

AN ASSOCIATION BETWEEN INCENSE SMOKE EXPOSURE AND
INCREASED CAROTID INTIMA-MEDIA THICKNESS
AMONG PEOPLE LIVING IN MUEANG DISTRICT,
SAKON NAKHON PROVINCE: A COHORT STUDY



บทคัดย่อและแฟ้มข้อมูลฉบับเต็มของวิทยานิพนธ์ตั้งแต่ปีการศึกษา 2554 ที่ให้บริการในคลังปัญญาจุฬาฯ (CUIR)
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ความสัมพันธ์ระหว่างการสัมผัสควีนรูปและการเพิ่มความหนาของอินทิมา- มีเดียของหลอดเลือด
คาโรติคในประชาชนที่อาศัยอยู่ในอำเภอเมือง จังหวัดสกลนคร: การศึกษาแบบไปข้างหน้า



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การจูดรูปเป็นแหล่งกำเนิดมลพิษอากาศในบ้านเรือน ซึ่งสามารถส่งผลกระทบต่อระบบหัวใจและหลอดเลือด การศึกษานี้มีวัตถุประสงค์เพื่อทดสอบความสัมพันธ์ของการสัมผัสควันธูปในบ้านเรือนระยะยาวกับการเพิ่มความหนาของอินทิมา-มีเดียของหลอดเลือดคาโรติด ระหว่างประชาชนที่อาศัยอยู่ในกลางเมืองของจังหวัดสกลนคร ประเทศไทย การศึกษาแบบไปข้างหน้า 1 ปี ได้ดำเนินการระหว่างเดือนกรกฎาคม 2559 ถึง เดือนกันยายน 2560 มีอาสาสมัครจำนวน 132 คนในช่วงแรกของการศึกษา และเหลืออาสาสมัคร 100 คน หลังจากติดตามผล 1 ปี อาสาสมัครได้ถูกแบ่งออกเป็น 3 กลุ่มตามความถี่ของการใช้ธูปในบ้านเรือน ได้แก่ กลุ่มที่ไม่ได้สัมผัสควันธูป, กลุ่มที่สัมผัสธูปไม่ทุกวัน และกลุ่มที่สัมผัสธูปทุกวัน อาสาสมัครทั้งหมดจะถูกสัมภาษณ์โดยแบบสอบถาม และได้รับการตรวจทางคลินิก ได้แก่ ตรวจเลือด และอัลตราซาวด์หลอดเลือดแดงที่ลำคอ รวมถึงตรวจวัดความเข้มข้นของฝุ่นละอองขนาดเล็กกว่า 10 ไมโครเมตร, อุณหภูมิ และความชื้นสัมพัทธ์ในบ้านเรือนในช่วงฤดูฝนและฤดูแล้ง การวิเคราะห์เพื่อหาความสัมพันธ์ของตัวแปรจะดำเนินการร่วมกับการปรับปัจจัยกวนบางตัวที่อาจเกิดขึ้น ผลการศึกษาแสดงให้เห็นว่า ในช่วงเริ่มการศึกษา มีความสัมพันธ์เชิงบวกระหว่างการจูดธูปในบ้านเรือนและความหนาของอินทิมา-มีเดียของหลอดเลือดคาโรติดเฉลี่ยรวมทั้งสองข้าง (CCA) และหลอดเลือดคาโรติดด้านซ้าย (LCCA) แต่ไม่พบความสัมพันธ์ในความหนาของอินทิมา-มีเดียของหลอดเลือดคาโรติดด้านขวา (RCCA) หลังจากติดตามผล 1 ปี พบว่า พัฒนาการของความหนาของอินทิมา-มีเดียของหลอดเลือดคาโรติดไม่พบความสัมพันธ์ในกลุ่มที่สัมผัสควันธูป สำหรับค่าเฉลี่ยความเข้มข้นของฝุ่นละอองขนาดเล็กกว่า 10 ไมโครเมตร เท่ากับ 24.2 ± 11.4 ไมโครกรัมต่อลูกบาศก์เมตร ความเข้มข้นของฝุ่นละอองขนาดเล็กกว่า 10 ไมโครเมตรในบ้านเรือนที่เพิ่มขึ้น 1 ไมโครกรัมต่อลูกบาศก์เมตรมีความสัมพันธ์กับการเพิ่มความสูงของการหนาขึ้นของค่าเฉลี่ย (mean) และค่าสูงสุด(maximum) ของความหนาอินทิมา-มีเดียในหลอดเลือดคาโรติดรวมทั้งสองข้าง (CCA) ร้อยละ 8 ร้อยละ 7 ตามลำดับ และเสี่ยงต่อการเพิ่มความหนาของค่าสูงสุด (maximum) ของความหนาอินทิมา-มีเดียในหลอดเลือดคาโรติดด้านซ้าย (LCCA) ร้อยละ 3 อย่างมีนัยสำคัญ ($p < 0.05$) แต่ไม่พบความสัมพันธ์กับความหนาของหลอดเลือดคาโรติดด้านขวา (RCCA) สรุปได้ว่า การสัมผัสควันธูปในระยะยาวมีความสัมพันธ์กับการเพิ่มความหนาของอินทิมา-มีเดียของหลอดเลือดคาโรติด แต่อย่างไรก็ตาม พัฒนาการของความหนาของอินทิมา-มีเดียของหลอดเลือดคาโรติดหลังจากติดตามผล 1 ปีนั้นไม่พบความสัมพันธ์กับการสัมผัสควันธูปแต่มีความสัมพันธ์กับฝุ่นละอองในบ้านเรือน ดังนั้น นโยบายด้านอนามัยสิ่งแวดล้อมควรมีการกำหนดค่ามาตรฐานของคุณภาพอากาศภายในอาคารหรือในบ้านเรือนในประเทศไทย

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RATANEE KAMMOOLKON: AN ASSOCIATION BETWEEN INCENSE SMOKE EXPOSURE AND INCREASED CAROTID INTIMA-MEDIA THICKNESS AMONG PEOPLE LIVING IN MUEANG DISTRICT, SAKON NAKHON PROVINCE: A COHORT STUDY. ADVISOR: ASST. PROF. NUTTA TANEAPANICHSKUL, Ph.D., CO-ADVISOR: ASSOC. PROF. VITTOOL LOHSOONTHORN, Ph.D., 119 pp.

Incense burning, a source of household indoor air pollution, is possible to effect on cardiovascular system. This study sought to examine the association of exposure to household incense smoke with increased Carotid intima-media thickness (CIMT) amongst people living in the central city of Sakon Nakhon province, Thailand. A one-year cohort study was conducted between July 2016 and September 2017. There were 132 participants at baseline and remained 100 participants after 1-year follow-up. Participants were stratified into three groups by frequency of incense use in their household; non-exposed group, non-daily exposed group, and daily exposed group. All participants were interviewed by questionnaire and underwent a clinical assessment, blood test and a carotid artery ultrasound. Household PM₁₀ concentrations, temperature, and relative humidity (RH) were measured inside all of the participants' home during the wet and the dry seasons. To find an association, multivariate analysis was performed with adjusted some potential confounding factors. The result showed the positive association between household burned incense and CIMT at common carotid artery (CCA) and left of common carotid artery (LCCA) but, not found in the CIMT of right of common carotid artery (RCCA) at baseline. After 1-year follow-up, the progression of CIMT in incense smoke exposure group were not found an association. For the average of PM₁₀ concentrations inside house was 24.2±11.4 µg/m³. An increasing of 1 µg/m³ average indoor PM₁₀ concentration were significant (p<0.05) associated with 8% increased risk of increased mean CCA (AOR = 1.08; 95%CI 1.01 - 1.15), 7% increased risk of increased maximum CCA (AOR = 1.07; 95%CI 1.01 - 1.12) and 3% increased risk of increased maximum LCCA (AOR = 1.03; 95%CI 1.01 - 1.09) but not for CIMT at RCCA. In conclusion, long-term exposure to incense smoke was associated with an increased CIMT. However, the progression of CIMT after a year of follow-up was not associated with incense smoke exposure but was associated with household particulate matter (PM₁₀). Therefore, the policy for environmental health should be considered to provide the standard level of indoor/ residential air quality in Thailand.

Field of Study: Public Health

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Student's Signature

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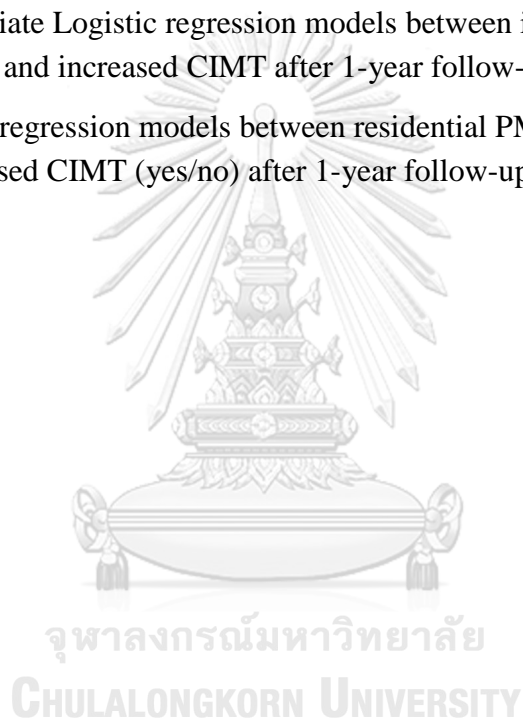
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LIST OF ABBREVEATIONS

AHA	= American Heart Association
ALRI	= Acute Lower Respiratory Disease
BMI	= Body Mass Index
CAC	= Coronary Artery Calcium
CAD	= Coronary Artery Diseases
CCA	= Common Carotid Artery
CHD	= Coronary Heart Disease
CIMT	= Carotid Intima-Media Thickness
COPD	= Chronic Obstructive Pulmonary Disease
CVD	= Cardiovascular Disease
DBP	= Diastolic Blood Pressure
HAP	= Household Air Pollution
HDL	= High Density Lipoprotein Cholesterol
Hs-CRP	= High Sensitivity C - reactive protein
IAQ	= Indoor Air Quality
ICA	= Internal Carotid Artery
IHD	= Ischemic Heart Disease
LCCA	= left common carotid arteries
LDL	= Low Density Lipoprotein Cholesterol
NIOSH	= The National Institute for Occupational Safety and Health
PAHs	= Polycyclic Aromatic Hydrocarbons
PM ₁₀	= Particular Matter less than 10 micrometer
PM _{2.5}	= Particular Matter less than 2.5 micrometer
RCCA	= right common carotid arteries
RH	= Relative Humidity
SBP	= Systolic Blood Pressure
TG	= Triglycerides
VOCs	= Volatile Organic Compounds
WHO	= World Health Organization

CHAPTER I

INTRODUCTION

1.1 Background and Rationale

World Health Organization reported that indoor air pollution contribute to 4.3 million deaths (WHO, 2012) due to health outcomes such as cerebrovascular diseases (stroke 34%), ischemic heart disease (IHD 26%), Chronic obstructive pulmonary disease (COPD 22%) and acute lower respiratory disease (ALRI 12%)(Smith et al., 2014). Primarily source of exposure to pollutants are cook stoves and open hearths. Incense burning release similar pollutants as cook stove, including particulate matter, carbon monoxide, sulfur dioxide and oxides of nitrogen (Cohen, Sexton, & Yeatts, 2013) which may effect on human health. According to the California Air Resources Board report, indoor air pollutant levels are greater than outside levels around 25 to 62 percent (ARB, 2005). The U.S. Environmental Protection Agency indicated that household indoor air and other buildings can be more seriously polluted than the outdoor air because people spend more than 90 percent amount of their time inside building. Infants and other vulnerable people may get a higher risk from indoor air pollution exposure than outdoor exposure (U.S.EPA, 1989).

Incense burning is a daily practice of Buddhism and Taoism which is normal religions in Asian countries, such as China, Thailand, and Taiwan. In Thailand, the average demand for incense is accountable for 1 million sticks per year (Department of industrial, ministry of industrial, 2016). A component of incense stick are 21% (by weight) of herbal and wood powder, 35% of fragrance material, 11% of adhesive powder, and 33% of bamboo stick (Lin, Krishnaswamy, & Chi, 2008). There are several pollutants from incense burning such as, particulate matter (PM), gas products (include CO, CO₂, NO₂, SO₂ and others) and many volatile organic compounds (VOCs). The PM produced form incense burning is greater than 45 mg/g burned when compared to cigarettes burning for 10 mg/g (Lin et al., 2008). In additional, incense burning also produces aldehydes and polycyclic aromatic hydrocarbons (PAHs) shown to harm human health (Lin et al., 2008).The study of United Arab Emirates (UAE) were

identified and measured the particles and gases emitted from two kinds of incense typically used in homes. Incense smoke exposure during the burn inside the home produced PM 1.42 mg/m³, CO 122 ppm, NO 0.3 ppm, and HCHO 85 ppb and several other carbonyls, resulting in the cellular inflammatory response. The result also found that the averages of PM, CO, and NO values exceeded current government regulation (Cohen et al., 2013). Study of Taiwanese were indicated that fine PMs contribute the majority of indoor particulate in 10 temples, the results showed that concentration of PM during incense burning in temples were 155.1 ±41.5 µgm⁻³ and PM₁/PM₁₀ ratios were estimated to be 81.2±5.3% (Chiang & Liao, 2006).

Several studies have shown a positive association between particulate matter concentrations and adverse health effects on cardiovascular mortality, accelerated atherosclerosis, vascular inflammation and stroke (Bornstadt, Kunz, & Endres, 2014; Du, Xu, Chu, Guo, & Wang, 2016; Lee, Kim, & Lee, 2014; Pope et al., 2004). There were a number of epidemiological and experimental studies confirmed an association between air pollution (PM) and cardiovascular disease (CVD) as risk factors. (Brook et al., 2010; Du et al., 2016; Gill et al., 2011; Pope et al., 2004) Some studies also established that “long term exposure to ambient and individual particular matter less than 2.5 µm in diameter (PM_{2.5}) is accountable for morbidity and mortality of CVD events”. (Miller et al., 2007; Pope et al., 2004) In addition, a chronic process of atherosclerosis is mainly affects the aorta, coronary artery and cerebral artery, due to lumen occlusion and plaque rupture which is the major pathological process of heart disease and stroke. Some studies have suggested that carotid artery intima-media thickness (CIMT) can identified level of atherosclerosis to estimate cardiovascular risk in population's future. (Chambless et al., 2000; Stein et al., 2008). A few cross-sectional studies observed significant associations between ambient PM or other markers of air pollution and the level of atherosclerosis, measured with CIMT, and both coronary and aortic calcifications. (Kunzli et al., 2010; Liu et al., 2015) . Long term exposure to particular matter were shown an associated with an increase of CIMT around 16.79 µm (95% CI, 4.95–28.63 µm) and 4.13 µm (95% CI, -5.79–14.04 µm) for an increase of 10 µg/m³ in PM_{2.5} and PM₁₀ respectively. The finding of this study suggested that an associated between higher PM exposed and increased CIMT results from the processes

of cumulative atherogenesis. “CIMT is a predictor of cardiovascular events and also has been linked to myocardial infarction, stroke and blood pressure”.(Liu et al., 2015).

A study of “chronic exposure to biomass fuel was strongly associated with increased carotid artery intima-media thickness and a higher prevalence of atherosclerotic plaque” demonstrated that indoor air pollution may increase carotid thickness. (Painschab et al., 2013). A prospective cohort study reported that higher long-term PM_{2.5} exposure were an association with increased IMT progression for a mean annual progression of 14 $\mu\text{m}/\text{y}$ per 2.5 mg/m^3 higher levels of residential PM_{2.5} (S. D. Adar et al., 2013). Similarly, the study of residential exposure to urban traffic was associated with CIMT in children (Armijos et al., 2015) and the study of long-term exposures to traffic-related air pollution (PM_{2.5}, PM₁₀, and NO_x) were positively associated with subclinical atherosclerosis middle-aged adults. The result showed the percentage increases of maximum left CIMT in one-year were 4.23% (95% CI: 0.32, 8.13) for increase in PM_{2.5} and 3.72% (95% CI: 0.32, 7.11) for 10- $\mu\text{g}/\text{m}^3$ increase in PM₁₀(Su, Hwang, Shen, & Chan, 2015).

Pollutants emitted from incense burning contains particulate matter (PM), gas products and many organic compounds are the great concern which may increase the risk of CVD and stroke (Armijos et al., 2015; Liu et al., 2015). The mechanisms of air pollution and CVD had included in the Multi-Ethnic Study of Atherosclerosis (Gill et al., 2011). However, only one studies from Chinese population in Singapore reported that long-term exposure to incense burning in home environment and cardiovascular mortality. (Pan et al., 2014) This study found that there was an associated with an increases risk of cardiovascular mortality in noncurrent users and current long-term users. Comparing between incense user and non-incense user found that in incense user had 12% higher risk of cardio vascular mortality, 19% higher risk of stroke and 10% higher risk of coronary heart disease.

To our knowledge, there is no study focused on an association between daily incense smoke exposure and increased CIMT. Sakon Nakhon province was chosen as a study area for this study because, there are a high rate of patients and death rate with ischemic heart disease and stroke. From the report in 2012, report that rates of patients admitted to hospital with ischemic heart disease and stroke were 1,496 and 1,332 patients with rate 129.19 and 115.03 respectively. In 2014, death rate per 1,000

populations from ischemic heart disease and stroke which were accountable for 22.53 and 31.77 respectively (Sakon Nakhon province public health office, 2014). Furthermore, burning incense inside home differentiate people behavior between the one living in the central city and the one living in other areas of this province in term of their occupation and ethnicity. Additionally, Sakon Nakorn province were selected to a study area for minimizing an effect of traffic-pollutants on CIMT (Armijos et al., 2015). Therefore, a current one-year cohort was investigated an association between daily exposed to incense smoke and increased CIMT by carotid artery ultrasound which can potentially lead to earlier progression to atherosclerosis and linked to CVD and stroke.

1.2 Research Question

1. Does incense smoke exposure increase Carotid Intima Media Thickness (CIMT) in people living in the central city of Sakon Nakhon province?
2. Does household particulate matter less than 10 micrometer (PM_{10}) related to incense burning increase of Carotid Intima Media Thickness (CIMT) among in people living in the central city of Sakon Nakhon province?

1.3 Objectives

1.3.1 General objective

To investigate an association between incense smoke exposure and increased Carotid Intima Media Thickness (CIMT) in people living in the central city of Sakon Nakhon province.

1.3.2 Specific Objectives

Phase I: Baseline characteristics

1. To compare residential environment among daily incense smokes exposed, non-daily incense and non-incense smoke exposed group in the dry season
2. To find an association between incense smoke exposure and the levels of CIMT at baseline.

Phase II: increased of CIMT after 1-year follow-up

1. To compare residential environment among daily incense smokes exposed, non-daily incense and non-incense smoke exposed group in the wet season
2. To find an association between incense smoke exposure and increased Carotid Intima Media Thickness (CIMT) after 1-year follow-up.
3. To find an association between the average of household PM₁₀ and increased of CIMT after 1-year follow-up.

1.4 Research Hypotheses

Exposure to incense smoke increases Carotid Intima Media Thickness (CIMT).

1.5 Variable of the study

1.5.1 Independent variable

1. Socio-demographic characteristics

- Age
- Gender
- Body Mass Index (BMI)
- Past medical
- Family history of CVD
- Level of Education
- Occupational
- Smoking status
- Alcohol consumption
- Physical activity

2. Household characteristics

- Type of resident
- Fuel cooking used
- mosquito repellent used

3. Incense used characteristics

- Type of incense
- number of incense used per time
- Duration of incense used
- History of incense used
- Location of incense use (close/Open)

4. Clinical assessments

Blood test

- Total cholesterol
- Triglycerides (TG)
- High density lipoprotein (HDL)
- Low density lipoprotein (LDL)
- Hemoglobin A1c
- High Sensitivity C-reactive protein (Hs-CRP)

Hemodynamics

- Heart rate
- Systolic blood pressure (SBP)
- Diastolic blood pressure (DBP)

5. Household particulate matter

- Particulate matter less than 10 micrometer (PM₁₀).
- Temperature (°C)
- Relative Humidity (% RH)

1.5.2 Dependent variables: Carotid intima-media thickness (CIMT).

1.6 Conceptual Framework

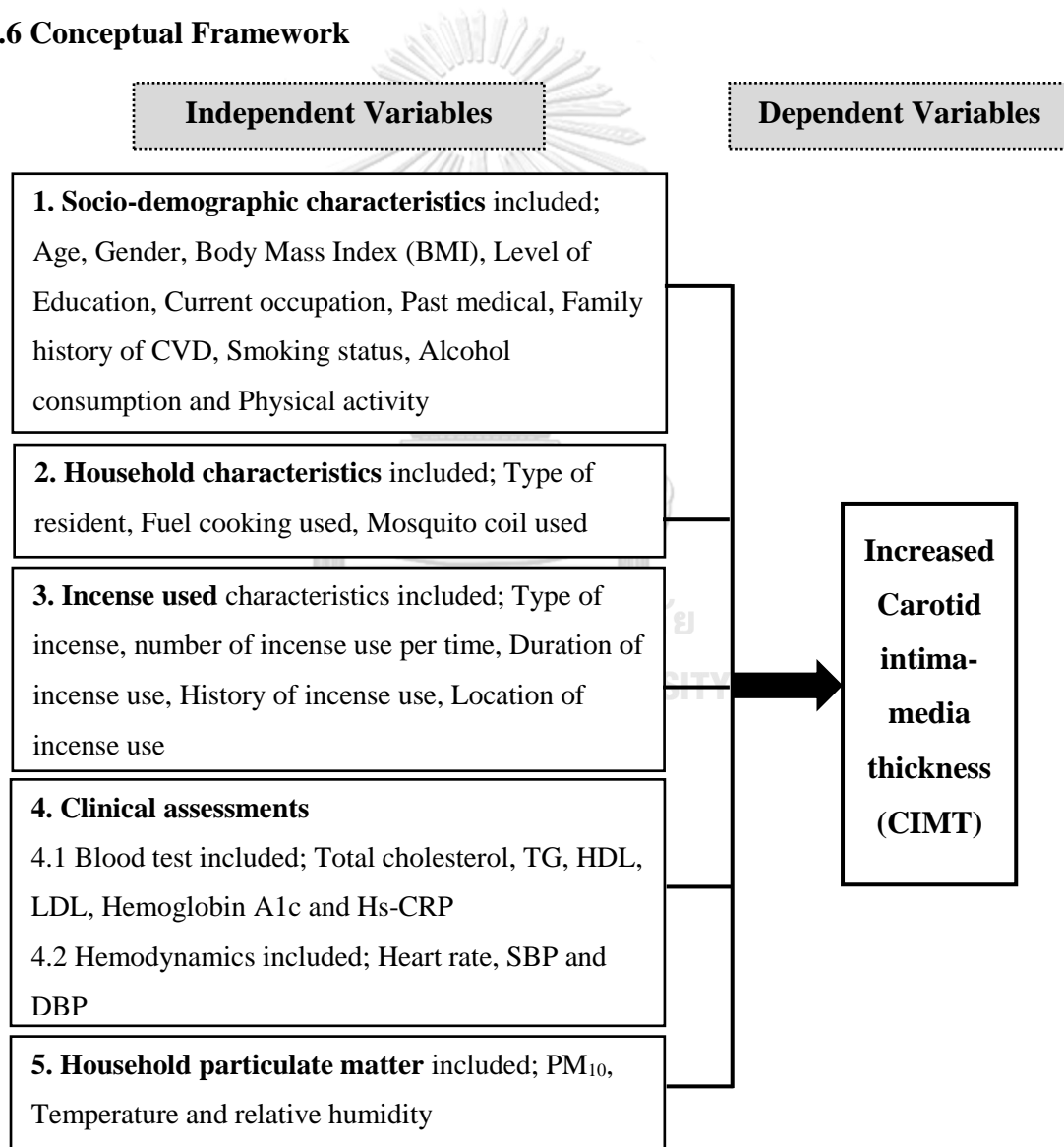


Figure 1 Conceptual Framework

1.7 Terms of Definitions

This study to measure and investigate an association between incense smoke exposure increase risks of Carotid intima-media thickness (CIMT) among people living in the central city of Sakon Nakhon Province, Thailand.

1.7.1 Incense is aromatic biotic material which release smoke when burned. The term refers to the material itself, rather than to the aroma that it produces. Incense is used for a variety of purposes, including “the ceremonies of religion, to overcome bad smells, repel insects, spirituality, aromatherapy, meditation, and for simple pleasure”. In this study refer to incense stick that use for burning in daily basis to worship inside participant’s home.

1.7.2 Incense smoke exposure is the level of emission smoke concentrations when burned inside home. There are several pollutants from incense burning. In this study focused on particulate matter (PM₁₀).

1.7.3 Incense smoke is the emission from burning incense. There are contains “particulate matter (PM), gas products and others. Incense burning also produces VOCs, such as benzene, toluene, and xylenes, as well as aldehydes and PAHs”. In our study, we are going to assess particulate matter (PM) and gas products include, CO, NO_x and SO₂

1.7.4 Particulate matter (PM) is the form of solid mixture particles and liquid droplets found in the indoor air. Some particles, such as duster smoke are large or dark enough to be seen with the naked eye. They can only be identified by an electron microscope. In this study, there are 2 type, coarse particles (PM₁₀) and Fine particles (PM_{2.5}) (WHO, 2004). In this study, source of particulate matter was focused on incense burning inside home in Mueang districts, Sakon Nakhon province.

1.7.5 Indoor particulate matter is particulate matter concentrations inside home. In our study, we are going to assess the particulate matter less than 10 µg/m³ (PM₁₀) levels in participant’s home by using particulate matter standard procedure recommended by NIOSH method (NIOSH, 1994).

1.7.6 Carotid intima-media thickness (CIMT) is “the screening measures the thickness of a patient’s arterial walls by ultrasound scanner. Increased thickness in the walls of CCA is associated with risk for CHD” (AHA, 2012).

1.7.7 CIMT measurement is an assessing the thickness as both the mean and maximum of three predefined angles (anterior, lateral, and posterior) capturing the media-adventitia interface of the near and far arterial walls, 1 cm proximal to the bulb from common carotid both right and left common carotid arteries, 10 mm length for proper location of CIMT measurement.

1.7.8 Cardiovascular disease (CVD) is “a general term that describes the heart disease or blood vessels, includes coronary artery diseases (CAD) such as angina and myocardial infarction (commonly known as a heart attack)” (Mendis, Puska, Norrving, & editors, 2011).

1.7.9 Personal factors is the characteristic of participant. In this study focus on socio-demographics, hemodynamics and blood test.

1.7.10 Socio-demographic information is the personal characteristic, there are age, gender, Body Mass Index (BMI), level of education, current occupational, past medicals, family history of CVD, smoking status, alcohol consumptions and physical activity.

1.7.11 Clinical assessments

- **Hemodynamics** is “the fluid dynamics of blood flow which important part of cardiovascular physiology dealing with the forces the pump (the heart) has to develop to circulate blood through the cardiovascular system”. In this study were measured a dynamics of blood flow included, heart and blood pressure (BP); in terms of the systolic (maximum) pressure over diastolic (minimum) pressure.

- **Blood test** was included, “total cholesterol, high density lipoprotein cholesterol, low density lipoprotein cholesterol, triglyceride, hemoglobin A1c and high sensitivity C-reactive protein (hs-CRP)”.

1.7.12 Home characteristics is the condition of participant’s home that related to indoor air pollution concentration included;

1. Type of resident is a kind of home such as shop house, townhouse, single house which may effect to the distribution of indoor air pollutants.

2. Fuel cooking use is any material that can be releases the energy as heat to be used for cooking such as biomass and liquid gas.

3. Mosquito coil used is “a mosquito repelling incense, usually made into a spiral, and typically made from a dried paste of pyrethrum powder”. “Burning usually

begins at the outer end of the spiral and progresses slowly toward the center of the spiral, producing a mosquito-repellent smoke” (McKean, Erin, et al, 2005)

4. Temperature is a qualified measure of hot or cold inside home by HOBO data logger at 5 minutes.

5. Humidity is “the amount of water vapor in the air. Higher humidity reduces the effectiveness of sweating in cooling the body by reducing the rate of evaporation of moisture from the skin”.

6. Indoor pollutants related to incense burning is the pollutants emitted from incense burning. In this study focused on PM₁₀.

1.7.13 Incense Used is the condition of incense using at home included, type of incense, number of incense use per day, duration of incense use, and history of incense use and location of incense use, included;

1. Type of incense is a composition of incense sticks that demonstration by form of incense such as stick, cone, coil and powder.

2. Number of incense use per day is amount of incense use in daily days. It's depend on the purpose of use such as 9 sticks for worship.

3. Duration of incense use is the period of the time to burning incense on a daily basis.

4. History of incense use is the duration of incense use in the past.

5. Location of incense use is the area that have incense burning (may be close or open room)

In this study, incense use characteristic was related into 3 levels according to behavior of incense burning inside home included;

1. Daily incense exposure is the burning of incense inside home in daily. This study, defined as using of incense stick for burning inside home ≥ 5 days per week. (Navasumrit et al., 2008)

2. Non-daily incense exposure is the burning of incense inside home as normal practice occasionally. In this study, define as participants who burned incense < 5 days/week.

3. Non-incense exposure is participants who had never burned incense inside their houses.

CHAPTER II

LITERATURE REVIEW

2.1 The indoor air quality (IAQ)

In all appreciate that homes IAQ can be worse very unhealthy and polluted. Several research is “pointing to very serious health significances from both short term and long term exposure to particulate matter (PM), caused by inside the house and carried in from outside’. A problem happens from buildup of pollutants from inside the home due to the homes are sealed from the outdoor air to rise heating or cooling efficiency. Some outdoor air is essential and also unavoidable no matter for sealed at home. Therefore, household PM will be a combination of particles from outdoor and those produced inside the house (Isaxon et al., 2015).

In our homes there are many diversities of the particulate contaminants. For outdoor air included “industrial sources, construction sites, combustion sources, pollen, and numerous others”. The particles are generated by "indoor activity" such as cooking, incense used, house cleansing, the carpet, and pet or even just sitting on the sofa, movement or vibration can produce airborne particles (air movement, even over a clean surface, apparently to particles), airborne allergens (mold spores, bacteria, and dust mite/insect feces) are also present. Including constant laser printer can be a high-level emitter of PM. Indoor particular matter can be increased seriously by automobile and truck exhaust into their home. A many result of studies showed “a relation between this particulate pollution and cardiovascular disease, respiratory disease, and cancer”. The studies of adolescent children showed slow down lung function growth can cause by particulate pollution “similar to cigarette smoking with predictable consequences for lung health later in life”. Some studies were showed the rise on days with high particulate pollution can cause to mortality rates from a variation in the short term. The high concentrations of particulates can be affected on health problems in to long-term (appear probably for many years after the damaging exposure), like respiratory illnesses, heart disease, stroke or cancer. Therefore, daily air pollution with higher levels are associated with an increased risk of acute cardiovascular events, including

myocardial infarction, cerebrovascular event and decompensation in patients with congestive heart failure. (Bardin, 2015)

2.2 Health effects of household (indoor) air pollution

2.2.1 Health effects of air pollution

Particulate matter concentration is “effects on breathing and respiratory systems, damage to lung tissue, cancer, and premature death”. The sensitivity group are included “the elderly, children, and people with chronic lung disease, influenza, or asthma, tend to be especially sensitive to be affected by particulate matter exposure”. Short and long-term exposures to PM can effected to heart and lung disease, also can lead to daily life activity, illness and hospital admission and deaths. In another, it can “cause early death, particularly among people who have a higher risk of being affected by particle pollution”.

2.2.2 Burden of disease from Household Air Pollution

World Health Organization were reported that the globally, “4.3 million deaths were attributable to household air pollution (HAP) in 2012 which almost all in low and middle income (LMI) countries”. The South East Asian and most of the burden with 1.69 and 1.62 million deaths in Western Pacific regions, respectively. “The large increase in burden compared with the previous estimate of 2 million deaths from HAP from 2004” (WHO, 2009) is mainly due to 1) the analysis included health outcomes such as cerebrovascular diseases and ischemic heart disease (Smith et al., 2014). 2) the evidence showed that an exposure and health outcomes and were associated with the integrated exposure response functions use (Burnett et al., 2014) and 3) non-communicable diseases were increased. While, a higher levels exposure in women due to their greater involvement in daily cooking activities. Therefore, women have a higher relative risk to develop adverse health outcomes than men, but the absolute burden in men is larger.

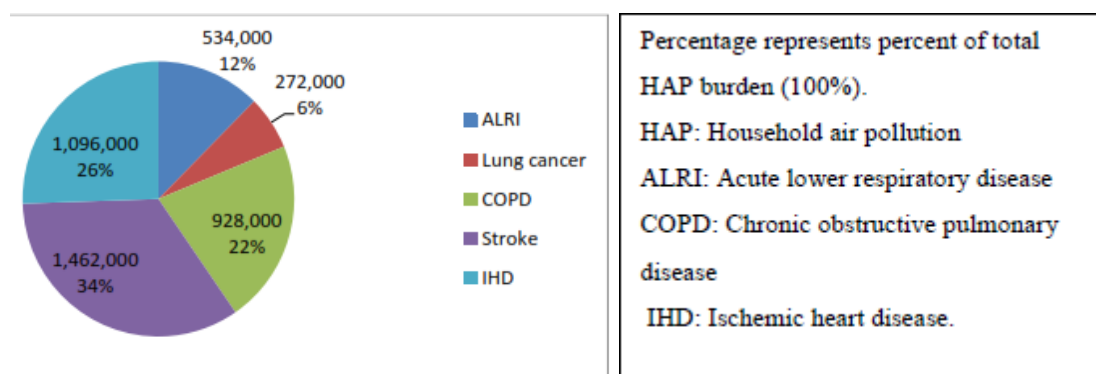


Figure 2 Deaths attributable to HAP in 2012, by disease (WHO, 2012)

2.2.3 Exposure-risk relationships

The Global Burden Disease 2010 were integrated exposure-response functions (IER) developed to use for ALRI (acute lower respiratory infections), lung cancer, stroke and IHD (ischemic heart disease) (Burnett et al., 2014) (Table 2.1). For COPD, the relative risks from the systematic review / meta-analysis were used for the GBD 2010 study (Smith et al., 2014)

Table 1 Relative risks of disease from Household Air Pollution exposure.

Disease	RR (95%CI)	RR (95%CI)	Reference
	women (≥ 25 years)	men (≥ 25 years)	
ALRI	2.9 (2.0-3.8) for children (under 5 years)	(Burnett et al., 2014; Smith et al., 2014)	(Burnett et al., 2014; Smith et al., 2014)
COPD	2.3 (1.7-3.1)	1.9 (1.2-3.1)	(Smith et al., 2014)
Lung cancer	2.3 (1.5-2.8)	1.9 (1.4-2.3)	(Burnett et al., 2014; Smith et al., 2014)
IHD	(1.4-2.2)	(1.4-2.2)	(Burnett et al., 2014; Smith et al., 2014)
Stroke	(1.4-2.4)	(1.3-2.4)	(Burnett et al., 2014; Smith et al., 2014)

(WHO, 2009)

2.2.4 Air Quality Index (AQI) and Health Concerns

Particles less than 10 micrometers in diameter which cause or worsen a number of health problems and have been linked with illnesses and deaths from heart or lung disease in short-term exposures and long-term exposures. The sensitive groups for particle pollution include “people with heart or lung disease, older adults and children.

The risk of heart attacks, and thus the risk from particle pollution, may begin as early as the mid-40s for men and mid-50s for women”. Also the people with heart or lung diseases and older adults who visit to emergency rooms, be hospitals admitted.

Particulate matter exposure may cause people with “heart disease to experience chest pain, palpitations, shortness of breath, and fatigue, and has also been associated with cardiac arrhythmias and heart attacks”. “A high levels of particle pollution concentration may not be able to breathe as deeply or vigorously in people with lung disease and also people who have a symptom such as coughing and shortness of breath can increase susceptibility to respiratory infections and can aggravate existing respiratory diseases”.

2.3 Characteristics of incense

2.3.1 Incenses and incense burning

Generally, there are several forms of incenses, for example, sticks, joss sticks, cones, coils, powders, rope, rocks/charcoal, and smudge bundles (James J. Jetter, Zhishi Guo , Jenia A. McBrian , & Flynn, 2001). The important disparity in the types of incense is a form of bamboo base. For some kind of incense, there is no the central base. Also, there is no the mixture of ingredients attached onto incense. Physically, the characteristics of these incenses are very lookalike, such as length, bamboo stick’s diameter, incense-coated part’s diameter, and weight. In general, “the incense sticks consists of 21% of herbal and wood powder, 35% of fragrance material, 11% of adhesive powder, and 33% of bamboo stick” (Lin et al., 2008). The process of producing a bamboo incense stick starts with soaking the mixture of fragrance, herbal and wood powders with adhesive materials. Then, the incense sticks are coated with the prepared mixtures. For the coating process, they should be coated repeatedly two more times. Eventually, all of incense stick from the coating process will be dried by sunlight.

Basically, the incense burns completely within 50 to 90 minutes. Moreover, the complete incense burning emits smokes or fumes. Also, these fumes normally contain particulate matter (PM), gasses (CO, CO₂, NO₂, SO₂, and others), and “other volatile organic compounds (VOCs), such as, benzene, toluene, and xylene. As well as, aldehydes and polycyclic aromatic hydrocarbons (PAHs) mostly are absorbed on particulate matter”.

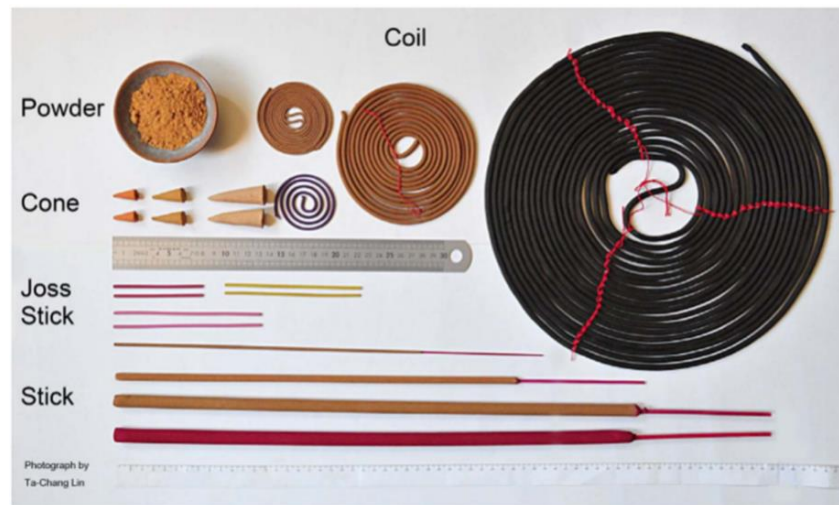


Figure 3 Five major forms of Asian incense; Photo by (Lin et al., 2008)

2.3.2 Main types of air pollutants from incense smokes and their toxicological effects

Inhalation is the major exposure route to the incense smokes. These smokes contain particulate matter, gas products and a variety of volatile organic compounds. Additionally, all of these chemical substances may cause adverse effects to human health.

1. Particulate matter (PM)

According to the previous studies, they reported that particulate matter is able to draw some negative effects to human health. There are many size of particulate matter, but the important size of particulate matter are 1) Coarse particles are greater than $10\ \mu\text{m}$ in diameter that are large enough to get into the human respiratory system, which are not able to pose a major threat to human health immediately. 2) Thoracic coarse particles ($\text{PM}_{10-2.5}$) size range in 10 to $2.5\ \mu\text{m}$ in diameter (WHO, 2004). 3) Fine particulate matters, which size are less than $2.5\ \mu\text{m}$ in diameter ($\text{PM}_{2.5}$). This kind of particles is able to enter in human body deeply, especially in alveoli, which possibly causes a terrible drawback of human health. 4) Ultrafine particles, its size is less than $0.1\ \mu\text{m}$ in diameter. However, breathing in the particles, which are less than $10\ \mu\text{m}$ in diameter, can accumulate in human's respiratory system and lead to some adverse effects after exposing for long term.

“The major sources of residential indoor particulate matter, especially in the size of $2.5\ \mu\text{m}$ are from the combustion of incense, wood, cigarette, and candles” (Fang,

Chu, Wu, & Fu, 2002). The previous research reported that incense burning emitted particulates matter greater than 45 mg/g burned when compared to 10 mg/g burned for cigarettes (Mannix, Nguyen, Tan, Ho, & Phalen, 1996). Previously, the research of indoor air pollution in Taiwan indicated that the emission rate of incense burning was of 0.038 ± 0.026 particles/second. About 62–92% of indoor particles sources are in the range from 0.5 to 5 μm , where were from cooking, incense burning, and other residential activities (Liao, Chen, Chen, & Liang, 2006b).

2. Gaseous emissions

2.1. Carbon monoxide (CO)

Generally, carbon monoxide is “produced from an incomplete combustion of organic substances, such as hydrocarbons, wood, incense, cigarette, and fossil fuels”. Carbon monoxide minimizes the capacity of blood oxygen carrying, especially in hemoglobin. Breathing in the low concentration of CO can lead to headache, dizziness, weakness and nausea, while the high concentration of CO can be fatal.

2.2. Sulfur dioxide (SO₂) and nitrogen dioxide (NO₂)

The exposures to SO₂, and NO₂ have the impacts on health. For example, decrease in work capacity, worsening of the existing CVD, effects on pulmonary function and respiratory system.

2.3. Volatile organic compounds (VOCs)

VOCs are chemicals that have low boiling points and also evaporates easily at room temperature included; benzene, toluene, xylenes, and isoprene. “Acute symptoms of exposed to VOCs; eye irritation or eye watering, nose irritation, throat irritation, headache, nausea/vomiting, dizziness, and asthma exacerbation and the chronic symptoms of VOCs exposure include cancer, liver damage, kidney damage, and central nervous system damage”.

2.4. Aldehydes

The combustion of incense burning is the source of aldehydes and ketones production. Also, it is able to generate aerosols and formaldehyde. Typically, aldehydes characteristics are a sort of volatile organic compounds, which can be exposures to irritating skin, eyes and the upper respiratory tract.

2.5. Polycyclic aromatic hydrocarbons (PAHs)

Several studies have been found that the incense smoke composed of polycyclic aromatic hydrocarbons (PAHs) (Lin et al., 2008). One study indicated the total mean of PAH concentrations indoor and outdoor air in temple were 6,258 ng/m³ and 231 ng/m³, respectively. The result showed 27 times higher concentration of PAH inside temples than outside air. The highest of individual PAHs concentrations were “acenaphthylene (3,583 ng/m³), naphthalene (1,264 ng/m³), acenaphthene (349 ng/m³), fluoranthene (243 ng/m³) and phenanthrene (181 ng/m³), orderly” (Lin et al., 2008). Another research in Thailand posted about incense smoke and human health. It pointed that the exposure to incense burning is likely to induce carcinogens in humanity, which elevates the risk of cancer development among temple workers (Navasumrit et al., 2008).

2.4 Incense smoke and health effects

In accordance with the former research, they presented that incense burning released pollutants, which are likely to be toxic to human health as same as second hand smoke. Owing to the fact that the constituent substances of incense smoke were particulate matters, VOCs, aldehydes, PAHs, and diethyl phthalate (DEP). All of these chemical substances are possibly dangerous to the lung and allergenic to the dermal and eyes (Lin et al., 2008). Recently, An Pan and colleagues found that the chronic exposure to incense burning in home was associated with posing the higher risk of cardiovascular mortality among Singaporean (Pan et al., 2014). Although, it has a difficulty to study the effects of incense smoke pollutants on human health, many research on epidemiology have proposed that the chemical substances of incense burning are probable to be the cause of health problems as the following;

1. Airway dysfunction

Various studies claimed that inhaling of pollutants from incense smoke probably originates respiratory dysfunction. For instance, a previously prospective cohort study Singapore indicated that “the duration and intensity of using incense were associated with an increased risk of squamous cell carcinomas in the entire respiratory tract” (p =0.004). Also, the researcher presented “the relative risk value of squamous cell carcinomas among chronic incense users was 1.8 (95% CI, 1.2-2.6; p=0.004) in the

entire respiratory tract” (Friborg et al., 2008). Another study has conducted an investigation into 109 temple workers in Kaohsiung, Taiwan.

2. Allergy and Dermatological Effects

Particulate matter from incense burning might be contaminated with lead, particularly in PM_{2.5} and PM_{2.5-10} that can induce lethal blood and modulated immune system with IgE production (Lin et al., 2008). Lin and colleagues showed the concentration of lead from incense burning in Taiwan temple, which detected PM_{2.5} and PM_{2.5-10} were 0.14 and 0.21 mg/g, respectively. One research found the link between incense burning fumes and dermatological problems. It claimed that using incense about 15 years or more led to have itchy de-pigmented macules on dorsum Manus, left shoulder, and abdomen (Hayakawa, Matsunaga, & Arima, 1987).

3. Carcinogenic

Incense smoke exposure is likely to be associated with many adverse health effects. One research reported that incense smoke could elevate the risk of leukemia in children, whose parents burned incense inside home (Lowengart et al., 1987). Another research showed a greater risk of lung cancer among Chinese females in Singapore who exposed to incense smoke (MacLennan et al., 1977). The chemical substances of incense smokes are able to induce genotoxic effects on human health and mammalian cells owing to sister chromatid exchange. It seemed to pose a higher than those of tobacco smoke condensates (Chen & Lee, 1996). Additionally, the study of incense smoke in Thailand indicated that “exposure to carcinogens that emitted from incense burning may risk of cancer among temple workers” (Navasumrit et al., 2008).

4. Cardiovascular (CVD) and Stroke

According to the previous research stated that the home environments, where had “a long-term exposure to incense burning was associated with an increased risk of CVD mortality” (Pan et al., 2014). Similarly, in another study, which found “the association between incense use and cardiovascular mortality”. A mortality of coronary heart disease (CHD) and stroke were difference as the potential pathophysiology of these two diseases (Hyvärinen et al., 2010; Wilhelmsen, Koster, Harmsen, & Lappas, 2005). Furthermore, one research revealed that indoor biomass combustion from solid fuels was associated with a higher risk of CHD (Lee et al., 2014) However, very few

research was found that incense burning as a source of indoor air associated with the risk of CVD.

2.5 Cardiovascular diseases (CVDs)

Cardiovascular disease was put into the first rank of death globally. In 2012, “an estimated 17.5 million people died from CVDs, representing 31% of all global deaths were approximately 7.4 and 6.7 million due to coronary heart disease and stroke, orderly”. Out of the 16 million deaths below the age of 70 owing to non-communicable diseases, about 82% were in low and middle income countries, and about 37% were caused by CVDs. To minimize the risk of cardiovascular diseases is “addressing behavioral risk factors such as tobacco use, unhealthy diet, obesity, physical inactivity, and harmful alcohol consumption amongst people who have cardiovascular disease or at the higher risk of cardiovascular (due to the presence of one or more potential risk factors such as hypertension, diabetes, hyperlipidemia, and so on)” need early detection, counseling to the specialist or even obtaining medication (WHO, 2009).

Cardiovascular diseases (CVDs) is “a group of disorder that concerned with heart and blood vessels including: 1) coronary heart disease; related to dysfunctional blood vessels supply to the heart muscle, 2) cerebrovascular disease which is linked to dysfunctional blood vessels supply to the brain 3) Peripheral arterial disease is connected to dysfunctional blood vessels supply to the arms and legs, 4) Rheumatic heart disease is concerned with an impairment of the heart muscle and heart valves from rheumatic fever, caused by streptococcal bacteria, 5) Congenital heart disease is a heart structure malformation, which exists at birth and 6) Deep vein thrombosis and pulmonary embolism is linked to blood clots in the leg veins that can move to the heart and lungs”.

Heart attacks and strokes are usually acute events. Also, they are mostly caused by a blockage of blood flow to the heart or brain. Since, a blood clot is an accumulation of fatty deposits on the inner walls of the blood vessels, which provide to the heart or brain. In addition, strokes are caused by losing blood from a blood vessel in the brain or from blood clots. Usually, unhealthy diet, tobacco use, obesity, less physical activity, alcohol consumption, hypertension, diabetes, and hyperlipidaemia run a risk of heart attack and stroke (WHO, 2011).

Risk factors of cardiovascular disease

There are severally potential risk factors of CVD, which are “high blood pressure (hypertension), high blood cholesterol, diabetes, smoking, shortage of exercise, overweight or obese, family history of heart disease, and ethnic background”. These risk factors are connected to cardiovascular diseases as the following;

1. High blood pressure (hypertension) is by far the most significant risk factor of CVD. It can destruct the artery walls. Also, high blood pressure induces a greater risk of blood clot development. Basically, the unit of blood pressure measurement is in millimeters of mercury (mmHg), and it is recorded as the below: systolic pressure is the blood pressure, the heart beats to pump blood out and diastolic pressure is the blood pressure, the heart rests in between beat that demonstrates the strength of arteries, while resisting to the blood flow. The normal value of blood pressure supposes to be under 130/80 mmHg.

2. Smoking also poses an important risk of CVD. The harmful chemicals in tobacco can impair and narrow coronary arteries. Therefore, smokers vulnerably expose to coronary heart disease.

3. High blood cholesterol is a fatty substance, which traveled through the blood by proteins. The combination of lipid and protein can be harmful or even protective. There are two kinds of a well-known lipoprotein that are LDL (bad cholesterol) and HDL (good cholesterol). In case of high blood cholesterol, it narrows arteries and raises the risk of blood clot. Additionally, blood test is able to show the result of cholesterol (both LDL and HDL). The recommended cholesterol level depends upon the overall risk of arterial disease.

4. Diabetes mellitus is the condition of a high blood sugar level. There are two main sorts of diabetes, which are diabetes type 1 and 2. The high level of blood glucose is related to diabetes. It results in the ruins of artery walls and it leads to grow fatty deposits (atheroma). In general, type 2 diabetes is linked to overweight or obesity.

5. Shortage of exercise can produce a high blood pressure, a greater level of cholesterol, an elevation of stress levels, and overweight. The physical activities are an alternative way of preserving a healthy heart and staying in the good shape, particularly when combines with consuming a healthy diet.

6. Overweight or obese has been found that it is related to elevate the risk of diabetes and high blood pressure as well. There are various methods that can classify health in relation to weight, but the most broadly way of measuring is body mass index (BMI). Of which can estimate whether a healthy weight for a height. Also, “waist circumference is used as an indicator of health problems such as if men have a waist circumference about 94 cm or more and women have a waist circumference about 80 cm or more, these tend to develop an obesity related to health problems”.

7. Family history of heart disease is an essential condition, which increases a chance of CVD. For instance, males tend to deal with CVD, even their age less than 55 years. While, it tends to exist in the age of 65 years in females.

8. Ethnic background has been considered as a potential risk factor of developing CVD. As claimed by the UK survey, found that “the rates of coronary heart disease are the highest in South Asian communities while, African Caribbean tends to have a chance of stroke or developing high blood pressure”. Also, compared with the rest of the population, type 2 diabetes commonly occur in African Caribbean and South Asian people.

9. Other risk factors are possible to have an influence on developing CVD as well, such as, “sex (males are more likely to have CVD at the earlier age than females), age, dietary (a high-fat die at probably causes fatty deposits, which its build up inside an arteries and lead to high blood cholesterol levels, also high blood pressure), alcohol consumption (excessive alcohol consumption possibly results in a cholesterol, blood pressure, and stress”. All these above risk factors are considered as an increase the risk of developing CVD. However, some risk factors are not easy to control or get rid of, for example, family history and ethnicity, but it is practicable to cut down on other risk factors as above and takes steps to keep the heart safe.

Furthermore, a reflection of the major forces driving social, economic and cultural change or globalization, urbanization, population aging, poverty, stress, or even hereditary can be an underling determinants of CVDs.

2.6 Carotid artery intima-media thickness (CIMT)

Lately, a surrogate image from Carotid IMT ultrasound screening is a broadly accepted as a marker of generalized atherosclerosis(Susanne Bartelsa, Angelica Ruiz

Francob, & Rundek, 2012). This technique is able to measure double-line pattern by illustrated in the two significant landmarks, where the lumen between intima, and the media layer on the near and the far wall of the carotid artery, which these were shown in the Figure 4 (Touboul, Hennerici, Desvarieux, & et, 2006). Even without the existence of atherosclerosis, the developing of age results in changes of biomechanical parameters, like blood flow and tension on the wall can elevate the intima and the media layer. The previous research claimed that CIMT was associated with subclinical atherosclerosis as it was involved in the formation of atherosclerotic plaque (C. M. Robertson, Gerry, Fowkes, & Price, 2012). Additionally, several studies have been published an association between CIMT measurements and risk of cardiovascular events (T. Z. Naqvi & M. S. Lee, 2014). The benefit of CIMT measurement by ultrasonography is applicable, convenient, non-invasive, and cost effective (Touboul et al., 2006). Additionally, CIMT technique is an interested method in research because it easily assesses vascular risk or the therapeutic effects of a specific treatment (Susanne Bartelsa et al., 2012).



Figure 4 Assessment of carotid IMT. (Bartelsa, Francob, & Rundek, 2012)

In general, mean of the IMT in the far wall of the normal carotid artery (CCA) is 0.625 ± 0.045 mm, which was measured by an automatic edge detection algorithm that represented by the yellow and purple lines (the green line in the lumen of the CCA stands for the reference value of the arterial wall echo gradient calculations).

Carotid intima-media thickness (CIMT) measurement is a practical outcome to subjects who have cardiovascular problems, since it is a non-invasive screening instrument, which provides the result by ultrasound. The initial plaque within the layers of the artery wall can be tracked down by the CIMT test, as there is enough

obstruction of blood flow to cause heart attack or stroke. Moreover, the arterial age can be calculated by an average of person's carotid artery thickness to a person of that age who is at a normal risk. In case of the arterial age is more than 5 years higher than an actual age, it can be interpreted that there is a significant increased risk of cardiovascular event.

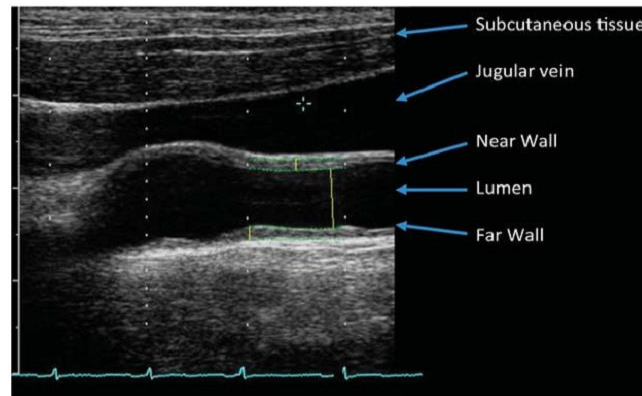


Figure 5 Image of CIMT Ultrasound scan in the distal 1 cm of the CCA. (O’Leary & Bots, 2010)

2.7 Effect of air pollutants to increased carotid artery intima-media thickness (CIMT) related to cardiovascular diseases (CVD) and stroke.

CVD is “strongly associated with increased CIMT that is a powerful intermediate phenotype of atherosclerosis plaques” (O’Leary & Bots, 2010). According to the Cardiovascular Health Study (CHS) showed “study of 4,476 subjects without clinical CVD followed for a median period of 6.2 years, using a combined 12 CCA and ICA CIMT measured”. The result of MI, the HR was 1.15. Splitting these populations into quintiles based on CIMT outcome, “CHS presented the 7-year rates for MI or stroke was over 25% for participants in the fifth quintile compared with less than 5% for those in the first quintile”(O’Leary et al., 1999). In accordance with “a systematic review and meta-analysis of 8 relevant general population-based studies that pointed the ability of CIMT to predict future CVD, which examined in 37,197 subjects followed by 5.5 years, the output revealed an absolute CIMT difference of 0.1 mm, the future risk of MI increased by 10–15%, and the stroke risk increased by 13–18%”. Presently, “over 20 cohort studies among subjects with or without previous vascular disease, and with and without CVD risk factors”. The outcome presented consistently

that “increased CIMT linked to increased CVD risk, independently of established vascular risk factors” (Lorenz, Markus, Bots, Rosvall, & Sitzer, 2007).

An average annual CIMT progression rates was 0.005–0.01 mm/year. Additionally, “the estimated rate of rate of change in CIMT was 0.0147 mm/year (95% CI: 0.0122–0.0173) in a pooled analysis of lipid lowering trials, for the mean of CCA and IMT was 0.0176 mm/year (95% CI: 0.0149– 0.0203) combined with mean maximum CCA, bulb, and internal carotid artery (ICA) IMT”. Of which these estimations reflect group averages, it explains that some participant develop rapidly than others, while some may not change at all. The axial resolution of an ultrasound unit is “between 0.1 and 0.3 mm and the error of measurement tool is consistently presented at least 5–10% of baseline CIMT”. Individually, “the CIMT measurement error prevents identification of real change over any reasonable time period” (O’Leary & Bots, 2010).

Furthermore, “a long-term exposure to fine particulate air pollution (PM_{2.5}) has been repeatedly associated with cardiovascular and ischemic heart disease” (Brook et al., 2010). Lately, Ta-Chen Su found that “the long-term exposures to traffic-related air pollution of PM_{2.5}, PM₁₀, NO₂, and NO_x were positively associated with subclinical atherosclerosis among middle-aged adults in Taiwan” (Su et al., 2015). The output indicated “the increased percentage in maximum left CIMT was 4.23% per $1.0 \times 10^{-5} \mu\text{g}/\text{m}^3$ increase in PM_{2.5}, 3.72% per $10 \mu\text{g}/\text{m}^3$ increase in PM₁₀, 2.81% per $20 \mu\text{g}/\text{m}^3$ increase in NO₂, and 0.74% per $10 \mu\text{g}/\text{m}^3$ increase in NO_x”. However, there was no an association evident for the right CIMT, and PM_{2.5} mass concentration also was not associated with the outcomes. Additionally, “a cross-sectional study among elder adults with a long term $10 \text{ mg}/\text{m}^3$ PM_{2.5} concentration were associated with a 1%–10% larger intima-medial thickness of the common carotid artery (IMT)” (Diez Roux et al., 2008). Similarly, the study of “a prospective cohort study from the Multi-Ethnic study of atherosclerosis and air pollution recommended that the higher long-term fine particulate (PM_{2.5}) concentrations are associated with increased IMT development, the greater PM_{2.5} lessening are related to slow IMT progression” (Sara D. Adar et al., 2013). Nonetheless, another prospective cohort study showed that there was no a significant association between the low level of traffic-related air pollution and the progression of carotid artery atherosclerosis (Gan et al., 2014).

Additionally, one research revealed that a long-term exposure to PM_{2.5} components at participants' homes showed the differences in CIMT, "which was associated with interquartile-range increases in sulfur, silicon. Also, OC predictions from the spatiotemporal model were 0.022 mm (95% confidence interval (CI): 0.014, 0.031), 0.006 mm (95% CI: 0.000, 0.012), and 0.026 mm (95% CI: 0.019, 0.034), respectively" (Kim et al., 2014). Another research indicated that black carbon concentration based on spatially resolved exposure estimates was associated with CIMT among elder men. These results supported "a relationship between long-term air pollution exposure and atherosclerosis" (Wilker et al., 2013).

From now on, the air pollution is a cause of morbidity, mortality, or even harmful to human health, particularly in cardiovascular and respiratory, has been a well-known issue. Recently, it has been shown that these damaging effects extend to the brain. As the previous study found that "the impact of air pollution upon the brain was the first considered as an increase in ischemic stroke (IS), which frequently found in individuals who exposed to indoor coal fumes".

However, an association between a cerebrovascular disease and an exposure to outdoor air pollutions (i.e., PM, O₃, CO, and NO₂) is still limited. The epidemiological study found that there was an association between outdoor air pollutions and an improvement of ischemic cerebrovascular events. Also, the recent reports published that there was an association between a progressions of ischemic stroke.

2.8 Possibility Potential mechanisms of cardiovascular and stroke event cause by air pollution exposures.

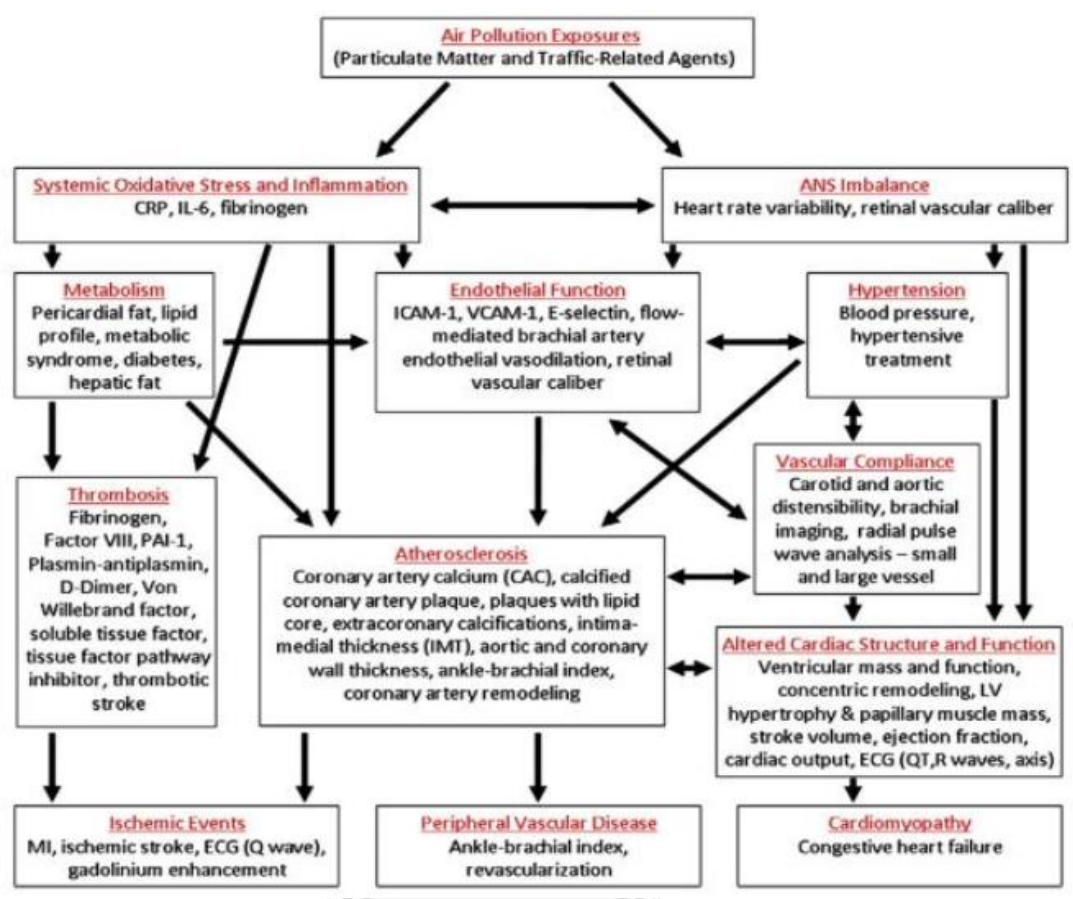


Figure 6 Potential mechanisms of cardiovascular and stroke event cause by air pollution. (Gill et al., 2011)

Edward A. Gill et al (2011) were studied Air Pollution and Cardiovascular Disease in the Multi-Ethnic Study of Atherosclerosis. They were showed Figure about physiologic pathways by which air pollution may impact on Cardiovascular Disease (CVD) which contain with potential mechanisms of ischemic stroke cause by air pollution (figure 6). This section was described about mechanisms/pathway that may impact to atherosclerosis and linked to cardiomyopathy caused by pathogenic mechanisms as follow;

1. Systemic Oxidative Stress and Inflammation

Oxidative stress occurs when there is “increased production of oxidizing species in cells and tissues, such as air pollution causes oxidative stress via both of these

pathways”. This can be enhanced when the pollutant itself is “highly oxidizing as in the case of ozone or PM_{2.5}, which contain organic chemicals, transition metals, high surface areas, all of which can contribute to local generation of reactive oxygen species and can become oxidative stressed” (Newby et al., 2015).

Pro-inflammatory and many of the pro-inflammatory genes are “oxidative stress responsive which leading to a vicious cycle that results in high levels of oxidative stress”. Chronic exposure to air pollutants can be logically related to the observed morbidity and mortality effects in a number of ways, it may cause by the pollution aggravate existing inflammatory lung disease. “Systemic inflammatory effects of cytokines or oxidizing molecules emanating from the lungs may also affect atherosclerotic plaques, leading to their progression, destabilization, or rupture, precipitating acute coronary syndrome”(Smeeth et al.).

Michelle L. Block and Lilian Calderon Garciduenas were studied Mechanisms of Neuro inflammation that make understanding well about “the effects of air pollution reach the brain”, and have also begun to indicate the cell types mediate air pollution-induced CNS pathology in recent studies (Block & Calderón-Garcidueñas, 2009). Air pollution “can impact to the vascular system, but blood vessels throughout the body display a large range of phenotypes differing in gross structure, function, cellular ultrastructure, and blood-tissue exchange properties, which may result in unique responses to air pollution, these small vessels within the brain parenchyma constitute the blood-brain barrier (BBB)”.

2. Thrombosis

Follow the study, “short-term associations between PM exposure and cardiovascular mortality suggest a rapidly inducible effector pathway such as thrombogenicity and exposure to traffic has been linked to triggering of myocardial infarction within hours”. The pathophysiological mechanisms of ischemic cerebrovascular and cardiovascular disease were similar, CVD has also been involved in the genesis of stroke (Anderson, Thundiyil, & Stolbach, 2012). In additional, “platelet activation by translocated ultrafine PM, platelets are also sensitized by mediators released into the circulation as a result of PM-induced lung inflammation” (Newby et al., 2015).

3. Atherosclerosis

Atherosclerosis is often referred to as “hardening of the arteries.” It’s the process in which credits of “fatty substances, cholesterol, cellular waste products, calcium and other substances build up in the inner lining of an artery”. This buildup is called “plaque”. The blood vessels were damaged by atherosclerotic plaque. Most of the damage occurs when “plaques become breakable and rupture cause of the blood clots that can block blood flow or break off and travel to another part of the body”. If it blocks a blood vessel that feeds the brain and heart, it causes to CVD and stroke events.

Concentration of ambient PM_{2.5} potentiates plaque burden and vascular dysfunction in murine models of atherosclerosis. “Exposure is also associated with features of plaque vulnerability, including enhanced innate immune cells, increased reactive oxygen species generating pathways and tissue factor expression” (Newby et al., 2015). The potentials risk factors of atherosclerosis and the crucial role of preexisting illness cause by PM. According, “the systemic inflammatory processes are closely linked to the pathogenesis of atherosclerosis”. Even “the first stages of vascular dysfunction are characterized by invasion of circulating macrophages into the arterial wall”. Later on, “during manifest atherosclerosis, both cellularity inside the plaques as well as systemic inflammatory status determines the risk of a plaque rupture”. In this context, “PM may accelerate systemic and local pro-inflammatory processes and thereby critically contribute to the progression of the atherogenic cascade” (Bornstadt et al., 2014).

In summary, possibility mechanisms of CVD cause by particulate matter (PM). When PM (we can call free radical or antigen) get into the body, oxidative and inflammation system have to produce the antibody for eradicate PM/ free radical/antigen and move out of the body. If we get PM over exposure, it might be imbalance for antibody production in cell and destroy the cell wall to die, it occurs on metabolism process and blood vessel that cause to thrombosis or atherosclerosis such as, coronary artery calcium (CAC), coronary artery plaque, intima-medial thickness (IMT) and wall thickness which mad the arteries that connect to the heart and the brain becoming blocked or narrowed and resulting in cardiomyopathy and stroke event.

2.9 Related Article

Table 2 Summary of particulate matter exposure increased risk of carotid artery intima-media thickness (CIMT) and carotid atherosclerotic plaques.

Reference	Particulate matter	Study Population	Study design	Results
Sara D. Adar et al, 2013	PM _{2.5}	5,362 participants	a prospective cohort study	<p>- After adjusted for confounders including age, sex, race/ethnicity, smoking, and socio-economic indicators. A mean annual IMT progression was 14 $\mu\text{m}/\text{y}$ for higher levels residential PM_{2.5} of 2.5 mg/m^3 during the follow-up period were associated with 5.0 $\mu\text{m}/\text{y}$ (95% CI 2.6 to 7.4 mm/y) greater IMT progressions among persons in the same metropolitan area.</p> <p>- All of the six areas showed positive associations. Greater reductions in PM_{2.5} over follow-up for a fixed baseline PM_{2.5} were also associated with slowed IMT progression (22.8 mm/y [95% CI 21.6 to 23.9 mm/y] per 1 mg/m^3 reduction).</p>
Matthew S Painschab et al, 2013	PM _{2.5}	266 adults aged ≥ 35 years in Puno, Peru	A cross-sectional study	<p>- Results that the biomass fuel group had greater unadjusted mean CIMT (0.66 vs 0.60 mm; $p < 0.001$), carotid plaque prevalence (26% vs 14%; $p = 0.03$), systolic blood pressure (118 vs 111 mm Hg; $p < 0.001$) and median household</p>

				<p>PM_{2.5} (280 vs 14 mg/m³; p<0.001).</p> <p>- In multivariable regression, the biomass fuel group had greater mean CIMT (mean difference=0.03 mm, 95% CI 0.01 to 0.06; p=0.02), a higher prevalence of carotid plaques (OR=2.6, 95% CI 1.1 to 6.0; p=0.03) and higher systolic blood pressure (mean difference=9.2 mm Hg, 95% CI 5.4 to 13.0; p<0.001).</p> <p>- There are associated between chronic exposure to biomass fuel was increased CIMT, increased prevalence of atherosclerotic plaques and higher blood pressure. That mean biomass fuel use as a risk factor for CVD.</p>
Rodrigo X. Armijos et al, 2014	PM _{2.5} and PM ₁₀	287 healthy children	A cross-sectional study	<p>- The children residing <100 meters from the nearest heavily trafficked road had CIMT mean and maximum measurements that were increased by 15% and 11% compared to those living = 200 meters away (<i>P</i> = 0.0001).</p> <p>- Children who resided 100–199 meters from traffic or in the middle DWTD textile also exhibited increased CIMT but these differences were not statistically significant.</p>

				<p>- This finding is important since even small increases in CIMT over time can potentially lead to earlier progression to atherosclerosis. It is also important because traffic-related pollution is potentially modifiable.</p>
Xiaole Liu et al, 2015.	PM _{2.5} and PM ₁₀	Among 56 identified studies, 11 articles satisfied the inclusion criteria.	A Systematic Review and Meta-Analysis	<p>- An increments of 10 µg/m³ in PM_{2.5}and PM₁₀were associated with an increase of CIMT (16.79 µm; 95% CI, 4.95–28.63 µm and 4.13 µm; 95% CI, -5.79–14.04 µm, respectively).</p> <p>- Exposure to PM_{2.5}had a significant association with CIMT and for women the effect may be more obvious.</p>
Ta-Chen Su et al, 2015	PM _{2.5abs} , PM ₁₀ , NO ₂ and NOx	689 volunteers 35–65 years of age	A Cross-Sectional Study	<p>- One-year average air pollution exposures were 44.21 ± 4.19 µg/ m³ for PM₁₀, 27.34 ± 5.12 µg/ m³ for PM_{2.5}, and (1.97 ± 0.36) × 10⁻⁵/m for PM_{2.5abs}.</p> <p>- The percentage increases in maximum left CIMT of 4.23% (95% CI: 0.32, 8.13) per 1.0 × 10⁻⁵/m increase in PM_{2.5abs}; 3.72% (95% CI: 0.32, 7.11) per 10- µg/ m³ increase in PM₁₀; 2.81% (95% CI: 0.32, 5.31) per 20- µg/ m³for increase in NO₂; and 0.74% (95% CI: 0.08, 1.41) per 10- µg/ m³ increase in NOx.</p>

				<ul style="list-style-type: none"> - Long-term exposures to traffic-related air pollution of $PM_{2.5}$, PM_{10}, and NO_x were positively associated with subclinical atherosclerosis in middle-aged adults.
Eline B. Provost et al, 2014.	$PM_{2.5}$	18,349 participants from eight cohorts for the cross-sectional association between CIMT and $PM_{2.5}$ 7,268 participants from three cohorts for the longitudinal analysis on CIMT progression and $PM_{2.5}$ exposure.	the Meta-Analytical Evidence	<ul style="list-style-type: none"> - The average exposure to $PM_{2.5}$ 20.8 $\mu g/m^3$ in the different study populations ranged from 4.1 to and CIMT averaged (SD) 0.73 (0.14) mm. - an increase of 5 $\mu g/m^3$ was associated with a 1.66 % (95% CI: 0.86 to 2.46; $P < 0.0001$) thicker CIMT, which corresponds to an average increase of 12.1 μm. - The combined longitudinal estimate showed for each 5 $\mu g/m^3$ exposure, a 1.04 μm per year (95% CI: 0.01 to 2.07; $P = 0.048$) greater CIMT progression. higher $PM_{2.5}$ - The meta-analysis supports the evidence of a positive association between CIMT, a marker of subclinical atherosclerosis, and long-term exposure to particulate air pollution.
An Pan et al, 2013	Incense burning inside home	63,257 Singapore Chinese 5–74 years	A cohort study	<ul style="list-style-type: none"> - 76.9% were current incense users, and most of the current users (89.9%) had burned incense daily for = 20 years.

				<p>Relative to non-current users, current users had a 12% higher risk of cardio vascular mortality [multivariable adjusted hazard ratio (HR) = 1.12; 95% CI: 1.04, 1.20]. The HR was 1.19 (95% CI: 1.03, 1.37) for mortality due to stroke and 1.10 (95% CI: 1.00, 1.21) for mortality due to coronary heart disease.</p> <ul style="list-style-type: none"> - The association between current incense use and cardiovascular mortality appeared to be limited to participants without a history of cardiovascular disease at baseline (HR = 1.16; 95% CI: 1.07, 1.26) but not linked to those with a history (HR = 1.00; 95% CI: 0.86, 1.17). - In addition, the association was stronger in never smokers (HR = 1.12; 95% CI: 1.02, 1.23) and former smokers (HR = 1.19; 95% CI: 1.00, 1.42) than in current smokers (HR = 1.05; 95% CI: 0.91, 1.22). - Long-term exposure to incense burning in the home environment was associated with an increased risk of cardiovascular mortality in the study population.
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Elissa H. Wilker et al, 2013	Black Carbon	380 participants	The Normative Aging Study	<p>- The average \pm SD age was 76 \pm 6.4 years and the mean \pm SD CIMT was 0.99 \pm 0.18 mm. A one-interquartile range increase in 1-year average black carbon (0.26 $\mu\text{g}/\text{m}^3$) was associated with a 1.1% higher CIMT (95% CI: 0.4, 1.7%) based on a fully adjusted model.</p> <p>- Annual mean black carbon concentration based on spatially resolved exposure estimates was associated with CIMT in a population of elderly men. These findings support an association between long-term air pollution exposure and atherosclerosis.</p>
Wilker EH et al, 2014	Black carbon, fine particles, nitrogen dioxide and nitric oxide.	509 participants aged 30–65 years	A prospective cohort study.	<p>- After follow-up, the differences in annual changes of these markers between these two groups were small and not statistically significant. Also, no significant associations were observed with concentrations of traffic-related air pollutants including black carbon, fine particles, nitrogen dioxide and nitric oxide.</p> <p>- This study did not find significant associations between traffic-related air pollution and progression of carotid artery atherosclerosis in a region with lower levels and smaller</p>

				contrasts of ambient air pollution.
Kim SY et al, 2014.	PM _{2.5}	5,488 Multi-Ethnic Study of Atherosclerosis participants residing in 6 US metropolitan areas	A Cross-Sectional Analysis	<p>- Long-term concentrations of PM_{2.5} components at participants' homes were predicted using both city-specific spatiotemporal models and a national spatial model.</p> <p>- The estimated differences in CIMT associated with interquartile-range increases in sulfur, silicon, and OC predictions from the spatiotemporal model were 0.022 mm (95% confidence interval (CI): 0.014, 0.031), 0.006 mm (95% CI: 0.000, 0.012), and 0.026 mm (95% CI: 0.019, 0.034), respectively.</p> <p>- Long-term concentrations of sulfur and OC, and possibly silicon, were associated with CIMT.</p>
Bauer M et al, 2010.	PM ₁₀ and PM _{2.5}	3,380 participants	Results from the HNR (Heinz Nixdorf Recall) study.	<p>Median CIMT was 0.66 mm (interquartile range 0.16 mm). An interquartile range increase in PM_{2.5} (4.2 µg/m³), PM₁₀ (6.7 µg/m³), and distance to high traffic (1,939 m) was associated with a 4.3% (95% confidence interval [CI]: 1.9% to 6.7%), 1.7% (95% CI: -0.7% to 4.1%), and 1.2% (95% CI: -0.2% to 2.6%) increase in CIMT, respectively.</p>

				<p>- There was clear association of long-term exposure to PM_{2.5} with atherosclerosis. This finding strengthens the hypothesized role of PM_{2.5} as a risk factor for atherogenesis.</p>
Nino Kunzli et al, 2010.	PM _{2.5}	1,483 participants	Cross-sectional studies	<p>- PM_{2.5} and traffic proximity were positively associated with CIMT progression. Adjusted coefficients were larger than crude associations and statistically significant for highway proximity while of borderline significance for PM_{2.5} (P = 0.08).</p> <p>- Annual CIMT progression among those living within 100 m of a highway was accelerated (5.5 micrometers/year [95%CI: 0.13–10.79; p = 0.04]) or more than twice the population mean progression. For PM_{2.5}, coefficients were positive as well, reaching statistical significance in the socially disadvantaged; in subjects reporting lipid lowering treatment at baseline; among participants receiving on-trial treatments; and among the pool of four out of the five trials.</p> <p>- This is the first study to report an association between exposure to air pollution and the</p>

				progression of atherosclerosis indicated with CIMT change in humans
Robert D. Brook et al, 2010.	PM _{2.5}		a comprehensive review of the literature for an Update to the Scientific Statement from the American Heart Association	<ul style="list-style-type: none"> - Exposure to PM_{2.5} <math>\mu\text{g}</math> in diameter (PM_{2.5}) over a few hours to weeks can trigger cardiovascular disease-related mortality and nonfatal events. - longer-term exposure (e.g., a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years. - Reductions in PM_{2.5} levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years.
Gregory A. Wellenius et al, 2012	PM _{2.5}	The 1,705 Boston area patients hospitalized	The time-stratified case-crossover study design to assess the association between the risk of ischemic stroke	<ul style="list-style-type: none"> - The estimated odds ratio (OR) of ischemic stroke onset was 1.34 (95% CI, 1.13-1.58) (P<.001) following a 24-hour period classified as moderate (PM_{2.5} 15-40 $\mu\text{g}/\text{m}^3$) by the U.S.EPA - The estimated odds ratio of ischemic stroke onset to be 1.11 (95% CI, 1.03-1.20) PM_{2.5} levels (6.4 $\mu\text{g}/\text{m}^3$).

			onset and PM _{2.5} concentrations.	- The increase in risk was greatest within 12 to 14 hours of exposure to PM _{2.5} most strongly associated with markers of traffic-related pollution.
Oudin A, Stromberg U, Jakobsson K and Stroh E, Bjork J, 2009.	PM ₁₀	Both first-time (N = 8,142) and recurrent (N = 2,982) strokes were included in the study.	case-crossover analysis Time series analysis and time-stratified	- An increase in risk of ischemic stroke was observed when levels of PM ₁₀ were above 30 µg/m ³ , compared with PM ₁₀ levels below 15 µg/m ³ , RR was 1.13 (95% CI: 1.04-1.22). - Daily mean temperature also associated with ischemic stroke; a decrease in risk when temperatures were above 16°C, RR of 0.88 (95% CI: 0.77-1.00).
Ravi Maheswaran, Tim Pearson and Nigel C. Smeeton et al, 2012.	PM ₁₀	1,832 ischemic and 348 hemorrhagic strokes in 1,995 to 2004	a small-area level ecological study design	- A resident population concentration was 25.1 (1.2) µg/m ³ (range, 35.4 –68.0 µg/m ³ (range, 23.3–36.4 µg/m ³) for PM ₁₀ . - For ischemic stroke, adjusted rate ratios per 10-µg/m ³ increase, for all ages, 40 to 64 and 65 to 79 years, respectively, were 1.22 (0.77–1.93), 1.12 (0.55–2.28), and 1.86 (1.10 – 3.13) for PM ₁₀ . - For hemorrhagic stroke, the corresponding rate ratios were 0.52 (0.20 –1.37), 0.78 (0.17– 3.51), and 0.51 (0.12–2.22) for PM ₁₀ .

Manuel A. Leiva G et al, 2013.	PM _{2.5}	Santiago reported 33,624 stroke admissions between January 1, 2002 and December 30, 2006	Time series study	<ul style="list-style-type: none"> - PM_{2.5} concentration was markedly seasonal, <u>increasing during the winter</u>. - This study found an association between PM_{2.5} exposure and hospital admissions for stroke; for every PM_{2.5} concentration increase of 10 µg/m³, the risk of emergency hospital admissions for Cerebrovascular causes increased by 1.29% (95% CI 0.552%-2.03%).
Martin J. O'Donnell et al, 2011.	PM _{2.5}	9,202 patients hospitalized with acute ischemic stroke	<p>Mixed method</p> <ul style="list-style-type: none"> - A time-stratified case-crossover design. - matching - Meta-analysis. 	<ul style="list-style-type: none"> - PM_{2.5} was associated with a -0.7% change in ischemic stroke risk per 10 µg/m³ increase in PM_{2.5} (95% CI = -6.3% to 5.1%). - PM_{2.5} Was associated with an 11% increase in ischemic stroke risk (1% to 22%) among patients with diabetes mellitus. - The association between PM_{2.5} and ischemic stroke risk, with the strongest associations observed for strokes due to large artery atherosclerosis and small vessel occlusion.

CHAPTER III

METHODOLOGY

3.1 Study design

A one-year cohort study was conducted in adults aged ≥ 35 years old who spent the majority of his/her time inside the house (>8 hours) and residing in the central city of Sakon Nakhon province, Thailand. The participants in each house were selected by purposive sampling and divided into three groups according to their long-term history of household incense use which is a common practice among this community; non-exposed group, non-daily exposed group and daily exposed group. The participants were Thai-Vietnamese. All household were located beside the main road of the central city to minimize outdoor air pollution effects. From June to August 2016, trained health volunteers conducted a face-to-face interview by standard questionnaire to evaluate the pattern of household incense burning, demographic status, and household characteristics. The presence of CVD, diabetes, hypertension, respiratory problems and dyslipidemia were assessed by a self-report during interview. Particulate matter concentrations (PM_{10}), temperature, and humidity which related to incense burning were measured inside participants' house. All participant were measured CIMT by ultrasound scanner and investigated some clinical assessment (Provost, Madhloum, Int Panis, De Boever, & Nawrot, 2015) in September 2016 and September 2017. This study protocol was approved by the institutional review board of Chulalongkorn university ethics committee for research involving human health subjects (COA No. 146/2016). A total of 150 participants were recruited at baseline. Eighteen individuals did not agree to perform a carotid ultrasound examination and clinical assessments. It remained 132 participants (88% of those recruited) with completed the data at baseline (phase 1). After a year of follow-up, only 100 residents were completed the data collection (phase 2). The loss of follow-up rate was 24%. All participants gave their written consent form before involving in the study.

3.2 Study Area

The central city of Sakon Nakhon Province was chosen purposively to be a study area because of a high rate of patients admitted to hospital and a high death rate. Specific causes of mortality were ischemic heart disease and stroke. Furthermore, burning incense inside house differentiate people behavior between the one living in the central city and the one living in other areas of this province in term of their occupation and ethnicity. Additionally, Sakon Nakorn province was selected to study area for minimizing an effect of traffic-pollutants on CIMT (Armijos et al., 2015). (Figure 7)



Figure 7 Topography map of the study area, the central city of Sakon Nakhon Province, Thailand.

3.3 Study Population

The people who lived in the central city of Sakon Nakhon province, Thailand.

3.4 Samples and Sample size

Study participants

We stratified participants by their long-term history of household incense use into 3 groups, namely,

1. Non-exposed group were selected from participants who had never burned incense inside their houses.
2. Non-daily exposed group were selected from participants who burned incense as normal practice occasionally burning incense; < 5 days per week.
3. Daily exposed group were selected from participants who were burned incense ≥ 5 days per week.

Sample size calculation

The n4 Studies version 1.4.1 was used to sample size calculation. Two independent means (two-tailed test) was selected to calculate the sample size of exposure and non-exposure group because this study was to find a mean difference between independent groups

From previous study about chronic exposure to biomass fuel (in term of indoor particulate matter) is associated with increased carotid artery intima-media thickness (CIMT) among people living in the city of Puno, Peru. The result found that the mean of CIMT was 0.66 mm. (SD 0.13) in biomass fuel group and 0.60 mm (SD 0.12) in clean fuel group. There are difference between biomass fuel and clean fuel group as statistical significantly ($p < 0.001$) (Painschab et al., 2013). The sample size calculation by n4Studies program as figure 8

Testing two independent means

Formula[ref]:
$$n_1 = \frac{(z_{1-\frac{\alpha}{2}} + z_{1-\beta})^2 [\sigma_1^2 + \frac{\sigma_2^2}{r}]}{\Delta^2}$$

$$r = \frac{n_2}{n_1}, \Delta = \mu_1 - \mu_2$$

Mean in group1 (μ_1) = 0.66

Mean in group2 (μ_2) = 0.60

SD. in group1 (σ_1) = 0.13

SD. in group2 (σ_2) = 0.12

Ratio (r) = 1

Alpha (α) = 0.01

Beta (β) = 0.1

Calculate **Clear**

Sample size:
group1 = 69, group2 = 69

Figure 8 sample size calculation on n4Studies program (Bernard, R. (2000) & Ngamjarus C., Chongsuvivatwong V. (2014)

Therefore, the whole sample in this study supposed to be 138 participants from calculation. However, the researcher added 10 percent for loss to follow-up so, the final number of participants were 150 participants.

Sampling Technique

Study area was purposively selected. These three group of participants were invited by inclusion and exclusion criteria from people who lived beside the main road in the central city of Sakon Nakhon province. If there were more than 1 participant in household who met criteria, selected participant in that household was obtained by using simple random sampling. The sampling procedure was shown in figure 9

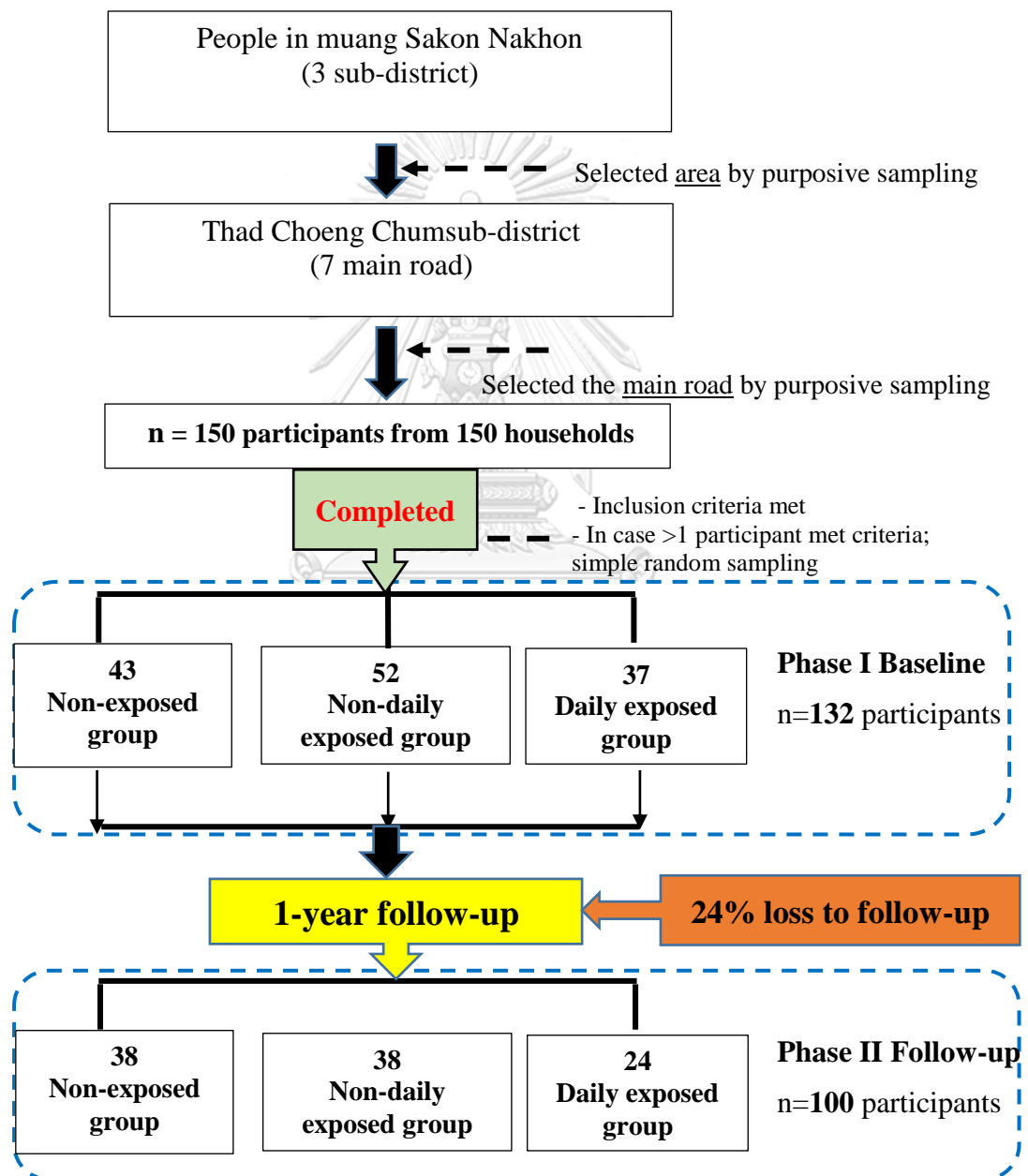


Figure 9 Sampling Technique.

3.5 Inclusion and exclusion criteria

Table 3 Inclusion and exclusion criteria

Criteria	Non-exposed	Non-daily exposed	Daily-exposed
Inclusion criteria			
1) Male and female aged ≥ 35 years	✓	✓	✓
2) No incense burning inside home and unrelated to incense stick	✓		
3) Have incense burning inside home < 5 day per week		✓	
4) Have incense burning inside home ≥ 5 day per week (Navasumrit et al., 2008)			✓
5) Live beside of main road in the central city of Sakon Nakhon.	✓	✓	✓
6) Register and lived in the same house as Sakon Nakhon province resident more than 5 years (Brook et al., 2010).	✓	✓	✓
7) Duration of spending time at home more than 8 hours/day (Wellenius et al., 2012).	✓	✓	✓
8) Willing to participate and to collect the air sampling in their home.	✓	✓	✓
Exclusion criteria			
1) Electronic incense use	✓	✓	✓
2) Pregnant	✓	✓	✓

3.6 Measurement Tools

Measurement tools in this study were used questionnaires and indoor air monitoring measurement in participant's home. Carotid ultrasound test for CIMT, medical laboratory test and hemodynamics were measured at Sakon Nakhon hospital. The details of method were used list as follow:

3.6.1 Face to face questionnaire interviewed

Face to face questionnaire interviewed were applied to all participants in this study. The questionnaire developed by researcher for evaluation the pattern of socio-demographic, household characteristics and incense burning behavior of all participants at the beginning of the study. All participants were requested to complete a questionnaire by face to face interview to find factors that might be risk of the increased CIMT. The questionnaires were consisted all factors which could affect to biomarker levels. There were 3 parts as following; (appendix A)

Part 1. Socio- demographics of participant included, age, gender, Body Mass Index (BMI), congenital disease, family history of CVD and stroke disease, level of education, occupational, smoking status, alcohol consumption and physical activity.

Part 2. Home characteristics included, type of resident, fuel cooking use, and mosquito repellent used.

Part 3. Incense used; were asked for condition of incense using at home included, type of incense, number of incense used per day, duration of incense used, history of incense used and location of use.

Validity and reliability of questionnaire

The validity was considered by 3 experts in the major of environmental and public health. "An evaluation using the index of item-objective congruence; IOC (Rovinelli & Hambleton, 1977) was a process where content experts rate individual items on the degree to which they do or do not measure specific objectives listed by the test developer". A content expert was evaluated each item by giving the item a rating of 1 (for clearly measuring), -1 (clearly not measuring), or 0 (degree to which it measures the content area was unclear) for each objectives. Index of Item Objective Congruence (IOC) score was over 0.5.

3.6.2 Measurement of household particulate matter concentration

There were various pollutants from incense smoke inside house. This study was focused on indoor particulate matter less than 10 micrometer (PM_{10}) that might cause to increase CIMT (Lin et al., 2008). It was monitored continuously in 24 hours. Relative humidity and temperature were recorded during indoor PM_{10} collection at home.

Household PM_{10} concentrations, temperature and relative humidity (RH) were collected inside participants' homes during the dry season (November-December 2016) and wet season (June-July 2017). Household PM_{10} samples were collected continuously for 24 hours following the National Institution's Occupational Safety And Health Guideline (NIOH, 1998). Briefly, a personal sampling pump (SKC 224-PCXR8 model) connected with aluminum cyclone (SKC model 37 mm- Cat No. 225-01-02) was calibrated before and after the sampling period to set a flow-rate at 2.5 L/min. The polyvinyl chloride filters (37 mm, 5.0 micrometer pore size, SKC Inc. USA) were pre- and post- weighed at controlled room conditions. The device was placed in a box together with a HOBO® tempt/RH data logger (Onset devices, Pocasset, MA). The data logger was programmed to detect, record temperature and RH every five minutes for 24 hours. A box of devices was placed by a researcher, in the middle of a room where participants spent most of their time each day at a height of 1-1.5 m above the floor. An average of temperature and RH were reported. (Figure 10)



(a)

(b)

Figure 10 Indoor particulate matter (PM_{10}), temperature and relative humidity measurements. (a) Calibrator, Personal sampling air pump and a HOBO® tempt/RH data logger (b) A box of devices placed at participant's home

Validity and reliability of tools

Particulate monitors were calibrated before and after collecting the sample in each time which follow by guidelines for air sampling and analytical method development and evaluation (NIOSH, 1994).

3.6.3 Clinical assessments

Measurement of Carotid Intima Media Thickness (CIMT) was a clinical assessment for defined the thickness of common carotid artery (CCA). All of participants were measured CIMT at baseline and followed after one year. The difference of thickness in CCA were provided as a result of this study. “Measurement of carotid intima-media thickness (CIMT) was reasonable for cardiovascular risk assessment in asymptomatic adults at intermediate risk and published recommendations on required equipment, technical approach, and operator training and experience for performance of the test must be carefully followed to achieve high-quality results” (Nishimura RA et al., 2014).

CIMT measurements followed by the American College of Cardiology (ACC) and the American Heart Association (AHA) guidelines on the assessment of cardiovascular risk (Goff et al., 2013). The CIMT measurement was performed using a high resolution B-mode ultrasound scanner (Toshiba Aplio 300). An adult cardiac 1.8-4.8 MHz linear array transducer with Micro-convex was utilized. We exported the images for offline viewing using Synapse PD-S Viewer Version 1.0. This method is a well-validated, inexpensive, non-invasive surrogate marker of both current and future coronary artery disease and atherosclerosis. Thickness was assessed as both the mean and maximum of the anterior capturing the media-adventitia interface of far arterial walls (Christine M Robertson, Fowkes, & Price, 2012). This was validated against histological specimens as representative of the true thickness of the vessel wall (T. Z. Naqvi & M.-S. Lee, 2014), as well as 10 millimeter manual measurements to the bulb from the common carotid on both right and left common carotid arteries. The mean of CIMT and maximum of CIMT in both the right common carotid arteries (RCCA) and left common carotid arteries (LCCA) for each participant were averaged to present the overall mean of CIMT and maximum of CIMT (Painschab et al., 2013). Each

participant's CIMT levels were measured three times. Their anonymized data was then submitted to a radiologist who produced an average rating of the 3 measurements for each participant's incense exposure criteria. Radiologist were blinded for CIMT diagnosis of incense exposed and non-exposed participants. (Figure 11)

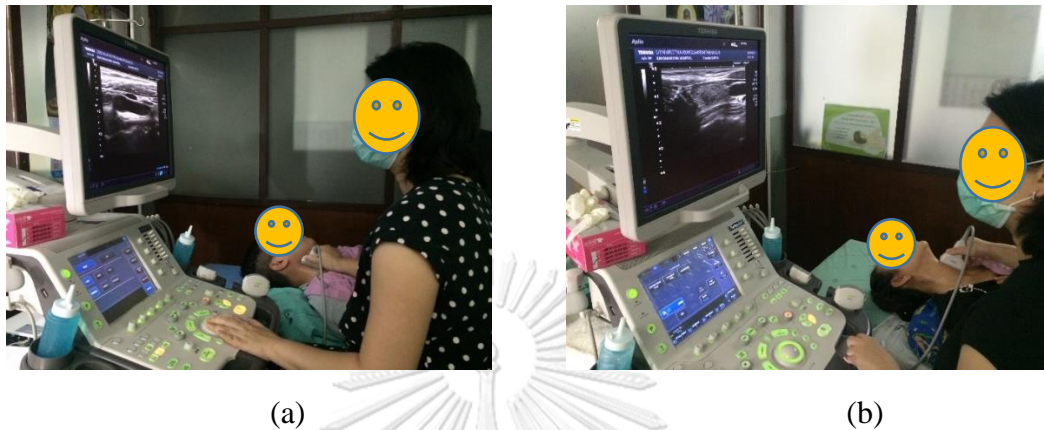


Figure 11 Carotid Intima-Media Thickness (CIMT), (a) CIMT at left common carotid artery (LCCA), (b) CIMT at right common carotid artery (RCCA)

Blood samples analysis was the confounding factors of CVD that may increase CIMT. It was collected from all participants with followed by a clinical assessment for test (Goff et al., 2013; Rosvall et al., 2015). The test was included, total cholesterol, high density lipoprotein cholesterol (HDL), low density lipoprotein cholesterol (LDL), triglyceride (TG), hemoglobin A1c, high sensitivity C-reactive protein (hs C-RP).

Blood samples were collected from all participants which followed by ACC/AHA Prevention Guideline, 2013 (Goff et al., 2013). All blood samples were analyzed immediately in Sakon Nakhon hospital medical laboratory. Laboratory researchers were blinded for blood analysis of exposed and non-exposed participants. (Figure 12)



Figure 12 Laboratory blood sample

Hemodynamics test was measured; 1) heart rate 2) systolic blood pressure (SBP) and 3) diastolic blood pressure (DBP). Hemodynamics were measured by OMRON blood pressure monitor with automatic cuff inflation and deflation was used to measure blood pressure and heart rates according to standard protocol. Heart rate, systolic blood pressure and diastolic blood pressure were reported. Each reported measurement were presented the value of blood pressure by nurse. Nurses was blinded for hemodynamics measurement of exposed and non-exposed participants. (Figure 13)



Figure 13 Blood pressure monitoring

A clinical assessment included, CIMT, blood test and hemodynamics were set as health check-up program for all participants in this study and analyzed immediately in Sakon Nakhon hospital medical laboratory for 2 times, at baseline (September, 2016) and 1-year follow-up (September, 2017).

3.7 Data Collection

All participants who eligible for study inclusion were asked for their willing to participate in the study during June 2016 to September 2017. Data were collected as followed figure 14.

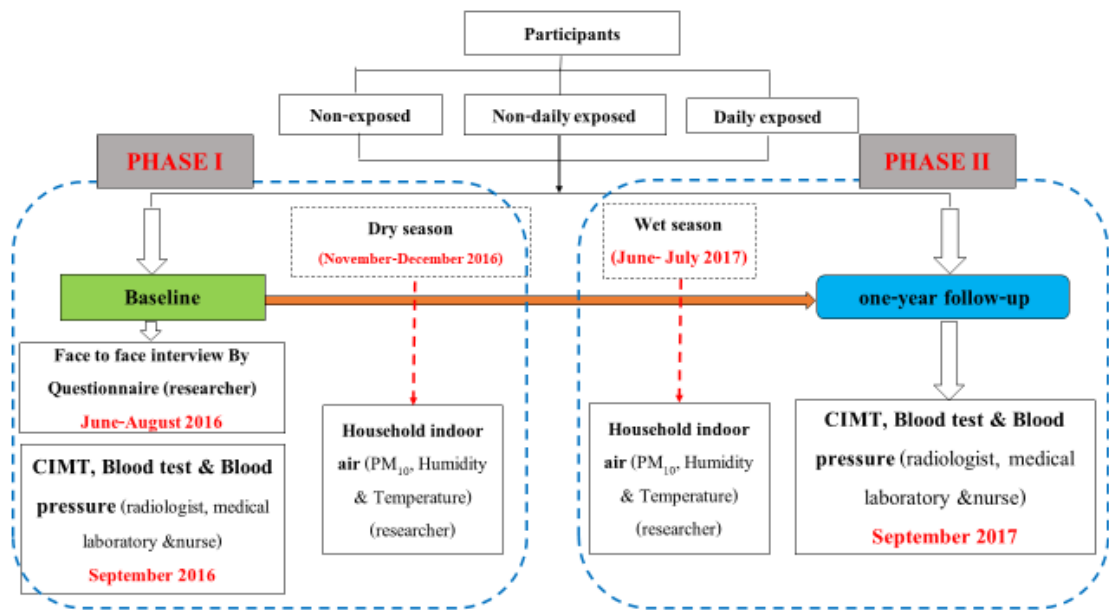


Figure 14 the data collection.

3.7.1 Field study

All participants were conducted by face to face interview using questionnaire to obtain personal characteristics, home characteristics and incense used characteristics. The questionnaire was collected at the beginning of data collection on June 2016. (Appendix A)

3.7.2 Household particulate matter assessment

Air sampling of incense smoke inside of participants' home were conducted by seasonality because there were different of air concentration in each season within year (Leiva, Santibanez, Ibarra, Matus, & Seguel, 2013). Therefore, the represent data for indoor air concentration should be average by seasonality. In this study, we were measured air samples in dry season (November-December 2016) and wet season (June-July 2017). After air sampling, samples were capped, transported to the laboratory for analysis with followed by guidelines for air sampling and analytical method development and evaluation (NIOH, 1998).

3.7.3 Clinical assessment collection

All participants were received check-up health program included, carotid ultrasound test, blood test and hemodynamics by physician of Sakon Nakhon hospital at baseline and followed-up for one year in September 2016 and September 2017.

Carotid artery ultrasound for IMT

1. Participants were placed in a supine position on scan bed with head resting comfortably and slightly rotated neck in direction opposite to probe.
2. Use 45-degree angle wedge pillow to help standardize lateral rotation (Stein et al., 2008)
3. The CIMT measurements were performed after participant had rested quietly for 10–15 minutes.
4. The thickness were reported measurement by radiologist.

Blood test and Hemodynamics

1. All participants were requested to measure their weight and height to obtain body mass index (BMI) using an automatic balance before collecting blood test.
2. Blood pressure and heart rates were measured using an OMRON blood pressure monitor with automatic cuff inflation and deflation, reported measurement by nurse with heart rate, systolic blood pressure and diastolic blood pressure by average of three time measurements.
3. Then, all participants were determined blood samples followed by a clinical assessment for laboratory test. Blood samples were used 5 milliliters for analysis, total cholesterol, triglyceride, high-density lipoprotein (HDL), low-density lipoprotein (LDL), haemoglobin A1c and high sensitivity C-reactive protein (hs-CRP).
4. All blood samples of participant were collected for analysis immediately in a standardized medical laboratory of Sakon Nakhon hospital by professional technicians.

3.8 Data analysis

Particulate matter concentration

The particulate matter concentration, especially repairable dust, was calculated by the below equation from NIOSH- method 0600 (NIOH, 1998).

$$C = \frac{(w_2 - w_1) - (B_2 - B_1)}{V(L)} \times 1000 \text{ mg/m}^3$$

Where: C = concentration of particulate matter (mg/m³)

W₁ = tare weight of filter before sampling (mg)

W_2 = post-sampling weight of sample-containing filter (mg)

B_1 = mean tare weight of blank filters (mg)

B_2 = mean post-sampling weight of blank filters (mg)

V = Air volume as sampled at flow (m^3)

Analytical statistics

The main outcome from this study was that incense burning does increase the risk of developing CIMT. All analyses were performed using IBM's SPSS Statistical Software for Windows (IBM SPSS, version 22, Chicago, IL, USA). The p-value below 0.05 defined statistical significance.

For the baseline characteristics of participants, continuous variables were expressed as mean \pm standard deviation (SD) and median (Interquartile range; IQR). Categorical variables were presented as number and percentage (%). Categorical variables were presented by percentage (percent; %). We used Chi-square (χ^2) or Fisher's exact tests for categorical data as appropriate. Analysis of variance (ANOVA) was performed to compare differences amongst the 3 exposure groups if normally distributed and Kruskal Wallis test were used if non-normally distributed. An independent t-test was analyzed to compare the difference between 2 exposure groups, indoor environment parameters between wet and dry season and the socio-demographic of participants between baselines to follow-up. The paired t-test was analyzed to compare the difference of CIMT between baseline and follow-up.

Multivariate linear regression was used to examine the association between exposure to incense smoke and CIMT at baseline adjustments for age, hypertension, cardiovascular disease, cholesterol, hs-CRP and heart rate. Covariate factors in the model were selected by recommended factors by the American Heart Association guidelines for CIMT (Greenland et al., 2010; Rosvall et al., 2015) and had a p-value of less than 0.2 in the bivariate analysis.

Multivariable logistic regression was used to estimate the risk of exposure to incense smoke and household PM_{10} exposure on an increase of CIMT (yes/no) after a one-year follow-up. We used the 75th percentile of annual change of mean CIMT (0.05 mm) and maximum CIMT (0.08 mm) both left and right of CCA to identify

participants with increased (yes) and not increased (no) of CIMT. For PM_{10} , the association was reported for $1 \mu\text{g}/\text{m}^3$ increase of an average concentration between the wet and the dry seasons. Covariate factors in the model were selected by recommended factors by the American Heart Association guidelines for CIMT (Greenland et al., 2010; Rosvall et al., 2015) and had a p-value of less than 0.2 in the bivariate analysis; including age, gender, HDL and SBP.

3.9 Ethical Consideration

This study protocol was approved by Institutional review boards (IRBs) of the Ethic Review Committee for Research Involving Human Research Subjects, Health Science Group, Chulalongkorn University (COA No. 146/2016; Date of approval: 15 August 2016). All participants were asked to provide written in the consent form prior to participate at the beginning of this study.

CHAPTER IV

RESULT

A one-year cohort study was conducted in adults aged ≥ 35 years old residing in the central city of Sakon Nakhon province, Thailand. The participants were selected by purposive sampling and divided into three groups according to their long-term history of household incense use; non-exposed group (participants who had never burned incense inside their houses), non-daily exposed group (participants who burned incense as normal practice occasionally burning incense; < 5 days per week) and daily exposed group (participants who were burned incense ≥ 5 days per week). During the data collection period, a total of 150 participants were recruited at baseline. Eighteen individuals did not agree to perform a carotid ultrasound examination and clinical assessments. It remained 132 participants (88% of those recruited) with completed the data at baseline (phase 1). After a year of follow-up, only 100 residents were completed the data collection (phase 2). Thirty two participants were loss to follow-up (24%). All participants were interviewed by questionnaires and underwent a clinical assessment, blood laboratory analyses and a carotid artery ultrasound. The household indoor air environment which related to incense burning, particulate matter concentrations (PM₁₀), temperature and relative humidity (RH), were measured inside participants' house. The result of the present study could show as following;

4.1 Phase I: Baseline characteristics

4.1.1 General information of participants

4.1.1.1 Socio-demographic characteristic

Table 4 presents general characteristic of participant in baseline. We carried out on 132 residents in the central city of Sakon Nakhon province. All participants completed a face-to-face interview, provided a blood sample and underwent CIMT measurements. The majority of the study population was female (75.0%) and median reported age (IQR) was 56(12) years old. After stratification by incense exposure assessment criteria, 32.6% of participants were in the non-incense exposed group

(n=43; median age 57(11) years), 39.4% were in the non-daily incense exposed group (n=52; median age 53(14) years) and 28.0% were placed in the daily incense exposed group (n=37; median age 60(10) years). The age was significantly different amongst the 3 groups but gender was similar. The average of Body mass index (BMI) was 23.7 (± 3.2) kg/m². The highest of BMI was found in daily incense exposed group (24.1 \pm 3.2 kg/m²). Most of them were finished high school (56.1%). In term of current occupational, 75.8% were merchant and it was found significantly association amongst the 3 groups (p=0.002).

According to cardiovascular risk factors, the daily incense exposed group had a higher self-report history of hypertension than others. As for the member history of cardiovascular and stroke disease was 16.7%. In the non-daily incense exposed group had highest an alcohol consumption (19.2%) while the non-exposed group were higher of smoking status (4.7%) and physical activity (81.4%) than others. However, it was not showed an association between cardiovascular risk factors and incense used (p>0.05).

Table 4 Baseline characteristics of the participants according to incense exposure (n=132)

Demographic	Total (n=132)	non- exposed (n=43)	non-daily exposed (n=52)	daily exposed (n=37)	p-value
Age (years) , median(IQR)	56(12.0)	57(11.0)	53(14.0)	60(10.0)	0.025* ^a
Gender, n (%)					0.833 ^c
Male	33(25.0%)	11(25.0%)	14(26.9%)	8(22.2%)	
female	99(75.0%)	33(75.0%)	38(73.0%)	28(77.8%)	
Body Mass Index (kg/m ²), mean \pm SD	23.7 \pm 3.2	23.5 \pm 3.4	23.7 \pm 2.9	24.1 \pm 3.2	0.622 ^b
Education, n (%)					0.119 ^c
Uneducated	29(22.0%)	6(14.0%)	14(26.9%)	9(24.3%)	
High school	73(56.1%)	22(51.2%)	29(55.8%)	22(62.2%)	
Bachelor and more	29(22.0%)	115(34.9%)	9(17.3%)	5(13.9%)	

Table 4.1 (Continue.)

Demographic	Total (n=132)	non- exposed (n=43)	non-daily exposed (n=52)	daily exposed (n=37)	p-value
Current occupation					0.002* ^c
Agricultural / Laborer/ Housewife	25(18.9%)	13 (30.2%)	8(15.4%)	4(10.8%)	
Merchant and trader	100(75.8%)	24(55.8%)	43(82.7%)	33(89.2%)	
Government / Company employee	7(5.3%)	6(14.0%)	1(1.9%)	0(0%)	
Past Medical, n (%)	54(40.9%)	12(27.9%)	23(44.2%)	19(51.4%)	0.086 ^c
Diabetes	10(7.6%)	3(7.0%)	4(7.7%)	3(8.1%)	0.967 ^d
Hypertension	30(22.7%)	5(11.6%)	13(25%)	12(32.4%)	0.076 ^c
Cardiovascular	5(3.8%)	0(0%)	3(5.8%)	2(5.4%)	0.269 ^d
Respiratory	7(5.3%)	1(2.3%)	4(7.7%)	2(5.4%)	0.553 ^d
Dyslipidemia	14(10.6%)	4(9.3%)	6(11.5%)	4(10.8%)	1.00 ^d
Member history of cardiovascular and stroke disease, n (%)	22 (16.7%)	7(16.3%)	8 (15.4%)	7 (18.9%)	0.904 ^c
Smoking, n (%)	4(3.0%)	2(4.7%)	1(1.9%)	1(2.7%)	0.825 ^d
Alcohol consumption, n (%)	20(15.2%)	6(14%)	10(19.2%)	4(10.8%)	0.532 ^c
Physical activity, n (%)	100 (75.8%)	35(81.4%)	36(69.2%)	29(78.4%)	0.352 ^c

* p<0.05

^aKruskal wallis test, ^bOneway-ANOVA, ^cChi-square (χ^2), ^dFisher's exact test

4.1.1.2 Household characteristic

Most of participant's home were shop house (56.8%), it was highest in daily incense exposed group (75.7%). The participants were used biogas fuel cooking in home (86.4%), highest in in daily incense exposed group (91.9%) and using of mosquito repellent was 20.0%. Type of resident was significantly different amongst the 3 groups (p=0.006) however, fuel cooking use in home and mosquito repellent use were not association with incense exposure (Table 5).

Table 5 Household characteristic of the participants according to incense exposed

Household characteristic	Total (n=132)	non-exposed (n=43)	non-daily exposed (n=52)	daily exposed (n=37)	p-value
Type of resident					0.006 ^{d*}
detached house	46 (34.8%)	24 (55.8%)	16 (30.8%)	6 (16.2%)	
Town house	6 (4.5%)	2 (4.7%)	2 (3.8%)	2 (5.4%)	
rented room	5 (3.8%)	1 (2.3%)	3 (5.8%)	1 (2.7%)	
Shop house	75 (56.8%)	16 (37.2%)	31 (59.6%)	28 (75.7%)	
Fuel cooking use in home					
Biogas fuel	114 (86.4%)	36 (83.7%)	44 (84.6%)	34 (91.9%)	0.059 ^c
biomass fuel	25 (18.9%)	9 (20.9%)	13 (25.0%)	3 (8.1%)	0.124 ^c
Electric stove	18 (13.6%)	8 (18.6%)	5 (9.6%)	5 (13.5%)	0.446 ^c
Microwave	35 (26.5%)	11 (25.6%)	12 (23.1%)	12 (32.4%)	0.607 ^c
Mosquito repellent, n (%)	29 (20.0%)	6 (14.0%)	12 (23.1%)	11 (29.7%)	0.229 ^c

* p<0.05

^cChi-square (χ^2), ^dFisher's exact test

4.1.1.3 Incense use characteristics

More than half of the non-daily exposed and daily exposed participants used a long incense stick (length > 20 cm; 64.0%) and burned incense at less 5 sticks (65.2%) each time. 92.1% of them spent more than 30 minutes for burning incense each time and 82% had used incense for more than 14 years. When they burning incense, most of them burned incense in semi-open room (33.3%), sometime they sat closely with incense burning (34.8%) and smelled to incense smoke (40.2%). Type of burning room, sit closely with incense burning and smell to incense smoke were significantly different amongst those 3 group however, type of incense, amount of burned incense sticks durations of burned incense sticks and years exposed to incense smoke were not showed any association (Table 6).

Table 6 Incense use behavior among non-daily incense exposed and daily incense exposed groups (n=89)

Household characteristic	Total (n=89)	non-daily exposed (n=52)	daily exposed (n=37)	p-value
Type of incense				.310 ^b
Short stick (length ≤ 20 cm)	32(36.0%)	18(34.6%)	14(37.8%)	
Long stick (length ≥ 20 cm)	57(64.0%)	34(65.4%)	23(62.2%)	
Number of burned incense sticks in each time (sticks)				.394 ^c
min-max = 3-30				
mean±SD = 6.9±5.4				
< 5 sticks	31(34.8%)	20(38.5%)	11(29.7%)	
≥ 5 sticks	58(65.2%)	32(61.5%)	26(70.3%)	
Durations of burned incense sticks in each time (minutes)				.100 ^d
min-max = 15-180				
mean±SD = 47.5±32.5				
< 30 minutes	7(7.9%)	4(7.7%)	3(8.1%)	
≥ 30 minutes	82(92.1%)	48(92.3%)	34(91.9%)	
Years exposed to incense smoke (years)				.450 ^e
min-max = 5-50				
mean±SD = 18.7±9.5				
< 14 years	16(18.0%)	8(15.4%)	8(21.6%)	
≥ 14 years	73(82.0%)	44(84.6%)	29(78.4%)	
Type of burning room				<0.001 ^{**c}
Open room	19 (14.4%)	13 (25.0%)	6 (16.2%)	
Semi-open room	44 (33.3%)	23 (44.2%)	21 (56.8%)	
Close room (one entrance)	26 (19.7%)	16 (30.8%)	10 (27.0%)	

Table 6 (Continued)

Household characteristic	Total (n=89)	non-daily exposed (n=52)	daily exposed (n=37)	p-value
Stay in the room during burning incense				<0.001 ^{**c}
Always	11 (8.3%)	6 (11.5%)	5 (13.5%)	
Sometimes	46 (34.8%)	28 (53.8%)	18 (48.6%)	
Never	32 (24.2%)	18 (34.6%)	14 (37.8%)	
Smelling to incense smoke				<0.001 ^{**c}
Always	26 (19.7%)	14 (26.9%)	12 (32.4%)	
Sometime	53 (40.2%)	31 (59.6%)	22 (59.5%)	
Never	10 (7.6%)	7 (3.5%)	3 (8.1%)	

* p<0.05, ** p<0.001

^bOneway-ANOVA, ^cChi-square (χ^2), ^dFisher's exact test

4.1.1.4 Clinical assessment (Blood parameters and hemodynamics) of participants at baseline

Table 7 shows blood test and hemodynamics at baseline. Mean reported the total cholesterol (\pm SD) and low density lipoprotein cholesterol (LDL) (\pm SD) were 212.3(\pm 36.1) mg/dl and 139.9(\pm 35.6) mg/dl respectively. Median reported triglyceride (IQR), high density lipoprotein cholesterol (HDL) (IQR), hemoglobin A1c (IQR) and high sensitivity C-reactive protein (hs-CRP) (IQR) were 101.0(91.8) mg/dl, 58.5(24.8) mg/dl, 5.3(0.5) mg/dl and 1.4(1.9) mg/l respectively.

Blood parameters indicated higher total cholesterol, HDL, LDL, hemoglobin A1c and hs-CRP levels in the non-exposed group than other groups. Triglyceride had higher in non-daily incense exposed group than others. Regarding to hemodynamics, heart rate, systolic blood pressure (SBP) and diastolic blood pressure (DBP) were 73.4 \pm 9.7 beats/min, 132.3 \pm 20.4 mmHg and 80.41 \pm 12.06 mmHg respectively. It was higher in non-daily incense exposed group than other. However, we found that the Hs-CRP level and heart rate were significantly different among the three groups (p<0.05).

Table 7 Blood test parameters and hemodynamics in non-exposed, non-daily exposed and daily exposed participants at baseline and follow-up

Blood test	Incense smoke exposure				p-value
	Total (n=132)	non-exposed (n=43)	non-daily exposed (n=52)	daily exposed (n=37)	
Total cholesterol, mean \pm SD, mg/dl.	212.3 \pm 36.1	220.6 \pm 33.4	208.8 \pm 37.2	207.5 \pm 36.7	0.182 ^b
Triglyceride, median (IQR), mg/dl.	101.0(91.8)	88.0(56.0)	119.0(11.3)	103(63.0)	0.359 ^a
HDL, median (IQR), mg/dl.	58.5(24.8)	60.0(20.0)	55.0(22.5)	59.0(24.0)	0.41 ^a
LDL, mean \pm SD, mg/dl.	139.9 \pm 35.6	147.4 \pm 38.1	135.6 \pm 33.5	137.2 \pm 34.9	0.237 ^b
Heamoglobin A1c, median (IQR), mg/dl.	5.3(0.5)	5.4(0.6)	5.2(0.6)	5.2(0.5)	0.537 ^a
Hs-CRP, median(IQR), mg/l.	1.4(1.9)	2.1(2.7)	1.4(1.7)	0.9(1.3)	0.045* ^a
Heamodynamics					
Heart rate, mean \pm SD, beats/min	73.4 \pm 9.7	70.9 \pm 9.1	75.9 \pm 10.2	72.8 \pm 9.1	0.039* ^b
Systolic blood pressure (SBP), mean \pm SD, mm Hg	132.3 \pm 20.4	130.6 \pm 21.8	135.0 \pm 20.3	130.7 \pm 18.8	0.482 ^b
Diastolic blood pressure (DBP), mean \pm SD, mm Hg	80.4 \pm 12.1	78.6 \pm 12.9	82.4 \pm 11.8	79.8 \pm 12.3	0.295 ^b

* p<0.05, ^aKruskal wallis test, ^bOneway-ANOVA

4.1.1.5 Carotid Intima Media Thickness (CIMT) and incense exposed group at baseline

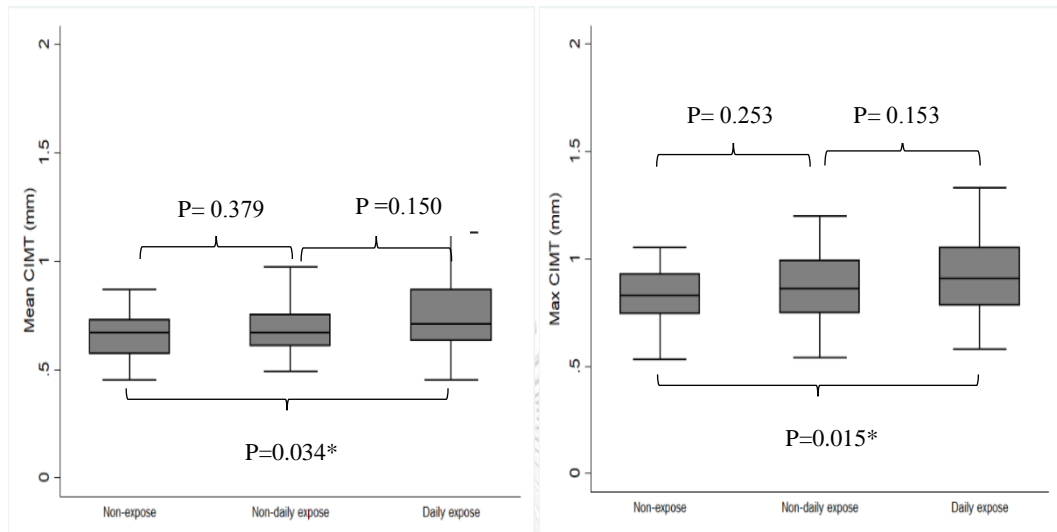
The daily incense exposed group had the greatest mean CIMT of common carotid artery (CCA) (mean \pm SD: 0.75 \pm 0.18 mm) and combined left and right maximum CIMT of CCA (mean \pm SD: 0.92 \pm 0.2 mm). The combined left and right mean CIMT of CCA (mean \pm SD: 0.69 \pm 0.13 mm) and maximum CIMT of CCA (mean \pm SD: 0.87 \pm 0.17

mm) of the non-daily exposed group were also greater than the non-exposed group, combined mean CIMT of CCA and combined maximum CIMT of CCA were 0.66 ± 0.14 mm and 0.83 ± 0.13 mm respectively. (Table 8) Significant differences of combined mean and max CIMT among those three groups were achieved (mean CIMT $p=0.034$; maximum CIMT $p=0.015$) (Figure 15). A comparison of the CIMT of the left common carotid artery (LCCA) and right common carotid artery (RCCA) among these 3 groups found that mean CIMT of LCCA (mean \pm SD: 0.75 ± 0.23 mm) and maximum CIMT of LCCA (mean \pm SD: 0.9 ± 0.273 mm) in the daily incense exposed group were greater than mean CIMT of LCCA (mean \pm SD: 0.68 ± 0.14 mm) and maximum CIMT of LCCA (mean \pm SD: 0.85 ± 0.18 mm) in the non-daily exposed group and non-exposed group (mean CIMT of LCCA (mean \pm SD: 0.64 ± 0.11 mm) and maximum CIMT of LCCA (mean \pm SD: 0.80 ± 0.14 mm). CIMT of LCCA was significantly different among the 3 groups of exposure (mean CIMT of LCCA $p=0.006$; maximum CIMT of LCCA $p=0.017$) (Figure 4.2). It was similar to mean and maximum CIMT of RCCA. The daily incense exposure group had the greatest mean CIMT of RCCA (mean \pm SD: 0.74 ± 0.21 mm) and maximum CIMT of RCCA (mean \pm SD; 0.91 ± 0.25 mm). The non-daily exposed group were higher mean CIMT of RCCA (mean \pm SD: 0.70 ± 0.14 mm) and maximum CIMT of RCCA (mean \pm SD; 0.88 ± 0.18 mm) than the non-exposed group, mean CIMT of RCCA (mean \pm SD: 0.67 ± 0.13 mm) and maximum CIMT of RCCA (mean \pm SD; 0.85 ± 0.16 mm) (Table 8). However, significant difference was not achieved amongst the three groups of exposure (Figure 16).

Table 8 Mean and maximum CIMT among incense exposed group at baseline.

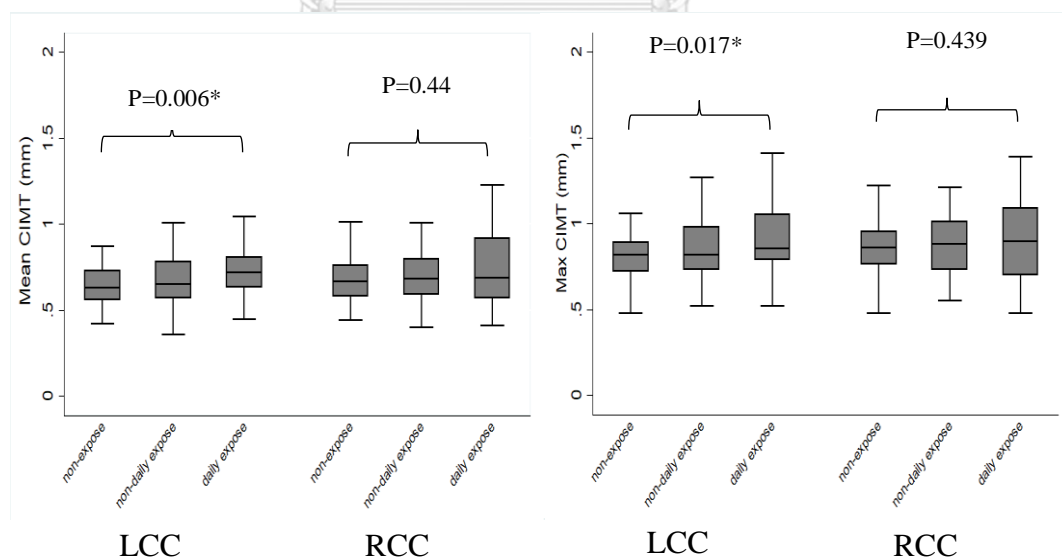
Carotid Intima Media Thickness (mm)	Incense exposure			
	Total (n=132)	non-exposed (n=43)	non-daily exposed (n=52)	daily exposed (n=37)
CCA, mean	0.69 ± 0.14	0.66 ± 0.10	0.69 ± 0.13	0.75 ± 0.18
maximum	0.87 ± 0.17	0.83 ± 0.13	0.87 ± 0.17	0.92 ± 0.2
RCCA, mean	0.70 ± 0.16	0.67 ± 0.13	0.70 ± 0.14	0.74 ± 0.21
maximum	0.87 ± 0.19	0.85 ± 0.16	0.88 ± 0.18	0.91 ± 0.25
LCCA, mean	0.69 ± 0.16	0.64 ± 0.11	0.68 ± 0.14	0.75 ± 0.23
maximum	0.86 ± 0.19	0.80 ± 0.14	0.85 ± 0.18	0.93 ± 0.23

Figure 15 Combined left and right mean CIMT and combined left and right maximum CIMT among the non-exposed, non-daily exposed, and daily exposed groups at baseline (n=132)



*independent t-test

Figure 16 Mean and maximum CIMT of left common carotid artery (LCCA) and right common carotid artery (RCCA) stratified by group (non-exposed, non-daily exposed, and daily exposed) at baseline (n=132)



* Oneway-ANOVA

4.1.2 An association of incense smoke exposure and increased Carotid Intima Media Thickness (CIMT) of all participants at baseline

In multivariable regression, in the daily exposure was significantly association with increased mean CIMT of CCA (mean difference = 0.05 mm; 95%CI 0.013, 0.087) and maximum CIMT of CCA (mean difference= 0.092 mm; 95%CI 0.018, 0.166) were greater than other group. The mean CIMT of LCCA (mean difference=0.110 mm; 95%CI 0.042, 0.178) and maximum CIMT of LCCA (mean difference=0.060 mm; 95%CI 0.019, 0.101) also found a significantly association with daily incense exposure.

After adjustments for age, self-reported hypertension, self-reported CVD, total cholesterol, hs-CRP and heart rate, the combined left and right mean CIMT of the daily exposed group (mean difference = 0.04 mm; $p = 0.016$) and non-daily exposed group (mean difference = 0.032 mm; $p = 0.037$) were greater than the non-exposed group. The combined left and right maximum CIMT of the daily exposed group (mean difference = 0.076 mm; $p = 0.025$) and non-daily exposed group (mean difference = 0.064 mm; $p = 0.039$) were greater than for the non-exposed group. Additionally, mean CIMT of LCCA of the daily exposed group (mean difference = 0.102 mm; $p = 0.002$) and non-daily exposed group (mean difference = 0.064 mm; $p = 0.031$) were greater than in the non-exposed group. The maximum CIMT on LCCA was found to be the same association as the mean CIMT of LCCA. However, mean and maximum CIMT of RCCA did not show any associations to household incense exposure (Table 9).

Table 9 Multivariate linear regression model for mean and maximum carotid intima-media thickness (CIMT) at baseline (n=132)

CIMT (mm)	non-exposed	non-daily exposed		Daily exposed	
	Ref.	β	95% CI	β	95% CI
Unadjusted model					
CCA, mean	Ref. category	0.017	-0.017, 0.051	0.050	0.013, 0.087*
maximum	Ref. category	0.036	-0.032, 0.104	0.092	0.018, 0.166*
RCCA, mean	Ref. category	0.015	-0.026, 0.055	0.034	-0.010, 0.078
maximum	Ref. category	0.023	-0.057, 0.103	0.056	-0.030, 0.143
LCCA, mean	Ref. category	0.035	-0.027, 0.098	0.110	0.042, 0.178*
maximum	Ref. category	0.021	-0.017, 0.060	0.060	0.019, 0.101*
Adjusted model^a					
CCA, mean	Ref. category	0.032	0.002, 0.063*	0.040	0.008, 0.073*
maximum	Ref. category	0.064	0.003, 0.126*	0.076	0.011, 0.142*
RCCA, mean	Ref. category	0.027	-0.012, 0.065	0.019	-0.022, 0.060
maximum	Ref. category	0.044	-0.032, 0.119	0.028	-0.053, 0.109
LCCA, mean	Ref. category	0.064	0.006, 0.123*	0.102	0.04, 0.165**
maximum	Ref. category	0.041	0.006, 0.076*	0.058	0.020, 0.096*

^aAdjusted for factor associated to CIMT in adult: age, hypertension (yes/no), CVD (yes/no), total cholesterol, hs-CRP, and heart rate.

* p<0.05, ** p<0.001

4.1.3 Household indoor air parameters (Particulate matter less than 10 μm (PM₁₀), relative humidity (RH) and temperature ($^{\circ}\text{C}$) in wet and dry seasons

4.1.3.1 Household indoor air parameter in dry seasons

In dry season, median reported particulate matter less than 10 μm (PM₁₀) (IQR) was 27.6(24.6) $\mu\text{g}/\text{m}^3$, it was highest in non-daily exposed group (29.4(27.3) $\mu\text{g}/\text{m}^3$). Mean temperature ($\pm\text{SD}$) and relative humidity ($\pm\text{SD}$) were 28.9(\pm 1.9) $^{\circ}\text{C}$ and 57.2(\pm 5.2)% respectively. PM₁₀ concentration and temperature were significantly different between the 3 groups (p<0.049 and p<0.001) (Table 10).

Table 10 Household indoor particulate matter assessments of the participants according to incense exposed group at baseline (n=132)

Particulate matter (Dry season)	Incense exposure				p-value
	Total (n=132)	non-exposed (n=43)	non-daily exposed (n=52)	daily exposed (n=37)	
PM ₁₀ (µg/m ³), median(IQR)	27.6(24.6)	21.5(22.6)	29.4(27.3)	28.3(23.9)	0.049 ^{*a}
Temperature (°C), mean±SD	28.9±1.9	26.2±1.6	26.8±1.6	27.9±2.1	<0.001 ^{*b}
Relative humidity (RH) (%), mean±SD	57.2±5.2	57.5±5.7	56.9±5.5	57.0±4.1	0.854 ^b

* p<0.05, ^aKruskal wallis test, ^bOneway-ANOVA

4.1.3.2 Household indoor air parameter in wet season

In wet season, we found median of PM₁₀ (IQR) was 16.1(11.9) µg/m³, highest in non-daily exposed group (17.7(8.8) µg/m³) and in the daily-exposed group (median (IQR): 16.1(11.6) µg/m³). The average of temperature (median (IQR) was 28.9(1.0)°C which highest in the non-daily exposed group (median (IQR): 29.2(0.9)°C). For RH average was 73.2(6.7) %, highest in non-exposed group (median (IQR): 74.0(6.4). However, they were no any significantly different among those three groups of incense users (Table 11).

Table 11 Household indoor particulate matter assessments of the participants according to incense exposed group at 1-year follow-up (n=100)

Particulate matter (Wet season)	Incense smoke exposure				p-value
	Total (n=100)	non-exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
M ₁₀ (µg/m ³), median(IQR)	16.1(11.9)	12.9(12.1)	17.7(8.8)	16.1(11.6)	0.168 ^a
Temperature (°C), median(IQR)	28.9(1.0)	28.8(1.0)	29.2(0.9)	28.8(1.1)	0.314 ^a
Relative humidity (%), median(IQR)	73.3(6.7)	74.0(6.4)	72.5(6.8)	72.5(9.2)	0.242 ^a

* p<0.05, ^aKruskal wallis test

4.1.3.3 An average of household indoor air parameters in dry and wet season

One year average of PM₁₀ (average of wet and dry season) (\pm SD) was 24.2 \pm 11.4 μ g/m³, which was highest in non-daily exposed group (mean \pm SD: 26.2 \pm 11.3 μ g/m³). The mean of temperature (\pm SD) was 27.9(\pm 1.2) °C, highest in daily exposed group (mean \pm SD: 28.5 \pm 1.2 °C) at significantly difference amongst these three group (p=0.003). The average of relative humidity (RH) (mean \pm SD) was 65.3 \pm 3.9%, it was highest in non-exposed group (mean \pm SD; 66.1 \pm 4.0 %) (Table 12).

Table 12 Average of household indoor particulate matter assessments of the participants according to incense exposed group (n=100)

Average of particulate matter (dry and wet season)	Incense smoke exposure				p-value
	Total (n=100)	non-exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
PM ₁₀ (μ g/m ³), mean \pm SD	24.2 \pm 11.4	21.6 \pm 10.7	26.2 \pm 11.3	25.2 \pm 12.4	0.190 ^b
Temperature (°C), mean \pm SD	27.9 \pm 1.2	27.5 \pm 1.0	27.9 \pm 1.2	28.5 \pm 1.2	0.003 ^{*b}
Relative humidity (RH) (%), mean \pm SD	65.3 \pm 3.9	66.1 \pm 4.0	64.7 \pm 3.9	64.7 \pm 3.8	0.252 ^b

* p<0.05, ^bOneway-ANOVA

4.1.3.4 Comparison of household particulate matter between dry and wet season

Table 13 shows a significant difference of PM₁₀ concentration, temperature and humidity between wet and dry season. Household indoor particulate matter assessments of the participants, the average of PM₁₀ (mean \pm SD) was 24.2 \pm 11.4 μ g/m³, the average of temperature (mean \pm SD) was 27.9 \pm 1.2 °C and the average of RH (mean \pm SD) was 65.3 \pm 3.9%. We found PM₁₀ in dry season (mean \pm SD; 31.4 \pm 18.2) higher than wet season (mean \pm SD; 17.1 \pm 8.8) while temperature (mean \pm SD; 28.9 \pm 1.1 °C) and relative humidity (RH) (mean \pm SD; 72.7 \pm 5.2%) in wet season were higher than dry season.

Additional, we found that there were strongly significant different between household indoor PM₁₀, temperature and RH in dry and wet season ($p < 0.001$).

Table 13 Household indoor assessment of the participants (n=100)

Parameters	Average	Dry season	Wet season	P-value (Dry & Wet)
		(n=100)	(n=100)	
PM ₁₀ ($\mu\text{g}/\text{m}^3$), mean \pm SD	24.2 \pm 11.4	31.4 \pm 18.2	17.1 \pm 8.8	<0.001**
Temperature ($^{\circ}\text{C}$), mean \pm SD	27.9 \pm 1.2	26.9 \pm 2.0	28.9 \pm 1.1	<0.001**
Relative Humidity (%), mean \pm SD	65.3 \pm 3.9	57.9 \pm 5.8	72.7 \pm 5.2	<0.001**

** $p < 0.001$, Paired t-test

4.2 Phase II: 1-year follow-up (increased of CIMT)

During the data collection period, a total of 132 participants from baseline and remained of 100 residents who completed for the data collection after a year of follow-up. Thirty-two participants loss to follow-up (24%), 11.6% were loss from the non-incense exposure group (n=5), 26.9% were loss from the non-daily incense exposed group (n=14) and 35.1% were loss from the daily incense exposed group (n=13). Most of them were female (84.4%) with mean age (\pm SD) was 55.81(\pm 7.2). All characteristics of participants (age, gender, BMI, educational, occupational, past medical, CVD history, smoking status, alcohol consumption and physical activity were no any difference between baseline and follow-up) who loss to follow-up were no any association with incense exposure (APPENDIX C).

4.2.1 General characteristics of participants according to incense exposed group at 1-year follow-up

Socio-demographic of participants at 1-year follow-up

At follow-up characteristics, we carried out on 100 residents in the central city of Sakon Nakhon province. All participants provided a blood sample and underwent CIMT measurements. The majority of the study population was female (75.0%) and median reported age (IQR) was 57.5(13) years old. After one-year follow-up, the non-

incense exposure group and the non-daily incense exposed group were equal to 38.0% of participants (n=38; median age 57(9) years and n=38; median age 53(17) years) and 24.0% were placed in the daily incense exposed group (n=24; median age 60(8) years). The age was significantly (p=0.015) different amongst the 3 groups but gender was similar. Mean reported the average of Body Mass Index (BMI) (\pm SD) was 23.6 (\pm 3.2), it was nearly level of BMI in those three group of incense exposed. Most of them were finished high school (54.0%). In term of current occupational, 79.0% were merchant/trader however there were no any association between BMI, education, occupational and incense exposure. According to cardiovascular risk factors, most of them were highest with hypertension (22.0%) especially, in the non-daily incense group (26.3%). Member history of cardiovascular and stroke disease were 18.0%, highest in the daily-incense exposed group (20.8%). Other cardiovascular risk factors included, smoking status, alcohol consumption and physical activity were average 1.0%, 15.0% and 77.0% respectively (Table 14).

Table 14 Characteristics of the participants according to incense exposure at 1-year follow-up (n=100)

Demographic	Incense smoke exposure				p-value
	Total (n=100)	non- exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
Age (years) , median(IQR)	57.5(13.0)	57(9)	53(17)	60(8)	0.015* ^a
Gender, n (%)					0.232 ^c
Male	25(25.0%)	8(21.1%)	13(34.2%)	4(16.7%)	
female	75(75.0%)	30(78.9%)	25(65.8%)	20(83.3%)	
Body Mass Index, mean \pm SD)	23.6 \pm 3.2	23.3 \pm 3.4	23.5 \pm 2.9	24.2 \pm 3.1	0.491 ^b
Education, n (%)					0.514 ^c
Uneducated	22(22.0%)	8(21.1%)	10(26.3%)	4(16.7%)	
High school	54(54.0%)	18(47.4%)	20(52.6%)	16(66.7%)	
Bachelor and more	24(24.0%)	12(31.6%)	8(21.1%)	4(16.7%)	
Current occupation					0.141 ^d
Agricultural / Laborer/ Housewife	16(16.0%)	9(23.7%)	4(10.5%)	3(12.5%)	
Merchant and trader	79(79.0%)	25(65.8%)	33(86.8%)	21(87.5%)	
Government / Company employee	5(5.0%)	4(10.5%)	1(2.6%)	0(0.0%)	

Table 14 (Continued)

Demographic	Incense smoke exposure				p-value
	Total (n=100)	non- exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
Past Medical, n (%)	45(45.0%)	13(34.2%)	19(50.0%)	13(54.2%)	0.225 ^c
Diabetes	10(10.0%)	3(7.9%)	4(10.5%)	3(12.5%)	0.913 ^d
Hypertension	22(22.0%)	6(15.8%)	10(26.3%)	6(25.0%)	0.498 ^c
Cardiovascular	4(4.0%)	0(0%)	2(5.3%)	2(8.3%)	0.179 ^d
Respiratory	8(8.0%)	2(5.3%)	4(10.5%)	2(8.3%)	0.734 ^d
Dyslipidemia	11(11.0%)	5(13.2%)	4(10.5%)	2(8.3%)	0.922 ^d
Member history of cardiovascular and stroke disease, n (%)	18(18.0%)	6(15.8%)	7(18.4%)	5(20.8%)	0.878 ^c
Smoking, n (%)	1(1.0%)	0(0%)	0(0%)	1(4.2%)	0.240 ^d
Alcohol consumption, n(%)	15(15.0%)	6(15.8%)	8(21.1%)	1(4.2%)	0.190 ^c
Physical activity, n(%)	77(77.0%)	30(78.9%)	28(73.7%)	19(79.2%)	0.827 ^c

* p<0.05,

^aKruskal wallis test, ^bOneway-ANOVA, ^cChi-square (χ^2), ^dFisher's exact test, ^eT-test

Comparison the socio-demographic of participants at baseline to follow-up

The socio-demographic of participants at baseline to follow-up. We carried out on 132 residents at baseline and 100 residents at follow-up. For baseline to follow-up, the majority of the study population was female and mean reported age (\pm SD) were 56 \pm 12.0 and 57.5 \pm 13 years old. Mean reported the average of Body Mass Index (BMI) (\pm SD) was equal as baseline to follow-up. Most of them were finished high school and careered on trader. According to past medical, most of them were highest with hypertension and dyslipidemia. However, all characteristics of participants included; age, gender, BMI, educational, occupational, past medical, CVD history, smoking status, alcohol consumption and physical activity were no any difference between baseline and follow-up (Table 15).

Table 15 Comparison the characteristics of the participants at baseline to follow-up

Demographic of participants	Incense exposed groups		p-value
	Baseline (n=132)	Follow-up (n=100)	
Age (years), mean \pm SD	56 \pm 12.0	57.5 \pm 13.0	0.955 ^e
Gender, n (%)			0.789 ^c
Male	31(23.5%)	25(25.0%)	
female	101(76.5%)	75(75.0%)	
Body Mass Index (kg/m ²), mean \pm SD	23.7 \pm 3.2	23.58 \pm 3.2	0.74 ^e
Education, n (%)			0.929 ^c
Uneducated	29(22.0%)	22(22.0%)	
High school	74(56.1%)	54(54.0%)	
Bachelor and more	29(22.0%)	24(24.0%)	
Current occupation			0.833 ^c
Agricultural / Laborer/ Housewife	7(5.3%)	5(5.0%)	
Merchant and trader	25(18.9%)	16(16.0%)	
Government / Company employee	100(75.8%)	79(79.0%)	
Past Medical, n (%)			0.533 ^c
Diabetes	10(7.6%)	10(10.0%)	0.515 ^c
Hypertension	30(22.7%)	22(22.0%)	0.895 ^c
Cardiovascular	5(3.8%)	4(4.0%)	0.934 ^c
Respiratory	7(5.3%)	8(8.0%)	0.408 ^c
Dyslipidemia	14(10.6%)	11(11.0%)	0.924 ^c
Member history of cardiovascular and stroke disease, n (%)	22(16.7%)	18(18%)	0.79 ^c
Smoking, n (%)	6(4.5%)	1(1.0%)	0.119 ^d
Alcohol consumption, n (%)	20(15.2%)	15(15.0%)	0.975 ^c
Physical activity, n (%)	100(75.8%)	77(77.0%)	0.826 ^c

^cChi-square (χ^2), ^dFisher's exact test, ^eindependent t-test

Clinical assessments of participants at 1-year follow-up

Table 16 shows the blood test level and hemodynamics at 1-year follow-up period. Mean reported the average of total cholesterol (\pm SD) were 210.9(\pm 37.0) mg/dl and LDL (\pm SD) were 142.3(\pm 35.6) mg/dl. Median reported of the average of triglyceride (IQR) were 114.0(84.3) mg/dl, HDL (IQR) 60.0(26.0) mg/dl, hemoglobin A1c (IQR) 5.4(0.6) mg/dl and for hs-CRP (IQR) 1.3(1.8) mg/l. The results indicated that total cholesterol, HDL, LDL, hemoglobin A1c levels and hs-CRP were higher in the non-exposed group than others while triglyceride had higher in non-daily incense exposed group. Regards to hemodynamics, median reported the average of heart rate (IQR), systolic blood pressure (SBP) (IQR) and diastolic blood pressure (DBP) (IQR) were 75.0(13.8) beats/min, 126.5(22.5) mm Hg and 76.0(14.8) mm Hg respectively. Heart rate and SBP were higher in daily exposed group while DBP was higher in non-daily incense exposed group. However, there were no any significantly different among those three groups of incense users.

Table 16 Blood test parameters and hemodynamics in non-exposed, non-daily exposed and daily exposed participants at follow-up (n=100)

Clinical assessments	Incense smoke exposure				p-value
	Total (n=100)	non- exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
Total cholesterol, mean \pm SD, mg/dl.	210.9 \pm 37.0	217.53 \pm 41.4	205.1 \pm 29.5	209.4 \pm 40.1	0.34 ^b
Triglyceride, median (IQR), mg/dl.	114.0(84.3)	103.5(83.3)	121.5(89.0)	108.0(81.0)	0.557 ^a
HDL, median (IQR), mg/dl.	60.0(26.0)	64.0(28.5)	56.0(15.3)	61.5(29.0)	0.112 ^a
LDL, mean \pm SD, mg/dl.	142.3 \pm 35.6	149.3 \pm 40.2	138.3 \pm 28.7	137.4 \pm 37.5	0.305 ^b
Heamoglobin A1c, median (IQR), mg/dl.	5.4(0.6)	5.4(0.6)	5.4(0.5)	5.3(0.7)	0.765 ^a
Hs-CRP, median(IQR), mg/l.	1.3(1.8)	1.5(1.6)	1.2(2.3)	1.4(1.3)	0.801 ^a

Table 16 (Continued)

Clinical assessments	Incense smoke exposure				p-value
	Total (n=100)	non- exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
Phase 2: Follow-up					
Heamodynamics					
Heart rate,					0.365 ^a
median(IQR), beats/min	75.0(13.8)	71.0(11.0)	75.0(12.0)	78.0(19.8)	
Systolic blood pressure,					0.448 ^a
median(IQR), mm Hg	126.5(22.5)	124.5(25.5)	125.5(15.0)	132.0(28.3)	
Diastolic blood					0.948 ^a
pressure, median(IQR), mm Hg	76.0(14.8)	76.0(15.8)	76.5(15.5)	76.0(13.5)	

^aKruskal wallis test, ^bOneway-ANOVA

Carotid Intima Media Thickness (CIMT) and incense exposed group at 1-year follow-up

After 1 year follow-up, we found the daily incense exposed group had the greatest combined left and right mean CIMT of common carotid artery (CCA) (mean±SD; 0.80±0.19 mm) and combined left and right maximum CIMT of CCA (mean±SD; 1.00±0.20 mm). Mean CIMT of CCA (mean±SD; 0.70±0.13 mm) and maximum CIMT of CCA (mean±SD; 0.89±0.16 mm) of the non-daily exposed group were also greater than the non-exposed group (mean CIMT of CCA (mean±SD; 0.71±0.13 mm) and maximum CIMT of CIMT (mean±SD; 0.89±0.16 mm). There was significantly different among incense exposure and mean CIMT at CCA (p=0.022) but not for maximum CIMT at CCA.

When comparing the CIMT of RCCA, we found the daily incense exposure group had the greater mean CIMT (mean±SD; 0.71±0.17 mm) and maximum CIMT (mean±SD; 0.89±0.19 mm) than mean and maximum CIMT of RCCA in the non-daily exposed group (mean±SD; 0.67±0.15 mm and 0.85±0.21 mm respectively) and the mean and maximum CIMT in non-exposed group were mean±SD; 0.69±0.15 mm and mean±SD; 0.87±0.20 mm respectively. However, we could observe a significantly

difference amongst the three groups of exposure and mean CIMT of RCCA ($p=0.037$) but, not for maximum RCCA.

For the CIMT of the left common carotid artery (LCCA), mean and maximum CIMT of LCCA in the daily incense exposed group (mean \pm SD; 0.80 ± 0.22 mm and mean \pm SD; 1.02 ± 0.24 mm respectively) were greater than the non-daily exposed (mean CIMT of LCCA (mean \pm SD; 0.77 ± 0.14 mm; maximum CIMT of LCCA (mean \pm SD; 0.88 ± 0.16 mm) and non-exposed group (mean CIMT (mean \pm SD; 0.71 ± 0.15 mm and maximum CIMT (mean \pm SD; 0.91 ± 0.19 mm). Both mean and maximum CIMT of LCCA were significantly different among these 3 group of incense exposed ($p=0.039$ and $p=0.027$) (Table 17).

Table 17 Mean and maximum of carotid intima-media thickness (CIMT) according to incense exposed group at 1-year follow-up. (n=100)

Carotid Intima Media Thickness (CIMT)	Incense exposure				p-value
	Total (n=100)	non- exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
CCA, mean	0.73 ± 0.15	0.71 ± 0.13	0.70 ± 0.13	0.80 ± 0.19	0.022^{*a}
maximum	0.92 ± 0.18	0.89 ± 0.16	0.89 ± 0.16	1.00 ± 0.20	0.052^a
RCCA, mean	0.75 ± 0.17	0.72 ± 0.15	0.72 ± 0.15	0.82 ± 0.19	0.037^{*a}
maximum	0.94 ± 0.19	0.92 ± 0.18	0.92 ± 0.19	1.01 ± 0.21	0.108^a
LCCA, mean	0.73 ± 0.17	0.71 ± 0.15	0.77 ± 0.14	0.80 ± 0.22	0.039^{*a}
maximum	0.92 ± 0.20	0.91 ± 0.19	0.88 ± 0.16	1.02 ± 0.24	0.027^{*a}

* P-value < 0.05, ^aOneway ANOVA

4.2.2 Increased of Carotid intima-media thickness (CIMT) from baseline to follow-up

Comparison of increased CIMT from baseline to follow-up

Table 18 shows that follow-up CIMT was higher than baseline CIMT ($p<0.001$). At baseline, mean reported the thickness of carotid intima-media at CCA (\pm SD) were $0.71 (\pm 0.15)$ mm and maximum CIMT at CCA (\pm SD) were $0.89 (\pm 0.18)$ mm. Then after 1-year follow-up, the mean and maximum CIMT (\pm SD) were 0.73

(± 0.15) and $0.92 (\pm 0.18)$ mm respectively. For left and right CCA, we observed that baseline of mean LCCA (mean \pm SD; 0.70 ± 0.18 mm) and maximum LCCA (mean \pm SD; 0.87 ± 0.21 mm) were less than mean RCCA (mean \pm SD; 0.72 ± 0.17 mm) and maximum RCCA (mean \pm SD; 0.89 ± 0.19 mm). After a year of follow-up, we still found that mean LCCA (mean \pm SD; 0.73 ± 0.17 mm) and maximum LCCA (mean \pm SD; 0.92 ± 0.20 mm) were less than mean RCCA (mean \pm SD; 0.75 ± 0.17 mm) and maximum RCCA (mean \pm SD; 0.94 ± 0.19 mm). Additionally, we also found a strongly significant difference between CIMT at baseline and follow-up ($p < 0.001$). Overall increment of CIMT, we found that after 1-year follow-up was greater than CIMT at baseline.

Table 18 An increased Carotid intima-media thickness (CIMT) from baseline to follow-up (n=100)

Carotid Intima Media Thickness (CIMT)	Total		p-value
	Baseline	Follow-up	
CCA, mean	0.71 ± 0.15	0.73 ± 0.15	$<0.001^{**}$
maximum	0.89 ± 0.18	0.92 ± 0.18	$<0.001^{**}$
RCCA, mean	0.72 ± 0.17	0.75 ± 0.17	$<0.001^{**}$
maximum	0.89 ± 0.19	0.94 ± 0.19	$<0.001^{**}$
LCCA, mean	0.70 ± 0.18	0.73 ± 0.17	$<0.001^{**}$
maximum	0.87 ± 0.21	0.92 ± 0.20	$<0.001^{**}$

** $p < 0.001$, Paired t-test

Comparison of CIMT among the three groups of incense exposed at baseline to 1-year follow-up.

Table 19 shows the comparison of carotid intima-media thickness (CIMT) between baseline and 1-year follow-up in each group of incense exposed. We found that after 1-year follow-up, mean and maximum of all CIMT in the non-exposure, the non-daily exposure and the daily exposure were higher than CIMT at baseline.

Table 19 Comparison of mean and maximum of carotid intima-media thickness (CIMT) according to incense exposed group at baseline to 1-year follow-up. (n=100)

CIMT level (±SD)	non-exposed		non-daily exposed		daily exposed	
	Baseline	Follow-up	Baseline	Follow-up	Baseline	Follow-up
CCA, mean	0.69 (±0.13)	0.71 (±0.13)	0.68 (±0.13)	0.70 (±0.13)	0.78 (±0.19)	0.80 (±0.19)
maximum	0.87 (±0.16)	0.90 (±0.16)	0.85 (±0.17)	0.90 (±0.16)	0.97 (±0.21)	1.00 (±0.19)
RCCA, mean	0.69 (±0.15)	0.73 (±0.15)	0.69 (±0.15)	0.72 (±0.15)	0.78 (±0.12)	0.82 (±0.19)
maximum	0.88 (±0.17)	0.92 (±0.18)	0.86 (±0.19)	0.92 (±0.19)	0.97 (±0.24)	1.01 (±0.21)
LCCA, mean	0.68 (±0.15)	0.71 (±0.15)	0.67 (±0.14)	0.70 (±0.14)	0.78 (±0.23)	0.80 (±0.22)
maximum	0.86 (±0.21)	0.91 (±0.19)	0.88 (±0.16)	0.88 (±0.16)	0.96 (±0.26)	1.02 (±0.24)

Paired t-test, *p<0.05, ** p<0.001

Mean difference of increased of Carotid Intima Media Thickness (CIMT) according to incense exposed group

Table 20 shown the mean difference of increased of Carotid Intima Media Thickness (CIMT) among the 3 group of incense exposed at baseline to follow-up. The mean difference of increased mean and maximum CIMT at CCA (±SD) were 0.022 (±0.04) mm and 0.034 (±0.06) mm. It was highest increase in the non-daily exposed group both mean and maximum of CCA (mean CCA; 0.023±0.05 mm and maximum CCA; 0.044±0.08 mm). However, there were no any significant difference among incense exposure and CIMT at CCA (p>0.05).

The mean difference of increased mean and maximum CIMT of RCCA (±SD) were 0.032 (±0.07) mm and 0.044 (±0.09) mm respectively. The mean difference of increased in mean CIMT of RCCA in the daily incense exposed group (mean±SD; 0.039±0.11 mm) were greater than other groups of exposed while mean difference of increased maximum CIMT of RCCA were greater in the non-daily exposed group mean±SD; 0.055±0.09mm) than other 2 group of exposed. However, we could not found any significant difference among incense exposure and CIMT at RCCA (p>0.05).

For the mean difference of increased mean and maximum CIMT at LCCA (\pm SD) were 0.029 (\pm 0.05) mm and 0.05 (\pm 0.07) mm respectively. We found the non-exposed and the non-daily exposure group had increased CIMT equally of 0.031 (\pm 0.04) mm which were greater than the daily exposed group (mean \pm SD; 0.024 \pm 0.06 mm). While the mean difference of maximum LCCA had greater in the daily-exposed group (mean \pm SD; 0.057 \pm 0.08 mm) than other 2 group of exposed. However, we could not observe a significantly difference amongst the three groups of exposure ($p>0.05$).

Table 20 Mean difference of increased carotid intima-media thickness (CIMT) after 1-year follow-up according to incense exposed group. (n=100)

Carotid Intima Media Thickness (CIMT)	Change of CIMT in incense exposure				p-value
	Total (n=100)	non- exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
CCA, mean	0.022 \pm 0.04	0.021 \pm 0.02	0.023 \pm 0.05	0.02 \pm 0.04	0.95 ^b
maximum	0.034 \pm 0.06	0.026 \pm 0.04	0.044 \pm 0.08	0.031 \pm 0.05	0.41 ^b
RCCA, mean	0.032 \pm 0.07	0.028 \pm 0.04	0.031 \pm 0.06	0.039 \pm 0.11	0.83 ^b
maximum	0.044 \pm 0.09	0.034 \pm 0.06	0.055 \pm 0.09	0.045 \pm 0.11	0.59 ^b
LCCA, mean	0.029 \pm 0.05	0.031 \pm 0.04	0.031 \pm 0.05	0.024 \pm 0.06	0.83 ^b
maximum	0.05 \pm 0.07	0.048 \pm 0.07	0.046 \pm 0.08	0.057 \pm 0.08	0.85 ^b

^bOne-way ANOVA

Number of participants whose increased CIMT according to incense exposed group

Table 21 showed an incensements CIMT of all participants after 1-year follow-up. We found that combined left and right CIMT at CCA were similarly increased in mean (10.0%) and maximum CIMT (19.0%). Combined mean maximum CIMT at CCA were highest increased in the non-daily exposed group (13.2% and 23.7% respectively). As for CIMT of RCCA were increased of mean (15.0%) and maximum CIMT (23.0%) which were highest increasing of mean RCCA in the daily exposed group (16.7%) and maximum RCCA were highest in the non-daily exposed group (34.2%). An increasing of CIMT at LCCA were 17.0% for mean LCCA and 28.0% for

maximum LCCA. The non-daily exposed group was highest increase in mean of LCCA (21.1%) and the daily exposed group was highest increased in maximum of LCCA (37.5%). However, we could not found any significant difference amongst the three groups of exposure ($p>0.05$).

Table 21 Number of participants whose increased of CIMT according to incense exposed group after 1-year follow-up (n=100)

Carotid Intima Media Thickness (CIMT)	Incense exposure				p-value
	Total (n=100)	non-exposed (n=38)	non-daily exposed (n=38)	daily exposed (n=24)	
CCA, mean	10 (10.0%)	2 (5.3%)	5(13.2%)	3(12.5%)	0.464
maximum	19 (19.0%)	5 (13.2%)	9 (23.7%)	5 (20.8%)	0.488
RCCA, mean	15 (15.0%)	6 (15.8%)	5 (13.2%)	4 (16.7%)	0.918
maximum	23 (23.0%)	6 (15.8%)	13 (34.2%)	4 (16.7%)	0.113
LCCA, mean	17 (17.0%)	7 (18.4%)	8 (21.1%)	2 (8.3%)	0.412
maximum	28 (28.0%)	9 (23.57%)	10 (26.3%)	9(37.5%)	0.477

Chi-square (χ^2) test

4.2.3 An association between incense smoke exposure and increased Carotid Intima Media Thickness (CIMT) after 1-year follow-up

An association between incense smoke exposure and increased CIMT

Multivariate logistic regression models of an association between an increment of 1 order of incense exposure (non-exposed, non-daily exposed and daily exposed) and risk of increased CIMT after a year follow-up. For unadjusted model, an increasing of 1 order of incense exposure was 1.55 (95%CI 0.67-2.53) fold increased odds of increasing mean CIMT at CCA and 1.33 (95%CI 0.7-2.53) fold increased odds of increasing maximum CIMT at CCA. After adjusted for age, gender, high density lipoprotein (HDL), Systolic blood pressure (SBP) and CIMT at baseline, we could observe that increased odds of increasing mean and maximum CIMT at CCA. An increasing of 1 order of incense exposed was increased risk of increased mean CCA (AOR = 1.05; 95%CI 0.48-2.31) and maximum CCA (AOR = 1.54; 95%CI 0.73-3.28). For LCCA and RCCA, we found increased risk of increasing maximum LCCA (1.72, 95%CI 0.91-3.25) and maximum RCCA (1.25, 95%CI 0.61-2.55) after adjusted model

but not for mean LCCA and RCCA. However, we could not observe an association between exposures to incense smoke and increased of mean and maximum CIMT both LCCA and RCCA (Table 22).

Table 22 Logistic regression models between incense smoke exposure and increased CIMT after 1-year follow-up (n=100)

Increased CIMT	Incense smoke exposure			
	Unadjusted model		Multivariate adjusted model ^a	
	OR	95% CI	AOR	95% CI
Increased CCA				
Mean	1.55	0.67-2.53	1.05	0.48-2.31
Maximum	1.33	0.7-2.53	1.54	0.73-3.28
Increased RCCA				
Mean	1.01	0.49-2.06	0.93	0.47-1.81
Maximum	1.12	0.62-2.04	1.25	0.61-2.55
Increased LCCA				
Mean	0.73	0.36-1.46	0.80	0.40-1.67
Maximum	1.38	0.79-2.43	1.72	0.91-3.25

^aAdjusted for factor associated to CIMT in adult: age, gender, high density lipoprotein (HDL), Systolic blood pressure (SBP) and CIMT at baseline

An association of increased Carotid Intima Media Thickness (CIMT) in the non-daily exposed group and daily-exposed group

In multivariate logistic regression models of an association between an incense smoke exposure and risk of increased CIMT after a year follow-up. For unadjusted model, exposure to daily incense and non-daily incense were increased odds of increased mean and maximum CIMT at CCA, RCCA and LCCA but not for mean RCCA in non-daily incense exposed and mean LCCA in daily exposure. However, we could not found any association between incense exposure and increased CIMT.

After adjusted for age, gender, high density lipoprotein (HDL), Systolic blood pressure (SBP) and CIMT at baseline, we could observe that increased odds of increased mean and maximum of CIMT. An exposure to daily incense had greatest

increased risk of increased mean CCA (AOR=9.67; 95%CI; 0.94, 99.76) than other of CIMT. For CIMT of RCCA, the non-daily exposure had greatest increased risk of increased maximum RCCA (AOR=5.04; 95%CI; 1.19, 21.36). While the maximum CIMT of LCCA was highest in daily exposure (AOR=2.88; 95%CI; 0.81, 10.22). However, we could not observe any an association between exposures to incense smoke and increased of CIMT after 1-year follow-up (Table 23).

Table 23 Multivariate Logistic regression models between incense smoke exposure and increased CIMT after 1-year follow-up (n=100)

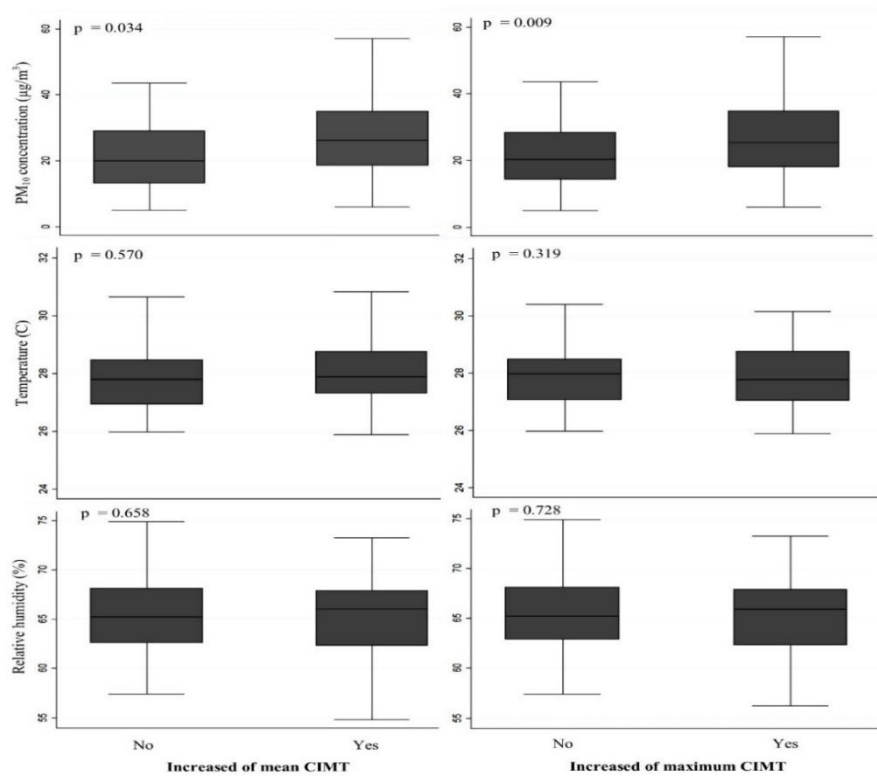
CIMT (mm)	Incense exposed groups				
	non-exposed	non-daily exposed		Daily exposed	
	Ref.	OR	95% CI	OR	95% CI
Unadjusted model					
CCA, mean	Ref. category	2.73	0.49, 15.03	2.57	0.39, 16.66
maximum	Ref. category	2.05	0.62, 6.81	1.74	0.45, 6.78
RCCA, mean	Ref. category	0.81	0.22, 2.91	1.07	0.27, 4.25
maximum	Ref. category	2.77	0.92, 8.33	1.07	0.27, 4.25
LCCA, mean	Ref. category	1.18	0.38, 3.66	0.40	0.08, 2.13
maximum	Ref. category	1.15	0.41, 3.25	1.93	0.63, 5.89
Adjusted model^a					
CCA, mean	Ref. category	1.93	0.27, 13.99	9.67	0.94, 99.76
maximum	Ref. category	1.73	0.45, 6.60	2.37	0.50, 11.13
RCCA, mean	Ref. category	0.86	0.21, 3.59	1.06	0.21, 5.25
maximum	Ref. category	5.04	1.19, 21.36	1.05	0.18, 6.10
LCCA, mean	Ref. category	1.14	0.32, 4.13	0.59	0.10, 3.36
maximum	Ref. category	1.27	0.39, 4.16	2.88	0.81, 10.22

^aAdjusted for factor associated to CIMT in adult: age, gender, high density lipoprotein (HDL), Systolic blood pressure (SBP) and CIMT at baseline

4.2.4 An increased mean and maximum of CIMT from baseline to follow-up and the average PM₁₀ concentration of participants

According to an increased mean CIMT from baseline to follow-up, the average PM₁₀ concentration of those participants whose CIMT had increased ($26.59 \pm 12.12 \mu\text{g}/\text{m}^3$) was significantly ($p=0.034$) higher than those who had not increased ($21.95 \pm$

9.83 $\mu\text{g}/\text{m}^3$). For maximum CIMT, the average PM_{10} concentration was also significantly ($p=0.009$) higher among those who had increased CIMT ($27.18 \pm 11.98 \mu\text{g}/\text{m}^3$) than those who had not ($21.48 \pm 9.77 \mu\text{g}/\text{m}^3$). An average 24-hours temperature of participants whose mean CIMT had increased was $27.86 (\pm 1.26)^\circ\text{C}$ which was slightly lower than participants whose had not increased ($27.99 \pm 1.22)^\circ\text{C}$. For relative humidity, an average 24-hours RH was not different among both groups of participant. Temperature and relative humidity could not find any associations with an increased mean and maximum CIMT. (Figure 17)



* Independent t-test

Figure 17 Increased mean and maximum carotid intima-media thickness (CIMT) (Yes/No) according to PM_{10} concentration, temperature, and relative humidity

4.2.5 An association between residential PM_{10} concentration and increased CIMT of all participants after 1-year follow-up

Table 24 shows multivariate logistic regression models of an association between an increment of 1 $\mu\text{g}/\text{m}^3$ indoor PM_{10} concentration and risk of increased CIMT after a year follow-up. For unadjusted model, an increasing of 1 $\mu\text{g}/\text{m}^3$ average

indoor PM₁₀ concentration were fold increased odds of increasing mean CCA (OR=1.07; 95%CI 1.01-1.14), maximum CCA (OR=1.07; 95%CI 1.02-1.12) and maximum LCCA (OR=1.04; 95%CI 1.00-1.08) at statistically significant (p<0.05). After adjusted for age, BMI, HDL and SBP, we could observe a stronger association between an average of indoor PM₁₀ concentration and mean and maximum CCA and maximum LCCA but not for RCCA. An increasing of 1 µg/m³ of an average indoor PM₁₀ concentration was significant (p<0.05) associated with 8% increased risk of increased mean CCA (AOR = 1.08; 95%CI 1.01 - 1.15), 7% increased risk of increased maximum CCA (AOR = 1.07; 95%CI 1.01 - 1.12) and 3% increased risk of increased maximum LCCA (AOR = 1.03; 95%CI 1.01 - 1.09). However, mean LCCA was not associated with indoor PM₁₀ concentration (AOR = 1.03; 95%CI 0.99-1.07). We also could not observe a risk of indoor PM₁₀ concentration on an increased CIMT of RCCA.

Table 24 Logistic regression models between residential PM₁₀ concentration and an increased CIMT (yes/no) after 1-year follow-up (n =100)

Increased CIMT	PM ₁₀ concentration (µg/m ³)			
	Unadjusted model		Multivariate adjusted model ^a	
	OR (95% CI)	p-value	AOR (95% CI)	p-value
Increased CCA				
Mean	1.07(1.01, 1.14)	0.021*	1.08(1.01, 1.15)	0.028*
Maximum	1.07(1.02, 1.12)	0.006*	1.07(1.01, 1.12)	0.012*
Increased LCCA				
Mean	1.03(0.92, 1.07)	0.245	1.02(0.97, 1.07)	0.38
Maximum	1.04(1.00, 1.08)	0.041*	1.03(1.01, 1.09)	0.031*
Increased RCCA				
Mean	1.01(0.96, 1.06)	0.782	1.00(0.96, 1.06)	0.88
Maximum	1.02(0.98, 1.07)	0.282	1.03(0.98, 1.07)	0.273

^aAdjusted for factor associated to CIMT in adult: age, high density lipoprotein (HDL), Systolic blood pressure (SBP), * p-value <0.05

CHAPTER V

DISCUSSION

Our study demonstrated the positive association between household burned incense and CIMT at CCA and LCCA among adults age more than 35 years in the central city of Sakon Nakhon province, Thailand. However, an associations were not found in the CIMT of RCCA. After stratifying incense exposure into 3 groups, we found CIMT of LCCA among daily exposure to household incense smoke was greater than non-daily exposure after controlling for others major CVD risk factors. Additionally, the non-exposure incense group yielded the least CIMT of LCCA amongst those three exposure groups. After a one year of follow-up, we found a positive association between incense exposed and increase CIMT but null statistically significant. In addition, our finding also found a positive association between exposures to household indoor particulate matter and increase of CIMT. An average of household indoor PM₁₀ concentration was significantly different between participants whose CCA had increased and those whose CCA had not. We did observe a risk increase LCCA associate with indoor PM₁₀ concentration. However, association was not find in RCCA.

5.1 General information of participants

General characteristics of participants

The general characteristics of participants at baseline and after 1-year follow-up were quite similar. This present showed that the majority of the study population was female and adults age (IQR) 56(12) years old which was the same previous study (Painschab et al., 2013; Su et al., 2015). The age was significant association with incense exposure and increased CIMT because CIMT value increased with advancing age (over the age of 45 years) in all carotid segments (Loboz-Rudnicka et al., 2016; Qu & Qu, 2015; Ren, Cai, Liang, Li, & Sun, 2015; Simova, 2015). All participants were stratification into; non-incense exposed, non-daily incense exposed and daily incense exposed group by long term incense exposure which may effected to human health (Navasumrit et al., 2008). Current occupational was found an association with incense

user because most of participants were trader, they burned incense at home for ritual or religious purpose which is a common practice among Thai-Vietnam community same as Chinese populations in China, Singapore (Friborg et al., 2008; Pan et al., 2014) and Taiwan (Liao, Chen, Chen, & Liang, 2006a). According to most of them were trader therefor type of residents were shop house which was a close room or some part of room open. When they burned incense, sometime they were sat closely to incense burning area and smell to incense smoke therefor, these characteristics were associated with exposure to incense smoke. Although, our study have 24% of participants loss to follow-up (n=32). All of socio-demographic characteristics of participants who loss to follow-up did not effected to incense exposed groups. Also, there were on any difference between the characteristics of participants at bassline and follow-up.

Clinical assessment (Blood parameters and hemodynamics) of participants

Our results showed the association between the cardiovascular risk factors such as hs-CRP level and heart rate and household incense exposure at baseline while, there were no any association after 1-year follow-up. However, the daily incense exposure group revealed lower cardiac inflammation (hs-CRP) than the other two groups. Hs-CRP together with traditional CVD risk factors (LDL, HDL and total cholesterol in each groups) might be a better predictor. Our study also needed further markers of endothelial dysfunction which was suggested by a previous study finding along similar lines (Painschab et al., 2013). Regarding heart rate, we found an association with incense exposed which was a source of household indoor air similar to the study of Huang et al reported that “personal exposure to household particulate matter, household activities especially, during stir-frying, cleaning with detergent and burning incense association with heart rate variability among housewives” (Huang et al., 2014). However, we acknowledge that traditional risk factors may be underreported in the incense exposed group.

5.2 Comparisons of Carotid intima-media thickness (CIMT) and household indoor particulate matter (PM₁₀) from baseline to follow-up

Comparisons of Carotid intima-media thickness (CIMT) from baseline to follow-up

We found a strongly significant difference between CIMT at baseline and follow-up. Overall, our baseline the average of mean CIMT on CCA (0.71 ± 0.15 mm) and annual change in mean CIMT 0.022 ± 0.04 mm/year were compared to those study by Kunzli et al (mean CIMT 0.78 ± 0.15 mm; 0.002 ± 0.013 mm/year) (Kunzli et al., 2010) and the study of Adar et al (0.678 ± 0.189 mm, 0.014 ± 0.053 mm/year) (Sara D. Adar et al., 2013). For average of maximum CIMT on CCA (0.89 ± 0.18 mm) and annual change in maximum CIMT (0.034 ± 0.06 mm/year) was greater than the previous study of Gan WQ et al (0.673 ± 0.122 mm; 0.0092 ± 0.0121 mm/year) (Gan et al., 2014). Additionally, we observed that baseline of mean LCCA and maximum LCCA were less than mean RCCA and maximum RCCA. This finding was opposite effect from the study of the differences in left and right CIMT and the risk factors association (Luo, Yang, Cao, & Li, 2011) and cross sectional study of CIMT and long term exposure to traffic related to air pollution in middle aged residents of Taiwan (Su et al., 2015) maybe cause of haemodynamic and biochemical changes of person had different effects on the CIMT depending on the side affected (Luo et al., 2011), these relations may be more affected by confounding by personal factors (Sara D. Adar et al., 2013) and other causes of changes in CIMT (Qu & Qu, 2015).

Comparison of household indoor particulate matter (PM₁₀) between dry and wet season

We conducted PM₁₀ inside of all participants' home depend on seasonality. Our result found the average of PM₁₀ was 24.2 ± 11.4 $\mu\text{g}/\text{m}^3$ which were compared to the study of Bauer et al showed 1-year exposure to PM₁₀ 20.8 ± 2.5 $\mu\text{g}/\text{m}^3$ (Bauer et al., 2012) and Su et al reported that 1-year outdoor PM₁₀ concentration was 44.21 ± 4.19 $\mu\text{g}/\text{m}^3$ (Su et al., 2015), there were different from the study of indoor/outdoor PM₁₀ and PM_{2.5} in Bangkok, Thailand reported that the average of PM₁₀ concentration in living room was 185 ± 42 $\mu\text{g}/\text{m}^3$ (Feng CT. et al., 2000). However, the level of household

indoor PM₁₀ was not exceed the National Ambient Air Quality Standards (NAAQSs) of outdoor air in Thailand (50 µg/m³) but it was exceed the level of Air quality guidelines of World Health Organization (20 µg/m³) (WHO, 2005, 2010).

Our finding also found a significant difference of PM₁₀ concentration, temperature and relative humidity (RH) between wet and dry season. Household indoor PM₁₀ in dry season (31.4 ± 18.2 µg/m³) higher than wet season (17.1 ± 8.8 µg/m³), because of particulate matter concentrations were affected by shifting seasons with lowest averages obtained during the rainy season and highest levels of particulate matter during the winters, due to the air exchange rate which was directly correlated with PM concentrations in the living rooms (Sidra, Ali, Ahmad Nasir, & Colbeck, 2015). The major contributor to source apportionment of indoor PM₁₀ in home was the outdoor contribution (Chao & C. Cheng, 2002). While, Sidra et al. were reported that “particulate matter in residential settings resulting from various routine activities such as cooking, floor sweeping, presence of people, smoking and space heating” (Sidra et al., 2015).

5.3 An association between incense smoke exposure and the average of residential PM₁₀ concentration and increase CIMT of all participants

An association of incense smoke exposure and Carotid Intima Media Thickness (CIMT) of all participants at baseline

We found an association between mean and maximum CIMT of common carotid artery (CCA) and incense exposure. Multivariable regression analysis after controlling for age, hypertension, cardiovascular disease, cholesterol, hs-CRP and heart rate remained a strong association between exposure to incense smoke and CIMT. Incense burning is composed of particulate matter (PM) and other air pollutants such as volatile organic compounds. Evidence demonstrated that exposure to air pollution is potentially linked to a progression of CIMT which is used as a marker of cardiovascular health (Sara D. Adar et al., 2013; Armijos et al., 2015; Provost et al., 2015; Su et al., 2015). Our finding supports previous studies about air pollution exposure and CIMT; long term smoking (Barnoya & Glantz, 2005), chronic exposure to fossil fuel combustion (Kunzli et al., 2010) solid fuel combustion in home (Mi-Sun Lee et al., 2012) and biomass fuel (Painschab et al., 2013) are all associated with CIMT

and atherosclerosis. A possible mechanism links between exposure to air pollution and increased CIMT is that “increases in oxidative stress, lung-mediated inflammation and stimulation of the autonomic nervous system are associated with the development of atherosclerosis” (Hoffmann et al., 2007). Since CIMT has well characterized surrogate markers for CVD, the findings of the study revealed that there was an important association between long-term exposure to incense smoke and increased risk for CVD.

Our study findings are strengthened by controlling potential confounding factors which reported specifically on effects of CIMT in adults, other cardiovascular risk factors, and outdoor air pollutant exposures by restricted to main road in the study area. In addition, we found that an incense exposed group has a stronger association with mean CIMT of LCCA compared to non-incense exposure group. The progression of atherosclerotic generally on both sides in the presence of traditional risk factors such as older age, hypertension, and hypercholesterolemia (Rosvall et al., 2015; Su et al., 2015). Our results also measured both the left and right CCAs for accessing the association with incense exposure. We found that the daily incense exposure groups were associated with mean LCCA compared with non-exposure groups while, RCCA did not show any association. The possible reason to support this finding is “the different origins of the left and right CCA, whereby they are subjected to different flow intensities from the aortic arch”. “The left CCA stems directly from the arch of the aorta and is affected by aortic arch pressure (hydrostatic pressure) while the right CCA stems from the innominate artery, which is an extension of the ascending aorta, and is subjected to significant pressure from ascending aortic blood flow (dynamic pressure)” (Luo et al., 2011). However, the reason for this phenomenon is not yet clear. We need further study to confirm these hypotheses. Moreover, the difference of CIMT on left and right CCA depends on the haemodynamic and biochemical changes on the CIMT. It was found that in adults between the ages of 35 and 65 years old, the left CIMT was thicker than the right (Luo et al., 2011), however the reason is still not clear.

An association between incense smoke exposure and increased Carotid Intima Media Thickness (CIMT) after 1-year follow-up

Although, our finding could observe that exposure to daily incense was increased risk of increased mean CIMT of CCA, LCCA and RCCA but there were no

any significantly association after 1-year follow-up. Incense burning is a source of household indoor air pollution (Huang et al., 2014). There were no evidence of exposure to incense used and CIMT but we could compare to the study of exposed to indoor air such as chronic exposure to biomass fuel (Painschab et al., 2013) and residential particulate matter (Sara D. Adar et al., 2013). Two previous study were significant associated with increased CIMT; our finding was contrast with Sara D. Adar et al (2013) which found an association between an increased CIMT and residential particulate matter. The reason to support is that the follow-up period was different between our study (1 year) and Sara D study (2.5 years). Moreover, the study of exposed to outdoor air pollutants (Kunzli et al., 2010; Provost et al., 2015; Su et al., 2015) were also showed a significant associated with increased CIMT. But, our finding was similar to long-term exposure to traffic pollutant of Gan WQ et al (2014) study which did not find an association between traffic-related air pollution and progression of carotid artery atherosclerosis. Gan's suggested that it might be an error of CIMT measurement. In addition, Hemodynamic, personal biochemical changes and personal activity factors may effect on the progression of CIMT (Sara D. Adar et al., 2013 & Qu & Qu, 2015). Additionally, a small sample size due to loss to follow up might potentially contributed to our finding. Therefore, our result could not observe any significant association between increasing of CIMT in these three of incense exposed groups. These differences may partly explain the null associations in our study.

An increased mean and maximum of CIMT from baseline to follow-up and the average PM₁₀ concentration of participants

According to an increased mean CIMT from baseline to follow-up, the average PM₁₀ concentration of those participants whose CIMT had increased ($26.59 \pm 12.12 \mu\text{g}/\text{m}^3$) was significantly higher than those who had not increased ($21.95 \pm 9.83 \mu\text{g}/\text{m}^3$) ($p=0.034$). For maximum CIMT, the average PM₁₀ concentration was also significantly higher among those who had increased CIMT ($27.18 \pm 11.98 \mu\text{g}/\text{m}^3$) than those who had not ($21.48 \pm 9.77 \mu\text{g}/\text{m}^3$) ($p=0.009$). Our result were fairly consistent with those from the study of Tonne et al showed median of PM₁₀ exposure was $24.4 \mu\text{g}/\text{m}^3$, after adjustment an interquartile range increase ($1.6 \mu\text{g}/\text{m}^3$) was associated with increase in CIMT (Tonne C, Yanosky JD, Beevers S, Wilkinson P, & FJ, 2012), the study of

Aguilera et al reported that an exposure contrast between the 10th and 90th percentile for PM₁₀ (PM average $20.2 \pm 2.3 \mu\text{g}/\text{m}^3$) was associated with percent change of CIMT (Aguilera et al., 2016), and Su et al indicated that one-year average of exposures to PM₁₀ ($44.21 \pm 4.19 \mu\text{g}/\text{m}^3$) were significantly increased for CIMT (Su et al., 2015). Another previous study were reported in range increase of $10 \mu\text{g}/\text{m}^3$ (Liu et al., 2015) and $6.7 \mu\text{g}/\text{m}^3$ (Bauer et al., 2010) of PM₁₀ were associated with increased CIMT but no significant and Perez et al also reported that PM₁₀ $27.8 \pm 1.8 \mu\text{g}/\text{m}^3$ was positively increased CIMT but not significantly association, results of Heinz Nixdorf Recall (HNR; Ruhr Area, Germany) (Perez et al., 2015)

An association between residential PM₁₀ concentration and increased Carotid Intima Media Thickness (CIMT) of all participants after 1-year follow-up

We found a positive association between exposures to household indoor particulate matter and increase of CIMT after a year of follow-up in the central city of Sakon Nakhon province, Thailand. A few studies have investigated the association between indoor particulate matter and CIMT (Sara D. Adar et al., 2013; Armijos et al., 2015; Painschab et al., 2013). CIMT results from the processes of cumulative atherogenesis. CIMT progression is a predictor of atherosclerosis and cardiovascular events (Chambless et al., 2000; Gepner et al., 2006; Liu et al., 2015; O'Leary et al., 1999; Stein et al., 2008). Our finding are in agreement with previous study (Bauer et al., 2012; Liu et al., 2015; Su et al., 2015; Tonne C et al., 2012) who found that those on household indoor PM₁₀ increased CIMT. The findings support the statement of the American Heart Association's expert panel regarding the biological mechanisms of the effects of particulate matter on cardiovascular events (Brook et al., 2010)

Our studies found a stronger association between indoor PM₁₀ concentration and mean of CIMT and maximum of CIMT at CCA particularly, maximum of CIMT at LCCA after controlling for others major CVD risk factors; age, BMI, HDL, LDL and SBP which is associated with progression of CIMT in CCA (Greenland et al., 2010; Qu & Qu, 2015; Rosvall et al., 2015). An average indoor PM₁₀ concentration was significantly different between participants whose CIMT had increased and those who's CIMT had not. We could observe a stronger association between average indoor PM₁₀ concentration (increments of $1 \mu\text{g}/\text{m}^3$) and increased risk of increased mean CCA (7%)

and maximum CCA (8%). This finding similar to the study of Tonne et al that an interquartile range increase ($5.2 \mu\text{g}/\text{m}^3$) in PM_{10} was significantly association with increased of CIMT 5% (95% CI 1.9%, 8.3%) after adjustment (Tonne C et al., 2012), but it was different from the numerous study with no significant such as; Lui et al indicated that overall analysis increments of $10 \mu\text{g}/\text{m}^3$ in PM_{10} was associated with an increase of CIMT ($4.13 \mu\text{m}$; 95% CI, -5.79 – $14.04 \mu\text{m}$) (Liu et al., 2015), An interquartile range increase in PM_{10} ($6.7 \mu\text{g}/\text{m}^3$) was associated with a 1.7% (95% CI: -0.7% to 4.1%) increase in CIMT (Bauer et al., 2012). And the study of Aguilera et al reported that an exposure to PM_{10} had percent change of CIMT 1.58% (95% CI: -0.30 , 3.47%) (Aguilera et al., 2016). It's maybe cause by pathways of air pollution effect to cardiovascular disease (Newby et al., 2015) which particulate matter is thought to influence atherogenesis include the oxidative stress and inflammation (Hoffmann et al., 2009).

The progression of atherosclerotic were generated on both sides in the presence of traditional risk factors. Our results also measured both the left and right CCA for considering the association with indoor PM_{10} exposure. We found that a risk increase LCCA was significantly associated with indoor PM_{10} concentration, it was similar to the study of Su et al was found an average percentage increases in maximum left CIMT of 3.72% (95% CI: 0.32, 7.11) per $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} (Su et al., 2015). However, we also could not observe a risk indoor PM_{10} concentration on an increased RCCA. The possible reason to support this finding is “the different origins of the left and right CCA may subjected to different flow intensities from the aortic arch”(Luo et al., 2011). “The left CCA stems directly from the arch of the aorta and is affected by aortic arch pressure (hydrostatic pressure) while the right CCA stems from the innominate artery, which is an extension of the ascending aorta, and is subjected to significant pressure from ascending aortic blood flow (dynamic pressure)” (Luo et al., 2011). However, the reason for this phenomenon is not yet clear. We need further study to confirm these hypotheses.

CHAPTER VI

CONCLUSION

6.1 Conclusion

To investigate an association between incense smoke exposure and increased Carotid Intima Media Thickness (CIMT), there were 2 periods study included; baseline characteristics and a one-year follow-up. From the finding in this study, it would be concluded that household particulate matter (PM₁₀) was associated to increase of CIMT at CCA and LCCA. However, the progression of CIMT in incense smoke exposure were small and null associations.

Most of participants were female in middle age (more than 35 years old) who were finished high school and were merchant. Current occupational was found an association with incense user because most of participants were trader who burned incense at home for ritual or religious purpose which is a common practice among Thai-Vietnam community. Regarding to incense used characteristics, most of them were in the non-daily exposed group. As for the incense exposed group, most of them used a long incense stick and burned incense at less 5 sticks each time. They spent more than 30 minutes for burning incense each time and had used incense for more than 14 years. As for clinical assessment (Blood parameters and hemodynamics) of participants, the relationship between the cardiovascular risk factors such as ages, hs-CRP level, HDL, SBP and heart rate were associated with household incense exposure.

The levels of CIMT at baseline, combined mean CIMT of CCA was 0.71 ± 0.15 mm and combined maximum CIMT of CCA was 0.88 ± 0.18 mm. For mean and maximum CIMT of RCCA were 0.72 ± 0.17 mm and 0.89 ± 0.19 mm respectively. Mean and maximum CIMT of LCCA were 0.70 ± 0.18 mm and 0.87 ± 0.21 mm respectively. The mean and maximum of combine CIMT, RCCA and LCCA were highest in the daily-incense exposed group. Combined mean and maximum CIMT of CCA and mean and maximum CIMT at LCCA were significantly ($p < 0.05$) different among the 3 groups of exposure but not for CIMT at RCCA. The mean difference of increased mean

and maximum CIMT at CCA (\pm SD) were 0.022 (\pm 0.04) mm and 0.034 (\pm 0.06) mm. Mean and maximum CIMT of RCCA (\pm SD) were 0.032 (\pm 0.07) mm and 0.044 (\pm 0.09) mm respectively. For mean and maximum CIMT at LCCA (\pm SD) were 0.029 (\pm 0.05) mm and 0.05 (\pm 0.07) mm respectively. However, we could not observe a significantly difference amongst the three groups of exposure ($p>0.05$).

Regarding to an association of incense smoke exposure and increased Carotid Intima Media Thickness (CIMT) at baseline. After adjusted for confounding factors, the daily exposed and non-daily exposed were significant ($p<0.05$) associated with combine CIMT of CCA and LCCA which compared to the non-exposed group. However, mean CIMT and maximum CIMT on RCCA did not show any associations to household incense exposure.

After 1-year follow-up, the levels of CIMT was higher than baseline CIMT ($p<0.001$). All CIMT were remained highest in daily-incense exposed group. The average levels of mean and maximum of combine CIMT of CCA were 0.73 ± 0.15 mm and 0.92 ± 0.18 mm respectively. For RCCA, mean and maximum CIMT were 0.75 ± 0.17 mm and 0.94 ± 0.19 mm respectively. The mean and maximum CIMT of LCCA were 0.73 ± 0.17 mm and 0.92 ± 0.20 mm respectively. There were significantly different in mean CCA, mean RCCA and both mean and maximum CIMT of LCCA among those three groups of exposed but not for maximum CIMT of CCA and RCCA.

An association between incense exposed and increased CIMT after 1-year follow-up, the differences in annual changes of CIMT between incense smoke exposures were small and also, no significant associations. An increment of 1 order of incense exposure (non-exposed, non-daily exposed and daily exposed) were highest increased risk of increased maximum CIMT of LCCA (AOR=1.72, 95%CI 0.91-3.25). The combine mean and maximum CIMT (AOR=1.05 (95%CI 0.48-2.31) and AOR=1.54 (95%CI 0.73-3.28) respectively) and maximum RCCA (AOR=1.25 (95%CI 0.61-2.55) were also risk factors but not for mean LCCA and RCCA. These findings suggest that incense burning inside the house is a form of indoor air pollution and may be a risk factor for cardiovascular disease development which is the main cause of morbidity and mortality in Asian countries.

The one year average of household indoor PM₁₀ was 24.2±11.4 µg/m³. PM₁₀ in dry season (27.6 ±24.6 µg/m³) were higher than wet season (16.1 ±11.9 µg/m³) with a significant difference of PM₁₀ concentration between wet and dry season (p<0.0001). An average indoor PM₁₀ concentration were stronger association with mean and maximum CCA and maximum LCCA but not for RCCA and mean LCCA. An increasing of 1 µg/m³ average indoor PM₁₀ concentration was significant (p<0.05) associated with 8% increased risk of increased mean CCA (AOR = 1.08; 95%CI 1.01 - 1.15), 7% increased risk of increased maximum CCA (AOR = 1.07; 95%CI 1.01 - 1.12) and 3% increased risk of increased maximum LCCA (AOR = 1.03; 95%CI 1.01 - 1.09). However, CIMT of RCCA and mean LCCA was not associated with indoor PM₁₀ concentration (p>0.05). Household indoor particulate matter (PM₁₀) is associated with increased CIMT of CCA and LCCA but not for CIMT of RCCA. These findings suggest that particulate matter inside the house may be a risk factor for cardiovascular disease morbidity and mortality.

6.2 Benefit of this study

1. Our study could suggest that long-term exposure to incense smoke may increase CVD risk and exposure to residential PM₁₀ may increase CVD risk. Therefore, the finding will use to support a further intervention study to improve residential environment.

2. It could predict the risk of CVD which is related to indoor air pollution exposure.

3. The results may support the progression of CIMT among participants since there is no previous study had been conducted among this population in Thailand.

6.3 Limitations of this study

Some potential limitations might affect our results as following;

1. Our sample size is small might potentially contributed in our study.
2. Different types and brand of incense may not produce the same air pollution concentration which may contribute to adverse effects on health.

3. The various types of household activities carried out in a routine day in a house such as cooking, floor sweeping, smoking, space heating and incense burning may contributed the difference of particulate matter and other indoor air pollutants (PM_{2.5}, CO, SO₂, NO₂ and VOCs) which may confounded our findings.

4. Particulate matter concentration in this study was based on a single sample collection for 24 hours in each season. It may not be a good representative of the concentrations.

5. Loss to follow-up was another limitation of the current study. Thirty-two participant (24%) were not complete for the clinical assessment at follow-up period, because of heavy flood in the central city of Sakon Nakhon province (on August 2016). Leaving a relatively small sample of 100 individuals might potentially contributed in our study.

6. In term of generalizability, participants of this study was limited to one main road and one city. Therefore, general characteristics may not be the same as other Thai population

6.4 Recommendations of this study

For study participants;

1. During burning incense, they should open the doors and/or windows and/or use exhausted fan to circulate the air. Or, they should burn incense outside the home to reduce an incense smoke. Regarding to our finding, the type of house was associated with incense burning.

2. They should not stay in the area of incense burning due to our study found that most of participant would like to stay close to incense burning area and smell an incense smoke.

3. Participants should cleaning their house frequently to reduce the concentration of particulate matter because we found an association between household PM₁₀ and increased CIMT.

For policy recommendation;

Regarding to our study results, the policy for environmental health should be considered to provide the standard level of indoor/ residential air quality of Thailand.

For further study;

1. Only household PM_{10} concentrations were considered in the present, however, there are several pollutants such as $PM_{2.5}$, CO, CO_2 , NO_x , SO_x , black carbon, VOCs and PAHs from household indoor air related to incense burning which may affect to increase CIMT. Therefore, the remains should be provided in further study.

2. One-year follow-up revealed a small change of CIMT. Therefore, a longer follow-up would be clearly seen the changing of CIMT and could provide the progression of atherosclerosis.



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จุฬาลงกรณ์มหาวิทยาลัย
CHULALONGKORN UNIVERSITY



APPENDIX

จุฬาลงกรณ์มหาวิทยาลัย
CHULALONGKORN UNIVERSITY

APPENDIX A

QUESTIONNAIRE (English Version)

This study will be collected the data with questionnaires about the factors that might be cause to increase CIMT. The data collection will consist with the personal characteristics, home characteristics and incense used factors. There are 3 parts as follow;

Part 1. Personal characteristics of participant included, age, gender, Body Mass Index (BMI), level of education, income, residing time in home (hour), alcohol consumption, physical activity and dietary.

Part 2. Home characteristics included, temperature, humidity, type of resident, house size (wide/length), fuel cooking use, using air conditioner, using the air purifier and fan

Part 3. Incense Used; will asked for condition of incense using at home included, type of incense, number of incense use per day, time of incense use and duration of incense use, history of incense use and location of incense use (close/Open).

Part 1: Personal characteristics

1. Age..... (Year).
2. Gender () male () female
3. Ethnic.....
4. Weight.....kilograms. Height centimeters.
5. Body Mass Index (BMI).....
6. Education level
 - High School/Diploma/Certificate
 - Bachelor's degree
 - Master's degree
 - Higher than Master's degree
7. Current occupation
 - Agricultural occupation
 - Merchant and trader
 - Government
 - Company employee

34. Did you use air ventilation in your home?
 Yes No (Move to number 36)
35. Which room did you use air ventilation?
 Bedroom living room work office
 Shop room other.....
36. Have you ever burned the mosquito repellent coils in the house?
 Yes No

Part 3 Incense used

37. Have you ever used incense burning inside home?
 Yes.....
How many time you have burning of incense.....time/day
How many incense stick did you burned per timesticks
 No (finished of questionnaire)
38. How often did you burn incense sticks?
 1-2 days/week 3-4 days/week
 \geq days/week every days
39. When you have burning of incense.....to..... (O'clock)
40. Which area in your home did you burn incense?
 Bedroom living room work office
 Shop room upstairs of home other.....
41. Did you sit closely to incense burning area?
 yes- during the time of incense burning.
 yes- but, sometime of incense burning
 never
42. Did you have a smell of incense?
 yes- during the time of incense burning.
 yes- but, sometime of incense burning
 never
43. In incense burning time, how far did you stay from incense burning area?.....meter
44. How long have you burned incense.....years?

45. Type of incense

 stick joss stick coil cone powder electronic incense

46. Do you inserting to participate for screen the risk of CVD and stroke?

 Yes No

APPENDIX B

QUESTIONNAIRE (Thai Version)

แบบคัดกรอง

แบบคัดกรองนี้จัดทำขึ้นเพื่อใช้ในการสอบถามข้อมูลเบื้องต้นเพื่อคัดเลือกผู้ร่วมวิจัยที่มีคุณสมบัติตามเกณฑ์การเข้าร่วมในงานวิจัยเรื่อง ความสัมพันธ์ระหว่างการสัมผัสควันรูปและการเพิ่มความหนาของผนังหลอดเลือดแดงแคโรติดที่ลำคอในประชาชนที่อาศัยอยู่ในเขตเมือง จังหวัด สกลนคร

กรุณาตอบแบบสอบถามทุกข้อตามความเป็นจริง โดยข้อมูลที่ท่านตอบในแบบสอบถามจะถูกเก็บเป็นความลับ การนำเสนอข้อมูลจะนำเสนอในภาพรวมเท่านั้น หากมีข้อสงสัยประการใด ท่านสามารถสอบถามเพิ่มเติมได้ที่ นางสาวรัตน์ คำมูลกร โทรศัพท์ 095-658-8095

ผู้วิจัยขอขอบคุณที่กรุณาให้ความร่วมมือในการตอบแบบคัดกรองนี้อย่างครบถ้วน

วิทยาลัยวิทยาศาสตร์สาธารณสุข จุฬาลงกรณ์

มหาวิทยาลัย

คำชี้แจง โปรดทำเครื่องหมาย ลงในช่อง หรือเติมคำในช่องว่างให้ตรงกับความจริงมากที่สุด
ส่วนที่ 1 ข้อมูลทั่วไปของผู้ตอบแบบสอบถาม

1. บ้านเลขที่..... หมู่..... ถนน..... ตำบล.....

2. อายุ.....ปี

3. ท่านมีโรคประจำตัวหรือไม่

มี

เบาหวาน

ความดันโลหิต

โรคหัวใจและหลอดเลือด

โรคระบบทางเดินหายใจ

โรคไขมันในเลือดสูง

อื่นๆ ระบุ.....

ไม่มี

4. ท่านใช้เวลาอยู่บ้านหลังนี้เฉลี่ยประมาณกี่ชั่วโมงในแต่ละวัน (24 ชั่วโมง).....ชั่วโมง
5. ท่านอาศัยอยู่บ้านหลังนี้มานานกี่ปี.....ปี
6. ปัจจุบันท่านสูบบุหรี่หรือไม่ สูบบุหรี่จำนวน.....มวนต่อวัน ไม่สูบบุหรี่
7. ลักษณะที่อยู่อาศัยของท่านในปัจจุบันเป็นอย่างไร
 บ้านเดี่ยว ทาวน์เฮ้าส์ ห้องเช่า ตึกแถวที่ใช้เป็นร้านค้าอย่างเดียว
- ตึกแถวที่มีลักษณะเป็นร้านค้าและบ้านพักในหลังเดียวกัน
- อื่นๆ ระบุ.....
8. ท่านได้มีการจัดรูปภายในบ้านหรือไม่
 มีการจัดรูป ไม่มีการจัดรูปในบ้าน
9. ชนิดของรูปที่ท่านใช้จุดในบ้าน
 รูปแท่งไม้ไฟ/รูปกำยาน/รูปขด รูปไฟฟ้า
10. ท่านจุดรูปบ่อยหรือไม่
 1-4 วันต่อสัปดาห์ 5 วันหรือมากกว่า 5 วันต่อสัปดาห์หากมี
11. โปรแกรมตรวจคัดกรองความเสี่ยงต่อการเป็นโรคหัวใจและหลอดเลือดเบื้องต้น ท่านสนใจที่จะเข้าร่วมตรวจหรือไม่
 สนใจเข้าร่วม ไม่สนใจเข้าร่วม

แบบสอบถาม

ความสัมพันธ์ระหว่างการสัมพัทธ์วันรูปและการเพิ่มความหนาของผนังหลอดเลือดแดงแคโรติดที่
ลำคอในประชาชนที่อาศัยอยู่ในเขตเมือง จังหวัดสกลนคร

แบบสอบถามนี้ได้จัดทำขึ้นเพื่อใช้ในการศึกษาวิจัยในประชาชนที่อาศัยอยู่ในเขตเมือง
จังหวัดสกลนคร โดยจะทำการเก็บข้อมูลปัจจัยที่มีผลต่อการเพิ่มความหนาของผนังหลอดเลือดแดง
ได้แก่ ข้อมูลทั่วไปของผู้ตอบแบบสอบถาม ข้อมูลลักษณะของที่อยู่อาศัย และการใช้รูป

กรุณาตอบแบบสอบถามทุกข้อตามความเป็นจริง โดยข้อมูลที่ท่านตอบในแบบสอบถามจะ
ถูกเก็บเป็นความลับ การนำเสนอข้อมูลจะนำเสนอในภาพรวมเท่านั้น หากมีข้อสงสัยประการใด
ท่านสามารถสอบถามเพิ่มเติมได้ที่ นางสาวรัตณี คำมูลกร โทรศัพท์ 095-658-8095

ผู้วิจัยขอขอบคุณที่กรุณาให้ความร่วมมือในการตอบแบบสอบถามอย่างครบถ้วน

วิทยาลัยวิทยาศาสตร์สาธารณสุข จุฬาลงกรณ์

มหาวิทยาลัย

คำชี้แจง โปรดทำเครื่องหมาย ลงในช่อง หรือเติมคำในช่องว่างให้ตรงกับความจริงมากที่สุด
ส่วนที่ 1 ข้อมูลทั่วไปของผู้ตอบแบบสอบถาม

1. เพศ ชาย หญิง
2. เชื้อชาติ ไทย จีน ญวน
 อื่นๆ ระบุ.....
3. น้ำหนัก.....กิโลกรัม ส่วนสูง.....เซนติเมตร
4. ระดับการศึกษา
 ไม่ได้เรียนหนังสือ ประถม/มัธยม
 ปริญญาตรี ปริญญาโทหรือมากกว่า
5. อาชีพ
 ข้าราชการ พนักงาน/ลูกจ้างของรัฐ
 พนักงาน/ลูกจ้างของรัฐวิสาหกิจ พนักงาน/ลูกจ้างของเอกชน
 เกษตรกร ค้าขาย/อาชีพอิสระ
 รับจ้างทั่วไป อื่นๆ ระบุ.....

6. ท่านมีโรคประจำตัวหรือไม่

มี

- เบาหวาน ความดันโลหิต โรคหัวใจและหลอดเลือด
 โรคระบบทางเดินหายใจ โรคไขมันในเลือดสูง
 อื่นๆ ระบุ.....

ไม่มี

7. สมาชิกในครอบครัวของท่านมีประวัติการป่วยด้วยโรคหัวใจและหลอดเลือดหรือไม่

มี (มีความสัมพันธ์เป็น.....)

ไม่มี

8. ท่านใช้เวลาอยู่บ้านหลังนี้เฉลี่ยประมาณกี่ชั่วโมงในแต่ละวัน (24

ชั่วโมง).....ชั่วโมง

9. ในแต่ละวัน ส่วนใหญ่ท่านอยู่บ้านหลังนี้ในช่วงเวลาใด

ช่วงเช้า ตั้งแต่เวลา.....น. ถึง.....น.

ช่วงบ่าย ตั้งแต่เวลา.....น. ถึง.....น.

ช่วงเย็น ตั้งแต่เวลา.....น. ถึง.....น.

10. ในช่วงกลางวัน บริเวณใดในบ้านที่ท่านใช้เวลาอยู่นานที่สุด และนานกี่ชั่วโมง

ห้องนอนชั่วโมง ห้องรับแขก/นั่งเล่น/ดูทีวีชั่วโมง

ห้องทำงานชั่วโมง ห้องขายของชั่วโมง

อื่นๆ ระบุ.....

11. ท่านอาศัยอยู่บ้านหลังนี้มานานกี่ปี.....ปี

12. ปัจจุบันท่านมีสมาชิกในบ้านที่อาศัยอยู่บ้านเดียวกันจำนวนกี่คน (ไม่นับรวมตัวท่าน).....คน

13. ในอดีตที่ผ่านมาท่านเคยสูบบุหรี่หรือไม่ เคย (เลิกสูบบุหรี่แล้วเป็นเวลา.....ปี)

ไม่เคย

14. ปัจจุบันท่านสูบบุหรี่หรือไม่ สูบบุหรี่จำนวน.....มวนต่อวัน ไม่

สูบบุหรี่

15. ปัจจุบันมีสมาชิกที่อาศัยอยู่บ้านเดียวกันกับท่านสูบบุหรี่หรือไม่

มี จำนวน.....คน

ไม่มี

16. ในอดีตที่ผ่านมาท่านดื่มเครื่องดื่มแอลกอฮอล์เป็นประจำหรือไม่

ดื่ม

ไม่ดื่ม

17. ปัจจุบันท่านยังดื่มเครื่องดื่มแอลกอฮอล์เป็นประจำหรือไม่
 ดื่ม.....วันต่อสัปดาห์ ครั้งละประมาณขวด/แก้ว
 ไม่ดื่ม (ข้ามไปข้อ 19)
18. ในระยะ 3 เดือนที่ผ่านมาท่านดื่มเครื่องดื่มแอลกอฮอล์ประเภทใด
 เบียร์ บรันดี เหล้าขาว
 ยาดอง ไวน์ อื่นๆ ระบุ.....
19. ในระยะ 3 เดือนที่ผ่านมาท่านได้มีการออกกำลังกายเป็นประจำหรือไม่ (มีการเคลื่อนไหวร่างกายอย่างต่อเนื่องเป็นเวลาอย่างน้อย 30 นาที)
 ใช่ สัปดาห์ละ.....วัน ไม่ใช่ (ข้ามไปข้อ 21)
20. ในระยะ 3 เดือนที่ผ่านมาท่านออกกำลังกายประเภทใด
 วิ่ง เต้นแอโรบิก ว่ายน้ำ
 เล่นกีฬา อื่นๆ ระบุ.....
21. อาหารประเภทใดที่ท่านชอบรับประทานมากที่สุด (ในระยะ 3 เดือนที่ผ่านมา)
 เนื้อสัตว์ อาหารทะเล ผักและผลไม้
 ธัญญาพืช (ถั่ว งา ข้าวโพด ข้าวฟ่าง) อาหารกระป๋อง หรืออาหารสำเร็จรูป
 อื่นๆระบุ.....
22. รสชาติของอาหารที่ท่านชอบทานเป็นประจำ (ในระยะ 3 เดือนที่ผ่านมา)
 จืด เปรี้ยว หวาน มัน เค็ม เผ็ด
23. ประเภทเครื่องดื่มที่ท่านชอบดื่มเป็นประจำ (ในระยะ 3 เดือนที่ผ่านมา)
 น้ำชา กาแฟ น้ำอัดลม
 น้ำผลไม้ เครื่องดื่มชูกำลัง (กระทิงแดง, M150 เป็นต้น)
 อื่นๆ ระบุ.....
24. ในระยะ 3 เดือนที่ผ่านมา ท่านไปวัด หรือศาลเจ้า บ่อยหรือไม่
 ไม่เคยไปวัดเลย 1-2 วันต่อสัปดาห์ 3-4 วันต่อสัปดาห์
 5 วันหรือมากกว่า 5 วันต่อสัปดาห์ ทุกวัน
25. ท่านได้อยู่ในวัดหรือศาลเจ้าขณะจูดรูปหรือไม่
 ใช่ อยู่ที่ชั่วโมง..... ท่านอยู่ห่างจากจุดที่จูดรูป.....เมตร
 ไม่มีการจูดรูป

ส่วนที่ 2 ข้อมูลลักษณะของที่อยู่อาศัย

26. ลักษณะที่อยู่อาศัยของท่านในปัจจุบันเป็นอย่างไร
 บ้านเดี่ยว ทาวน์เฮ้าส์ ห้องเช่า ตึกแถวที่ใช้เป็นร้านค้าอย่างเดียว
 ตึกแถวที่มีลักษณะเป็นร้านค้าและบ้านพักในหลังเดียวกัน อื่นๆ ระบุ.....
27. ขนาดของบ้านท่าน กว้าง.....ตร.ม. ยาว.....ตร.ม. หรือมีพื้นที่ประมาณ.....ตารางวา
28. จำนวนชั้นของบ้านท่าน.....ชั้น
29. ท่านใช้อุปกรณ์ชนิดใดที่ใช้ในการหุง ต้มอาหารในบ้านเป็นประจำ
 เตาแก๊ส เตาถ่าน เตาไฟฟ้า
 ไมโครเวฟ อื่นๆ ระบุ.....
30. ในระยะ 3 เดือนที่ผ่านมาท่านเป็นผู้ปรุงอาหารเองหรือไม่
 ปรุงเองทุกวัน ปรุงเองบางครั้ง (3-5 วันต่อสัปดาห์) ไม่ได้ปรุงเอง
31. ท่านใช้เครื่องปรับอากาศในบ้านหรือไม่ ใช่ ไม่ใช่ (ข้ามไปข้อ 32)
32. ท่านใช้เครื่องปรับอากาศในห้องใดของบ้านท่าน (ตอบได้มากกว่า 1 ข้อ)
 ห้องนอน ห้องรับแขก/นั่งเล่น/คูทิว ห้องทำงาน
 ห้องชายของ อื่นๆ ระบุ.....
33. ท่านใช้เครื่องฟอกอากาศในบ้านท่านหรือไม่ ใช่ ไม่ใช่ (ข้ามไปข้อ 34)
34. ท่านใช้เครื่องฟอกอากาศในห้องใดของบ้านท่าน (ตอบได้มากกว่า 1 ข้อ)
 ห้องนอน ห้องรับแขก/นั่งเล่น/คูทิว ห้องทำงาน
 ห้องชายของ อื่นๆ ระบุ.....
35. ท่านใช้พัดลมระบายอากาศในบ้านท่านหรือไม่ ใช่ ไม่ใช่ (ข้ามไปข้อ 36)
36. ท่านใช้พัดลมระบายอากาศในห้องใดของบ้านท่าน (ตอบได้มากกว่า 1 ข้อ)
 ห้องนอน ห้องรับแขก/นั่งเล่น/คูทิว ห้องทำงาน
 ห้องชายของ อื่นๆ ระบุ.....
37. บ้านท่านมีหน้าต่างในที่ใดบ้าง และมีจำนวนกี่บาน (ตอบได้มากกว่า 1 ข้อ)
 ห้องนอน จำนวน.....บาน ห้องรับแขก/นั่งเล่น/คูทิว จำนวน.....บาน
 ห้องทำงาน จำนวน.....บาน ห้องชายของ จำนวน.....บาน
 ชั้นบนของบ้าน จำนวน.....บาน อื่นๆ ระบุ.....

38. ในระยะ 3 เดือนที่ผ่านมา ท่านมีการจุดยากันยุงชนิดใดในบ้านหรือไม่

ใช่ ท่านจุดยากันยุงประมาณ.....นาทิจำชั่วโมง

ท่านนั่งห่างจากตำแหน่งที่จุดประมาณเมตร

ไม่ใช่

ส่วนที่ 3 การใช้รูปในบ้าน

39. ท่านได้มีการจุดรูปภายในบ้านหรือไม่

มีการจุดรูปครั้งต่อวัน ครั้งละ.....ดอก

ไม่มีการจุดรูปในบ้าน(เสร็จสิ้นการตอบแบบสอบถามไม่ต้องทำข้อต่อไป)

40. ชนิดของรูปที่ท่านใช้จุดในบ้าน

รูปแท่งไม้ไผ่ยาว



รูปแท่งไม้ไผ่สั้น

รูปแท่งไม้มีก้านไม้ไผ่



รูปกำยาน หรือรูปหอม



รูปขด หรือรูปกันยุง



รูปไฟฟ้า



41. ท่านจุดรูปบ่อยหรือไม่

1-2 วันต่อสัปดาห์

3-4 วันต่อสัปดาห์

5 วันหรือมากกว่า 5 วันต่อสัปดาห์

ทุกวัน

42. ท่านจุดธูปในช่วงเวลาใดในแต่ละวัน ตั้งแต่เวลา.....น. ถึง.....น.
43. ท่านจุดธูปในบริเวณใดของบ้าน
 ห้องนอน ห้องรับแขก/นั่งเล่น/คูทีวี ห้องทำงาน
 ห้องชายของ ชั้นบนของบ้าน อื่นๆ ระบุ.....
44. ท่านได้นั่งอยู่ในบริเวณที่จุดธูปในช่วงที่มีการจุดธูปหรือไม่
 ใช่-ตลอดช่วงเวลาที่มีการจุดธูป ใช่-แต่ไม่ตลอดช่วงเวลาที่มีการจุดธูป
 ไม่เคยอยู่ในบริเวณที่มีการจุดธูป อื่นๆ ระบุ.....
45. ช่วงเวลาที่จุดธูปท่านได้กลิ่นควันธูปหรือไม่
 ได้กลิ่นควันธูปตลอดช่วงเวลาที่มีการจุดธูป
 ได้กลิ่นธูป-แต่ไม่ตลอดช่วงเวลาที่มีการจุดธูป
 ไม่ได้กลิ่นควันธูปเลย อื่นๆ ระบุ.....
46. ในช่วงเวลาจุดธูป ท่านนั่งห่างจากตำแหน่งที่จุดประมาณเมตร
47. ท่านจุดธูปเป็นประจำแบบนี้มานานเท่าใด.....ปี
48. ข้อเสนอแนะอื่นๆ

.....

.....

APPENDIX C

Characteristics of the participants who loss to follow-up after 1-year follow-up (n=32)

Demographic	Incense smoke exposure				p-value
	Total (n=32)	non- exposed (n=8)	non-daily exposed (n=13)	daily exposed (n=11)	
Age (years), mean ±SD (min=38, max =68)	55.81±7.2	57.1±6.7	54.9±7.2	56.0±7.9	0.785 ^b
Gender, n (%)					0.49 ^d
Male	5(15.6%)	1(12.5%)	1(7.7%)	3(27.3%)	
female	27(84.4%)	7(87.5%)	12(92.3%)	8(72.7%)	
Body Mass Index (kg/m ²), mean±SD (min=17.6, max=31.1)	24.04±3.0	24.1±2.3	24.1±3.0	23.9±3.6	0.982 ^b
Education, n (%)					0.39 ^d
Uneducated	7(21.9%)	0(0.0%)	3(23.1%)	4(36.4%)	
High school	22(68.8%)	7(87.5%)	9(69.2%)	6(54.5%)	
Bachelor and more	3(9.4%)	1(12.5%)	1(7.7%)	1(9.1%)	
Current occupation					0.063 ^d
Agricultural / Laborer/ Housewife	8(25.0%)	4(50.0%)	3(23.1%)	1(9.1%)	
Merchant and trader	23(71.9%)	3(37.5%)	10(76.9%)	10(90.9%)	
Government / Company employee	1(3.1%)	1(12.5%)	-	-	

(Continued)

Demographic	Incense smoke exposure				p-value
	Total (n=32)	non- exposed (n=8)	non-daily exposed (n=13)	daily exposed (n=11)	
Past Medical, n (%)	11(34.4%)	3(37.5%)	4(30.8%)	4(36.4%)	1.00 ^d
Diabetes	-	-	-	-	-
Hypertension	9(28.1%)	2(25.0%)	3(23.1%)	4(36.4%)	0.88 ^d
Cardiovascular	1(3.1%)	-	1(7.7%)	-	1.00 ^d
Respiratory	1(3.1%)	1(12.5%)	-	-	0.25 ^d
Dyslipidemia	3(9.4%)	-	2(15.4%)	1(9.1%)	0.77 ^d
Member history of cardiovascular and stroke disease, n (%)	5(15.6%)	2(25.0%)	1(7.7%)	2(18.2%)	0.585 ^d
Smoking, n (%)	1(3.1%)	-	1(7.7%)	-	1.00 ^d
Alcohol consumption, n (%)	5(15.6%)	-	2(15.4%)	3(27.3%)	0.32 ^d
Physical activity, n (%)	25(78.1%)	8(100%)	13(100%)	11(100%)	0.138 ^d

^bOneway-ANOVA, ^dFisher's exact test

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Khon Kean research Journal, 3(1), January - April 2015, 69-82.



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