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Original Research

Outdoor and Indoor Air Pollution and Myocardial Infarction among Women in Kaunas, Lithuania: a Case-Control Study

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Abstract

A population-based case-control study among 35–61-year-old women in Kaunas, Lithuania, was conducted in 1997-2005. In total 368 myocardial infarction cases and 725 healthy controls were interviewed using a standardized questionnaire containing information on gas stove use, environmental tobacco smoke, high traffic, household crowding, traditional ischemic heart disease risk factors, socioeconomic position (education, occupation), and perceived stress. Nitrogen dioxide was selected for analysis as an indicator of traffic-related air pollution. In the fully adjusted model, the Odds ratio of outdoor nitrogen dioxide pollution exposure for the third tertile was 1.10; 95% CI 0.57-2.15 as compared to the first tertile. The adjusted OR for gas stove usage was 1.20; 95% CI 0.72-1.98, environmental tobacco smoke 1.28; 95% CI 0.71-1.66. The study has demonstrated that outdoor and indoor air pollution exposure might have trace associations with coronary heart disease.

Keywords: air pollution, myocardial infarction, case-control study, risk evaluation, logistic regression

Introduction

Investigations in recent years have confirmed the associations between ambient and indoor air pollution and health outcomes [1]. Studies on long-term air pollution by nitrogen dioxide (NO_2) found a slight increase in coronary heart disease and myocardial infarction risk [2, 3]. The studies on ambient air pollution and coronary atherosclerosis are based on long-term observations, thus the epidemiological evidence of the associations is unequivocal [4]. However, the associations between health outcomes and gender in relation to air pollution by NO_2 as indicator of exhaust emissions from vehicles is controversial. The study on long-term NO_2 effect on myocardial infarction risk among men indicates consistent associations [5]. But the study on residence close to high traffic and prevalence

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of coronary heart disease found stronger associations for residents younger than 60 years and no effect for women [6].

Because of the increasing emphasis on environmental protection, indoor air pollution has become of great concern. Major sources of indoor air pollution are by-products of combustion (carbon monoxide (CO), carbon dioxide (CO_2), nitrogen oxides (NO_x), environmental tobacco smoke (containing 4,000 chemicals), and bioaresols [7]. Using a gas stove to is hazardous due to the possible occurrence of incomplete combustion (increase of CO emissions and/or soot formation). Both NO_2 and nitrogen monoxide (NO) generally rise with thermal input or load height to flame length ratio [7].

Passive smoking is the inhalation of smoke, called secondhand smoke or environmental tobacco smoke. Sufficient evidence is reported in epidemiological research to support a causal association between secondhand smoking exposure and increased risk of cardiovascular morbidity among both men and women [8]. The risk of developing an acute cardiac syndrome or chronic lifetime coronary events is at least 30 percent. The risk for current smokers and ex-smokers is even higher.

Though the traditional ischemic heart disease risk factors were disclosed many decades ago, the incidence rates for myocardial infarction are not lowering as quickly as public health professionals could expect. The studies revealing the non-traditional risk factors in the etiology of myocardial infarction are of great importance.

We investigated the associations between outdoor (ambient NO_2 exposure) and indoor (gas stoves usage, environmental tobacco smoke) air pollution and myocardial infarction among 34–61-year-old women in Kaunas, Lithuania, taking into account the possible effects from age, the traditional ischemic heart disease risk factors (arterial hypertension, increased body mass index, low physical activity), education level, socioeconomic position, household crowding, perceived stress, sleep disorders, family stress, and high residential traffic.

Subjects and Methods

The effect of ambient NO_2 pollution on the risk of first-time myocardial infarction within the municipality of Kaunas, the second largest city in Lithuania, was analyzed using the population-based case-control study design. The study base population comprised all 35–61-year-old women residing in the city. All hospitalized women with a first-time myocardial infarction (I21 code of the 10th version of the International Classification of Diseases – ICD-10) from 1997 to 2005 were eligible for the study. Altogether, 421 first-time myocardial infarction cases were registered and 368 (87.4%) were interviewed in the hospital during the first week of hospitalization. Controls without signs of ischemic heart disease were randomly selected from the study base, stratified by age and gender. Altogether, 725 controls were interviewed (response rate 71.2%).

The NO_2 measurements were taken from 12 municipal monitoring posts in each residential district of the city. The monitors were located outside, primarily near schools and kindergartens. We used all the daily measurements of NO_2 (photometric method) to assess the annual mean residential exposure and grouped the residential districts according to pollutant concentrations into three categories (tertiles). The mean NO_2 concentration was 25.0 (SD 5.4) μg/m^3 for the study period. The group of the participants with exposure in the first tertile was used as the reference category (low exposure area with NO_2 concentrations bellow 15 μg/m^3). Three areas with different exposure were determined: medium exposure area (15-35 μg/m^3) and high exposure area, where NO_2 concentrations exceeded 35 μg/m^3.

Trained physicians interviewed the cases and controls using identical standardized questionnaires, including information on demographic, socioeconomic, psychological factors, health behaviors, and residence history. High residential traffic exposure was evaluated as living less than 100 m from a major road. Household cooking was assessed as by usage of electrical or gas stoves. Crowding was defined as less than half a room per person. Arterial blood pressure measured as ≥160/95 mm Hg was defined as arterial hypertension. Body mass index was calculated as weight (kg) divided by height squared (m^2). Smoking was assessed as passive (secondhand), current, and former (smoking cessation more than 2 years). The respondent's perceived stress was measured by a set of seven questions, adapted from L. Reeder stress scale [9]. Education was categorized into university, college, secondary, and incomplete secondary. The occupations were categorized into white-collar and blue-collar. Marital stress was measured by a set of questions, evaluating the frequency and severity of conflicts and presence of permanent tension. Physical activity was assessed as "high" if it was performed more often than weekly.

The software SPSS 13.0 for Windows was used in statistical analysis. The logistic regression analysis was performed, including data on outdoor (ambient NO_2 levels) and indoor (gas stove usage, smoking) air pollution and all other risk factors, adjusting for age. All study variables (NO_2>35 μg/m^3, gas stove, smoking, high traffic, crowding, education, occupation, arterial blood pressure, body mass index, physical activity, perceived stress, insomnia, family stress) were included simultaneously in the final model. The results of the models are presented as adjusted odds ratios (OR) and their 95% confidence intervals (CI).

Results

Table 1 presents the distribution of the potential myocardial infarction risk factors among 35–61-year-old women in Kaunas. More myocardial infarction cases lived in the third NO_2 tertile, used gas stoves, and smoked (including passive smoking) as compared to controls.

The results from the logistic regression analysis are presented in Table 2. We found a tendency in myocardial

Table 1. Distribution of potential myocardial infarction risk factors among cases and controls.

Risk factors	Cases (368)		Controls (725)	
	N	%	N	%
Age				
< 50 yrs	40	10.9	94	13.0
50- <54 yrs	45	12.2	114	15.7
54- <60 yrs	109	29.6	229	31.6
60-61 yrs	174	47.3	288	39.7
Occupation				
white-collar	174	47.9	407	57.3
blue-collar	189	52.1	303	42.7
Education				
Higher and secondary	287	78.0	618	85.2
Incomplete secondary	81	22.0	107	14.8
Smoking				
non-smoker	253	68.8	561	77.4
passive smoker	58	15.7	100	13.8
current smoker	51	13.9	50	6.9
former smoker	6	1.6	14	1.9
Blood pressure				
< 140/90 mmHg	137	37.2	488	67.3
≥140/90-<160/95 mm Hg	134	36.4	157	21.7
≥160/95 mm Hg	97	26.4	80	11.0
Body mass index				
normal (20.1-25.0 kg/m^2)	98	26.6	291	40.2
increased (25.1-30.0 kg/m^2)	123	33.4	230	31.7
obesity (>30.0 kg/m^2)	147	39.9	204	28.1
Physical activity				
weekly	26	7.1	100	13.8
occasional	115	31.3	198	27.3
never	227	61.7	427	58.9
Family stress				
no	310	84.2	655	90.3
yes	58	15.8	70	9.7
Psychological status				
no stress	73	19.8	417	57.5
moderate stress	195	53.0	243	33.5
severe stress	100	27.2	65	9,0
Insomnia				
no	248	67.4	596	82.2
yes	120	32.6	129	17.8
Crowding				
≥ 0.5 room /person	355	96.5	708	97.7
< 0.5 room /person	13	3.5	17	2.3
High traffic				
no	213	57.9	454	62.6
yes	155	42.1	271	37.4
Gas stove				
no (electrical)	31	8.4	74	10.2
yes	337	91.6	651	89.8
NO_2 exposure				
1st tertile	325	88.3	625	86.2
2nd tertile	25	6.8	69	9.5
3rd tertile	18	4.9	31	4.3

infraction risk increase for ambient air pollution by NO_2 exceeding 35 μg/m^3, gas stove usage and environmental tobacco smoke. Crowding (<0.5 rooms per person) and high traffic showed a tendency for increase in myocardial infarction risk. While the associations with active smoking, obesity, arterial hypertension, perceived stress, insomnia, and low physical activity remained significant in the final model. Socioeconomic factors (education, occupation), as well as family stress lost statistical significance in the final model.

Discussion

The associations between outdoor and indoor air pollution and first non-fatal myocardial infarction among 35–61-year-old women in Kaunas, Lithuania were investigated. NO_2 as a measure of outdoor, mainly traffic-generated long-term air pollution and kitchen gas stove usage and second-hand smoke were chosen as indicators of indoor air pollution.

A tendency for increase in the first myocardial infarction risk in the third versus first tertile of ambient NO_2 exposure level among 35–61-year-old women was found. In 2009 a published review found moderate evidence that long-term exposure to an annual mean below 40 μg NO_2/m^3 is associated with adverse health effects [1], and some studies report no effects of long-term exposure to NO_2 on myocardial infarction risk [2, 3], especially non-fatal [10] hospital admissions from coronary heart disease [11].

Significant human exposure to NO_2 can occur in indoor settings [12]. Gas-burning settings, such as unvented furnaces and stoves, are the principal sources of indoor NO_2,

Table 2. Logistic regression model in the associations between myocardial infarction and outdoor, indoor air pollution, and other risk factors among 35–61-year-old women.

Risk factors	Age – adjusted OR		Fully – adjusted OR*	
	OR	95% CI	OR	95% CI
NO_2 > 35 μg/m^3	1.15	0.63-2.09	1.10	0.57-2.15
Gas stove	1.24	0.78-1.96	1.20	0.72-1.98
Passive smoking	1.29	0.89-1.86	1.28	0.71-1.66
Active smoking	2.56	1.67-3.92	2.98	1.84-4.81
Crowding (<0.5 room/p)	1.53	0.73-3.20	1.14	0.49-2.65
High traffic	1.21	0.94-1.57	1.22	0.91-1.63
Arterial hypertension	4.23	2.97-6.02	4.23	2.85-6.28
Low physical activity	2.08	1.32-3.26	2.09	1.25-3.48
Perceived stress	4.01	2.83-5.69	3.91	2.63-5.83
Insomnia	2.21	1.65-2.96	1.49	1.06-2.08
Family stress	1.73	1.19-2.52	1.41	0.91-2.83
Education (incomplete secondary)	1.55	1.12-2.14	1.48	0.98-2.24
Occupation (blue-collar)	1.42	1.10-1.83	1.13	0.82-1.56

*Adjusted for all the variables in the table.

although kerosene space heaters and tobacco smoke may also play a role.

The underlying biological mechanisms linking long-term exposure to NO_2 with cardiovascular disease is still a subject of research. Heart rate variability is a measure of cardiac autonomic tone. A negative association between exposure to ambient NO_2 and heart rate variability was found in women [13], especially with underlying cardiovascular problems.

Mechanisms involving oxidative stress and inflammation have been proposed to explain associations of ambient air pollution with cardiovascular morbidity and mortality. Recent investigations have confirmed that traffic-related air pollutants are associated with increased platelet activation and decreased erythrocyte antioxidant enzyme activity [14], and increased systemic inflammation marker (serum interleukin-6) levels [15].

The extent to which ambient air pollution affects the individual's coronary arteries depends on pollution concentrations, exposure duration, and many underlying factors such as genetic susceptibility, psychological state, socioeconomic position, nutritional habits, food additive usage, level of physical activity, leisure time, and even the availability for recreational weekends. As we found no consistent associations between NO_2 exposure and first myocardial infarction among 35–61-year-old women in Kaunas, probably some of these factors were not taken into account. Other potential limitations of the study arise from the possible misclassification of exposure. We estimated exposure to pollutants using average measurements of the entire residential district, but this possible bias is inherent to all epidemiological studies. Exposure levels were estimated at the home address, whereas employed women do not spend all of their time at home.

The associations between indoor air pollution and myocardial infarction taking into account passive or secondhand smoke and use of gas stoves in household were investigated among women in our study. Previously our study on myocardial infarction risk factors among men indicated that gas stove usage was not associated with a first myocardial infarction risk increase.

Because more than half of all households in the United States use gas, gas appliances or gas-fueled cooking and unvented kerosene heaters are the primary source of indoor CO and NO_2 [16]. Elevated indoor air pollution levels are more prevalent in lower-income housing developments because of poor ventilation, small apartment size, and frequent use of gas stoves for supplemental heating. Only 0.5% of the indoor environment is being affected by outdoor CO concentration [17]. The acute health risks of CO and NO_2 exposure have been well established in the general population and for certain high-risk groups, including infants, the elderly and people with heart disease or asthma [18]. The Federal-Provincial Advisory Committee on Environmental and Occupational Health recommended that acceptable short-term exposure ranges for CO in residential indoor air not exceed an average concentration of 11 ppm over 8 hours and 25 ppm over 1 hour [19]. However, CO levels of about 100 ppm have been detected in the kitchens of some houses immediately after gas stoves were used for cooking. The level of 70 ppm is equivalent to a carboxyhemoglobin concentration in blood of 10% [20]. In non-smokers the concentration of carboxyhemoglobin is normally around 1% of

total hemoglobin [21]. Blood carboxyhemoglobin levels of 2.5-4% were associated with decreased short-term maximal exercise duration in young healthy men and levels of 2.7-5.1% in patients with ischemic heart disease due to increased chest pain (angina) [22].

The difficulty in determining whether very low levels of CO produce long-term health effects arises from the fact that these levels generally do not produce consistent symptoms. People with blood carboxyhemoglobin levels of around 5% or bellow generally experience no symptoms; this is true to cigarette smokers in whom the carboxyhemoglobin concentrations may reach 12% [22]. Studies of exposure to pollutants found in the indoor domestic environment are increasing, but results on health effects of non-fatal, long-term, low-dose indoor exposure to CO and other pollutants are still inconclusive and too infrequently documented [23]. On the other hand, epidemiological studies have shown that levels of CO in the air were mildly correlated with mortality and hospital admissions for coronary heart disease [11]. The proposed mechanism underlying this correlation includes effects on inflammation, coagulation factors, and oxidative stress, which could increase the risk of coronary events and atherosclerosis [24]. Chronic low concentrations of CO poisoning result in generalized tissue hypoxia. Preexisting cardiovascular disease makes tissue hypoxia more likely to present clinically, and may result in myocardial infarction [21]. Acute CO exposure seems to modify cardiac autonomic function as measured by heart rate variability in patients with stable coronary artery disease [25]. Myocardial injury (defined by cardiac troponin and creatine kinaze-MB levels) was found in 37% of patients with moderate-to-severe CO poisoning, and 38% of those with myocardial injury had died at a median follow-up of 7.6 years [26, 27]. In our study we found a tendency for increase in myocardial infarction risk among women using gas stoves as compared to electrical ones. Those women might have been exposed to levels of CO in excess of those found in ambient air.

CO poisoning is almost entirely preventable by the correct installation, maintenance, and operation of devices that emit CO [28]. In Lithuania the majority of gas stoves are unvented and it is a national public health problem. The use of gas assemblers is ineffective because of improper installation and maintenance (filters are not being changed over the years). It has been estimated that appropriate use of CO detectors (also called CO alarms) could prevent at least half of all deaths attributable to CO poisoning [29].

Difficulty regarding regulation of domestic indoor environments is its inherent privacy. Individual behavior patterns and activities have the greatest influence on pollutant levels in indoor air. Monitoring levels of pollutants in the home and ensuring regulation is a charge to encourage the reduction of pollutant levels in indoor domestic air. In the present study, tendencies toward elevated odds ratios among household gas stove users for the first myocardial infarction among 35–61-year-old women were observed. The possible limitation of the study occurs from the fact that the majority of myocardial infarction cases were employed women, probably spending only a little daytime near gas stoves on working days. The study among older retired women probably would show more consistent associations. Nevertheless, the results of our study show possible myocardial injury risk factors in the household environment.

Exposure to secondhand smoke, a major air pollutant, is linked to increased cardiovascular morbidity and mortality, including cardiac arrhythmias. A vast number of studies on the unfavorable effects of secondhand smoke have been conducted, the majority of which evaluated longitudinal epidemiological data. Although limited, animal and human experimental studies that assess the acute and short-term effects of exposure to secondhand smoke indicate a number of pathophysiological mechanisms through which the deterious effects of secondhand smoke may arise [30]. Impaired cardiac autonomic function, indexed by reduced heart rate variability, may represent the underlying cause. There is some evidence that small exposures to tobacco smoke such as with secondhand smoke or smoking 1 or 2 cigarettes a day substantially affect heart disease risk because of the nonlinear dose-response effects on platelet activation, endothelian dysfunction, and other factors [31]. New studies have found significant reductions in acute myocardial infarction admissions associated with the implementation of smoke-free laws [32].

Poor-quality housing and overcrowding seem to have a direct relationship to poor mental health, development delay, and heart disease [33]. Though crowding (more than one person per room) was not related to myocardial infarction in a former communist country [34], material factors, including household conditions, contributed more to educational differences in incidences of acute myocardial infarction than behavioral factors in the Globe study [35].

Conclusions

Tendencies in first myocardial infarction risk increase related to outdoor and indoor air pollution exposure among 35–64-year-old women were found in Kaunas. Though air pollution exposure evaluation methods used were crude and routine, the study results indicate the necessity of more precise studies on the relationship between myocardial infarction and outdoor and indoor air pollution. Direct personal measurements of nitrogen dioxide and carbon monoxide should be implemented.

Difficulties in outdoor and indoor air pollution exposure assessment should be solved and new methodologies in household air pollution exposure measurement should be implemented.

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