International Journal of Research in Advent Technology, Vol.4, No.7, July 2016
E-ISSN: 2321-9637
Available online at www.ijrat.org

Effect of Endotoxin in Wood Dust to Increased Levels of TNF-α Nasal lavage on Furniture Workers

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Abstract-Endotoxin in wood dust is inhaled in the workplace can cause inflammatory reactions in the respiratory tract, characterized by increased levels of TNF-α in the surrounding tissue inflammation. This research aimed to analyze the differences in the levels of TNF-α in nasal lavage and the effect of endotoxins in wood dust to the increase level of TNF-α nasal lavage in furniture workers.. This research was analytic observational with cross sectional before-after study design. The sample size in the study was 12 samples. Measurement of levels of TNF-α performed before and after work using ELISA technique. The results showed that there were differences ($p < 0.05$) levels of TNF-α nasal lavage. Multiple linear regression analysis showed levels of endotoxin in the dust wood influence ($p < 0.05$) in the increased levels of TNF-α. It is concluded that exposure to endotoxin effect on TNF-α increase in nasal lavage. It is suggested to furniture workers to always wear personal protective equipment while working to reduce exposure to endotoxin and wood dust.

Keywords- wood dust, Endotoxin, TNF-α nasal lavage

1. INTRODUCTION

Exposure to chemical, biological, and physical work environment in the air may caused occupational diseases. Various disorders airway and lungs can occur, ranging from mild to cause disability in workers [1][2]. In the wood processing industry, wood dust and endotoxin are main pollutants for workers [3][4], including for workers furniture manufacturer.

Endotoxin is often associated with dust allergens. It is a complex lipopolysaccharide (LPS), the outer membrane of the cell wall of gram-negative bacteria, such as Escherichia coli cell can contain 2 million LPS. Gram-negative bacterial endotoxin release into the environment in small amounts when of their actively growing, and in large quantities after death or cell lysis [5][6].

Inhalation of wood dust and endotoxins will be deposited in the airways and lungs, through the mechanism of impaction, sedimentation and diffusion. Excessive particle deposition will cause in inflammation of the airways. Dust particles will stimulate alveolar macrophages to release a product that constitute an inflammatory response mediators such as cytokines and begin the process of fibroblast proliferation and collagen deposition [7][8].

Inflammation is an early sign of an immune response to infection or injury in the body, which is detected by an increase in the concentration of biomarkers in the body as a product of activation of inflammatory cells. The degree of inflammation and activation of inflammatory cells will affect the concentration of biomarkers [9][10]. Tumor Necrosis Factor-alpha (TNF-α) is known as a biomarker to assess respiratory inflammation[9][11].

TNF-α is main cytokine in acute inflammatory response to gram-negative bacteria, microbes, and other stimuli that play a role in nonspecific immunity. TNF-α is not usually detected in healthy people, but in conditions of inflammation and infection was found increased in serum and tissues [12][13]. When the inflammatory process occurs excessively or continuously, it may cause tissue injury or organ dysfunction and may contribute to the pathogenesis of diseases [14]. Some studies show that exposure of wood dust and endotoxin affect the increased levels of TNF-α, both in animals and in workers exposed [15][16]. If the levels of TNF-α increased excessively, it can have an impact on health, which is known to play a role in some respiratory diseases and lung disorders, such as chronic bronchitis, chronic obstructive pulmonary disease, asthma, acute lung injury (ALI) and acute respiratory distress syndrome (ARDS), allergic and non-allergic rhinitis [17][18].

The purpose of this study was to analyze the differences in the levels of TNF-α in nasal lavage and the effect of endotoxins in the dust wood to the increase of TNF-α in nasal lavage in furniture workers.

2. RESEARCH METHODE

This research was analytic observational research with cross sectional before-after study design, the time of observation and measurements were taken at the time before and after work (cross shift). The study population was furniture workers in Simogunung

street, Surabaya, Indonesia. The sample size this study was 12 samples.

The research variables are divided into: endotoxin levels as independent variables, levels TNF-α as dependent variables and confounding variables that include age, packyears, and tenure. confounding variables that include age, packyears, and tenure. Techniques and procedures for data collection consisted of: (1) Measurement of levels dust personal using a personal dust sampler, (2) sampling of nasal lavage [23], (3) measurement of endotoxin and TNF-α levels using enzyme-linked immunosorbent assay (ELISA) technique.

The measurement results will be analyzed using statistical tests. Differences in levels TNF-α are analyzed using paired t test if the normal distribution of data or Wilcoxon test if the data are not normally distributed. Multiple linear regression analysis was used to analyze the effect of levels endotoxin in wood dust to increased TNF-α.

3. RESULT

Respondent in this research is furniture workers by criteria: sex male, willing to become respondents, and did not have work history that may caused respiratory illness, and did not have a history related treatment of respiratory diseases.

Table 1. Characteristics of Respondents Research

Characteristics	n	Mean ± (SD)	min/max
Age (years)	12	34.75 ± (7.74)	21/49
Packyears	12	8.33 ± (7.46)	0/21
Work period (years)	12	14.75 ± (7.92)	2/27

In Table 1 it is known that the mean age of respondents was 34.75 years, with a minimum age of 21 years and maximum age 24 years. Packyears mean was 8.33 with packyears maximum was 29. The mean tenure of respondents was 14.75 years, with the quickest work period was 2 years and the longest work period was 27 years.

Measurement of personal wood dust using personal dust sampler, the filter is then used for endotoxin testing using ELISA techniques with methods Limulus Amebocyte Lysat (LAL). The results of the measurement of endotoxin and personal dust contained in Table 2.

Table 2. Measurement of personal wood dust and endotoxins

Parameter	Mean ± (SD)	min/max
Wood dust (mg/m^3)	1.57 ± 1.61	0.20/5.93
Endotoxin (ng/m^3)	2.18 ±1.13	0.92/5.40

Table 2 showed the mean levels personal wood dust was 1.57 mg/m^3, with the smallest levels 0.20 mg/m^3 and the greatest levels was 5.93 mg/m^3. While the mean measurement of endotoxin in the respondents was 2.18 ng/m^3, was smallest levels 0.92 ng/m^3 and the greatest levels was 5.40 ng/m^3.

TNF-α levels were measured from nasal lavage samples were analyzed using ELISA techniques. The following measurement results:

Table 3. Distribution Levels of TNF-α nasal lavage furniture workers

Levels of TNF-α nasal lavage	f	%
Increase	9	75,0
Decrease	2	16,7
Fix	1	8,3
Total	12	100
Mean ± (SD)	4.16 ± (7.33)	
Min	-1.69	
Max	25.17	

Table 4. Test results levels of TNF-α differences nasal lavage furniture workers (cross shift).

Levels of TNF-α (pg/ml)	Before	After
Min	3.78	6.15
Max	14.43	33.35
Mean ± (SD)	7.75 ± (2.79)	11.92 ± (7.62)
p-value	0.030	

p<0.05: significant

Table 3 showed that 75% workers have elevated levels of TNF-α after working with a mean increase of 4.16 pg/ml. The result of paired t-test in Table 4 showed there significant differences (p<0.05) between levels TNF-α before and after work (p = 0.030).

Multiple linear regression was conducted to determine the effect of endotoxin levels in wood dust and the characteristics of workers to increased TNF-α nasal lavage Furniture Workers.

Table 4. Multiple regression analysis effect of endotoxin levels in wood dust and respondent characteristic to increased TNF-α nasal lavage furniture workers

Variable	Increased TNF-α	
	B	p
Levels of personal wood dust	1.972	0.021*
Levels of endotoxin	-1.444	0.036*
Age	0.647	0.065
Packyears	-1.164	0.029*
Work period	0.177	0.742

* = Significant (p<0.05)

Table 4 shows that personal wood dust, endotoxin and packyears affect the increased TNF-α nasal lavage Furniture Workers.

4. DISCUSSION

The results of analyzes differences in levels TNF-α nasal lavage current workers before and after work showed that there significant difference (p = 0.030), where there is 75% workers have elevated levels of TNF-α nasal lavage with a mean increase of 4.16 pg / ml. Multiple linear regression analysis to determine the effect of endotoxin levels in the dust wood to increased TNF-α nasal lavage furniture workers. In Table 4 it is known that levels endotoxin (p = 0.036), wood dust (p = 0.021) and packyears (p = 0.029) significantly affects increased levels of TNF-α.

Inhalation of endotoxin, wood dust, and cigarette smoke causes inflammation in the airways, stimulates macrophages to release TNF-α and other proinflammatory cytokines [13][22][23]. TNF-α levels will determine the inflammatory effect caused. At low levels will cause acute inflammation and higher can cause pathologic abnormality of septic shock [13].

Wood dust and endotoxin are main pollutant source in of wood processing [3][4]. Some studies show that exposure to wood dust and endotoxin may increase levels of TNF-α. Animal studies showed that exposure wood dust or dust wood furniture increase levels of TNF-α in experimental animals [15][19], exposure endotoxin in experimental animals caused an increase TNF-α in the first hour and reaches a maximum at the sixth hour [24].

Wood dust is organic dust. Similar research on organic dust showed that exposure endotoxin in organic dust affect the increased serum levels of TNF-α and TNF-α levels nasal lavage rice milling operator [16][20]. In another study of 50 ug LPS exposure increases the concentration of TNF-α in sputum after 6 hours [25]. LPS aerosol inhalation (30 µg for 90 minutes) caused increased levels of TNF-α in the group of smokers and smokers [26]. Cigarette smoke may caused smokers macrophages to release cytokines such as TNF-α [27].

Endotoxin is main stimulator of TNF-α, but other agents (such as dust) including cytokines alone also could be a stimulator [12][13][28]. Endotoxin exposure may cause effects (local and systemic) biological and clinical in humans. The lungs are the main target endotoxin exposure, by inducing an inflammatory response in the lungs. Acute inflammatory reaction may cause effects such as fever, chills, dry cough, chest tightness, joint pain, influenza-like symptoms, which are all symptoms of organic dust toxic syndrome (ODTS). Chronic exposure to endotoxins can cause chronic bronchitis, asthma, COPD and reduced lung function due to chronic inflammation [29].

5. CONCLUSION

The results showed that there were significant difference (p <0.05) levels of TNF-α nasal lavage before and after working on furniture workers. Multiple linear regression analysis showed that endotoxin significant effect (p <0.05) increase in TNF-α in nasal lavage furniture workers.

To reduce exposure endotoxins in the workplace, furniture worker is advisable to always use personal protective equipment such as masks during work.

Acknowledgments

The author would like to thank furniture workers at Simo Gunung Raya street which has been willing to become respondents in this research.

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