



Effect of Smoking on the Mineralizing Ability of Oral Fluid

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Abstract: The article examines the influence of smoking on the state of human teeth, reveals the influence of tobacco on the state of tissues in the oral cavity, conducted a study on the state of teeth in smoking and non-smoking patients, indicated the results of a survey on awareness of the harm of smoking among students. Groups of smokers and non-smokers (control group) students were taken samples of oral tissues of the oral cavity. Using the spectrophotometric method, the concentration of mucin in the oral fluid was determined.

Keywords: oral fluid, mucin, smoking, mineralizing ability.

Introduction

One of the most significant problems of our society that persists for a long time is smoking. Russia ranks fifth in the number of smokers [6]. Recent studies claim that smoking begins as early as 15 years of age, and the first experience of "testing" cigarettes even earlier - up to seven years [3]. The fact that smoking causes great harm to a person has long been proven and is not in doubt.

Even in the last century, the adverse effects of tobacco on the state of the dentoalveolar system were established. The effectiveness of treatment of certain oral diseases is reduced due to active long-term smoking of patients, as tobacco smoke contains hundreds of different compounds, such as carbon monoxide, polonium-210, hydrogen cyanide, nitrogen oxides, hexavalent chromium, cadmium, and more than 76 metals. These substances have a negative impact on the treatment process. Various studies show that tobacco smoking negatively affects the organs and tissues of the oral cavity, which leads to the development of diseases specific to smokers, such as ulcerative-necrotic gingivitis, smokers' melanoma, etc., and also contributes to the progression of major dental diseases, causing [1,2,3].

staining of teeth, dental restorations, and dentures [13]. Smoking disrupts complex enzymatic processes occurring in the oral cavity.

The process of smoking is the burning of tobacco, accompanied by the release of a number of complex chemicals and the interaction of chemicals not only with air oxygen, but also with each other [7]. When smoking, exposure to the human body of the constituent components of tobacco smoke can occur directly through the mucous membrane of the mouth, nose and bronchi. The oral cavity is primarily exposed to tobacco products due to its anatomical and topographical features табакокурения. When smoking increases the content of organic acids, in particular, lactate in the oral fluid and plaque, which contributes to the demineralization of enamel and the development of caries. Tobacco smoke also releases nitrates and nitrites into the oral fluid. [8]

A number of researchers have shown that smoking affects the microcrystallization of oral fluid by reducing the amount of mucin in it. Confirmation of this experience has shown that smoking changes the biological properties of oral fluid, which means that it violates its mineralizing and immunological properties, leading to the development of carious process and inflammatory diseases of the oral mucosa [9, 10]. Mucin is an acidic protein (glycoprotein). It is an essential component of the secretions of the mucous membranes of the gastrointestinal, respiratory tracts, genitourinary system and conjunctiva, serving as a factor of innate immunity.

Mucin belongs to the micelles of the oral cavity. Micelles are colloidal formations that maintain calcium salts in a pseudo-dissolved state, stabilize them, maintaining the constancy of the mineral composition of oral fluid, and form a protective film on the enamel of teeth - a pellicle. It is believed that the ionic bonds formed between calcium and proteins prevent the precipitation of calcium salts. In the presence of mucin, Ca^{2+} and HPO_4^{2-} ions are unable to form supersaturated solutions. Due to the presence of a carbohydrate component, mucin gives the oral fluid a thick, mucous character. Mucins of oral fluid cover and lubricate the surface of the mucous membrane. Their large molecules prevent bacteria from sticking and colonizing, protect tissues from physical damage, and allow them to resist thermal changes [3]. The composition of oral fluid varies throughout the day, depending on food intake and the state of the body. Taking into account the functions of mucin in the oral fluid, we can assume what changes will be observed in the oral cavity of a smoking person: widespread hard and soft plaque, carious lesions of the hard tissues of the tooth, mechanical injuries of the oral mucosa, traces of teeth on the tongue and cheek mucosa, inflammatory diseases of the oral mucosa. [4,5].

The aim of the study was to study the effect of smoking on the condition of human teeth.

Methodology

Students of Smolensk State Medical University - 30 non-smokers (mean age 18.7 ± 1.2 years, control group) and 30 smokers (mean age 18.7 ± 1.2 years, study group) were recruited to participate in the study on a voluntary basis. The average smoking experience was 3.3 ± 1.7 years.

The content of mucin in the oral fluid was studied experimentally and experimentally (chemically). The presence of complaints among the surveyed contingent was revealed by means of a questionnaire (Appendix 1). To assess the mucin content in the oral fluid, an analysis of the oral fluid of students in the study and control groups was performed. The amount of mucin in saliva was determined by spectrophotometric method from the difference in protein concentration in the initial material and above the sedimentary liquid formed after acid precipitation of mucin [7]. For this purpose, two experimental samples were prepared, the first containing saliva and a working reagent, the second containing a sedimentary liquid and a working reagent; a standard sample containing an aqueous solution of albumin with a concentration of 0.25 g / l and a working reagent; and a control sample containing distilled water and a working reagent. The working reagent was prepared by mixing a bromine-phenolic blue solution at a concentration of 1.2 g / l and a buffer solution (pH 3.0) containing 320 mmol / l citric acid and 160 mmol/l sodium phosphate in a ratio of 2: 23. The contents of each sample were mixed and incubated for 10 minutes. The optical density (OP) of the experimental and standard samples was determined against the control sample at a wavelength of 620 nm. The protein concentration in saliva and надосадочной supernatant (g/l) was calculated by the formula: $C = (\text{OP}_{\text{Pop}} : \text{OP}_{\text{St}}) \cdot 0.25$, where OP_{Pop} - the optical density of the experimental sample, OP_{St} - the optical density of the standard sample, 0.25 - the protein concentration in the albumin solution. After obtaining the results, mucin precipitation was performed. For this purpose, 0.05 ml of 20% acetic acid was added to 0.5 ml of oral fluid acid, after 5 minutes, was centrifuged at a speed of 2000 revolutions per minute for 10 minutes. The mucin concentration in the oral fluid was calculated as the difference between the protein concentrations in the starting material and in надосадочной the supernatant formed after mucin deposition. [6,7].

Statistical processing was carried out using descriptive statistics (mean, standard deviation), in accordance with the accepted algorithms for testing hypotheses about differences in central trends,

the nonparametric Mann-Whitney U-test was used (data are presented as a median with a confidence interval of 95%), and the exact Fisher test, the differences were considered significant at $p < 0.05$ [12].

Research results and discussion

According to the results of the spectrophotometric method, the OP of the standard sample was 0.36 kg / m³. The OP of the experimental sample before precipitation in the smoking contingent reached 0.21 kg / m³. The OP of the experimental sample before precipitation in the students of the control group was 0.57 kg / m³. The OP of the experimental sample after mucin deposition in the smoking contingent was only 0.004 kg / m³. This indicator of the experimental sample after precipitation in the control group was 0.05 kg / m³. Since the study was conducted for several days at long time intervals, the albumin was inactivated, so for more accurate indicators, the method was repeated with a new albumin solution. The results obtained after repeated experiments are presented in Table 1.

Table 1. Optical density of samples (kg / m³)

	. Op smoking	op counter	Op smoking	op counter	. Op smoking	op counter	. Op standard
Before precipitation	0.25	0.57	0.27	0.67	0.25	0.5959	0.5
after precipitation	0,004	0,5	0,007	0,04	0,009	0,06	0,05

Optical density Smokers Non-smokers (control group) Criterion p

As a result of calculations, it was revealed that the concentration of mucin in the oral fluid of the examined group of non-smoking students was 2.4 times higher than in the oral fluid of the smoking contingent and amounted to 0.31 g / l versus 0.13 g/l ($p < 0.05$). We present the results of dental examinations of several students.

Student L., 19 years old, does not smoke. Among chronic diseases, he notes chronic gastritis, vegetative-vascular dystonia. Previous inflammatory diseases of the oral cavity: stomatitis. At the time of the examination, he does not complain; he brushes his teeth irregularly-1 time a day, visits the dentist 2 times a year. At the time of the examination, student L. had no visible significant changes in the oral mucosa and carious lesions of the teeth (Fig.A).

1. A - oral mucosa of student L., 19 years old. B-student A., 21 years old, hypertrophic gingivitis

Student A., 21 years old, has been smoking 10-15 cigarettes a day for 5 years. He denies any chronic diseases. Previous inflammatory diseases of the oral mucosa: stomatitis, gingivitis. Claims that inflammation occurs at least once a year. At the time of the examination, he complained of bleeding gums. Irregular brushing of teeth - 1 time a day; visits the dentist once a year or less. At the time of examination, hypertrophic gingivitis in the upper jaw (Fig. 1B) and carious lesions of the teeth 2.6, 3.4, 3.5 were detected.

Student V., 20 years old, has been smoking 10 cigarettes a day for 6 years. Denies chronic diseases. Previous inflammatory diseases of the oral mucosa: tonsillitis. Claims that inflammation occurs at least 2 times a year. At the time of examination, complaints of discoloration of teeth, caries; irregular brushing of teeth-1 time a day. I have visited the dentist only once in the last 5 years. At the time of examination, multiple carious processes and hard dental deposits were detected (Fig. 2). The results of the survey are presented in Table 2.

Smoking history	Number of cigarettes smoked per day	Have chronic diseases (%) of the oral cavity	inflammatory diseases of the oral cavity(%)	Gingivitis (%)	. Stomatitis (%)
3±1,7 0	8±3 0	17	41	33,1	23
Complaints	. Bleeding gums 5	Caries 5	Discoloration of teeth 11.7	HNe related to smoking 23%	NotNe related to smoking 67% %

Student A., 21 years old, hard dental deposits Table 2. Anamnestic and clinical data_

Note. * - statistically significant differences between non-smokers and smoking students

Smoking students were more likely to have inflammatory diseases of the oral cavity. Every third smoker was found to have gingivitis, and every 10th smoker had bleeding gums. Signs of caries are noted somewhat more often among smokers compared to non-smokers. Thus, a decrease in the concentration of mucin in the oral fluid can be considered as one of the factors contributing to the development of oral diseases in smoking students.

Conclusion

In the course of our work, it was found that smoking has a significant impact on the qualitative composition of human oral fluid by reducing the amount of mucin in it, which can lead to a decrease in the mineralizing ability of oral fluid and the most frequent development of carious processes in hard dental tissues, as well as to an increased risk of developing inflammatory diseases of oral tissues.

Smoking reduces the concentration of mucin in the oral fluid, which leads to frequent development of the carious process.

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