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Secondhand smoke, obesity, and risk of type II diabetes among California teachers

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Abstract

Purpose: To examine if secondhand smoke (SHS) is associated with elevated risk of type II diabetes among California teachers. We also aim to determine if overall and central obesity are mediators or effect modifiers of this association.

Methods: Using data from the California Teachers Study, conducted in 1995–2013 in California public schools, we obtained information on SHS exposure among 39,887 lifetime nonsmokers. The association between SHS and incident diabetes after 17 years of follow-up was assessed using Cox regression models. The mediation and modification effects of BMI and waist circumference on this association were tested.

Results: At baseline, 70.2% of the nonsmokers reported exposure to SHS. Higher intensity, duration, and intensity-years of exposure to SHS were associated with higher multivariate adjusted risk of incident diabetes in a dose-response manner (hazard ratio = 1.28; 95% confidence interval, 1.11–1.48 for highest quartile vs. lowest quartile of exposure; $P = .001$ for trend). Participant's waist circumference (measured 2 years after baseline) could explain greater than 50% of the association between SHS and diabetes.

Conclusions: SHS exposure is associated with increased risk of type II diabetes among nonsmokers of California teachers with obesity being a potentially important mediator but not an effect modifier for this association.

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Authors' contributions: L.J. designed the study, researched the data, contributed to the discussion, and wrote, reviewed, and edited the article. J.C. researched the data, performed data analysis, and reviewed and edited the article. A.Z., D.D., P.R., L.B., and H.A.C. participated in the study design and data collection of the CTS study, contributed to the discussion, and reviewed and edited the article. L.J. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

The authors claim no conflict of interests.

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.annepidem.2019.01.011.

Keywords

Abdominal adiposity; Central obesity; Environmental tobacco smoke; Mediation; Passive smoking; Secondhand smoke; Type II diabetes; Waist circumference

Despite falling prevalence in most developed countries in the past few decades, smoking remains to be one of the leading causes of avoidable morbidity and mortality worldwide [1]. Meanwhile, many nonsmokers are exposed to the detrimental effects of tobacco smoke due to secondhand smoke (SHS) exposure. It is well known that SHS causes respiratory infections in children as well as cardiovascular disease and lung cancer among adults [2].

The rising prevalence of diabetes also is a serious public health concern globally, with the highest prevalence and associated health expenditures found in North America and the Caribbean Islands [3]. According to the most recent National Diabetes Statistics Report, 30.3 million people in the United States had diabetes in year 2015, and the estimated diabetes costs were $245 billion in 2012 [4]. To address this global epidemic, it is important to identify potentially modifiable risk factors for this chronic disease.

Recently, evidence regarding a significant association between type II diabetes and active smoking has been documented. In a 2015 meta-analysis of 84 studies, the risk of developing type II diabetes among current smokers was reported to be 37% higher than that among never smokers [5]. A large European prospective investigation suggested the association between active smoking and type II diabetes was slightly stronger among normal weight than overweight men [6], indicating obesity might be an effect modifier for this association. Meanwhile, because obesity has been linked to higher risk of developing insulin resistance and diabetes, overall or abdominal obesity might be important mediators for the association between active smoking and diabetes.

In addition to the potential causal link between active smoking and diabetes risk, burgeoning evidence has appeared, suggesting SHS is also associated with elevated risk of type II diabetes [5,7–10]. However, most previous studies of SHS and diabetes [11–17] lacked detailed information on characteristics of SHS or had relatively short follow-up periods. Furthermore, the potential mediating and/or modifying roles of obesity in the association between SHS and diabetes remain unclear. Using data from the California Teachers Study (CTS), a large prospective cohort study of female California public school employees, we examined the association of SHS exposure with incidence of type II diabetes among lifetime nonsmokers. We also explored the degree to which body mass index (BMI) and waist circumference may be potential mediators or effect modifiers of this association.

Methods

Study population

A detailed description of the CTS has been published elsewhere [18]. Briefly, in 1995–1996, 133,479 female California public school employees completed a self-administered baseline questionnaire that collected information on disease histories and demographic, anthropometric, reproductive, and lifestyle factors. CTS participants have been followed up

annually to collect information on cancer diagnosis, death, and change of address. Participants have completed four follow-up questionnaires to update information collected previously and to collect new or more detailed information on various topics: 1997–1998 (wave 2), 2000–2001 (wave 3), 2005–2006 (wave 4), and 2012–2013 (wave 5).

The analyses presented here were restricted to nonsmoking women with known diabetes status at wave 5. Therefore, we sequentially excluded women who, at baseline (1995–1996), were current (n = 6779) or past smokers (n = 38,263) or did not report smoking status (n = 909); who reported diabetes (n = 2507), cardiovascular disease (n = 1776), or cancer (n = 6197); or who were aged 80 years or older (n = 3174). A total of 73,874 baseline responders remained in the analytical sample. Among these participants, 39,887 nonsmoking women responded to a series of questions about each respondent's diabetes status in the 2012–2013 questionnaires. These 39,887 (54% of the baseline nonsmokers) participants were used as the primary analytical sample of the present study.

Measures

Type II diabetes—Incident type II diabetes was ascertained using the 2012–2013 questionnaires, which asked the following question: "Has a health professional ever told you that you have diabetes?" Those who answered yes to this question subsequently reported whether they had type I or type II diabetes and the age of diagnosis.

Measures of exposure to SHS—At baseline, participants reported if they ever lived with a smoker during childhood or adulthood. We grouped the participants into four categories: no household SHS exposure, childhood exposure only, adulthood exposure only, and both childhood and adulthood exposure.

In 1997–1998, the questionnaire was used to collect detailed information on exposure to SHS in the household, the workplace, and social settings during six age periods. Around 97% baseline never smokers reported household SHS exposure at baseline also reported household SHS exposure in 1997–1998. For each combination of setting and age period, participants were asked whether they were exposed to tobacco smoke from others. If affirmative, they were further asked about the duration and intensity of this exposure. Duration was estimated by asking the number of years of exposure within specific age periods. SHS intensity for each age period was estimated by a qualitative description: a little smoky, fairly smoky, or very smoky.

As described previously [19], we created variables for duration (years) and intensity (smokiness) of lifetime exposure to SHS from all three settings combined, as well as for the combination of duration and intensity (intensity-years). We assigned a numerical score to represent the intensity of SHS (1 for a little smoky, 2 for fairly smoky, and 3 for very smoky). The lifetime intensity for each exposure setting was calculated by averaging the numerical scores across all age periods. An overall lifetime intensity score was obtained by summing the intensity scores from the three settings. All cumulative exposure measures were categorized into quartiles based on the distribution among all women.

Anthropometric measurements—The CTS participants reported their current weight and height at baseline. They also reported their current weight and height in the 2005–2006 questionnaire. In addition, they self-measured waist and hip circumference following instructions on the 1997–1998 questionnaire. Each participant was asked to measure her waist and hip circumference twice and reported both values. The average of those two values was used in this study.

Covariate assessment—Data from the baseline questionnaire include age (continuous), race (non-Hispanic white vs. other), family history of diabetes (yes vs. no), physical activity (average hours of moderate and strenuous physical activity per week over lifetime, in quartiles), and alcohol consumption (grams per day). Dietary intake in the year before baseline was assessed using an early version of the 112-item Block95 food frequency questionnaire, which was validated in the CTS [20]. The food frequency questionnaires data were assigned nutrient values based on an updated version of the Block95 nutrient database. The dietary factors relevant to diabetes risk included here are daily caloric intake and intake of magnesium, calcium, vitamin D, dietary fiber, total fat, and saturated fat (all in quartiles).

Statistical analysis

Age-adjusted anthropometric measurements by SHS exposure were calculated using predicted values from linear regression of each anthropometric measure with age and SHS exposure as the only covariates. To determine the association between SHS and risk of incident type II diabetes in CTS participants, hazard ratios (HR) were calculated using Cox proportional hazards regression models. Age at cohort entry or at the time of the second questionnaire, as appropriate, and age at the end of the individual's follow-up were used as the underlying time metric. Subjects were censored either at the year of type II diabetes diagnosis or at the end of CTS follow-up (year completing the 2012–2013 questionnaire). Deaths before 2012–2013 were not considered as censored observations because it is impossible for them to complete the 2012–2013 questionnaires and hence were excluded from our final analytical sample. For each SHS exposure variable, we began with a regression model with only exposure and age at cohort entry as independent variables. We then fit models after adding baseline BMI and including other potential cofounding variables that have been previously associated with incident diabetes: race, family history of diabetes, physical activity, alcohol consumption, and dietary factors (daily caloric intake, magnesium, calcium, vitamin D, dietary fiber, total fat, and saturated fat).

Potential nonlinearity for the association between SHS exposure and diabetes risk was tested using natural cubic splines. None of the nonlinearity tests were statistically significant. Thus, the dose–response relationship between SHS exposure and diabetes was assessed by testing the linear trend of the association. Potential mediating effects of BMI at different time points (baseline and wave 4), waist circumference, hip circumference, and waist–hip ratio at wave 2 for the association of SHS intensity and duration with type II diabetes were evaluated using a SAS macro that can calculate the percent of exposure effect explained by each intermediate variable (Mediate SAS) [21]. This macro estimates the mediation proportion using the "difference method" [22]. For Cox regression model, a data duplication algorithm and a generalized estimation equations approach were used to estimate the mediation

proportion and variance [23]. Effect modifications by obesity status were assessed by fitting Cox regression models stratified by BMI categories and median of waist circumference. We also tested the interaction terms of SHS status with BMI (both continuous and categorical) and waist circumference (continuous).

Results

At baseline, 70.2% of the nonsmokers reported childhood and/or adult exposure to SHS in their households. Compared with nonsmokers without exposure to SHS, women exposed to SHS were older, more likely to have family history of diabetes, had higher BMI, and consumed more alcohol (Table 1). In Table 2, age-adjusted anthropometric measurements by SHS exposure among lifetime nonsmokers are presented. It shows the age-adjusted average values of BMI at baseline and wave 4, waist circumference at wave 2, and hip circumference at wave 2 all increased with increasing levels of SHS exposure.

As shown in Table 3, by year 2012–2013, a total of 2495 nonsmoking women reported incident type II diabetes. The estimated incidence of diabetes was 3.85 per 1000. The incidence rate of type II diabetes was the lowest among those without household exposure to SHS and was the highest among the women with both childhood and adult exposure. Compared with nonsmokers without exposure to SHS, the age-adjusted HRs for nonsmoking women with exposure during childhood, adult years, and both childhood and adult years were 1.12, 1.12, and 1.36, respectively. After adjusting for potential confounders, those with both childhood and adult exposure had significantly higher risk for diabetes than those without any exposure to SHS (HR = 1.25, 95% confidence interval [CI]: 1.11–1.41). Detailed SHS exposure measurements assessed in 1997–1998 showed that the age-adjusted risk of developing type II diabetes 17 years later significantly increased with higher intensity, duration, and intensity-years of total exposure to SHS in all three settings combined. After multivariate adjustment, the risk in the highest quartile of exposure was significantly higher than that among those without exposure (HR = 1.18, 95% CI, 1.04–1.33 for intensity of exposure; HR = 1.28, 95% CI: 1.11–1.48 for years of exposure; HR = 1.22, 95% CI: 1.06–1.41 for intensity-years of exposure).

When we stratified the SHS exposure into two age groups, exposure happened when the participants were younger than 20 years old and when they were 20 years or older. The results were similar to those found above except that the magnitude of the association between SHS and diabetes was a little lower for SHS exposure occurred before 20 years old (Appendix 2). And when we analyzed the SHS exposure (as continuous variables) in the household, workplace, and social settings separately, as shown in Appendix 3, the associations of diabetes with SHS exposures in each of the three settings were similar before adjusting for SHS exposure in the other two settings, but the associations were slightly stronger in social setting (and in household setting for years of SHS exposure) after adjusting for the exposure in the other two settings.

Table 4 reveals that BMI at baseline and wave 4 (year 2005–2006) and waist and hip circumference reported at wave 2 (in 1997–1998) all had potential mediation effects on the association between SHS and risk of type II diabetes. Among them, waist circumference

measured at wave 2 had the strongest mediation effect with more than 50% of the exposure effects explained ($P < .0001$), followed by BMI at wave 4, which explained 49.5%–55.0% of the excess risk of diabetes associated with SHS. Hip circumference at wave 2 and baseline BMI also explained a substantial proportion of those associations, but at a lower magnitude (36.5%–51.5%). Waist-to-hip ratio did not explain a significant proportion of the association of interest.

As shown in Table 5, the HRs across different BMI and waist strata were similar to each other, and none of the interactions between SHS and BMI or waist circumference were statistically significant.

Discussion

In this large cohort study of female California public school teachers who were followed for up to 17 years, we found exposure to SHS to be significantly associated with higher risk of incident type II diabetes in a dose–response relationship after adjusting for baseline BMI and other potential confounders. This is consistent with previous studies that reported a positive association between SHS and risk of diabetes [7–10]. The multivariable-adjusted HRs in the present study were consistent with the pooled relative risk from recent meta-analyses [5], ranging from 1.18 to 1.28 for those in the upper quartile of SHS exposure defined by intensity and/or duration, adding another piece of supportive evidence to establish SHS as a causal risk factor for type II diabetes.

More importantly, our findings expand the existing literature on the association between SHS and type II diabetes by providing evidence for the potential mediator role of body size measures in this association. It is well recognized that the relationship between active smoking and obesity is complicated. Many cross-sectional studies reported that body weight or BMI is lower in current smokers than that in nonsmokers [24–31]. However, ample evidence indicated that heavy smoking might be associated with a greater risk of obesity [28–31]. Furthermore, multiple studies have shown that smoking is associated with abdominal obesity as measured by waist-to-hip ratio or waist circumference, even after adjusting for BMI [24,32]. This may partially explain the apparently paradoxical observation of increased metabolic risk among active smokers, despite their overall lower BMI [33].

Many studies investigating the association between SHS and obesity focused on prenatal exposure and discovered a positive association between maternal smoking in pregnancy and obesity in childhood [34]. It is hypothesized that prenatal exposure to tobacco smoking “imprints” the fetus to a higher likelihood of becoming obese [35]. Another study reported that adolescents exposed to environmental tobacco smoke had four times greater risk of being overweight than those not exposed [36]. Here we found that participants with higher levels of accumulative SHS exposure had significantly higher age-adjusted BMI at baseline and larger waist circumference 2 years after baseline in a dose–response manner. In animal models, early life nicotine exposure was found to induce adipocyte hypertrophy and leptin resistance [37]. Also, nicotine-exposed neonates with growth retardation *in utero* exhibited “catch-up’ growth and an expansion of adipose stores when provided with excess calories

[38]. These are all potential biological pathways to explain the association of SHS and higher level of overall body size and central obesity observed in this study.

When adjusting BMI in the regression models for active smoking and type II diabetes, the association usually became stronger [6,39,40]. This may be caused by negative confounding as BMI is usually lower among current smokers. However, in most studies examining the association between SHS and diabetes, the association was attenuated after controlling for participants' BMI and waist circumference [11,13,14,17]. We also found the HRs of SHS for type II diabetes were reduced after adding BMI to the regression models, consistent with our hypothesis that obesity may be an intermediate variable in the pathway from SHS to type II diabetes. Among the BMI measures, BMI at wave 4 (i.e., 10 years after baseline) appeared to have the strongest mediating effect on the association of interest. Meanwhile, waist circumference at wave 2 (i.e., 2 years after baseline) had an even stronger mediating effect, explaining 58% of this association. These imply visceral fat accumulation may be an important mechanism explaining the increased risk of type II diabetes among nonsmokers with SHS exposure [29].

The strengths of this study include its large sample size, availability of many potential confounders or mediators for the association of interest, as well as comprehensive evaluation of lifetime exposure to SHS from various settings. It is the first study to report a dose–response relationship between years of SHS exposure and risk of type II diabetes, whereas all the previous studies have only reported categorical SHS status or an intensity measure of a person's exposure on a typical day [17]. Furthermore our data allowed us to estimate the magnitude and statistical significance of the mediating effects of BMI and central obesity measures on the association between SHS and diabetes.

This study also has several limitations. First, both SHS, diabetes incidence, BMI, and waist circumference were all self-reported, and the intensity of SHS exposure was based on subjective classification. Yet, self-reported diabetes status has been demonstrated to have substantial to almost perfect agreement with diagnosis from medical records or claim-based data [41,42] and has been validated in several large longitudinal cohort studies [43,44]. Similarly, SHS history reporting has been shown to have high reliability [45] and validity, especially the self-reported assessment in one's own home [46]. The measurement errors in self-reported exposure likely underestimated the association between SHS and diabetes, whereas the measurement errors in potential mediators (i.e., BMI and waist circumference) could have biased the estimated mediational proportion in either direction.

Second, the year of diabetes diagnosis was also self-reported in the 2012–2013 questionnaires, which was subject to memory recall errors and limited the inference of this study to those who completed the 2012–2013 questionnaires. Thus, due to loss to follow-up, information on incident diabetes was only available for 54% of the nonsmokers identified at baseline, which might be a biased sample if nonrandom censoring happened. However, a comparison of nonsmokers included in and excluded from the final analytical sample revealed comparable participant characteristics (Appendix 1). Furthermore, the estimated diabetes incidence rate based on our final analytical sample was 3.85 per 1000, which is very

close to the diabetes incidence of nonsmokers in the Nurse Health Study (1902/505438 = 3.76 per 1000) [11].

Another limitation is that the CTS only recruited female participants who had worked in California with the majority being non-Hispanic White, so that our findings may not be generalizable to males, race/ethnic minority populations, blue collar workers, or individuals never worked in California. California is a unique state to study the effects of SHS due to the existence of the highly successful California Tobacco Control Program, which resulted in substantial reduction in adult cigarette consumption and introduction of legislation that restricted smoking in workplace and public in California in the past two to three decades [47]. Yet, all our SHS measures were collected between 1995 and 1998. Thus, it is impossible for us to assess the time-varying effects of SHS exposure in California.

Finally, the “difference method” we used to estimate the mediation proportion does not prove the existence of a mediational relationship. Several other possibilities might cause similar changes in regression coefficients after adding a variable into the regression model, such as positive confounding, selection bias, or a combination of them. Furthermore, multiple assumptions are required when estimating mediation proportion based on the “difference method,” including no uncontrolled exposure–outcome and mediator–outcome confounding, and no mediator–exposure interaction. Although our data satisfy the no interaction assumption as shown by Table 5, the other two assumptions cannot be tested. However, extensive simulation study has shown the point estimator for the mediation proportion is consistent under a rare outcome assumption for Cox model, which is fulfilled in most chronic disease incidence studies [23]. In addition, the temporal order of the exposure, potential mediator, and outcome of our mediational analysis is not completely clear. Although the exposure was reported at baseline or 2 years after baseline and the measurement time of potential mediators was at or after baseline, body size may have been characteristic of that measure before baseline or SHS exposure. Conversely, some of the potential mediators might have occurred after the diagnosis of diabetes.

In summary, our results provide further support for the hypothesis that SHS exposure is associated with an increased risk of incident type II diabetes among nonsmokers. They also suggest that central obesity may be an important mediator but not an effect modifier for this association. This study is the first that calculated the magnitude of mediation by waist circumference and BMI for the association between SHS and diabetes, which provides important insight into the potential mechanisms explaining the metabolic effects of SHS exposure. Future research to further elucidates the pathways explaining the association between smoking, SHS, and metabolic disorders is important for designing innovative interventions to reduce the heavy burden of this pandemic chronic disease.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Baseline (1995) characteristics by secondhand smoke exposure among lifetime nonsmokers who responded to the 2012–2013 questionnaires in the California Teachers Study

Baseline characteristic	**Household secondhand smoke exposure**				***P****
	No exposure	**Childhood only**	**Adult only**	**Childhood and adult**	
N	11,744	13,554	5068	9084	
Non-Hispanic white (%)	86.4	88.5	84.7	87.5	<.0001
Family history of diabetes (%)	17.7	19.2	20.2	20.7	<.0001
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	
Age (y)	45.8 (12.1)	46.8(10.2)	52.6 (11.9)	52.3 (10.6)	<.0001
BMI (kg/m^2)	24.0 (4.8)	24.5 (4.9)	24.5 (4.6)	25.0 (5.0)	<.0001
Strenuous or moderate exercise (h/wk)	4.6 (3.9)	4.7 (4.0)	4.3 (3.9)	4.2 (3.8)	<.0001
Alcohol consumption (g/d)	5.2 (7.5)	6.4 (7.7)	6.9 (8.9)	7.6 (8.9)	<.0001
Dietary factors (per day)					
Total calories (kcal)	1628.5 (547.1)	1636.0 (550.8)	1610.5 (554.2)	1613.8 (560.0)	.0067
Magnesium (mg)	261.9 (95.8)	259.3 (94.1)	259.4 (93.8)	255.0 (91.9)	<.0001
Calcium (mg)	825.1 (459.5)	796.4 (446.7)	775.9 (434.9)	758.4 (426.4)	<.0001
Vitamin D (IU)	192.2 (131.1)	183.2 (126.6)	184.1 (124.5)	178.4 (122.3)	<.0001
Total fat (mg)	56.8 (25.9)	57.3 (26.2)	56.9 (26.4)	57.5 (27.1)	.1834
Total saturated fat (mg)	18.8 (9.4)	18.8 (9.4)	18.6 (9.4)	18.8 (9.7)	.6064
Total fiber (g)	15.7 (6.5)	15.4 (6.3)	15.6 (6.4)	15.2 (6.4)	<.0001l
	Intensity-years of secondhand smoke exposure in three settings[†]				***P****
	<5	**5.1–25**	**25.1–50**	**>50**	
N	8052	10,885	7575	7787	
Non-Hispanic white (%)	86.5	87.6	88.2	89.4	<.0001
Family history of diabetes (%)	16.9	18.2	20.2	22.3	<.0001
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	
Age (y)	44.8 (12.0)	47.2 (11.1)	49.6 (10.9)	53.3 (10.5)	<.0001
BMI (kg/m^2)	23.7 (4.5)	24.2 (4.7)	24.6 (4.8)	25.2 (5.2)	<.0001
Strenuous or moderate exercise (h/wk)	4.6 (3.9)	4.5 (3.9)	4.4 (3.8)	4.5 (4.0)	.0123
Alcohol consumption (g/d)	5.1 (7.1)	6.2 (7.9)	7.0 (8.8)	7.4 (8.9)	<.0001

Dietary factors (per day)					
Total calories (kcal)	1627.9 (539)	1632.3 (550.8)	1619.2 (547.6)	1634.6 (566.2)	.3327
Magnesium (mg)	261.3 (94.8)	261.7 (95.3)	258.7 (93.5)	259.3 (94.1)	.1089
Calcium (mg)	825.8 (454.1)	809.6 (453.6)	783.2 (440.9)	770.9 (438.9)	<.0001
Vitamin D (IU)	191.6 (129.6)	187.7 (129.0)	182.5 (126.3)	180.9 (124.7)	<.0001
Total fat (mg)	56.6 (25.4)	57.0 (26.2)	56.8 (26.2)	58.2 (27.3)	.0006
Total saturated fat (mg)	18.7 (9.1)	18.8 (9.5)	18.7 (9.5)	19.0 (9.7)	.1603
Total fiber (g)	15.6 (6.5)	15.6 (6.4)	15.4 (6.4)	15.6 (6.5)	.1082

*P of χ^2 test assessing distribution of categorical variable in secondhand smoke exposure groups or analysis of variance test assessing the distribution of continuous variable among secondhand smoke exposure groups.

†The three settings are the household, workplace, and social settings.

Table 2

Average age-adjusted anthropometric measurements by secondhand smoke exposure among lifetime nonsmokers who responded to the 2012–2013 questionnaires in the California Teachers Study

Anthropometric measurements	**Household secondhand smoke exposure**				***P****
	No exposure	**Childhood only**	**Adult only**	**Childhood and adult**	
N	11,744	13,554	5068	9084	
BMI at baseline (kg/m^2)	24.1	24.5	24.3	24.9	<.0001
BMI at wave 4 (kg/m^2)	25.3	25.8	25.6	26.2	<.0001
Waist-to-hip ratio at wave 2[//]	0.793	0.793	0.794	0.797	.0028
Waist circumference at wave 2[//] (inches)	31.4	31.7	31.6	31.9	<.0001
Hip circumference at wave 2[//] (inches)	39.5	39.9	39.7	40.0	<.0001
	Secondhand smoke exposure intensity in three settings[¶]				***P**†*
	≤1.0	**1.1–2.0**	**2.1–3.0**	**>3.0**	
N	12,520	7419	5977	8456	
BMI at baseline (kg/m^2)	24.0	24.3	24.5	24.9	<.0001
BMI at wave 4 (kg/m^2)	25.2	25.52	25.82	26.32	<.0001
Waist-to-hip ratio at wave 2[//]	0.793	0.793	0.795	0.797	.0056
Waist circumference at wave 2[//] (inches)	31.3	31.6	31.7	32.1	<.0001
Hip circumference at wave 2[//] (inches)	39.5	39.7	39.9	40.2	<.0001
	Years secondhand smoke exposure in three settings[¶]				***P**‡*
	≤5.0	**5.1–20**	**20.1–40**	**>40**	
N	9004	10,990	8866	6111	
BMI at baseline (kg/m^2)	23.8	24.2	24.7	25.1	<.0001
BMI at wave 4 (kg/m^2)	25.0	25.5	26.1	26.4	<.0001
Waist-to-hip ratio at wave 2[//]	0.793	0.792	0.795	0.798	.0002
Waist circumference at wave 2[//] (inches)	31.3	31.5	31.9	32.2	<.0001
Hip circumference at wave 2[//] (inches)	39.4	39.7	40.1	40.3	<.0001

	Intensity-years of secondhand smoke exposure in three settings[¶]				$p^{\S}$
	≤5.0	**5.1–25**	**25.1–50**	**>50**	
N	8052	10,885	7575	7787	
BMI at baseline (kg/m^2)	23.8	24.2	24.5	25.0	<.0001
BMI at wave 4 (kg/m^2)	24.9	25.5	25.8	26.4	<.0001
Waist-to-hip ratio at wave 2[//]	0.793	0.792	0.794	0.799	<.0001
Waist circumference at wave 2[//] (inches)	31.3	31.5	31.7	32.3	<.0001
Hip circumference at wave 2[//] (inches)	39.4	39.7	39.9	40.3	<.0001

[*] *P* from linear regression models adjusted by continuous age, with null hypothesis: $\beta_{\text{no exposure}} = \beta_{\textit{childhood only}} = \beta_{\textit{adult only}} = \beta_{\textit{childhood and adult}} = 0$.

[†] *P* from linear regression models adjusted by continuous age, with null hypothesis: $\beta_{\leq 1.0} = \beta_{1.1\text{–}2.0} = \beta_{2.1\text{–}3.0} = \beta_{>3.0} = 0$.

[‡] *P* from linear regression models adjusted by continuous age, with null hypothesis: $\beta_{\leq 5.0} = \beta_{5.1\text{–}20} = \beta_{20.1\text{–}40} = \beta_{>40} = 0$.

[§] *P* from linear regression models adjusted by continuous age, with null hypothesis: $\beta_{\leq 5.0} = \beta_{5.1\text{–}25} = \beta_{25.1\text{–}50} = \beta_{>50} = 0$.

[//] Wave 2 questionnaires were completed in year 1997–1998.

[¶] The three settings are the household, workplace, and social settings.

Table 3

Association of secondhand smoke exposure and risk of incident type II diabetes after 17 y of follow-up among lifetime nonsmokers who responded to the 2012–2013 questionnaires in the California Teachers Study

Hazard ratio (HR)	Household secondhand smoke exposure				*P* of trend test*
	No exposure	Childhood only	Adult only	Childhood and adult	
Number of participants	11,691	13,468	5033	9032	
Number of cases (%)	616 (5.3)	796 (5.9)	348 (6.9)	735 (8.1)	
Age-adjusted HR (95% CI)	l (ref)	1.12 (1.00–1.24)	1.12 (0.98–1.29)	1.36 (1.22–1.52)	
Age- and BMI-adjusted HR	l (ref)	1.05 (0.95–1.18)	1.09 (0.94–1.25)	1.22 (1.09–1.36)	
Multivariable-adjusted HR†	l (ref)	1.08 (0.97–1.21)	1.09 (0.95–1.26)	1.25 (1.11–1.41)	.0002
	Secondhand smoke exposure intensity in three settings‡				
	≤1.0	**1.1–2.0**	**2.1–3.0**	**>3.0**	
Number of participants	12,420	7342	5907	8363	
Number of cases (%)	612 (4.9)	406 (5.5)	356 (6.0)	607 (7.3)	
Age-adjusted HR (95% CI)	l (ref)	1.09 (0.96–1.24)	1.20 (1.05–1.38)	1.40 (1.25–1.57)	
Age- and BMI-adjusted HR	l (ref)	1.03 (0.90–1.17)	1.17 (1.02–1.34)	1.23 (1.10–1.39)	
Multivariable-adjusted HR†	l (ref)	0.97 (0.85–1.11)	1.09 (0.95–1.26)	1.18(1.04–1.33)	.0035
	Years of secondhand smoke exposure in three settings‡				
	≤5	**5.1–20**	**20.1–40**	**>40**	
Number of participants	8935	10,891	8784	6014	
Number of cases (%)	404 (4.5)	546 (5.0)	553 (6.0)	520 (8.7)	
Age-adjusted HR (95% CI)	l (ref)	1.05 (0.92–1.19)	1.27 (1.11–1.45)	1.56 (1.35–1.79)	
Age- and BMI-adjusted HR	l (ref)	0.96 (0.84–1.10)	1.10 (0.97–1.27)	1.29 (1.12–1.49)	
Multivariable-adjusted HR†	1 (ref)	0.95 (0.83–1.09)	1.08 (0.94–1.25)	1.28 (1.11–1.48)	.0001
	Intensity-years of secondhand smoke exposure in three settings‡				
	≤5	**5.1–25**	**25.1–50**	**>50**	
Number of participants	7990	10,786	7509	7676	
Number of cases (%)	364 (4.6)	558 (5.2)	441 (5.9)	614 (8.0)	
Age-adjusted HR (95% CI)	1 (ref)	1.07 (0.94–1.23)	1.20 (1.04–1.38)	1.50 (1.31–1.72)	

Age- and BMI-adjusted HR	1 (ref)	1.00 (0.87–1.15)	1.08 (0.93–1.25)	1.26 (1.09–1.44)	
Multivariable-adjusted HR[†]	l (ref)	0.98 (0.85–1.13)	1.05 (0.91–1.22)	1.22 (1.06–1.41)	.0014

[*] *P* of trend test using multivariable-adjusted model.

[†] Multivariable models were adjusted for age, baseline BMI, race (non-Hispanic white vs. other), family history of diabetes, physical activity, alcohol consumption, and dietary factors (daily dietary calories, magnesium, calcium, vitamin D, dietary fiber, total fat, and saturated fat).

[‡] The three settings are the household, workplace, and social settings.

Table 4

Mediation by BMI and waist-to-hip circumference at different time points for the association of secondhand smoke exposure with risk of type II diabetes among lifetime nonsmokers who responded to the 2012–2013 questionnaires in the California Teachers Study

Potential mediator	Explained proportion of effect estimates (95% CI)	*P*
Secondhand smoke exposure intensity in three settings		
BMI at baseline	46.8% (25.9%, 67.8%)	<.0001
BMI in year 2005–2006	55.0% (27.1%, 82.8%)	.0001
Waist circumference in year 1997–1998[*]	*56.4% (28.2%, 84.7%)*	*<.0001*
Hip circumference in year 1997–1998	43.2% (20.3%, 66.1%)	.0002
Waist-to-hip ratio in year 1997–1998	−9.4% (−24.7%, 5.8%)	.2259
Years of secondhand smoke exposure in three settings		
BMI at baseline	36.5% (19.2%, 53.7%)	<.0001
BMI in year 2005–2006	49.5% (24.4%, 74.6%)	.0001
Waist circumference at in year 1997–1998[*]	*50.0% (25.4%, 74.7%)*	*<.0001*
Hip circumference in year 1997–1998	43.9% (21.3%, 66.5%)	.0001
Waist-to-hip ratio in year 1997–1998	−6.5% (−20.0%, 7.1%)	.3479
Intensity-years of secondhand smoke exposure in three settings		
BMI at baseline	46.2% (26.5%, 65.8%)	<.0001
BMI in year 2005–2006	53.1% (26.0%, 80.2%)	<.0001
Waist circumference at in year 1997–1998[*]	*57.9% (32.0%, 83.7%)*	*<.0001*
Hip circumference in year 1997–1998	51.5% (27.1%, 76.0%)	<.0001
Waist-to-hip ratio in year 1997–1998	−3.6% (−16.1%, 8.9%)	.574

[*] Italic font indicates the variable that explained the highest percent of the association between secondhand smoke exposure and diabetes.

Table 5

HRs for diabetes for secondhand smoking exposures across strata of waist circumference and BMI among lifetime nonsmokers who responded to the 2012–2013 questionnaires in the California Teachers Study*

Strata	Household secondhand smoke exposure				*P* for interaction†
	No exposure	Childhood only	Adult only	Childhood and adult	
Waist < median	l (ref)	1.22 (0.82, 1.81)	0.97 (0.56, 1.67)	1.28 (0.82, 1.97)	.78
Waist ≥ median	l (ref)	1.05 (0.91, 1.21)	1.05 (0.88, 1.26)	1.23 (1.06, 1.43)	
BMI <25	l (ref)	1.10 (0.89, 1.37)	1.20 (0.93, 1.56)	1.38 (1.11, 1.72)	.42
BMI ≥25	l (ref)	1.04 (0.91, 1.19)	1.02 (0.86, 1.21)	1.16 (1.01, 1.34)	
	Secondhand smoke exposure intensity in three settings‡				
	≤1.0	**1.1–2.0**	**2.1–3.0**	**>3.0**	
Waist < median	l (ref)	1.13 (0.74, 1.73)	1.25 (0.80, 1.96)	1.29 (0.86, 1.94)	.72
Waist ≥ median	l (ref)	0.93 (0.80, 1.09)	1.06 (0.91, 1.24)	1.14 (0.99, 1.30)	
BMI <25	l (ref)	0.96 (0.75, 1.23)	0.91 (0.70, 1.19)	1.21 (0.97, 1.51)	.19
BMI ≥25	l (ref)	0.95 (0.81, 1.12)	1.13 (0.96, 1.33)	1.15 (0.99, 1.33)	
	Years of secondhand smoke exposure in three settings‡				
	≤5	**5.1–20**	**20.1–40**	**>40**	
Waist < median	l (ref)	0.96 (0.64, 1.45)	0.95 (0.61, 1.47)	1.12 (0.69, 1.82)	.95
Waist ≥ median	l (ref)	0.92 (0.78, 1.08)	1.09 (0.93, 1.28)	1.25 (1.06, 1.47)	
BMI <25	l (ref)	0.89 (0.69, 1.14)	1.08 (0.85, 1.39)	1.27 (0.97, 1.66)	.52
BMI ≥25	l (ref)	0.96 (0.81, 1.13)	1.05 (0.89, 1.25)	1.24(1.04, 1.47)	
	Intensity-years of secondhand smoke exposure in three settings‡				
	≤5	**5.1–25**	**25.1–50**	**>50**	
Waist < median	1 (ref)	0.95 (0.62, 1.46)	1.07 (0.68, 1.69)	1.08 (0.68, 1.73)	.94
Waist ≥ median	l (ref)	0.95 (0.81,1.12)	1.02 (0.86, 1.20)	1.17 (0.99, 1.37)	
BMI <25	l (ref)	0.97 (0.76, 1.25)	1.07 (0.82, 1.40)	1.21 (0.93, 1.58)	.31
BMI ≥25	1 (ref)	0.96 (0.80, 1.14)	1.01 (0.84, 1.21)	1.18 (0.99, 1.40)	

*Multivariable Cox proportional hazards regression models were adjusted for age, baseline BMI, race (non-Hispanic white vs. other), family history of diabetes, physical activity, alcohol consumption, and dietary factors (daily dietary calories, magnesium, calcium, vitamin D, dietary fiber, total fat, and saturated fat). Waist circumference was reported in year 1997–1998. Median of waist was 30.5 inches. BMI was reported at baseline questionnaire.

† *P* for joint test for interaction for secondhand smoking exposure categories with waist circumference and BMI as binary variables.

‡ The three settings are the household, workplace, and social settings.