THE ASSOCIATION BETWEEN PARTICULATE MATTER 10 AND SEVERITY OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE, CHIANG RAI PROVINCE, IN 2011-2012

PEEYAWAN PRAMUANSUP

MASTER OF SCIENCE
IN
PUBLIC HEALTH

SCHOOL OF HEALTH SCIENCE
MAE FAH LUANG UNIVERSITY

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ACKNOWLEDGEMENTS

I would like to express my sincere thanks to all who have helped me in accomplishing this task successfully. First of all, I would like to thank my advisor and co-advisor, Dr. Phairoj Jantaramanee, Dr. Tawatchai Apidechkul, Dr. Nittaya Pasukphun and, Dr. Monchai Wongkarnka for their kindness help and encouragement throughout the course of this research.

I would like to thank all COPD clinic staff and patients who participated in the study sites from Mae Sai, Mae Chan, Phaya Meng Rai, Somdejprayanasungwon and Chiang Saen hospitals for their cooperation.

Finally, I am most grateful to my family and relatives for their encouragement, and to my friends for supporting me on all needs for this study.

Peeyawan Pramuansup

Thesis Title	The association between particulate matter 10 and severity of chronic obstructive pulmonary disease, Chiang Rai Province, in 2011-2012
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ABSTRACT

This research determines the association between particulate matter with ≤ 10 μ/m^3(PM_{10}) and the severity of Chronic Obstructive Pulmonary Disease (COPD). A retrospective cohort study design was conducted and collected data by using completed and tested questionnaires. The data were collected from the medical records among the COPD cases from 5 local hospitals in Chiang Rai Province, Thailand: Mae Chan, Mae Sai, Prayameng Rai, Sodejprayanasungwon, and Chiang San Hospitals. The PM_{10} was calculated by the setting of PM_{10} monitoring system in Chiang Rai Province. The severity was measured by the Modified Medical Research Council Dyspnea Score (mMRC) method. Logistic regression model was applied to test the association between independent variables and dependent variable. The determinations of statistical significance levels were 0.10 and 0.05 in the univariate and multivariate models respectively.

The different PM_{10} exposing level was found in the group for exposure to PM_{10} ($p>0.05$) in dry season. Multiple logistic regression analysis was found that those people who exposed PM_{10} would increasing to be resulted in severity of COPD with 6.03 times when compare to un-exposed period, OR= 6.03 (95% CI: 4.13-8.63). Increasing of PM_{10} level is directly associated with the severity of the COPD. Increasing of people awareness to avoid and protect from the PM_{10} are necessary for increasing quality of life among the COPD.

Keywords: COPD/ PM_{10}/ Severity of COPD

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ABBREVIATIONS AND SYMBOLS

AAT	Alpha-1 Antitrypsin
AECOPD	Admitted to hospital with acute exacerbation of COPD
API	Airborne Particle Index
AQI	Air Quality Index
Cd	Cadmium
CO	Carbon monoxide
CO_2	Carbon dioxide
COPD	Chronic Obstructive Pulmonary Disease
CRD_s	Chronic Respiratory Disease
Cu	Copper
DM	Diabetes Mellitus
ED	Emergency Department
Fe	Iron
GOLD	Gold Initiative for Obstructive Lung Disease
HIV	Human Immunodeficiency Virus
HT	Hypertension
IOC	Item objective Congruence Index
mMRC	The modified Medical Research Council Dyspnea Scale
Mn	Manganese
NH_3	Ammonia
Ni	Nickel
NO_2	Nitrogen dioxide
NO_x	Nitrogen oxide
O_3	Ozone
OPD	Out-Patient Department

ABBREVIATIONS AND SYMBOLS (continued)

PM	Particulate Matter
$PM_{2.5}$	Particulate Matter less than 2.5 micrometer size
PM_{10}	Particulate Matter less than 10 micrometer size
SO_x	Sulfur oxide
$SVOC_s$	Semi Volatile Organic Compound
TSP	Total Suspended Particulate
V	Vanadium
VOC_s	Volatile Organic Compound
WHO	World Health Organization
Zn	Zinc

CHAPTER 1

INTRODUCTION

1.1 Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a common disease characterized by airflow obstruction. In population-based studies, COPD affects 5.00% to 19.00% of adult population above 40 years old (Rabe et al., 2007). Most of the information available on COPD prevalence, morbidity and mortality comes from high-income countries. Even in those countries, accurate epidemiologic data on COPD is difficult and expensive to collect. It is known that almost 90.00% of COPD deaths occurred in low- and middle-income countries (World Health Organization, 2011a). According to the latest WHO estimates (2011a), 64 million people have COPD and 3 million people died of COPD. WHO predicts that COPD will become the third leading cause of death worldwide by 2030 (World Health Organization, 2011b). The economic costs for the management of COPD are staggering and do not take into account the burden a reduced quality of life.

The total number of COPD cases in the 12 countries in Asia–Pacific were 56.6 million with an overall prevalence rate of 6.30%. The COPD prevalence rates was ranged from 3.50% in Hong Kong and Singapore, 6.70% in Vietnam and 5.00% in Thailand (Tan & Ng, 2008). The proportion of deaths from COPD was found at its lowest at 4.00% in New Zealand to more than 40.00% in Sri Lanka and Thailand (Lopez, Mather, Ezzati, Jamison& Murray, 2006). COPD is predominantly caused by smoking, however, other factors, particularly occupational exposures, such as air

pollution are recognized as significant contributors to the development and progression of COPD.

The burden of COPD in Asia is currently greater than in developed countries with the number of cases and deaths increasing. This is mainly linked to the epidemic of indoor (such as cigarette smoke or propane) and outdoor air pollution exposure.

The outdoor air pollution, smoke from forest fires has a potential to affect millions of people. Therefore, it has been identified as a major public health problem.

More than 30 years parts of large forests in Thailand have been cleared for agriculture. In addition, forest fires (with a natural cause vs. man-made) in Thailand have occurred during the dry season (December-May), in the deciduous forest. In 2010 in Chiang Rai Province, there were 25 recorded forest fires (Forest Fire Control Division, 2010). Chiang Rai Province is located in the northern-most part of Thailand, with its northern and western border being Mynmar. Northern Thailand and parts of neighbouring Myanmar suffer from a smoke-filled haze caused by the burning of the forest and agricultural waste. During forest fires, large areas of land can be covered in layers of smoke, hundreds of kilometers away from the actual fires.

The very significant issue of forest fire smoke risk is a serious problem in Southeast Asia (Forest Fire Control Division, 2010). The smoke that is produced by forest fires, which lingers in the atmosphere. The smoke produced by forest fires, when it enters the respiratory tract into the body causes the anti-bodies to deteriorate and causes various respiratory diseases including: COPD, asthma, cough, pneumonia, cataracts, and tuberculosis and is likely a cause of of other dangerous diseases,such as, heart disease, problems with blood transfusions and cancer (Brauer, Goh, Schwela, &Goldammer, 1999). The part of air pollution increases most significantly as a result of forest fire smoke is Particulate Matter (PM), which can affect respiratory health and cause COPD.

Of all the air pollutants, inhalable particulate matter is less than 10 micrometer size (PM_{10}) and shows an association with adverse respiratory health effects. Acute exacerbations of COPD has been associated with short-term exposure to air pollution (Atkinson et al., 2001). One study (Abbey, Hwang, Burchette, Vancuren& Mills, 1995) found a positive association between PM_{10} and development of symptoms of

COPD productive cough and increased severity of airway obstructive diseases and asthma, stronger than who were exposed occupationally to dust and fumes.

Even a small increase in risk could cause large-scale respiratory health problems. In Chiang Rai Province,the number of cases of COPD has increased every year since 2006 to 2008, 368.32, 405.45 and 423.50, respectively per 100,000 population (Chiang Rai Provincial Public Health Office, 2008). In 2008, Chiang Rai and other districts in the northern provinces were declared disaster zones because of the choking smoke, which plagued Chiang Mai, Mae Hong Son and Lamphun. Chiang Rai Province has the highest PM_{10} concentrations in Thailand, the measurements of air pollution in the northern provinces found dust particles smaller than 10 microns at 100.4 micrograms per cubic meter, which is close to what is considered the dangerous level. The public health standard was a maximum 120 micrograms per cubicmeter (Pollution Control Department, 2012).

Many people were admitted to the hospital for care and treatment due to the air pollution problem, affects not only the environment, but also affects human health especially the elderly and children. COPD is a major cause of morbidity in the elderly. The second most common cause of disability in the elderly is respiratory disease (Hunt,Enquest& Bowen, 1976). Reports from Mae Chan hospital (located in Chiang Rai Province) between September 2010 to March 2011 found COPD was the fifth-most common cause of out-patient treatment and the most-common cause of being admitted to the hospital, costing the hospital 1,705,092 baht. COPD was found to be the fifth leading cause of death in Mae Chan Hospital. (Mae Chan Hospital, 2011).

The purpose of this study is focused on the affect of air particulate pollution due to PM_{10} and the severity of COPD in Chiang Rai, Thailand.

1.2 Hypotheses

Particulate Matter 10 (PM_{10}) is dominant cause of severity of Chronic Obstructive Pulmonary Disease (COPD).

1.3 Objective

To determine the association between Particulate Matter 10 (PM_{10}) and the severity of Chronic Obstructive Pulmonary Disease (COPD).

1.4 Variables

1.4.1 Independentvariables

1.4.1.1 Demographic and socio-economic factors: sex, age, education, occupation, and income.

1.4.1.2 Risk-behavior factors: history of smoking, indoor and outdoor activities.

1.4.1.3 Environmental factors: $PM_{10,}$ biomass burning, waste burning, factories which produce air pollution, and forest fires

1.4.2 Dependentvariable

Severity of COPD

1.5 OperationDefinitions

Particulate Matter$_{10}$ is defined as: Particulate matter which is less than 10 microns in diameter and forms when gasses emitted from forest fires are expelled into the atmosphere.

COPD is defined as: Diagnosed by physician as Chronic Obstructive Pulmonary Disease and found in their respective medical records.

The severity of COPD refers to the level of impairment of lung function and level of symptoms that is determined by the following questions:

A. How many times you go to see the doctor for COPD?

B. How many times have you been admitted to the hospital for COPD?

C. What type of medications were used to treat COPD?

D. When did complications of COPD begin to develop?

mMRC refers to the modified Medical Research Council dyspnea scale that measure the severity of COPD .This score counts by taking history of dyspnea.

Biomass burningrefers to the burning of living and dead vegetation. It includes the human-initiated burning of vegetation for land clearing and land-use change as well as natural, lightning-induced fires.

Waste burningrefers to the burning of household garbage and/or domestic waste.

Transportation refers to the movement of people, animals and goods from one location to another, including air, rail, road and water.

Pollution from Factories refers to air pollution given off by factories

Forest fire refers to a natural or man-made fire which destroys a forested area, and can be a great danger to people who live in the forest area as well as to wildlife. Forest fires are generally started by lightning, but also by human negligence or season.

Education refers to each subject's level of education at the date of interview.

Occupation refers to the occupation or career of the subjects.

Income refers to the annual income of the subjects. An annual family income was measured in Thai "Baht" currency based on the subjects' income earned in the previous year.

History of smoking refers to the history of smoking currently or an ex-smoker. History of smoking was measured by asking the subject: "Have you ever smoked?" Dichotomous of "Yes" or "No" would result.

Indoor air pollutionrefers to the quality of air in the subjects' household.

Outdoor air pollution refers to the quality of air outdoors.

1.6 Keywords

PM_{10}, COPD

1.7 Scope of the Study

This study is a retrospective cohort study to determine the risk factors of Chronic Obstructive Pulmonary Disease for the population who live in Chiang Rai, Thailand. The association between PM_{10} and severity of COPD is determined. The variables of this study were divided into 3 groups. Firstly, demographic and socio-economic factors: sex, age, education, occupation and income. Secondly, risk behaviors such as: history of smoking, indoor and outdoor air pollution, and the last group is environmental factors such as PM_{10} from biomass burning, waste burning, transportation, factories, and forest fires. All of these factors were evaluated by questionnaire and the subjects were selected using lists of medical records of those who were diagnosed with COPD by clinics and hospitals in Chiang Rai, Thailand.

CHAPTER 2

LITERATURE REVIEW

The air pollution that increases as a result of forest fires smoke is particulate matter 10 (PM_{10}) therefore the forest fires are major problem in Northern Thailand, especially during the dry season. One major cause of forest firesis agricultural burning before and after harvest, resulting in smokes and air pollutions and affect the people health who living in that area.

2.1 Forest Fires Caused and Description

2.1.1 Occurrence

Forest fires in Thailand annually occur during the dry season from December to May with their peak in February-March. Fires, Mostly classified as surface fires, mainly take place in Mixed Deciduous Forest, Dry Dipterocarp Forest, and Forest Plantations, and to some extent in Dry Evergreen Forest, Hill Evergreen Forest or event in some parts of the Tropical Rain Forest. In certain extremely dry areas, double burning in one season is common. These surface fires consume surface litter, other loose debris on the forest floor and small vegetation (Forest Fire Control Division, 2010).

2.1.2 Duration

Forest fires in Thailand annually occur during the dry season from December to May with their peak in February-March. (Forest Fire Control Division, 2010)

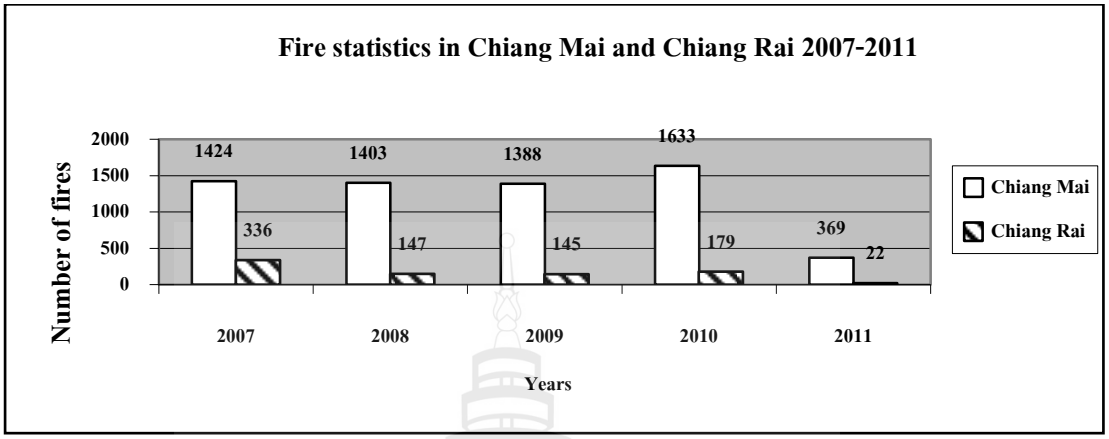


Figure 2.1 Fire Statistics in Chiang Mai and Chiang Rai 2007-2011

2.2 Forest Fires Smoke

Smokes produced by burning of biomass play an important role in atmospheric chemistry and in the climate of the earth, as well as affecting local and regional air pollution. (Radke, Hegg, Hobbs & Penner, 1995)

2.2.1 AQI (Air Quality Index)

The AQI is an index for reporting daily air quality. It tells how clean or polluted the air is, and what associated health effects might be a concern for people. The AQI focuses on health effects. People may experience within a few hours or days after breathing polluted air. The AQI is calculated for five major air pollutants regulated by the Clean Air Act: ground-level ozone, particle pollution (also known as particulate matter), carbon monoxide, sulfur dioxide, and nitrogen dioxide. (U.S. Environmental Protection Agency, 2011)

Table 2.1 Classifications and Cautionary Statements in Thailand (Pollution Control Department, 2012)

AQI	Classifications	Color	Cautionary statements
0-50	Good	Blue	Air quality is considered satisfactory, and air pollution poses little or no risk.
51-100	Moderate	Green	Air quality is considered satisfactory, and air pollution poses little or no risk.
101-200	Unhealthy for sensitive group	Yellow	People with lung disease, older adults and children are at a greater risk from exposure to ozone, whereas persons with heart and lung disease, older adults and children are at greater risk from the presence of particles in the air.
201-300	Very unhealthy	Orange	A health alert signifying that everyone may experience more serious health effects.
>300	Hazardous	Red	A health warnings of emergency conditions. The entire population is more likely to be affected.

2.2.2 Calculated the AQI from Pollutant Concentration Data

Calculated the AQI by using the pollutant concentration data the following equation (U.S. Environmental Protection Agency, 2011).

$$I_p = \frac{I_{Hi} - I_{Lo}}{BP_{Hi} - BP_{Lo}} (C_p - BP_{LO}) + I_{LO}$$

Where

Ip	=	the index for pollutant p
Cp	=	the rounded concentration of pollutant p
BPHi	=	the breakpoint that is greater than or equal to Cp
BPLo	=	the breakpoint that is less than or equal to Cp
BPHi	=	the breakpoint that is greater than or equal to Cp
IHi	=	the AQI value corresponding to BPHi
ILo	=	the AQI value corresponding to BPLo

Table 2.2 Breakpoint for the AQI

AQI	**PM_{10} (24 hr.) $\mu g/m^3$**
50	40
100	120
200	350
300	420
400	500
500	600

Source Pollution Control Department (2012)

2.2.3 Forest Fires Composition

Forest fires can be considered as physical and chemical stressor, as long as it consists of components with physical and chemical properties are as following: (Dokas, Statheropolos & Karma, 2007)

2.2.3.1 VOCs (Volatile organic compound): VOCs inclued methane and other hydrocarbon, which can be aliphatic (alkanes,alkenes,alkynes), such as ethane, heptane, decane, propene, 1-nonene, 1-undecene, acetylene or aromatic hydrocarbons.

2.2.3.2 SVOCs (Semi volatile organic compound): SVOCs canbe polyaromatic hydrocarbons such as benzene.

2.2.3.3 Particulate matter: Particulate matter from forest fires (also calledwildfires, vegetation fires, or biomass fires) can be coarse(>PM_{10}) or fine, depending on their size.The major amount of particles produced in forest fire,(over 90.00%), are 10μm or less in diameter (Dokas, Statheropoulos & Karma, 2007).

2.2.3.4 Permanent gas include CO_2, CO, NO_x, SO_x and NH_3: SO_x areusually produced in small quantities because generally forest fuel sulfur content is low. However, high amount of sulfur-based compounds can be produced when sulfur-rich vegetation or soil are burned.

Table 2.3 IndicativeForestFiresSmoke Compounds and How They are Transferred Through the Environment

Compound	Example	Notes
Permanent gases	CO, CO_2	Transported over distances.
	NO_2	Reactive.Concentrations decrease with distrance from bushfire.
Particles	PM_{10}	Coarse particle are ususlly deposited. They contain mostly soil and ash.
	$PM_{2.5}$	Fine particles are transported over long distances.

Source Brauer et al.(1999)

2.3 Definition of Particulate Matter

Particulate matter (PM) is the term used for a mixture of solid particles and liquid droplets suspended in the air. These particles originate from a variety of sources, such as power plants, industrial processes, and diesel trucks, and they are formed in the atmosphere by transformation of gaseous emissions. Their chemical and physical compositions depend on location, time of year, and weather. Particulate matter is composed of both coarse and fine particles. (Marian, 2000)

2.3.1 Coarse Particles (PM_{10})

Coarse particles (PM_{10}) have an aerodynamic diameter between 2.5 μm and 10 μm. They are formed by mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays, andsuspension of dust. PM_{10} is composed of aluminosilicate and other oxides of crustal elements, andmajor sources including fugitive dust from roads, industry, agriculture, construction and demolition,and fly ash from fossil fuel combustion. The lifetime of PM_{10} is from minutes to hours, and its traveldistance varies from < 1 km to 10 km (Marian, 2000).PM_{10} fraction that causes significant health impacts is dominated by particles from three sources. First is fine particles from industrial, Second is aerosol, mostly ammonium sulphate and ammonium nitrate and third is wind-blown soil and street dust. (Harrison, Deacon, Jones & Appleby, 1997)

2.3.2 Fine Particles ($PM_{2.5}$)

Fine particles have an aerodynamic diameter less than 2.5 μm ($PM_{2.5}$). They differ from PM_{10} in origin and chemistry. These particles are formed from gas and condensation of high-temperature vapors during combustion, and they are composed of various combinations of sulfate compounds, nitrate compounds, carbon compounds, ammonium, hydrogen ion, organic compounds, metals (Pb, Cd, V, Ni, Cu, Zn, Mn, and Fe), and particle bounded-water. The major sources of $PM_{2.5}$ are fossil fuel combustion, vegetation burning, and the smelting and processing of metals.

Their lifetime is from days to weeks and travel distance ranges from 100s to >1,000s km. (Marian, 2000)

2.4 The Effects of PM_{10} from Forest Fires Smoke on Respiratory Health

There are different terms around the world for forest fires, which are also called wildfires, bushfires, or vegetation fires, by which that mean fires that occurs in the forest, scrub or grassland anywhere in the world except Antarctica. During bushfires, large area of lands can be covered in layers of smoke, hundreds of kilometers away from the actual fire.

Forest fires smoke has the potential to affect millions of people and is therefore a major public health problem. The air pollutant that increases most significantly as a result of forest fires smoke is PM. During forest fires smoke episodes, PM concentrations are usually much higher than urban background concentrations, at which effects on respiratory health have been observed. The smoke even small quantity increases in the risk of respiratory health effects and cause large public health problems.

Many studies have summarized the literature on the respiratory health effects of forest fires smoke. Results from studies in Asia-Pacific region and North America. These studies are reliable study designs with rigorous analysis and have risk estimates studies which measured PM concentrations and its related to respiratory health diseases.

Tham, Esperanza andAkram (2009) (used a time series approach to investigate the effects of PM_{10}, Airborne Particle Index (API representing fine particulate pollution 0.10–1.00 mm in diameter) and ozone on respiratory-related emergency department (ED) visits and hospital admissions during a 6-month bushfire season (2002–2003). No associations were found in the Gippsland region of Victoria because those in rural areas lived further away from hospitals and were therefore more likely to go to primary health-care providers. However, this study found an association between PM_{10} and ED visits after adjusting for day-of the week, temperature and

humidity. An increase of 9.10 mg/m^3 in PM_{10} was associated with an increase in respiratory ED attendances of 1.80%.

Cooper, Mira and Danforth(1994) compared numbers of asthma patients visiting ED in three inner-city hospitals in January and divided this into three time periods. They did not find any significant difference between before, during or after the forest fires.

Smith, Jalaludin and Byles (1996) using ED presentations in western Sydney, the authors used two different analytical methods which would have been able to detect an increase in asthma presentations of 50.00% for the bushfire period. However, they also did not find any increase in ED presentations for the bushfire period for the week of the forest fires compared to the same week the previous year.

Morgan, Sheppeard and Khalaj (2010) investigated respiratory hospital admissions with an extended time series analysis from 1994 to 2002. They divided PM_{10} into forest fires PM_{10} and 'non-forest fires' (background) PM_{10}. It was assumed that on days where city-wide 24 hours average PM_{10} was greater than the 99th percentile for the study period, the PM_{10} was primarily from forest fires and also found forest fire PM_{10} was associated with a small and sustained increase in elderly respiratory admission at lag 0 (1.72[0.12 to 3.34]), lag 1, and lag 2 days and also found PM_{10} was associated with a moderate and sustained increase in elderly COPD admission at lag 0 (3.29[0.86 to 5.78]), lag 1, lag 2 and lag 3 days.

On these days forest fire PM_{10} was calculated to be the difference between total PM_{10} and estimated background PM_{10}. Over the 8.5-year study period, 32 forest fire days were identified, resulting from 14 different forest fires events. Analyses were done for three age groups, and models adjusted for temperature, humidity, day of the week and presence of an influenza epidemic. They found no significant associations with 24 hours average concentration on 1, 2 and 3 days before the hospital admission. However, the effect between bushfire PM_{10} and background PM_{10} were in admissions for COPD among those over 65 years of age and for asthma admissions for those between 15 and 64 years of age. No associations were found with background PM_{10}, but significant associations were found for both outcomes with forest fire PM_{10}. The COPD admissions for those over 65 years were consistently associated with forest

fires PM_{10}, with a 10mg/m^3 increase in forest fires smoke resulting in an increase in hospital admissions of 3.30%.

Chen, Verrall and Tong(2006) had done the time series study from Brisbane to investigate the association between forest fires PM_{10} on respiratory hospital admissions. The results had shown that an increase in respiratory hospital admissions of 19.00% for forest fire days and of 13.00% for days without the presence of forest fires smoke.

Johnston, Bailie and Pilotto(2007) from Darwin investigated the association between hospital admissions for all respiratory diseases and forest fires during the 2000, 2004 and 2005 seasons combined. Unfortunately, no PM_{10} measurement data were available for the years 2001 to 2003. This is the only study on forest fire smoke which has used the case-crossover design that eliminates confounding factors by day of week and seasonal trends in the exposure variables.

An increase of 10 µg/m^3 in $PM_{10}$was most strongly associated with COPD and asthma. For indigenous people all effect sizes were larger, in particular for COPD presentations.

Several studies investigated the effect on respiratory health of the populations living in cities across South-East Asia. Aditama (2000), only one study was done in Indonesia, where the actual fires occurred. Routine data from government health facilities were used to compare cases between September 1997 and June 1998 with the same period in 1995–1996 and increases were reported for acute respiratory infection and bronchial asthma. The study found that the number of acute respiratory infections increased by 1.80 times, and in south Sumatra this was 3.80 times. Emmanuel (2000), study the time series analysis adjusting for temperature, relative humidity, rainfall and wind speed and exposure with PM_{10} found that increasing in 100 µg/m^3 of PM_{10} was significantly associated with a 12.00% increase in outpatient department for upper-respiratory tract illness, 19.00% for asthma and 26.00% for rhinitis. There were also increases in Emergency Departmentfor haze related conditions.

Buckeridge and other (2002) found that PM_{10} had significant effect on hospital admission rates for a subset of respiratory diagnose (asthma, bronchitis, COPD, pneumonia, upper respiratory and lower respiratory tract infections).

2.5 Chronic Respiratory Diseases

Chronic respiratory diseases (CRDs), affect the airways and other structures of the lungs, and represent a wide array of serious diseases. Preventable CRDs include asthma and respiratory allergies, COPD, occupational lung diseases, cancer, sleep apnoea syndrome and pulmonary hypertension. Many risk factors for preventable CRDs have been identified such astobacco smoke and other forms of indoor air pollution eg; pollutants, outdoor pollution and allergen (Brauer et al., 1999).

2.5.1 Definition of COPD

The working definition of COPD, as noted in the 2006 update of the Global Initiative for Obstructive Lung Disease (GOLD) guidelines, is that COPD is "a preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The air flow limitation is usually progressive and associated with an abnormal inflammatory response of lung to noxious particle or gases".(David & Buist, 2007)

Irritation of the lungs can lead to asthma, emphysema, and chronic bronchitis and, in fact, many people develop two or three of these together. This constellation is known as COPD (Kimball, 2009).

COPD is a leading cause of morbidity and mortality in many countries; high, middle and low income. According to WHO's Global Burden of Disease and Risk Factors project in 2001, COPD was the fifth leading cause of death in high-income countries and it was the sixth leading in middle and low income countries.

2.5.2 Burden of COPD

COPD is the fourth leading cause of death in the U.S. in 2006 (Centers for disease control and preventation, 2009). COPD prevalence, morbidity, and mortality is directly related to the prevalence of tobacco smoking, outdoor air pollution, indoor air pollution and also the burning of wood and biomass fuels that are the risk factors of COPD (Salvi & Barnes, 2009). The burden of COPD in the Asia-Pacific region is

higher than Western countries in terms of the numbers of death, years spent living with disability and years of life lost (Lopez et al., 2006).

2.5.3 Prevalence of COPD

The lowest estimation of prevalence are those based on self-reporting of a doctor diagnosis of COPD. The prevalence of COPD that studied in China, Hong Kong, Taiwan, Japan, Korea and Thailand have shown COPD range from 2.50% 10.90% (Zhong, Wang & Yao, 2007; Zielinski, Bednarek & Gorecka, 2006; Wang et al., 2007; Fukuchi, Nishimura & Ichinose, 2004; Kim, D. S., Kim, Y. S. & Jung, 2005 & Maranetra, Chuaychoo & Dejsomritrutai, 2002). In America found chronic bronchitis was about twice as common among women (57.6 per 1,000) as men (28.6 per 1,000). It was also more common among those over 65 years of age (56 per 1,000) and 45 to 64 years of age (54.9 per 1,000) than among those 18 to 44 years of age (31.6 per 1,000) (Centers for Disease Control and Prevention, 2009). In general, men are affected more than women. Such variations in prevalence across the region are probably due to differences in the methods of subject recruitment, age of the subjects and definitions of COPD. Taiwan has a good health insurance program with a coverage rate of more than 90.00% of the population, and it is therefore possible to assess COPD prevalence using the insurance database (Wang et al., 2007).

A systematic review and meta-analysis of studies carried out in 28 countries between 1990 and 2004, and additional study from japan, provide evidences that the prevalence of COPD was higher in smokers and ex-smokers than in nonsmokers, in age over 40 years than under 40 years also in men than in women (Halbert, 2006; Fukuchi et al., 2004).

2.5.4 Morbidity and Mortality of COPD

COPD is a disease with high morbidity and mortality, as illustrated by several studies conducted in Singapore, Japan, Taiwan and Hong Kong (Ng, Niti & Tan, 2008; Izumi, 2002; Ko, Ip & Chan, 2007; Wang et al., 2007). A study of subjects admitted to hospital with acute exacerbation of COPD (AECOPD) in Hong Kong found that the re-admissions and mortality rates over a 12-month period were respectively 56.30% and 18.80% (Ko, Ip & Chan, 2007). Another study that looked

specifically at patients whose AECOPD was severe enough to warrant non-invasive ventilatory support found that the mortality rate at 1 year was very high, at 49.10% (Chu et al., 2004). Nearly 50.00% of cases requiring home mechanical ventilation in recent years were COPD patients (Chu et al., 2004)

In Taiwan, COPD mortality rates decreased between 1981 and 1993 and increased thereafter (age standardization mortality decreased from 8.26 to 4.91 per 100,000 population from 1981 to 1993 and 716 then increased to a peak of 7.36/100 000 in 1999).The latter was largely attributable to increased mortality rates in men (Kuo, Yang &Kuo, 2005). In Singapore, COPD accounted for 4.60% of total deaths between 1991 and 1998. Although the mortality rate of COPD had fallen steeply by more than 40.00% since 1991, hospitalization rates showed little change (Ng et al., 2004). In Hong Kong, there was a downward trend in mortality rate from 37/100000 in 1997 to 29/100000 in 2005. This trend was observed in both sexes (M Chan-Yeung, personal communication). The reason for this downward trend is not clear, but the recent increase in COPD prevalence seen in Hong Kong (Ko, Ip & Chan, 2006). suggests that better recognition and treatment of COPD may have contributed.

2.5.5 Economic Burden of COPD

COPD is a costly conditions due to the large number of people who have it and how much it impairs their everyday functioning. It is estimated that the total economic costs of COPD will be $49.9 billion in 2010. This includes $29.5 billion in direct health care expenditures and $20.4 billion in indirect costs (National Heart Lung and Blood Institute, 2009).

The total direct costs of respiratory disease in Europe are estimated about 6.00% of the total health care budgets and COPD costs for 56.00% (38.6 billion Euros) (European Respiratory Society, 2003). The direct medical costs in developing countries are less important than the impact of COPD on workplace and home productivity. The health sector might not provide long-term supportive care service for severely disabled individual. The indirect costs of COPD represent a serious threat to their economies.

Table 2.4 Differential Diagnosis of COPD

	Suggestive features
COPD	Onset in midlife
	Slowly progressive symptoms
	Tobacco smoking history
Asthma	Onset early in childhood
	Variable symptoms
	History of allergy
Bronchiectasis	Large volumes of purulent sputum
	Radiograph shows bronchial dilatation or wall thickening

Source David & Buist (2007)

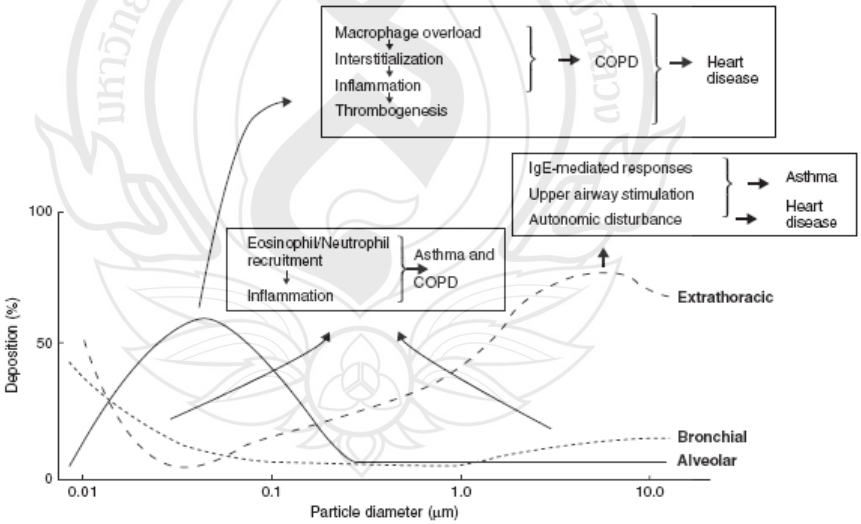


Figure 2.2 Deposition of Particles at Various Levels of the Respiratory Tract by Particle Size and Likely Mechanisms of Health Effect

Table 2.5 Staging of a COPD Exacerbations Based on Health- Care Utilization

Severity	Level of health – care Utilization
Mild	Patient has an increased need for medication, which he/she can manage in own normal environment.
Moderate	Patient has an increased need for medication and feels the need to seek additional medical assistance.
Severe	Patient/caregiver recognizes obvious and/or rapid deterioration in condition, requiring hospitalization.
Very sever	At this stage, complications such as respiratory failure and heart failure begin to develop. Quality of life is extremely impaired and the symptoms become life threatening.

Source Tan & Ng (2008)

2.6 Factors Associated with COPD

The pathogenesis of COPD is related to an interaction between host factor (*ie*, genes, airway hyperresponsivenes, and lung growth) and many different environmental exposures (*ie*, tobacco smoke, occupational dust and fumes, respiratory infection, outdoor air pollution, and indoor air pollution cause by biomass or traditional fuels and coal) and socioeconomic status, which could also be affected by comorbid diseases (David et al., 2007).

2.6.1 Genetic Factor

Many genetic factors may influence an individual likelihood of COPD developing. Studies have demonstrated an increased risk of COPD within families with COPD probands. The best known genetic factor linked to COPD is a deficiency of serine protease α-1 antitrypsin, which arises in 1.00-3.00% of patients with COPD (Stoller&Aboussouan, 2005). Alpha-1 antitrypsin (AAT) is the main inhibitor of

serum proteases. The inherited deficiency of AAT is the only genetic variation truly proven to be associated with development of COPD (Dimov & Viaykova, 2010).

Alpha-1 antitrypsin is a secretory glycoprotein produced by the liver and is the most abundant serum antiprotease in circulation (Kueppers, 1971).While the majority of AAT in the body is hepatocyte-derived, it is actively transcribed and secreted by other cell types including monocytes (Carroll, McElvaney & Greene, 2010), macrophages (Mornex et al., 1986), neutrophils (Bergin et al., 2010), intestinal epithelial cells (Perlmutter et al.,1989), and various epithelial cells in the lung (Hu & Perlmutter 2002; Venembreet al., 1994; Cichy, Potempa & Travis 1997), albeit in smaller quantities.

Other mutations affecting the structure of Alpha-1 antitrypsin or the regulation of gene expression have been identified as risk factors. Genes, including those for alpha-1 antichymotrypsin, alpha-2 macroglobulin, vitamin D-binding protein and blood group antigens, have also been associated with the development of COPD. Variants of the cystic fibrosis transmembrane regulator gene have been identified as risk factors for disseminated bronchiectasis (Sandford, Weir & Pare, 1997).

2.6.2 Tobacco Smoke

According to WHO, estimation in high-income countries, 73.00% of COPD mortality is related to smoking and with 40.00% related to smoking in low and middle income countries.(Lopez et al., 2006)

Tobacco smoking or cigarette smoking is the main risk factor and main method by which tobacco exposure is involve in the development of COPD. Smoking has been shown to accelerate the progression of the disease and increase mortality. (Pelkonen et al., 2001)

Although cigarette smoking is the most commonly encountered tobacco-related risk factors for COPD (Global Strategy for the Diagnosis, Management and Prevention of Chronic Obstructive Pulmonary Disease, 2012), other types of tobacco smoking popular in various countries are also risk factors for COPD (Jindal et al., 2006; Al-Fayez et al., 1988)and air pollution resulting from the burning of woods and other biomass fuels, has also been identified as a COPD risk factors (Global Strategy for the Diagnosis, Management and Prevention of Chronic Obstructive Pulmonary

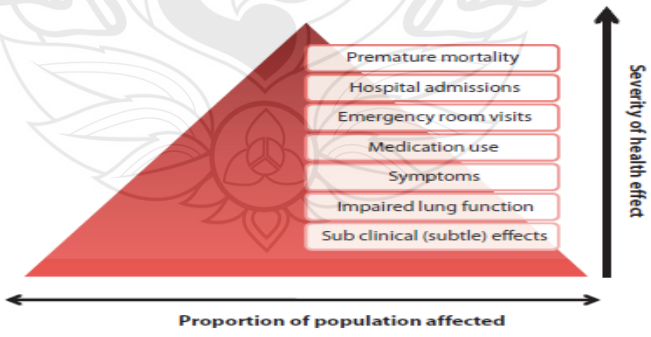
Disease, 2012). Passive exposure to cigarette's smoke may also contribute to the development of COPD by increasing the lung total burden of inhaled particles and gases. (Eisner et al., 2005)

In addition, smoking cessation is associated with a significant reduction of COPD exacerbations (Au, Bryson & Chien, 2009.) and hospital admissions. (Godtfredsen, Vestbo, Osler & Prescott, 2002)

2.6.3 Occupational Dust, Vapours and Fumes

Exposure to dusts, chemicals, vapors, and fumes in the area of workplace is a factor for many people with COPD. Although incompletely defined, the role of occupational dust and fumes in the development of chronic airflow limitation is well recognized in Asia.(LeVan,Koh & Lee, 2006)

The risk is less than that of smoking and interactions between smoking and occupational exposure to various agents are relevant. Occupational exposures to various dusts, chemicals, vapors and fumes act additively to increase (Dennekamp & Abramson, 2011) a person's risk of developing COPD. The fraction of COPD attributable to work was estimated as 19.20% overall and 31.10% in never smokers (Hnizdo, Sullivan, Bang & Wagner 2002). Specific occupational exposures that contribute to development of COPD have accumulated over the past two decades.(Hendrick, 1996)



Source American Thoracic Society (2000)

Figure 2.3 The Air Pollution Health Effects Pyramid

2.6.4 Indoor Air Pollution

The combustion of biomass fuel in the form of wood, coke, charcoal, coal, or animal dung for cooking and for heating in poorly homes, is an important risk factors for COPD in nonsmokers in developing countries including Asia (Smith et al., 1996).

Exposure to indoor air pollution from the combustion of solid fuels has been implicated, with varying degrees of evidence, as a causal agent of several diseases in developing countries, including acute respiratory infections and otitis media (middle ear infection), chronic obstructive pulmonary disease (COPD), lung cancer, asthma, cancer of the nasopharynx and larynx, tuberculosis, perinatal conditions and low birth weight, and diseases of the eye such as cataract and blindness (Smith et al., 1996). Almost 3 billion world population use biomass and coal as the major source of energy for cooking, heating and other household needs, so the population at risk worldwide is large. (Torres-Duque, Maldonado, Perez-Padilla, Ezzati & Viegi, 2008)

WHO estimates that, in countries of low and middle income, 35.00% of people with COPD developed the disorder after exposure to indoor smoke from biomass fuel (Lopez et al., 2006). Furthermore, WHO suggests that 36.00% of mortality from lower respiratory disease is also related to indoor smoke exposure (Lopez et al., 2006). Even in some developed countries, such as Canada, Australia, and western states of USA, the persistent rise in the cost of energy has prompted an increasing number of households to use wood or biomass products for heating (Smith et al., 1996). Findings of a report from China showed that COPD prevalence in never-smoking women is two to three times higher in a rural area where women are exposed to biomass smoke compare with urban women without this exposure. (Torres-Duque et al., 2008)

Tone and others (2009), Study a total of 504 rural Mayan women in highland Guatemala aged 15–50 years, all usingtraditional indoor open fires, were randomized to either receive a chimney woodstove (plancha) or continue using the open fire. Assessments of chronic respiratory symptoms and lung function and individual measurements of carbon monoxide exposure were performed at baseline and every 6 months up to 18 months. Use of a plancha significantly reduced carbon monoxide exposure by 61.60. For all respiratory symptoms, reductions in risk were observed in the plancha group during follow-up; the reduction was statistically significant for

wheeze (relative risk = 0.42, 95% confidence interval: 0.25, 0.70). The number of respiratory symptoms reported by the women at each follow-up point was also significantly reduced by the plancha odds ratio = 0.7, 95% confidence interval: 0.50, 0.97).

However, no significant effects on lung function were found after 12–18 months. Reducing indoor air pollution from household biomass burning may relieve symptoms consistent with chronic respiratory tract irritation.

2.6.5 Outdoor Air Pollution

In many Asian countries, the ambient air pollution is long-term problem, which are undergoing rapid industrialization and urbanization. The risk attributable to outdoor pollutants in development of COPD is much smaller than indoor air pollution. WHO estimates the urban air pollution causes 1.00% of COPD cases in high-income countries and 2.00% in low and middle income (Lopez et al., 2006).

Most epidemiological air pollution studies conducted in high-income countries on short-term exposure and effects on the general population or patients suffering from COPD used the time series method to evaluate the risk of mortality due to cardio respiratory diseases, or COPD hospital admissions temporally related to air pollution levels (Liu, Shou, Wang, X., Wang,D. & Lu, 2007), few studies have been conducted prospectively to evaluate the long-term effects of air pollution on COPD. Although outdoor air pollution levels tend to be higher in low-income countries than in high-income countries, fewer studies were conducted.

Viegi and other (2006) found that exposure to PM by internal combustion and industrial emission recognized as significant contributors to exacerbation of COPD.

Moolgavkar and Luebeck (1997) found that the higher outdoor levels of O_3, PM_{10}, SO_2 and NO_2 were associated with the significantly higher rates of hospital COPD admission in Minneapolis, Minnesota.

Peel and others studies 4 million hospital emergency visits in Atlanta found that higher outdoor levels of PM_{10}, NO_2 and CO were associated with significantly higher emergency room visits for upper respiratory infections and COPD (Peel et al., 2006).

The study in Denver, Colorado found that study of adults with advanced COPD in two winter that higher ambient levels of CO, PM_{10} and NO_2 were associated with poorer lung function and more rescue medication use in one of the two winter. (Silkoff et. al, 2005)

Katsouyanni and other found that each 10μg/m3 increase in PM_{10} resulted in the statistically significant increases in hostital admissions for COPD of 1.00% in Europe and 1.50% in US. (Katsouyanni et al., 2001)

The study of Anderson and others found that PM may impair ventilation in COPD patients by causing airway narrowing and increasing the function of breathing. (Anderson et. al, 1990)

The seven studies in French found that urban air pollution was associated with increased in total mortality over 25 years. (Filleul et al., 2005)

In some studies of a consecutive cross sectional study conducted between 1985 and 1994,involving 4,757 women living in the Rhine-Ruhr Basinof Germany, it was found that the prevalence of COPD (Global Initiative for Chronic Obstructive Lung Disease stages 1–4) was 4.50%, whereas COPD and pulmonary function were the strongest affected by particulates with an aerodynamic diameter <10 mm (PM_{10}) and traffic-related exposure. (Schikowski, Sugiri & Ranft, 2005)

2.6.6 Aging

Age is a risk factor for COPD but it is unclear if healthy aging as such leads to COPD or if age reflects the sum of exposures throughout life. COPD prevalence, morbidity, and mortality increase with age. Lung function, which reaches in peak level in young adults, starts to decline in the third and fourth decades of life (Ran, Wang, & Yao, 2006). Some researchers have reported that elderly people with high levels of lung function live longer than do those with low level of lung function. (Fletcher,Peto, Tinker & Speizer, 1976)

2.6.7 Socioeconomic and Related Factors

Poor populations tend to have a higher risk of developing COPD and its complications than their wealthier counterparts. (Mannino & Davis, 2006)

Many studies from developed countries suggested that socioeconomic status (SES), measured by income and educational level, was associated with lung function and COPD in terms of exacerbation, prevalence and mortality (Peng & Mei, 2011; Bakke, Hanoa & Gulsvik, 1995; Prescott, Lange & Vestbo, 1999). This association may be partly explained by the greater proportion of smokers among people in lower socioeconomic groups, but smoking may not explain all of the association.

2.6.8 Sex

2.6.8.1 Women and COPD: In low-income countries exposure to indoor air pollution, such as the use of biomass fuels for cooking and heating, causes the COPD burden. Almost 3 billion people worldwide use biomass and coal as their main source of energy for cooking, heating, and other household needs. In these communities, indoor air pollution is responsible for a greater fraction of COPD risk than smoking or outdoor air pollution (WHO, 2011a). Biomass fuels used by women for cooking account for the high prevalence of COPD among nonsmoking women in parts of the Middle East, Africa and Asia. Indoor air pollution resulting from the burning of wood and other biomass fuels is estimated to kill two million women and children each year.

Using biomass fuels for cooking and heating is the highest risk factor for developing COPD in low and middle income countries.

Recent research indicates that estrogen plays a role in maintaining the lung function in women, putting postmenopausal women at higher risk of developing COPD, having a severe form, and dying from COPD. (WHO, 2011b)

Ekici and others (2005), the study of Turkish in non-smoking women found that a quarter of cases with chronic airflow obstruction could be attributed to exposure to biomass smoke. The another study in Pakistan, those with biomass exposure in 2,500 female subjects were 205 times more likely to develop COPD than the controls. (Akhtar, Ullah, Khan &Nazli, 2007)

Schikowaski, Sugiri and Ranft (2005) studied in women and used the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria to define COPD found a 7 $\mu g/m^3$ increase in ambient PM_{10} was associated with a 5.10% more rapid decline in FEV1 and an odds ratio of 1.33 for the development of COPD.

2.6.9 Infection

A history of severe childhood respiratory infection has been associated with decreased lung function and increased respiratory symptoms in adulthood (Barker & Godfrey, 1991). Chronic obstructive pulmonary disease affects an estimated 600,000 people in the UK and accounts for 30,000 deaths per year. In the USA it is estimated to affect 24 million people and is the 4th leading cause of morbidity and mortality. (Rennard et al., 2002)

On a worldwide basis, it is estimated that COPD will be the 3rd leading cause of mortality by the year 2020 (Murray & Lopez, 1997). Much of the morbidity and mortality associated with COPD are due to acute exacerbations. Historically COPD exacerbations have been considered as being predominantly caused by bacteria, however recent evidence has suggested that respiratory viruses are associated with 40.00%–60.00% of COPD exacerbations. (Seemungal et al. 2001; Rohde et al., 2003; Tan et al., 2008)

The presence of HIV infection has been shown to accelerate the development of emphysema, but the impact on the development of COPD in Asians is unknown. (Bakke et al., 1995)

Ko and others (2007), they studied in Hong Kong found that among the 530 episodes of acute exacerbation of COPD admissions with sputum saved 13.00%, 6.00% and 5.50% had positive growths of *Haemophilusinfluenzae*, *Pseudomonas aeruginosa* and *Streptococcus pneumoniae*.

Previous studies of the role of respiratory viruses in COPD exacerbations suggested that they are responsible for a minority of exacerbations only, with viruses detected in 18.00% of episodes of acute respiratory illness (Smith et al., 1996).

Among respiratory viruses, influenza has the greatest impact in terms of both the morbidity and mortality that it causes. Although influenza affects all age groups, much of the morbidity and mortality are concentrated in high-risk groups such as the elderly and those with comorbid disease, particularly cardiovascular and pulmonary disease. (Patrick & Sebastian, 2007)

Among patients hospitalized with acute respiratory disease during an influenza epidemic chronic pulmonary disease is the most common underlying disease (Glezen,

Decker & Perrotta, 1987) suggesting that pulmonary disease including COPD is the most important risk factor for an adverse outcome with influenza infection.

Recent studies have also re-evaluated the role of bacterial infection in the 1918 pandemic. It is now thought that much of the mortality may have been due to bacterial pneumonia with organisms such as *S. pneumonia* and *H. influenzae*, rather than primary viral pneumonia (Brundage, 2006). These organisms are common colonizing organisms in COPD patients.

In addition the mortality and requirement for intensive care of community acquired bacterial pneumonia is higher in COPD patients. (Restrepo, Mortensen, Pugh & Anzueto, 2006)

2.7 Pathogenesis of Particulate Matter Induced COPD

The major mechanisms of deposition of particulate matter in the lungs are impaction, sedimentation and diffusion. (Stuart, 1976)

2.7.1 Impaction

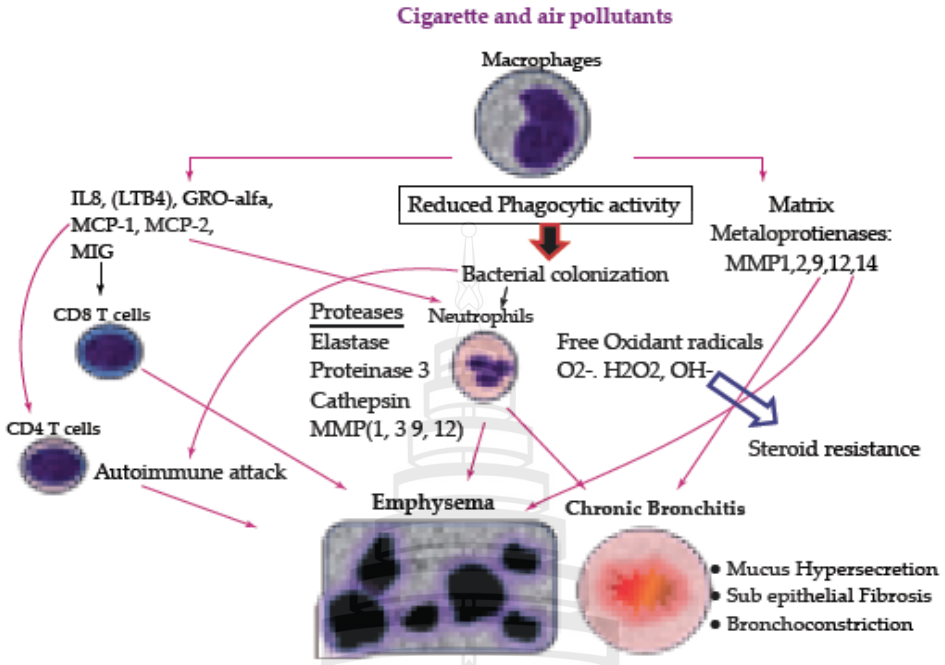
Impaction is the main mechanism of large PM deposition. The larger particles usually travel on an initial path until they are deflected by branching airway. (Ling & van Eeden, 2009)

2.7.2 Sedimention

The particles fall under their force of gravity. The mechanism affects the particles size between diameter of 0.10 μm and 50 μm. (Ling et al., 2009)

2.7.3 Diffusion

The diffusion affects the smallest particles that they are displaced by random gas motion and occur in the small airway of the lung. (Ling et al., 2009)



Source William (2000)

Figure 2.4 Pathogenesis of COPD

2.8 Assessment of COPD

The goals of COPD assessment are to determine the severity of the disease, its impact on the patient's health status and the risk of future events.

2.8.1 Assessment of Symptoms

There are several validated questionnaires to assess symptoms in patient with COPD.

2.8.1.1 The modified medical Research Council Dyspnea scale: This questionnaire relates well to other measures of health status and predicts future mortality risk. (Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease, 2011)

Table 2.6 The Modified Medical Research Council Dyspnea Questionnaire of Assessing the Severity of Breathlessness

Grade	Description of Breathlessness
0	I only get breathless with strenuous exercise.
1	I have shortness-of-breath when hurrying on level ground or walking up a slight hill.
2	On level ground, I walk slower than other people of the same age because of breathlessness, or have to stop for breath when walking at my own pace.
3	I stop for breath after walking about 100 yards or after a few minutes on level ground.
4	I am too breathless to leave the house or I am breathless when dressing.

2.8.2 Spirometric Assessment

The classification of airflow limitation severity in COPD shows in Table 2.7.Sepecificspirometric cut-points are used for purposes of simplicity. Spirometry should be performed after the administration of an adequate dose of a short-acting inhaled bronchodilator in order to minimize variability (Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease, 2011).

Table 2.7 Classification of Severity of Airflow Limitation in COPD

GOLD	Description of Breathlessness
1 Mild	$FEV_1 \geq 80\%$ predicted
2 Moderate	$50\% \leq FEV_1 < 80\%$ predicted
3 Severe	$30\% \leq FEV_1 < 50\%$ predicted
4 Very severe	$FEV_1 < 30\%$ predicted

2.9 Conceptual Framework

Independent Variables **Dependent Variable**

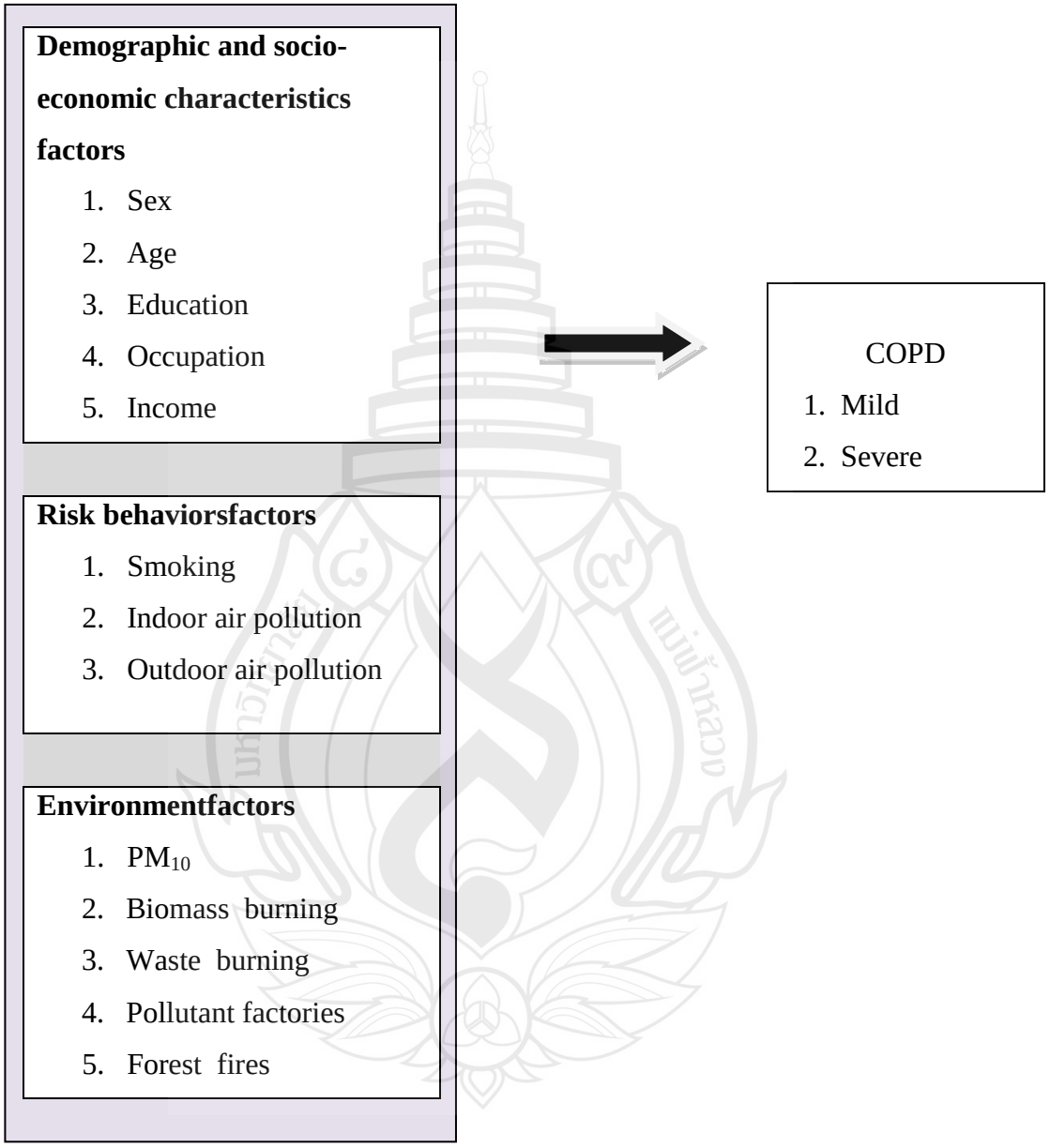


Figure 2.5 Conceptual Framework

CHAPTER 3

MATERIALS AND METHODS

A retrospective cohort study design was conducted in five hospitals in ChiangRai Province, Thailand. The purpose of this study was to determine the association between PM_{10} and the severity of COPD in Chiang Rai, Thailand. The target population was COPD cases who lived in Chiang Rai Province. The subjects were recruited from Out-Patient Department (OPD) lists who recently diagnosed with COPD. The study settings were in five Chiang Rai hospitals: Mae Chan, Mae Sai, Weing Chai, PraYaMengRai and Somdejprayanasungwon Hospitals.

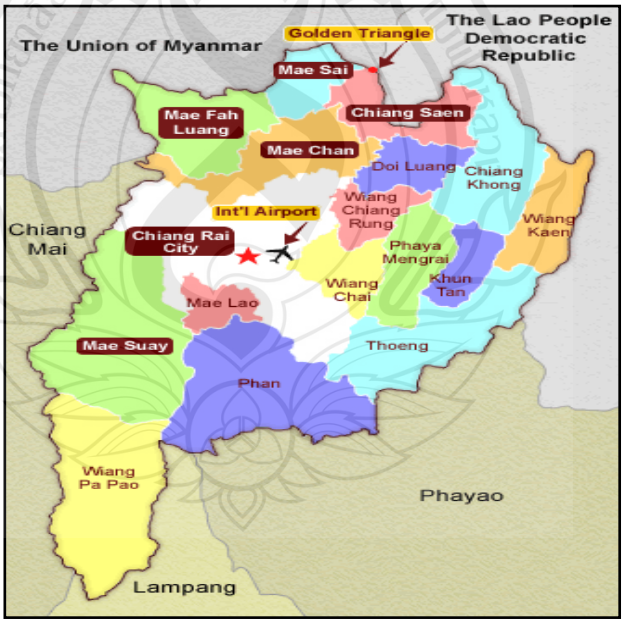


Figure 3.1 Map of Study Settings, Chiang Rai Province, Thailand

3.1 Study Design

The retrospective cohort study design was used to evaluate the association between PM_{10} level and severity of COPD. The exposure of PM_{10} by the subjects was evaluated for the two sites monitoring $PM_{10.}$ The evaluation of air quality was recorded clearly from two sites at Natural Resources and Environment, Muang, Chiang Rai and Mae Sai District Public Health Office. However, the data from air quality mornitoring at Mae Sai station had less than 75.00%, the researcher chose the data from Muang Chiang Rai station was representative of all air quality data. The classification of the exposure period (considered to be during March–May 2012) shows the mean level of PM_{10} taken from air pollution monitoring station. The un-exposure period (considered to be from August–October 2011) shows the mean level of PM_{10}, taken from air pollution monitoring station. The mean level of PM_{10} in each period was used for evaluation of the dependent variable in the analysis. For this study, both the exposure and un-exposure period was measured in the each subject.

3.2 Study Population

The study population were Thai people who had been diagnosed with COPD by a physician and lived in Chiang Rai Province, Thailand.

3.3 Study Samples

The subjects were COPD patients who were recently diagnosed with COPD from five local hospitals. The sample size was calculated by the following formula.

3.4 Sample Size Estimation

The estimated sample size is calculated as following formula:(Fleiss, 1981)

$$m' = [Z_{\alpha/2}\sqrt{(r+1)\overline{PQ}} + Z\beta\sqrt{rP1Q1 + P2Q2}]^2$$

$$r = \frac{n1}{n2}$$

$$P = (P_1 + rP_2) / (r+1)$$

$$Q = 1-P$$

$$m = \frac{m'}{4}\left(1+\sqrt{1+\frac{2(r+1)}{m'r\,|P_2 - P_1|}}\right)^2$$

$$n = 304$$

Where

α = Level of type I error set at 0.05 (5.00%)

Z_α = Standard score for α ($Z_{0.95}$ =1.96)

β = Level of type II error set at 0.20 (20.00%)

Z_β = Standard score for power of test ($Z_{0.20}$ = 0.80)

n_1 = m (Size of sample from population 1)

n_2 = rm (Size of sample from population 2)

p_1 = Proportion of outcome in non-exposed group 15%

p_2 = Proportion of outcome in exposed group 20%

1-β = Power (0.80)

r = ratio of exposed to non-exposed group

The study conducted by Schwartz found that the risk ratio of hospital admissions for respiratory disease was 1.54 (Schwartz, 1994). The data based on COPD cases in Mae Chan hospital found that COPD cases were exposed to PM_{10}.The probability of exposed group was 20.00% (Mae Chan Hospital, 2011).The estimation of the sample size needed for this study is 276 cases, increased 10.00% for margin of error, totaling 304 cases.

3.5 Criteria

3.5.1 Inclusion Criteria

3.5.1.1 Having been diagnosed with COPD and visited COPD clinics in Chiang RaiProvince.

3.5.1.2 Found complete medical records.

3.5.2 Exclusion Criteria

3.5.2.1 Could not speak Thai.

3.5.2.1 Refused to participate.

3.5.2.3 Had respiratory co-morbidities: lung cancer, tuberculosis, lung fibrosis,sarcoidiosis, and cystic fibrosis.

The simple random sampling technique was used for selecting the subjects after determining whether the subject met the inclusion criteria from the lists of the five hospitals in Chiang Rai. After providing the information by verbal consent to the subjects about the objective and process of the study.Informed consent had been obtained before interview.

The subjects were recruited by simple random sampling technique. All subjects had their clinical signs and symptoms recorded two times each. The first time their clinical signs and symptoms were evaluated during August-October 2011, and the second time their clinical signs and symptoms were evaluated during March to May 2012. All data was collected from medical records and face-to-face interviews. The classification of exposure was clarified by high and low peak periods of PM_{10} in Chiang Rai. The subjects in the exposed period and un-exposed period were the same people.

3.6 Research Instruments

The constructed questionnaire was used as a research instrument. Before being used in the field all instruments had been tested for reliability and validity. The

questionnaire had been tested in the pilot phase and refined again before implementing.

The questionnaire was divided into four parts. The first part was composed of the questions about general information and socio-economic characteristics. The second part was composed of questions of exposure to various kinds of smoke. The third part was composed of questions of their medical history. The last part was composed questions on the characteristics of their immediate environment. The PM_{10} concentration data was obtained from secondary information. The High Volume Air Sampler machine for detecting PM_{10} levels was used from The Pollution Control Department, Ministry of Natural Resources and Environment, Thailand.

3.6.1 Measurement of the Independent Variables

As mentioned above, the questions for evaluation of independent variables were divided into four parts. The first part of the questionnaire includes: sex, age, education, occupation and income. The second part includes questions of smoking habits, history of exposure to indoor and outdoor air pollutants. The third part of the questionnaire includes the environmental factors of exposure to biomass burning, transportation, pollutants from factories and forest fires.The forth part of the questionnaire includes the subjects' medical records. For this part, the researcher gathered data by examining their medical records. Part one through part three were completed and compiled by face-to-face interview.

Questions regarding environmental factors were collected by asking about the following risk factors:"Have you lived in or do you live now in a household where you were/are exposed to cigarette smoke?" The answer provided was either "yes" or "no". If the answer provided was "yes" then the subjects were asked the next question: "How many hours a day were/are you exposed to cigarette smoke?"

For the question: Do you use gas,wood or coal to warm your household? The answer provided was either "yes" or "no". If the answer provided was "yes" then follow-up question was asked: How many hours, on average, does the subject use gas, wood or coal to keep warm?

For the question: How do you manage waste in your household? The subjects answered how they manage waste in their household.

For the question:Is your residence near a factory?The answer provided was either “yes” or “no”. If answer was “yes” the subjects were asked what kind of factory is nearby.

For the question:Is there the agricultural burning near your residence? The answer provided was either “yes” or “no”. If the answer was “yes” the next question was: “Can the patient smell the smoke from agricultural burning?”

For the question: Are there forest fires near your residence? The answer provided was either “yes” or “no”. If the answer was “yes” the next question was “Can the patients smell smoke from the forest fires?”

3.6.2 Measurement of the Dependent Variable

The severity of COPD was measured by the modified Medical research Council dyspnea scale (mMRC), used a simple grading system to clarify the level of severity of COPD. (Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease., 2011)

Table 3.1 The Modified Medical Research Council Dyspnea Scale (mMRC)

Grade	Description of Breathlessness
0	I only get breathless with strenuous exercise.
1	I have shortness-of-breath when hurrying on level ground or walking up a slight hill.
2	On level ground, I walk slower than other people of the same age because of breathlessness, or have to stop for breath when walking at my own pace.
3	I stop for breath after walking about 100 yards or after a few minutes on level ground.
4	I am too breathless to leave the house or I am breathless when dressing.

Symptoms and/or health status using the modified Medical Research Council Dyspnea Scale (mMRC) (see Table 3.1) AnmMRC score of ≥2 is indicative of high impact of symptoms.

3.6.3 Testing Validity of Instruments

There were two steps for testing the validity of the questionnaire. The first was test by IOC test; the second was the pilot test.

3.6.3.1 Step 1 (IOC test)

The validity of the questionnaire was tested using IOC technique. This study used three external experts to give an opinion for each question (see appendix D for profiles of the three external experts).

Finally, after receiving the assessment from all three of the external experts, the researcher pooled all the scores together and made an interpretation. If any item had a total score of zero (0), it was excluded from the questionnaire. If the total score less than 0.50, these items were corrected according to the suggestion of the expert panel. If the items had the total score ≥ 0.50 and there were suggestions made by the panel these items were corrected before pooled into the final draft.

IOC was calculated by the following formula

$$IOC = \frac{\Sigma R}{n}$$

Where

R = score from each expert

n = number of experts

In this study, all the items in the questionnaire were found to have a total score equal 0.7 that more than 0.50.

3.6.3.2 Step 2 (Pilot test)

Before data collection, a pilot test was conducted at TambonMengRai(a primary care hospital) in August 2011. The pilot test was an essential component for implementing the questionnaire under realistic conditions to give practical experience in all aspects of data collection. Thirty subjects were chosen by simple random

sampling. Based on these results, some questions in the questionnaire were revised by the researcher.

The pilot test was conducted in the following stages:

Stage 1: Coordinated with the director of TambonMengRai health primary care hospital.

Stage 2: Simple Random Sampling of COPD cases from COPD lists.

Stage 3: Made appointments with the selected subjects.

Stage 4: Face-to-face interview with each COPD case asking questions in a private and confidential room.

Stage 5: Researcher collected the questionnaire which was completed by the subjects.

3.7 Step of the Data Gathering

The following was the steps were used to gather data in this study:

1. Coordinatedwith thehospital directorfor permission to interview their patients.
2. Coordinatedwith the COPD clinic staff who are responsible for the COPD clinics in each hospital and making appointments with the clinical staff.
3. Identified subjects according to inclusion and exclusion criteria.
4. Made a random sampling list to find subjects.
5. Providedallinformation,such as the purpose of the study, to the subjects.
6. Obtained the consent form from the subjects.
7. Interviewed usingthe completequestionnaire, which lasted 15 minutes with each subject in the private and confidential room.
8. Reviewed the medical records of each subject.
9. Coded and entered data into the excel program.
10. Cleaned the data.

11. Analyzed the data

1) Descriptive statistics (mean, median, percentile, standard deviation)

2) Inferential statistics (unconditional logistic regression model, Chi-square test)

12. Interpreted data.

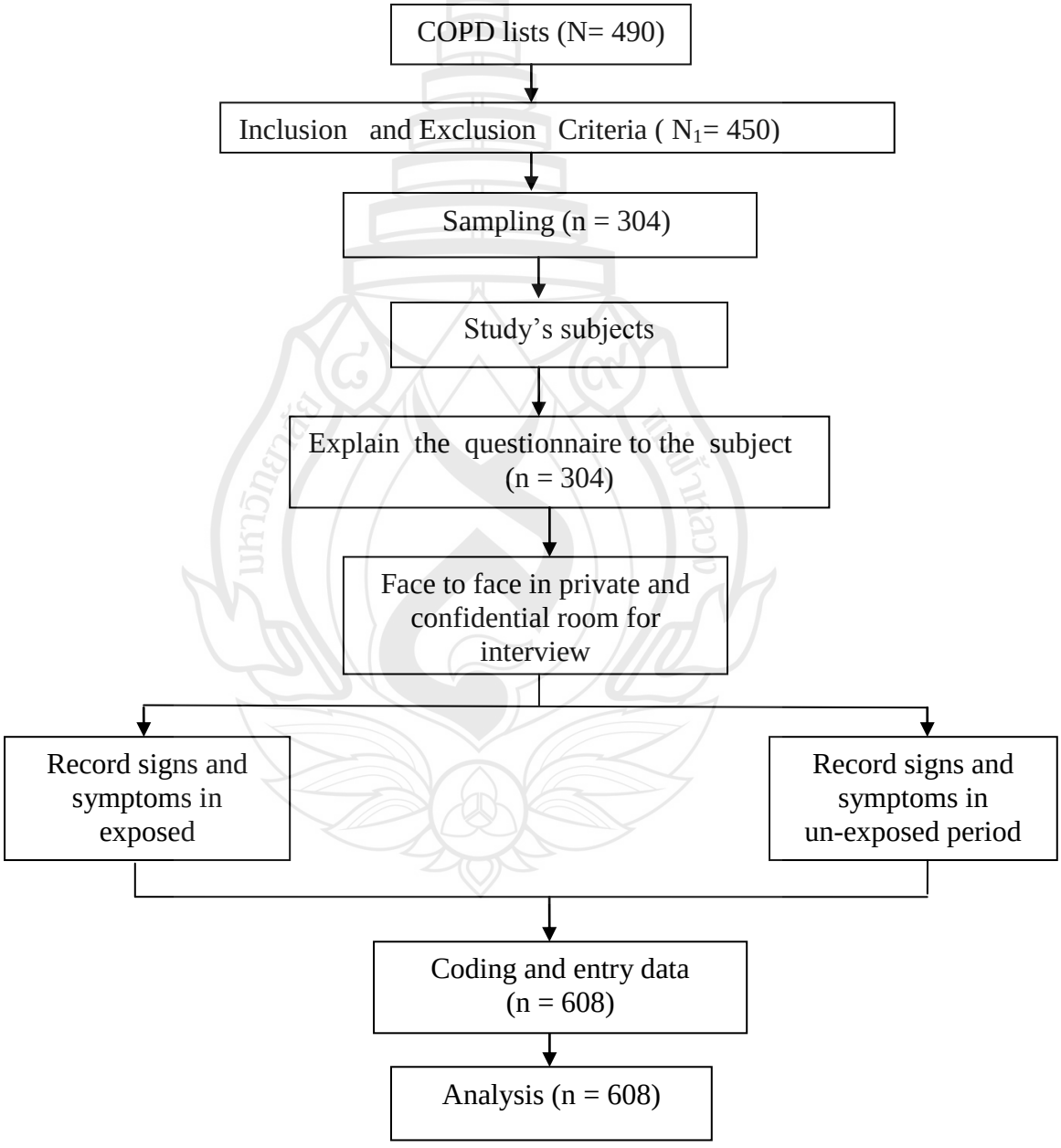


Figure 3.2 The Process of Sampling Subjects and Analysis

3.8 Statistical Analysis

Data collected was double-entry and validated by using Microsoft Excel.

3.8.1 Descriptive Statistics included mean, median, percentile and standard deviation were used to explain the general characteristics of the sample.

3.8.2 Inferential Statistics were selected the unconditional logistic regression model for finding the association between the severity of COPD and PM_{10} concentrations and the chi - square test to compare the characteristics and severity of COPD. The level of statistical association for multivariate was considered significant at $p<0.05$.

3.9 Ethical Consideration

All instruments of the study were approved by the Committee for the Protection of Human Subjects of Mae Fah Luang University, Thailand, no.REH-55025-25/2555

CHAPTER 4

RESULTS

A retrospective cohort study design was applied to describe the association between PM_{10} and severity of COPD. The subjects were COPD cases that were recently diagnosed with COPD. The selection of subjects was recruited from COPD clinics in five Chiang Rai hospitals in Chiang Rai Province: Mae Chan, Mae Sai, Prayameng Rai, Chiang San and Somdejprayanasungwon Hospitals.

The independent variables were collected using face–to–face interviews and the questionnaire, and was recorded from the profiles of the subjects' medical history. The information of PM_{10}levels was collected from two PM_{10} monitoring sites.

The results were described according to 6 issues below:

4.1 Daily concentrations of PM_{10} levels;

4.2 General Characteristics of subjects;

4.3 Risk behaviors and medical history;

4.4 Environmental factors;

4.5 Comparisons of general characteristics of subjects;

4.6 Analysis of risk factors.

4.1 Daily Concentrations of PM_{10} Levels

The daily concentrations of PM_{10} levels were observed from August–October 2011 (during rainy season) and again from March–May 2012 (during dry season). As expected, the rainy season revealed low-peak PM_{10}levels, and the dry season revealed high-peak PM_{10} levels. Twenty-four days during the dry season showed higher than the accepted standard "safe" PM_{10} levels (120 µg/m^3). Those higher than accepted "safe"days were found in March 2012.

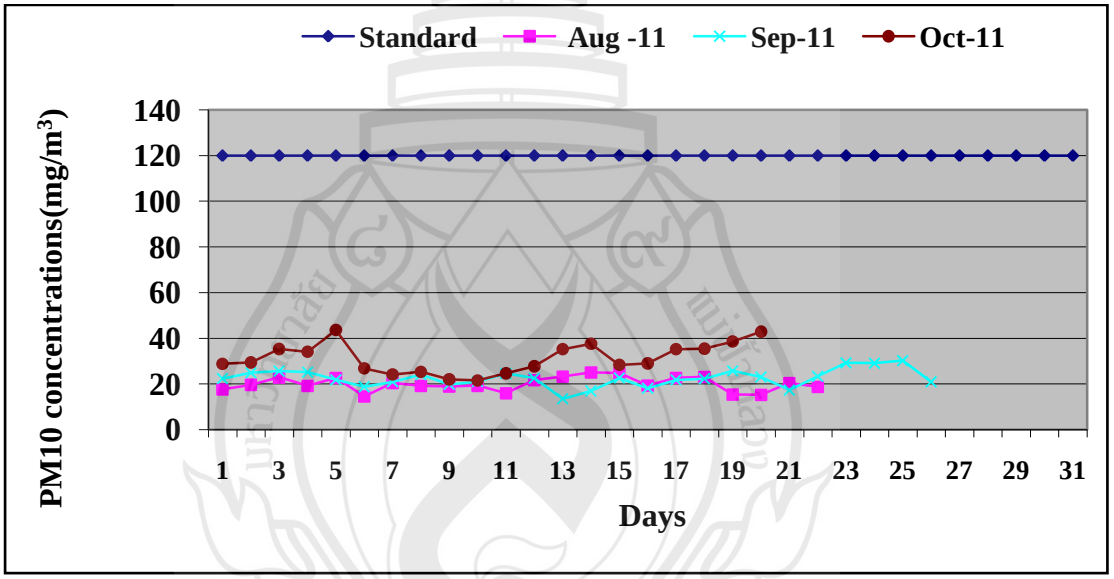


Figure 4.1 The Daily of PM_{10} Concentrations in Chiang Rai Province During Low Peak Period

Figure 4.1 shows the PM_{10} concentrations in low peak 3 months period (August–October 2011) of PM_{10} level. In August 2011 the highest PM_{10} level was 33.70 µg/m^3, the lowest was 14.50 µg/m^3, with an average level of PM_{10} at 20.37 µg/m^3. The standard deviation is 12.66.

In September, 2011 the maximum PM_{10} level was 33.10 µg/m^3,the lowest was 13.90 µg/m^3, with an average was 22.92 µg/m3. The standard deviation is 1.06.

In October, 2011the maximum PM_{10} level was 47.40 µg/m3 and the minimum was 20.40 µg/m^3, with the average being 31.15 µg/m^3. The standard deviation is 2.33.

However, PM_{10} concentrations in these 3 months were not over the accepted standard PM_{10} level (120 µg/m^3).

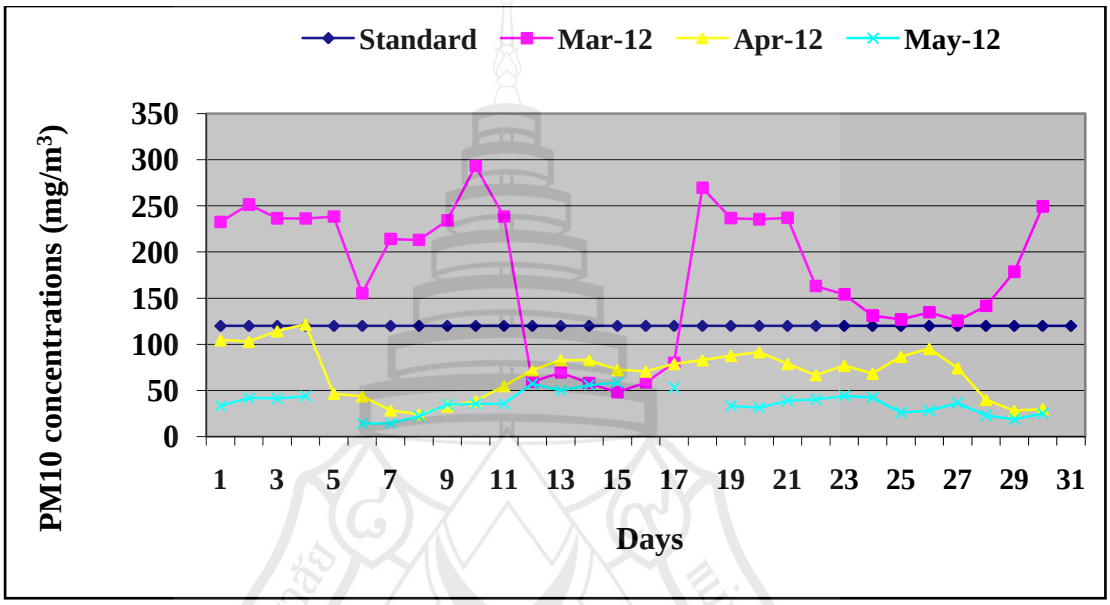


Figure 4.2 The Daily of PM_{10} Concentrations in Chiang Rai Province During High Peak Period

Figure 4.2 shows the PM_{10} concentrations at their peak in this three month period (March–May, 2012) of PM_{10} level.

In March 2012, the highest PM_{10} level was 293.38 µg/m^3, the lowest was 48.13µg/m^3, with the average of 173.99 µg/m^3 and the standard deviation is 73.15.However, the average PM_{10} concentrations for this month was over the PM_{10}accepted "safe" level (120 µg/m^3) for 24 days.

In April 2012,the maximum of PM_{10} level was 122.79 µg/m^3, the minimum was 24.33µg/m^3, and the average was 69.41µg/m^3. The standard deviation is 27.70. The average PM_{10} concentrations for this month was over the PM_{10}"safe" level (120 µg/m^3) for 1 day.

In May 2012, the maximum of PM_{10} level was 58.46 µg/m^3, the minimum was 14.42µg/m^3, the average was 36.47µg/m^3, and the standard deviation is 12.40. There were no days that showed to be over the PM_{10}"safe" level.

4.2 PM_{10} Concentrations in Chiang Rai Province

Table 4.1 shows the PM_{10} concentrations in Chiang Rai Province, Thailand in the year 2011-2012. The highest PM_{10} level was 293.38 µg/m^3 in March 2012, whereas the lowest level was 13.90 µg/m^3 in September 2011. The highest mean of PM_{10} was 173.99 with SD 73.15 µg/m^3 in March 2012. The average level of PM_{10} during the exposure period (March-May 2012) had a higher than average PM_{10} level than the un-exposure period (August-October 2011). March and April reported a higher than standard"safe" level of PM_{10}.

Table 4.1 Twenty–Four Hour Average PM_{10} Concentrations in Chiang Rai Province, Thailand, 2011-2012

Months	**PM_{10} concentrations**	
	Range (µg/m^3)	**Mean/SD**
August 2011	14.50-33.70	20.37/12.66
September 2011	13.90-33.10	22.92/1.06
October 2011	20.40-47.40	31.15/2.33
March 2012	48.13-293.38	173.99/73.15
April 2012	24.33-122.79	69.41/27.70
May 2012	14.42-58.46	36.47/12.40

4.3 Characteristics of Subjects

Table 4.2 shows general characteristics of subjects who participated in this study. There were one hundred sixty subjects (of which 52.60% were female), 37.30% aged 71-80 years old, 81.90% had an income ≤ 5,000 baht/month, and 63.10% unemployed, 69.10% married, and 96.71% Buddhist.Sixty subjects were selected from each of the following hospitals: (19.74%) Mae Sai, Prayameng Rai, Chiang San and, Somdejproyanasungwon hospitals, whereas sixty-four (21.04%) were selected from Mae Chan hospital.

Table 4.2 General Characteristics of Subjects

Characteristics	n	%
Total	304	100.00
Sex		
Male	144	47.40
Female	160	52.60
Age (years old)		
≤50	14	46.00
51-60	40	13.20
61-70	104	34.30
71-80	113	37.30
≥81	33	10.60
Number of COPD case in each hospitals		
Mae Chan	64	21.04
Mae Sai	60	19.74
Prayameng Rai	60	19.74
Chiang San	60	19.74
Somdejprayanasungwon	60	19.74

Table 4.2 (continued)

Characteristics	n	%
Marital status		
Single	2	0.70
Married	210	69.10
Widowed	1	0.30
Divorced	91	29.90
Religion		
Buddhism	294	96.70
Other	10	3.30
Income (baht/month)		
≤5,000	249	81.90
≥5,001	55	18.10
Family member (person)		
≤2	99	32.50
3-4	134	44.10
5-6	52	17.10
≥7	19	6.30
Occupation		
Unemployed/Retired	192	63.10
Housewife	24	7.90
Day-laborer	16	5.30
Farmer	64	21.10
Business Owner	7	2.30
Office Worker	1	0.30

4.4 Risk Behaviors and Medical History of Subjects

Table 4.3 shows the risk behaviors among the subjects, 85.20% smoked, 67.40% used beedi (hand-rolled cigarettes), 72.70% were exposed passive smoke from at least one family member. Two hundred fifty-nine subjects (85.20%) had been diagnosed with COPD for 1-5 years, 80.80% got influenza vaccine. One hundred thirty-five subjects (44.40%) had co-morbidities, and 61.20% had acute exacerbation during winter season.

Table 4.3 Risk Behaviors and Medical History

Characteristics	n	%
Smoked		
Yes	9	3.20
No	35	11.60
Quit	260	85.20
Types of smoking		
Beedi (hand-rolled cigarettes)	205	75.93
Cigarettes	26	9.63
Both	39	14.44
Second-hand smoke		
Yes	83	27.30
No	221	72.70
Diagnosis of COPD (years)		
≤5	259	85.20
6-10	15	4.90
11-15	16	5.20
≥16	14	4.60

Table 4.3 (continued)

Characteristics	n	%
Received Influenza vaccine		
Yes	246	80.80
No	58	19.10
Co-morbidities		
Yes	135	44.40
No	169	55.60
Acute exacerbation		
Yes	263	86.50
No	41	13.50
Duration during which subjects had acute exacerbation	42	16.30
Rainy season	186	70.70
Winter season	35	16.00
Both of rainy and winter season		

4.5 Environment Factors

Table 4.4 shows the environmental factors. Generally, (77.00%) typically cook at their residence, 38.50% used propane gas, wood or coal to keep warm, and 65.80% used charcoal, gas and wood for cooking. Eighty-eight subjects (28.90%) managed their household garbage by burning, 28.60% burned their household garbage 1-2 times a week, and 13.80% reported factories near the residence. One hundred seventy-seven subjects (58.20%) reported at least one forest fire near their house.

Table 4.4 Environmental Factors

Characteristics	n	%
Household typically cook		
Yes	234	77.00
No	70	23.00
Using gas, wood or coal to keep warm		
Yes	117	38.50
No	187	61.50
Types of fuel in kitchen		
Coal	4	1.30
Propane Gas	60	19.70
Wood	1	0.30
Coal, Propane Gas, Wood	200	65.80
Coal, Wood	39	12.80
Managing household waste		
Burned	88	28.90
Landfill	46	15.10
Municipality	170	55.90
Forest fires occurring near their residence		
Yes	177	58.20
No	127	41.80
Waste household burning (time/week)		
1	56	18.40
2	31	10.20
≥3	9	3.00
No	208	68.40
Factories located near their residence		
Yes	42	13.80
No	262	86.20

Table 4.5 shows the differentiation of the six general characteristics in the low peak PM_{10} period and compares them with the severity level of their COPD. There were no variables found to be statistically significantly different.

Table 4.5 Differentiation of General Characteristics of Severity of COPD in the Low Peak PM_{10} Period

Characteristics	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Sex						
Male	106	50.48	38	40.43	2.631	0.105
Female	104	49.52	56	59.57		
Age (years)						
≤50	9	4.29	5	5.32	2.516	0.642
51 – 60	26	12.38	14	14.89		
61 – 70	74	35.24	30	31.91		
71 – 80	75	35.71	38	40.43		
≥81	26	12.38	7	7.45		
Income (baht/month)						
≤5,000	171	8.14	78	80.98	0.105	0.746
≥5,001	39	91.86	16	17.02		
Marital status						
Married	141	67.14	69	73.40	1.192	0.275
Others	69	32.86	25	26.60		

Table 4.5 (continued)

Characteristics	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Family member (person)						
≤2	72	34.29	27	28.72	2.046	0.530
3-4	88	41.90	46	48.94		
5-6	38	18.10	14	14.89		
≥7	12	5.71	7	7.45		
Occupations						
Unemployed/retired	154	73.34	62	65.96	1.718	0.190
Other	56	26.21	32	34.04		

Note. Significant level at $\alpha = 0.05$

Table 4.6 shows the differentiation of the risk factors in the low peak PM_{10} period and compares them with the severity level of their COPD. There were no variables found to be statistically significantly different.

Table 4.6 Differentiation of the Risk Factors in the Low Peak PM_{10} Period

Characteristics	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Smoking						
Yes	8	3.81	1	1.06	1.704	0.19
No and quit smoking	202	96.19	93	98.94		
Household typically cook						
Yes	164	78.10	70	74.47	1.040	0.61
No	46	21.90	24	25.53		
Managing household						
Burning	57	27.14	31	32.98	1.119	0.57
Landfill	32	15.24	14	14.89		
Municipality	121	57.62	49	52.13		
Forest fires occurring near their residence						
Yes	121	57.62	56	59.57	0.102	0.75
No	89	42.38	38	40.43		
Waste household burning (time/week)						
1	39	61.90	17	51.52	1.063	0.59
2	19	30.16	12	36.36		
≥3	5	7.94	4	12.12		
Factories located near their residence						
Yes	20	9.52	22	23.40	10.507	0.01
No	190	90.48	72	76.60		

Note. Significant level at $\alpha = 0.05$

The differentiation of general characteristics in the high peak PM_{10} period and severity of COPD shows in table 4.7. The result of chi-square test shows none statistically significant different in 6 general characteristics.

Table 4.7 Differentiation of General Characteristics of Severity of COPD During High Peak PM_{10} Period

Characteristics	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Sex						
Male	38	45.24	106	48.18	0.211	0.646
Female	46	54.76	114	51.82		
Age (years)						
≤50	6	7.14	8	3.64	4.538	0.333
51 – 60	13	15.48	27	12.27		
61 – 70	25	29.76	79	35.91		
71 – 80	34	40.48	79	35.91		
≥81	6	7.14	27	12.27		
Income (baht/month)						
≤5,000	68	80.95	181	80.27	0.072	0.789
≥5,001	16	19.05	39	19.73		
Marital status						
Married	60	71.43	150	68.18	0.300	0.584
Others	24	28.57	70	31.82		

Table 4.7 (continued)

Characteristics	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Family member (person)						
≤2	31	36.90	68	30.91	3.352	0.341
3-4	36	42.86	98	44.55		
5-6	10	11.90	42	19.09		
≥7	7	8.33	12	5.45		
Occupations						
Unemployed/retired	61	72.62	155	70.46	1.380	0.710
Others	23	27.38	65	29.54		

Note. Significant level at $\alpha = 0.05$

Table 4.8 shows the differentiation of the risk factors in the high peak PM_{10} period and compares them with the severity level of their COPD. There was found smoking history to be statistically significantly different.

Table 4.8 Differentiation of the Risk Factors in the High Peak PM_{10} Period

Characteristics	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Smoking						
Yes	6	7.14	3	1.36	7.067	0.01*
No and quit smoking	78	92.86	217	98.64		
Household typically cook						
Yes	63	75	171	77.73	0.712	0.80
No	21	25	49	22.27		
Managing household						
Burning	24	28.57	64	29.10	1.979	0.37
Landfill	9	10.71	37	16.81		
Municipality	51	60.72	119	54.09		
Forest fires occurring near their residence						
Yes	50	59.92	127	57.73	0.081	0.78
No	34	40.48	93	42.27		
Waste household burning (time/week)						
1	19	65.52	37	55.22	0.925	0.67
2	8	27.59	23	34.34		
≥3	2	6.89	7	10.44		
Factories located near their residence						
Yes	9	10.71	33	15	0.938	0.33
No	75	89.29	187	85		

Note. *Significant level at $\alpha = 0.05$

Table 4.9 shows the outcome of severity of COPD in low-peak PM_{10} period and high-peak PM_{10} period. The study found 30.90% of subjects had severe stage in low-peak PM_{10} level and 72.40% in high-peak PM_{10} period.

Table 4.9 Outcome of Severity of COPD During the Two Periods

Periods	Severity of COPD				χ^2-test	p-value
	Mild		Severe			
	n	%	n	%		
Low-peak PM_{10} period	210	69.10	94	30.90	1.046	<0.001*
High-peak PM_{10} period	84	27.60	220	72.40		

Note. *Significant level at $\alpha = 0.05$

Univariate logistic regression analysis found that PM_{10} was found to have a statistically significant association with the severity of COPD with 5.85 times comparing between exposure and un-exposure periods. COPD had a greater severity stage during the dry season with 5.85 times compared to rainy season (95%CI=4.12-8.30).

Table 4.10 Univariate Logistic Regression Analysis of PM_{10} and the Severity of COPD

Factor	OR	95%CI	p-value
PM_{10}	5.85	4.12–8.30	<0.001*
Smoking	0.73	0.44-1.21	0.219
Factories	1.79	1.05-3.04	0.031*

Note. *Significant level at $\alpha = 0.05$

Multivariate logistic regression model found that PM_{10} was found to have a statistically significant association with the severity of COPD with 6.03 times comparing between exposure and un-exposure periods. COPD had a greater severity stage during the dry season with 6.03 times compared to rainy season (95%CI=4.21-8.63).

Table 4.11 Multivariable Logistic Regression Analysis of PM_{10} and the Severity of COPD

Factor	OR	95%CI	p-value
PM_{10}	6.03	4.21–8.63	<0.001*
Factories	2.02	1.13-3.61	0.018*

Note. *Significant level at $\alpha = 0.05$

CHAPTER 5

CONCLUSION AND DISCUSSION

This study aimed to describe the association between particulate matter 10 and severity of chronic obstructive pulmonary disease, northern Thailand between low peak PM_{10} period (August–October, 2011) and high peak PM_{10} period (March–May, 2012). Totally, 304 subjects from 5 hospitals in Chiang Rai province were recruited into the study and face-to-face interview. For the medical history data were collected from each hospital. For logistic regression analysis with 95% confident interval was used to determine the association of significant PM_{10} level with the severity of COPD.

5.1 Conclusion

This present study determined the association between particulate matter 10 and the severity of chronic obstructive pulmonary disease (COPD).The sample are 304 patients who recently diagnosed of COPD from 5 hospitals in Chiang Rai Province. The exposure and un-exposure groups were the same individual. For the information of PM_{10} concentrations were collected from secondary data source from The Pollution Control Department, Ministry of Natural Resources and Environment Thailand. It can be concluded from the present study that, increasing of PM_{10} level significantly associated with the severity of COPD. Awareness and motivation people to avoid and protect themself from exposure to PM_{10} are necessary for increasing quality of life among the COPD patients.

5.2 Research Methodology Discussion

The research was design as a retrospective cohort to determine the association between PM_{10} and severity of COPD in Chiang Rai Province, Thailand.

The study samples in this study were the COPD cases, and the the data were collected through interviews about the risk factors of COPD between 2 periods. Firstly, low peak PM_{10} period (August–October, 2011). Secondly, high peak PM_{10} period (March–May, 2012).

However, the study was a retrospective cohort study design. Therefore, some information could not collect completly. Especially, the assessment of severity of COPD. However, the assessment data of severity of COPD by mMRC scale were collected completely.

Another important factor affecting the outcomes of this study had a few recall-bias. Since the questions were asked after the event had occurred. Therefore, the data elicited from the subjects had not been affected by recall-bias. However, the researcher made necessary attempts to increase the confidential of statistic from the research methodology, by high validating the instruments and the procedures.

The assessment of severity of COPD in this study was used the methods as following;

1. The data from spirometer,
2. Medication using,
3. The frequency of admission or re-admission,
4. Assessment from signs and symptoms clinical history. This method was used the modified Medical Research Council Dyspnea scale (mMRC)for assessment of severity of COPD (Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease, 2011).

From the assessment of severity of COPD in previous paragraph in each data collection and found the problem below:

5.2.1 The Assessment of Severity of COPD by Using Spirometer

The spirometer measured the severity of the subjects with the most reliable because the value of the estimation was quite accurate. The problem in this method is spirometer was not use in every hospital because some hospitals lack of the equipment and criteria for using the spirometer were different in each hospital. Therefore, each time of the medical treatment of subjects with COPD, the data of the spirometers was not recorded in every treatment in each hospital.

5.2.2 Assessment from Medication Using

COPD is a chronic lung disease that characterized by chronic obstruction of lung airflow which interferes with normal breathing and is not fully reversible. Medications used in COPD patients were depend on the severity of the disease. Therefore, this method can use to evaluate the severity of COPD. The medication using data were collected from the medical records of subjects found that the level of medication using of subjects in each hospital with minor changes in the high peak PM_{10} period of the study and found that the majority of subjects received as same as treatment even the clinical symptoms of subjects were changed. Therefore, this method made it impossible to assess the severity of COPD in subjects.

5.2.3 The Frequency of Admission or Re-Admission

This method estimated the frequency of subjects who went to the hospital. According to the incoming treatment of subjects with COPD were not depending on the severity of the disease because the subjects returned to the hospitals by appointed from the physician only so if the subjects returned for treatment repeatedly it was not mean that the subjects had symptoms worse.

5.2.4 Assessment by Asking Signs and Symptoms Clinical History

This method used the modified Medical Research Council Dyspnea scale (mMRC) (Global Strategy for Diagnosis, Management and Prevention of Chronic Obstructive Pulmonary Disease, 2012) for assessment of severity of COPD. Dyspnea and respiratory distress syndrome are conditions that is often found in patients with

COPD, the patients had more symptoms on the severity of the disease increased the symptoms of the disease had a history of clinical symptoms.

The researchers found that the treatment of subjects with COPD in every first visit to the physician, The subjects were asked a history of dyspnea and daily life which the assessment was done in every hospital. Therefore, the researchers collected the data completely in every hospital and in every case of COPD subjects also assessed the severity of COPD by medical history.

The mMRC scale was used in this study which had 5 grades of evaluation from 0–4 grades. The mMRCscale were collected from 2 periods. Firstly, the low peak PM_{10} concentrations and another in high peak PM_{10} concentrations. Therefore, the value of mMRC scale were mean for data analysis between the association between PM_{10} and severity of COPD.

The data from this study had been collected from medical records and the database of PM_{10} concentrations that collected the secondary data so this study could reduce the bias during data collection.

In each data collection, the researcher interviewed the subjects in private and confidential room so that the information would be the truth. However, in each interview was supported by clinical staffs in COPD clinics.

Face-to-face interview was done by only the researcher and all questionnaires were checked for any missing questionnaire or mistake by 3 experts before leaving so this study could control missing value and content validity effectively.

The subjects were simple randomed from the lists of COPD patients in COPD clinics that had the medical record history and the patients had been entitled to free treatment from the government. For this reason these patients were appropriate the subjects.

The exposure and un-exposure groups were the same individual. The subjects were measured 2 periods. Therefore this study could control of individual factors.

5.3 Characteristics of Subjects

Three hundred and four subjects were patients who diagnosed of COPD from 5 hospitals in Chiang Rai Province. Additionally, all subjects were interviewed by the questionnaires in the private and confidential room.

Tan and Ng (2008), found that the proportion of COPD hospitalization by sex in Singapore was 94:16 (male to female). From the report of Center of Disease Control and Prevention (2009) found that death rate of COPD between male and female was 48.6:36.6. This proportion was difference from ours which shown 47:53. Because of smoking habits in Singapore are much higher in male than female.

In this study found COPD prevalence was the highest among male aged between 71-80 years 40.00%. However, we found the prevalence of COPD among the female aged between 51-60 and 61-70 years was higher than male in the same group. The another study of the Nippon COPD Epidemiology(NICE) Study of Japan found COPD prevalence was 3.50% in subjects aged 40-49 years and 24.40% in those aged >70 years (Fukuchi et al., 2004). Therefore, age is also an important determinant for COPD prevalence. The data from Japan was different from this study because of information obtained from Japan was reported.

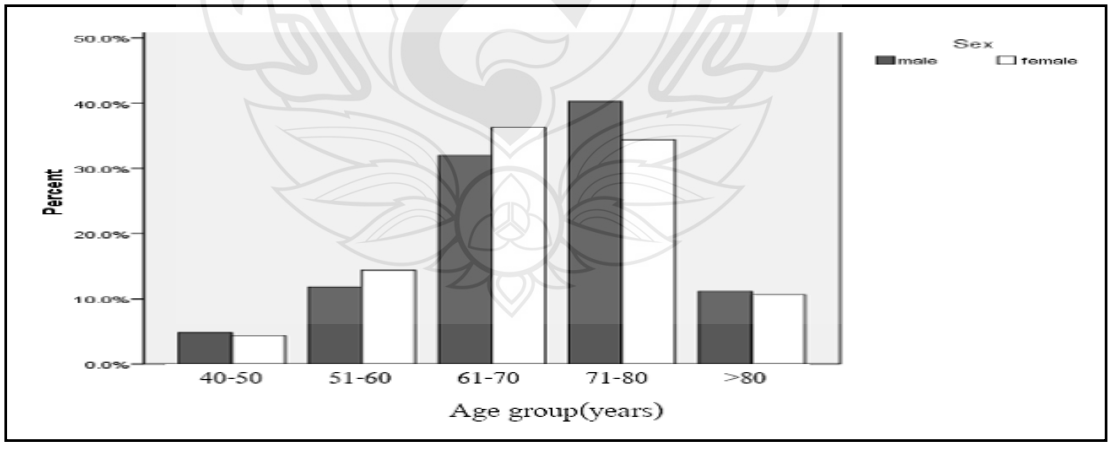


Figure 5.1 Prevalenceof COPD Among Adults Aged 40 and over, by Age Group and Sex, Chiang Rai, Thailand

5.4 Risk Behaviors and Medical History of Subjects

WHO estimates that in high-income countries, 73.00% of COPD mortality is related to smoking, with 40.00% related to smoking in nations of low and middle income (Torres et al., 2008). However, in our study found that the factors influenced the severity of COPD were cigarette smoking with 88.40%.But this value included both active smokers and ex-smokers so the value would be higher than other studies.

Since the outcome of COPD is always occurring when people reach old age, and most of the COPD cases had the comorbidity. This is might be the factors influencing to the severity of COPD. In our study, we found that many people had a comorbidity either Diabetes Melliteus (DM) or Hypertension (HT). The result also consisting with the study of Manen and Davis (2011) reported that over 50.00% of 1,145 patients with COPD had 1 to 2 comorbidities, 15.80% had 3 to 4 comorbidities, and 6.80% had 5 or more comorbid conditions.

Liu et al. (2007) found the significant association between the prevalence of COPD and exposing to biomass fuel for cooking in rural China. Our study also found that 4.00% were typically cook by charcoal, 1.00% used wood, and 39.00% used in both. For this study found non significant association between biomass fuel exposing and severity of COPD.

In our study found that the patients, who had diagnosed with COPD between 1 to 5 years, had been received influenza vaccine in every year. This is a good indicator of accessing health care system for people who need help by vaccination. Donaldson et al. (2012) found exacerbations in the cold seasons (November to February), of which 42.50% and 50.60% than in warm seasons (May to August), of which 31.40% and 45.40%. This could make conclusion that the weather is related to the exacerbation. Another study in 1996 that study of survival following hospital admission for acute exacerbations reported inhospital mortality rates of 11.00% and 1year mortality rates of 43.00% (Connors et al., 1996) Outcomes following acute exacerbations of severe chronic obstructive lung disease.

Cao, Ong and Eng (2006) found that the COPD patients who required frequent hospitalizations, the prevalence of influenza vaccination is less than 12.00%.

However, in this study found 80.80% had got influenza vaccination since the government has been supporting all the COPD patients theinfluenza vaccine.

5.5 Concentrations of PM_{10} in Chiang Rai Province, Thailand

In early of March every year, there was a big environmental problem in the northern of Thailand. The air pollution due to the forest fires covered many kilometers in this area.The study found that the concentrations of PM_{10}in March 2012 higher than others and foundthat the lowest PM_{10} concentrations in September 2011. Usanee and others measured the PM_{10} concentrations in Chiang Mai Province and found that PM_{10} concentrations in March 2005 higher than PM_{10} concentrations in August and September 2004 (Usanee, Taneyhill, Chewonarin, Chumram, Vinitketkumnuen & Tansuwanwong, 2007) as same as the study in Malaysia found the results that the amounts of PM_{10} in dry season are higher than those in rainy season in stations (Amanollahi, Abdullah, Pirasteh, Ramli & Rashidi, 2011). But a little different in the study of Jorge and others (2013) in Maxico found the most of the sampling sites, PM_{10} concentrations were lower during the summer season and increased gradually in the rainy season due to wind patterns. However, the winter season in Maxico had the highest PM_{10}concentrations as same as this study.

5.6 The Association between PM_{10} and Severity of COPD

This study found that the association between PM_{10} and severity of COPD statistically significant with 6.03 times comparing between exposure and un-exposure periods, our study had controlled the individual factors by collecting the data in two periods same individual subjects that nearly other studies.

Zhu and other (2013) had done a meta-analysis study from China to investigate the relationship between PM_{10} and hospitalizations and mortality rate of COPD. The search yielded 31 studies for the meta-analysis during the period from January 1, 2000 to October 31, 2011. The results had shown a 10 $\mu g/m^3$ increase in

PM_{10} was associated with a 2.70% (95%CI = 1.90%-3.60%) increase in COPD hospitalizations with an OR of 1.027 (95%CI: 1.019-1.036), and a 1.10% (95%CI: 0.80%-1.40%) increase in COPD mortality with an OR of 1.011 (95%CI: 1.008-1.014).

Harre and others (1997) studied time series to investigate the relationship between air pollution levels and respiratory symptoms and peak expiratory flow rate (PEFR) in subjects with chronic obstructive pulmonary disease (COPD) living in Christchurch, New Zealand. This study found a rise in the PM_{10} concentration equivalent to the interquartile range was associated with an increase in night time chest symptoms (relative risk 1.38, 95% CI 1.07 to 1.78).

5.7 Limitation of the Study

The study was retrospective study design therefore we could not collect all of factors completely and classified the severity of COPD clearly. In some hospital had not the instruments for assessment the severity of COPD so this study had to collect the data from clinical symptoms in medical record cards and self-interview instead then assessed the severity of COPD by Modified Medical Research Council Dyspneoa Scale; mMRC scale (Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease, 2011). and found that the PM_{10} concentration level changing in different season especially high in dry season (exposure period) had associated with the severity of COPD in this study.

The standard assessment of severity of COPD was different in each hospital but the best assessment of COPD is spirometry values. However, this assessment did not use in some hospitals. Therefore, some hospitals used other assessment instead.

The information of PM_{10} concentrations level were collected from one site at Muang district in two periods that were used for secondary data. Therefore, the research had randomed the hospitals that near the PM_{10} stations but it could not cover for 5 hospitals.PM_{10} collection in these periods had mean of PM_{10} concentrations level lower than previous years because of rain.

5.8 Recommendations

5.8.1 Recommendations for Government or Authorities

The information in this study found that PM_{10} concentrations level had been affected statistically significant the severity of COPD. Therefore, the measures of government should take responsibility to reduce the concentrations of PM_{10} level in the atmosphere, especially during the forest fires occurred in dry season. Through the cooperation between local administration and the people in the area.

For the problem of smoke with the high PM_{10} concentrations is partly a result of forest fires in the north of Thailand and neighboring Myanmar and Laos. From this reason, Thailand and neighboring should be collaborative solving this problem.

5.8.2 Recommendations for Further Studies

There are many conditions that need to be investigated to improve awareness of PM_{10} concentrations and to affect respiratory health in the north of Thailand. More studies also need to be done in other province, especially the provinces in the north of Thailand that had been effected by PM_{10} concentrations level from the forest fires such as Chiang Mai, Phayao and Lampang Province. The criteria of assessing the severity of COPD should be the same standard in each hospital. This study should monitoring air quality by the equipment with high specific to obtain the information that is accurate before analyzing the data.

5.8.3 Recommendations for Health Care and Setting

From the study we suggest that hospitals in northern provinces should have the standard guideline and line of management of COPD patients in every emergency rooms. So that the clinical staffs in each hospital will do the same standard treatment in every condition of COPD such as acute exacerbation or respiratory distress syndrome. By doing this will increase the efficacy in treatment COPD patients and will reduced the morbidity and mortality. Not to forget that every COPD patients should have the knowledge about the disease in aspect of prevention of exacerbation as well. Furthermore, The hospitals should set the COPD patients care team to

improve the efficacy COPD treatment for reducing the morbidity and mortality in COPD patients.

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APPENDICES

APPENDIX A

THE RESEARCH ETHICAL APPROVAL DOCUMENT

เอกสารเลขที่ ๒๕/๒๕๕๕

หนังสือยืนยันการยกเว้นการรับรอง
คณะกรรมการจริยธรรมการวิจัยในมนุษย์ มหาวิทยาลัยแม่ฟ้าหลวง
ขอรับรองว่า

โครงการวิจัย : ความสัมพันธ์ระหว่างอนุภาคฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรัง
(The Association Between Particulate Matter 10 And Severity of Chronic Obstructive Pulmonary Disease, Northern Thailand)

โครงการเลขที่ : REH-๕๕๐๒๕

ชื่อหัวหน้าโครงการ : นางสาวปิยะวรรณ ประมวลทรัพย์

สังกัด : สำนักวิชาวิทยาศาสตร์สุขภาพ

เป็นโครงการวิจัยที่เข้าข่ายยกเว้น (Research with Exemption Determination) ที่ไม่ขัดต่อหลักจริยธรรมสากลตามคำปฏิญญาเฮลซิงกิ (The Declaration of Helsinki) และแนวทางจริยธรรมการวิจัยในคนแห่งชาติ พ.ศ. ๒๕๔๕

จึงเห็นสมควรให้ดำเนินการวิจัยในขอบข่ายของโครงการที่เสนอต่อคณะกรรมการจริยธรรมการวิจัยในมนุษย์ มหาวิทยาลัยแม่ฟ้าหลวงได้ ณ วันที่ ๘ เดือน มิถุนายน พ.ศ. ๒๕๕๕

ลงชื่อ.....

(รองศาสตราจารย์ ดร.ชยาพร วัฒนศิริ)
ประธานคณะกรรมการจริยธรรมการวิจัยในมนุษย์
มหาวิทยาลัยแม่ฟ้าหลวง

APPINDIX B

CONSENT FROM

หนังสือยินยอมเข้าร่วมโครงการวิจัย

วันที่.....เดือน..... พ.ศ.....

ข้าพเจ้า (นาย/นาง/นางสาว)..... อายุ.....ปี

อยู่บ้านเลขที่..... หมู่ที่.....ถนน.....ตำบล.....อำเภอ.....จังหวัด

..... รหัสไปรษณีย์.....

ขอทำหนังสือแสดงความยินยอมเข้าร่วมโครงการวิจัยเพื่อเป็นหลักฐานแสดงว่า

1. ข้าพเจ้ายินยอมเข้าร่วมโครงการวิจัยของ นางสาวปิยะวรรณ ประมวลทรัพย์

เรื่อง ความสัมพันธ์ระหว่างอนุภาคฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรังด้วยความสมัครใจ โดยมิได้มีการบังคับหลอกลวงแต่ประการใด และพร้อมจะให้ความร่วมมือในการวิจัย

2. ข้าพเจ้าได้รับการอธิบายและตอบข้อสงสัยจากผู้วิจัยเกี่ยวกับวัตถุประสงค์การวิจัย วิธีการวิจัย ความปลอดภัย อาการ หรืออันตรายที่อาจเกิดขึ้น รวมทั้งประโยชน์ที่จะได้รับจากการวิจัย โดยละเอียดแล้วตามเอกสารชี้แจงผู้เข้าร่วมการวิจัยแนบท้าย

3. ข้าพเจ้าได้รับการรับรองจากผู้วิจัยว่าจะเก็บข้อมูลส่วนตัวของข้าพเจ้าเป็นความลับ จะเปิดเผยได้เฉพาะในรูปแบบของการสรุปผลการวิจัยเท่านั้น

4. ข้าพเจ้าได้รับทราบจากผู้วิจัยแล้วว่า หากเกิดอันตรายใดๆ จากการวิจัยผู้วิจัยจะรับผิดชอบค่ารักษาพยาบาลที่เป็นผลสืบเนื่องจากการวิจัยนี้

5. ข้าพเจ้าได้รับทราบว่า ข้าพเจ้ามีสิทธิที่จะถอนตัวออกจากการวิจัยครั้งนี้เมื่อใดก็ได้ โดยไม่มีผลกระทบใดๆ ต่อการรักษาพยาบาลตามสิทธิ์ที่ข้าพเจ้าควรได้รับ

ข้าพเจ้าได้อ่านและเข้าใจข้อความตามหนังสือนี้แล้ว จึงได้ลงลายมือชื่อไว้เป็นสำคัญ พร้อมกับหัวหน้าโครงการวิจัยและพยาน

ลงชื่อ..... ผู้ยินยอม/ผู้ปกครอง

(.....)

ลงชื่อ..... หัวหน้าโครงการ

(.....)

ลงชื่อ..... พยาน

(.....)

ลงชื่อ..... พยาน

(.....)

APPINDIX C

PARTICIPANT INFORMATION SHEET

เอกสารชี้แจงผู้เข้าร่วมการวิจัย

ในเอกสารนี้อาจมีข้อความที่ท่านอ่านแล้วยังไม่เข้าใจ โปรดสอบถามหัวหน้าโครงการวิจัย หรือผู้แทนให้ช่วยอธิบายจนกว่าจะเข้าใจดี

ชื่อโครงการวิจัย ความสัมพันธ์ระหว่างอนุภาคฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรัง

ชื่อผู้วิจัย นางสาวปิยะวรรณ ประมวลทรัพย์

วัตถุประสงค์ การศึกษานี้มีวัตถุประสงค์เพื่อหาความสัมพันธ์ระหว่างอนุภาค ฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรังในผู้ป่วยที่มารับการรักษาที่โรงพยาบาลในจังหวัดเชียงราย

วิธีการวิจัย แบบ Retrospective Cohort Study Design

สถานที่วิจัย โรงพยาบาลแม่จัน, โรงพยาบาลแม่สาย, โรงพยาบาลเชียงแสน, โรงพยาบาลสมเด็จพระญาณสังวร, โรงพยาบาลพญาเม็งราย จังหวัดเชียงราย

ระยะเวลาในการทำงานวิจัย 12 เดือน

โครงการวิจัยนี้ทำขึ้นเพื่อศึกษาความสัมพันธ์ความสัมพันธ์ระหว่างอนุภาค ฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรังใน ผู้ป่วยที่มารับการรักษาที่โรงพยาบาลในจังหวัด เชียงราย ซึ่งมีประ โยชน์ที่คาดว่าจะ ได้รับคือ

1. ทำให้ทราบข้อมูลเกี่ยวกับความสัมพันธ์ระหว่างอนุภาค ฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรัง
2. เพื่อนำข้อมูลที่ได้มาใช้พัฒนาแนวทางในการส่งเสริมสุขภาพของประชากรที่อาศัยในพื้นที่เสี่ยงต่อมลพิษ

ผู้เข้าร่วมงานวิจัยนี้ได้รับเชิญให้เข้าร่วมงานวิจัยเนื่องจากท่านได้รับการวินิจฉัยจากแพทย์ว่าเป็นโรคปอดอุดกั้นเรื้อรัง ซึ่งมีคุณสมบัติตามเกณฑ์ที่ ผู้วิจัยกำหนด และการวิจัยนี้ทำขึ้นเพื่อให้ได้ทราบข้อมูลของความสัมพันธ์ระหว่างอนุภาค ฝุ่นเล็กกว่า 10 ไมครอนกับความรุนแรงของโรคปอดอุดกั้นเรื้อรัง ในผู้ป่วยที่มารับการรักษาที่โรงพยาบาลในจังหวัดเชียงราย

โครงการนี้เป็นศึกษาวิจัยในระดับมหาบัณฑิตศึกษา สาขาวิชาสาธารณสุขศาสตร์(หลักสูตรนานาชาติ) มหาวิทยาลัยแม่ฟ้าหลวง จะรับผู้เข้ ร่วมวิจัยทั้งสิ้น 304 ราย โดยจะใช้ระยะเวลาที่จะทำการวิจัยทั้งสิ้นประมาณ 12 เดือน

โดยมีขั้นตอนการวิจัยดังนี้

1. ขั้นเตรียมการ ผู้วิจัยคัดเลือกผู้เข้าร่วมวิจัยที่มีคุณสมบัติตามเกณฑ์ที่กำหนด จากนั้นผู้เข้าร่วมวิจัยจะได้รับเอกสารดังนี้ ,เอกสารชี้แจงผู้เข้าร่วมการวิจัย, หนังสือเจตนายินยอมเข้าร่วมการวิจัย, แบบสอบถามเกี่ยวกับข้อมูลส่วนตัวและประวัติทางสุขภาพ
2. ขั้นตอนการเก็บข้อมูล ผู้เข้าร่วมการวิจัยจะได้รับแบบสอบถาม ผู้วิจัยจะอธิบายถึงขั้นตอนการสัมภาษณ์แก่ผู้เข้าร่วมการวิจัยในสถานที่มิดชิดและมีความเป็นส่วนตัวจนเข้าใจอย่างละเอียดโดยมีรายละเอียดในการสัมภาษณ์ดังนี้
   - 2.1 แบบสอบถามส่วนที่ 1 ประกอบด้วย ข้อมูลส่วนตัว ลักษณะครัวเรือนและสังคมเศรษฐกิจ
   - 2.2 แบบสอบถามส่วนที่ 2 ประกอบด้วย ข้อมูลเกี่ยวกับพฤติกรรมและปัจจัยเสี่ยง
   - 2.3 แบบสอบถามส่วนที่ 3 ประกอบด้วย ข้อมูลเกี่ยวกับสิ่งแวดล้อม
   - 2.4 แบบสอบถามส่วนที่ 4 ประกอบด้วย ข้อมูลเกี่ยวกับประวัติการรักษาทางการแพทย์

ความเสี่ยงในอาจเกิดขึ้นเมื่อเข้าร่วมการวิจัย ระหว่างการสัมภาษณ์ท่านอาจเกิดอาการเหนื่อยหอบอันเนื่องมาจากอาการของโรค เมื่อพักแล้วอาการยังไม่ทุเลาลง ส่งผลให้ผู้วิจัยไม่สามารถถูกสัมภาษณ์ต่อไปได้ ผู้วิจัยจะยุติการสัมภาษณ์ทันที และจะส่งผู้เข้าร่วมการวิจัยปรึกษาแพทย์ผู้เชี่ยวชาญต่อไป ข้อมูลทั้งหมดที่สัมภาษณ์จะไม่ถูกเปิดเผยต่อสาธารณะเป็นรายบุคคลแต่จะรายงานผลการวิจัยเป็นข้อมูลส่วนรวม

หากเกิดผลข้างเคียงที่ไม่พึงประสงค์จากการวิจัย ท่านจะได้รับการช่วยเหลือจากผู้วิจัยและส่งปรึกษาแพทย์ผู้เชี่ยวชาญต่อไป หากมีข้อสงสัยที่จะสอบถามเกี่ยวข้องกับการวิจัยสามารถติดต่อผู้วิจัย (นางสาวปิยะวรรณ ประมวลทรัพย์)ได้ตลอดเวลาที่เบอร์โทรศัพท์ของผู้วิจัย 08-9931-2739

ผู้เข้าร่วมการวิจัยมีสิทธิ์ถอนตัวออกจากโครงการวิจัยเมื่อก็ได้ โดยไม่ต้องแจ้งให้ทราบล่วงหน้า และการไม่เข้าร่วมการการวิจัยหรือถอนตัวออกจากโครงการวิจัยนี้จะไม่มีผลกระทบต่อการบริการและการรักษาที่สมควรได้รับแต่ประการใด

หากท่านได้รับการปฏิบัติที่ไม่ตรงตามที่ระบุไว้ในเอกสารชี้แจงนี้ท่านจะสามารถแจ้งให้ประธานคณะกรรมการจริยธรรมฯ ทราบได้ที่ส่วนบริการงานวิจัย มหาวิทยาลัยแม่ฟ้าหลวง 333 หมู่ 1 ต.ท่าสุด อ.เมือง จ.เชียงราย เบอร์โทร 0-5391-6387

ข้าพเจ้าได้อ่านรายละเอียดในเอกสารนี้ครบถ้วนแล้ว

ลงชื่อ.....

(.....)

วันที่.....

APPINDIX D

DATA COLLECTION FORM

ENGLISH

Direction Please mark ✓ in the blank ( ) that is your real situation

Part 1 General information history

1. Gender

( ) male ( ) female

2. Age _____ years

3. Education

( ) Primary school ( ) High school

( ) Diploma ( ) Bachelor's degree

( ) Higher than bachelor's degree ( ) 6. No

4. Occupations

( ) Unemployed/retired ( ) Housewife (female)

( ) Unskilled worker ( ) Skilled worker

( ) Business ( ) Government/private officer

( ) Others

5. Income

( ) <5,000 Baht/ month ( ) 5,001-10,000 Baht/ month

( ) 15,001-20,000 Baht/ month ( ) 10,001-15,000 Baht/ month

( ) >20,000 Baht/ month

6. Hometown

6.1 If you immigrated, how many years did you live here?

( ) Yes_____ years ( ) No

7. Religion _____

8. Race

( ) Thai

( ) Others

9. Marital Status

( ) Single ( ) Married

( ) Widowed ( ) Divorced

10. Member in households_____

Part 2 Medical and smoking history

1. Smoking

( ) No

( ) Yes ( ) quit ( ) smoking

1.1 How many years did you smoke ? _____ years

1.2 Type of smoking

( ) Beedi

( ) Cigaratte

( ) Others

1.3 How many cigarettes do you currently smoke each day ?

_____ cigarettes

(If you are an ex-smoker, how many did you smoke each day?)

1.4 How old were you when you started smoking? _____ Year old

2. Passive smoking

( ) No

( ) Yes _____hours/day

For_____ years_____ months

3. Did you get passive smoking for household typically cook?

( ) No

( ) Yes _____hours/day

For_____ years_____ months

4. Do you use fuel to keep warm?

( ) No ( ) Yes _____months

5. Did you kitchen connect to your house ?

( ) No

( ) Yes

6. What types of flues did you use in the kitchen?

( ) Coal ( ) Propane Gas

( ) Wood ( ) Animal dung ( ) Others

7. How did you manage the waste in your household?

( ) Burning _____ times/week

( ) Landfill in house area

( ) Municipality

Part 3 Environment Characteristics

1. Are there the industries as follow near your residence not far more than 50 km ?

( ) No

( ) Yes ( ) Coal mine

( ) Cotton mill

( ) Tobacco factory

( ) Rice mill

( ) Sawmill

( ) Cement plant

( ) Others

2. Are there the agricultural burning near your residence?

( ) Yes ( ) smell ( ) No smell

( ) No

3. Are there the forest fires near your residence?

( ) Yes ( ) smell ( ) No smell

( ) No

4. Are there the waste burning near your residence?

( ) Yes ( ) smell ( ) No smell

( ) No

5. How long the distance between your residence and main road?

( ) < 50 meters ( ) 50 – 100 meters

( ) 101 -150 meters ( ) > 150 meters

Part 4 Medical history

1. When did you had a diagnosis of COPD ? _____/_____/_____
2. Influenza vaccine

( ) No

( ) Yes

3. Body Mass Index (BMI)

3.1What is your weight in kilograms ? _____kg

3.2 What is your height in meters ? _____meters

4. Did you get influenza ?

( ) No

( ) Yes When____/_____/____

5. Co-morbidities ?

( ) Yes ( ) DM ( ) Hypertension

( ) Thalasemia ( ) G6PD

( ) Lung cancer

( ) No

6. Complications

( ) Yes ( )Acute exacerbation

When_____/_____/_____

( ) Respiratory failure When_____/_____/_____

( ) No

7. Medical history

Items	1 August to 31 October 2011	1 March to 31 May 2012
1. Number of admission	.....	.....
2. On O_2	.....	.....
3. β_2–agonist	.....	.....
4. Corticosteroids	.....	.....
5. Aminophylline	.....	.....
6. On ventilator	.....	.....
7. mMRC score	.....	.....
7.1 Short of breath when	.....	.....
hardly exercise.	.....	.....
7.2 Short of breath when	.....	.....
walking or climbing stairs.	.....	.....
7.3 Slowly walking than	.....	.....
others in the same age	.....	.....
because of short of breath.	.....	.....
7.4 Short of breath when	.....	.....
walking more than 100	.....	.....
meters.	.....	.....
7.5 Short of breath when	.....	.....
doing social activities	.....	.....
such as talking, visiting	.....	.....
friends/relatives.	.....	.....

APPINDIX D

LIST OF EXTERNAL EXPERTS

List of the external experts who validate the questionnaires

1. Associate Professor Dr. Kulaya Narksawat.
   Department of Epidemiology, Faculty of Public Health, Mahidol University, Bangkok, Thailand
2. Associate Professor Dr. Saipin Kasemkitwattana
   School of Nursing, Mae Fah Luang University, Chiang Rai, Thailand.
3. Dr. Haluataya Kusayanunt Department of Internal Medicine, Buddhachinaraj Hospital, Phitsanulok, Thailand.

CURRICULUM VITAE

CURRICULUM VITAE

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WORK EXPERIENCE

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