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ORIGINAL RESEARCH

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Sex-specific association between secondhand smoke exposure and acute coronary syndrome using KoGES epidemiological cohort data

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Abstract

While numerous studies highlight sex differences in the adverse effects of direct smoking, research on sex-specific risks from secondhand smoking (SHS)-induced diseases is sparse. We hypothesized that acute coronary syndrome (ACS) could be influenced by SHS exposure, with effects varying by sex. We utilized the Korea Genome Epidemiology Study (KoGES) data which was collected starting from 2001 and follow-up for 19 years, to conduct our study. Our primary exposure was secondhand smoke (SHS), and the main outcome of interest was acute coronary syndrome (ACS). We conducted Cox proportional logistic regression analysis and performed an interaction analysis to investigate the interaction between SHS and sex in relation to ACS incidence. SHS exposure was not associated with an increased risk of ACS, exhibiting a hazard ratio (HR) of 1.44 (95% confidence interval (CI), 0.88–1.49) in comparison to the non-exposed group. In the interaction analysis between SHS exposure and sex on the incidence of ACS, in females, SHS was not a risk factor for ACS (HR: 0.97 (95% CI: 0.70–1.35)), whereas, in males, SHS was a significant risk factor for ACS (HR: 1.63 (1.02–2.60). We identified a significant association between SHS exposure and an increased risk of ACS in males. Our findings emphasize the importance of minimizing secondhand smoke exposure, particularly among the male demographic.

Keywords

Secondhand smoke; Sex; Acute coronary syndrome

1. Introduction

Acute coronary syndrome (ACS) signifies a spectrum of conditions that reflect a grave obstruction to coronary blood flow, embodying unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI) [1]. Representing a foremost cause of morbidity and mortality worldwide, ACS imposes a significant public health burden [2]. According to the Global Burden of Disease Study, ischemic heart disease, in which ACS is a major contributor, was responsible for an estimated 9 million deaths in 2021, symbolizing a striking 15.5% rise in the past decade [3]. ACS is not only a predominant driver of sudden cardiac events but is also critically implicated in the subsequent development of heart failure and related complications [4, 5]. The urgency of actively addressing Acute Coronary Syndrome (ACS) is underscored by its substantial impact on public health, necessitating directed efforts to reduce its incidence and enhance management strategies, particularly in populations bearing elevated risks due to various factors [6].

Secondhand smoke (SHS), comprising over 7000 chemicals, is a recognized risk factor for cardiovascular disease, including ACS [7, 8]. The toxic constituents of SHS lead to endothelial dysfunction, increased oxidative stress, and a pro-atherosclerotic environment, making even brief exposures hazardous [9]. Epidemiological data indicates that non-smokers exposed to SHS face a 25–30% higher risk of heart disease [10]. This highlights the urgent need for protective measures against SHS exposure, especially in high-risk environments [11].

Although there are ample studies detailing the sex differences in the harmful effects of direct smoking on both humans and animals [12, 13], research on sex disparities concerning the risks of diseases induced by SHS remains limited. We hypothesized that ACS, known to be significantly triggered by active smoking, might also be induced by SHS, with varying effects across sex. Utilizing prospective cohort data, we conducted an 18-year longitudinal study to determine whether SHS acts as a risk factor for ACS and to analyze if such a risk is modified by sex.

2. Methods

2.1 Study design and data sources

Established in 2001, the Korean Genome Epidemiology Study (KoGES) embarked on two unique prospective cohort studies in distinct regions: Ansung, a rural community with an approximate 2010 population of 176,000, and Ansan, an urban region housing nearly 715,000 individuals that same year. These cohorts comprised Korean males and females aged 40 to 69, sharing a consistent ethnic lineage. Detailed methods, including participant selection and sampling strategies for these ongoing investigations, have been described elsewhere [14]. During 2001–2002, 7129 individuals from Ansung and 10,957 from Ansan were deemed eligible. From this, 5018 subjects in Ansung (2239 males and 2779 females) and 5020 in Ansan (2523 males and 2497 females) underwent baseline evaluations in their respective locales. Follow-ups occurred routinely, culminating in the 9th assessment from 2019 to 2020. Interviewers, adhering to a consistent protocol, received biennial training updates. These cohort members underwent structured evaluations at two-year intervals.

2.2 Study population and definition of ACS

KoGES data included biennial follow-up assessments of patient information, beginning with the baseline survey in 2001–2002. In this study ACS was defined as myocardial infarction or angina pectoris diagnosed during the biennial follow-up assessments conducted from 2003 to 2020. For the analysis, we excluded participants with physician-diagnosed ACS or with no SHS exposure history at the time of baseline survey.

2.3 SHS and other risk factors

Participants completed an interviewer-administered questionnaire. On the 2001–2002 questionnaire, exposure to secondhand smoke was assessed based on whether participants answered "yes" to the question, "Are you regularly exposed to secondhand smoke at home or in the workplace?" We also collected demographic characteristics, including age, sex, education year, and socioeconomic status measured by the house income; comorbidities, including hypertension, diabetes mellitus (DM), coronary artery disease, dyslipidemia, and kidney disease; and health-related behavior, including body mass index (BMI), alcohol intake and insomnia. Additionally, we measured and presented the serum levels of total cholesterol, high density lipoprotein (HDL) and triglycerides (TG).

2.4 Statistical analysis

We calculated descriptive statistics for the baseline characteristics of the study participants by SHS exposure and sex. The baseline characteristics of participants from the KoGES were compared using the Wilcoxon rank sum test for continuous variables and the chi-square test for categorical variables. The crude 19-year (2001 to 2020) incidence rates of ACS were calculated as the number of risk cases per 1000 person-years based on the SHS exposure and sex. The hazard ratios (HRs) and 95% confidence interval (CIs) obtained from Cox proportional hazard regression models with fixed covariates were used to estimate the relative risks for 19-year cumulative ACS incidence based on SHS exposure and sex. Additionally, the inter effect of SHS exposure and alcohol intake was assessed by taking individuals with neither SHS exposure nor alcohol intake as the reference group. We tested the multicollinearity between covariables in the model. All statistical analyses were performed using SAS version 9.4 software (SAS Institute Inc., Cary, NC, USA).

3. Results

3.1 Demographic findings

During the 18 years of follow-up, we documented 244 cases of new-onset ACS (2.64 cases per 1000 person-years). The characteristics of the study population according to history of SHS exposure are presented in Table 1. The group exposed to SHS is younger with a predominant female presence. A noteworthy association was observed between SHS exposure and alcohol consumption, with the former being more common in individuals who alcohol intake (41.9% *vs.* 35.1%). However, the overall incidence of ACS did not exhibit a significant difference between the groups. The characteristics of study population according to sex are present in Table 2. In males, there was a significant higher prevalence of diabetes mellitus and dyslipidemia, and higher proportion of alcohol intake. The incidence of ACS was greater in males compared to females (4.4% *vs.* 3.6%), but this difference was not statistically significant (p value = 0.13).

3.2 Main outcomes

In the group without SHS exposure, there were 150 cases of ACS, reflecting an incidence rate of 2.7 per 1000 person-years and the group with SHS exposure demonstrated an ACS incidence rate of 2.5 per 1000 person-years. To explore the impact of exposure variables on ACS incidence, a Cox proportional logistic regression analysis was conducted, adjusting for all confounding variables. Following adjustment, SHS exposure was not associated with an increased risk of ACS, exhibiting a hazard ratio (HR) of 1.44 (95% CI, 0.88–1.49) in comparison to the non-exposed group. However, Male present a significant HR (1.67 (1.23–2.28)) on the risk of ACS (Table 3). In the interaction analysis between SHS exposure and sex on the incidence of ACS, in females, SHS was not a risk factor for ACS (HR: 0.97 (95% CI: 0.70–1.35)), whereas, in males, SHS was a significant risk factor for ACS (HR: 1.63 (1.02–2.60) (Table 4).

4. Discussion

Our study provides an in-depth examination of the sex-specific relationships between SHS exposure and the risk of ACS. Over an 18-year follow-up, we identified that males exposed to SHS had a significantly elevated risk of developing ACS with an HR of 1.63, while females did not exhibit a significant association. Interestingly, even though SHS-exposed groups were younger with a larger female proportion and a higher prevalence of alcohol intake, the overall incidence of ACS did not display a significant difference between those exposed to SHS and those not. These findings underscore the importance of sex-specific approaches when evaluating the risk factors associated

TABLE 1. Demographic findings of study population according to second-hand smoking.

Variables	All	Second-hand smoking		
	N (%)	Yes	No	*p*-value
All	6346 (100.0)	2559 (100.0)	3787 (100.0)	
Age, years, mean (SD)	52.3 (8.9)	50.5 (8.4)	53.5 (9.0)	<0.001
Sex, female	4336 (68.3)	1841 (71.9)	2495 (65.9)	0.003
Educational period <9 years	3747 (59.0)	1488 (58.1)	2259 (59.7)	0.231
House income, lower half	3199 (50.4)	1197 (46.8)	2002 (52.9)	<0.001
Comorbidity				
Hypertension	1042 (16.4)	413 (16.1)	629 (16.6)	0.621
Medication	808 (12.7)	299 (11.7)	509 (13.4)	0.042
Diabetes mellitus	404 (6.4)	164 (6.4)	240 (6.3)	0.914
Medication	227 (3.6)	78 (3.0)	149 (3.9)	0.061
Dyslipidemia	154 (2.4)	61 (2.4)	93 (2.5)	0.851
Kidney disease	201 (3.2)	90 (3.5)	111 (2.9)	0.189
Cerebrovascular disease	66 (1.0)	24 (0.9)	42 (1.1)	0.512
Body mass index				
<18.5 (underweight)	88 (1.4)	25 (1.0)	63 (1.7)	
18.5–24.9 (normal weight)	3343 (52.7)	1322 (51.7)	2021 (53.4)	0.024
>25.0 (overweight)	2915 (45.9)	1212 (47.4)	1703 (45.0)	
Alcohol intake, yes	2403 (37.9)	1072 (41.9)	1331 (35.1)	<0.001
Insomnia, yes	1086 (17.1)	432 (16.9)	654 (17.3)	0.691
Lipid profile				
Total cholesterol, mean (SD)	191.7 (35.1)	191.6 (35.5)	191.7 (34.9)	0.434
HDL, mean (SD)	45.0 (9.9)	45.1 (9.9)	45.0 (34.9)	0.195
Triglyceride, mean (SD)	155.1 (99.7)	154.5 (98.7)	155.6 (100.4)	0.151
Total ACS cases	244 (3.8)	94 (3.7)	150 (4.0)	0.562

SD: standard deviation; HDL: high density lipoprotein; ACS: acute coronary syndrome.

with ACS. For males, the pronounced risk associated with SHS exposure suggests an urgent need for targeted public health interventions, especially given the established harmful effects of direct smoking. The lack of association in females, on the other hand, offers avenues for further research to understand the underlying mechanisms and factors contributing to this discrepancy. Moreover, our research highlights the importance of considering other interacting factors, such as alcohol intake, when assessing the comprehensive effects of SHS. It is paramount for health professionals and policymakers to recognize these distinctions and tailor preventive strategies accordingly.

Several studies have established the association between smoking and an increased risk of Acute Coronary Syndrome (ACS). A study by Teo *et al.* [15] in 2006 demonstrated that smoking is a significant risk factor for ACS across different populations globally. Another study by Ambrose and Barua in 2004 explored the pathophysiological mechanisms linking smoking to coronary artery disease, further solidifying the relationship between smoking and ACS [16]. However, there are limited studies that specifically explore the sex differences in the effects of smoking on ACS risk. A study by Huxley and Woodward in 2013 did find that women smokers have a higher risk of coronary heart disease compared to their male counterparts, but this was focused on direct smoking rather than secondhand smoke (SHS) exposure [17].

In a comprehensive assessment of the global burden of disease from SHS, Öberg *et al.* [18] found that as of 2004, 40% of children, 35% of women, and 33% of men were regularly exposed to SHS worldwide. This widespread exposure was implicated in an estimated 603,000 deaths, making up about 1.0% of global mortality. Additionally, the link between SHS exposure and coronary heart disease has been established in prior studies. For instance, a 2015 meta-analysis revealed that exposure to SHS significantly increased the risk for all-cause mortality and cardiovascular disease (CVD) [19]. Another study noted that the risk of coronary heart disease almost doubled in those with the highest levels of SHS exposure compared to those unexposed [20]. While the deleterious effects of SHS are clear, the literature still has gaps in understanding sex-specific risks. Some investigations, like the one by Tan *et al.* [21], suggested a potentially heightened risk for women in relation to certain conditions, such as lung cancer, due to SHS exposure. The overarching narrative underscores the need

TABLE 2. Demographic findings of study population by sex.

Variables	All	Sex		
	N (%)	Male	Female	*p*-value
All	6346 (100.0)	2010 (100.0)	4336 (100.0)	
Second-hand smoking, yes	2559 (40.3)	718 (35.7)	1841 (42.5)	<0.001
Age, years, mean (SD)	52.3 (8.9)	52.0 (8.8)	52.4 (8.9)	0.069
Educational period <9 years	3747 (59.0)	816 (40.6)	2931 (67.6)	0.004
House income, lower half	3199 (50.4)	772 (38.4)	2427 (56.0)	<0.001
Comorbidity				
Hypertension	1042 (16.4)	321 (16.0)	721 (16.6)	0.511
Medication	808 (12.7)	231 (11.5)	577 (13.3)	0.041
Diabetes mellitus	404 (6.4)	155 (7.7)	249 (5.7)	<0.001
Medication	227 (3.6)	88 (4.4)	139 (3.2)	0.024
Dyslipidemia	154 (2.4)	70 (3.5)	84 (1.9)	<0.001
Kidney disease	1042 (16.4)	321 (16.0)	721 (16.6)	<0.001
Cerebrovascular disease	66 (1.0)	23 (1.1)	43 (1.0)	0.581
Body mass index				
<18.5 (underweight)	88 (1.4)	32 (1.6)	56 (1.3)	
18.5–24.9 (normal weight)	3343 (52.7)	1108 (55.1)	2235 (51.5)	0.014
>25.0 (overweight)	2915 (45.9)	870 (43.3)	2045 (47.2)	
Alcohol intake, yes	2403 (37.9)	1313 (65.3)	1090 (25.1)	<0.001
Insomnia, yes	1086 (17.1)	213 (10.6)	873 (20.1)	<0.001
Lipid profile				
Total cholesterol, mean (SD)	191.7 (35.1)	193.3 (35.0)	190.9 (35.2)	0.032
HDL, mean (SD)	45.0 (9.9)	43.7 (9.7)	45.7 (10.0)	<0.001
Triglyceride, mean (SD)	155.1 (99.7)	172.4 (120.4)	147.1 (87.4)	<0.001
Total ACS cases	244 (3.8)	88 (4.4)	156 (3.6)	0.131

SD: standard deviation; HDL: high density lipoprotein; ACS: acute coronary syndrome.

TABLE 3. Cox proportional logistic regression analysis for study outcome.

Potential risk factors	Numbers at risk	ACS events	Person-years	Incidence rate per 1000 PYS	Model 1	Model 2	Model 3
					aHR (95% CI)	aHR (95% CI)	aHR (95% CI)
Second-hand smoking							
No	3787	150	54,863.7	2.7	1.00 (reference)	1.00 (reference)	1.00 (reference)
Yes	2559	94	37,487.5	2.5	1.15 (0.88–1.49)	1.14 (0.87–1.48)	1.14 (0.88–1.49)
Sex							
Female	4336	156	63,431.6	2.5	1.00 (reference)	1.00 (reference)	1.00 (reference)
Male	2010	88	28,919.7	3.0	1.32 (1.01–1.71)	1.58 (1.18–2.12)	1.67 (1.23–2.28)

PYS: person-years; aHR: adjusted hazard ratio; ACS: acute coronary syndrome; CI: confidence interval.
Model 1: adjusted for age and sex. Model 2: adjusted for Model 1 + hypertension, diabetes mellitus, dyslipidemia, kidney disease, and cerebrovascular disease. Model 3: adjusted for Model 2 + education period, house income, body mass index, and insomnia.

TABLE 4. Interaction analysis between sex and second-hand smoking on study outcome.

	Second-hand smoking		
	No	Yes	*p* value
		aHR (95% CI)	<0.001
Male	1.00	1.63 (1.02–2.60)	
Female	1.00	0.97 (0.70–1.35)	

aHR: adjusted hazard ratio; CI: confidence interval.

for more refined studies, especially those exploring potential gender disparities in SHS-related health outcomes.

In our study, while SHS did not emerge as a principal risk factor for ACS across the entire cohort, it was discernibly a significant risk factor among male. This observed distinction between sexes can be attributed to the variable nature of SHS exposure, which, unlike active smoking, is less potent and more inconsistent [22]. This observed distinction can be elucidated by considering three main factors. Firstly, pathophysiological differences between male and female must be acknowledged. Specifically, the cardiovascular system in males might respond more adversely to certain components of SHS than females, potentially due to biological variations like hormonal differences or vascular reactivity [23]. Secondly, particular lifestyle habits prevalent among male could amplify the detrimental effects of SHS. For instance, male might be more exposed to occupational hazards or other harmful environments or may adopt specific dietary or activity patterns that enhance the risks associated with SHS [24]. Thirdly, sociological factors play a pivotal role. Male might, due to socio-cultural contexts, be more frequently exposed to SHS-rich environments or be influenced by other behavioral factors that coalesce to heighten the risk of ACS alongside SHS. Furthermore, social norms or expectations could shape the health-related behaviors and stress-coping mechanisms of male differently than female, and such distinctions could influence the ACS risk associated with SHS [25].

Our study conclusively demonstrated that SHS exposure serves as a risk factor for ACS specifically in men. Such findings provide substantial theoretical groundwork for healthcare and public health policymakers. It underscores the urgent need to tailor preventive strategies and interventions targeting male populations exposed to SHS.

Our study has several limitations. Firstly, our reliance on self-reported SHS exposure introduces potential recall bias, potentially leading to an underestimation or overestimation of actual SHS exposure levels. Secondly, we did not incorporate information on the severity or specific type of ACS, limiting the comprehensiveness of our results regarding the effects of SHS on particular ACS categories or severities. Thirdly, a critical limitation of our study is the lack of quantification regarding the level of SHS exposure. While we acknowledge the absence of specific data on the duration, frequency and concentration of SHS, the inability to precisely quantify the exposure level further impedes our capacity to assess its impact on ACS risk accurately. This quantification is essential for a more detailed and accurate evaluation of the relationship between SHS exposure and ACS risk, as the extent of exposure may vary significantly among individuals and can be a decisive factor in disease development. Fourthly, while we attempted to control for many variables, potential confounding factors such as diet, physical activity, and other environmental exposures that might interact with or influence the effects of SHS were not fully accounted for. Fifthly, the homogeneity of our cohort, specifically being Korean, could limit the generalizability of our findings to other ethnic or racial populations. Lastly, we did not consider potential changes in SHS exposure or smoking habits over time, which might influence the cumulative risk assessment of ACS related to SHS. Despite these limitations, our research provides substantial evidence for the role of SHS as a significant risk factor for ACS, particularly among males.

5. Conclusions

Our study provides compelling evidence that SHS exposure significantly elevates the risk of ACS in men. These findings underscore the urgent need for rigorous public health interventions and policies aimed at reducing secondhand smoke exposure, especially among male populations.

AVAILABILITY OF DATA AND MATERIALS

Data in this study were from the Korean Genome and Epidemiology Study (KoGES; 6635-302), National Institute of Health, Korea Disease Control and Prevention Agency, Republic of Korea.

AUTHOR CONTRIBUTIONS

EJ and HHR—conceptualization, investigation, software, writing original draft, writing review and editing; EJ and HLK—data curation; EJ and HYK—formal analysis, supervision. All authors have read and agreed to the published version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study protocol was reviewed and approved by the Institutional Review Board of Chonnam National University Hospital (CNUH-2018-297). The requirement for informed consent from patients was waived.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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