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RESEARCH ARTICLE

Secondhand Smoke Exposure in Childhood and Adulthood in Relation to Adult Mortality Among Never Smokers

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Introduction: Secondhand smoke is known to have adverse effects on the lung and vascular systems in both children and adults. It is unknown if childhood exposure to secondhand smoke is associated with adult mortality.

Methods: The authors examined associations of childhood and adult secondhand smoke exposure with death from all causes, ischemic heart disease, stroke, and chronic obstructive pulmonary disease among 70,900 never smoking men and women, predominantly aged ≥50 years, from the Cancer Prevention Study−II Nutrition Cohort in 1992−1993. There were 25,899 participant deaths during follow-up through 2014. During 2016−2017, Cox proportional hazards regression models were used to calculate multivariable-adjusted hazard ratios and 95% CIs.

Results: Childhood secondhand smoke exposure was not associated with all-cause mortality. However, childhood secondhand smoke (living with a smoker for 16−18 years during childhood) was associated with higher mortality from chronic obstructive pulmonary disease (hazard ratio=1.31, 95% CI=1.05, 1.65). Adult secondhand smoke exposure of ≥10 hours/week at enrollment was associated with a higher risk of all-cause (hazard ratio=1.09, 95% CI=1.04, 1.14); ischemic heart disease (hazard ratio=1.27, 95% CI=1.14, 1.42); stroke (hazard ratio=1.23, 95% CI=1.04, 1.45); and chronic obstructive pulmonary disease (hazard ratio=1.42, 95% CI=0.97, 2.09) mortality.

Conclusions: These results suggest that childhood secondhand smoke exposure, as well as adult secondhand smoke exposure, increase the risk of chronic obstructive pulmonary disease death in adulthood. Consistent with previous studies, the results also show that adult secondhand smoke is meaningfully associated with higher mortality from vascular disease and all causes. Overall, these findings provide further evidence for reducing secondhand smoke exposure throughout life.

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INTRODUCTION

Secondhand smoke (SHS), also referred to as environmental tobacco smoke, is the combination of smoke exhaled by smokers and the smoke from the burning tip of the cigarette. The U.S. Surgeon General finds no risk-free level of SHS exposure.[1] Although U.S. exposure to SHS has been declining over the past 2 decades, 25% percent of nonsmokers are still exposed to SHS.[2,3] Therefore, SHS remains an important preventable risk factor for many diseases.

Parental smoking and SHS exposure in the home is associated with adverse health effects in childhood,[4] including increased risk of chronic respiratory symptoms, asthma, and poorer lung function. There is also evidence supporting developmental effects on the cardiovascular system including atrial septal heart defects, poor vascular endothelial function, and increased blood pressure.[5]

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Exposure to SHS during adulthood is associated with diseases of the lungs and vascular system.[1] Since 1986, the U.S. Surgeon General has considered SHS to be a cause of lung cancer,[6] and more recently found convincing evidence for an association with fatal coronary heart and cerebrovascular disease.[1] Associations of SHS with adult-onset asthma; chronic obstructive pulmonary disease (COPD); and other lung diseases are suggestive of a relationship, but not yet considered causal.[1]

Although there has been extensive research on the associations of both childhood SHS exposure with health outcomes among children and adult SHS exposure with health outcomes among adults, there are few established associations with childhood SHS exposure in relation to adult mortality.[4] It is plausible that childhood SHS exposure resulting in damage to the lung and cardiovascular system would increase the risk for fatal outcomes in adults.

The American Cancer Society's Cancer Prevention Study–II (CPS–II) Nutrition Cohort is a large nationwide prospective study of men and women that includes more than 70,000 never smokers. Information on intensity and duration of childhood SHS exposure, and intensity of adult SHS exposure was collected from participants in this cohort. This information was used to examine childhood and adult SHS exposure at enrollment in relation to risk of death during adulthood from all causes; ischemic heart disease (IHD); stroke; and COPD.

METHODS

Study Population

Men and women included in this analysis were selected from the CPS–II Nutrition Cohort, a prospective study of cancer incidence and mortality among 184,185 men and women from the U.S., described in detail elsewhere.[7] Briefly, the Nutrition Cohort is a subcohort of the ≅1.2 million subjects in CPS–II, a mortality study established by the American Cancer Society in 1982. Participants in the larger study were recruited nationally and completed a four-page questionnaire at enrollment. In 1992–1993, participants, primarily aged 50–74 years, were recruited for the Nutrition Cohort from 21 states. Participants completed a ten-page mailed questionnaire that included information on demographic, medical, behavioral, environmental, occupational, and dietary factors. Follow-up questionnaires were sent to cohort members every 2 years beginning in 1997 to update exposure information. Responses to all surveys were received from >87% of living participants after multiple mailings. The current study includes mortality follow-up through December 31, 2014, which was made available in March 2017. All aspects of the CPS–II Nutrition Cohort study have been approved by the Emory University IRB.

This analysis excluded participants from the CPS–II Nutrition Cohort who ever previously reported cigarette/cigar/pipe smoking or had unknown smoking status at baseline in 1992 (n=111,078), or were missing information on childhood or adult exposure to SHS in 1992 (n=2207). The final analytic cohort included 70,900 men and women who had never smoked.

Measures

In 1992, participants were asked: *During the period from birth to age 18, did you ever live for more than 1 year with someone who smoked on a daily basis?* Participants who answered *yes* to this question were asked: *For how many years?* Response options included: *1–3*, *4–6*, *7–9*, *10–12*, *13–15*, or *16–18 years*. And: *Was this usually. . .* Response options: *One*, *two*, *or more than two persons*. To be included in the analysis, subjects with childhood exposure to SHS were required to answer questions for years living with and number of smokers in the home.

At enrollment in 1992, participants were asked the question: *Whether or not you smoke, about how many hours per week are you currently exposed to the smoke of others. . .*, followed by 2-digit write-in answers for *At home (hours/week)* and *In other places (hours/week)* and asked to enter *0* if unexposed. To be included in the analytic cohort, participants were required to have answered at least one of the 1992 questions with a value of ≥0. Values from the two responses were summed to determine total hours/week of SHS exposure in 1992.

Vital status of CPS–II participants was determined using linkage with the National Death Index to identify deaths[8] that occurred from September 1992 through December 2014. Cause of death was obtained for >99% of all known deaths. Specific causes of death were coded using ICD versions 9 and 10 as IHD (ICD-9 410–414 and 429.2, ICD-10 I20–I25); stroke (ICD-9 430–438, ICD-10 I60–I69); COPD (ICD-9 490–492 and 496, ICD-10 J40–J44), and all other causes of death combined.

Statistical Analysis

Childhood SHS exposure was examined as any exposure (no, yes); years through age 18 living with a smoker (<16, 16–18 years); number of smokers in the household (one, two or more); and combinations of the years and number of smokers. Adult SHS exposure at enrollment was examined as any exposure (no, yes); as quartiles of the total hours/week in the exposed (1, 2, 3–9, ≥10 hours/week); and location of current exposure (in home, outside of home, or both inside and outside of home). Descriptive statistics were calculated using distributions of categorical variables.

Person-years of follow-up for each participant were calculated as the amount of time from completion of the questionnaire in 1992/1993 to date of (1) death; (2) a questionnaire including a report of smoking; (3) the end of mortality follow-up. The stratified Cox model was used to compute multivariable-adjusted hazard ratios (HRs) and 95% CIs for the association between SHS variables and mortality. All models were stratified on year of age and additionally adjusted for gender (man, woman); race (white, non-white); education (less than high school, high school graduate, some college, college graduate); BMI (<18.5, 18.5 to <25, 25 to <30, ≥30); physical activity (0, >0 to <17.5, ≥17.5 MET hours/week); time spent sitting (<3, 3–5, ≥6 hours/day); alcohol use (non-drinker, less than one, one to two, two or more drinks/day); healthy eating diet score (low, moderate, high); and marital status (married, not married). Models were not adjusted for prevalent lung and vascular disease because they are on the causal pathway. Probability values for linear trend were calculated using continuous terms with values of 0 for the unexposed, midpoints were assigned to categories when continuous data were unavailable. Effect modification was evaluated using multiplicative interaction terms. The proportional hazards assumption was evaluated

graphically and using multiplicative interaction terms with the log of fail-time; no violations were observed. All *p*-values are two-sided. All analyses were performed using SAS, version 9.4, between October 2016 and August 2017.

RESULTS

Approximately 52% of participants lived with a smoker at some point in their childhood, and 74% of these lived with a smoker their entire childhood. Few lived with more than one smoker, but the proportion living with two or more smokers during childhood was greater with each consecutive birth cohort (Figure 1). This reflects increased smoking among adults, particularly women, in the late 20th century.[9] Thus, the youngest participants, born in the 1930s to 1940s, were more likely than older participants to be exposed to SHS as children. Participants who reported living with smokers as children were more likely to be women, less educated, alcohol drinkers, and exposed to SHS as adults, whereas the distribution of other factors was comparable (Table 1). Only 36% of the cohort reported exposure to SHS at enrollment.

Associations of childhood SHS exposure with death from all causes and specific causes are presented in Table 2. There were no statistically significant associations of any measure of childhood SHS exposure with death from all causes, IHD, or stroke, although HRs for participants who lived with two or more smokers for their entire childhood (16–18 years) were >1 for all-cause mortality and each category of cause-specific mortality examined.

For COPD mortality, the HR for any childhood exposure was 1.21 (95% CI=0.97, 1.50) and the HR for living with a smoker throughout childhood (16–18 years) was slightly higher and statistically significant (HR=1.31, 95% CI=1.05, 1.65). When stratified by adult SHS exposure (Appendix Table 1, available online), the HR for living with a smoker throughout childhood was limited to participants with no adult SHS exposure at enrollment (HR=1.48, 95% CI=1.12, 1.96), although the difference was not statistically significant (p=0.18 for interaction).

Associations of adult SHS exposure at enrollment with death from all causes and specific causes are shown in Table 3. Adult SHS exposure showed a significant dose–response relationship with overall mortality (p<0.0001 for trend). The strongest association was for ≥10 hours/week of SHS exposure compared with no exposure (HR=1.09, 95% CI=1.04, 1.14). SHS exposure at home also was associated with higher risk of death from all causes. There was a strong positive trend with increasing adult SHS exposure in relation to IHD death

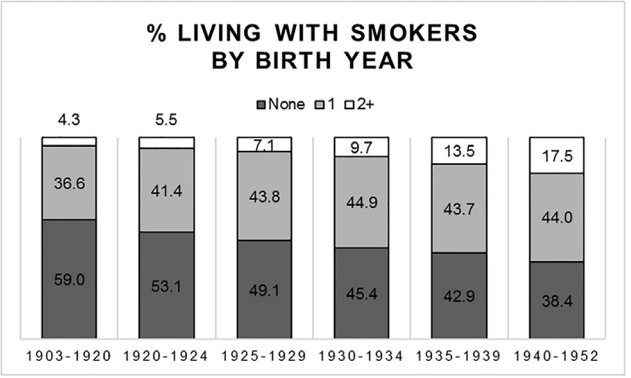

Figure 1. Percentage of subjects living with smokers in the house during childhood by birth year.

(p<0.0001 for trend) culminating in a significant association among those with ≥10 hours per week of exposure (HR=1.27, 95% CI=1.14, 1.42). Any exposure to SHS was associated with a higher risk of fatal stroke (HR=1.14, 95% CI=1.04, 1.26), and there was also a trend with increasing hours of exposure (p=0.02 for trend). Associations of adult SHS exposure with death from COPD were of a higher magnitude than the associations with death from IHD or stroke, although only exposure to SHS both outside and inside the home was significantly associated with COPD death (HR=1.76, 95% CI=1.06, 2.90).

DISCUSSION

This study's large size and prospective design provide an opportunity to examine associations of childhood and adult exposure to SHS with subsequent death from all causes and specific causes in never smokers. Childhood SHS was not associated with all-cause mortality, however exposure to SHS throughout childhood was associated with a higher risk of death from COPD. Higher levels of adult exposure to SHS was associated with death from all causes, as well as with death from IHD, stroke, and COPD.

To the authors' knowledge, this is the first study to identify an association between childhood SHS and COPD death among adults. Childhood SHS exposure has been associated with adverse lung function in children, young adults,[1,10] and older adults.[11] Eisner et al.[12] examined maternal smoking during pregnancy and found a nonsignificant relationship with incidence of adult COPD (OR=1.36, 95% CI=0.86, 2.41) of a magnitude that was similar to this study. A study in China found no association between number of smokers in the house during childhood and risk of COPD, but did

Table 1. Baseline Characteristics of Never Smokers by Childhood Secondhand Smoke Exposure in the CPS-II Nutrition Cohort

Variable	Lived with smoker as child	
	No, % (*n*=34,049)	Yes, % (*n*=36,851)
Age group (years) in 1992		
<55	7.6	10.7
55–59	20.0	23.4
60–64	26.0	27.5
65–69	25.3	22.9
70–74	16.3	12.6
≥75	4.8	2.9
Gender		
Male	34.2	27.3
Female	65.8	72.7
Race		
White	96.7	97.7
Non-white/missing	3.3	2.3
Education		
Less than high school	5.5	5.3
High school graduate	28.3	32.0
Some college	26.9	27.5
College graduate	39.3	35.3
Marital status		
Married	87.8	87.3
Not married	11.4	12.1
BMI		
<18.5	1.5	1.4
18.5–<25	46.8	45.6
25–<30	36.2	35.8
≥30	13.9	15.7
Physical activity (METs)		
No activity	9.1	9.0
>0–<17.5	64.3	63.5
≥17.5	25.4	26.6
Time spent sitting (hours/day)		
<3	50.4	46.5
3–5	38.2	41.4
≥6	9.3	9.9
Current alcohol use		
Non-drinker	56.7	48.7
<1/day	31.4	37.4
1–2/day	5.3	7.3
>2/day	2.4	3.5
Diet quality score		
Low	23.7	26.2
Moderate	43.0	43.7
High	23.8	22.3
Current exposure to SHS		
No	68.5	59.2
Yes	31.5	40.8
History of lung disease		
No	90.7	89.6
Yes	9.3	10.4
History of vascular disease		
No	61.2	61.4
Yes	38.8	38.6

Note: Columns may not sum to 100% because of rounding or missing data.
CPS-II Nutrition Cohort, American Cancer Society's Cancer Prevention Study II; SHS, secondhand smoke.

observe significant associations with respiratory symptoms in adulthood.[13]

It is established that SHS exposure in childhood can result in asthma, chronic wheezing, respiratory infections, and decreased lung function and growth in children.[1,14,15] These respiratory illnesses in early life are associated with worse lung function in adolescence and adulthood, as indicated by a lower forced expiratory volume in a 1 second plateau, and ultimately diagnosis of COPD.[16] This evidence, with results from this study, suggest childhood exposure to SHS may be the first step in a chain of events that starts with poor lung development and asthma in childhood, leading to development of COPD, and ultimately death from COPD. Although the analysis focused on mortality outcomes, it should be noted that an association between childhood SHS and adult COPD mortality implies that childhood SHS likely has effects on respiratory disease morbidity in adulthood.

Beyond lung effects, there is compelling evidence that SHS exposure in children is associated with arterial endothelial function, increased arterial stiffness, autonomic dysfunction, and other vascular endpoints.[5] This led to a hypothesis that early life exposure to SHS might manifest itself in greater risk of IHD and stroke mortality in adults, which was not observed. These results are similar to a study of nonsmoking Chinese women.[17] Most men and women with childhood SHS exposure in this study lived with only one smoking parent, likely the father. The number of cigarettes smoked in the home is the largest predictor of SHS exposure in children,[18] suggesting that associations could be more apparent in

Table 2. Childhood Exposure to Secondhand Smoke and Adult Mortality in CPS-II Nutrition Cohort, 1992–2014

		All-causes		Ischemic heart disease		Stroke		COPD		Other causes	
Categories	**Person-years**	**Deaths**	**HR (95% CI)**	**Deaths**	**HR (95% CI)**	**Deaths**	**HR (95% CI)**	**Deaths**	**HR (95% CI)**	**Deaths**	**HR (95% CI)**
Any exposure											
No	631,534	13,308	1.00	2,244	1.00	992	1.00	162	1.00	9,910	1.00
Yes	702,227	12,591	0.99 (0.96, 1.01)	1,944	0.96 (0.90, 1.02)	957	1.01 (0.93, 1.11)	184	1.21 (0.97, 1.50)	9,506	0.99 (0.96, 1.02)
Number of smokers in house											
1	580,572	10,751	0.98 (0.96, 1.01)	1,661	0.95 (0.89, 1.01)	820	1.01 (0.92, 1.11)	161	1.21 (0.97, 1.51)	8,109	0.98 (0.96, 1.01)
≥2	121,656	1,840	1.02 (0.97, 1.07)	283	1.02 (0.90, 1.16)	137	1.07 (0.89, 1.29)	23	1.18 (0.75, 1.83)	1,397	1.01 (0.95, 1.07)
p-trend			0.91		0.44		0.41		0.18		0.84
Years lived with smoker											
<16	184,529	3,283	1.00 (0.96, 1.03)	518	0.98 (0.89, 1.07)	233	0.96 (0.84, 1.11)	35	0.90 (0.62, 1.30)	2,497	1.00 (0.96, 1.05)
16–18	517,699	9,308	0.98 (0.96, 1.01)	1,426	0.95 (0.89, 1.02)	724	1.03 (0.94, 1.14)	149	1.31 (1.05, 1.65)	7,009	0.98 (0.95, 1.01)
p-trend			0.16		0.21		0.49		0.06		0.14
Years and number of smokers											
1 smoker, <16 years	160,435	2,893	1.01 (0.97, 1.05)	455	0.99 (0.89, 1.09)	204	0.97 (0.83, 1.13)	29	0.85 (0.57, 1.26)	2,205	1.02 (0.97, 1.07)
1 smoker, 16–18 years	420,136	7,858	0.97 (0.95, 1.00)	1,206	0.94 (0.87, 1.01)	616	1.02 (0.92, 1.13)	132	1.34 (1.06, 1.69)	5,904	0.97 (0.94, 1.00)
≥2 smokers, <16 years	24,093	390	0.92 (0.83, 1.02)	63	0.90 (0.70, 1.16)	29	0.94 (0.65, 1.36)	6	1.29 (0.57, 2.93)	292	0.92 (0.82, 1.03)
≥2 smokers, 16–18 years	97,562	1,450	1.05 (0.99, 1.11)	220	1.06 (0.92, 1.22)	108	1.11 (0.91, 1.36)	17	1.14 (0.69, 1.89)	1,105	1.04 (0.97, 1.11)

Note: Models are stratified on single-year of age, and adjusted for sex, race, education, BMI, recreational physical activity, sitting time, alcohol use, diet score, marital status, and exposure to SHS at enrollment.

COPD, chronic obstructive pulmonary disease; CPS-II, Cancer Prevention Study II; HR, hazard ratio; SHS, secondhand smoke.

Table 3. Adult Exposure to Secondhand Smoke at Enrollment and Adult Mortality in CPS-II Nutrition Cohort, 1992–2014

Categories	Person-years	All-causes		Ischemic heart disease		Stroke		COPD		Other causes	
		Deaths	HR (95% CI)	Deaths	HR (95% CI)	Deaths	HR (95% CI)	Deaths	HR (95% CI)	Deaths	HR (95% CI)
Any exposure											
No	841,738	17,193	1.00	2,775	1.00	1,259	1.00	224	1.00	12,935	1.00
Yes	492,023	8,706	1.00 (0.98, 1.03)	1,413	1.03 (0.97, 1.10)	690	1.14 (1.04, 1.26)	122	1.15 (0.92, 1.44)	6,481	0.98 (0.95, 1.01)
Hours of exposure											
1 hour/week	152,545	2,479	0.94 (0.90, 0.98)	366	0.88 (0.79, 0.98)	182	1.00 (0.85, 1.16)	34	1.04 (0.72, 1.49)	1,897	0.94 (0.90, 0.99)
2 hours/week	91,509	1,647	0.98 (0.94, 1.04)	270	1.02 (0.90, 1.15)	148	1.27 (1.07, 1.51)	20	0.97 (0.61, 1.53)	1,209	0.95 (0.90, 1.01)
3–9 hours/week	137,691	2,547	1.02 (0.98, 1.06)	411	1.04 (0.93, 1.15)	201	1.16 (1.00, 1.35)	37	1.22 (0.85, 1.73)	1,898	1.00 (0.95, 1.05)
≥10 hours/week	110,278	2,033	1.09 (1.04, 1.14)	366	1.27 (1.14, 1.42)	159	1.23 (1.04, 1.45)	31	1.42 (0.97, 2.09)	1,477	1.04 (0.98, 1.09)
p-trend			<0.0001		<0.0001		0.02		0.43		0.07
Location of SHS exposure											
Outside of home only	395,814	6,931	0.99 (0.96, 1.01)	1,102	0.98 (0.92, 1.05)	542	1.12 (1.01, 1.24)	94	1.11 (0.87, 1.41)	5,193	0.97 (0.94, 1.00)
In home only	47,141	911	1.08 (1.01, 1.16)	164	1.31 (1.12, 1.54)	77	1.24 (0.98, 1.57)	11	0.99 (0.54, 1.83)	659	1.02 (0.95, 1.11)
In and out of home	49,068	864	1.07 (1.00, 1.15)	147	1.23 (1.04, 1.46)	71	1.27 (0.99, 1.62)	17	1.76 (1.06, 2.90)	629	1.02 (0.94, 1.10)

Note: Models are stratified on single-year of age, and adjusted for sex, race, education, BMI, recreational physical activity, sitting time, alcohol use, diet score, marital status, and exposure to SHS as a child.
COPD, chronic obstructive pulmonary disease; CPS-II, Cancer Prevention Study II; HR, hazard ratio; SHS, secondhand smoke.

studies of participants born in the 1950s and 1960s that include more exposure from a smoking mother in the home.[9]

The findings on adult exposure to SHS and its association with death are consistent with previous research.[4] The study identified an increased risk of all-cause mortality associated with in home exposure (7%–8%) and exposure of 10 or more hours/week (9%). A recent meta-analysis estimated an 18% increased risk of death from all causes among nonsmokers exposed to SHS compared with those who are not.[19] The threshold for SHS exposure in this study is quite small (1 hour/week) compared with the regular home exposure measured in most other studies, possibly explaining the lower effect estimates. Surprisingly, exposure to 1 hour/week of SHS was generally associated with lower mortality. Among those exposed to 1 hour/week of SHS, 96% were exposed outside the home. The authors speculate that 1 hour/week exposure is indicative of occasional exposure (e.g., bars and restaurants) more often experienced by participants in better health. There are limited data on the dose–response relationship of SHS exposure and all-cause mortality.[19] In this study, there was a significant linear trend in mortality risk with increasing exposure to SHS ($p<0.0001$). Similar to death from all causes, previous SHS associations with IHD and cardiovascular disease diagnosis and death observed in other studies[1] were replicated in this population.

This study identified an association between adult exposure to SHS and increased death from COPD. The most recent Surgeon General's report on smoking and health stated that the evidence was "suggestive but not sufficient,"[1] and that further research is needed. A recent meta-analysis examined studies of SHS with COPD and other respiratory conditions through 2010 and estimated a relative risk of 1.56 (95% CI=1.40, 1.74) with little heterogeneity among studies[20]—similar to this estimate for in-home and out-of-home exposure (HR=1.76). The meta-analysis included approximately 7,000 subjects from prospective cohorts, thus this study of more than 70,000 participants is a substantial addition to the literature. The associations observed with both childhood exposure to SHS and adult exposure to SHS add to the mounting data relating SHS to COPD.

A strength of this study is the large size and 20 years of follow-up that provided an adequate number of outcomes to evaluate death from specific causes in a never smoking population. The prospective design reduces the possibility of recall bias. The detailed questionnaires on active smoking history during follow-up ensured that the SHS and mortality relationship was not being confounded by active smoking later in life. This is currently

the only study that the authors are aware of to evaluate childhood SHS exposure on all-cause adult mortality.

Limitations

The measure of childhood SHS exposure in this analysis has certain limitations. Childhood SHS exposure was self-reported at least three decades after childhood and undoubtedly was recalled imperfectly by some participants. Nonetheless, self-reported recall of childhood exposure to SHS has been shown to be reliable,[21] and details on the number of smokers in the household and years of exposure captured the entire childhood period. In addition, information distinguishing between maternal and paternal SHS was unavailable. Maternal smoking during pregnancy has been shown to be associated with cardiovascular and respiratory outcomes in children.[22] Therefore, it is possible that observed associations with childhood exposure represent in utero exposure from the mother. However, most participants with childhood SHS exposure reported only one smoker in their household during childhood. This smoker is more likely to have been their father than their mother given the much higher prevalence of smoking among U.S. men than women through the 1950s.[9]

The measure of adult SHS exposure used in this analysis also has limitations. Adult SHS exposure was based on current exposure reported at enrollment in 1992, when the average age of participants was 63 years. This measure may not fully reflect exposure earlier during adult life. In addition, a participant's SHS exposure could have changed after enrollment because of changes in their living or work conditions. To the extent adult SHS exposure measured at enrollment does not fully capture etiologically relevant exposure, this study may underestimate the full impact of adult SHS exposure. Additionally, there are concerns about generalizability, because the CPS–II Nutrition Cohort is predominantly white and somewhat better educated than the general population. Other limitations include the possibility of residual confounding, and possible false positives as a result of multiple testing.

CONCLUSIONS

This study identified an association between long-term childhood SHS exposure and death from COPD. Additionally, these findings support previous research on adult SHS exposure and risk of death from all causes, IHD, and stroke. Active smoking is a more important factor in increased mortality; however, SHS is a moderate risk factor comparable with other secondary risk factors.[23] More than 50 years after the publication of the first Surgeon General report on smoking and health, these findings suggest that researchers and scientists still do not fully understand the long-term health consequences of smoking, particularly, the potential delayed effects of childhood SHS exposure in later adulthood. Understanding these long-term effects are relevant in the U.S., and potentially even more relevant in countries where smoking rates and SHS exposure are higher. Finally, the results of this research provide further support for implementation of smoke-free air laws,[24] smoke-free home policies,[25] and clinical interventions[26,27] to reduce SHS exposure.

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SUPPLEMENTAL MATERIAL

Supplemental materials associated with this article can be found, in the online version at https://doi.org/10.1016/j.amepre.2018.05.005.

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