



## Vital Lung Capacity in Active Smokers

Edy Sapada<sup>1</sup>, Wita Asmalinda<sup>2\*</sup>)

<sup>1,2</sup>STIK Siti Khadijah Palembang, Indonesia

### ARTICLE INFO

#### Article history:

Received 19 March 2023  
Accepted 15 June 2023  
Published 30 June 2023

#### Keyword:

Vital Lung Capacity  
Active Smokers

### ABSTRACT

The prevalence of Indonesian smokers is increasing, the age of smokers is getting younger every day, and the number of cigarettes consumed is increasing. Smoking behavior or habits can have an impact on the emergence of pulmonary ventilation disorders due to irritation and excessive mucus secretion in the bronchi. The study aimed to determine the correlation between age, number of cigarettes, and duration of smoking with the vital capacity value of the lungs of active smokers. This type of research is analytic with a cross-sectional approach. This research was conducted for 30 days at the Ibnu Sina Clinic in Palembang. The sample of this study was active smokers who met the inclusion, and exclusion criteria. The number of samples is 50 respondents. Data analysis used the SPSS version 16 program. Sample characteristic data and measurement results were assessed using the Spearman test. The results of the study found there was a strong correlation between age variables in smoking duration and the vital lung capacity of the respondents. It was concluded that there was a significant correlation between the variables of age, number of cigarettes and duration of smoking with active smokers vital lung capacity values. It is suggested to conduct further research on cytokine variables

This open access article is under the [CC-BY-SA](https://creativecommons.org/licenses/by-sa/4.0/) license.



### Kata kunci:

Kapasitas Vital Paru  
Perokok aktif

#### \*<sup>)</sup> corresponding author

Wita Asmalinda, SST., M.Kes

Faculty of Midwifery  
Jl H.M.Saleh Perumahan Alam Raya  
residence Blok E1 No 14 Kec. Sukarami  
Kota Palembang-Indonesia 30153

Email: wita\_asmalinda@yahoo.co.id

DOI: 10.30604/jika.v8i2.2032  
Copyright 2023 @author(s)

### ABSTRAK

Prevalensi perokok Indonesia makin meningkat, umur perokok tiap harinya semakin muda, dengan jumlah rokok yang dikonsumsi semakin banyak. Perilaku atau kebiasaan merokok dapat berdampak pada timbulnya gangguan ventilasi paru akibat iritasi dan sekresi mucus yang berlebihan pada bronkus Tujuan penelitian adalah untuk mengetahui korelasi antara umur, jumlah rokok dan lamanya mengkonsumsi rokok dengan nilai kapasitas vital paru perokok aktif. Jenis penelitian ini bersifat analitik dengan pendekatan *cross sectional*. Penelitian ini dilaksanakan selama 30 hari di Klinik Ibnu Sina Palembang. Sampel penelitian ini adalah perokok aktif yang memenuhi kriteria inklusi dan eksklusi. Jumlah sampel sebanyak 50 responden. Analisis data menggunakan program SPSS versi 16. Data karakteristik sampel dan hasil pengukuran dinilai menggunakan uji *spearman*. Hasil penelitian didapatkan ada korelasi yang kuat antara variabel umur dan lama merokok dengan nilai kapasitas vital paru responden. Disimpulkan bahwa umur, jumlah rokok dan lama merokok berpengaruh terhadap nilai kapasitas vital paru perokok aktif. Disarankan untuk melakukan penelitian lebih lanjut pada variabel sitokin.

This open access article is under the [CC-BY-SA](https://creativecommons.org/licenses/by-sa/4.0/) license.



## INTRODUCTION

The last decade revealed that there was a significant increase in the prevalence of active smokers in Indonesia (Nadia, L., 2016; Sapada, 2020). The cause of death due to smoking worldwide is 1:10 (Hanewinkel, R., 2010; Nadia, L., 2016). There has been a large increase in smoking behavior in Indonesia, which tends to smoke at a young age (Rahmat, M., 2013). Adolescents who consume cigarettes are increasing every year, with the total consumption of cigarettes increasing per day (Nadia, L., 2016). There are 2.0% of teenagers starting to smoke at the age of 10-14 years. As many as 0.7% of these adolescents regularly smoke every day, with an average consumption of 10 cigarettes per day (Rahmat, M. 2013). It is also concerning that the largest group of cigarette consumers is people with low incomes. Allegedly the factors of environmental influence and the association of adolescents are the originators of this (Atmajaya, 2007; Sapada, 2021). Starting from association and then continuing to become nicotine addiction (Larasati, A., 2016; Abdurahman, WF, 2010). Smoking habits are an interaction of physiological, psychological and cognitive aspects (Siregar, 2020). Another supporting factor is that cigarette users do not directly feel the bad effects of smoking in the short term so there is a tendency to reject health appeals about the dangers of smoking (Ministry of Health RI., 2019; Larasati, A., 2016; Susana, 2003). According to the World Health Organization (WHO) in developing countries around 50-60% of men and 10% of women have smoking habits while in developed countries around 30% of men and 30% of women have smoking habits (Buchari, 2007; Sapada, 2020). The prevalence of smokers in Indonesia includes 67% of men smoking, and 2.7% of women smoking. From the smoker's data, 80.4% of the current smoking population only smokes kretek cigarettes. 1.7% of the population consumes chewing tobacco (Asmalinda, 2021; Ministry of Health RI., 2015; Abdurahman WF, 2010; Ramos V, 2009; Bakhtiar, A., 2016).

The Active smokers are individuals who consume cigarettes regularly and periodically (Ardam, 2015; Sapada, 2020; Asmalinda, 2021). According to Asmalinda (2021), active smokers are on average able to spend 10-20 cigarettes per day. When active smokers carry out smoking activities, the cigarette smoke released into the environment is 4-6 times more nicotine than the smoke inhaled by the smoker himself. It is assumed that when smoking, the smoke produced by a cigarette that is still burning will continue and be released into the air even though it is not inhaled (Susana, 2003; Asmalinda, 2021). Smoking behavior or habits can have an impact on the emergence of pulmonary ventilation disorders due to irritation and excessive mucus secretion in the bronchi (Abdurahman, WF., 2010; Alsagaff, H & Mukty. 2008; Nadia, L., 2016). Toxic substances in cigarettes over a long period time will increasingly accumulate in the body, causing the exchange of oxygen with carbon dioxide in the alveoli to be disrupted and in severe conditions will cause damage to the alveoli (Barakati et al., 2015; American Lung Association, 2017). This damage will reduce the number of alveoli that function in the respiration process, causing a decrease in the function of the lung organs and a decrease in lung vital capacity (Barakati et al., 2015).

The content in cigarette smoke and inhaled cigarettes can accumulate in the body cumulatively so that the longer a person smokes, the more harmful substances in cigarettes will enter the body (Adeniyi, B. O., & Erhabor, G. E. 2011). In the lungs, there is *Glutathione (GSH)* which is an antioxidant compound that protects the lungs. In active smokers, it is

reported that there is a decrease in *Glutathione* metabolism (Nadia, L., 2016). This is thought to activate the decreased elasticity of the lungs resulting in emphysema (widening of the alveoli) (Nadia, L., 2016; Budiman, 2018). During their journey, patients with emphysema take a long time for their breathing activities (Nadia, L., 2016). If this condition is allowed to continue, it will cause interference with the vital capacity of the lungs and at certain stages will cause further effects in the form of disorders or diseases in the lungs such as chronic obstructive pulmonary disease (COPD) to cancer (Adeniyi, B.O., & Erhabor, G. E. 2011; Antao, 2015).

The Vital lung capacity (KVP) is the amount of air that can be expelled by voluntary effort after deep inspiration (Adeniyi, B.O., 2011; Guyton and Hall, 2012; Bakhtiar, A., 2016; Asmalinda, 2021). Vital lung capacity is the sum of the tidal volume, inspiratory reserve volume, and in-expiratory reserve volume (Guyton and Hall, 2012; Bakhtiar, A., 2016; Asmalinda, 2021). Vital lung capacity reflects the change in maximal lung volume which is useful for ascertaining the picture of functional lung capacity. The vital lungs capacity can be measured using a pulmonary function test kit. The most basic test used is spirometry (Sherwood, 2014; Pellegrino, R., 2010; Smeltzer, SC., 2002; Sapada, 2020). The measurement of the vital capacity of the lungs is the largest volume of air that can be exhaled after the deepest inspiration. During inspiratory breathing, there will be suction pressure which will affect the increase in lung volume and capacity (Bakhtiar, A., 2016; Pinugraha, B., 2017). Vital capacity values are influenced by age, sex, body weight, (Pujiastuti, BE., 2012; Zulfrianiangrum, H., 2016; Sapada, 2020), body position, respiratory muscle strength, the ability of the lungs and chest cavity to expand (Bakhtiar, A., 2016) The vital capacity of the lungs is reduced if there is lung and heart disease which causes lung congestion and respiratory muscle weakness (Hapsari, R., 2009; Gunther, AC., 2006; Citizen, 2015).

Factors that affect the quality of lung capacity by showing interference in the form of restrictive, obstructive, or a combination of both. Several internal and external factors can influence lung disease. Internal factors include the body's defense system, anatomically and physiologically, age, gender, history of disease, nutritional status, and individual susceptibility. External factors include exposure to dust, exercise habits, work history, work environment, use of respiratory protective equipment, and smoking habits (Dwiputra, 2019; Sapada, 2020). One of the indicators in assessing the level of lung health is the vital lung capacity (VAD). According to Pellegrino & Antonelli (2010) the change in maximal lung volume which is useful for ascertaining the functional capacity of the lung or what is called the vital capacity of the lung is an important measurement to determine lung restrictive abnormalities which are shown by decreased lung function. To check the condition of the lungs, the first thing the subject has to do is maximal inspiration, then the next thing to do is maximal expiration. According to research by Hapsari, R (2009) every cigarette puff can increase heart rate and blood pressure resulting in a lack of oxygen in the bloodstream. The existence of toxic substances contained in cigarettes will inhibit gas exchange in the alveoli. This of course will reduce the number of functional alveoli that play a role in the respiration process, and as result there will be a decrease in lung function. The purpose of this study was to determine the vital capacity of active smokers' lungs.

**METHODS**

This research is analytic observational research that is based on natural events without any treatment of the object under study. The research design based on the time of implementation is a cross-sectional study that aims to analyze the effect of age and length of smoking and the number of cigarettes consumed per day on lung vital capacity in objects that are actively smoked. This research was carried out at the Palembang Ibnu Sina Clinic which is located at Jalan Kasnariansyah number 1627 RT 21 RW 07 Kelurahan 20 Ilir DIV Ilir Timur I KM 4.5 District Palembang City for 1 month from 01 September to 30 September 2022. The sample in the study This is part of the active smokers who visited the Ibnu Sina Palembang Clinic totaling 50 respondents. The sampling method used Consecutive Sampling which means that all active smokers who meet the inclusion criteria will become the research sample. To all respondents explained the purpose, procedure, benefits, and risks as a sample in this study. After obtaining the respondent's consent, they then signed an informed consent to become the respondent in this study. The independent variables in this study were the age of active smokers, duration of smoking, and the number of cigarettes consumed per day. While the dependent variable is the value of lung vital capacity.

The data collected is primary data obtained through the process of filling out questionnaires to determine age, duration of smoking, number of cigarettes consumed per day and direct assessment to assess lung function using a lung function measuring instrument, namely a spirometer that uses electrical energy. Examination of lung vital capacity values was carried out by health workers at the Ibnu Sina

Clinic in Palembang. Equipment and materials used for assessing lung function using lung function measuring devices, namely a spirometer that uses electrical energy, mouthpieces, alcohol cotton, trays, oval bowls (*nierbekken*), handsconds, tables, chairs, trash cans, and chlorine solution. The procedure for collecting lung vital capacity data is as follows: the respondent is in the correct position, sitting upright or standing, with feet flat on the floor, and not crossing. Asking the respondent to loosen their clothing, because clothes that are too tight, can give a limited view on the spirometer giving inaccurate results, it could be that the lung vital capacity results obtained are lower than they are. Ask the respondent to massage the nose using the hand or nose clip, and perform a spirometry examination using a spirometer and mouthpiece. Respondents were asked to breathe normally first, then take a deep breath and exhale maximally, record the results displayed on the spirometer screen, and include vital capacity (VC).

This research has received an ethical approval recommendation from the Palembang Health Polytechnic Research Ethics Commission Number. 0196/KEPK/Adm.2/II/2022.

**RESULTS AND DISCUSSION**

An analytic observational study of Lung Vital Capacity in active smokers has been carried out with a cross sectional design. The data obtained in this study were then analyzed statistically using the SPSS version 16 program, which included univariate and bivariate analysis of the independent variables and the dependent variable

**Table 1. Characteristics of Respondent**

Variable	N	Min	Max	Mean	Median	SD
Age (year)	50	17	62	37.44	36.00	14.224
Number of Cigarettes (cigarettes/day)	50	9	27	15.58	13.00	5.230
Long Smoking (year)	50	1	35	14.20	13.00	9.480
Vital Lung Capacity (ml)	50	3367	4624	4140.82	4183.00	303.999

From Table. 1 above it is known that of the 50 respondents, the lowest age was 17 years and the highest age was 62 years. The average number of cigarettes consumed by respondents was 15.58 cigarettes per day with an SD value of 5,230. Of the 50 respondents, the average smoking duration was 14.20 years, the minimum smoking duration was 1 year and the longest was 35 years. The lowest respondent's lung vital capacity value was 3367 ml and the highest was 4624 ml, with a mean value of 4140.82 ml with an SD value of 303.99. Active smokers are people who consume cigarettes regularly every day (Saputri, et al., 2018; Asmalinda, W; 2021). The criteria for smokers include: light smokers can spend 1-10 cigarettes/day, moderate smokers

can to smoke 11-19 cigarettes/day, and heavy smokers can spend >20 cigarettes/day (Sapada, E., 2020). Age is related to the aging process, the older a person is, the higher the chance for decreased lung function (Fikriyah, S., 2012) Asmalinda, W., 2021; Saputri, et al., 2018). Decreased lung function begins at the age of 30 years and accelerates after the age of 40 years. Another supporting factor for decreased lung function is if a person has a habit of smoking for a long period time and consumes large amounts of cigarettes every day. This process is exacerbated when active smokers rarely exercise and lack activity. (Gunther, 2006; Ganesha, 2020; Juarfianti, 2015; Asmalinda, W., 2021).

**Table 2: Correlation between Agen, Number of Cigarettes, and Duration of Smoking with Respondent's Vital Lung Capacity**

Variable	Vital Lung Capacity		p-value
	N	Correlation Coefficient	
Age (year)	50	-0.830	0.0001
Number of Cigarettes (cigarettes/day)	50	-0.540	
Duration of Smoking (year)	50	-0.832	

From Table 2 above, it can be seen that based on the results of the correlation test using the Spearman test on the variables of age and lung vital capacity, a significance value (p-value) of 0.0001 was obtained, which indicated that the correlation between the respondent's age and lung vital capacity was significant. The Spearman correlation value for age and lung vital capacity is -0.830 indicating that the direction of the correlation is negative with a very strong correlation strength. The Spearman correlation test between the number of cigarettes consumed per day by the respondent and the vital lung capacity value obtained a significance value of 0.0001, this indicates that the correlation between the number of cigarettes consumed per day by the respondent and the vital lung capacity value is significant. The Spearman correlation value is -0.540 indicating that the direction of the correlation is negative with moderate correlation strength. The results of the Spearman correlation test between smoking duration and lung vital capacity obtained a significance value of 0.0001 which indicated that the correlation between the number of cigarettes consumed per day by respondents and the value of lung vital capacity was significant. The Spearman correlation value is -0.832 indicating that the direction of the correlation is negative with a very strong correlation strength. A negative value means that the greater the value of one variable, the smaller the value of the other variable.

Cigarettes are processed tobacco wrapped in cigars produced from the plants *Nicotiana tabacum*, *Nicotiana rustica* and other species or their synthetic counterparts which contain nicotine and tar with or without additives (European Respiratory Society, 2019). The tar content contained in cigarettes is a polynuclear aromatic hydrocarbon compound which is part of the cigarette particles after the nicotine content and water vapor are removed. surfaces of teeth, respiratory tract and lungs. This deposition varies between 3-40 mg per cigarette, while the tar content in cigarettes ranges from 24-45 mg. (Ardam, 2015; Dwiputra, 2019). Tar in cigarette smoke contains carcinogenic ingredients that can paralyze the cilia in the lungs thereby contributing to the occurrence of emphysema, chronic bronchitis, and lung cancer, besides that, it also increases the risk of squamous cell carcinoma of the larynx, and disrupts the function of the organs of the mouth, vocal cords, throat, , kidneys, bladder, uterus, and ovaries (Barakati, RV., 2015; Sapada, 2020).

The next ingredient in cigarettes is carbon monoxide, which is a colorless, odorless, tasteless, and toxic gas. This gas is the result of incomplete combustion of carbon-containing materials or combustion under high pressure and temperature. Carbon monoxide contained in cigarettes can bind to blood hemoglobin. As a result, the oxygen in hemoglobin will be eliminated and cannot be used by the body. The effect caused by the carbon monoxide content is that the blood vessel tissue will narrow and harden resulting in blood vessel blockage. One cigarette that is burned contains 3-6% carbon monoxide and 400 ppm smoked by smokers can increase carboxyhemoglobin levels in the blood by 2-16% (Khan, 2010; Nadia, L., 2016).

The high concentration of nicotine in the arteries is obtained through inhalation of cigarette smoke and the nicotine balance between blood and brain (Susana, 2003). Nicotine induces the formation of active compounds that can interfere with blood flow by inducing inflammation in blood vessels (Sharon et al., 2001; Jennifer et al., 2001; Nadia, L., 2016). Disturbances caused to these blood vessels can cause vasoconstrictive and cellular pathology such as disturbances in the balance of Ca<sup>2+</sup> in the arterioles of the brain which can

cause strokes (Sharon et al., 2001; Jennifer et al., 2001; Nadia, L., 2016). Vasoconstriction occurs in the blood vessels of the brain, presumably due to thromboxane A2 which is activated by nicotine. The occurrence of increased *NO* (*nitric oxide*) production and activation of K<sup>+</sup> junction pathways and increased thromboxane A2 caused by nicotine have a significant effect on the occurrence of vasoconstriction in the blood vessels of the brain, thereby increasing the occurrence of stroke due to smoking (Nadia, L., 2016; Taufick, ALK., 2023).

Nicotine is a chemical that can poison the nerves. One cigarette contains 1 mg of nicotine which is absorbed through the lung epithelium. Nicotine, which has low concentrations, is a stimulant, which can increase blood pressure, activity, and memory, constrict peripheral vessels, and cause dependence on the wearer. Meanwhile, at high concentrations, it can act as a depressant (Jennifer, M.K., 2001; Siregar, 2020). The binding of nicotine and acetylcholine to nicotine receptors causes conformational changes that can open and close ion channel receptors thereby affecting neuron activity, synapse communication and behavior. The addiction process begins with the interaction between nicotine, and nicotine receptors in the brain in the mesolimbic area of the dopamine system in the Ventral Tegmental 11-neuron area. This interaction supports the activation of the Central Nervous System (CNS) including the dopamine Mesoaccumbens system. Activation of this nerve will result in the release of dopamine. Increased dopamine in the brain can stimulate the brain and activate reward pathways, namely feelings and behavior regulation systems caused by feedback mechanisms in the brain. It is strongly suspected that this mechanism encourages cigarette addicts to continue using nicotine and triggers extreme physical dependence on nicotine (Jennifer, M.K., 2001; Siregar, 2020). In addition, dopamine itself is a chemical compound produced by the body that is responsible for feelings of pleasure, joy, motivation and self-confidence. in humans. It is this effect desired by smokers that causes addiction. So if someone consumes cigarettes continuously it will increase dopamine levels in the body which results in a feeling of addiction. If the work of the nervous system is disrupted, it will cause various changes in the body's systems, especially in the respiratory system. The function of the respiratory system and blood circulation will increase during childhood and will reach its peak at the age of 19-21 years (Siregar, 2020)

The accumulation of cigarette consumption per day among active smokers is increasing every year, this is suspected because the nicotine content in cigarettes has an addictive effect (physical addiction), namely the failure of active smokers to control smoking behavior, which is indicated by the increasing number of cigarettes consumed day per day. Smoking is considered a social trend and provides pleasure (Siregar, T., 2020). According to Siregar (2020) the process of addiction due to repeated and continuous exposure activates the release of the neurotransmitter dopamine in the mesocorticolimbic dopaminergic cycle, which has a reward effect. The body responds by altering circulatory function in the typical compulsive behavior. In a state of addiction, cigarette addicts experience a lack of control, namely increasing the number of cigarettes consumed, because of the strong dependence on cigarettes and the convenience of smoking for a long time. Smoking behavior becomes anticipation, in the form of the assumption that smoking is a problem-solving or coping strategy. Cigarette addicts at the Neglect social life stage, ignore social life, focus more on their interests, and lose

awareness of the effects of smoking on environmental health and family health and passively also inhale cigarette smoke (Siregar, T., 2020). Cigarette addicts, if they want to stop consuming cigarettes, are constrained by the effects of nicotine, namely the feeling of always feeling uneasy, anxious, uncomfortable, and loss of concentration, emotions that are sometimes out of control, which activates, and stimulate them to consume cigarettes again in the hope of reducing the effects of nicotine they feel (Taufick, 2023).

Smoking habits cause oxidative stress due to cigarette smoke and the 4000 chemicals contained therein such as Tar, Ammonia, nicotine, phenol, acrolein, carbon monoxide, carbon, nitrogen oxides, and hydrogen cyanide (Rahma, 2019) are exogenous-free radicals for the body (Sinaga, 2016; Budiman 2018; Rahma, 2019). The body responds to oxidative stress by producing Reactive Oxygen Species (ROS) and Reactive Nitrogen species (NOS), which are chemicals that play an active role in reducing endurance (Sinaga, 2016). Oxidative stress is the main trigger for inflammatory reactions (Rahma, 2019). The inflammatory process does not only take place in the lungs but also occurs systemically, which is characterized by increased levels of *C-reactive protein* (CRP), *tumor necrosis factor- $\alpha$*  (TNF- $\alpha$ ), *interleukin 6* (IL-6) and *interleukin 8* (IL8). This systemic response reflects the progression of lung disease and subsequently develops into decreased skeletal muscle mass (muscle wasting), coronary heart disease and atherosclerosis. Gas exposure from cigarette smoke activates alveolar macrophages and airway epithelial cells to form chemotactic factors. The release of this chemotactic factor induces an infiltration mechanism for hematopoietic cells in the lung which can cause structural damage to the lung. Infiltration of these cells can be a source of new chemotactic factors and prolong the pulmonary inflammatory reaction into chronic and progressive disease. Organs that are at high risk of being affected by inhaled cigarette smoke are the lungs (Ganesha, 2020). Cigarette smoke also reduces the antioxidant capacity in plasma due to a decrease in sulfhydryl protein or glutathione (GSH). This decrease in GSH causes an increase in lipid peroxidase and transcription of proinflammatory cytokine genes that play a role in pulmonary obstruction. Several body cells that have been shown to be damaged by free radicals are the lungs, blood vessel endothelial cells, and the heart (Ganesha, 2020; Sinaga, 2016; Budiman 2018; Rahma, 2019).

The response of the airway epithelium to exposure to cigarette smoke is in the form of an increase in the number of cytokines such as IL-8, *macrophage inflammatory protein-1  $\alpha$*  (MIP1- $\alpha$ ), and *monocyte chemoattractant protein-1* (MCP-1). An increased number of T lymphocytes dominated by CD8+ was found not only in lung tissue but also in paratracheal lymph nodes. CD8+ cytotoxic cells in the central airways are a source of IL-4 and IL-3 which cause mucus hypersecretion in patients with chronic bronchitis and also cause destroys of lung parenchyma by releasing perforin and granzymes. Smoking activity, which is routinely carried out with an increasing number of cigarettes over a long period time until old age, has an impact on the smoker's immune system (Ganesha, 2020; Asmalinda, 2021).

## CONCLUSION AND SUGGESTIONS

Based on the results of the analysis, there is a significant effect between age, number of cigarettes, and duration of smoking on the Vital Lung Capacity of active smokers, it can

be concluded that age, number of cigarettes consumed, and duration of smoking in active smokers affect decreasing the value of vital capacity It is recommended to conduct research further on the molecular level on cytokine parameters

## CONFLICT OF INTEREST STATEMENT

The authors declared that no potential conflict of interest concerning for the authorship and publication of this article

## REFERENCES

- Abdulrahman, WF. (2010). Effect of Smoking on peak expiratory flow rate in Tikrit University. *Tikrit Medical Journal*, 17(1); 11-18.
- Adeniyi, B. O., & Erhabor, G. E. (2011). The Peak Flow Meter and Its Use in Clinical Practice. *African Journal of Respiratory Medicine*, 5-8.
- Alsagaff, H & Mukty. (2008). *Fundamentals of Lung Disease*. Airlangga University Press. Jakarta.
- American Lung Association. (2017). *Lung Function Test*.<https://www.lung.org/lung-health-and-diseases/lung-procedures-and-tests/lung-function-tests.html>. 31 Juli 2022.
- Antao, Vinicius C & Pinheiro, Germania. (2015). Surveillance for Occupational Respiratory Diseases in Developing Countries. *Semin Respir Crit Care Med*, 36(3), 449-454. <https://www.ncbi.nlm.nih.gov/pubmed/26024351>. 31 Juli 2019.
- Ardam, Kiky Aunillah Y. (2015). Relationship of Dust Exposure and Exposure Time with Lung Physiological Disorders of Overhaul Power Plant Workers. *The Indonesian Journal of Occupational Safety and Health*. 4(2): 155-166. <https://ejournal.unair.ac.id/index.php/IJOSH/article/view/1746>. July 22, 2021.
- Asmalinda W, Sapada E, Agustin Y. (2021). Increasing the Saliva pH of Active Smokers Using Xylitol Gum. *Journal of Health*, 12(3), 427-434.
- Atmajaya, Aditya Surya & Ardyanto, Denny. (2007). Identification of Dust Levels in the Work Environment and Subjective Complaints of Respiratory Workers in the Finish Mill Section. *Journal of Environmental Health*. 3(2), 161-172.
- Bakhtiar, A and Amran, WS. (2016). Static Lung Physiology. *Journal of Respiration*, 2(3): 91-98.
- Barakati, RV, Lintong, F, Moningka, MEW. (2015). Comparison of Pulmonary Forced Vital Capacity in Student Smokers and Non-Smokers at the Faculty of Medicine, University of Sam Ratulangi Manado. *Journal of e-Biomedik (eBM)*, 3(1): 350-354.
- Buchari. (2007). *Occupational Diseases and Work-Related Diseases*. Medan : *Repository of the University of North Sumatra*.
- Budiman HM, Berawi KN, Bustomi EC, Medicine F, Lampung U, Physiology B, et al. (2018). Smoking Mechanism in Increasing Risk of Alzheimer's Disease. *Medical Journal*, 7(3), 234-240.
- Dwiputra, Edmundo C. (2019). *Factors Affecting Lung Function in Stone Breaking Workers in Bandar Lampung City*. Thesis at the Faculty of Medicine, University of Lampung.
- Citizen, RK. (2015). The Comparison of Lung Vital Capacity in Various Sports Athletes. *Majority Journal*, 4(2); 96

- European Respiratory Society. European Lung White Book : *Occupational Lung Disease Chapter 24*. (<https://www.erswhitebook.org/chapters/occupational-lung-diseases>), diakses tanggal 1 Agustus 2019.
- Fikriyah, S and Febrijanto, Y. (2012). Factors Influencing Smoking Behavior in Male Students in Boys' Dormitory. *STIKES Journal*, 5(1), 99-109.
- Ganesha IGH, Linawati NM, Satriyasa BK. (2020). Administration of Purple Cabbage (Brassica Oleraceae L.) Ethanol Extract Reduced Malondialdehyde Levels and the Number of Lung Tissue Macrophages of Rats Exposed to Cigarette Smoke. *Medical Science Journal*, 6(1), 1-9.
- Guyton AC, Hall JE. (2012). *Textbook of Medical Physiology*. 9th Edition. Jakarta, EGC.
- Gunther AC, Bolt D, Borzekowski DLG, Liebhart JL, Diliar JP. (2006). Presumed influence on peer norms: how mass media adversely affects adolescent smoking. *Journal of Communication* [Influence of Tobacco Marketing on Smoking Behavior, Monograph 19. The Role of the Media], 56 (1), 52 – 68. Available from: [http://cancercontrol.cancer.gov/ctcr/monographs/19/m19\\_complete.pdf](http://cancercontrol.cancer.gov/ctcr/monographs/19/m19_complete.pdf).
- Hapsari, R. (2009). The Effect of Gambling Dust Exposure on Lung Vital Capacity in Gambling Workers at UD Telaga Agung Blora, Central Java. *Thesis at the UNNES Faculty of Public Health*.
- Hanewinkel R, Isensee B, Sargent JD, Morgenstern M. (2010). Cigarette advertising and adolescent smoking. *Am Journal Prev Med* [serial on the internet]. 38(4), 359-366 [cited 2022 Sep 3]. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/20307803>.
- Jennifer, M.K., McIntosh, J.M., Doju, Y., & Baldomero, M.O. (2001). Nicotine- evoked transmitter release from synaptosomes: functional association of specific presynaptic acetylcholine receptors and voltage-gated calcium channels. *Journal of Neurochemistry*; 77, 1581-1589
- Juarfianti, Engka, J.N.A., and Supit, S. (2015). Lung Vital Capacity in Highland Residents of Rurukan Tomohon Village. *Journal of e-Biomedik (eBm)*, 3(1), 430-434.
- Khan GJ, Javed M, Ishaq M. (2010). Effect of smoking on salivary flow rate. *Gomal Journal of Medical Sciences*, 8(2), 22-27
- Larasati, A. (2016). Differences in the Degree of Acidity (pH) of Saliva in Cigarette and Non-Cretek Smokers. [Thesis]. *Jakarta: Faculty of Medicine and Health Sciences, Syarif Hidayatullah State Islamic University*.
- Ministry of Health of the Republic of Indonesia. (2019). Occupational Health Situation. InfoDATIN: Data and Information Center of the Indonesian Ministry of Health, accessed 12 September 2019.
- Nadia L. (2016). The Negative Influence of Smoking on Health and Awareness of Urban Communities. *Negative Effects of Smoking on Health and Awareness of Urban Society* [Internet]. 28(02), 77-104. Available from: <http://repository.ut.ac.id/id/eprint/7088>
- Pellegrino, R., Antonelli, A., & Mondino, M. (2010). Bronchodilator Testing: an Endless Story. *European Respiratory Journal*, 35(5), 952-954.
- Pinugroho, B. Setyo & Kusumawati, Yuli. (2017). Age Relationship, Length of Dust Exposure, Use of PPE, Smoking Habits with Impaired Lung Function of Mabel Workers in Kec. Kalijambe Sragen. *Journal of Health*, 10(2), 1979-7621.
- Pujiastuti, BE., Sulastris, Zulaicha. (2012). Analysis of Factors Affecting Lung Vital Capacity in Pregnant Women at RB Sri Lumintu Jajar Laweyan Surakarta. *Thesis at the Faculty of Health Sciences, Muhaamdiyah University, Surakarta*.
- Rahma F, Ardriaria M, Panunggal B. (2019). The Effect of Giving Purple Sweet Potatoes on Total Leukocyte Levels in Male Wistar Rats Exposed to Cigarette Smoke, 8, 2-9.
- Rachmat M, Thaha RM, Syafar M. (2013). Smoking Behavior of Junior High School Adolescents. *Public Health Natl Public Health Journal*, 7(11):502.
- Ramos V, Germain D, Ross N. (2009). Smoking behavior, knowledge and attitudes among the spanish speaking community in Victoria, Australia [manuscript on the internet]. Available from: [http://www.quit.org.au/downloads/NESB/Spanish\\_Smoking\\_Survey.-pdf](http://www.quit.org.au/downloads/NESB/Spanish_Smoking_Survey.-pdf)
- Sapada IE, Asmalinda W. (2020). The Vital Lung Capacity of Employees with Risk Factors for Potential Exposure to Ammonia Gas. *Journal of Health Science and Technology*, 8(1), 1-13
- Sharon, R.G., Natalie, M.M., Jian, C., Andrew, M.R., Marina, R.P., Jean-Pierre, C., McIntosh, J.M., Michael, J.M., & Allan, C.C. (2001). Nicotinic agonists stimulate acetylcholine release from the mouse interpeduncular nucleus: a function mediated by a different nAChR than dopamine release from the striatum. *Journal of Neurochemistry*; 77: 258-268.
- Sherwood, Lauralee. (2014). *Human Physiology from Cells to Systems* (Edition 8). Jakarta, EGC.
- Sinaga FA. (2016). Oxidative stress and antioxidant status on maximal physical activity. *Gener Campus Journal* [Internet], 9(2), 176-189. Available from: <https://jurnal.unimed.ac.id/2012/index.php/gk/article/view/7823>
- Siregar TA, Hamdan SR. (2020). Relationship between Internet Addiction and Smoking Behavior in Adolescents. *Psychostudy Journal of Psychology*, 9(3), 214-224.
- Smeltzer, S.C and Bare, B.G. (2002). *Textbook of Medical-Surgical Nursing* (Brunner & Suddarth) Edition 8 Vol. 1. Jakarta: E.G.C.
- Susana, D., Hartono, B., Fauzan, H. (2003). Determination of Nicotine levels in Cigarette Smoke. *Journal of Health Ecology*, 2(3), 272-274.
- Taufick ALK., Nugroho AD, Wulandari AA., Budiman DT, Wibisono AB, et al. (2023). The dynamics of smoker addiction. *Seurune. Unsyiah Journal of Psychology*, 6(1):1-28.
- Zulfrianiangrum, H. (2016). Relationship between Hemoglobin Levels and Vital Lung Capacity with Cardiorespiratory Endurance of Students Participating in Basketball Extracurricular at S.M.P. Negeri I Jetis, Bantul Regency. *Tesis. Yogyakarta: Department of Sports Education, Universitas Negeri Yogyakarta*.