necessity for cigarettes are constant and intense, and the treatment for smoking addicts is long and difficult. Smoking is a worldwide epidemic, which is closely connected to other addictions such as coffee, alcohol, drugs, and gambling.

Keywords: smoking, nicotine, addiction

## 1. Introduction

Smoking is a chronic non-contagious disease that causes a variety of diseases leading to premature death and a significant reduction in the quality of life. The way to consume tobacco is by inhaling smoke from a stunned tobacco leaf in cigarettes, cigars, or tobacco pipe. The occurrence of addiction is massive and it is the most widespread social disease. Tobacco smoke is one of the biggest risk factors responsible for 63% of the total mortality. By 2030, it is estimated that 10 million people per year will die of the consequences of smoking [1]. According to World Health Organization data, tobacco smoking is among the first causes of death that can be prevented. Epidemiological studies around the world have shown that there is a strong link between smoking and incidence of cigarettes, nonsmoking and chronic diseases such as



cardiovascular disease, lung disease, stroke [2, 3], and cancer. The global epidemic of smoking worldwide causes about 6 million deaths every year, including about 6,000,000 people who were nonsmokers but were exposed to inhalation of tobacco smoke. Every 8 s, a person dies from tobacco addiction. [4]. The International Cancer Research Organization has included more than 60 substances [5], which tobacco cigarettes contain, in the carcinogen category. Studies show that smoking prevalence comes from low- or middle-income countries.

Tobacco is the most accessible drug, and its consumption has its history. There are data that tobacco smoking had been practiced somewhere between 5000 and 3000 B.C. In the archeological finds in areas where the Indians from the Mayan tribe lived, the priests were shown smoking a tobacco pipe during a ceremony [6]. A common pipe was smoked during various ceremonies, as a welcome sign, for the end of warfare and peacemaking, as the famous peace flag. Tribal healers used tobacco, dry leaves, or powdered leaves as a healing remedy for wounds, pain relief, fatigue, thirst, or hunger [7]. There was an overwhelming belief that tobacco was a sacred plant. Tobacco is considered to be abundant in North America [6]. The history of tobacco dates back to 1492 when Christopher Columbus wrote in his journal: here, in the new country there are men and women with a half-burnt weed in their hands, being the herbs they are accustomed to smoke... [8]. Colombian sailors transferred tobacco to Spain and Portugal, and from there it was spread across European countries and other continents. In 1560, French Ambassador of Portugal Jean Nicot brought tobacco seeds to the royal court and gave it to Queen Katarina Medici as a miraculous and healing plant. Tobacco got its Latin name after his surname Nicotiana. There are 70 different species of Nicotiana, and the best known is the one from the Solanaceae family—Nicotiana tabacum [9]. It is a 1-year plant that can grow up to 2 meters, and tobacco is produced in the root and deposited in the leaves [9]. The name is derived from the Arabic word tobacco, meaning smoke. From 1600 to 1700, smoking spread across Europe in all social classes [10]. During the seventeenth century, tobacco planting was extended to other European countries: Germany, Sweden, Denmark, Austria, and then the Far East and China [9]. The first laws prohibiting smoking were adopted in the seventeenth century. Sultan Murat brought a law that punished anyone who smoked with a death sentence because it was suspected that smokers caused a major fire in Constantinople. In the same period with the support of the church, anti-smoking laws were being adopted in Europe [11]. British King James I condemned the first tobacco importer in England with a death penalty. In the mid-nineteenth century, industrial cigarette production began, and smoking became fashionable. Tobacco expansion and the number of smokers increased especially after the end of World War I where a large number of smokers recorded were among women.

Since the appearance of tobacco and smoking in Europe, we have witnessed the first conflicts between two sides, those who advocate smoking and tobacco as a healing plant, and others who consider it harmful and dangerous to health. None of the two parties could prove that they were right or wrong until the beginning of the twentieth century.

During the 1920s, the dangers of tobacco smoking leading to mortality and the many consequences of smoking on the economic and social level were recognized. From the early 1930s to the mid-1950s of the twenty-first century, the first experimental evidence confirms the association between tobacco smoke and lungs, bronchus diseases, and the carcinogenicity of cigarette smoke substances. Epidemiological research has shown that there is a causal

link between excessive cigarette smoking and lung cancer. In 1960, a statutory obligation was issued to highlight the health hazard warning on each cigarette box. From 1962 to 1964, Luther L. Terry had published more than 7000 articles on smoking and health and ended with a famous report titled *Terry's Bomb*. Thereafter, the scientific approach to the smoking problem intensified, and in 1967, the First World Conference on Smoking and Health was held by the World Health Organization (WHO) as a systematic action plan. In 1971, WHO began a systematic anti-smoking campaign and adopted the first anti-smoking resolution and first action plans. In 1975, smoking was first listed in the International Classification of Diseases, and in 1995, it was classified under mental disorders and tobacco-related disorders under code F17.

# 2. Nicotine dependence

Smoking is initially a habit leading to physical and psychological dependence. An important role in the smoking-related phenomenon is a number of repeated movements in the smoking act of the hand and oral muscles, cigarette outbreaks, lighting a cigarette, blowing smoke, holding, and extinguishing the cigarette butt. All these movements are repeated a few hundred times a day [6] and become a ritual that is repeated. A smoker thus makes about 500-600 kisses daily by activating their oral muscles. The smell of smoke, fire, or lighter, and the way cigarette is lit also affect dependence. Cigarettes are like magic wands that are used to relax, calm down, relieve stress, or as a substitute for enhanced food enjoyment. The act of smoking contributes toward self-destruction and pollution of the environment. Therefore, smoking is not just the problem of the person who smokes, but it creates problems for the entire community and society where the smoker lives and works. People who smoke cigarettes can be divided into four groups: permanent smoker, occasional smoker, former smoker, and nonsmoker. The consequences of smoking can affect non-smokers, who we call passive smokers. The largest group of victims is children whose parent(s) smoke because cigarette smoke remains in the air for the next 8 h [12] after a person has finished the task of smoking, and is called environmental tobacco smoke or Second-Hand Tobacco Smoke (SHS) [13].

### 2.1. Harmful effects of nicotine

Nicotine is the main tobacco alkaloid, isolated in 1828. It is a colorless, oily liquid, odorless, and one of the strongest herbal poisons and neurotoxins that act on the central and peripheral nervous system. The most dangerous ingredient is tobacco smoke, which stays in the air, and is connected with oxygen with a characteristic smell of tobacco. The fast "dose" of nicotine is obtained from 7 to 10 s and is one reason why smoking becomes a hard-core addiction that speeds up heart activity, elevates blood sugar levels, increases blood pressure, triggers an apparent fatigue condition that leads to slowing down of heart causing uneasiness and the desire to recycle the cigarette so that the discomfort disappears and nicotine accumulates in the blood. This pattern cyclically repeats and promotes the sustenance of nicotine addiction, called *nicotinism*. Its effect on the central nervous system makes smoking comfortable and acts on brain cells in a way that regulates emotions, alleviates anxiety, irritability, and improves mood. The smoker is able to control how much the brain receives nicotine by blowing the smoke at his own pace [14]. From lighting the cigarette, the smokers inhale about 15–20%

smoke on average. Acute nicotine poisoning causes symptoms such as vomiting, weakness, blush, dizziness, drowsiness, headache, and sweating. Abdominal pain, cramps, anxiety disorder, low blood pressure, poorly filled pulse, salivation, epileptic cramps, collapse, loss of consciousness, cessation of breathing, and death are the result of larger quantities of poisoning. The smoker's body launches various defense mechanisms against tobacco poisoning that overwhelm the inhaled poisons, reduce or prevent their adverse effects, and reduce toxic effects on the human body, and therefore no symptoms of acute poisoning appear, as with the novice smoker. With regular smokers, symptoms of poisoning can occur if the smoker smokes double or multiple cigarettes, or more cigarettes than usual.

### 2.2. Tobacco smoke

Tobacco smoke contains more than 4000 different compounds such as alkaloids; alcohols; phenols; ethers; ketones; quinones; esters; nitrites; hydrocarbons; sulfur organic compounds; various inorganic compounds of lead, iron, copper, manganese, nickel, hydrocarbon molybdenum; and other metabolites. The composition of tobacco smoke depends on the type of tobacco, the temperature and the rate of combustion, various supplements, the length of cigarettes, the technological processing, and the production of cigarettes. The most potent tobacco smoke ingredients introduced into the human body are nicotine, cyanide compounds, and carbon monoxide. Nicotine, inhaled with other tobacco smoke ingredients or absorbed in the mucous membrane of the mouth, penetrates the blood and enters all parts of the body. In 7 s, it reaches the brain cells and increases dopamine gain, which causes euphoria and relaxation. Smoke pollutes all parts of the nasal cavity with various poisons and makes the gas exchange process difficult. The nasal mucus destroys and prevents viruses and bacteria growing, and smoking tobacco reduces this function. When excessive smoking occurs, the mucous membrane is formed in the throat, speech becomes difficult, and the voice lurks. With frequent smoking, smokers cough more and more, thereby releasing smoking mucus from lung bronchi into the mouth cavity with harmful ingredients. Chronic persistent cough is very common with smokers, and it is considered that about 75% of smokers have chronic coughs. Sinus cavities are constantly coated with tobacco smoke particles, causing the mucous membranes to be chronically inflated and thickened, and the openings are reduced. Many smokers therefore have occasional headaches, especially during colds and other diseases of the upper respiratory organs. The outer part of a smoker's lungs is dark gray to black. Plush tissue looks like a black sponge dipped in diluted tar. Due to the accumulation of tobacco particles on the bronchi, bronchial walls, and stronger bronchial mucus, their diameter narrows and reduces the flowability of the gases. Tobacco smoking causes increased bronchial hyperreactivity [15]. Oxidants and free radicals cause seizure and accumulation of neutrophils in pulmonary microcirculation, as well as accumulation of macrophages in respiratory bronchioles, whereby macrophages are a new potential oxidant reservoir that damages the tissue found [16]. Disordered harmony between ventilation and perfusion of alveolar spaces causes obstructive pulmonary diseases such as bronchitis, emphysema, asthma, bronchitis and bronchiectation. Smoking is a predisposing factor for respiratory infections [17-19] and exacerbation of asthma [20], as well as exposure to tobacco smoke from the environment that increases asthma and its exacerbation [21]. Lung cancer in men occurs between 35 and 75 year of age. According to world surveys, 95% of throat cancer patients were smokers, and laryngeal cancer grows due to a rise in the number of smokers among women [22, 23]. Smoking increases the risk of obesity and stomach cancer, as well as the development of osteoporosis that changes the physical appearance of a person. Smoking is a risk factor for the development of cardiovascular diseases and adverse effects on endothelial blood vessels [24, 25]. In families with hereditary characteristics of tumor development associated with smoking, there is a high probability of autosomal dominant inheritance of the predisposition for cancer [26–28]. The effect of smoking on reproductive system in men leads to erectile dysfunction [29] and sperm quality [30]. Sexual glands are very sensitive to nicotine and if consumed with alcohol leads to reduced sexual activity. Tobacco poisons damage the molecules inside the cell nucleus, especially the DNA that contains the chromosomes. Tobacco smoke poisons also slow the transmission of nerve impulses and reflex mechanisms that may be delayed or slower. Weight loss proves that tobacco poisons interfere with metabolism because smoking requires more energy and reduces appetite. It affects calcium and insulin activity (insulin resistance), and plasma cortisol is associated with the amount of cigarette smoked in 1 day. People exposed to passive smoking have higher IgE production overall and specific for allergenic patients [31]. Smokers have a higher level of total E immunoglobulin than non-smokers [32]. There is a cumulative effect of early and late exposure to smoking, including tobacco smoke from the environment and the appearance of atopic dermatitis [33].

Because of the impassibility of the smallest blood vessels, a person's appearance is changed, and the skin is pale—yellowish tone, aging faster, does not get the necessary food and oxygen, face and nails become yellow, the lips become blue, body has an unpleasant smell, wrinkles on the face, yellow teeth, and a rough voice.

Nicotine and other components of the cigarette smoke cause different endocrine imbalances and have a negative effect on the pituitary, thyroid, and adrenal glands; testes, egg cells; and their function. It significantly affects the probability of successful pregnancy in healthy women, not just in assisted reproduction cases. Women have adverse effects on reproductive ability, pregnancy-related difficulties, the use of oral contraceptives, menstrual cramps, and urinary bladder. Smoking destroys the human embryo and fetus throughout the entire perinatal development [34, 35], after delivery, during breastfeeding, and throughout the life of the baby. The consequences of its effect are complications in pregnancy, premature birth, reduced birth weight [36], and increased perinatal mortality. Children, during later stages of development after birth, may be prone to allergies, asthma, neurodermatitis, reduced sense of smell, tumor, disruption of intellectual maturation, frequency of aggression, and behavioral disorders. With pregnant women who smoke, a fetus gets a number of harmful substances and poisons through the bloodstream, and it is the only source of nutrients and oxygen. The most common are nicotine and carbon monoxide. Nicotine causes narrowing of blood vessels throughout the mother's body, as well as those in the umbilical cord and causes disturbances in the maturation of the placenta function leading to intrauterine oxygen deficiency. Carbon monoxide produced by smoking is bound to pigments in red blood cells suppressing oxygen, which in turn limits the supply of oxygen to the fetus. After a newborn's birth, the lungs are the most endangered organ, as they are underdeveloped and are unable to supply the whole body with oxygen due to which the newborn experiences breathing problems. Passive smoking with pregnant women in a smoking family or exposure to tobacco smoke is equally as dangerous as smoking. It has been demonstrated that children whose mothers were smoking during pregnancy have an increased risk of developing respiratory disease later in childhood [37]. It is estimated that 165,000 children die from the consequence of passive smoking per year [38], and it has been shown that passive exposure to tobacco smoke is associated with increased risk of sudden infant death syndrome, malignant diseases, particularly leukemia and lymphoma, cardiovascular disease, slowed psycho-motor development, difficulties in learning, problem behaviors, and obesity [39]. The damaging effects of the mother's active and passive smoking during pregnancy on growth and development of the child are directly related to the number of cigarettes smoked [40, 41].

People begin smoking generally during puberty or early adult age [42], which act as predictors of smoking in adulthood [43, 44]. Companies marketing cigarette brands are aware of their role in the creation of child smokers because there is a high likelihood that they will become lifelong buyers. Children and young people begin to smoke for fun, curiosity, imitation of friends or parents who are smoking, or because of the aspiration to be accepted in society. Their increased use of cigarettes on weekends may be triggered by factors such as socialization with peers, alcohol, and drug consumption at the same time [45, 46]. Studies have shown that adolescents smoke cigarettes even though they know and understand all the adverse health effects [14, 47].

# 3. Measures in the implementation of tobacco smoking reduction

Health-care systems and surveillance systems can contribute to and monitor efforts to change behaviors. A useful framework for policymakers, advocacy groups, researchers, clinicians, community, and other stakeholders is to understand and implement the most effective lifestyle changes to improve health. The effectiveness of different population strategies should help to inform policy priorities in different countries. Evidence-based interventions could be carried out in combinations, either at the same time or in stages, providing access to improved food, increased physical activity, and reduced tobacco use. If more interventions are made simultaneously, they are much more effective in smoking cessation. At the population level, changes in risk behavior and risk factors can significantly alter health outcomes and the risk of disease. Lifestyle behavior affects a multitude of individual, social, economic, regular mass media, and other environmental factors. Population-based interventions can affect many of these factors. Due to limited funding and other resources for preventive efforts, knowledge and assessment of most evidence-based strategies are essential for promoting priorities.

Academic research centers should focus on projects that would implement knowledge to set policy rules that promise political support and encourage further development.

Smoking behavior is a complex phenomenon, and without help in behavioral interventions, very few people are capable of getting rid of this habit. Behavioral change is divided into the different phases through which people pass to change. Non-effectiveness in taking away a habit is due to the lack of knowledge about behavior, interest, negative experience of

termination, and lack of motivation. Social support of persons plays a key role in adopting health behavior and defines the enjoyment of love, support, and care by family members and others [48]. Emotional support includes love, compassion, acceptance, and respect for the individual. Instrumental means material, real and objective assistance from others [49]. Research has shown that the most common types of social support are information, emotional, and instrumental support.

Schools and educational systems play an important role in the prevention and education of the harmful consequences of tobacco smoking. Investigations have found that the earliest people begin to smoke is at the age of 11–16. The level of knowledge of this population about the harmful consequences of smoking is relatively low. It is important to educate and raise awareness of the need for smoking cessation for educators, as they should demonstrate with their example and point out to children and young people the smoking hazards.

Public and media are included in the national anti-smoking program under the leadership and coordination of governmental institutions as well as other independent professional associations and individuals. Their role, particularly television, radio, Internet, daily press, and film industry, is extremely important in terms of systematic information gathering and educating citizens about the adverse consequences of smoking tobacco, promoting health education, and anti-smoking messages.

# 4. Tobacco laws and policies

Legislation should precede an organized public education strategy, implementation plan and infrastructure, and appropriate mechanisms of control and evaluation of implementation. A legislative policy positively affects smoking reduction and tobacco smoke exposure in public places if strictly implemented [50].

Smoking prohibitions or "smoke-free" laws imply public policies, including crime laws and regulations on occupational health, which prohibit the use of tobacco at work and other public places. The smoke-free environment appears to be effective as a control mechanism in reducing the number of current and future smokers [51]. The smoke-free environment protects the non-smoker's health and has a favorable outcome in reducing smoking. Smoking restriction in public places reduces average tobacco consumption by 4-10% [52]. The aim of the tobacco control strategy is not only to stimulate smoking cessation but also to protect nonsmokers from passive exposure to tobacco smoke in public places, as well as to protect the rights of non-smokers in a non-tobacco smoke environment [53]. Interventions toward young people and the realization of difficulty in availing tobacco, have no effect on the prevalence of smoking, as opposed to a completely smoke-free environment that is an effective measure of reducing smoking in young people [54]. Young people living in a home without tobacco smoke are more likely (74%) to be non-smokers when compared to peers living with parents who smoke [55]. According to World Medical Association, governments are tasked with helping smokers stop smoking and choosing a healthier way of life, not to be scared by the tobacco industry [56].

### 5. Conclusion

Smoking is rightly considered one of the biggest public health problems of today. Noxiousness of smoking and tobacco smoke is associated with loss of treatment costs, loss of productivity due to illness, increased disability, premature death, and environmental damage. Public cooperation in the implementation of the smoking ban laws positively affects smoking reduction and tobacco smoke exposure and conveys the message that smoking is not socially acceptable. By using effective tobacco smoking cessation interventions, health professionals and public health professionals should promote smoking cessation by joint efforts. Contemporary smoking tobacco laws are a prerequisite for a systematic anti-smoking campaign. Their application shows good results if they are systematically enforced along with other key factors such as health, education, media, religious communities, and others. They are aimed at banning the promotion of tobacco and tobacco products, promoting health and quality of life, and systematically and permanently informing citizens of the consequences of smoking as well as providing systematic deterrence assistance.

### Conflict of interest

The authors received no support from any organization for the submitted chapter, no financial relationships with any organizations that might have an interest in the submitted chapter, and no other relationships or activities that could appear to have influenced the submitted chapter.

# **Funding**

None.

# Declaration of authorship

Juranić B, Mikšić Š: *literature search, writing paper*. Rakošec Ž, Vuletić S: *literature search*.

### **Author details**

Brankica Juranić<sup>1\*</sup>, Štefica Mikšić<sup>1</sup>, Željko Rakošec<sup>2</sup> and Suzana Vuletić<sup>3</sup>

- \*Address all correspondence to: juranicbrankica@gmail.com
- 1 Faculty of Dental Medicine and Health Osijek, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
- 2 Department of Culturology, Josip Juraj Strossmayer University of Osijek, Osijek, Croatia
- 3 Catholic Faculty of Theology in Đakovo, Josip Juraj Strossmayer University of Osijek, Đakovo, Croatia

# References

- [1] Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protectional Agency. Smoking and Health Monograph 10. Bethesda, MD: National Cancer Institute; 1999
- [2] Smith Jr SC, Greenland P, Grundy SM. AHA Conference Proceedings. Prevention conference V: Beyond secondary prevention: Identifying the high-risk patient for primary prevention: Executive summary. American Heart Association. Circulation. 2000;101(1): 111-116. DOI: 10.1161/01.CIR.101.1.111. PMID: 10618313
- [3] US Department of Health and Human Services. Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2012. p. 3
- [4] WHO report on the global tobacco epidemia, 2008. The mpower package
- [5] Tobacco Smoke and Involuntary Smoking. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol. 83. Lyon, France: International Agency for Research on Cancer; 2004
- [6] Šimunović M. Zašto (ne) pušiti? : priručnik za suzbijanje pušenja. 3. dopunjeno izdanje ed. Zagreb: vlast. nakl.; 2000. pp. 15-113
- [7] Jonjić A. Zašto pušiti i piti? Rijeka; 1993. str.9
- [8] Blaž-Čop N, Đorđević V. SOS za (ne)pušaće. Zagreb: global komunikacije; 1991. str.21
- [9] Duhan GD. u medicinska encikolpedija, sv.6. zagreb: Jugoslavenski leksikografski zavod; 1970. str.3
- [10] Poljak Ž. Neprijatelj čovječanstva osvaja svijet, u Život i zdravlje. Vol. 3-4. Zagreb; 1987. str.4
- [11] Tadić M. Ovisnost o pušenju u moralno -etičkoj prosudbi.Obnov.život. 2000;55:107-128
- [12] Popović-Grle S. Alergijske bolesti i pušenje. Acta Medica Croatica. 2011;65:141-146
- [13] World Health Organization. Protection from Exposure to Second–Hand Tobacco Smoke: Policy Recommendations. Geneva; 2007
- [14] Sarafino PE. Health Psychology: Biopsychosocial Interactions. New York: John Wiley and Sons; 1994
- [15] Willemse BMW, Postma DS, Timens W, Hackenn HT. The impact of smoking cessation on respiratory symptoms, lung function, airway hyperresponsiveness and inflammation. The European Respiratory Journal. 2004;23:464-476
- [16] Nishikawa M, Nobumasa K, Ito T, Kudo M. Superoxide mediates cigarette-smoke Infiltration of Neutrophils into airways through nuclear factor-K B activation and Il-8 Mrna expression in Guinea pig in vivo. American Journal of Respiratory Cell and Molecular Biology. 1999;20:189-198

- [17] Arcavi L, Benowitz NL. Cigarette smoking and infection. Archives of Internal Medicine. 2004;**164**:2206-2216
- [18] Almirall J, Gonzales CA, Balanzo X, et al. Proportion of community-acquired pneumonia cases attributable to tobacco smoking. Chest. 1999;**116**:375-379
- [19] Kolappan C, Gopi PG. Tobacco smoking and pulmonary tuberculosis. Thorax. 2002;57:
- [20] Leson S, Gershwin ME. Risk factors for asthmatic patients requiring intubation: A comprehensive review. Allergologia et Immunopathologia. 1995;23:235-247
- [21] Perzanowski MS, Divjan A, Mellind RB, et al. Exhaled no among inner-city children in New York City. The Journal of Asthma. 2010;47:1015-1021
- [22] Shah JP, Patel SG. (ur.) Head and Neck Surgery and Oncology. Edinburgh: Mosby; 2003. pp. 173-394
- [23] Sasco AJ, Secretan MB, Straif K. Tobacco smoking and cancer: A brief review of recent epidemiological evidence. Lung Cancer. 2004;45:3-9
- [24] Puranik R, Celermajer DS. Smoking and endothelial function. Progress in Cardiovascular Diseases. 2003;45(6):443-458. DOI: 10.1053/pcad.2003.YPCAD13
- [25] Rigotti NA, Clair C. Managing tobacco use: The neglected cardiovascular disease risk factor. European Heart Journal. 2013;34:3259-3267. DOI: 10.1093/eurheartj/eht352
- [26] Chin D, Boyle GM, Theile DR, Parsons PG, Coman WB. Molecular introduction to head and neck cancer (HNSCC) carcinogenesis. British Journal of Plastic Surgery. 2004;57: 595-602
- [27] Papadimitrakopulou VA. Carcinogenesis of head and neck cancer and the role of chemoprevention in its reversal. Current Opinion in Oncology. 2000;12:240-245
- [28] Quon H, Liu FF, Cummings BJ. Potential molecular prognostic markers in head and neck squamous cell carcinomas. Head & Neck. 2001;23:147-159
- [29] Natali A, Mondaini N, Lombardi G, Del Popolo G, Rizzo M. Heavy smoking is an important risk factor for erectile dysfunction in young men. International Journal of Impotence Research. 2005;17:227-230
- [30] Trummer H, Habermann H, Haas J, Pummer K. The impact of cigarette smoking on human semen parameters and hormones. Human Reproduction. 2002;17:1554-1559
- [31] Kimata H. Selective induction of total and allergen-specific IGE production by passive smoking. European Journal of Clinical Investigation. 2003;33811:1024-1025
- [32] Popović-Grle S. Kriteriji alergološke dijagnostike Bronhalne senzibilizacije u kroničnih opstruktivnih bolesti Pluća (magistarski rad). Medicinski fakultet: Zagreb; 1989
- [33] Lee CH, Chiang HY, Hong CH, et al. Lifetime exposure to cigarette smoking and development of adult-onset atopic dermatitis. The British Journal of Dermatology. 2010. DOI: 10.1111/j.1365-2133.2010.10116x

- [34] Andersen ML, Simonsen U, Uldbjerg N, Aalkjaer C, Stender S. Smoking cessation early in pregnancy and birth weight, length, head circumference, and endothelial nitric oxide synthase activity in umbilical and chorionic vessels: An observational study of healthy singleton pregnancies. Circulation. 2009;119:857-864
- [35] Miyake Y, Tanaka K, Arakawa M. Active and passive smoking during pregnancy and birth outcomes: The Kyushu Okinawa Maternal and Child Health Study. BMC Pregnancy and Childbirth. 2013;13:157. DOI: 10.1186/1471-2393-13-157 [Accessed: Dec 27, 2017]
- [36] Lambers DS, Clark KE. The maternal and fetal physiologic effects of nicotine. Seminars in Perinatology. 1996;20:115-126. DOI: 10.1016/S0146-0005(96)80079-6 [Accessed: Dec 27, 2017]
- [37] Duijts L, Jaddoe VWV, van der Valk R, et al. Fetal exposure to maternal and paternal smoking and the risks of wheezing in preschool children: The generation R study. Chest. 2012;141:876-885. DOI: 10.1378/chest.11-0112 [Accessed: Dec 31, 2017]
- [38] Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010. Lancet. 2012;380:2224-2260. DOI: 10.1016/S0140-6736(12)61766-8 [Accessed: Dec 24, 2017]
- [39] Cheraghi M, Salvo S. Environmental tobacco smoke (ETS) and respiratory health in children. European Journal of Pediatrics. 2009;168:897-905
- [40] Kharrazi M, DeLorenze GN, Kaufman FL, et al. Environmental tobacco smoke and pregnancy outcome. Epidemiology. 2004;15:660-670. DOI: 10.1097/01.ede. 0000142137.39619.60 [Accessed: Dec 24, 2017]
- [41] Cui-Jin MS, Rossignol AM. Effects of passive smoking on respiratory illness from birth to age eighteen months in Shanghai, People's Republic of China. The Journal of Pediatrics. 1993;**123**:553-558. DOI: 10.1016/S0022-3476(05)80949-7 [Accessed: Dec 24, 2017]
- [42] Stanton WA. Longitudinal study of the influence of parents and friends on children's initiation of smoking. Journal of Applied Developmental Psychology. 1992;13:423-434
- [43] Choi WS, Pierce JP, Gilpin EA, et al. Which adolescent experimenters progress to established smoking in the United States? American Journal of Preventive Medicine. 1997;13(5):385-391
- [44] Ellickson PL, Tucker JS, Klein DJ. High-risk behaviors associated with early smoking: Results from a 5-year follow-up. The Journal of Adolescent Health. 2001;28(6):465-473
- [45] Colder CR, Lloyd-Richardson EE, Flaherty BP, et al. The natural history of college smoking: Trajectories of daily smoking during the freshman year. Addictive Behaviors. 2006;31:2212-2222
- [46] Dierker L, Lloyd-Richardson E, Stolar M, et al. The proximal association between smoking and alcohol use among first year college students. Drug and Alcohol Dependence. 2006;81:1-9

- [47] Cook A, Bellis MA. Knowing the risk: Relationships between risk behaviour and health knowledge. Public Health. 2001;115:54-61
- [48] Cobb S. Presidential Address-1976. Social support as a moderator of life stress. Psychosomatic Medicine. 1976;38(5):300-314. DOI: 10.1097/00006842-197609000-00003. PMID: 981490
- [49] Drentea P, Clay OJ, Roth DL, Mittelman MS. Predictors of improvement in social support: Five-year effects of a structured intervention for caregivers of spouses with Alzheimer's disease. Social Science & Medicine. 2006;63(4):957-967. DOI: 10.1016/j.socscimed.2006. 02.020. PMID: 16616406
- [50] Ceronja I. Zašto ograničiti uporabu duhanskih proizvoda. Liječnički Vjesnik. 2013;**135**: 104-109
- [51] WHO. WHO Report on the Global Tobacco Epidemic, 2009: Implementing smoke-free environments. Available from: http://www.who.int/tobacco/resources/publications/en/[Accessed: Dec 24, 2017)
- [52] Jha P. Curbing the Epidemic: Governments and the Economics of Tobacco Control: Development in Practice. Washington: The World Bank; 1999
- [53] Serra C, Cabezas C, Bonfill X, Pladevall-Vila M. Interventions for Preventing Tobacco Smoking in Public Places. The Cochrane Library; 2008
- [54] Fichtenberg CM, Glantz SA. Effect of smoke-free workplaces on smoking behaviour: systematic review. British Medical Journal. 2002;325:188. PMCID:PMC117445
- [55] Ong M, Glantz S. Free nicotine replacement therapy programmes vs implementing smoke free workplaces: A cost effectiveness comparison. American Journal of Public Health. 2005;95(6):969-975. DOI: 10.2105/AJPH.2004.040667 [Accessed: Dec 31, 2017]
- [56] World Medical Association Internet. Geneva: World Medical Association; 2011. Available from: http://www.wma.net/en/40news/20archives/2011/2011\_30/index.html [Accessed: Dec 24, 2017]