COMPARISON OF DAILY MORTALITY EFFECTS OF OUTDOOR (AMBIENT) AIR POLLUTION BETWEEN 1999-2001 AND 2006-2008 IN BANGKOK :TIME STRATIFIED CASE-CROSSOVER STUDY

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การเปรียบเทียบการเสียชีวิตในเขตพื้นที่กรุงเทพมหานคร จากผลกระทบของมลพิษทางอากาศ ในช่วงปี พ.ศ. ๒๕๔๒-๒๕๔๔ กับปี พ.ศ. ๒๕๔๕-๒๕๕๑ ด้วยรูปแบบการศึกษา Time Stratified Case Crossover

นายไพโรจน์ จันทรมณี

วิทยานิพนธ์นี้เป็นส่วนหนึ่งของการศึกษาตามหลักสูตรปริญญาสาธารณสุขศาสตรดุษฎีบัณฑิต
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Thesis Title COMPARISON OF DAILY MORTALITY EFFECTS OF OUTDOOR (AMBIENT) AIR POLLUTION BETWEEN 1999-2001 AND 2006-2008 IN BANGKOK :TIME STRATIFIED CASE-CROSSOVER STUDY

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ไพโรจน์ จันทรมณี: การเปรียบเทียบการเสียชีวิตในเขตพื้นที่กรุงเทพมหานคร จากผลกระทบของมลพิษทางอากาศในช่วงปี พ.ศ. ๒๕๔๒-๒๕๔๔ กับปี พ.ศ. ๒๕๔๙-๒๕๕๑ ด้วยรูปแบบการศึกษา TIME STRATIFIED CASE CROSSOVER (COMPARISON OF DAILY MORTALITY EFFECTS OF OUTDOOR (AMBIENT) AIR POLLUTION BETWEEN 1999-2001 AND 2006-2008 IN BANGKOK :TIME STRATIFIED CASE-CROSSOVER STUDY) อ.ที่ปรึกษาวิทยานิพนธ์หลัก: ศ.นพ.สุรศักดิ์ ฐานีพานิชสกุล, 186 หน้า.

รูปแบบการศึกษาด้วยวิธีการศึกษาไขว้ ระหว่างกลุ่มที่ศึกษาและ กลุ่มควบคุม แบบ Case-Crossover (CCO) แสดงผลการศึกษาให้เห็นว่า มลพิษทางอากาศมีความสัมพันธ์กับการเสียชีวิตด้วยโรคระบบทางเดินหายใจ และโรคระบบหมุนเวียนโลหิต ซึ่งประเทศไทย ยังไม่มีการนำเอาวิธีการของ Case-Crossover มาใช้กับระบบเฝ้าระวังเพื่อติดตามผลกระที่เกิดจากมลพิษทางอากาศกับการเสียชีวิต การศึกษานี้มีวัตถุประสงค์ เพื่อศึกษา 1.) การเสียชีวิตที่สัมพันธ์กับมลพิษทางอากาศในเขตพื้นที่กรุงเทพมหานคร 2.) เปรียบเทียบผลกระทบต่อสุขภาพที่เกิดจากมลพิษทางอากาศในช่วงปี พ.ศ. 2542-2544 และ ปี พ.ศ. 2549-2551 3.) เปรียบเทียบผลการศึกษาของ CCO ในครั้งนี้ กับผลการศึกษาด้วยวิธีการแบบ Time series design การศึกษาครั้งนี้ใช้วิธีการเลือกกลุ่มควบคุมแบบ Time-stratified ซึ่งเป็นการแบ่งช่วงเวลาโดยใช้วันของสัปดาห์เป็นเดียวกัน และเลือกเฉพาะเดือนเดียวกันกับกลุ่มศึกษา ตัวแปรที่เป็นปัจจัยเสริมเช่น อุณหภูมิ (Lag0) และ ความชื้น (Lag1) ถูกนำมาเข้าสมการเพื่อควบคุมปัจจัยดังกล่าว การศึกษาครั้งนี้เป็นการศึกษาผลกระทบระยะสั้น ของ ไนโตรเจนไดออกไซด์ (NO_2) โอโซน (O_3) คาร์บอนมอนอกไซด์ (CO) ซัลเฟอร์ไดออกโซด์ (SO_2) และ ฝุ่นละอองขนาดเล็กกว่า 10 ไมครอน (PM_{10}) ผลการศึกษาพบว่า ในช่วงเวลาที่ศึกษามีผู้เสียชีวิตที่ไม่ใช่เกิดจากอุบัติเหตุ จานวน 228,103 ราย จากการศึกษาพบว่า PM_{10} NO_2 และ CO มีความสัมพันธ์เชิงบวกกับการเสียชีวิตโดยธรรมชาติ adjusted OR 1.008 (95 %CI 1.004-1.012) adjusted OR 1.002 (95 %CI 1.001-1.004) และ adjusted OR 1.008 (95 %CI 1.003-1.013) ตามลาดับ CO มีความแต่ต่างระหว่าง 2 ช่วงปีการศึกษาอย่างมีนัยสำคัญทางสถิติ (P-value 0.01) เมื่อทำการควบคุมปัจจัยเสริมในสมการ พบว่า PM_{10} CO NO_2 และ O_3(ค่าเฉลี่ย 8 ชั่วโมง) มีความสัมพันธ์เชิงบวกกับการเสียชีวิตด้วยระบบไหลเวียนโลหิตล้มเหลวอย่างมีนัยสำคัญทางสถิติ แต่ไม่พบนัยสำคัญกับระบบทางเดินหายใจ ปัญหามลพิษทางอากาศในช่วงปี พ.ศ. 2549-2551 มีผลกระทบมากกว่าปี พ.ศ. 2542-2544 และมีความสัมพันธ์กับการเสียชีวิตอย่างมีนัยสำคัญทางสถิติ เมื่อเปรียบเทียบผลการศึกษาระหว่าง Time series กับ CCO ผลปรากฏว่ามีความสอดคล้องกัน เนื่องจากพบความสัมพันธ์ของมลพิษทางอากาศกับการเสียชีวิตในเขตพื้นที่กรุงเทพมหานครเช่นเดียวกัน จึงยังคงไม่สามารถระบุให้ชัดเจนได้ว่าวิธีการใดเหมาะสมกว่ากัน แต่เพื่อเป็นการตรวจสอบความถูกต้องของข้อมูล ควรเลือกใช้ทั้งสองวิธี เพราะทำให้การประมาณการมีความแม่นยำมากยิ่งขึ้น

สาขาวิชา สาธารณสุขศาสตร์ ลายมือชื่อนิสิต .....

ปีการศึกษา 2554 ลายมือชื่อ อ.ที่ปรึกษาวิทยานิพนธ์หลัก .....

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KEYWORDS: AMBIENT AIR POLLUTION / MORTALITY / CASE-CROSSOVER DESIGN / BANGKOK

PHAIROJ JANTARAMANEE: COMPARISON OF DAILY MORTALITY EFFECTS OF OUTDOOR (AMBIENT) AIR POLLUTION BETWEEN 1999-2001 AND 2006-2008 IN BANGKOK :TIME STRATIFIED CASE-CROSSOVER STUDY. ADVISOR: PROF. SURASAK TANEEPANICHSKUL, M.D. M.MED. CO-ADVISOR: ROBERT SEDGWICK CHAPMAN, M.D, M.P.H , 186 pp.

Several case-crossover studies have shown association between ambient air pollution and cardiovascular or respiratory mortality. Thailand has not been done Epidemiological Surveillance System by case-crossover design to continuous monitoring air pollution and mortality. This study aimed 1) to examine the association between ambient air pollution and daily mortality in Bangkok, 2) to compare health effect of air pollution among years 1999-2001 and 2006-2008. 3) to compare the applicability of case-crossover design in this study with other design as time series study. Time-stratified case-crossover analysis was used to evaluate the short term effects of ambient air pollution (Nitrogen dioxide (NO_2), Ozone (O_3), Carbon monoxide (CO), Sulfur dioxide (SO_2) and Particulate matter 10 $\mu g/m^3$ (PM_{10})) on natural, cardiovascular and respiratory mortality. Controls period were selected by matched days of the week in the same month. Time dependent variables such as temperature today (lag0) and humidity yesterday (lag1) for non-external mortality model, circulatory mortality and respiratory mortality we used moving average of humidity as adjustor in the model of conditional logistic regression analyses. 228,103 non-external deaths in study periods were included. The findings for non-external mortality showed statistically significant associations with PM_{10} CO and NO_2 in adjusted models, adjusted OR 1.008 (95 %CI 1.004-1.012) adjusted OR 1.002 (95 %CI 1.001-1.004) and adjusted OR 1.008 (95 %CI 1.003-1.013) respectively. Carbon monoxide has a difference effect between 2 periods with statistically significant (P-value 0.01), specially for period two has effect on non-external mortality than period one. Ozone level was significant increasing. PM_{10} CO NO_2 and O_3 (8 hours) have effected on circulatory mortality in adjusted model but respiratory mortality did not. This study showed precise air pollutions in 2006-2008 were more problems than 1999-2001 and statistic significant association. The both methods, time series study and case-crossover study provide generally similar evidence. The using both methods to estimated adverse health effect from air pollution are increasing the strong estimation models.

Field of the Study Public Health Student's Signature .....

Academic Year 2011 Advisor's Signature .....

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CONTENTS

Page

Page

LIST OF TABLES

Table Page

Table Page

Table Page

Table Page

LIST OF FIGURES

LIST OF ABBREVIATIONS

µ	Micrometer/micron
µg	Microgram
ANOVA	Analysis of Variance
ARIMA	Autoregressive integrated moving average models
ASSBI	Adjusted semi-symmetric bi-directional method
AQI	Air Quality Index
C°	Celsius (centigrade)
CCO	Case-crossover
CH_4	Hydrocarbons
CI	Confidential Interval
CLR	Conditional logistic regression
CO	Carbon monoxide
CO_2	Carbon Dioxide
COPD	Chronic obstructive pulmonary disease
C-R	Concentration-response
CVD	Cardiovascular diseases
df	Degree of freedom
FEV	Forced expiratory volume
FSBI	Full stratum bi-directional
FVC	Forced vital capacity
GAMs	Generalized additive models
H_2CO_3	carbonic acid
HCO_3^-	Bicarbonate ions
hr	Hour
ICD-10	The International Classification of Diseases, tenth vision
lag0	Air pollution level today
lag1	Air pollution level yesterday
JIA	Juvenile idiopathic arthritis
m^3	Cubic meter
MH	Mantel-Haenszel
MI	Myocardial Infarction

mm.Hg	The millimetre of mercury
MOPH	Ministry of Public Health
MV01	Moving average today and yesterday
nm	Nanometer
NO_2	Nitrogen dioxides
O_3	Ozone
OR	Odds Ratio
Pa	Pascal
PAI-1	Plasminogen activator inhibitor-1
PAPA	The study of Public Health and Air Pollution in Asia
PCD	Pollution control Department
PM	Particulate Matter in air
PM_{10}	Particulate Matter with a 50% cut-off aerodynamic diameter of 10 micron
Po_2	Prtial oxygen pressure
ppb	Part per billion
ppm	Part per million
rH	Relative Humidity
RR	Relative Risk
SBI	Symmetric bi-directional
SD	Standard Deviation
Sig	Significance
SO_2	Sulfur dioxide
sP-selectine	Soluble P-selectine
SSBI	Semi-symmetric bi-directional
t	t-test
TS	Time-stratified
TEOM	Tapered Element Oscillating Microbalance
TS-CCO	Time stratified case-crossover
UV	Ultraviolet
UFPs	Ultrafine particles
VA	Verbal Autopsy
WHO	World Health Organization

CHAPTER I

INTRODUCTION

1.1 Background and Rationale

Air is essential for the survival of all the living organisms on earth that include humanity. Air supplies us with oxygen which is essential for our bodies to live. Air is made up of 78 percent nitrogen 21 percent oxygen, 0.93 percent argon, 0.038 percent carbon dioxide, and trace amounts of other gases. The clean Air is decrease because of consumption has largely been ignored in the trade and environment debate, despite the fact that it accounts for a large portion of total emissions. Human activities can release chemicals and particulates into the atmosphere, some of which can cause problems for humans, animals, plants, and environment. A substance in the air that causes harm to humans and the environment we know as Air pollutant (Frederik, 1994).

Air pollution is a serious threat to public health and the environment. Air pollutants can be in the form of solid particles, liquid droplets, or gases. In addition, they may be natural or man-made. Among the major sources of pollution are power and heat generation, the burning of solid wastes, industrial processes, transportation and especially household such as cooking, candle burning and tobacco smoking. There are several types of air pollution which are commonly discussed. The major types of pollutants are carbon monoxide (CO), hydrocarbons (CH_4), nitrogen dioxides (NO_2), particulate matter (PM), sulfur dioxide (SO_2), Ozone (O_3) and photochemical oxidants (Stephen et al., 1999).

Ambient air pollution has the effect to physical, psychology, environments and economic problems. Air pollutants are both indoor and outdoor, and can cause morbidity and mortality. In the studies between 1990 and 2010 they found many kinds of cardiovascular diseases and respiratory diseases that occurred because of air pollutants effect, such as angina and ischemic heart disease, acute myocardial infarction, Asthma, Cancer, Cerebrovascular, Conduction disorders and dysrhythmia, Congestive heart failure, Chronic obstructive pulmonary disease (COPD), Diabetes,

Hypertension, Atherosclerosis, Bronchiolitis, Cardiac anomalies, Cardiac arrest, Gastroenteric Disorders, Ischemic stroke, Hyperlipidemia, Other kidney or renal disease, Otitis Media, Postneonatal (Eduardo et al., 2010).

Epidemiological studies of air pollutants are essential in prevention and control of public health problems. There are several kinds of research design to estimate air pollutant and mortality. The two designs used most to evaluate effects of short-term air pollution exposures are the time series design and the case-crossover (CCO) design. These short-term effects include daily mortality and daily hospital admissions for respiratory and cardiovascular diseases. In time series, daily pollutant levels and daily counts of adverse health effects (e.g., mortality) are correlated to see whether these counts are associated with pollutant levels. Several time series studies, but as yet no CCO studies, have been done in Bangkok. Poisson regression models with flexible smoothing methods have been developed for time-series data that appropriate to the acute effects of air pollution on daily mortality.

The case-crossover designs is useful to approaches in environmental epidemiology (Nitta et al., 2010). Case-crossover (CCO) design is complement with crossover of subjects between periods of exposure and non exposure, so each subject serves as his or her own control. Case-crossover is conceptually similar to matched case-control. But in case-control the controls are people. In CCO, the "case" is the pollution level on the day when the health event occurred in a person, and the "controls" (referents) are pollution levels on days when this event did not occur. The CCO design is useful for studying transient effects in relation to intermittent exposures (Janes et al., 2005). Nitta (2010), showed study in Japanese, that case-crossover designs are useful to approaches in environmental epidemiology.

In Asian Cities the effects of air pollution appear to be similar to or greater than in North American and Western European cities (Wong et al., 2008). The study of Public Health and Air Pollution in Asia (PAPA) which found out the effect from 4 cities Bangkok, Hong Kong, Shanghai and Wuhan are as high or higher than west cities, especially in Bangkok Thailand. Modeled air pollution effects on mortality were considerably higher in Bangkok than in the other 3 cities. It is not yet clear whether these stronger effects are real. One major goal of the proposed study is to compare risks of air pollution exposure as determined in the time series research to risks as determined using the case-crossover design.

Bangkok is the capital city that has the most air pollutants in Thailand. The situation of air quality in Bangkok between 2008 and 2009 data from Air Quality and Noise Management Bureau Bangkok Thailand showed O_3 and PM_{10} which are still more problem. The study daily time-series analysis of particulate matter air pollution effects on health in Bangkok found the percentage change in daily mortality estimated for each 10 $\mu g/m^3$ change in daily PM_{10} concentration. They used a 2 or 3 day lag in PM_{10} and 5 day moving average (Lauraine et al., 1998).

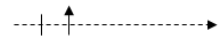
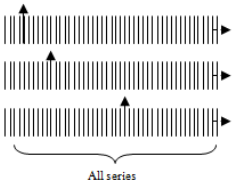
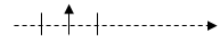
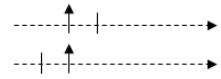
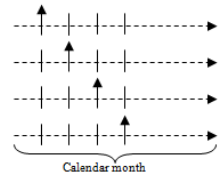
The association between short term effected of air pollution and daily mortality has been well established in national and international scientific literature. Another main objective of this dissertation is to find out the relative effects of air pollution on mortality in Bangkok during 1999-2001 and 2006-2008 by use time stratified case crossover design to estimate this hypothesis. The association between particulate matter PM_{10} and gases (NO_2, SO_2, CO, and O_3), and all natural (non-accidental) mortality, as well as cardiovascular and respiratory mortality was presented. Specific issues have been investigated, such as the comparison effect of air pollution in the last decade and recently year. The method of research design time series and case crossover was compared. The study population consisted of 221,444 subjects, which non accidental death in Bangkok during study period. For each subject, information was collected on cause of death, demographical variables and doctor diagnoses (ICD 10) for cause of death.

The case-crossover design was created by Malcolm Maclure in 1991 (Maclure et al., 1991). He attempted to test hypotheses concerning the immediate determinants of myocardia infarction using Case-control study to study the onset date. However, Maclure and colleagues found the choice of control group was not straightforward because in the general population are not easy to recruit the healthy representatives. The case-control design is subject to confounding by many factors, such as physical exertion, alcohol intake, anger and heavy eating. Maclure and colleagues asked the question “who would be the best representatives of the population base that produced the cases?” and they found the answer was the cases themselves, leading to the case-crossover design.

There have been important developments in the CCO design since Maclure. An important issue in case-crossover studies (as in case-control studies) is selection of

referent days (days on which the event did not occur for each subject). Different CCO referent strategies are shown in table 1.

Table 1: Comparison of different CCO designs.

Reference	Type	Selection	Advantage	Factors that can introduce bias	Selection of controls diagram
Maclure 1991	CCO	One control point before the effect	All possible confounding factors undergoing no change between control periods and effect, automatically controlled for by design	Long-term trends or seasonality	
Navidi 1998	Full-stratum bidirectional	For each case, all the days of the series other than that of the event taken as controls	Provides control for long-term trends	Long-term trends (only partially controlled for) or seasonality	
Bateson and Schwartz 1999	Symmetric bidirectional	Two at equal distance of the event	Provides adequate control for long-term trends and seasonality		
Navidi and Weinhandi 2002	Semisymmetric bidirectional	One chosen at random from the two used for symmetric bidirectional CCO	Provides adequate control for long-term trends and seasonality		
Lumley and Levy 2000	Time stratified	One (or several) within the same time stratum in which the event occurred	Provides adequate control for long-term trends and seasonality		

Arrows pointing up indicate case periods; horizontal arrows represent direction of time wihtin 1 month; dashed lines indicate time periods of 1 day; Vertical lines indicate control periods.
Source: Eduardo et al., 2010.

Several alternative CCO referent strategies have been developed after Maclure. Navidi (1998), compared unidirectional with bidirectional full-stratum CCO design, the result showed bidirectional design had less bias than unidirectional study. Bateson and Schwartz (1999), compared Poisson time-series regression design with different CCO designs, instance for unidirectional, bidirectional, random matched pair, and symmetric, by control periods ranging from 1–4 weeks both before and after the index time. They found the symmetric CCO design performed best in terms of bias.

Lumley and Levy (2001), compared symmetric CCO designs with time-stratified CCO designs, the result showed time-stratified CCO design better performance than symmetric CCO designs, even though both appear a small bias. Lee et al. (2000), compared unidirectional design with symmetric CCO, the result showed symmetric CCO did better. Levy compared unidirectional with symmetric design, they used

different numbers of control periods and at different intervals from the event period, as well as the influence of autocorrelation between control periods and overlapping (Levy et al., 2001). They provided that the symmetric CCO design did better performance than unidirectional study. Navidi and Weinhandl compared Poisson time series design with the following CCO designs such as symmetric, semisymmetric, random matched pair, and full stratum (Navidi and Weinhandl, 2002). They found the semisymmetric design did the best performed from all of designs in the study. Fung compared Poisson time-series design with unidirectional, symmetric, and semisymmetric CCO designs (Fung et al., 2003). The result showed the symmetric design had a better performance in terms of bias than all designs. Figueiras compared the Poisson time-series design with a number of CCO, they provided semisymmetric CCO had fewer biases than did symmetric or time-stratified CCO (Figueiras et al., 2005). Janes et al. (2005) showed that the time-stratified CCO design is more generally valid than any other CCO design. Thus, the proposed research was employ the time-stratified design.

1.2 Knowledge gaps

The CCO study has frequently been used to investigate associations between Air pollution and daily mortality, but Thailand has still not used CCO to assess the effect of air pollutants with public health issue. Using CCO study has a slowly growth (Carracedo-Eduardo et al., 2010). The present study focuses on outdoor (ambient) air pollution by using time stratified case crossover study to find out the effects of air pollution to public health problem in Bangkok, and to compare results to existing time series results.

1.3 Research Questions

1.3.1. Does Air pollutions associated with daily mortality in Bangkok could be identified by time stratified case-crossover (TS-CCO) study?

1.3.2. Does Health effect of air pollution in years 1999-2001 less than years 2006-2008?

1.3.3. How do estimates of air pollution effects using CCO design compare to time series design, as seen in other studies?

1.4 Objectives

1.4.1. To examine the association between ambient air pollution and daily mortality in Bangkok, by using Time Stratified Case-Crossover design.
1.4.2. To compare health effect of air pollution among years 1999-2001 and 2006-2008, by using TS-CCO design.
1.4.3. To compare the applicability of TS-CCO design in this study with time series design as PAPA study.

1.5 Research Hypotheses

Hypothesis I: There are significant association between Air pollutants (O_3, SO_2, PM_{10}, NO_2, CO) and daily mortality (non-accidental, respiratory, circulatory) in Bangkok, by using Time Stratified Case-Crossover study.
Hypothesis II: There is different in health effect on air pollution between years 1999-2001 and 2006-2008.
Hypothesis III: There is no different results in health effect on air pollution between two study designs (Time Stratified Case-Crossover and Time-Series).

1.6 Expected Benefit & Application

1.6.1. The results of this study are useful for the policy maker to planning strategy reduce mortality rate from air pollution (Zeka et al., 2006).

1.6.2. Bureau Epidemiology of Ministry of Public Health Thailand consider to using CCO analysis for continue monitoring air pollution effect on health in surveillance system (Cadum et al., 2009).

1.6.3. The cardiovascular and respiratory patients aware to protect themselves from air pollution.

1.7 Operational definitions

Ambient Air pollution; (World Bank, 1998) Air pollution outside and surrounding air pollution. The air pollutants were collected by Bangkok Pollution Control Department (PCD). For instance, 10 sites in Bangkok are collected air sample for PM_{10}, SO_2, O_3, CO, and NO_2. The average of air pollutions were calculated to represent the problem of air pollutant in Bangkok, by using CCO design with comparison to other design such as Time Series design.

Daily mortality; (MOPH Thailand, 2011) The non-accidental mortality, Respiratory system (ICD-10 = J00 – J99) e.g. Chronic obstructive pulmonary diseases (COPD), Circulatory system (ICD-10=I00 – I99) e.g. Cardiovascular diseases (CVD).

Referent selection strategy; (Eduardo et al., 2010) The type of study design of CCO that appropriate for the data collection to referent the time in the control period, e.g. unidirectional, full stratum bidirectional, symmetric, semi-symmetric, or time stratified.

Index time; (Maclure et al., 1991) The exposure just before the event that the same as case group in match case control.

Referent time; (Maclure et al., 1991) The control period that expose or non expose in the random time.

Overlap bias; (Levy et al., 2001) bias resulting from the use of incorrect referent periods. The bias can occur in Conditional Logistic Regression analysis. Overlap is misleading cause of individual design set of possible referent windows overlapping is subject to overlap bias, the design which not in the windows is not subject to such bias.

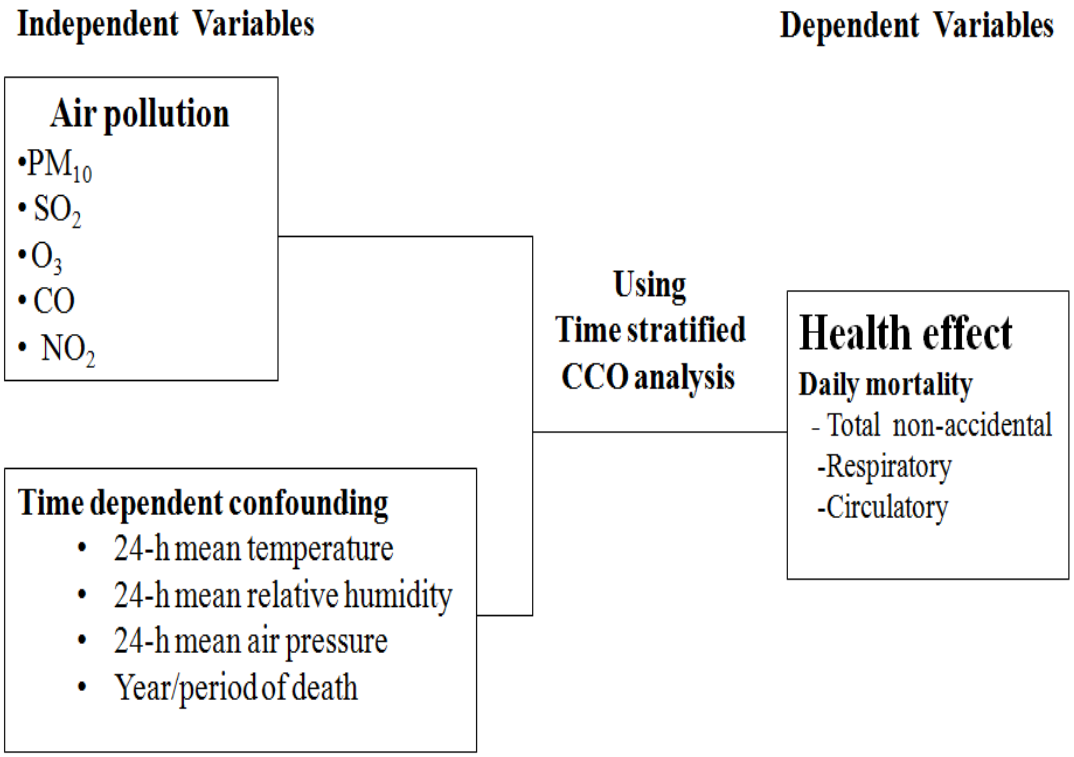
Short term effect; (Maclure et al., 1991) The acute health effect after get the exposure of air pollutants in a short time.

Autocorrelation; (Levy et al., 2001) The correlation of a temporal series variable with its own previous or posterior values.

Case crossover study; (Eduardo et al., 2010) The design can control time dependent by restricting referents time to the same day of the week, month, and year as the index day. Conditional logistic regression analysis of shared exposure series with time stratified by year, month, and day of the week was used.

Time series study; (Merz, 1972) The design is to collected the observations by repeated measurements over time.

1.8 Conceptual Framework*



*** In the CCO design there is no need to adjust for time-constant variables such as sex, age, and other sociodemographic variables. Effects of these variables was examined and compared in stratified analyses (see chapter 3).**

CHAPTER II

LITERATURE REVIEW

This chapter presents specific literatures relevant to the research effort. Seven principle areas including Air pollution, Anatomy, Air pollution effect on health, Case crossover study, Time series study and comparing between time-series and Case crossover study.

2.1 Air pollution

We all know that air is very essential for the survival of all the living organisms on earth that include humanity. Air supplies us with oxygen which is essential for our bodies to live. The earth's atmosphere is roughly 79% nitrogen, 21% oxygen and another gas as carbon dioxide, helium, argon, krypton, xenon, organic and inorganic gas. The clean Air is decrease because of consumption has largely been ignored in the trade and environment debate, despite the fact that it accounts for a large portion of total emissions. Human activities can release of chemicals and particulates into the atmosphere, some of which can cause problems for humans, animals, plants, and environment. Instance for traffic and powerplant those are source of Particulate matter that associated with increased deaths (Maynard et al., 2007). A substance in the air that causes harm to humans and the environment we know as Air pollutant. In the past air pollution may come from variances of component in atmosphere by nature such as volcano when they expose, the smoking, ash or gas release to the ambient air. It cause of human and animal die in large numbers. (Frederik, 1994).

Air pollution is a serious threat to public health and the environment. Air pollutants can be in the form of solid particles, liquid droplets, or gases. In addition, they may be natural or man-made. Among the major sources of pollution are power and heat generation, the burning of solid wastes, industrial processes, transportation and especially household such as cooking, candle burning and tobacco smoking. There are several types of air pollution which are commonly discussed. The major types of pollutants are carbon monoxide (CO), hydrocarbons (CH_4), nitrogen dioxides (NO_2),

particulate matter (PM), sulfur dioxide (SO_2), Ozone (O_3) and photochemical oxidants (Stephen et al., 1999). Primary pollutants are the pollutants which are emanation directly into the atmosphere (e.g. CO, SO_2). Secondary pollutants are the air those are produced by chemical reactions with other gas (e.g. O_3). Some pollutants are be the both primary and secondary because emitted directly into the atmosphere, and formed from other pollutants (e.g. NO_2, PM). Instance for nitrogen dioxide emanate directly from vehicle exhaust and power station, while oxidation of nitric oxide (NO) can transform NO_2 into the atmosphere. Another sample is PM, it emitted directly from a number of natural and anthropogenic sources. The transform of PM is mainly from the oxidation of SO_2 and NO_2 that produce secondary PM.

Ambient air pollution has the effect to physical, psychology, environments and economic problems. The socioeconomic status is the factor of exposed to the air pollutant as a less advantaged people (Forastiere, 2007), the lower socioeconomic status people more susceptible exposed to air pollutants and effect on their health (Malig et al., 2009). Air pollutions are both indoor and outdoor that can cause of morbidity and mortality (Chapman et al., 1988). The poor ambient air quality area should be concern (Luginaah et al., 2005). In the studies between 1990 and 2010 they found many kinds of disease in cardiovascular diseases and respiratory diseases that occurred because of air pollutants effect, such as Angina and ischemic heart disease, Acute myocardial infarction, Asthma, Cancer, Cerebrovascular, Conduction disorders and dysrhythmia, Congestive heart failure, Chonic obstructive pulmonary disease (COPD), Diabetes, Hypertension, Atherosclerosis, Bronchiolitis, Cardiac anomalies, Cardiac arrest, Gastroenteric Disorders, Ischemic stroke, Hyperlipidemia, Other kidney or renal disease, Otitis Media, Postneonatal et. (Eduardo et al., 2010). For example, the exposure as NO_2, CO, and BC are strong association with myocardial infaction and pneumonia this source is from traffic exposure that responsible to heart attack (Zannobetti and Sxhwartz, 2006). Short term exposure to particulate air pollution has been linked to an increased risk of ischemic stroke hospital admission (Oudin et al., 2010)

In Asian Cities the effects of air pollution are similar or greater than in the North American and Western European cities (Wong et al., 2008). The study of Public Health and Air Pollution in Asia (PAPA) which found out the effect from 4 cities Bangkok, Hong Kong, Shanghai and Wuhan are as high or higher than west cities

especially Bangkok Thailand is the city that have the most strong effect at older ages for air pollutants support the validity of this estimates because the populations spend more time outdoor and less time in air conditions. Time series analysis was used to estimate air pollution and health effect outcome in this study, but data did not clear whether these higher effects were real. In central Bangkok (Vichit-Vadakan et al., 2008), a short-term effect on air pollution increases in daily levels of PM_{10} and O_3 associate with the number of daily emergency hospital visits for cardiovascular diseases (CVD), particularly among in elderly age more than 65 years (Buadong et al., 2009).

Epidemiological studies of air pollutants are essential in prevention and control of public health problems. Epidemiology is the study to find out exposures in associate with disease. The key question is either a given exposure or set of exposures that cause a exactly disease. If can find the exposure cause of diseases, it the chance to implement the intervention, prevention, and controlling disease spread that is ultimate goal of epidemiological study. The association between health effects and air pollution are weak, so observed in the large populations to considerable variation of air pollutant would have to monitor because individuals who close to risk of exposure cannot be identified accurate a *priori* (Hill, 1965). They have many kind of research design to estimate air pollutant and mortality. Two designs used to evaluate short-term air pollution exposures are the time series design and the case-crossover (CCO) design. In time series, daily pollutant levels and daily counts of adverse health effects (e.g., mortality) are correlated to see whether these counts are associated with pollutant levels. Several time series studies, but as yet no CCO studies, have been done in Bangkok. In epidemiological studies not only design but method to collects the sample of air pollutant also is important. The selection of different exposure settings as individual stations and regional averages may affect the observed epidemiological concentration-response (C-R) relationships. (Sajani et al., 2010).

Bangkok is the capital city that has the most air pollutants in Thailand. The situation of air quality in Bangkok between 2008 and 2009 data from Air Quality and Noise Management Bureau Bangkok Thailand showed O_3 and PM_{10} which are still more problem. The study health effects of particulate matter air pollution in Bangkok found the percentage change in daily mortality estimated for each 10 $\mu g/m^3$ change in daily PM_{10} concentration. They used a 2 or 3 day lag in PM_{10} and 5 day moving average

(Lauraine et al., 1998). Bangkok is urban area the condition of air pollution similar most of urban atmospheric pollution around the world that motor vehicle emissions are the main source than other (Maitre et al., 2006). Bangkok have condition with heavy traffic that may expose to particulate and gaseous emissions from diesel trucks and light-duty vehicles (Subramanian et al., 2009). Crowds of people on roadside and under skytrain could be had effect from PM_{10} and bacterial in the air because of insufficient air ventilation (Luksamijarulkul and Kongtip, 2010). The tourists who visit Bangkok where may effect on the air pollution as a smoking, they have be more awareness about the Thai regulation for reducing air pollution (Viriyachaiyo and Lim, 2009), that mean most of them recognize to protect themselves from exposure of air pollutants.

Table 2: Air pollution in Bangkok, 2004

Pollutant	Min-Max	95 Percentile	Standards	Number of over standard/Number of measure (%)	Average /year
TSP 24-h (mg/m^3)	0.02-0.32	0.21	0.33	0/436 (0)	0.11
PM_{10} 24-h ($\mu g/m^3$)	19.3-183.8	116.4	120	56/1,665 (3.7)	58.1
Pb 1-m ($\mu g/m^3$)	0.02-0.34	0.22	1.5	0/107 (0)	0.09
CO 1-h (ppm)	0-8.3	2.0	30	0/71,616 (0)	0.7
CO 8-h (ppm)	0-5.2	1.8	9	0/74,282 (0)	0.7
O_3 1-h (ppb)	0-173.0	53.0	100	102/58,081 (0.18)	15.7
SO_2 1-h (ppb)	0-103.0	14.0	300	0/70,886 (0)	5.0
SO_2 24-h (ppb)	0-19.7	10.3	120	0/2,884 (0)	5.0
NO_2 1-h (ppb)	0-170.0	58.0	170	0/69,752 (0)	24.3

Source: Pollution Control Department (2004)

Table 3: Reporting of Daily Air Quality - Air Quality Index (AQI) in Bangkok.

AQI	PM_{10} (24 hr.)	O_3 (1 hr.)		SO_2 (24 hr.)		NO_2 (1 hr.)		CO (8 hr.)	
	µg./m^3	µg./m^3	ppb	µg./m^3	ppb	µg./m^3	ppb	µg./m^3	ppb
50	40	100	51	65	25	160	85	5.13	4.48
100	120	200	100	300	120	320	170	10.26	9.00
200	350	400	203	800	305	1,130	600	17.00	14.84
300	420	800	405	1,600	610	2,260	1,202	34.00	29.69
400	500	1,000	509	2,100	802	3,000	1,594	46.00	40.17
500	600	1,200	611	2,620	1,000	3,750	1,993	57.50	50.21

Source: Pollution Control Department (2010)

2.2 Instrumental Method

2.2.1 Non-dispersive Infrared Detection is the method that measure CO_2 by using infrared.

2.2.2 Chemiluminescence is the technique to measure NO_2 and O_3. NO_2 measurement by using O_3 to reactive with Nitric oxide change to NO_2. Concentration of emitting species and emitted light intensity is proportional to the concentration of reactant at wavelengths greater than 600 nanometers. O_3 measurement by using Ethylene reactive with O_3 and measure from emitted light intensity at wavelengths between 350 to 550 nanometer.

$$NO + O_3 \rightarrow NO_2^* + O_2$$

$$NO_2^* \rightarrow NO_2 + hV$$

2.2.3 UV-Fluorescence is sulfur dioxide measurement by using ultraviolet into reaction cell where it is irradiated by a beam of ultraviolet energy at a known wavelength. SO_2 molecules present in the sample air become excited to produce a metastable higher energy molecule. This high energy molecule, on returning to its ground state, releases energy at wavelengths between 120 to 190 nanometer. The

equations or reaction is

$$SO_2 + UV\ (190\text{-}230\ nm) \rightarrow SO_2^*$$

$$SO_2^* \rightarrow SO_2 + Energy\ (120\text{-}190\ nm)$$

2.2.4 Pararosaniline is the technique to measurement SO_2 volume bubble through a solution of 0.04 M potassium tetrachloromercurate (TCM). The SO_2 present in the air stream reacts with the TCM solution to form a stable monochlorosulfonatomercurate, is Potassium Tetrachloromerculate to form dichlorosulfito merculate complex and reaction to Pararosaniline and formaldehyde to form an intensely colored pararosaniline methyl sulfonic acid. The optical density of this species is determined spectrophotometrically at 548 nanometer which related to SO_2 collection.

2.2.5 Ultraviolet Absorption Photometry is the technique that using Ultraviolet to reactive with Ozone and measure absorption light from reaction at wavelengths 254 nanometer.

2.2.6 Gravimetric is method which are used for PM_{10}. Ambient air are pulled through filter media and using a high volume. Particle matter size of 10 micron are represented by the 99th percentile, the weight of PM deposited on the filter can be used to calculate the PM_{10} level.

2.2.7 Beta Ray using increasing of beta radiation absorption with mass of particle matter which size less than 10 micron. Particles are collected on filter tape in beta gauge, measurement the co-efficiency beta radiate and PM_{10} concentration on filter tape. Transformation the light to digital and display on monitor.

2.2.8 Tapered Element Oscillating Microbalance (TEOM) is technique using piezoelectric quartz. Particles are collected on quartz and make the frequency of oscillating change then equipment analyze the output. This is real time measurement that can continuous collect PM_{10} and $PM_{2.5}$

2.2.9 Dichotomous is the method that collect $PM_{2.5}$ by separate from part of PM_{10} because of different in mass, size and inertial. Acceleration jet plumb particle drown into middle plenum, coarse particulate catch on the filter but fine particle are pulled by vent jet and thought to vent tube flow to virtual impactor.

Table 4: National Ambient Air Quality standards analyzer of Thailand.

Pollutants	**Standard**	**Time average**	**Method**	
			Reference Method	**Equivalent Method**
CO	- 30 ppm(34.2 mg/m^3) - 9 ppm(10.26 mg/m^3)	1 hour. 8 hour.	-Non-dispersive Infrared Detection	
SO_2	- 0.30 ppm(0.78 mg/m^3) - 0.12 ppm(0.30 mg/m^3) - 0.04 ppm(0.10 mg/m^3)	1 hour. 24 hour. Annual	-UV Fluorescence -Pararosaniline -Pararosaniline	-Pararosaniline -Ultraviolet Fluorescence -Ultraviolet Fluorescence
NO_2	- 0.17 ppm(0.32 mg/m^3) - 0.03 ppm(0.057 mg/m^3)	1 hour. Annual	-Chemiluminescence -Chemiluminescence	
O_3	- 0.10 ppm(0.20 mg/m^3) - 0.07 ppm(0.14 mg/m^3)	1 hour. 8 hour.	-Chemiluminescence	-Ultraviotlet Absorption Photometry -Ultraviotlet Absorption Photometry
PM_{10}	- 0.12 mg/m^3 - 0.05 mg/m^3	24 hour. Annual	-Gravimetric -Gravimetric	-Beta Ray -Tapered Element Oscillating Microbalance: TEOM

Source: Pollution Control Department, Thailand (2010).

Table 5: Sources of air pollutions, Health and Welfare Effects for Criteria Pollutants 2010.

Pollutant	Description	Sources	Health Effects	Welfare Effects
Sulfur Dioxide (SO_2)	Colorless gas that dissolves in water vapor to form acid, and interact with other gases and particles in the air.	Electric power companies that burn coal are a major source of sulfur oxides. The burning of large quantities of wood in fireplaces and stoves. Natural Volcanoes and Hot Springs. Diesel fuel and gasoline.	It irritates the nose, throat, and airways to cause coughing, wheezing, shortness of breath, or a tight feeling around the chest.	Contribute to the formation of acid rain, visibility impairment, plant and water damage, aesthetic damage.
Nitrogen Dioxide (NO_2)	Reddish brown, highly reactive gas.	Vehicles burning diesel or petrol. Domestic fires. Power stations burning fossil fuels. Major industry.	NO_2 is a pulmonary irritant affecting primarily the upper respiratory system. Individuals with asthma, respiratory disorders, and lung diseases are more sensitive to the effects of NO_2.	Contribute to the formation of smog, acid rain, water quality deterioration, global warming, and visibility impairment.

Pollutant	Description	Sources	Health Effects	Welfare Effects
Ozone (O_3)	Gaseous pollutant when it is formed in the troposphere.	Vehicle exhaust and certain other fumes. Formed from other air pollutants in the presence of sunlight.	Eye and throat irritation, coughing, respiratory tract problems, asthma, lung damage.	Plant and ecosystem damage.
Carbon Monoxide (CO)	Colorless, odorless and tasteless gas.	Motor vehicle exhaust, kerosene or wood burning stoves.	Headaches, reduced mental alertness, heart attack, cardiovascular diseases, impaired fetal development, death.	Contribute to the formation of smog.
Particulate Matter (PM_{10})	Very small particles of soot, dust, or other matter, including tiny droplets of liquids.	Volcanoes, dust storms, forest and grassland fires, living vegetation, and sea spray. Burning of fossil fuels in vehicles, power plants and various industrial processes	Asthma, lung cancer, cardiovascular issues, and premature death. Particulate matter rupturing, blocking and/or passing through alveoli, leading to CA, atherosclerosis	Visibility impairment, atmospheric deposition, aesthetic damage.

Source: Pollution Control Department, Thailand (2010).

2.3 Anatomy

2.3.1 Respiratory System

The aim of respiratory function is to bring oxygen to cell. Cell use oxygen to metabolize and diffusion carbon dioxide to the environment. Cell does not contact with air directly then they have the co ordination function that is respiratory system, muscle system, circulatory system, and nervous system to bring the oxygen in and release the carbon dioxide out.

2.3.1.1 Respiratory processes are to convert gas have 2 steps.

2.3.1.1.1 External respiration is the process to convert air between external and internal body where occur in lung. The lung take carbon from vein and give the oxygen to vein, component with 3 processes.

2.3.1.1.1.1Ventilation is the process to bring air outside come into the lung and release air from lung to environment because of pressure are different between external and internal lung

2.3.1.1.1.2 Perfusion is the process when blood come in alveoli to take oxygen and release carbon dioxide.

2.3.1.1.1.3 Diffusion is the process to convert oxygen and carbon dioxide through alveolar capillary membrane

2.3.1.1.2 Internal respiration or cellular respiration is the process of cell to bring oxygen inside for burn down and release carbon dioxide that is product from this process.

2.3.1.2 Respiratory structures have 2 part of function.

2.3.1.2.1 Conduction air way when air passes the nostril or oral to pharynx larynx trachea bronchi bronchiole and terminal bronchiole. The air way dose not exchange but also bar the way from particle matter (PM) to lung with slime, hair in nose blow the PM bigger than 10 micron at Nasopharynx and 2-10 micron at wall of larynx and trachea the particle matter 0.3-2 micron catch at alveolar duct and alveoli if smaller 0.3 micron breath out.

2.3.1.2.2 Respiratory unit start at bronchiole that respiratory bronchiole alveolar duct alveolar sac and alveoli. Gas is exchanged between gas in alveoli and gas in vein.

2.3.1.3 Mechanism of Breathing are combination 2 parts first is Inspiration and second is expiration this function use muscle of respiratory system, chest and lung

movement so air pressure different between outside and inside alveoli make air move in and move out the lung

2.3.1.3.1 Inspiration process need energy and using muscle of respiratory system as diaphragm muscle and external intercostals muscle that are activated by signal of nervous ranic and intercostal when diaphragm retract, it's shape like a dome, it will flat and move down to the abdominal cavity make high of chest increase and widen below of the ribs that make diameter of lower chest is wider. The simple inspiration make diaphragm down 1 – 1.5 centimeter and bring air to lung 75 percentage. Full inspiration, diaphragm will down 6 -11 centimeter and sternum widen increase air in to lung 25 percentages. Air will moved to lung when chest is extend make air pressure in lung decrease and lower than outside. The strong breath use more muscle such as trernoclidomastoid serratus anterior scalene and trapezius make ribs higher. The volume of chest cavity will increase while inspiration the intrapleural pressure and intrapulmonary pressure will change because air pressure in lung lower than outside or negative pressure (- 3 mm.Hg.). Lung will huddle up to the end of breathing pressure in lung proximately -2.5 mm.Hg. Until the deep breathing about -6 mm.Hg. when lung become normal. The strong inspiration will reduce pressure in lung -30 mm.Hg. when lung most huddle will effect to extend the vein in the lung and make blood cannot move while strong expiration the pressure in lung will positive, artery vein will pressed blood cannot move to heart then brain lack of oxygen make the patient unconscious. The pressure in lung relates to respiratory when breath in chest will extend and make lung expand. Pressure in lung about -1 mm.Hg. will move air volume 500 ml. per 2 second into lung.

2.3.1.3.2 Expiration, especially normal expiration does not need energy but use the loosen of diaphragm muscle and muscle in cavity between external intercostals, it will reduce cavity. The lung tissue that extend while breath in will huddle and pressure in lung increase so difference from air pressure outside proximately 1-3 mm.Hg., air in lung will pushed out to environment that is expiration. Until air pressure is balance, air pressure in lung about 757 mm.Hg. or -3 mm.Hg. fast breath will use more muscle to expiration more than particular the internal intercostals huddle pull the sternum and reduce cavity and muscle abdominal namely internal oblique external oblique and transverse abdominis these huddle will make the organ in abdominal push the

diaphragm higher to increase vertical volume or cavity sternum and push the air in lung out.

2.3.1.2 Pulmonary Perfusion

Two system of Blood circular transfer to respiratory system. First is Bronchial circulation, this system bring food and oxygen to feed organs in respiratory system compose to Bronchial vein and artery. Artery bronchial transport high oxygen to lung and bronchi and low oxygen enter vein to heart after used. Second is Pulmonary circulation, this system transport blood from heart to lung to exchange oxygen and carbon dioxide, it takes oxygen to heart and refer to organs. Pulmonary circulation from pulmonary artery from right heart to bronchi thought capillary cover alveoli for gas exchange that suitable ventilation/perfusion matching. Pulmonary circulation are different other circulation in organs, cause of short vassal resistant, low pressure, thin cell wall, more extend

2.3.1.3 Gases exchange

Ventilation supplies atmospheric air to the alveoli when inhale the ambient air, O_2 is the most necessary need for an organic. The next step in the process of respiration is the diffusion of gases between the alveoli that bring O_2 to area and the blood in the pulmonary capillaries that bring CO to here. The respiratory membrane is all of the areas in which gas exchange between O_2 in the air and CO in the blood. The major area of gas exchange is in the alveoli, event if some appear in the respiratory bronchioles and alveolar ducts. Gas exchange between blood and air does not appear in as other areas of the respiratory passageways are thus called this area is dead space. The exchange of gases across the respiratory membrane is influenced by the thickness of the membrane, the total surface area of the membrane, and the partial pressure of gases across the membrane.

2.3.1.4 Gases Transport in the blood

Oxygen Transport; after inhale air when O_2 diffuses through the respiratory membrane into the blood, around 98.5% of the O_2 combines reversibly with the iron-containing heme groups of hemoglobin in the alveoli. around 1.5% of the O_2 still remains dissolved in the plasma. Hemoglobin with O_2 bundle to its heme groups that we called Oxyhemoglobin. The function of hemoglobin is to bind to O_2 but it depends on the partial oxygen pressure (Po_2) hemoglobin liberated O_2. In the lungs, Po_2 normally is sufficiently high that hemoglobin can holds as much O_2 as it can. In the

tissues, Po_2 is lower to holds O_2 because the cells of the tissues are using O_2. Abridging, hemoglobin liberates O_2 into the tissues. O_2 then diffuses into cells and is used the cells in aerobic metabolism. Approximately 23% of the O_2 are picked up by hemoglobin in the lungs is aim to the tissues. The amount of O_2 liberated from oxyhemoglobin is influenced by many factors. More O_2 is liberated from hemoglobin if the Po_2 is low, the Po_2 is high, the pH is low, and the temperature is high. Increased muscular activity which is the results in a decreased Po_2, an increased Po_2, a decreased pH, and an increased temperature. Abridging, more than 73% of the O_2 are picked up by hemoglobin in the lungs is liberated in skeletal muscles when physical exercise.

Carbon Dioxide(CO_2) diffuses from cells, where it is produced and leased into the tissue capillaries. After CO_2 enters the blood, it is transported in three principal ways (1) about 7% is transported as CO_2 dissolved in the plasma, (2) 23% is transported in combination with bold proteins, primarily hemoglobin, and (3) 70% is transported in the form of bicarbonate ions. CO_2 reacts with water to form carbonic acid (H_2CO_3). The carbonic acid can dissociates to form H^+ and bicarbonate ions (HCO_3^-). carbonic anhydrase in an enzyme that can found inside red blood cells and on the surface of capillary epithelial cells. Carbonic anhydrase increases the rate tissue capillaries. Therefore, carbonic anhydrase promotes the uptake of CO_2 by red blood cells. In the capillaries of the lungs, the process is revered so that the HCO_3^- and H^+ combine to produce H_2CO_3 and forms CO_2 and H_2O. The CO_2 diffuses into the alveoli and is expired, it has an important effect on the pH of blood. The blood pH decreases when CO_2 levels increase because CO_2 reacts with H_2O to form H_2CO_3. The dissociation of H_2CO_3 is responsible for the decrease in pH can release H^+. It can conversely, as blood levels of CO_2 decline, the blood pH increases.

2.3.2 Circulatory System

Circulatory system is an organ which has function to passes nutrients, gases, hormones, blood cells, etc. The process that vessels and muscles control the flow of the blood around the body is called circulation system. The main organs of the circulatory system are heart, arteries, veins and capillaries. This system helps body to fight diseases and stabilize body temperature and maintain homeostasis. The heart is

major source of force that causes of blood flowing in circulate, and the peripheral circulation functions to carry blood, blood vessels carry blood leave the heart from the left ventricle and thought to aorta. The aorta is the largest artery where the blood leaves and take full of oxygen to all tissues of the body and brain that to do work and back to the heart with carbon dioxide. The circulatory system' functions are Exchange nutrients, waste products, and gases. Nutrients and oxygen scatter from blood vessels to all necessary cells of the body. Waste products and carbon dioxide are out of the cells and black to blood vessels. Circulatory system transports, Hormones, components of the immune system, Molecules required for coagulation, enzymes, nutrients, gases, waste products, and other substances in the blood to all areas of the body. The blood through a system of vein and reaches to the lungs where carbon dioxide is removed from the blood and exchange with oxygen that process is inhalation through the lungs. The circulatory system composes of the cardiovascular system, lymphatic system. These are fluids move through the circulatory system. The cardiovascular system has blood, heart, and blood vessels to form. The lymphatic system has lymph, lymph nodes, and lymph vessels to form.

Heart, veins, and blood vessels are the main components of the human cardiovascular system. The system comprises pulmonary circulation as a loop through the lungs where gas exchange. The average blood in adult contains about 4.7 to 5.7 liters, which compound of plasma, platelets, white blood cells, and red blood cells. Including the digestive system, that is function organ works with the circulatory system to provide the nutrients to necessary cells of body, especially to keep the heart continues pumps blood to save the live. The right ventricle pumps blood into the pulmonary trunk, as the shortness vessel branches where blood pass to left and right pulmonary arteries. The left and right lungs are the destination of blood travel. These arteries carry poorly oxygenated blood to the pulmonary capillaries in the lungs for exchange gas, such oxygen is taken up by the blood and carbon dioxide is released. The rich oxygenated blood flows from the lungs to the left atrium. The four pulmonary veins are out of the lungs and carry the oxygenated blood to the left atrium and distribution to body as a network.

Systemic circulation is a part of the cardiovascular system that transports oxygenated blood take away from the heart throughout the body and returns poorly oxygen with carbon dioxide blood back to the heart. Systemic circulation is distance longer than

pulmonary circulation when transporting blood to every tissues and cells. The coronary circulatory system provides a blood supply with oxygenated to the heart. The heart pumps rich oxygenated blood to the body and poor oxygenated blood to the lungs. In the human heart there is one ventricle and one atrium for each circulation, for human body have both a systemic and a pulmonary circulation, total of there are four chambers such as right atrium, right ventricle, left atrium and left ventricle. The left atrium receives refresh oxygenated blood from the lungs as well as the pulmonary vein which is passed into the strong left ventricle to be pumped through the aorta to every part of the organs in the body. The right atrium is the upper chamber of the right side of the heart. The blood that is returned to the right atrium is poor in oxygen and passed into the right ventricle. It pump through the pulmonary artery to the lungs for exchange gas carbon dioxide and oxygen . the circulatory system closed to cardiovascular system. Because the blood never leaves the network of blood vessels. The other component of the circulatory system is the lymphatic system, but this system is not closed. The location of heart is in the center of the body, it stay between the two lungs. The reason of the heart beat is felt on the left side, because the left ventricle is pumping harder. Cardiovascular Effects as once toxic substances reach the cardiovascular system, a number of reactions can occur. Therefore air pollution has the effected on cardiovascular system include physical changes, degeneration, and inflammation of the heart and other areas. Pollutants can also cause heart arrhythmias, which can cause of morbidity and mortality. Instance for air pollution and cardiovascular hospital admissions are association (Barnett, 2006)

This dissertation aim to study air –pollution and Circulatory system effect, so temperature should be controlled cause of it is the confounder for study. Temperature is association with healthy especially in elder (Barnett, 2007). The study in Sao Paulo about temperature and mortality risk showed strongly relationship, the elder are targeted for impact (Bell, 2008). Ambient temperature in Beijing China has association with hospital visit for cardiovascular disease (Gou et al., 2009). Both of high and low temperatures are relate increases in mortality (McMichael et al., 2008). Between sex variable are get the different exposure level and different health effect (Kolb et al., 2007)

2.4 Air pollution Effect on health

2.4.1 Non-accidental mortality

Air pollution relates to the non-accidental mortality, as several study showed in the case of CCO design. For examples is Ren's study, Ren and colleague studied the modifiers of short term effects of ozone on mortality, which socioeconomic status coded at the tract in eastern Massachusetts, US. The study's period was May-September, 1995-2002. The subjects were 157,197 non-accident deaths aging more than 35 years. They used moving averages of maximal 8 hour concentrations of ozone monitored at 8 stationary stations as individual exposure. The results showed ozone was associated with changes in all natural deaths, respiratory disorders, diabetes, cardiovascular diseases, heart diseases, acute myocardial infarction and stroke. This study was not found association significantly of the ozone's effects on mortality and modified by socioeconomic status, even individual characteristics (Ren et al., 2010). The temperature is not confounder for association between O_3 and mortality risk (Schwartz, 2005).

Kan studied the association between SO_2 and daily mortality in Bangkok, Hong Kong, Shanghai, and Wuhan (4 cities in Asia). The result showed increment 10 $\mu g/m^3$ 2 day moving average concentrations of SO_2 was relevant to daily mortality in four cities of this study. These appearance may be referent able to SO_2 serving as an indicator of other substances (Kan et al., 2010).

Ueda and other studied the short term effects of $PM_{2.5}$ on daily mortality in Japan. The results showed that increased in $PM_{2.5}$ the short-term effects of air pollutants had significantly association with daily mortality in Japan. and information was when study in multipollutant, using the differences in estimates obtained from different statistical models should be done (Ueda et al., 2009).

Stafoggia studied relationship between the short term effected of air pollution and mortality in ten Italian cities 2001-2005. The subjects were 276,205 who died in study areas, aged more than 35 years old, resident in one of the 10 Italian cities studied. The results showed that the strong association between a short term effect both of NO_2 and O_3 with mortality for all death causes. The air pollution emitted from the

original by vehicular traffic is the most environmental problem in Italian (Stafoggia et al., 2009).

The distance from air pollution has the effect to health, as Sajani's study. They studied different exposure settings on air pollution and daily mortality. Six main cities areas were selected in the central-western part of Emilia-Romagna region. They used four approaches to assign exposure to air pollutants for each individual considered in the study. They did not find any change in the C-R estimates. Within a geographically homogeneous region, the pollutant total of monitoring station data leads to higher and strong risk estimates for PM_{10} and NO_2, even though the correlation between distances of monitors showed a few decrease. Using of individual stations is larger aggregation improves the representative of the exposure estimates by decreasing exposure misclassification than regional averages (Sajani et al., 2010).

2.4.2 Respiratory mortality

Respiratory system is the most first organ that get an adverse event, so many investigators use case-crossover to find out the effect of air pollution and mortality in respiratory (Colais et al., 2009). Instance for Faustini and colleague studied the relationship between ambient particulate matter and respiratory mortality in 2001-2005. Their subjects were to assess association between PM_{10} and respiratory mortality in Italy, the second was to examine potentially susceptible groups. They selected 19,629 who were respiratory death and age over 35 year olds in ten northern of central and southern Italian cities. They corrected data for PM_{10}, nitrogen dioxide and ozone to be indicators. They found the effect of particulate 2.29% (CI 95%=1.03; 3.58) increase in respiratory mortality at 0-3 lags and stronger than on natural mortality. Females and chronic disease sufferers die more than males and healthy people. So the effects of air pollution on respiratory mortality are significantly resulting (Faustini et al., 2011).

Carbajal studied the effect of PM_{10} and O_3 on infant's respiratory mortality among residents in the Mexico City Metropolitan Area 1997-2005. The subjects 3,903 infant age between 1-11 months had respiratory deaths and resided in the Mexico City Metropolitan Area were included. The results showed that PM_{10} air pollutant had statistically significant positive association for increase in risk of respiratory

mortality. The infant, who exposed to O_3 was significantly association to respiratory mortality in low socioeconomic status (Carbajal-Arroyo et al., 2010).

2.4.3 Cardiovascular mortality

Mortality in Cardiovascular has the association with air pollution that showed in many studies, such as Serinelli's study, they evaluated the association between daily air pollutant concentration and daily data regarding mortality and hospital admissions among residents of Brindisi, in Southern Italy in the years 2003-2006. 6,925 subject who hospital admissioned for acute conditions as cardiac, cerebrovascular and respiratory diseases were recruited. They used time-stratified case-crossover analysis and a conditional logistic regression models. They controlled confounders as mean temperature, relative humidity, influence of epidemics, summer decrease of resident population and holidays. the models had been fitted with the cause of death or hospital admission, gender, age and season. They found the statistically significant associated between PM_{10} and mortality from all natural causes (10.36%; 95% CI 1.83-19.61 at lag 0-1), especially the risk of cardiovascular mortality was highest. This study showed strong and consistent associations between outdoor air pollution which coming from both industrial emissions and urban traffic and short-term increases in both mortality and morbidity in elderly age over 75 years (Serinelli et al., 2010).

Chen studied association between ambient air pollution and daily mortality in Anshan, China 2004-2006. Using TS-CCOapproach to assessment the effect of air pollutants as PM_{10}, SO_2, NO_2 and CO on cardiopulmonary mortality in Anshan. The results showed that significant relationship between short term effect of air pollution and daily mortality from cardiovascular diseases in Anshan. The total and respiratory mortality were generally positive association but statistically was not significant (Chen et al., 2010). Ambient air pollution has effect to increase the cardiovascular mortality in Barcelona especially PM_{10} and $PM_{2.5}$ (Perez et al., 2009).

2.4.4 Other respiratory

Mobility in some disease are relate to air pollutants that show in the CCO design, as study of Su and colleague. They studied the association between ambient air pollution and hospital emergency room visits for respiratory diseases in Beijing, China. Their objective was to assess the association between ambient air pollution and the hospital

emergency room visits for respiratory diseases. They used the International Classification of Diseases, tenth vision (ICD-10: J00-J99). They corrected the data from the daily Peking University Third hospital emergency room visits of the respiratory diseases were obtained in January 1st 2004 - December 31st 2005, and using meteorological factors from the local municipal environmental monitoring center and meteorology bureau of Beijing, respectively. They used Time-stratified case-crossover technique to evaluate the relationships between ambient air pollution and hospital emergency room visits for respiratory. The results showed that higher levels of ambient air pollutants increased the risk of hospital emergency room visits for respiratory diseases (Su et al., 2010).

Pereira studied the traffic-related air pollution and emergency department presentations for asthma in Perth, Western Australia. The subjects were 603 children and youths aged between 0-19 years who had presented with asthma and had resident in a south west metropolitan area of Perth, between January 1st 2002 to December 31st 2006. The results showed 1-day lagged exposure to NO and CO was significant risk to emergency presentation for asthma on patient's age between 0-4 years old. the day before emergency department presentation is association to The period of exposure (Pereira et al., 2010). As Barnett's study, he found air pollution had association with childhood hospital admissions (Barnett, 2005). In Toronto Canada, Children who exposed to ambient air pollution had effect on respiratory infections but small association (Lin et al., 2005).

Zeft studied association between exposure of $PM_{2.5}$ air pollution and systemic inflammation and the clinical presentation of various cardiopulmonary heath events juvenile idiopathic arthritis (JIA) 1993-2006. The subjects were 338 JIA cases living on Utah's Wasatch Front. The results showed that significantly associated with increased concentrations of $PM_{2.5}$ and risk of JIA onset in preschool aged children, but not in older age (Zeft et al., 2009). Bagheri Lankarani studied the exposure of NO air pollution effects on peak expiratory flow rate in primary school students in District 12 of Tehran. The subjects were results 356 female and 206 male students who were included and test for the lung function over the 6 weeks. The results showed that strong associations between students whose poor lung function and NO air pollutants exposure (Bagheri et al., 2010). PM_{10} are association with respiratory hospital admission in Darwin Australia (Johnston et al., 2007). Orazzo and colleague studied

triggering of wheezing in children, they found associated between air pollution in six Italian cities and wheezing (Orazzo, 2009).

2.4.5 Other cardiovascular

Mobility in cardiovascular relates to air pollution. Investigators studied by using CCO study and provide the results is significant, instance for Yi studied Seasonal effect of PM_{10} concentrations on mortality and morbidity by used a temperature-matched case-crossover analysis that same rounded to degrees Celsius, month, and year. The periods of study was 2000-2006 and 2001-2006. 238,826 deaths were identified in Seoul, Korea. The inpatient admissions 98,570 for cardiovascular and 93,553 for respiratory diseases. They found the association between effect of PM_{10} on mortality and morbidity that varies with season and increases in the summer season period (Yi et al., 2010).

Chang studied air pollution and hospital admissions for cardiovascular disease in Taipei during 1997-2001. The conclusion was exposure to higher levels of ambient air pollution association with cardiovascular hospital admission statistically significant Chang et al., 2005). It different from surveillance of the short term health effect of $PM_{2.5}$ on cardiovascular in New York State, that showed as small relationship and difficult to measure with precision (Haley et al., 2009). In Perth Australia had similar result as Haley, they found a small number of significant relation between cardiovascular disease and air pollution (Hinwood et at., 2006). Expose air pollution in Sensitive group has the effect on health adverse as cardiovascular which strong association than other group (Peel et al., 2007).

2.4.6 All others

They are more diseases which relates to ambient air pollution including mobility and mortality. In the term of CCO design, in which provides the evident that support the hypothesis of this dissertation.

Otitis Media and air pollution significant association was showed in Zemek's research. This study they aimed to investigate the association between ambient air pollution exposure and Emergency Department Visits for Otitis Media in Edmonton, Canada. They used ten years of Emergency Department data in Edmonton. 14,527 Emergency Department visits for Otitis Media over 10 years in children aged 1-3

years were recruited. They applied a time-stratified case-crossover technique to analyze. Statistic used Conditional logistic regression analysis with the subject's ID as a stratum variable and adjustment for meteorological factors. The results showed statistically significant positive associations between Emergency Department visits and Otitis Media, which interquartile increases in carbon monoxide (CO) and nitrogen dioxide (NO_2), Specially in the warmer months (Zemek et al., 2010).

Myocardial Infarction (MI) can occur because exposure to the air pollution. Such as, Rich studied association between acute increases in fine $PM_{2.5}$ and increased risk of MI in both transmural and nontransmural infarctions. The subjects were all hospital admissions from 2004 – 2006 who came for first acute MI. 5,864 adult residents of New Jersey who lived no more than 10 km from a $PM_{2.5}$ monitoring site were enrolled. The results showed that overall of MI was not relation to each interquartile-range increase in $PM_{2.5}$ concentration, but was associated with an increased of a transmural infarction. This study found no association between $PM_{2.5}$ and nontransmural infarction. So earlier studies of PM and MI should be stratified by infarction type (Rich et al., 2010). Hsieh studied air pollution and hospital admissions for MI in a subtropical city Taipei, Taiwan, 1996-2006. The results showed that on the warm days were statistically significant positive associations with all pollutants except SO_2. On cool days were significantly associated between increased all pollutants and increased MI admissions except SO_2. Increasing in O_3 and NO_2 were significant both on warm and cool days effect to admissions for MI. Conclusion that increase of ambient air pollutants levels significantly associate with increase the risk for MI (Hsieh et al., 2010). Berglind studied the association between ambient air pollution exposure and first time for myocardial infarction in Stockholm 1993-1994. The subjects were 660 first-time myocardial infarction cases who were interviewed shortly after diagnosis. The results showed that no relationship between trigger the first time of myocardial infarction onset and 2-hour or 24-hour air pollution exposure (Berglind et al., 2010). CO is the pollutant that associated to hospital admission for myocardial infarction (Cheng et al., 2009). Myocardial infarction in Rome was found increasing relevant to air pollutant level, especially in the warm season (DIppoliti et al., 2003). O_3 and NO_2 were association to higher hospital admissions for myocardial infarction in Taipei Taiwan (Hsieh, 2010). Ruidavets showed the result, O_3 was association to myocardial infarction but NO_2 and SO_2 no relation (Ruidavets et al.,

2005). Control for the confounder has the effect for study, if study did with a small confounding the result could be no consistent association (Sullivan et al., 2005).

Cardiac relates to the level of air pollution, as Silverman and colleague examined association between ambient fine particles (of aerodynamic diameter less than or equal 2.5 mum, or $PM_{2.5}$) with out-of-hospital cardiac arrests in New York City during the years 2002-2006. 8,216 subjects who out-of-hospital cardiac arrests of primary cardiac etiology were recruited. The variables season, day-of-week, same-day, and delayed/apparent temperature were controlled. They found that an increased risk of cardiac association significant with $PM_{2.5}$ in the warm season (RR = 1.09, 95% CI: 1.03, 1.15) but not the cold season (RR = 1.01, 95% CI: 0.95, 1.07) (Silverman et al., 2010). Anderson studied air pollution and activation of implantable cardioverter defibrillators in London. The subjects were 705 patients experienced 5,462 activation days that were included. The results showed that was little evidence for relationship between air pollutants exposure and activation of implantable cardioverter defibrillators in London. The pollutants, which had positive associations to this adverse effect from secondary sources, more than primary sources as from transportation (Anderson et al., 2010). Tsai and other studied the association between air pollution level and emergency room visits for cardiac arrhythmia in a subtropical city Taipei, Taiwan 2000-2006. The results showed that increased in all air pollutants had statistically significant positive associations with emergency room visits for cardiac arrhythmia in Taipei, but except SO_2. These provided evidence for the two pollutants model as O_3 and NO_2 remained association on both warm and cool days with significant statistic (Tsai et al., 2009) which similar Chiu's study. Dennekamp studied association between outdoor particulate matter (PM) air pollution and out-of-hospital cardiac arrests Melbourne, Australia 2003-2006. The subjects were 8,434 adult's age over 35 years who were identified through the Victorian Cardiac Arrest Registry were include and excluded arrests with an manifest preceding non-cardiac diseases. The results showed that increase in an interquartile range of 4.26 $\mu g/m^3$ in $PM_{2.5}$ over 2 days (lag 0-1) was relevant to an increase in risk presentation for an out-of-hospital cardiac arrest which strongest in $PM_{2.5}$ exposure. The subject who age between 65-74 years old were most sensitive to $PM_{2.5}$ exposure, while people who age more than 75 years old had the lowest risk for out-of-hospital cardiac arrests

(Dennekamp et al., 2010). Particulate air pollution was associated with 13% (95%CI 1.3-26.2) increase in heart failure (Pope et al., 2008).

Ischaemic Stroke associate with short term effects of traffic exposure to ultrafine particles (UFPs), was showed in Copenhagen, Denmark that Andersen studied in 7,485 stroke admissions, 6,798 were ischaemic and 687 haemorrhagic, 3485 mild, and 2248 severe all of them were included. The results showed that was significant positive association between exposure to UFPs and ischaemic strokes (Andersen et al., 2010). The exposed O_3 was relate to ischaemic stroke in Dijon France (Henrotin et al. 2007). Congestive heart failure relate to ambient particulate air pollution, increase in PM associated with increase in hospital admission for congestive heart failure (Wellenius et al., 2006). O_3 and NO_2 were significant associate with congestive heart failure (Yang et al., 2008).

Coronary relates to air pollution in CCO study done by Serinelli and other. They evaluated the association between Particulate matter (PM_{10}) and out-of-hospital coronary deaths in eight Italian cities during 1997-2004.16,989 subjects aged more than 35 years who died out-of-hospital from coronary causes were recruited. The subjects were hospital admissions in the previous 2 years identified. They studied the effect of the mean of current and previous day PM_{10} levels (lag 0-1). They used a time-stratified case-crossover analysis to approach which controlled confounders for weather, holidays, influenza epidemics, and summer decrease in population. They estimated the pooled percentage increase (95% CI) in mortality per 10 microg/m3 increase in PM_{10}. This study they found statistically significant relationship between short term exposure to PM_{10} and coronary mortality. The subjects who were the lowest socio-economic had a stronger effect than the highest socio-economic people, especially among the elderly and low socio-economic (Serinelli et al., 2010).

Cerebral hemorrhage and air pollution are relationship. Such as Ye studied the association between air pollution and acute onset of cerebral hemorrhage in Hangzhou city. This study found that there was significantly relationship between short effect of exposure on air pollutants and the acute onset representative for cerebral hemorrhage. Both in the spring and in the first half of the year had a strong association (Ye et al., 2009).

Hypoxia associates with air pollution in Schreijer's study, in the study They determined activation after air travel involved with hypoxia, stress, inflammation or

viral infection. The subjects were 71 healthy volunteers who exposed to an 8 hours flight, 8 hours movie marathon and 8 hours of regular activities, compared to markers for several hypothetical pathways as plasminogen activator inhibitor-1 (PAI-1), stress, plasma factor (F)VIII coagulant activity (FVIIIc), soluble P-selectine (sP-selectine), interleukin-8 (IL-8) and neutrophil elastase. Earlier an activated clotting system, as evidenced by thrombin generation, in 17% of volunteers after the flight were reported. Our results do not support the hypotheses that stress, infection or air pollution are involved in the development of a prothrombotic state in air travellers. The result showed that after long air travel may be the risk caused to hypoxia (Schreijer et al., 2010).

suicide attempts associates with air pollution, was showed by Szyszkowicz's study. The subjects were emergency department visits for in vancouver, Canada. They collected emergency visit hospital data in Vancouver, Canada. They found both hierarchical and case-crossover methods had statistically significant positive associations among carbon monoxide (CO), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), and particulate matter (PM_{10}) for all suicide attempts in the cold period. were not statistically significant in the warm period (Szyszkowicz et al., 2010). Kim studied the association between exposure to ambient particulate matter and suicide in urban settings during a 1-year period in 2004. In which 4,341 suicide cases in seven cities in the Republic of Korea were subjects of research. Hourly average concentrations of PM_{10} at 106 sites in the 7 cities areas and $PM_{2.5}$ at 13 sites in one city were measured. The results showed large associations were increase in suicide risk associated with an interquartile range increase in PM_{10} and $PM_{2.5}$, especially for personal with presentation for cardiovascular disease (Kim et al., 2010).

Asthma relates to air pollution, Yamazaki studied the association between modifying effect of age on short-term exposure to outdoor air pollution and night-time primary care visits representative for asthma in metropolitan Tokyo. The subjects were 308 children aged 0-14 years and 95 adolescents and adults aged 15-64 years, who visited to for an asthma attack at 7 PM and 12 AM time periods. The results showed that significantly association between ozone and night-time primary care visits for asthma attack in warmer months. The study evidence associated was stronger in preschool children, and no relationship between O_3 and health outcome of study in adults (Yamazaki et al., 2009). It different form Boutin's study, that O_3 was association with

emergency room visit for asthma (Boutin-Forzano et al., 2004). Children in Australian who asthma emergency visit had association with air pollution as PM, O_3 and NO_2 (Jalaludin et al., 2008). The emergency outpatient in Soul who came for asthma had association with air pollution, strongly for NO_2 (Kim et al., 2007). Children age less than 15 years old who hospital admission for asthma relation with PM_{10} level significantly effects (Tecer et al., 2008). NO_2 relevant to hospital admissions for asthma, although on cool days remained statistically significant (Tsai et al., 2006). Outdoor air pollution levels positive associate with asthma and strongest in young children (Villeneuve et al., 2007).

Hypertension and air pollution are association. As study of Guo and colleague, that they studied gaseous (SO_2,NO_2) air pollution and emergency hospital visits for hypertension in Beijing, China. They found positive associated between urban gaseous air pollution and increased emergency hospital visits for hypertension in Beijing, China. Other study the association between explore the risk effect of PM air pollutants and emergency hospital visits for hypertension in Beijing, China, 2007 of Guo's study. In this study period, 1,491 subjects were included cause of hypertension emergency hospital visits. The results showed that an increase in 10 $\mu g/m^3$ in $PM_{2.5}$ and PM_{10} was significantly positive associated with emergency hospital visits for hypertension (Guo et al., 2010).

Chronic obstructive pulmonary relates to air pollution level, was showed in CCO study such as Sauerzapf studied association between environmental factors and hospitalisation admissions representative for chronic obstructive pulmonary disease in Norfolk (a rural county of England) 2006-2007. The subjects were 1050 admissions for COPD during the study period. The results showed that significantly positive association between increase in CO and increase in the COPD hospital admissions in the rural area of England (Sauerzapf et al., 2009). Infant bronchiolitis had a little number associate to ambient air pollution (Karr et al., 2006). The study in Kaohsiung Taiwan showed association between COPD hospital admission and ambient air pollution specially CO and O_3 (Lee, 2007).

Pneumonia in the subpopulation are greater risk to air pollution exposed. The evidence was showed in Cheng's study. They found air pollution levels association with hospital admission for pneumonia (Cheng et al., 2009). Chiu controled confounder as whether and found out ambient air pollution related to the risk of

hospital admission for pneumonia (Chiu et al., 2009). Increasing in O_3 related to pneumonia admission was confirmed by Medina's study (Medina-Ramon et al., 2006)

Ventricular arrhythmias is association with air pollution level. The moderate increases of air pollutants display relationship to arrhythmias (Ljungman et al., 2008). O_3 was associations with 21 % increased arrhythmias (Rich et al., 2005).

Bronchiolitis cases relates to the ambient air pollution level such as PM_{10}, SO_2, and NO_2 when control for long term trend, seasonality, holiday, public holiday, weekday and meteorological variables (Segala et al., 2008).

2.5 Case crossover methods

The beginning of epidemiologic studies on the impact of air pollution on morbidity and mortality were initial consequence in the decades 1930 to 1960 which used simple graphic to representation or comparisons of mortality rates for association between air pollution and health effects (Carracedo-Eduardo, 2009). Long time series are required for air pollution level cause of the fallen substantial to estimate and consequence that have the effects on health. In 1970s the epidemiologists began to use regression equation to consisted the association between health effects and exposures of air pollutant that distributed over time. The autoregressive integrated moving average models (ARIMA) was used to control residual auto-correlation. The problem of ARIMA is dependent variable is not distributed normally because extremely rare in the daily morbidity and mortality events count. Poisson regression model is the parametric approach was used to control for time trend in 1990s. the distribution of event counts have a Poisson distribution, so time variable and its transforms, quadratic and sinusoidal functions of the different frequency and amplitude. This model used to control the effect on outcome variables of unmeasured variables that vary with time trend, e.g. PM concentration, meteorological variables, influenza outbreaks, and population change, or that may crisis of politics as Bangkok Thailand in 2009 and 2010. The problem of Poisson regression is unmeasured variables a cyclical component of varying frequency and width cannot be easily adapted in the parametric functions, So generalized additive models (GAMs) were used because functions of the variable time is nonparametric and appropriate for irregular cyclic components of unmeasured variables. GAMs can use for reduce the confounding factors cause of allow flexible fits variables, e.g. temperature, relative humidity, and barometric pressure in to the models. Some variable as heatwaves have effect on health, such as Tong used Case-crossover analyses to Assess the effect of heat that related health impacts in Brisbane, Australia. The daily data on climate, air pollution, and emergency hospital admissions in Brisbane between January 1996 and December 2005; and mortality between January 1996 and November 2004 were used. The result showed that a statistically significant During heatwaves for increase in emergency hospital admissions and mortality, although a small change in the heatwave definition had effect on the estimated health impact (Tong et al., 2010). The problem of GAM is

number of degrees of freedom must be specified by researcher, so it can lead to bias in the model, Therefore, the epidemiologists use case-crossover to control for the time trends and individual confounding such as sex, age, physical exertion, or smoking (Maclure, 1991).

Case-crossover (CCO) design was created by Malcolm Maclure in 1991. He attempted to test hypotheses concerning the immediate short-term determinants of myocardial infarction. His question was "who would be the best representatives of the population base that produced the cases?" and found the answer was the cases themselves. The design referents are sampled from the case their own history. The simplification control selection is an advantage of this study. The case-crossover design is a class of designs in air pollution epidemiology that aim to estimate the short acute effects of air pollution. Referred to as short term health effect studies, CCO designs enable to evaluation the effect of day-to-day variation in exposure of air pollution on morbidity and mortality. Various adverse health events such as cardiovascular disease, chronic obstructive pulmonary disease have been studied, including deaths of disease as myocardial infarctions. Ambient air pollution concentrations are measured at centrally of the city and monitors in particular geographic regions to represent as the exposure of air pollutants in that city. Therefore, the same exposure is used for all index time in the study. The referent to this subject as a shared exposure series are in the exposure period. Particulate matter has been the most popularly air pollutant to studied the impacted of health. There are many challenges to study the short term health with including error in the measurement of exposure air pollution and the discordance between ambient and personal exposures of air pollution. Confounding is also a major concern such as Time-independent confounding that can occur in the same as a shared exposure series, if subjects are measured at different points in time. Time-dependent confounders have majority problems in CCO's study (Checkoway et al., 2000), instance for seasonality or other pollutant's concentrations in the period of studying. These factors are very strongly association with day-to-day variation between exposure of air pollution and many morbidities or mortalities. The effect of air pollution exposure in general population is very small, therefore biases are especially troublesome can occur in studies because the effects of time-dependent confounders many times larger. For example to estimate the risk of cardiovascular disease associated with variety of air

pollution exposures, for CCO study, one or more referent time are chosen at pre-specified intervals prior to the index time.

The case-crossover study is design to represents novel approaches that can be control for confounding variables as time tend. Instance for subjects who experienced the event of interest like got a diseases after expose to air pollutants in the short term effected, exposure just prior to the event, this period is the index time. CCO is used index time to compare with exposure at comparable control (referent times). Case-crossover is conceptually similar to matched case-control. The case and control are perfectly matched on potential confounders. Subjects in case-control the controls are people, but subjects In CCO the "case" is the pollution level on the day when the health event occurred, and the controls (referents) are days when this event did not occur. The exposure of air pollution at each index time, event occur which is part of a matched (referent window) set of exposures consisting of exposures for the subject at his/her referent times. Referent window can make within-subject comparisons, time-independent confounders, such as sex, age, smoking history, occupational history, and genetics, are controlled by design. For example, the investigators interested in study of CO and myocardial infarction. The time when event of myocardial infarction occur (case) is called the index time. The referent time (control) is able to be the same time of day in 24 hours and 48 hours prior to the index time. Then case was exposed to CO at the time of event and at either of the referent times, these time together form a matched case-control set. Statistic to analysis of this matched sets from different individuals and used conditional logistic regression similar to use in standard matched case-control study. The advantages of CCO, the referent times are matched to the index time with control to time-independent confounders, e.g. the referents time are restricted to the same season as the index time; these effects are controlled by design of the study. This should used other approaches to estimation the acute pollution effects, instance for time series studies in which time-independent confounding is controlled by modelling. The selection of control or referent times is an important issue in the case-crossover design, if choose the wrong referent times that the estimate value could be fail. This problem can called referent selection strategy or referent scheme that mean the choosing of design in CCO. So it is important to control for time-dependent confounding. Including, the CCO makes the confident assumption that no time trend in exposure within the referent window. Besides, only with certain

referent strategies are the assessment model normally used the conditional logistic regression (CLR) to estimating the equations that unbiased. This bias is called overlap bias in the conditional logistic regression estimating equations. Although in the large samples this Bias can be happen in estimating equation that is problematic. The bias began since it implies the associated parameter estimates. Using of the CCO method is limited by the requirement that exposures should appear for non time tend biases. Selection control or referent time will be failure of the condition. The strong condition need to evasive bias. The essential to control for time trends in exposures of air pollution is to propose a variant of the CCO design (Navidi, 1998). This design is a full stratum bi-directional (FSBI) case-crossover method. The method allows for referent times (control) are chosen before and after the index time. Navidi's FSBI method approach is based on an analogy same cohort studies which sampling scheme. Even Maclure's case-crossover approach is similar to case-control studies with time tend bias. For FSBI the method is the ordered time series of exposures of air pollution treat as fixed, and index times are randomly. The full stratum bi-directional CCO method is similar to a special case of the self-controlled case series method earlier developed by Farrington in 1995. The distinction between case-control and cohort sampling schemes is necessary to comprehension the diversity between case-crossover designs, such CCO's method lead to truth likelihoods with truth properties. FSBI's model supposes that the underlying index time rate, the situation is constant over during time of data collection. It is not appropriate for events that may appear on time dependent variation. Investigator conducted a range of methods by pretence, developed a new method that called the symmetric bi-directional (SBI) design. This method choose neighbouring referent by times symmetrically before and after of the index time. The referent time are chosen in fixed and relates to index times. This method conduct similar Maclure's original referent sampling scheme strategy while unification Navidi's advice of using referent times on either sides of the index time. the symmetric bi-directional method is not yield steady estimates like FSBI method does, So the resulting bias in the SBI method is called overlap bias. The new method becomes assumed for necessary accommodation on FSBI design to short time windows preferably than application to the all period of data collection. This method is called the time-stratified (TS) case-crossover study that can avoids overlap bias because it is assumed that index time rates are constant only over short term effect. TS

method observes in the short periods that is the same as another special case of the case series method. A further approach is proposed that called the semi-symmetric bi-directional (SSBI) method. It can remove the resolution dependence of referent and index times by conducting a random element in the selection of referent periods. SSBI method is not based on a standard CLR likelihood. Janes proposed the adjusted semi-symmetric bi-directional method (ASSBI), it a simplified that attempted a classification of the various methods in order to characterize those that produce overlap bias (Janes et al., 2005). but conclude that a satisfactory heuristic explanation of overlap bias still eludes them. It has been claimed that the time-stratified case-crossover method controls for time dependent confounding, but residual confounding from temporal effects are consistently bias to estimates the obtain by using the TS method. Case-crossover methods are used to studying the environmental exposures that are frequently contrasted with time series methods. Both of the FSBI and TS models are equivalent to Poisson regression models with dummy time variables. Some of case-crossover models are equivalent to time series models. It has a different choices of adjustments for temporal confounders. Formulation of distinction between overlap bias and bias cause of un-model temporal trends is less display.

The CCO method appropriate for air pollution exposures and outcomes. The design can assess the association between a short term effect of exposure and the risk of an acute event. It suitable to analyze transient exposure effects. The relevant of air pollution exposure is shared among all individuals, therefore, the likelihood of the data is appropriate to condition on this fixed exposure series that mean likelihood can be thought of as the probability model for the data. However, when estimate the properties of the effect should condition on the exposure. Because has only one exposure series that the estimate and the corresponding estimating equations are unbiased for this specific exposure series. If has a little value of averaged over all possible exposure series, it can be unbiased. The standard regression analyses, instance for, linear and logistic regression are condition on exposure. The ambient air pollution exposure data is exogenous; they are generated independent of the individual underlining study. If compare with endogenous exposures, the observe of individual have influent for endogenous exposure when it change as a result of having experienced the event. Therefore, personal air pollutant exposure is an endogenous exposure. Attributes of the CCO design is very useful in the Air Pollution study. The

strengths of the case-crossover design are no requirement a control sample, so can avoids bias associated with improper control selection. It makes effect modification estimation the relatively simple and controls for fixed confounders by design. The time-dependent confounders can be controlled by CCO design and matching referents time to the index time. The methods are used to assess the association between short-term air pollution exposure and health effects are time series study and CCO. The difference between time series regression and conditional logistic regression in CCO is that the time series requires modelling to control confounders. In which, a conditional logistic regression analysis can control the independent confounding effects of all matching variables by design. The time dependent confounders are controlled by modelling in the conditional logistic regression model.

The time-varying exposure model in CCO is the statistical model. The first postulated for the CCO study was a precipitating event model. This model assumes that time can be the process of making mathematically discrete into exposed and unexposed periods. It conditions that a subject is at high risk for a fixed time or index time following an exposed period and thereafter returns to background risk until the next exposed period. This mean the under certain conditions of the model is subject to length bias. However, the proportional hazards model for a rare disease in first proposed for CCO studies with a constant baseline hazard for each individual is appropriate for air pollution exposures which are not binary. This is called the time-varying exposure model. The time-varying exposure model states is only one event for each subject that meaning is a legitimate assumption for a rare event and specifies risk such a function of time and exposure. CLR is used to estimate coefficient in the time-varying exposure model. This method has been motivated by the analogy to matched case-control designs. The conditional logistic regression likelihood is exactly the likelihood of the data for matched case-control. CCO design is to control for confounding by making comparisons within referent windows and the condition on the referent windows in the analysis are required. Other model as Mantel-Haenszel (MH) estimator was used in early case-crossover studies with binary exposures. MH analyzes only one referent for each case. The CLR is a better choice than MH. Because of non-binary exposures can be used with this model. The control of additional confounders not used in the matching. Therefore these factors can be included in the regression model.

The potential biases associate with the choice of referent selection strategy (e.g. symmetric bidirectional referents) in the CCO studies. The likelihood of the data for the CCO study depends on the choice of referent selection strategy. The CLR estimating equations are only unbiased for certain referent selection strategies. For most of the commonly used referent selection strategies, some design could be overlap bias as the conditional logistic regression estimating equations are not unbiased. The CCO design relies on the assumption that there is no trend in exposure within the referent window. It is required because the effect of exposure is assessed by contrasting exposures at the event time and referent times. If referents are prior to the event time and there is a decreasing trend in exposure over time, the effect estimate will be negatively biased. In which, the strong long-term time trends, bias due to time trend is a concern to present in air pollution data. Holly Janes said "the case-crossover design assumes that the referent exposures are representative of the usual distribution of exposure, and that the index exposure represents the exposure that generated the event." The classes of referent selection strategies, for example unidirectional, full stratum bidirectional, symmetric, semi-symmetric, and time stratified as mentioned, a number of fundamentally different types of referent selection strategies have been used in air pollution studies. The proposal of taxonomy for referent selection strategies is groups that correspond to the statistical properties in CCO designs. Localizable or non-localizable, ignorable or non-ignorable are the way to classify designs. Localizable designs are the design that likelihood of the index times conditional on the referent windows contains information about coefficient. In the different of a non-localizable design, the conditional likelihood is uninformative for coefficient. Instance for non-localizable design is the symmetric bidirectional design because the index time is fixed in the centre of the referent window. The index time's location within the referent window is no information about coefficient. For example, the referent sampling strategy of CCO which are localizable as the time stratified, full stratum bidirectional, and semi-symmetric bidirectional designs.

Localizability is wishful when assessment can be based on comparisons to the referent windows. This comparison is assumption matching on time dependent confounders, that mean these confounders are controlled. Only the localizable designs are classified as either ignorable or non-ignorable. Ignorable design has a referent sampling scheme that can be ignored when analyzing the data. CLR can be used to obtain unbiased

estimates. The other method, a non-ignorable design, the likelihood of the data depends on the referent sampling scheme and this likelihood must be used for an unbiased analysis. this definition of ignorability that use in the missing data context. It implies the data can be analyzed as if the observed data are the complete data. The TS and FSBI designs are localizable and ignorable. The referent sampling strategies are relate to choosing statistical analysis. CLR yields unbiased estimates for localizable or ignorable designs. although, the CLR estimates have overlap bias under non-localizable or localizable for non-ignorable referent selection. In which, the likelihood of the data in non-localizable design must be used to obtain unbiased effect estimates, but application is impractical. In case of a localizable and non-ignorable referent scheme can be again the likelihood of the data that must be used for unbiased estimation, but there is a simple way as described subsequently to obtain these method. They have many kind of selection referent strategy, So the detail in each referent selection strategy will explanation more as below.

2.5.1 Unidirectional CCO design

Maclure proposed the CCO unidirectional design which selects the control only one point before the index time (Maclure, 1991). A unidirectional design restricts to selects and control confounding due to season and day of the week that are controlled by selecting referents time for on the same day of the week as the index day. All possible confounding factors are undergoing no change between referent time and index time, automatically these controlled for by design. This design is non-localizable. Using conditional logistic regression to estimate but have overlap bias. Unidirectional sampling has a major disadvantage in air pollution studies, such as when selecting referents only prior to the index time it can lead to time trend (long term trend) bias. The bias will be larger the further referent time are from the index time, So unidirectional sampling strategy is not commonly for air pollution and health effects studies.

2.5.2 Full stratum Bidirectional CCO design

Greenland's study was the first to recognize the trap of unidirectional referent selection in the presence of the time trend. Navidi proposed CCO full stratum bidirectional design which selects control for each case, all the days of the series other

than that of the event taken as controls (Navidi, 1998). The time trend or long term trend bias could be eliminated by choosing referents time both before and after the index time. This strategy called bidirectional referent selection. Validity of the technically of bidirectional sampling is only cases which are still at risk after index time and assumption that is certainly not valid when the event is death. The advantage of FSBI is provided control for long term trends. The bidirectional sampling was justified by air pollution data. In which the exposure is exogenous and available for all cases both before and after the index time. A strong justification was proposed by Lumley and Levy, They showed the bias cause of sampling referents after the at risk period is small in a rare event. More importantly, the bias associated with sampling referents in full stratum bidirectional design after the time at risk is smaller than the bias in unidirectional referent selection for a time trend. Navidi proposed FSBI referent selection in which the referents are all days in the exposure series other than the event day. Interestingly, Lumley and Levy showed that CCO analysis with FSBI referents and a shared exposure series is equivalent to a Poisson regression analysis. However, while bias due to time trend is controlled, but time dependent confounding is not controlled for example seasonality, therefore time dependent confounding must be controlled by modelling. Because the referent window is large than index time that confounding cannot be controlled by the design of FSBI study. The full stratum bidirectional design is a localizable, ignorable design; consequently that the overlap bias is not an appeared in its design.

2.5.3 Symmetric Bidirectional CCO design

Bateson and Schwartz (1999) proposed CCO symmetric bidirectional design which selection of control in the two points at equal distance of the event, that provides adequate control for long term trends and seasonality. SBI is a popular alternative to the full stratum bidirectional design. This design can controls for bias cause of time trend and confounding by both season and day of the week, if referents time are within the same season and the same day of the week as the index time. The imitation studies show confounding bias in the shorter lags are less than longs lags, but confounding is not well controlled when the seasonality pattern of exposure is not symmetric. The disadvantage of the SBI design is that it is non-localizable. Therefore, CLR estimates are subject to overlap bias.

2.5.4 Semi-Symmetric Bidirectional CCO design

Navidi and Weinhandl proposed the semi-symmetric bidirectional design which selection of control by chosen one from the two randomly (Navidi and Weinhandl, 2002). That mean the semi-symmetric bidirectional design, the method is randomly one referent from days at a fixed lag before and after the event occur. If only one of these days is available (due to the case being at either end of the exposure series), it serves as the referent. If the lag is small and a multiple of seven, confounding by season and day of the week can be controlled by design. That advantages is provides adequate control for long term trends and seasonality. There is no bias cause of time trend. The referents are bidirectionally sampled; therefore, this design is a localizable and non-ignorable. The likelihood of the data must be used in order to obtain an unbiased effect estimate, and standard CLR estimates have overlap bias. however, in this case estimates based on likelihood can be obtained by using standard conditional logistic regression.

2.5.5 TS-CCOdesign

Lumley and Levy proposed the time stratified design which selection control from one or several within the same time stratum in which the event occurred (Lumley and Levy, 2000). The design is not subject to bias because time trend for this design is no pattern in the placement of referents associate to the index time. In addition, the design is used matching time dependent confounding to controls biases. For example, restricting referent periods are selected with the same day of the week, month, and year as the index day. TS CCO design can controls for seasonality and day of the week, it is a localizable and ignorable design. Therefore, Conditional logistic regression (CLR) can be used to obtain unbiased effect estimates. So, the advantages of time stratified design has provides adequate control for long term trends and seasonality, in addition, some interesting relationships with other designs, such as the full stratum bidirectional design that is a special case of a time stratified design in which there is one large stratum, although in this case confounding must be controlled by modelling. Conditional logistic regression analysis of a shared exposure series with time stratified by year, month, and day of the week that method is the same as Poisson regression analysis with dummy variables have been adjusted for day of the week within each month, and month within each year.

Eduardo used A systematic review analysis to studying effects of air pollution on adverse health events, CCO was used to application in this subject. They review 105 papers that fulfilled the inclusion criteria, 24 addressed methodological aspects of CCO design in 1999-2008. The result showed that symmetric bidirectional and TS-CCOdesign were used more than others methods. Eduardo reviewed of methodological aspects revealed a trend in CCO bidirectional designs with referent selection strategy to the choice of control periods and trend in use of CCO methods for analysis association between the short term health effect and air pollutants level (Figure 1). Therefore, in this dissertation focus on TS-CCOdesign (Eduardo et al., 2010).

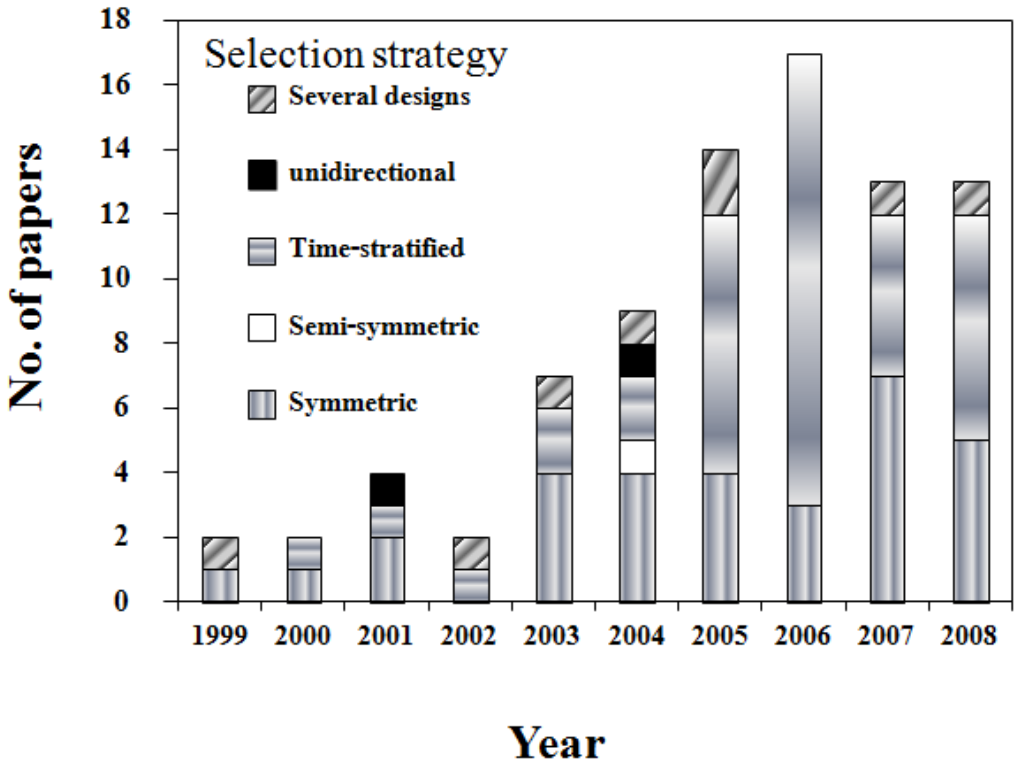


Figure 1: Trend in the use of different CCO methods for analyzing the short-term relationship between air pollution and health.

Source: Eduardo et al., (2010).

2.5.6 Overlap Bias on CCO design

Lumley and Levy, who are the first that used the term, overlap bias. They found CCO overlap bias same as matched case-control studies. The meaning of overlap is something misleading in which an individual's set of possible referent windows, these overlapping to subject is called overlap bias. In the full stratum bidirectional and symmetric both of them are not in the case of overlap bias. The referent windows of bidirectional designs is overlap, But for the full stratum bidirectional design is free from overlap bias when the symmetric bidirectional design is not overlap bias. Overlap bias is same as the bias that relationship with using unconditional logistic regression in a match case control study. The phenomenon of overlap bias is undefined by the index time, even though it fixed or no fixed within the referent window. Inspite of the existing referent sampling strategies with index times fixed within the referent window are subject to overlap bias, but with random index times are not such as time stratified design. It would be possible to design a referent sampling strategy with a random index time that is subject to overlap bias. therefore, The bias is purely mathematical; whether or not bias exists for a particular referent sampling strategy depends on the form the likelihood of data.

This dissertation focus on time stratified case-crossover design because it suitable to the study of the association between short terms effect of air pollution exposure and acute adverse health event as mortality in Bangkok. The design makes within person comparisons confounding by time independent confounders is eliminated. Even though, referents are matched on potential time dependent confounders, these effects are also controlled by modelling. Effect modification can be estimated and standard conditional logistic regression methods can be used for analysis. Most of time dependent confounders and time trends and autocorrelation in air pollution exposure series, the CCO design with consistency of referent selection is particularly essential. Because air pollution exposures have confounders that tend to dominate the exposure effects. Referents were matched on the most dominant time varying confounders, and sampled including before and after the index times. Sampling referents period too near to the index time result in a loss of power due to autocorrelation in the exposure series, this study used length of sampling is 7 day in each lags. To increase efficiency used more referents (Kunzli N., 2005), If there remains a choice as to the number of referents after these concerns are taken into account. In the analysis was condition on

the fixed exposure series and used conditional logistic regression yields biased estimates. Therefore, this study wise to avoid overlap bias entirely. Localizable, ignorable referent schemes as TS CCO (assume that the exposure is exogenous and the outcome is rare) allow for unbiased estimation and using a standard conditional logistic regression analysis, it was appropriate for this dissertation. The advantage of TS CCO is to avoid the overlap bias and bias due to time trend. The design can be tailored to match on the most important time dependent confounders such as season, temperature, and humidity. Even though, the semi symmetric bidirectional method can achieve these goals, but it requires modification of the traditional conditional logistic regression analyse data that was less efficient than a time stratified design because semi symmetric bidirectional use a fewer referents period. Cox proportional hazards model with time dependent covariates is appropriate to estimate short term effect of air pollution on health (Lepeule et al., 2006). Epidemiologic studies in short term health effect of air pollution by using date of hospitalization had have under-estimate due to misclassification of time of event onset (Lokken et al., 2009). 8 hour onset period relevant to air pollution level than 24 hour onset period (Symon et al., 2006). The referent sampling approaches should be greatly for CCO analysis, if sampling did not greatly alter it could be no significant (Xu et al., 2008).

2.6 Time Series analysis

Herman Wold Swedish mathematician edited a Bibliography on time Series in 1965 that was published by The International Statistical institute. Herman compiled the bibliography from 30 countries. in 1938, Wold's monograph on stationary time series analysis that complete frequencies, relative frequencies, and cross tabulations for each study. Time series analysis has flourished since 1980. In 1970-1990 Time Series used as the techniques for economic analysis that treatise was edited by Jose Carvalho (Judy et al., 1997).

Article about air pollution and Time Series was found in 1972, Egorov studied the aliphatic alcohols in work zone (Egorov, 1972), Merz studied aerometric by use time series to forecast (Merz, 1972). These information was searched from PubMed on EndNote program.

The model of time series provide estimates exponential smoothing, univariate autoregressive integrated moving average (ARIMA), and multivariate ARIMA models, and produces forecasts in the analysis. The performance combine an expert modeller which automatically identifies and estimates the best fitting ARIMA or exponential smoothing model for one or more outcome variable series, therefore excluding the necessity to identify an fit model through trial and error. The Statistics as goodness of fit measures such stationary R-square, root mean square error, mean absolute error, mean absolute percentage error, maximum absolute error, maximum absolute percentage error, normalized Bayesian information criterion. Residuals can present by autocorrelation function, partial autocorrelation function, Ljung-Box Q. For ARIMA models: ARIMA orders for dependent variables, transfer function orders for independent variables, and outlier estimates. Also, smoothing parameter estimates for exponential smoothing models.

Smoothing methods are fit when a time series appear no statistically significant effects of a stable. The purpose is to smooth out the non regular component of the time series by using an averaging process. Therefore the time series is smoothed when it is used to produce forecasts. The moving averages method use smoothing technique, such as smooth the time series. The method uses the average of a number of contiguous data points. The overlap of observations are used to generate by averaging process. Instance for predictors want to generate three period moving averages. They must lead the first three observations of the time series and calculate the average. After that they have to drop the first observation and calculate the average of the last observations. The term of moving is the way to averages are calculated. In analysis may experiment with different length moving averages that choose the length which yields the highest accuracy for the forecasts generated.

The forecast errors can be positive and negative. Therefore, calculation a simple average of forecast errors over time may not capture the true magnitude of forecast errors. The large positive errors may simply erasure out large negative errors. The provided a misleading susceptible about the correctly of forecasts generated, thus using the commonly of the mean squares error to measure the forecast error. The mean squares error is the average of the sum of squared forecasting errors. This measure, by calculation the squares of forecasting errors and exclude the chance of negative and positive errors erasure out. In choosing the length of the moving

averages that can use the mean squares error measure to examine the number of values to be included in computation the moving averages. The trail of different lengths to generate moving averages after that analyzes forecast errors or the associated mean squares errors for each length. Choosing the length that minimizes the mean squared error of forecasts generated is taken. Exponential smoothing may more difficult mathematically. However, exponential smoothing uses the weighted average concept in the form of the weighted average of all past observations, such contained in the association between time series and generate forecasts for at the last stage of period. Exponential smoothing comes from the truth that is method using a weighting scheme for the historical values of data. This scheme defines the maximum weight to the recent observation and the weights decline in a systematic manner as former and former observations are included. The accuracies of forecasts using exponential smoothing are determined in a manner similar to that for the moving averages method. Anyway, time Series Modeler Data Considerations are the dependent variable and any independent variables should be numeric.

Assumptions of the time series; the outcome variable and any independent variables are treated as time series. So, each case represents a time point or time period and successive cases eliminate by a constant time interval. Stationarity in the time series; they use for created ARIMA models. The time series to be modeled should be stationary. Forecasts model for producing forecasts using models with independent variables that are the predictors. For example World Health Organization (WHO, 2004), studied the association between Particulate Matter and Ozone by using Meta-analysis of time-series studies, the result showed that time series studies is appropriate to study the short-term effect and provide the convincingly results.

2.7 Comparing time-series and time stratified case-crossover results

Vigotti evaluated the association between the daily pollutants of ambient air pollution and children who daily hospital admissions for respiratory causes in Pisa during 1998-2002. Subjects were residents, by compared the results obtained with two methods of statistical analyses that research designs were both time-series and case-crossover analysis. Temperature, holidays, influenza epidemics, rain, and relative humidity were controlled. The results with both analyses showed that significantly positive

association between children's hospital admissions for respiratory conditions and increasing daily levels of PM_{10} and CO at different time lags. Time series analyses provides similar results in Case-crossover, although the estimates were lower than case-crossover, in terms of percentage increment and length of confidence intervals. The study indicated ambient air pollution risk to increase respiratory health of children who living in urban environment. The results of both analyses case-crossover and time series were consistent (Vigotti et al., 2010). Son and other studied infant daily mortality and air pollution, they found both of time series and CCO obtained similar result (Son et al., 2008).

Guo studied the short-term effect of air pollution on cardiovascular mortality in Tianjin, China from 2005 to 2007. The results showed that both case crossover and time series analyses were positively associated between air pollution and cardiovascular disease. When see in each model, the estimates from the TS-CCOvaried greatly with changing strata length. The estimates from the time series analyses varied slightly with changing degrees of freedom per year for time. The residuals from the time series analyses had less autocorrelation than those from the CCO analyses indicating a better fit. So Time series analyses conducted better resulted than the TS-CCO analyses in which the terms of residual autocorrelation analysis (Guo et al., 2010). Biggeri studied short term effect of air pollutants and daily mortality, the results did not differ between Case-crossover and Poisson regression approach (Biggeri et al., 2009).

CHAPTER III

METHODOLOGY

The research objective was to examine daily mortality in relation to short-term ambient air pollutant concentrations in Bangkok in 1999-2001 and 2006-2008, using the case-crossover design. This chapter focuses on the methodological approach. The material includes research design, study area, population, inclusion and exclusion criteria, sample size, sampling technique, measurement tools, data collection, data analysis, and ethical considerations.

3.1 Research design

This research design was time-stratified case-crossover study (January 1, 1999 to December 31, 2001 and January 1, 2006 to December 31, 2008). This 6 year period was the data collection period. This dissertation examines the association between air pollution and daily mortality in Bangkok, Thailand. Daily mortality, air pollution, and weather data in 1999-2001 and 2006-2008 in Bangkok were collected. Time-stratified case-crossover approach was used to estimate the effect of the air pollutants PM_{10}, O_3, SO_2, NO_2 and CO on daily total non external, cardiovascular, and respiratory mortality. The CCO design in air pollution research is conceptually somewhat similar to the matched case-control design. However, in the latter, a case person is matched with one or more control persons. In contrast, in the CCO design the air pollution level on the day on which a person died are matched with levels on days on which that person did not die. In time-stratified matching, controls are selected as matched days of the week in the same month as the death occurred. For example, if the death occurred on Wednesday, 8 August, 2007, the referent days would be all other Wednesdays in that same month (1, 15, 22, and 29 August 2007). If the death occurred on Tuesday, 20 June, 2000, the referent days would be the Tuesdays 6, 13, and 27 June, 2000). Thus, the number of referents was not the same for all cases. However, this did not bias the analysis. Time dependent variables were controlled by entering them in conditional logistic regression models. Effects of time-constant

variables such as gender and age was examined and compared in stratified analyses (for example, one model for males and a separate model for females). There was no need to enter time-constant variables into analytical models.

3.2 Study Area

Study sites in Bangkok were collected air sample for PM_{10}, SO_2, O_3, CO, and NO_2. The average of ambient air pollution was used to represent the whole Bangkok (Figure 2).

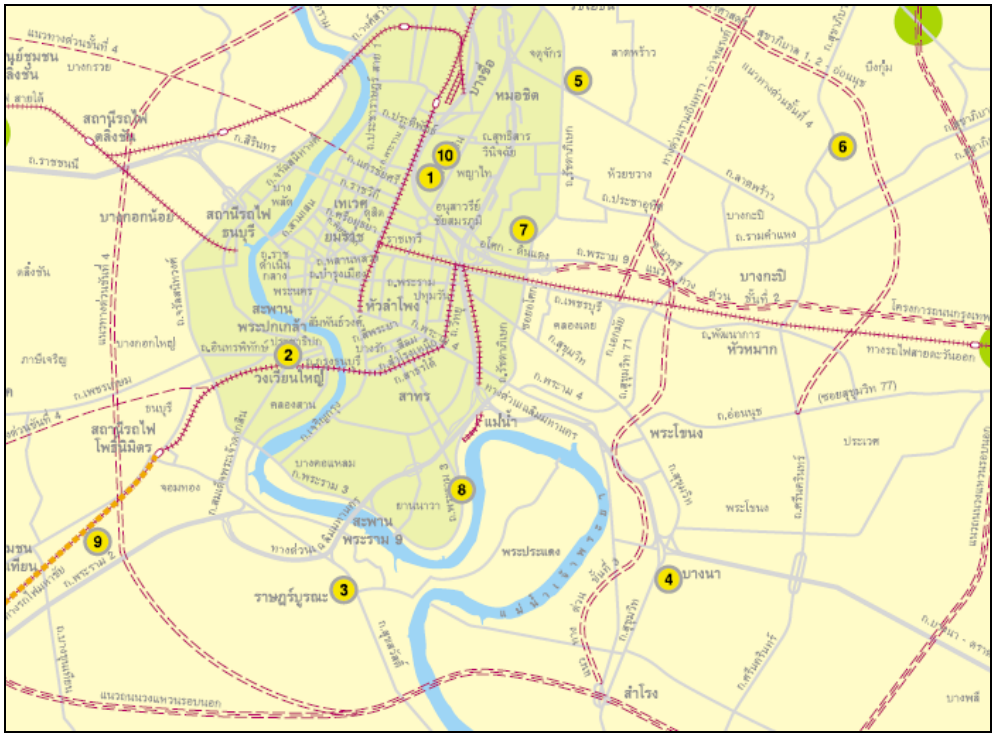


Figure 2: Map of collection sites for air pollution samples in Bangkok
Source: Pollution Control Department (2010)

Site 1: Office of Natural Resources and Environmental Policy and Planning, Phayathai, Bangkok.

Site 2: Bansomdejchaopraya Rajabhat University, Thon Buri, Bangkok.

Site 3: Ratburana Post office, Ratburana, Bangkok.

Site 4: Bangna Meteorological Department, Bangna, Bangkok.

Site 5: Chandrakasem Rajabhat University, Jatuchak, Bangkok.

Site 6: Klongchan Avenue National Housing Authority, Bangkapi, Bangkok.

Site 7: Huai Khwang Avenue National Housing Authority Stadium, Huai Khwang, Bangkok.

Site 8: Nonsi Withaya School, Yannawa, Bangkok.

Site 9: Singharaj Pittayakom School, Bangkhuntien, Bangkok.

Site 10: The Government Public Relations Department, Phayathai, Bangkok.

Table 6: The study sites that collected ambient air samples in each year.

Year	Site1	site2	Site3	Site4	Site5	Site6	Site7	Site8	Site9	Site10
1999	X	/	/	/	/	/	/	/	/	X
2000	X	/	/	/	/	/	/	/	/	X
2001	X	/	/	/	/	/	/	/	/	X
2006	/	/	/	/	/	/	/	/	/	/
2007	/	/	/	/	/	/	/	/	/	/
2008	/	/	/	/	/	/	/	/	/	/

X = not measure in that year

/ = measure in that year

Site 2 did not measure PM_{10} and O_3 in 1999, 2000, 2001,2006 and 2007.

Site 3 did not measure PM_{10}

Site 4 did not measure O_3 in 1999, 2000, 2001, 2006 and 2007, but they started on July 2008.

Site 5 did not measure PM_{10} and NO_2 in 1999.

Site 10 did not measure O_3 in 1999, 2000, 2001, 2006 and 2007.

There are many reasons of the losing data instance for water leak on the roof , machine did not work, out of electricity, so Pollution Control Department considered to repaired or maintenance that station, sometime some station had to establish the new equipments to measure air pollution. All kinds of these reasons made data missing.

3.3 Study Population

Non external, cardiovascular, and respiratory deaths in Bangkok 2 periods from January 1st, 1999 to December 31st, 2001 and January 1st, 2006 to December 31st, 2008.

3.4 Inclusion and Exclusion Criteria

Inclusion Criteria:

1. All non external mortality was enrolled to analysis. Also, the 2 parts of mortality as below was included for the CLR model.

2. Respiratory system (ICD-10 = J00 – J99)

e.g. Chronic obstructive pulmonary disease (COPD)

3. Circulatory system (ICD-10=I00 – I99)

e.g. Cardiovascular diseases (CVD)

Exclusion Criteria: the subjects who die with accidental and poisoning was excluded to analysis. Accidental mortality (ICD-10 > R99)

3.5 Sample size

Table 7: The number of death in Bangkok between 1999-2001 and 2006-2008.

Year	Thailand	Bangkok
1999	362,607	36,799
2000	365,741	37,257
2001	369,493	38,963
2006	391,126	38,825
2007	393,254	38,174
2008	397,327	38,090
Total	**1,518,929**	**228,108**

Source: Bureau of Policy and strategy Ministry of Public Health Thailand.

3.6 Sampling Technique

All deaths in Bangkok, for which date of death and ICD-10 code were received from the Ministry of Health, were included in analysis. Time stratified case-crossover design was referent selection strategy that was used for this study. The main objective was to find out the association between short terms effect of air pollution and daily non external mortality in Bangkok. Effects of time dependent confounders, such as daily temperature and humidity, was controlled by modeling. Data were analyzed by conditional logistic regression (figure 3).

An example of the time-stratified referent strategy was given in figure 3, in which the hypothetical death occurred on Monday, 8 January.

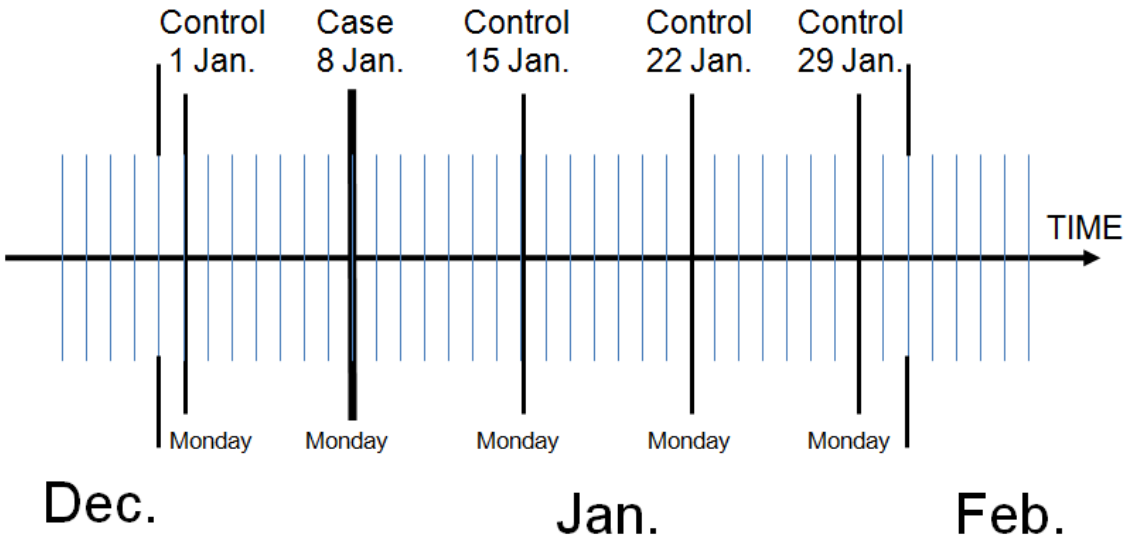


Figure 3: Time-stratified selection of referents for control periods. (Nitta et al., 2010)

3.7 Measurement Tools

Researcher used secondary data that were received from 3 departments as Ministry of Public Health Thailand, Meteorological Bureau, and Pollution Control Department. So the quality of equipments depend on each department, and the researcher cannot control for this. Potential limitations are discussed in chapter 6.

All hospitals in Bangkok used the same version of ICD 10 both two periods in 1999-2001 and 2006-2008. Bangkok has not verified the causes of death by using Verbal Autopsy (VA). Instrument in Air station in 1999 was the same as in 2008. The technicians test instrument every 2 months for the quality of data.

Real-time pumped systems were used for air pollution colleting such as

3.7.1 Pulsed fluorescence was used for SO_2

3.7.2 UV absorption was used for O_3

3.7.3 Chemiluminescence was used for NO

3.7.4 Infrared absorption was used for CO_2

3.7.5 Beta ray was used for PM_{10}

3.8 Data Collection and processing

3.8.1 Researcher sent the request form to 3 department.

3.8.1.1 Death certificates data from Ministry of Public Health (MOPH) Thailand,

3.8.1.2 Weather data such as temperature, humidity from Bangkok Meteorological Bureau.

3.8.1.3 Air pollution concentration data from Bangkok Pollution Control Department (PCD)

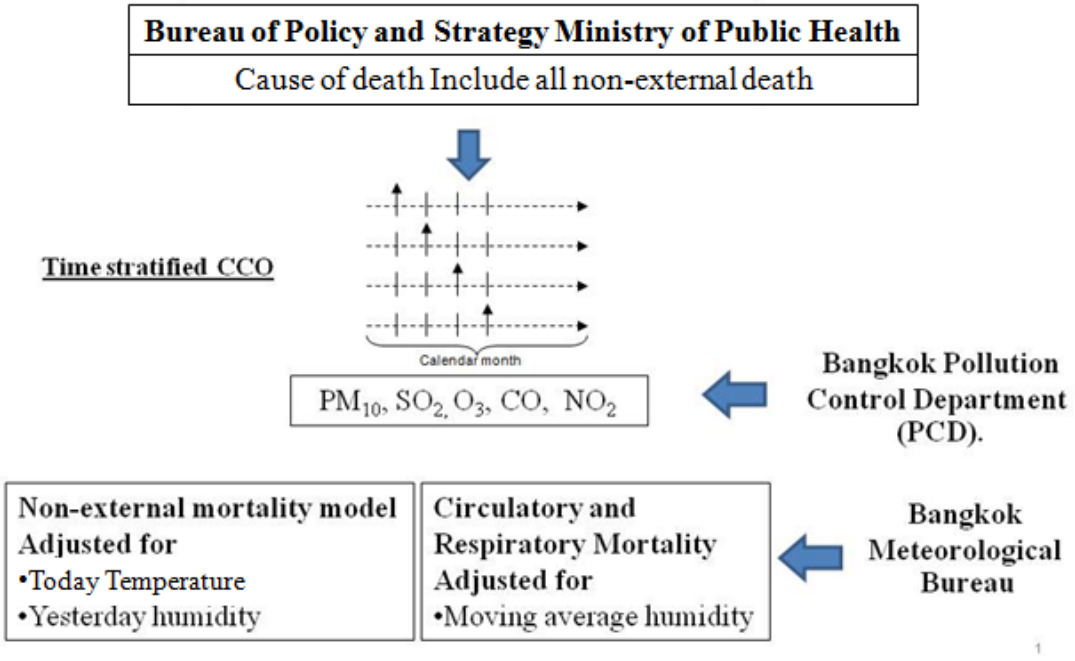


Figure 4: Source of data collecting.

3.8.2 Checking and correcting data to confirm that study variables meet the conditions for being able to study association by using a case crossover design. If data do not correct researcher co-operated for return to official and looking for the corrected data.

3.8.3 Event that use in the study was acute event e.g. respiratory systems on short term effect.

3.8.4 Air pollution level for reference periods was selected by time stratified design.

3.8.5 Checking for the proportion of missing data should be small.

3.8.6 24-hour average air pollutant levels were calculated for analysis. The same rules was used as in the PAPA time series study reported in Wong et al., 2008. Specifically, 75% of hourly values must be present for each included day for example CO was collected very hours (24 times a day) if they conducted less than 17 time that parameter was excluded for the day. If more than 25% of daily values of a given pollutant were missing for any station, that station was excluded from analysis for that

pollutant, instance for in the first period had 1096 days if one parameter loss more than 274 days then that station was excluded for this parameter.

3.8.7 Exposure variables was computed the individual or combine the different lags. These depend on the nature of the dependent variable, for this study used mortality as outcome variable, therefore longer lags and moving average were needed to compare with time series analysis.

3.8.8 The database was transmuted into a matrix with a case crossover structure which was as many strata as there were deaths. The periods of exposure at the time of the event in each stratum was called a case period (index time). Other periods were selected by sampling technique (time stratify case crossover) that was a control period.

3.8.9 The statistical analysis as the following steps below was used to analyze the association between dependent and independent variables.

3.8.9.1 Build a baseline equation by introducing time-depenent variables instance for temperature, relative humidity, and atmospheric pressure.

3.8.9.2 Build the single air pollutant models by adding one air pollutant to the standard model.

3.8.9.3 Build the multi air pollutant models by adding more than one air pollutant to the standard model.

3.8.9.4 Analyze effects of time-constant potential confounders or effect modifiers (e.g., age, sex), by doing stratified analysis.

3.8.10 Completed models by recheck with 2 programs such as STATA and SPSS.

3.9 Data Analysis

The association between the daily mortality and daily pollutant concentration were analyzed by using a conditional logistic regression. Bi-directional control periods were selected by using a time-stratified approach. Models include time dependent variable such as mean of temperature, relative humidity, and air pressure. As hazard periods the following lags have been considered: single lag (from 0 to 1) and moving average of air pollution concentration (lag 0-1, lag 0-2 for mortality)

Descriptive statistics of mortality outcomes, air pollution levels were used mean, SD and percentage. ANOVA was used to compare mean air pollutant levels in each year.

Single-pollutant models: Conditional Logistic regression was used to analyze association between mortality risk and air pollution.

Unadjusted for weather

$$Log(OR) = \beta O_3 \quad \ldots.(1)$$

$$Log(OR) = \beta PM_{10} \quad \ldots.(2)$$

$$Log(OR) = \beta NO_2 \quad \ldots.(3)$$

$$Log(OR) = \beta SO_2 \quad \ldots.(4)$$

$$Log(OR) = \beta CO \quad \ldots.(5)$$

When: Log (OR) was the outcome that variables can be non external, respiratory, or circulatory mortality.

Adjusted for weather on non-external mortality models

$$Log(OR) = \beta O_3 + \beta Temperature\ lag0 + \beta Humidity\ lag1 \quad \ldots.(6)$$

$$Log(OR) = \beta PM_{10} + \beta Temperature\ lag0 + \beta Humidity\ lag1 \quad \ldots.(7)$$

$$Log(OR) = \beta NO_2 + \beta Temperature\ lag0 + \beta Humidity\ lag1 \quad \ldots.(8)$$

$$Log(OR) = \beta SO_2 + \beta Temperature\ lag0 + \beta Humidity\ lag1 \quad \ldots.(9)$$

$$Log(OR) = \beta CO + \beta Temperature\ lag0 + \beta Humidity\ lag1 \quad \ldots.(10)$$

Adjusted for weather on Circulatory mortality models

$$Log(OR) = \beta O_3 + \beta Humidity\ mv0\text{-}1 \quad \ldots.(6)$$

$$Log(OR) = \beta PM_{10} + \beta Humidity\ mv0\text{-}1 \quad \ldots.(7)$$

$$Log(OR) = \beta NO_2 + \beta Humidity\ mv0\text{-}1 \quad \ldots.(8)$$

$$Log(OR) = \beta SO_2 + \beta Humidity\ mv0\text{-}1 \quad \ldots.(9)$$

$$Log(OR) = \beta CO + \beta Humidity\ mv0\text{-}1 \quad \ldots.(10)$$

When: Humidity mv0-1 was the moving average of humidity level during today and yesterday (lag0 to lag1).

Adjusted for weather on Respiratory mortality models

$$Log(OR) = \beta O_3 + \beta Humidity\ mv0\text{-}1 \quad \ldots.(11)$$

$$Log(OR) = \beta PM_{10} + \beta Humidity\ mv0\text{-}1 \quad \ldots.(12)$$

$$Log(OR) = \beta NO_2 + \beta Humidity\ mv0\text{-}1 \quad \ldots.(13)$$

$$Log(OR) = \beta SO_2 + \beta Humidity\ mv0\text{-}1 \quad \ldots.(14)$$

$$Log(OR) = \beta CO + \beta Humidity\ mv0\text{-}1 \quad \ldots.(15)$$

When: Humidity mv0-1 was the moving average of humidity level during today and yesterday (lag0 to lag1).

The results of this study was compared to results of time series studies conducted by other researchers. As mentioned above, time-series results suggest stronger effects of air pollution in Bangkok than in other Asian cities for the time period 1999-2003 (Wong et al., 2008). It was the special interest to examine whether results of CCO analysis show effects in Bangkok during 1999-2000 that were as strong as those in time series analysis. It was also of interest to compare results between the 1999-2001 period and the 2006-2008 period.

The time stratum of the referent time was the same month and the same year as the index time. Referent days were chosen within this same month. Times Stratified Case crossover Design (TSD), time was divided *a priori* into strata $s(t) = 1,\ldots,S$. The reference window for day t was the set of days in its stratum (Lumley and Levy, 2000). Conditional logistic regression models were stratified on each set of the index time and its referent days (possibly with one or more lags for the day of death). This was similar to conditional logistic regression for matched case-control, in which model strata were the sets of matched cases and controls. Models included one or more air pollutants, and the time-dependent adjustor variables listed in the conceptual framework. We select temperature today (lag0) and humidity yesterday (lag1) for non-external mortality model. Circulatory mortality and Respiratory mortality we used moving average of humidity that was humidity today combined humidity yesterday then divided by two (humidity mv0-1) as adjustor. Analysis was used both adjusted and unadjusted for weather in single pollution model and co pollutants model. We tested for strongest statistic significant in each lag of weather before used as the adjustor. The dependent variables or outcome variables were total non-external mortality, cardiovascular mortality, and respiratory mortality. The conditional logistic analyses were conducted using Cox models, structured to ensure that the event (mortality) always occurs for the mortality days, and never occurs for the referent days. Stata program was used to recheck the correction value. Differences in pollutant effects between the 2 study periods were tested, in models that included data from both study periods, by entering variables for pollutant level over both periods, and pollutant level in period 2 (period 1 pollutant level set to zero). The Wald chi-square for the latter variable gave a test of significance of the difference in pollutant effect between the 2 periods.

3.10 Ethical Consideration

This study passed the Ethical Review committee for Research Involving Human Research Subjects, Health Sciences Group, Chulalongkorn University (IRB approval No.117/2011), although the researcher used secondary data. The researcher also obtained permission from the director of each department to access the data.

CHAPTER IV

RESULTS

The study was conducted in 2011. All data were secondary data which came from 3 departments of Thai government. Multidimensional databases were the method to collection and combination data with the different source that were linked by date. The 2 periods of study were 1999-2001 and 2006-2008, 3 years per period, total 6 years. The results are presented in 10 parts:

4.1 Sociodemographic characteristics of the population

4.2 Characteristics of air pollution in Bangkok

4.3 Correlations of Air pollution and Time-dependent variables in each year

4.4 Seasonality and air pollution

4.5 Single-pollutant concentrations with different lags and moving average

4.6 Association between Air pollution and Non-external Mortality in Bangkok.

4.7 Association between Air pollution and Circulatory Mortality in Bangkok.

4.8 Association between Air pollution and Respiratory Mortality in Bangkok.

4.9 Analyses stratified on time independent variables

4.10 Comparison the current CCO study and PAPA time series

4.1 Sociodemographic characteristics of the population

A total of 207,693 deaths were non-external in six years of the study, 91.05 % of the deaths in Bangkok. More males than females died in each year, with median of percentages were 55.49% and 44.51% of the total number when calculate median from percentage in each year.

Table 8: Characteristics of population for non-external mortality

Variables	Years						Over all
	1999	2000	2001	2006	2007	2008	
Death per day	91.86	92.71	97.91	96.52	94.92	94.83	94.88
Gender (n)	33,521	33,933	35,652	35,231	34,647	34,709	207,693
Male	19,104	19,207	20,016	19,322	18,856	18,867	115,372
(n/%)	56.99%	56.60%	56.14%	54.84%	54.42%	54.36%	55.49%
Female	14,417	14,726	15,636	15,909	15,791	15,842	92,321
(n/%)	43.01%	43.40%	43.86%	45.16%	45.58%	45.64%	44.51%
Age (n)	33,473	33,863	35,586	35,140	34,594	34,679	207,335
0-10 years	1,232	1,264	1,328	1,188	1,104	1,031	7,147
(n/%)	3.68%	3.73%	3.73%	3.38%	3.19%	2.97%	3.53%
11-20 years	464	488	469	376	349	320	2,466
(n/%)	1.39%	1.44%	1.32%	1.07%	1.01%	0.92%	1.20%
21-30 years	2,346	2,316	2,332	1,275	1,149	1,095	10,513
(n/%)	7.01%	6.84%	6.55%	3.63%	3.32%	3.16%	5.09%
31-40 years	3,613	3,633	3,758	2,704	2,449	2,363	18,520
(n/%)	10.79%	10.73%	10.56%	7.69%	7.08%	6.81%	9.13%
41-50 years	3,828	3,973	4,196	4,219	4,090	4,057	24,363
(n/%)	11.44%	11.73%	11.79%	12.01%	11.82%	11.70%	11.76%
51-60 years	4,267	4,285	4,490	5,005	5,199	5,271	28,517
(n/%)	12.75%	12.65%	12.62%	14.24%	15.03%	15.20%	13.50%
> 60 years	17,723	17,904	19,013	20,373	20,254	20,542	115,809
(n/%)	52.95%	52.87%	53.43%	57.98%	58.55%	59.23%	55.71%
Minimum	0	0	0	0	0	0	0
Maximum	117	112	112	118	114	111	118
average	58.24	58.16	58.55	61.62	62.23	62.69	60.09
SD	22.20	22.19	22.21	21.34	21.08	20.79	21.77
Place of death	33,521	33,933	35,652	35,231	34,647	34,709	207,693
Outside hospital	11,494	11,435	12,216	10,030	9,764	9,144	64,083
(n/%)	34.29%	33.70%	34.26%	28.47%	28.18%	26.34%	31.09%
Inside hospital	22,027	22,498	23,436	25,201	24,883	25,565	143,610
(n/%)	65.71%	66.30%	65.74%	71.53%	71.82%	73.66%	68.92%

The age group with the highest number of deaths was more than 60 year old with average 55.70% of the total number when calculate means from percentage in each years. The median of average age in each year was 60.08 year old. Most of them died inside hospital, median of the annual percentage was 68.92% (Table 8).

Table 9: Characteristics of population for cardiovascular (circulatory) mortality

Variable	Year						Over all
	1999	2000	2001	2006	2007	2008	
Death per day	10.43	12.70	14.57	13.58	13.47	13.28	13.38
Gender (n)	3,802	4,648	5,313	4,958	4,917	4,859	28,497
Male	2,103	2,581	2,944	2,838	2,800	2,797	16,063
(n/%)	55.3%	55.5%	55.4%	57.2%	56.9%	57.6%	56.20%
Female	1,699	2,067	2,369	2,120	2,117	2,062	12,434
(n/%)	44.7%	44.5%	44.6%	42.8%	43.1%	42.4%	43.80%
Age (n)	3,795	4,645	5,302	4,944	4,911	4,852	28,449
0-10 years	37	41	76	50	36	23	263
(n/%)	1.0%	0.9%	1.4%	1.0%	0.7%	0.5%	0.95%
11-20 years	51	45	55	48	34	34	267
(n/%)	1.3%	1.0	1.0%	1.0%	0.7%	0.7%	1.00%
21-30 years	109	137	183	98	99	110	736
(n/%)	2.9%	2.9%	3.4%	2.0%	2.0%	2.3%	2.60%
31-40 years	230	239	303	256	271	257	1,556
(n/%)	6.0%	5.1%	5.7%	5.2%	5.5%	5.3%	5.40%
41-50 years	458	536	650	611	605	586	3,446
(n/%)	12.0%	11.5%	12.2%	12.3%	12.3%	12.2%	12.20%
51-60 years	673	763	921	874	823	821	4,875
(n/%)	17.7%	16.4%	17.3%	17.6%	16.7%	16.9%	17.10%
> 60 years	2,237	2,884	3,114	3,007	3,043	3,021	17,306
(n/%)	58.8	62.0%	58.6%	60.6%	61.9%	62.2%	61.95%
Minimum	0	0	0	0	0	0	0
Maximum	117	111	104	105	104	109	117
average	61.90	63.07	61.89	63.62	64.22	64.52	63.345
SD	17.26	17.14	18.03	17.37	17.19	16.94	17.225
Place of death							
Outside hospital	583	664	870	1,050	1,067	960	5,194
(n/%)	15.3%	14.3%	16.4%	21.2%	21.7%	19.8%	18.10%
Inside hospital	3,219	3,984	4,443	3,908	3,850	3,899	23,303
(n/%)	84.7%	85.7%	83.6%	78.8%	78.3%	80.2%	81.90%

A total of 28,479 deaths were due to cardiovascular causes in the six years of the study. More males died than females in each year, with median of percentages were 56.20% and 43.80% of the total number when calculate median from percentage in each year. The highest death in age group was more than 60 year old with median of average 61.95% of the total number when calculate median from percentage in each years (Table 9).

Table 10: Characteristics of population for respiratory mortality

Variables	Years						Over all
	1999	2000	2001	2006	2007	2008	
Death per day	6.78	7.39	8.48	9.05	8.85	9.12	8.67
Gender (n)	2,473	2,704	3,079	3,304	3,231	3,340	18,131
Male	1,730	1,858	2,048	1,977	1,920	1,986	11,519
(n/%)	70.0%	68.7%	66.5%	59.8%	59.4%	59.5%	63.15%
Female	742	846	1,031	1,327	1,311	1,354	6,611
(n/%)	30.0%	31.3%	33.5%	40.2%	40.6%	40.5%	36.85%
Age (n)	2,473	2,703	3,074	3,290	3,224	3,339	18,103
0-10 years	62	62	145	91	95	64	519
(n/%)	2.5%	2.3%	4.7%	2.8%	2.9%	1.9%	2.65%
11-20 years	28	25	31	34	37	32	187
(n/%)	1.1%	0.9%	1.0%	1.0%	1.1%	1.0%	1.00%
21-30 years	297	328	330	152	135	109	1,351
(n/%)	12.0%	12.1%	10.7%	4.6%	4.2%	3.3%	7.65%
31-40 years	413	486	501	303	253	229	2,185
(n/%)	16.7%	18.0%	16.3%	9.2%	7.8%	6.9%	12.75%
41-50 years	292	333	348	317	304	286	1,880
(n/%)	11.8%	12.3%	11.3%	9.6%	9.4%	8.6%	10.45%
51-60 years	244	236	250	290	282	327	1,629
(n/%)	9.9%	8.7%	8.1%	8.8%	8.7%	9.8%	8.75%
> 60 years	1,130	1,233	1,469	2,103	2,118	2,292	10,345
(n/%)	45.7%	45.6%	47.7%	63.7%	65.6%	68.0%	55.70%
Minimum	0	0	0	0	0	0	0
Maximum	101	102	108	118	105	104	118
average	55.29	55.28	55.04	64.21	65.00	67.04	59.75
SD	22.64	22.72	23.91	22.46	22.30	20.85	22.55
Place of death							
Outside hospital	645	703	731	456	429	348	3,312
(n/%)	26.1%	26.0%	23.7%	13.8%	13.3%	10.4%	18.75%
Inside hospital	1,827	2,001	2,348	2,848	2,802	2,992	14,818
(n/%)	73.9%	74.0%	76.3%	86.2%	86.7%	89.6%	81.25%

The median of average age was 63 year old. Many of them died inside hospital, median of percentage was 81.90% (Table 9).

A total of 18,130 deaths were due to respiratory causes in six years of the study. Male was death more than female every years with median of percentages were 63.15% and 36.85% of the total number when calculate median from percentage in each year. The highest death in age group was more than 60 year old with median of average 55.7% of the total number when calculate median from percentage in each years. The median of average age was 59 year old. Most people died inside hospital, the median of the percentage was 81.25% (Table 10).

Table 11: Counts and percentages of mortality in Bangkok due to non-external, circulatory, and respiratory causes, by year

Year	Non-external		Circulatory		Respiratory		Unclassified*		Senility†	
	n	%	n	%	n	%	n	%	n	%
1999	33,440	100	3,795	11.3	2,469	7.4	14,662	43.8	4,929	14.7
2000	33,933	100	4,648	13.7	2,704	8.0	11,756	34.6	4,639	13.7
2001	35,149	100	5,232	14.9	3,043	8.7	10,873	30.9	4,856	13.8
2006	35,231	100	4,958	14.1	3,304	9.4	9,244	26.2	2,200	6.2
2007	34,647	100	4,917	14.2	3,231	9.3	8,802	25.4	2,910	8.4
2008	34,709	100	4,859	14.0	3,340	9.6	8,535	24.6	1,969	5.7
Total	207,109	100	28,409	13.7	18,091	8.7	63,872	30.8	21,503	10.4

* Unclassified includes all ICD codes beginning with R.

† Senility is a subset of unclassified (R54).

Table 11 shows the counts of mortality by years and types of diagnoses. We included the eligible data 207,109 natural deaths.The median of Circulatory mortality's percentage is 14.05% higher than median of Respiratory mortality's percentage. The total controls were 703,884 that mean case per control was 1: 3.4 were included in the analysis.

4.2 Characteristics of air pollution in Bangkok

Distribution of carbon monoxide (CO (μg/m^3)) for the time period 1999-2001 was higher than period 2006-2008. The tendency show decreasing both 2 periods. The standard of air pollution quality identify that should not over 10,350 μg/m^3/8hours. The average of CO concentration did not exceed the standard line (Figure 5 and 6).

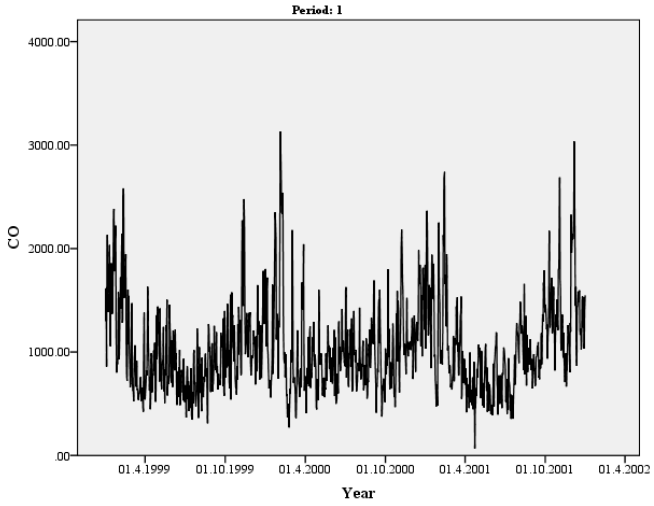


Figure 5: 24-hour average CO concentrations (μg/m^3) in 1999-2001

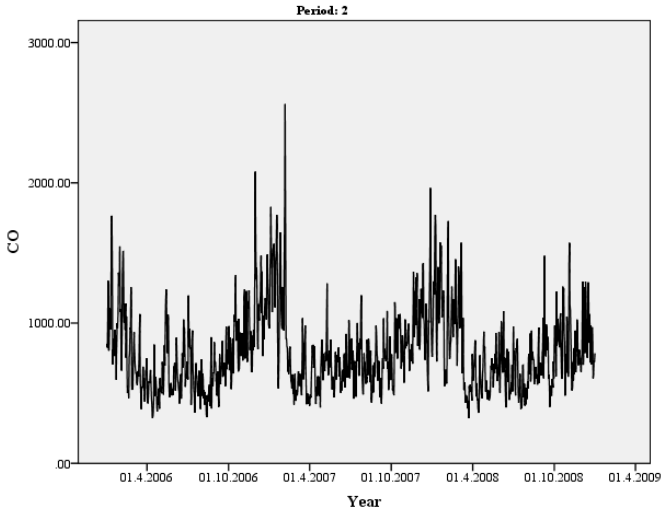


Figure 6: 24-hour average of CO concentration (μg/m^3) in 2006-2008

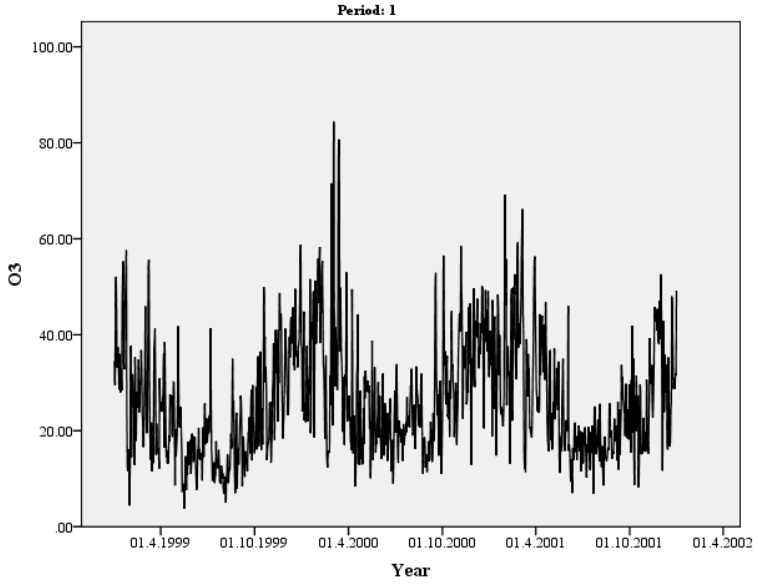


Figure 7: 24-hour average of O_3 concentration ($\mu g/m^3$) in 1999-2001

Distribution of the O_3 concentration ($\mu g/m^3$) for the time period 2006-2008 was higher than period 1999-2001. There was fluctuation in both periods. The standard of air pollution quality identify that should not over 137.2 $\mu g/m^3$/8hours. The average O_3 concentration did not exceed the standard line (Figure 7 and 8).

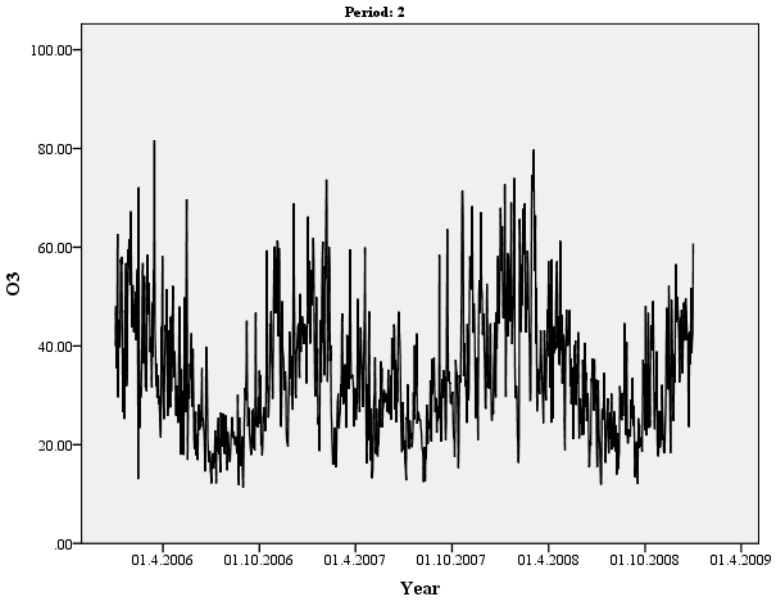


Figure 8: 24-hour average of O_3 concentration ($\mu g/m^3$) in 2006-2008

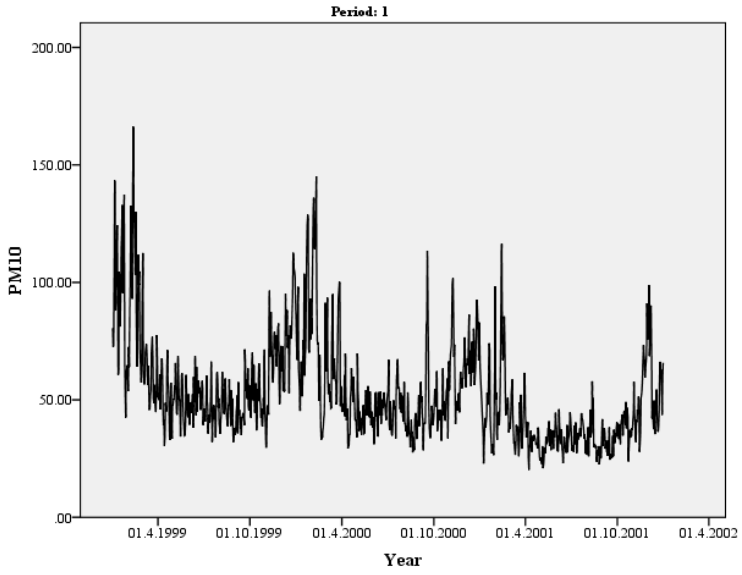


Figure 9: 24-hour average of PM_{10} concentration ($\mu g/m^3$) in 1999-2001

Distribution of the PM_{10} concentration ($\mu g/m^3$) for the time period 1999-2001 was higher than period 2006-2008. The tendency show decreasing in first period. In the second period PM_{10} was not stable. The standard of air pollution quality identify that PM_{10} concentration should not over 120 $\mu g/m^3$ /24 hour. The average of PM_{10} concentration had some days over the standard line (Figure 9and10).

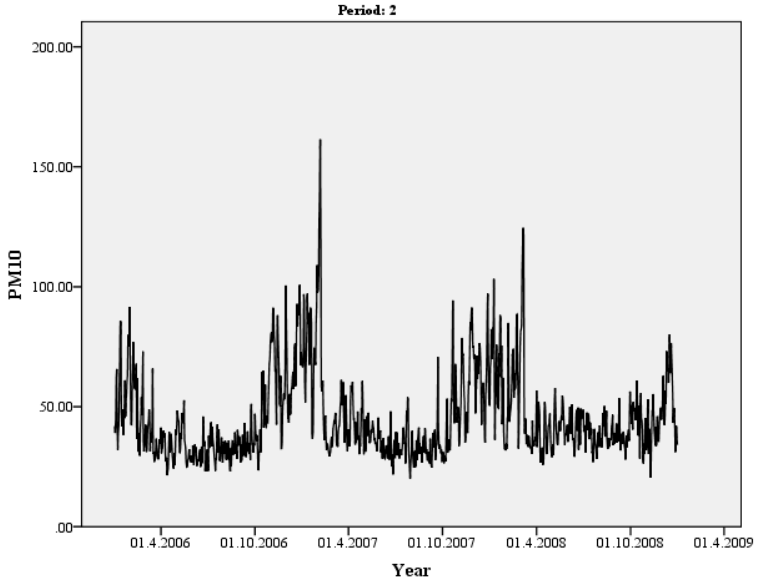


Figure 10: 24-hour average of PM_{10} concentration ($\mu g/m^3$) in 2006-2008

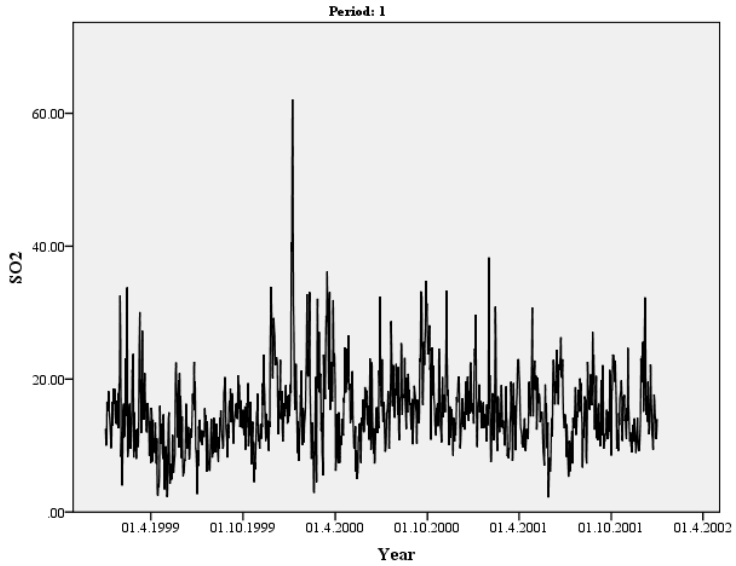


Figure 11: 24-hour average of SO_2 concentration ($\mu g/m^3$) in 1999-2001

Distribution of the SO_2 concentration ($\mu g/m^3$) for the time period 1999-2001 was higher than period 2006-2008. The tendency show similar in both period. The standard of air pollution quality identify that SO_2 concentration should not over 314.4 $\mu g/m^3$/1 hour. The average of SO_2 concentration did not exceed the standard line (Figure 11and12).

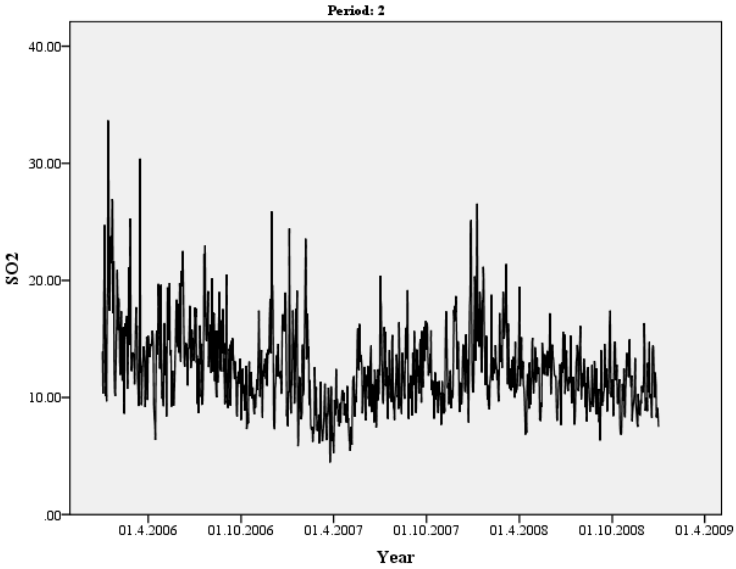


Figure 12: 24-hour average of SO_2 concentration ($\mu g/m^3$) in 2006-2008

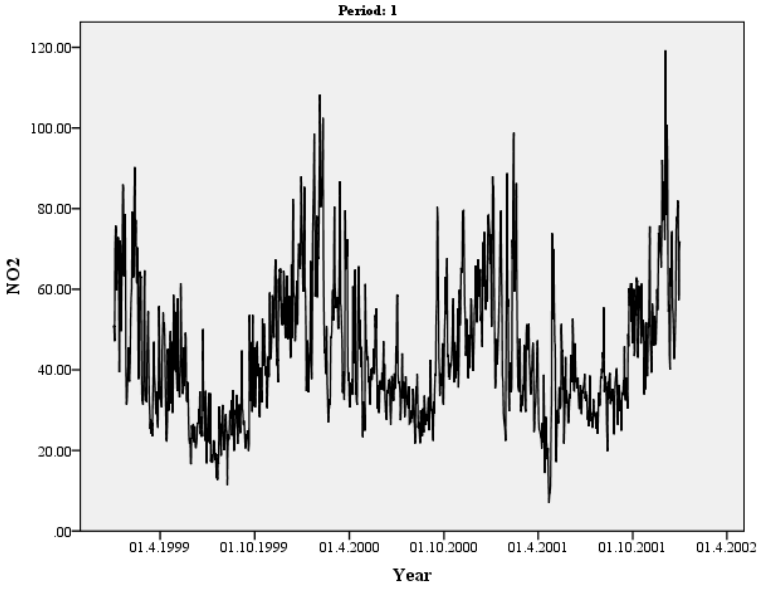


Figure 13: 24-hour average of NO_2 concentration ($\mu g/m^3$) in 1999-2001

Distribution of the NO_2 concentration ($\mu g/m^3$) for the time period 1999-2001 was a little higher period 2006-2008. Thre was fluctuation in both periods. The standard of air pollution quality identify that NO_2 concentration should not over 319 $\mu g/m^3$/hour. The average of NO_2 concentration did not exceed the standard line (Figure 13and14).

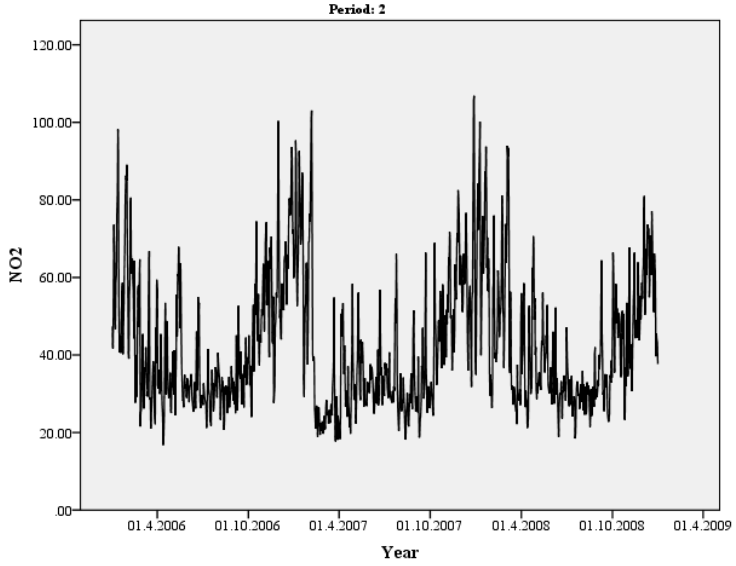


Figure 14: 24-hour average of NO_2 concentration ($\mu g/m^3$) in 2006-2008

Table 12 show all data set, PM_{10} was loss 5 days measurement. Maximum of CO and PM_{10} were over the standard limited. CO had largest standard deviation.

Table 12: Means of air pollutant concentrations and meteorological conditions in 6 years of study

	Years 1999-2001 and 2006-2008				
	n	Mean	SD	Min	Max
CO(µg/m^3)	2192	913	392.6	66.6	3131.9
O_3(µg/m^3)*24hr*	2192	30.3	13.1	3.8	84.4
O_3(µg/m^3)*8hr*	2192	60.4	29.3	6.8	184.2
NO_2(µg/m^3)	2192	43.0	17.0	7.0	119.3
SO_2(µg/m^3)	2192	13.8	5.1	2.2	62.1
PM_{10}(µg/m^3)	2187	49.0	20.2	20.1	166.3
Temperature(C°)	2192	29.0	1.7	18.6	33.1
Humidity(rH)	2192	71.0	8.2	39.0	95.7
Air pressure(Pa)	2192	1008.9	2.9	962.6	1019.1

This study used moving average 0-1 (average of current day and previous day) for air pollutants, per increment of 10 µg/m^3 for O_3, NO_2, SO_2 and PM_{10}, and per increment of 100 µg/m^3 for CO. The mean difference between two periods was shown CO, SO_2, and PM_{10} significant decreasing. O_3 was statistically significant increase. Only NO_2 did not differ significantly between two periods (Table 13).

Table 13: Mean difference of air pollutants concentration moving average 0-1 in two periods

	Mean Difference (period 1- period2)	Std. Error Difference	df	t	Sig. (2-tailed)
CO(µg/m^3)	2.46	0.15	1884	16.5	<.001
O_3(µg/m^3)*24hr*	-0.79	0.05	2179	-16.0	<.001
O_3(µg/m^3)*8hr*	-1.23	0.11	2190	-10.8	<.001
NO_2(µg/m^3)	0.13	0.07	2190	1.8	0.061
SO_2(µg/m^3)	0.28	0.02	1780	14.8	<.001
PM_{10}(µg/m^3)	0.76	0.08	2100	9.2	<.001
Temperature(C°)	-0.36	0.07	2190	-5.2	<.001
Humidity(rH)	3.02	0.33	2175	9.2	<.001
Air pressure(Pa)	2.46	0.15	1884	16.5	<.001

Table 14 show average of air pollutants level as CO, PM_{10}, O_3, and SO_2. The study show decreasingly tendency of CO, SO_2, and $PM_{10.}$ But show increasingly tendency on O_3 with mean different were significantly statistic ($P < .001$) between 2 periods of the study. The mean differences of NO_2 in each year were not significant.

Table 14: Mean differences of air pollutants moving average 0-1 in each year

	Period 1 (1999-2001)						**Period 2 (2006-2008)**						**P-value**
	1999		**2000**		**2001**		**2006**		**2007**		**2008**		
	Average	SD	Average	SD	Average	SD	Average	SD	Average	SD	Average	SD	
CO ($\mu g/m^3$)	10.08	3.88	10.33	4.03	10.67	4.44	7.75	2.64	8.10	2.83	7.84	2.61	<.001
O_3 *24hr* **($\mu g/m^3$)**	2.35	1.06	2.87	1.13	2.65	1.08	3.34	1.21	3.38	1.13	3.53	1.24	<.001
NO_2 ($\mu g/m^3$	4.06	1.58	4.64	1.57	4.39	1.58	4.05	1.72	4.34	1.58	4.30	1.63	0.171
SO_2 ($\mu g/m^3$)	1.34	0.47	1.71	0.62	1.51	0.44	1.40	0.34	1.13	0.29	1.19	0.25	<.001
PM_{10} ($\mu g/m^3$)	6.11	2.21	5.65	1.99	4.06	1.41	4.34	1.66	4.65	1.97	4.56	1.45	<.001

The air pollutant in first period most of them were higher than second period excepting O_3. The temperature in second period average was higher than first period (Table 15).

Table 15: Mean of air pollutants concentration and meteorological conditions in first period of study

	Year 1999-2001				
	n	Mean	SD	Min	Max
CO ($\mu g/m^3$)	1096	1036.4	442.7	66.6	3131.9
O_3 ($\mu g/m^3$)*24hr*	1096	26.3	12.0	3.8	84.4
O_3 ($\mu g/m^3$)*8hr*	1096	54.3	27.8	6.8	179.1
NO_2 ($\mu g/m^3$)	1096	43.7	17.0	7.0	119.3
SO_2 ($\mu g/m^3$)	1096	15.3	5.9	2.2	62.1
PM_{10} ($\mu g/m^3$)	1091	52.8	21.7	20.2	166.3
Temperature (C°)	1096	28.9	1.7	18.6	32.9
Humidity (rH)	1096	72.5	8.4	39.0	95.7
Air pressure (Pa)	1096	1008.7	3.1	962.6	1019.1

Air pollution concentrations in the PAPA time series study were generally similar to those in this CCO study. Exception for O_3 in PAPA the average from 8 hours but CCO was 24 hours that make PAPA's O_3 had mean higher than CCO study 2 fold. CO was the pollutant which using in CCO study but did not in PAPA study (Table 16)

Table 16: Percent differences in mean air pollutant concentrations and meteorological conditions in PAPA time series study in Bangkok (Wong et al., 2008)

	Year 1999-2003			% different
	Mean	Min	Max	(CCO-PAPA)
O_3 (µg/m^3) *8h*	59.4	8.2	180.6	-8.6
NO_2 (µg/m^3)	44.7	15.8	139.6	-2.2
SO_2 (µg/m^3)	13.2	1.5	61.2	15.9
PM_{10} (µg/m^3)	52.0	21.3	169.2	1.5
Temperature	28.9	18.7	33.6	0
Humidity	72.8	41.0	95.0	-0.4

The second period had average of air pollutants less than first period such as CO, NO_2, SO_2 and PM_{10} (Table 17). The meteorological condition between two period were similarly.

Table 17: Mean of air pollutants concentration and meteorological conditions in second period of this case-crossover study

	Year 2006-2008				
	n	Mean	SD	Min	Max
CO (µg/m^3)	1096	789.7	286.4	322.9	2561.8
O_3 (µg/m^3)*24hr*	1096	34.2	12.9	11.3	81.7
O_3 (µg/m^3)*8hr*	1096	66.6	29.6	12.1	184.2
NO_2 (µg/m^3)	1096	42.4	17.0	16.8	106.8
SO_2 (µg/m^3)	1096	12.4	3.5	4.5	33.7
PM_{10} (µg/m^3)	1091	45.2	17.8	20.1	161.5
Temperature	1096	29.2	1.6	23.1	33.1
Humidity	1096	69.4	7.7	47.0	90.5
Air pressure	1096	1009.2	2.7	1001.3	1018.3

4.3 Correlations between air pollutants and time-dependent variables

Analysis of relationships between the time-dependent variables (meteorological factors) and air pollution in 6 years show positive correlations between air pressure and air pollutants (NO_2, PM_{10}, O_3, CO, SO_2). The correlation between NO_2 and air pressure was stronger when compared with the correlation between the other air pollutants and air pressure. Temperature and Humidity were negative correlation with air pollutants and significantly, especially O_3 was stronger negative relationship with Humidity than others (Table 18).

Table 18: Spearman Correlations between air pollutants and time-dependent meteorology variables in all 6 year

		O_3 mv01*	NO_2 mv01	SO_2 mv01	PM_{10} mv01	Temperature mv01	Humidity mv01
CO mv01	Correlation Coefficient	0.220	0.742	0.319	0.640	-0.546	-0.076
	P-value	<.001	<.001	<.001	<.001	<.001	<.001
	n	2192	2192	2192	2188	2192	2192
O_3 mv01	Correlation Coefficient	1	0.563	0.047	0.431	-0.058	-0.592
	P-value		<.001	0.028	<.001	0.006	<.001
	n		2192	2192	2188	2192	2192
NO_2 mv01	Correlation Coefficient		1	0.294	0.679	-0.519	-0.292
	P-value			<.001	<.001	<.001	<.001
	n			2192	2188	2192	2192
SO_2 mv01	Correlation Coefficient			1	0.232	-0.044	-0.061
	P-value				<.001	0.040	0.004
	n				2188	2192	2192
PM_{10} mv01	Correlation Coefficient				1	-0.346	-0.371
	P-value					<.001	<.001
	n					2188	2188
Temperature mv01	Correlation Coefficient					1	-0.104
	P-value						<.001
	n						2192
Humidity mv01	Correlation Coefficient						1
	P-value						
	n						

* mv01 indicates moving average of current day and previous day

In year 1999-2001 show positive correlations between air pressure and air pollutants (NO_2, PM_{10}, O_3, CO, SO_2), NO_2 was stronger relative with air pressure than other air pollutants.

Table 19: Spearman correlations between air pollutants and time-dependent variables, 1999-2001

		O_3 mv01	**NO_2 mv01**	**SO_2 mv01**	**PM_{10} mv01**	**Temperature mv01**	**Humidity mv01**
CO mv01	Correlation Coefficient	0.337	0.721	0.227	0.484	-0.522	-0.100
	P-value	<.001	<.001	<.001	<.001	<.001	<.001
	n	1096	1096	1096	1092	1096	1096
O_3 mv01	Correlation Coefficient	1	0.625	0.198	0.484	-0.101	-0.521
	P-value		<.001	<.001	<.001	<.001	<.001
	n		1096	1096	1092	1096	1096
NO_2 mv01	Correlation Coefficient		1	0.274	0.599	-0.507	-0.257
	P-value			<.001	<.001	<.001	<.001
	n			1096	1092	1096	1096
SO_2 mv01	Correlation Coefficient			1	0.177	0.008	-0.119
	P-value				<.001	0.791	<.001
	n				1092	1096	1096
PM_{10} mv01	Correlation Coefficient				1	-0.287	-0.409
	P-value					<.001	<.001
	n					1092	1092
Temperature mv01	Correlation Coefficient					1	-0.124
	P-value						<.001
	n						1096
Humidity mv01	Correlation Coefficient						1
	P-value						
	n						

Temperature was negative correlation with air pollutants and significantly, except that SO_2 show some positive correlations. Humidity was negative correlation with air pollutants (Table 19).

Table 20: Spearman Correlations between Air pollutions and Time-dependent variables 2006-2008

		O_3 mv01	NO_2 mv01	SO_2 mv01	PM_{10} mv01	Temperature mv01	Humidity mv01
CO mv01	Correlation Coefficient	0.427	0.825	0.208	0.752	-0.573	-0.190
	P-value	<.001	<.001	<.001	<.001	<.001	<.001
	n	1096	1096	1096	1096	1096	1096
O_3 mv01	Correlation Coefficient	1	0.627	0.126	0.635	-0.110	-0.580
	P-value		<.001	<.001	<.001	<.001	<.001
	n		1096	1096	1096	1096	1096
NO_2 mv01	Correlation Coefficient		1	0.313	0.777	-0.529	-0.346
	P-value			<.001	<.001	<.001	<.001
	n			1096	1096	1096	1096
SO_2 mv01	Correlation Coefficient			1	0.162	-0.018	-0.111
	P-value				<.001	0.544	<.001
	n				1096	1096	1096
PM_{10} mv01	Correlation Coefficient				1	-0.358	-0.459
	P-value					<.001	<.001
	n					1096	1096
Temperature mv01	Correlation Coefficient					1	-0.066
	P-value						0.029
	n						1096
Humidity mv01	Correlation Coefficient						1
	P-value						
	n						

In year 2006-2008 show positive correlations between air pressure and air pollutants (NO_2, PM_{10}, O_3, CO, SO_2). PM_{10} and NO_2 was stronger relative with air pressure than other air pollutants. Temperature was negative correlation with air pollutants and significantly. Humidity was negative correlation with air pollutions, especially for O_3 (Table 20).

Analysis of relationships between the time-dependent variables (meteorological factors) and air pollution each year is shown in the appendix.

4.4 seasonality and air pollution

CO was highest level in winter (median of average = 1180.2 μg/m^3), lowest concentration of pollutants in the rainy season (median of average = 761.9 μg/m^3). Summer was the moderate (median of average = 783.5 μg/m^3). The tendency in very year was similar (Figure 15).

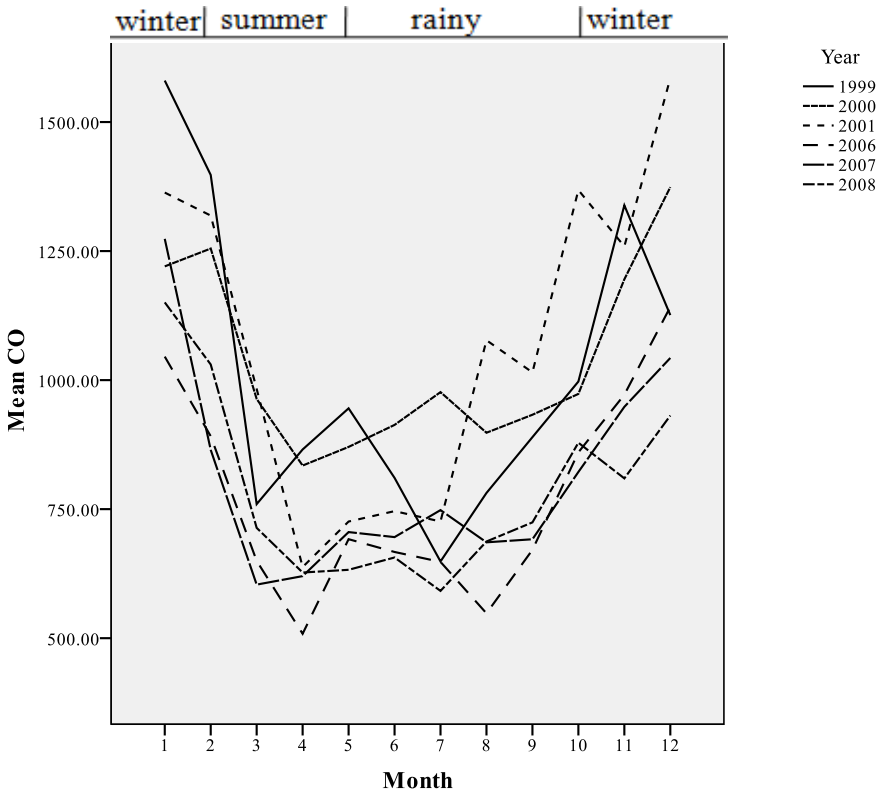


Figure 15 The mean of CO (μg/m^3), by month and year

O_3 was air pollutant which highest in winter (median of average = 38.5 µg/m^3), O_3 as CO that lowest concentration in the rainy season (median of average = 23.1 µg/m^3). The concentration of ozone was the moderate in summer (median of average = 31.8 µg/m^3).The tendency was similarly in very year (Figure 16).

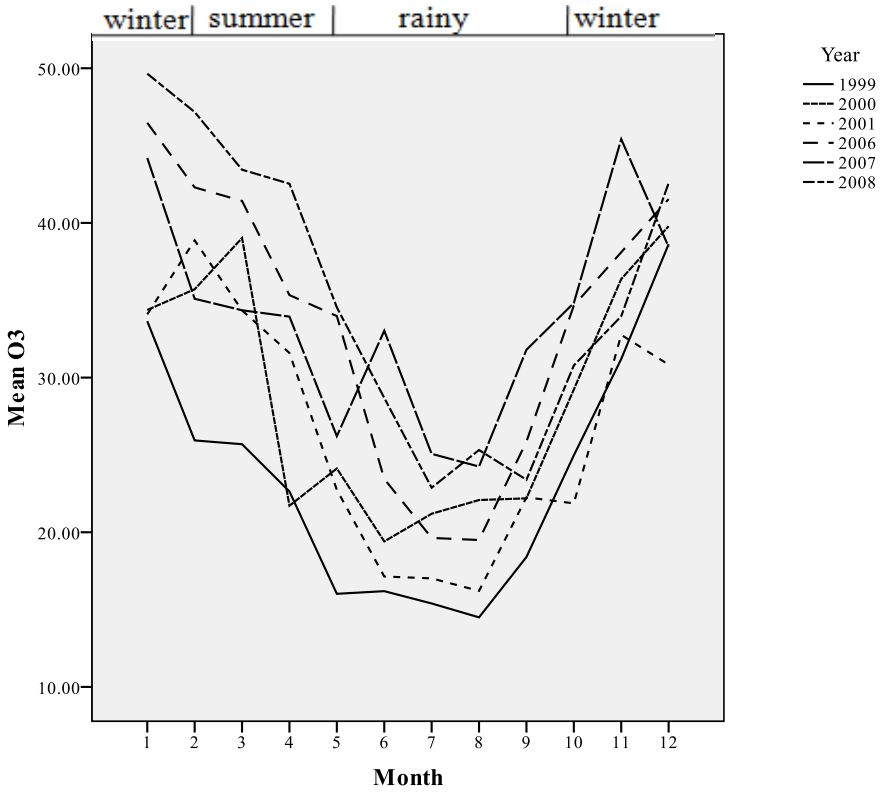


Figure 16 The mean of O_3 (µg/m^3), by month and year

They had the association between season and NO_2 concentration. There were similar condition among NO_2 CO and O_3 that was highest in winter (median of average NO_2 = 58 µg/m^3). The lowest concentration of NO_2 was in the rainy season (median of average = 33.7 µg/m^3). The moderate concentration was in summer (median of average = 38.8 µg/m^3).The characteristic of situation was similar in every year (Figure 17).

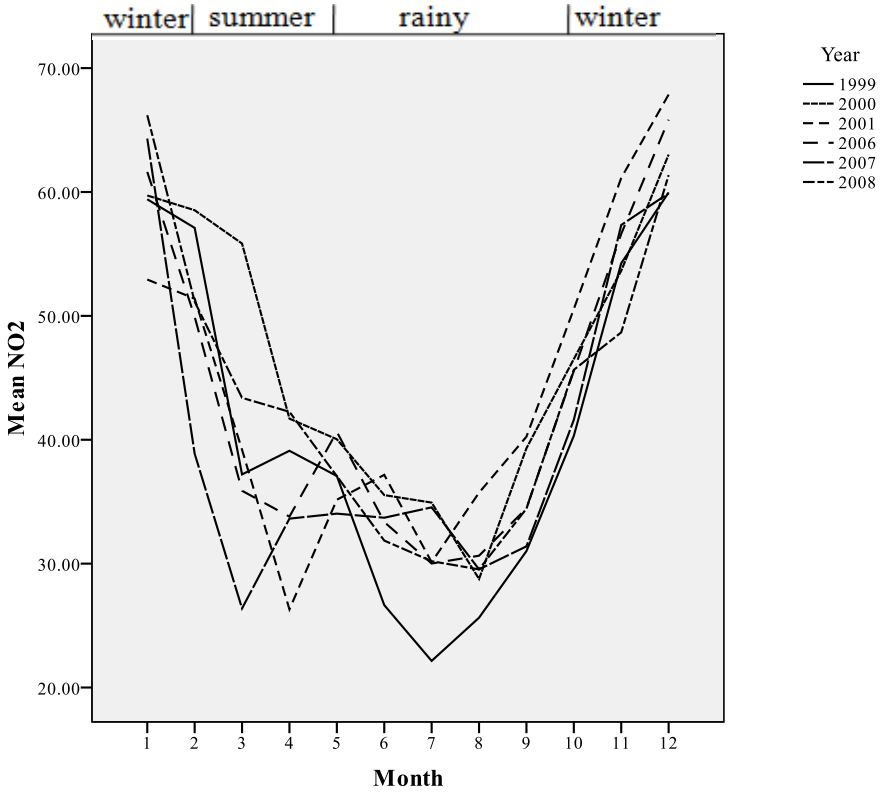


Figure 17 The mean of NO_2 (µg/m^3), by month and year

SO_2 was similar situation every year. The tendency appeared to low in the summer season (median of average = 13.2 µg/m^3). Especially in the end of summer but the rainy season remained lowest on median average for 6 year consolidate (median of average NO_2 = 13.1 µg/m^3). The high concentration of SO_2 was in the winter season (median of average = 14.9 µg/m^3). The characteristic of condition of SO_2 was swing in every year (Figure 18).

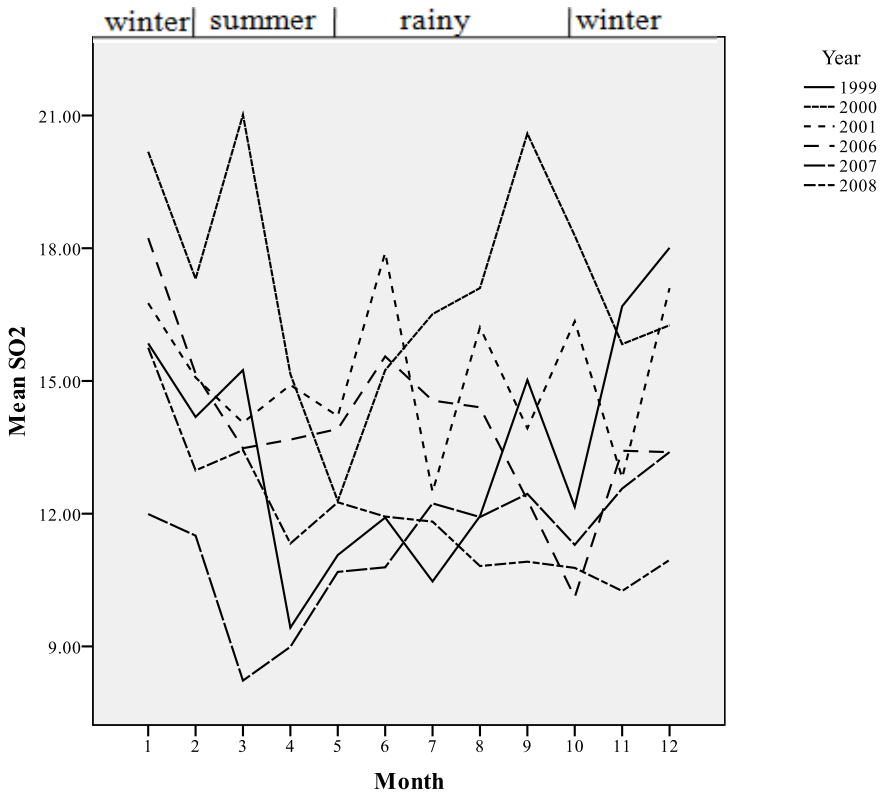


Figure 18 The mean of SO_2 (µg/m^3), by month and year

PM_{10} was highest in winter (median of average = 63.8 µg/m^3), lowest concentration of pollutants in the rainy season (median of average = 37.3 µg/m^3). Summer was the moderate (median of average = 44.4 µg/m^3).The tendency in every year was similar to CO (Figure 19).

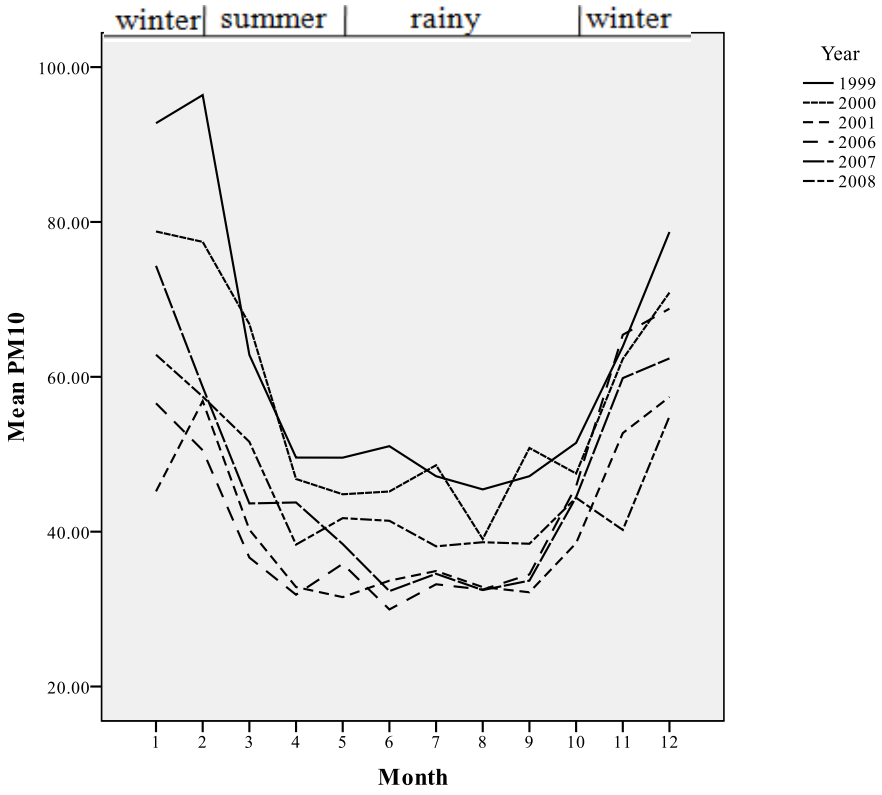


Figure 19 The mean of PM_{10} (µg/m^3), by month and year

The meteorological information had characteristic different between Temperature and Air pressure but Temperature had the similar direction with Humidity. Humidity was high in summer and rainy season but lower in the winter season. The condition were shown on figure 20 to figure 22. This study did not find association between air pressure and mortality in Bangkok, so air pressure was excluded from model. Temperature in the same day of event and Humidity on the day before event were included for non-external mortality model. The Humidity moving average of day before and the same day when event occurred was used in circulatory mortality and respiratory mortality.

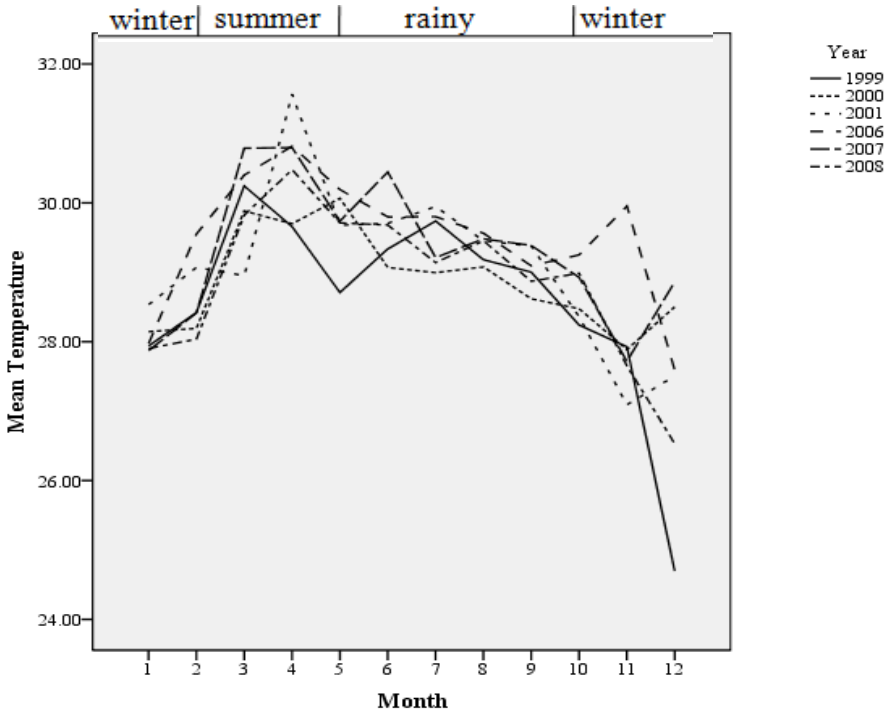


Figure 20 The mean of Temperature (C°), by month and year

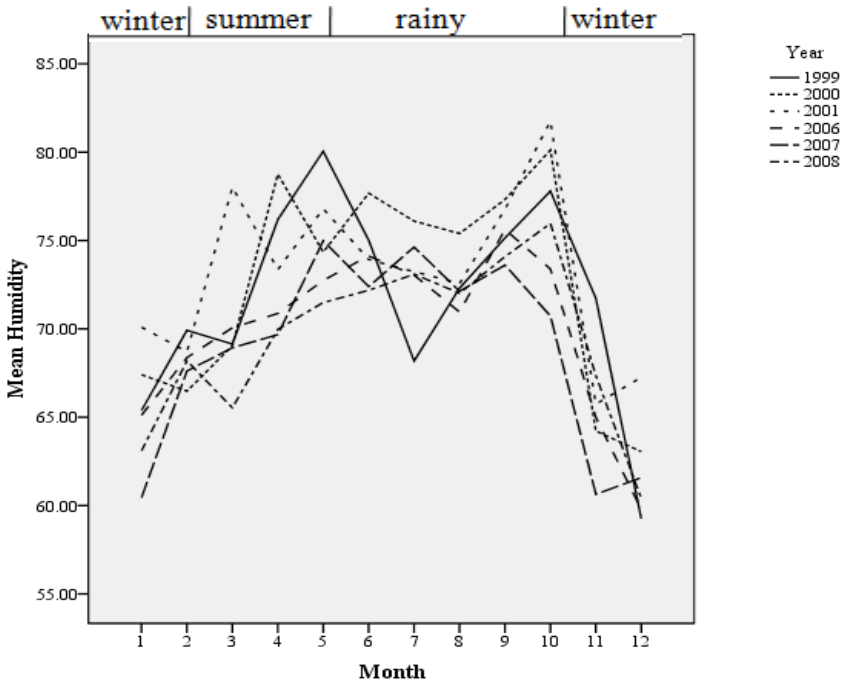


Figure 21 The mean of Relative Humidity (rH), by month and year

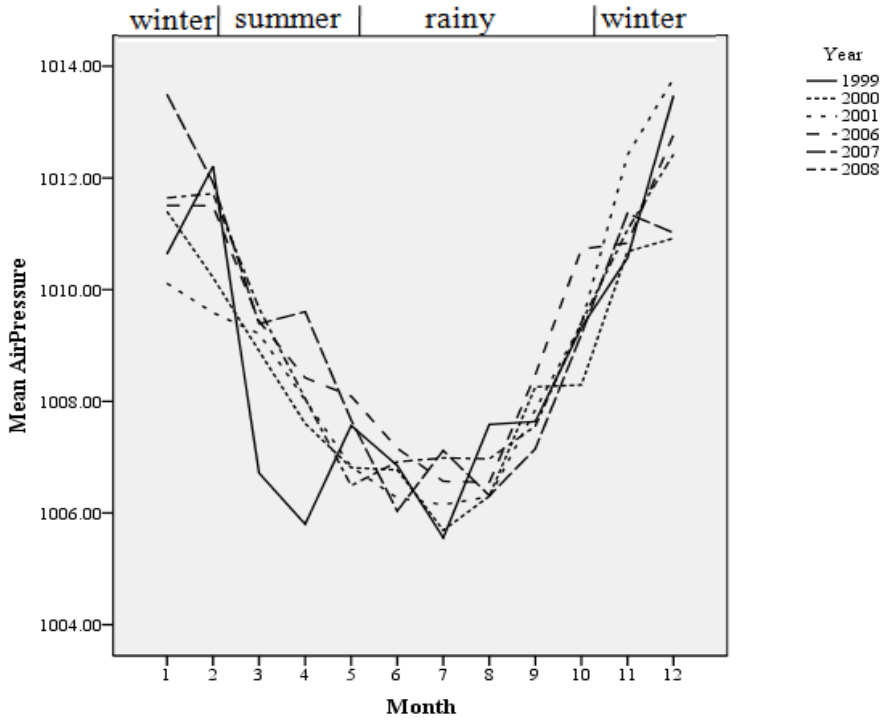


Figure 22 The mean of Air Pressure (Pa), by month and year

4.5 Single-pollutant with different lags and moving average

The purpose of study as determine the different sex, age group and place of death. The station with more than 25 percentage missing air pollution level was removed and Increase 10 μg/m^3 in O_3, NO_2 SO_2 and PM_{10} but 100 μg/m^3 for CO. using air pollution lag1 lag2 and moving average (lag0 – 1, lag0-2) the results were show as below.

Table 21: Crude Odds ratios (95%) for single-pollutant different lags and moving average (MV) by type mortality in all 6 years

Air Pollution	Non-external			Circulatory			Respiratory		
	OR	95%CI		OR	95%CI		OR	95%CI	
		lower	upper		lower	upper		lower	upper
CO (per 100μg/m^3)									
Lag 0	1.0018	1.0002	1.0033	1.0072	1.0030	1.0114	1.0009	0.9956	1.0063
Lag 1	1.0016	1.0001	1.0032	1.0058	1.0016	1.0100	0.9994	0.9941	1.0048
Lag 2	1.0026	1.0010	1.0041	1.0085	1.0043	1.0128	1.0006	0.9952	1.0061
MV 0-1	1.0020	1.0003	1.0037	1.0078	1.0032	1.0124	1.0002	0.9944	1.0061
MV 0-2	1.0028	1.0009	1.0046	1.0099	1.0050	1.0149	1.0005	0.9941	1.0068
O_3 (per 10μg/m^3)									
Lag 0	1.0066	1.0017	1.0115	1.0178	1.0047	1.0312	1.0243	1.0078	1.0412
Lag 1	1.0074	1.0026	1.0123	1.0161	1.0029	1.0295	1.0108	0.9944	1.0274
Lag 2	1.0097	1.0047	1.0146	1.0173	1.0039	1.0308	1.0223	1.0057	1.0392
MV 0-1	1.0089	1.0034	1.0145	1.0218	1.0068	1.0369	1.0224	1.0038	1.0414
MV 0-2	1.0119	1.0059	1.0179	1.0260	1.0096	1.0426	1.0289	1.0085	1.0498
NO_2 (per 10μg/m^3)									
Lag 0	1.0053	1.0014	1.0092	1.0143	1.0038	1.0249	1.0151	1.0020	1.0283
Lag 1	1.0053	1.0014	1.0093	1.0140	1.0034	1.0246	1.0082	0.9951	1.0215
Lag 2	1.0083	1.0043	1.0122	1.0170	1.0063	1.0277	1.0067	0.9935	1.0201
MV 0-1	1.0061	1.0020	1.0103	1.0163	1.0050	1.0277	1.0135	0.9994	1.0277
MV 0-2	1.0082	1.0037	1.0127	1.0197	1.0076	1.0319	1.0135	0.9994	1.0277
SO_2 (per 10μg/m^3)									
Lag 0	1.0176	1.0066	1.0287	1.0338	1.0039	1.0646	1.0390	1.0006	1.0788
Lag 1	1.0139	1.0029	1.0250	0.9936	0.9644	1.0236	1.0423	1.0037	1.0824
Lag 2	1.0062	0.9951	1.0173	1.0116	0.9818	1.0422	1.0504	1.0110	1.0912
MV 0-1	1.0193	1.0071	1.0316	1.0167	0.9840	1.0504	1.0500	1.0071	1.0947
MV 0-2	1.0177	1.0046	1.0310	1.0184	0.9832	1.0548	1.0619	1.0152	1.1107
PM_{10} (per 10 μg/m^3)									
Lag 0	1.0088	1.0055	1.0120	1.0173	1.0083	1.0263	1.0139	1.0028	1.0251
Lag 1	1.0088	1.0055	1.0121	1.0152	1.0062	1.0243	1.0124	1.0013	1.0237
Lag 2	1.0097	1.0064	1.0130	1.0166	1.0076	1.0257	1.0146	1.0034	1.0260
MV 0-1	1.0100	1.0065	1.0135	1.0185	1.0089	1.0282	1.0149	1.0031	1.0270
MV 0-2	1.0116	1.0079	1.0153	1.0207	1.0106	1.0310	1.0173	1.0048	1.0301

Table 22: Crude Odds ratios (95%) for single-pollutant different lags and moving average (MV), by type mortality in year 1999-2001

Air Pollution	Non-external			Circulatory			Respiratory		
	OR	95%CI		OR	95%CI		OR	95%CI	
		lower	upper		lower	upper		lower	upper
CO (per 100µg/m^3)									
Lag 0	1.0011	0.9993	1.0029	1.0083	1.0034	1.0132	0.9965	0.9902	1.0029
Lag 1	1.0008	0.9989	1.0026	1.0056	1.0007	1.0106	0.9977	0.9913	1.0042
Lag 2	1.0023	1.0005	1.0041	1.0098	1.0048	1.0148	0.9992	0.9927	1.0058
MV 0-1	1.0011	0.9991	1.0031	1.0083	1.0030	1.0137	0.9965	0.9895	1.0036
MV 0-2	1.0019	0.9998	1.0041	1.0108	1.0050	1.0166	0.9970	0.9894	1.0046
O_3 (per 10µg/m^3)									
Lag 0	1.0084	1.0012	1.0156	1.0146	0.9949	1.0346	1.0346	1.0088	1.0612
Lag 1	1.0131	1.0059	1.0203	1.0197	0.9999	1.0398	1.0153	0.9899	1.0413
Lag 2	1.0088	1.0016	1.0161	1.0238	1.0038	1.0443	1.0024	0.9773	1.0282
MV 0-1	1.0137	1.0056	1.0220	1.0221	0.9996	1.0450	1.0320	1.0028	1.0620
MV 0-2	1.0156	1.0066	1.0247	1.0301	1.0052	1.0557	1.0268	0.9950	1.0596
NO_2 (per 10µg/m^3)									
Lag 0	1.0048	0.9992	1.0104	1.0149	0.9996	1.0304	0.9992	0.9797	1.0190
Lag 1	1.0044	0.9988	1.0100	1.0171	1.0018	1.0326	0.9984	0.9789	1.0184
Lag 2	1.0077	1.0021	1.0135	1.0255	1.0100	1.0413	0.9914	0.9717	1.0115
MV 0-1	1.0054	0.9993	1.0114	1.0186	1.0021	1.0354	0.9986	0.9775	1.0201
MV 0-2	1.0074	1.0009	1.0139	1.0251	1.0074	1.0431	0.9951	0.9727	1.0180
SO_2 (per 10µg/m^3)									
Lag 0	1.0163	1.0038	1.0290	1.0355	1.0012	1.0710	1.0329	0.9885	1.0792
Lag 1	1.0144	1.0019	1.0271	1.0042	0.9706	1.0389	1.0457	1.0009	1.0924
Lag 2	1.0057	0.9932	1.0184	1.0198	0.9854	1.0553	1.0453	0.9997	1.0929
MV 0-1	1.0186	1.0048	1.0325	1.0239	0.9866	1.0626	1.0475	0.9984	1.0992
MV 0-2	1.0168	1.0020	1.0318	1.0274	0.9873	1.0691	1.0565	1.0033	1.1124
PM_{10}(per 10µg/m^3)									
Lag 0	1.0089	1.0044	1.0134	1.0247	1.0120	1.0375	1.0027	0.9867	1.0190
Lag 1	1.0082	1.0037	1.0127	1.0197	1.0071	1.0325	1.0055	0.9894	1.0219
Lag 2	1.0080	1.0035	1.0126	1.0231	1.0104	1.0360	1.0036	0.9875	1.0200
MV 0-1	1.0098	1.0050	1.0147	1.0255	1.0119	1.0393	1.0047	0.9875	1.0223
MV 0-2	1.0109	1.0058	1.0161	1.0291	1.0146	1.0438	1.0054	0.9870	1.0241

The differen lags and moving averages of unadjusted weather exhibited different odds ratios. The data set of 6 years were found more related between air pollution and mortality than separate periods (Table 21 and Table 22). PM_{10} related with non-external mortality, circulatory mortality and respiratory mortality where lag 2 had concentration level higher than lag 1, moving average 0-2 was more relate than moving average 0-1 (Table 20).

Table 23: Crude Odds ratios (95%) for single-pollutant different lags and moving average (MV), by type mortality in year 2006-2008

Air Pollution	Non-external			Circulatory			Respiratory		
	OR	95%CI		OR	95%CI		OR	95%CI	
		lower	upper		lower	upper		lower	upper
CO (per 100µg/m^3)									
Lag 0	1.0036	1.0006	1.0066	1.0044	0.9965	1.0124	1.0109	1.0013	1.0206
Lag 1	1.0040	1.0009	1.0070	1.0061	0.9981	1.0141	1.0033	0.9936	1.0130
Lag 2	1.0033	1.0002	1.0063	1.0052	0.9972	1.0133	1.0038	0.9941	1.0136
MV 0-1	1.0046	1.0013	1.0079	1.0064	0.9976	1.0152	1.0086	0.9980	1.0193
MV 0-2	1.0051	1.0015	1.0087	1.0075	0.9979	1.0171	1.0085	0.9970	1.0201
O_3 (per 10µg/m^3)									
Lag 0	1.0050	0.9985	1.0117	1.0205	1.0028	1.0385	1.0171	0.9957	1.0391
Lag 1	1.0026	0.9960	1.0092	1.0133	0.9957	1.0313	1.0076	0.9863	1.0293
Lag 2	1.0103	1.0036	1.0171	1.0120	0.9943	1.0301	1.0369	1.0148	1.0594
MV 0-1	1.0049	0.9974	1.0124	1.0215	1.0016	1.0419	1.0157	0.9916	1.0405
MV 0-2	1.0089	1.0008	1.0170	1.0228	1.0011	1.0449	1.0304	1.0039	1.0576
NO_2 (per 10µg/m^3)									
Lag 0	1.0058	1.0004	1.0113	1.0138	0.9994	1.0284	1.0276	1.0101	1.0455
Lag 1	1.0062	1.0008	1.0117	1.0111	0.9967	1.0258	1.0160	0.9984	1.0338
Lag 2	1.0087	1.0033	1.0142	1.0094	0.9949	1.0241	1.0186	1.0010	1.0366
MV 0-1	1.0069	1.0011	1.0127	1.0143	0.9988	1.0299	1.0249	1.0062	1.0440
MV 0-2	1.0089	1.0027	1.0151	1.0149	0.9984	1.0316	1.0267	1.0067	1.0471
SO_2 (per 10µg/m^3)									
Lag 0	1.0220	0.9990	1.0455	1.0285	0.9685	1.0921	1.0563	0.9815	1.1369
Lag 1	1.0121	0.9892	1.0355	0.9601	0.9031	1.0206	1.0326	0.9582	1.1128
Lag 2	1.0077	0.9849	1.0309	0.9866	0.9287	1.0481	1.0646	0.9886	1.1466
MV 0-1	1.0218	0.9958	1.0485	0.9927	0.9270	1.0629	1.0573	0.9723	1.1498
MV 0-2	1.0212	0.9929	1.0504	0.9874	0.9163	1.0639	1.0792	0.9847	1.1828
PM_{10}(per 10µg/m^3)									
Lag 0	1.0087	1.0039	1.0135	1.0096	0.9969	1.0225	1.0239	1.0086	1.0395
Lag 1	1.0096	1.0048	1.0144	1.0106	0.9979	1.0236	1.0185	1.0032	1.0340
Lag 2	1.0117	1.0068	1.0165	1.0099	0.9971	1.0229	1.0245	1.0090	1.0403
MV 0-1	1.0102	1.0051	1.0153	1.0114	0.9978	1.0251	1.0238	1.0076	1.0403
MV 0-2	1.0123	1.0070	1.0177	1.0125	0.9982	1.0270	1.0275	1.0104	1.0448

The pollutants that has same direction association between two periods were PM_{10} and O_3 which effected on non-external mortality (Table 21 and Table 22). The respiratory in 2006-2008 was relation to all PM_{10} categorical when un-adjustment for time-dependent variable was conducted on single model (Table 23).

Table 24: Adjusted Odds ratios (95%) for single-pollutant different lags and moving average (MV), by type mortality in all 6 years

Air Pollution	Non-external[a]			Circulatory[b]			Respiratory[b]		
	OR	95%CI		OR	95%CI		OR	95%CI	
		lower	upper		lower	upper		lower	upper
CO (per 100µg/m^3)									
Lag 0	1.0028	1.0012	1.0044	1.0070	1.0028	1.0112	1.0004	0.9951	1.0058
Lag 1	1.0019	1.0003	1.0034	1.0055	1.0013	1.0097	0.9989	0.9935	1.0043
Lag 2	1.0023	1.0007	1.0039	1.0082	1.0040	1.0124	0.9999	0.9945	1.0053
MV 0-1	1.0028	1.0011	1.0046	1.0075	1.0029	1.0121	0.9996	0.9937	1.0055
MV 0-2	1.0033	1.0014	1.0051	1.0095	1.0046	1.0145	0.9996	0.9933	1.0060
O_3 (per 10µg/m^3)									
Lag 0	0.9981	0.9927	1.0034	1.0137	0.9984	1.0292	1.0113	0.9923	1.0307
Lag 1	0.9981	0.9925	1.0039	1.0116	0.9967	1.0267	0.9950	0.9767	1.0136
Lag 2	1.0050	0.9998	1.0103	1.0137	0.9997	1.0280	1.0137	0.9962	1.0314
MV 0-1	0.9974	0.9909	1.0039	1.0177	0.9998	1.0359	1.0041	0.9821	1.0266
MV 0-2	1.0010	0.9939	1.0081	1.0223	1.0030	1.0420	1.0116	0.9878	1.0359
NO_2 (per 10µg/m^3)									
Lag 0	1.0082	1.0039	1.0126	1.0120	1.0012	1.0230	1.0095	0.9961	1.0231
Lag 1	1.0062	1.0021	1.0103	1.0119	1.0012	1.0228	1.0032	0.9899	1.0167
Lag 2	1.0084	1.0043	1.0125	1.0153	1.0045	1.0262	1.0025	0.9891	1.0161
MV 0-1	1.0084	1.0038	1.0130	1.0139	1.0023	1.0257	1.0074	0.9929	1.0220
MV 0-2	1.0102	1.0053	1.0151	1.0173	1.0049	1.0299	1.0066	0.9912	1.0221
SO_2 (per 10µg/m^3)									
Lag 0	1.0087	0.9976	1.0198	1.0307	1.0006	1.0616	1.0309	0.9925	1.0708
Lag 1	1.0051	0.9940	1.0162	0.9915	0.9624	1.0216	1.0377	0.9991	1.0778
Lag 2	1.0000	0.9889	1.0111	1.0112	0.9814	1.0419	1.0493	1.0099	1.0902
MV 0-1	1.0084	0.9962	1.0208	1.0135	0.9808	1.0473	1.0422	0.9993	1.0869
MV 0-2	1.0064	0.9932	1.0198	1.0157	0.9805	1.0522	1.0554	1.0087	1.1042
PM_{10}(per 10µg/m^3)									
Lag 0	1.0077	1.0039	1.0114	1.0157	1.0058	1.0258	1.0058	0.9936	1.0182
Lag 1	1.0066	1.0030	1.0103	1.0132	1.0035	1.0230	1.0051	0.9932	1.0173
Lag 2	1.0081	1.0046	1.0116	1.0149	1.0054	1.0244	1.0092	0.9974	1.0210
MV 0-1	1.0084	1.0044	1.0124	1.0169	1.0062	1.0276	1.0064	0.9933	1.0196
MV 0-2	1.0100	1.0059	1.0143	1.0192	1.0081	1.0305	1.0090	0.9952	1.0229

[a]Adjusted temperature lag0 and humidity lag1 for non-external mortality.
[b]Adjusted humidity MV 0-1 for circulatory mortality and respiratory mortality.

The association was weak or lower when adjusted meteorological variables but CO and NO_2 were similar statistically significant to non-external mortality and circulatory mortality. The association between respiratory mortality and PM_{10} was low when adjusted weather on single model (Table 24).

Table 25: Adjusted Odds ratios (95%) for single-pollutant different lags and moving average (MV), by type mortality in year 1999-2001

Air Pollution	Non-external[a]			Circulatory[b]			Respiratory[b]		
	OR	95%CI		OR	95%CI		OR	95%CI	
		lower	upper		lower	upper		lower	upper
CO (per 100µg/m^3)									
Lag 0	1.0018	0.9999	1.0036	1.0081	1.0032	1.0130	0.9962	0.9899	1.0026
Lag 1	1.0007	0.9989	1.0025	1.0054	1.0005	1.0103	0.9973	0.9908	1.0037
Lag 2	1.0018	0.9999	1.0036	1.0095	1.0045	1.0145	0.9985	0.9920	1.0051
MV 0-1	1.0015	0.9995	1.0035	1.0081	1.0027	1.0135	0.9961	0.9891	1.0031
MV 0-2	1.0020	0.9998	1.0041	1.0105	1.0047	1.0163	0.9963	0.9887	1.0039
O_3 (per 10µg/m^3)									
Lag 0	0.9971	0.9892	1.0051	1.0076	0.9844	1.0313	1.0202	0.9899	1.0514
Lag 1	1.0021	0.9935	1.0108	1.0149	0.9924	1.0379	0.9964	0.9678	1.0257
Lag 2	1.0028	0.9951	1.0106	1.0205	0.9996	1.0418	0.9914	0.9654	1.0181
MV 0-1	0.9992	0.9894	1.0090	1.0163	0.9889	1.0445	1.0110	0.9759	1.0473
MV 0-2	1.0011	0.9904	1.0119	1.0263	0.9967	1.0567	1.0023	0.9654	1.0406
NO_2 (per 10µg/m^3)									
Lag 0	1.0071	1.0010	1.0132	1.0126	0.9969	1.0285	0.9927	0.9729	1.0129
Lag 1	1.0045	0.9987	1.0104	1.0151	0.9996	1.0309	0.9932	0.9734	1.0134
Lag 2	1.0074	1.0015	1.0132	1.0240	1.0083	1.0400	0.9871	0.9672	1.0074
MV 0-1	1.0069	1.0004	1.0134	1.0163	0.9993	1.0335	0.9918	0.9704	1.0136
MV 0-2	1.0085	1.0016	1.0155	1.0229	1.0048	1.0413	0.9879	0.9651	1.0112
SO_2 (per 10µg/m^3)									
Lag 0	1.0060	0.9933	1.0188	1.0327	0.9983	1.0684	1.0253	0.9808	1.0717
Lag 1	1.0052	0.9925	1.0180	1.0027	0.9690	1.0375	1.0421	0.9973	1.0890
Lag 2	0.9988	0.9862	1.0116	1.0197	0.9853	1.0554	1.0452	0.9994	1.0930
MV 0-1	1.0068	0.9928	1.0209	1.0213	0.9839	1.0601	1.0409	0.9916	1.0926
MV 0-2	1.0046	0.9896	1.0198	1.0253	0.9852	1.0671	1.0513	0.9980	1.1073
PM_{10}(per 10µg/m^3)									
Lag 0	1.0059	1.0010	1.0108	1.0244	1.0105	1.0385	0.9919	0.9745	1.0095
Lag 1	1.0038	0.9990	1.0087	1.0182	1.0046	1.0319	0.9967	0.9796	1.0142
Lag 2	1.0047	1.0000	1.0094	1.0218	1.0086	1.0351	0.9973	0.9806	1.0142
MV 0-1	1.0057	1.0004	1.0111	1.0251	1.0101	1.0403	0.9933	0.9746	1.0124
MV 0-2	1.0066	1.0009	1.0123	1.0288	1.0130	1.0449	0.9939	0.9742	1.0141

[a]Adjusted temperature lag0 and humidity lag1 for non-external mortality.

[b]Adjusted humidity MV 0-1 for circulatory mortality and respiratory mortality.

The two periods of study show non-constant air pollutant effects when look the overall table 24 and table 25 after adjust the models for weather. Example for relationship between circulatory mortality and CO or PM_{10} that significant in first period but did not display in second period (Table 25 and Table 26).

Table 26: Adjusted Odds ratios (95%) for single-pollutant different lags and moving average (MV), by type mortality in year 2006-2008

Air Pollution	Non-external[a]			Circulatory[b]			Respiratory[b]		
	OR	95%CI		OR	95%CI		OR	95%CI	
		lower	upper		lower	upper		lower	upper
CO ($100\mu g/m^3$)									
Lag 0	1.0058	1.0026	1.0090	1.0040	0.9961	1.0121	1.0101	1.0005	1.0198
Lag 1	1.0052	1.0021	1.0082	1.0057	0.9977	1.0138	1.0025	0.9928	1.0122
Lag 2	1.0037	1.0006	1.0068	1.0048	0.9968	1.0129	1.0028	0.9931	1.0126
MV 0-1	1.0067	1.0033	1.0102	1.0059	0.9971	1.0148	1.0076	0.9970	1.0184
MV 0-2	1.0070	1.0033	1.0107	1.0069	0.9974	1.0166	1.0073	0.9958	1.0189
O_3 ($10\mu g/m^3$)									
Lag 0	0.9986	0.9914	1.0059	1.0182	0.9980	1.0389	1.0056	0.9814	1.0304
Lag 1	0.9948	0.9872	1.0025	1.0090	0.9892	1.0292	0.9942	0.9705	1.0184
Lag 2	1.0069	0.9997	1.0141	1.0082	0.9893	1.0275	1.0312	1.0077	1.0551
MV 0-1	0.9956	0.9870	1.0044	1.0187	0.9952	1.0428	0.9997	0.9717	1.0286
MV 0-2	1.0006	0.9911	1.0101	1.0195	0.9940	1.0455	1.0181	0.9873	1.0498
NO_2 ($10\mu g/m^3$)									
Lag 0	1.0093	1.0030	1.0156	1.0116	0.9967	1.0268	1.0233	1.0052	1.0418
Lag 1	1.0077	1.0018	1.0135	1.0090	0.9942	1.0241	1.0115	0.9935	1.0297
Lag 2	1.0093	1.0036	1.0151	1.0075	0.9927	1.0225	1.0148	0.9969	1.0331
MV 0-1	1.0099	1.0033	1.0165	1.0119	0.9959	1.0282	1.0200	1.0006	1.0397
MV 0-2	1.0118	1.0049	1.0187	1.0123	0.9953	1.0297	1.0216	1.0009	1.0426
SO_2 ($10\mu g/m^3$)									
Lag 0	1.0160	0.9930	1.0396	1.0240	0.9640	1.0876	1.0458	0.9713	1.1261
Lag 1	1.0033	0.9804	1.0266	0.9565	0.8996	1.0169	1.0248	0.9507	1.1046
Lag 2	1.0025	0.9798	1.0257	0.9853	0.9274	1.0467	1.0611	0.9852	1.1428
MV 0-1	1.0123	0.9864	1.0390	0.9874	0.9219	1.0576	1.0455	0.9610	1.1375
MV 0-2	1.0111	0.9828	1.0401	0.9825	0.9116	1.0590	1.0680	0.9741	1.1709
PM_{10}($10\mu g/m^3$)									
Lag 0	1.0100	1.0043	1.0159	1.0065	0.9923	1.0210	1.0192	1.0021	1.0367
Lag 1	1.0101	1.0046	1.0156	1.0080	0.9942	1.0221	1.0130	0.9964	1.0299
Lag 2	1.0121	1.0069	1.0174	1.0076	0.9941	1.0213	1.0204	1.0041	1.0371
MV 0-1	1.0118	1.0057	1.0180	1.0084	0.9933	1.0237	1.0184	1.0003	1.0369
MV 0-2	1.0143	1.0080	1.0207	1.0095	0.9938	1.0256	1.0225	1.0036	1.0417

[a]Adjusted temperature lag0 and humidity lag1 for non-external mortality.

[b]Adjusted humidity MV 0-1 for circulatory mortality and respiratory mortality.

This study aimed to use air pollutants on moving average because requirement to comparison with time series study, even though the moving average 0-2 was very high effect on any kind of mortality.

4.6 Association between Air pollution and Non-external mortality

Moving average for air pollution concentration during the day before event occurred and the same day of event was used because they were very strong significant when put into model. The station was removed if more than 25 percentage missing air pollution level for each parameter. The unit conversion was used in the study period and increased 10 $\mu g/m^3$ for O_3, NO_2 SO_2 and PM_{10} but increased 100 $\mu g/m^3$ for CO. PM_{10} was highest statistic significant association with Non-external mortality and positive relationship, Wald chi-square was 31.65 (p-value<.001). SO_2 was shown with strongest OR than others air pollutions (OR 1.0193, 95%CI 1.0071-1.0316). There were statistically significant association between all air pollutions and non-external mortality in unadjusted model analysis (Table 27).

Table 27: Single pollutant model unadjusted for weather effected on non external mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (100µg/m^3)	0.0020	0.0009	5.54	1	0.019	1.0020	1.0003	1.0037
O_3 (10µg/m^3)*24hr*	0.0089	0.0028	10.19	1	<.001	1.0089	1.0034	1.0145
O_3 (10µg/m^3)*8hr*	0.0050	0.0012	17.30	1	<.001	1.0050	1.0027	1.0074
NO_2 (10µg/m^3)	0.0061	0.0021	8.31	1	0.004	1.0061	1.0020	1.0103
SO_2 (10µg/m^3)	0.0191	0.0061	9.63	1	0.002	1.0193	1.0071	1.0316
PM_{10} (10µg/m^3)	0.0100	0.0018	31.65	1	<.001	1.0100	1.0065	1.0135

In the first period, PM_{10} SO_2 and O_3 were significant relate to Non-external mortality. The second period PM_{10} CO and NO_2 were significant relate to Non-external mortality. PM_{10} was the air pollution which association to Non-external mortality in both period (1999-2001 and 2006-2008) with significant statistically positive relationship (OR 1.0098 95%CI 1.0050-1.0147 and OR 1.0102 95%CI 1.0051-1.0153). PM_{10} Log likelihood in second period was -154252.44, Chi-square was 15.54

(P-value <.001), was smaller than PM_{10} in the first which Log likelihood of was -150745.32, Chi-square was 16 (P-value <.001) for unadjusted model analysis between 2 periods (Table 28).

Table 28: Single pollutant model unadjusted for weather effected on non external mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (100µg/m^3)	1.0011	0.9991	1.0031	1.0046	1.0013	1.0079
O_3 (10µg/m^3)*24hr*	1.0137	1.0056	1.0220	1.0049	0.9974	1.0124
O_3 (10µg/m^3)*8hr*	1.0068	1.0033	1.0103	1.0035	1.0003	1.0068
NO_2 (10µg/m^3)	1.0054	0.9993	1.0114	1.0069	1.0011	1.0127
SO_2 (10µg/m^3)	1.0186	1.0048	1.0325	1.0218	0.9958	1.0485
PM_{10} (10µg/m^3)	1.0098	1.0050	1.0147	1.0102	1.0051	1.0153

The single-pollutant model was adjusted by Temperature lag0 and Humidity lag1. Results were shown PM_{10} CO and NO_2 were be significant. PM_{10} was highest statistic significant association with Non-external mortality and positive relationship, wald was 17.23 (p-value<.001) but less than single model unadjusted for weather. SO_2 and O_3 was not association to air pollutions. There were statistically significant associations between some air pollutions and natural mortality for single model analysis (Table 29).

Table 29: Single pollutant model adjusted for weather effected on non external mortality in 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (100µg/m^3)	0.0028	0.0009	10.33	1	<.001	1.0028	1.0011	1.0046
O_3 (10µg/m^3)*24hr*	-0.0026	0.0033	0.63	1	0.428	0.9974	0.9909	1.0039
O_3 (10µg/m^3)*8hr*	0.0004	0.0014	0.08	1	0.772	1.0004	0.9976	1.0032
NO_2 (10µg/m^3)	0.0084	0.0023	12.89	1	<.001	1.0084	1.0038	1.0130
SO_2 (10µg/m^3)	0.0084	0.0062	1.81	1	0.179	1.0084	0.9962	1.0208
PM_{10} (10µg/m^3)	0.0084	0.0020	17.23	1	<.001	1.0084	1.0044	1.0124

Adjust for Temperature lag0 and Humidity lag1

In the first period (Table 30), PM_{10} and NO_2 were significantly related to non-external mortality. The second period PM_{10} CO and NO_2 were significant relate to Non-external mortality similar to first period. PM_{10} and NO_2 were the air pollutions which association to Non-external mortality in both period (1999-2001 and 2006-2008) with significant statistically positive relationship. PM_{10} had OR strong in second period than first period as the NO_2. Log likelihood of PM_{10} in second period was -154230.1, Chi-square was 60.22(P-value <.001), was smaller than PM_{10} in the first which Log likelihood of was -150715.12, Chi-square was 76.41 (P-value <.001) for single model analysis between 2 periods but Chi-square was higher than simple model. NO_2 Log likelihood in second period was -154232.94, Chi-square was 54.54 (P-value <.001), was smaller than NO_2 in the first which Log likelihood of was -150715.2, Chi-square was 76.25 (P-value <.001) for single model analysis between 2 periods show the difference was higher than in the unadjusted model.

Table 30: Single pollutant model adjusted for weather effected on non external mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (100µg/m^3)	1.0015	0.9995	1.0035	1.0067	1.0033	1.0102
O_3 (10µg/m^3)*24hr*	0.9992	0.9894	1.0090	0.9956	0.9870	1.0044
O_3 (10µg/m^3)*8hr*	1.0012	0.9970	1.0054	0.9997	0.9958	1.0035
NO_2 (10µg/m^3)	1.0069	1.0004	1.0134	1.0099	1.0033	1.0165
SO_2 (10µg/m^3)	1.0068	0.9928	1.0209	1.0123	0.9864	1.0390
PM_{10} (10µg/m^3)	1.0057	1.0004	1.0111	1.0118	1.0057	1.0180

Adjust for Temperature lag0 and Humidity lag1

Table 30 shows non statistic significant different between two periods of air pollutants (O_3 NO_2 SO_2 and PM_{10}) effect on non-external mortality but CO had the statistic significant different between 2 periods (Table 31) the details are shown in table 29.

Table 31: Comparison between two periods for non-external mortality

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (P2 - P1)	0.0052	0.002	6.57	1	0.010	1.0052	1.0012	1.0092
O_3 (P2 - P1)*24hr*	-0.0035	0.007	0.28	1	0.597	0.9965	0.9835	1.0096
O_3 (P2 - P1)*8hr*	-0.0015	0.003	0.28	1	0.596	0.9985	0.9928	1.0041
NO_2 (P2 - P1)	0.0030	0.005	0.40	1	0.525	1.0030	0.9938	1.0122
SO_2 (P2 - P1)	0.0030	0.005	0.40	1	0.525	1.0030	0.9938	1.0122
PM_{10} (P2 -P1)	0.0060	0.004	2.16	1	0.142	1.0061	0.9980	1.0142

Adjust for dummy variable Temperature lag0 period 1, 2 and Humidity lag1 period 1, 2.

P1 was the study period 1999-2001, P2 was the study period 2006-2008.

The all six years non-external mortality data set on table 32 show O_3 was the co-pollution of CO that made CO strong significant (P-value <.001) and Wald equalled 12.7, larger than the co-pollutants. NO_2 and PM_{10} were co-pollution that made CO did not reach statistic significant, especially for PM_{10}.

Table 32: Co-pollution of CO effect estimation on non-external mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (single)	0.0028	0.0009	10.33	1	<.001	1.0028	1.0011	1.0046
CO + O_3 *24hr*	0.0033	0.0009	12.70	1	<.001	1.0033	1.0015	1.0051
CO + NO_2	0.0011	0.0013	0.67	1	0.413	1.0011	0.9985	1.0037
CO + SO_2	0.0027	0.0009	8.78	1	0.003	1.0027	1.0009	1.0045
CO + PM_{10}	0.0003	0.0013	0.04	1	0.839	1.0003	0.9977	1.0028

Adjust for Temperature lag0 and Humidity lag1

Table 33: Co-pollution of CO effect estimation on non-external mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (single)	1.0015	0.9995	1.0035	1.0067	1.0033	1.0102
CO + O_3 *24hr*	1.0016	0.9996	1.0037	1.0085	1.0048	1.0123
CO + NO_2	0.9999	0.9970	1.0028	1.0079	1.0016	1.0142
CO + SO_2	1.0014	0.9993	1.0034	1.0072	1.0035	1.0110
CO + PM_{10}	0.9997	0.9966	1.0028	1.0039	0.9986	1.0092

Adjust for Temperature lag0 and Humidity lag1

Ttable 33 shows co-pollution of CO between two periods. O_3 made CO strong significant by OR equalled 0.0085 bigger than others co-pollution in the second period. PM_{10} were co-pollution that made CO did not reach statistic significant in both periods.

Table 34: Co-pollution of NO_2 effect estimation to on non-external mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
NO_2 (single)	0.0084	0.0023	12.89	1	<.001	1.0084	1.0038	1.0130
NO_2 + CO	0.0063	0.0035	3.23	1	0.072	1.0063	0.9994	1.0132
NO_2 + O_3 *24hr*	0.0118	0.0026	20.05	1	<.001	1.0119	1.0067	1.0171
NO_2 + SO_2	0.0082	0.0025	11.10	1	<.001	1.0083	1.0034	1.0132
NO_2 + PM_{10}	0.0019	0.0038	0.25	1	0.618	1.0019	0.9944	1.0094

Adjust for Temperature lag0 and Humidity lag1

Table 35: Co-pollution of NO_2 effect estimation to on non-external mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
NO_2 (single)	1.0069	1.0004	1.0134	1.0099	1.0033	1.0165
NO_2 + CO	1.0071	0.9976	1.0166	0.9974	0.9857	1.0093
NO_2 + O_3 *24hr*	1.0088	1.0016	1.0161	1.0150	1.0075	1.0226
NO_2 + SO_2	1.0065	0.9998	1.0133	1.0110	1.0034	1.0185
NO_2 + PM_{10}	1.0036	0.9934	1.0139	0.9986	0.9874	1.0099

Adjust for Temperature lag0 and Humidity lag1

The table 35 show co-pollution of NO_2 between two periods. O_3 made NO_2 strong significant by OR was 1.5% bigger than others co-pollution in the second period, OR was 0.88% in the first period. PM_{10} and CO were co-pollution that made NO_2 did not reach significance in either period.

The all six years non-external mortality data set on table 34 show O_3 was the co-pollution of NO_2 that made NO_2 strong significant (P-value <.001) and Wald was 20.05 bigger than others co-pollution. CO and PM_{10} were co-pollution that made NO_2 did not reach statistic significant, especially for PM_{10}.

The all six years non-external mortality data set on table 36 show O_3 was the co-pollution of PM_{10} that made PM_{10} strong significant (P-value <.001) and Wald was 24.81, larger than co-pollutants. NO_2 were co-pollution that made PM_{10} was smallest association to non-external mortality.

Table 36: Co-pollution of PM_{10} effect estimation to on non-external mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
PM_{10} (single)	0.0084	0.0020	17.23	1	<.001	1.0084	1.0044	1.0124
PM_{10} + CO	0.0079	0.0030	6.92	1	0.009	1.0079	1.0020	1.0139
PM_{10} + O_3 *24hr*	0.0111	0.0022	24.81	1	<.001	1.0112	1.0068	1.0156
PM_{10} + NO_2	0.0071	0.0033	4.57	1	0.033	1.0071	1.0006	1.0136
PM_{10} + SO_2	0.0083	0.0021	15.42	1	<.001	1.0083	1.0042	1.0125

[a] Adjust for Temperature lag0 and Humidity lag1

Table 37 shows co-pollution of PM_{10} between two periods. O_3 made PM_{10} strong significant by OR was 1.81% bigger than others co-pollution in the second period, OR was 0.11% in the first period. CO was co-pollution that made PM_{10} did not reach statistic significant in both periods. NO_2 was co-pollution than made PM_{10} did not significant in the first period.

Table 37: Co-pollution of PM_{10} effect estimation on non-external mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
PM_{10} (single)	1.0057	1.0004	1.0111	1.0118	1.0057	1.0180
PM_{10} + CO	1.0064	0.9982	1.0146	1.0066	0.9973	1.0160
PM_{10} + O_3 *24hr*	1.0067	1.0011	1.0125	1.0181	1.0110	1.0253
PM_{10} + NO_2	1.0034	0.9951	1.0119	1.0129	1.0023	1.0236
PM_{10} + SO_2	1.0054	1.0000	1.0109	1.0136	1.0066	1.0206

Adjust for Temperature lag0 and Humidity lag1

4.7 Association between Air pollution and Circulatory mortality

Table 38 presented estimates of the coefficient correlation and odds ratio from single pollutant analyses. O_3 was highest association with circulatory mortality and positive relationship statistically significant, wald was 8.18 (p-value=0.004). SO_2 was not reach statistic significant but association show positive direction. There were statistic significant association between CO O_3 NO_2 and PM_{10} concentration and circulatory mortality for single model analysis with unadjusted for meteorological information (Table 38).

Table 38: Single pollutant model unadjusted for weather effected on circulatory mortality in 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (100µg/m^3)	0.0078	0.0023	11.16	1	<.001	1.0078	1.0032	1.0124
O_3 (10µg/m^3)*24hr*	0.0215	0.0075	8.18	1	0.004	1.0218	1.0068	1.0369
O_3 (10µg/m^3)*8hr*	0.0096	0.0033	8.64	1	0.003	1.0096	1.0032	1.0161
NO_2 (10µg/m^3)	0.0162	0.0057	8.03	1	0.005	1.0163	1.0050	1.0277
SO_2 (10µg/m^3)	0.0166	0.0167	0.99	1	0.320	1.0167	0.9840	1.0504
PM_{10} (10µg/m^3)	0.0183	0.0048	14.35	1	0.000	1.0185	1.0089	1.0282

Table 39 show results in the first period, PM_{10} NO_2 and O_3 were significant relate to circulatory mortality. The second period only O_3 was significant association to circulatory mortality for single pollutant model with significant statistically positive direction (OR 1.0215 95%CI 1.0016-1.0419). Log likelihood of O_3 in second period was -21716.77, Chi-square was 4.46 (P-value 0.035), was smaller than O_3 in the 6 year model (Table 36) which Log likelihood of was -41808.437, Chi-square was 8.16 (p=0.004).

Table 39: Single pollutant model unadjusted for weather effected on circulatory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (100µg/m^3)	1.0083	1.0030	1.0137	1.0064	0.9976	1.0152
O_3 (10µg/m^3)*24hr*	1.0221	0.9996	1.0450	1.0215	1.0016	1.0419
O_3 (10µg/m^3)*8hr*	1.0115	1.0019	1.0211	1.0081	0.9994	1.0169
NO_2 (10µg/m^3)	1.0186	1.0021	1.0354	1.0143	0.9988	1.0299
SO_2 (10µg/m^3)	1.0239	0.9866	1.0626	0.9927	0.9270	1.0629
PM_{10} (10µg/m^3)	1.0255	1.0119	1.0393	1.0114	0.9978	1.0251

Table 40 was single pollutant model adjusted for weather by Temperature lag0 and Humidity lag1. Results show PM_{10} CO and NO_2 associated to circulatory and significant similar to non-external mortality cause. CO was highest statistic significant association with circulatory mortality and positive relationship, wald was 10.32 (p-value<.001) but less than single pollutant model unadjusted.

Table 40: Single pollutant model adjusted for weather effected on circulatory mortality in 6 years of study

	Coef.	**SE**	**Wald**	**df**	**P-value**	**OR**	**95%CI**	
							lower	**upper**
CO (100µg/m^3)	0.0075	0.0023	10.32	1	<.001	1.0075	1.0029	1.0121
O_3 (10µg/m^3)*24hr*	0.0175	0.0091	3.74	1	0.053	1.0177	0.9998	1.0359
O_3 (10µg/m^3)*8hr*	0.0080	0.0040	4.07	1	0.044	1.0080	1.0002	1.0159
NO_2 (10µg/m^3)	0.0138	0.0059	5.50	1	0.019	1.0139	1.0023	1.0257
SO_2 (10µg/m^3)	0.0134	0.0167	0.64	1	0.424	1.0135	0.9808	1.0473
PM_{10} (10µg/m^3)	0.0167	0.0054	9.69	1	0.002	1.0169	1.0062	1.0276

Adjust for Humidity mv01

SO_2 and O_3 was not association to circulatory mortality for single model adjustment analysis (Table 40).

In the first period (Table 41), PM_{10} and CO were significant relate to circulatory mortality. The second period all pollutants were not significant for circulatory mortality. The association between PM_{10} and circulatory mortality was found in first period (1999-2001) with statistically significant positive relationship, log likelihood was -20086.698, Chi-square was 13.64(P-value <.001) for single model adjusted weather analysis. The study show significant association between CO and circulatory mortality in first period as PM_{10} , log likelihood of CO was -20087.754, Chi-square was 11.52 (P-value 0.003) for single pollutant model (Table 41).

Table 41: Single pollutant model adjusted for weather effected on circulatory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (100µg/m^3)	1.0081	1.0027	1.0135	1.0059	0.9971	1.0148
O_3 (10µg/m^3)*24hr*	1.0163	0.9889	1.0445	1.0187	0.9952	1.0428
O_3 (10µg/m^3)*8hr*	1.0101	0.9984	1.0220	1.0064	0.9960	1.0170
NO_2 (10µg/m^3)	1.0163	0.9993	1.0335	1.0119	0.9959	1.0282
SO_2 (10µg/m^3)	1.0213	0.9839	1.0601	0.9874	0.9219	1.0576
PM_{10} (10µg/m^3)	1.0251	1.0101	1.0403	1.0084	0.9933	1.0237

Adjust for Humidity mv01

Table 42 show non statistic significant different all air pollutants that effect to circulatory mortality between two periods on CO O_3 NO_2 SO_2 and PM_{10}. Even though CO in first period was statistic significant positive association with circulatory mortality when second period was not significant.

Table 42: Comparison between two periods for circulatory mortality

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (P2 - P1)	-0.0021	0.005	0.17	1	0.683	0.9979	0.9877	1.0082
O_3 (P2 - P1)*24hr*	0.0024	0.018	0.02	1	0.898	1.0024	0.9669	1.0391
O_3 (P2 - P1)*8hr*	-0.0037	0.008	0.21	1	0.646	0.9963	0.9808	1.0121
NO_2 (P2 - P1)	-0.0043	0.012	0.13	1	0.716	0.9957	0.9729	1.0190
SO_2 (P2 - P1)	-0.0337	0.040	0.72	1	0.398	0.9668	0.8941	1.0454
PM_{10} (P2 -P1)	-0.0164	0.011	2.32	1	0.128	0.9837	0.9632	1.0047

Adjust for dummy variable Temperature lag0 period 1, 2 and Humidity lag1 period 1, 2.

P1 was the study period 1999-2001, P2 was the study period 2006-2008.

The all six years circulatory mortality data set on table 43 show SO_2 was the co-pollution of CO that made CO strong significant (P-value 0.002) and Wald was 9.66 bigger than others co-pollution. PM_{10} were co-pollution that made CO did not reach statistic significant.

Table 43: Co-pollution of CO effect estimation on circulatory mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (single)	0.0075	0.0023	10.32	1	<.001	1.0075	1.0029	1.0121
CO + O_3 *24hr*	0.0068	0.0024	7.82	1	0.005	1.0068	1.0020	1.0116
CO + NO_2	0.0076	0.0035	4.80	1	0.028	1.0076	1.0008	1.0145
CO + SO_2	0.0075	0.0024	9.66	1	0.002	1.0075	1.0028	1.0123
CO + PM_{10}	0.0047	0.0035	1.79	1	0.181	1.0047	0.9978	1.0116

Adjust for Humidity mv01

The table 44 show co-pollution of CO between two periods. NO_2 made CO strong significant by OR was 0.91% bigger than others co-pollution in the first period. PM_{10} was co-pollution that made CO did not reach statistic significant in both periods. All co-pollutions were made CO did not significant in the second period.

Table 44: Co-pollution of CO effect estimation on circulatory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (single)	1.0081	1.0027	1.0135	1.0059	0.9971	1.0148
CO + O_3 *24hr*	1.0078	1.0023	1.0133	1.0038	0.9943	1.0134
CO + NO_2	1.0091	1.0013	1.0169	1.0014	0.9851	1.0180
CO + SO_2	1.0078	1.0023	1.0133	1.0076	0.9981	1.0172
CO + PM_{10}	1.0029	0.9948	1.0112	1.0054	0.9915	1.0195

Adjust for Humidity mv01

Associations with circulatory mortality are shown in Table 45. SO_2 was the co-pollution of NO_2 that made NO_2 strong significant (P-value 0.027) and Wald was 4.87, bigger than co-pollutants. O_3, CO and PM_{10} were co-pollution that made NO_2 did not reach statistical significance.

The table 46 show co-pollution of NO_2 between two periods. All co-pollutions were made NO_2 did not significant in the both periods.

Table 45: Co-pollution of NO_2 effect estimation on circulatory mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
NO_2 (single)	0.0138	0.0059	5.50	1	0.019	1.0139	1.0023	1.0257
NO_2 + CO	-0.0005	0.0088	0.00	1	0.957	0.9995	0.9824	1.0169
NO_2 + O_3 *24hr*	0.0109	0.0066	2.71	1	0.100	1.0110	0.9979	1.0242
NO_2 + SO_2	0.0136	0.0062	4.87	1	0.027	1.0137	1.0015	1.0260
NO_2 + PM_{10}	-0.0024	0.0099	0.06	1	0.805	0.9976	0.9785	1.0171

Adjust for Humidity mv01

Table 46: Co-pollution of NO_2 effect estimation on circulatory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
NO_2 (single)	1.0163	0.9993	1.0335	1.0119	0.9959	1.0282
NO_2 + CO	0.9957	0.9718	1.0201	1.0097	0.9800	1.0404
NO_2 + O_3 *24hr*	1.0146	0.9961	1.0335	1.0075	0.9892	1.0260
NO_2 + SO_2	1.0148	0.9975	1.0325	1.0163	0.9985	1.0345
NO_2 + PM_{10}	0.9872	0.9618	1.0133	1.0151	0.9856	1.0455

Adjust for Humidity mv01

The all six years circulatory mortality data set on table 47 show SO_2 was the co-pollution of PM_{10} that made PM_{10} strong significant (P-value 0.003) and Wald was 9.04 bigger than others co-pollution, but remained less than single pollutant. CO was co-pollution that made PM_{10} did not reach statistic significant.

Table 47: Co-pollution of PM_{10} effect estimation on circulatory mortality in all 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
PM_{10} (single)	0.0167	0.0054	9.69	1	0.002	1.0169	1.0062	1.0276
PM_{10} + CO	0.0087	0.0081	1.17	1	0.279	1.0088	0.9930	1.0248
PM_{10} + O_3*24hr*	0.0150	0.0059	6.39	1	0.011	1.0151	1.0034	1.0270
PM_{10} + NO_2	0.0185	0.0090	4.23	1	0.040	1.0187	1.0009	1.0368
PM_{10} + SO_2	0.0169	0.0056	9.04	1	0.003	1.0170	1.0059	1.0282

Adjust for Humidity mv01

Table 48 shows co-pollution of PM_{10} between two periods. NO_2 made PM_{10} strong significant by OR was 3.4% bigger than others co-pollution in the first period. CO was co-pollution that made PM_{10} did not reach statistic significant in both periods. All co-pollutions were made PM_{10} no significant in the second period.

Table 48: Co-pollution on PM_{10} effect estimation to circulatory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
PM_{10} (single)	1.0251	1.0101	1.0403	1.0084	0.9933	1.0237
PM_{10} + CO	1.0188	0.9961	1.0421	1.0012	0.9776	1.0255
PM_{10} + O_3*24hr*	1.0249	1.0090	1.0411	1.0031	0.9857	1.0208
PM_{10} + NO_2	1.0340	1.0106	1.0579	0.9965	0.9690	1.0247
PM_{10} + SO_2	1.0244	1.0090	1.0400	1.0119	0.9951	1.0291

Adjust for Humidity mv01

4.8 Association between Air pollution and Respiratory mortality

The association between PM_{10} and respiratory mortality in 6 years was the highest statistic significant and positive relationship, Wald was 6.10 (p-value=0.014). O_3 was shown with strongest OR than others air pollutions (OR 1.0224, 95%CI 1.0038-1.0414). There were statistic significant association between PM_{10} O_3 SO_2 and respiratory mortality for single pollutant model analysis (Table 49).

Table 49: Single pollutant model unadjusted for weather effected on respiratory mortality in 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (100µg/m^3)	0.0002	0.0030	0.01	1	0.940	1.0002	0.9944	1.0061
O_3 (10µg/m^3)*24hr*	0.0222	0.0094	5.57	1	0.018	1.0224	1.0038	1.0414
O_3 (10µg/m^3)*8hr*	0.0104	0.0041	6.51	1	0.011	1.0104	1.0024	1.0185
NO_2 (10µg/m^3)	0.0134	0.0071	3.53	1	0.060	1.0135	0.9994	1.0277
SO_2 (10µg/m^3)	0.0488	0.0213	5.25	1	0.022	1.0500	1.0071	1.0947
PM_{10} (10µg/m^3)	0.0148	0.0060	6.10	1	0.014	1.0149	1.0031	1.0270

There was association between O_3 and respiratory mortality in the first period for single pollutant model which unadjusted for meteorological information (Table 50). PM_{10} and NO_2 had a positive association that reached statistical significant at moving average of 2 days (OR 1.0238 95%CI 1.0076-1.0403 and OR 1.0249 95%CI 1.0062-1.0440). Log likelihood of PM_{10} was -14545.856, Chi-square was 9.82 (P-value 0.007) in the second period. Log likelihood of NO_2 was -14545.806, Chi-square was 9.92 (P-value 0.007) in second period, was bigger than NO_2 in the first period for single pollutant model analysis (Table 50).

The single model was adjusted by Temperature today and Humidity yesterday. Results were shown no statistic significant for all air pollutants. Table 51 shows SO_2

was highest association positive direction to respiratory mortality, wald was 3.72 (p-value=0.054).

Table 50: Single pollutant model unadjusted for weather effected on respiratory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (100µg/m^3)	0.9965	0.9895	1.0036	1.0086	0.9980	1.0193
O_3 (10µg/m^3)*24hr*	1.0320	1.0028	1.0620	1.0157	0.9916	1.0405
O_3 (10µg/m^3)*8hr*	1.0139	1.0015	1.0264	1.0079	0.9974	1.0186
NO_2 (10µg/m^3)	0.9986	0.9775	1.0201	1.0249	1.0062	1.0440
SO_2 (10µg/m^3)	1.0475	0.9984	1.0992	1.0573	0.9723	1.1498
PM_{10} (10µg/m^3)	1.0047	0.9875	1.0223	1.0238	1.0076	1.0403

Table 51: Single pollutant model adjusted for weather effected on respiratory mortality in 6 years of study

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (100µg/m^3)	-0.0004	0.0030	0.02	1	0.892	0.9996	0.9937	1.0055
O_3 (10µg/m^3)*24hr*	0.0041	0.0113	0.13	1	0.717	1.0041	0.9821	1.0266
O_3 (10µg/m^3)*8hr*	0.0027	0.0049	0.29	1	0.589	1.0027	0.9930	1.0124
NO_2 (10µg/m^3)	0.0073	0.0074	0.99	1	0.319	1.0074	0.9929	1.0220
SO_2 (10µg/m^3)	0.0414	0.0214	3.72	1	0.054	1.0422	0.9993	1.0869
PM_{10} (10µg/m^3)	0.0064	0.0067	0.91	1	0.340	1.0064	0.9933	1.0196

Adjust for Humidity mv01

In the first period (Table 52) there were no statistic significant between air pollution and respiratory but significant in the second period, PM_{10} and NO_2 were significant relate to respiratory mortality in second period. NO_2 were stronger than PM_{10} a little,

log likelihood was -14546,421 Chi-square was 8.69(P-value 0.033), PM_{10} was smaller than NO_2 in the second period which log likelihood of was -14546.277, Chi-square was 8.98 (P-value 0.029) for single pollutant model adjusted meteorological analysis.

Table 52: Single pollutant model adjusted for weather effected on respiratory mortality between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (100µg/m^3)	0.9961	0.9891	1.0031	1.0076	0.9970	1.0184
O_3 (10µg/m^3)*24hr*	1.0110	0.9759	1.0473	0.9997	0.9717	1.0286
O_3 (10µg/m^3)*8hr*	1.0052	0.9901	1.0204	1.0010	0.9884	1.0137
NO_2 (10µg/m^3)	0.9918	0.9704	1.0136	1.0200	1.0006	1.0397
SO_2 (10µg/m^3)	1.0409	0.9916	1.0926	1.0455	0.9610	1.1375
PM_{10} (10µg/m^3)	0.9933	0.9746	1.0124	1.0184	1.0003	1.0369

Adjust for Humidity mv01

Table 53 shows no different air pollutants effect to respiratory mortality between two periods on CO O_3 NO_2 SO_2 and PM_{10}. Even though NO_2 and PM_{10} table 51 show significant in first period and no significant in second period.

Table 53: Comparison between two periods for respiratory mortality

	Coef.	SE	Wald	df	P-value	OR	95%CI	
							lower	upper
CO (P2 - P1)	0.0115	0.006	3.15	1	0.076	1.0116	0.9988	1.0246
O_3 (P2 - P1)*24hr*	-0.0112	0.023	0.24	1	0.628	0.9888	0.9450	1.0347
O_3 (P2 - P1)*8hr*	-0.0042	0.010	0.17	1	0.679	0.9959	0.9765	1.0156
NO_2 (P2 - P1)	0.0280	0.015	3.57	1	0.059	1.0284	0.9990	1.0587
SO_2 (P2 - P1)	0.0045	0.050	0.01	1	0.928	1.0045	0.9114	1.1071
PM_{10} (P2 -P1)	0.0250	0.013	3.50	1	0.061	1.0253	0.9988	1.0524

Adjust for dummy variable Temperature lag0 period 1, 2 and Humidity lag1 period 1, 2.

P1 was the study period 1999-2001, P2 was the study period 2006-2008.

4.9 Stratified time independent variables

Table 54 show PM_{10} effected on mortality in female 4.11% more than male. Elderly age at least 65 year was higher risk than another group, excess risk was 1.05% increase of chance on non-external mortality. The subjects who died outside hospital, exposed PM_{10} and CO, increase risk on non-external mortality.

Table 54: Excess risk (ER;%) of mortality for single-pollutant mv0-1 with 10 μg/m^3 increase on NO_2, SO_2, O_3, PM_{10} and 100 μg/m^3 increase on CO in all 6 years

	CO			O_3			NO_2			SO_2			PM_{10}		
	ER	95%CI		ER	95%CI		ER	95%CI		ER	95%CI		ER	95%CI	
Non-external**															
Gender															
Male	0.22	-0.01	0.46	-0.44	-1.30	0.43	0.73	0.11	1.35	-0.21	-1.83	1.43	0.74	0.20	1.27
Female	0.36	0.10	0.62	-0.03	-1.00	0.95	0.99	0.30	1.69	2.22	0.35	4.11	0.98	0.38	1.58
Age group															
Less than 65	0.24	0.00	0.49	-0.27	-1.19	0.65	0.62	-0.03	1.28	0.05	-1.64	1.77	0.64	0.08	1.20
At least 65	0.32	0.07	0.57	-0.24	-1.15	0.68	1.07	0.41	1.72	1.73	-0.04	3.54	1.05	0.49	1.62
At least 75	0.34	0.02	0.66	0.05	-1.12	1.24	1.00	0.16	1.84	0.68	-1.60	3.00	0.93	0.20	1.67
Place of death															
In hospital	0.23	0.02	0.44	-0.42	-1.19	0.36	0.76	0.21	1.32	1.35	-0.15	2.87	0.72	0.24	1.21
Out hospital	0.38	0.08	0.68	0.07	-1.10	1.25	1.00	0.18	1.84	-0.22	-2.34	1.94	1.07	0.36	1.78
Circulatory‡															
Gender															
Male	0.68	0.07	1.29	2.20	-0.18	4.64	1.25	-0.29	2.82	-0.06	-4.34	4.41	1.97	0.55	3.41
Female	0.85	0.15	1.54	1.22	-1.48	3.99	1.57	-0.19	3.37	3.18	-1.80	8.42	1.32	-0.28	2.95
Age group															
Less than 65	0.73	0.07	1.39	3.87	1.22	6.58	1.59	-0.10	3.30	-0.34	-4.99	4.54	1.93	0.39	3.50
At least 65	0.77	0.13	1.41	-0.09	-2.51	2.40	1.22	-0.39	2.85	2.89	-1.65	7.65	1.46	-0.01	2.95
At least 75	0.76	-0.12	1.66	1.51	-1.81	4.94	1.46	-0.72	3.69	-0.30	-6.39	6.18	1.71	-0.29	3.76
Place of death															
In hospital	0.75	0.24	1.25	2.30	0.31	4.34	1.40	0.11	2.70	1.72	-1.87	5.43	1.58	0.41	2.77
Out hospital	0.77	-0.35	1.91	-0.50	-4.49	3.66	1.36	-1.36	4.16	-0.51	-8.29	7.92	2.17	-0.35	4.75
Respiratory‡															
Gender															
Male	-0.02	-0.74	0.71	0.18	-2.59	3.03	0.95	-0.87	2.80	3.45	-1.79	8.96	1.01	-0.64	2.68
Female	-0.09	-1.08	0.91	0.82	-2.76	4.52	0.38	-1.97	2.78	5.71	-1.58	13.55	0.02	-2.13	2.20
Age group															
Less than 65	-0.09	-0.91	0.73	-1.46	-4.63	1.83	0.33	-1.77	2.48	5.77	-0.23	12.13	-0.65	-2.53	1.27
At least 65	0.01	-0.82	0.86	2.01	-1.01	5.12	1.08	-0.89	3.08	2.61	-3.42	9.01	1.78	-0.02	3.62
At least 75	0.41	-0.62	1.45	2.23	-1.43	6.03	1.97	-0.44	4.43	1.62	-5.71	9.53	2.94	0.72	5.22
Place of death															
In hospital	0.06	-0.59	0.73	0.80	-1.62	3.29	0.68	-0.91	2.29	5.90	1.00	11.04	0.72	-0.73	2.19
Out hospital	-0.45	-1.73	0.85	-1.42	-6.51	3.95	1.00	-2.36	4.48	-1.65	-10.20	7.70	0.29	-2.68	3.36

** Adjusted temperature lag0 and humidity lag1 for non-external mortality.

‡ Adjusted humidity mv0-1 for circulatory mortality and respiratory mortality

Table 55 shows first period NO_2 and SO_2 effects on non-external mortality in female 1.21% and 2.38% more than male. Elderly age at least 65 year was higher risk than another group, excess risk was increase of chance on non-external mortality but no significant association. The subjects died out side hospital who exposed PM_{10} and CO had the increase risk on non-external mortality in the first period.

Table 55: Excess risk (ER;%) of mortality for single-pollutant mv0-1 with 10 µg/m^3 increase on NO_2, SO_2, O_3, PM_{10} and 100 µg/m^3 increase on CO 1999-2001

	CO			O_3			NO_2			SO_2			PM_{10}		
	ER	95%CI		ER	95%CI		ER	95%CI		ER	95%CI		ER	95%CI	
Non-external**															
Gender															
Male	0.07	-0.20	0.33	-0.47	-1.76	0.83	0.29	-0.57	1.16	-0.62	-2.46	1.25	0.41	-0.29	1.12
Female	0.25	-0.05	0.56	0.44	-1.04	1.95	1.21	0.22	2.20	2.38	0.25	4.56	0.78	-0.02	1.60
Age group															
Less than 65	0.17	-0.11	0.44	-0.15	-1.48	1.20	0.66	-0.23	1.56	0.14	-1.76	2.07	0.56	-0.17	1.29
At least 65	0.12	-0.17	0.42	-0.02	-1.44	1.42	0.70	-0.24	1.66	1.29	-0.75	3.37	0.58	-0.19	1.36
At least 75	0.12	-0.26	0.50	1.10	-0.78	3.01	0.69	-0.55	1.95	0.60	-2.05	3.32	0.70	-0.32	1.73
Place of death															
In hospital	0.01	-0.23	0.26	-0.02	-1.21	1.19	0.30	-0.50	1.11	1.29	-0.43	3.05	0.25	-0.41	0.91
Out hospital	0.41	0.07	0.75	-0.22	-1.88	1.47	1.42	0.31	2.54	-0.49	-2.85	1.92	1.19	0.28	2.11
Circulatory‡															
Gender															
Male	0.60	-0.11	1.32	2.43	-1.25	6.24	0.61	-1.63	2.90	-0.05	-4.93	5.08	2.27	0.27	4.32
Female	1.07	0.27	1.89	0.65	-3.41	4.89	2.93	0.37	5.56	4.97	-0.75	11.02	2.81	0.56	5.10
Age group															
Less than 65	0.82	0.06	1.59	3.83	-0.12	7.94	2.00	-0.40	4.46	1.00	-4.31	6.60	3.61	1.46	5.81
At least 65	0.79	0.04	1.55	-0.50	-4.26	3.42	1.26	-1.11	3.69	3.18	-2.02	8.64	1.45	-0.63	3.57
At least 75	0.60	-0.47	1.68	2.18	-3.23	7.89	1.42	-1.92	4.88	1.46	-5.68	9.14	2.72	-0.25	5.78
Place of death															
In hospital	0.72	0.14	1.31	2.65	-0.36	5.74	1.39	-0.45	3.26	2.06	-1.99	6.28	2.24	0.62	3.89
Out hospital	1.27	-0.10	2.66	-3.80	-10.32	3.20	2.95	-1.35	7.43	2.52	-6.87	12.85	4.01	0.15	8.01
Respiratory‡															
Gender															
Male	-0.47	-1.32	0.39	0.02	-4.19	4.41	-1.14	-3.74	1.54	2.51	-3.37	8.76	-0.41	-2.68	1.91
Female	-0.23	-1.47	1.02	3.42	-2.79	10.02	-0.19	-3.90	3.66	7.45	-1.29	16.97	-1.22	-4.50	2.17
Age group															
Less than 65	0.03	-0.90	0.97	-0.50	-5.04	4.25	0.79	-2.08	3.74	5.27	-1.32	12.30	-0.86	-3.32	1.66
At least 65	-0.96	-2.03	0.12	3.29	-2.14	9.03	-2.94	-6.13	0.36	2.58	-4.69	10.41	-0.42	-3.28	2.52
At least 75	-0.81	-2.15	0.54	4.79	-2.12	12.18	-2.05	-6.10	2.18	1.57	-7.42	11.44	1.07	-2.57	4.86
Place of death															
In hospital	-0.40	-1.21	0.41	2.70	-1.39	6.96	-1.71	-4.17	0.80	6.64	0.85	12.75	-1.03	-3.19	1.19
Out hospital	-0.35	-1.76	1.07	-3.63	-10.27	3.50	1.84	-2.46	6.33	-3.32	-12.38	6.67	0.36	-3.31	4.17

** Adjusted temperature lag0 and humidity lag1 for non-external mortality.

‡ Adjusted humidity mv0-1 for circulatory mortality and respiratory mortality

Table 56 show second period, PM_{10} effected on non-external mortality in female 1.27% more than male. Elderly age at least 75 year was higher risk than another group, excess risk was increase 4.01% of chance on respiratory mortality and significant association when exposed to PM_{10}. The subjects who died outside hospital and exposed to PM_{10}, had the increasing risk on non-external mortality in the second period.

Table 56: Excess risk (ER;%) of mortality for single-pollutant mv0-1 and 10 μg/m^3 increase on NO_2, SO_2, O_3, PM_{10} and 100 μg/m^3 increase on CO 2006-2008

	CO			O_3			NO_2			SO_2			PM_{10}		
	ER	95%CI		ER	95%CI		ER	95%CI		ER	95%CI		ER	95%CI	
Non-external**															
Gender															
Male	0.69	0.22	1.16	-0.48	-1.64	0.70	1.14	0.26	2.04	0.85	-2.63	4.46	1.11	0.28	1.94
Female	0.65	0.14	1.17	-0.39	-1.67	0.91	0.80	-0.17	1.78	1.71	-2.13	5.70	1.27	0.36	2.19
Age group															
Less than 65	0.49	-0.01	1.00	-0.44	-1.69	0.83	0.54	-0.41	1.50	-0.49	-4.18	3.33	0.72	-0.16	1.61
At least 65	0.83	0.35	1.31	-0.43	-1.62	0.78	1.39	0.48	2.30	2.83	-0.78	6.58	1.60	0.75	2.45
At least 75	0.85	0.25	1.46	-0.66	-2.16	0.87	1.26	0.11	2.41	0.63	-3.84	5.30	1.19	0.13	2.27
Place of death															
In hospital	0.81	0.40	1.21	-0.79	-1.81	0.23	1.12	0.35	1.90	1.05	-1.99	4.18	1.18	0.46	1.91
Out hospital	0.31	-0.34	0.97	0.51	-1.13	2.18	0.65	-0.59	1.91	1.78	-3.11	6.91	1.20	0.04	2.37
Circulatory‡															
Gender															
Male	0.87	-0.29	2.04	2.04	-1.06	5.25	1.84	-0.28	4.00	-0.19	-8.87	9.32	1.67	-0.34	3.71
Female	0.21	-1.13	1.57	1.64	-1.93	5.35	0.31	-2.11	2.80	-2.74	-12.41	7.99	-0.27	-2.56	2.07
Age group															
Less than 65	0.46	-0.83	1.77	3.89	0.37	7.55	1.18	-1.17	3.59	-4.92	-14.13	5.27	0.10	-2.10	2.36
At least 65	0.70	-0.49	1.91	0.20	-2.94	3.43	1.20	-0.97	3.41	1.92	-7.14	11.86	1.48	-0.59	3.58
At least 75	1.12	-0.45	2.72	1.13	-3.03	5.46	1.53	-1.33	4.47	-5.30	-16.33	7.17	0.88	-1.81	3.65
Place of death															
In hospital	0.80	-0.18	1.80	2.04	-0.62	4.77	1.43	-0.37	3.25	0.44	-7.02	8.49	0.86	-0.84	2.58
Out hospital	-0.25	-2.21	1.74	1.25	-3.73	6.49	0.27	-3.22	3.89	-7.52	-20.48	7.55	0.77	-2.53	4.19
Respiratory‡															
Gender															
Male	1.17	-0.21	2.56	0.31	-3.33	4.08	2.85	0.33	5.43	6.62	-4.44	18.97	2.51	0.15	4.92
Female	0.18	-1.48	1.86	-0.50	-4.83	4.03	0.75	-2.25	3.85	1.64	-10.93	15.97	0.90	-1.90	3.78
Age group															
Less than 65	-0.51	-2.22	1.23	-2.37	-6.77	2.23	-0.20	-3.29	2.98	8.03	-5.79	23.88	-0.36	-3.26	2.62
At least 65	1.55	0.20	2.92	1.46	-2.15	5.19	3.35	0.87	5.88	2.49	-7.91	14.06	3.18	0.88	5.54
At least 75	2.16	0.54	3.80	1.25	-3.03	5.72	3.98	1.01	7.03	1.58	-10.59	15.40	4.01	1.23	6.87
Place of death															
In hospital	0.98	-0.15	2.13	-0.23	-3.22	2.86	2.34	0.27	4.46	3.87	-5.08	13.65	2.09	0.16	4.07
Out hospital	-0.88	-3.90	2.25	1.45	-6.28	9.83	-0.51	-5.81	5.10	9.67	-13.77	39.48	0.07	-4.91	5.31

** Adjusted temperature lag0 and humidity lag1 for non-external mortality.

‡ Adjusted humidity mv0-1 for circulatory mortality and respiratory mortality.

4.10 Comparison this CCO study to PAPA Time series study

The Poisson log linear model need to adjust for time independent variable such as age, gender, occupation et. That made non-external mortality model was higher estimation if without them. The result of non-external mortality was shown in table 56, PM_{10}, O_3 and SO_2 for unadjusted model were more similar PAPA than adjusted for weather in the year 1999-2001 (Table 57).

Table 57: Comparison excess risk (ER;%) of mortality between PAPA (Time series) with Time stratify case-crossover (CCO in years 1999 - 2001)*

Air pollutions†	PAPA(Time series)			CCO Single Pollution Models					
				Unadjusted			Adjusted		
	ER	95%CI		ER	95%CI		ER	95%CI	
Non-external**									
O_3¶	0.63	0.30	0.95	1.37	0.56	2.20	-0.08	-1.06	0.90
NO_2	1.41	0.89	1.95	0.54	-0.07	1.14	0.69	0.04	1.34
SO_2	1.61	0.08	3.16	1.86	0.48	3.25	0.68	-0.72	2.09
PM_{10}	1.25	0.82	1.69	0.98	0.50	1.47	0.57	0.04	1.11
Circulatory‡									
O_3¶	0.82	0.03	1.63	2.21	-0.04	4.50	1.63	-1.11	4.45
NO_2	1.78	0.47	3.10	1.86	0.21	3.54	1.63	-0.07	3.35
SO_2	0.77	-2.98	4.67	2.39	-1.34	6.26	2.13	-1.61	6.01
PM_{10}	1.90	0.80	3.01	2.55	1.19	3.93	2.51	1.01	4.03
Respiratory‡									
O_3¶	0.89	-0.10	1.90	3.20	0.28	6.20	1.10	-2.41	4.73
NO_2	1.05	-0.60	2.72	-0.14	-2.25	2.01	-0.82	-2.96	1.36
SO_2	1.66	-3.09	6.64	4.75	-0.16	9.92	4.09	-0.84	9.26
PM_{10}	1.01	-0.36	2.40	0.47	-1.25	2.23	-0.67	-2.54	1.24

* Excess risk (ER;%) of mortality for single-pollutant moving average 0-1.
† per 10 μg/m^3 increase on NO_2, SO_2, O_3, PM_{10}, and 100 μg/m^3 increase on CO.
** Adjusted temperature lag0 and humidity lag1 for non-external mortality.
‡ Adjusted humidity mv0-1 for circulatory mortality and respiratory mortality.
¶ In the PAPA study and the present study, ozone was averaged over 8 hr and 24 hr, respectively.

The single model of CCO in all 6 years of the study showed the results very similar between unadjusted meteorology information on non-external mortality and circulatory mortality. The respiratory mortality model of CCO study had more

significant association to exposures in PM_{10}, O_3 and SO_2 for unadjusted model than PAPA study (Table 58).

Table 58: Comparison excess risk (ER;%) of mortality between PAPA (Time series) with Time stratify case-crossover (CCO in all 6 years)*

Air pollutions†	PAPA(Time series)			CCO Single Pollution Models					
				Unadjusted			Adjusted		
	ER	95%CI		ER	95%CI		ER	95%CI	
Non-external**									
O_3¶	0.63	0.30	0.95	0.89	0.34	1.45	-0.26	-0.91	0.39
NO_2	1.41	0.89	1.95	0.61	0.20	1.03	0.84	0.38	1.30
SO_2	1.61	0.08	3.16	1.93	0.71	3.16	0.84	-0.38	2.08
PM_{10}	1.25	0.82	1.69	1.00	0.65	1.35	0.84	0.44	1.24
Circulatory‡									
O_3¶	0.82	0.03	1.63	2.18	0.68	3.69	1.77	-0.02	3.59
NO_2	1.78	0.47	3.10	1.63	0.50	2.77	1.39	0.23	2.57
SO_2	0.77	-2.98	4.67	1.67	-1.60	5.04	1.35	-1.92	4.73
PM_{10}	1.90	0.80	3.01	1.85	0.89	2.82	1.69	0.62	2.76
Respiratory‡									
O_3¶	0.89	-0.10	1.90	2.24	0.38	4.14	0.41	-1.79	2.66
NO_2	1.05	-0.60	2.72	1.35	-0.06	2.77	0.74	-0.71	2.20
SO_2	1.66	-3.09	6.64	5.00	0.71	9.47	4.22	-0.07	8.69
PM_{10}	1.01	-0.36	2.40	1.49	0.31	2.70	0.64	-0.67	1.96

* Excess risk (ER;%) of mortality for single-pollutant moving average 0-1.
† per 10 $\mu g/m^3$ increase on NO_2, SO_2, O_3, PM_{10}, and 100 $\mu g/m^3$ increase on CO.
** Adjusted temperature lag0 and humidity lag1 for non-external mortality.
‡ Adjusted humidity mv0-1 for circulatory mortality and respiratory mortality.
¶ In the PAPA study and the present study, ozone was averaged over 8 hr and 24 hr, respectively.

CHAPTER V

DISCUSSION

This study evaluated air pollution and daily mortality with the case-crossover design, in which time-independent variables were controlled, in a fashion similar to controlling matching variables in matched case-control studies. Adjustment was made for time-dependent meteorologic variables. Specifically, non-external mortality was adjusted for same-day temperature and previous-day humidity, and cardiovascular and respiratory mortality were adjusted for the moving average of same-day and previous-day humidity.

5.1 Sample Collection

The subjects of this study were similar number in each year. The non external deaths were around 33,000-36,000 in each year. There were more deaths in males than females, especially for respiratory mortality. As expected, most deaths occurred in the elderly. The relationship between ambient particulate matter and respiratory mortality was studied in Italy. The authors found females had the suffering from chronic disease and death of respiratory than males. That is, the air pollution effect was stronger in females (Faustini et al., 2011). Serinelli found the elderly was high risk group who exposed NO_2 had positive association with increased mortality (Schneider et al., 2010). Our study showed the place of death was inside hospital more than outside hospital. We arranged the cause of death by sort cases ascending and selected ICD10 code was A00 to R99 for non external mortality group. Cardiovascular and respriatory deaths were coded beginning with I and J, respectively.

Air pollution data were collected by the Pollution Control Department (PCD). PCD officials check data every day. Under PCD quality control procedures, when more than 25% of daily values of a given pollutant were missing for any station, that station was excluded from analysis for that pollutant. Air pollutants was calculated every hour and calibration 1 hour per day that meaning was air pollutants were collected 23 times per day. They calculated for the average of air pollution concentration per hour, per 8 hours and per 24 hours. The air pollutants in this study most of them were 24 hour averages, and 8-hour ozone was also considered. Technicians of PCD monitored

and checked station including instrument once a week. Sometimes the station monitoring equipment had technical problems. Then data could not be collected. Despite these limitations, air pollutant data were available for nearly all days included in this study.

Bureau of Policy and Strategy Department, Ministry of Public Health used program database for the past and changed to Access program to collecting data for the whole country. They receive data file from hospitals and merge for the Thailand Death Certificate. We used the population of the deaths to analyze that appropriate to represent the people who live in Bangkok city.

5.2 Air pollution levels

Our study found positive associations between air pollutant levels and non external mortality in Bangkok. O_3 level in second period was significantly higher than in the first period. Air pollution is an important public health problem as capital city in the world because they increased levels of environmental pollution (Dashdendev et al., 2011). Bangkok is the capital of Thailand that have more cars and transportation vehicle. These source of ambient air pollution which risk of respiratory and circulatory morbidity and mortality in the general population (Wong et al., 2008).

CO concentration in the first period (1999-2001) was higher than second period (2006-2008). This decreasing of air pollution level may in part result from campaigns of motivating using natural gas for vehicles as examined in Barcelona showed using NGV related to reduce air pollutant concentrations (Goncalves et al., 2009). CO level was high in the January or February in the first month of the year and November or December in the late month of the year. This season was winter which people had more consumption and more burning, as seasonal has the strong effects on mortality during winter in China (Qian et al., 2010)

O_3 concentration in the first period were lower than second period. Ozone was the only pollutant that increased in this study. The sources of ozone about thousands types of sources **(**EPA, 2011**)** for example vehicle exhaust, ionic air purifiers, laser printers, photocopiers, elevators or hydraulic pumps those kinds of generated ozone. Ozone in upper troposphere acts as a greenhouse gas. Ozone Absorb the infrared radiation then protect the earth from global warming . This gas around us can harm to lung function

and irritate the respiratory system. Pehnec examined the short term exposed to O_3 and lung function, they found O_3 may affect forced vital capacity (FVC) and forced expiratory volume (FEV) (Pehnec et al., 2011). Further tendency of ozone cite increase slowly as transient effect. Exposed to low concentration of O_3 was risk for severe vascular (Henrotin et al., 2010)

PM_{10} concentration in the first period (1999-2001) was higher than second period (2006-2008). The tendency of PM_{10} was similar to CO but level was less than. The present time, particular matter 2.5 $\mu g/m^3$ is the interesting pollutant for the short term health affect. Bangkok resident release chemicals and particulates into the atmosphere, especially PM_{10} that cause problems for humans as person who died outside hospital was relationship with non external mortality in Bangkok. Schwartz studied the association among airborne particles and daily death and hospital admission, he found deaths outside of hospital was increased in daily deaths (Schwartz, 2001) Sources of Particulate matters were traffic and power plant those are associated with increased adverse health effect (Linares et al., 2010). The major sources in Bangkok city were automotive traffic and residential biomass burning (Chuersuwan et al., 2008). The roadside and under skytrain station in Bangkok sometime has PM_{10} concentration more over 120 $\mu g/m^3$ that is the recommended level (Luksamijarulkul and Kongtip, 2010). PM could be emitted directly from a number of natural and anthropogenic sources as oxidation of SO_2 and NO_2 produce PM. This study show the results as Laurine and others studied on health effects of particulate matter air pollution in Bangkok found the percentage change in daily mortality estimated for each 10 $\mu g/m^3$ change in daily PM_{10} concentration. (Lauraine et al., 1998).

SO_2 concentration in the first period was higher than second period. To study air pollution and harmful health effects should find out the combine exposure because in reality of humans life are continuously exposed to multi air pollution level (Ren et al., 2010). Even though Bangkok is not high level of SO_2 in our study.

NO_2 concentration in both periods of this study vary in every year. Nitrogen dioxide emanate directly from vehicle exhaust and power station, while oxidation of nitric oxide (NO) can transform NO_2 into the atmosphere. Bangkok is not a place of high NO_2 concentration, but sometimes when the traffic is stuck for long time periods

nitrogen dioxide could be high. Exposure to NO_2 is related to traffic (Setton et al., 2008)

Seasonality was association to air pollution level instance for air pollution growing up in winter and low in the rainy season most of them are the same direction as the study in Chaimai they found PM_{10} was increase at dry season and decreased on end of April that is the beginning of rainy season (Pengchai et al., 2009), but SO_2 was different because concentration was highly in each season. In Zagreb, Croatia found seasonality related to air pollution levels (Toth et al., 2011). The PM_{10} positive associated with circulatory disease in winter at Adelaide, South Australia (Hansen et al., 2012). These show season effect on health.

5.3 Air pollution effect on mortality

5.3.1 Non external mortality

Non external mortality was positively associated with all ambient air pollutants that show on Air pollutant single model which did not adjusted for weather, but when we adjusted meteorological data as temperature and humidity then O_3 and SO_2 show no relation to the non external mortality. Several study showed in the case of CCO design both different and similar results. For examples similarly Ren's case-crossover study, Ren and colleague studied the modifiers of short term effects of ozone on mortality and the results showed ozone was associated with changes in all non external deaths (Ren et al., 2010). The daily mortality relevant the increased air pollutants (Ueda et al., 2009). The short term effected of air pollution both of NO_2 and O_3 relative with mortality for all death causes (Stafoggia et al., 2009), this evidence close to our result when whole dataset were analyzed in unadjusted single-pollutant models.

5.3.2 Cardiovascular mortality

The number of cardiovascular deaths was higher than the number of respiratory deaths. We included ICD10 from the code I00-I99 for Cardiovascular mortality. We found the positive association between CVD mortality and air pollution level CO, PM_{10}, O_3 and NO_2 statistical significantly positive direction. The Bangkok residents are high risk for ambient air pollutants because their consumptions are more than rural regions. They use more gas to cooking, burning and transportation. They travel

between home and business place by car, bus and train. The open air windows buses are especially widely used by the low-income portion of Bangkok's population.

Circulatory mortality related to air pollution level instance for CO and PM_{10} in year 1999 to 2001 after adjust for weather, it show precise the short term effect on people risky healthy, most of people died on the days when air pollution level was going up. The higher concentration of the ambient air pollution are followed by transient effect up to short time, but for longer lags there appear to be decreasing in risk (Krishnan et al., 2011) Bangkok is the most ambient air polluted city in Thailand, the monitoring surveillances are still remain conducted to preserve environmental air pollutants in Bangkok. Mortality in cardiovascular disease who hospital were admission for acute conditions as cardiac had the association with air pollutant such as PM_{10} (Serinelli et al., 2010). The ambient air pollution as PM_{10}, SO_2, NO_2 and CO showed that significant positive relationship between short term effect of air pollution and daily mortality from cardiovascular diseases effect (Chen et al., 2010) similar to our model for unadjusted weather.

5.3.3 Respiratory mortality

The percentage of respiratory mortality was increase, the total number was 18,131 deaths in six year of the study. We used ICD codes J00-J99 for respiratory mortality. Many studies show respiratory relationship with air pollution concentration but the result of study was not association when we adjusted model, It might be the subject who die because of air pollution were very rare in Bangkok. Our study had the result similar to the study in Metro Vancouver which showed predictor of population mortality and air pollution did not reliability (Goran et al., 2011). Much evidence indicates that air pollution has effect on respiratory morbidity and mortality. Respiratory system is the first organ system that encounters inhaled air pollution as PM_{10} association with mortality in respiratory especially for the people who were age over 35 year old (Colais et al., 2009), as we found respiratory mortality in elderly who age at least 75 years was increasing 2.94 percent chance of death when PM_{10} growing up 10 $\mu g/m^3$. The effect of particulate increase in respiratory mortality at 0-3 lags and significant stronger than non external mortality (Faustini et al., 2011). The infant get the effect from PM_{10} and O_3, these group were respiratory deaths cause of exposed to gaseous and higher in low socioeconomic status (Carbajal-Arroyo et al., 2010).

respiratory mortality were generally positive association with air pollution concentration (Perez et al., 2009) that we found out in unadjusted model for the all 6 year data.

5.4 PM_{10} Concentrations and mortality in Bangkok

PM_{10} associated with non external mortality in whole dataset and the two period do not statistic different, that mean PM_{10} are the strong effect on non-external mortality. The relevant to cardiovascular mortality was shown in first period but seem to be similar when we compared two period of study. The positive associated between air pollution and respiratory mortality is shown in second period a little different to circulatory mortality. PM_{10} has a stronger impact associated with non external mortality for single pollutant model after adjusted for the weather. The elderly is higher risky to exposed and get the averse health effect than other age group (Ma et al., 2011). The death case who die outside the hospital with non external mortality significantly positive association in second period. Particulate matter was the major effect, the components are sulfate, nitrates, ammonia, sodium chloride, carbon, mineral dust and water which the mixture of solid and liquid particles of organic and inorganic substances suspended in the air. After PM is inhaled, it could reach the bronchioles then interfere with gas exchange inside the alveoli. The short term effect of exposure to PM can cause of death in Bangkok. The evidence showed increasing PM_{10} was relevant to mortality in Bangkok (Vichit-Vadakan et al., 2010), the similar results is shown in our study. The Western European cities such as Spain, there was contribution the risk increasing of mortality linked to PM where high levels of PM were linked to major PM sources traffic and construction dust (Ostro et al., 2011). PM is a worldwide public health problem. In Latin American cities, PM_{10} also had a short term effect on mortality, especially at Sao Paulo (O'Neill et al., 2008).

5.5 NO_2 Concentrations and mortality in Bangkok

NO_2 associated with non external mortality in the whole dataset and both in separate the periods, when we compared two periods NO_2 remained had the similar effect on non-external mortality. The relationship between NO_2 and circulatory mortality was statistical significant positive direction for single model with adjusted meteorological

variables as relative humidity moving average days 0 to 1, but was no statistic significant when we separated in 2 periods. We found positive association between NO_2 and respiratory mortality only in second period. The main source of NO_2 was nitrate aerosols, the major sources of anthropogenic emissions of NO_2 are combustion processes such as engines in vehicles like most the traffic problem in Bangkok that may cause of heart dysfunction (Weichenthal et al., 2010). The traffic released high NO_2 level will positive association with acute risk of circulatory and respiratory mortality (Bhaskaran et al., 2011). The Epidemiological Surveillance and Primary Prevention in Italy found the association between NO_2 and cause-specific mortality as non-external, cardiac and respiratory mortality (Chiusolo et al., 2011) that was similar our unadjusted model in the whole 6 years dataset.

5.6 SO_2 Concentrations and mortality in Bangkok

We found positive association between SO_2 and non-external mortality if there was no adjustment for weather, that result was similar the SO_2 effect on respiratory mortality. The concentration of SO_2 was very oscillating, although in the seasonality. In urban district of Beijing, China SO_2 was associated with daily cardiovascular and respiratory mortality (Zhang et al.,2011). In urban area of Lucknow, India, SO_2 effect on cardiovascular and respiratory disease, these may increase even of mortality (Barman et al., 2010).

5.7 O_3 Concentrations and mortality in Bangkok

The investigator was unable to obtain 8-hour O_3 average levels, so 24-hour average O_3 levels were used in this study. O_3 was positive association to non external mortality in single pollutant model unadjusted for meteorology information when analyzed combination of 6 years data. The cardiovascular mortality related to O_3 that resemblance non external mortality. Respiratory show the same result as another cause. O_3 was an air pollution that increasing but the effect on health had smaller than PM_{10} and CO. In Canada, O_3 was effect to mortality with large number of deaths but low in United States and Europe (Katsouyanni et al., 2009). In Pearl River Delta, Southern China showed ambient oxidants as O_3 effect on acute mortality (Tao et al., 2011). The health effect vary in air pollutant may has different impact such as

ischemic heart disease mortality associated to O_3 especially in low-walkability neighborhoods (Hankey et al., 2012), but the CCO design was great to control this kind of confounder factors.

5.8 CO Concentrations and mortality in Bangkok

CO was positively associated with non external mortality which show on single pollutant model adjusted for weather. Both periods were different because there had positive direction significant only second period for non external mortality with. The result show CO effected on circulatory mortality and two periods had similar direction when we compared the period of study. The association between CO and circulatory mortality and respiratory mortality was statistically significant positive direction in Chinese population at mega-city Guangzhou (Tao et al., 2011) close to our result that show CO very precise relate to mortality in Bangkok. Even though season and temperature are very strong to modified CO for the adverse health effect (Park et al., 2011) but we remain provided the significant positive association between CO and mortality in Bangkok. CO is the greenhouse effect to climate change, the evidence release contribute to premature circulatory and respiratory mortality (Dennekamp, 2010). Reducing CO level is important to public health profitableness.

5.9 Comparing with the results of the PAPA time series study in Bangkok

This study (CCO) show the estimates were less precise (the 95% CIs were wider) than time series study from PAPA project both unadjusted and adjusted weather in first period (1999-2001) but CCO show more precise than time series for unadjusted in all 6 years data set. The meaning of this observation is not entirely clear, because the PAPA study included 5 years, whereas the present study, period 1, included only 3 years. Nevertheless, the bias from time stratify case-crossover designs is small (Lumley and levy, 1999). The time series design results are similar to unadjusted CCO model. That is the technique of time series method has complexity to analysis and more potential confounder variables in design (Son and Cho, 2008). The best strength of CCO study is about selection controls period as our study use time stratified that air pollution and time dependent variables (temperature, humidity or

seasonal) are controlled in the stratum, the especially individuals' personal covariate variables are controlled like a match case control (Bateson and Schwartz, 2001). Choosing between the two methods may difficult because both methods provide the reasonable estimates air pollutants which are effect on health adverse (Fung et al., 2003). Also, despite extensive previous research on short-term effects of air pollution on mortality, modeling data is very complex, and optimal modeling methods are not yet completely understood.

Finally, verbal autopsy (VA) is the method to analyze cause of death which can increase reliability of death certificate data(Porapakkham et al., 2010) and otherwise improve the quality of mortality data (Lozano et al., 2011). VA was not used in Bangkok to verify the underlying cause of death. We did not use VA in our study because research design was served to comparing two periods; using VA may not appropriate for the last decade memory because subjects could not remember and answer could be bias. Recall bias might be occurring when asked the respondent in the past ten year. Bangkok city have people move in and move out all the time it very hard to find the subject closer, family or their friend to interviewed. Thus, VA was not feasible, and in the researcher's opinion was not appropriate, in the present study. Further research should find out how to use VA in Bangkok to check and verify the routine public health statistics (Franca et al., 2011).

CHAPTER VI

CONCLUSIONS

6.1 Conclusions

6.1.1 We found positive association between gaseous pollutants (CO, NO_2, PM_{10}) and mortality (non-external, cardiovascular, respiratory) in Bangkok by using the time-stratified case-crossover method. Regarding non-external mortality, PM_{10} and NO_2 showed similar results between the two study periods but the CO effect differed between periods. Ambient air pollution was fluctuation due to many sources were not stable because there could change all the time. There are seriously increases risks of mortality when exposed to high level of air pollutants. Air pollution is the public health problem issue with transient averse health effect, so surveillance system to detect the epidemic is the most important.

6.1.2 The motivated of using natural gas in Bangkok, such as Natural Gas for Vehicles (NGV) Liquefied Petroleum Gas (LPG) or Compressed Natural Gas (CNG) may cause of reduce air pollution level, for each air pollutants was aimed at promoting a process to change from high level to lower level that the right way to practice. Future research should also evaluate health effects of airborne volatile organic compounds (VOC).

6.1.3 The both methods, time series and case-crossover provide generally similar evidence. We cannot say at present which method is better. CCO design save more cost than time-series design, because time-series require more information than case-crossover for appropriate controlling of potential confounders in analysis.

6.2 Potential Biases and limitations

6.2.1 Monitoring stations may provide some error air pollution level because new constructions were built around the stations that may cause of change or exhibit direction of air flowing in some season.

6.2.2 Many deaths were coded as non-specific and/or "senility/old age." Not specific, introduces uncertainty, and could introduce bias, into results for respiratory and cardiovascular mortality.

6.2.3 Death certificate were no clear in the underlying diagnose cause of death. The first period had more unclassified cause of death than second period, so the results in second period showed more precise than first period.

6.3 Recommendations

6.3.1 These CCO study results strongly suggest that reducing ambient air pollution levels would lead to public health benefit.

6.3.2 Air pollution is a major environmental hazard to public health. If reducing air pollution concentration in Bangkok or the province where more problems with air pollution should take action to reducing air pollution level together both of the local and national level. Government can reduce the burden of disease from cardiovascular, respiratory morbidity and mortality.

6.3.3 The cardiovascular and respiratory patients are high risk of acute adverse health effect when expose to air pollution. However, government and non-government who concern about hazard of air pollution should provide the knowledge to high-risk-group.

6.3.4 Monitoring of Air pollution and health effect should be continuously conducted by epidemiology surveillance systems, using both Time series and case-crossover model will increasing the validity of estimation health impact.

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APPENDIX

Appendix A: Form data collecting

Air Pollution data collecting form

Day/month/year	PM_{10}	NO_2	O_3	SO_2	CO
Total					
Missing					

Meteorological data collecting form

Day/month/year	Temperature	Humidity	Air pressure
Total			
Missing			

Death certificate data collecting form

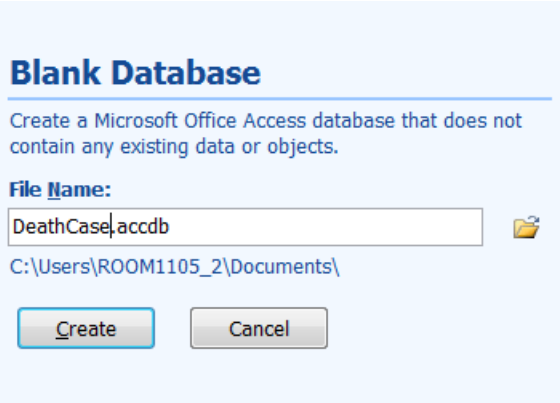
No.	ddate	sex	age	ncause	lccaattmm	codepro	hosname	place	addeath
Total									
Missing									

Variable Code

Field	Type	Variable name	Code
1.Date of death	Numeric	Ddate	
2. Month of death	Numeric	Dmon	
3. Year of death	Numeric	Dyear	
4. Sex	String	Sex	1 = Male 2 = Female
5. Age	Numeric	Age	
6. Cause of death	String	Ncause	ICD 10
7. Address in ID	String	Lccaattmm	
8. Province	String	Codepro	10 = Bangkok
9. Hospital name	String	Hosname	
10. Place of death	String	Place	
11. Address when living	String	Addeath	

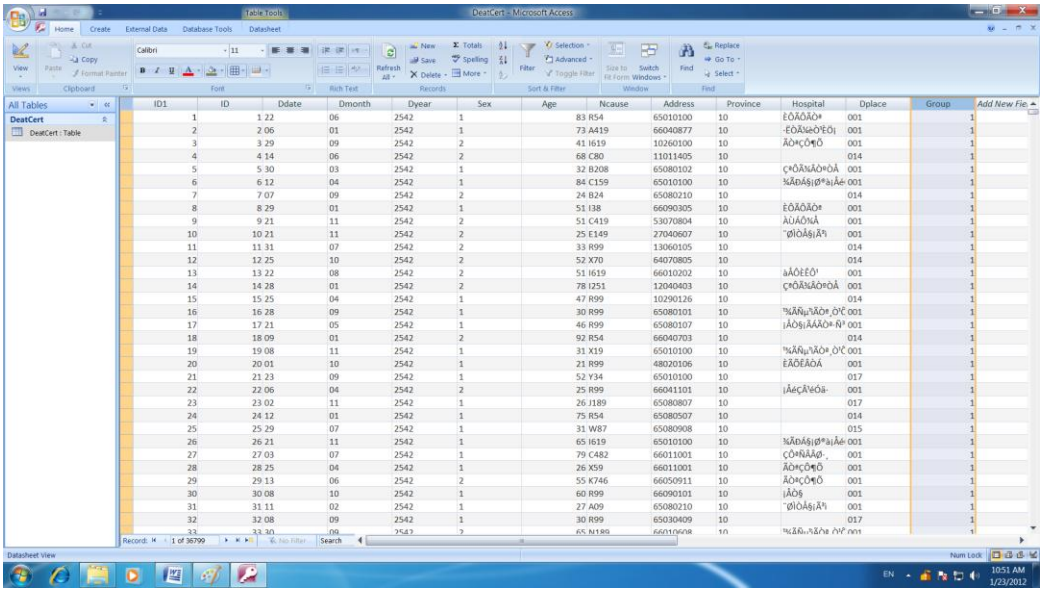
Appendix B: How to manage data

1. Creative the Death data for control group (reference group) by using Access 2007



Create new database file. File name was DeathCase.accdb

Make variable group = 1

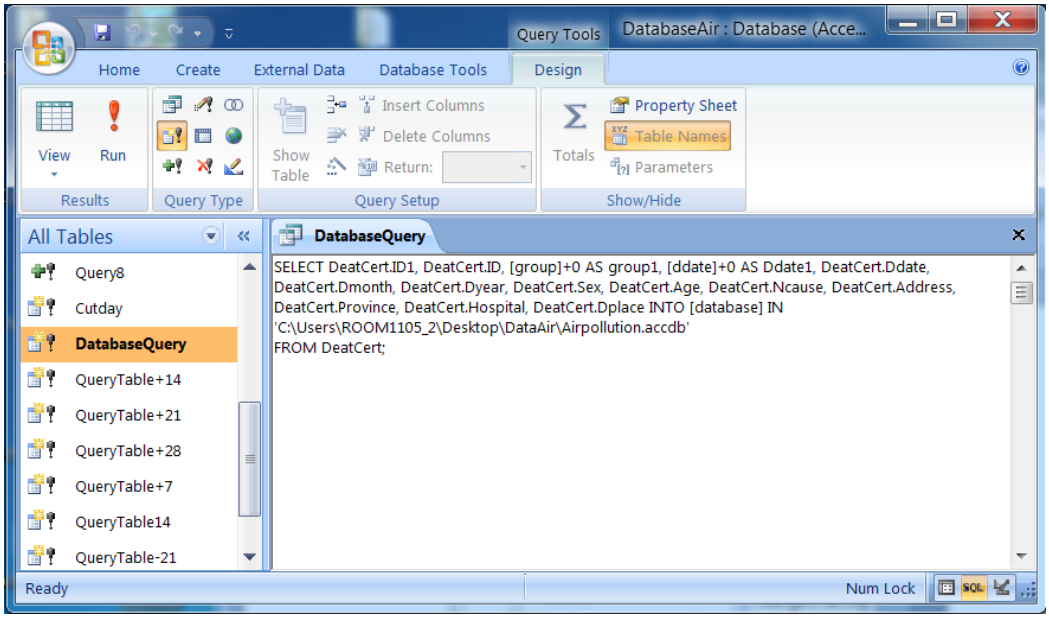


Open New file “DatabaseAir”. Imports Deathcase table then set date month year variable by using

Command DatabaseQuery

SELECT DeatCert.ID1, DeatCert.ID, [group]+0 AS group1, [ddate]+0 AS Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex, DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province, DeatCert.Hospital, DeatCert.Dplace INTO [database] IN 'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'

FROM DeatCert;



Made Query1-Query8 to split table into each control day.

Command for Query1

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable+141].ID1, [QueryTable+141].ID, [QueryTable+141].group1, [QueryTable+141].Ddate1, [QueryTable+141].Ddate, [QueryTable+141].Dmonth, [QueryTable+141].Dyear, [QueryTable+141].Sex, [QueryTable+141].Age, [QueryTable+141].Ncause, [QueryTable+141].Address, [QueryTable+141].Province, [QueryTable+141].Hospital, [QueryTable+141].Dplace
FROM [QueryTable+141];

Command for Query2

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable+211].ID1 AS Expr1, [QueryTable+211].ID AS Expr2, [QueryTable+211].group1 AS Expr3, [QueryTable+211].Ddate1 AS Expr4, [QueryTable+211].Ddate AS Expr5, [QueryTable+211].Dmonth AS Expr6, [QueryTable+211].Dyear AS Expr7, [QueryTable+211].Sex AS Expr8, [QueryTable+211].Age AS Expr9, [QueryTable+211].Ncause AS Expr10, [QueryTable+211].Address AS Expr11, [QueryTable+211].Province AS Expr12, [QueryTable+211].Hospital AS Expr13, [QueryTable+211].Dplace AS Expr14
FROM [QueryTable+211];

Command for Query3

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable+281].ID1 AS Expr1, [QueryTable+281].ID AS Expr2, [QueryTable+281].group1 AS Expr3, [QueryTable+281].Ddate1 AS Expr4, [QueryTable+281].Ddate AS Expr5, [QueryTable+281].Dmonth AS Expr6, [QueryTable+281].Dyear AS Expr7, [QueryTable+281].Sex AS Expr8, [QueryTable+281].Age AS Expr9, [QueryTable+281].Ncause AS Expr10, [QueryTable+281].Address AS Expr11, [QueryTable+281].Province AS Expr12, [QueryTable+281].Hospital AS Expr13, [QueryTable+281].Dplace AS Expr14
FROM [QueryTable+281];

Command for Query4

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable+71].ID1 AS Expr1, [QueryTable+71].ID AS Expr2, [QueryTable+71].group1 AS Expr3, [QueryTable+71].Ddate1 AS Expr4, [QueryTable+71].Ddate AS Expr5, [QueryTable+71].Dmonth AS Expr6, [QueryTable+71].Dyear AS Expr7, [QueryTable+71].Sex AS Expr8, [QueryTable+71].Age AS Expr9, [QueryTable+71].Ncause AS Expr10, [QueryTable+71].Address AS Expr11, [QueryTable+71].Province AS Expr12, [QueryTable+71].Hospital AS Expr13, [QueryTable+71].Dplace AS Expr14
FROM [QueryTable+71];

Command for Query5

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable-14].ID1 AS Expr1, [QueryTable-14].ID AS Expr2, [QueryTable-14].group1 AS Expr3, [QueryTable-14].Ddate1 AS Expr4, [QueryTable-14].Ddate AS Expr5, [QueryTable-14].Dmonth AS Expr6, [QueryTable-14].Dyear AS Expr7, [QueryTable-14].Sex AS Expr8, [QueryTable-14].Age AS Expr9, [QueryTable-14].Ncause AS Expr10, [QueryTable-14].Address AS Expr11, [QueryTable-14].Province AS Expr12, [QueryTable-14].Hospital AS Expr13, [QueryTable-14].Dplace AS Expr14
FROM [QueryTable-14];

Command for Query6

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable-211].ID1 AS Expr1, [QueryTable-211].ID AS Expr2, [QueryTable-211].group1 AS Expr3, [QueryTable-211].Ddate1 AS Expr4, [QueryTable-211].Ddate AS Expr5, [QueryTable-211].Dmonth AS Expr6, [QueryTable-211].Dyear AS Expr7, [QueryTable-211].Sex AS Expr8,

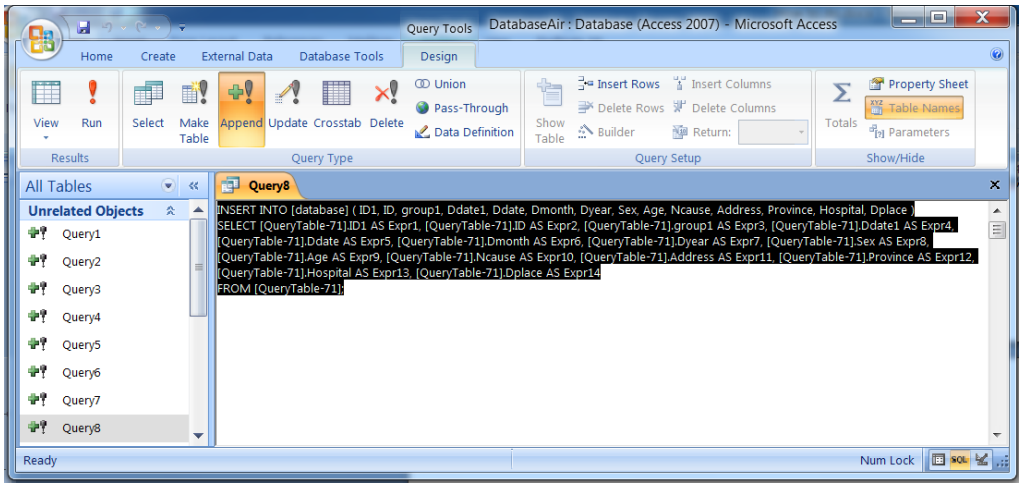
[QueryTable-211].Age AS Expr9, [QueryTable-211].Ncause AS Expr10, [QueryTable-211].Address AS Expr11, [QueryTable-211].Province AS Expr12, [QueryTable-211].Hospital AS Expr13, [QueryTable-211].Dplace AS Expr14
FROM [QueryTable-211];

Command for Query7

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable-281].ID1 AS Expr1, [QueryTable-281].ID AS Expr2, [QueryTable-281].group1 AS Expr3, [QueryTable-281].Ddate1 AS Expr4, [QueryTable-281].Ddate AS Expr5, [QueryTable-281].Dmonth AS Expr6, [QueryTable-281].Dyear AS Expr7, [QueryTable-281].Sex AS Expr8, [QueryTable-281].Age AS Expr9, [QueryTable-281].Ncause AS Expr10, [QueryTable-281].Address AS Expr11, [QueryTable-281].Province AS Expr12, [QueryTable-281].Hospital AS Expr13, [QueryTable-281].Dplace AS Expr14
FROM [QueryTable-281];

Command for Query8

INSERT INTO [database] (ID1, ID, group1, Ddate1, Ddate, Dmonth, Dyear, Sex, Age, Ncause, Address, Province, Hospital, Dplace)
SELECT [QueryTable-71].ID1 AS Expr1, [QueryTable-71].ID AS Expr2, [QueryTable-71].group1 AS Expr3, [QueryTable-71].Ddate1 AS Expr4, [QueryTable-71].Ddate AS Expr5, [QueryTable-71].Dmonth AS Expr6, [QueryTable-71].Dyear AS Expr7, [QueryTable-71].Sex AS Expr8, [QueryTable-71].Age AS Expr9, [QueryTable-71].Ncause AS Expr10, [QueryTable-71].Address AS Expr11, [QueryTable-71].Province AS Expr12, [QueryTable-71].Hospital AS Expr13, [QueryTable-71].Dplace AS Expr14
FROM [QueryTable-71];



Import all table for Merge Control group into DatabaseAir file the use command to merge case to an active file.

Command QueryTable+28

```
SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]+28 AS
Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex,
DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province,
DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable+28] IN
'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;
```

Command QueryTable+21

```
SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]+21 AS
Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex,
DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province,
DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable+21] IN
'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;
```

Command QueryTable+14

```
SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]+14 AS
Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex,
DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province,
DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable+14] IN
'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;
```

Command QueryTable+7

```
SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]+7 AS
Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex,
DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province,
DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable+7] IN
'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;
```

Command QueryTable-7

```
SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]-7 AS Ddate1,
DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex, DeatCert.Age,
DeatCert.Ncause, DeatCert.Address, DeatCert.Province, DeatCert.Hospital,
DeatCert.Dplace INTO [QueryTable-7] IN
'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;
```

Command QueryTable-14

SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]-14 AS Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex, DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province, DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable-14] IN 'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;

Command QueryTable-21

SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]-21 AS Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex, DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province, DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable-21] IN 'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;

Command QueryTable-28

SELECT DeatCert.ID1, DeatCert.ID, [group]+1 AS group1, [ddate]-28 AS Ddate1, DeatCert.Ddate, DeatCert.Dmonth, DeatCert.Dyear, DeatCert.Sex, DeatCert.Age, DeatCert.Ncause, DeatCert.Address, DeatCert.Province, DeatCert.Hospital, DeatCert.Dplace INTO [QueryTable-28] IN 'C:\Users\ROOM1105_2\Desktop\DataAir\Airpollution.accdb'
FROM DeatCert;

Checking and cut the fault date using command Cutday

SELECT database.ID1 AS Expr1, database.ID AS Expr2, database.group1 AS Expr3, database.Ddate1 AS Expr4, database.Ddate AS Expr5, database.Dmonth AS Expr6, database.Dyear AS Expr7, database.Sex AS Expr8, database.Age AS Expr9, database.Ncause AS Expr10, database.Address AS Expr11, database.Province AS Expr12, database.Hospital AS Expr13, database.Dplace AS Expr14 INTO Ttol1
FROM [database]
WHERE ((([database].[Ddate1])<32 And ([database].[Ddate1])>0));

2. **Creative the Air pollution data in Excel file**. Instance for unit conversion, 75% of daily air pollution concentration loss that parameter was exclud. and 25% missing data in the period of study that station was deleted for that parameter loss. Unit conversion

CO (ppm) x 1150 = CO($\mu g/m^3$)
NO_2 (ppb) x 1.88 = NO_2($\mu g/m^3$)
O_3 (ppb) x 1.96 = O_3($\mu g/m^3$)

SO2(ppb) x 2.62 = SO2(μg/m^3)

3. **Creative the Meteorological information in Excel file**. For example fill the average with 25% missing data was excluded.
4. **Merge variable air pollution and meteorological into file** which preparing for the whole file by using SPSS
5. **Creative the preparing file with increasing variables Lag1 Lag2 Lag01 Lag02**

Command to created Lag1 Lag2 Lag01 Lag02

```
CREATE

 /HumidiLag1=LAG(Humidity 1)/TemperLag1=LAG(Temperature
1)/AirPreLag1=LAG(AirPressure

   1)/COLag1=LAG(CO 1)/O3Lag1=LAG(O3 1)/NO2Lag1=LAG(NO2
1)/SO2Lag1=LAG(SO2 1)/PM10Lag1=LAG(PM10 1).


CREATE

 /HumidiLag2=LAG(Humidity 2)/TemperLag2=LAG(Temperature
2)/AirPreLag2=LAG(AirPressure

   2)/COLag2=LAG(CO 2)/O3Lag2=LAG(O3 2)/NO2Lag2=LAG(NO2
2)/SO2Lag2=LAG(SO2 2)/PM10Lag2=LAG(PM10 2).


COMPUTE HumidiMv01=mean(Humidity, HumidiLag1).

EXECUTE.


COMPUTE TemperMv01=mean(Temperature, TemperLag1).

EXECUTE.


COMPUTE AirPreMv01=mean(AirPressure, AirPreLag1).

EXECUTE.


COMPUTE COMv01=mean(CO, COLag1).
```

```
EXECUTE.

COMPUTE O3Mv01=mean(O3, O3Lag1).
EXECUTE.

COMPUTE NO2Mv01=mean(NO2, NO2Lag1).
EXECUTE.

COMPUTE SO2Mv01=mean(SO2, SO2Lag1).
EXECUTE.

COMPUTE PM10Mv01=mean(PM10, PM10Lag1).
EXECUTE.

COMPUTE HumidiMv02=mean(Humidity, HumidiLag1, HumidiLag2).
EXECUTE.

COMPUTE TemperMv02=mean(Temperature, TemperLag1, TemperLag2).
EXECUTE.

COMPUTE AirPreMv02=mean(AirPressure, AirPreLag1, AirPreLag2).
EXECUTE.

COMPUTE COMv02=mean(CO, COLag1, COLag2).
EXECUTE.
```

COMPUTE O3Mv02=mean(O3, O3Lag1, O3Lag2).

EXECUTE.

COMPUTE NO2Mv02=mean(NO2, NO2Lag1, NO2Lag2).

EXECUTE.

COMPUTE SO2Mv02=mean(SO2, SO2Lag1, SO2Lag2).

EXECUTE.

COMPUTE PM10Mv02=mean(PM10, PM10Lag1, PM10Lag2).

EXECUTE.

6. **Merge Variable Air pollution, meteorology and Death case together**.

Command to Merge Variable in SPSS

```
DATASET ACTIVATE DataSet2.
MATCH FILES /FILE=*
 /TABLE='DataSet1'
 /RENAME (Ddate = d0)
 /BY sequence
 /DROP= d0.
EXECUTE.
```

7. **Analysis Conditional logistic regression by STATA**

Command to analysis

Set memory 1g

Set maxvar 5000

Recode group (1=1) (2=0), gen(ngroup)

Clogit ngroup CO O3 NO2 SO2 PM10 Humidity Temperature AirPressure, group(ID)

Clogit ngroup CO O3 NO2 SO2 PM10 Humidity Temperature AirPressure, group(ID) or

8. Analysis Cox proportional hazards model by using SPSS

Command to analysis

COXREG time

/STATUS=group(1)

/STRATA=ID

/METHOD=ENTER CO O3 NO2 SO2 PM10 Humidity Temperature AirPressure

/PRINT=CI(95) CORR

/CRITERIA=PIN(.05) POUT(.10) ITERATE(20).

9. Analysis Poisson loglinear model from GLM by using SPSS

Command to analysis

* Generalized Linear Models.

GENLIN NumberDeath WITH CO O3 NO2 SO2 PM10 Humidity Temperature AirPressure

/MODEL CO O3 NO2 SO2 PM10 Humidity Temperature AirPressure INTERCEPT=YES

DISTRIBUTION=POISSON LINK=LOG

/CRITERIA SCALE=1 COVB=MODEL PCONVERGE=1E-006(ABSOLUTE) SINGULAR=1E-012 ANALYSISTYPE=3(WALD)

CILEVEL=95 CITYPE=WALD LIKELIHOOD=FULL

/MISSING CLASSMISSING=EXCLUDE

/PRINT CPS DESCRIPTIVES MODELINFO FIT SUMMARY SOLUTION.

Appendix C: How to select Time-dependent Variable for the models

For Example

Outcome is total Non-accidental mortality (Non-external mortality)
Checking meteorology for all non-external deaths
Adjust for same-day temperature and humidity lag 1.

Separate Periods

Case Processing Summary[b]

		N	Percent
Cases available in analysis	Event[a]	102522	22.7%
	Censored	346548	76.8%
	Total	449070	99.6%
Cases dropped	Cases with missing values	0	.0%
	Cases with negative time	0	.0%
	Censored cases before the earliest event in a stratum	2024	.4%
	Total	2024	.4%
Total		451094	100.0%

a. Dependent Variable: time

b. period = 1

Case Processing Summary[b]

		N	Percent
Cases available in analysis	Event[a]	104587	22.7%
	Censored	355312	77.3%
	Total	459899	100.0%
Cases dropped	Cases with missing values	0	.0%
	Cases with negative time	0	.0%
	Censored cases before the earliest event in a stratum	0	.0%
	Total	0	.0%
Total		459899	100.0%

a. Dependent Variable: time

b. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.017	.003	43.543	1	.000	1.017

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.013	.003	22.534	1	.000	1.013

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
templag1	.010	.003	15.594	1	.000	1.010

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
templag1	.007	.003	7.108	1	.008	1.007

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
tempmv01	.015	.003	32.019	1	.000	1.015

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
tempmv01	.012	.003	15.608	1	.000	1.012

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.020	.004	29.936	1	.000	1.021
templag1	-.005	.004	1.920	1	.166	.995

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.018	.004	17.690	1	.000	1.018
templag1	-.006	.004	2.251	1	.134	.994

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humidity	-.003	.001	27.888	1	.000	.997

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humidity	-.003	.001	26.723	1	.000	.997

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humdlag1	-.003	.001	36.410	1	.000	.997

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humdlag1	-.003	.001	25.212	1	.000	.997

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humdmv01	-.003	.001	37.473	1	.000	.997

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humdmv01	-.003	.001	29.642	1	.000	.997

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humidity	-.001	.001	2.022	1	.155	.999
humdlag1	-.002	.001	10.544	1	.001	.998

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
humidity	-.002	.001	4.463	1	.035	.998
humdlag1	-.001	.001	2.953	1	.086	.999

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.015	.003	35.359	1	.000	1.015
humdlag1	-.003	.001	28.176	1	.000	.997

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.012	.003	20.562	1	.000	1.012
humdlag1	-.003	.001	23.238	1	.000	.997

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.003	30.338	1	.000	1.014
humdmv01	-.003	.001	24.194	1	.000	.997

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.011	.003	16.315	1	.000	1.011
humdmv01	-.003	.001	23.416	1	.000	.997

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
pressure	-.004	.002	6.329	1	.012	.996

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
pressure	-.001	.002	.267	1	.605	.999

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
preslag1	-.002	.002	.941	1	.332	.998

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
preslag1	.001	.002	.500	1	.480	1.001

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
presmv01	-.003	.002	3.805	1	.051	.997

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
presmv01	.000	.002	.010	1	.920	1.000

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
pressure	-.005	.002	5.836	1	.016	.995
preslag1	.001	.002	.429	1	.513	1.001

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
pressure	-.008	.004	4.455	1	.035	.992
preslag1	.008	.004	4.687	1	.030	1.008

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.015	.003	28.836	1	.000	1.015
humdlag1	-.003	.001	28.403	1	.000	.997
pressure	.000	.002	.250	1	.617	.999

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.013	.003	18.958	1	.000	1.013
humdlag1	-.002	.001	20.628	1	.000	.998
pressure	.002	.002	.501	1	.479	1.002

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.015	.003	33.450	1	.000	1.015
humdlag1	-.003	.001	27.598	1	.000	.997
preslag1	.000	.002	.055	1	.814	1.000

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.003	22.539	1	.000	1.014
humdlag1	-.002	.001	18.578	1	.000	.998
preslag1	.003	.002	2.127	1	.145	1.003

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.015	.003	30.350	1	.000	1.015
humdlag1	-.003	.001	28.071	1	.000	.997
presmv01	.000	.002	.021	1	.884	1.000

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.003	20.955	1	.000	1.014
humdlag1	-.002	.001	19.271	1	.000	.998
presmv01	.003	.002	1.309	1	.252	1.003

a. period = 2

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.015	.003	29.195	1	.000	1.015
humdlag1	-.003	.001	27.623	1	.000	.997
pressure	-.002	.002	.581	1	.446	.998
preslag1	.001	.002	.383	1	.536	1.001

a. period = 1

Variables in the Equation[a]

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.013	.003	18.824	1	.000	1.013
humdlag1	-.002	.001	18.803	1	.000	.998
pressure	-.003	.004	.677	1	.411	.997
preslag1	.006	.004	2.303	1	.129	1.006

a. period = 2

For periods 1 and 2 combined

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.015	.002	65.132	1	.000	1.015

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
templag1	.009	.002	22.210	1	.000	1.009

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
tempmv01	.014	.002	46.743	1	.000	1.014

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.019	.003	47.183	1	.000	1.019
templag1	-.006	.003	4.194	1	.041	.994

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
humidity	-.003	.000	54.608	1	.000	.997

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
humdlag1	-.003	.000	61.310	1	.000	.997

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
humdmv01	-.003	.000	66.977	1	.000	.997

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
humidity	-.001	.001	6.005	1	.014	.999
humdlag1	-.002	.001	12.709	1	.000	.998

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.002	55.638	1	.000	1.014
humdlag1	-.003	.000	51.783	1	.000	.997

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.013	.002	46.138	1	.000	1.013
humdmv01	-.003	.000	47.919	1	.000	.997

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
pressure	-.003	.001	5.296	1	.021	.997

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
preslag1	.000	.001	.104	1	.747	1.000

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
presmv01	-.002	.001	2.036	1	.154	.998

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
pressure	-.005	.002	8.353	1	.004	.995
preslag1	.003	.002	3.095	1	.079	1.003

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.002	46.733	1	.000	1.014
humdlag1	-.003	.000	50.916	1	.000	.997
pressure	.000	.001	.000	1	.997	1.000

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.002	54.858	1	.000	1.015
humdlag1	-.003	.000	47.876	1	.000	.997
preslag1	.001	.001	1.126	1	.289	1.001

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.002	50.353	1	.000	1.014
humdlag1	-.003	.000	49.061	1	.000	.997
presmv01	.001	.001	.352	1	.553	1.001

Variables in the Equation

	B	SE	Wald	df	Sig.	Exp(B)
temperat	.014	.002	47.816	1	.000	1.014
humdlag1	-.003	.000	48.161	1	.000	.997
pressure	-.002	.002	.824	1	.364	.998
preslag1	.002	.002	1.940	1	.164	1.002

The last output was show Temperature lag0 and Humidity lag1 is still significant and Wald very large than Air pressure, so we selected Temperature lag0 and Humidity lag1 to put into the model of Non-external mortality model both period 1 and period 2.

Showing how we chose the method of adjustment for meteorology for each kind of death. For non-external deaths, adjust for same-day temperature and humidity lag 1. For cardiovascular and respiratory deaths, adjust for moving average humidity only.

Appendix D: Output original unit and no delete station

Table 59: Odds ratios between all natural mortality and CO (ppm) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0463	1.0165	1.0771
2000	1.0011	0.9706	1.0325
2001	1.0079	0.9724	1.0447
2006	1.0912	1.0252	1.1614
2007	1.0169	0.9598	1.0775
2008	1.0268	0.9682	1.0889

Table 60: Odds ratios between all natural mortality and O_3 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0012	0.9986	1.0038
2000	1.0008	0.9986	1.0032
2001	1.0027	1.0001	1.0053
2006	1.0017	0.9994	1.0041
2007	0.9991	0.9969	1.0012
2008	1.0025	1.0001	1.0048

Table 61: Odds ratios between all natural mortality and NO2(ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0004	0.9984	1.0025
2000	1.0008	0.9992	1.0025
2001	1.0006	0.9988	1.0024
2006	1.0015	0.9997	1.0034
2007	0.9997	0.9981	1.0014
2008	1.0021	1.0003	1.0038

Table 62: Odds ratios between all natural mortality and SO_2 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0043	0.9971	1.0118
2000	1.0064	1.0011	1.0117
2001	1.0053	0.9985	1.0121
2006	1.0030	0.9942	1.0119
2007	1.0031	0.9926	1.0137
2008	1.0147	1.0021	1.0275

Table 63: Odds ratios between all natural mortality and PM_{10} ($\mu g/m^3$) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0009	1.0002	1.0016
2000	1.0005	0.9998	1.0012
2001	1.0014	1.0004	1.0024
2006	1.0015	1.0005	1.0025
2007	1.0003	0.9996	1.0011
2008	1.0009	1.0001	1.0018

Table 64: Odds ratios between cardiovascular mortality and CO (ppm) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.1320	1.0365	1.2364
2000	1.1043	1.0171	1.1989
2001	1.1680	1.0610	1.2857
2006	1.0959	0.9313	1.2897
2007	1.0020	0.8584	1.1697
2008	1.0653	0.9100	1.2472

Table 65: Odds ratios between cardiovascular mortality and O_3 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0047	0.9967	1.0128
2000	0.9987	0.9925	1.0050
2001	1.0049	0.9983	1.0115
2006	1.0078	1.0016	1.0140
2007	0.9999	0.9943	1.0056
2008	1.0053	0.9990	1.0117

Table 66: Odds ratios between cardiovascular mortality and NO_2 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	0.9992	0.9928	1.0056
2000	1.0040	0.9996	1.0084
2001	1.0058	1.0011	1.0106
2006	1.0033	0.9984	1.0082
2007	0.9996	0.9951	1.0041
2008	1.0052	1.0005	1.0098

Table 67: Odds ratios between cardiovascular mortality and SO_2 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0278	1.0052	1.0509
2000	1.0020	0.9878	1.0163
2001	1.0172	1.0003	1.0342
2006	0.9936	0.9709	1.0169
2007	1.0082	0.9805	1.0366
2008	1.0361	1.0019	1.0714

Table 68: Odds ratios between cardiovascular mortality and PM_{10} ($\mu g/m^3$) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0032	1.0009	1.0054
2000	1.0017	0.9998	1.0035
2001	1.0035	1.0010	1.0060
2006	1.0017	0.9992	1.0043
2007	1.0002	0.9982	1.0021
2008	1.0013	0.9991	1.0036

Table 69: Odds ratios between respiratory mortality and CO (ppm) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	0.9994	0.8971	1.1133
2000	0.9934	0.8900	1.1087
2001	0.9692	0.8507	1.1042
2006	1.1459	0.9380	1.3999
2007	1.1899	0.9919	1.4275
2008	1.0620	0.8792	1.2829

Table 70: Odds ratios between respiratory mortality and O_3 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	1.0047	0.9950	1.0145
2000	1.0050	0.9967	1.0133
2001	1.0079	0.9992	1.0168
2006	1.0003	0.9927	1.0081
2007	1.0024	0.9956	1.0093
2008	1.0077	1.0000	1.0155

Table 71: Odds ratios between respiratory mortality and NO_2 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	0.9945	0.9869	1.0022
2000	1.0015	0.9956	1.0073
2001	1.0017	0.9954	1.0080
2006	1.0019	0.9959	1.0080
2007	1.0064	1.0011	1.0118
2008	1.0064	1.0009	1.0120

Table 72: Odds ratios between respiratory mortality and SO_2 (ppb) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	0.9921	0.9653	1.0197
2000	1.0237	1.0052	1.0427
2001	0.9955	0.9739	1.0176
2006	1.0000	0.9717	1.0293
2007	1.0466	1.0119	1.0825
2008	0.9985	0.9593	1.0394

Table 73: Odds ratios between respiratory mortality and PM_{10} ($\mu g/m^3$) concentration stratify by year

Years	OR	95% CI	
		Lower	Upper
1999	0.9986	0.9959	1.0013
2000	1.0020	0.9996	1.0045
2001	1.0004	0.9971	1.0038
2006	1.0022	0.9990	1.0054
2007	1.0024	1.0002	1.0046
2008	1.0022	0.9995	1.0049

Table 74: Adjusted Odds ratios between non-accidental mortality and air pollutant levels in 1999

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0552	1.0180	1.0938
O_3 (ppb)	1.0001	0.9965	1.0036
NO_2 (ppb)	0.9954	0.9918	0.9990
SO_2 (ppb)	1.0029	0.9952	1.0107
PM_{10} ($\mu g/m^3$)	1.0011	0.9998	1.0024
Humidity	0.9979	0.9959	1.0000
Temperature	0.9977	0.9882	1.0073
Air pressure	0.9987	0.9911	1.0064

Table 75: Adjusted Odds ratios between non-accidental mortality and air pollutant levels in 2000

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.9430	0.8856	1.0041
O_3 (ppb)	0.9970	0.9937	1.0003
NO_2 (ppb)	1.0043	0.9997	1.0088
SO_2 (ppb)	1.0046	0.9990	1.0102
PM_{10} (µg/m^3)	1.0005	0.9988	1.0022
Humidity	0.9978	0.9956	1.0001
Temperature	1.0175	1.0060	1.0291
Air pressure	0.9952	0.9875	1.0030

Table 76: Adjusted Odds ratios between non-accidental mortality and air pollutant levels in 2001

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.9624	0.9019	1.0269
O_3 (ppb)	0.9992	0.9958	1.0027
NO_2 (ppb)	1.0032	0.9992	1.0072
SO_2 (ppb)	0.9988	0.9907	1.0069
PM_{10} (µg/m^3)	1.0010	0.9995	1.0026
Humidity	0.9994	0.9969	1.0020
Temperature	1.0260	1.0139	1.0382
Air pressure	0.9977	0.9900	1.0055

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Table 77: Adjusted Odds ratios between non-accidental mortality and air pollutant levels in 2006

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.1406	1.0139	1.2831
O_3 (ppb)	0.9993	0.9962	1.0024
NO_2 (ppb)	0.9962	0.9917	1.0007
SO_2 (ppb)	0.9982	0.9881	1.0083
PM_{10} (µg/m^3)	1.0020	1.0001	1.0039
Humidity	0.9984	0.9959	1.0008
Temperature	1.0097	0.9986	1.0210
Air pressure	1.0000	0.9998	1.0001

Table 78: Adjusted Odds ratios between non-accidental mortality and air pollutant levels in 2007

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0706	0.9640	1.1890
O_3 (ppb)	0.9958	0.9929	0.9987
NO_2 (ppb)	0.9973	0.9933	1.0014
SO_2 (ppb)	0.9986	0.9856	1.0117
PM_{10} (µg/m^3)	1.0020	1.0002	1.0038
Humidity	0.9983	0.9960	1.0005
Temperature	1.0232	1.0107	1.0360
Air pressure	1.0051	0.9974	1.0129

Table 79: Adjusted Odds ratios between non-accidental mortality and air pollutant levels in 2008

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.9231	0.8220	1.0366
O_3 (ppb)	0.9990	0.9958	1.0023
NO_2 (ppb)	1.0056	1.0012	1.0101
SO_2 (ppb)	1.0027	0.9864	1.0193
PM_{10} (µg/m^3)	0.9997	0.9978	1.0015
Humidity	0.9989	0.9966	1.0012
Temperature	1.0199	1.0055	1.0344
Air pressure	0.9994	0.9900	1.0089

Table 80: Adjusted Odds ratios between cardiovascular mortality and air pollutant levels in 1999

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.1309	1.0128	1.2627
O_3 (ppb)	1.0090	0.9983	1.0197
NO_2 (ppb)	0.9778	0.9675	0.9882
SO_2 (ppb)	1.0301	1.0060	1.0548
PM_{10} (µg/m^3)	1.0059	1.0021	1.0098
Humidity	1.0012	0.9952	1.0072
Temperature	0.9859	0.9584	1.0141
Air pressure	1.0043	0.9811	1.0279

Table 81: Adjusted Odds ratios between cardiovascular mortality and air pollutant levels in 2000

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0881	0.9175	1.2904
O_3 (ppb)	0.9940	0.9851	1.0029
NO_2 (ppb)	1.0040	0.9917	1.0164
SO_2 (ppb)	0.9989	0.9840	1.0141
PM_{10} (µg/m^3)	0.9999	0.9954	1.0045
Humidity	0.9990	0.9930	1.0050
Temperature	1.0008	0.9706	1.0319
Air pressure	0.9826	0.9619	1.0037

Table 82: Adjusted Odds ratios between cardiovascular mortality and air pollutant levels in 2001

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0842	0.9163	1.2829
O_3 (ppb)	1.0043	0.9958	1.0129
NO_2 (ppb)	0.9998	0.9907	1.0090
SO_2 (ppb)	1.0057	0.9861	1.0257
PM_{10} (µg/m^3)	1.0023	0.9982	1.0064
Humidity	1.0049	0.9981	1.0119
Temperature	1.0221	0.9908	1.0543
Air pressure	1.0129	0.9923	1.0339

Table 83: Adjusted Odds ratios between cardiovascular mortality and air pollutant levels in 2006

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0875	0.7949	1.4878
O_3 (ppb)	1.0081	0.9998	1.0163
NO_2 (ppb)	0.9999	0.9880	1.0119
SO_2 (ppb)	0.9897	0.9636	1.0165
PM_{10} ($\mu g/m^3$)	0.9996	0.9945	1.0047
Humidity	1.0005	0.9940	1.0071
Temperature	1.0013	0.9722	1.0312
Air pressure	1.0001	0.9996	1.0005

Table 84: Adjusted Odds ratios between cardiovascular mortality and air pollutant levels in 2007

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0152	0.7703	1.3380
O_3 (ppb)	1.0007	0.9930	1.0085
NO_2 (ppb)	0.9951	0.9847	1.0056
SO_2 (ppb)	1.0156	0.9812	1.0512
PM_{10} ($\mu g/m^3$)	1.0008	0.9961	1.0054
Humidity	1.0006	0.9948	1.0065
Temperature	0.9840	0.9525	1.0165
Air pressure	0.9969	0.9770	1.0173

Table 85: Adjusted Odds ratios between cardiovascular mortality and air pollutant levels in 2008

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.8807	0.6445	1.2034
O_3 (ppb)	1.0007	0.9922	1.0093
NO_2 (ppb)	1.0133	1.0013	1.0254
SO_2 (ppb)	1.0338	0.9892	1.0803
PM_{10} ($\mu g/m^3$)	0.9948	0.9900	0.9997
Humidity	0.9964	0.9903	1.0025
Temperature	1.0146	0.9769	1.0537
Air pressure	1.0147	0.9891	1.0410

Table 86: Adjusted Odds ratios between respiratory mortality and air pollutant levels in 1999

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.0905	0.9543	1.2461
O_3 (ppb)	1.0098	0.9967	1.0231
NO_2 (ppb)	0.9927	0.9796	1.0060
SO_2 (ppb)	0.9925	0.9642	1.0217
PM_{10} ($\mu g/m^3$)	0.9980	0.9933	1.0027
Humidity	0.9982	0.9909	1.0056
Temperature	1.0150	0.9779	1.0536
Air pressure	0.9948	0.9675	1.0229

Table 87: Adjusted Odds ratios between respiratory mortality and air pollutant levels in 2000

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.8464	0.6773	1.0576
O_3 (ppb)	1.0029	0.9910	1.0149
NO_2 (ppb)	0.9924	0.9763	1.0087
SO_2 (ppb)	1.0191	0.9996	1.0389
PM_{10} (µg/m^3)	1.0076	1.0017	1.0136
Humidity	1.0033	0.9954	1.0113
Temperature	1.0169	0.9771	1.0584
Air pressure	1.0071	0.9797	1.0353

Table 88: Adjusted Odds ratios between respiratory mortality and air pollutant levels in 2001

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.8928	0.7157	1.1138
O_3 (ppb)	1.0054	0.9940	1.0170
NO_2 (ppb)	1.0161	1.0042	1.0281
SO_2 (ppb)	0.9863	0.9618	1.0114
PM_{10} (µg/m^3)	0.9965	0.9912	1.0019
Humidity	1.0002	0.9911	1.0093
Temperature	1.0569	1.0151	1.1006
Air pressure	0.9935	0.9675	1.0202

Table 89 Adjusted Odds ratios between respiratory mortality and air pollutant levels in 2006

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	1.3719	0.9351	2.0127
O_3 (ppb)	0.9974	0.9872	1.0078
NO_2 (ppb)	0.9872	0.9725	1.0020
SO_2 (ppb)	1.0001	0.9677	1.0337
PM_{10} (µg/m^3)	1.0038	0.9975	1.0101
Humidity	0.9956	0.9878	1.0036
Temperature	0.9789	0.9436	1.0155
Air pressure	1.0001	0.9995	1.0006

Table 90: Adjusted Odds ratios between respiratory mortality and air pollutant levels in 2007

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.9760	0.6935	1.3735
O_3 (ppb)	0.9952	0.9860	1.0045
NO_2 (ppb)	1.0056	0.9925	1.0188
SO_2 (ppb)	1.0318	0.9889	1.0765
PM_{10} (µg/m^3)	1.0006	0.9949	1.0063
Humidity	0.9988	0.9916	1.0060
Temperature	1.0051	0.9651	1.0467
Air pressure	0.9971	0.9723	1.0225

Table 91: Adjusted Odds ratios between respiratory mortality and air pollutant levels in 2008

Air Pollution	Adj. OR	95% CI	
		Lower	Upper
CO (ppm)	0.7757	0.5302	1.1348
O_3 (ppb)	0.9993	0.9889	1.0097
NO_2 (ppb)	1.0209	1.0064	1.0355
SO_2 (ppb)	0.9560	0.9066	1.0081
PM_{10} ($\mu g/m^3$)	0.9975	0.9916	1.0035
Humidity	0.9945	0.9871	1.0018
Temperature	1.0076	0.9630	1.0543
Air pressure	0.9911	0.9615	1.0216

Table 92 Odds ratios between all natural mortality and air pollutants level between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (ppm)	1.0205	1.0021	1.0392	1.0424	1.0071	1.0788
O_3 (ppb)	1.0015	1.0001	1.0030	1.0010	0.9997	1.0023
NO_2 (ppb)	1.0007	0.9997	1.0018	1.0011	1.0001	1.0021
SO_2 (ppb)	1.0056	1.0020	1.0093	1.0057	0.9997	1.0117
PM_{10} ($\mu g/m^3$)	1.0009	1.0004	1.0013	1.0008	1.0004	1.0013

Table 93: Adjusted Odds ratios between all natural mortality and air pollutants level between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	Adj. OR	95% CI		Adj. OR	95% CI	
		Lower	Upper		Lower	Upper
CO (ppm)	1.0149	0.9878	1.0427	1.0365	0.9721	1.1052
O_3 (ppb)	0.9992	0.9973	1.0011	0.9980	0.9963	0.9998
NO_2 (ppb)	0.9993	0.9971	1.0014	0.9999	0.9975	1.0024
SO_2 (ppb)	1.0029	0.9990	1.0067	0.9979	0.9909	1.0050
PM_{10} ($\mu g/m^3$)	1.0008	1.0001	1.0016	1.0011	1.0001	1.0022
Humidity	0.9982	0.9969	0.9994	0.9983	0.9970	0.9996
Temperature	1.0107	1.0045	1.0169	1.0164	1.0098	1.0231
Air pressure	0.9968	0.9924	1.0012	0.9999	0.9998	1.0001

Table 94: Odds ratios between cardiovascular mortality and air pollutant levels between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (ppm)	1.1311	1.0749	1.1902	1.0521	0.9602	1.1525
O_3 (ppb)	1.0023	0.9984	1.006	1.0041	1.0005	1.0075
NO_2 (ppb)	1.0037	1.0008	1.0065	1.0025	0.9998	1.0052
SO_2 (ppb)	1.0119	1.0022	1.0218	1.0074	0.9916	1.0233
PM_{10} ($\mu g/m^3$)	1.0025	1.0013	1.0038	1.0009	0.9996	1.0021

Table 95: Adjusted odds ratios between cardiovascular mortality and air pollutant levels between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	Adj. OR	95% CI		Adj. OR	95% CI	
		Lower	Upper		Lower	Upper
CO (ppm)	1.1087	1.0263	1.1976	1.0007	0.8440	1.1867
O_3 (ppb)	1.0012	0.9960	1.0065	1.0031	0.9984	1.0077
NO_2 (ppb)	0.9920	0.9862	0.9979	1.0033	0.9969	1.0097
SO_2 (ppb)	1.0091	0.9986	1.0198	1.0042	0.9857	1.0230
PM_{10} ($\mu g/m^3$)	1.0031	1.0008	1.0054	0.9984	0.9956	1.0011
Humidity	0.9995	0.9960	1.0030	0.9980	0.9946	1.0014
Temperature	0.9956	0.9790	1.0125	0.9967	0.9797	1.0140
Air pressure	0.9947	0.9827	1.0069	1.0001	0.9997	1.0005

Table 96: Odds ratios between respiratory mortality and air pollutant levels between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	OR	95% CI		OR	95% CI	
		Lower	Upper		Lower	Upper
CO (ppm)	0.9893	0.9258	1.0571	1.1321	1.0143	1.2634
O_3 (ppb)	1.0059	1.0007	1.0111	1.0033	0.9991	1.0076
NO_2 (ppb)	0.9998	0.9961	1.0036	1.0051	1.0018	1.0083
SO_2 (ppb)	1.0076	0.9951	1.0204	1.0144	0.9951	1.0341
PM_{10} ($\mu g/m^3$)	1.0004	0.9988	1.0021	1.0022	1.0007	1.0037

Table 97: Adjusted Odds ratios between respiratory mortality and air pollutant levels between 1999-2001 and 2006-2008

year	1999-2001			2006-2008		
Air Pollution	Adj. OR	95% CI		Adj. OR	95% CI	
		Lower	Upper		Lower	Upper
CO (ppm)	0.9876	0.8964	1.0882	0.9762	0.7924	1.2025
O_3 (ppb)	1.0071	1.0002	1.0141	0.9976	0.9921	1.0033
NO_2 (ppb)	0.9988	0.9912	1.0066	1.0052	0.9973	1.0131
SO_2 (ppb)	1.0052	0.9918	1.0189	1.0002	0.9776	1.0233
PM_{10} ($\mu g/m^3$)	0.9999	0.9970	1.0029	1.0002	0.9968	1.0035
Humidity	0.9997	0.9953	1.0042	0.9964	0.9922	1.0006
Temperature	1.0193	0.9971	1.0421	0.9991	0.9781	1.0206
Air pressure	0.9931	0.9835	1.0144	1.0001	0.9995	1.0006

CURRICULUM VITAE

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Areas of Expertise

Public Health/ Epidemiology over 22 years

Working experience

1990-1997	Public health official level 2, Working with government in ministry of public health at health center village and sub district. Job descriptions were prevention and control diseases in rural area.
1997-1998	Customers service at IBM and Nippon Express
1998-2006	Public health official level 7, Working with government in ministry of public health at health office Nakhonnahyok and Nong Khai province.
2006-2007	Public health official level 7, at Department of disease control and prevention, Ministry of public health. Job description was reviewer researches for Ethic Committee DDC Thailand.
2009	Statistic in Public Health Lecturer at Chalermkarnchana College for 2 months. Resignation because passed doctoral examination and had to studying course work for 2 semesters.

Academics history

19-21 January 2005	prevention motorcycle accident in community (oral presentation in motor accidental national conference)
25-27 May 2005	Participation of health volunteer to survey H5N1 (oral presentation in Epidemiology national conference)
4-6 September 2006	meditation for happiness (oral presentation in public health national conference)
19-23 August 2007	Sexual behavior in sex worker (poster presentation in 8th International Congress on AIDS in Asia & the Pacific)