Original Article

The Relationship between Household Particulate Matter and an Increase of Carotid Intima-Media Thickness [CIMT]: A One-Year Follow-Up Study

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Background: Outdoor particulate matter [PM] has been associated with cardiovascular [CVD] morbidity and mortality. Epidemiological and experimental studies confirmed an association between outdoor PM and carotid artery intima-media thickness [CIMT], which can potentially lead to progression of CVD. However, evidence on the effects of indoor PM on the CVD system is still limited.

Objective: To examine the association of long-term exposure to household indoor PM of less than 10 micrometer [PM_{10}] and increased carotid intima-media thickness [CIMT] of the common carotid artery [CCA].

Materials and Methods: A one-year follow-up study was conducted in 104 households located in the central city of Sakon Nakhon province, Thailand. Clinical testing and a carotid artery ultrasound of participants were undertaken between September 2016 and September 2017. Household PM_{10} concentrations, temperature, and relative humidity [RH] were measured inside the participants' home during the wet and dry seasons.

Results: The average of PM_{10} concentrations was 24.2±11.2 μg/m^3. Mean and maximum CIMT at CCA was different between baseline and follow-up (p-value <0.001). Logistics regression models showed that an increase of 1 μg/m^3 average household PM_{10} concentrations were associated with 6% increased risk of increased maximum CIMT of CCA (AOR 1.06; 95% CI 1.02 to 1.11).

Conclusion: Household PM_{10} concentration is associated with increased CIMT of CCA. This finding suggests that household PM_{10} may be a risk factor for CVD morbidity and mortality.

Keywords: Household air pollution, PM less than 10 micrometer [PM_{10}], Carotid intima-media thickness [CIMT], Common carotid artery [CCA], Cardiovascular disease [CVD]

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According to the World Health Organization, indoor air pollution contributes to 4.3 million premature deaths including cerebrovascular diseases, ischemic heart disease, chronic obstructive pulmonary disease, and acute lower respiratory disease[1,2]. The air within homes and other buildings is a greater cause for concern than outdoor air because people spend more than 90 percent of their time indoors[3]. Air pollution is linked to cardiovascular [CVD] mortality and morbidity[4]. A positive relationship between particulate matter [PM] concentrations and adverse health effects on CVD mortality, accelerated atherosclerosis, vascular inflammation and stroke have been found in previous studies[4-7]. A chronic process of atherosclerosis 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[8-10].

A few studies observed significant associations between outdoor PM and the degree of atherosclerosis measured by carotid artery intima-media thickness [CIMT][8,11]. CIMT is used to identify the level of atherosclerosis to estimate CVD risk in the population's future[12,13]. It is a predictor of CVD events and has been linked to myocardial infarction, stroke, and blood pressure. Long-term exposure to PM showed

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an association with an increase of CIMT around 16.79 micrometer and 4.13 μm for an increase of 10 μg/m^3 in PM less than 2.5 micrometer [$PM_{2.5}$] and PM less than 10 micrometer [PM_{10}] respectively. The finding suggested that an association between higher PM exposure and increased CIMT resulted from the processes of cumulative atherogenesis[11].

An investigation of indoor PM and CIMT has suggested a connection. For example, the effect of chronic exposure to cooking biomass fuel presented strong associations with increased CIMT and a higher prevalence of atherosclerotic plaque[14]. A prospective cohort study in the United States reported that long-term residential $PM_{2.5}$ exposure was associated with increased IMT progression of 14 micrometer/year[15]. However, there is no study on the effect of indoor PM concentration and progression of CIMT in Thailand. Therefore, a current one-year cohort investigated an association between household indoor PM and increased CIMT by carotid artery ultrasound, which can potentially lead to earlier progression to atherosclerosis and possible CVD and stroke.

Materials and Methods

Study design and participants

A one-year longitudinal study was conducted in a main street of the inner city of Sakon Nakhon province, in the northeast of Thailand between September 2016 and September 2017. This main street was purposively selected because of its high mortality rate of ischemic heart disease and stroke. In addition, the present study was restricted to only one main street to minimize outdoor and traffic air pollution effects on CIMT[9].

One participant from each household aged above 30 years who spent the most time (more than eight hours) in the house was invited to participate in the study[16]. Trained health volunteers conducted a face-to-face interview for demographic status including age, gender, body mass index [BMI], smoking status, and drinking status. Underlying diseases including CVD (yes/no), diabetes (yes/no), hypertension (yes/no), and respiratory problems (yes/no) were investigated during the interview. One hundred four participants from 104 households completed the CIMT assessment at the baseline in September 2016 and followed-up in September 2017. The present research was approved by the Ethics Review Committee for Research involving human subjects, Health Science Group, Chulalongkorn University (COA No. 146/2016). All participants submitted their written consent forms before being enrolled in the present study.

Exposure assessment

Household indoor particulate matter concentrations (PM_{10}), temperature, and relative humidity [RH] were collected inside participants' homes during the dry season (November to December 2016) and wet season (June to July 2017). Indoor PM_{10} samples were collected continuously for 24 hours following the National Institution's Occupational Safety And Health Guideline[17]. Briefly, a personal sampling pump (SKC 224-PCXR8 model) connected with an 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/minute. 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 to 1.5 m above the floor. An average of temperature and RH were reported.

Carotid artery ultrasound and clinical assessment

All participants underwent clinical assessment and CIMT ultrasounds at Sakon Nakhon Hospital. Blood samples was collected by professional nurses on the same day as the CIMT ultrasound and were analyzed for high-density lipoprotein [HDL] and low-density lipoprotein [LDL]. Participants' weight and height were measured by an automatic balance to obtain BMI. An OMRON blood pressure monitor with automatic cuff inflation and deflation was used to measure blood pressure and heart rates according to standard protocol. An average baseline and follow-up of HDL, LDL, BMI, and blood pressure was reported.

CIMT measurements were conducted according to the American College of Cardiology [ACC] and the American Heart Association [AHA] guidelines in the assessment of CVD risk[18]. The measurement was performed using a high-resolution B-mode ultrasound scanner (Toshiba Aplio 300). An adult cardiac 1.8 to 4.8 MHz linear array transducer with a Micro-convex was utilized. The images for offline viewing using Synapse PD-S Viewer Version 1.0 was exported. 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[19]. This was validated against histological specimens as representative of the true thickness of the vessel wall[20], as well as 10 millimeter manual measurements to the bulb from the common carotid on both right and left common carotid arteries [CCA]. 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[14]. Each participant's CIMT levels were measured three times. Their anonymized data were then submitted to a radiologist who produced an average rating of the three measurements for each participant.

Statistical analysis

Multivariable logistic regression was used to estimate the risk of household indoor PM_{10} exposure on an increase of CIMT after a one-year follow-up (yes/no). For PM_{10}, the association was reported for 1 $\mu g/m^3$ increase of an average concentration between wet and dry seasons. Continuous variables were expressed as mean ± standard deviation [SD] and median (interquartile range [IQR]). Categorical variables were presented by percentage (%). The paired and independent t-test was analyzed to compare the difference of CIMT between baseline and follow-up and indoor environment parameters. Covariate factors in the model were selected by recommended factors of the AHA guidelines for CIMT[21,22] including age, HDL, LDL, and systolic blood pressure [SBP]. All analyses were performed using IBM SPSS Statistical software for Windows (IBM SPSS, version 22, Chicago, IL, USA). The *p*-value less than 0.05 defined as statistical significance.

Results

Participants' characteristics

The present study was carried out on 104 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. Most of the study population was female (75.0%) and the median reported age (IQR) was 58 (50 to 62.75) years old. The average BMI was 23.74±3.2. Most of them (54.8%) had completed high school. Forty-five percent of them reported an underlying disease, which was predominantly hypertension (23.1%). The average baseline and follow-up blood analysis, HDL, LDL, and SBP were 60.9±18.6 mg/dl, 138.0±37.3 mg/dl, and 133.5±21.3 mmHg, respectively (Table 1).

Carotid intima-media thickness

Table 2 shows that the follow-up CIMT was higher than baseline CIMT (p<0.001). At baseline, mean thickness of carotid intima-media at CCA was 0.71±0.15 mm while maximum CIMT was 0.89±0.18 mm. The mean and maximum follow-up CIMT was 0.74±0.15 and 0.93±0.18 mm respectively. For left

Table 1. General information and medical history of the participants (n = 104)

Demographic	Total (n = 104)
Age (years), median (IQR)	58 (50 to 62.75)
Gender, n (%)	
Female	78 (75.0)
Body mass index [BMI] (kg/m^3), mean ± SD	23.74±3.2
Education, n (%)	
Uneducated	22 (21.2)
High school	57 (54.8)
Bachelor and more	25 (24.0)
Current occupation, n (%)	
Agricultural/laborer/housewife	18 (17.3)
Merchant and trader	79 (76.0)
Government/company employee	7 (6.7)
Underlying disease, n (%)	47 (45.2)
Diabetes	11 (10.6)
Hypertension	24 (23.1)
Cardiovascular [CVD]	4 (3.8)
Respiratory	8 (7.7)
Current smoking (yes), n (%)	1 (1.0)
Current alcohol consumption, n (%)	15 (14.4)
Regular exercise, n (%)	80 (76.9)
Blood test, mean ± SD	
High density lipoprotein [HDL] (mg/dl)	60.9±18.6
Low density lipoprotein [LDL]: (mg/dl.)	138.0±37.3
Systolic blood pressure [SBP] (mm Hg)	133.5±21.3

Table 2. Increased carotid intima-media thickness [CIMT] from baseline to follow-up (n = 104)

CIMT	Total, mean ± SD		*p*-value	Increased CIMT after 1 year follow-up, n (%)
	Baseline	Follow-up		
CCA (mm)				
Mean	0.71±0.15	0.74±0.15	0.004*	51 (49.0)
Maximum	0.89±0.18	0.93±0.18	<0.001**	50 (48.1)
RCCA (mm)				
Mean	0.72±0.17	0.75±0.17	<0.001**	50 (48.1)
Maximum	0.89±0.19	0.93±0.21	<0.001**	49 (47.1)
LCCA (mm)				
Mean	0.70±0.18	0.73±0.17	<0.001**	52 (50.0)
Maximum	0.88±0.21	0.93±0.21	<0.001**	59 (56.7)

CCA = common carotid artery; RCCA = right common carotid artery; LCCA = left common carotid artery

* p<0.05, ** p<0.001

and right CCA, the authors observed that the baseline of mean LCCA (0.70±0.18 mm) and maximum LCCA (0.88±0.21 mm) were less than mean RCCA (0.72±0.17 mm) and maximum RCCA (0.89±0.19 mm). After a year of follow-up, the authors found that maximum CIMT of LCCA was equal to RCCA (0.93±0.21 mm). Regarding the overall increment of CCA, almost half the participants had increased mean and maximum CCA levels, 48.1% of them were observed as having an increase of mean RCCA whereas 50% had an increase in mean LCCA. For LCCA, the authors found that most participants (56.7%) had increased LCCA after follow-up. However, 47.1% of them found an increment of RCCA.

Household indoor particulate matter

Table 3 showed a significant difference of PM_{10} concentration, temperature, and humidity between the wet and dry season. The average PM_{10} of the participants was 24.2±11.2 μg/m^3. The authors found PM_{10} in the dry season (31.4±18.0 μg/m^3) was significantly higher than during the wet season (17.1±8.64 μg/m^3), while temperatures and RH during the wet season (28.9±1.18°C and 72.5±5.33%) were higher than the dry season.

According to an increased mean CIMT from baseline to follow-up, the average PM_{10} concentration of those participants whose CIMT had increased (26.59±12.12 μg/m^3) was significantly higher than those who had not increased (21.95±9.83 μg/m^3) (p = 0.034). For maximum CIMT, the average PM_{10} concentration was also significantly higher among those who had increased CIMT (27.18±11.98 μg/m^3) than those who had not (21.48±9.77 μg/m^3) (p = 0.009). An average 24-hours temperature of participants whose mean CIMT had increased was 27.86±1.26°C, which was slightly lower than participants whose levels had not increased 27.99±1.22°C. For RH, an average 24-hours RH was not different among both groups of participants. Regarding temperature and RH, no associations with an increased mean and maximum CIMT (Figure 1) were found.

Table 3. Household indoor assessment of the participants (n = 104)

Parameters	Average mean ± SD	Dry season mean ± SD	Wet season mean ± SD	p-value$^+$
PM_{10} (μg/m^3)	24.2±11.2	31.4±18.0	17.1±8.64	<0.001*
Temperature (°C)	27.9±1.2	26.9±1.99	28.9±1.18	<0.001*
Humidity (%)	65.3±4.0	58.1±5.90	72.5±5.33	<0.001*

PM_{10} = particulate matter less than 10 micrometer

$^+$ Dry and Wet seasons, * p<0.001

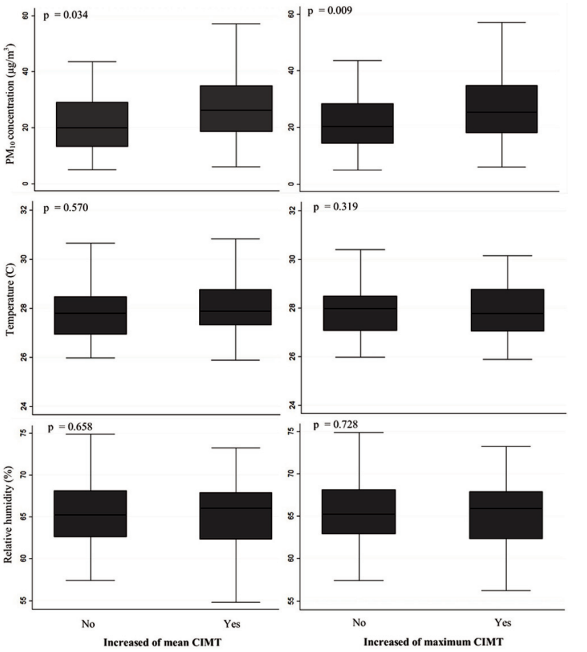

Figure 1. Increased mean and maximum carotid intima-media thickness [CIMT] (Yes/No) according to PM_{10} concentrations (μg/m^3), temperature (°C), and relative humidity (%).

An association between household indoor particulate matter and increased CIMT

Table 4 shows multivariate logistic regression models with an association between an increment of 1 μg/m^3 indoor PM_{10} concentration and risk of increased CIMT after a one-year follow-up. For the unadjusted model, an increase of 1 μg/m^3 average indoor PM_{10} concentration of 1.04 (95% CI 1.00 to 1.08) fold increased odds of increasing mean CCA and 1.05 (95% CI 1.01 to 1.09) fold increased odds of increasing maximum CCA. After adjusting for age, BMI, HDL, LDL, and SBP, the authors could observe a stronger association between average indoor PM_{10} concentrations and maximum CCA but not for the mean CCA. An increase of 1 μg/m^3 average indoor PM_{10} concentrations was associated with 6% increased risk of increased maximum CCA (adjusted odds ratio [AOR] 1.06; 95% CI 1.02 to 1.11). Only maximum LCCA was associated with indoor PM_{10} concentrations. An increase of 1 μg/m^3 average indoor PM_{10} concentrations was 1.06 (95% CI 1.02 to 1.11) fold increased odds of increasing maximum LCCA. However, the mean LCCA was not associated with indoor PM_{10} concentrations (AOR 1.03; 95% CI 0.99 to 1.07). The authors also could not observe a risk to

Table 4. Logistic regression models between household indoor PM_{10} concentrations (μg/m3) and an increased carotid intima-media thickness [CIMT] (yes/no) after 1 year follow-up (n = 104)

Outcome	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	1.04 (1.00 to 1.08)	0.04*	1.04 (1.00 to 1.08)	0.04*
Maximum	1.05 (1.01 to 1.09)	0.01*	1.06 (1.02 to 1.11)	0.007*
Increased LCCA				
Mean	1.03 (0.99 to 1.07)	0.07	1.03 (0.99 to 1.07)	0.09
Maximum	1.06 (1.02 to 1.10)	0.008*	1.06 (1.02 to 1.11)	0.006*
Increased RCCA				
Mean	1.02 (0.98 to 1.06)	0.29	1.02 (0.99 to 1.06)	0.24
Maximum	1.02 (0.99 to 1.06)	0.24	1.03 (0.99 to 1.07)	0.15

OR = odds ratio; AOR = adjusted odds ratio; CCA = common carotid artery; RCCA = right common carotid artery; LCCA = left common carotid artery

[a] Adjusted for factors associated to CIMT in adult: age, body mass index [BMI], high density lipoprotein [HDL], low density lipoprotein [LDL], systolic blood pressure [SBP]; * *p*-value <0.05

indoor PM_{10} concentrations on an increased RCCA (p>0.05).

Discussion

The authors found a positive association between exposures to household indoor PM and an increase of CIMT after a one-year follow-up in the central city of Sakon Nakhon province, Thailand. An average indoor PM_{10} concentration was significantly different among participants whose CCA had increased and those whose CCA had not. The authors observed a risk of increased LCCA associated with indoor PM_{10} concentrations. However, association was not found in the RCCA.

A few studies have investigated the association between indoor PM and CIMT[9,14,15]. CIMT results from the processes of cumulative atherogenesis. CIMT progression is a predictor of atherosclerosis and CVD events[11-13,23,24]. The authors' findings are in agreement with previous studies[11,25,26], which found those on household indoor PM_{10}-increased CIMT. The findings support the statement of the AHA's expert panel regarding the biological mechanisms of the effects of PM on CVD events[27].

The atherosclerotic process accelerates generally on both sides in the presence of traditional risk factors[26]. The authors' results also underscore the importance of measuring both the left and right CCA for assessing the association with indoor PM_{10} exposure. The authors observed that the baseline of mean LCCA and maximum LCCA were less than mean RCCA and maximum RCCA. This finding was contrary to the study of the differences in left and right CIMT and the associated risk factors[28]. It may be a cause of hemodynamic and biochemical changes of person who had different effects on the CIMT depending on the side affected. These relations may be more affected by confounding personal factors[15] and other causes of changes in CIMT[29].

The authors' study found a stronger association between indoor PM_{10} 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 such as age, BMI, HDL, LDL, and SBP, which is associated with progression of CIMT in CCA[21,22,29]. However, mean LCCA was not associated with indoor PM_{10} concentrations and differed from the study of Su et al[26], which reported that the mean LCCA was associated with traffic air pollution (PM_{10}) concentration. Additionally, the authors could not observe any risk of indoor PM_{10} concentration on the increased RCCA. The possible reason to support this finding is in 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). 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)[28]. However, the reason for this phenomenon is not yet clear. The authors need further study to confirm these hypotheses.

The authors conducted PM_{10} inside of all participants' homes depending on the season. The average of PM_{10} was 24.2±11.2 μg/m^3, which were different from the study of indoor/outdoor PM_{10} and $PM_{2.5}$ in Bangkok, Thailand where it was reported that the average of PM_{10} concentration in the living

room was 185±42 μg/m^3[30]. Additionally, the level of household indoor PM_{10} exceeded the level of air quality guidelines (20 μg/m^3 annual mean)[31].

The authors also found a significant difference of PM_{10} concentration, temperature, and RH between the wet and dry season. Household indoor PM_{10} during the dry season (31.4±18.0) was higher than during the wet season (17.1±8.64). PM concentrations were affected by shifting seasons with lowest averages obtained during the rainy season and highest levels of PM during the winter because the air exchange rate was directly correlated with PM concentrations[32]. The major contributor to source apportionment of indoor PM_{10} in residential settings results from various routine activities such as cooking, floor sweeping, presence of people, smoking, and space heating[32].

Some potential limitations might also have an effect on the results. Firstly, the authors' sample size is small; however, despite this limitation, we were still able to detect important differences between household PM_{10} and an increased CIMT. Secondly, the various types of household activities carried out on a routine day such as cooking, floor sweeping, presence of people, smoking, space heating, and incense burning were not collected in the present study. The activities may contribute to the difference of PM concentration. Additionally, only PM_{10} was collected in the present study. Other pollutants including $PM_{2.5}$, carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and total volatile organic compounds [VOCs], which can possibly increase CIMT levels, are suggested for further studies. Lastly, PM concentration in the present study was based on a single sample collection for 24 hours in each season. It may not be a good representative of household PM concentrations. Further study should include a sampling of days and hours. In terms of generalizability, participants of the present study were limited to one main road and one city. Therefore, general characteristics may not be the same as for other Thai populations.

Conclusion

Household indoor PM is associated with increased CIMT of CCA and LCCA but not for CIMT of RCCA. These findings suggest that PM inside the house may be a risk factor for CVD disease morbidity and mortality.

What is already known on this topic?

Air pollution is linked to atherosclerosis, which is a predictor for CVD morbidity and mortality. Several epidemiological and prospective cohort studies found an association between outdoor PM and the degree of atherosclerosis measured by CIMT. Indoor PM predicted by cooking fuel use was associated with an increased CIMT.

What this study adds?

This study suggested that indoor PM_{10} concentration was linked to a progression of CIMT after a one-year follow-up. An increase 1 μg/m^3 of PM_{10} concentration was associated with 6% increased risk of increased maximum CIMT of CCA after adjustments for age, BMI, HDL, LDL, and SBP. The findings support the previous studies on the potential risk of PM and increased CIMT that leads to earlier atherosclerosis and can predict the progression of cardiovascular disease.

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Potential conflicts of interest

The authors declare no conflict of interest.

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