Gambaran Epitel Mukosa Bukal Pada Perokok Wanita Kota Bandung

(Buccal Mucosal Epithelium Female Smokers in Bandung)

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Abstrak

Kekhawatiran muncul bahwa semakin banyak wanita muda yang mungkin terkena penyakit gigi dan mulut akibat meningkatnya jumlah perokok wanita di Kota Bandung. Banyak penelitian telah menunjukkan bahwa merokok dapat membahayakan berbagai organ, dan bahwa wanita lebih rentan daripada pria. Penelitian ini bertujuan untuk mengetahui perbedaan morfologi gusi, menghitung persentase abnormalitas sel epitel mukosa bukal, dan membandingkan cytomorphometric sel epitel mukosa bukal pada wanita perokok dan bukan perokok. Subjek penelitian ini adalah 60 wanita usia 17-25 tahun dengan teknik pengambilan sampel yang digunakan adalah jenis purposive sampling. Kriteria dari responden adalah wanita berusia 17-25 tahun yang tidak sedang berpuasa, dan tidak sedang sakit pada bagian rongga mulut. Jumlah responden teridiri dari 30 wanita bukan perokok dan 30 wanita perokok. Hasil penelitian menunjukkan bahwa 36,67% perokok yang mengalami smoker's melanosis dengan prevalensi 20% mengalami pigmentasi tunggal dan 16,67% mengalami pigmentasi meluas. Hasil penelitian menunjukkan bahwa persentase abnormalitas sel epitel mukosa bukal pada kelompok perokok adalah 12,2% dan persentase abnormalitas sel epitel mukosa bukal pada kelompok bukan perokok adalah 3,5%. Kesimpulannya, morfologi gusi pada perokok terdapat smokers melanosis (pigmentasi yang lebih gelap). Abnormalitas sel epitel mukosa bukal pada wanita perokok dan bukan perokok berbeda secara signifikan ($p<0,05$). Lalu terdapat perbedaan yang signifikan antara cytomorphometric sel epitel mukosa bukal pada wanita perokok dan bukan perokok pada parameter diameter sel dan luas sel. Temuan ini menunjukkan bahwa kebiasaan merokok dapat berdampak negatif terhadap kesehatan mukosa bukal pada wanita

Kata kunci: Abnormalitas sel, *Cytomorphometric*, Rokok, Smoker's melanosis.

Abstract

Concerns are emerging that young female may get dental and oral illnesses as a result of Bandung City's growing female smoking population. Numerous studies have demonstrated that smoking cigarettes can harm a variety of organs, and that female are more susceptible than men. This study aims to determine the difference in gum morphology, calculate the percentage of buccal mucosal epithelial cell abnormalities, and compare cytometric buccal mucosal epithelial cells in female smokers and nonsmokers. The subjects of this study were 60 females with the sampling technique used being purposive sampling type. The criteria of the respondents were female aged 17-25 years who were not fasting and were not sick in the oral cavity. The number of respondents consisted of 30 smokers and 30 non-smokers. The results showed that 36.67% of smokers who experienced smoker's melanosis with a prevalence of 20% experienced single pigmentation and 16.67% experienced widespread pigmentation. The results showed that the percentage of buccal mucosal epithelial cell abnormality in smokers was 12.2%, and the percentage of buccal mucosal epithelial cell abnormality in nonsmokers was 3.5%. In conclusion, the morphology of the gums in smokers is that smoker's melanosis (darker pigmentation). Abnormalities of buccal mucosal epithelial cells in smoking and non-smoking female were significantly different ($p < 0.05$). Then there is a significant difference between cytometric buccal mucosal epithelial cells in female smokers and nonsmokers in the parameters of cell diameter and cell area. These findings suggest that smoking habits may negatively impact the health of the buccal mucosa in female.

Keywords: *Cell abnormalities, Cytometric, Smoking, Smoker's melanosis*

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Cigarettes are rolls of tobacco wrapped in cigarette paper and contain about 4,000 types of chemicals, 40 of which are known to cause cancer because they are carcinogenic.[1] Smoking is a major health problem globally because the number of smokers aged over 15 years worldwide reached 1.3 billion respondents, with a prevalence of 942 million men and 175 million females. [2] Meanwhile, the death rate from smoking in 2018 reached 30%, or the equivalent of 17.3 million respondents, and according to WHO, this figure will continue to increase until 2030.[3] When they first start smoking, female in the Indonesian city of Palembang endure headache pain and menstrual pain. Because the pain they experience does not last long the habit of smoking remains they do until now.[4] According to research, 11 out of 20 female smokers in Bandung believe they are unable to avoid smoking, thus they become a part of them.[5] Compared to male smokers, female smokers are more likely to experience health issues. According to Yang et al. (2024) female who smoke are 25% more likely than men to suffer a coronary heart attack,[6]

Cigarette smoke can cause dysplasia or abnormal but not necessarily cancerous development of cells and tissues.[7] Mild to severe changes towards malignancy in the histology of epithelial cells due to cigarette smoke can be seen from hyperkeratosis and carcinoma in situ that occur in oral epithelial cells. Oral signs of dysplasia are loss of epithelial cell lines or layers, accumulation of basal cells, irregular layers, increased abnormal cell images, rapid keratinization, hyperchromatic and pleomorphic in the cell nucleus, and increased ratio of cell nucleus and cytoplasm.[8]

Exposure to cigarette smoke also has a strong relationship with DNA damage because the substances inhaled from cigarette combustion trigger oxidative stress and carcinogenesis.[9] Evidence of DNA oxidation includes cell death,

DNA mutations, replication errors, and genomic instability can occur if oxidative DNA damage is not repaired prior to DNA replication.[10] The continuous occurrence of physical and chemical trauma can also cause cell and DNA damage.[11] Meanwhile, the buccal mucosa is often used as an indicator if the periodontal tissue is affected by the disease because most periodontal disease starts from the buccal mucosa.[11] The study parameters of buccal mucosal epithelial cells that are usually observed are cell length and width and cell nucleus length and width. [12]

The gingiva is extremely susceptible to the effects of the oral cavity environment since it is a component of the outer periodontal tissue. Mucosa buccal epithelial tissue is a labile structure, new cells are constantly being produced during mitotic division. Function of mucosa buccal epithelial tissue is to cover, shield, and coat the body's exterior or interior surfaces.[13] Male smokers in the Iranian city of Babul, who were between the ages of 20 and 40, had larger epithelial cell nuclei and smaller cytoplasm in the buccal mucosa.[14] There is currently no research on how cigarettes affect the shape of female smokers' buccal mucosa in Bandung. This study aims to determine the difference in gum morphology, calculate the percentage of buccal mucosal epithelial cell abnormalities, and compare cytometric buccal mucosal epithelial cells in female smokers and nonsmokers.

METHODS

Sampling was carried out in the city of Bandung, and observations were made at the Biology Laboratory of the University of Muhammadiyah Bandung. Interviews were conducted before sampling, and then sampling was carried out after participants signed informed consent. Ethical approval with number 665 / KEP.01/UNISA-BANDUNG/VIII / 2023 (Research Ethics Committee of Universitas 'Aisyiyah Bandung). This study used a survey method with female subjects. The sampling technique used is purposive sampling. The criteria of the respondents were female aged 17-25 years who were not fasting and were not sick in the oral cavity. The number of respondents consisted of 30 smokers and 30 non-smokers.

Data sampling was conducted after the respondents agreed and signed informed consent. Morphological observation is done before sampling by observing the color of the gums and also the presence of smoker's melanosis with the naked eye, then a gum shot. Sampling of epithelial cells of the buccal mucosa by scraping or taking cells up to the parabasal section. Scraping is carried out by means of a cotton swab moistened with 0.9% NaCl, which is gently rubbed horizontally into the buccal mucosa. This sampling process will not cause pain and will not cause injury. After that, the cotton swab is applied slowly and in one direction to the glass object horizontally, and then we wait for one minute. Then fixation or immersion with 96% ethanol for approximately one minute to kill the cells without damaging the cell structure. Furthermore, cell staining will be done using Giemsa dyes and decolorization or rinsing using distilled water to clear preparations (15).

Observation of epithelial cells was carried out using a microscope at 400x magnification. A total of fifty clearly defined cells with good staining are selected by gradual systematic sampling (16) (17). The movement of the microscope is regulated from left to right, then up and down slowly to avoid repeated measurements of the same cells (18). Buccal mucosal epithelial cells are measured using a micrometer and then calibrated to determine the actual size. Calibration is done by aligning the two scale shadows, the eyepiece micrometer scale and the objective micrometer scale, by rotating the top of the eyepiece lens (19).

The observed study parameters include cell length and width as well as cell nucleus length and width (12). Recorded measurement data. Ratio calculation is also done according to Primasari & Cynthia in 2018. The ratio of nucleus to cytoplasm is calculated by the formula:

$$\text{Formula} = \frac{nuclei\ size}{cytoplasm\ size}$$

The area of the cell and the area of the cell nucleus are calculated using the formula of the area of the circle π x r2. The average values of the cell nucleus length and width diameter, as well as the cell length and width diameter, are calculated in advance. The criteria of normal epithelial cells are the shape of the cell and the shape of the cell nucleus is regular and uniform, the distribution of homogeneous color intensity so that it has a smooth texture, the color intensity in the cell nucleus tends to be higher so that the cell nucleus is darker, then the color of the micronucleus is the same as the color of the main cell nucleus, and the diameter of the micronucleus is less than 1/3 of the main cell nucleus, and the micronucleus does not overlap with the main cell nucleus. Meanwhile, the criteria of abnormal epithelial cells are to have the shape of the cell and the shape of the cell nucleus is larger and irregular, the distribution of color intensity is wider so that almost all parts of the cell are dark and have a rougher texture (20). According to Kurniawan et al. in 2013, the formula for calculating the percentage of epithelial cell abnormalities is as follows:

$$\text{Percentage} = \frac{number\ of\ abnormal\ cells\ observed}{total\ cell} \times 100\%$$

There are two types of data to be analyzed, namely qualitative data and quantitative data. The qualitative data contain the elaboration or description of the cell structure of the epithelium of the buccal mucosa to be displayed in the form of a micrograph. Meanwhile, quantitative data containing the results of cytometric analysis data will then be carried out with statistical tests using the T test with the level of error or alpha= 0.05. This statistical analysis was done using SPSS software.

RESULTS

In this study, the average smoker is still included in the light category because they only consume 1-10 cigarettes a day and 3-11 dripping liquid vapes a day. In general, there are three

categories of smokers based on the length of smoking, namely less than one year, one to two years, and three to four years. There are smokers who use machine-made kretek cigarettes, e-cigarettes, or vapes, and there are also those who use both.

Morphology Of The Gums Of Female Smokers

Healthy gums are usually an even pink color, have a firm and dense texture, and have an even contour and follow the shape of the teeth well (figure 3.1.a). On the gums of smokers, the presence of smoker's melanosis can be found. Smoker's melanosis usually appears in the form of uneven patches of pigment, and pigmentation is usually more clearly visible on the front gums, especially on the bottom of the front teeth, but can appear anywhere along the gum line. Smoker's melanosis is assessed based on the melanin modification index in Hanioka with a value interval of zero to two. A value of zero indicates the absence of pigmentation, a value of one indicates a single pigmentation (figure 1.b), and a value of two indicates widespread pigmentation (figure 1.c).

In this study, smoker's melanosis was not found in the gums of non-smoking female. Single pigmentation, the color of the smoker's melanosis looks younger, and the location of the pigmentation fills the small space between the teeth (interdental gingiva). Whereas in widespread pigmentation, the color of smoker's melanosis tends to be darker and located on the gums that are not attached to the teeth (attached gingiva). According to Vieta et al. 2017, smoker's melanosis occurs in 25-31% of smokers and increases significantly during the first year of smoking. In this study, there were 36.67% of smokers who experienced smoker's melanosis, with a prevalence of 20% experiencing single pigmentation and 16.67% experiencing widespread pigmentation (Table 1).

Buccal Mucosal Epithelial Cell Abnormalities In Smoking And Non-Smoking Female

In this study, we also observed the presence of abnormal epithelial cells with different types of abnormalities (Figure 3.2.). The explanation is in Table 2. The results showed that the percentage of buccal mucosal epithelial cell abnormality in smokers was 12.2%, and the percentage of buccal mucosal epithelial cell abnormality in nonsmokers was 3.5% (Table 3). Judging from these data, the percentage of buccal mucosal epithelial cell abnormalities in smokers was higher than the percentage of buccal mucosal epithelial cell abnormalities in nonsmokers. This is in accordance with the results of an independent t-test that showed a significance value of 0.003 ($p<0.05$), which means that there is a significant difference between buccal mucosal epithelial cell abnormalities in the group of female smokers and non-smokers.

Cytometry Of Buccal Mucosal Epithelial Cells In Smoking And Non-Smoking Female

The results of measuring the nuclei of epithelial cells of the buccal mucosa in smoking female ranged from 7.54 to 10.07 (9.59±1.33 µm). The results of measuring the nuclei of the epithelial cells of the buccal mucosa in non-smoking female ranged from 7.7 to 10.06 (9.58-1.35 µm). Then the cytoplasmic measurement of buccal mucosal epithelial cells in smoking female ranged from 37.58 to 67.46 (50.17±7.54 µm). The results of cytoplasmic measurements of buccal mucosal epithelial cells in non-smoking female ranged from 36.3 to 59.29 (46.82±5.99 µm). The average size of the nucleus in smoking female was 72.55 µm, while the average size of the nucleus in non-smoking female was 72.44 µm. The mean cytoplasmic area of cells in smoking female is 2747.56 µm², while the mean cytoplasmic size of cells in non-smoking female is 1731.08 µm² (Table 4).

The ratio of the area of the nuclei of cells of the epithelium of the buccal mucosa to the area of the cytoplasm (N/C) in the group of smoking females was 1:5.2, which means that for every 1 unit of nucleus size, there are 5.2 units of cytoplasmic size. Then the ratio of the area of the nuclei of the cells of the epithelium of the buccal mucosa to the area of the cytoplasm (N/C) in the group of non-smoking female was 1:4.9, which means that for every 1 unit of size of the nucleus, there are 4.9 units of cytoplasmic size. This shows that in smoking cells, the size of the cytoplasm is larger than the nucleus, while in non-smoking cells, the size of the nucleus is larger than the cytoplasm (Table 4).

Independent t-test results for cell diameter and cell area data showed a significant difference between cell diameter and cell area in smoking and non-smoking female ($p < 0.05$). Meanwhile, independent t-test results for cell nucleus diameter and cell nucleus area showed no significant difference between cell nucleus diameter and buccal mucosal epithelial cell nucleus area of smoking and non-smoking female ($p>0.05$). The nuclei of the epithelial cells of the buccal mucosa of smokers have a smaller size than the nuclei of the epithelial cells of the buccal mucosa of non-smokers (Table 4).

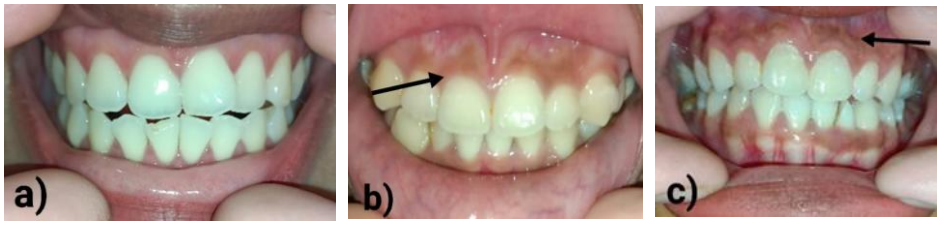

Figure 1. Results Of Research On Types Of Pigmentation: (A) No Pigmentation, (B) Single Pigmentation, (C) Widespread Pigmentation.

Table 1. Types Of Pigmentation

Type of pigmentation	Frequency	Percentage
No pigmentation	19	63,33%
Single pigmentation	6	20%
Widespread pigmentation	5	16,67%
Total	30	100%

Table 2. Normal And Abnormal Buccal Mucosal Epithelial Cell Structure

Type of cells	Description
1. Normal epithelial cells	Polygonal or round cells with smooth edges, the nucleus is in the middle of the cell, round and has evenly distributed chromatin and the cytoplasm looks homogeneous and transparent (Figure 2.a)
2. Chromatin condensation	The chromatin in the nucleus becomes denser and looks darker due to the accumulation of genetic material. Between the dense and dark areas, there are white or lighter spots which are areas of the nucleus that are less dense or free of chromatin (Figure 2.b)
3. karyorrhexis	The nucleus is fragmented leaving the largest fragments in the cell nucleus (Figure 2.c)
4. karyo-lysis	The nucleus begins to dissolve and disappear so that it looks like an empty cell (Figure 2.d)
5. Micronucleus	There is an additional nucleus next to the main nucleus (Figure 2.e)
6. *Nuclear buds*	A small protrusion is formed on the surface of the nucleus which is like a micronucleus (Figure 2.f)
7. *Broken egg*	The nucleus looks as if it has broken and some genetic material comes out of the nucleus (Figure 2.g)

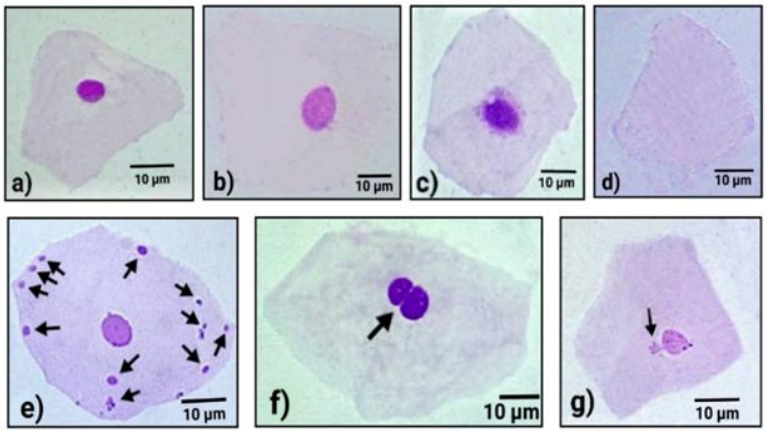

Figure 2. Normal And Abnormal Buccal Mucosal Epithelial Cell Structure

Description: a) normal epithelial cells, b) chromatin condensation, c) karyorrhexis, d) karyo-lysis, e) epithelial cells containing many micronuclei, f) epithelial cells with nuclear buds, g) epithelial cells with broken egg nuclei.

Table 3. Percentage Of Cell Abnormalities

Jenis abnormalitas sel	Smokers	Nonsmokers
chromatin condentation	5%	0.94%
karyorrhexis	3.85%	1.82%
karyo-lysis	0.46%	0.58%
Micronucleus	2.54%	0%
Nuclear bud	0.077%	0.073%
Broken egg	0.23%	0.073%
Total (Rerata ± SD)%	12,2% ± 4,47^{B}	3,5% ± 9,68 ^{A}

Description: The numbers shown are the mean ± standard deviation (STDEV), numbers with different letters are significantly different using an independent t-test.

Table 4. Cytometric Of Buccal Mucosal Epithelial Cells In Female Smokers And Non-Smokers

Sample	Ø nuclei (mean±SD) µm	Ø cell (Mean±SD) µm	Nuclei area (Mean±SD) µm^2	Cell area (Mean±SD) µm^2	Ratio N/C
Smokers	9.59 ± 1.33 ^{A}	50.17±7.54 ^{B}	72.55 ± 10.21 ^{A}	2747.56± 524.79 ^{B}	1 : 5.2
Nonsmokers	9.58 ± 1.35 ^{A}	46.82±5.99 ^{A}	72.44 ± 9.18 ^{A}	1731.08± 259.67 ^{A}	1 : 4.9

Description: The numbers shown are the mean ± standard deviation (STDEV), numbers followed by the same letter are not significantly different and numbers followed by different letters are significantly different.

DISCUSSION

The degree of pigmentation is influenced by various factors, some of which are the most influential, such as the duration of smoking and the number of cigarettes smoked by respondents in a day. The onset of this pigmentation is basically due to the stimulant material in cigarette smoke reaching the melanocytes of the buccal mucosa. The first way is through the mucosa and saliva, while the second way is through breathing. In the first way, nicotine and benzopyrene reach melanocytes in the buccal mucosa through the mucosa and saliva. Hot cigarette smoke containing nicotine and benzopyrene stimulates melanocytes to produce more melanosomes, resulting in an increase in melanin pigment in the lamina propria and deposition in the basal cells of the epithelial layer of the oral mucosa. In the second way, nicotine and benzopyrene present in the blood circulation affect melanocytes indirectly. [14]

In 25-31% of smokers, smoker's melanosis will be seen, according to Zarra & Utami in 2023. However, in this study, we also found respondents without pigmentation smoker's melanosis, which amounted to 60.33%. The absence of pigmentation is not only influenced by the length of smoking and the number of cigarettes consumed in a day, but also the environment around the smoker. Active smokers can act as passive smokers. When respondents become passive smokers, the mechanism of pigmentation formation occurs through the second way. In respondents without pigmentation, they may not be passive smokers, so that the mechanism of formation of smoker's melanosis does not occur. [15] The colour of healthy gingiva is pink. Good dental health is essential to maintain a high quality of life and boost self-confidence.

Abnormalities of the cell nucleus are biomarkers that are often used to observe exposure to harmful agents in individuals that can cause mutagenetic, genotoxic, or carcinogenic [15]. Changes in the shape of the nucleus are also closely related to important cell functions, such as cell motility and polarization. The morphology of the nucleus is also related to the shape of the cell, so an elongated cell usually has a nucleus that is also elongated. Changes and volume of the nucleus can affect the concentration of nuclear proteins, as well as the regulation and transcription of genes.[16]

There are two types of cell death that can be in the form of necrosis (external factors) or apoptosis (internal factors). Necrosis is a process of cell death that occurs in living organisms caused by pathological conditions, such as infection or inflammation. Meanwhile, normal cells will experience programmed cell death, or apoptosis, so that apoptosis is a normal occurrence in the development and maintenance of health in multicellular organisms. Apoptosis is cell death per individual cell, whereas necrosis involves a group of cells.[17] Both types of cell death are characterized by the typical edge changes in the nucleus, namely Pyknosis, karyorrhexis and karyo-lysis. [18]

According to Hadi 2011, cells undergoing apoptosis can also undergo several changes, including size reduction, as well as chromatin condensation (Figure 3.2.a).The nuclei of cells that undergo pyknosis will shrink, dark in color, with irregular boundaries due to solidified chromatin. Meanwhile, in the karyorrhexis stage, the cell nucleus will disintegrate into fragments of chromatin in the cell so as to leave the largest fragments in the nucleus and finally the chromatin into lysis, which is called karyo-lysis, so that on observation it will appear as an empty cell.[19] In this study, no pyknosis cells were found in both smokers and non-smokers cells (table 3). Meanwhile, cells that undergo karyorrhexis and karyo-lysis are not only found in the group of smokers but also found in the group of non-smokers (table 3). Keep in mind, karyorrhexis and karyo-lysis are indicators of apoptosis, so it is very natural that in the group of non-smokers, we also found cells that undergo karyorrhexis and karyo-lysis because all physiologically normal cells will undergo the process of apoptosis.[15]

Observations using a microscope with a magnification of 400x obtained a picture of micronuclei measuring between 1/3 to 1/6 of the nucleus (figure 2.e). This is in accordance with the research of Leonardi et al. in 2020, which states that in cells with micronuclei, there will be a main nucleus and one or more micronuclei that are smaller, between 1/3 and 1/6 of the diameter of the main nucleus.[15] According to Husein 2014, the formation of micronuclei indicates the occurrence of DNA damage and cell death that can cause mutations and cancer.[20] This is in line with the results of the study, where in this study, micronucleus was only found in the cells of the smoker group. Research conducted by Widiani et al. 2022 also found the presence of micronuclei in smokers' buccal mucosal epithelial cells (28). Micronucleus is a small, nucleus-like structure located near the main nucleus and in the cytoplasm. Micronuclei are formed as a result of chromosomal abnormalities, that is, when cells divide in anaphase, chromosome fragments or intact chromosomes fail to be pulled by the spindle threads to the opposite poles, so that the chromosomes that are not pulled successfully still undergo the formation of the nuclear membrane in

telophase, and a separate nucleus is formed in the cytoplasm.

The results of Rahmah et al.'s research in 2016 showed that exposure to factors such as radiation, chemicals, alcohol, and tobacco consumption (smoking, hosts) can increase the number of micronuclei so that this is in line with the results of a study where micronuclei were only found in cells of the smoker group[21]. The more intense the smoking by respondents, the higher the frequency of micronucleus formation.[28] This is in accordance with the results of the Gangadharan study in 2016, which showed that the group that smoked more than 6 cigarettes per day had a higher frequency of micronucleus formation than the group that smoked less than 6 cigarettes per day.[22]

The mean number of buccal mucosal micronuclei in smokers with a smoking history of more than 10 years also had a higher prevalence of micronuclei compared to smokers with a smoking history of less than 10 years, although it did not differ significantly.[21] Furthermore, there is an abnormality of the cell type nuclear buds, or nuclear buds that have a structure similar to the micronucleus, attached to the main nucleus through a thin connection of nucleoplasm. These structures can be formed through a mechanism similar to the formation of micronuclei during cell division. It occurs when chromosomes or chromosome fragments are not properly distributed to daughter nuclei during anaphase, the stage in which chromosomes are pulled toward opposite poles of a dividing cell.[23]

In this study, cell nuclei with broken egg type or broken eggs were found in both groups of smokers and non-smokers (Figure 2.g). This is in line with the results of Roberts' 1997 study, where generally cell nuclei with broken egg type or broken eggs were found in both groups of smokers and also control groups that were not smokers. The phenomenon of a broken egg is explained as a nucleus divided into two parts and connected by a thin ribbon of matter that does not react with Feulgen dyes. Feulgen dyes are used to identify DNA because these dyes react specifically with aldehyde groups formed in DNA after treatment with acids, so it can be concluded that the thin band connecting the two parts of the nucleus does not contain DNA, and this band consists of other materials other than DNA, possibly proteins or other cytoplasmic components. [24]

Although statistically the diameter of the cell nucleus in the group of smoking and non-smoking female did not differ significantly ($p > 0.05$), the ratio value showed that in the group of non-smoking females, the cells had a larger nucleus diameter and a smaller cytoplasmic diameter (1:4.9). This is because the active cell proliferation process shows that the diameter of the nucleus is increasing while the diameter of the cytoplasm is decreasing. Repeated exposure to cytotoxic agents can result in chronic trauma to cells, compensated cell proliferation, and end in cancer.[15]

Cell proliferation is the process by which the body's cells divide and multiply. This is an important part of the network maintenance and repair process. In smokers, cell proliferation is often inhibited or impaired due to several factors related to exposure to harmful substances in cigarettes. Impaired or slowed cell proliferation can lead to a buildup of intermediate cells.[25] This is in line with the results of the study where the size of the nucleus is relatively smaller compared to the size of the cytoplasm in the group of female smokers, which can indicate that the cell is in the intermediate stage, and it indicates disruption of cell proliferation.

The conclusion of this study is that the morphology of the gums in female smokers and non-smokers is different because the gums of smokers can show darker pigmentation, or better known as smoker's melanosis. Then the percentage of buccal mucosal epithelial cell abnormalities in smoking female was 12.2%, and the percentage of buccal mucosal epithelial cell abnormalities in non-smoking female was 3.5%. Abnormalities of buccal mucosal epithelial cells in female smokers and nonsmokers differ significantly ($p < 0.05$), and there are also cytometric differences of buccal mucosal epithelial cells in female smokers and nonsmokers in the parameters of cell diameter and cell area. It was determined that the detrimental effects of smoking result in a decrease in the size of epithelial cells and an increase in cell abnormalities in female smokers.

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REFFERENCE

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