LITERATURE REVIEW

Work Lung Disease Due to Grain Dust

Ariani Permatasari[1], Reagen Irwan Kolibu[1,2]*

[1] Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Universitas Airlangga/Dr. Soetomo General Hospital, Surabaya, Indonesia.
[2] Wamena Jayawijaya Hospital, Papua, Indonesia.

ARTICLE INFO

Article history:
Received 03 July 2020
Received in revised form 07 September 2020
Accepted 13 September 2020
Available online 30 September 2020

Keywords:
Work lung diseases,
Grain dust,
Farmers,
Lung fungtion

ABSTRACT

Indonesia is one of the third largest grain producers globally; therefore, grain dust-related lung diseases will be more significant. Grain dust is easily exposed to humans, triggers lung damage, and decline the lung function. Grain dust exposure is associated with occupational lung diseases, for instance, acute reversible airflow obstruction, chronic obstructive pulmonary disease, grain fever, organic dust syndrome, and hypersensitivity pneumonitis. Smoking and the environment might aggravate grain dust-related respiratory problems. The average tolerated dose of grain dust is about less than 3 mg/m3. Avoiding exposure to grain dust is primer prevention.

INTRODUCTION

Indonesia is one of the thirs largest grain producers globally that harvested by around 25,9 million farmers (77% of total farmers), so grain is the Indonesian staple food. Grain mill dust exposure among farmers is one of the occupational lung diseases. International labor organizations (ILO) describe occupational disorder is diseases caused by work, work tools, materials, processes, and work environment. Furthermore, it divides based on agents, target organs, and malignancy.[1,2] It was estimated that around 2.78 million workers die every year due to work accidents and occupational diseases.[3,4]

Characteristics of grain dust

Grain dust is the product of various processes (seeding, drying, storing, and grinding) widely spread and easily exposed to humans.[5] It is biologically composed of plant material, fungi, parts of insects, bacteria, endotoxin, and soil.[6]

Microorganisms in grain dust

Fungi is the most predominant microorganism of grain dust; therefore, grain dust exposure will be followed by inhalation of fungi. The binding of fungus and grain dust is determined by factors abiotic (climate and geographic) and biotic (agricultural system).[6] A study assessed the association between exposure to grain dust and fungal diseases during harvest in Switzerland showed an increase in respiratory disorders caused by allergenic fungi such as the genus Epicocum and Cladosporium, and toxic genus Fusarium.[7] In line with the previous study, Ewa K et al. conducted a study of fungal concentration in grain dust and found an increase in the concentration of species Fusarium Poae, source of harmful mycotoxins, in grain dust samples.[8]

Dust mites in grain

More than 250 species of mites are known to cause human health problems; for instance, dust mites-associated allergy in a gricultural areas. Two groups of dust mites, house dust mites (Dermathophagoides

**Correspondence:* kolibu.reagen@gmail.com

Jurnal Respirasi, p-ISSN: 2407-0831; e-ISSN: 2621-8372.
Available at https://e-journal.unair.ac.id/JR. DOI: 10.20473/jr.v6-I.3.2020.79-84

Figure 1: Grain dust is one of the causes of lung disease due to work[2]

pteronyssinus) and storage mites (Acarus siro), bind to grain dust because of high humidity and multiplies faster to induce allergic reactions (house-dust allergy) and domestic mite dermatitis, respectively.[9] Both of them can cause airway inflammation through the activation of innate and adaptive immunity, lead to an asthma attack.[9,10]

Animal materials bound to grain dust

Fragments of animal bodies (cats, dogs, horses, cows, and sheep) mixed heterogeneously with grain dust often interfere with the respiratory tract. By focusing on the relationship between cat and dog exposure to asthma attacks, Lodge Cj et al. found that asthma attacks significantly increase in groups with and without asthma history.[11] Recently, various studies demonstrated grain dust bound to animal materials-related asthma attack in grain farmers.[12]

Pathogenesis mechanism of acute bronchoconstriction due to exposure to grain dust

The grain dust enters the human airway and triggers the defense processes of the human body.[12] As an extrinsic antigen, it will stimulate plasma cells and lymphoid tissue to produce IgE. IgE's binding to mast cells leads to asthma attacks characterized by the release of histamine and bradykinin, narrowing of the respiratory tract, thickening of the mucosa, and increase of mucous production.[13]

An elevation of serum IgE levels will induce the respiratory tract's obstruction to result in a decrease of the FEV1 value. An extensive mucosal obstruction in the segmental, subsegmental, and peripheral bronchial airways leads to lung hyperinflation; however, the pulmonary parenchyma's anatomy is relatively normal.[14]

Bronchoconstriction occurs in someone who has atopy allergen. After allergen inhalation, macrophages will capture and present its specific antigenic characteristics to the activated T lymphocytes (Th2). The allergen-specific Th2 will induce B lymphocytes to produce IgG and IgM to allergen-specific immunoglobulin (IgE).[13] IgE will be bound by a cell with surface receptors for IgE, eosinophil cells, and macrophages such as mastocytes in the tissue and basophils in the circulation.[13,14] This bond will cause ca ++ influx and a change in the cell, which decreases cAMP levels.[14]

A decrease of cAMP levels will cause histamine-mediated degranulation of cells to stimulate the respiratory tract's obstruction.[15] Physiologically, the respiratory tract's epithelial cells, from the upper airway (nose, nasopharynx, and larynx) to the lower airway (tracheobronchial branches), contribute to keep the airway open continuously and defend actively against infection in the early stage. Mechanical or chemical

JURNAL RESPIRASI, MAY 2020, VOL 06 (02); 79-84

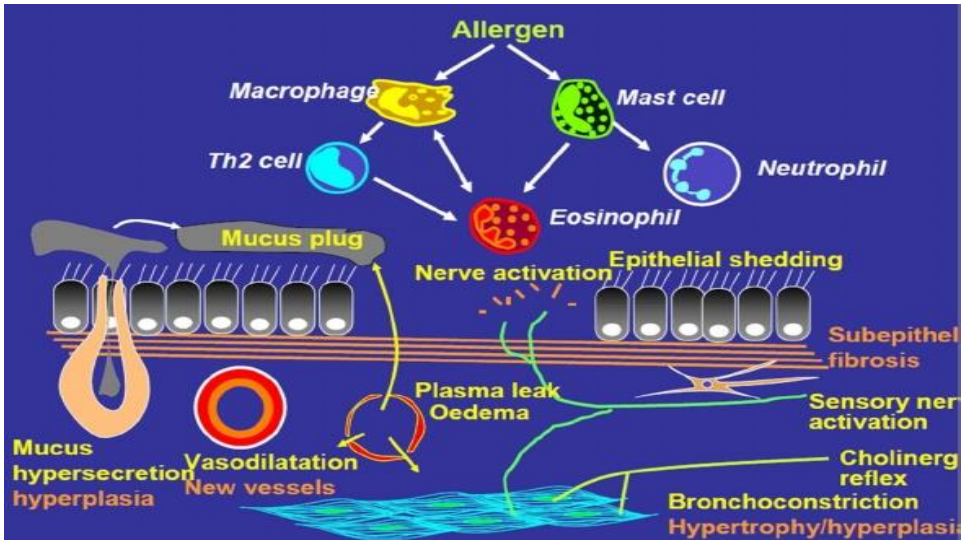

Figure 2: Mechanisms of respiratory tract obstruction.[14]

irritation will trigger receptors in the airways and result in bronchoconstriction, reducing the penetration of toxic substances or dust particles in the airways.[14,15]

Grain Dust-related lung diseases

a. Acute reversible obstruction

Individuals with the risk of allergies and asthma often experience asthma attacks as they visit dusty places and exposure to irritants, such as grain dust.[15] Many grain farmers with a history of asthma no longer work in the paddy fields. Exposure to grain dust can also cause asthma reactions in healthy individuals. Some researchers have shown evidence of specific bronchial reactions to inhalation with grain dust or grain dust extracts in grain workers.[15] Bronchial reactions usually occur immediately after exposure and are followed by spontaneous recovery.[16] Some workers' direct reactions are followed by slow reactions of several hours after exposure, usually lasting less than 24 hours. [15,16]

Manfeda et al. conducted a study in southern Manitoba, Canada, by measuring skin patch tests, total IgE, hematology, and spirometry in permanent residents between the ages of 20 and 65 in agricultural areas. Grain dust is more irritating, especially respiratory obstruction in the upper respiratory tract, than other agricultural crop products.[15] Furthermore, Max et al. conducted a study in 154 farmers at four grain milling factories in Cape Town, South Africa. The spirometry data were collected three times, start from Monday morning after two days without exposure to grain dust, before beginning work, and Thursday evening. There was an increase in the prevalence of acute airway obstruction, marked by a decrease in FEV1 of workers who performed spirometry examinations on Thursday.[16]

Gorge et al. conducted a hypothesis test by exposing endotoxin-sensitive (C3H / HeBFeJ) and endotoxin (C3H/ HeJ) grain dust extracts to rats eight weeks. The mice were evaluated before exposure to grain dust extract (GDE), four weeks after exposure, and eight weeks after exposure. The physiological response to exposure to grain dust was assessed with a body-weight chart to estimate airway resistance. C3H/ HeBFeJ mice showed significantly lower airway inflammation eight weeks after exposure to grain dust. This inflammatory response is entirely resolved after the recovery period.[17]

Symptoms of acute reversible airflow obstruction appear when in the work environment and manifest such as fever, fatigue, cough accompanied by sputum production. Symptoms usually improve when individuals are far from the work environment, and sometimes it takes several days for the symptoms to disappear.[15]

Several studies have found that grain farmers show a decrease in lung function during one work shift. The occurrence of acute reversible airflow obstruction depends on dust concentration (the higher the concentration of dust exposed to the respiratory tract causes more remarkable changes in lung function), environmental, and individual factors.[15,16]

b. Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is characterized by progressive obstruction of the respiratory tract when a patient breathes in harmful gases or particles like grain dust.[18,19] The most critical risk factor for developing COPD is smoking; however, occupational exposure, air pollution, and infection can also be associated with COPD.[18]

According to two experimental animal models, exposure to several kinds of dust, metals, and endotoxins might result in chronic bronchitis with air obstruction.[18] In human models, only alpha 1 anti-triptisin deficiency and an emphysema model confirmed this findings.[18] Centrilobular emphysema causes COPD related to dust eksposure.[19] Centrilobular emphysema due to occupationally hazardous materials might result from alveolar macrophages that can clear material from alveolars and bronchioles. Several experimental studies have suggested that lack of macrophage metalloelastase might play a protective role against emphysema development.[18,19]

Angeles et al. reported that subjects exposed to dust or smoke for more than 15 years were likely to have lower lung function than those exposed to less than 15 years with an average difference in FEV1 / FVC ratio was -1.7% (- 3,3–0,2). The FEV1 value is -0.6 mL (-2.1-0.8) in individuals exposed to less than 15 years and those who have never been exposed.[20]

c. Grain fever organic dust syndrome

Grain fever organic dust syndrome caused by inhaling dust containing fungi from damaged plant material is a common respiratory disease for farmers, especially those who work with seeds, straw, silage, and animals. Symptoms are fever and flu-like symptoms that occur four to 12 hours after exposure to contaminated dust.[21]

DoPico et al. explained the symptoms of grain fever characterized by heartburn, headaches, weakness, myalgia, fever, chills, chills, burning sensation in the throat and trachea, and tightness in the chest, shortness of breath, and coughing. Physiological changes in the lungs are characterized by obstruction of airflow without restrictive changes.[21]

d. Hypersensitivity pneumonitis

Hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis is a pulmonary disorder syndrome characterized by diffuse inflammation of the interstitial lung, terminal bronchioli, and alveoli. Inflammatory reactions are caused by prolonged and repeated exposure to inhaled antigens (grain dust, poultry dust, mold, straw dust, rat urine, (gerbils) tobacco and wheat dust), with particles generally measuring less than five μm.[22] This syndrome varies significantly in terms of the severity of symptoms, clinical features, prognosis, and depends on the nature of the causative agent, duration of exposure, host factors and characteristics of antigens.[23]

Many substances can be linked to conditions such as poultry dust, mold, straw dust, rat urine, (gerbils) tobacco, and wheat dust.[23] After antigen exposure, most individuals do not experience the other inflammatory response needed to diagnose hypersensitivity pneumonitis.4 The inflammatory response that arises is the formation of mild lymphocytic alveolitis even though it will not show symptoms. In hypersensitivity pneumonitis, regulatory T cells will suppress the Th1 and Th2 cells' immune response. The proliferation of T cells that cannot suppress the immune response can be associated with pneumonitis development. [22,23]

Symptoms of HP are acute, subacute, and chronic. Acute HP usually appears between four to six hours after exposure to grain dust. Symptoms are usually accompanied by fever, chills, malaise, dry cough, and tightness that gradually subside in the next 18 hours. Subacute symptoms are characterized by productive cough, dyspnea, fatigue, anorexia, weight loss, and pleurisy. At the chronic stage, the symptoms usually exist are productive cough, progressive tightness, fatigue, weight loss, and clubbed fingers.[24]

Host predisposing factors

The environment and smoking habits are the most predisposing factors for the emergence of respiratory disorders due to grain dust that lead to lung function decline. Kayaba et al. demonstrated an increase of IgE of the population living around grain farming areas, comparing those living in cities. They concluded that grain husk dust and smoke from burning straw had a significant role in exacerbating airway disease in agricultural areas.[25] Another

study by Manfreda revealed that smoking could increase an individual's susceptibility to grain dust exposure-related respiratory problems.[16]

Grain dust threshold value in humans

The occupational diseases due to grain dust exposure depend on the constitution, concentration of dust, and exposure duration. Grain dust exposure among workers in agricultural industries has been studied extensively in different countries. Enerson et al. found that exposure to grain dust at concentrations greater than 5 mg / m3 reduces FEV1 in grain farmers; approximately 10% of workers significantly decreased FEV1 during the workweek than before work. Indeed, there was increased respiratory distress in the third week.[26]

In 1978 the Ministry of Labor determined the value of 10 mg/m^3 as the maximum permissible grain dust concentration in the workplace.[27] Corey et al. showed changes in lung function and respiratory symptoms in grain workers when the dust concentration was less than 10 mg/m^3.[28] DoPico et al. demonstrated the effects of acute respiratory disturbances due to exposure to grain dust for an eight-hour shift on 248 workers.[22] Others researchers revealed a decrease in lung function at the concentration of 10 mg/m^3.

All of the studies and research have concluded that average grain dust can be tolerated in humans 3 mg/m3. However, the duration of exposure grain dust must also be assessed.

Prevention

The primary prevention related to lung disease due to grain dust is to avoid exposure to grain dust. Workers who work in an environment full of grain dust are always expected to wear protective equipment such as masks, always carry out regular health checks, wash their hands before eating and always change clothes exposed to grain dust.

SUMMARY

Grain dust exposure is associated with occupational lung diseases, for instance, acute reversible airflow obstruction, chronic obstructive pulmonary disease, grain fever, and pneumonia hypersensitivity. Smoking and the environment might aggravate grain dust-related respiratory problems. The primary prevention is to avoid exposure to allergens.

REFERENCES

1. Pusat pengendalian dan pencegahan penyakit, kecelakaan kerja dan kematian di antara pekerja muda. Amerika Serikat 1998-2007. 2010;449–55.
2. Badraningsih L, Enny Z. Kecelakaan dan penyakit akibat kerja [Internet]. 2015 [cited 2019 Apr 25]. Available from: http://www.staffnew. Uny.ac.id
3. Dewi S. Identifikasi dan rehabilitasi kerja. Jakarta: Departemen IKK Fakultas Kedokeran Universitas Airlangga; 2012. 1–6 p.
4. The Global Burden of Non-Malignant Respiratory Disease Due to Occupational Airborne Exposures. Changing behaviour: Electronic cigarettes. American Journal of Industrial Medicine. 2005;48:432–445.
5. Margaret R. Grain Dust and Lung Health Not Just a Nuisance Dust. Departement of medicine, epidemiology, biostatics and occupoational health McGill university health Quebec. Canadian respiratory journal. 2007;2:423–5.
6. Jhon S, Paul E, Stevie K. Human Health Concern from Grain Dusts and Molds During Harvest. Madison: Agricultural safety and Health, University of Wiscon sin-Madison; 2016. 1–6 p.
7. Lois P. The composition of airborne and grain dust fungal comonities is shaped at regional scale by plant genotypes and farming practices. University of Swiszerland; 2016. 1–25 p.
8. Sheng J, En-Chih L. Levels of Fungi and Mycotoxins In The Samples of Grain and Dust Collected From Five Various Cereal Crops In eastern Poland. 159-167: Departement oif Occupational BioHazards Polandia.Ann Agric Med; 2007.
9. Yu S-J, Liao E-C, Tsai J-J. House dust mite allergy: environment evaluation and disease prevention. Asia Pac Allergy. 2014;4(4):241.
10. Gary M, Barry M. Mites. Departement of Entomoly and Plant Patology. Auburn University USA. 2019: Elseiver; 533–596 p.
11. Ownby D, Johnson CC. Recent Understandings of Pet Allergies. F1000Res. 2016 Jan 27;5:108.
12. Lodge CJ, Allen KJ, Lowe AJ, Hill DJ, Hosking CS, Abramson MJ, et al. Perinatal Cat and Dog Exposure and the Risk of Asthma and Allergy in the Urban Environment: A Systematic Review of Longitudinal Studies. Clinical and Developmental Immunology. 2012;2012:1–10.
13. Fall T, Lundholm C, Örtqvist AK, Fall K, Fang F, Hedhammar Å, et al. Early Exposure to Dogs and Farm Animals and the Risk of Childhood Asthma. JAMA Pediatr. 2015 Nov 2;169(11):e153219.
14. Kudo M, Ishigatsubo Y, Aoki I. Pathology of asthma. Front Microbiol [Internet]. 2013 [cited 2020 Sep 14];4. Available from: http://journal.frontiersin.org/article/10.3389/fmicb.2013.00263/abstract

15. Iwasaki A, Foxman EF, Molony RD. Early local immune defences in the respiratory tract. Nat Rev Immunol. 2017 Jan;17(1):7–20.
16. Manfreda J, Holford-Strevens V, Cheang M, Warren CPW. Acute Symptoms Following Exposure to Grain Dust in Farming. Environmental Health Perpectives. 1986;66:78–80.
17. Bachmann M, Myers JE. Grain dust and respiratory health in South African milling workers. Occupational and Environmental Medicine. 1991 Oct 1;48(10):656–62.
18. George CLS, Jin H, Wohlford-Lenane CL, O'Neill ME, Phipps JC, O'Shaughnessy P, et al. Endotoxin responsiveness and subchronic grain dust-induced airway disease. American Journal of Physiology-Lung Cellular and Molecular Physiology. 2001 Feb 1;280(2):L203–13.
19. Cho Y, Lee J, Choi M, Choi W, Myong J-P, Kim H-R, et al. Work-related COPD after years of occupational exposure. Ann of Occup and Environ Med. 2015 Dec;27(1):6.
20. Boschetto P, Quintavalle S, Miotto D, Lo Cascio N, Zeni E, Mapp CE. Chronic obstructive pulmonary disease (COPD) and occupational exposures. J Occup Med Toxicol. 2006;1(1):11.
21. Bhatt S, Brown A, Hitchcock J, Schumann C, Wells M, Dransfield M. Determinants of successful completion of pulmonary rehabilitation in COPD. COPD. 2016 Feb;11:391–7.
23. Riario Sforza GG, Marinou A. Hypersensitivity pneumonitis: a complex lung disease. Clin Mol Allergy. 2017 Dec;15(1):6.
22. Dopico GA, Jacobs S, Flaherty D, Rankin J. Pulmonary Reaction to Durum Wheat: A Constituent of Grain Dust. Chest. 1982;81(1):55–61.
24. Vasakova M, Morell F, Walsh S, Leslie K, Raghu G. Hypersensitivity Pneumonitis: Perspectives in Diagnosis and Management. Am J Respir Crit Care Med. 2017 Sep 15;196(6):680–9.
25. Kayaba H, Meguro H, Muto H, Kamada Y, Adachi T, Yamada Y, et al. Activation of Eosinophils by Rice-Husk Dust Exposure: A Possible Mechanism for the Aggravation of Asthma during Rice Harvest. Tohoku J Exp Med. 2004;204(1):27–36.
26. Enarson D, Vedal S, Chan-Yeung M. Rapid decline in FEV1 in grain workers: Relation to level of dust exposure. Am Rev Respir Dis. 1985;132:814–817.
27. Guidelines for Environmental and Medical Surveillance Program in Grain Industry. 895th-7th–11th ed. Ottawa: Occupational Safety and Health Branch, Dept of Labour; 1978.
28. Corey P, Hutcheon M, Broder I, Mintz S. Grain elevator workers show work-related pulmonary function changes and dose-effect relationships with dust exposure. Occupational and Environmental Medicine. 1982 Nov 1;39(4):330–7.