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EFFECT AMOUNT OF CIGARETTE SMOKING ON GINGIVAL EPITHELIUM THICKNESS
(EFEK JUMLAH ROKOK SIGARET TERHADAP KETEBALAN EPITHEL GINGIVAL)

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Abstract
Smoking is one of the most important risk factors for oral diseases. Tobacco smoking produces more than 4000 chemical materials. Gingival protects underlying tissue to oral environment. Cells of gingival epithelium are metabolic actively, and can be reacted to internal and external stimuli. The thickness of oral mucosa epithelium is related with the amount of cell or proliferation activity. This study aimed to investigate effect amount of cigarette smoking on thickness of gingival epithelium. This study was animal experimental laboratory study. This study used 40 males Rattus Norvegicus strain Sprague Dawley, and were divided into 4 groups. They were control, mild, moderate and severe smoker groups. The animal models were exposed 2 grams by pump and were euthanized with ketamine in the 7th day. Measurement of gingival epithelium thickness used hematoxylin eosin that was measured from base to granular layer and 3 sites (in μm). Data were analyzed by one way anova. The results of this research showed that there was an increasing thickness of gingival epithelium of animal models. The highest of gingival epithelium thickness was in severe smoker groups. In conclusion, cigarette smoking influenced on gingival epithelium thickness.

Key words: cigarette smoking, cell proliferation, gingival thickness

INTRODUCTION
Smoking is now recognized as the most important cause of preventable death and disease.1 Cigarette consists of numerous vaporized chemicals (92 %) and particulates (8 %) suspended in a gaseous medium.2 Exposure to environmental tobacco contains more than 4000 chemicals, including nicotine and at least 40 known carcinogens.3 Tobacco smoking exerts a substantial destructive effect on the periodontal tissues and increases the rate of periodontal disease progression. Smokers with periodontal disease seem to show less sign of clinical inflammation and gingival bleeding compared to non-smokers. The process could be explained by the fact that nicotine exerts local vasoconstriction, reducing blood flow, edema and clinical signs of inflammation.4

The function of the gingival epithelium is to protect the deep structures while allowing a selective interchange with the oral environment. This is achieved by proliferation and differentiation of the keratinocyte. Proliferation of keratinocyte takes places by mitosis in the basal layer and less frequently. In the suprabasal layers, where a small proportion of cells remains as a proliferative compartment while a larger number begins to migrate to the surface. Proliferating cells are related with the increasing of thickness of tissue.5 This study aimed to investigate effect amount of cigarette smoking on gingival epithelium thickness.

MATERIALS AND METHODS
This study was an animal experimental laboratory study. All of animal models were bought and cared in Pharmacology Laboratory Medicine Faculty Gadjah Mada University Yogyakarta Indonesia. The ethical clearance for experimental procedure was approved by ethical commission of Faculty of Dentistry Gadjah Mada University. All of animal models were adapted on new environment for a
week. This study used 20 males *Rattus norvegicus* strain *Sprague Dawley*, and divided into 4 groups. They were control, mild (2 gr/day), moderate (4 gr/day) and severe smoker (8 gr/day) and exposed for 5 days. The animal models were exposed 2 grams/exposed by chip blower and in the cage. The animal models were euthanized with ketamine in the 7th day. After having been sacrificed, the gingival specimen obtained from the incisors area of the mandible, washed by water and then fixed in 10% phosphat buffered formalin pH 7.4. After alcohol dehydration, the gingival specimens were embedded in paraffin. Measurement of gingival epithelium thickness used hematoxylin eosin that was measured from base to granular layer and 3 sites (in μm). Data was analyzed by one way anova.

**RESULTS**

The measurement of gingival epithelium thickness in smoker and non smoker showed as in the Table 1. It was shown that the severe smoker has the most thickness in gingival epithelium.

The statistical analysis was continued by Least Significant Differences (LSD) to know probability of the dose and duration of smoking effect in gingival epithelium thickness between groups.

Table 1. Mean and standard deviation of gingival epithelium thickness measurement in smoker and non smoker rats

<table>
<thead>
<tr>
<th>Group</th>
<th>Gingival epithelium thickness Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>1.92 ± 0.41</td>
</tr>
<tr>
<td>Mild smoker</td>
<td>2.99 ± 0.15</td>
</tr>
<tr>
<td>Moderate smoker</td>
<td>3.56 ± 0.49</td>
</tr>
<tr>
<td>Severe smoker</td>
<td>4.91 ± 0.08</td>
</tr>
</tbody>
</table>

Table 2 showed that was significant difference of thickness of gingival epithelium between smoker and non smoker.

Table 2. Summary of Anova test of gingival epithelium thickness measurement

<table>
<thead>
<tr>
<th>Group</th>
<th>Sum of Square</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>23.197</td>
<td>3</td>
<td>7.732</td>
<td>70.089</td>
<td>0.000</td>
</tr>
<tr>
<td>Within groups</td>
<td>1.765</td>
<td>16</td>
<td>0.110</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>24.962</td>
<td>19</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There was significant difference between amounts of smoking to gingival epithelium thickness (Tab-

le 3). Figure 1 showed the picture of gingival thickness change.

![Figure 1. Microscopic examination of gingival epithelium thickness in control (a), mild (b), moderate (c), and severe smoker groups. Arrow sign was the width of thickness (400x)](image)

**DISCUSSION**

Based on the results of research, there was an increasing of gingival thickness and the highest gingival thickness was in severe smoker groups. Smoking was associated with a variety of dangerous changes in oral cavity, such as oral cancer, periodontal disease, etc. Tobacco smoking contained 4000 dangerous chemical substances and 40 of them are carcinogenic. Inhaled tobacco smoking contained gas and particulate. Gas components were carbon monoxide, carbon dioxide, oxygen, cyanide hydrogen, ammonia, nitrogen, and hydro carbon compound. Particulate components were tar, nicotine, benzopirene, phenol and cadmium.

Heat and poisonous component from smoking caused cell morphology changing, especially cell in oral mucosa. Stimulation of smoking would be in long time and continuously. Stimulation of smoking caused cell will make adaptation process. The adaptation process in this study was the increasing of gingival epithelium thickness and activity of cell
proliferation. Smoking habits can change normal cytology of oral mucosa. The changes were such as hypertrophy, hyperplasia, total thickening epithelial and trigger an increase in progenitor cell keratin proliferation number.\(^7\)

Chemical substances and heat in smoking caused oral mucosa changing. They can change vascularization of gingival and salivary secretion. Smoking stimulation for long time can cause thickening of oral epithelium. Amount and duration of smoking were important factor for determining of hyperkeratinization degree and characteristic.\(^7\) Heavy smoker might have grayish discoloration and increasing number of keratinocyte cells in their gingival.\(^1\)

Cigarette contained tar less than 1.35 mg. In developing country, tar in cigarette was high. In Indonesia, cigarette contains 19-33 mg of tar. Tar was carcinogenic compound.\(^8\) Tar caused carcinogenic in epithelium by increasing of cell proliferation. In cigarette, tar would be condensation in tissue and penetrate into tissue and cell, it would increase cell activity. Previous study showed that tar in cigarette smoking can cause hyperkeratosis on animal model skin.\(^10\)

Nicotine was one of the components in cigarette smoking that has addictive stimulation. Nicotine would be condensation and rapidly absorbed into the bloodstream, where 30% remains in its free form. It is highly lipid soluble and readily penetration to cell membrane. Nicotine can also be absorbed through oral mucosa, and caused the changing of oral mucosa proliferation. In blood stream, nicotine would stimulate sympathetic ganglia to produce neurotransmitter and catekoline. They can influence \(\alpha\)-receptor in blood vessel and cause vasoconstriction in blood vessel of periodontal tissue. It caused the decreasing of blood supply in gingival, so it influenced in revascularization and cell activity in periodontal tissue.\(^1\)

Effect of smoking depends on duration and amount of cigarette that smoked by smoker. Type of cigarettes also influenced effect of smoking, such as type of tobacco, cigarette design (with or without filter) and pattern of smoking. In this study used one of the cigarettes that is tar, nicotine and carbon monoxide were more than the others, also without filter. This type caused more harmful effect in gingival.\(^8\) Smoking can increase thickness of gingival epithelium. The increasing of gingival epithelium thickness was caused by keratinocyte proliferation. Chemical substance in smoking influenced proliferation degree of cell in gingival.\(^11\)

The conclusion of this study, there were effects of cigarette smoking in gingival epithelium thickness.

References