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Chronic Respiratory Symptoms and Lung Function of Farmer and Breeder in UTU Village, Tabanan, Bali

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Abstract

AIM: The objective of the study was to determine chronic respiratory symptoms and lung function of farmers.

METHODS: The study was conducted in Utu Village, Tabanan, Bali with 84 subjects. This research was observational analytic cross sectional study.

RESULTS: Three dominant chronic respiratory symptoms in farmers were coughing (15.1%), dyspnea (13.1%), and phlegm (13.1%). Average values of forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC), and FEV1/FVC were 83.75 ± 34.42, respectively, 81.62 ± 34.30 and 104.90 ± 13.90, respectively. Cough was dominant experiencing by smokers ($p = 0.008$). Mean of FEV1% prediction and FVC% prediction value was lower in passive smoker group than no smoker group ($p = 0.005$ and $p = 0.03$).

CONCLUSION: Occupational exposure while farming and raising livestock can cause chronic respiratory symptom and lung function decline in farmers and can be influenced by smoking history and secondhand smoke exposure.

Introduction

Agriculture is occupational sub-sectors that dominate in Indonesia with approximately 13 million of farmers. In general, farmers in Indonesia combine farming and breeding activities everyday [1], [2]. Those behaviors can increase risk of environmental noxious substance exposure to farmers by increasing variability of noxious substance type and quantity. Risk of exposure from inorganic dust; organic dust; gas decomposition; and pesticides caused agriculture is one of the occupations with a high risk of occupational lung disease [3].

Soil processing and harvesting activities tend to inorganic dust exposure. Main source of agricultural inorganic dust exposure is silica in the soil. Exposure to silica can cause pneumoconiosis with clinical manifestations of pulmonary restriction and opacity on chest radiographs. Risk of agriculture organic dust exposure comes from rice dust grains containing plant products, insect fragments, endotoxins, and pollen. Exposure risk to organic substances also occurs while breeding activities, like animal waste, especially poultry containing intestinal mucosa excretion particle and immunoglobulin A and G which are highly antigenic [4].

There have been several studies conducted on lung function in farmers. The study conducted by Stoleski *et al*. in 2014 about chronic respiratory symptom and lung function in Northern Europe agriculture farmer found that respiratory symptoms prevalence were higher in agriculture workers than office workers ($p < 0.05$). That study also found small obstructive airway changes in agriculture workers were significantly affected by the duration of exposure and smoking history [5]. Another study conducted by Buralli *et al*. in 2018 in Brazil found that there was a strong relationship between pesticide exposures to pathologic process in respiratory tract. Respiratory symptoms such as cough were found 40% in farmers during the harvest season, nasal allergies were found in 30.7% of farmers, and chest pain was found in 24% of farmers [6]. A study conducted by Lamprecht *et al*. in 2007 in Austria using 228 farmer subjects found that agriculture was a risk factor of persistent airway obstruction. That study also found 30.2% of subjects experienced persistent airway obstruction with relative risk of chronic obstructive pulmonary disease in farmers was 1.5 times higher than non-farmers (95% confidence interval [CI] 1.1–2.0) [7]. Chronic respiratory function limitation in farmers was not only caused by respiratory tract obstruction but can also by loss compliance pulmonary compliance due to fibrosis as occurs in hypersensitivity pneumonitis (HP).

In an article written by Oshimo *et al*. in 2012 about HP was said that 0.5–19% of farmers experienced HP [8].

All facts above show that high risk of occupational lung disease in farmers and breeder due to noxious environmental substance exposure. Therefore, research on the description of lung function in areas with high numbers of farmers such as Bali Province is urgency. This research was conducted in Utu Village, Tabanan Regency, Bali with the aim to see a picture of chronic airway symptoms and lung function in farmers associated with duration of exposure, smoking history, and age. Utu Village is one of the villages in Bali which is famous for its agricultural products. It is hoped that the results of this study can help provide an overview of the planning of prevention and treatment programs for occupational lung diseases in farmers, especially in Bali.

Methods

This study was conducted in Utu Village, Tabanan Regency, Bali on 17 and 18 November 2018. This study was cross-sectional analytic.

Study sample and sample selection

The number of subjects was 84. Subjects were selected by total sampling by taking all of Utu Village farmers. All research subjects carried out farming and breeding activities such as (1) soil processing with animal or machine, (2) planting rice, (3) irrigation, (4) using pesticides, (5) harvesting rice, (6) digging the soil, (7) feeding livestock, and (8) clean the cattle pen.

All research subjects were given information about the study and gave written informed consent on the form that was provided by researcher.

Data collection

Data were collected in 2 days. First day subjects filled questionnaire and assisted by researchers. The 2nd day, spirometry examination was carried out on research subjects.

Questionnaire

Questionnaire consist of four parts: (1) Subject's identity (gender and age); (2) Nutritional status (height, weight, and body mass index); (3) Smoking status (active smokers, passive smokers, and no smoking); (4) Job description (duration of work); and (5) Chronic respiratory symptom over past 12 months (cough, phlegm, dyspnea, chest pain,and wheezing).

Smoking status is divided into active smokers, passive smokers, and not smoking. Active smoker is subjects who smoke at least once every day, including on the day of examination except religious fasting day. Passive smoker is subjects who until the time of the examination get exposure to secondhand smoke from the environment every day. No smoking is a subject that does not fit the criteria of active and passive smokers.

Information regarding chronic respiratory symptom over the past 12 months was evaluated by a modified European Community Respiratory Health Survey questionnaire.

Spirometry

Spirometry was used to measure forced vital capacity (FVC), forced expiratory volume in 1 s (FEV 1), and the FEV1/FVC ratio. Measurements were done 3 times and best value was used as a result. Spirometry results were presented as a percentage of predicted value.

Data analysis

Data were analyzed using statistical software. Normality testing was performed for numerical data by Kolmogorov–Smirnov. Demographic characteristics, nutritional status, smoking status, job descriptions, and respiratory symptom for 12 months, and spirometry results data were analyzed descriptively. We analyzed distribution difference of respiratory symptom based on age, smoking status, and duration of exposure with Chi-square or Fisher's exact. Spirometry results were abnormal distribution data because of that average comparative spirometry test results based on age, smoking status, and duration of exposure were analyzed with Mann–Whitney.

Results

The number of study subjects was 84 farmers. Demographic characteristics of subjects can be seen in Table 1. Respiratory symptom such as coughing, phlegm, dyspnea wheezing, and chest pain was evaluated. Spirometry examination was performed to evaluate values of FVC, FEV1, and FEV1/FVC (Table 1).

Comparison of respiratory symptom distribution data based on age can be seen in Table 2. Subjects age was grouped into <65 years and ≥ 65 years. Respiratory symptom percentage was higher ≥65 year's group except for dyspnea, but there were no significant differences in both of groups.

Comparison of respiratory symptom distribution based on working duration can be seen in Table 2.

Working duration data were abnormally distributed with a median of 29.5. Therefore, working duration data were grouped into two groups, they were working duration <29.5 years and working duration >29.5 years. Respiratory symptom percentage was higher in the >29.5 years of work, but there were no significant differences in the two groups.

Comparison of respiratory symptom distribution based on smoking history can be seen in Table 2. Respiratory symptom percentage was higher in the smokers group, except for chest pain. A significant difference was seen in coughing and phlegm production ($p < 0.05$).

Table 3 shows comparison of spirometry results based on age group, working duration, smoking history, and secondhand smoke exposure. The number of subjects for spirometry results analysis based on environmental secondhand smoke exposure was 60 due to the selection of subjects who smoked.

Table 1: Demography characteristic of subjects

Demography characteristic	Frequency/Mean
n	84
Gender (%)	
Male	40.5
Female	59.5
Age (years)	58.29 ± 12.09
BMI (kg/m^2)	23.51 ± 3.61
Length of work (years)	25.95 ± 15.34
Safety equipment	
Mask	8.3
No safety equipment	91.7
Smoking status (%)	
Active smoker	28.6
Passive smoker	36.9
Without history of smoke exposure	34.5
Respiratory symptom (%)	
Dyspnea	13.1
Wheezing	7.1
Coughing	15.5
Phlegm	13.1
Chest pain	2.4
Spirometry parameter	
FEV 1(%)	83.75 ± 34.24
FVC (%)	81.63 ± 34.30
FEV 1/FVC	104.90 ± 13.90

BMI: Body mass index, FEV1: Forced expiratory volume in 1 s, FVC: Forced vital capacity.

Discussion

Table 3 shows comparison of spirometry results based on age group, working duration, smoking history,

Table 2: Respiratory symptom based on age, length of work, smoking history, and secondhand smoke exposure history

Respiratory symptom based on age				
Respiratory symptom	≥65 year (n=23) (%)	<65 year (n=61) (%)	Prevalence difference (Δ) (%)	p
Dyspnea	8.69	14.75	6.06	0.719
Wheezing	8.70	6.56	2.14	0.663
Cough	17.39	14.75	2.64	0.745
Phlegm	17.39	11.48	5.91	0.483
Chest pain	4.35	1.64	2.71	0.475
Respiratory symptom based on length of work				
	>29.5 year (n=42)	<29.5 year (n=42)	Prevalence difference (Δ)	p
Dyspnea	14.29	11.90	2.39	0.746
Wheezing	9.52	4.76	4.76	0.676
Cough	21.43	9.52	11.91	0.131
Phlegm	16.67	9.52	7.15	0.332
Chest pain	4.76	0	4.76	0.494
Respiratory symptom based on smoking history				
	Smoker (n=24)	No smoker (n=60)	Prevalence difference (Δ)	p
Dyspnea	25%	8.33	16.67	0.069
Wheezing	16.67	3.33	13.34	0.053
Cough	33.33	8.33	25	0.008
Phlegm	29.17	6.67	22.5	0.011
Chest pain	0	3.33	3.33	1.00
Respiratory symptom based on secondhand smoke exposure history				
	Passive smoker (n=31)	No passive smoker (n=29)	Prevalence difference (Δ)	p
Dyspnea	16.13	0	16.13	0.053
Wheezing	6.45	0	6.45	0.492
Cough	9.68	6.90	2.78	1.00
Phlegm	6.45	6.90	0.45	1.00
Chest pain	6.45	0	6.45	0.492

Table 3: Spirometry result based on age, length of work, smoking history, and secondhand smoke exposure history

Spirometry result based on age				
Spirometry result	<65 year (n=61)	≥65 year (n=23)	Mean difference (Δ)	p
FVC (%)	79.47 ± 25.02	87.72 ± 52.73	8.25	0.911
FEV 1 (%)	83.54 ± 29	84.32 ± 46.80	0.78	0.684
FEV1/FVC (%)	105.01 ± 13.97	104.60 ± 14.05	0.41	0.919
Spirometry result based on length of work				
	< 29.5 year (n=42)	>29.5 year (n=42)	Mean difference (Δ)	p
FVC (%)	79.57 ± 29.90	83.69 ± 38.47	4.12	0.671
FEV 1 (%)	84.43 ± 35.43	83.07 ± 33.42	1.36	0.929
FEV1/FVC (%)	106.39 ± 13.36	103.42 ± 14.44	2.97	0.447
Spirometry result based on smoking history				
	Smoker (n=24)	No smoker (n=60)	Mean difference (Δ)	p
FVC (%)	72.50 ± 20.36	85.28 ± 38.03	12.78	0.671
FEV 1 (%)	74.04 ± 25.29	87.63 ± 36.69	13.59	0.929
FEV1/FVC (%)	101.24 ± 14.71	106.37 ± 13.41	5.13	0.447
Spirometry result based on secondhand smoke exposure history				
	Passive smoker (n=31)	No passive smoker (n=29)	Mean difference (Δ)	p
FVC (%)	72.80 ± 21.35	98.62 ± 46.94	25.82	0.005
FEV 1 (%)	78.06 ± 23.90	97.86 ± 44.89	19.08	0.03
FEV1/FVC (%)	105.82 ± 13.69	106.97 ± 13.34	1.15	0.589

FEV1: Forced expiratory volume in 1 s, FVC: Forced vital capacity.

and environmental secondhand smoke exposure. The number of subjects for spirometry results analysis based on environmental secondhand smoke exposure history was 60 due to the selection of subjects who smoked.

Research conducted by Stoleski *et al*. in 2015 on agricultural farmers in Republic of Macedonia found similar results. That study used subjects with an average age characteristic similar to this study, which is 50 year old. Result of that study was three dominant respiratory symptom experienced by farmers during the past 12 months were cough, sputum, and dyspnea with prevalence of 20%, 10.7%, and 12%, respectively. The average value of FVC and FEV1 in the study also obtained more than 80% that were 84.2 ± 8.6% and 82.7 ± 8.3%, respectively [5]. Other studies conducted by Khane and Arora in 2015 on farmers with an average age of 32 years found that dominant respiratory symptoms on farmer were dyspnea (33.3%), coughing (20%), wheezing (15.2%), and phlegm (8%). In that study, the prevalence of respiratory symptom was higher compared to our study and wheezing was one of the four dominant respiratory symptoms experienced by farmer [9]. Research conducted by Buralli *et al*. in 2018 on Brazil population who work as farmer found that coughing (40%) and chest tightness (24%) as two dominant respiratory symptoms experienced by farmer in the past 12 months. The average prediction value of FVC, FEV1, and FEV1/FVC during the planting season was obtained above 80%. In that study, it was found that the prevalence of respiratory symptom was higher in farmers compared to our study and symptoms such as coughing and chest tightness were the dominant symptoms [6].

Above facts show that each study has variations in the types of dominant respiratory symptoms by farmers' subjects. These variations can be caused by environmental factors and subject characteristics [10]. One of the environmental factors is the type of exposure [11]. Farmers and breeder have high risk of getting noxious exposure from variety sources when farming and raising livestock. Each working environment has different combination of noxious substances sources and noxious particle components [12]. Noxious substance can be in the form of organic dust, inorganic dust, pesticides, and decomposition gas. Each of those substances can cause different pathological reactions to the respiratory tract that make variation of clinical symptoms in exposed individuals [13].

Agricultural and livestock organic dust can come from rice dust grains containing plant products, insect fragments, endotoxins, pollen, and animal waste [14]. Organic dust associated with the occurrence of Hypersensitivity Pneumonitis (HP) in farmers or often referred to as Farmer's Lung [3], [15]. HP is a pulmonary disease that has a spectrum involving interstitial, alveolar, and bronchial tissue in response to inhalation of organic dust especially low molecular dust weight that can cause permanent tissue damage [16]. HP occurs due to an immunological response to allergens that cause lung inflammation. Those reactions generally occur in susceptible individuals. Chronic exposure can lead to expansion and activation of fibroblasts and accumulation of extracellular matrix which then cause pulmonary fibrosis. There are three types of HP that is determined by the pathological mechanism that occurs, they are acute, sub-acute, and chronic HP. Acute HP is mediated by an immune complex and characterized by an increase in immunoglobulin G titers and neutrophils. Sub-acute and chronic PH phases are characterized by an increase in T cell migration and proliferation and a decrease in T cell apoptosis which subsequently leads to T-lymphocytic alveolitis [17].

Clinical features that can occur in HP patients depend on HP type. In acute HP symptoms that can occur for 1 week after exposure include fever, coughing, dyspnea, and weakness. On lung examination can be found additional sounds in the form of rhonchi or crackle. Sub-acute PH is difficult to identify and provides a clinical picture of productive cough, dyspnea, and weakness. Chronic PH provides clinical features of progressive shortness of breath with episodes of wheezing, cough with phlegm, recurrent mild fever, anorexia, weight loss, and respiratory failure. Chest radiograph examination can show interstitial fibrosis and spirometry examination can show restriction [18], [19]. Clinical variations in each type of HP can be one cause of symptom variation in various studies and lung function examination results. Individuals who have different types of HP will provide different clinical variations and individuals with sub-acute PH can show normal lung function examination results.

Inorganic dust from farming and raising livestock activities can come from silica exposure when cultivating agricultural soil [20]. Continuous exposure to silica dust can cause silicosis. Silicosis is a form of pneumoconiosis due to silica deposited in the bronchoalveolar duct and induces fibrosis. Silica particles that are inhaled and enter the alveoli will be phagocyted by alveoli macrophages. The toxic nature of silica particles to macrophages causes apoptosis of macrophages which then activates reactive oxygen species, reactive nitrogen species, and other free radical factors. That substance then triggers the release of pro-inflammatory factors such as tumor necrosis factor-α, interleukin (IL)-1β, IL-6, proteases, and arachidonic acid metabolites that activate the migration of leukocytes to the lung tissue. Macrophages also produce fibrogenic factors such as PDGF, transforming growth factor (TGF)-α, TGF-β, epidermal growth factor, and insulin-like growth factor-2 which recruit type-II pneumocytes and fibroblasts to produce fibronectin and collagen as the basic ingredients of scar tissue in the lung parenchyma [21]. Pulmonary fibrosis due to silica particles will provide a clinical picture of dyspnea on effort and progressive dyspnea. Chronic silicosis sometimes also does not provide a clear clinical picture, so it is often diagnosed as an aging process. The discovery of silicotic nodules in radiological examination is a typical sign of silicosis. In this study, there were 13.1% of subjects who had symptom of dyspnea without wheezing during the past 12 months, but further testing is still needed to ensure definitive diagnosis [22]. The clinical features of silicosis are sometimes non-specific and can also contribute to the possibility of finding varied clinical features in the subjects of several studies.

Exposure to pesticides is also one of the respiratory diseases risk factor in farmer. Various studies had shown relationship of respiratory symptoms and pathological results on spirometry examination with a history of pesticide used [6], [23]. Effects of exposure on the respiratory system are related to local and systemic toxic reactions. Most pesticides have small molecular weight and thus require hapten mechanisms to cause pathological reactions. Exposure to pesticides can cause local and systemic toxicity reactions that induce exacerbation of atopic symptoms in patients, exacerbations of asthma, and contact dermatitis. Respiratory disorders by pesticides can be through the mechanism of Type I hypersensitivity, Type IV hypersensitivity, and oxidative stress. Organophosphate insecticide toxicity occurs through decrease in function of the Muscarinic 2 receptor on the nerve, which causes inhibition loss of the acetylcholinesterase enzyme resulting in respiratory hyperreactivity [24], [25]. In this study, there are no data on the history of farmers' exposure to pesticides, but because pesticides are still commonly used in Indonesia, risk of farmers' exposure to pesticides is still high. The results showed 7.1% of subjects experienced wheezing complaints during the past 12 months. This can be caused by pesticide exposure, beside exposure to other noxious substances. In this case, lung function can be normal if examined in non-exacerbated subject.

Risk of exposure to organic substances also occurs in breeding activities, especially chickens breeder or other animals such as cows and pigs. The intended organic matter exposure is animal waste either due to breeding or fertilizing activity [13]. Several studies found individual who was exposed to animal waste for long time had higher risk for experiencing clinical symptom such as asthma or chronic bronchitis. Research conducted by Viegas *et al*. in 2013 found prevalence of asthma symptom in breeder reaching 45.2% [26]. Other respiratory symptoms also had high prevalence such as coughing (29.8%) and wheezing (19.1%). Another study conducted by Hamid *et al*. in 2018 found similar thing. That study found that respiratory symptoms had a high prevalence in farmers, wheezing was experienced by 18.3%, chest tightness (16.9%), dyspnea (14.1%), and coughing (15.5%). Based on spirometry examination, 65% of farmers experienced restrictive lung disease and 21% had obstructive abnormalities [27]. Research conducted by Radon *et al*. in 2007 found that populations who live around 500 m from livestock with more than 12 animals had higher risk of experiencing wheezing (odds ratio 2.45; 95% CI 1.22–4.90) compared to livestock with <12 animal [28].

Respiratory symptom due to exposure by animal waste can be caused by several mechanisms. First mechanism is animal waste containing intestinal mucous excretion particles and immunoglobulin A and G that has antigenic properties and can trigger allergic reactions [29], [30]. Second mechanism is airway inflammation due to exposure by hydrogen sulfide (H2S) and ammonia. H2S at low level is an airway irritant and at high levels is an asphyxiant chemical. Mechanism of H2S toxicity is related to the inhibition of oxidative phosphorylation which causes decreased of cellular energy. Pulmonary edema can occur at 250 ppm H2S level and confusion and death can occur at 500 ppm H2S level [31]. Ammonia is an irritant gas of the respiratory membrane and mucosa. Tissue damage occurs due to ammonia – tissue reaction that produces ammonium hydroxide which has strong alkaline properties. Tissue damage due to strong bases is caused by liquefaction necrosis and the ability of deep tissue penetration. In the respiratory tract, ammonia can damage the cilia and mucosa which are innate immunity (innate immunity) of the respiratory tract. Development of pathological conditions such as mucous hypersecretion, edema, and reactive smooth muscle contractions can cause significant airway obstruction. Respiratory symptoms experienced by the subjects in this study can also be caused by exposure to animal waste. Normal spirometry results can be affected by the time of examination and the history of exposure. High frequency of exposure and high concentrations of noxious particle can increase the risk of respiratory symptom and decreased lung function [32], [33].

Individual characteristics are another factor that can influence variation of respiratory symptom and pulmonary function in several studies. The individual characteristics are age, sex, smoking history, environmental secondhand smoke exposure, and genetic [34]. Comparative analysis result was significant differences in respiratory complaints between the smoking and nonsmoking groups. The percentage of farmers who cough and increase phlegm production was higher in smoker group than not smoker. There were significant differences in FEV1 and FVC values in the group with a history of environmental secondhand smoke exposure and without a history of exposure, where the exposed group had lower FEV1 and FVC values. In a study conducted by Stoleski *et al*. in 2015 also obtained similar results. That study found chronic respiratory symptoms associated with smoking habits ($p < 0.01$) [13]. Based on spirometry results, obstructive changes in the small respiratory tract were significantly related to smoking habits ($p < 0.01$) but were not influenced by a history of environmental second hand smoke exposure. The article written by Omland in 2002 supports this study results. The article explained that smoking is one of the risk factors for farmers to experience chronic bronchitis and asthma. Decrease of FEV1 and FVC also occurs more rapidly in farmers who smoke than those who did not smoke. Chronic inflammatory processes in the respiratory tract caused by smoking or exposure to secondhand smoke can aggravate airway pathological damage due to exposure to agricultural and livestock noxious substances [35]. This process is one of the causes of higher prevalence of respiratory symptom and lower lung function in subjects who smoke and had history of exposure to secondhand smoke than those who did not [36].

Another factor that can influence the emergence of respiratory symptom and lung function in farmers and breeder is the duration of exposure. In a study conducted by Stoleski *et al*. in 2015 found that farmers who had worked ≥16 years had a higher prevalence of respiratory tract symptom like phlegm production than those who worked for <16 years. In that study, there were no significant differences in the results of FEV1, FEV, and FEV1/FVC in groups with secondhand smoke exposure ≥16 years and exposure of <16 years [13]. In a study conducted by Soumagne *et al*. in 2017, exposure duration was the only variable that influenced emphysema incidence in Farmer's Lung ($p < 0.05$). Farmer's lung with emphysema occurred in farmers with an average length of work of 33.3 ± 11.1 months. In this study, no significant differences were found in respiratory symptoms and spirometry results based on exposure duration. That can occur due to differences in the frequency of exposure per day and substance exposed [37]. Content of the noxious substance also contributes to the exposure effect which mainly related to the toxicity of the noxious substance component [13], [11]. This study also did not have information about mobility and subjects history of exposure with other noxious sources such as the use of firewood during cooking, exposure to traffic, and oil combustion. Future studies are needed to look at the effects of agricultural and animal noxious exposure with a larger sample size, adequate observation time, complete information about exposure sources and content in working area, as well as information on the individual exposure history to another noxious source.

Conclusion

Based on the results, it can be concluded that dominant respiratory symptoms experienced by farmers were cough, sputum, and dyspnea with prevalence 15.5%, 13.1%, and 13.1%, respectively. The mean value of the subject's FVC%, FEV1%, and FEV1/FVC% predicted was more than 80%. Comparative analysis showed that percentage of farmers who coughing and phlegm production was higher in the smoker group than nonsmoker group. There were significant differences in FEV1% and FVC% predicted values in the group with a history of secondhand smoke exposure and without exposure history, where the exposed group had lower FEV1% and FVC% predicted values.

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