CARIES PREVALENCE IN HEROIN ADDICTS

Nives Protrka[1], Marina Katunarić[2], Irina Filipović[3] and Željko Verzak[4]

[1]Private Dental Practice; [2]Department of Endodontics and Restorative Dentistry, [3]Department of Oral Surgery, [4]Department of Pediatric Dentistry, School of Dental Medicine, University of Zagreb, Zagreb, Croatia

SUMMARY – The aim of the study was to determine the prevalence of caries in patients who are heroin addicts and the effects of salivary flow and saliva pH on its prevalence. The study included 200 subjects (100 subjects diagnosed with heroin addiction as the test group and 100 subjects taking no addictive substances as the control group). In both groups, saliva status was established by determining flow rates and pH of stimulated and unstimulated saliva. The total number of dental caries cases was determined using the DMFT index and their more specific detection in the context of localization on the tooth using the DMFS index. The DMFT index was higher in the test group (mean 18.78) than in the control group (mean 5.32), with a statistically significant difference ($P<0.000$). The highest caries prevalence in both groups was recorded on occlusal dental surfaces (44.70% in the test group and 16.20% in the control group). The prevalence of caries of vestibular dental surfaces was higher in the test group (9.40%) than in the control group (2.34%), and the difference was statistically significant ($P<0.000$). A statistically significant correlation was established between heroin addiction and reduced production of unstimulated saliva ($P<0.001$) and stimulated saliva ($P<0.002$), as well as pH values of stimulated saliva ($P<0.001$) and unstimulated saliva ($P<0.001$). In conclusion, a higher prevalence of caries of hard dental tissue was found in the test group. The flow of stimulated and unstimulated saliva as was as saliva pH was lower in the test group.

Key words: *Dental caries; Heroin dependence – complications; Saliva – physiology; Tooth demineralization*

Introduction

Caries is a unique multifactorial disease of hard dental tissues. Understanding of the etiologic factors in caries formation and the efficiency of prophylactic methods has made it possible to put the disease under control. However, answers to important questions such as how to predict who will develop caries, why immunity to caries has not developed, etc., are yet unavailable. Previous as well as present concepts of caries formation are based on the chemical process of fermentation of carbohydrates (glucose, fructose, sucrose) by bacteria, which results in acid formation, thus lowering pH of the plaque, which, in turn, leads to demineralization of the hard dental tissues[1-5]. The bacterial composition of the plaque is relatively stable thanks to the synergic and antagonist microbiological interaction. However, frequent exposure of the plaque to low pH may lead to a reduction in the number of the bacteria sensitive to low pH with the resulting increase in the count of *Streptococcus mutans* and *Lactobacillus*[6]. This changes the microbiological flora from gram-positive to gram-negative, chiefly anaerobic saccharolytic microorganisms[7-9]. Even though *Streptococcus mutans* is the most frequently researched cariogenic microorganism, it is only one of over 500 types of microorganisms that can be found in dental plaque[5].

Saliva has a protective effect on the health of hard and soft oral cavity tissues[10]. Chronically reduced salivation is one of the most important factors that

Correspondence to: *Assoc. Prof. Marina Katunarić, MSc, PhD*, Department of Endodontics and Restorative Dentistry, School of Dental Medicine, University of Zagreb, Gundulićeva 5, HR-10000 Zagreb, Croatia
E-mail: katunaric@sfzg.hr

Received March 19, 2013, accepted October 30, 2013

increase the risk of caries[11]. Determining the parameters of saliva is therefore an important factor in determining caries development risks. It is a generally accepted fact that saliva and its components play an important role in the preservation of oral health, but caries development depends on a number of factors, prominent among which are defense mechanisms of the entire body, the surface morphology of the tooth, general physical health, diet, hormonal status, oral hygiene, bad habits, and so on[4]. The authors of previous studies believed that genetic factors such as tooth morphology, occlusions, time of tooth eruption, etc. played a lesser role in caries development than dietary and microbiological factors did[12,13]. However, results of more recent studies in twin populations have shown that genetic factors also play an important role in caries development risk[14,15]. Smoking and alcohol intake can also increase the risk of caries development, as can the presence of various diseases (diabetes, Sjögren's disease, etc.) that can result in xerostomia, as well as medicine intake (antihistamines, antidepressants)[16-18]. In the past several years, various authors have described pathologic changes in soft and hard tissues in the oral cavity in drug addicts and correlated higher prevalence of caries with the intake of certain addictive substances[19-24]. It is well known that opioids reduce gastric secretion but whether they reduce salivation[25], one of the most important protective factors in defense against caries, has not been proven. Caries may also appear as a result of disregard of one's general health[26], including poor oral hygiene[27]. Excessive intake of carbohydrates[28,29] is a consequence of excessive need for sugar intake in addicts[30]. However, the exact mechanism of the higher prevalence has not been fully determined. Considering the increasingly widespread intake of various addictive substances, drug addiction represents a problem in modern society. The consequences of drug abuse are highly detrimental to the individual's physical and mental health. Reference to the available literature indicates the lack of information on the oral status of addicts, including those in Croatia. This study included only the patients who are addicted to heroin with the goal to have a higher quality sample and more accurate research results. The objective of this study was to determine the prevalence of caries in heroin addicts in the test group and control group. Moreover, it was necessary to analyze the influence of stimulated and unstimulated saliva pH in the test group and control group as well as the influence of the stimulated salivary flow on the prevalence of caries in both groups.

Subjects and Methods

Study population

The study included 200 subjects, 100 in the test group and 100 in the control group. The total sample included 94 women and 106 men. The mean age was 30.15 years in the test group and 27.5 years in the control group. The subjects in both groups signed an informed consent to taking part in the study. Test group subjects were on therapy for addiction to opiates at Department of Drug Addiction, Sestre milosrdnice University Hospital Center in Zagreb, Croatia, the Ethics Committee of which provided approval for the study. Control group consisted of patients attending Department of Endodontics and Restorative Dentistry, School of Dental Medicine, University of Zagreb, Zagreb, Croatia, who had never been taking drugs. The study protocol for the control group was obtained from the Ethics Committee of the Zagreb University Hospital Center. Data were collected by general examination of the subjects and filling in a questionnaire for each subject. The questionnaire included the following: general information (name, age, occupation); medical history; intake of sweetened and alcoholic beverages; smoking; and intake of various addictive substances (test group patients also answered questions related to the most frequently taken addictive substance, the period of intake, and mode of intake).

Clinical examination

Wearing a mask, an experienced dental practitioner (N. P.) conducted clinical examination by using a dental explorer and dental mirror under standard dental operating lights. Ten patients were examined for tooth decay on two separate occasions to test reproducibility and agreement between the scores. The intra-examiner reliability had a kappa value of 0.857, which indicated a very high level of agreement. Following compressed air drying, all surfaces were examined in the same order in all subjects, from tooth 18 to 48.

The codes set by the World Health Organization (WHO) are as follows[31] (Table 1):

0 Intact crown without evidence of treated or untreated clinical caries. Since caries preceding cavitation cannot be positively detected, it was ruled out. Intact crowns therefore may include the following defects:
- white stains
- discolored stains hard to the probe
- colored cavities or fissures in the enamel without visible signs of damaged enamel or being soft to the probe
- dark, glossy, firm, spotted enamel areas showing signs of moderate to severe fluorosis lesions due to abrasion

1 Caries recorded as lesion on softened tooth surface, visible cavitation and undermined enamel. This includes teeth with temporary fillings

2 Tooth with filling and caries

3 Tooth with filling without caries – includes teeth restored with crowns because of caries

4 Tooth missing as a consequence of caries

5 Tooth missing for other reasons – used for congenital deficiencies or tooth extraction due to periodontal disease, trauma, or for orthodontic reasons

6 If a stamped tooth includes caries, it is marked as carious

7 Bridge, crown, or shell carrier – used for teeth that are restored or replaced for reasons other than caries; if shell is involved, the tooth is not carious

8 Unerupted tooth crown does not include congenital tooth absence or tooth lost due to trauma

T Trauma represents fracture of a tooth surface as a result of trauma, without evidence of caries

9 Tooth not recorded – used for any erupted tooth that cannot be examined for a certain reason (e.g., orthodontic dividers).

The data obtained from dental status verification allowed for the calculation of two very important indices, Decayed, Missing and Filled Teeth (DMFT) index and Decayed, Missing, and Filled Surfaces (DMFS) index. The D-component encompasses categories 1 and 2, the M-component relates to 4 and 5, and the F-component only includes teeth under code 3. All the teeth and third molars were included, while teeth marked with 6 (dental filling stamps) and 7 (bridge carriers, special crowns or shells) were not included in the DMFT index.

Table 1. Codes used to mark dental status of the tooth[31]

Code	Tooth status
0	Intact
1	Caries
2	Filling with caries
3	Filling without caries
4	Missing due to caries
5	Missing due to other reasons
6	Fissure stamp
7	Prosthetic supplement (bridge, crown, faceted shell)
8	Unerupted tooth
T	Trauma
9	Not recorded

The DMFT shows the representation of dental caries, and the numerical value of caries prevalence is obtained by adding the numbers of carious teeth (D or K), missing teeth (M or E) and teeth with fillings (F or P). If a tooth has both a filling and caries, it is considered to be carious only (D). A more detailed index is the DMFS index, which includes dental surfaces, five in the molars and premolars and four in the front teeth. Registration was conducted by way of filling in a form especially created for the purposes of this study.

Establishing and recording salivary status

Salivary status was determined by measuring salivary flow and determining pH of unstimulated and stimulated saliva in each subject. Buffer capacity was not established since a linear correlation exists between pH of saliva and buffer capacity of saliva, as well as salivary flow[32]. Salivary status was established first in the morning without prior tooth brushing. We asked each patient to take a seat and, from a relaxing position, spit out, without swallowing, the saliva collected within a one-minute period into a graded test tube through a glass funnel. The salivary flow was entered in the form, immediately after which the saliva pH was determined applying the colorimetric method and using a universal pH indicator paper

0-14 (Panpeha, Schleicher&Schuell, Sigma-Aldrich GmbH, Germany). After that, the stimulated salivary flow rate was established by asking the patient to begin chewing 1.5 g of pure paraffin wax (Parafilm, American National Can., Greenwich, Ct., USA) for five minutes and, also without swallowing, spitting all the saliva into a graded test tube through a glass funnel. After entering the stimulated salivary flow in the form, the pH of the sample was immediately established using the same colorimetric method. It was necessary to measure pH of each sample immediately after entering the salivary flow because pH of the saliva exposed to air quickly changes toward alkaline as saliva loses carbon dioxide[33-35].

The unstimulated salivary flow rates were grouped into three categories and thus recorded:

1 = <0.5 mL/min
2 = 0.5-1.0 mL/min
3 = >1.0 mL/min

The stimulated salivary flow rates were also grouped into three categories and thus recorded:

1 = <1.0 mL/min
2 = 1.0-2.0 mL/min
3 = <2.0 mL/min

pH levels of unstimulated saliva were recorded with pH values as follows:

1 = <3
2 = 4
3 = 5
4 = 6
5 = 7
6 = >8

pH levels of stimulated saliva were recorded with pH values as follows:

1 = <5
2 = 5-7
3 = >7.

Data analysis

The data obtained were entered in a database and processed using the SPSS 11.0 statistical package. The database comprised all the data obtained by the questionnaire and clinical examination of the subjects. The sample structure was described in relative frequencies (percentages). The data were initially described in a descriptive statistical manner, depending on the type, and with the use of the χ^2-test. Frequency distribution was provided for qualitative data, while for quantitative data the mean value and standard deviation were calculated, as a dispersion measure. Moreover, the minimum and maximum values of the results were provided. Differences between the groups were tested using t-test on the mean value differences. The accepted value of statistical significance was 95% ($P<0.05$).

Results

Questionnaire results

Analysis of data from the questionnaire showed a statistically significant difference between the test group and control group in the frequency of cigarette smoking, with a mean of 20.58 (SD=10.00) cigarettes *per* day in the test group and 6 cigarettes *per* day (SD=8.43) in the control group. A statistically significant difference was recorded in the frequency of coffee drinking, with a mean of 1-2 cups of coffee *per* day in the test group and only occasional coffee taking in the control group (χ^2=2.482; df=198; $P<0.05$). Data analysis revealed 45% of the test group subjects to suffer from not only their main illness (addiction to opiates) but also from other, mostly mental illnesses (depression, personality disorder, schizophrenia, obsessive-compulsive disorder). Further data analysis included processing the parameters of the type of the drugs taken by the subjects, which revealed heroin, methadone, marijuana, and hashish to be the drugs taken most frequently (several times a day) for the longest period (over five years).

Salivary flow rate and pH

There was significant correlation between the test group and control group according to unstimulated ($P<0.001$) and stimulated ($P<0.002$) salivary flow rates (Table 2). Moreover, significant correlation was established between pH values of unstimulated and stimulated ($P<0.001$ both) salivary flow rates between the test group and control group (Tables 3 and 4).

DMFT and DMFS indices

Dental status of both groups is shown in Table 5. The established frequency of caries, extracted teeth and filled teeth in the test group was 58.70%, as opposed to the control group, in which the frequency

Table 2. Absolute and relative frequencies of unstimulated and stimulated salivary flow rates in test group and control group

Group	Unstimulated salivary flow rate			
	<0.5 mL/min (%)	0.5-1 mL/min (%)	>1 mL/min (%)	Total (%)
Test	38 (38)	61 (61)	1 (1)	100 (100)
Control	21 (21)	64 (64)	15 (15)	100 (100)
	Stimulated salivary flow rate			
Group	<1 mL/min (%)	1-2 mL/min (%)	>2 mL/min (%)	Total (%)
Test	23 (23)	72 (72)	5 (5)	100 (100)
Control	20 (20)	54 (54)	26 (26)	100 (100)

Table 3. Absolute and relative frequencies of some unstimulated saliva pH values in test group and control group

Group	Unstimulated saliva pH					Total (%)
	3 (%)	4 (%)	5 (%)	6 (%)	7 (%)	
Test	2 (2)	1 (1)	41 (41)	49 (49)	7 (7)	100 (100)
Control	0 (0)	0 (0)	1 (1)	20 (20)	79 (79)	100 (100)

Table 4. Absolute and relative frequencies of some stimulated saliva pH values in test group and control group

Group	Stimulated saliva pH			Total (%)
	<5 (%)	5-7 (%)	>7 (%)	
Test	5 (5)	94 (94)	1 (1)	100 (100)
Control	1 (1)	80 (80)	19 (19)	100 (100)

Table 5. Dental disease characteristics in test group and control group

Group	Variable	Mean	SD
Test	DMFT	18.78	7.50
	Decay	12.57	6.30
	Missing	4.59	4.37
	Filling	1.60	2.93
Control	DMFT	5.32	7.84
	Decay	2.00	4.04
	Missing	0.68	1.54
	Filling	2.31	4.52

DMFT = Decayed, Missing and Filled Teeth index

was 16.63%, yielding a statistically significant difference ($P<0.000$). In the test group, the prevalence of caries was 39.40%, extraction 14.34%, and teeth with fillings 5.0%. In the control group, the prevalence of caries was 6.25%, extraction 2.13%, and teeth with fillings 7.21%. Moreover, a statistically significant between-group difference was recorded in the prevalence of caries and extracted teeth ($P<0.000$). Concerning the number of teeth with fillings, there was no statistically significant between-group difference ($P<0.20$), although a higher mean number of teeth with fillings was found in the control group as compared with the test group (7.21% *vs*. 5.0%).

Analysis of the results obtained by using the DMFS index showed caries to occur most frequently on occlusal surfaces in both groups, with a frequency of 16.2% in the control group and 44.7% in the test group. On smooth dental surfaces, it most frequently occurred on the proximal surfaces in both the test group (15.5%) and control group (8.60%). However, although the prevalence of caries was lower on the vestibular surfaces than on other dental surfaces in both groups, a statistically significant difference ($P<0.000$) was established between the groups. The frequency of vestibular surface caries was higher in the test group (9.4%) than in the control group (2.34%).

Discussion

A statistically significant difference was established according to smoking habits, i.e. the test group subjects smoked considerably more than those in the control group ($P<0.01$). The results are consistent with those reported from earlier studies[36]. Cigarettes may have a drying effect on the mouth, that is, they may reduce salivary flow and saliva pH[37]. Although this study did not show a statistically significant difference ($P<0.56$) between the two groups according to consumption of alcoholic beverages, the fact that 5% of the test group subjects had previously suffered from alcoholism should be taken into account because a correlation has been established between alcohol consumption and dental caries development[38].

Considering that the test group subjects were in the rehabilitation process, it was necessary to include the intake of methadone as an opiate agonist in the analysis. Methadone was most frequently ingested, which mode of intake provides direct contact between the drug and oral mucosa. Although the authors had previously established a link between methadone intake and xerostomia, this study did not show a statistically significant difference in the flow of stimulated and unstimulated saliva between the test group subjects having ingested methadone for more than five years and those having done it for less than five years[39]. However, it should be noted that this study used a different sample and different methodology.

Study results showed that the test group subjects secreted on average smaller amounts of unstimulated and stimulated saliva than the control group. Considering that opiate addicts take a number of other addictive substances in addition to heroin, previous literature states various addictive substances that may lead to xerostomia, with hallucinogens and cannabis as the leading representatives[20].

Moreover, the value of unstimulated and stimulated saliva pH in the test group was lower, i.e. their saliva was more acidic. Consequently, caries potential was higher in the test group.

This type of research has not been conducted in Croatia, although the authors of a previous study established the mean DMFT of 7.76[40], and another mean of 9.53[41] in the rest of the population in Croatia. However, they did not assess the prevalence of caries in addicts.

The higher prevalence of caries progression in the test group may be attributed to reduced salivary secretion and a reduced effect of its protective role and a decrease in saliva pH, which promotes the process of enamel demineralization. These results are consistent with the results of a previous study, in which Angelillo *et al*. established a mean DMFT of 12.9 and mean DMFS of 36.2 in the addict group, whereas in our subjects the respective figures were 18.78 and 40.43[27]. As the reason for such results, Angelillo *et al.*[27] state poor Oral Hygiene Index (OHI). However, this study used a different methodology and did not assess OHI but salivary status because reduced saliva secretion can be one of the potential factors in higher caries prevalence. Bearing in mind that addicts take a number of other addictive substances in addition to heroin, previous studies attempted to establish whether a difference existed between amphetamine abusers and heroin abusers and caries prevalence, but no statistically significant difference was established[42]. In 1967, Lowenthal found a higher prevalence of caries on cervical tooth surfaces[43]. This was confirmed by Angelillo *et al.* in their study from 1991[27]. The results obtained in the present study showed the highest prevalence of caries on occlusal surfaces in both groups, but the prevalence of caries in cervical area was higher in the test group, where the difference was statistically significant. This result is consistent with literature data.

Previous studies report that addicts neglect their health[26,44], have a high sugar intake[28,29] and, if the fact is taken into account that they seek help when the situation is very serious, i.e. when symptoms of pain and swelling in the mouth appear[30], it may be said that a different approach is necessary in the addicts' oral cavity repair to alleviate the symptoms as much as possible.

Moreover, previous studies have established that opioids reduce gastric secretion, although not proving whether they reduce salivary secretion as well[25,45]. The results obtained in our study showed that salivary secretion was reduced, which, in addition to poor oral hygiene, may have a powerful effect on caries development. Future studies should therefore pay more attention to establishing the cause of hyposalivation in opiate addicts.

In addition to their main disease, addicts also suffer from other mental disorders and pains, which is believed to be a potential reason for increased fear of dental intervention[46]. Dentists should be aware of the mental state of such patients in order to make cooperation with the patient more successful and to fully implement the therapy plan.

As the latest studies have shown that early detection of drug substance in urine and new therapy approach in the future can have an important role in decreasing the number of drug addicts, it is also important to understand the complexity oral disease development in drug addicts to achieve better prevention and therapy[47,48].

Conclusions

Stimulated and unstimulated salivary flow was lower in the test group, and saliva pH was more acidic. Caries prevalence was considerably higher in the test group than in control group, which may be attributed to reduced salivary secretion and a decrease in saliva pH. Moreover, a higher prevalence of atypical caries of the cervical area on smooth tooth surfaces was recorded in the addicts.

References

1. MILLER W. The presence of bacterial plaques on the surface of teeth and their significance. Dent Cosmos 1902;44:425-46.
2. WILLIAM LJ. A contribution to the study of pathology of enamel. Dent Cosmos 1897;39:169-96.
3. BLACK GV. Dr. Black's conclusions reviewed again. Dent Cosmos 1898;40;440-51.
4. LENANDER-LUMIKARI M, LOIMARANTA V. Saliva and dental caries. Dent Res 2000;14:40-7.
5. ZERO DT, FONTANA M, MARTINEZ-MIER EA *et al*. The biology, prevention, diagnosis and treatment of dental caries. JADA 2009;140(1):255-345.
6. BRADSHAW DJ, McKEE AS, MARSH PD. Effects of carbohydrate pulses and pH on population shifts within oral microbial communities *in vitro*. JDR 1989;68(9):1298-302.
7. Van HOUTE J. Role of micro-organisms in caries etiology. JDR 1994;73(3):672-81.
8. MARSH PD. Microbial ecology of dental plaque and its significance in health and diesase. ADR 1994;8(2):263-71.
9. Van HOUTE J. Microbiological predictors of caries risk. ADR 1993;7(2):87-96.
10. MANDEL ID, WOTMAN S. The salivary secretions in health and disease. Oral Sci Rev 1976;(8):25-47.
11. LEONE CW, OPPENHEIM FG. Physical and chemical aspects of saliva as indicators of risk for dental caries in humans. J Dent Educ 2001;65(10):1054-62.
12. HASSELL TM, HARRIS EL. Genetic influences in caries and periodontal diseases. Crit Rev Oral Biol Med 1995;6(4):319-42.
13. REICH E, LUSSI A, NEWBRUN E. Caries-risk assessment. Int Dent J 1999;49(1):15-26.
14. CONRY JP, MESSER LB, BORAAS JC, AEPPLI DP, BOUCHARD TJ Jr. Dental caries and treatment characteristics in human twins reared apart. Arch Oral Biol 1993;38(11):937-43.
15. BRETZ WA, CORBY PM, MELO MR *et al*. Heritability estimates for dental caries and sucrose sweeteners preference. Arch Oral Biol 2006;51(12):1156-60.
16. MUHAMMAD J, SYED MS, QADER SA, AZHAR A. Dental caries in diabetes mellitus: role of salivary flow rate and minerals. J Diabetes Complicat 2011;25(3):183-6.
17. GUGGENHEIMER J, MOORE PA. Xerostomia: etiology, recognition and treatment. JADA 2003134(1):61-9; quiz 118-9.
18. PETERSEN PE, BOURQEOIS D, OGAWA H, ESTUPINAN-DAY S, NDIAYE C. The global burden of oral diseases and risks to oral health. Bull World Health Organ 2005;83(9):661-9. Epub 2005 Sep 30.
19. Di CUGNO F, PEREC CJ, TOCCI AA. Salivary secretion and dental caries experience in drug addicts. Arch Oral Biol 1981;26(5):363-7.
20. FAZZI M, VESCOVI P, SAVI A, MANFREDI M, PERACCHIA M. The effects of drugs on the oral cavity. Minerva Stomatol 1999;48(10):485-92.
21. DU M, BEDI R, GUO L, CHAMPION J, FAN M, HOLT R. Oral health status of heroin users in a rehabilitation centre in Hubei province, China. Community Dent Health 2001;18(2):94-8.
22. COLON Jr. PG. Dental disease in the narcotic addict. Oral Surg 1972;33(6):905-10.
23. MOLENDIJK B, Ter HORST G, KASBERGEN M, TRUIN G-J, MULDER J. Comm Dent Oral Epidemiol 1996;24(22):117-9.
24. TITSAS A, FERGUSSON MM. Impact of opioid use in dentistry. ADJ 2002;47(2):94-8.
25. ODEH M, OLIVEN A, BASSAN H. Morphine and severe dryness of the lips. Postgrad Med J 1992;68:303-4.
26. AFFINNIH YH. A preliminary study of drug abuse and its mental health and health consequences among addicts in Greater Accra, Ghana. J Psychoactive Drugs 1999;31:395-403.
27. ANGELILLO IF, GRASSO GM, SAGLIOCCO G, VILLARI P, D'ERRICO MM. Dental health in a group of drug addicts in Italy. Community Dent Oral Epidemiol 1991;19(1):36-7.
28. ZADOR D, LYONS WALL PM, WEBSTER I. High sugar intake in a group of women on methadone maintenance in South Western Sydney, Australia. Addiction 1996;91:1053-61.
29. MORABIA A, FABRE J, CHEE E, ZEGER S, ORSAT E, ROBERT A. Diet and opiate addiction: a quantitative assessment of the diet of non-institutionalized opiate addicts. Br J Addict 1989;84:173-80.

30. SANTOLARIA-FERNANDEZ FJ, GOMEZ-SIRVENT JL, GONZALEZ-REIMERS CE *et al.* Nutritional assessment of drug addicts. Drug Alcohol Depend 1995;38:11-8.
31. World Health Organization. Oral Health Surveys; Basic methods. 4th edn. Geneva: World Health Organization, 1997.
32. MILOSEVIC A, AGRAWAL N, REDFEARN P, MAIR L. The occurrence of toothwear in users of Ecstasy methylenedioxymethamphetamine). Community Dent Oral Epidemiol 1999;2(4):283-7.
33. JENKINS GN. The physiology and biochemistry of the mouth. 3rd edn. Oxford, England: Wiley-Blackwell, 1987:311-27.
34. JENKINS G. Salivary effects on plaque pH. In: KLEINBERG I, ELLISON SA, MANDEL ID, editors. Saliva and dental caries. Microbiology Abstracts. New York City: IRL Press, 1979:307.
35. MÄKINEN KK. The use of xylitol in nutritional and medical research with special reference to dental caries. In: SHAW JH, ROUSSOS GG, editors. Proceedings: Sweeteners and Dental Caries. Feeding, Weight & Obesity Abstracts. Washington: Information Retrieval, 1978:193-224.
36. GOSSOP M, MARSDEN J, STEWART D *et al.* Substance use, health and social problems of service users at 54 drug treatment agencies. Intake data from the National Treatment Outcome Research Study. Br J Psychiatry 1998;173:166-71.
37. PARVINEN T. Stimulated salivary flow rate, pH and lactobacillus and yeast concentrations in non-smokers and smokers. Scand J Dent Res 1984;92(4):315-8.
38. JANSSON L. Association between alcohol consumption and dental health. J Clin Periodontol 2008;35(5):379-84.
39. GRAHAM CH, MEECHAN JG. Dental management of patients taking methadone. Dent Update 2005;32(8):477-88, 481-2, 485.
40. BADEL T, AZINOVIĆ Z, KEROS J *et al.* Caries prevalence in the Croatian army. Acta Stomatol Croat 2007;37(4);433-7.
41. BEGO K, NJEMIROVSKIJ V, PELIVAN I. Epidemiological research on oral health in central Dalmatia: a pilot study. Acta Stomatol Croat 2007;41(4):337-44.
42. BROWN C, KRISHNAN S, HURSH K, YU M, JOHNSON P, PAGE K, SHIBOSKI CH. Dental disease prevalence among methamphetamine and heroin users in an urban setting: a pilot study. JADA 2012;143(9):992-1001.
43. LOWENTHAL AH. Atypical caries of the narcotic addicts. Dent Survey 1967;43:44-7.
44. ZAHRANI AA. Dental health status of institutionalized Saudi male chronic drug abusers. Saudi Dent J 2006;18(1):25-31.
45. REISINE T, PASTERNAK G. Opioid analgesics and antagonists. In: HARDMAN JG, LIMBIRD LE, MOLINOFF PB, RUDDON RW, GOODMAN GILLMAN A, editors. Goodman and Gillman's The pharmacological basis of therapeutics. 9th edn. New York: McGraw Hill, 1996:521-55.
46. SCHEUTZ F. Anxiety and dental fear in a group of parenteral drug addicts. Scand J Dent Res 1986;94:241-7.
47. LASIĆ D, UGLEŠIĆ B, ŽULJAN-CVITANOVIĆ M *et al.* False-positive methadone urine drug screen in a patient treated with quetiapine. Acta Clin Croat 2012;51(2):269-72.
48. DOROTIĆ M. Use of monoclonal antibodies in cocaine and heroin addicts. Acta Clin Croat 2005;44(2):237.

Sažetak

UČESTALOST KARIJESA KOD PACIJENATA OVISNIH O HEROINU

N. Protrka, M. Katunarić, I. Filipović i *Ž. Verzak*

Provedena ja studija kako bi se odredila pojavnost karijesa kod pacijenata ovisnih o heroinu i utvrdio utjecaj količine sline i pH sline na njihovu pojavnost. U studiju je bilo uključeno 200 ispitanika (100 ispitanika kojima je dijagnosticirana ovisnost o heroinu činili su testnu skupinu i 100 ispitanika koji nisu uzimali nikakva sredstva ovisnosti činili su kontrolnu skupinu). Status sline obiju skupina ispitanika određen je tako da je utvrđena količina i pH stimulirane i nestimulirane sline. Ukupan broj karijesnih promjena na zubima odredio se pomoću indeksa DMFT, a njihova preciznija identifikacija u smislu lokalizacije na zubu pomoću indeksa DMFS. Indeks DMFT bio je veći kod testne skupine ispitanika (srednja vrijednost 18,78) nego kod kontrolne skupine (srednja vrijednost 5,32) sa statistički značajnom razlikom ($P<0,000$). Kod ispitanika obiju skupina utvrđena je najveća pojavnost karijesa na okluzalnim plohama zuba (testna skupina 44,70% i kontrolna skupina 16,20%). Učestalost karijesa vestibularnih ploha zuba bila je veća kod testne skupine ispitanika (9,40%) nego kod kontrolne skupine ispitanika (2,34%), a razlika se pokazala statistički značajnom ($P<0,000$). Utvrđena je statistički značajna povezanost ovisnosti o heroinu i smanjene količine stvaranja nestimulirane ($P<0,001$) i stimulirane ($P<0,002$) sline te pH stimulirane ($P<0,001$) i nestimulirane ($P<0,001$) sline. U zaključku, kod ispitanika testne skupine utvrđena je veća pojavnost karijesa tvrdih zubnih tkiva. Količina stimulirane i nestimulirane sline bila je manja, a pH sline niži kod testne skupine ispitanika.

Ključne riječi: *Zubni karijes; Heroin, ovisnost – komplikacije; Slina – fiziologija; Zubi, demineralizacija*