# Parodontopatija kod pušača

# Periodontal disease in smokers

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KRATAK SADRŽAJ

*Duvan sadrži oko 4000 različitih toksičnih supstanci, od kojih je 40 kancerogeno. Nikotin, toksični alkaloid, najaktivnija je supstanca u duvanu i od njega potiče najveći broj štetnih posledica po organizam, pa i po parodontalna tkiva.*

*Cilj rada bio je da ukaže na štetno delovanje pušenja na nastanak i tok parodontopatija, odnosno, na probleme koje pušenje uzrokuje u toku i posle terapije ovog oboljenja. Parodontopatija se kod pušača češće javlja u težem obliku nego kod nepušača. Karakteriše se manjim stepenom inflamacije gingive, većim i bržim gubitkom alveolarne kosti i gingivalnog pripoja, dubljim parodontalnim džepovima i većim brojem recesija gingive.*

*Osim toga, kod pušača je smanjen imunološki odgovor koji se izražava kroz poremećaj različitih vidova odbrane. Pušenje negativno utiče i na uspeh nehirurške i hiruruške terapije parodontopatije. Efekat pušenja na uspeh terapije parodontopatije zahteva primenu antiseptika i antibiotika u toku terapija.*

*Parodontolog bi trebalo edukativno da utiče na pacijente u smislu odvikavanja od pušenja i da, na taj način, deluje preventivno na nastanak i progresiju parodontopatije.*

**Ključne reči: parodontopatija, pušenje, terapija parodontopatije**

SUMMARY

*Tobacco contains about 4000 different toxic substances from which almost 40 are proven to be cancerogenic. Nicotine, toxic alkaloid, is the most active substance in tobacco causing major number of harmful consequences for human organism as a whole, and for periodontal tissues as well.*

*The aim of the paper was to show harmful effects of smoking on periodontal disease development, and to point out the problems caused by smoking during and after the periodontal treatment. Periodontal disease occurs in smokers more frequently as opposed to non-smokers. Typically, smokers have lower level of gingival inflammation, more excessive and accelerated loss of alveolar bone and epithelial insertion, deeper periodontal pockets and numerous gingival recessions.*

*Along with that, smokers are carrying a decreased immune response that is expressed through various defense mechanisms. Smoking has negative impact on the outcome of conservative and surgical periodontal therapy. Effects of smoking on periodontal therapy success rate are requiring administration of antiseptic solutions and antibiotics throughout the treatment course.*

*Every periodontologist must influence patients to stop smoking and thus act preventively on occurrence and progress of periodontal disease.*

**Key words: periodontal disease, smoking, periodontal therapy.**

Prema podacima Ministarstva zdravlja Republike Srbije iz 2003 godine, skoro svaki drugi odrasli muškarac (48%) i svaka treća žena (36%) u Srbiji su pušači. Zabrinjavajuće je što je među učenicima od petog do osmog razreda osnovne škole 23,3% pušača.[1]

Kod stanovnika Evropske Unije procenat pušača je 29%. Zastupljenost muškarca je veća ( 34%) u odnosu na žene (24%).[2]

Istraživanja sprovedena između 1995 i 1999 godine pokazuju da 23,5% Amerikanaca su pušači, od toga su 43% srednjoškolci.[3] Poslednjih godina procenat pušača u

According to the data of Ministry of Health of Republic of Serbia from 2003, almost every other adult man (48%) and every third woman (36%) in Serbia were smokers. It is worrisome that among pupils of fifth to eighth grade at the elementary schools, there are 23.3% smokers.[1]

In European Union 29% of the population are smokers. Men are more numerous (34%) compared to women (24%).[2]

Studies conducted between 1995 and 1999 have shown that 23.5% of Americans are smokers, and high-school teenagers are 43% of that number.[3] During the last years, percent of smokers is increasing following aggres-

svetu se povećava, najviše zahvaljujući marketingu, koji je posebno usmeren prema adolescentima.

Navika pušenja se lako i brzo stiče, ali se od pušenja teško odvikava. Statistike pokazuju, da 70% pušača želi prekinuti, ali samo 25 do 34% uspe da se oslobodi pušenja.[4]

Duvanski dim sadrži oko 4000 različitih toksičnih supstanci, od čega su 40 kancerogene. Zato se pušenje povezuje sa nizom bolesti kao što su: kardiovaskularne, plućne, cerebrovaskularne, i posebno sa rakom pluća, grla, usne duplje, mokraćne bešike i pankreasa.Pušenje se dovodi u vezu sa povećanim brojem pobačaja i sve većim brojem novorođenčadi male porođajne težine.[5,6,7]

Nikotin je najaktivnija farmološka komponenta u duvanu. To je otrovni alkaloid koji se apsorbuje inhalacijom preko pluća, preko oralne sluzokože i kože i dospeva u krvotok, a za samo nekoliko minuta i u mozak. Dokazano je da izaziva neku vrstu spokojstva, antidepresivno dejstvo i smanjuje stres kod pušača.[8] Kada osoba jednom postane zavisna na nikotin, želja za stalnim korištenjem nikotina postaje sve veća. Nikotin je jedna od supstanci koja stvara najveću zavisnost, pa većina ljudi koji prestanu da puše, ponovo počinju za nekoliko dana.[9]

Pušenje mnogo košta savremenu civilizaciju. Podaci SZO pokazuju da je pušenje odgovorno za oko 4 miliona smrtnih slučajeva godišnje. To je više od onoga što odnesu sida, tuberkuloza, saobraćajni udesi, ubistava i samoubistava.[10] U Americi 1 od 5 smrtnih slučajeva se pripisuje pušenju. Smatra se da će duvan do 2030 godine biti glavni uzrok smrtnosti i invalidnosti kod svetske populacije od koga će stradati više od 10 miliona ljudi.[11] Ovi statistički podaci su alarmantni, čak ako se uzme u obzir da se pušenje u jednom velikom procentu može sprečiti.

Smoking habit is acquired easy and quickly, but reverse effect is very difficult to achieve. Statistics have shown that 70% of smokers are willing to stop smoking, but only 25% to 34% have succeeded in that attempt.[4]

Tobacco smoke contains around 4000 different toxic substances from which 40 are carcinogenic. For that reason, smoking is associated with many diseases like cardiovascular, lung, cerebro-vascular and, especially, is involved in causation of lung cancer, throat, oral cavity, bladder and pancreatic cancers. Smoking is also associated with an increased number of miscarriages and low birth weight.[5,6,7]

Nicotine is the most active pharmacological component in tobacco. It is a toxic alkaloid absorbed by inhaling through lungs, throuh oral mucosa and skin it reaches blood flow and within minutes reaches brain tissues. It is proven that it has calming effect, anti-depressive and stress relieving effects in smokers.[8] Once a person becomes addicted to nicotine, desire for permanent nicotine use is growing.[9]

WHO data reveal that smoking is death causing in more than 4 million cases per year. It is far more than AIDS, tuberculosis, car accidents, homicides and suicides.[10] In USA one of five deaths is due to smoking. It is considered that by the year 2030 smoking will be the main death and invalidity cause in entire world population affecting more than 10 million people.[11] This statistical data should be taken as a warning even though smoking can be to the great extent prevented.

The aim of this paper was to show harmful effects of smoking on periodontal diseases development, and on problems caused by smoking during the treatment of the disease.

## Faktori koji utiču na učestalost i težinu Kliničke slike parodontopatije kod pušača

Parodontopatija je tri puta češće kod pušača nego kod nepušača, bez obzira na nivo oralne higijene.[10,11] Pored toga, ona kod pušača ima mnogo težu kliničku sliku nego kod nepušača.[12]

Bergstrom i sar.[13] su ispitivali stanje parodoncijuma pomoću kliničkih i rendgenoloških parametara kod 257 pušača, starosti između 20 i 60 godina. Ustanovili su da je ono kod pušača mnogo lošije nego kod nepušača. Takođe je ustanovljeno da je efekat pušenja na parodoncijum kumulativan. Zato, negativne posledice pušenja na potporni aparat zuba zavise od dužine pušenja i broja cigareta.

Clasina i sard.[14] su ispitivali uticaj pušenja na parodontalna tkiva. Ispitivanje je sprovedeno kod 240 pacijenata, a praćeni su dubina parodontalnih džepova, gingivalne recesije, nivo kliničkog pripoja, labavljenje

## Factors affecting prevalence and clinical symptoms of periodontal disease in smokers

Studies have shown that periodontal disease is three times more frequent in smokers compared to non smokers no matter the oral hygiene level.[10,11] In addition, periodontal disease in smokers is characterized by heavier clinical symptoms than in non smokers.[12] Bergstrom et al.[13] were investigating periodontal tissues by clinical and radiological parameters in 257 smokers, ages 20 to 60 years old, and have assessed that they were much more impaired than in non smokers. They have also observed cumulative effect of smoking on periodontal tissues. That is the reason why negative consequences of smoking on periodontium depend on smoking duration and number of cigarettes.

Clasina et al.[14] also have studied influence of smoking on periodontal tissues. The study was conducted in 240 patients and followed up were the depth of periodon-

zuba, indeks krvarenja i plak indeks. Sve vrednosti praćenih kliničkih parametara su bile veće kod aktivnih i bivših pušača, nego kod nepušača. Ustanovljeno je da aktivni pušači imaju 2,7 puta, a bivši pušači 2,3 puta veću mogfućnost da dobiju parodontopatiju nego nepušači.

Parodontopatija kod pušača, u upoređenju sa nepušačima, ima neke posebne kliničke karakteristike. Ona ima težu kliničku sliku, koja se manifestuje većim brojem dubokih džepova, većim gubitkom pripoja i prisustvom većeg broja recesija gingive[10,11,14]

Kao posebna karakteristika parodontopatije kod pušača izdvaja se brz gubitak alveolarne kosti i veća zahvaćenost furkacija, što rezultira gubitkom većeg broja zuba u kraćem vremenskom periodu.[15]

Šveđani Jonsson i Lavsted[16] su prospektivnoj studiji, koja je trajala 20 godina, (od 1970 do 1990 godine), pratili gubitak marginalne alveolarne kosti i zuba kod pušača. Ustanovili su da pušenje ubrzava gubitak alveolarne kosti kod aktivnih pušača. Osobe koje su u periodu ispitivanja prestale da puše , imale su manji gubitak kosti. Nađen je veći broj izgubljenih zuba kod pušača nego kod nepušača, mada razlika nije bila statistički značajna.

Pušači imaju dva puta veći gubitak gingivalnog pripoja (4,75 mm u odnosu na nepušače 2,05 mm), i gubitak kosti (7,28 mm u odnosu 3,25)[17]

Interesantno je da kod pušača postoji manji stepen inflamacije gingive, uključujući smanjen stepen krvarenja, za razliku od drugih kliničkih znakova parodontopatije, koji su značajno izraženi.[18] Smatra se da je to posledica vazokonstrikcije krvnih sudova gingive, koja je izazvana nikotinom. Histološka istraživanja pokazuju da postoji manji broj krvnih sudova u gingivi pušača nego u gingivi nepušača.[19]

Štetno delovanje duvana na tkiva parodoncijuma uglavnom potiče od toksičnog delovanja nikotina. Istraživanja pokazuju da se nikotin apsorbuje u oralna tkiva. Nađen je i na površini korena zuba pušača. Najveća količina nikotina, a takođe i najznačajnija, nađena je u serumu, pljuvačci i gingivalnoj tečnosti.[8]

Dokazano je da fibroblasti izloženi različitim koncentracijama nikotina pokazuju smanjenu proliferaciju, migraciju i smanjenu mogućnost pripajanja za površinu korena zuba.[20] Kada je poremećena funkcija fibroblasta dolazi do smanjene produkcije proteina i sinteze kolagena, što se negativno odražava na metabolizam svih parodontalnih tkiva. Najverovatnije da citotoksično delovanje duvanskih sastojaka, posebno nikotina, na parodontalna tkiva rezultira pojavom težih oblika parodontopatije kod pušača.[11,12,13]

Istraživanja pokazuju da pušači, za razliku od nepušača, imaju više vrsta bakterija koje se mogu dovesti u vezu sa parodontopatijom: *A. actinomycetemcomitans, B forsytthus, Porphyromonas gingivalis*.[21] *Pervotela intermedia, Fusobacterium nucleatum, Streptococcus micros, Streptococcus aureus* [22]

tal pockets, gingival recessions, epithelial insertion, teeth luxation, index of gingival bleeding and plaque index. All values of clinical parameters were higher in active and former smokers than in non smokers. It was established that active smokers are at a 2.7 times and former smokers 2.3 times higher risk of periodontal disease compared to non smokers.

Periodontal disease in smokers, when compared to non smokers, has some special clinical characteristic. Clinical symptoms are heavier and manifested by higher number of deep pockets, more excessive loss of epithelial insertion and presence of higher number of gingival recessions.[10,11,14]. Particular characteristic of periodontal disease in smokers is accelerated alveolar bone loss and greater impact on furcation which is resulting in a loss of more teeth in a shorter time period.[15]

Swedish authors Jonsson and Lavsted[16] in prospective study over 20 years (1970-1990) were following up the loss of marginal alveolar bone and teeth in smokers. They have assessed that smoking accelerates alveolar bone loss in active smokers. Persons who have stopped smoking during the study suffered less bone loss. Greater number of lost teeth was in smokers than in non smokers though the difference was not statistically significant. Gingival insertion loss was two times higher in smokers (4.75mm compared to non smokers 2.05mm) and bone loss (7.28mm compared to 3.25mm).[17]

Interestingly, as opposed to other clinical periodontal signs, in smokers gingival inflammation and gingival bleeding are decreased.[18] It is considered to be the consequence of gingival vasoconstriction caused by nicotine. Histological studies have shown that there is a lower number of blood vessels in the gingival of smokers than non smokers.[19] Harmful effects of tobacco on periodontal tissues originate from toxic effects of nicotine and studies show that nicotine is absorbed in oral tissues. It was found on the root surface of teeth in smokers. The highest amount, however, was detected in blood serum, saliva and gingival fluid.[8]

It was proven that fibroblasts that were exposed to different nicotine concentrations have lower proliferation, migration and impaired capability of attaching to the dental root surface.[20] When fibroblasts function is impaired protein production and collagen synthesis are decreased which is resulting in decreased metabolism of all periodontal tissues. It is most probable that cytotoxic effect of tobacco components, especially nicotine on periodontal tissues is to be blamed for aggravated forms of periodontal disease in smokers.[11,12,13]

Studies have shown that smokers unlike non smokers harbor more bacterial species associated with periodontal disease: *Actinomyces actinomycetemcomitans, Bacillus forsythus, Porphyromonas gingivalis*[21], *Prevotela intermedia, Fusobacterium nucleatum, Streptotoccus micros, Staphylococcus aureus*.[22]

Neke promene u ustima pušača,kao što je smanjenje kiseonika, pogoduju razvoju anaeroba u subgingivalnom sulkusu i parodontalnom džepu. Još jedna pogodnost postoji za razvoj bakterija u ustima pušača a to je njihova povećana adherencija za ćelije i površinu epitela[23]

Ustanovljeno je da pušači imaju manju količinu gigivalne tečnosti od nepušača. Gingivalna tečnost, koja cirkuliše u sulkusu ili džepu, sadrži različite obrambene komponente, koje su važne za zaštitu parodontalnih tkiva. Jedna od osnovnih odbrambenih faktora parodontalnih tkiva su PMN leukociti, ali njihov broj je smanjen u gingivalnoj tečnosti kod pušača.[24]

Osnovni faktor koji utiče na destrukciju parodontalnih tkiva kod pušača je imunosupresija, koja se odvija kroz poremećaj različitih vidova odbrane.

Aktivnost PMN leukocita,hemotaksa i fagocitoza su značajno smanjene kod pušača,a to predstavlja veoma važnu prvu liniju odbrane od patogenih oralnih mikroorganizama.[25] Pušači imaju i smanjenu produkciju imunoglobulina, koji su veoma važni za opsonizaciju bakterija. Takođe imaju smanjen broj imunoregulatora T ćelija. Kao rezultat toga kod pušača je poremećen specifični i nespecifični imuni odgovor.[26]

Takođe je ustanovljeno da se oslobađa veća količina elastaze,matriks metalo proteinaza i njihovih inhibitora alfa 1 antitripsina i alfa 2 makroglobulina u tkivu pušača, što može da vodi povećanoj destrukciji parodontalnih tkiva. U najnovijim istraživanjima je ustanovljeno da su matriks metalo proteinaze moćani enzimi koji vrši destrukciju vezivnog tkiva parodoncijuma.[27]

Pušene smanjuje nivo većine klasa imunoglobulina, osim IgE i stimuliše produkciju citokina među kojima su najvažniji interleukini.[28] Interleukini su citokini koji vrše itercelularnu transmisiju, dajući odrećene signale ostalim ćelijama u tkivu. Ta komunikacija je najčešća između leukocita i drugih ćelija koje učestvuju u upalnim procesima, kao što su ćelije endotela i fibroblasti.[29]

Grupa interleukina N1 gena je najznačajniji genetski faktor rizika za parodontopatiju. Produkcija interleukina je delimično regulisana ovim genima. Spoljni faktori, kao pušenje, mogu da utiču na polimorfizam gena, i time neke osobe učine podložnijim za parodontopatiju od drugih.[30]

Kormman i sar.[31] su ustanovili povećan rizik za parodontopatiju u osoba koje su imale genotip grupe gena IL1-alfa i beta, kao i IL1-RN. Dokazano je da IL1 alfa i beta pokazuju značajnu pozitivnu korelaciju sa pušenjem, povećanim gubitkom gingivalnog pripoja i alveolarne kosti.

Bonstrom i sar[32] su ispitivali nivo proinflamatornih citokina IL-6 i TNF alfa u gingivalnoj tečnosti kod 75 pušača i 35 nepušača. Ustanovili su da se nivo IL-6 u gingivalnoj tečnosti pušača i nepušača nije značajno razlikovao, dok je nivo TNF alfa bio mnogo veći kod pušača nego kod nepušača. Tumor nekrozis faktor ima važnu ulogu u destrukciji parodotalnog tkiva.

There are certain changes in smokers' oral cavity, like decreased oxygen levels that are convenient for anaerobs development in subgingival sulcus and periodontal pocket. Bacterial adherence to cells and epithelial surface is enhanced in smokers' oral cavity.[23]

It is known that smokers have lower amount of gingival fluid compared to non smokers. Gingival fluid, circulating in crevix or in the pocket, contains various defense components important for periodontal tissues. One of the main defense factors in periodontal tissues are PMN leukocytes but their number is decreased in gingival fluid of smokers.[24]

The main factor influencing destruction of periodontal tissues in smokers is immunosuppression reflected through disturbed defense mechanisms.

Activity of PMN leukocytes, chaemotaxis and fagocitosis are significantly decreased in smokers and those are the first line of defense against oral pathogens.[25] Immunoglobulins production, important for opsonization of bacteria is also decreased in smokers. Smokers have lower number of T-cell immunoregulators. As a result of that, specific and non specific immune response is altered in smokers.[26]

It was also found that higher amounts of elastase, matrix metal-proteinase, and their inhibitors – α-1-antitrypsin and α-2-macroglobuline are excreted in smokers' tissues which can consequently lead to periodontal tissue destruction. In recent studies it was shown that that matrix metal-proteinase is powerful enzyme capable of destructing periodontal connective tissues.[27]

Smoking is decreasing levels of majority of immunoglobulins classes, except IgE and is stimulating production of cytokines among which the most important are interleukins.[28] Interleukins are cytokines responsible for intercellular transmission and for certain signals to the other cells in tissues. That communication is most common between leukocytes and other cells participating in inflammatory processes, like endothelial cells or fibroblasts.[29] Group of interleukins of N1 gene is the most important genetic risk factor for periodontal disease. Production of interleukins is partially regulated by these genes. Smoking can influence genes' polymorphism and make some persons prone for periodontal diseases.[30]

Komman et al.[31] have found higher risk for periodontal disease in persons with genotype of genes' group IL1-α and β, as well as IL 1-RN. It is proven that IL 1-α and β are in positive correlation with smoking incidence, increased loss of the gingival insertion and alveolar bone.

Bonstrom et al.[32] were studying the level of proinflammatory cytokines IL-6 and TNF-α in gingival fluid in 75 smokers and 35 non smokers. Levels of IL-6 in gingival fluid of smokers and non smokers did not differ significantly while TNF-α was much higher in smokers than in non smokers. Tumor necrosis factor has, also, an important role in destruction of periodontal tissues.

Dokazano je da pušači, koji imaju parodontopatiju, imaju ubrzan gubitak alveolarne kosri. Henemyre i sar.[33] su ispitivali delovanje nikotina na resorpciju kosti. Eksperimentalni model su predstavljali svinjski osteoklasti, koji su postavljeni na kalcijumfosfatnu podlogu. Njima je dodavan čist nikotin u različitim razblaženjima. Određenim metodama merena je resorpcija kalcijumfosfata. Ustanovljeno je da nikotin stimuliše diferencijaciju osteoklasta a time i resorpciju kalcijumfosfata, glavnog sastojka kostiju. Uočeno je da se pri većoj koncentraciji nikotina povećava broj osteoklasta,ćelija koje su odgovorne za resorpciju i remodelaciju kosti kod hirurških zahvata na parodoncijumu i u toku parodontopatije.

U jednoj obimnijoj studiji[34] razmatran je uticaj pušenja na pojavu kardiovaskularnih oboljenja i infarkta miokarda i parodontopatije i njihova međusobna povezanost. Sugerisano je da je pušenje značajan faktor rizika i za kardiovaskularna oboljenja i za parodontopatiju, ali su srčani infarkti, posebno kod mlađih osoba koje su imale parodontopatiju, mnogo dramatičniji nego kod onih koji nisu imali parodontopatiju. U diskusiji ove studije je ostalo nerazjašnjeno pitanje uticaja pušenja na mikrocirkulaciju srčanog mišića i parodoncijuma. Može li to, zapravo biti zajednički faktor rizika za jedno i drugo oboljenje. Stoga se sugerišu dalja istraživanja ovog problema.

## Uticaj pušenja na uspeh terapije parodontopatije

Parodontalna tkiva imaju veliki regenerativni potencijal, što je vrlo važno za uspeh u terapiji parodontopatije. Međutim, pušenje je važan faktor koji usporava zarastanje rana u opštoj hirurgiji.Posebno je taj negativan uticaj izražen u oralnoj implantologiji i parodontalnoj hirurgiji, jer su oralna tkiva direkto izložena štetnim sastojcima duvanskog dima.[35]

Pušenje ima negativan uticaj i u toku primene nehirurške terapije parodontopatije. Kod primene nehirurške terapije parodontopatije želi se postići eliminacija inflamacije gingive, povećanje nivoa epitelnog pripoja kao i poboljšanje njegovog kvaliteta i smanjenje dubine parodontalnih džepova. Na ovaj način se zaustavlja destrukcija parodntalnih tkiva.

Istraživanja pokazuju da se kod pušača, u toku nehirurške terapije parodontopatije postiže manja redukcija dubine parodontalnih džepova i manji nivo epitelnog pripoja.[36,37,38] Postignuta redukcija dubine džepova u toku nehirurške terapije parodontopatije, u proseku je za 0,5mm manja kod pušača za razliku od nepušača. Rezultati nehirurškog lečenja parodontopatije mogu se donekle poboljšati primenom antibiotika.[39]

It is proven that smokers with periodontal disease have accelerated loss of alveolar bone. Henemyre et al.[33] were investigating effects of nicotine on bone resorption. Experimental model were pig's osteoclasts planted on calcium-phosphate matrix. They were immersed in pure nicotine in different concentrations. Resorption of calcium-phosphate was measured by applied methods. It was estimated that nicotine is stimulating differentiation of osteoclasts and, thus, the resorption of calcium-phosphate, the main bone component. It was observed that with increasing nicotine concentration the number of osteoclasts is increasing as well, and osteoclasts are cells responsible for bone resorption and remodeling in periodontal surgical attempts and during the periodontal disease process.

In a large study[34], effect of smoking on cardiovascular diseases, heart infarction and periodontal diseases occurrence and their correlation was investigated. It was suggested that smoking is important risk factor, both for cardiovascular and periodontal diseases, but heart infarctions, especially in younger patients with periodontal diseases, are with much heavier course comparing to patients without periodontal disease. In discussion, authors have not clarified the question of smoking influence on microcirculation of the heart muscle and periodontal tissues, and if there can be found common risk factors for both diseases. For that, further investigations are recommended.

## Effects of smoking on periodontal therapy outcome

Periodontal tissues harbor a great regenerative potential which is very important in periodontal therapy. However, smoking is important in impairing wound healing in surgery. That is particularly obvious in oral implantology and periodontal surgery, because oral tissues are directly exposed to harmful effects of tobacco smoke.[35]

Smoking has negative impact during conservative periodontal treatment. During conservative procedures, the aim is to eliminate gingival inflammation, increase the level of epithelial insertion and enhance its quality, as well as decrease the depth of periodontal pockets. In this way, destruction of periodontal tissues can be arrested.

Studies have shown that in smokers, during conservative periodontal therapy, less reduction of periodontal pockets depth and lower level of epithelial insertion are achieved.[36,37,38] Depth of periodontal pockets achieved during non surgical periodontal therapy is 0.5mm less in smokers. Results of this kind of treatment can be supported to some extent with antibiotic administration.[39]

Considering that periodontal treatment in smokers is less successful, some authors suggest introduction of antiseptics and antibiotics. For that purpose, effect of triclosan

Kako parodontopatija kod pušača slabije reaguje na terapiju, neki autori predlažu da se ona dopuni sa antiseptičima i antibioticima. U tom cilju je ispitan uticaj antiseptika triclosana u toku nehirurške terapiji parodontopatije kod 60 pušača.[40] Uticaj triklosana, koji je primenjen u pasti za zube, na terapiju parodontopatije praćen je pomoću parodontalnih parametara. U dvogodišnjem eksperimentu ustanovljeno je da triklosan značajno utiče na pozitivan uspeh u terapiji parodontopatije kod pušača.

Kod primene raznih hirurških metoda u terapiji parodontopatije, negativno dejstvo pušenja je još više izraženo, zbog usporenog zarastanja rana. Kod pušača kod kojih je urađena Modifikovana Widmanova režan operacija, postiže se mnogo manja redukcija dubine parodontalnih džepova i nivoa pripoja nego kod nepušača.[41] Dokazano je da pušenje usporava zarastanje posle primene regenerativne terapije gde se koriste implantati i membrane za vođenu regeneraciju tkiva. Oko 80% neuspeha kod tretmana furkacionih defekata pripisuje se dejstvu štetnih sastojaka duvanskog dima. Ovi neuspesi se manifestuju usporenim zarastanjem hirurške rane ili gubitkom kosti i implantata, za razliku od nepušača gde je taj neuspeh svega 10%. Čak se smatra da pušenje više utiče na gubitak kosti nego loša oralna higijena.[42]

Pušenje značajno smanjuje procenat prekrivenosti ogolićenih korenova zuba posle izvedenog prekrivanja.[43] Ovo najverovatnije iz razloga što se nikotin taloži na površini korena zuba delujući toksično na fibroblaste i onemogućujući formiranje novog kvalitetnijeg pripoja između vezivnog tkiva gingive i površine korena zuba.[44]

## Zaključak

Nesumnjivo je dokazano da u duvanu i duvanskom dimu ima veliki broj toksičnih i kancerogenih supstanci. Ove supstance, među kojima je farmakološki najaktivniji nikotin, štetno utiču i na tkiva parodoncijuma. Zbog toga se parodontopatije češće javljaju i u težem obliku kod pušača nego kod nepušača. Pušenje, takođe, negativno deluje i na uspeh nehirurške i hirurške terapije parodontopatije. Negativni efekat pušenja je više izražen kod hirurške nego kod nehirurške terapije, posebno u slučajevima primene regenerativne terapije parodontopatije. Terapiju parodontopatija kod pušača treba dopuniti primenom antiseptika i antibiotika.

Parodontolog bi trebalo, pre svega, da edukativno utiče na pacijente, u smislu odvikavanja od pušenja i da, na taj način, deluje preventivno na nastanak i progresiju parodontopatije.

over a non surgical periodontal therapy was investigated in 60 smokers.[40] The effect of triclosan, in a toothpaste was followed up according to various periodontal parameters. In a two-year experiment, it was observed that triclosan is significantly influencing the successful outcome of periodontal therapy in smokers.

When surgical methods in periodontal treatment are applied, negative impact of smoking is even more expressed for impaired wound healing. In smokers who underwent modified Widmann flap procedure, less reduction of pocket depth can be achieved and lower insertion level than in non smokers.[41] It was proven that smoking is affecting wound healing after regenerative therapy when implants and membranes are used for guided tissue regeneration. Almost 80% of failure in treating furcational defects is due to negative effects of components from tobacco smoke. These failures are manifested by impaired wound healing or bone and implant loss comparing in non smoking patients where failure rate does not exceed 10%. It is considered that smoking is affecting bone loss more than poor oral hygiene.[42]

Smoking is decreasing significantly percentage of covered dental roots following covering procedures.[43] This is due to nicotine precipitation on the root surfaces and its negative effect on fibroblasts and formation of the new insertion between connective gingival tissue and dental root surface.[44]

## Conclusion

It is proven without doubt that in tobacco and tobacco smoke there are many toxic and cancerogenic substances. These components, among which the most active pharmacologically is nicotine, influence periodontal tissues as well. Because of this, periodontal diseases occur more frequently in smokers than in non smokers. Smoking is also negatively affecting the outcome of non surgical and surgical periodontal treatment. Negative impact of smoking is more evident after surgical than following non surgical therapy, especially when periodontal regenerative procedures were applied. Periodontal therapy in smokers should be added by antiseptic and antibiotic administration.

A periodontologist should educate patients to stop smoking and act preventively on occurrence and progression of periodontal disease.

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