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***Editorial***

Tobacco has been traded around the world for centuries as a very profitable commodity thus leading to grow a very strong economical industry. After the catastrophic effect of smoking on human health have been revealed by some pioneer researchers like Sir Richard Doll, this industry counter-attacked every possible way to protect this high profit business. Industry accused people who underlined the effects of tobacco on human health with pseudoscience, support and finance smoking favorable studies, sued the researchers who studied effects of smoking on health and most importantly using political power they avoided governments to constitute anti-smoking laws until early eighties. However the truth became as clear as it can be with the efforts of independent doctors cooperating with WHO. When the industry is sued by people who had been suffered from smoking (people who have cancer etc.) they are shielded back two main arguments usually. Firstly they claimed that to start smoking is a personal initiative (people make a choice to smoke) and secondly is that smokers could stop smoking if they really want to. Today we strongly know that both arguments are wrong. Tobacco is one of the most potent addictive substances in the world and it can be accessed very easily. To cease smoking is hard as some very potent narcotics (cocaine etc.). It is now that the industry is shifting its future enterprises to electronic cigarettes or hookahs (It is discussed in this special issue with a review) especially they aim children and teenagers. It has been estimated that there are at least 18 millions of people who smokes regularly in our country. The danger is clear and present. Smoking cannot be considered as a problem of an exact specialty (Internal Medicine, Chest and lung Diseases etc.). Every effort to solve this problem without primary care professionals (Family practice specialists, family physicians, general practitioners etc.) is likely doomed to fail. However many medical faculties around the world (Including Turkey), standard undergraduate medical education gives a little effort to teach medical students how to help their patients to stop cessation (One of our articles is about a cessation lesson in medical education). In the recent years there are several positive steps are taken for controlling smoking in our country. New laws about smoking prohibition is legalized and a motivation in general public to cease smoking is raised with television commercials. Many physicians are trained for smoking cessation by ministry of health and a free smoking cessation line (171) have been established. With two major brief projects people got free drugs in order to stop smoking. However it can be argued that, this type of approach is the best for public health. To avoid teenagers to start smoking should be the number one priority. Secondary priority should be given to ex-smokers to remain in that status. Lastly to help smokers to quit smoking. For all of this priorities education and prevention is essential. Instead of big budgeted short session cessation projects, low cost and continuous and steady programs (Public education, children and teenager prevention, motivational interventions and nicotine replacement and very well indicated drug therapy) should be more successful.

In this special issue we tried to reveal some of the important aspects of smoking. We have a short report about Fagerstrom Nicotine Addiction Test which is widely used, and reviews about Transteoretical Model, the effects of smoking on cardiovascular and oral health. Apart from several important national and international analytic descriptive studies there are several different we have received several interesting studies analyzing the cessation rates with different interventions. We hope this issue will increase interest of health professionals in this topic.

Prof. Dr. Bektas Murat Yalcin
Editor of Family Medicine Special Issue

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**Review**

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# Effects of smoking on oral cavity

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**ABSTRACT**

Smoking is a common practice and damages almost all organs and systems of the body. Oral cavity is often overlooked yet such an important region. Its rich flora contains many microorganisms that cause local and systemic diseases if microbiological flora is altered. Cigarette smoke renders oral mucosa epithelium to be susceptible for colonization of pathogens. These pathogens can cause or contribute formation of systemic diseases such as diabetes and obesity. Also smoking causes mutations that can lead to cancers. Many cancerous or precancerous lesions and bad breath attributed to smoking. This review focuses smoking related oral cavity conditions and their mechanisms.



## Introduction

Smoking is one of the most important public health problems in the world. Tobacco usage (Cigarette, cigar, pipe, hookah etc) results in a very strong addiction syndrome. This syndrome reflects all basic features of addiction according to DSM V. Although smoking rate is decreasing in developed countries, sadly, smoking is a common practice in developing countries. The most important reasons for this situation are marketing strategies of international tobacco companies and lack of education in developing countries (Caliskan, 2015). It is a known fact that cigarette smoking causes adverse effects on the whole body (Ozturk et al., 2014). While struggling against smoking primary care physicians often neglect the effect of tobacco on oral health. This review aims to help primary care physicians to gain knowledge and improve their perspective about this topic.

## Smoking and oral health

Oral cavity is the initial portion of the digestive tract and it is surrounded by the lips, cheeks, palate, tongue and the mouth floor. The section between teeth, gums, lips and cheeks is called “vestibulum oris”. “Cavitas oris propria” is the inner section surrounded by teeth and gums includes the tongue. Oral cavity is an important structure that hosts both soft and rigid surfaces washed by saliva and open to the external

environment. Smoking causes cancers, mucosal lesions and periodontal diseases in all regions of oral cavity. It increases coronal and root caries. Smokers are notorious for large carries and missing teeth as well as bad breath. (Heintze, 1984; Sayed and Stephen, 2000; Wanda et al., 2007; Aguilar-Zinser et al., 2008; Yıldırım et al., 2010). Oral lesions associated with smoking is shown in Table 1.

**Table 1.** Oral lesions and conditions associated with tobacco use

| |
|---|
| **Oral precancerous lesions** |
| Leukoplakia |
| Erythroplakia |
| Smokeless tobacco keratosis |
| **Oral cancers** |
| ***Squamous cell carcinomas of the*** |
| Tongue |
| Floor of the mouth |
| Lip |
| Gingiva |
| ***Verrucous carcinomas of the*** |
| Buccal mucosa |
| Gingiva |
| Alveolar ridge |
| **Periodontal diseases** |
| Increased plaque and calculus depositions |
| Ischaemia |
| Enfections |
| Periodontal pockets |
| Gingival recession |
| Alveolar bone loss |
| Root caries |
| Peri-implantitis |
| Halitosis |
| Taste derangement |
| Stained teeth and restorations |
| Black hairy tongue syndrome |

(Tomar and Asma, 2000; Gurvits and Tan, 2014; Çetin Kargın and Marakoğlu, 2015)

## The effect of smoke on oral health

Chemical carcinogens in cigarettes corrupts protein and lipid-A- derived 3-OH fatty acid profile in the salivary and causes mutations and chromosome breakages in the DNA (Jeng et al., 2001; IARC, 2004; Borojevic, 2012). Oral lesions associated with smoking in general, is caused by various toxins and carcinogens made from the burning tobacco. With the burning of tobacco, various carcinogens i.e., tar, carbon monoxide, benzopyrene, Cd complex-nitrogen oxide are released or formed besides nicotine. In fact around 4000 chemicals, most of them irritants if not carcinogen, are released from burning tobacco. Cotinine, the most important metabolite of nicotine, is detected in blood, urine and gingival fluid. Cotinine levels of regular smokers in the saliva are found to be more than 100 ng/ml (Özbek and Karabıyıkoğlu, 1996; Tangada et al., 1997; Mızrak and Acun Kaya, 2005).

## Smoking and oral flora

Nicotine affects proliferation, binding and chemotaxis of periodontal ligament cells negatively. Pro-inflammatory cytokines produced by gingival fibroblasts have synergistic relationship with lipo-polysaccharides of *Escherichia coli* and *Porphyromonas gingivalis P. gingivalis* (Giannopoulou et al., 2001). The effects of smoking on oral cavity immunity is shown in Fig. 1. Smoking affects directly periodontopathogen colonies, sub-gingival ecology and increases colonization of the mouth by potential pathogen microorganisms (Blackwell et al., 1992; Grossi et al., 1996). Some of these frequently found pathogens are Actinobacillus actinomycetemcomitans (A.A.), *P. gingivalis*, *Prevotella intermedia* (*P. intermedi* A), Eikenella corrodens (*E. corrodens*) and Fusobacterium nucleatum (*F. nucleatum*) (Kinane and Radvar, 1997).

Smoking makes binding of some pathogen microorganisms to the epithelism easier. The ability of binding to the epithelium is important for colonization of bacteria in the oropharyngeal mucous membranes and stops the destruction of the bacteria. Yetkin at al. isolated pathogen bacteria in 43% of smokers and 20% of non-smokers ($p<0.05$) (Yetkin et al., 2010). Greenberg et al. (2006) showed that more of Haemophilus influenzae and Streptococcus pneumoniae carriage is found in individuals exposed to cigarette smoke.

## Smoking and oral neoplastic effects

Smoking can lead to precancerous lesions and oral cancers related to p53 mutations (Chen et al., 2008; Gibbons et al., 2014; Yeh et al., 2016). Many molecular and immunohistochemical studies found that main p53 mutations occur in 220, 245-248 and 278-281 codons as G —> A, G —> T or G —> C transversions or deletions (Iggo et al., 1990; Somers et al., 1992; Fıeld et al., 1993; Brennan et al., 1995).

"Volatile sulphur compounds" (VSCs) in tobacco smoke are the dominant components that cause halitosis. Moreover smoking contributes to halitosis by causing hyposalivation and periodontal diseases (Al-Atrooshi and Al-Rawi, 2007; Scully and Greenman, 2012). In a study where oral hygiene and smoking evaluated, 82% of smokers and 52% of controls complained of halitosis (Soylu Özler and Akoğlu, 2014). Smoking is an important risk factor for the prevalence, the affected area size and severity of the periodontal diseases (Chambrone et al., 2010). In the United States smoking is responsible for almost half of the cases of periodontitis (Tomar and Asma, 2000). The risk of periodontitis in smokers is increased to 2-7 folds (Susin et al., 2004; César Neto et al., 2012). Pathogens related to periodontal diseases, are shown to be associated with


**Fig. 1.** The effect of tobacco smoking on periodontal tissues (Sham et al., 2003) PMN polymorphonuclear leukocytes **IgA:** Immunoglobulin A; **IgG:** Immunoglobulin; **GIL-1:** Interleukin-1; **TNF-α:** Tumour necrosis factor-alpha; **MMPs:** Matrix metalloproteinase

systemic conditions of cardiovascular disease, stroke, premature or low-weight infants, upper respiratory tract infections, diabetes, obesity, rheumatoid arthritis and renal diseases (Külekçi and Gökbuget, 2009). Therefore tobacco related mouth diseases affects whole body directly or indirectly.

Second hand smoke is thought to be associated with periodontal diseases. Erdemir at al. (2010) studied 109 children and concluded that passive exposure to smoke is a risk in terms of periodontal diseases compared to those without any exposure (Tanner et al., 2005). Real time PCR is shown that smoking increases amount and depth of bacterial invasion (Gomes et al., 2006; Teixeira et al., 2009). Hairy tongue is an interesting finding in smokers. A study has revealed the frequency of hairy tongue in 32.3% of smokers and 16.5% of non-smokers ($p<0.05$) (Özeç et al., 2008).

The damages of Smoking on teeth and oral tissues are related to the amount and duration of usage. Some studies purport that after 10 years of smoking cessation, the risk of oral cancer is equalized to those of nonsmokers but some say despite the reduction, the risk is still higher than non-smokers (Macfarlane et al., 1995).

In conclusion, Oral cavity is an important region overshadowed by cardiovascular or respiratory studies. Smoking may affect oral cavity in different levels from a simple complaint to life threatening conditions. Cessation of smoking will prevent many oral cavity conditions and systemic diseases.

## REFERENCES

Aguilar-Zinser, V., Irigoyen, M.E., Rivera, G., Maupome, G., Sanchez-Perez, L., 2008. Cigarettes smoking and dental caries among professional truck drivers in Mexico. Caries Research. 4, 255-262.

Al-Atrooshi, B.A., Al-Rawi, A.S., 2007. Oral halitosis and oral hygiene practices among dental students. J. Bagh. Coll. Dent. 19, 72-76.

Blackwell, C.C., Tzanakaki, G., Kremastinou, J., 1992. Factors affecting carriage of Neisseria meningitidis among Greek military recruits. Epidemiol Infect. 108, 441-448.

Borojevic, T., 2012. Smoking and periodontal disease. Mat. Soc. Med. 24, 274-276.

Brennan, Ja., Boyle, Jo., Koch, Wm., et al., 1995 Association between cigarette smoking and mutation of the p53 gene in squamous celi carcinoma of the head and neck. N. Eng. J. Med. 332, 712-717.

Caliskan, S., 2015. Üniversite öğrencilerinin sigara kullanımını etkileyen faktörler (ekonometrik bir yaklaşım). Uşak Üniversitesi Sosyal Bilimler Dergisi. 22, 23-48.

César Neto, J.B., Rosa, E.F., Pannuti, C.M., Romito, G.A., 2012. Smoking and periodontal tissues: A review. Braz. Oral. Res. 26 Suppl. 1, 25-31.

Chambrone, L., Chambrone, D., Lima, L.A., 2010. Predictors of tooth loss during long-term periodontal maintenance: A systematic review of observational studies. J. Clin. Periodontol. 37, 675-684.

Chen, Y.J., Chang, J.T., Liao, C.T., Wang, H.M., Yen, T.C., Chiu, C.C., Lu, Y.C., Cheng, A.J., 2008. Head and neck cancer in the betel quid chewing area: Recent advances in molecular carcinogenesis. Cancer Sci. 99, 1507e14.

Çetin Kargın, N., Marakoğlu, K., 2015. Sigara içmeyle ilişkili nadir bir olgu: Siyah dil gelişimi. Türk Aile Hek. Derg. 19, 112-113.

Erdemir, E.O., Sönmez, I.S., Oba, A.A., Bergstrom, J., Caglayan, O., 2010. Periodontal health in children exposed to passive smoking. J. Clin. Periodontol. 37, 160-164.

Fıeld, J.K., Pavelıc, Z.P., Spandıdos, D.A., Stambrook, P.J., Jones, A.S., Gluckman, J.L., 1993. The role of p53 tumor suppressor gene in squamous cell carcinoma of the head and neck. Arch. Otolaryngol. Head Neck Surg. 119, 1118-1122.

Giannopoulou, C., Roehrich, N., Mombelli, A., 2001. Effect of nicotine- treated epithelial cells on the proliferation and collagen production of gingival fibroblasts. J Clin Periodontol. 28, 769-775.

Gibbons, Don L., Byers, Lauren A., Kurie, Jonathan M., 2014. Smoking, p53 Mutation, and Lung Cancer. Mol. Cancer Res. 12, 3-13.

Gomes, S.C., Piccinin, F.B., Oppermann, R.V., Susin, C., Nonnenmacher, CI., Mutters, R., Marcantonio, R.A., 2006. Periodontal status in smokers and never-smokers: Clinical findings and real-time polymerase chain reaction quantification of putative periodontal pathogens. J. Periodontol. 77, 1483-1490.

Greenberg, D., Givon-Lavi, N., Broides, A., Blancovich, I., Peled, N., ÊDagan, R., 2006. The contribution of smoking and exposure to tobacco smoke to Streptococcus pneumoniae and Haemophilus influenzae carriage in children and their mothers. Clin. Infect Dis. 42, 897-903.

Grossi, S.G., Skrepcinski, F.B., DeCaro, T., Zambon, J.J., Cummins, D., Genco, R.J., 1996. Response to periodontal therapy in diabetics and smokers. J Periodontol. 67, 1094-1102.

Gurvits, Grigoriy E., Tan, A., 2014. Black hairy tongue syndrome. World J. Gastroenterol. 20, 10845-10850.

Heintze, U., 1984. Secretion rate, buffer effect and number of lactobacilli and Streptococcus mutans of whole saliva of cigarette smokers and non-smokers. Scand. J. Dent. Res. 92, 294-301.

IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2004. Betel-quid and areca-nut chewing and some arecanut derived nitrosamines. IARC Monogr Eval Carcinog Risks Hum. 85:1e334.

Iggo, R., Gatter, K., Bartek, J., Lane, D., Harrıs, Al., 1990. Increased expression of mutant forms of p53 oncogene in primary lung cancer. Lancet, 675.

Jeng, J.H., Chang, M.C., Hahn, L.J., 2001. Role of areca nut in betel quidassociated chemical carcinogenesis: Current awareness and future perspectives. Oral Oncol. 37, 477e92.

Kinane, D.F., Radvar, M., 1997. The effect of smoking on mechanical and antimicrobial periodontal therapy. J. Periodontol. 68, 467-472.

Külekçi, G., Gökbuget, A., 2009. Ağız mikroflorasının genel sağlığa etkisi. Ankem Derg. 23, 137-145.

Macfarlane, G.J., Zheng, T., Marshall, J.R., Boffetta, P., Niu, S., Brasure, J., Merletti, F., Boyle, P., 1995. Alcohol, tobacco,diet and the risk of oral cancer: A pooled analysis of three case control studies. Eur. J. Cancer (B). 31B, 181-187.

Mızrak, T., Acun Kaya, F., 2005. Sigara Kullanımının periodontal dokular üzerine olan etkisi. Dicle Tıp Dergisi. 32, 102-107.

Ozturk, O., Yalcin, B.M., Unal, M., 2014. The quality of life Difference between smoker and non smoker rheumatoid arthritis and ankylosing spondylitis patients. J. Addict. Res. Ther. 5, 4.

Özbek, M., Karabıyıkoğlu T., 1996. Sigara ve çaya bağlı, tükürükteki kalsiyum, fosfat konsantrasyonları ile optik dansite ve PH değerleri arasındaki farklılıkların biyokimyasal olarak incelenmesi ve değerlendirilmesi. Atatürk Ü. Diş Hek. Fak. Dergisi. 6, 18-22.

Özeç, İ., Taşveren, S., Yeler, D., Kılıç, E., 2008. Sivas'ta 40 Yaş üzeri bireylerde ağız mukozası lezyonlarının yaygınlığının değerlendirilmesi. Cumhuriyet Üniversitesi Diş Hekimliği Fakültesi Dergisi. 11, 10-15.

Sayed, M., Stephen, I., 2000. Tobacco-associated lesions of the oral cavity: Part I. Nonmalignant lesions. J. Can. DentAssoc. 66, 252-256.

Scully, C., Greenman, J., 2012. Halitology (breath odour: aetiopathogenesis and manage-ment). Oral Dis. 18, 333-345.

Sham, A.S., Cheung, L.K., Jin, L.J., Corbet, E.F., 2003. The effects of tobacco use on oral health. Hong Kong Med. J. 9, 271-277.

Somers, K.D., Merrıck, M.A., Looez, M.E., Incognıto, L.S., Schechter, G.L., Casey, G., 1992. Frequent p53 mutations in head and neck cancer. Cancer Res. 52, 5997-6000.

Soylu Özler, G., Akoğlu, E., 2014. Correlation of oral hygiene habits, smoking and nutritional habits with halitosis. J. Ann. Eu. Med. 2, 9-12.

Susin, C., Oppermann, R.V., Haugejorden, O., Albandar, J.M., 2004. Periodontal attachment loss attributable to cigarette smoking in an urban Brazilian population. J. Clin. Periodontol. 31, 951-958.

Tangada, S.D., Califano, J.V., Nakashima, K., Quinn, S.M., Zhang, J.B., Gunsolley, J.C., Schenkein, H.A., Tew, J.G., 1997. The effect of smoking on serum IgG2 reactive with Aa. in early-onset periodontitis patients. J. Periodontol. 68, 842-850.

Tanner, A.C., Kent, R Jr., Van Dyke, T., Sonis, S.T., Murray, L.A., 2005. Clinical and other risk indicators for early periodontitis in adults. J. Periodontol. 76, 573-581.

Teixeira, SRL., Mattarazo, F., Feres, M., Figueiredo, L.C., de Faveri, M., Simionato, M.R., Mayer MP., 2009. Quantification of Porphyromonas gingivalis and fimA genotypes in smoker chronic periodontitis. J. Clin. Periodontol. 36, 482-487.

Tomar, S.L., Asma, S., 2000. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. J. Periodontol. 71, 743-751.

Wanda, C., Gonsalves, W.C., Chi, A.C., Neville, B.W., 2007. Common oral lesions: Part I. Superficial mucosallesions. Am. Fam. Physician. 7, 501-506.

Yeh, C.Y., Lin, C.L., Chang, M.C., Chen, H.M., Kok, S.H., Chang, S.H., Kuo, Y.S., Hahn, L.J., Chan, C.P., Lee, J.J., Jeng, J.H., 2016. Differences in oral habit and lymphocyte subpopulation affect malignant transformation of patients with oralprecancer. J. Formos Med. Assoc. 115, 263-268.

Yetkin, G., Ay, S., Yetkin, Ö., Taştekin, N., Güçlüer, N., 2010. Sigara içiminin nazofarinks'de potansiyel patojenlerin taşıyıcılığına etkisi. Erciyes Medical Journal. 32, 009-014.

Yıldırım, G., Demirbaş Kaya, A., Ateş, M., 2010. Sigara içen ve içmeyen genç bireylerde bazı tükürük özelliklerinin karşılaştırılması. İstanbul Üniversitesi Diş Hekimliği Fakültesi Dergisi. 44, 167-173.

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Review

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# Transtheoretic model in smoking cessation

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**ABSTRACT**

Smoking is one of the major public health problems and a major cause of preventable diseases. Today there are many ways to combat with tobacco use which is the chief risk factor for avoidable diseases. Pharmacotherapy and other supportive therapies based on motivation and cognitive-behavioral approaches are used in treatment. Among those, concentrating on behavioral changes are gaining more popularity as number of people who stop smoking using behavioral therapies are increasing, so is the interest on psychological models. Transtheoretic model is known as behavioral changes model which is widely used in smoking cessation and developed for the first time by Prochaska and DiClemente. It uses appropriate intervention according to the stage of the individual. According to Transtheoretic model, five stages are to be passed for behavior change. Motivational techniques are important for succesfull passing of a stage and should be structured for preparation to the next stage. Each stage should be evaluated for the transition to the next stage. Transtheoretic model is a significant tool for smoking cessation with its ability to use different models of behavior changes. This flexibilitaly of Transtheoretic model makes the model treatment of choice in different addictions. In this review we focus on the features of Transtheoretic model.



## 1. Introduction

Smoking is a major public health problem and a major cause of preventable diseases. Smoking kills more than five million people every year which will exceed eight million in 2030 if current trend continues. According to the Global Adult Tobacco Survey 2012 results, prevalence of smoking among men is 41.4% and among women is 13.1% in Turkey (Turkey Statistical Institute, 2012). In contrast to previous years, smoking is increasing among women causing significant problems. Therefore, various preventive and theruapatic measures are taken to curb the dangers of smoking. Pharmacotherapy and other supportive therapies based on motivation and cognitive-behavioral approaches are used in treatment (Manfredi et al., 1999; Bilir, 2005; Le Foll et al., 2005; Karlıkaya et al., 2006; Koyun, 2013).

Behavior modification is a difficult and complex procedure. One of the reasons that behavior change attempts fail is that the person is not ready for change (Prochaska and DiClemente, 1982; Beck Institue, 2012).

One theory or combination of different theories can be applied for behavior change (Arkin et al., 2002). However explanation of health behavior does not seem possible with a single theory. Existing theories are used for understanding of a behavour and planning to change it. Commonly used models are: Health Beliefs Model, Health Focus of Control, Diffusion of Innovation, Pender's Health Promotion Model, Transtheoretical

Model/Stages of Change Model (Redding et al., 2000; Özvarış, 2011).

Transtheoretic model 'TTM' also known as 'stages of change' was developed in 1982 by psychologist James O. Prochaska and Carlo DiClemente (Greene et al., 1999; Özvarış, 2011; Pro-change behavior system, 2012). This model is formed from different psychotherapy theories (Prochaska and Norcross, 2010).

In TTM, change of the behavior stages are; 1-Precontemplation (not ready or thinking of change), 2-Contemplation (getting ready), 3-Preparation (ready), 4-Action, 5-Maintenance. For the success individual should be supported according to the stages and personnel needs (Prochaska and DiClemente, 1982). The superiority of TTM is using models from different theories and moreover it directs the therapies according to the stage of the individual (Prochaska and Velicer, 1997).

Surgeon General of the United States used 'nicotine addicition' term for the first time in 1964, 'very strong addictive substances' in 1979 and 'addictive substance in tobacco is nicotine that causes pharmacological and psychological addiction similar to heroin and cannabis' in 1988 (US Department of Health and Human Services, 1964, 1979, 1988). Cigarette addiction is classified as a chronic disease according to the ICD-10 International Classification of Diseases hence requires treatment (Öztuna, 2005; WHO, 2005).

**Transtheoretical model**

TTM uses appropriate intervention according to the stage of the individual. Each stage should be evaluated for the transition to the next stage. It could progress linear or spiral pattern hence can return to the previous stage (Erol and Erdoğan, 2007). Giving same treatment information to everyone in different stages leads to resistance therefore is not recommended (Cingözbay et al., 2011). Giving advanced stage treatment informations on the earlier stages leads to the development of resistance when that stage is reached.

Instead of information on treatment approaches and other issues, motivational techniques are recommended for the people who decided to quit smoking. TTM is appropriate for this purpose as methods are chosen for the individual's stage (Velicer et al., 1998). TTM uses many behavioral, cognitive techniques (Miller and Rollnick, 2002).

TTM is uptaded regularly (Prochaska and DiClemente 1983; DiClemente et al., 1985; Velicer et al., 1985; Prochaska and DiClemente, 1986; DiClemente et al., 1991; Velicer et al., 1992; Prochaska et al., 1993; Prochaska et al., 1994; McConnaughy et al., 1998; Prochaska et al., 2001). Contemplation stage is the most important stage for the efficacy of the model. Many factors from the amount of cigarettes to previous quit attempts are important for this stage (Woodruff et al., 2006).

According to the model; person goes through various stages until behavior change. These stages are described with several concepts (Table 1) (Prochaska et al., 1993; Prochaska and Velicer, 1997; Velicer et al., 2000; Cancer Prevention Research Center, 2012; Koyun, 2013).

TTM is increasingly being used for various unwanted behavioral changes including smoking, diet, weight loss, stress management, drug addiction, obesity, routine pelvic examination and condom use. Unwanted behaviors have the potential to affect qulity of life of individuals and public health (Prochaska and DiClemente, 1983; Prochaska et al., 1994; Prochaska and Velicer, 1997).

**Stages of change**

According to TTM five stages are to be passed for behavior change. Motivational techniques are important for succesfull passing of a stage and should be structured for preparation to the next stage (Velicer et al., 1998).

The individual usually progresses to the next stage but for various resons he may return to a previous stage (relapse). In stopping smoking without assistance these stages often follow spiral patterns. Smokers who reach to Action and Maintenance stages revert to previous stages. Studies indicate that only 5% of those who thought of quitting reach to the maintenance stage. In those who started smoking again, 15% reverts to precontemplation, 85% to Contemplation and preperation stages. In TTM each stage has different properties therefore; completing each stage is important (Anczak and Nogler, 2003). Future stage informations create resistance. In various studies smokers are shown to be in the stages of precontemplation (50-60%), contemplation (30-40%) and preparation (%10-15) (Rodgers et al., 2001). These and other studies suggest that identification of stages is as important as the treatment. Inappropriate treatment for the stages cause failure and creates bad tales before next attempts.

**Step 1: Precontemplation (not ready)**

In the mind of the person at this stage, there is no thought about changing behavior within six months (Woodruff et al., 2006). The person either does not know the harms of unwanted behavior or does not care. Usually he/she does not like to receive information that will help to change the behavior and escapes. Friends and family pressure may reinforce the behavior rather than avoid it. Becouse of the previous failed change attempt, the morale and motivation is lacking. That leads resistance to change behavior and a serious drop in confidence (Velicer et al., 1998).

**Table 1**. Structure of The Transtheoretical Model

| Structures | Descriptions |
|---|---|
| **Stage of change** | |
| Precontemplation | Not thinking of quitting tobacco in the next 6 months. |
| Contemplation | Thinking of quitting tobacco in the next 6 months |
| Preparation | Thinking of taking action within 30 days |
| Action | Changed the behavior within the past 6 months |
| Maintenance | Behavior change more than 6 months |
| **Decisional Balance** | |
| Gains | Benefits of change |
| Costs | Costs of change |
| **Self-Efficiacy** | Self confidence to maintain healthy behavior when face temptation in trying situations |
| **Process of change** | |
| **Experiential** | |
| 1. Consciousness Raising [Increasing awareness] | Get the Facts *"I recall information people had given me on how to stop smoking"* |
| 2. Dramatic Relief [Emotional arousal] | Pay Attention to Feelings *"I react emotionally to warnings about smoking cigarettes"* |
| 3. Environmental Reevaluation [Social reappraisal] | Notice Your Effect on Others *"I consider the view that smoking can be harmful to the environment"* |
| 4. Social Liberation [Environmental opportunities] | Notice Public Support *"I find society changing in ways that make it easier for the nonsmoker"* |
| 5. Self Reevaluation [Self reappraisal] | Create a New Self-Image *"My dependency on cigarettes makes me feel disappointed in myself"* |
| **Behavioral** | |
| 1. Stimulus Control [Re-engineering] | *"I remove things from my home that remind me of smoking"* |
| 2. Helping Relationship [Supporting] | *"I have someone who listens when I need to talk about my smoking"* |
| 3. Counter Conditioning [Substituting] | *"I find that doing other things with my hands is a good substitute for smoking"* |
| 4. Reinforcement Management [Rewarding] | *"I reward myself when I don't smoke"* |
| 5. Self Liberation [Committing] | *"I make commitments not to smoke"* |

**Step 2: Contemplation (Getting ready)**
The person is aware of the problem and wants to start the behavioral changes within six months. He/she tries to gather information and calculates gains and costs. Unable to take action he searches methods of change. Person can remain in this stage for a long time (Prochaska and Velicer, 1997; Koyun, 2013).

**Step 3: Preperation (Ready)**
In this stage the person is ready to take action soon (within the next month). There are usually unsuccessful small attempts. In their head they prepare an action (starting a gym, getting professional help or making individual change plan). In this period they fear failure and need support from friends (Prochaska and Velicer 1997; Woodruff et al., 2006).

**Step 4: Action**
Unwanted problematic behaviors have changed in the last six months and even began to obtain healthy behaviors. In this stage care should be taken to prevent return of the unwanted behavior and get used to the new bevavior. People are generally proud of sharing their success hence increase motivation (Prochaska and Velicer, 1997; Koyun, 2013).

**Step 5: Maintenance**
The person on this stage is free of unwanted behavior more than six months. The aim is make the bevoviour change permenant. He/she is now more resistant to the unwanted behavior. Therefore, motivation and confidence is increasing. The possibility of a return to the previous steps or changed behavior gets smaller by the time but caution is necessary at all times (Woodruff et al., 2006).

**Decisional balance (DB)**
One of the key elements in TTM is DB which is evaluation according to costs and benefits, proposed for the first time in 1977 by Janis and Mann (Miller et al., 2001). It is based on comparisons between pros and cons. In the initial stages losses or cons might outweigh the benefits or pros, i.e precontemplation stage. As the stages progress the balance should shift in favor of gains if the intended behavior change be permenant. In the contemplating stage balance should be equal between gains and losses so when the balance tipped towards gains than he/she can move into preparation or action stage. In the maintenance stage gains should outweigh costs in order to prevent relapse.

**Self-efficacy**
TTM uses self-efficiacy theory of Bandura, 1982. The relation between behavior change and self-efficiacy is defined clearly. Prochaska et al. (1997) described two components in self-efficiacy:

**1- Self-confidence** is the main component that prevents a return to the previous step or unwanted behavior in relaps triggering situations (Bandura, 1982).
**2-Temptation** is defined as the degree of desire for relapse.

The balance between these to should be tipped in favour of confidence to prevent relapse. In the precontemplation and contemplation stages temptation is greater than self confidence. But in action stage these are almost equal and behavior change occurs (Plummer et al., 2001). Self-efficiacy is important in all stages. Lack of motivation results in returning previous stages (Miller and Rollnick, 2002).

**Processes of change**

The methods and techniques used have compatible structures with behavior change stages. The application of methods and techniques is significantly less in the precontemplation stage. Their use increases in contemplation and preparation stages and peaks in action stage. There is a decrease again in maintenance stage. In the early stages cognitive methods are recommended. In later phases (preparation, action, maintenance) behavioral methods are recommended (Cancer Prevention Research Center, 2012). Behavior change is realized using 10 methods; 5 cognitive and 5 behavioral (Tablo 1) (Prochaska et al., 1993; Prochaska and Velicer, 1997; Cancer Prevention Research Center, 2012; Koyun, 2013).

In smoking addiction TTM measurement tools can be considered for evaluation. According to the stages of change, smoking cessation should be planned. Behovior change models should be taught to all health workers and included in the curriculum. By training primary care staff more of the smokers can be reached.

TTM is a significant tool for smoking cessation with its ability to use different models of behavior changes. This flexibilitaly of TTM makes the model treatment of choice in different addictions.

**REFERENCES**

Anczak, J.D., Nogler, R.A., 2003. Tobacco cessation in primary care: Maximizing intervention strategies. Clin. Med. Res. 3, 201-216.

Arkin, E., Maibach, E., Parvanta, C., 2002. General public: Communicating to Persuade, D., Nelson, R., Brownson, P. Remington, C., (Ed.). Communicating public health information effectivelly, Washington: American public health association. pp: 17-21.

Bandura, A., 1982. Self-efficacy mechanism in human Aaency. Am. Psychologist. 37, 122-147.

Beck Institute, 2012. Cognitive behavior therapy. http://www.beckinstitute.org/what-is-cognitive-behavioral-therapy/?gclid=CIeH9pXD_K0CFQGFDgodzWA8rg. 15.03.2016.

Bilir, N., 2005. Sigara Kullanımının Kadın Sağlığına Etkileri ve Kontrolü. s.1-7. Access: 22.03.2012.

Cancer Prevention Research Center, 2012. Transtheoretical model. http://www.uri.edu/research/cprc/TTM/detailedoverview.htm. Access: 15.03.2016.

Cingözbay, B.Y., Işılak, Z., Tokatlı, A., Uzun, M., 2011. Editöre mektuplar. Anadolu Kardiyoloji Dergisi, 11, 467.

Di Clemente, C.C., Prochaska, J.O., Fairhurst, S., Velicer, W.F., Rossi, J.S., Velasquez, M., 1991. The process of smoking cessation: An analysis of precontemplation, contemplation and contemplation/action. J. Consult. Clin. Psychol. 59, 295-304.

Di Clemente, C.C., Prochaska, J.O., Gibertini, M., 1985. Self-efficacy and the stages of self-change of smoking. Cognit Ther Res. 9, 181-200.

Erol, S., Erdoğan, S., 2007. Sağlık davranışlarını geliştirmek ve değiştirmek için transteoretik modelin kullanılması. Atatürk Üniversitesi Hemşirelik Yüksekokulu Dergisi. 10, 86-94.

Greene, G.W., Rossi, S.R., Rossi, J.S., Velicer, W.F., Fava, J.L., Prochaska, J.O., 1999. Dietary applications of the stages of change model. J. Am. Diet. Assoc. 99, 673-678.

Karlıkaya, C., Öztuna, F., Solak, Z.A., Özkan, M., Örsel, O., 2006. Tütün kontrolü. Toraks. 7, 51-64.

Koyun, A., 2013. Yetişkin kadınlara değişim aşamaları modeli (transteoretik model) ile yapılan eğitimin sigarayı bırakma davranışı üzerine etkisi. Hacettepe Üniversitesi Sağlık Bilimleri Enstitüsü. Doğum, Kadın Hastalıkları Hemşireliği Programı. Doktora Tezi. Ankara. pp: 32-40.

Le Foll, B., Melihan-Cheinin, P., Rostoker, G., Lagrue, G., AFSSAPS, 2005. Smoking cessation guidelines: Evidence-based recommendations of the French Health Products Safety Agency. Eur. Psych. 20, 431-441.

Manfredi, C., Crittenden, K.S., Warnecke R, Engler J, Cho Y, Shaligram C., 1999. Evaluation of a motivational smoking cessation intervention for women in Public Health Clinics. Preventive Med. 28, 51-60.

McConnaughy, E.A., Prochaska, J.O., Velicer, W.F., 1998. Stages of change in psychotherapy-measurement and sample profiles. Psychotherapy: Theory, Research and Practice. 20, 368-375.

Miller, M.P., Gillespie, J., Billian, A., Davel, S., 2001. Prevention of smoking behaviors in middle school students: Student nurse interventions. Public Health Nurs. 18, 77-81.

Miller, W.R., Rollnick, S., 2002. Motivational interviewing. The Guilford Press. New York. London. pp: 274-275.

Öztuna, F., 2005. Sigara bırakma polikliklerinde tedavi ve izlem. J. Med. Sci. 25, 546-550.

Özvarış, Ş.B., 2011. Sağlığı geliştirme ve sağlık eğitimi. Hacettepe Üniversitesi Yayınları. Ankara. pp: 40-59.

Plummer, B.A., Velicer, W.F., Redding, C.A., Prochaska, J.O., Rossi, J.S., Pallonen, U.E., Meier, K.S., 2001. Stage of change, decisional balance, and temptatios for smoking measurement and validation in a large, school-based population of adolescent. Addict. Behav. 26, 551-557.

Pro-change Behavior System, 2012. Transtheoretical Model. http://www.prochange.com/ttm. Access: 28.03.2012.
Prochaska, J.M., Prochaska, J.O., Levesque, D.A., 2001. A transtheoretical approach to changing organizations. Adm. Policy. Ment. Health. 28, 247-261.
Prochaska, J.O., DiClemente, C.C., 1982. Transtheoretical therapy toward a more integrative model of change. Psychotherapy: Theory, Research, Practice. 19, 276-288.
Prochaska, J.O., DiClemente, C.C., 1983. Stage and processes of self change of smoking: Toward and integrative model. J. Consult. Clin. Psychol. 51, 390-395.
Prochaska, J.O., DiClemente, C.C., 1986. Toward a comprehensive model of change. Miller, W.R., Heather, N., Ed. Treating addictive behaviors: Processes of change. Plenum Press. New York. pp: 3-27.
Prochaska, J.O., DiClemente, C.C., Velicer, W.F., Rossi, J.S., 1993. Standardized, individualized, interactive, and personalized self-help programs for smoking cessation. Health Psychol. 12, 399-405.
Prochaska, J.O., Norcross, J.C., 2010. Systems of psychotherapy: A transtheoretical analysis. 7th Ed. Brooks and Cole. pp: 64-73.
Prochaska, J.O., Redding, C.A., Evers, K.E., 1997. The transtheoretical model and stages of change. Glanz, K., Lewis, F.M., Rimer. B.K., Ed. Health behavior and health education: Theory, research, and practice. Jossey-Bass. San Francisco. pp: 60-84.
Prochaska, J.O., Velicer, W.F., 1997. The transtheoretical model of health behavior change. Am. J. Health Promot. 12, 38-48.
Prochaska, J.O., Velicer, W.F., Rossi, J.S, Goldstein, M.G., Marcus, B.H.,1994. Stages of change and decisional balance for 12 problem behaviors. Health Psychol. 13, 39-46.
Redding, C.A., Rossi, S.J., Rossi, R.S., Velicer, W.F., Prochaska, J.O., 2000. Health behavior models. Int. Elec. J. o Health Edu. 3, 180-193.
Rodgers, W.M., Courneya, K.S., Bayduza, A.L., 2001. Examination of the transtheoretical model and exercise in 3 populations. Am. J. Health. Behav. 25, 33-41.
Turkey Statistical Institute, 2012. Global Adult Tobacco Survey . http://www.tuik.gov.tr/PreHaberBultenleri.do?id=13142. Access: 15.11. 2013.
US Department of Health and Human Services, 1964. Reducing the health consequences of smoking: A Report of the surgeon general. Atlanta, Georgia US, Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promontion, Office on Smoking and Health. Surgeon Generals Report. pp: 34.
US Department of Health and Human Services, 1979. Smoking and health: A Report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promontion, Office on Smoking and Health. Surgeon Generals Report. pp: 15.
US Department of Health and Human Services, 1988. The health concequences of smoking: Nicotine addiction. A Report of the surgeon general. Rockville, US Department of Health and Human Services, Public Health Service, Centers for Health Promotion and Education. Office on Smoking and Health. DHHS Publication. 88, 8406.
Velicer, W.F., DiClemente, C.C., Prochaska, J.O., Brandenburg, N., 1985. Decisional balance measure for assessing and predicting smoking status. J. Pers. Soc. Psychol. 48, 1279-1289.
Velicer, W.F., Prochaska, J.O., Fava, J.L., Norman, G.J., Redding, C.A., 1998. Smoking cessation and stress management: Applications of the transtheoretical model of behavior change. Homeostasis. 38, 216-233.
Velicer, W.F., Prochaska, J.O., Rossi, J.S., Snow, M.G., 1992. Assessing outcome in smoking cessation studies. Psychol. Bul. 111, 23-41.
Woodruff, S., Lee, J., Conway, T., 2006. Smoking and quitting history correlates of readiness to quit in multiethnic adolescents. Am. J. Health Behav. 30, 663-674.
World Health Organization (WHO), 2005. Tütün kontrolünde sağlık profesyonellerinin rolü. Bilir, N. Ed. Tütünsüz Yaşam Derneği. Ankara. pp:1-20.

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# Are electronic cigarettes saviors or new culprits?

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**ABSTRACT**

Electronic cigarette (EC) usage in the world is increasing. It might outsell the other tobacco products in the future with this rate of selling increase. Promoters of EC argue that using them will bring additional benefits in the fight against tobacco. By switching from cigarettes to ECs, smokers will not be exposed to a number of dangerous substances thus; morbidity and mortality of smoking would decrease. This may cause major epidemiologic changes. Opponents argue that none of these claims are proven. Nicotine itself is not only addictive but also a harmful substance. Besides it may disseminate tobacco use young people into nicotine addiction with its new harmless image. These devices imitate the hits of nicotine, therefore feed the addiction rather than prevent it. People simply may continue smoking and use EC where cigarettes are socially or legally unacceptable. The solutions of nicotine carry the risk of using unhealthy mixtures and inappropriate nicotine dosing. Users simply may abuse these devices to use other substances such as cocaine. ECs are not recognized as a form of nicotine replacement therapy. In fact most health authorities are negative on ECs. Legal issues like using ECs in public places are not clear. Health professionals are increasingly facing the questions regarding EC usage and fail to provide satisfactory answers.



## 1. Introduction

After Herbert A Gilbert got a patent for a device for a non-tobacco cigarette at 1963 he intended that he had made a discovery replacing burning tobacco and paper. This invention made little interest because the harmful effects of smoking was not fully understood at those days. Another inventor Hon Lik from China designed a different device basically depending on the principles of piezoelectric ultrasound at 2001. This device got commercial success in both eastern and western markets afterwards. In the past decade there is an increasing trend for electronic cigarettes. In this review we tried to discuss the current usage, advantages and disadvantages of electronic cigarettes in the view of current literature.

### What is an electronic cigarette?

Electronic cigarette (EC) is a battery powered electronic device that heats and vaporizes solutions mostly with nicotine. The main parts of the device is presented in Fig. 1. Some have replaceable cartridge, some are disposable. When the liquid evaporate the user inhales this vapor to receive nicotine similar to cigarette' reach to brain ratios. This

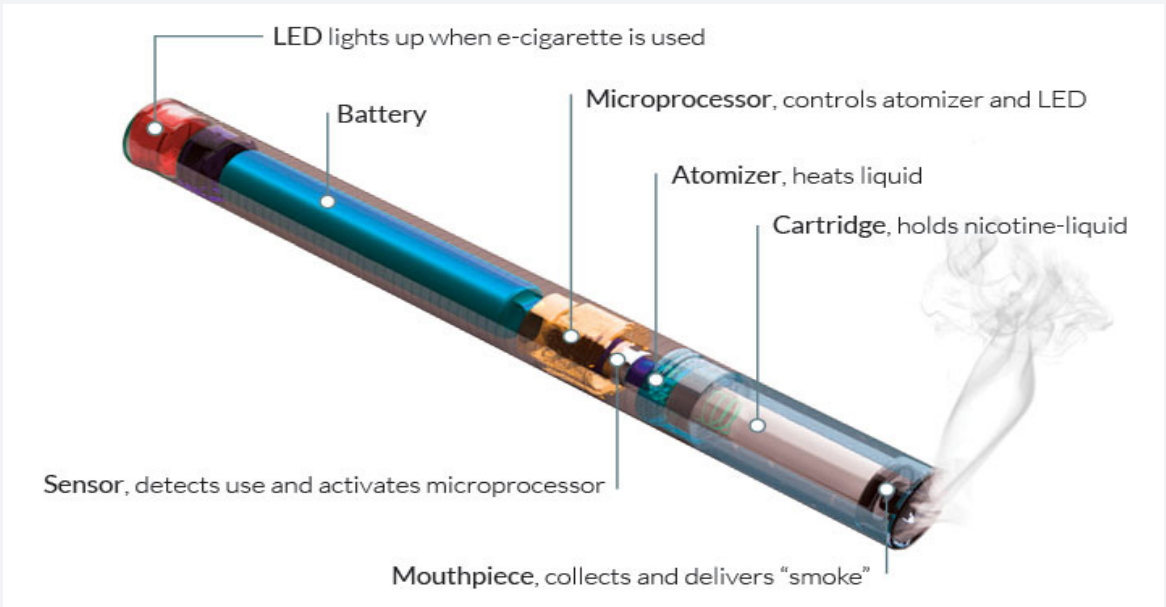


**Fig. 1.** Illustration of electronic cigarette

liquid has drugs, chemicals, flavoring and coloring agents which some of them aim to attract young users. Known agents in these liquids up to date are: Nicotine, glycerol, propylene glycol, Ethylene glycol, 1,3 popandiol, thujone, tobacco specific nitrosamines (N-nitrosonicotinine, N-nitrosoanabasine, N-nitrosonabatine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone), tobacco-specific impurities (cotinine, anabasine and myosmine), flavouring agents (ethyl vanillin, vanilla extract, malic acid, linalool, menthol beta-daascenone, acetyl pryazine, tabanone, ethyl ecetate, ethyl maltol, 2,3,5,6-Tetramethylpryazine, 2-Hydroxy-3-Methylcyclopent-2-en-1-one, 2-Acetylpryidine, 4-(2,6,6-Trimethylcyclohex-1-ene-1-yl)-but-3-3-ene-3-one, L-Menthan-3-one) (Hahn et al., 2014). EC liquids can contain rimonabant and amino-tadalafil. This makes EC a libido enhancing device (Hadwiger et al., 2010).

**How are they marketed?**

Marketing ECs seems to be another success story for tobacco companies as the market grows rapidly. Tobacco companies initiated EC sales in 2006 and reached to estimate billion dollar profits. Global EC market is expected to grow over $50 billion by 2015 to 2025 (Research and Market, 2015). The companies are achieving these figures despite negative approach of most governments. They are in intense campaign to portray EC as harmless product. Even use term of 'vaping' instead of smoking. It is marketed online or in local suppliers as an aid to smoking cessation or a safe alternative for smoking. Smoking EC cost less than cigarettes. This pricing policy and being able to use indoors are making EC popular. This popularity created a new generation with nicotine addiction. Authorities are taking a number of steps to limit dissemination of ECs (applying same rules for tobacco i.e., sale ban to people under age 18). Banning the use of flavored nicotine liquid is also considered (FDA, 2015). Some of the powerful cigarette companies are also producing ECs (Reynolds American Plc, Altria, Imperial Brands Plc). Industry can put a lot of resources on marketing and public relations.

**Rose project**

Inhalation is very favorite way of delivering substances aimed for brain. Cigarette is an ingenious way of delivering nicotine as it reaches brain in seconds after a puff. Developing a way to deliver nicotine as efficient as a cigarette is a goal for some scientists. Rose Project is developing a device that does not use heat. When Smokers take a puff pyruvic acid and nicotine mix and form nicotine-pyruvate cloud. Pyruvic acid is a naturally occurring substance in the body so it won't add toxicity to nicotine. Smokers will be free of tars and other harmful substances in the cigarettes (Rose et al., 2010). But using nicotine as a recreational substance contradicts the philosophy of stopping addictions.

**Nicotine inhalers**

Nicotine inhalers (Nls) are established form of nicotine replacement therapy (NRT). However they are not ECs and they have different action mechanisms. The nicotine inhaler consists of a plastic mouthpiece and plastic tube with a cartridge containing nicotine. People can practice the hand-to-mouth ritual of smoking.

When take a puff the inhalator releases nicotine into mouth then absorbed through the lining of the mouth, throat and upper esophagus just like nicotine gum. There is no risk of passive nicotine exposure. Each puff contains about 10 times less nicotine than a puff of a cigarette hence no risk of addiction to nicotine. Peak nicotine dose occur after 10-15 minutes.

**The rationale behind the use of EC**

E-cigarettes are cheaper, cleaner, more flavorful, and less harmful than tobacco. They seem to be viable alternative to smoking cigarettes. Almost one-fifth of smokers who try ECs once go on to become regular users (Etter and Bullen, 2014). Cigarette smokers may switch to ECs in order the decrease the risks of smoking. If ECs are used instead of tobacco, users will not be exposed to many harmful chemicals and tar normally present in cigarettes. Around 4000 different chemicals with 40 known cancerogens make tobacco a very dangerous substance. If only nicotine is responsible for addiction supplying only nicotine with similar delivery system seems to make sense. The ideal goal is stopping cigarette all together. But unfortunately fighting nicotine addiction is difficult task with high relapse rate. ECs may help prevention of relapse in people with severe cravings. It may help to those who want to stop smoking. Smokers are addicted to the behaviors associated with smoking besides nicotine. ECs may fulfill these hands to mouth rituals and fight psychological components of smoking.

The most common reason for using ECs is to reduce consumption of cigarettes. ECs may be useful in cutting the number of cigarettes when compared to nicotine patch and placebo. EC may also protect those exposed to frequent secondhand smoke. If ECs were to replace conventional cigarettes, it could have a substantial impact on public health. Smoking related morbidity and mortality would decrease.

People with depression and schizophrenia use nicotine to self medicate themselves. In fact many psychiatric wards have a smoking room. While there may be some short term benefits of using cigarettes for psychiatric patients the health risks are too great. ECs may provide alternative solution for these people. No serious adverse event (SAEs) in the short term related to EC use is reported. All these benefits seem to make EC a good alternative to smoking. However the grades of quality of these studies are classed as low and very low (Boyce et al., 2016).

**Arguments against EC**

EC promoters wants to create an image that EC is less dangerous than smoking in the same way they did in 1950s when they introduced filtered cigarettes and claimed that filtered cigarettes do not cause cancer. Two main arguments against ECs are: Lack of evidence of efficacy and safety and the unknown long term effects. ECs are not an accepted form of NRT. Quality and ingredients of ECs needs monitoring. The vapor in these devices is not proved to be safe. ECs expose both users and bystanders to particulate matter (Schober et al,. 2014). Lung and immune system damage is being documented on mice (Sussan et al., 2015). They may trigger curiosity and encourage young people for usage. It still is not clear whether EC may help smokers to quit, or make people user of EC and tobacco cigarettes (CDC, 2015). No country is endorsed ECs and they are bringing same legislation as tobacco.

**Nicotine**

Nicotine is a strong addictive substance. Cigarettes are the most efficient nicotine-delivery device. Inhaling smoke loaded with nicotine disseminates through the lungs and rapid nicotine boost into the bloodstream occurs. The acute effects of nicotine diminish in a few minutes and the smoker should continue dosing for pleasure and prevention of withdrawal symptoms. No NRT can imitate this action. Only ECs are capable of delivering nicotine in the vapor. Therefore ECs are proposing recreational use of nicotine. If medical establishment accepts EC as a form of NRT, it will have endorsed and legitimized an addictive substance. This may disseminate nicotine addiction.

Nicotine has toxic effects. Exposure to nicotine during pregnancy harms the developing fetus, and causes lasting consequences for the developing brain and lung function in newborns. Nicotine exposure also affects maternal and fetal health during pregnancy, and can result in low birth weights, preterm delivery and stillbirth. Nicotine has a negative impact on adolescent brain development. Human brain development continues far longer than was previously realized, and nicotine use during adolescence and young adulthood has been associated with lasting cognitive and behavioral impairments, including effects on working memory and attention (Surgeon General Report, 2014). Nicotine may be carcinogen and appears to promote the spread of existing tumors (Sanner and Grimsrud, 2015). Nicotine is toxic when ingested. The lethal dose is 6-13 mg/kg. Side effects of the nicotine with different forms are: Dyspepsia, nausea, diarrhea, dry mouth, hiccup, flatulence, increased salivation, stomatitis, oral blistering, tooth disorder, glossitis, unpleasant taste, decrease in lower esophageal sphincter pressure, heartburn, chest discomfort, bronchitis, bronchospasm in patients with asthma, sore throat, increase in heart rate and blood pressure, new ventricular and supraventricular tachycardia, ST- or T-wave changes, rare MI, local skin irritation with patches, lightheadedness, headache, sleep disturbances, abnormal dreams, irritability, dizziness, and tremor. Stroke due to severe cerebral artery vasospasm has

been reported in a patient with a recent history of subarachnoid hemorrhage shortly after applying a 10 mg nicotine. Longterm side effect are included as arthralgia, myalgia, jaw pain, increased platelet aggregation and thrombus formation, hyperinsulinemia and insuline resistance (Drugs.com, 2016).

**Nicotine solutions and safety of the device**

Nicotine solutions are not regulated yet, their labels may be inaccurate, their contents and inhalation devices are prone to contamination. They are available usually in 100 mg bottles with various concentrations. The lethal dose when ingested is 6-13 mg/kg. One sip of 5 ml can render a child 500 mg of nicotine. Nicotine acts like succinylcholine causing seizures and paralysis in 15-30 minutes. Rapid response with treatment of benzodiazepins and induced coma is required. Succinylcholine should not be used for intubation.

Propylene glycol, glycerol or ethylene glycol in these solutions may form toxic or cancer-causing compounds when vaporized. ECs produces formaldehyde in similar level or higher than cigarettes. Anabisine, myosmine and beta nikotryinne also detected in ECs. Anabisine is present in tobacco to protect the plant from harmful pests. Some food additives are generally considered safe when eaten; this does not mean that these chemicals are safe when inhaled, as a vapor (Hutzler et al., 2014). Further problem might occur when people starts experimenting with different recreational chemicals in these solutions. Possibilities are endless.

Diacetyl, a buttery flavored chemical often added to food products such as popcorn, caramel, and dairy products, has also been found in some ECs with flavors. Diacetyl can cause a serious and irreversible lung disease commonly known as "popcorn lung." Many of these chemicals are produced for oral consumption. We don't know effects of these chemicals when they are inhaled (Farsalinos et al., 2014).

**Dopamine and addiction mechanisms**

Dopamine is critically important in reward and motivation. It is released as a reward in taking drugs, smoking, having sex, and eating food. In addicted persons the phasic dopamine release is heightened, compelling person the pursuit of abused substances. When a smoker stops smoking low dopamine levels occur as a result of withdrawal. This is the main reason for relapse due to unpleasant feelings of withdrawal (Wonnacott et al., 2005). Withdrawal symptoms are; Anhedonia, anxiety-related behavior, conditioned fear, irritability, anger, difficulty concentrating, sleep disturbance, increased appetite, weight gain and threat-induced anxiety (Hogle et al., 2010). As long as this abnormal dopamine conducting system in the brain continues the addiction and relapse risk will continue. Therefore the aim should be vane of nicotine addiction altogether. ECs seem to feed these addiction pathways to remain.

**Young people and EC**

Limbic system is matured before prefrontal cortex control systems. This renders developing youth brains more susceptible for addictions. Therefore it is dangerous to encourage young people to use an EC (Villanti et al., 2015). In fact Use of ECs is increasing rapidly (CDC, 2016). Even it has been reported that ECs are associated with more cigarette smoking (Rigotti, 2015). Youths are using EC when they cannot use cigarettes in social settings. They don't see it as an aid to stop smoking. People who do not smoke may be attracted to EC for its less harmful, socially more acceptable image. For a youth it may be a way to seek attention and simple out of curiosity. The appeal of flavored e-cigarettes to children and adolescents are alarming. Nicotine is highly addictive substance and once they are hooked they may easily switch to regular cigarette. When EC is accepted as a viable and healthier option for smoker's nicotine addiction may disseminate easier. It may have an image of socially acceptable, harmless activity. These images may act like magnets for youths.

There may be some benefits of ECs namely quitting or decreasing the number of cigarettes. Moreover no significant adverse effect is reported with EC use. However these claims are not proven and quality of evidences are poor. Arguments against ECs seem to have better rationale. ECs are running the dangers of luring young people into nicotine addiction. People simply may use EC and cigarettes at the same time. EC may feed addiction rather than stop it by providing dopamine peaks. Official view of most health authorities on EC negative. Until now there is not sufficent evidence to recommend EC as a first-line option in smoking cessation services.

**REFERENCES**

Boyce, J.H., Mc Robbie, H., Bullen, C., Begh, R., Stead, L.F., Hajek, P., 2016. Electronic cigarettes for smoking cessation. Cochrane database of systemic reviews. doi: 10.1002/14651858.CD010216.pub3.

CDC., 2016. Tobacco use among middle and high school students-United States, 2011-2015. Morbidity and mortality weekly report 2016. 65, 361-367. www.fda.gov/tobaccoproducts/labeling/productsingredientscomponents/ucm456610.htm. Accessed: 10.11.2016.

CDC., 2015. percent distribution of cigarette smoking status among adult current e-cigarette users, by age group, national health interview survey, United States, 2015. Morbidity and mortality weekly report 2016. 65, 1177. http://www.fda.gov/

TobaccoProducts/Labeling/ProductsIngredientsComponents/ucm456610.htm. Accessed: 01.11.2016.

Drugs.com., 2016. https://www.drugs.com/sfx/nicotine-side-effects.html. Accessed: 02.11.2016.

Etter, J.F., Bullen, C., 2014. A longitudinal study of electronic cigarette users. Addict Behav. 39; 491-494. doi: 10.1016/j.addbeh.2013.10.028.

Farsalinos, K.E., Kistler, K.A., Gillman, G., Voudris, V., 2014. Evaluation of electronic cigarette liquids and aerosol for the presence of selected inhalation toksins. Nicotine Tob. Res. 17, 168-174.

FDA., 2015. Vaporizers, e-cigarettes, and other electronic nicotine delivery systems (ENDS). http://www.fda.gov/TobaccoProducts/Labeling/ProductsIngredientsComponents/ucm456610.htm. Accessed: 01.11.2016.

Hadwiger, M.E., Trehy, M.L., Ye, W., Moore, T., Allgire, J., Westenberger, B.J., 2010. Identification of amino-tadalafil and rimonabant in electronic cigarette products using high pressure liquid chromatography with diode array and tandem mass spectrometric detection. J. Chromatogr. A. 1217, 7547-7555. doi: 10.1016/j.chroma.2010.10.018.

Hahn, J., Monakhova, B.M., Hengen, J., Himmelseher, M.K., Schüssler, J., Hahn, H., Kuballa, T., Lachenmeier, D.W., 2014. Electronic cigarettes: Overview of chemical composition and exposure estimation. Tob. Induc. Dis. 12, 23. doi: 10.1186/s12971-014-0023-6.

Hogle, J.M., Kaye, J.T., Curtin, J.J., 2010. Neuroadaptation in human addiction. Biol. Psychiatry. 2010. 68, 719-725.

Hutzler, C., Paschke, M., Kruschinski, S., Henkler, F., Hahn, J., Luch, A., 2014. Chemical hazards present in liquids and vapors of electronic cigarettes. Arch. Toxicol. 88, 1295-1308.

Kralikova, E., Novak, J., West, O., Kmetova, A., Hajek, P., 2013. Do e-cigarettes have the potential to compete with conventional cigarettes?: A survey of conventional cigarette smokers' experiences with e-cigarettes. Chest. 144, 1609-1614. doi: 10.1378/chest.12-2842.

Research and Market; 2016. Global e cigarette and vaporizer market 2015-2025-analyses and forecast for 50 billion industry. http://www.researchandmarkets.com/research/vq366f/global_e (Last updated at: 5th / 8/ 2015).

Rigotti, N.A., 2015. E-Cigarette use and subsequent tobacco use by adolescents: New evidence about a potential risk of e-cigarettes. JAMA. 314, 673-674.

Rose, J.E., Turner, J.E., Murugesan, T., Behm, F.M., Chris, J., Wynne, C.J., Murray Laugesen, M., 2010. Pulmonary delivery of nicotine pyruvate: Sensory and pharmacokinetic characteristics. Poster, Society For Research On Nicotine And Tobacco 16th Annual Conference. Baltimore, 27 February 2010.

Sanner, T., Grimsrud, T.K.,. 2015. Nicotine: Carcinogenicity and effects on response to cancer treatment-a review. Front Oncol. 5, 196. doi: 10.3389/fonc.2015.00196.

Schober ,W., Szendrei, K., Matzen, W., Fuchs, H.O., Heitmann, D., Schettgen, T., Jorres, R.A., Fromme, H., 2014. Use of electronic cigarettes (e-cigarettes) impairs indoor air quality and increases FeNO levels of e-cigarette consumers. Int. J. Hygiene Env. Health. 217, 628-637.

Surgeon General Report, 2014. The health consequences of smoking-50 years of progress. http://www.surgeongeneral.gov/library/reports/50-years-of-progress. Accessed 10.15.2016.

Sussan, T.E., Gajghate, S., Thimmulappa, R.K., Ma, J., Kim, J.H., Sudini, K., Consolini, N., Cormier, S.A., Lomnicki, S., Hasan, F., Pekosz, A., Shyam Biswal, S., 2015. Exposure to electronic cigarettes impairs pulmonary anti-bacterial and anti-viral defenses in a mouse model. Plos one. http://dx.doi.org/10.1371/journal.pone.0116861.

Villanti, A.C., Rath, J.M., Williams, V.F., Pearson, J.L., Richardson, A., Abrams, D.B., Niaura, R.S., Vallone, D.M., 2015. Impact of exposure to electronic cigarette advertising on susceptibility and trial of electronic cigarettes and cigarettes in US young adults: A randomized controlled trial. Nicotine Tob. Res. 18, 1331-1339.

Wonnacott, S., Sidhpura, N., Balfour, D.J., 2005. Nicotine: From molecular mechanisms to behavior. Curr. Opin. Pharmacol. 5, 53-59.

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http://dergipark.ulakbim.gov.tr/omujecm

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# Smoking and cardiovascular diseases

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**ABSTRACT**

Cardiovascular diseases are the foremost cause of morbidity and mortality in the world and in our country. What is favorable in terms of epidemic cardiovascular diseases is that they are substantially "preventable". Smoking is one of the leading preventable risk factors. Smoking causes development and progression of cardiovascular diseases through different pathophysiological processes such as endothelial dysfunction, development and progression of atherosclerosis, hemodynamic effects, inflammation, hypercoagulable state, and dyslipidemia. Cardiologists, who encounter the clinical consequences of smoking, play a central role in informing patients regarding the hazards of smoking, supporting them for smoking cessation, providing psychological and pharmacological treatments as well as cardiovascular effects of these treatments. Starting the fight against smoking, which seriously threatens public health, seems to be the most appropriate start regarding fight for the prevention and control of cardiovascular diseases.



## 1. Introduction

Cardiovascular diseases are the foremost cause of morbidity and mortality in the world and in our country. According to Turkish Statistical Institute and based on the main ICD 10 codes, diseases of the circulatory system were by far the leading cause of death with a rate of 40.3% in 2015 (TİK, 2015). Of the deaths resulting from circulatory system disorders, 40.5% were due to ischemic heart disease, 24.3% were due to cerebrovascular disease, 20.4% were due to other cardiac diseases and 9.7% were due to hypertensive diseases (TİK, 2015).

According to 2012 World Health Organization (WHO) data, 46.2% (17.5 million) of all deaths globally were the result of cardiovascular diseases. Among these, 7.4 million were associated with ischemic cardiac diseases while 6.7 million were due to stroke. Cardiovascular diseases are responsible for 37% of deaths below the age of 70 years. Deaths due to cardiovascular diseases are estimated to be 22.2 million in 2030, 8 million of which are related directly to smoking. Furthermore, cardiovascular diseases are estimated to remain the leading cause of death for a long period of time (http://www.sbu.saglik.gov.tr; www. apps.who.int/iris.com; www.who.int/healthinfo/globalburdendisease). However, what is favorable in terms of cardiovascular diseases is that they are substantially "preventable". WHO stated that the incidence of cardiovascular diseases may be reduced by half with the control of unfavorable behaviors such

as smoking, sedentary lifestyle, and unhealthy nutrition (http://www.sbu.saglik.gow.tr; www.apps.who.int/iris. com.; www.whqlibdoc.who.int/publications/2011).

In pathophysiology and development of cardiovascular diseases, smoking is the leading factor among preventable risk factors. In risk calculation systems for cardiovascular diseases and mortality, particularly in the most frequently used SCORE system, the risk doubles in smokers (http://www.sbu.saglik.gov. tr). Nearly half of regular smokers die due to diseases associated with smoking. Life expectancy decreases by 10 years on average in smokers compared to non-smokers (Rigotti and Clair, 2013). Twenty-nine percent of deaths associated with cardiovascular diseases are attributed directly to smoking (Piepoli et al., 2016). According to 2012 data, 1.1 billion individuals are known to be smokers worldwide (www. apps.who.int/ iris.com). Six million individuals die due to tobacco use or passive smoking each year (www. apps.who.int/ iris.com).

According to the results of a 2012 survey on global tobacco use among adults in Turkey, 14.8 million individuals (27.1%) were found to be users of tobacco products in 2012. Frequency of tobacco use is 41.5% in men, and 13.1% in women. Among all tobacco users, 23.8% (37.3% in men and 10.7% in women) use tobacco every day. Age of onset for smokers who smoke everyday is 17.1 years. Of all users of tobacco products, 94.8% use manufactured cigarettes and 1% use hookah (http://www.sbu.saglik.gov.tr; KYTA, 2012). Current data may provide an explanation why all kinds of cigarettes must be avoided owing to the destructive effects of smoking and the incidence of cardiovascular disease.

Cardiovascular diseases such as coronary heart disease (CHD) [including myocardial infarction (MI) and sudden death], cerebrovascular disease (stroke), peripheral artery disease (PAD) and abdominal aortic aneurysm, and hypertension are identified to be directly related to smoking (Rigotti and Clair, 2013). Development of heart failure increases by two-fold in smokers compared to non-smokers. Prognosis is even worse in smoking patients with heart failure (Rigotti and Clair, 2013). Additionally, smoking may trigger cardiac arrhythmia or increase existing arrhythmia (D'Alessandro et al., 2012).

**Tobacco and smoking**

Tobacco was discovered in America and Australia followed by a rapid spread across the world. In Turkey, tobacco use and cultivation was started in 1600s (Yetkin, 1992). More than 90% of tobacco is used in manufacturing cigarettes; and only small amounts are used for medicinal purposes in veterinary medicine, agriculture and medicine (Yetkin, 1992). After the discovery of cigarette rolling machine in 1881, it became cheaper and resulted in a more widespread use. Tobacco was provided to American soldiers free of charge during World War I and II. Precautions against smoking because of increasing cardiovascular diseases were initiated between 1940 and 1960 (http://www. healthliteracy.worlded. org/docs/tobacco). Cigarettes contain more than 4000 cytotoxic, carcinogenic and mutagenic chemical substances, particularly nicotine, carbon monoxide (CO) and oxidant chemical gases (Behr and Nowak, 2002). There are several substances such as nitrosamines, polycyclic hydrocarbons and inorganic compounds, which enter the body in the form of particles with cigarette smoke. With each cigarette consumed, 2-3 mg nicotine and 20-30 ml CO enter the body. One g of tar of cigarette smoke contains 1018 oxygen radicals while the gas phase of the smoke contains 1015 oxygen radicals (Behr and Nowak, 2002). Free oxygen radicals result in DNA mutations with oxidative stress, progression of atherosclerosis, and chronic inflammation. These chemical substances cause endothelial dysfunction, insulin resistance, dyslipidemia, inflammation, hemodynamic changes, hypercoagulable state and atherothrombosis associated with smoking (Salahuddin et al., 2012).

**Pathophysiological changes induced by smoking**

Nicotine, CO and oxidant chemical substances which generate free oxygen radicals are involved in the pathophysiology of cardiovascular diseases resulting from smoking. These factors increase atherothrombosis and its progression through several mechanisms (Salahuddin et al., 2012; Fig. 1).

**Endothelial dysfunction and Atherosclerosis**

Nicotine causes structural changes in vascular endothelium. It stimulates DNA synthesis in vascular endothelial cells and causes vascular proliferation (Benowitz, 2003). Free oxygen radicals increase in endothelium via the oxidant toxic molecules in cigarette. The increase in cytokines as well as increased inflammation, lipid oxidation, platelet activation and vasospasm due to increased endothelium are observed alongside the decrease in production and levels of endothelial vasodilator nitric oxide (NO). Endothelial damage plays a significant role in the development and acceleration of atherosclerosis with the migration of lymphocytes, macrophages, monocytes and smooth muscle cells into the intima as well as increased chemotaxis molecules and thrombocyte adhesion leading to accumulation of oxidized LDL in intima and foam cell formation (Smith and Fischer, 2001; Benowitz, 2003; Salahuddin et al., 2012).

**Hemodynamic effects of smoking**

Nicotine increases the heart rate, blood pressure, cardiac output, myocardial oxygen demand and

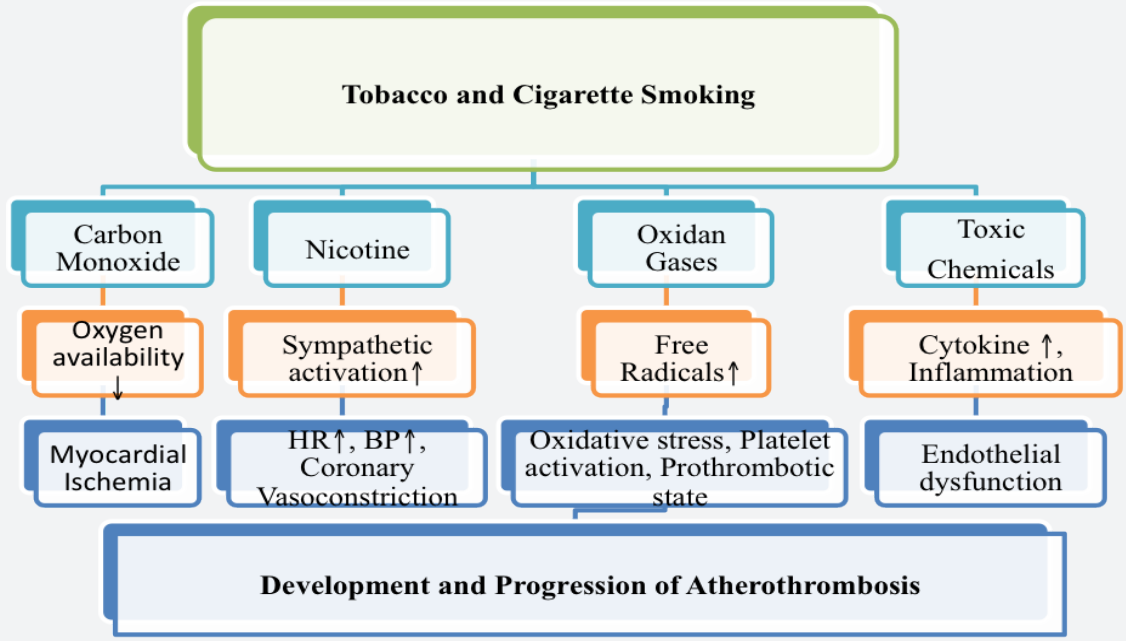


**Fig. 1.** Pathophysiological effect of cigarette smoking on cardiovascular disease. **HR:** Heart rate; **BP:** Blood pressure; **NO:** Nitrogen monoxide. Figure reprinted with permission from Salahuddin, S., Prabhakaran, D., Roy, A. Pathophysiological mechanisms of tobacco-related CVD. Global Heart. 2012. 7, 113-119

consumption via sympathetic activation. It causes coronary vasoconstriction. CO decreases oxygen transfer to all tissues, particularly to myocardium by binding proteins such as hemoglobin, myoglobin, and cytochrome oxidase (Benowitz, 2003; Salahuddin et al., 2012). For this reason, diastolic and systolic myocardial functions are affected by chronic smoking and are shown in clinical researchs. (Gulel et al., 2007).

**Inflammation**

Smoking causes chronic inflammation, which is also the basis of atherosclerosis. Smoking increases proinflammatory cytokines and adhesion molecules such as interleukin-6, C-reactive protein (CRP), tumor necrosis factor alpha (TNF-a), soluble vascular cell adhesion molecule 1, intracellular adhesion molecule 1 and E-selectin (Mazzone et al., 2001; Bermudez et al., 2002; Salahuddin et al., 2012).

**Hypercoagulable state**

Smoking creates a predisposition to thrombosis with the increase in thrombocyte activation, adhesion, and aggregation. It leads to a prothrombotic state by increasing plasma viscosity, coagulation factors and serum fibrinogen levels while decreasing the levels of fibrinolytic molecules such as tissue plasminogen activators. The thrombosis/fibrinolysis balance on endothelium shifts in favor of thrombosis (Benowitz, 2003; Salahuddin et al., 2012).

**Lipid abnormalities**

Serum cholesterol, triglyceride, very low-density lipoprotein cholesterol (VLDL-C), low-density lipoprotein cholesterol (LDL-C) levels are high in smokers while serum high-density lipoprotein cholesterol (HDL-C) and apolipoprotein A-1 levels tend to be lower. Again, endothelial permeability and subendothelial accumulation increase with the increase of oxidized LDL-C (Craig et al., 1989; Salahuddin et al., 2012).

**Smoking and insulin-resistance**

Smoking is thought to cause insulin resistance by various mechanisms such as the increase in corticosteroids, growth hormone, endothelial dysfunction and oxidative stress by means of sympathetic nervous system activation. Smoking increases visceral fat. Also, waist/hip ratio is detected to be higher in smokers (Tahtinen et al., 1998; Canoy et al., 2005; Salahuddin et al., 2012).

**Passive smoking and cardiovascular effects**

It is called passive smoking when non-smokers inhale the smoke in smoking zones. The CO in cigarette smoke, nicotine, nitrosamines, heavy metals, polycyclic hydrocarbons are involved in the development of cardiovascular diseases. Passive smoking is known to create risks for cardiovascular diseases at least as much as smokers (Rigotti and Clair, 2013).

**Water-pipe smoking and cardiovascular effects**

The waterpipe tobacco smoking, also known as narghile, shisha, hookah, goza, and hubble bubble, has become prevalent in the world, especially among youth (Shihadeh et al., 2015). Waterpipe users are exposed to significant levels of carbon monoxide, nicotine, nitrosamines, carcinogenic aromatic hydrocarbons and volatile aldehydes over the duration of the smoking session, despite a common opion that waterpipe smoking is less harmful than cigarette smoking (Neergaard et al., 2007; Shihadeh et al., 2015). So waterpipe smoking has create risks for cardiovascular diseases at least as much as smokers. Using waterpipe to smoke is not a safe alternative to cigarette smoking and less harmful (Neergaard et al., 2007).

**Electronic cigarettes and cardiovascular effects**

Complete smoking cessation is the best outcome for smokers, but the powerful addictive effects of nicotine and the ritualistic behavior of smoking create a huge obstacle. Electronic cigarettes are devices that can vaporize a nicotine solution combined with liquid flavors and as marketed to help smoking cessation that require further investigation for advers effects (Nelluri et al., 2016). But their benefits in smoking cessation still have not been proven by adequate scientific evidence, also, they uphold nicotine addiction and may increase the risk of starting conventional cigarette. Electronic cigarettes have sympathomimetic cardiovascular effects related to nicotine exposure, also contain other chemicals that require further investigation for advers effects (Farsalinos and Polosa, 2014; Nelluri et al., 2016).

**Smoking cessation treatment and cardiovascular effects**

Smoking cessation must be recommended to all smokers regardless of period and amount of smoking, age, and existence of diseases (Rigotti and Clair, 2013). Quitting smoking before the age of 40 years may reduce deaths associated with smoking by 90% (Jha et al., 2013). In a survey conducted in USA, 70% of adult smokers stated that they wanted to quit smoking and more than half of these individuals reported trying to quit; however, only 6% of them were successful (Jamal et al., 2012). The major hindrance for smoking cessation is related to the addictive effect of nicotine. Cardiovascular symptoms may also be clinically apparent as well as irritability, anger, restlessness, anxiety, depressed mood, difficulty concentrating, insomnia, and increased hunger as nicotine withdrawal symptoms (Rigotti and Clair, 2013). Pharmacological treatments and psychosocial treatments are used for smoking cessation treatment. NRT (nicotine replacement therapy) such as patch, gum, and spray as well as varenicline and bupropion are the main pharmacological agents used for smoking cessation (Rigotti and Clair, 2013).

Nicotine replacement therapy (NRT) reduces nicotine withdrawal symptoms. Different results have been obtained with its use in patients with cardiovascular diseases. Nicotine patches are reported to be not as thrombogenic as cigarettes and they also do not contain CO (Rigotti and Clair, 2013). They are reported to be safely used in patients with stable cardiovascular disease, and may also be used by taking into consideration the benefit/risk ratio in patients who experienced myocardial infarction, unstable angina or and ventricular arrhythmia in the last 2 weeks (Rigotti and Clair, 2013).

Varenicline is the partial agonist of the alpha4-beta2 nicotine receptor. While it reduces nicotine withdrawal symptoms by binding nicotine receptors, it also blocks nicotine from binding to receptors again. It is reported to be safely used in patients with cardiovascular disease (Rigotti and Clair, 2013).

Bupropion is an atypical antidepressant which exerts its effects by increasing the levels of dopamine and norepinephrine. It is reported to be safely used in stable CVD. Bupropion is metabolized with the cytochrome P450 2B6 enzyme; and caution should be exercised with this agent as it may result in increased drug levels when used with medications metabolized through the same pathway, especially clopidogrel (Rigotti and Clair, 2013).

**Conclusions and recommendations: Cardiologist's role**

Smoking is involved in the development and progression of cardiovascular diseases through various pathophysiological mechanisms and is also the leading preventable risk factor. Cigarettes are addictive and have serious effects on individual and public economy due to treatment of resulting diseases, smoking costs, early deaths as well as the costs of behavioral and pharmacological treatments used for smoking cessation. Cardiovascular effects may be reversed by preventing cigarette use, and by smoking cessation among smokers. Cardiologists, who encounter the clinical consequences of smoking, must have a leading role in informing patients about the harms of smoking at every stage including medical and pharmacological support for smoking cessation. Starting the fight against smoking, which seriously threatens public health, seems to be the most appropriate start regarding fight for the prevention and control of cardiovascular diseases.

**REFERENCES**


Behr, J., Nowak, D., 2002. Tobacco smoke and respiratory disease. ERS Journals Ltd. European Respiratory Monograph. 161-179.

Benowitz, N.L., 2003. Cigarette smoking and cardiovascular disease: pathophysiology and implications for treatment. Prog. Cardiovasc. Dis. 46, 91-111. doi: http://dx.doi.org/10.1016/S0033-0620(03)00087-2.

Bermudez, E.A., Rifai, N., Buring, J.E., Manson, J.E., Ridker, P.M., 2002. Relation between markers of systemic vascular inflammation and smoking in women. Am. J. Cardiol. 89, 1117-1119. doi: http://dx.doi.org/10.1016/S0002-9149(02)02284-1.

Canoy, D., Wareham, N., Luben, R., Welch, A., Bingham, S., Day, N., Khaw, K.T., 2005. Cigarette smoking and fat distribution in 21, 828 British men and women: A population-based study. Obes. Res. 13, 1466-1475. doi:10.1038/oby.2005.177.

Craig, W.Y., Palomaki, G.E., Haddow, J.E., 1989. Cigarette smoking and serum lipid and lipoprotein concentrations: An analysis of published data. BMJ. 298, 784-788.

D'Alessandro, A., Boeckelmann, I., Hammwhoner, M., Goette, A., 2012. Nicotine, cigarette smoking and cardiac arrhythmia: An overview. Eur. J. Prev. Cardiol. 19, 297-305. doi: 10.1177/1741826711411738.

Erhardt, L., 2009. Cigarette smoking: an undertreated risk factor for cardiovascular disease. Atherosclerosis. 205, 23-32.

Farsalinos, K.E., Polosa, R., 2014. Safety evaluation and risk assessment of electronic cigarettes as tobacco cigarette substitutes: A systematic review. Ther. Adv. Drug. Saf. 5, 67-86. doi: 10.1177/2042098614524430.

Gulel, O., Soylu, K., Yazici, M., et al., 2007. Longitudinal diastolic myocardial functions are affected by chronic smoking in young healthy people: A study of color tissue doppler imaging. Echocardiography. 24, 494-498. doi:10.1111/j.15408175.2007.00421.

Http://www. apps.who.int/iris/bitstream/10665/148114/1/9789241564854_eng. WHO, Global Status Report on Noncommunicable Diseases 2014.

Http://www.healthliteracy.worlded. org/docs/tobacco/Unit1/2history_of.html. From the First to the Last Ash: The History, Economics, and Hazards of Tobacco [online curriculum].

Http://www.sbu.saglik.gov.tr/Ekutuphane/kitaplar/t14.pdf. Türkiye Kalp ve Damar Hastalıklarını Önleme ve Kontrol Programı 2015-2020, Basım 2015 Ankara.

Http://www.whqlibdoc.who.int/publications/2011/9789241564373_eng. World Health Organization, Global Atlas on Cardiovascular Disease Prevention and Control 2011, World Heart Federation and World Stroke Organization. (Erişim tarihi: 21.12.2016)

Http://www.whqlibdoc. who.int/publications/2012/9789241564434_eng.pdf. WHO Global Report: Mortality Attributable to Tobacco. World Health Organisation, 2012.

Http://www.who.int/healthinfo/globalburdendisease. WHO, Global Health Risks: Mortality and burden of disease attributable to selected major risks. World Health Organization, 2009.

Jamal, A., Dube, S.R., Malarcher, A.M., Shaw, L., Engstrom, M.C., 2012. Tobacco use screening and counseling during physician office visits among adults-National Ambulatory Medical Care Survey and National Health Interview Survey, United States, 2005-2009. MMWR Morb. Mortal Wkly. Rep. 61, 38-45.

Jha, P., Ramasundarahettige, C., Landsman, V., Rostron, B., Thun, M., Anderson, R.N., McAfee, T., Peto, R., 2013. 21st century hazards of smoking and benefits of cessation in the United States. N. Engl. J. Med. 368, 341-350. doi: 10.1056/NEJMsa1211128.

Mazzone, A., Cusa, C., Mazzucchelli, I., Vezzoli, M., Ottini, E., Ghio, S., Tossini, G., Pacifici, R., Zuccaro, P., 2001. Cigarette smoking and hypertension influence nitric oxide release and plasma levels of adhesion molecules. Clin. Chem. Lab. Med. 39: 822-826. doi:10.1515/CCLM.2001.136.

Neergaard, J., Singh, P., Job, J., et al., 2007. Waterpipe smoking and nicotine exposure: A review of the current evidence. Nicotine Tob. Res. 9, 987-994. doi:10.1080/14622200701591591.

Nelluri, B., Murphy, K., Mookadam, F., et al., 2016. The current literature regarding the cardiovascular effects of electronic cigarettes. Future Cardiol. 12, 167-179. doi:10.2217/fca.15.83.

Piepoli, M.F., Hoes, A.W., Agewall, S., et al. 2016. European Guidelines on cardiovascular disease prevention in clinical practice: The sixth joint task force of the European society of cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of 10 societies and by invited experts) Developed with the special contribution of the European Association for cardiovascular prevention&rehabilitation (EACPR). Atherosclerosis. 252, 207-274. doi:10.1016/j.atherosclerosis.2016.05.037.

Rigotti, N.A., Clair, C., 2013. Managing tobacco use: The neglected cardiovascular disease risk factor. Eur. Heart. J. 34, 3259-3267. doi: 10.1093/eurheartj/eht352.

Salahuddin, S., Prabhakaran, D., Roy, A., 2012. Pathophysiological mechanisms of tobacco-related CVD. Glob. Heart. 7, 113-120. doi:10.1016/j.gheart.2012.05.003.

Shihadeh, A., Schubert, J., Klaiany, J., et al. 2015. Toxicant content, physical properties and biological activity of waterpipe tobacco smoke and its tobacco-free alternatives. Tob. Control. 24, i22-i30. doi: 10.1136/tobaccocontrol-2014-051907.

Smith, C.J., Fischer, T.H., 2001. Particulate and vapor phase constituents of cigarette mainstream smoke and risk of myocardial infarction. Atherosclerosis. 158, 257-267. doi: http://dx.doi.org/10.1016/S0021-9150(01)00570-6.

Tahtinen, T.M., Vanhala, M.J., Oikarinen, J.A., Keinanen-Kiukaanniema, S.M., 1998. Effect of smoking on the prevalence of insulin resistance-associated cardiovascular risk factors among Finnish men in military service. J. Cardiovasc. Risk. 5, 319-323.

T.C. Sağlık Bakanlığı, Küresel Yetişkin Tütün Araştırması (KYTA)-2012, Yönetici Özeti: Türkiye.

Türkiye İstatistik Kurumu, Ölüm Nedeni İstatistikleri, 2015 http://www.tuik.gov.tr/Haber Bulteni. doi=21526.

Yetkin, Y., 1992. Fizyolojik çevrenin korunması ve sigara içimi ile tütün üretimi ve biyoloji eğitimi arasındaki ilişki. SBAD. 3, 99-110.

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# Smoking status of pregnant women in Elazig, an eastern province of Turkey

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**ABSTRACT**

This study was conducted to determine the smoking situation of pregnant women in Elazig. Pregnant women registered at 173 family medicine centers in all the districts of Elazig comprise this descriptive research. 12.3% (n=77) of pregnant women had smoked regularly before pregnancy, 84.6% (n=531) of them stated that they never smoked and, 3.2%(n=20) had quit smoking before pregnancy. 87.7% (n=551) of pregnant women stated that they did not smoke at all during pregnancy, 4.2% (n=26) continued smoking during pregnancy, 6.7% (n=42) quit smoking as soon as they learned of their pregnancy, and 1.4% (n=9) smoking for a while and then quit smoking during pregnancy. 72.5% (n=37) of pregnant women who quit smoking, did so by themselves without any advice, 21.6% (n=11) by nurse advice and 5.9% (n=3) by doctor advice. 35.0% of pregnant women stated that they were exposed to second-hand smoking during pregnancy. The rate of smoking during pregnancy increased with decreasing income ($p<0.05$). The rate of smoking in pregnant women is low. The rate of pregnant women taking advice from health personnel about smoking cessation is low. The risk of smoking during pregnancy should be dwelt on by health personnel and relevant education should be given to pregnant women.



## 1. Introduction

It was reported that the smoking rate among men is 37.3% and women is 10.7% in our country (Turkish Statistical Institute Health Survey, 2013). In our country, 110.000 people die due to tobacco related diseases each year (Doğanay et al., 2012). Tobacco is the most common addictive substance. Unlike other addictive substances, cigarette smoke may affect other living things in the environment. There are toxic substances in cigarette smoke that threaten the health of individuals who occupy the same environment with the smoker such as polycyclic aromatic hydrocarbons, aromatic amines, nitrosamines, heavy metals, pesticide residues, and radioactive elements. The fetus being subject to detrimental substances originating from the mother using tobacco by means of the placenta is a special example of this threat (Toyran, 2005).

Smoking is a very common addiction in society. 90% of smokers start smoking before the age of 20 and while the number of male smokers is declining the number of female smokers is increasing (Ozmen, 2004). Smoking during pregnancy is a very serious health problem in our country and in many other countries as well. In a study into smoking during pregnancy in the USA in 2002, 11.4% of pregnant women were determined to smoke. Smoking during pregnancy requires several measures due to both preventing the growth of healthier generations and leading to maternal and infant

problems (Ozmen, 2003; Marakoglu and Sezer, 2003; Dawn and Nan, 2005)

There are many studies about increasing the amount of daily tobacco use during pregnancy being in direct proportion to increases in the delivery of low birth weight infants, preterm delivery and risk of abortion (Cook and Strachan, 1999; Agrawai, 2010). Yeltekin et al. (2005) declared that exposure to cigarette smoke during pregnancy had an adverse effect on birth weight. This study was conducted to determine the smoking situation of pregnant women in Elazig.

## 2. Material and methods

### 2.1. Design

This cross-sectional survey study was done between March and April 2015. The research population included pregnant women registered at 173 family medicine centres in all the districts of Elazig who had routine check-ups between the dates determined for the conduct of this research. The survey was offered to 692 pregnant women but it was only administered to 628 since participation was voluntary. The response rate was 90.7%. The surveys were administered in face-to-face interviews by trained pollsters. The survey included questions about socio demographic information and the pregnant women's opinions about childbearing and childbirth methods as well as smoking. The comprehensibility of the questionnaire was verified by a pilot test with 15 pregnant women who were not included in the research and was revised accordingly.

### 2.2. Statistical analysis

The data obtained from the research were recorded to SPSS package program and checked for errors. Tables and statistical analyses were analysed using this program. The statistical analysis used percentages, means and chi-square tests depending on the quality of the variables. Means include their standard deviations and $p<0.05$ was used as the threshold of significance.

### 2.3. Ethics

The T.C. Public Health Institution had approved ethical permission for this study.

## 3. Results

The mean age of pregnant participants was 28.82±5.43 (min=17, max=45) years, the mean marriage age of participants was 22.29±0.165 (min=14, max=39) years and average parity was 1.11±1.08 (min=0, max=6) delivery. 55.6% of pregnant women had elementary education, 91.1% were housewives, 67.2% had 1500 TL or lower income, and 76.8% resided in a city centre. The feature of the socio demographic characteristics of the participants are presented at Table 1. The smoking status or exposure to smoke of the particiipants before and after their pregnancy is presented at Table 2. 72.5% (n=37) of pregnant women who quit smoking, did so by themselves without any advice, 21.6% (n=11) by nurse advice and 5.9% (n=3) by doctor advice. When smoking cessation reasons were examined, 96.0% (n=603) of pregnant women replied "I thought smoking would be harmful for my baby", 0.4% (n=25) "because of nausea and vomiting". Moreover, 80.4% (n=505) of pregnant women who quit smoking during pregnancy didn't intend to resume smoking, 19.6% (n=123) intended to resume smoking after pregnancy. The comparison of the socio demographic and obstetric features of the smoker and non-smoker participants are presented at Table 3. The mean number of cigarettes smoked daily by pregnant women who smoked during pregnancy was 6.58±5.16 (min=1, max=20) pcs. When educational background was examined, while the proportion of smoking among illiterate pregnant women was 2.3% (n=1), this proportion was 4.3% (n=25) among pregnant women who had a primary-school degree or above ($p>0.05$). The rate of smoking during pregnancy increased with decreasing income ($p<0.05$). Moreover, when the smoking rates were compared in accordance with age, while the proportion of smoking during pregnancy was 5.6% (n=11) among those 25 younger, this proportion was 3.5% (n=15)

**Table 1.** Features of the socio demographic characteristics of the pregnant women

| Socio demographic characteristics | n | % |
|---|---|---|
| **Age of pregnant women** | | |
| 17-26 | 233 | 37.1 |
| 27-31 | 341 | 54.3 |
| 37 and over | 54 | 8.6 |
| **Educational levels of pregnant women's** | | |
| Illiterate | 44 | 7.0 |
| Primary school graduate | 349 | 55.6 |
| High school and above | 235 | 37.4 |
| **The educational levels of pregnant women's husbands** | | |
| Illiterate | 11 | 1.8 |
| Primary school graduate | 266 | 42.4 |
| High school and above | 351 | 55.9 |
| **Pregnant women's jobs*** | | |
| Housewife | 536 | 91.1 |
| State official | 73 | 11.6 |
| Self-employed | 19 | 3.0 |
| **Income levels of pregnant women**** | | |
| 1500 TL and lower | 422 | 67.2 |
| 1501-3000 TL | 131 | 20.9 |
| 3001 TL and higher | 75 | 11.9 |
| **Marriage age** | | |
| 18≥ | 121 | 19.2 |
| 19-34 | 499 | 79.5 |
| 35≤ | 8 | 1.3 |

* Tailors and hairdressers are considered to be self-employed
** 1 $=2.8 TL (Turkish Lira)

**Table 2.** The features of smoking status and exposure to smoke of the participants

| | n | % |
|---|---|---|
| **Smoking status** | | |
| **Smoking before pregnancy** | | |
| Smoked daily | 77 | 12.3 |
| Quit before pregnancy | 20 | 3.2 |
| Never smoked | 531 | 84.6 |
| **Smoking during pregnancy** | | |
| Smoked during pregnancy | 26 | 4.1 |
| Quit during pregnancy | 42 | 6.7 |
| Kept up smoking for a while and then quit smoking during pregnancy | 9 | 1.4 |
| Never smoked | 551 | 87.7 |
| **Status of intending to resume smoking after pregnancy*** | | |
| Smoked during pregnancy and intended to keep up smoking | 26 | 4.1 |
| Intended to resume smoking after pregnancy | 42 | 6.7 |
| Not intended to resume smoking after pregnancy | 9 | 1.4 |
| Never smoked | 551 | 87.7 |
| **Status of exposure to second-hand smoking** | | |
| Exposed | 220 | 35.0 |
| Not exposed | 408 | 65.0 |
| **Exposure to second-hand smoking through whom (n=220)** | | |
| Husbands | 194 | 30.9 |
| Relatives | 25 | 4.0 |
| House guests | 1 | 0.1 |

*Only pregnant women who quit smoking during pregnancy

among those aged 26 or older (p>0.05). 2.6% (n=5) of pregnant women who experienced their first pregnancy smoked during pregnancy, while 4.8% (n=21) of pregnant women who experienced their second or more pregnancies smoked during pregnancy (p>0.05). 4.2% (n=26) of pregnant women who went for a check-up regularly during pregnancy smoked during pregnancy, while pregnant women who did not go for a check-up did not smoke at all during their pregnancy (p>0.05). All of the participants answered the harms of smoking on health. Their answers was classified at Table 4.

## 4. Discussion

Overall 250 million women smoke tobacco all over the world. Prevalence of smoking among women in developed countries is 22% and 9% in developing countries. Evidence indicates that women find it harder to quit smoking than men. Tobacco companies target women (Mackay and Ericsen, 2002; Mackay and Amos, 2003). 12.3% of pregnant women who attended our study were determined to smoke regularly before pregnancy. In the Global Adult Tobacco Survey that was conducted in our country in 2012, it was determined that smoking prevalence among women was 13.1% (Turkish Statistical Institute, 2014). In a report prepared by WHO in (2014) the proportion of women aged 15 and over who smoked regularly every day was 13%.The Turkish Statistical Institute stated that the proportion of every day smoking among women aged 15 and over was 10.7% (Turkish Statistical Institute Health Survey, 2013). These results correspond with our study. The prevalence of smoking among pregnant women was 4.1% in our study. The prevalence of smoking among pregnant women was 3.0% in a similar study conducted in Afyonkarahisar, 6.2% in Edirne and 3.0% in Erzurum (Semiz et al., 2006; Kılıçarslan, 2008; Taşkıran, 2009). Of pregnant women was found that the rate of smoking cessation by physician or nurse advice and assistance was low. In the study of Marakoglu and Sezer (2003), they stated that the rate of smoking cessation through the advice and assistance of nurses or doctors was low among pregnant smokers. In a study conducted in Istanbul in 2013 it was determined that the rate of smoking cessation by physician or nurse advice and assistance was low (Aydin and Ergul, 2015). This case shows us that routine check-ups of pregnant women had not been done regularly and properly or decent questioning and enlightenment about smoking tobacco were not conducted during routine check-ups. In our study, 96.0% of pregnant women stated that they had quit smoking with the thought that smoking would be harmful to the baby and 0.4% of pregnant women stated that they quit smoking because of nausea and vomiting. Another study determined that 70.4% of pregnant women quit smoking with the thought that smoking would be harmful to the baby, 22.2% quit because of nausea and aversion and 7.4% quit because of both reasons (Marakoglu and Duygu, 2007). A study performed by Kocak et al. (2015) determined that the rate of pregnant women who quit smoking with the thought that smoking would be harmful to the baby was 77.6%, and the rate of pregnant women who quit smoking both with the thought that smoking would be harmful to the baby and because of nausea was 8.7%. Both of these studies mentioned above correspond with our study in terms of the high rate of quitting smoking with the thought that smoking would be harmful to the baby. The proportion of exposure to second-hand smoke among pregnant women who attended the study was 35.0%. In the study of Demirkaya, it was proved that 26.0% of pregnant women were passive smokers in 2004 (Özmen, 2004). In the study conducted in Brazil the proportion of passive smoking during pregnancy was determined as 35.9% by Nakamura et al. (2004). These findings are similar to ours. There are many factors that affect the growth and development of the fetus, and among these smoking and exposure to second-hand smoke are crucial in terms of their prevalence and avoidability (Di Franza and Lew, 1995; Andres and Day, 2000). There are numerous studies stating that pregnancy complications increase with smoking tobacco. These complications can be sorted

**Table 3.** Smoking status during pregnancy according to sociodemographic characteristics

| | Smoker | | Non-smoker | | Total | | Test |
|---|---|---|---|---|---|---|---|
| **Smoking status during pregnancy** | **n** | **%** | **n** | **%** | **n** | **%** | |
| **Age** | | | | | | | |
| 25> | 11 | 5.6 | 184 | 94.4 | 195 | 31.1 | $\chi^2$=1.60 |
| 26 and over | 15 | 3.5 | 418 | 96.5 | 433 | 68.9 | p=0.205 |
| **Place of residence** | | | | | | | |
| City center | 20 | 4.1 | 462 | 95.5 | 482 | 76.8 | $\chi^2$=0.01 |
| Rural region | 6 | 4.1 | 140 | 95.5 | 146 | 23.2 | p=0.983 |
| **Education status** | | | | | | | |
| Illiterate | 1 | 2.3 | 43 | 97.7 | 44 | 7.1 | $\chi^2$=0.41 |
| Primary education and above | 25 | 4.3 | 559 | 95.7 | 584 | 92.9 | p=0.812 |
| **Income groups** | | | | | | | |
| 1500 TL and below | 24 | 5.7 | 398 | 94.3 | 422 | 67.2 | $\chi^2$=8.03 |
| 1501-3000 TL | 2 | 1.5 | 129 | 98.5 | 131 | 20.9 | p=0.018 |
| 3001 TL and above | 0 | 0.0 | 75 | 100 | 75 | 11.9 | |
| **Profession** | | | | | | | |
| Housewife | 22 | 4.1 | 515 | 95.9 | 537 | 85.5 | $\chi^2$=0.01 |
| Employed | 4 | 4.4 | 87 | 95.6 | 91 | 14.5 | p=0.895 |
| **Number of pregnancy** | | | | | | | |
| 1 | 5 | 2.6 | 186 | 97.4 | 191 | 30.4 | $\chi^2$=1.60 |
| 2 and above | 21 | 4.8 | 416 | 95.2 | 437 | 69.6 | p=0.206 |
| **Status of abortion** | | | | | | | |
| Yes | 5 | 3.7 | 129 | 96.3 | 134 | 21.3 | $\chi^2$=0.07 |
| No | 21 | 4.3 | 473 | 95.7 | 494 | 78.7 | p=0.789 |
| **Status of going to regular check-up** | | | | | | | |
| Yes | 26 | 4.2 | 591 | 95.8 | 617 | 98.2 | $\chi^2$=0.48 |
| No | 0 | 0 | 11 | 100 | 11 | 1.8 | p=0.522 |
| **Stillbirth in previous pregnancies** | | | | | | | |
| Yes | 2 | 8.0 | 23 | 92.0 | 25 | 4.0 | $\chi^2$=0.97 |
| No | 24 | 4.0 | 579 | 96.0 | 603 | 96.0 | p=0.323 |
| **Paying attention to sleeping pattern** | | | | | | | |
| Yes | 11 | 2.6 | 407 | 97.4 | 418 | 66.6 | $\chi^2$=7.16 |
| No | 15 | 7.1 | 195 | 02.9 | 210 | 33.4 | p=0.007 |

as increased risk of abortion, prenatal growth failure, premature rupture of membranes, premature birth, stillbirth, placenta previa and decollement placenta (Olsen, 1992; Ananth et al., 1996; Mathews, 2004). In addition to this, there are studies indicating that infants whose mothers were exposed to environmental tobacco smoke, although they did not smoke themselves, have similar symptoms to infants whose mothers smoked tobacco (Olsen, 1992; Goel et al., 2004). Side-stream smoke that is inhaled from second-hand smokers contains all the same carcinogens that smokers inhale directly and because it is not filtered it contains one hundred times the amount of carcinogens of main-stream smoke. Consequently, although they do not smoke, pregnant women who are exposed to environmental tobacco smoke and their infants are affected by tobacco in the same way as pregnant women who smoke tobacco and their infants (Windham et al., 2000; Hofhuis et al., 2002).

When some traits of pregnant women were compared according to smoking status, there was no significant correlation between smoking status and age, place of residence, education level, or working status. There was a significant difference between smoking status and income levels, and sleeping patterns. Smoking rate increased with increasing education level but correlation between them was not statistically significant. In a study conducted in Sivas, the smoking rate was found to be highly statistically significant in pregnant women who had high education levels (Marakoglu and Sezer, 2003). When income and rate of smoking in pregnancy were compared, smoking rate was found to be highly statistically significant in pregnant women who had lower income. There are studies that correspond with our study and indicate that smoking rate increases with decreasing income level in the literature (Lantz et al., 1998; Karatay and Kubilay, 2004).

Consequently, it was determined that there were mothers who had smoked before pregnancy and also smoked and had been exposed to second-hand smoking during pregnancy, who had some knowledge about the

**Table 4.** Answers to the question "what is the harm of smoking on health?"

| | n | % |
|---|---|---|
| Lung problems | 274 | 43.7 |
| Mental and growth deficiency | 27 | 4.3 |
| Low birth weight infant | 24 | 3.8 |
| Threatened abortion | 15 | 2.3 |
| Harmful for mother | 12 | 1.9 |
| Treatment of premature birth | 18 | 2.8 |

harmful effects of smoking on the health of both the baby and mother and in spite of this knowledge smoking was kept up during pregnancy. It was also determined that there were pregnant women who quit smoking during pregnancy but intended to resume smoking after pregnancy and they were unable to get proper support or advice from doctors or nurses for smoking cessation. It is obvious that not to start smoking is the most efficient method. The gestation period is an important opportunity for women who are addicted to smoking to quit. Thus, in-service training programs should be prepared for health personnel, and more importance must be placed on relaying information during routine check-ups about the subject and being supportive about smoking cessation to increase the rate of smoking cessation of addictive pregnant women. It should not be forgotten that such efforts will reduce perinatal mortality and morbidity and contribute to the creation of healthier society in both physical and psychological terms.

## REFERENCES


Agrawal, A., Scherrer, J.F., Grant, J.D., Sartor, C.E., Pergadia, M.L., Duncan, A.E., Madden, P.A., Haber, J.R., Jacob, T.,Bucholz, K.K., Xian, H., 2010. The effects of maternal smoking during pregnancy on offspring outcomes. Prev. Med. 50, 13-18. doi: 10.1016/j.ypmed.2009.12.009. Epub 2009 Dec 21.

Ananth, C.V., Savitz, D.A., Luther, E.R., 1996. Maternal cigarette smoking as a risk factor for placental abruption, placenta previa, and uterine bleeding in pregnancy. Am. J. Epidemiol. 144, 881-889. doi: 10.1093/oxfordjournals.aje.a009022.

Andres, R.L., Day, M.C., 2000. Perinatal complications associated with maternal tobacco use. Semin. Neonatol. 5, 231-241. doi: 10.1053/siny.2000.0025.

Aydın, Y., Ergül, A., 2015. Determining the degree of cigarette addiction of pregnant smokers Journal of Dependence. 16, 49-56.

Cook, G.D., Strachan, D.P., 1999. Health effects of passive smoking-10: Summary of effects of parental smoking on the respiratory health of children and implications for research. Thorax. 54, 357-366. doi: 10.1136/thx.54.4.357.

Dawn, P., Nan, A., 2005. Maternal smoking and birth weight: Interaction with parity and mother's own in utero exposure to smoking. Epidemiology. 16, 228-293. doi: 10.1097/01.ede.0000158198.59544.cf.

Di Franza, J.R., Lew, RA., 1995. Effect of maternal cigarette smoking on pregnancy complications and sudden infant death syndrome. J. Fam. Pract. 40, 385-394. doi: 10.3109/13814789509160303.

Doğanay, S., Sözmen, K., Kalaça, et al. 2012. How has the prevalence of smoking changed in Turkey? Turk. J. Public. Health. 10, 93-115.

Goel, P., Radotra, A., Singh, I., Aggarwal, A., Dua, D., 2004. Effects of passive smoking on outcome in pregnancy. J. Postgrad. Med. 50, 12-16.

Hofhuis, W., Merkus, P.J., de Jongste, J.C., 2002. Negative effects of passive smoking on the child. Ned. Tijdschr. Geneeskd. 146, 356-359.

Karatay, G., Kubilay, G., 2004. The determination of substance use and influenced factors at two highschool; which have different socio-economic status. Journal of Research and Development in Nursing. 1, 57-70.

Kilicarslan, S., 2008. Sociodemographic characteristics, health qualities and anxiety levels of third trimester pregnant women living in Edirne. University of Trakya, Medical School Family Medicine Department. Dissertation Thesis. Edirne.

Koçak, C., Kurçer, M.A., Arıkan I.I., 2015. Knowledge, attitudes, and behaviours of pregnant women regarding smoking who were admitted to the obstetrics clinic of the Bulent Ecevit University Hospital. Istanbul. Med. J. 16, 133-136. doi: 10.5152/imj.2015.66588.

Lantz, P.M., House, J.S., Lepkowski, J.M., Williams, D.R., Mero, R.P., Chen, J., 1998. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of us adults. JAMA. 279, 1703-1708. doi:10.1001/jama.279.21.1703.

Mathews T.J., 2004. Div of vital statistics, national center for health statistics; CC Rivera, Div of reproductive health, National Center for Chronic Disease Prevention and Health.

Mackay, J., Ericsen, M., 2002. The tobacco atlas. World Health Organization.

Mackay, J., Amos, A., 2003. Women and tobacco. Respirology. 8, 123-130. doi: 10.1046/j.1440-1843.2003.00464.x.

Marakoğlu, K., Duygu, E., 2007. Attitudes and behaviours of pregnant women about smoking in Konya. Erciyes. Med. J. 29, 047-055.

Marakoğlu, K., Sezer, R., 2003. Smoking in pregnancy in Sivas. Cumhuriyet. Tıp. Dergisi. 25, 157-164.

Nakamura, M.U., Alexandre, S.M., Kuhn dos Santos, J.F., et al.2004. Obstetric and perinatal effects of active and/or passive smoking during pregnancy. Sao. Paulo. Med. J. 122, 94-98. doi: 10.1590/S1516-31802004000300004.

Olsen, J.,1992. Cigarette smoking in pregnancy and fetal growth. Does the type of tobacco play a role? Int. J. Epidemiol. 21, 279-284. doi: 10.1093/ije/21.2.279.

Ozmen, D.B., 2004. The effects of smoking during pregnancy on the human placenta and newborn infant health. Ministry of

Health of Republic of the Turkey Sisli Etfal Training and Research Hospital. Gynaecology and Obstetrics. Dissertation Thesis. Istanbul.

Promotion, CDC. Smoking during pregnancy-United States, 1990-2002. MMWR Morb.Mortal. Wkly. Rep. 8, 911-915.

Semiz, O., Sözeri, C., Cevahir, R., et al. 2006. Some characteristics of the smoking profiles of pregnant women who applied to a health center in Sakarya. STED. 15, 149-152.

Taşkıran, N., 2009. Assessment of sleep quality in pregnant women. Nursing of Department of Gynaecology and Obstetrics. Master's Thesis. Afyon.

Toyran, M., 2005. The effects of smoking during pregnancy on infant health. Clinic. Pediatri. 4, 17-23.

Turkish Statistical Institute, 2014. Global Adult Tobacco Survey Micro Data Set 2012. Ministry of Health Publication No: 948, Ankara, 2014.

Turkish Statistical Institute Health Survey Ankara; 2013.

Windham, G.C., Hopkins, B., Fenster, L., Swan, S.H., 2000. Prenatal active or passive tobacco smoke exposure and the risk of preterm delivery or low birth weight. Epidemiology. 11, 427-433. doi: 10.1097/00001648-200007000-00011.

World Health Statistics, 2014. World Health Organization. http://apps.who.int/iris/bitstream/10665/112738/1/9789240692671_eng.pdf.

Yeltekin, S., Karasimav, D., Yalçın, S., 2005. Effects of passive tobacco exposure on birth weight at Gümüşdere Health Station. STED. 14, 90-93.

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# Some sociodemographic factors on smoking cessation rate in Konak smoking cessation outpatient clinic

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**ABSTRACT**

The aim of this study was to describe the sociodemographic characteristics and evaluate effects of attitudes and knowledge about smoking on the effectiveness of smoking cessation in patients attending to Konak Smoking Cessation Outpatient Clinic (SCOC). In this retrospective study, files of patients that admitted to İzmir Konak SCOC between 2009 and 2010 were investigated. Patient files consisted of; i) the "Patient Evaluation Form" ii) the Fagerstrom Test, iii) the Hospital Anxiety and Depression Scale (HADS). A patient who didn't smoke for a year was accepted as cessation and other cases were accepted as recurrences. Chi-square, Mann-Whitney, and Student's t-test were used for statistical analyses. 1.508 patient files were examined. 50.0% of the patients were female, mean age was 42.37±12.17, dependency score was high/very high in 52.9% of the cases. The "cessation" rate was 38.7% for the whole group. Cessation rate of the 15-19 age group was lower compared to other age groups ($p<0.05$). Cessation rate was lower among singles compared to married or divorced/widows ($p<0.05$). The cessation rate of students was lower compared to working and non-working patients ($p<0.05$). The average age of starting smoking was 16.87±4.86. The risk of recurrence was higher in the presence of other people smoking at home or at work (RR: 1.4, $p<0.001$). Increase in the Fagerstrom dependency anxiety and depression scores were observed in the case of recurrence ($p<0.001$). The cessation rates to quit smoking were lower, the younger the age the patient started smoking ($p<0.05$). Cessation rates of patients attending to Konak SCOC after one year follow ups are high. Interventions to increase the application rates of young people and the promotion of integrated health activities for adolescents will increase the efforts toward the tobacco epidemic.



## 1. Introduction

Tobacco use is a risk factor for the six leading causes of death in the world; coronary heart diseases (CHD), cerebral vascular diseases, lower respiratory tract infections, chronic obstructive pulmonary disease, tuberculosis, and tracheal-bronchial-lung cancer. Worldwide, tobacco use is responsible for 12% of all deaths in people 20 years and older (World Health Organization (WHO), 2008). In 2014, around 5 million adults died due to tobacco use. This means one death in every six seconds. Among all deaths, the two WHO regions with the highest tobacco usage rates are America (16%) and Europe (16%). In all WHO regions, deaths due to tobacco usage are higher in man according to women (WHO, 2012).

Tobacco use is quite common in Turkey. In Turkey,

approximately two out of five men and one out of five women are smokers (Bilir, 2009). According to the "Turkey Chronic Respiratory Diseases (Asthma-COPD) Prevention and Control Program Action Plan (2009-2013)" one of the strategies under the title "To prevent the development of chronic airway disease, reduce morbidity and mortality" is to reduce tobacco use (Ministry of Health of Turkey, 2009). In the Strategic Plan (2013-2017) of the Ministry of Health of the Turkish Government, the current situation and tobacco control targets are stated as follows: Rate of tobacco use among adults (15 years and older) was 37.3% for male population in 2012. The target is to reduce tobacco use of men to 30% by 2017 and to 22% by 2023. The same rate for women was 10%.7 in 2011. The target is to reduce tobacco use among women to 10% by 2017 and to 8% by 2023 (Ministry of Health of Turkey, 2012).

All the activities of this legislation and practices are reflected in the survey results. When results of the Global Adult Tobacco Survey held in 2008 conducted by the Turkey Statistical Institute (TUIK) are compared to results of 2006, a decrease from 33.4% to 31.3% is observed in general population. About 7% of smokers quit smoking and tendency to quit tobacco use is more common among young people (TUIK, 2008).

The most important achievement in the fight against smoking is to prevent people start smoking (primary prevention). However, when current smoking rates are put into consideration, cessation policies also come to the fore. Therefore, Smoking Cessation Outpatient Clinics (SCOC) were established in Turkey in order to support the cessation of the smoking habit. In 2012 the number of these clinics was 413 and served to 498.294 people between January 2011 and March 2012 (Ministry of Health of Turkey 2012b).

In our study, the aim was to describe the sociodemographic characteristics and evaluate effects of attitudes and knowledge about smoking on the effectiveness of smoking cessation in patients attending to Konak SCOC.

## 2. Matherial and methods

### 2.1. Design of the study

In this retrospective study files of patients that admitted to İzmir Konak SCOC between 2009 and 2010 were reviewed. Treatments and follow-ups were conducted by three certified doctors. In this study, sampling was not used. All of the 1.508 patient files were examined.

### 2.2. Definition of study area

Konak SCOC is a health unit consisting of one education room, two examination rooms and patient waiting area. Patients attending the SCOC first are invited to join a group meeting, consisting of groups between 10-20 people and are given information about the hazards of smoking, and the system of the SCOC. Patients who decide to "quit" are given individual appointments. Follow ups are planned according to the patient. Patients who don't visit the clinic regularly are called by phone after three months, six months and one year and are asked about their status. Cessation treatment is given by doctors, certified for "tobacco treatment and training". This certificate is approved by the Ministry of Health. Medical and behavioral therapy are used for all patients. Patients are expected to continue the follow ups for at least one year. CO measurements were conducted at the beginning of the treatment and once or twice at some follow ups, in order to show the patients their "success" and ensure motivation.

### 2.3. Tools

Patient files consisted of; i) the "Patient Evaluation Form" containing questions about demographic characteristics, smoking attitudes and behaviors, ii) the Fagerstrom Test, used for detecting the level of dependency, and iii) the Hospital Anxiety and Depression Scale (HADS). Fagerstrom Test for Nicotine Dependence has been developed to determine the level of physical dependence on nicotine. This test consists of six simple questions. Each question has 2-4 answers (yes/no, multiple choice). Each answer is scored between 0 and 3, and the total score of the test varies from 0 to 10. While 0-3 points show a low degree of addiction, 4-6 points stand for intermediate, and 7-10 points stand for a high degree of nicotine addiction (Fagerstrom et al., 1990).

The HADS has been developed by Zigmond and Snaith in 1983 to determine the risk and measure the level and change of anxiety and depression in patients. The Turkish validity and reliability study for this scale was conducted by Aydemir et al. (1997). This test does not intend to diagnose patients physically ill or patients attending to primary health care units, but to determine risk groups for anxiety and depression as soon as possible. The test consists of 14 questions. While seven questions assess anxiety, the other seven questions aim to assess depression. Questions about anxiety are given with even numbers and questions about depression are given odd numbers. Responses are collected using a Likert scale, scored between 0-3. Scoring of each item in this scale is different. Questions 1, 3, 5, 6, 8, 10, 11 and 13 gradually decrease in power and scoring is from 3 towards 0 (3, 2, 1, 0). On the other hand items 2, 4, 7, 9, 12 and 14 are scored from 0 towards 3. For the anxiety subscale, scores of questions numbered 1, 3, 5, 7, 9, 11 and 13, and for the depression subscale 2, 4, 6, 10, 12 and are collected. For both subscales, the range of scores varies between 0 (lowest) through 21 (highest). The cut-off point of the Turkish version of HADS is determined as 10 for the anxiety subscale and as 7 for depression (Dönmez et al., 2012).

### 2.4. Definitions

Having not smoked for a year was considered as "cessation" and continuing/re-starting smoking as "recurrence". Information about the "cessation or recurrence status" of patients was obtained by telephone follow ups or by declaration of patients visiting the SCOC after completing one year follow ups. Necessary permits to carry out the study were taken from the Public Health Directorate of Izmir.

### 2.5. Statistical analyses

SPSS 15.0 was used for statistical analyses. Descriptive data is given by number, percentage and mean values (with standard deviation and minimum-maximum) and median values (with interquartile range [IQR]). Chi-square, Mann-Whitney, and Student's t-test were used for bilateral comparisons.

### 2.6. Ethics

Approval for the study was granted by Izmir Public Health Directorate and Konak Community Health Center.

### 2.7. Limitations

In this study only the files of Konak SCOC were used. Patient files were manually filled by the doctors and no detailed electronic records were available. Therefore it was not possible to group patients according to their medical treatment time and type of medication.

Another limitation might be the group education given at the beginning of the treatment. We must accept that patients deciding to take an appointment after this pre-education were probably more motivated and more likely to "quit".

## 3. Results

In this study 1.508 patient files were examined. %50.0 (n=754) of the patients were female, %67.7 (n=1.02) were married, mean age was 42.37±12.17 (min=15, max=80) and median age was 42 (IQR25=33, IQR75: 51). There was no statistically significant difference between mean age of men (42.93±13.14) and women (41.80±11.09) (t=1.808; p=0.07).

High school and/or higher educational attainment rate was 61.0% (n=920) and the rate of patients actively working in a job was 55.8% (n=841). 3.3% of these applicants (n=50) were students.

Some socio-demographic characteristics of the study group are presented in Table 1.

The dependence score was high/very high in 52.9% (n=797) cases, medium in 14% (n=221) and low / very low in 33.2% (n=500) of the cases.

The "cessation" rate was 38.7% (n=584) for the whole group. There was no significant difference between the cessation rates of men (%40.7, n=307) and women (%36.7, n=277) (p>0.05).

**Table 1.** Sociodemographic characteristics of the study group

| Characteristics (N: 1.508) | n (%) |
|---|---|
| **Gender** | |
| Male | 754 (50.0) |
| Female | 754 (50.0) |
| **Age (years)** | |
| 15-19 | 34 (2.3) |
| 20-29 | 199 (13.2) |
| 30-39 | 391 (25.9) |
| 40-49 | 463 (30.7) |
| 50-59 | 292 (19.4) |
| 60 and over | 129 (8.6) |
| **Marital status** | |
| Married | 1021 (67.7) |
| Single | 449 (29.8) |
| Widow/divorced | 38 (2.5) |
| **Educational level** | |
| Illiterate | 20 (1.3) |
| Literate | 26 (1.7) |
| Primary school | 356 (23.6) |
| Junior high school | 186 (12.3) |
| High school | 434 (28.8) |
| University | 486 (32.2) |
| **Working status** | |
| Employed | 841 (55.8) |
| Unemployed | 617 (40.9) |
| Student | 50 (3.3) |

There was a statistically significant difference between cessation and recurrence among different age groups; the cessation rate of the 15-19 age group was found to be lower compared to other age groups ($p<0.05$). We also found that the rate of quitting smoking was lower among singles (never married before) when compared to married or divorced/widows ($p<0.05$). The risk of "recurrence" was significantly higher among singles compared to married (RR=1.4; %95 CI=1.11-1.78, p=0.005).

There was no correlation between cessation/recurrence and educational level (p>0.05).

The cessation rate of students was lower compared to working and non-working patients ($p<0.05$). Also the risk for "recurrence" was higher in students compared to the rest of the group (RR=2.8; %95CI=1.3-5.9, p=0.006).

Distributions of cessation and recurrence status of the study group according to some socio-demographic features are presented in Table 2.

The average age of starting smoking in the study group was 16.87±4.86 (min: 4 max: 48) and the median age of starting smoking was 17 (IQR25=14, IQR75=19). There were 12 cases who started smoking under the age of seven, and five patients started smoking over 40 years of age.

Emulation (48.8%, n=736), curiosity (35.5%,

**Table 2.** Distribution of cessation and recurrence of smoking cessation according to some sociodemographic characteristics

| | Cessation (%) | Recurrence (%) | $x^2$; p |
|---|---|---|---|
| **Gender** | | | |
| Male | 307 (40.7) | 447 (59.3) | 2.515; 0.113 |
| Female | 277 (36.7) | 477 (63.3) | |
| **Age group (years)** | | | |
| 15-19 | 6 (17.6) | 28 (82.4) | |
| 20-29 | 69 (34.7) | 130 (65.3) | |
| 30-39 | 147 (37.6) | 244 (62.4) | 12.912; 0.024 |
| 40-49 | 177 (38.2) | 286 (61.8) | |
| 50-59 | 127 (43.5) | 165 (56.5) | |
| 60 and over | 58 (45.0) | 71 (55.0) | |
| **Marital status** | | | |
| Married | 413 (40.5) | 608 (59.5) | |
| Single | 151 (33.6) | 298 (66.4) | 9.290; 0.010 |
| Divorced/widow | 20 (52.6) | 18 (47.4) | |
| **Educational level** | | | |
| Illiterate | 5 (25.0) | 15 (75.0) | |
| Literate | 14 (53.8) | 12 (46.2) | |
| Primary school | 132 (37.1) | 224 (62.9) | 10.328; 0.066 |
| Junior high school | 79 (42.5) | 107 (57.5) | |
| High school | 151 (34.8) | 283 (65.2) | |
| University | 203 (41.8) | 283 (58.2) | |
| **Working status** | | | |
| Employed | 321 (38.2) | 520 (61.8) | |
| Unemployed | 253 (41.0) | 364 (59.0) | 8.849; 0.012 |
| Student | 10 (20.0) | 40 (80.0) | |
| **Total** | 584 (38.7) | 924 (61.3) | |

and stress/sadness (18.0%, n=271) were the main reasons to begin smoking.

Fear of future illnesses (%67.3, n=1.015), the idea of giving harm to others (%42.3, n=638) and economic reasons (%35.6, n=537) on the other hand, were the main reasons for wanting to quit smoking.

Having cancer him/herself or knowing a family member having cancer, having a respiratory system disease or any other systemic disease did not affect cessation p>0.05 for each).

The risk of recurrence was higher in the presence of other people smoking at home or at work (RR= 1.4, %95 CI=1.1-1.7, p<0.001). "Having tried to quit before" had no impact on cessation (p>0.05).

The relation between smoking cessation cessation/recurrence, Fagerstrom dependency score, anxiety and depression scores and the age of starting smoking were assessed.

Increase in the Fagerstrom dependency anxiety and depression scores were observed in case of recurrence p<0.001 for each). In addition, the cessation rates to quit smoking were lower, the younger the age the patient started smoking (p<0.05) (Table 3).

## 4. Discussion

In this study, smoking cessation cessation/recurrence points were evaluated according to certain sociodemographic characteristics. Gender and education level had no effect on smoking cessation (p>0.05). However, recurrence to quit smoking appears to be higher in the 15-19 age group than other age groups. In this study, being a student and being single has also been identified as a risk of recurrence in smoking cessation (p<0.05). It can be said that the "age" variable reflects the status of being "unmarried" and being a "student" since most of the younger participants were students and singles.

Although no evidence was shown between the age of starting smoking and cessation/recurrence on quitting smoking in this study (p>0.05), low cessation rates of smoking cessation among younger age groups demonstrates the importance of interventions held to prevent starting tobacco use at these ages. This study was conducted on the records of the people, who decided to quit smoking and admitted to SCOC. The median age for starting smoking in this group was 17 and this is a late age to start smoking, considering the general population of Turkey. By the Psychiatric Association of Turkey, it is estimated that the age of starting smoking is 10-11 years old. It should be considered that this very young group don does not think about quitting smoking and therefore don't apply to any healthcare provider. In a study by Arguder et al. (2013) conducted in Ataturk Training and Research Hospital, the median age of starting smoking among patients who admitted the SCOC was reported 16 years which is similar to our study. In the study above, gender, educational level, marital status and age of starting smoking was similar in both groups (cessation/recurrence). It must be considered that "cessation" was defined as "three months for the cessation of smoking cessation", while in our study the criteria was "one year". However, results of our work were similar to the results of Arguder and friends (2013). In a study by Fidan et al. (2005) smoking cessation rate was lower among patients who started smoking 15 years and earlier which is compatible with our results.

In studies investigating the reasons for starting smoking, main causes among college/university students are found to be; distress/anxiety, friends, psychological problems, stress, emotional space and emulation (Picakciefe et al., 2007; Hassoy et al., 2011). In a study among High School students, curiosity, peer pressure, and stress have been reported as the most common reasons for starting smoking (Golbasi et al., 2011). In another study conducted in primary schools, curiosity came to the fore as the main reason. In our study, emulation, curiosity, and stress/sadness causes

**Table 3.** Relationship between cessation/recurrence status and Fagerstrom dependency, anxiety and depression scores

| Spearman correlation (r; p) | Fagerström score | Anxiety score | Depression score | Starting age of smoking |
|---|---|---|---|---|
| Cessation/recurrence | r=0.156<br>p<0.01 | r=0.118<br>p<0.01 | r=0.108<br>p<0.01 | r=- 0.058<br>p<0.05 |

have been identified as the main reasons for starting smoking. We can say that results are consistent with literature. Some publications outside of Turkey also state similar results (Hamzacebi et al., 2008; Taheri et al., 2014; Povlsen et al., 2016).

Fear of future illnesses, idea of giving harm to others and economic reasons were the main reasons for wanting to quit smoking in our study. Similarly in Fidan's study, fear of future illnesses, existing disease, idea of giving harm to others, public pressure and economic reasons were reasons why patients wanted to quit smoking (Fidan et al., 2005).

The presence of other people smoking at home or at work was another risk factor affecting cessation negatively in our study. Fichtenberg and Glanz (2002) reported that working in establishments that restrict smoking, decreased frequency and daily cigarette consumption. In a study with participants from Canada, USA, Britain, and Australia, it was shown that no-smoking family members increase the frequency of attempts to quit smoking as well as the cessation of smoking cessation (Borland et al., 2006). These results are also similar to our study.

In a study conducted on 1.567 students at Kocaeli University, it was reported that close to half the students tried to quit smoking at least once, but could not be cessationful (Boyaci et al., 2003). Mayda et al. (2007) also reported in a study that among medical school students in Duzce, three-quarters of the students tried but could not succeed quit smoking. Literature results indicate that attempts to quit smoking show recurrence in the absence of professional support. In our study, the impact of having tried to quit smoking earlier on "cessation" was not observed. This result can count as evidence that smoking cessation can be successful on the "first attempt" if professional support can be provided.

In our study, the withdrawal of the age of starting smoking at an early age resulted to lead to a decrease in smoking cessation. Boyaci et al. (2003) obtained similar results in their study.

The role of negative effects of smoking on the cardiovascular and respiratory systems and on the pathogenesis of cancer is well known (Şahin and Güven, 2011). Therefore, smokers with chronic diseases or having sick friends/family are expected to be more motivated to quit cigarettes. But in this study, having cancer him/herself or knowing a family member having cancer, having a respiratory system disease or any other systemic disease did not affect cessation. The reason for this may be that the study population included also young people.

"Recurrence" was higher when Fagerstrom dependency, anxiety and depression scores showed increase. Teneggi and colleagues (2002) reported that nicotine addiction affected treatments to quit smoking. The association of smoking addiction with mental illnesses such as depression and anxiety are known. In a one-year follow-up study by Yasar and friends (2014), "cessation" rates showed decrease with high dependency scores, but no evidence was shown for the effect of anxiety and depression. Although anxiety and depression usually show comorbidity in smokers, the fact that anxiety and depression can be influenced by many factors may affect cessation negatively. However, as we expected in our study, the presence of anxiety and depression decreased cessation of smoking cessation.

To conclude, one year "cessation" rates of patients attending to Konak SCOC are high. Interventions to increase the application rates of young people (promotional activities, collaboration with family physicians, educators, employers, etc.) and the promotion of integrated health activities for adolescents will increase the efforts toward the tobacco epidemic. Anxiety and depression must be taken under consideration due to comorbidity and adverse effects on therapy.

## REFERENCES

Argüder, E., Karalezli, A., Hezer, H., Kılıç, H., Er, M., Hasanoğlu, H.C., Demir, P., 2013. Factors affecting the success of smoking cessation. Turkish Thoracic Journal. 14, 81-87. doi: 10.5152/ttd.2013.18.

Aydemir, Ö., Güvenir, T., Küey, L., Kültür, S., 1997. Validity and reliability of Turkish version of hospital anxiety and depression Scale. Turkish Journal of Psychiatry. 8, 280-287.

Bilir, N., 2009. Level of tobacco control in Turkey. Turkish Thoracic Journal. 10, 31-34.

Borland, R., Yong, H.H., Cummings, K.M., Hyland, A., Anderson, S., Fong, G.T., 2006. Determinants and consequences of smoke-free homes: findings from the International Tobacco Control (ITC) Four Country Survey. Tobacco Control. 15(Suppl 3):iii42-iii50.

Boyaci, H., Çorapçıoğlu, A., Ilgazlı, A., Başyiğit, İ., Yıldız, F., 2003. The evaluation of smoking habits in Kocaeli University students. Solunum Hastalıkları. 14, 169-175.

Dönmez, S., Dağ, H., Kazandı, M., 2012. The determination of pregnant women's anxiety and depression risk levels before amniocentesis. Journal of the Acibadem University of Health Sciences. 3, 255-261.

Fagerstrom, K.O., Heatherton, T.E., Kozlowski, L.T., 1990. Nicotine addiction and its assessment. Ear Nose Throat J. 69, 763-765.

Fichtenberg, C.M., Glantz, S.A., 2002. Effect of smoke-free workplaces on smoking behavior: Systematic review. BMJ. 325(7357):188.

Fidan, F., Pala, E., Ünlü, M., Sezer, M., Kara, Z., 2005. Factors affecting smoking cessation and success rates of the treatment methods used. The Medical Journal of Kocatepe. 6, 27-34.

Golbasi, Z., Kaya, D., Cetindag, A., Capik, E., Aydogan, S., 2011. Smoking prevalence and associated attitudes among high school students in Turkey. Asian Pac. J. Cancer Prev. 12, 1313-1316.

Hamzaçebi, H., Ünsal, M., Dabak, Ş., Bilgin, S., Aker, S., 2008. Smoking prevalence of smoking and affecting factors in primary school students in Tekkeköy, Samsun. Toraks Dergisi. 9, 34-39.

Hassoy, H., Ergin, I., Davas, A., Durusoy, R., Karababa, A.O., 2011. Determining the factors effecting the cigarette, narghile and hand-rolled tobacco smoking among medical technology vocational training school students and evaluation of their opinions about starting and continuing with their habits of smoking. Respiratory. 13, 91-99.

Mayda, A.S., Tufan, N., Baştaş, S., 2007. Attitudes towards smoking and frequency of smoking among students of Düzce Medical school. TAF. Prev. Med. Bull. 6, 364-370.

Picakciefe, M., Keskinoglu, P., Bayar, B., Bayar, K., 2007. [Smoking prevalence among Muğla school of health sciences students and causes of leading increase in smoking]. TAF Prev. Med. Bull. 6, 267-272.

Povlsen, L., Aryal, U.R., Petzold, M., Krettek, A., 2016. Adolescents' knowledge and opinions about smoking: A qualitative study from the Jhaukhel-Duwakot Health Demographic Surveillance Site, Bhaktapur District, Nepal. Int. J. Adolesc. Med. Health. 2016 Apr 9.

Smoking and Mental Health. 2008. file:///C:/Users/Acer/Downloads/f-11-sigara-ve-ruh-sagligi-32.pdf (Access: 25.05.2016).

Şahin, U., Güven, G.S., 2011. Smoking in elderly and efforts to cessation Is it worth dealing? Academic Geriatrics. 3, 1-12.

Taheri, E., Ghorbani, A., Salehi, M., Sadeghnia, H.R., 2014. Cigarette smoking behavior and the related factors among the students of Mashhad University of medical sciences in Iran. Iran Red. Crescent. Med. J. 17, e16769.

The Psychiatric Association of Turkey. Nicotine (smoking) addiction. http://www.psikiyatri.org.tr/pagepublic.aspx?menu=50 (Access: 25.05.2016).

Teneggi, V., Squassantre, L., Iavarone L, Milleri, S., Bye, A., Gömeni, R., et al., 2002. Correlation and predictive performances of saliva and plasma nicotine concentration on tobacco with drawal-induced craving. Br. J. Clin. Pharmacol. 54, 407-414.

Turkey Statistical Institute. 2008. Global Adult Tobacco Survey-2008.

Uysal, M.A., Kadakal, F., Karşıdağ, Ç., Bayram, N.G., Uysal, Ö., Yılmaz, V., 2004. Fagerstrom test for nicotine dependence: Reliability in a Turkisk sample and factor analysis. Tüberküloz ve Toraks Dergisi. 52, 115-121.

WHO. 2008. WHO Report on the Global Tobacco Epidemic.

WHO. 2012. WHO Global Report: Mortality Attributable to Tobacco.

Yaşar, Z., Kurt, Ö.K., Talay, F., Kargı, A., 2014. One-Year Follow-up results of smoking cessation outpatient clinic: Factors affecting the cessation of smoking. Eurasian. J. Pulmonol. 16, 99-104.

Zigmond, A.S., Snaith, R.P., 1983. The hospital anxiety and depression scale. Acta Psychiatrica Scandinavica. 67, 361-370.

Journal of Experimental and Clinical Medicine
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# The association between smoking and obesity in Iranian adult population: A Study based on third national surveillance of the risk factors of the noncummunicable diseases (SuRFNCD-2007)

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**ABSTRACT**

Smoking and smoking cessation are presumed to have associations with body weight and central adiposity. This study aims to investigate the relationship between these factors in a large sample of the Iranian adult population. We collected the data regarding smoking status, weight, height and waist circumference (WC) from 5287 Iranian individuals aged 15-64 years who participated in the third national surveillance of risk factors of non-communicable diseases (SuRFNCD) in March 2007. The BMI and WC values were investigated in smoker, ex-smoker and never-smoker groups using univariate and multivariate analyses. In the univariate analysis, ex-smokers had significantly higher rate of general obesity (p=0.002), central obesity (P<0.001), mean BMI (p<0.001) and mean WC (p<0.001) compared to the current smoker group. Although smokers had significantly lower rate of obesity (p=0.003) and mean BMI (p<0.001) compared to non-smokers, they had significantly higher WC (p=0.016). Interestingly, among female subjects, smokers had higher rate of obesity (p=0.006) and BMI (p=0.006) than non-smokers, while ex-smokers were not more obese than smokers. However, smoking status was not independently associated with obesity or central obesity in the multivariate regression analysis. Since smoking seems to be associated with higher risk of central obesity, the misleading notion that smoking causes weight loss should be avoided. Iranian women should be more cautious if they tend to stay slim by the false belief that smoking induces weight loss.



## 1. Introduction

Smoking and obesity are important preventable risk factors of non-communicable diseases in the world and particularly, in Iran (Esteghamati et al., 2010; Meysamie et al., 2010). Smoking and obesity can together increase the risk of cerebrovascular, cardiovascular and systemic atherosclerotic diseases (Honjo et al., 2010). Life expectancy of overweight smokers is estimated to be 13 years lower than normal weight non-smokers (Peeters et al., 2003). According to available body of evidence, smoking is presumed to be associated with central adiposity despite decreased weight (Barrett-Connor and Khaw, 1989). Significant changes in both waist circumference (WC) and waist-to-hip ratio (WHR) along with lipid profile disturbances have been seen among smokers (Caks and Kos, 2009; Meysamie

**Table 1**. Prevalence of central obesity in different smoking strata divided by participant characteristics; Iran 2007

| | | Central obesity -IR | | | | |
|---|---|---|---|---|---|---|
| | | Smoker | Ex-smoker | Nonsmoker | p | |
| Gender | Male | 38.8% (34.1%-43.8%) | 50.6% (41.7%-59.4%) | 36.1% (33.2%-39.1%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.021<br>0.002<br>0.335 |
| | Female | 47.3% (32.2%-62.8%) | 75.3% (41.9%-92.8%) | 34.8% (32.5%-37.1%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.199<br>0.038<br>0.094 |
| | p | 0.283 | 0.237 | 0.490 | | |
| Habitant | Rural | 31.7% (23.7-40.8) | 38.6% (25.6-53.5) | 29.8% (26.7-33.1) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.367<br>0.173<br>0.660 |
| | Urban | 42.3% (37.0-47.9) | 58.2% (47.7-68.1) | 37.7% (35.5-40.0) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.009<br><0.001<br>0.111 |
| | p | 0.036 | 0.024 | <0.001 | | |
| Age | =<40 | 34.2% (28.5-40.3) | 39.0% (24.2-56.0) | 26.1% (24.0%-28.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.544<br>0.061<br>0.006 |
| | >40 | 47.5% (40.4%-54.7%) | 60.8% (51.6%-69.3%) | 64.8% (62.1%-67.4%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.027<br>0.405<br><0.001 |
| | p | 0.004 | 0.016 | <0.001 | | |
| Obesity | Non Obese | 31.7% (27.4%-36.2%) | 39.5% (30.5%-49.2%) | 23.8% (22.1%-25.6%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.116<br><0.001<br><0.001 |
| | Obese | 95.1% (88.3%-98.0%) | 100.0% (---) | 89.7% (86.9%-92.0%) | S vs. ES<br>ES vs. NS<br>S vs. NS | <0.001<br><0.001<br>0.278 |
| | p | <0.001 | <0.001 | <0.001 | | |
| Total | | 39.3% (34.7%-44.0%) | 52.3% (43.7%-60.8%) | 35.4% (33.6%-37.2%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.009<br><0.001<br>0.116 |

**S:** Smoker**; ES:** Es-smoker**; NS:** Non-smoker

et al., 2012). Not only smoking increases central fat accumulation, but also is usually clustered with some other risk factors like poor diet, alcohol consumption and low physical activity (Chiolero et al., 2008), which may lead to weight gain. On the other hand, smoking can cause weight loss by increasing daily energy expenditure by 10% (Hofstetter et al., 1986), and reducing appetite (Chiolero et al., 2008). However, contributing pathophysiologic factors in the association between smoking and obesity are yet to be elucidated (Chiolero et al., 2008). Additionally to quit smoking by itself may affect metabolic processes in the human body. Some explanations have been presented, for example it has been shown that pancreatic β-cell secretion increases in response to decreased fasting insulin sensitivity shortly after smoking cessation (Stadler et al., 2014). Also, decreased level of Neuropeptide-Y (NPY) in smoking and its increase after cessation was found to correlate with body weight, WC and BMI. NPY plays a major role in energy homeostasis (Hussain et al., 2012).

Although several researchers have addressed this topic in our country, there is no national representative data regarding the association between smoking and obesity in Iran. Thus, we aimed to clarify this association in the adult population of Iran.

## 2. Material and methods

The third national surveillance of risk factors of non-communicable Diseases (SuRFNCD) was conducted in March 2007 and included 5.287 non-institutionalized individuals aged 15-64 years. The survey was devised in accordance with the STEPs guidelines of the WHO (Esteghamati et al., 2009b). Study sample consisted of clusters of 10 men and 10 women who lived in adjacent residents which were randomly chosen according to the postal codes. Verbal consents were obtained before recording the data. Interviewers asked information about tobacco use among other questions and measured weight, height and WC of the participants in addition to some other examinations. Weight and height were measured using portable calibrated digital weighing scale and portable inflexible measurement tapes. WC was measured using constant-tension tape device, halfway between the lowest border of the ribs (the mid rib 12) and the upmost part of the hip (iliac crest) on the midle axillary line, at the end of normal expiration while the arms were extended and aligned with body.

**Table 2**. Prevalence of central obesity in different smoking strata divided by participant characteristics; Iran 2007

| | | Central obesity -IDF | | | | |
|---|---|---|---|---|---|---|
| | | Smoker | Ex-smoker | Nonsmoker | p | |
| Gender | Male | 25.6% (21.5%-30.2%) | 42.2% (34.0%-50.9%) | 26.0% (23.6%-28.6%) | S vs. ES<br>ES vs. NS<br>S vs. NS | <0.001<br><0.001<br>0.869 |
| | Female | 70.3% (55.0%-82.1%) | 94.2% (66.7%-99.2%) | 58.8% (56.3%-61.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.376<br>0.213<br>0.178 |
| | p | <0.001 | 0.036 | <0.001 | | |
| Habitant | Rural | 19.6% (13.1-28.3) | 32.4% (20.8-46.7) | 39.3% 36.0-42.6) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.046<br>0.278<br><0.001 |
| | Urban | 31.5% (26.7-36.7) | 51.6% (41.4-61.7) | 47.0% (44.7-49.2) | S vs. ES<br>ES vs. NS<br>S vs. NS | <0.001<br>0.389<br><0.001 |
| | p | 0.007 | 0.018 | <0.001 | | |
| Age | =<40 | 23.3% (18.4%-29.1%) | 36.8% (23.1%-53.1%) | 35.7% (33.5%-38.0%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.046<br>0.875<br><0.001 |
| | >40 | 35.8% (29.6%-42.6%) | 51.6% (42.4%-60.7%) | 73.2% (70.8%-75.4%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.005<br><0.001<br><0.001 |
| | p | 0.002 | 0.085 | <0.001 | | |
| Obesity | Non Obese | 19.2% (15.8%-23.1%) | 32.2% (23.9%-41.7%) | 33.9% (32.0%-36.0%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.002<br>0.699<br><0.001 |
| | Obese | 91.8% (81.3%-96.6%) | 96.2% (77.4%-99.5%) | 95.2% (91.4%-97.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.775<br>0.929<br>0.486 |
| | p | <0.001 | 0.008 | <0.001 | | |
| Total | | 39.3% (34.7%-44.0%) | 52.3% (43.7%-60.8%) | 35.4% (33.6%-37.2%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.009<br>0.001<br>0.116 |

**S:** Smoker**; ES:** Es-smoker**; NS:** Non-smoker

### 2.1. Authropometric measurements

We defined the obesity status by Body Mass Index (BMI), determined as weight (Kg)/height ($m^2$). We considered BMI level ≥30 $kg/m^2$ as obesity and BMI level in the 25-30 $kg/m^2$ range as overweight. Central obesity was defined with 3 different available criteria: WC ≥90 cm for both genders according to optimal cut-off for Iranian citizens (Esteghamati et al., 2009a); WC ≥80 for women and WC ≥94 cm for men according to the International Diabetes Federation (IDF) criteria (Alberti et al., 2005); and WC ≥88 cm in females and ≥102 cm in males according to National Cholesterol Education Program-Third Adult Treatment Panel (ATP III) criteria (2001). Current cigarette smokers who had smoked at least 7 cigarettes during the week before recruitment were defined zas smokers. We defined Ex-smokers as those who had not smoke for at least one year. Nonsmokers were those who hadn't smoked.

### 2.2. Statistical analysis

Statistical analysis was conducted by complex sample survey analysis using SPSS statistical package (V20) and STATA Portable (V12). Total prevalence rates were presented with 95% confidence intervals (CI 95%). We used two proportion and two mean comparison tests based on complex sample survey analysis. Finally adjusted Odds Ratios based on complex sample logistic regression analysis were calculated via multivariate analysis.

### 2.3. Ethics

This study has been ethically approved by Iranian Ministry of Health, Treatment, and Medical Education as a national study (SuRFNCD-2007).

## 3. Results

The analysis included the data of 5227 adults for this study excluding 60 participants without valid BMI values from the original database. Out of all, 2631 were male comprising 733 smokers, 166 Ex-smokers and 1795 nonsmokers; and 2596 were female comprising 52 smokers, 11 Ex-smokers and 2533 non-smokers.

The prevalence of central obesity in different smoking strata with Iranian central obesity criteria in Table 1, with IDF criteria in Table 2 and with ATP3 criteria in Table 3.

Based on ATP III criteria, the total prevalence of abdominal obesity was 13.5%, 23.0% and 27.4% among smokers, ex-smokers and nonsmokers, respectively (p<0.001). According to the IDF criteria

**Table 3**. Prevalence of central obesity in different smoking strata divided by participant characteristics; Iran 2007

| | | Central obesity -ATP3 | | | | |
|---|---|---|---|---|---|---|
| | | Smoker | Ex-smoker | Nonsmoker | p | |
| Gender | Male | 11.5% (8.5%-15.4%) | 18.3% (12.7%-25.5%) | 9.8% (8.4%-11.4%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.024<br><0.001<br>0.294 |
| | Female | 47.3% (32.2%-62.8%) | 86.1% (55.3%-96.9%) | 40.7% (38.3%-43.2%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.101<br>0.039<br>0.390 |
| | p | <0.001 | <0.001 | <0.001 | | |
| Habitant | Rural | 5.5% (3.3-8.9) | 13.1% (6.2-25.4) | 23.8% (20.9-27.0) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.009<br>0.020<br><0.001 |
| | Urban | 16.8% (12.8-21.7) | 27.3% (19.2-37.1) | 28.9% (27.0-30.8) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.014<br>0.711<br><0.001 |
| | p | <0.001 | 0.013 | 0.005 | | |
| Age | =<40 | 9.0% (6.3%-12.8%) | 18.0% (8.2%-35.0%) | 19.5% (17.7%-21.4%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.031<br>0.774<br><0.001 |
| | >40 | 20.7% (15.0%-27.9%) | 26.2% (19.2%-34.5%) | 52.2% (49.3%-55.2%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.228<br><0.001<br><0.001 |
| | p | <0.001 | 0.210 | <0.001 | | |
| Obesity | Non Obese | 5.5% (3.9%-7.6%) | 10.2% (5.6%-17.6%) | 15.3% (13.9%-16.9%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.024<br>0.075<br><0.001 |
| | Obese | 71.8% (57.8%-82.5%) | 69.3% (49.9%-83.7%) | 84.0% (80.2%-87.2%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.836<br>0.074<br>0.053 |
| | p | <0.001 | <0.001 | <0.001 | | |
| Total | | 13.5% (10.5%-17.2%) | 23.0% (16.7%-30.7%) | 27.4% (25.8%-29.0%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.003<br>0.208<br>0.001 |

**S:** Smoker**; ES:** Es-smoker**; NS:** Non-smoker

a similar pattern was seen (28.1% in smokers vs. 44.7 in non-smokers, p<0.001). Abdominal obesity based on Iranian cut-offs was significantly more prevalent among Ex-smokers (52.3%) than smokers (39.3%) and nonsmokers (35.4%), p<0.001. Female ex-smokers had significantly higher prevalence of central obesity compared to non-smokers (86.1% vs. 40.7%, p=0.039 based on ATP III criteria, 75.3% vs. 34.8%, p=0.038 based on Iranian cut-offs). However, this difference despite seen was not significant between ex and current smokers.

Table 4 shows the prevalence of obesity according to BMI definitions in different smoking strata. Ex-smokers had the highest prevalence of obesity (22.0%), and smokers had the lowest (12.1%), (p=0.002). Obesity was also significantly (p<0.001) more prevalent among Ex-smoker males than smoker and nonsmoker males. Among female participants, Obesity was significantly more prevalent (p=0.006) in smokers (38.8%) than non-smokers (22.4%). The pattern of BMI in different smoking strata is depicted in Fig. 1.

Information on the mean WC is provided in Table 5. Mean WC differed significantly (p<0.001) between Ex-smokers (91.6; CI 95%=89.2-94.0) and smokers (86.2; CI 95%=85-87.5). This amount was 84.6 (CI 95% 84.0-85.1) for nonsmokers, with a significant difference (P=0.016) from the mean for smokers. In females, mean WC was significantly higher (p=0.021) in smokers (89.9, CI 95%=85.3-94.5) than nonsmokers

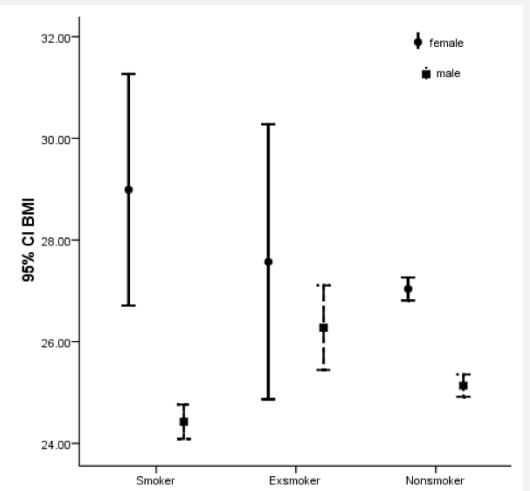


**Fig. 1.** The pattern of body mass index in males and females between different smoking strata

**Table 4**. Prevalence of obesity in different smoking strata divided by participant characteristics; Iran 2007

| | | Obesity | | | | |
|---|---|---|---|---|---|---|
| | | Smoker | Ex-smoker | Nonsmoker | p | |
| Gender | Male | 10.5% (7.7%-14.3%) | 21.8% (15.5%-29.9%) | 11.1% (9.3%-13.1%) | S vs. ES<br>ES vs. NS<br>S vs. NS | <0.001<br><0.001<br>0.727 |
| | Female | 38.8% (25.3%-54.4%) | 25.1% (5.9%-64.1%) | 22.4% (20.6%-24.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.277<br>0.773<br>0.006 |
| | p | <0.001 | 0.737 | <0.001 | | |
| Habitant | Rural | 7.6% (4.5%-12.7%) | 16.2% (7.7%-31.1%) | 14.0% (11.8%-16.6%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.025<br>0.606<br>0.004 |
| | Urban | 13.9% (10.2%-18.6%) | 24.5% (16.8%-34.2%) | 19.0% (17.4%-20.7%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.008<br>0.136<br>0.024 |
| | p | 0.013 | 0.175 | 0.001 | | |
| Age | =<40 | 10.3% (7.3%-14.5%) | 21.0% (10.9%-36.5%) | 12.9% (11.4%-14.6%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.015<br>0.063<br>0.155 |
| | >40 | 14.9% (9.8%-22.0%) | 22.7% (15.6%-31.9%) | 32.2% (29.7%-34.8%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.074<br>0.024<br><0.001 |
| | p | 0.108 | 0.790 | <0.001 | | |
| Total | | 12.1% (9.2%-15.7%) | 22.0% (15.8%-29.9%) | 17.5% (16.2%-18.9%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.002<br>0.133<br>0.003 |
| | | **Overweight** | | | | |
| | | Smoker | Ex-smoker | Nonsmoker | | p |
| Gender | Male | 31.8% (27.6%-36.4%) | 32.8% (24.9%-41.7%) | 31.4% (28.7%-34.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.826<br>0.740<br>0.875 |
| | Female | 21.0% (11.6%-35.0%) | 33.5% (11.9%-65.2%) | 30.7% (28.6%-32.9%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.263<br>0.798<br>0.079 |
| | p | 0.063 | 0.953 | 0.688 | | |
| Habitant | Rural | 25.7% (17.8%-35.7%) | 25.8% (13.3%-44.1%) | 26.0% (23.3%-28.9%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.989<br>0.976<br>0.944 |
| | Urban | 33.4% (28.9%-38.3%) | 35.7% (26.9%-45.7%) | 33.2% (31.1%-35.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.645<br>0.582<br>0.937 |
| | p | 0.112 | 0.224 | <0.001 | | |
| Age | =<40 | 26.8% (21.5%-32.8%) | 23.4% (11.7%-41.4%) | 28.2% (26.1%-30.3%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.616<br>0.456<br>0.634 |
| | >40 | 38.4% (32.5%-44.6%) | 38.7% (30.1%-48.0%) | 40.2% (37.3%-43.1%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.955<br>0.748<br>0.593 |
| | p | 0.004 | 0.055 | <0.001 | | |
| Total | | 31.2% (27.2%-35.6%) | 32.8% (25.3%-41.3%) | 31.0% (29.3%-32.8%) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.710<br>0.642<br>0.928 |

**S:** Smoker**; ES:** Es-smoker**; NS:** Non-smoker

(84.2, CI 95%=83.5-84.9). Mean WC was 86.0 (CI 95% 84.7-87.3) for smoker males and 85.0 (CI 95% 84.3-85.8) for nonsmoker males, but 90.9 (CI 95% 88.6-93.3) for ex-smoker males, which is significantly higher than smoker and non-smoker groups (p=0.001).

As presented in Table 6, mean BMI was also calculated for all subgroups and yielded similar significant differences. Ex-smokers had significantly higher BMI than smokers (p=0.001), and smokers had significantly lower BMI than non-smokers (p=0.001). Smoker females had significantly higher BMI than non-smokers females (p=0.006) and ex-smoker males had significantly higher BMI than smoker males (p=0.001).

While in under 40 age group the mean BMI value and prevalence of obesity are significantly higher in ex-smokers than smokers (p=0.024 and 0.015

**Table 5**. Mean Body mass index and waist circumference among study subjects

| | | BMI | | | | |
|---|---|---|---|---|---|---|
| | | **Smoker** | **Ex-smoker** | **Nonsmoker** | **p** | |
| **Gender** | **Male** | 24.3 (23.8-24.7) | 26.1 (25.1-27.2) | 24.5 (24.2-24.7) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.001<br>0.001<br>0.523 |
| | **Female** | 28.5 (26.0-30.9) | 27.9 (24.6-31.3) | 26 (25.7-26.2) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.854<br>0.308<br>0.006 |
| | **p** | <0.001 | 0.381 | <0.001 | | |
| **Habitant** | **Rural** | 23.6 (22.8-24.3) | 24.7 (23.4-26.0) | 24.4 (24.1-24.7) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.165<br>0.717<br>0.050 |
| | **Urban** | 24.9 (24.3-25.5) | 26.9 (25.6-28.2) | 25.7 (25.5-25.9) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.003<br>0.051<br>0.007 |
| | **p** | 0.006 | 0.039 | <0.001 | | |
| **Age** | **=<40** | 24 (23.5-24.6) | 26.1<br>(23.8-28.5) | 24.5<br>(24.2-24.7) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.024<br>0.076<br>0.206 |
| | **>40** | 25.3 (24.6-26.0) | 26.3 (25.5-27.1) | 28.1 (27.8-28.4) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.146<br>0.002<br>0.001 |
| | **p** | 0.011 | 0.859 | <0.001 | | |
| **Obesity** | **Non Obese** | 23.3 (22.9-23.6) | 24.2 (23.6-24.8) | 23.5 (23.4-23.7) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.038<br>0.120<br>0.191 |
| | **Obese** | 33.4 (32.3-34.5) | 33.6 (31.4-35.8) | 33.7 (33.4-34.0) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.847<br>0.904<br>0.544 |
| | **p** | <0.001 | <0.001 | <0.001 | | |
| **Total** | | 24.5 (24.1-25.0) | 26.3 (25.2-27.3) | 25.3 (25.1-25.5) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.001<br>0.062<br>0.001 |
| | | **Waist circumference** | | | | |
| | | **Smoker** | **Ex-smoker** | **Nonsmoker** | **p** | |
| **Gender** | **Male** | 86 (84.7-87.3) | 90.9 (88.6-93.3) | 85 (84.3-85.8) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.001<br><0.001<br>0.203 |
| | **Female** | 89.9 (85.3-94.5) | 100.9 (91.5-110.3) | 84.2 (83.5-84.9) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.051<br>0.002<br>0.021 |
| | **p** | 0.111 | 0.033 | 0.107 | | |
| **Habitant** | **Rural** | 83.7 (81.6-85.8) | 87.9 (84.7-91.1) | 82.5 (81.6-83.4) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.036<br>0.024<br>0.342 |
| | **Urban** | 87.2 (85.8-88.7) | 93.2 (90.2-96.2) | 85.4 (84.8-86.0) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.001<br><0.001<br>0.027 |
| | **p** | 0.008 | 0.030 | <0.001 | | |
| **Age** | **=<40** | 84.7 (83.1-86.3) | 88.8 84.1-93.5) | 81.7 (81.0-82.3) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.095<br>0.005<br>0.001 |
| | **>40** | 88.7 (86.8-90.6) | 93.4 (91.1-95.7) | 93.8 (93.1-94.4) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.011<br>0.796<br>0.001 |
| | **p** | 0.002 | 0.078 | <0.001 | | |
| **Obesity** | **Non Obese** | 83.8 (82.7-84.8) | 87.1 (85.0-89.2) | 81 (80.5-81.5) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.010<br><0.001<br><0.001 |
| | **Obese** | 104.7 (102.5-106.8) | 107.7 (103.3-112.2) | 101.4 (100.5-102.3) | S vs. ES<br>ES vs. NS<br>S vs. NS | 0.171<br>0.006<br>0.027 |
| | **p** | <0.001 | <0.001 | <0.001 | | |
| **Total** | | 86.2 (85.0-87.5) | 91.6 (89.2-94.0) | 84.6 (84.0-85.1) | S vs. ES<br>ES vs. NS<br>S vs. NS | <0.001<br><0.001<br>0.016 |

**S:** Smoker**; ES:** Es-smoker**; NS:** Non-smoker

**Table 6**. Multivariate Logistic regression analysis for central obesity based on IDF criteria and Iranian cut-offs

| Parameter | IDF criteria | | IR criteria | |
|---|---|---|---|---|
| | p | OR (CI 95%) | p | OR (CI95%) |
| Age >40 vs. =<40 | 0.001 | 2.935 (2.487-3.463) | 0.003 | 3.13 (2.688-3.645) |
| Smoker vs. non-smoker | 0.115 | 0.835 (0.638-1.094) | 0.085 | 1.046 (0.814-1.344) |
| Ex-smoker vs. non-smoker | 0.089 | 1.455 (0.941-2.250) | 0.074 | 1.181 (0.729-1.914) |
| Female vs. male | 0.002 | 7.379 (6.019-9.046) | 0.01 | 0.554 (0.463-0.663) |
| Urban vs. rural | 0.068 | 1.127 (0.927-1.370) | 0.091 | 1.063 (0.891-1.269) |
| Normal vs. obese | < 0.001 | 0.009 (0.005-0.014) | < 0.001 | 0.003 (0.001-0.006) |
| Normal vs. overweight | < 0.001 | 0.058 (0.048-0.071) | < 0.001 | 0.068 (0.056-0.081) |

respectively), these significant differences do not exist in the over 40 age group. Also, the prevalence of obesity and the mean BMI value is not significantly different between smokers and non-smokers in the under 40 age group.

Table 6 demonstrates results of a multivariate logistic regression analysis of the relationship between prevalence of central obesity and covariates. Table 7 demonstrates similar information about the prevalence of obesity. Table 6 shows that the only independent predictors of central obesity are age more than 40 years, female sex and obesity. Table 7 shows that the only independent predictors of obesity are urban residency, female sex and central obesity.

**Table 7.** Multivariate Logistic regression analysis for general obesity based on IDF criteria and Iranian cut-offs

| Parameter | p | OR (CI 95%) |
|---|---|---|
| Age >40 vs. =<40 | 0.075 | 1.124 (0.936-1.351) |
| Smoker vs. non-smoker | 0.256 | 0.931 (0.686-1.263) |
| Ex-smoker vs. non-smoker | 0.122 | 1.438 (0.877-2.358) |
| Female vs. male | 0.001 | 3.425 (2.800-4.189) |
| Urban vs. rural | 0.032 | 1.299 (1.057-1.597) |
| Central obese vs. normal | < 0.001 | 31.969 (24.494-41.725) |

## 4. Discussion

In the present study, we found that average BMI and WC values were higher among ex-smokers. Prevalence of obesity and central obesity were also higher among ex-smokers than smokers. The result is consistent with previous studies reporting higher prevalence of obesity and central obesity among ex-smokers and weight gain after abstinence from smoking (Klesges et al., 1997; Ferrara et al., 2001; Filozof et al., 2004; Janzon et al., 2004; Sulander et al., 2007; Chiolero et al., 2008; Matsushita et al., 2011; Aubin et al., 2012). Pisinger and Jorgensen (2007) reported an almost 4 cm increase in WC of ex-smokers compared to smokers, after one year follow up, which is comparable with our findings (Table 3).

Smokers had significantly higher WC than non-smokers; 86.2 cm (CI 95%, 85-87.5) for smokers vs. 84.6 cm (CI 95%, 84.0-85.1) for non-smokers; while the rate of obesity and the mean BMI value were significantly lower in smokers. This finding indicates that although smoking leads to weight loss, it is associated with higher rates of central adiposity, which is an even more prominent risk factor for cardiovascular diseases (Mendelson et al., 2008; Dhaliwal and Welborn, 2009). Therefore it is necessary to consider smoking as a dual risk factor for cardiovascular disease, and not to be misled by the weight loss it causes. Saarni et al. (2009) reported the same results in their study of 4296 Finnish twins, concluding that smoking is a risk factor for central obesity but not for general obesity. Kim et al. (2012) also reported that although smokers have lower mean BMI values than never smokers, they tend to show more abdominal and visceral obesity. The CT-measured visceral obesity of 4656 Korean men was significantly higher in the smoker group in the study of Lee et al. (2012). Yet some studies have failed to find a positive relationship between smoking and central obesity (Clair et al., 2011; de Oliveira Fontes Gasperin et al., 2014).

When comparing the mean BMI and general obesity estimates between smokers and nonsmokers, gender played a discriminative role. Therefore, among men, smoking was correlated with a lower risk of obesity and lower BMI, whereas among females, smoking was linked with higher risk of obesity and BMI. These results are in agreement with with the results of Cooper et al. (2003) and Saarni et al. (2009). Stice et al. (2015) also reported that female smokers gained more weight than non-smokers (2.9 Kg vs. 0.9 kg) after 2 years of follow-up. This finding is partly because of riskier life style among female smokers which is accompanied by more alcohol consumption and lower physical activity leading to weight gain (Rabaeus et al., 2013). Considering the settings of our study design we could not determine whether smoking by itself caused the higher BMI levels or general obesity.

We also found that the mean BMI value and the prevalence of obesity is significantly higher in under 40 ex-smokers compared to smokers; which is not true about the over 40 age group. This indicates that smoking cessation is more likely to cause weight gain in younger smokers which make them the target for national screening programs. These findings are concordant with Locatelli et al. (2014), but opposed to results of Kasteridis and Yen (2012).

Considering lower BMI and weight among smokers

vs nonsmokers, the role of age should be mentioned, as in under 40 population smoking was not associated with less body weight. Mackay et al. (2013) reported the same age-related pattern in their recent study. Since losing weight is one of the main motivations of young new smokers (Jang et al., 2012; Penzes et al., 2012; Hong and Johnson, 2013), it is crucial to run educational campaigns targeted at younger population in order to inform them that smoking does not guarantee persistent decreased weight.

As discussed, smoking cessation is associated with higher BMI, weight, and central obesity. Thus, in order to assure that beneficial effects of smoking cessation are not attenuated by weight gain and central obesity (Sulander et al., 2007; Inoue et al., 2011; Travier et al., 2012; Komiyama et al., 2015) and to omit the discouraging outlook of weight gain on smokers dissuading them from quitting (Chiolero et al., 2008; Bush et al., 2014; Veldheer et al., 2014; Landrau-Cribbs et al., 2015) we strongly recommend interventions to prevent weight gain and central obesity be included in smoking cessation strategies. Another important finding of our study is that, unlike men, female ex-smokers have lower rate of obesity and BMI value than female smokers. This indicates that smoking cessation in females may not be followed by weight gain. Therefore, female smokers who are discouraged by the concept of gaining weight in case of quitting should be informed that if they quit smoking they will not face the fear of becoming more obese (John et al., 2005).

As smoking is associated with weight gain and central obesity among females, this fact negates the common belief that smoking is a way to lose weight or stay thin among lots of females (Honjo and Siegel, 2003; Penzes et al., 2012; White, 2012). On the other hand, smoking and the correlated general and central obesity are major predictors of cardiovascular and metabolic disorders (Manson et al., 2000; Saarni et al., 2009) and they subsequently decrease life expectancy (Peeters et al., 2003).

Furthermore, smoking cessation is correlated with weight gain and central obesity which in turn put ex-smokers at greater risk of the cardiovascular diseases (Mendelson et al., 2008; Dhaliwal and Welborn, 2009). Smoking prevention seems to be the best option to reduce prevalence of smoking and related morbidities (Saarni et al., 2009). It should be noted that although smoking cessation may lead to weight gain and central obesity, evidence indicate that the benefits of smoking cessation exceed its disadvantages, and smoking cessation does decrease the risk of CVD events (Clair et al., 2013).

However, our multivariate logistic regression analyses showed that smoking status is not an independent predictor of central or general obesity. Confounding factors such as age, sex and general/central obesity may have caused us to overestimate the effect of smoking or smoking cessation on central/general obesity. This issue suggests the necessity of conducting further studies of prospective design in order to better elucidate the causal effect of smoking and smoking cessation on obesity and central obesity.
One of the limitations of this study as a cross-sectional study was that the causal and temporal relationship between different variables could not be established. Another limitation of this study is that we didn't control the confounding effect of some other variables, such as physical activity and diet. We also did not stratify the smoker group based on their amount of exposure, so that light, moderate and heavy smokers were all put into a single group.

Higher prevalence of abdominal and general obesity along with higher mean BMI and WC among Ex-smokers highlights the need to carry out screening measures in this mainly aged high risk population. The authors would like to conclude that, smoking is correlated with higher prevalence of obesity and increased BMI among women and the youth, making to smoke to stay slim a false belief in these fractions of Iranian population. Therefore, we recommend targeted educational interventions at women and young adults, with the aim of informing them that if they intend to lose weight or stay slim, smoking is not a safe option. Female smokers should also be assured that they will not gain more weight if they try to quit smoking.

**REFERENCES**

Alberti, K.G., Zimmet, P., Shaw, J., 2005. The metabolic syndrome a new worldwide definition. Lancet. 366, 1059-1062.

Aubin, H.J., Farley, A., Lycett, D., Lahmek, P., Aveyard, P., 2012. Weight gain in smokers after quitting cigarettes: Meta-analysis. BMJ. 345, 4439.

Barrett-Connor, E., Khaw, K.T., 1989. Cigarette smoking and increased central adiposity. Ann. Intern. Med. 111, 783-787.

Bush, T., Hsu, C., Levine, M.D., Magnusson, B., Miles, L., 2014. Weight gain and smoking: Perceptions and experiences of obese quitline participants. BMC Public Health. 14, 1229.

Caks, T., Kos, M., 2009. Body shape, body size and cigarette smoking relationships. Int. J. Public Health. 54, 35-39.

Chiolero, A., Faeh, D., Paccaud, F., Cornuz, J., 2008. Consequences of smoking for body weight, body fat distribution, and insulin resistance. Am. J. Clin. Nutr. 87, 801-809.

Clair, C., Chiolero, A., Faeh, D., Cornuz, J., Marques-Vidal, P., Paccaud, F., Mooser, V., Waeber, G., Vollenweider, P., 2011. Dose-dependent positive association between cigarette smoking, abdominal obesity and body fat: Cross-sectional data from a

population-based survey. BMC Public Health. 11, 23.
Clair, C., Rigotti, N.A., Porneala, B., Fox, C.S., D'Agostino, R.B., Pencina, M.J., Meigs, J.B., 2013. Association of smoking cessation and weight change with cardiovascular disease among adults with and without diabetes. JAMA. 309, 1014-1021.
Cooper, T.V., Klesges, R.C., Robinson, L.A., Zbikowski, S.M., 2003. A prospective evaluation of the relationships between smoking dosage and body mass index in an adolescent, biracial cohort. Add. Behav. 28, 501-512.
de Oliveira Fontes Gasperin, L., Neuberger, M., Tichy, A., Moshammer, H., 2014. Cross-sectional association between cigarette smoking and abdominal obesity among Austrian bank employees. BMJ. 4, 004899.
Dhaliwal, S.S., Welborn, T.A., 2009. Central obesity and multivariable cardiovascular risk as assessed by the Framingham prediction scores. Am. J. Cardio. 103, 1403-1407.
Esteghamati, A., Abbasi, M., Rashidi, A., Meysamie, A., Khalilzadeh, O., Haghazali, M., Asgari, F., Nakhjavani, M., 2009a. Optimal waist circumference cut-offs for the diagnosis of metabolic syndrome in Iranian adults: Results of the third national survey of risk factors of non-communicable diseases (SuRFNCD-2007). Diabetic Med. 26, 745-746.
Esteghamati, A., Khalilzadeh, O., Mohammad, K., Meysamie, A., Rashidi, A., Kamgar, M., Abbasi, M., Asgari, F., Haghazali, M., 2010. Secular trends of obesity in Iran between 1999 and 2007: National surveys of risk factors of non-communicable diseases. Metab. Syndr. Relat. Disord. 8, 209-213.
Esteghamati, A., Meysamie, A., Khalilzadeh, O., Rashidi, A., Haghazali, M., Asgari, F., Kamgar, M., Gouya, M.M., Abbasi, M., 2009b. Third national surveillance of risk factors of non-communicable diseases (SuRFNCD-2007) in Iran: Methods and results on prevalence of diabetes, hypertension, obesity, central obesity, and dyslipidemia. BMC Public Health. 9, 167.
Expert panel on detection, evaluation, and treatment of high blood cholesterol in Aadults, 2001. Executive summary of the third report of the national cholesterol education program (ncep) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA. 285, 2486-2497.
Ferrara, C.M., Kumar, M., Nicklas, B., McCrone, S., Goldberg, A.P., 2001. Weight gain and adipose tissue metabolism after smoking cessation in women. Int. J. Obes. Relat. Metab. Disord. 25, 1322-1326.
Filozof, C., Fernandez Pinilla, M.C., Fernandez-Cruz, A., 2004. Smoking cessation and weight gain. Obesity Reviews. 5, 95-103.
Hofstetter, A., Schutz, Y., Jequier, E., Wahren, J., 1986. Increased 24-hour energy expenditure in cigarette smokers. Eng. J. Med. 314, 79-82.
Hong, T., Johnson, C., 2013. A longitudinal analysis of adolescent smoking: Using smoking status to differentiate the influence of body weight measures. J. Sch. Health. 83, 314-321.
Honjo, K., Iso, H., Tsugane, S., Tamakoshi, A., Satoh, H., Tajima, K., Suzuki, T., Sobue, T., 2010. The effects of smoking and smoking cessation on mortality from cardiovascular disease among Japanese: Pooled analysis of three large-scale cohort studies in Japan. Tobacco Control. 19, 50-57.
Honjo, K., Siegel, M., 2003. Perceived importance of being thin and smoking initiation among young girls. Tobacco Control. 12, 289-295.
Hussain, T., Al-Daghri, N.M., Al-Attas, O.S., Draz, H.M., Abd Al-Rahman, S.H., Yakout, S.M., 2012. Plasma neuropeptide Y levels relate cigarette smoking and smoking cessation to body weight regulation. Regulatory Peptides. 176, 22-27.
Inoue, K., Takeshima, F., Kadota, K., Yoda, A., Tatsuta, Y., Nagaura, Y., Yoshioka, S., Nakamichi, S., Nakao, K., Ozono, Y., 2011. Early effects of smoking cessation and weight gain on plasma adiponectin levels and insulin resistance. Intern. Med. 50, 707-712.
Jang, S.Y., Kim, J.H., Lim, M.K., Kim, H.J., Jee, S.H., Namkoong, K., Cho, W.H., Park, E.C., Lee, S.G., 2012. Relationship between BMI, body image, and smoking in Korean women as determined by urine cotinine: Results of a nationwide survey. Asian Pac. J. Cancer Prev. 13, 1003-1010.
Janzon, E., Hedblad, B., Berglund, G., Engstrom, G., 2004. Changes in blood pressure and body weight following smoking cessation in women. J. Intern. Med. 255, 266-272.
John, U., Hanke, M., Rumpf, H.J., Thyrian, J.R., 2005. Smoking status, cigarettes per day, and their relationship to overweight and obesity among former and current smokers in a national adult general population sample. Int. J. Obes. 29, 1289-1294.
Kasteridis, P., Yen, S.T., 2012. Smoking cessation and body weight: Evidence from the Behavioral Risk Factor Surveillance Survey. Health Serv. Res. 47, 1580-1602.
Kim, J.H., Shim, K.W., Yoon, Y.S., Lee, S.Y., Kim, S.S., Oh, S.W., 2012. Cigarette smoking increases abdominal and visceral obesity but not overall fatness: An observational study. PloS One. 7, 45815.
Klesges, R.C., Winders, S.E., Meyers, A.W., Eck, L.H., Ward, K.D., Hultquist, C.M., Ray, J.W., Shadish, W.R., 1997. How much weight gain occurs following smoking cessation? A comparison of weight gain using both continuous and point prevalence abstinence. J. Consult. Clin. Psychol. 65, 286-291.
Komiyama, M., Wada, H., Ura, S., Yamakage, H., Satoh-Asahara, N., Shimada, S., Akao, M., Koyama, H., Kono, K., Shimatsu , A., Takahashi, Y., Hasegawa, K., 2015. The effects of weight gain after smoking cessation on atherogenic alpha1-antitrypsin-low-density lipoprotein. Heart Vessels. 30, 734-739.
Landrau-Cribbs, E., Cabriales, J.A., Cooper, T.V., 2015. General and smoking cessation weight concern in a Hispanic sample of light and intermittent smokers. Addictive Behaviors. 41, 29-33.
Lee, K., Lee, C.M., Kwon, H.T., Oh, S.W., Choi, H., Park, J.H., Cho, B., 2012. Associations of smoking and smoking cessation with CT-measured visceral obesity in 4656 Korean men. Prev. Med. 55, 183-187.
Locatelli, I., Collet, T.H., Clair, C., Rodondi, N., Cornuz, J., 2014. The joint influence of gender and amount of smoking on weight gain one year after smoking cessation. International journal of environmental research and public health. 11, 8443-8455.
Mackay, D.F., Gray, L., Pell, J.P., 2013. Impact of smoking and smoking cessation on overweight and obesity: Scotland-wide,

cross-sectional study on 40, 036 participants. BMC Public Health. 13, 348.
Manson, J.E., Ajani, U.A., Liu, S., Nathan, D.M., Hennekens, C.H., 2000. A prospective study of cigarette smoking and the incidence of diabetes mellitus among US male physicians. Am. J. Med. 109, 538-542.
Matsushita, Y., Nakagawa, T., Yamamoto, S., Takahashi, Y., Noda, M., Mizoue, T., 2011. Associations of smoking cessation with visceral fat area and prevalence of metabolic syndrome in men: The Hitachi health study. Obesity. 19, 647-651.
Mendelson, J.H., Goletiani, N., Sholar, M.B., Siegel, A.J., Mello, N.K., 2008. Effects of smoking successive low and high-nicotine cigarettes on hypothalamic-pituitary-adrenal axis hormones and mood in men. Neuropsychopharmacology. 33, 749-760.
Meysamie, A., Ghaletaki, R., Haghazali, M., Asgari, F., Rashidi, A., Khalilzadeh, O., Esteghamati, A., Abbasi, M., 2010. Pattern of tobacco use among the Iranian adult population: Results of the national Survey of Risk Factors of Non-Communicable Diseases (SuRFNCD-2007). Tobacco Control. 19, 125-128.
Meysamie, A., Ghaletaki, R., Zhand, N., Abbasi, M., 2012. Cigarette smoking in Iran. Iran J. Public Health. 41, 1-14.
Peeters, A., Barendregt, J.J., Willekens, F., Mackenbach, J.P., Al Mamun, A., Bonneux, L., 2003. Obesity in adulthood and its consequences for life expectancy: A life-table analysis. Ann. Intern. Med. 138, 24-32.
Penzes, M., Czegledi, E., Balazs, P., Foley, K.L., 2012. Factors associated with tobacco smoking and the belief about weight control effect of smoking among Hungarian adolescents. Cent. Eur. J. Public. Health. 20, 11-17.
Pisinger, C., Jorgensen, T., 2007. Waist circumference and weight following smoking cessation in a general population: The Inter99 study. Prev. Med. 44, 290-295.
Rabaeus, M., Salen, P., de Lorgeril, M., 2013. Is it smoking or related lifestyle variables that increase metabolic syndrome risk? BMC Medicine. 11, 196.
Saarni, S.E., Pietilainen, K., Kantonen, S., Rissanen, A., Kaprio, J., 2009. Association of smoking in adolescence with abdominal obesity in adulthood: A follow-up study of 5 birth cohorts of Finnish twins. Am. J. Public Health. 99, 348-354.
Stadler, M., Tomann, L., Storka, A., Wolzt, M., Peric, S., Bieglmayer, C., Pacini, G., Dickson, S.L., Brath, H., Bech, P., Prager, R., Korbonits, M., 2014. Effects of smoking cessation on beta-cell function, insulin sensitivity, body weight, and appetite. Eur. J. Endocrinol. 170, 219-227.
Stice, E., Marti, C.N., Rohde, P., Shaw, H., 2015. Young woman smokers gain significantly more weight over 2-year follow-up than non-smokers. How Virginia doesn't slim. Appetite. 85, 155-159.
Sulander, T., Rahkonen, O., Nissinen, A., Uutela, A., 2007. Association of smoking status with obesity and diabetes among elderly people. Archives of Gerontology and Geriatrics. 45, 159-167.
Travier, N., Agudo, A., May, A.M., Gonzalez, C., Luan, J., Wareham, N.J., Bueno-de-Mesquita, H.B., van den Berg, S.W., Slimani, N., Rinaldi, S., Clavel-Chapelon, F., Boutron-Ruault, M.C., Palli, D., Sieri, S., Mattiello, A., Tumino, R., Vineis, P., Norat, T., Romaguera, D., Rodriguez, L., Sanchez, M.J., Dorronsoro, M., Barricarte, A., Huerta, J.M., Key, T.J., Orfanos, P., Naska, A., Trichopoulou, A., Rohrmann, S., Kaaks, R., Bergmann, M.M., Boeing, H., Hallmans, G., Johansson, I., Manjer, J., Lindkvist, B., Jakobsen, M.U., Overvad, K., Tjonneland, A., Halkjaer, J., Lund, E., Braaten, T., Odysseos, A., Riboli, E., Peeters, P.H., 2012. Longitudinal changes in weight in relation to smoking cessation in participants of the EPIC-PANACEA study. Prev. Med. 54, 183-192.
Veldheer, S., Yingst, J., Foulds, G., Hrabovsky, S., Berg, A., Sciamanna, C., Foulds, J., 2014. Once bitten, twice shy: Concern about gaining weight after smoking cessation and its association with seeking treatment. Int. J. Clin. Practice. 68, 388-395.
White, M.A., 2012. Smoking for weight control and its associations with eating disorder symptomatology. Comp. Psychiatry 53, 403-407.

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# Evaluation of the relation between reasons for initiating smoking and dependence level

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**ABSTRACT**

The aim was to evaluate the dependence level and relation condition by examining the reasons to start smoking. Scanning the people in Family Health Center, personal information, smoking conditions and reasons to start smoking were asked to 359 smokers over 18 years of age using a questionnaire prepared. Fagerström Test for Nicotine Dependence (FTND) was used to determine the dependence level in smoking individuals. Then statistical analysis was made using the acquired data. 269 people, 181 males (50.4%) and 178 females (49.6%), were included in our study. While the package/year average of smokers is 9.0±8.4 right now, FTND score average was found 6.9±1.7 points. According to FTND, the ratio of low level, medium level and high level of dependents were 6.7%, 40.4% and 52.9% in order. Reasons for starting to smoke were grouped under 11 different answers. The cases stated the most common reason for starting smoking as "imitating" (21.2%) and boredom/stress (13.6%) followed this. There was a significant relation between the ages and reasons for starting smoking among the participants in our study ($F=4.067$, $p<0.001$). A statistically significant difference was not found in smoking dependence levels among FTND scores and smoking package/year condition. No significant relation was found between nicotine dependence level and reason to start smoking. "Imitation" as the most common reason to start smoking. Thus applications increasing the information level of individuals may decrease the smoking starting frequency of individuals.



## 1. Introduction

Tobacco which is the most important reason for preventable causes of death kills nearly half of its users. More than one billion of people on the world which constitutes 1/4 of adult population, use tobacco products and tobacco use causes the death of more than 5 million people every year (Mathers and Loncar, 2006). On the other hand, tobacco use in developing countries increases day by day due to the increase in population and aggressive marketing efforts of tobacco industry (Murray and Anthonisen, 1999; Atılgan et al., 2008).

The increase in consumption and production of cigarettes and other tobacco products in the whole world causes a very severe burden on people and national health system. Smoking is a serious addiction and quitting smoking is an extremely difficult process. While struggling against smoking, three strategies gain importance. The first of these is the smoking cessation of smokers, the second and third are preserving the condition for those who quitted and never started smoking. Yearly smoking cessation ratios even in

the presence of pharmacological approach together with professional help in smoking individuals are rarely above 25% (Ozturk et al., 2015). The most promising fact for future when these three strategies are considered is unchanging of non-smokers' condition. Thus the precautions required for individuals not to start smoking can be taken only by understanding how smoking is started. So the reasons for starting smoking were examined in individuals registered to a family health center in our city were investigated in our study for this reason. Also whether there was a relation between the dependence degrees and smoking starting reason was investigated in our study.

## 2. Material and method

This observational, cross-sectional and analytic study was made on the population registered in Family Health Center (FHC) between 28.03.2016 and 15.04.2016. 369 current smokers who are over 18 years of age, registered in FHC, have no psychiatric or other chronic diseases known participated voluntarily in the study after taking informed consent. No one rejected to participate in the study. Some demographical characteristics (age, gender, marital status, socioeconomic and education level, etc), smoking information [Fagerstrom Test for Nicotine Dependence (FNDT) and package/year] of these people were questioned. After a questionnaire was given to every individual to question their reasons for starting to smoke. Then acquired information was statistically analyzed.

### 2.1. Fagerstrom Test for Nicotine Dependence

This test is a questionnaire used to determine the degree of smoking addiction commonly in the world. It was developed by Karl O. Fagerström in order to determine the level of physical smoking addiction and contains six questions (Fagerström et al., 1996). The patient can both be applied face to face and can be filled individually. A score between 1 and 10 can be taken from the questionnaire and the dependence level is considered to increase as the amount of the score taken increases. Those who take a score below 5 points from the test are defined as low nicotine addicts, and those who take 7 points and above are defined as severe nicotine addicts. In the adaptation made for our country, test and retest correlations were found 0.85 and 0.88 in order (Fidancı et al., 2015). A standardization study is available for the Turkish sampling (Uysal et al., 2004) and this scale was applied only to smoking participants (Şenyüz and Coştur, 2010).

### 2.2. Statistical analyses

The acquired data were examined and evaluated using SPSS 16.0 statistics program. Characteristics of the study group were presented with definitive type of analyses (number, percentage, average and standard deviation). Data were evaluated using mutual independent group comparisons Mann-Whitney-U groups test and Pearson chi-square and Kruskall Wallis test analysis methods. Statistical significance level 'p' value was accepted as those below 0.05.

### 2.3. Ethic board

Ethic board consent for this study was taken from Turkish Republic of Health Clinical Studies Ethic Board.

## 3. Results

### 3.1. Demographic features

The demographic and smoking features of the study group are given in Table 1. 269 people, 181 males (50.4%) and 178 females (49.6%), were included in our study. The average age of the participants was found 52.6±12.2 years. There was no significant difference between the average ages of both genders. In the classification according to the educational status of the participants, 21 people (5.8%) stated that they were literate, 32 (8.9%) were elementary school, 156

**Table 1.** The demographic and smoking features of the participants according to their gender

| | Male | Female | |
|---|---|---|---|
| Age (years) (mean) | 52.95±12.63 | 52.34±11.83 | t=0.470, p=0.636 |
| Occupation | Not occupied 14 (7,7%)<br>Farmer 33 (18.2%)<br>Worker 29 (16.0%)<br>Retired 68 (37.6%)<br>State worker 35 (19.3%)<br>Student 2 (1.1%) | Not occupied 15 (8.4%)<br>Farmer 16 (9.0%)<br>Worker 23 (12.9%)<br>Retired 49 (27.5%)<br>State worker 25 (14.0%)<br>Student 3 (1.7%)<br>Housewife 47 (26.4%) | $x^2$=1.345 p<0.05 |
| Education level | Unschooled 10 (5.5%)<br>Elementary 15 (8.3%)<br>Secondary 49 (27.1%)<br>High school 94 (51.9%)<br>University 13 (7.2%) | Unschooled 11 (6.2%)<br>Elementary 17 (9.6%)<br>Secondary 83 (46.6%)<br>High school 62 (34.8%)<br>University 5 (2.8%) | $x^2$=2.307<br>p<0.001 |
| FNBT scores (mean) | 7.07±1.7 | 6.74±1.7 | t=1.804 p=0.072 |
| Package/year (mean) | 8.57±8.1 | 9.6±8.6 | t=1.157, p=0.248 |
| Years of smoking (Mean) | 9.5±8.6 | 10.46±9.3 | t=1.008, p=0.314 |

were (43.5%) high school and 18 (5.0%) were college graduates. Most of the participants in the study were married [64 (17.8%) were single and 295 (82.2%) were married]. When occupational groups are considered, 49 people (13.6%) were workers, 52 (14.5%) were government employees, 60 (16.7%) were freelancers, 5 (1.4%) were students, 47 (13.1%) were housewives and 117 (32.6%) were retired. 29 (8.1%) people stated that they were not working.

### 3.2. Smoking characteristics

Average FTND scores of the cases participating in the study were calculated as 6.90±1.740. There was no difference between FTND test score averages of males and females (t=1.804, p=0.072). Package/year value of the participants was calculated as 9.08±8.4. There was no difference between package/year averages of males and females (t=1.157, p=0.248). No correlation was detected between the ages and both total smoking duration (r=0.053, p=0.314), package/years (r=0.029, p=0.581) and FNTD scores of the cases participating in the study (r=0.082, p=0.122) A strong correlation was detected only between total smoking duration and package/year of the patients (r=0.850, p<0.001)

### 3.3. Reasons for starting to smoke

Reasons for starting to smoke grouped according to the answers given by the participants are given in Table 2. According to this, no statistical difference was found among the genders when reasons for starting to smoke were considered ($x^2$=9.903, p=0.272). When the reasons for smoking and smoking durations, FNTD scores (p=0.301) and package/year values (p=0.245) of the cases participating in the study are compared, no statistical relation was detected. But there was a significant relation between the ages and reasons for starting among the participants in our study (F=4.067, p<0.001). According to this, average ages of the people who stated that they were influenced by their friends (44.7±1.5 years) and movies or television (48.3±1.6 years) were 12 years less in average than those who were curious 56.9±1.2) or influenced by their families (54.89±1.8).

## 4. Discussion

The period when smoking trials are most common is between 16-19 years of age which is called adolescence. All factors affecting the life of the adolescent (physical, social and psycho-social factors) affect the individual. Especially with the effect of environmental circumstances, most adolescents are accepted as the group under risk when dangerous behaviors and habits are considered. It is quite common for the adolescents who cannot cope with the problems they face to use substances as a way to cope. Peer influence and impulse control problem are among the reasons for use and nearly 1/4 of smokers smoked their first cigarette before they reached 10 years of age. Especially in different studies abroad, the smoking trial frequency among high school students was reported as 70.4% and 78% (Warren et al., 2008). In studies made on participants less than 18 years of age in different cities of Turkey, the smoking frequency interval was found to be larger (9.5%-41.2%) (Dogan and Ulukol, 2010). Similar to other recent studies made in Turkey (Mayda et al., 2007), when smoking starting age is considered, it took place in late adolescence period (16-19 years of age) in our study, too.

Adolescence is accepted as a period in which people don't hesitate from taking risks. Adolescents think that risks which may take place in the future are too far away and would never affect them. When they don't care about health problems which may take place in their future lives, it is more common for them to start this risky behavior. Smoking starting age was found to be in adolescence in our study, too and the answer given for the reason was "imitation" which supports the studies available in literature (Akgül and Kutluk, 2015).

It is known that smoking behavior generally starts during the period up to the end of adolescence and although more rarely, older individuals may also start this behavior. Effective factors in starting to smoke are accepted as socio-demographical factors, socio-economical condition, personal characteristics, affect of family and friends and reachability of tobacco products (WHO, 2010; Aslan and Aşut, 2015).

**Table 2.** Comparison of most common reasons to start smoking according to gender

| Reasons | Male | | Female | | Total | |
|---|---|---|---|---|---|---|
| | n | (%) | n | (%) | n | (%) |
| I imitated smoking individuals | 39 | (21.5) | 37 | (20.8) | 76 | (21.2) |
| I started in order to reduce my discomfort or stress | 31 | (17.1) | 18 | (10.1) | 49 | (13.6) |
| I was influenced by my family | 26 | (14.4) | 19 | (10.7) | 45 | (12.5) |
| I was influenced by my friends and social environment | 21 | (11.6) | 21 | (11.8) | 33 | (9.2) |
| I was influenced by the characters in movies or television | 19 | (10.5) | 24 | (13.5) | 43 | (12) |
| I started to prove that ı grew up | 17 | (9.4) | 20 | (11.2) | 37 | (10.3) |
| I was curious | 14 | (7.7) | 13 | (7.3) | 27 | (7.5) |
| I started to influence opposite sex | 12 | (6.6) | 25 | (14.0) | 37 | (10.3) |
| Other | 2 | (1.1) | 1 | (6) | 3 | (0.8) |

In a study made by Mayda and friends (Mayda et al., 2007) on the students of medical faculty, the reasons for starting to smoke were stated as friend effect (54.4%), imitation (28.0%), curiosity (28.8%) and loneliness (20.6%) (Aslan and Aşut, 2015). In our study, "friend effect" was lower among the reasons (9.2%), imitation was the first (21.2%) and the reason "I was influenced by my family" (12.5%) was the second.

As a result, in addition to the effect of many reasons, it shouldn't be forgotten that personal and environmental characteristics are also important in addition to many reasons and its relation with nicotine dependence level is not clear. It should be remembered that the group which should be given highest importance is adolescent age group and education, seminary and similar activities should be increased to reach and inform young individuals.

**REFERENCES**

Akgül, S., Kutluk, T., 2015. Çocuk ve Ergenlerde Tütün Kontrolü. STED. 24, 1-5.

Aslan, D., Aşut, Ö., 2015. Dünyada Kadınlar Arasında Tütün Kullanımı ve Mücadelede Ayrıntılar. STED. 24, 6-9.

Atılgan, Y., Gürkan, S., Şen, E., 2008. Hastanemizde çalışan personelin sigara içme durumu ve Etkileyen Faktörler. Tur. Toraks. Der. 9, 160-166.

Bilir, N., Doğan, B.G., Yıldız, A.N., Emri, S., Kalyoncu, A.F., 1979. Smoking behaviour and attitudes, Hacettepe Public Health Foundation. 14, 1-5.

Doğan, D.G., Ulukol, B., 2010. Ergenlerin sigara içmesini etkileyen faktörler ve sigara karşıtı iki eğitim modelinin etkinliği. İnönü Üniversitesi Tıp Fakültesi Der. 17, 179-185.

Fagerström, K.O., Kunze, M., Schoberberger, R., Breslau, N., Hughes, J.R., Hurt, R.D., Puska, P., Ramstrom, L., Zatonski, W., 1996. Nicotine dependence versus smoking prevalence: comparisons among countries and categories of smokers. Tobacco Control, 5, 52-56.

Fidancı, İ., Tekin, O., Arslan, İ., Eren, Ş.Ü., Dinçer, G., Kızıltaş, Ö., 2015. Sigara alışkanlığının HbA1c üzerine etkisinin değerlendirilmesi. Ankara Med. J. 15, 127-133.

Mathers, C.D., Loncar, D., 2006. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Medicine, 3, e442.

Mayda, AS., Tufan, N., Baştaş, S., 2007. Düzce Tıp Fakültesi öğrencilerinin sigara konusundaki tutumları ve içme sıklıkları. Kor Hek. 6, 364-370.

Murray, R.P., Anthonisen, NR., 1999. Interventions for smoking cessation; the outcomes are determined by the questions asked. Eur Respir J. 13, 231-232.

Ozturk, O., Ozturk, G., Yazicioglu, B., Yalcin, BM., Unal, M. 2015. Smoking frequency, cessation knowledge; attitudes and beliefs among internal and surgery residents. J. Exp. Clin. Med. 32, 171-175.

Şenyüz, L., Coştur, R., 2010. Sigara kullanan ve kullanmayan üniversite öğrencisi genç yetişkinlerin sigara paketlerinde yer alan uyarı yazılarıyla ilgili değerlendirmeleri. Journal of Faculty of Letters. 27, 253-270.

Uysal, M.A., Kadakal, F., Karida, C., Bayram, NG., Uysal, Ö., Yılmaz, V., 2004. Fagerstrom test for nicotine dependence: Reliability in a Turkish sample and factor analysis. Tüberküloz ve Toraks. 52, 115-121.

Warren, C.W., Jones, N.R., Peruga, A., Chauvin, J., Baptiste, J-P., Costa, dSV., 2008. Global youth tobacco surveillance, 2000-2007. Morbidity and mortality weekly report Surveillance summaries (Washington, DC: 2002). 57, 1-28.

World Health Organization (WHO): Tobacco use and its impacts on health. 2010. [http://www.who.int/tobacco/publications/gender/en_tfi_gender_women_prevalence_tobacco_use.pdf?ua=1] (Accessed March 2016).

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# The carbon monoxide measurements' effect on smokers to give cessation decision in primary care

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**ABSTRACT**

The aim of this study was to investigate the levels of ExpCO and its effect on giving decision to stop smoking in primary care. The study was held in Tekkeköy Family Health Center. A total of 853 current smokers (391 in study group and 462 in control group) included into the study. Both group of smokers got first two steps of "5 A" method (Ask, Advice, Assist, Assess and Arrange) for smoking cessation however the ExpCO levels were measured only in the study group. CO levels in expiratory air were measured by the single breath method using a calibrated carbon monoxide monitor and the time of the last smoked cigarette (TLC) of the participants were examined. The mean ExpCO level was 16.9±7.7 ppm in study group. The mean Fagerstrom Nicotine Dependency Test (FNDT) score was 5.0±2.7 in both groups. The mean package/year value in the both participants were 16.0±14.8 (min=1, max=100) and the mean Fagerstrom Nicotine Dependence Test (FNDT) score was 5.0±2.7. The mean ExpCO level was 15.7±8.7 ppm in study group. A significant positive correlation was determined between FTND score and ExpCO. TLC values in males were significantly lower than females ($p<0.001$) in both groups. In a linear regression model it was seen that age, package/year value, FNTD scores and TLC are independent risk factors for elevation of ExpCO values. 23 (5%) smokers in the control group and 47 (12%) in the study group decided to quit smoking ($x^2=15.412$, $p<0.001$). This measurement might have an effect on motivating smokers to quit smoking.



## 1. Introduction

According to World Health Organization (WHO), smoking is the leading preventable cardiovascular risk factor for mortality (WHO, 2008). Huge efforts are spent to decrease the ratio of smokers in developed countries after its negative effect on human health is well understood. In the last two decade in spite of its established detrimental effects, widespread use of smoking is decreased at western countries. However today tobacco industry aims to increase the smoking rates in the developing countries which have limited resources for health care (Pineros et al., 2016). The most recent descriptive overview by WHO estimates that 29% of adults in Europe is smoking (Alwan, 2011).

Most of the case many primary care physicians prefer to use Fagerstrom Nicotine Dependence Test Scale (FNDT) in their daily practice in order to determine the dependency level of the smokers (Fagerstrom et al., 1996). This test is self-administered, and depends on smoker's subjective claims. In dealing with cessation therapies with smokers sometimes it is a necessity to determine objectively the level

of cigarette smoke that the smoker is exposed. This is true in cases in order to control the claims of the smoker or to identify other environmental risk factor cessation and relapse. It is well known than there are at least 3984 chemical substances can be identified in cigarette smoke which the most ones can be identified are nicotine, carbon monoxide (CO) and tar (Margolis et al., 2016). For objective screen in order to determine whether the individual is currently exposing to cigarette smoke (active or passive) several tests including detecting metabolites of nicotine (Cotinine) and tar in blood or urine and being used widely in practice. The disadvantages of these tests can be identified as they may be invasive, expensive, requiring special kits and time consuming. Because of this reasons evaluation of CO levels (ExpCO) in expiratory air is becoming very popular in smoking cessation clinics. Although CO exposure by occupational and environmental pollution the ExpCO levels may increase in normal individuals, this measurement reflects the exposure of cigarette smoke in term of smoking addiction and second hand exposure (Ripoll et al., 2012; Lindson-Hawley et al., 2016). The measurements one or two hours after a cigarette gives the best results with a high rate of reliability (Okutan, 2007). CO levels in expiratory air (ExpCO) is an effective and easy and actual indication of consumption and addiction (Middleton and Morice, 2000). Besides its reliability of reflecting the exposed amount of cigarette smoke there are several advantages of this method. First of all this technique is very cheap and simple to perform which requires little technical knowledge and skill. The other advantage of this method is results taken immediately (Middleton and Morice, 2000). Measuring ExpCO levels in primary care units may have other advantages. While using 5 A (Ask, Advise, Assist, Assess, Arrange) method for their smoker population primary care physicians use several feedback and motivation techniques on their patients (Quinn et al., 2009). These interventions are based on the hypothesis that one of the reasons why people continue smoking, in spite of knowing the harmful effects of tobacco, is that they underestimate the personal risk of becoming ill because of it. In this sense, the measurements in currents smokers interventions will offer motivational feedback to promote awareness of the risk (Weinberg et al., 1981). It has been suggested that some smokers who manage to quit smoking are more aware of the adverse effects of tobacco or to have had their health seriously compromised (Mclure, 2001). For this reason CO measurements may help primary care physicians to motivate their smoker patients with the damage of smoking to their health in the first hand.

Although there are many advantages in smoking cessation there are limited data about the effects of ExpoCO measurements in primary care in our country. In our study we aimed to investigate the mean values of ExpCO and its relation between several factors just like the time of the last cigarette (TLC) and the addiction levels of the smoking (FNDT) in participants. The other aim of our study is to investigate the effect of the ExpCO measurements on giving cessation decision on the smokers in primary care.

## 2. Matherial and method

### 2.1. Study design

This is a randomized controlled study. The study is held at the Tekkeköy Family Health Center between 1st August to 1 September 2015. Before the study the researcher (HP) at Tekkeköy Family Health Center was trained about the principles and skills of using an ExpCO device in a two hours clinical skills workshop. After his training he practiced the device with performing it to at least ten test subjects in supervision of their instructor (BMY). The smokers who had applied to Tekkeköy in the study period were included into the study. A total of 853 participants included into the study. After their informant constant were taken the smokers were divided into two groups with a basic random pattern (First smoker to the study group, second to the control etc.) as the smokers who get just 5 "A" approach (control group) while the other group get 5 "A" approach after measuring their ExpCO measurements are performed. The researcher gave advice the participants in the both groups to stop smoking. He also gave information about Smoking Cessation Clinic of Ondokuz Mayis University Department of Family Practice and referred the affirmative smokers. Overall there was 391 participants in the study group and 462 in control group. The volunteered smoker participants who were over 18 years, had been smoking until a year, with no condition/disease effecting his/her cognitive abilities (depression, stroke etc.), patients without lung diseases (Tuberculosis, Chronic Obstructive Lung Disease etc) were included into the study. The individuals who may be exposed to CO with occupational risk (Welders etc) were also omitted from the study (A total of 25 individuals). FNDT was applied to determine dependence level of the smokers with face-to-face interviews to all of the participants. CO levels in expiratory air were measured by the single breath method using a calibrated carbon monoxide monitor (PICO+ Smokerlyzer, Bedfont Scientific Ltd. UK). The subjects were instructed to take a deep breath, hold their breath, and exhale fully into the mouthpiece of the detector. The time of the last smoked cigarette (TLC) of the participants were examined in the study group.

### 2.2. Statistical analyses

The acquired data were examined and evaluated using SPSS 16.0 statistics program. Characteristics of the study group were presented with definitive type of analyses (number, percentage, average and

standard deviation). Data were evaluated using mutual independent group comparisons Mann-Whitney-U groups test and Pearson chi-square and Kruskall Wallis test analysis methods. The quantitative variables were presented as mean ± standard deviation (SD), and the categorical variables with figures and percentages (%). The chi-square test and multivariable logistic regression analysis were used in the assessment of data. The Odds Ratios (OR) from the regression analysis were presented with 95% confidence intervals (CI). A p value of $p<0.05$ was considered to be significant.

## 3. Results

The sociodemographic and smoking features of the both groups are presented at Table 1. The A total of 462 people participated in the control group and 391 in the study group. Mean age of the participants in both groups were 36.2±12.9 year, with age range of 17 to 82 years old. There was no difference between the mean of age between both genders (Men: 33.12±14.1, women=32.88±11.7) (t=0.189, p=0.850) in the study group. The mean package/year value in the both participants were 16.0±14.8 (min=1, max=100). There was a statistically significant difference between package/year values of in different genders (Men=17.56±17.1, women=14.57±14.7) (t=2.563, p=0.011) in the study group. The mean FTND score was 5.0±2.7 in all of the participants. There was no difference between the mean FTND scores between different genders (t=1.116, p=0.265) in study group. There was a correlation between the FNDT scores and package/ year values (r=0.398, p<0.001) in both groups.

The mean ExpCO level was 16.9±7.7 ppm in study group. The mean ExpCO values were significantly higher in men (18.07±8.2) compared with women (13.9±8.9) (t=3.785, p<0.001). A significant positive correlation was determined between FTND score and ExpCO (Men equals to r=0.402, p<0.001; women equals to r=0.484, p<0.001). TLC values in men were significantly lower than women (t=3.428, p<0.001). A mid-level negative correlation was detected between ExpCO and TLC for both sexes man (r=-0.507, p<0.001); Female (r=-0.612, p<0.001). In a linear regression model the factors which might have effect on ExpCO value is investigated. This model has a value of 0.315 value of 0.315 and Durbin Watson value as 1.630. It was seen that age, package/year value, FNTD scores and TLC are independent risk factors for elevation of ExpCO values. The model is shown at Table 2.

23 (5%) smokers in the control group (12 women 50%) and 47 (12%) in the study group (15 women 31.9%) decided to quit smoking ($x^2$=15.412, p<0.001) and applied to Ondokuz Mayis University Family Medicine Smoking Cessation Clinic.

## 4. Discussion

Our study is designed as a pilot study and investigated the mean ExpCO levels in smokers and the factors effecting its measurements. Our measurements revealed that the mean ExpCO level is s 15.7±8.7 ppm among in our participants. In some international studies it is documented that the mean of ExpCO levels in smokers varies between 9.5-21.6 ppm (Zayasu et al., 1997; Low et al., 2004; Chatkin et al., 2007) and 1.3-4.3 ppm in nonsmokers. Some researchers suggested the cutoff ExpCO levels as 6 ppm and 6.5 ppm in smokers and non-smokers respectively (Middleton and Morice, 2000; Doruk et al., 2012). However there are some

**Table 1.** Comparison of sociodemographic and smoking features of all the smokers in both groups

| Variables | Control (n=462) | Study (n=391) | p |
|---|---|---|---|
| Gender | Women 159, 37.8%<br>Men 303, 62.2% | 149, 35.7%<br>242, 64.3% | $x^2$=1.005<br>P=0.451 |
| Age (mean) | 35.50±13.66 | 36.22±11.6 | t=0.54<br>p=0.562 |
| Mean time spent in education (years) | 14.40±1.17 | 14.78±3.3 | |
| Occupation<br>Housewife<br>Student<br>Farmer<br>Manual laborer<br>White collar (teacher etc.)<br>Private (small trader etc.) | <br>110, 23, 8%<br>28, 6%<br>112, 24.2%<br>124, 26.8%<br>41, 8.8%<br>35, 7.5% | <br>108, 27.6%<br>41, 10.4%<br>97, 24.8%<br>96, 24.8%<br>29, 7.4%<br>20, 5.1% | <br><br><br>$x^2$=0.265<br>p=0.658 |
| Age of starting to smoke | 18.00±3.6 | 18.70±9.7 | t=0.058<br>p=0.275 |
| Package/year (mean) | 16.8±1.2 | 17.1±2.2 | t=6.897<br>p=0.107 |
| The mean score of FNDT* | 4.9±1.7 | 5.5±0.8 | t=0.154<br>p=0.241 |
| Number of quit attempts (mean) | 2.1±1.4 | 1.8±1.8 | t=0.874<br>p=0.987 |
| Total number of quit attempts so far* | 137 | 71 | $x^2$=1.215<br>P=0.007 |

**Table 2.** The independent factors for affecting ExpCo measurements

| Mode 1 | | Unstandardized Coefficients | | Standardized Coefficients | | | 95% Confidence Interval for B | |
|---|---|---|---|---|---|---|---|---|
| | | B | Std. error | Beta | t | Sig. | Lower bound | Upper bound |
| 1 | (Constant) | 17.753 | 1.508 | | 11.771 | 0.000 | 14.788 | 20.718 |
| | Package/year | 0.073 | 0.032 | 0.137 | 2.259 | 0.024 | 0.010 | 0.137 |
| | Age | -0.193 | 0.037 | -0.289 | -5.195 | 0.000 | -0.266 | -0.120 |
| | FNDT | 1.107 | 0.151 | 0.340 | 7.334 | 0.000 | 0.810 | 1.404 |
| | TLC | -0.007 | 0.001 | -0.293 | -7.082 | 0.000 | -0.008 | -0.005 |

**ExpCO:** The carbon monoxide in Expiratory; **FNTD:** Fagerstrom Nicotine Dependence Test; **TLC:** The Last Cigarette Time

studies pointing out lower cutoff values as 5 ppm (Low et at., 2004).

One of the important factor that effects the measurement of ExpCO in smokers is found as age in our study. As age is mostly correlated with calculating the package/year values it is not surprising that it is a depended factor for levels of ExpCO. As the time of the smokers using cigarette increases the negative effects of cigarette smoke increases the damage in lungs. The package/year is reflecting the amount of the total cigarette smoke that have been exposed it is not surprising to see that it is an independent risk factor for ExpCO. However it is surprising to see that this relation is not very strong (OR=0.073, 95% CI; 0.01-0.137, p=0.024). This result may be explained that the ExpCO measurement is more related with actual smoke exposure rather than past. This concept is more interesting as the package/year and FNDT values have a correlation in our study FNDT seems to be more important (OR= 1.107, 95% CI; 0.8-1.4. p<0.001) than package/year values. Our results were confirmed with the results of (Low et at., 2004). In their study Low et al. found the mean ExpCO level is 11.6 ppm and it has a positive correlation between FNDT scores. Similar with our study two studies held in Turkey by Deveci et al. (2004) and Temel et al. (2009) found positive correlation ExpCO levels with FNDT scores (Doruk et al., 2012). To analyze objectively the results of the FNDT it is imperative to remember that the two questions is most important ones. First one is about the time of the first cigarette (3 points) and the other the amount of the cigarettes taken in a day (3 points). Especially the time intervals between cigarettes decreases with the number of cigarettes smoked in a day. In order to support this concept Hung et al. (2006) found the mean exhaled CO level of those consuming 1-10 cigarettes a day was significantly lower than the mean exhaled CO level of those consuming >10 cigarettes a day in their study.

The smoker's individual smoking conditions are assessed by ExpCO levels and the time period the last cigarette smoked is the important determinant of ExpCO levels. Because of the half-life of CO being 5 to 6 hours, the last cigarette smoked is affected the ExpCO levels significantly (Peterson and Steward et al., 1970; Crowley et al., 1989). The amount of CO in the expiratory air starts to decline after 3 hours from smoking, measures at that moment naturally come out as low and may not give an exact result In our study it is seen that TLC values in men participants were significantly lower than women participants (p<0.001). A mid-level negative correlation was detected between ExpCO and TLC for both sexes: man (r=-0.434, p<0.001); women (r=-0.535, p<0.001). Parallel to our results, Low et al. (2004) found a significant negative correlation between the time of the last cigarette smoked and levels of ExpCO in their study. They revealed a cigarette which is smoked 5 hours ago might indicate a high level of ExpCO. Terao et al. (1998) showed that the time elapsed since last smoke had effects on the expired air carbon monoxide level. Our study revealed that TLC is an independent factor for ExpCO measurements.

One of the most important finding in our study is the positive effect of performing ExpCO measure on the cessation decision in study population. In the literature the success of different interventions for smoking often measured with cessation rate. In recent approach it has been recommended to the primary care physicians to accept smoking as a chronic disease which requires a life long struggle (Rao and Pilot, 2014). "5 A" method is born from that need. One of the most important factor that this method work is depends on the relationship between smoker and physician. When the physician motivates, encourages, supports and offers unlimited help to their patients it has the best possibility to work. It is known that the just using the first two steps of the "5 A" method app. 5% of the smokers quit smoking (Dorothy et al., 2008). Our study revealed measuring ExpCO in study doubled the cessation decision compared with control group compared with our control group. It can be argued that giving decision doesn't mean cessation of smoking. However it is an important to thing to get the attraction of the smokers tip the balance of their decision in favor of cessation.

This pilot study has limitations. First of all it was conducted in a single Family Health Center which may affect the features of our study universe. The individuals who were enlisted to this center may not represent to other parts of our country. The population of this area is mostly workers and farmers. Although

the decision of smokers about quit smoking is decided upon their approval to our clinic, it no way to investigate the real effect of measuring ExpCO levels in smokers. To stop smoking is complex decision for smokers. As the smoking might be considered as a chronic disease the effect of this measurement might not be isolated. However our study gave us important clues about ExpCO measurements and factors which might have effect on it. Our linear regression model had a R2 value of 0.315 which means that 31% differences in the ExpCO measurements can be explained by this model. As a easy, cheap and effective method increase in ExpCO measurements may have positive effects for smoking cessation activities and follow-up. More information is needed in this topic.

**REFERENCES**


Alwan, A., 2011. Global status report on non-communicable diseases 2010 Geneva: World Health Organization.

Chatkin, J., Fritscher, L., de Abreu, C., Cavalet-Blanco, D., Chatkin, G., Wagner, M., Fritscher, C., 2007. Exhaled carbon monoxide as a marker for evaluating smoking abstinence in a Brazilian population sample. Prim. Care Respir. J. 16, 36-40.

Crowley, T.J., Andrews, A.E., Cheney, J., Zerbe, G., Petty, J.L., 1989. Carbon monoxide assessment of smoking in chronic obstructive pulmonary disease. Addict. Behav. 14, 493-502.

Deveci, S.E., Deveci, F., Acik, Y., Ozan, A.T., 2004. The measurement of exhaled carbon monoxide in healthy smokers and nonsmokers. Respir. Med. 98, 551-556.

Hatsukami, D.K., Stead, L.F., Gupta, P.C., 2008. Tobacco Addiction: Diagnosis and Treatment. Lancet. 14, 2027-2038.

Doruk, S., Demirtaş, İ., Akşit, H., Erkorkmaz, U., Seyfikli, Z., 2012. Assessment of exposure to tobacco smoke: Measurement of exhaled carbon monoxide and hair nicotine. Turk J. Med. Sci. 42, 739-745.

Fagerström, K.O., Kunze, M., Schoberberger, R., Breslau, N., Hughes, J.R., Hurt, R.D., Puska, P., Ramström, L., Zatoríski, W., 1996. Nicotine dependence versus smoking prevalence: Comparisons among countries and categories of smokers. Tobacco Control. 5, 52-56.

Hung, J., Lin C., Wang J., Chan, C., 2006. Exhaled carbon monoxide level as an indicator of cigarette consumption in a workplace cessation program in Taiwan. J. Formos. Med. Assoc. 105, 210-213.

Lindson-Hawley, N., Hartmann-Boyce, J., Fanshawe, T.R., Begh, R., Farley, A., Lancaster, T., 2016. Interventions to reduce harm from continued tobacco use. Cochrane Database Syst. Rev. 13;10.

Low, E.C., Ong, M.C., Tan, M., 2004. Breath carbon monoxide as an indication of smoking habit in the military setting. Singapore Med. J. 45, 578.

Mala, R., and Pilot, E., 2014. The missing link-the role of primary care in global health. Glob. Health Action. 7, 10.

Margolis, K.A., Bernat, J.K., Keely O'Brien, E., Delahanty, J.C., 2016. Online information about harmful tobacco constituents: A Content Analysis. Nicotine Tob. Res. 30. pii: ntw220.

Middleton, E.T., Morice, A.H., 2000. Breathe carbon monoxide as an indication of smoking habit. Chest. 117, 758-763.

McClure, J.B., 2001. Are biomarkers a useful aid in smoking cessation? A review and analysis of the literature. Behav Med. 27, 37-41.

Okutan, O., Taş, D., Kaya, H., Kartaloğlu, Z., 2007. Sigara içen sağlık personelinde nikotin bağımlılık düzeyini etkileyen faktörler (Turkish). Tüberküloz ve Toraks Dergisi. 55, 356-363.

Peterson J.E., Stewart R.D., 1970. Absorption and elimination of carbon monoxide by inactive young men. Arch. Environ. Health. 21, 165-171.

Piñeros. M., Sierra, M.S., Forman, D., 2016. Descriptive epidemiology of lung cancer and current status of tobacco control measures in Central and South America. Cancer Epidemiol. 44 Suppl 1, S90-S99.

Ripoll, J., Girauta, H., Ramos, M., Medina-Bombardó, D., Pastor, A., Alvarez-Ossorio, C., Gorreto, L., Esteva, M., García, E., Uréndez, A., Buades, A., Torres, E., 2012. Clinical trial on the efficacy of exhaled carbon monoxide measurement in smoking cessation in primary health care. BMC Public Health. 12, 322.

Temel, O., Coşkun, A.Ş., Gök, Ş., Celik, P., Yorgancıoğlu, A., 2009. The effect of nicotine among active, passive smoker health personel (Turkish). Tüberküloz ve Toraks Dergisi. 10, 107-111.

Terao, A., Konishi, M., Baba, S., Mannami, T., 1998. Exposure to tobacco smoke in a Japanese urban population. An analysis using biochemical markers of smoking [Japanese]. Nippon Koshu Eisei Zasshi. 45, 3-14.

Quinn, V.P., Hollis, J.F., Smith, S.K., Rigotti, N.A., Solberg, L.I., Hu, W., Stevens, V.J., 2009. Effectiveness of the 5-A's Tobacco Cessation Treatments in Nine HMOsJ. Gen. Intern. Med. 2, 149-154.

Weinberger, M., Green, J.Y., Hamlin, J.J., Jerin, M.J., 1981. Health beliefs and smoking behavior. Am. J. Public Health. 7, 1251-1255.

WHO. 2008. Report On the global tobacco epidemic (MPOWER), WHO, Geneva

Zayasu, K., Sekizawa, K., Okinaga, S., Yamaya, M., Ohrui, T., Sasaki H., 1997. Increased carbon monoxide in exhaled air of asthmatic patients. Am J. Respir. Crit. Care Med. 156, 1140-1143.

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# Results of a selective smoking cessation counseling and prevention course

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**ABSTRACT**

Our aim was to investigate the effect of a selective smoking cessation counseling class on the skills and knowledge of medical students. Sixty medical students from Ondokuz Mayis University attended a selective smoking cessation counseling and prevention class (total 96 hours) at 2011-2012 academic year. After attending an initial 8 weeks of lectures, problem-based sessions, case presentations, patient videos and workshops, the students then assisted with the counseling of real smokers in the remaining 4 weeks, under supervision. Students' knowledge of tobacco dependence, treatment and counseling strategies was scored before (pretest) and after (post-test) the course using a 50-item questionnaire. The students' skills were evaluated in an Objective Structured Clinical Exam (OSCE). A significant difference was determined between the pretest (12.7±7.6) and post-test (35.8±7.8) results ($p<0.001$). The mean OSCE score was 89.2±2.7. The smoking cessation counseling and prevention selective class is highly effective in improving students' cessation counseling skills.



## 1. Introduction

Smoking is the single most important preventable risk factor for global mortality and morbidity from many diseases, from cardiovascular diseases to cancer (Mathers and Loncar, 2006; WHO, 2013). Regardless of their specialty, physicians' responsibilities in the fight against smoking can be grouped under three main goals; to ensure that non-smokers do not start (especially children and teenagers), to help smokers to quit (especially more vulnerable individuals such as pregnant women and children) and lastly to encourage ex-smokers not to relapse (Zwar et al., 2014). However, several studies have shown that many physicians lose motivation and interest in promoting these services (McAvoy et al., 1999; Ellerbeck et al., 2001; Ferguson et al., 2005). A heavy work load and insufficient time, a lack of systems to support cessation services and an absence of financial incentives are some factors that have been investigated to account for this (Rigotti and Thorndike, 2001; Brotons et al., 2005). Many physicians report feeling insufficient confidence in their counseling skills and believe that the most important obstacle to promoting these activities is a lack of adequate training and skills (Conroy et al., 2005; Warner et al., 2013). Despite the impact of smoking on human health, undergraduate medical

education fails to devote proper attention to improving cessation and prevention skills and knowledge (Ferry et al., 1999; Montalto et al., 2004; Richmond, 2009). Although it has been suggested that undergraduate education is the optimal time for skills training in tobacco cessation, most physicians manage to graduate from medical schools with no or only minimal formal training in cessation and prevention (Richmond et al., 2009). Many medical schools around the world still prefer to imbed didactic information regarding smoking and tobacco dependence into the curriculum (Frank et al., 2007). Special modules, tasks or courses concentrating on the subject are rare, and individual cessation or prevention skills training is of low priority in undergraduate or postgraduate medical education (Frank et al., 2007; Richmond et al., 2009, ). Without understanding the importance of the topic, students rarely have an opportunity to provide counselling for real smokers during this period, leaving them untrained and unprepared after graduation (Chatkin and Chatkin, 2009). It is not surprising that there are many calls for urgent changes to under- and postgraduate medical education, which currently fails to respond to major public health problems in many parts of the world (Springer et al., 2008; Ponciano-Rodrigez, 2010).

Promoting the smoking and cessation counselling skills of undergraduate students through specialized courses may pose various advantages. Such courses will not only prepare students for their professional lives, but will also encourage them to focus on this topic at a very early stage. The aim of the class was to provide early clinical contact (Students are evaluated through an objective structured clinical exam with simulated patients) after they have counseled a volunteer real smoker under the supervision of an academic. The aim of this study was to investigate the effect of this class on students' knowledge and skills in the area of smoking cessation. We also analyzed the effectiveness of counseling activities provided for their own social circles by students who had participated in this class.

## 2. Materials and methods

### 2.1.The design of the study

This is a descriptive and analytic study. We designed a pilot selective smoking quit counseling and prevention class lasting 12 weeks (every Wednesday for 8 hours for a total of 96 hours) for 1st year medical students at Ondokuz Mayis University, Turkey, based on current evidence-based medicine (Richmond, 1999; Springer et al., 2008; Fiore et al, 2008; NIH, 2008; Richmond et al., 2009; Lai et al., 2010; Cahill et al., 2013; Stead et al., 2013; Hartman-Boyce et al., 2014) at 2011-2012 academic year. The class content and learning objectives are presented in Appendix 1. The schedule and program of the class is summarized in Appendix 2. In order to achieve the greatest efficiency from the class, the number of students was limited to 60. After the content of the class had been announced, the first 60 volunteers out of 210 1st year students were enrolled. The participants first answered a survey about their demographic data and their own and their family smoking status. All participants described their exposure to second-hand smoke in a five-point Likert type question (5=very often, 4=often, 3=sometimes, 2=occasionally, 1=never), while those students who stated that they had smoked more than 100 cigarettes in their lives to date also took the Fagerstrom Nicotine Dependence Test (FNDT). Students' knowledge of tobacco dependence and treatment and counseling strategies was evaluated before (pretest) the beginning of the class. Students attended the first half of the program (1st to 4th weeks) at the medical faculty and the second half (5th to 12th weeks) at the smoking cessation clinic of the Ondokuz Mayıs University Department of Family Medicine. Once the program had come to an end, students were evaluated through a written exam, OSCE, a clinical interview and a special task.

### 2.2. The smoking cessation counseling and prevention class

The aim of the class was to increase the knowledge and skills of the medical students in order to help smokers quit smoking, to maintain ex-smokers as non-smokers and to prevent non-smokers starting smoking at all. The class consisted of didactic, skills training and applied skills training elements.

### 2.3. Didactic educational activities

The didactic educational activities (active presentations and two different problem-based scenario sessions) lasted for 20 hours. In the problem-based sessions (total 8 hours) students encountered two different scenarios.

### 2.4. Skills training

For skills training, role-plays (three role-play sessions), workshops (three workshops), watching and discussing patients' videos with different counseling techniques (videos of six different real smoker visits), were used for a total 30 hours of education. In role-plays, each student participated by assuming the role of a physician and various smoker roles selected by chance from a range of different scenarios. In the workshops, the students were divided into five separate groups. In the first workshop, we asked them to prepare different patient education handouts, in the second they designed a poster which might be used in primary care settings to motivate smokers to quit, and finally each group was asked to design an imaginary nationwide public anti-smoking campaign. The groups then presented their work to the other groups. The patient videos were selected from different range of real patient interventions.

### 2.5. Applied skills training

Applied skills training was given for a total of 46 hours. Between the 5th and 8th weeks (after most of the learning objectives had been achieved from didactic educational and skills training) the entire group and one academic together counseled different patients every day (planetary sessions) and then discussed them. After the 8th week, each student was appointed to an academic on a one-to-one basis in order to counsel patients. During applied skills training, medical students had an opportunity to experience many different problems (prevention or cessation counseling for smokers or ex-smokers) at first hand with academic counseling.

### 2.6. Evaluation of the class

The students were evaluated in three steps. The first, pretest, was taken before the class began. At the end of the class students were readministered the same test, the post-test. The results of the post-test are taken as written exam scores and were used to evaluate their informative knowledge. The difference between the pre- and post-test results was analyzed in order to investigate the increase in the knowledge of the subject by the end of the class. The tests were scored between 0 and 100, with 2 points given for each correct answer to 50 multiple choice questions. The day after the post-test, students were tested with simulated patients in an Objective Structured Clinical Exam (OSCE). The simulated patients were provided by our university's drama club. Students were tested with standardized scenarios in one-way mirrored rooms. While they were counseling these simulated patients they were themselves being evaluated by researchers with a standardized check list in the adjacent room on the other side of the mirror. Each student had approximately 20 minutes for interviews in the OSCE.

In the third step, students provided counseling sessions for volunteer patients. The students were responsible for applying a standardized approach, described elsewhere, to these patients (Raupach, 2015). They were mainly responsible for taking smoking histories and discussing personalized session plans with patients. Their performance was evaluated by the same supervisor sitting next to them in the same room who had been appointed at the 8th week, using a standardized checklist (the same one as also used in the OSCE). Each student was allowed approximately 20 minutes for interviews. After the patient had left the room, the supervisor provided immediate feedback about students' performances. These patients received another visit with a different clinician after the first visit on the same day. These patients were selected from smokers who were determined to quit smoking and who were on their first visit to our clinic. They were aged over 18 years, were not pregnant and had no known psychiatric diseases or other drug/alcohol addiction.

The total class score was calculated with these three activities. The post-test scores were weighted as 35%, the OSCE as 35%, clinical counselling as 30% of the total score. All the activities were scored between 0 and 100 points, and the pass mark was 75 or above.

### 2.7. Statistical analysis

All analyses were performed on SPSS version 15 (Chicago IL) and Minitab version 15 software. Several parametric and non-parametric analytic techniques, including the Chi-square and Independent samples t-test were used. A p value of $<0.05$ was regarded as significant.

Approval for the study and the class content was granted by the dean of the Ondokuz Mayis University Medical Faculty.

## 3. Results

Students' demographic and smoking characteristics are shown in Table 1. Although there was no difference between the sexes in terms of direct experience of smoking, male students were more exposed to secondhand smoking ($p<0.001$). There was a statistically significant difference between the mean pre- (22.78±7.6) and post-test (44.8±2.1) correct answer scores (50 items) ($t=7.562$, $p<0.001$). Detailed pre- and post-test results showing students' answers to different items are presented in Table 2. Students' knowledge levels had increased in all areas according to the post-test results. Detailed evaluation methods and mean scores from different items from the OSCE and Clinical Interview are presented in Table 3. The mean score for the OSCE was 89.2±2.7. Students scored 90.0±4.8 on

**Table 1.** Demographic and smoking characteristics of the students participating in the class

| Variable | Male n=28 (46%) | Female n=32 (54%) | p |
|---|---|---|---|
| Age (years) | 23.14±1.5 | 22.7±6.9 | t=0.0125 p=0.417 |
| Have you ever smoked? Never <100 to date >100 to date | 18 (64.2%) 4 (14.2%) 8 (28.5%) | 20 (62.5%) 6 (18.75%) 4 (12.5%) | $x^2$=0.214 p=0.548 |
| FNDT Score* | 4.1±0.2 | 2.1±0.3 | Z=1.125 P=0.02 |
| Do your parents smoke? Father Mother | 11 (38%) 4 (15%) | 12 (36%) 3 (9.3%) | $x^2$=0.954 p=0.258 |
| Have you ever been exposed to secondhand smoke? Mean value (5=very much, 4=much, 3=sometimes, 2=rarely 1=never) | 2.45±3.2 | 1.8±2.7 | t=2.045 p<0.01 |

* Mean Fagerstrom Nicotine Dependence Test score of students who had smoked more than 100 cigarettes to date.

**Table 2.** Students' pre- and post-test score results

| ITEM | Pretest n, % | Post-test n, % |
|---|---|---|
| Number of smokers age over 18 in Turkey (1 item) | | |
| Underestimated | 35, 58.3% | 7, 11.6% |
| Overestimated | 5, 8.3% | 3, 6% |
| Answers within acceptable range (35-45%) | 20, 33.3% | 50, 83.3% |
| Risk of smoking to general health (10 items) | | |
| 0-3 correct answers | 35, 58.3% | 1, 1.6% |
| 4-7 correct answers | 12, 20% | 6, 10% |
| ≥ 8 correct answers | 13, 21.6% | 53, 88.3% |
| Health risk of secondhand smoke (4 items) | | |
| 0-2 correct answers | 56, 93.3% | 4, 6.6% |
| 3 correct answers | 3, 5% | 5, 8.3% |
| All answers correct | 1, 1.6% | 51, 85% |
| Health risk of smoking during pregnancy (3 items) | | |
| 0-1 correct answers | 20, 33.3% | 2, 3.2% |
| 2 correct answers | 28, 46.6% | 3, 5% |
| All answers correct | 12, 20% | 55, 91.6% |
| Risk of starting smoking before age 18 (1 item) | 3, 5% | 56, 93,3% |
| Benefits of cessation in terms of heart disease risk (1 item) | | |
| Underestimated | 5, 8.3% | 0, 0% |
| Overestimated | 35, 58.3% | 4, 6.6% |
| Answers within acceptable range (35-45%) | 20, 33.3% | 56, 93.3% |
| Benefits of smoking cessation in terms of lung cancer risk (1 item) | | |
| Underestimated | 14, 23.3% | 6, 10% |
| Overestimated | 35, 58.3% | 6, 10% |
| Answers within acceptable range (35-45%) | 11, 18.3% | 48, 80% |
| Benefits of cessation in terms of other cancers and diseases (2 items) | | |
| Correct answers for cancers | 22, 36.6% | 55, 91.6% |
| Correct answers for other diseases | 19, 31.6% | 56, 93.3% |
| Benefits of cessation in terms of premature death (1 item) | 15, 25% | 58, 96.6% |
| Percentage of Turkish smokers who want to quit (1) | | |
| Underestimated | 18, 30% | 2, 3.2% |
| Overestimated | 26, 43.3% | 5, 8.3% |
| Answers within acceptable range (60-70%) | 16, 26.6% | 53, 88.3% |
| Percentage of smokers expecting to quit on their own within a year (1 item) | | |
| Underestimated | 1, 1.6% | 0, 0 |
| Overestimated | 48, 80% | 4, 6.4% |
| Answers within acceptable range (<5%) | 11, 18.3% | 56, 93.3% |
| The role of the primary care physician (4 items) | | |
| Correct answer concerning asking each patient about smoking status | 2, 3.2% | 56, 93.3% |
| Correct answer concerning opportunistic smoking counseling | 6, 9.6% | 57, 95% |
| Correct answer for steps of 5As | 2, 3.2% | 58, 96,6% |
| Correct answer for steps of 5Rs | 1, 1.6% | 57, 95% |
| Model of stages of readiness to change (1 item) | 5, 8.3% | 58, 96.6% |
| Short- and middle-term nicotine craving symptoms (5 items) | | |
| 0-3 correct answers | 37, 61.6% | 2, 3.2% |
| 4 correct answers | 12, 20% | 4, 6.4% |
| All answers correct | 11, 18.3% | 54, 90% |
| Principles of motivational interview (1 item) | 2, 3.2% | 57, 95% |
| Principles of life style changes (4 item) | | |
| Correct answer concerning modifying smoking routines till quit day | 4, 6,4% | 58, 96.6% |
| Correct answer concerning features of a healthy diet | 2, 3,2% | 59, 98.3% |
| Correct answer concerning features exercises | 2,3,2% | 57, 95% |
| Correct answer concerning features hobbies | 3, 5% | 58, 96.6% |
| Nicotine replacement therapy (NRT) (4 items) | | |
| Correct success ratio | 0, 0% | 55, 91.6% |
| Correct answer concerning features of nicotine gum | 1, 1.6% | 56, 93.3% |
| Correct answer concerning features of nicotine patches | 2, 3.2% | 58, 96.6% |
| Correct answer concerning contraindication | 0, 0% | 58, 96.6% |
| Pharmacological therapy (Bupropion and Varenicline) (5 items) | | |
| Correct success ratio for Bupropion | 2, 3.2% | 52, 86.6% |
| Correct success ratio for Varenicline | 3, 5% | 54, 90% |
| Correct success ratio for combinations | 4, 6.4% | 58, 96.6% |
| Correct answer concerning features of Bupropion therapy | 2, 3.2% | 56, 93.3% |
| Correct answer concerning features of Varenicline therapy | 1, 1.6% | 58, 96.6% |
| Correct answer concerning contraindications for both | 1, 1.6% | 57, 95% |

**Table 3.** Detailed evaluation methods and mean scores from OSCE, Clinical Interview, and patient files (task)

| | | Mean Score | | |
|---|---|---|---|---|
| **Steps** | **Evaluation** | **OSCE** | **Clinical Interview** | **p** |
| Benefits of individual health gains if the subject stops smoking | 2 points max.<br>2 points=more than 3 examples are discussed<br>1 point=1-3 examples are discussed<br>0 points=If none are discussed | 1.2±0.5 | 1.1±0.9 | 0.155 |
| Calculation of Fagerstrom Nicotine Dependence Test score | 3 points in total if correctly calculated<br>1 point is subtracted from the total for every mistake | 1.7±0.5 | 1.8±0.3 | 0.214 |
| Calculation of package/year score | 3 points in total if it is correctly calculated<br>With every mistake 1 point is extracted from the total | 2.1±0.2 | 2.0±0.1 | 0.317 |
| Former quit attempts by the smoker and the methods used | 2 points in total<br>1 points for asking attempts<br>1 points for asking former used methods | 1.2±0.2 | 1.3±0.1 | 0.541 |
| Factors triggering smoking (minimum of three) | 5 points in total<br>5 points=3 or more examples are discussed<br>4 points=2 examples are discussed<br>3 points=at least 1 example is discussed<br>0 points=If none are discussed | 3.5±0.1 | 3.3±0.1 | 0.678 |
| Life style modifications (until quit day)<br>Change the last brand of cigarette consumed<br>Change the place where you smoke<br>Avoid smoking with or after tea<br>Avoid smoking with or after any meal<br>Avoid smoking in your social surroundings<br>Wait as long as you can for the first morning cigarette (at least 30 minutes)<br>Change the place where you smoke at home<br>Increase physical activity levels<br>Increase amount of daily water consumption<br>Try to find a hobby | 20 points<br>2 points for discussion of each modification | 15.8±0.8 | 16.5±0.5 | 0.147 |
| Enlist the support of family and friends | 2 points if asked and listed | 1.1±0.1 | 1.4±0.3 | 0.142 |
| Information about NRT*<br>General data about different types of NRT<br>Use of NRT<br>Teaching smokers who had chosen nicotine gum how to teach chew and park<br>Teaching smokers who had chosen patches how to use them<br>Side-effects of NRT | 15 points in total*<br>5 points=if general information is given<br>5 points=if the use of NRT is discussed properly<br>5 points=if the side-effects are discussed | 12.4±0.2 | 11.9±0.4 | 0.087 |
| Information about pharmacological therapy**<br>General data about different types of drug<br>Data about indications and side-effects of the drug<br>Use of drugs (dosage/time schedule) | 15 points in total**<br>5 points for each item discussed with the patient | 11.09±0.7 | 12.2±0.9 | 0.108 |
| Draw up a personalized treatment plan<br>Motivational interview (MI) only<br>MI+NRT<br>Bupropion<br>Bupropion+NRT<br>Varenicline | 20 points in total if the treatment plan is discussed with the patient | 16.9±0.1 | 17.5±0.2 | 0.097 |
| Individualized plan for quit day<br>Appoint a quit date<br>Provide information about the symptoms of nicotine withdrawal<br>Establish a plan for nicotine cravings<br>Remove the smell of nicotine from the house, clothes or car<br>Remove all tobacco products and equipment from house/work | 5 points in total<br>1 points for every item discussed with the patient | 3.6±0.8 | 3.7±0.9 | 0.078 |
| Establish a reward system for not smoking<br>Short term<br>Long term | 3 points in total<br>1.5 points for every item discussed with the patient | 1.7±0.4 | 1.8±0.2 | 0.215 |
| Avoid relapse<br>Provide information about slips and relapse<br>Establish a plan for relapse management | 5 points in total<br>2.5 points for every item discussed with the patient | 3.0±0.2 | 3.0±0.5 | 0.321 |
| Total | | 89.2±2.7 | 90.0±4.8 | 0.109 |

clinical counseling. Students scored a mean 89.2±0.4 from the class based on their post-test, OSCE, clinical counselling.

**4. Discussion**

In designing this class our main concern was to provide medical students with the essential knowledge and skills they would require to counsel smokers in their professional lives. From that point of view the class was designed as one of the most intense and integrated smoking cessation counseling classes in current undergraduate medical training. Students spent 96 hours on the class, nearly half of which represented clinical interviews. This length of time is very high compared to many other medical schools around the world (Richmond, 1999; Frank et al., 2007; Richmond et al., 2009; Chatkin and Chatkin, 2009; Raupach et al., 2015). Many countries devote an average of 7 to 8 hours of education focused on tobacco provided throughout the entire medical curriculum. One of the most important aspects of the class is giving students the opportunity for very early clinical contact with real smokers. Every student has the opportunity to counsel many different types of smokers (first attempt, relapsed smokers, teenagers, pregnant women etc.) under the supervision of academics. Richmond et al. (1999) concluded that only 45% of medical students worldwide have the opportunity to interface with real smokers.

Students' smoking cessation and prevention knowledge and skills increased after participation in this selective class. We evaluated different aspects of the class using various evaluation methods. Students' informative knowledge was evaluated with written exams (pre- and post-test results), and their skills and applied skills were tested with OSCE and Clinical Interview. There were two reasons for evaluating the students' skills and applied skills using two different skill evaluation methods. First, although OSCE is a well proven and effective way of evaluating students' skills, the smoking status of simulated patients or students (members of the drama club) might affect the entire process. A non-smoker pretending to be a patient might lack the experience of a real smoker, and a smoker who had frequent relapses might be poorly motivated. The objectivity and performance of the simulated patients might limit the accuracy of the scenario and the accuracy of the evaluation (Mounsey et al., 2006). Second, in OSCE, while educators can control every factor in a fictional clinical environment (features of the simulated patient, time schedule, etc.) which provides a good idea of a medical student's performance, real clinical practice is full of unforeseen factors to which the physician must quickly adapt and find solutions. Clinical interviews have the advantage of evaluating a student's performance and reaction to real-life situations. We therefore elected to use not overly-complicated cases rather than extreme cases (patients with psychotic symptoms or other psychological problems, pregnancy, teenagers or individuals with a history of many relapses, etc.) in order to avoid confronting them with a situation beyond their abilities. OSCE and clinical interview pose advantages and disadvantages which perfectly compensate for one another.

We apply the selective smoking cessation and prevention knowledge and skills class as early as possible in the early period of medical education for a number of reasons. The importance of the philosophy of primary prevention may best be seeded before students are concentrating on other specialties which attach high priority to interventions in their learning objectives. Frank et al. (2007) stated that students' attitudes shift towards prevention rather than intervention if they receive the appropriate instruction earlier in their medical training. However, in order to use these knowledge and skills properly in their professional lives, medical students must have occasional opportunities to practice and sharpen their skills during their medical education (spiral education principle).

Although our class covered many areas of tobacco cessation and prevention using a range of learning methods (lectures, discussions, case studies, problem-based learning sessions, etc.), as recommended in other publications (Springer et al., 2008), we did not employ web-based learning. That method offers many advantages, such as instant access to data, the formation of discussion groups or the opportunity to consult patients. However, we decided to devote some of our problem-based scenarios to an interactive web-based model. We believe that this method is more important in classes with a large number of participants when the number of PBL instructors is limited. The same problem can be overcome in clinical settings if there are also enough clinicians who can work one-to-one with medical students. If this class is included in the curriculum as a standard class instead of a selective one, the need for educators qualified in field of tobacco cessation will be critical. In order to ensure that our program ran smoothly we worked with real smokers receiving treatment in our clinic. This left the students to deal first-hand with patients who were very highly motivated to quit smoking. Although primary care mostly advocates horizontal health care services (Holmberg et al., 2014), in countries with very high rates of smoking, longitudinal organizations (specialized centers) can be very useful in terms of education and referral of selected cases.

In a country such as Turkey where 19 million adults smoke regularly, the first priority in medical education should be cessation and prevention (Bilir et

al., 2009). New and effective methods or techniques should be investigated in order to promote these skills and knowledge. Strategies to overcome some common problems (such as a lack of motivated and qualified instructors, resources and time, and inflexible curricula) in this field should be also investigated. In conclusion, this study describes a successful model for promoting tobacco cessation knowledge and skills. The advantage of this study is that the class can be given not only to undergraduates but also to postgraduate students (residents) or in the form of continuing medical education for professionals. This will inevitably result in new volunteers for a smoke-free world.

**REFERENCES**


Bilir, N., Cakır, B., Daglı, E., et al. 2009. Tobacco control in Turkey. Copenhagen, World Health Organization. Available at: http://www.euro.who.int/__data/assets/pdf_file/0004/98446/E93038.pdf (Accesed at 27.2.2015).

Brotons, C., Björkelund, C., Bulc, M., Ciurana, R., Godycki-Cwirko, M., Jurgova, E., Kloppe, P., Lionis, C., Mierzecki, A., Piñeiro, R., Pullerits, L., Sammut, M.R., Sheehan, M., Tataradze, R., Thireos, E.A., Vuchak, J; EUROPREV network, 2005. Prevention and health promotion in clinical practice: The views of general practitioners. Prev. Med. 40, 595-601. doi:10.1016/j.ypmed.2004.07.020.

Cahill, K., Stevens, S., Perera, R., Lancaster, T., 2013. Pharmacological interventions for smoking cessation: An overview and network meta-analysis. Cochrane. Database. Syst. Rev. 5, CD009329. doi: 10.1002/14651858.CD009329.pub2.

Chatkin, G., Chatkin, J. 2009. Learning about smoking during medical school: Are we still missing opportunities? Int. J. Tuberc. Lung. Dis. 13, 429-437. Available at: http://docstore.ingenta.com/cgibin/ds_deliver/1/u/d/ISIS/80949387.1/iuatld/ijtld/2009/00000013/00000004/art00003/18192C1EE8C437BA14250434701598DF2690045CB8.pdf (Accessed on 18.02.2014).

Conroy, M.B., Majchrzak, N.E., Regan, S., Silverman, C.B., Schneider, L.I., Rigotti, N.A., 2005. The association between patient-reported receipt of tobacco intervention at a primary care visit and smokers' statisfaction with their health care. Nicotine. Tob. Res. 7, 29-34. doi: 10.1080/14622200500078063.

Ellerbeck, E.F., Ahluwali, J.S., Jolicoeur, D.G., Gladden, J., Mosier, M.C., 2001. Direct observations of smoking cessation activities in primary care services. J. Fam. Pract. 50, 688-693.

Ferguson, J., Bauld, L., Chesterman, J., Judge K. 2005. The English smoking treatment services: One-year outcomes. Addiction. 100, 59-69. doi: 10.1111/j.1360-0443.2005.01028.x.

Ferry, L.H., Grissino, L.M., Runfola, P.S., 1999. Tobacco dependence curricula in USA undergraduate medical education. JAMA. 282, 825-829. doi:10.1001/jama.282.9.825.

Fiore, M.C., Jaén, C.R., Baker, T.B., Bailey, W.C., Benowitz, N.L., Curry, S.J., Dorfman, S.F., Froelicher, E.S., Goldstein, M.G., Healton, C.G., Henderson, P.N., Heyman, R.B., Koh, H.K., Kottke, T.E., Lando, H.A., Mecklenburg, R.E., Mermelstein, R.J., Mullen, P.D., Orleans, C.T., Robinson, L., Stitzer, M.L., Tommasello, A.C., Villejo, L., Wewers, M.E., Murray, E.W., Bennett, G., Heishman, S., Husten, C., Morgan, G., Williams, C., Christiansen, B.A., Piper, M.E., Hasselblad, V., Fraser, D., Theobald, W., Connell, M., Leitzke, C., 2008. Treating tobacco use and dependence: 2008 update U.S. Public Health Service Clinical Practice Guideline executive summary. Respir. Care. 53, 1217-1222.

Frank, E., Carrera, J.S., Elon, L., Hertzberg, V.S., 2007. Predictors of US medical students' prevention counselling practices. Prev. Med. 44, 76-81. doi:10.1016/j.ypmed.2006.07.018.

Hartmann-Boyce, J., Lancaster, T., Stead, L.F., 2014. Print-based self-help interventions for smoking cessation. Cochrane. Database. Syst. Rev. 6, CD001118. doi: 10.1111/add.12633.

Holmberg, C., Sarganas, G., Mittring, N., Braun, V., Dini, L., Heintze, C., Rieckmann, N., Muckelbauer, R., Müller-Nordhorn, J., 2014. Primary prevetion in general practice-views of German general practitioners: a mixed-methods study. BMC. Fam. Pract. 15, 103. doi: 10.1186/1471-2296-15-103.

Lai, D.T., Cahill, K., Qin, Y., Tang, J.L., 2010. Motivational interviewing for smoking cessation. Cochrane. Database. Syst. Rev. 20, CD006936. doi: 10.1002/14651858.CD006936.pub2.

Mathers, C.D., Loncar, D., 2006. Projections of global mortality and burden of disease from 2002 to 2030. PloS. Med. 3,442. doi:10. 1371/journal.pmed.0030442.

McAvoy, B.R., Kaner, E.F., Lock, C.A., Heather, N., Gilvarry, E., 1999. Our healthier nation: Are general practioners willing and able to deliver? A survey of attributes to and involvement in health promotion and life style counseling. Br. J. Gen. Practice. 49, 187-190.

Mounsey, A.L., Bovbjerg, V., White, L., Gazewood, J., 2006. Do students develop better motivational interviewing skills through role-play with standardized patients or with student's colleagues. Med. Educ. 40, 775-780. doi: 10.1111/j.1365-2929.2006.02533.x

Montalto, N.J., Ferry, L.H., Stanhiser, T., 2004. Tobacco dependence curricula. J. Am. Osteopath. Assoc. 104,317-23.

National Institute for Health and Clinical Excellence. Smoking cessation services. Quick reference guide, 2008.

Ponciano-Rodrigez, G., 2010. The urgent need to change the current medical approach on tobacco cessation in Latin America. Salud. Publica. Mex. 52, 366-372. http://dx.doi.org/10.1590/S0036-36342010000800035.

Rigotti, N.A., Thorndike, A.N., 2001. Reducing the health burden o tobacco use: What's the doctors role? Mayo. Clin. Proc. 76, 121-123. doi: http://dx.doi.org/10.1016/S0025-6196(11)63116-9.

Warner, J.J., Lawson, P.J., Panaite, V., et al. 2013. Comparing primary care physicians' smoking cessation counseling techniques to motivational interviewing. J. Addict. Med. 7, 139-142. doi: 10.1097/ADM.0b013e3182879cc5.

Raupach, T., Al-Harbi, G., McNeill, A., Bobak, A., McEwen, A., 2015. Smoking cessation education and training in U.K. medical schools. Nicotine. Tob. Res. 17, 372-375. doi: 10.1093/ntr/ntu199.

Richmond, R., 1999. Teaching medical students about tobacco. Thorax. 54, 70-78. doi:10.1136/thx.54.1.70.

Richmond, R., Zwar, N., Taylor, R., Hunnisett, J., Hyslop, F., 2009. Teaching about tobacco in medical schools: A worldwide study. Drug and Alcohol Rev. 258, 484-497. doi: 10.1111/j.1465-3362.2009.00105.x.

Springer, C.M., Tannert Niang, K.M., Matte, T.D., Miller, N., Bassett, M.T., Frieden, T.R., 2008. Do medical students know enough about smoking to help their future patients? Assessment of New York City fourth-year medical student's knowledge of tobacco cessation and treatment for nicotine addiction. Acad. Med. 83, 982-989. doi: 10.1097/ACM.0b013e3181850b68.

Stead, L.F., Buitrago, D., Buitrago, D., Preciado, N., Sanchez, G., Hartmann-Boyce, J., Lancaster, T., 2013. Physician advice for smoking cessation. Cochrane. Database. Syst. Rev. 5,CD000165. doi: 10.1002/14651858.CD000165.pub4.

Young J., Ward, J., 1998. Influence of physician and patient gender on provision of smoking cessation advice in general practice. Tob. Cont. 7, 360-363. doi:10.1136/tc.7.4.360.

World Health Organization. WHO report on global tobacco epidemic, 2013. Enforcing bans on tobacco advertising, promotion and sponsorship. The MPOWER package. Geneva: World Health Organization, 2013.

Zwar, N.A., Mendelson, C.P., Richmond, R.L., 2014. Tobacco smoking: Options for helping smokers to quit. Aust. Fam. Physician. 43, 348-354.

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# The comparison of insulin levels between over weighted and non-obese smokers

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**ABSTRACT**

Cardiovasculer diseases are the most common reason for morbidity and mortality in the world. Smoking and obesity are among the most important avoidable reasons for these diseases. However, in Turkey, there are not enough studies about the effect of obesity (central and androgenic) on insulin levels of smokers. With this aim; oral glucose tolerance test (OGTT) was performed on 84 patients who meet the study criteria among 211 patients treated at the second internal diseases clinic of Taksim Hospital between November 1997 and May 1998, who were volunteers, smokers and older than 18. In addition, the total cholesterol, HDL, LDL and triglicerid levels of these patiens were checked. Heights, weights and waist circumferences of patients were measured. From these datas, body mass indexes (BMI) and waist circumferences (WC) were calculated. Nicotine addiction levels of patients were evaluated by Fagerstrom Nicotine Dependency Test (FNDT) and package/year amounts were calculated. According to the results based on the data obtained from the studies, no difference were determined ($p>0.05$), in ages, FNDT scores, insulin, TC, LDL and HDL levels between overweighted and normal weighted patients. On the other hand; there was statistically significant difference between two groups in BMI values, WC measurements (between different genders) and TG levels. In the regression analysis; BMI [OR: 1.512, (95% CI min=0.928, max=2.069)] and WC [OR: 1.912, (95% CI min=1.051, max=2.125)] was founded as a risk factor for the insulin increment. Additional information about the subject for the large participation cross-sectional studies. More action about life style modification (smoking cessation and an effective weight control) may increase risk of cardiovascular diseases in populations.



## 1. Introduction

Around the globe various studies on the effects of smoking on the insulin levels are started to be done recently (Hinnouho et al., 2013). There is increasing evidence that smoking is conducive to a greater accumulation of visceral fat tissue and greater insulin resistance (Istvan et al., 1982). Smoking also increases the risk of metabolic syndrome and type 2 diabetes. It is well known that smokers' blood glucose rise to levels higher than normal promoting insulin resistance. This condition was often tried to be explained by the mechanism of rising the stress hormone, cortisol by cigarette smoking. As cortisol excess is known to induce insulin resistance, it has been suggested that glucocorticoids, such as cortisol, may be the missing link between cigarette smoking and insulin resistance

(Harris et al., 2016). Although smokers tend to lose weight and have less fat tissue compared to nonsmokers they may still develop prediabetes and have high levels of cortisol in their blood and tissues. This situation may aggravates the cardiovascular disease and atherosclerosis which reflects the high rate of mortality from cardiovascular diseases in smokers. According to the Framingham study, the life expectancy of obese smokers was approx. 13 years shorter than that of normal-weight nonsmokers. In the same cohort, one third to one-half of obese smokers died between the ages of 4t0 and 70 years, whereas only 10% of normal-weight nonsmokers did so (Fox et al., 2004).

The global trend in increasing rates of obesity around the globe puts challenging tasks for the physicians in developing world. The aim of this study is to determine the relationship between insulin levels in over-weighted and normal weighted smokers in a Turkish sample.

## 2. Material and method

### 2.1. Study design

This study is designed as a case control study. It was conducted among Taksim State Hospital 2nd Internal Diseases Clinic between November 1997 to May 1998. The study sample was selected between this time among volunteered obese and non-obese smokers who had presented to 2nd Internal Diseases Clinic who are currently smoking more than a year (A total of 211). The patients who are <18 years, with Diabetes Mellitus (DM), or not using medication which impairs glucose-insulin metabolism (steroids etc.), patients with severe kidney (Acute/chronic renal failure) or liver diseases were omitted the study (n=119). Oral Glucose Tolerance Test (OGTT) was performed to these patients in accordance with WHO criteria's.

In order to achieve this goal, 75 grams of glucose were given to the patients orally after 12 hours of hunger. The blood glucose levels were obtained at pre-prandial; 30. 60. 90. and 120. minutes. From all the patients whose pre-prandial glucose level >140 mgr. / dl, 1. hour >200 mgr/dl and 2. hour >200 mgr/dl were omitted from the study and were diagnosed as DM (n=8). Remaining 84 patients (over weighted n=48, normal weighted n=36) were included in the study. Every participant's blood plasma who were included into study was obtained after 12 hours night hunger in sitting position, and sent to laboratory for evaluating their insulin, LDL, HDL and triglyceride levels. Diagnostic Systems Laboratories, DSL-1600 İnsülin Radioimmunassey Kits were used at evaluating insulin levels.

Interviewers asked information about tobacco use to the participants, investigated the age that they started to smoke, calculated their package/year. All of the participants were administered the Fagerström Nicotine Dependency Test (FNTD) (Fagerstrom et al., 1996). The interviewers also measured weight, height and waist circumference (WC) of the participants and calculated their Body Mass Index (BMI). Weight and height were measured using standard calibrated digital weighing scale and portable inflexible measurement tapes. WC was measured after the patients exhale breath from halfway between the lowest border of the ribs (the mid of the 12th rib) and the upmost part of the hip (iliac crest) on the middle axillary line using constant-tension tape device. The BMI is calculated by dividing weight (Kg) to the square of the height (cm). The patients who had BMI between 25 to 30 $Kg/m^2$ is accepted as over weighted. The patients who had BMI≥30 $kg/m^2$ is accepted as obese and WC ≥88 cm in females and ≥102 cm in males according to National Cholesterol Education Program-Third Adult Treatment Panel (ATP III) criteria (NIH, 2002).

### 2.2. Fagerstrom test for nicotine dependence

This test is a questionnaire used to determine the degree of smoking addiction commonly in the world. It was developed by Karl O. Fagerström in order to determine the level of physical smoking addiction and contains six questions (Fagerström et al., 1996). The patient can both be applied face to face and can be filled individually. A score between 1 and 10 can be taken from the questionnaire and the dependence level is considered to increase as the amount of the score taken increases. Those who take a score below 5 points from the test are defined as low nicotine addicts, and those who take 7 points and above are defined as severe nicotine addicts. In the adaptation made for our country, test and retest correlations were found 0.85 and 0.88 in order (Fidancı et al., 2015). A standardization study is available for the Turkish sampling (Uysal et al., 2004) and this scale was applied only to smoking participants (Şenyüz and Coştur, 2010).

### 2.3. Statistical analyses

The acquired data were examined and evaluated using SPSS 16.0 statistics program. Characteristics of the study group were presented with definitive type of analyses (number, percentage, average and standard deviation). Data were evaluated using mutual independent group comparisons Mann-Whitney-U groups test and Pearson chi-square and Kruskall Wallis test analysis methods. Statistical significance level 'p' value was accepted as those below 0.05.

### 2.4. Ethics

Ethical Approval was obtained from Minister of Health of Turkish Republic

## 3. Results

A total of 84 participants (43 men 51.2% and 41 women

**Table 1.** The comparison between overweighed and normal weighted smokers FNTD scores, package year, insulin, TG, HDL and LDL levels

| Variables | Overweighted N=43 | Normal weighted N= 41 | t | p |
|---|---|---|---|---|
| FNDT | 4.52±1.23 | 4.56±1.25 | 0.258 | 0.624 |
| Pack/year | 12.5±2.5 | 12.9±2.3 | 0.154 | 0.754 |
| BMI (kg/m$^2$) | 28.6±2.4 | 23.04 | 12.739 | <0.001 |
| WC (cm) | Women 98±1.2<br>Men 120.2±1.8 | Women 90±1.5<br>Men 116.1±1.3 | 10.247<br>9.245 | <0.001<br><0.001 |
| Insulin ($\mu$IU/ml) | 25.16±5.3 | 24.97±6.2 | 0.153 | 0.878 |
| TC (mgr/dl) | 146±2.2 | 143±2.8 | 0.541 | 0.154 |
| LDL (mgr/dl) | 139.8±32.6 | 127.72±27.1 | 1.813 | 0.074 |
| TG (mgr/dl) | 219.5±77.7 | 169.2±106.1 | 2.509 | 0.014 |
| HDL (mgr/dl) | 42.7±9.4 | 44.5±9.6 | 0.694 | 0.489 |

**FNDT:** Fagerstrom nicotine dependency test; **BM**: Body, mass index; **WC:** Waist circumference; **TC:** Total cholesterol

48.8%) volunteered to participate into the study. There was no difference between the ages of the women and men participants in different groups (t=0.425, p=0.672). The comparison between two groups' FNDT, package/year, BMI, Insulin, total cholesterol, LDL, TG and HDL values are presented at Table 1. Only the TG levels in over weighted participants were high when compared to other group (p=0.014). It was seen that BMI (r=0.3) and WC (r=0.36) is correlated with insulin levels in both groups. In a linear regression model the BMI [OR: 1.512, (95% CI min=0.928, max=2.069)] and WC [OR: 1.912, (95% CI min=1.051, max=2.125)] was founded as a risk factor for the insulin increment. The model had a Durbin Watson value of 1.045, and its $r^2$ value was 0.296. The linear regression analyses are presented at Table 2.

## 5. Discussion

We obtained interesting results in this study in which we researched for the factors that can affect the insulin levels in overweight and normal weight smoker individuals. There is detailed information about insulin secretion, resistance, obesity and metabolic syndrome in the non-smokers. De Fronzo et al. (1985) had studied insulin resistance, glucose response and insulin response as dependent variables in over weighted and obese participants. They have reported that insulin response has only significant relation with decrease of leptin level independently. More importantly in the same study it was concluded that there is correlation between leptin concentration and a decrease in insulin response after weight loss. Also this decrease had no correlation regardless of which BMI measurement that patients have (De Fronzo et al., 1985). There are serious information that WC which is another anthropometric measurement method, is a risk factor for insulin level, insulin resistance and prediabetes (Li et al., 2016). Carantoni et al. (1999) reported that local distribution of body fats are important signs for NIDDM and cardiovascular disease. In a study performed on premenapausal women, visceral adipose tissue level has relation with lipoprotein rate that is used in cardiovascular disease risk analysis. In the same study it was revealed that high visceral fat level has relation with decrease in glucose tolerance and also it remains importantly after total body fat levels are taken under

**Table 2.** The Linear regression model investigating the relation of dependent variables of FNDT, with dependent variables of BMI, WC, FNDT, package year, gender, age, LDL, TG and HDL

| Model | Unstandardized coefficients | Standardized coefficients | | | 95% Confidence interval for B | |
|---|---|---|---|---|---|---|
| | B | Std. error | Beta | t | Sig. | Lower bound |
| (Constant) | -40.067 | 11.265 | | -3.557 | 0.001 | -62.503 |
| GENDER | -2.039 | 1.200 | -0.179 | -1.699 | 0.093 | -4.429 |
| BMI | 1.512 | 0.293 | 0.907 | 5.154 | 0.000 | 0.928 |
| AGE | 0.024 | 0.065 | 0.036 | .369 | 0.713 | -0.106 |
| LDL | 0.022 | 0.019 | 0.116 | 1.111 | 0.270 | -0.017 |
| TG | 0.004 | 0.006 | 0.068 | 0.650 | 0.518 | -0.009 |
| HDL | 0.034 | 0.065 | 0.057 | 0.527 | 0.600 | -0.095 |
| WC | 1.912 | 1.927 | 0.728 | 4.338 | 0.000 | 1.051 |
| FNDT | 0.251 | 0.021 | 0.521 | 0.541 | 514 | 0.621 |
| Package/year | 0.317 | 0.841 | 0.745 | 0.127 | 0.754 | 0.124 |
| TC | 0.08 | 0.148 | 0.124 | 0.05 | 0.147 | 0.08 |

**INSULIN:** Dependent variable; **FNDT:** Fagerstrom nicotine dependency test; **BM**: Body, mass Index; **WC:** Waist circumference; **TC:** Total cholesterol

control. More interestingly that there are significant relations between abdominal visceral obesity, insulin resistance and dyslipidemia and that visceral obesity is an important component for Syndrome X (Carantoni et al., 1999).

Only recently that data has started to be gathered about smoking addiction and insulin secretion, insulin resistance, central obesity (BMI) and/or androgenic obesity (WC) and metabolic syndrome. It is well known that nicotine acutely increases energy expenditure and could reduce appetite, which may explains why smokers tend to have lower BMI (Williamson et al. 1991). However in several cross sectional studies there has been clustering evidence of relationship between smoking and central (BMI) or androgenic (WC) obesity (Wild and Byrne, 2006). It is observed that; in overweight smokers there is a strong relation between obesity, metabolic syndrome, insulin resistance and diabetes (Chiolero et al., 2008). It has been argued that the increased insulin resistance in smokers may be related to their tendency towards increased abdominal fat accumulation (Canoy et al., 2005). Cross-sectional studies indicate that smokers tend to have both a larger waist circumference and a smaller hip circumference, compared to nonsmokers (Leite at al., 2006). This situation may be partly explained by the increase at visceral fat tissue. However this topic need more investigation as the association between smoking and visceral fat accumulation may be partly explained by a confounding with the low degree of physical activity and unhealthy diet frequently encountered among smokers.

In a study with a participating 729 dyslipidemia patients (143 of them were smokers) it has been revealed that insulin resistance is more common in smokers compared to non-smokers (Cibickova et al., 2014). In study which was conducted in Japan 1199 men who were at the baseline of impaired insulin secretion and insulin resistance were investigated (Morimoto et al., 2013). This study revealed that cigarette smoking is a modifiable risk factor for impaired insulin secretion. The interesting factor was this study showed that ex-smokers were at danger for impaired insulin secretion compared with non-smokers [1.06 (95% CI, 0.84-1.33)]. The most risky group was the current smokers [1.95 (95% CI, 1.44-2.63)]. The number of pack-years was positively associated with the risk for impaired insulin secretion in a dose-dependent manner (P-values for trend <0.001). The multivariable-adjusted HRs for insulin resistance were 0.95 (95% CI 0.56-1.61) in ex-smokers and 1.11 (95% CI 0.67-1.79) in current smokers compared with never-smokers. Szulinska et al., (2013) conducted a study investigating the potential influence of obesity and smoking on insulin resistance. In this study it was stated that smoking has a significant effect on insulin resistance, TNF-α concentration. Moreover, the coexistence of smoking and obesity significantly aggravates the abnormalities observed. The insulin levels ($\mu$İU/ml) of obese nonsmokers were (36.1±19.3), obese smokers (45.7±15.5) and normal weight smokers (7.5±2.8). The normal weighted non-smokers had the lowest insulin levels . Compared with that study our results confirmed lower insulin levels in both groups however they are both higher than the normal weight smokers. Although there were no differences between two groups' (over weighted and normal weighted) mean insulin levels there was a positive correlation between insulin levels and BMI and WC measurements in linear regression analyses.

Our study may have some flaws. First of all in our study we didn't included a healthy nonsmoker normal weight group. The results of this group might help us better understand the relationship and effect of smoking to insulin levels in smokers. Our aim in this study was to reflect that the insulin levels of normal weighted smokers may be as high as obese smokers. Although our design was a case control study so large cross sectional studies may confirm our results.

As a conclusion both smoking and obesity (increased BMI and WC) may be contributed to the levels of insulin, insulin resistance which is a key factor and significant predictive factor cardiovascular disease. More action about life style modification (smoking cessation and an effective weight control) may increase risk of cardiovascular diseases in populations.

**REFERENCES**

Canoy, D., Wareham, N., Luben, R., 2005. Cigarette smoking and fat distribution in 21, 828 British men and women: A population-based study. Obes. Res. 13, 1466-1475.

Carantoni, M., Abbas, F., Azhar, S., Schaaf, P., Reaven, G.M., 1999. References in insulin resistance: The link between obesity and cardiovascular disease. J. Clin. Endocrinol. Metab. 84, 869-872.

Chiolero, A., Faeh, D., Paccaud, F., Cornuz, J., 2008. Consequences of smoking for body weight, body fat distribution, and insulin resistance. Am. J. Clin. Nutr. 8, 87, 801-809.

Cibičková, Ľ., Karásek, D., Langová, K., Vaverková, H., Orság, J., Lukeš, J., Novotný, D., 2014. Correlation of lipid parameters and markers of insulin resistance: does smoking make a difference? Physiol. Res. 3, 387-393.

De Fronzo, R.A., Lnogmore, J.M., Hodgetts, T.J., Ramrakha, P.S., 1985. Oxford Handbook of Clinical Medicine. Thirteenth edition. Oxford Press. Oxford.

Fagerström, K.O., Kunze, M., Schoberberger, R., Breslau, N., Hughes, J.R., Hurt, R.D., Puska, P., Ramstrom, L., Zatonski, W.,

1996. Nicotine dependence versus smoking prevalence: Comparisons among countries and categories of smokers. Tobacco Control. 5, 52-56.

Fidancı, İ., Tekin, O., Arslan, İ., Eren, Ş,Ü., Dinçer, G., Kızıltaş, Ö., 2015. The effect of smoking dependency on HbA1C. Ankara Med. J. 15, 127-133.

Fox C.S., Evans J.C., Larson M.G., Kannel, W.B., Levy, D., 2004. Temporal trend in coronary heart disease mortality and sudden cardiac death from 1950 to 1999: The Framingham Study. Circulation. 110, 522-527.

Harris, K.K., Zopey, M,. Friedman, TC., 2016. Nat. Rev. Endocrinol, 12, 299-308

Hinnouho, G., Czernichow, S., Dugravot, A., Batty, G.D., Kivimaki, M., Singh-Manoux, A., 2013. Metabolically healthy obesity and risk of mortality: Does the definition of metabolic health matter? Diabetes Care. Epub ahead of print. 2.

Istvan, J., Cunningham, T., Garfinkel, L., 1992. Cigarette smoking and body weight in the cancer prevention study I. Int. J. Epidemiol. 21, 849-853.

Li, L., Yin, X., Yu, D., Li, H., 2016. Impact of physical activity on glycemic control and insulin resistance: A study of community-dwelling diabetic patients in eastern China. Intern. Med. 55, 1055-1060.

Lwite, M.L., Nicolasi, A., 2006. Lifestyle correlates of anthpometric estimates of body adiposity in an Italian middle-aged and elderly population: A covariance analysis. Int. J. Obes. 30, 926-934.

Mandeep, B., 2012. Nicotine and insulin resistance: When the Smoke Clears. Diabates. 61, 3079-3080.

Morimoto, A., Tatsumi, Y., Deura, K., Mizuno, S., Ohno, Y., Watanabe, S., 2013. Impact of cigarette smoking on impaired insulin secretion and insulin resistance in Japanese men: The Saku Study. J. Diabetes Investig. 4, 274-280.

NIH (National Institute of Health), 2002, Third Report of National Cholesterol Education Program (NCEP) Expert Panel. Detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). NIH publication No.02-5215. 2002. http://www.nhlbi.nih.gov.resources.ATPIII.pdf.

Schroeder, S., 2013. Smoking-related mortality in the United States. N. Engl. J. Med. 368, 1753-1754.

Şenyüz, L., Coştur, R., 2010. The assessment of smoker and non-smoker university students of the warning words on the cigarette packages. J. Faculty Lett. 27, 253-270.

Szulinska, M., Piorunek, T., Suliburska, J., Pupek-Musialik, D., Kupsz, J., Drzymala-Czyz, S., Bogdanski, P., 2013. Evaluation of insulin resistance, tumor necrosis factor alpha, and total antioxidant status in obese patients smoking cigarettes. Eur. Rew. Med. Pharmacol. Sci.17,1916-1922.

Wild, S.H., Byrne, C.D., 2006. ABC of obesity. Risk factors for diabetes and coronary heart disease. BMJ. 333, 1009-1011.

Williamson, D.F., Madans, J., Anda, R.F., Kleinman, J.C.,Giovino, G.A., Byers,T., 1991. Smoking cessation and severity of weight gain in a national cohort. N. Engl. Med. 324, 739-745.

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# The mid-term effect of grandchildren and children motivation to quit rates of smokers

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**ABSTRACT**

We aimed to investigate the mid-term quit rates of smokers who were motivated to quit smoking directly by their children or grandchildren. Thousand one hundred and forty-eight smokers who had attended to the Ondokuz Mayis University smoking cessation clinic were investigated for their initial motivation for quit smoking. Among them 80 participants were accepted as study group who claimed that their primary motivation for smoking cessation was their children or grandchildren's wish to see them as non-smokers. 200 other smokers were accepted as control group randomly. An individualized therapy cessation technique was selected for each participant (combination of behavioral counseling, nicotine replacement therapy, and/or pharmacotherapy). All of the participants in both groups attended a standard quittance program. The smoking statuses of both groups were investigated at the end of 1st and 3rd month after. Although there was no difference between the sociodemographic and smoking features of the both groups the study group had a better quit rate after 1st (45% versus 35%) and 3rd (37% versus 29%) month compared to control group ($p<0.001$ respectively). To get motivation from grandchildren or children had an independent effect on cessation (O.R=1.094, 95%CI, $p<0.001$). The smokers who were motivated to quit by their children or grandchildren may have an increased chance of quittance.



## 1. Introduction

Smoking is one of the most important health care problems around the world (WHO, 2012). Smoking is a very complex problem for the primary care physician as this epidemic has very different facets. One of the main problems dealing with smoking for the primary care physicians is to motivate smokers for a behavioral change and initiate a cessation attempt. Transtheoretic Model (Prochaska and Norcross, 2010), explains the change of behavior in five steps each have different rate of importance (Precontemplation [not ready or thinking of change], contemplation [getting ready], preparation [ready], action, maintenance). The most important steps are considered as the first three steps. There are several broad factors effects smokers to take cessation decision just like increasing health problems contributed to their smoking, negative impact on their social relations, the legal prohibitions in society, or just the burden of economical cost for smoking (Ross, 2016). Until know the effect of social support on first three steps of Transtheoretic Model is not studied in detail. Many researchers gave social support a theoretical importance especially on the last step (Maintenance) of this model (Westmaas et al., 2010). The importance of

social support is investigated in different sources, terms and conditions (Burns et al., 2014). Social support is typically defined as "the social resources that persons perceive to be available or that are actually provided to them by nonprofessionals in the context of both formal support groups and informal helping relationships (Cohen., 2004). The researchers tries to find answers to two important questions in order to formulate the most effective type of support. First one is who can deliver best social support to the smokers and second one is what is the best way to do this is. Different people from the smokers social circle regarding to their emotional or intimate bond (spouse, partner, and "buddy" etc) is investigated (May and West, 2000). In the second topic integrated counselling, telephone calls and even computer assisted cessation is researched (Rindal et al., 2013).

In Turkish society being a parent and/or grandparent is a very important social role (Çelik et al., 2012). In this social role parents and grandparents may have positive intimate and emotional relationships with their descendant. This positive bond may have a very powerful effect on both sides' behavioral patterns (Roopnarine and Carter., 1992). It may not be a rare event that grandchildren and children get concern about their smoker parent's health outcomes. It is possible that they may motivate their parents with this emotional bond. So far the effect of children and grandchildren's motivation on their smoker parents are not studied in our country. We investigated the effect of children and grandchildren's motivation on their parent/grandparents smoking quittance rate in mid-term.

## 2. Materıals and methods

### *Design*

The study was designed as a randomized case-controlled trial between May 2011 and December 2012. It was performed at the Ondokuz Mayis University (OMU) Medical Faculty Department of Family Practice Smoking Quittance Clinic, Turkey.

1148 smokers who had attended to the Ondokuz Mayis University smoking cessation clinic were asked to list their three prime motivation reason to quit smoking from most important to the lesser ones face to face by the researchers. Among these smokers who stated that their main motivation is related with their children or grandchildren in the first place were accepted as study group. Other smokers who didn't state any knowledge about their children and grandchildren motivation in their list is accepted as control group.

The criteria for inclusion in the study were willingness to take part and attendance at all sessions, age >18 years, intending to quit smoking within six months, Fagerstrom Nicotice Dependency Score>5 points, smoking more than 10 cigarettes a day, not being on any psycho-regulatory medication (antidepressant, anxiolytic or antipsychotic), not having any psychiatric illnesses, not being pregnant or breast-feeding and applying all the program session content for three months. A total of 80 smokers in the study group (out of 98 smokers) who meet inclusion criteria were accepted as study group. Among 1050 smokers 880 of them met study's' inclusion criteria. Two hundred control cases were selected randomly from them. Every fourth patient were selected from the list which were organized by their alphabetic surname order.

At the beginning of their program, each participant in the study and control subgroups was asked to respond to the Fagerstrom Test for Nicotine Dependence (FNDT). A full physical examination is performed and their anthropometric measurements were recorded. After that every smoker was applied our clinics standard cessation program which were described elsewhere in detail (Yalçın et al., 2012). At the end of 1st month and 3rd smoking status of patients was established by self-report and assessment of carbon monoxide (CO) with an inhaler. Participants who relapsed on just one or two occasions were not excluded from the study. Participants with readings of ≤10 ppm CO were regarded as smoking-free. The smoker who didn't attended to clinic between these two time schedules were called by telephone and invited to clinic to confirm their condition. 12 smokers in the study group and 42 smokers in the control group were called by phone. The cessation rate of two groups were compared with each other.

### *Tools*

#### *Fagerstrom Test for Nicotine Dependency*

The FNDT is a six-item self-report scale frequently used around the world to determine levels of nicotine addiction (Hearton et al., 1991). Although the test is actually modified from the Fagerstrom Tolerance Questionnaire, it has better internal consistency and is more easily answered. In terms of the overall logic of this test, it is based on number of cigarettes smoked and length of smoking-free periods. The instrument yields a dependency score between 0 (low) and 10 (high).

### *Statistical Analyses*

The cessation rates of both groups were regarded as independent variables. The relations between demographic, smoking features and results of the items were investigated using the Chi-Square test, Pearson correlation analysis, the Independent Samples T-Test and Two-way Repeated Measures ANOVA. Minitab version 10 was used for power analyzes and the two proportions test. All the remaining statistical analyzes were performed on SPSS version 13.0. A $p$ value of $<0.05$ was regarded as statistically significant.

## 3. Results

### *Demographic and Smoking Features*

The demographic features of both groups were presented at Table 1. There was no difference between the genders of the participants compared with each group ($x^2$=0.754, p=0.125). The age of the participants in the study group was higher than the ones in the control group (t=1.845, p=0.002). They were 3 years older than the control group. Although there were single participants in the control group there were no dEifference in the marital status of the participants between two groups. However most of the participants were married within both of the groups (p<0.001 respectively).

**Table 1.** The demographic features of the two groups

| Variables | Study Group N, % | Control Group N, % | p |
|---|---|---|---|
| Gender | Men= 48<br>Women= 32 | Men= 120<br>Women= 80 | >0.05 |
| Age (Years) | 39.22±27.25 | 36.19±51.14 | =0.002 |
| Marital Status<br>Single<br>Married<br>Widow<br>Divorced | <br>0, 0%<br>68, 85%<br>6, 7.5%<br>6, 7.5% | <br>10, 5%<br>165, 83.5%<br>12, 6%<br>11, 5.5% | <br><br><0.05<br><br> |
| Total education year (Mean) | 8.75±1.8 | 8.8±1.0 | >0.05 |

The smoking and their treatment features were presented in Table 2. There was no difference between the mean score of FNDT, package/year and mean of former quits attempts of study and control group (t=0.421, p=0.245; t=0.987, p=0.785; t=0.514, p=0.624 respectively). There was no difference between the treatment method ratio of the both groups were received ($x^2$=0.712, p=0.524).

**Table 2.** The Smoking and treatment features of the both groups

| | Study Group | Control Group | p |
|---|---|---|---|
| FNDT* (Mean) | 5.84±2.32 | 5.67±1.78 | >0.05 |
| Package/Year | 24.2±2.4 | 22.1±3.9 | >0.05 |
| Mean number of quit attempts | 1.7±1.3 | 1.8±1.5 | >0.05 |
| Cessation method that selected<br>NRT only<br>Bupropion+NRT<br>Bupropion only<br>Varenicline | <br>13, (16.5%)<br>23, (30.8%)<br>25, (32.3%)<br>29, (36.9%) | <br>35, (17.7%)<br>48, (24.0%)<br>52, (25.7%)<br>65, (32.6%) | <br><br><br><br> |

**FNDT*:** Fagerstrom nicotine dependency test score; **NRT**:** Nicotine replacement therapy

The total quit rate in the study group was 45% (n=36) at the end of the first month and 37% (n=30) at the end of the third month. The quit rate was 35% (n=70) at the end of the first month and 29% (n=58) at the end of the third month. The smokers at the study group had better quit rates at the end of the 1st month ($x^2$=2.568, p=0.008) and at the end of 3rd month ($x^2$=2.248, p<0.001). In a binary logistic regression model, it was seen that children and grandchildren motivation was an independent factor for quit smoking (O.R=1.094, 95%CI, p<0.001). The binary logistic regression model is presented at Table 3.

**Table 3.** The binary logistic regression model

| | B | SE | Wald | P | Exp (B) | 95% CI for Exp (B) |
|---|---|---|---|---|---|---|
| Study group* | 0.745 | 0.320 | 5.741 | 0.001 | 1.094 | 0.998-1.458 |
| FNDT** | 0.025 | 0.30 | 3.245 | 0.085 | 0.954 | 0.869-1.010 |
| Package/ year | 0.120 | 0.047 | 6.407 | 0.01 | 0.887 | 0.808-0.973 |
| Treatment method | 0.029 | 0.026 | 1.178 | 0.278 | 1.029 | 0.997-1.084 |
| Age | 0.020 | 0.034 | 0.081 | 0.776 | 1.008 | 0.934-1.087 |

**Study Group*:** To get children or garndchildren motivation to quit smoking; **FNDT**:** Fagerstrom Nicotine Dependency Test Score

## 4. Discussion

In our study it is revealed that the motivation from children and grandchildren has a potent effect on quit rates of the smokers. For our knowledge this is the first study on this topic. In former studies the importance of social support from different resources just like partners, spouses and close friends are generally studied (Burns et al., 2014). Similar to our results there is evidence that social relations within a family member has also very strong effect on smoking addiction. Also this effect may be either positive or negative on smoking cessation. For instance Gibbons et al. (1996) showed that positive support from spouse's increases quit rates of the smokers at short term. It is also understood that the recurrence of the smokers was also correlated with negative support of the spouses. In this study this phenomena is explained as if the smokers can't find effective behavioral solutions to some long term withdrawal problems such as increased agitation and anger, spouses generally lose their sympathy for their partners 'cessation. They stop giving emotional support for cessation and prefer the person to smoke again in order to balance the same marital relationship. The importance of the content of structured and positive support from spouses is underlined by Mermelstein et al. (1983). They found that the smokers who received support from their spouses had better quit ratios compared with single smokers at 1st, 3rd and 6th months. However we investigated our patients at mid-term (1st and 3rd months). In another study it was also seen that the spouses (or partners) social support was perceived as efficient by the smoker at end of the 1st month after cessation (Cohen and Lichtenstein., 1990). These smokers claimed that this support was vital although there were indications of expectations influencing the effectiveness of received support, none of critical

analyses reached statistical significance.

In a recent Cochrane review it is underlined that the difficulty of investigating the effect of social support is comes from the topics nature and content (Park et al., 2002). In this review it was noted that the recent nine studies about social support of relatives (mostly spouses) in smoking cessation didn't support enough evidence for increasing smoking cessation rate mostly because of their design flaws. The odds ratio for self-reported abstinence at 6-9 months was 1.08 (CI 95%, 0.81-1.44); and at 12 months post-treatment was 1.0 (CI 95%, 0.75-1.34). Similar to these results we find odd ratio for cessation at three months as 1.094 (CI 95%, 0,998-1.458). Most of the studies that mentioned above are based on Partner Interaction Questionnaire (PIQ-20) which is a very effective and reliable tool measuring the negative and positive support of spouses. However Barrera et al. (1986) stated that the level of emotional support which involves providing empathetic, caring, and reassuring communication and its perception by smokers is very individualized. We didn't use PIQ-20 in our study for some reasons. First of all PIQ-20 is designed for spouses (or partners). Also this questionnaire is mostly investigated the effect and amount of social support after a smoker gave cessation decision.

The common point of participants in the study group was somehow their motivation was directly related with a child or grandchild. The nature, type or style of motivations that our study group get was very heterogeneous. The motivation type or style was mostly depended on the descendants' age. Some of the smokers in the study group (All man) stated that their wife was pregnant and the baby's delivery would be soon. They didn't want to give any harm to their unborn child and they want to be nonsmoker when the baby is born. Very young toddlers (Mostly grandchildren) tended to behave a negative attitude against their grandparents smoking. They mostly refused to socialize with their grandparents (refuse to kiss, play with or sit on the knee etc.) because of bad tobacco smell on the clothes. This was a very strong motivation for some grandparents. However the older children or grandchildren were mostly concerned about their parent's health status. During face to face interview these smokers stated that the most important sentence their children used to motivate them was "Dad/Mom I don't want you die, please stop smoking". This situation may be attributed to the education in pre-kindergarten and primary school about the smoking's effect on health. Educative television commercials underlining the negative effect of smoking on health may be also effective for public opinion about smoking. A decade ago before several prohibitions about smoking is taken as a policy around the world multimedia had a powerful effect on motivating children for smoking (Ford Jones., 2003). Today this media instrument can be used against smoking.

This study had some flaws. First of all we didn't investigated the content and nature of the motivation that the study group had received for the whole cessation period. As stated above the nature of this initial prime motivation nature may be very different from each other. Our knowledge about this nature and content was rather subjective however providing very important and powerful clues about the topic. Also we don't know the effect of this kind of motivation on the rate of smokers to give their decision to quit. What we learned from this study was the smokers who had this motivation had better cessation ratios compared to others. Primary care physicians may use this relation in order to motivate their patients. "What does your grandchild think about your tobacco smell?" or "What does your child think about your smoking?" might be good questions for motivating smokers in primary care. More quantitative and qualitative research is needed to understand the relation of this motivation with cessation properly.

**Ethics**

Approval for the study was granted by Ondokuz Mayıs Üniversity Ethical Board.

**REFERENCES**

Barrera, M., 1986. Distinctions between social support concepts, measures, and models. Am. J. Community Psychol. 14, 413-445.

Brothers, B.M., and Borrelli, B., 2011. Motivating Latino smokers to quit: Does type of social support matter? Am. J. Health Promot. 25, 96-102.

Cohen, S., 2004. Social relationships and health. Am. Psychol. 59, 676-684.

Cohen, S., Lichtenstein, E., 1990. Partner behaviors that support quitting smoking. J. Consul. Clin. Phsycol. 58, 304-309.

Çelik, İ., Halmatov, M., Sarıçam, H., 2012. Attidues of parents toward child-rearing: A case study. BJSEP. 6, 12-20.

Ford-Jones, A., 2003. Impact of Media use on children and youth. Paediatr. Child Health. 8, 301-306.

Gibbons, F., Gerrard, M., Lando, H., McGovern, P., 1991. Social comparison and smoking cessation: The role of the "typical smoker." J. Exp. Soc. Psychol. 27, 239-258.

Heatherton, T.F., Kozlowski, L.T., Frecker, R.C., Fagerstrom, K.O., 1991. The fagerstrom test for nicotine dependence: A revision of the fagerstrom tolerance questionnaire. Br. J. Addict. 86, 1119-11127.

May S., West R., 2000. Do social support interventions ("buddy systems") aid smoking cessation? A review. Tob. Control. 9:415-422.

May, S., West, R., Hajek, P., McEwen, A., McRobbie, H., 2006. Randomized controlled trial of a social support ('buddy') intervention for smoking cessation. Patient Educ. Couns. 64, 235-241.

Mermelstein, R., Lichtenstein, E., Mclyntre, K., 1983. Partner support and relapse in smoking cessation programs. J. Consul. Clin. Phsycol. 51,456-466.

Park, E.W., Tudiver, F., Schultz, J.K., Campbell, T., 2004. Does enhancing partner support and interaction improve smoking cessation? A meta-analysis. Ann Fam. Med. 2, 170-174.

Park, E., Schultz, J.K., Tudiver, F., Campbell, T., Becker, L., 2002. Enhancing partner support to improve smoking cessation. Cochrane Database Syst. Rev. (1):CD002928.

Prochaska, J.O., Redding, C.A., Evers, K.E., 1997. The transtheoretical model and stages of change. K., Glanz, F.M., Lewis, B.K., Rimer. (Ed.). Health Behavior and Health Education: Theory, Research, and Practice, (p. 60-84). (2. Eds). San Francisco: Jossey-Bass.

Burns, R.J., Rothman, A.J., Fu, S.S., Lindgren, B., Joseph, AM., 2014. The relation between social support and smoking cessation: Revisiting an established measure to improve prediction. Ann Behav. Med. 47, 369-375.

Rindal, D.B., Rush, W.A., Schleyer, T.K., Kirshner, M., Boyle, R.G., Thoele, M.J., Asche, S.E., Thyvalikakath, T., Spallek, H., Durand, E.C., Enstad, C.J., Huntley, C.L., 2013. Computer-assisted guidance for dental office tobacco-cessation counseling: A randomized controlled trial. Am J. Prev. Med. 44, 260-264.

Roopnarine, J.L., Carter, D.B,. 1992. Socialization in diverse cultures. Ablex Publishing Corporation. 1st edition. New Jersey.

Ross, K.C., Gubner, N.R., Tyndale, R.F., Hawk, L.W., Jr, Lerman, C., George, T.P., Cinciripini, P., Schnoll, R.A., Benowitz, N.L., 2016. Racial differences in relationship between rate of nicotine metabolism and nicotine intake from cigarette smoking. Pharmacol. Biochem. Behav. 148, 1-7.

Westmaas, J.L., Bontempts-Jones, J., Bauer, J.E. 2010. Social support in smoking cessation: Reconciling theory and evidence. Nicotine Tob. Res. 12, 695-707.

WHO. 2008. Report on the global tobacco epidemic (MPOWER), WHO, Geneva.

Yalcin, B.M., Unal, M., Pirdal, H., Karahan, T.F., 2014. Effects of an anger management and stress control program on smoking cessation: A randomized controlled trial. J. Am Board Fam. Med. 27, 645-660.

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**Technical/Special note**

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# Does the Fagerstrom Nicotine Dependency Test suit every smoker in the waiting room?

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**ABSTRACT**

Fagerstrom Nicotine Dependency Test (FNTD) is a vital test in smoking cessation services. It determines the nicotine addiction level of the patients. The test consists of six items, and a smoker can get a score between 1 to 10. A higher score indicates a higher level of dependency. The result of this test is one of the key components that we use to determine and discuss the best possible individualized cessation strategy (life style modifications, motivation interview, nicotine replacement therapy or other drugs) with patients according to evidence-based medicine principles. It is known that FNTD is a very effective tool for primary care and has several advantages over other self / physician-rated tests. However in several cases we have encountered several flaws and problems in each of the six items with extreme or unusual cases in our experiences. Also such problematic cases are not uncommon in our patient population. To our experience clinicians have to question every item of FNTD with special care in order to prevent and misjudge. This condition may be resulted to under- or over-rate dependency levels of the smokers. In this short report we attempt to share and discuss some of these problematic situations and our own experiences.



As academics specializing in the field of family medicine, one of our most difficult tasks in the context of health promotion activities is assisting with smoking cessation (Fig. 1). We employ a behavioral change model for our patients and determine the best cessation strategy through discussion with them. We use evidence-based practice (best available knowledge, experience and patent choices) in order to take effective clinical decisions. The Fagerstrom Test for Nicotine Dependence (FNTD) constitutes an important part of "best available knowledge" as we prefer to intensify our approach (adding drugs for cessation) as levels of dependency increase. In providing smoking cessation services for our patients we routinely use FTND face-to-face while collecting information concerning subjects' smoking history. This six-item test is rated between 0 to 10 and indicates the subject's level of nicotine addiction (Heatherton et., 1991). The result of this test is one of the key components that we use to determine and discuss the best possible individualized cessation strategy (life style modifications, motivation interview, nicotine replacement therapy or other drugs) with patients according to evidence-based medicine principles (Anczak and Nogler., 2003; Cahill et al.,

**Fig. 1.** To help smokers to cease smoking is one of the top priorities in primary health care

2013; Stead et al., 2013). Although there are data suggesting that the FNTD correlates with self-rated addiction, some withdrawal symptoms and frequent urges to smoke, we have noted some flaws and problems in using this test with different patient profiles (Heatherton et al., 1989; Chabrol et al., 2005).

The first item in the test is "How soon after you wake up do you smoke your first cigarette?" Nicotine and its metabolites are known to be detoxified by the renal system during sleep, and a smoker's lowest nicotine level will usually be in the mornings. However, this level (the nicotine metabolite ratio) depends on several factors, including race and CYP2A6 enzyme activity rate (Ross et al., 2016). This item tests the patient's withdrawal symptoms based on these data. However, we have been surprised to note that, contrary to expectations, some of our very heavy smokers (>30 cigarettes/per day) do not smoke immediately after they wake up. This situation correlates with patients who wake up and smoke during the night. It may be that due to nicotine boosts at night, these patients' base blood nicotine levels never drop sufficiently to produce cravings just after they wake up. In addition, the perception of time in the morning on the part of some of our patients (mostly retired people) is shaped by the idea of breakfast, and it is not unusual for them to state in reply to this question that they do not smoke until after breakfast (something they are proud of), instead of citing a specific time period. In this case we try to establish what time they usually have breakfast. The second item concerns refraining from smoking in prohibited places. Although this item is intended to test craving symptoms, some smokers interpret it as referring to their attitude to smoking prohibitions and restrictions. Many smokers adopt a defensive posture toward this question and merely respond that they do not break the law. The fourth item, which asks how many cigarettes smokers smoke a day, may not elicit the amount of nicotine in fact consumed over the day. Several of our patients (mostly women) use light cigarettes, which contain lower nicotine levels, but they nevertheless smoke a greater number of cigarettes in order to achieve a specific daily dose of nicotine. This item is also imprecise in terms of smokers who use e-cigarettes, pipes, cigars or hookahs or those who simply do not inhale deeply. Additionally, some smokers in Turkey buy unprocessed tobacco from shops and roll their own cigarettes with no filters. The fifth item inquiries into intensive smoking in the first two hours of the day. Some of our very heavy smokers are people who work at night (such as police officers, security staff, firefighters and health workers). University students who study at night also tend to smoke more at night than in the day. These subjects' carbon dioxide measurements do not correlate with their FNTD scores in our clinic.

As a clinician we prefer to use face-to-face tests instead of lab tests (urine cotinine) or carbon dioxide measurements in order to establish a relationship of trust with our patients. Although the FNTD is a very effective tool (highly valid, reliable and simple) for primary care and has several advantages over other self / physician-rated tests, in several cases we believe that it under- or over-rates dependency levels. Our experience is that it may not be free of all imperfections or include all possible situations in smoking addiction.

**REFERENCES**

Anczak, J.D., Nogler, R.A., 2003. Tobacco cessation in primary care: Maximizing intervention strategies. Clin. Med. Res. 1, 201-216.

Cahill, K., Stevens, S., Perera, R., Lancaster, T., 2013. Pharmacological interventions for smoking cessation: An overview and network meta-analysis. Cochrane Database Syst. Rev. 31, 5:CD009329. doi: 10.1002/14651858.CD009329.pub2.

Chabrol, H., Niezborala, M., Chastan, E., de Leon, J., 2005. Comparison of the heavy smoking index and of the fagerstrom test for nicotine dependence in a sample of 749 cigarette smokers. Addict. Behav. 30, 1474-1477. doi: 10.1016/j.addbeh.2005.02.001.

Heatherton, T., Kozlowski, L., Frecker, R., Fagerström, K., 1991. The Fagerström test for nicotine dependence: A revision of the Fagerström tolerance questionnaire. British J. Adict. 86, 1119-11127. doi: 10.1111/j.1360-0443.1991.tb01879.x.

Heatherton, T.F., Kozlowski, L.T., Frecker, R.C., Rickert, W., Robinson, J., 1989. Measuring the heaviness of smoking: Using self-reported time to the first cigarette of the day and number of cigarettes smoked per day. Br. J. Addict. 84, 791-799. doi: 10.1111/j.1360-0443.1989.tb03059.x.

Ross, K.C., Gubner, N.R., Tyndale, R.F., Hawk, L.W., Lerman, C., George, T.P., 2016. Racial differences in relationship between

rate of nicotine metabolism and nicotine intake from cigarette smoking. Pharmacol. Biochem. Behav. 148, 1-7. doi: 10.1016/j.pbb.2016.05.002.

Stead, L.F., Buitrago, D., Preciado, N., Sanchez, G., Hartmann-Boyce, J., Lancaster, T., 2013. Physician advice for smoking cessation. Cochrane Database Syst. Rev. 31, 5:CD000165. doi: 10.1002/14651858.CD000165.pub4.

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