Angina Outcomes in Secondhand Smokers: Results from the National Health and Nutrition Examination Survey 2007–2018

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Abstract

Objective The aim of the study is to examine the relationship between secondhand smoke (SHS) and angina using the National Health and Nutrition Examination Survey database over a 12-year period.

Methods Self-reported smoking status and cotinine levels were used to identify exposure groups (smokers, nonsmokers, and secondhand smokers), and medical history of angina was also collected via a self-report survey. The association between exposure to SHS and angina was analyzed using odd ratios with 95% confidence intervals calculated using two logistic regression models.

Results The study found that when aggregating data from all 12 years, secondhand smokers are 42.9% significantly more likely to report having experienced angina, while smokers were 97.7% significantly more likely to report having angina compared with nonsmokers.

Conclusions This study is the first of its kind to examine data from a national database over a 12-year period to determine an association between SHS and angina outcomes, thus highlighting the importance of reducing SHS exposure to improve cardiovascular health.

Keywords

- secondhand smoke
- cardiovascular health
- environmental tobacco smoke
- public policy
- angina pectoris
- coronary heart disease

Introduction

Secondhand smoke (SHS), also known as passive or environmental tobacco smoke, is the smoke-comprised air produced from the burning of tobacco products, such as cigarettes, pipes, hookahs, and e-cigarettes.[1,2] According to the World Health Organization, over one-third of all people are regularly exposed to SHS across the world, including nonsmoking

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children (40%), men (33%), and women (35%).[1] Additionally, during 2013 to 2014, the Centers for Disease Control and Prevention (CDC) estimated that 58 million people—roughly one in four nonsmokers—were exposed to SHS in the United States, even with tobacco bans in public places.[3]

SHS is a known risk factor of many diseases in adults and children, including stroke, lung cancer, and cardiovascular disease (CVD).[3] Previous work between 1965 and 2014 showed that more than 20 million Americans died due to SHS-related causes, including nearly 2.5 million who died due to lung cancer or CVDs.[2–4] In 2005 to 2009, 32% of all deaths from coronary heart disease (CHD) were attributed to active smoking and exposure to SHS.[3] The narrowing of blood vessels due to CHD limits blood flow to the heart and can increase the risk of myocardial infarction and other CVDs.

A study in Beijing, China, found that among 1,209 women who never smoked, 39.5% were exposed to SHS at home or in the workplace.[5] Compared with nonsmoking women, secondhand smokers were 69% more likely to develop CHD and 56% more likely to develop ischemic stroke.[5] Similarly, a study in Greece found that, compared with participants not exposed to SHS, secondhand smokers had a 61% increased risk of having an event of acute coronary syndromes during the first 30 days after hospitalization.[6] Another study from Greece found that regular exposure to SHS is associated with a 99% increased risk of developing acute coronary syndromes (e.g., acute myocardial infraction or unstable angina).[7]

Although there is a clear association between exposure to SHS and CHD, few studies have examined the specific association for angina pectoris, which is one of the most common symptoms of CHD. Angina is chest pain or discomfort that occurs when the myocardium does not get enough oxygenated blood. Early work by Aronow[8] found that exposure to SHS in a sample of 10 male participants who experienced exercise-induced angina after exposure to the smoke of 15 cigarettes resulted in elevated venous carboxyhemoglobin, increased heart rate, increased systolic and diastolic blood pressure while resting, and decreased heart rate and systolic blood pressure during the angina episode. The study concluded that exposure to SHS aggravates angina.[8]

In the United States, data from the 2011–2012 cycle from the National Health and Nutrition Examination Survey (NHANES) was analyzed and concluded that SHS exposure was highest among respondents with respiratory disease (72.1%) followed by respondents with CVDs (70.6%).[9] The study characterized CVDs as congestive heart failure, CHD, angina, heart attack, or stroke. However, the specific association between SHS and angina was not presented. Other U.S. studies examined the impact of the Clean Indoor Air Acts (CIAA)[10] and statewide smoking bans[11] on the prevalence of various CVDs, including acute myocardial infarction, stroke, and angina. A year after CIAA was implemented, 10 states and/or territories had a significant decrease in the prevalence of angina, or acute myocardial infarction.[10] Additionally, 33% fewer angina cases were reported in hospital admissions a year after statewide smoking bans were implemented in Arizona.[11]

A handful of international studies has previously examined the association between exposure to SHS and angina. One study[12] in Korea found that the prevalence of angina significantly increases with exposure to SHS at home and work (relative risks: 1.016 and 1.006, respectively). Similarly, recent analyses in Scotland modeled the effects of indoor exposure to SHS at home, work, and other households. The study found that exposure to SHS in more than two places (i.e., home, work, other households) increases the likelihood of angina by 50% and of total CVD by 35%.[13] These studies demonstrate that exposure to SHS increases the likelihood of developing or worsening angina.

Overall, the literature demonstrates a meaningful association between exposure to SHS and CVDs in general, with limited information about the effects on angina in the United States., especially given that more than 40 years have passed since Aronow's seminal study.[8] Therefore, the paucity of research regarding SHS and its association with angina warrants further research. The present study addresses the gap in the literature by using a large, nationally representative survey over 12 years to generate robust and generalizable estimates of the likelihood of angina in secondhand smokers.

Methods

The NHANES is a cross-sectional survey among the U.S. civilian noninstitutionalized population conducted every 2 years by the CDC's National Center for Health Statistics. Our study included U.S. adults (18 years or older) who answered questions on tobacco use, had recorded laboratory values for serum cotinine, answered demographic questions on their age, gender, and race/ethnicity, and had a personal history of angina in the six NHANES cycles that spanned between 2007 and 2018 ($N = 59{,}842$). We excluded participants who were missing any of the variables of interest ($n = 25{,}177$; 42.07%), for a final analytic sample of 34,655 unweighted observations.

Exposure Groups and Outcomes

Participants were categorized into three exposure groups: nonsmokers, secondhand smokers, and active smokers using a combination of self-reported smoking in the prior 5 days ("During the past 5 days, including today, did you smoke cigarettes, pipes, cigars, little cigars or cigarillos, water pipes, hookahs, or e-cigarettes?") and blood serum cotinine levels. Active smokers were participants who responded "yes" to the question and/or whose cotinine levels were greater than 3.0 ng/mL. Nonsmokers were participants who responded "no" to the question and/or had blood cotinine levels $\leq$0.050 ng/mL. Participants were considered exposed to SHS if they responded "no" to the question and had blood cotinine levels between 0.051 and 3.0 ng/mL. The blood cotinine cut-off values were selected from prior studies that have used these values.[14–16]

The outcome of interest, angina, was self-reported based on yes/no answers to the question "Has a doctor or other health professional ever told you that you had angina, also called angina pectoris?"

Statistical Analyses

Raw NHANES data for the six cycles of interest were downloaded and match-merged one to one, incorporating the variables of interest: participant number, mobile examination center (MEC) exam sampling weight,[17] age, gender, race/ethnicity, blood serum cotinine levels, and history of smoking in the prior 5 days and lifetime angina. All analyses were conducted in the Statistical Package for Social Sciences (SPSS) v. 28.0 (The IBM Corporation; Armonk, New York, United States) in duplicate, using unweighted observations and then weighting these analyses using the MEC exam sampling weights.

Descriptive statistics (frequencies and means with standard deviations [SDs]) were estimated for the analytic sample. Weighted and unweighted backward stepwise logistic regressions were conducted to estimate the strength of associations (reported as odds ratios [ORs] with their respective 95% confidence intervals) between smoking status and angina. In each regression, the first layer entered smoking status while the second layer included a stepwise backward inclusion of gender, age, and race/ethnicity. In both models, nonsmokers were used as a reference group and Nagelkerke's R^2 was reported as a measure of model fit.[18] Briefly, this pseudo-coefficient of determination can be interpreted as the percentage of variance in the angina outcomes that can be accounted for by the variances of the variables included in the regression models.

Ethical Considerations

NHANES is a publicly available dataset without any identifiable information available for use by the research community. The Institutional Review Board at the University of Central Florida reviewed the study protocol (STUDY00003449) and determined that this study did not meet the federal regulatory definition of research involving humans.

Results

Demographics

Unweighted and weighted demographic data for the 34,655 study participants are presented in ►Table 1 based on smoking status groups. In the weighted analyses, most respondents were female (51.2%), nonsmokers (56.7%), non-Hispanic white (63.4%), with a mean (SD) age of 40.02 (22.11) years. A history of angina was reported by 2.2% of respondents.

Unadjusted Model Estimation

In the overall, unadjusted, weighted model (►Table 2), compared with nonsmokers, both secondhand smokers and smokers were significantly more likely to report a history of angina (8.1 and 12.4% more likely, respectively). However, compared with nonsmokers, secondhand smokers were twice as likely to report having angina during the 2015–2016 cycle while smokers are 76.8% more likely to report having angina during this cycle. Additionally, the model found a statistically significantly reduced likelihood of a history of angina between respondents exposed to SHS during the 2007–2008, 2013–2014, and 2017–2018 cycles and in smokers during the 2007–2008 and 2011–2012 cycles.

All Nagelkerke's R^2 were lower than 0.012 for this model, which indicates a very poor explanation of the variance. This model was deemed not a good fit for the data.

Adjusted Model Estimation

The adjusted model took into account age, sex, and race/ethnicity (►Table 3). Overall, compared with nonsmokers not exposed to SHS, secondhand smokers were 42.9% more likely to report having angina while smokers were 97.7% more likely. Of note, during the 2015–2016 cycles, secondhand smokers were 3.5 times more likely to report a history of angina compared with nonsmokers. Smokers, on the other hand, were 2.5 and 2.7 times more likely to report a history of angina during 2013–2014 and 2015–2016, respectively. Like the unadjusted model, the adjusted model found a statistically significant association between exposure to SHS and a reduced likelihood of angina in secondhand smokers only during the 2007–2008 cycle, while smokers were more likely to have angina in all the cycles.

Nagelkerke's R^2 was also calculated for the adjusted model, which improved the overall fit compared with the unadjusted model. The R^2 values ranged from 0.117 to 0.153. Even though the adjusted model improved the overall fit, there is still 87% of the variance in the history of angina unaccounted for, even after adjusting for age, sex, and race/ethnicity differences.

►Table 4 presents the final adjusted, weighted model predicting the likelihood for angina. As hypothesized, the likelihood of angina is 97.7% higher in smokers and 42.9% higher in subjects exposed to SHS. Keeping all elements equal, the demographics at increased likelihood of angina are non-Hispanic black, male, and Mexican American participants (50.9, 43.7, and 34.2% increased likelihood). The model fit was small ($R^2 = 0.130$).

Discussion

The purpose of this study was to assess the likelihood of a history of angina in secondhand smokers. Compared with nonsmokers, our study demonstrated that both secondhand smokers and smokers were significantly more likely to report a history of angina with ORs compatible with a dose–response relationship. These results are also consistent with the handful of international studies that have previously examined the association between exposure to SHS and angina.[12,13] Based on our extensive review, it is believed that this study is the first of its kind to use data from a 12-year period (2007–2018) to assess angina history and SHS exposure in the United States.

Interestingly, while smokers were more likely to have angina across all NHANES' yearly cycles compared with nonsmokers, individuals exposed to SHS were more likely to have angina across all cycles except for the 2007–2008 cycle. A potential explanation for these data is environmental

Table 1 Weighted demographic characteristics of the analytic sample

Demographic characteristics	Total			Nonsmokers			Exposed to SHS			Smokers			*p*-Value
	N	Weighted *N*	Weighted %	*N*	Weighted *N*	Weighted %	*N*	Weighted *N*	Weighted %	*N*	Weighted *N*	Weighted %	
Age, y, mean (SD)	37.48	40.02	21.11	39.07	41.96	22.09	30.79	33.96	21.76	41.21	40.74	16.40	<0.001[a]
Sex													<0.001[b]
Male	23,841	138,317,441	48.8	12,009	71,456,752	44.4	5,849	29,103,043	49.5	5,983	37,757,646	59.2	
Female	24,470	145,037,045	51.2	14,524	89,322,934	55.6	5,986	29,668,615	50.5	3,960	26,045,496	40.8	
Race/ethnicity													<0.001[c]
Mexican American	8,550	28,613,696	10.1	5,897	19,182,197	11.9	1,660	5,453,311	9.3	987	3,978,188	6.2	
Other Hispanic	5,210	17,847,104	6.3	3,338	11,245,237	7.0	1,128	3,644,699	6.2	744	2,957,168	4.6	
Other[d]	6,086	23,707,219	8.4	3,851	14,119,832	8.8	1,376	5,246,975	8.9	859	4,340,412	6.8	
Non-Hispanic Black	10,814	33,597,030	11.9	4,235	13,099,433	8.1	3,712	10,415,687	17.7	2,867	10,081,910	15.8	
Non-Hispanic White	17,651	179,589,438	63.4	9,212	103,132,988	64.1	3,953	34,010,986	57.9	4,486	42,445,464	66.5	
History of angina													<0.001[e]
Yes	834	4,926,688	2.2	437	2,665,053	2.1	171	868,017	2.2	226	1,393,618	2.4	
No	31,940	218,535,290	97.8	17,320	122,985,358	97.9	6,165	38,446,480	97.8	8,455	57,103,452	97.6	

Abbreviations: ANOVA, analysis of variance; SD, standard deviation; SHS, secondhand smoke.
[a]For a one-way ANOVA with $F(2, 283\ 354\ 483) = 3{,}203{,}713$.
[b]For a test of independence with $\chi^2(2) = 3{,}983{,}623$.
[c]For a test of independence with $\chi^2(8) = 6{,}967{,}740$.
[d]Includes multi-racial.
[e]For a test of independence with $\chi^2(2) = 12{,}648$.

Table 2 Unadjusted, weighted, logistic regression models for angina by smoking status

NHANES cycle	Exposed to SHS		Smokers		R^2
	OR	95% CI	OR	95% CI	
2007–2008	0.497	0.493–0.500	0.677	0.672–0.681	0.007
2009–2010	1.397	1.386–1.408	0.996	0.991–1.002	0.002
2011–2012	1.177	1.168–1.186	0.973	0.969–0.978	<0.001
2013–2014	0.924	0.918–0.930	1.282	1.275–1.288	0.002
2015–2016	2.680	2.659–2.702	1.776	1.768–1.785	0.012
2017–2018	0.987	0.981–0.993	1.187	1.182–1.193	0.001
Overall	1.081	1.078–1.084	1.126	1.124–1.129	<0.001

Abbreviations: CI, confidence interval; OR, odds ratio; SHS, secondhand smoke.

Table 3 Adjusted,[a] weighted, logistic regression models for angina by smoking status

NHANES cycle	Exposed to SHS		Smokers		R^2
	OR	95% CI	OR	95% CI	
2007–2008	0.760	0.755–0.765	1.287	1.279–1.295	0.153
2009–2010	1.850	1.836–1.865	1.721	1.711–1.731	0.122
2011–2012	1.417	1.406–1.428	1.799	1.789–1.809	0.148
2013–2014	1.230	1.222–1.238	2.509	2.495–2.522	0.149
2015–2016	3.596	3.566–3.366	2.748	2.734–2.762	0.117
2017–2018	1.186	1.178–1.193	1.949	1.939–1.958	0.134
Overall	1.429	1.425–1.433	1.977	1.972–1.981	0.130

Abbreviations: CI, confidence interval; OR, odds ratio; SHS, secondhand smoke.
[a]Model adjusted by age, sex, and race/ethnicity; reference group is nonsmokers.

Table 4 Final adjusted,[a] weighted,[b] model predicting the likelihood of angina

Predictor	OR	95% CI	R^2
Age	0.933	0.933–0.933	0.130
Sex			
Male	1.437	1.435–1.440	
Female (Ref.)	–		
Race/ethnicity			
Mexican American	1.342	1.336–1.349	
Other Hispanic	1.095	1.090–1.100	
Other[c]	1.111	1.107–1.115	
Non-Hispanic Black	1.509	1.504–1.515	
Non-Hispanic White (Ref.)	–		
Smoking status			
Active smoker	1.977	1.972–1.981	
Secondhand smoker	1.429	1.425–1.433	
Nonsmoker (Ref.)	–		

Abbreviations: CI, confidence interval; OR, odds ratio; Ref., reference.
[a]Model adjusted by age, sex, and race/ethnicity; reference group is nonsmokers.
[b]The sampling weight used was the mobile examination center (MEC) weight.
[c]Includes multi-racial.

factors from the 2007–2009 Economic Recession. Two studies found that the decline in economic resources during the 2007–2009 Economic Recession was associated with an increased likelihood of smoking in the Midwestern region of the United States.[19,20] In a meta-analysis of 141 cohort studies, the risk for CHD was 1.48 for smoking one cigarette per day and 2.04 for 20 cigarettes per day, and when the relative risk was adjusted for confounding variables, it was 1.74 and 2.27, respectively.[21] Therefore, increasing smoking can lead to increased risk for CHD, which potentially explains increased likelihood of having angina since it is a symptom of CHD. Other explanations to this counterintuitive finding can be a statistical artifact that results in an outlier or cyclical instability of data: while most ORs ranged between 1.2 and 1.8, the 2007–2008 and 2015–2016 cycles were outside these ranges. While the latter's OR (3.6) is also an outlier, it errs on the side of the pathophysiology of secondhand smoking and angina, perhaps opening the door for additional explanations to the OR identified in 2007–2008.

The present study found an increased likelihood of angina in males, and amongst non-Hispanic blacks and Mexican American participants. These results can be explained by a previous work examining the socioeconomic status (SES) and smokers,[22] exposure to SHS,[23,24] and CVDs.[25] For example, one study used Wave 1 (2002) and Wave 2 (2003) of the International Tobacco Control Project from Australia, Canada, United Kingdom, and the United States to investigate smokers of different SES affiliations and whether one's SES affiliation had any impact on how many smoker friends they had as time progressed.[22] They concluded that smokers with low SES affiliation reported gaining more smoking friends over time and additionally reported generally having a larger number of smoking friends compared with their high SES-affiliated smoker counterparts.[22] Similarly, a study which used the NHANES III 1988–1994 and NHANES 1999–2010 to examine socioeconomic disparities and SHS exposure among U.S. never-smoking adults found that men, young participants (e.g., those aged <40 years), non-Hispanic black participants, and participants with lower education or lower poverty-to-income ratio levels were more likely to be exposed to SHS.[23] Usually, non-Hispanic black and Latino participants tend to be from low SES' and face many health disparities.[26,27] Additionally, these populations tend to have more CVDs[25] and be exposed to SHS more often.[24] Although the current study did not examine SES, our demographic findings showed increased likelihood of having angina and is consistent with the literature.

Implications for Clinicians, Public Health, and Policies

These findings impact the way SHS should be addressed in both clinical and policy contexts. Currently, most clinicians do not implement screening procedures to examine exposure to SHS. For example, the U.S. Preventive Services Task Force does not include any recommendations or interventions for SHS exposure regarding smoking cessation.[28] Also, both the national guidelines for smoking cessation in primary care[29] and the American Academy of Family Physicians[30] do not ask about SHS exposure when discussing tobacco use and cessation. The American Academy of Pediatrics is the only organization identified that includes SHS exposure in their recommendations for clinical practice.[31]

Since our findings suggest that secondhand smokers are more likely to exhibit angina across most cycles compared with nonsmokers, clinicians should ask patients about their exposure to SHS. By doing so, clinicians can more thoroughly assess patients for angina and possible CVD, including CHD, myocardial infarction, or heart failure. Previous studies suggest that exposure to SHS increases the risk of a heart attack[32,33] and death among patients with heart failure.[34,35] Since angina is a symptom among patients with CHD, myocardial infarction, and heart failure, screening for SHS can enable clinicians to implement preventative measures to curb these negative diseases down the line.

Multiple studies have looked at the impact of policy implication to regulate SHS exposure among nonsmokers in the United States.[36,37] In Michigan, urine cotinine levels among nonsmoking bar employees decreased from 35.9 ng/mL to a negligible level only 2 months after a statewide comprehensive smoke-free law went into effect. Also, most bar employees reported a significant improvement in general health and respiratory symptoms such as cough, phlegm production, shortness of breath, wheezing, and allergic symptoms.[36] In New York, salivary cotinine levels among nonsmoking adult workers in restaurants, bars, and bowling facilities decreased by 85% 1 year after a smoke-free law went into effect in 2003.[37] These legislative and public policy restrictions are critical in reducing SHS exposure and, consequently, reducing angina and CVDs in the general population.

Legislative change restricting SHS exposure in public places can significantly improve the prevalence of preventative diseases and free up economic resources. Literature suggests that from 1965 to 2014, an estimated 20 million Americans experience SHS-related disease onsets and fatalities.[4] Additionally, it is estimated that 32% of all deaths from CHDs from 2005 to 2009 are attributed to smoking and exposure to SHS.[3] Limiting the exposure of SHS in public places may prevent the onset of SHS-related diseases and reduce the increased financial burden secondhand smokers experience compared with their nonsmoker counterparts. Previous findings suggest that patients exhibiting chronic angina have more than a twofold increase in medical resource utilization and an increased financial burden of $4,000 over a period of 8 months.[38] Additionally, a systematic review examined 17 studies and the relationship between management of chronic angina and overall health care expenditure. Of the $1,550 billion the United States spent in health care in 2002, angina accounted for 1.3% ($20 billion) of this budget.[39] Restricting SHS exposure in public places allows the fiscal budget otherwise spent on angina to be redistributed to targeting and advancing other disease outcomes.

Limitations and Future Directions

The study has some limitations worth discussing. History of angina and smoking status was based on self-reported data, which may be prone to participant bias. Since angina presence was determined by: "has a doctor or other health professional ever told you that you have angina or angina pectoris," respondents could have unknowingly experienced angina without either knowing the terminology to describe their symptom or may not have been questioned by their health care provider whether they have experienced angina or not. Parallel to this, the data collection method of angina presence was susceptible to recall and/or social-desirability biases.[40] However, these biases were limited since cotinine levels were also assessed to ensure correct group categorization in addition to patients' self-reported (yes/no) response to the smoking-status question. Blood cotinine levels were used to determine whether patients fell into the nonsmoker category ($\leq$0.050 ng/mL), secondhand smoker category (between 0.051 and 3.0 ng/mL), or smoker category ($>$3.0 ng/mL). Another limitation of the current study was the exclusion of a notable number of patients due to missing variables for smoking status or history of angina. Finally, environmental factors that may have served as a confounding variable when assessing SHS exposure and angina prevalence (e.g., higher obesity among secondhand smokers, dietary regulation, etc.) were not assessed in the current study.

Despite these limitations, the present study was the first of its kind to use data from a nationally representative sample, over a 12-year span, to examine the relationship SHS has with angina in the United States. This study serves as a bridge between the literature gap that exists for SHS and particularly its effects on angina, as the most recent study exploring this relationship in the United States was published in 1978 and examined 10 participants.[8] Another strength of the study was the addition of objective measures of blood cotinine levels, to confirm self-reported smoking status. Future studies should examine how lifestyle factors such as physical activity, sedentary behavior, nutrition, and alcohol use may attenuate or exacerbate the association between SHS and angina. Also, future studies should examine the effects of SHS with CVDs to determine whether SHS exposure results in increased CVDs such as CHD, myocardial infarction, and heart failure. Future studies should also target variables not accounted for in this study, such as medical history of other diseases, genetic composition, SES, and the duration of exposure to SHS among patients.

Conclusion

This study examined the relationship between SHS and angina and was the first of its kind to examine data from a national database over a 12-year period. Ultimately, the study found that when aggregating data from all 12 years, secondhand smokers are 42.9% significantly more likely to report having experienced angina, while smokers were 97.7% significantly more likely to report having angina. However, when examining yearly cycles from the NHANES database individually, secondhand smokers were more likely to exhibit angina across most cycles compared with nonsmokers. Similarly, the present study found that smokers are significantly more likely to experience angina when viewed in aggregate data and across all individual yearly cycles. These findings suggest that reducing SHS exposure can improve the overall health of the general public and prevent various disease onsets, such as CHD, myocardial infarction, and heart failure. Additionally, our findings suggest that restricting SHS exposure can allow the $20 billion in angina-related health care expenditure to be used to address other diseases. Although future studies can further explore this topic by examining other national databases and filling the literature gap that exists for SHS and angina, our study serves as a breakthrough in the modern-day examination of SHS and angina, and the legal and clinical implications they may have in health care expenditure, clinical assessment, and preventative medicine.

Ethical Approval

The study used data from NHANES, which is a publicly available dataset without any identifiable information available for use by the research community. The Institutional Review Board at the University of Central Florida reviewed the study protocol (STUDY00003449) and determined that this study did not meet the federal regulatory definition of research involving humans.

Funding

None.

Conflict of Interest

None declared.

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