mutiara medika
Jurnal Kedokteran dan Kesehatan
Vol 23 No 1 Page 34-41, January 2023

Mutiara Medika: Jurnal Kedokteran dan Kesehatan
http://journal.umy.ac.id/index.php/mm

# Epithelial Mucosal Changes and An Inflammation Process of the Cavum Nasi Due to Inhaled Sawdust

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**DATE OF ARTICLE:**
Received: 24 Oct 2022
Reviewed: 09 Dec 2022
Revised: 26 Jan 2023
Accepted: 27 Jan 2023

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**DOI:**
10.18196/mmjkk.v22i2.16623

**TYPE OF ARTICLE:**
Research

**Abstract:** Woodworkers are particularly susceptible to inhalation of sawdust exposure. Exposure to sawdust for a long time causes various health problems and causes the damage of the epithelium to a barrier. This study aims to identify whether inhaled sawdust can cause changes in the nasal mucosa and the occurrence of an inflammatory process. This study was a cross-sectional analytical study. Data were collected from July 2021 to January 2022 from the nasal cavity swab of 35 woodworkers who had worked for 1 to >5 years. The assessment was carried out by cytological examination to identify epithelial changes in the form of mild and moderate dysplasia, while the inflammation was found in the form of acute and chronic inflammation. Twenty-six (74.3%) samples were normal, 7 (20%) samples had mild dysplasia, and 2 (5.7%) samples had moderate dysplasia. Whereas 20 (57.1%) were found to have no inflammation, 8 (22.9%) were with acute inflammation, and 7 (20.0%) were found to have chronic inflammation). Fisher's exact test showed a relationship between the inflammatory process and epithelial changes (p 0.020). In conclusion, sawdust inhaled showed changes in the epithelial mucosa and inflammatory processes of the nasal cavity.

**Keywords:** cavum nasi; epithelial mucosal changes; inflammation process; wood sawdust

## INTRODUCTION

Data from the International Labor Organization (ILO) in 2018 reported that more than 1.8 million deaths due to labor occur yearly in Asia and the Pacific region. In fact, two-thirds of work-related deaths in the world occur in Asia. Globally, more than 2.78 million people die yearly from occupational diseases and accidents. In addition, 374 million work absences are reported due to non-fatal work injuries and illnesses yearly.[1–3] Referring to data from the Ministry of Manpower, work accident data in the first quarter of 2018 increased to 5,318 cases of work accidents with 87 dying workers. The causes of the mortality were related to work, such as cancer 34%, accidents 25%, diseases in the workplace, respiratory tract 21%, cardiovascular disease 15%, and several other factors 5%. Meanwhile, 52 workers were disabled, and another 1361 workers recovered after treatment. The International Labor Organization in 2013 reported that the risk of acute respiratory infections was high, especially among workers in the industrial sector.[1–4]

Epithelial changes that occur will cause an inflammatory reaction due to the activation of inflammatory mediators. The American Journal of Rhinology and Allergy reports that there is a strong influence between epithelial changes and the occurrence of inflammation. In addition, several studies have also reported epithelial changes due to inhalation of sawdust, in which the epithelial changes that occur have the potential for rhinitis, rhinosinusitis, nasal polyps, and even cancer.[5–12] A meta-analysis study discovered a relationship between inhalation of sawdust on time and duration of work as well as on the inflammatory process and the incidence of epithelial changes, which are a high risk of nasopharynx carcinomas.[13] The research measured the levels of inhaled sawdust at 3 different work locations using a personal dust sampler. The results of each location showed wood dust being inhaled and finding upper respiratory tract complaints indicating inflammation.[14]

A study involved 50 woodworkers with 25 as controls and reported a significant relationship between working time and ciliary changes in the respiratory epithelium, but the incidence of dysplasia was also not reported.[15] Different results involving 100 research subjects with homogeneity of working time in each day of 8 hours found epithelial changes in the respiratory tract due to fungal and bacterial infections contained in the wood. However, dysplasia was not reported, indicating that the type of wood and the time of taking the swab can affect the results of the relationship between exposure of time and changes in the nasal mucosal epithelium. [16] In this study, the selection of wood species was not carried out, and the swab was carried out before the research subjects started working.

Furthermore, this study showed no relationship between the inflammatory process and epithelial changes in the nasal mucosa. It does not align with several studies involving 50 research subjects with an average age of 64 years and an average length of service of 47 years who worked in apparently healthy sawmills and found an association between the inflammatory process and epithelial remodeling in the study.[17]

Research conducted on mucosal epithelial changes and an inflammation process of the cavum nasi due to inhaled sawdust was first performed at the Faculty of Medicine, Universitas Muhammadiyah Sumatera Utara. It is expected that the problems caused by inhaled sawdust can be a significant concern of the timber industry to the health and safety of workers. This research identifies whether inhaled sawdust cause changes in the nasal mucosa and the occurrence of an inflammatory process.

**MATERIAL AND METHOD**

This study used a cross-sectional research design. It is an analytical observational, categorical-categorical, unpaired study. This research did not give treatment to variables, only taking nasal swab smears and making cytology preparations to identify changes in the epithelium and the inflammatory process. This research was conducted at Pahlawan Street and Pahlawan district, Medan Perjuangan, Medan City, North Sumatera Province, from July 2021 to January 2022, with inclusion criteria including all wood craftsmen working in the wood cutting and refining section and agreeing to fill out informed consent. The exclusion criteria were workers with nasal anatomy abnormalities and all workers who had complaints of smell or were diagnosed with diseases related to the nasal cavity.

This research employed a descriptive-analytical cross-sectional design and was approved by the Health Research Ethics Committee, Faculty of Medicine, Universitas Muhammadiyah Sumatera Utara, No: 656/KEPK/FKUMSU/2021. This study employed 35 wood craftsmen in Pahlawan Street and Pahlawan District, Medan Perjuangan, Medan City.

Sampling was carried out directly on the mucosa of the nasal cavity using a cotton swab, and a smear was made and then stained with May Grunewald Giemsa staining. Epithelial changes were assessed by microscopic cytology in normal conditions (nasal mucosa is the pseudo-stratified ciliated epithelium and contains mucinous cells responsible for the continuous mucous secretion). In healthy conditions (healthy individuals without nasal diseases), only four cytotypes can be identified at NC: ciliated cells, mucinous cells, basal cells/striated cells, and sparse neutrophils. In this case, only sparse neutrophils can be found occasionally, with mild (abnormality confined to the basal layer), moderate (the lesion extends more than half of the epithelial layer), and severe conditions (all epithelial layers are involved) of dysplasia. Meanwhile, the picture of inflammation is assessed as normal (if the number of each type of inflammatory is <5 cells in 1 field of view), acute (predominantly polymorphonuclear cells), and chronic (predominantly mononuclear cells) inflammation—sawdust inhalation measured by Personal Dust Sampler. The tool was installed in section smoothing or cutting wood as high as flat nose worker, and the measurement of sawdust was carried out during working hours: Calibrating the tool first, then Installing the filter with the coarse part placed in front/top. The tool was then placed at the average height of the worker's nose for samples taken with tweezers and put in a desiccator for 24 hours. The filter was then weighed with an analytical balance, and the measurement values were 6.40 g/m$^3$ and 8.24 g/m$^3$.

The relationship between the working period and changes in the epithelium of the nasal mucosa and the inflammatory process, as well as the relationship between exposure time and changes in the epithelium of the nasal mucosa and the inflammatory process, were analyzed. The descriptive data were presented in the frequency table. Besides, categorical variables were correlated using Chi-square, with the alternative of Fisher's exact test. In this case, $p<0.05$ was considered statistically significant. Statistical analysis was then executed utilizing SPSS Statistics for Windows.

**RESULT**

The characteristics of 35 workers are presented in Table 1. Seven workers (20.0%) had epithelial change with mild dysplasia, 8 patients (22.9%) had acute inflammation, and 7 patients (20%) had chronic inflammation (Fig 1 and Fig 2). Based on these results, it can be concluded that inhalation of sawdust for a long time can cause epithelial changes and inflammation in the nasal mucosa. This situation can be exacerbated by age, length of work, and working time per day.

**Table 1. The Main Characteristics of Workers**

| Characteristics | | n | Percentage |
|---|---|---|---|
| Age | 19-25 years old | 10 | 28.6% |
| | 26-45 years old | 20 | 57.1% |
| | 46-65 years old | 5 | 14.3% |
| Working Time (hours/day) | <7 | 14 | 40.0% |
| | >=7 | 21 | 60.0% |
| Working Period (years) | <5 | 19 | 54.3% |
| | >=5 | 16 | 45.7% |
| Inhaled Sawdust | 6.40 g/m3 | 21 | 60.0% |
| | 8.24 g/m3 | 14 | 40.0% |
| Epithelial Changes | Normal | 26 | 74.3% |
| | Mild dysplasia | 7 | 20.0% |
| | Moderate dysplasia | 2 | 5.7% |
| Inflammatory Process | Normal | 20 | 57.1% |
| | Acute inflammation | 8 | 22.9% |
| | Chronic inflammation | 7 | 20.0% |

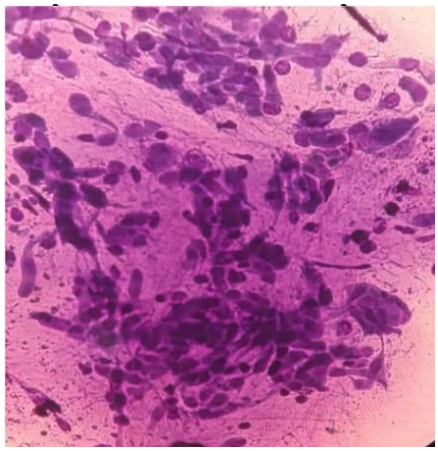

**Figure 1. Cytologic Features of Chronic Inflammation and Mild Dysplasia. Thickening and enlarged epithelium with an irregular shape accompanied by a scattering of inflammatory cells, Hyperkeratosis (May-Grunwald-Giemsa staining, 400x)**

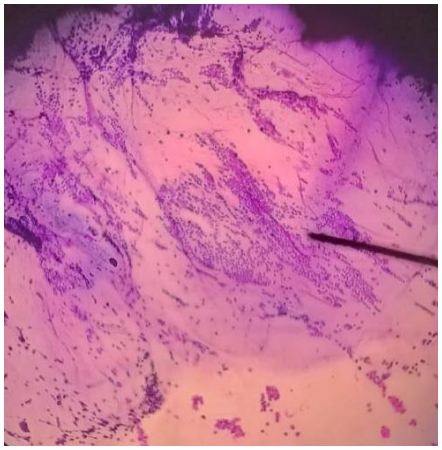

**Figure 2. Cytological Features of Acute Inflammation with Predominant Neutrophil Cells. Inflammatory cells were found to be dominated by neutrophils as a sign of acute inflammation (May-Grunwald-Giemsa staining, 100x)**

The relationship between inhaled sawdust and the working period can be seen in Table 2. It appears that workers who have worked <5 years (17; 81.0%) inhaled 6.40 g/m3 of sawdust, and workers who have worked > 5 years (12; 85.7%) inhaled 8.24 g/m3.

**Table 2. The Relationship between Inhaled Sawdust and Working Period**

| Inhaled Sawdust | Working Period | | | | p-value* |
|---|---|---|---|---|---|
| | < 5 years | | >= 5 years | | |
| | n | Percentage | n | Percentage | |
| **6.40 g/m3** | 17 | 81.0% | 4 | 19.0% | 0.000 |
| **8.24 g/m3** | 2 | 14.3% | 12 | 85.7% | |
| **Total** | 19 | 54.3% | 16 | 45.7% | |

*Fisher's exact test: p<0.05

The relationship between inhaled sawdust and epithelial changes can be seen in Table 3. Sixth cases (20.0%) of 8.24 g/m$^3$ of sawdust inhaled were epithelial changes in the form of mild dysplasia. Sawdust inhalation of 6.40 g/m$^3$ had a mild effect (2.6% mild dysplasia) and was not associated with moderate dysplasia.

**Table 3. The Relationship between Inhaled Sawdust and Epithelial Changes**

| Inhaled Sawdust | Epithelial Changes | | | | | | p-value* |
|---|---|---|---|---|---|---|---|
| | Normal | | Mild dysplasia | | Moderate dysplasia | | |
| | n | Percentage | n | Percentage | n | % | |
| 6.40 g/m3 | 20 | 57.16% | 1 | 2.6% | 0 | 0.00 | 0.02 |
| 8.24 g/m3 | 6 | 17.14% | 6 | 17.4% | 2 | 5.7 | |
| **Total** | 26 | 74.3% | 7 | 20.0% | 2 | 5.7 | |

*Fisher's exact test: p<0.05

The relationship between inhaled sawdust and the inflammatory process can be seen in Table 4. In seven cases (20.0%) of 8.24 g/m$^3$ of sawdust inhaled, an inflammatory process was found in the form of acute inflammation. Inhalation of sawdust in workers causes changes in the epithelium and the occurrence of an inflammatory process. In general, epithelial changes occur through two mechanisms: direct trauma (non-inflammatory) and activation of the immune system (inflammation). Inflammatory events are initiated by activating dendritic processes, macrophages, and other innate cells. As for dendritic and macrophage cells as APCs, APCs present the antigen earlier to MHC class II, then MHC class II activates CD4, and CD4 activates T helper cells to call other mediators. At the same time, macrophages which are also APCs will present antigens, in this case, wood powder, to MHC class I. MHC class I then activates cytotoxic T cells to attack the incoming antigen directly. If exposure to sawdust occurs for a long time, changes in the epithelium of the nasal mucosa will occur due to an increase in the extracellular matrix induced by cytokine activation due to continuous exposure. Cytokine activation can also cause inflammation, so it is not uncommon for wood craftsmen to find rhinitis, sinusitis, rhinosinusitis, nasal polyps, and others. The acute inflammatory process is initiated by cells already in the tissue, especially macrophages, dendritic cells, histiocytes, Kupffer, and mast cells. These cells have receptors on the surface or inside of cells called pattern recognition receptors *(PRR)*.

**Table 4. The Relationship between Inhaled Sawdust and Inflammatory Process**

| Inhaled Sawdust | Inflammatory Process | | | | | | p-value* |
|---|---|---|---|---|---|---|---|
| | Normal | | Acute Inflammation | | Chronic Inflammation | | |
| | n | Percentage | n | Percentage | n | Percentage | |
| 6.40 g/m3 | 19 | 54.2% | 1 | 2.9% | 1 | 2.9% | 0.000 |
| 8.24 g/m3 | 1 | 2.9% | 7 | 20.0% | 6 | 17.1% | |
| Total | 20 | 57.1% | 8 | 22.9% | 7 | 20% | |

*Fisher's exact test: p<0.05

The relationship between the inflammatory process and epithelial changes can be seen in Table 5. Four cases (57.1%) were found to have chronic inflammation with mild dysplasia. Inflammatory processes can occur due to the activation of the immune system. Activation of the immune system can lead to remodeling airway structures in blood vessels, epithelial and subepithelial layers, and smooth muscle. However, epithelial

changes can also occur without being preceded by an inflammatory state (epithelial changes through non-inflammatory mechanisms).

**Table 5. The Relationship between Inflammatory Process and Epithelial Changes**

| Inflammatory Process | Epithelial Change | | | | | | p-value* |
|---|---|---|---|---|---|---|---|
| | Normal | | Mild Dysplasia | | Severe Dysplasia | | |
| | n | % | n | % | n | % | |
| Normal | 19 | 95.0 | 1 | 5.0 | 0 | 0.0 | 0.002 |
| Acute Inflammation | 6 | 75.0 | 2 | 25 | 0 | 0.0 | |
| Chronic Inflammation | 1 | 14.3 | 4 | 57.1 | 2 | 28.6 | |
| **Total** | 26 | 74.3 | 7 | 20.0 | 2 | 5.7 | |

*Fisher's exact test: $p<0.05$

## DISCUSSION

The results in this study are also in line with a systematic review revealing that there was a relationship between inhalation of sawdust on time and duration of work as well as on the inflammatory process and the incidence of epithelial changes, which are a high risk of NPC (nasopharyngeal carcinomas).[13] Research measured the levels of inhaled sawdust at 3 different work locations using a personal dust sampler with the results of each location being inhaled and finding upper respiratory tract complaints indicating inflammation.[18]

The study measured sawdust using the EPAM-5000 tools installed at 3 points at the PT. Arumbai Kasembadan location. 2 installation points discovered the results of sawdust levels exceeding the NAV (value threshold).[16] Research at PT. Surya Sindoro Sumbing Wood Industry Wonosobo, with some research subjects of as many as 70 people, taken by random sampling who worked in the furniture component section (47 subjects) and woodworking area (37 subjects) had sawdust content exceeding the threshold value (>13,17–20).[14]

Research on the presence or absence of sawdust exposure to woodworkers was also carried out in 2011 at Company X using a Personal Dust Sampler, which was carried out in all work units, namely in the division, shaving, and cutting of wood. Furthermore, it was found that the average value of sawdust concentration in the shaving unit was 0.016 mg/m3. In the cleavage unit, it was 1.389 mg/m3. Moreover, the result in the cleavage unit, which was 1.765 mg/m3.[21] was also mentioned by Khumaidah in her 2009 research conducted by PT. Kota Jati Furnindo in Jepara with 44 research subjects. The results revealed 24 respondents (54.5%) were individual dust levels above NAV, and as many as 20 respondents (45.5%) with individual dust content below the NAV.[34]

This study is also in line with research conducted by Chadwiya et al. with the literature review method involving 100 articles with the results of a relationship between the incidence of inflammation in the respiratory tract and nasal cancer and exposure to sawdust.[7] However, this study is not in line with the research conducted by Borm et al. on woodworkers by including a homogeneous type of wood, namely the type of meranti wood, and involving 1000 woodworkers in 2002. The result revealed no relationship between the incidence of inflammation and epithelial changes and exposure to sawdust.[27]

Inhalation of sawdust in workers causes changes in the epithelium and the occurrence of an inflammatory process. In general, epithelial changes occur through two mechanisms: direct trauma (non-inflammatory) and activation of the immune system (inflammatory). Inflammation begins with the activation of dendritic cells, macrophages, and other innate cells. The dendritic and macrophage cells act as APCs. Then the APCs present the antigens to MHC class II, and MHC class II activates CD4. CD4 then activates T helper cells to summon other mediators, while macrophages and APCs will present antigens, in this case, and wood powder to MHC class I. MHC class I then activates cytotoxic T cells to attack directly on incoming antigens. If exposure to sawdust occurs for a long time, the nasal mucosal epithelium will change due to an increase in the extracellular matrix induced by cytokine activation due to continuous exposure. Cytokine activation can also cause inflammation, so it is not uncommon for woodworking workers to find rhinitis, sinusitis, rhinosinusitis, nasal polyps, and others. The acute inflammatory process begins with cells already present in the tissue, especially macrophages, dendritic cells, histiocytes, Kupffer, and mastocytes. These cells have receptors on the surface or inside cells called pattern recognition receptors (PRR).[4,19-30]

Neutrophils, eosinophils, and macrophages/monocytes have a certain time in the blood before migrating to the tissue, and each tissue has a certain time. Neutrophils can survive in the blood for 10 hours,

then migrate to tissues and survive in tissues for 1-2 days. Eosinophils can survive in the blood for 2 days, then migrate to tissues and can survive in tissues for 4-10 days. Meanwhile, macrophages can survive in the blood for 1 day, later migrate to the tissue as monocytes, and can survive in the tissue for 4-12 days to months.[20,21]

Based on table 5 with a p 0.002, there is a relationship between the inflammatory process and epithelial changes in the nasal mucosa. It aligns with several studies involving 50 research subjects with an average age of 64 years and an average length of service of 47 years working on a sawmill that looked healthy and found a relationship between the inflammatory process and epithelial remodeling.[31]

The research involved 70 research subjects who were woodworkers and experienced inflammation and mucosa. 3 people had normal conditions, while the rest had mild dysplasia, loss of cilia and goblet cell hyperplasia, and squamous cell metaplasia.[32] The inflammatory process can occur due to the activation of the immune system. Activation of the immune system can cause the remodeling of airway structures in blood vessels, epithelial and sub-epithelial layers, and smooth muscle. However, epithelial changes can also occur without being preceded by an inflammatory state (epithelial changes through non-inflammatory mechanisms).[33]

Inhaled sawdust will cause epithelial changes and inflammatory processes in the nasal mucosa that can occur within a working period of 6 years. Epithelial changes were also found in research subjects with a working period of 6 months and a 3-hour working time but the age of 57 years. It indicated that changes in the epithelium of the nasal mucosa were closely related to the working period and age of wood craftsman.[35]

The duration of action is related to the incidence of inflammation and epithelial changes induced by increased extracellular matrix due to chronic inflammation. Inflammatory processes and epithelial changes are unrelated to working time as they can occur quickly. Inflammation can cause changes in mucosa nasal epithelium due to the activation of inflammatory cytokines and increased extracellular matrix.[36]

## CONCLUSION

Inhaled sawdust was correlated with epithelial changes in the form of mild and moderate dysplasia, and inflammatory processes in the nasal mucosa, including acute and chronic inflammation.

## CONFLICT OF INTEREST

All co-authors have seen and agreed with the manuscript's contents, and there is no conflict of interest.

## REFERENCES

1. Ridasta BA. Penilaian Sistem Manajemen Keselamatan dan Kesehatan Kerja di Laboratorium Kimia. Higeia J Public Heal Res Dev. 2020;4(1):64-75. http://journal.unnes.ac.id/sju/index.php/higeia
2. Darwis AM, Naiem MF, Latief AWL, Maharja R, Noviponiharwani, Rahim MR, et al. Kejadian Cedera pada Karyawan Industri Percetakan di Kota Makassar Occurance of Injury of Worker in the Printing Industry in Makassar City. Jkmm. 2020;3(1). https://doi.org/10.30597/jkmm.v3i1.10281
3. Haworth N, Hughes S. The International Labour Organization. In Handbook of Institutional Approaches to International Business; 2012. https://doi.org/10.4337/9781849807692.00014
4. Yunus M, Raharjo W, Fitriangga A. Faktor-faktor yang berhubungan dengan kejadian infeksi saluran pernapasan akut (ISPA) pada pekerja PT . X. Kesehatan. 2020;6(1):21-30. https://doi.org/10.26418/jc.v6i1.43349
5. Mescher AL. Junqueira ' s Basic Histology Text and Atlas. Fourteenth Edition. Mc Graw Hill; 2016. p. 292–308
6. Eroschenko VP. DiFiore's atlas of histology with functional correlations. 11th ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2008.
7. Kasangana KK, Chadwiya MC, Masekameni D, Makonese T. Exposure to Wood Dust and Health Effects: A Review of Epidemiological Evidences from Developing Countries. 2017;(October). https://www.researchgate.net/publication/323826306_Exposure_to_Wood_Dust_and_Health_Effects_A_Review_of_Epidemiological_Evidences_from_Developing_Countries
8. Gallo S, Bandi F, Preti A, Facco C, Ottini G, Candia FD. et al. Exploring the role of nasal cytology in chronic rhinosinusitis. Acta Otorhinolaryngol Ital. 2020;40(5):368-376. https://doi.org/10.14639/0392-100X-N0711

9. Pendolino AL, Scarpa B, Ottaviano G. Relationship Between Nasal Cycle, Nasal Symptoms and Nasal Cytology. Am J Rhinol Allergy. 2019;33(6):644-649. https://doi.org/10.1177/1945892419858582
10. Heffler E, Landi M, Caruso C, Fichera S, Gani F, Guida G. et al. Nasal cytology: Methodology with application to clinical practice and research. Clin Exp Allergy. 2018;48(9):1092-1106. https://doi.org/10.1111/cea.13207
11. Herbert RA, Janardhan KS, Pandiri AR, Cesta MF, Miller RA. Nose, Larynx, and Trachea. Boorman's Pathology of the Rat; 2018. p. 391–435. https://doi.org/10.1016/B978-0-12-391448-4.00022-8
12. Watts AM, Cripps AW, West NP, Cox AJ. Modulation of allergic inflammation in the nasal mucosa of allergic rhinitis sufferers with topical pharmaceutical agents. Front Pharmacol. 2019;10(MAR). https://doi.org/10.3389/fphar.2019.00294
13. Beigzadeh Z, Pourhassan B, kalantary S, Golbabaei F. Occupational exposure to wood dust and risk of nasopharyngeal cancer: A systematic review and meta-analysis. Environ Res. 2019;171(December 2018):170-176. https://doi.org/10.1016/j.envres.2018.12.022
14. Suryani M, Setiani O, Nurjazuli N. Analisis Faktor Risiko Paparan Serbuk Kayu terhadap Gsngguan Fungsi Paru pada Pekerja Industri Pengolahan Kayu PT. Surya Sindoro Sumbing Wood Industry Wonosobo. J Kesehat Lingkung Indones. 2005;4.
15. Özler GS, Akoğlu E. Impairment of nasal mucociliary clearance time in wood industry workers. Eur Arch Otorhinolaryngol. 2020;277(2):493-496. https://doi.org/10.1007/s00405-019-05729-6
16. Neghab M, Jabari Z, Kargar Shouroki F. Functional disorders of the lung and symptoms of respiratory disease associated with occupational inhalation exposure to wood dust in Iran. Epidemiol Health. 2018;40:e2018031. https://doi.org/10.4178/epih.e2018031
17. Kherde PM, MishraNV, Chitta SS, Gahukar SD. Influence of sawdust on peak expiratory flow rate in sawmill workers of central India working in unprotected environment and its correlation with duration of exposure. National Journal of Physiology, Pharmacy and Pharmacology. 2017;1(Issue 1):68-73.
18. Indriyani D, Darundiati Y, Dewanti N. Analisis Risiko Kesehatan Lingkungan Pajanan Debu Kayu Pada Pekerja Di Industri Mebel Cv. Citra Jepara Kabupaten Semarang. J Kesehat Masy. 2017;5(5):571–80.
19. Sinaga JM. Hubungan antara Kadar Debu Kayu dengan Faal Paru Sebelum, Saat, Sesudah Bekerja pada Pekerja Industri Pengolahan Kayu Perusahaan X. Tesis. Universitas Sumatera Utara; 2019:2-3. URL https://repositori.usu.ac.id/handle/123456789/16950
20. Karnen Garna Baratawidjaja IR. Imunologi Dasar. Eleventh edition. Jakarta: Fakultas Kedokteran Universitas Indonesia; 2014. p. 50-75.
21. Grubbs H, Kahwaji CI. Physiology, Active Immunity [Internet]. StatPearls - NCBI Bookshelf. Published online 2021. Available from: https://www.ncbi.nlm.nih.gov/books/NBK513280/
22. Harkema JR, Carey SA, Wagner JG. The nose revisited: a brief review of the comparative structure, function, and toxicologic pathology of the nasal epithelium. Toxicol Pathol. 2006;34(3):252-269. https://doi.org/10.1080/01926230600713475
23. Nina Marliana RMW. Imunoserologi. 1st ed. Vol. 1. Kementerian Kesehatan Republik Indonesia; 2018.
24. Prasad ML, Perez-Ordonez B. Chapter 3 - Nonsquamous Lesions of the Nasal Cavity, Paranasal Sinuses, and Nasopharynx. In: Diagnostic Surgical Pathology of the Head and Neck (Second Edition). W.B. Saunders; 2009. P. 111-189. https://doi.org/10.1016/B978-1-4160-2589-4.00003-6
25. Genter MB, Doty RL. Toxic exposures and the senses of taste and smell. Handb Clin Neurol. 2019;164:389-408. https://doi.org/10.1016/B978-0-444-63855-7.00022-8
26. Nho R. Pathological effects of nano-sized particles on the respiratory system. Nanomedicine Nanotechnology, Biol Med. 2020;29:102242. https://doi.org/10.1016/j.nano.2020.102242
27. Löfstedt H, Hagström K, Bryngelsson IL, Holmström M, Rask-Andersen A. Respiratory symptoms and lung function in relation to wood dust and monoterpene exposure in the wood pellet industry. Ups J Med Sci. 2017;122(2):78-84. https://doi.org/10.1080/03009734.2017.1285836
28. Hough KP, Curtiss ML, Blain TJ, et al. Airway Remodeling in Asthma. Front Med (Lausanne). 2020;7:191. Published 2020 May 21. https://doi.org/10.3389/fmed.2020.00191
29. Akhouri S, House SA. Allergic Rhinitis [Internet]. StatPearls - NCBI Bookshelf. Published online 2021. Available from: https://www.ncbi.nlm.nih.gov/books/NBK538186/
30. Galet P, Nguyen DT, Russel A, Jankowski R, Vigouroux C, Rumeau C. Intestinal and non-intestinal nasal cavity adenocarcinoma: Impact of wood dust exposure. Eur Ann Otorhinolaryngol Head Neck Dis. 2018;135(6):383-387. https://doi.org/10.1016/j.anorl.2018.08.012
31. Kherde PM, Mishra N V, Chitta SS, Gahukar SD. Influence of sawdust on peak expiratory flow rate in sawmill workers of central India working in unprotected environment and its correlation with duration of exposure. Natl J Physiol Pharm Pharmacol. 2017;7(1):68-73. https://doi.org/10.5455/njppp.2016.6.0616525072016

32. Bussi M, Gervasio CF, Riontino E, Valente G, Ferrari L, Pira E. et al. Study of ethmoidal mucosa in a population at occupational high risk of sinonasal adenocarcinoma. Acta Otolaryngol. 2002;122(2):197-201. https://doi.org/10.1080/00016480252814225
33. Golden R. Identifying an indoor air exposure limit for formaldehyde considering both irritation and cancer hazards. Crit Rev Toxicol. 2011;41(8):672-721. https://doi.org/10.3109/10408444.2011.573467
34. Khumaidah, Khumaidah. Analisis Faktor-faktor yang berhubungan gangguan fungsi paru pada pekerja mebel PT Jati Furnindon Desa Suwawal Kecamatan Mlonggo Kabupaten Jepara. Thesis. Universitas Diponegoro; 2016. http://eprints.undip.ac.id/25008/
35. Ghelli F, Bellisario V, Squillacioti G, Grignani E, Garzaro G, Buglisi M. et al. Oxidative stress induction in woodworkers occupationaly exposed to wood dust and formaldehyde. J Occup Med Toxicol. 2021;16(1). https://doi.org/10.1186/s12995-021-00293-4
36. Lovato A, Staffieri C, Ottaviano G, Cappellesso R, Giacomelli L, Bartolucci GB et al. Woodworkers and the inflammatory effects of softwood/hardwood dust: evidence from nasal cytology. Eur Arch Oto-Rhino-Laryngology. 2016;273(10):3195-3200. https://doi.org/10.1007/s00405-016-3989-2