The Relationship between Smoking and Obstructive Sleep Apnea (Osa) in Students at the Faculty of Law, Indonesian Christian University

Abstract

Obstructive Sleep Apnea (OSA) is a condition of obstruction/narrowing of the upper airway during sleep, which results in cessation of breathing for some time. OSA can be experienced in all age groups, and as many as 56% reported having symptoms of excessive daytime sleepiness (EDS). Smoking can cause abnormal changes in the upper respiratory tract, both histologically and physiologically. Several mechanisms have been hypothesized to explain how smoking increases the risk of developing OSA. This study aimed to determine the relationship between smoking and OSA in law faculty students at the Indonesian Christian University Class of 2018-2021. The method used in this research is analytic observational with a cross-sectional study design. The sampling technique used simple random sampling of 94 respondents. The primary data was obtained using the ESS questionnaire and smoking history, and then the data were analyzed using the Chi-Square statistical test. Statistical test results obtained p value = 0.214. There is no relationship between smoking and OSA in law faculty students at the Indonesian Christian University class 2018-2021, which is the conclusion of this study.

***Keywords**: OSA, smoking*

Introduction

Sleeping and breathing are part of daily human physiological processes. Nearly a third of human life activities are sleep, which affects various physiological regulations of the body. However, Obstructive Sleep Apnea (OSA) can disrupt this physiological process. OSA is a condition where the upper airway narrows/obstructs during sleep; this condition results in cessation of breathing for some time and, in fatal cases, can lead to death due to lack of oxygen. [1]

All age groups can experience OSA, and it has a varying prevalence in various countries in the world, ranging from 9% to 38%, and is higher in men. Data on the prevalence of OSA in Indonesia is currently limited, but one study on the general population in Jakarta showed that the prevalence of OSA in the area was 49.5%. [2]

Symptoms that are often found in OSA sufferers are loud snoring, waking up at night, not feeling refreshed after waking up, nocturia, and continuous sleepiness during the day (excessive daytime sleepiness/EDS). [3] Apart from that, other symptoms can also be found, such as impaired concentration, headaches, intellectual disorders, depression, and decreased libido.

Excessive daytime sleepiness (EDS) is a symptom that can appear at any time when the individual feels like falling asleep or wanting to stay awake. [4] EDS is the main symptom of OSA, which causes a decrease in quality of life. The prevalence of EDS in students at the Faculty of Medicine and Health Sciences at Hidayatullah Islamic University in 2013 was assessed using the Epworth Sleepiness Scale (ESS) questionnaire, which assessed 55% of the entire population. Symptoms of EDS can be diagnosed with the Epworth Sleepiness Scale (ESS) questionnaire, which has been widely used by researchers, clinicians, and specialists as a screening for OSA. [5]

Male gender, obesity, and old age are the three main risk factors for the occurrence of OSA, but currently, many studies have found other risk factors that can contribute to the occurrence of OSA, one of which is smoking. Research conducted by

Kahsyap [6] found that the prevalence of OSA in smokers was higher than in non-smokers. In fact, in this study, it was also stated that active smokers were 2.5 times more likely to get OSA than the combination of former smokers and non-smokers. Several mechanisms have been suggested to explain this, one of which is inflammation of the upper respiratory tract due to inhalation of cigarette smoke, thus disrupting sleep stability. However, smoking as a risk factor for OSA is still a matter of debate because, in other studies, the correlation results were not significant enough.

Based on the description above, it appears that the relationship between smoking and OSA is still a complicated topic. Therefore, researchers are interested in the relationship between smoking and OSA in students at the Faculty of Law, Indonesian Christian University Class 2018-2021. The formulation of the research problem answered in this study is "Is there a relationship between smoking and OSA in FH UKI students class 2018-2021? The research aims to determine the relationship between smoking and OSA in FH UKI students in the class of 2018-2021. Meanwhile, the specific objectives of this research are: a) To determine the distribution of smoking frequency among FH UKI students class 2018-2021; b) To determine the frequency distribution of OSA among FH UKI students class 2018-2021; c) To determine the relationship between smoking and OSA in FH UKI students class 2018-2021; and d) to determine the relationship between the degree of smoking and OSA among FH UKI students class 2018-2021.

Literature Review

The upper respiratory tract in humans consists of the nose, nasal cavity, sinuses, pharynx, larynx, and trachea. When related to studies on obstructive sleep apnea (OSA) or sleep-disordered breathing, this focuses on the potential for upper airway obstruction, which refers to the pharynx and surrounding structures. The pharynx is a muscular tube located posterior to the nasal cavity, oral cavity, and larynx and anterior to the cervical vertebrae. The pharynx has three parts: the nasopharynx, oropharynx, and laryngopharynx (hypopharynx). The oropharynx has two regions, the retropalatal region, and the retroglossal region, while the laryngopharynx/hypopharynx starts from the base of the tongue, the epiglottis, to the larynx. Patients with breathing disorders during sleep usually manifest narrowing in the retropalatal region, retroglossal region, or both. [7]

Sleeping and breathing are physiological processes necessary for human life. There are differences in the respiratory control process during sleep and wakefulness. Disease conditions such as anatomical abnormalities/disorders in the upper respiratory tract, intercostals, or diaphragm muscles can affect breathing. Disturbances in any of these factors can cause gas exchange abnormalities, resulting in hypoxemia, hypercapnia, and breathing disorders during sleep (such as snoring, OSA, hypoventilation syndrome, etc.).

The ventilatory response to hypoxia and hypercapnia decreases during sleep, especially during the rapid eye movement/REM sleep phase. In addition, during sleep, the muscle tone of the upper respiratory tract also decreases, resulting in increased upper airway resistance and anatomical structures that are more susceptible to collapse.

Decreased ventilatory response to hypoxia, hypercapnia, and increased upper airway resistance can all contribute to sleep-disordered breathing, including OSA. [8]

Obstructive Sleep Apnea (OSA) is a sleep disorder caused by obstruction/narrowing of the airways during sleep so that breathing stops for a while.

OSA is characterized by repeated episodes of collapse of the airways (especially the orophangingeal tract), either partial (hypopnea) or total (apnea). [9] This narrowing of the airway will result in progressive asphyxia, thereby stimulating respiratory efforts during sleep and usually until the person wakes up. The apnea-hypopnea index (AHI) is a measurement commonly used to assess breathing disorders during sleep, and this index can be used to determine the severity of OSA. According to the American Academy of Sleep Medicine, OSA can be classified into a) mild OSA (AHI value 5-15), which appears as a feeling of drowsiness when doing activities that require little concentration, such as reading or watching; b) moderate OSA (AHI value 15-30) which appears in the form of drowsiness when carrying out activities that require concentration, such as presentations or meetings; and c) severe OSA (AHI value > 30) which appears in the form of drowsiness when doing activities that require more active concentration, such as talking or driving. [10]

OSA can be experienced in all age groups, but its prevalence increases with age. The prevalence rate in adults from each country varies, but for the general population, it is estimated to be around 3-7% for adult men and 2-5% for adult women, and the highest prevalence is reported in middle-aged men. [11] It is estimated that the prevalence of OSA based on the severity of the disease as measured by the apnea-hypopnea index overall is between 9% and 38% experiencing sleep disorders with an AHI value ≥ five and higher in the male group. [12] The prevalence of OSA in children is estimated to be 2- 3.5% with two peak periods. The first peak period occurs at the age of 2-8 years when children experience enlarged tonsils and goiters, and then the second peak period occurs at prepuberty when body weight increases. [13]

Until now, there have not been many reports on the prevalence of OSA in Indonesia, but there was research by Wiadnyana in 2010 on taxi X drivers in Jakarta with a Berlin questionnaire showing that 25% of drivers had a high risk of OSA. [14] In a different study by Susanto et al., using the Berlin questionnaire method in congestive heart failure (CHF) patients, it was found that 42 out of 70 patients (60%) had a high risk of OSA. [15] Another study by Pahlesia found that 25% of patients with chronic obstructive pulmonary disease (COPD) had a high risk of OSA based on the Berlin questionnaire. [16] Another research on asthma patients found an OSA prevalence of 9.8% with polysomnography examination. [17]

Many risk factors are known to be involved in the occurrence of OSA, such as obesity, genetics, male gender, middle age, menopause, retro or micro-nagzia, macroglossia, nasal obstruction, large neck circumference (more than 42.5 cm), and enlarged tonsils/adenoids. [18] Of the various risk factors, obesity is the main factor that can increase the risk of OSA. Apart from that, it is also known that asthma sufferers often experience sleep disturbances, and this can be a risk factor for OSA. Smoking habits and consuming alcohol can also trigger OSA. Cigarette smoke triggers inflammation and mechanical damage to the upper airway nerves and increases the risk of pharyngeal muscle collapse during sleep. Acutely, the habit of consuming alcohol can cause increased nasal and pharyngeal resistance. Consuming alcohol close to bedtime can increase the risk of hypopnea and apnea during sleep. [19]

Gradually and fundamentally, OSA occurs when the airway narrows, which results in apnea, then asphyxia, up to a period of arousal or a brief process of awakening from sleep as compensation to improve upper airway patency by increasing the activity of the dilator muscles so that airflow can be resumed. With improvement in asphyxia, the sufferer will return to sleep, and the same incident can happen again. In addition,

when entering the arousal period, sufferers generally experience nightmares until they finally wake up. [20]

It is known that three mechanisms can occur in the pathophysiology of OSA. The first is the pushing of the tongue and palate backward during sleep, which causes occlusion of the nasopharynx and oropharynx, narrowing airflow. The second is when the size of the upper airway lumen formed by the pharyngeal dilator muscles (m. Medial pterygoid, m. Tensor veli palatini, m. genioglossus, m. geniohyoid, and m. sternohyoid) experiences narrowing when negative intrathoracic pressure occurs. The narrowing of the airways can be caused by adenotonsil hypertrophy, micrognathia, retrognathia, and macroglossia. When negative intrathoracic pressure occurs, the upper airway tends to collapse if the size of the oropharynx is reduced. The third is anatomical abnormalities in the nasal area and hypopharyngeal area. Increased airway length, lateral wall thickness, and tongue volume can increase the risk of OSA. Obesity can play a role in this process; excessive body weight on the chest wall can cause diaphragm dysfunction so that it interferes with ventilation efforts during sleep, and fatty tissue in the neck and tongue causes the diameter of the airway to become narrower; this predisposes to closure when muscle tissue relaxes over time sleeping. [21]

OSA symptoms can be grouped into daytime/diurnal symptoms and nocturnal/nocturnal symptoms. Daytime symptoms include excessive daytime sleepiness (EDS), headaches in the morning, dry mouth or sore throat in the morning, reduced memory, decreased libido, fatigue, depression, and difficulty concentrating.Nocturnal symptoms include snoring, nocturia, dyspnea, diaphoresis, insomnia, and choking at night. Of the many symptoms of OSA that can occur, OSA has three main symptoms: excessive sleepiness during the day, snoring, and struggling to breathe while asleep. [22]

Polysomnography is the gold standard examination for diagnosing OSA, but due to limited numbers, requiring a trained operator, the recording process throughout the night, and the high cost, OSA sufferers often go undiagnosed. Therefore, an effective and efficient screening tool is needed to increase the identification of OSA patients, which can lead to earlier intervention that reduces morbidity and mortality. Currently, there are many kinds of screening tools in the form of questionnaires, such as the Berlin questionnaire, Stanford Sleepiness Scale, STOP-BANG questionnaire, and Epworth Sleepiness Scale (ESS). [23]

The Epworth Sleepiness Scale is a simple questionnaire to measure daytime sleepiness, the main symptom of OSA. The Epworth Sleepiness Scale consists of 8 questions, each with a score of 0-3 (according to the degree of sleepiness). The total score is the sum of the scores from the eight questions, with a score range of 0-24. A total score $\geq$ 10 indicates the presence of OSA.

OSA management is divided into non-surgical management (lifestyle changes, weight loss, and CPAP) and surgical management.

The volume and function of the upper airways of OSA sufferers can be improved by reducing weight, especially those with risk factors for obesity. Obesity causes fat accumulation around the neck, contributing to throat collapse (AAFP and Budiarsa). OSA patients who are overweight are strongly advised to reduce their weight until they reach a BMI of 25 kg/m2 or less. A 10% weight loss can reduce the Respiratory Disturbance Index (RDI) by 26% by reducing blood pressure, lung function, sleep and snoring structure, and CPAP pressure. Lying on your side can be a recommendation

because research shows that sleeping on your back can increase the apnea-hypopnea index (AHI) up to 2 times.

CPAP is a first-line and highly effective treatment for OSA. With 4-5 hours of CPAP use every night, OSA symptoms can be reduced by 90 – 95%. CPAP has not only been shown to improve sleep quality and quality of life in OSA patients, but it can also reduce blood pressure. CPAP in OSA patients can improve subjective and objective sleepiness, cognitive impairment, mood alertness, and quality of life.

There is no evidence of a specific type of surgery for treating OSA. However, various types of surgery can be performed to correct anatomical obstruction in patients with OSA, such as septoplasty, uvulopalatopharyngoplasty, tongue reduction, epiglottoplasty, and maxillomandibular advancement. Surgery may be indicated when non-surgical therapies such as nasal CPAP fail to effectively treat OSA patients or are rejected by the patient. [24]

Cigarettes are paper tubes between 70 and 120 mm long and about 10 mm in diameter, filled with shredded tobacco produced by the plants Nicotiana Tabacum, Nicotiana Rustica, and other species, or their synthetic counterparts containing nicotine and tar with or without additives. Smoking is the act of burning tobacco and then smoking it, either using a cigarette or pipe. The Indonesian Minister of Health launched the Indonesian Global Adult Tobacco Survey (GATS) in Jakarta in 2011, which revealed that Indonesia had the highest prevalence of active smoking, with 67% in men and 2.7% in women. Based on data from GATS in the same year, it was reported that Indonesia was the third country with the largest number of smokers globally, after China and India. [25]

Active smokers are those who smoke every day for at least six months and continue to do so until the time the research is conducted. According to the World Health Organization (WHO), based on the number of cigarettes smoked per day, smokers can be classified into three categories, namely: a) Light smokers consume 1-10 cigarettes per day; b) Moderate smokers consume 11-20 cigarettes per day, and c) Heavy smokers consume more than 20 cigarettes per day. [26]

A cigarette contains approximately 4,000 types of chemicals, of which 60 types are carcinogenic and 200 of them can harm the body. The main component, which can also be called the "main poison" in cigarettes, is a dangerous and addictive substance called nicotine. Then there is tar, which is carcinogenic, and carbon dioxide, which can reduce the oxygen content in the blood. Apart from that, cigarettes can also cause irritation to the eyes, nose, and throat, stimulate the recurrence of asthma, lung cancer, respiratory problems, cough up phlegm, and pose a risk of coronary heart disease, stroke, and cancer.

The upper respiratory tract mucosa can easily change due to smoking. Continuous cigarette exposure can result in abnormal histological changes in the upper respiratory tract mucosa. Histological changes that can occur include epithelial thickening, cellular hyperplasia, mucosal edema, and cilia damage. These abnormal histological changes can be one of the basic mechanisms for the occurrence of OSA. Not only that, several other mechanisms have been hypothesized to explain the relationship between smoking and OSA; the first is changes in the sleep cycle, also known as sleep architecture; the second is relaxation of the upper airway muscles and disruption of neural reflexes due to nicotine, the third is increased arousal threshold, and fourth is inflammation of the upper airway due to inhalation of cigarette smoke. [27]

Research Method

This research is an analytical observational study with a cross-sectional study design to determine the relationship between smoking and the risk of obstructive sleep apnea (OSA) in Indonesian Christian University Faculty of Law students class 2018-2021. This research was carried out at the Faculty of Medicine, Indonesian Christian University, Jl. Major General Sutoyo No.2, RT.2/RW.11, Cawang, Kec. Kramat Jati, East Jakarta City. Data collection was carried out from January 2021 to February 2022. The population of this study was 695 law faculty students at the Indonesian Christian University class of 2018-2020. inclusion criteria. The Slovin formula was used to determine the members of the population sampled in this study. Thus, the sample used, namely 94 students, is needed for this research. A simple random sampling method was used. In this method, samples are obtained randomly without paying attention to strata in the population. The research instrument used was an online questionnaire (g-form) containing a smoking habit questionnaire and the ESS (Epworth Sleepiness Scale). The steps taken in collecting data for research are: a) Selecting samples based on inclusion criteria using simple random sampling techniques; b) Collect data using a questionnaire distributed online; c) Manage and analyze data using IBM SPSS (Statistical for Social Science) and Microsoft Office Excel programs; and d) create a report on the results of the analysis

Data collected through questionnaires is processed using the IBM SPSS (Statistical for Social Science) program and the Microsoft Office Excel 2010 program.

Result and Discussion

This research was conducted in February 2021 involving 94 students from the 2018-2021 class of Indonesian Christian University, Faculty of Law, who were willing to be research samples and met the inclusion and exclusion criteria. All subjects were asked to agree by filling out a Google form containing informed consent before the research. This research was carried out by giving a questionnaire and asking respondents to fill out the questionnaire using a Google form.

Table 1. Characteristics of Research Respondents

Characteristics	Frequency (n)	Percentage (%)
Gender		
Male	59	62,8
Female	35	37,2
Age		
17	2	2,1
18	19	20,2
19	21	22,3
20	19	20,2
21	23	24,5
22	6	6,4
23	3	3,2
24	1	1,1
Smoking History		
Smoke	51	54,3
Do not Smoke	43	45,7
Total	**94**	**100**

Table 1 shows that of the 94 respondents, 59 were male (62.8%), and 35 were female (37.2%). The average age of most respondents in this study was 21 (24.5%). The distribution of respondents who smoke is greater than those who do not, namely 54.3%.

Table 2. Characteristics of Smoker Respondents

Characteristics	Frequency (n)	Percentage (%)
Gender		
Male	44	86,3
Female	7	13,7
Age		
17-19 years	18	35,3
20-24 years	33	64,7
Degree of Smoker		
Light	41	80,4
Moderate	10	19,6
Total	**51**	**100**

From Table 2 above, there are more men smokers than women (86.3%). Based on age range, 35.3% of smoker respondents were aged 17-19, and 64.7% were aged 20-24. Of the total respondents who smoke, the majority are light smokers (80.4%).

Based on research that has been carried out, an overview of the prevalence of OSA in the population is as follows:

Table 3. Distribution of Respondents Based on OSA

Characteristics	Frequency(n)	Percentage (%)
OSA	45	47,9
No OSA	49	52,1
Total	94	100

Table 3 shows that 47.9% of students experienced OSA compared to students who did not experience OSA.

Table 4.4 Bivariate analysis of the relationship between gender and OSA

Gender	OSA		Total	P
	OSA	No OSA		
	n (%)	n (%)	n (%)	
Male	44 (74,6)	15 (25,4)	59 (100)	0,000
Female	7 (20)	28 (80)	35 (100)	
Total	51	43	94 (100)	

From Table 4 above, it was found that the sample of men who experienced OSA was 44 people (74.6%), and 15 people did not experience OSA (25.4%). Meanwhile, the sample of women who experienced OSA was seven people (20%), and those who did not experience OSA were 28 people (80%). The results of the chi-square analysis showed a value of $p = 0.000$ ($p < 0.05$), which means that there is a significant relationship between gender and OSA in law faculty students at the Indonesian Christian University. The following are the results of the analysis using chi-square with a 2x2 table:

Table 5. Bivariate analysis of the relationship between smoking and OSA

Smoke	OSA		Total	P
	OSA	No OSA		
	n (%)	n (%)	n (%)	
Smoke	21 (41,2)	30 (58,8)	51 (100)	0,214
Do not Smoke	24 (55,8)	19 (44,2)	43 (100)	
Total	45	49	94	

From Table 5 above, it was found that 21 people (41.2%) of the sample with smoking habits experienced OSA, and 30 people (58.8%) did not experience OSA. Meanwhile, 24 people (55.8%) of the sample who did not have a smoking habit experienced OSA, and 19 people (44.2%) did not experience OSA. The results of the chi-square analysis showed a value of $p = 0.214$ ($p > 0.05$), which shows no significant relationship between smoking habits and OSA among law faculty students at Indonesian

Christian University. The following are the results of the analysis using chi-square with a 2x2 table:

Table 6. Bivariate Analysis of the Relationship between Degree of Smoker and OSA

Degree of Smoker	OSA		Total	P
	OSA	No OSA		
	n (%)	n (%)	n (%)	0,728
Light	17 (41,5)	24 (58,5)	41 (100)	
Moderate	5 (50)	5 (50)	10 (100)	
Total	22	29	51	

From Table 6 above, it was found that 17 people (41.5%) in the sample of mild smokers experienced OSA, and 24 people (58.5%) did not experience OSA. Meanwhile, five people (50%) in the sample of moderate smokers experienced OSA, and five people (50%) did not experience OSA. The results of the chi-square analysis gave a value of $p = 0.728$ ($p > 0.05$), which can be interpreted as meaning that there is no significant relationship between the degree of smoking and OSA in law faculty students at the Indonesian Christian University.

The results of this study showed that the prevalence of OSA in law faculty students at the Indonesian Christian University was 47.9%, dominated by men (74.6%). It aligns with research conducted by Brilliana et al., which found an OSA prevalence of 46.8% in a sample of high school students. [28]

In this study, all respondents had a normal body mass index (BMI) ranging from 18.5 to 24.9 kg/m2, so the incidence of OSA would not be affected by BMI. Obesity is the main risk factor for OSA, and in general, OSA sufferers are often overweight. The mechanism that can explain this is that obese individuals experience an increase in fat mass in the lateral walls of the pharynx, which causes the pharynx to become more susceptible to collapse, leading to the narrowing of the pharynx. A study in a Japanese population showed that neck circumference was associated with excess BMI and was also associated with the severity of OSA. [29]

The results of this research showed that 54.3% of Indonesian Christian University law faculty students were smokers, and 64.7% of them were aged 20-24 years. Based on the number of cigarettes smoked per day, smoking levels are divided into two categories, namely light smokers, who smoke 1-10 cigarettes per day, and moderate smokers, who smoke 11-20 cigarettes per day. The majority of respondents in this study fell into the light smoker category (80.4%). It is in line with research by Sawitri et al. [30], which concluded that the majority of students who smoke are light smokers, and most are aged 20-24 years. Based on gender, men constituted the largest number of smoking respondents (86.3%). It is in line with data from The Tobacco Atlas, which states that Indonesia has the largest number of male teenage smokers (over 15 years of age), namely 66% of men; next in second place is Russia at 60%, then in third place followed by China (53%), Philippines (48%), Vietnam (47%), Malaysia (44%), India (24%), and Brazil (22%). [31]

The research found a significant relationship between gender and OSA with a p-value of 0.000 (p-value < 0.05). It is supported by several epidemiological studies which state that the prevalence of OSA sufferers in men is 2-3 times greater than in women. [43] In line with research by Sasongko et al., a significant relationship was

found between gender and OSAS in a sample of ischemic stroke patients. [44] Research conducted by Duarte and Silveira also revealed that of the various factors related to OSA that they studied (gender, age, BMI, snoring symptoms, and hypertension), gender was the main predictor for diagnosing OSA. [32]

Several hypotheses can explain the male gender having a greater risk of developing OSA, namely differences in fat deposition, differences in anatomy and function of the upper airway, and hormonal effects between men and women. Deposition of fat and adipose tissue in the upper airway tends to be greater in men than women, leading to increased upper airway resistance and making it more susceptible to collapse. Also, men have larger upper airway soft tissue structures than women. Hormonal differences in men and women also affect the upper airway muscles. Hormones in women seem to have a protective role in the occurrence of OSA. This explanation is supported by the prevalence of OSA, which is 2 to 3 times higher in post-menopausal women than in pre-menopausal women.

The pathogenesis of OSA is a combination of various factors, such as anatomical factors and dysfunction of the muscles responsible for airway patency. Based on epidemiological data, OSA is often experienced by men. Various potential mechanisms could explain this event. First, after puberty, men secrete the hormone testosterone, which increases the risk of upper airway collapse. Meanwhile, the hormones estrogen and progesterone secreted by women have a protective function against upper airway collapse. Second, the weight gain that usually occurs with age has different characteristics between the sexes; obese men tend to have central obesity, which can predispose to upper airway collapse, while female obesity is generally peripheral and does not endanger airway mechanics.

At the same body size (men and women), men have several upper respiratory tract anatomical characteristics, namely a higher base of the nose and a larger, longer, and narrower nasal cavity than women. In addition, men also have a longer, more flexible oropharynx and a larger, fatter posterior tongue. It makes men more susceptible to nighttime collapse (which will develop into OSA) compared to women. Another characteristic of men is that they have a larger neck circumference than women. In the upper airway of men, it was revealed that there was higher pharyngeal and supraglottic resistance than in women, which could indicate that men had lower upper airway potency. It can predispose to upper airway collapse, so men are more at risk of experiencing obstructive sleep apnea than women. [11]

Upper airway dilator muscle activity contributes to airway patency. Many variables, including blood gases, sleep-wake state, sex hormones, blood pressure, temperature, lung inflation, pharyngeal airflow, and negative intrapharyngeal pressure, influence pharyngeal dilator muscle activity. As one of the largest pharyngeal dilator muscles, the genioglossus has been widely studied about OSA. Studies have shown that the decrease in genioglossus activity after the transition from wakefulness to non-REM sleep is greater in patients with OSA than in normal patients. Geniglossal activity in women during inspiration and expiration is greater than in men, indicating that women's airways are more stable and less prone to collapse. [11]

Longer airway length and greater soft tissue volume in the lateral pharyngeal wall are thought to be associated with greater susceptibility to collapse in men. It was previously reported that genioglossus muscle activity was positively correlated with progesterone levels and increased significantly with combined estrogen and

progesterone replacement in post-menopausal women. It suggests a hormonal role in maintaining upper airway stiffness by the dilator muscles. [12]

This research examines the relationship between smoking and OSA in law faculty students at the Indonesian Christian University. The data obtained shows that 51 respondents (54.3%) are smokers. There were 21 respondents (41.2%) who had a smoking habit and experienced OSA; in other words, almost half of the total students who had a smoking habit experienced OSA. The relationship between smoking and OSA in this study was analyzed using the Fisher's Exact test, and based on this test, a p-value of 0.724 ($p > 0.05$) was found, where this can be interpreted to mean that the relationship between smoking and OSA in students at the law faculty of the Indonesian Christian University is statistically non-existent meaningful relationship.

The hypothesis that there is a relationship between smoking and OSA in this study was not proven; this is in line with research by Hsu et [33] al, which showed that there was no significant relationship between smoking and OSA after BMI, gender, and age had been adjusted. This study confirmed that the common cause of OSA is the abnormal anatomical shape of the upper airway and dysfunction of the surrounding dilator muscles. Likewise, research conducted by Ioannidou et al., as well as meta-analysis conducted by Taveira et al., concluded that there is no significant relationship between smoking and OSA. [34]

However, this is different from research conducted by Kashyap et al., which stated that there was a relationship between smoking and OSA. In this study, it was concluded that smoking could be a risk factor for OSA, and in this study, it was stated that a smoker had 2.5 times the risk of experiencing OSA compared to a non-smoker. [8] It is also supported by research by Lin et al., which states that there is a significant relationship between smoking and OSA. Several hypotheses can explain the mechanism of smoking as a risk factor for OSA. The first is increased nasal resistance/airflow resistance due to inflammation caused by inhalation of cigarette smoke. Another mechanism that can explain how smoking can be related to OSA is due to the dangerous substance contained in it, namely nicotine. The muscles in the upper airway have an important role in maintaining upper airway patency while sleeping, especially the pharyngeal dilator muscles. Nicotine can interfere with upper airway neuromuscular reflexes, resulting in decreased activity of the airway dilator muscles and causing dysfunction of the dilator muscles so that the airways will narrow and be susceptible to upper airway collapse. [4]

This study also examined the relationship between smoking levels and OSA. The smoking degree category is divided into light smokers, who consume 1-10 cigarettes per day, and moderate smokers, who consume 11-20 cigarettes per day. From the data that has been obtained, the majority of students who smoke are light smokers (76.9%), and only a small portion fall into the moderate smoker category (13.1%). Statistically, the bivariate analysis using the Fisher exact test results showed a value of $p = 0.379$ ($p > 0.05$), which shows no significant relationship between the degree of smoking and OSA.

The hypothesis that there was a relationship between the degree of smoking and OSA in this study was also not proven. Likewise, research conducted by Esen et al. stated that there was no significant relationship between the degree of smoking and OSA. [35]

On the other hand, similar research was also carried out by Rahmadati, where she concluded that there was a significant relationship between the degree of smoking and OSA. In his thesis, it is explained that as a person's degree of smoking increases, the

risk of experiencing OSA will increase. It is also in line with research by Anggara, which concluded that there was a significant relationship between the level of cigarette consumption and sleep disorders. One of the mechanisms that can explain this is histological changes in the upper respiratory tract, namely epithelial thickening, cellular hyperplasia, mucosal edema, and disruption of the mucociliary system. The thickening of the mucosa is also caused by an inflammatory process played by Calcitonin Gene-Related Peptide (CGRP). [39]

The relationship between smoking and OSA is currently not known with certainty. Data from the Wisconsin Sleep Cohort Study reported that, after confounding factors were adjusted for, active smoking was associated with a greater likelihood of OSA risk, especially among heavy smokers. Additionally, another study by Valor et al. reported that heavy smokers had more severe OSA and that smoking was associated with disease diagnosis at an earlier age. However, in the absence of samples of heavy smokers, this cannot be confirmed in this study. [10]

There are several limitations to this research, namely: a) Collecting primary data using a questionnaire; b) The research was carried out during the Covid-19 pandemic so that the distribution of questionnaires was carried out online; c) The number of samples obtained is limited; d) There were no samples of heavy smokers; and e) The type of cigarette consumed by the respondent was not studied.

Conclusion

The conclusions from this research are: a) The prevalence of OSA in law faculty students at the Indonesian Christian University is 47.9%; b) The majority of Indonesian Christian University law faculty students have a smoking habit (54.3%); c) Most of the law faculty students at the Indonesian Christian University who have a smoking habit are light smokers (80.4%); d) There is no relationship between smoking and OSA in law faculty students at the Indonesian Christian University; e) There is no relationship between the degree of smoking and OSA in law faculty students at the Indonesian Christian University. Thus, it is recommended to conduct further research with a larger sample of smokers ranging from light smokers, moderate smokers, to heavy smokers; conduct further research regarding the relationship between length of smoking and type of cigarette on OSA, and in the next study, the diagnosis of OSA was confirmed using the gold standard tool, namely polysomnography.

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