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ORIGINAL INVESTIGATION

Early Life Exposure to Cigarette Smoke and Depressive Symptoms Among Women in Midlife

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ABSTRACT

Introduction: Active cigarette smoking has consistently been associated with depression, but little is known about the association between other cigarette smoke exposures, particularly in early life, and depression. We investigated whether exposures to maternal smoking during pregnancy (MSP) and childhood secondhand smoke (SHS) are associated with depressive symptoms in midlife.

Methods: Pregnant mothers were enrolled and were provided data on maternal smoking and other parental characteristics. Female offspring were followed through age 7 years and again in midlife (age range = 38–44 years), when they provided data on smoking history, SHS across the life course, and current depressive symptoms using the Center for Epidemiological Studies Depression Scale (CES-D).

Results: Participants exposed to MSP had a higher risk for depression (risk ratio [*RR*] = 1.83, 95% confidence interval [CI] = 1.08, 3.09) than those without MSP exposure. Relative to those with no MSP and no childhood SHS exposures, participants with MSP and childhood SHS had more than twice the risk of depressive symptoms (*RR* = 2.40, 95% CI = 1.07, 5.41). Further adjustment for adult factors, particularly current smoking, substantially reduced these associations (e.g., MSP vs. no MSP exposure: *RR* = 1.36 [95% CI = 0.75, 2.45]).

Conclusions: Early life exposure to cigarette smoke is associated with increased risk for depression in midlife, with the association largely mediated by active smoking. These findings support a role for early life cigarette exposures in shaping smoking and depression risks in later life, and they provide some support for the direction of smoke exposure influence on depression.

INTRODUCTION

Depression is estimated to affect 7%–9% of adult Americans (Centers for Disease Control Prevention, 2010a; Kessler, Chiu, Demler, & Walters, 2005), with its prevalence projected to increase in the next 15 years (World Health Organization, 2008). A relatively large body of research has identified a range of predictors of depression, which include stressful life events, gender, age, family history, alcohol and drug use disorders, and childhood adversity (Kendler & Prescott, 1999; Kessler & Magee, 1993; Maughan, 2002; Mirowsky & Ross, 1992; Piccinelli & Wilkinson, 2000; Silberg et al., 1999; Sullivan, Neale, & Kendler, 2000). Available evidence also points to cigarette smoking as a risk factor for depression among adults (Boden, Fergusson, & Horwood, 2010; Breslau, Peterson, Schultz, Chilcoat, & Andreski, 1998; Covey, 1998; Fergusson, Goodwin, & Horwood, 2003; Flensborg-Madsen, 2011; Glassman et al., 1990; Kendler et al., 1993; Kinnunen et al., 2006; Nakata et al., 2008; Pasco et al., 2008). Less research, however, has explored the relationship between other forms of or indirect exposures to cigarette smoke, including secondhand smoke (SHS), and depression. Furthermore, due to the paucity of long-term studies with reliable data on cigarette smoke exposures over the life course, even less is known about the potential effect of timing and/or accumulation of these exposures on depression in adulthood. Such information can substantially improve our understanding of the underlying mechanisms (e.g., directionality of the association between cigarette smoke exposure and depression) and provide important information for primary prevention of depression.

Recent declines in smoking prevalence and restrictive regulations on public smoking have led to an overall decline in SHS exposure among adults (Callinan, Clarke, Doherty, & Kelleher, 2010; Centers for Disease Control Prevention, 2010b; Graham & Geoff, 1999; Pickett, Schober, Brody, Curtin, & Giovino, 2006). Children and adolescents, however, have the highest prevalence of SHS exposure (Graham & Geoff, 1999; Pirkle, Bernert, Caudill, Sosnoff, & Pechacek, 2006; U.S. Department

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of Health and Human Services, 2007), with approximately 35%–80% of children in the United States exposed to SHS in public areas (Kum-Nji, Meloy, & Herrod, 2006), and 22% exposed at home (U.S. Department of Health and Human Services, 2007). Exposure to SHS has been associated with increased risk of depression and other mental disorders among youth (Bandiera, Richardson, Lee, He, & Merikangas, 2011; Herrmann, King, & Weitzman, 2008), and exposure to SHS in adulthood has been associated with increased depression and psychological distress among never-smokers in adulthood (Bandiera et al., 2010; Nakata et al., 2008). Another form of cigarette smoke exposure, in utero exposure to maternal smoking during pregnancy (MSP), has been examined in relation to depression among children and young adult offspring in only two studies, which have yielded mixed results (Fergusson, Woodward, & Horwood, 1998; Weissman, Warner, Wickramaratne, & Kandel, 1999). Clearly, more research is needed to simultaneously examine active smoking, MSP and SHS exposures across the life course in relation to adult depression.

We used data from an adult follow-up study of a female birth cohort that enrolled pregnant mothers and their offspring to examine the association between early life exposures to cigarette smoke, assessed through MSP and presence of adult smoker(s) in childhood households (childhood SHS), and depression symptoms in mid-adulthood. We further investigated whether any associations between early life cigarette smoke exposures and depressive symptoms in midlife would be explained by adult exposures to cigarette smoke, including household SHS exposure in adulthood and adult active smoking.

METHODS

Study Population and Data Collection

We used data from the New York Women's Birth Cohort, an adult follow-up study of former child participants in the National Collaborative Perinatal Project (CPP). The CPP collected extensive clinical and epidemiologic data on more than 55,000 pregnant women and their offspring across 12 U.S. sites (Broman, 1984; Terry, Wei, & Esserman, 2007). In 2001, the New York Women's Birth Cohort began to trace and recruit female offspring who were born between 1959 and 1963 in the New York site of CPP and prospectively followed through age 7. In total, 375 female offspring were successfully traced, and 262 (70% of those traced) completed an adult follow-up questionnaire. Here, we used data from 178 women (47% of those traced or 68% of those enrolled) who completed a more extensive questionnaire that included reports of depressive symptoms (average age at adult follow-up = 41 years, *SD* =1.5 years). Tracing and recruitment of adult participants were not associated with maternal and pregnancy characteristics including maternal smoking, and infant or early childhood growth, but we were more successful in tracing women whose mothers provided social security numbers in the childhood phase of the study and whose family had higher childhood socioeconomic status (for more details on study design, please see Terry, Flom, Tehranifar, & Susser, 2009).

The original CPP collected sociodemographic, behavioral, and clinical data from mothers and their offspring following a protocol standardized across all sites; details have been previously published (Broman, 1984; Hardy, 2003). These early life data included parental socioeconomic factors, maternal characteristics, and smoking behavior. Adult offspring provided additional data as part of the New York Women's Birth Cohort follow-up study, which included smoking behavior, exposure to household tobacco smoke in childhood and adulthood, childhood family structure, and indicators of adult socioeconomic status (Tehranifar, Liao, Ferris, & Terry, 2009; Terry et al., 2007, 2009).

This study was approved by the institutional review board of Columbia University Medical Center.

Measures

Early Life Exposures to Cigarette Smoke

We used two measures of early life smoke exposure. Our primary measure of interest was exposure to MSP, which was assessed using maternal self-reports of their own smoking status collected at the time of pregnancy with the offspring in the study. Data on childhood SHS exposure was collected from adult offspring in response to the following question: "As a child, did any member of your household, including caregivers, smoke in your presence?" In addition to using MSP and childhood SHS separately, we combined these variables to create an overall measure as follows: no MSP and no childhood SHS exposures, childhood SHS with no MSP exposure, and MSP with or without childhood SHS exposures. The latter category included both MSP with and without childhood SHS, because only three participants with MSP exposures did not have childhood SHS exposure.

Adult Exposures to Cigarette Smoke

Similar to childhood SHS, exposure to adult SHS was measured by self-reported responses to the following question: "As an adult, did any member of your household smoke in your presence?" Current active smoking status was assessed through standard questions on smoking history. Participants who reported having smoked at least one cigarette per day for one month or longer and participants who reported currently smoking were categorized as current smokers; all others were categorized as current nonsmokers.

Depressive Symptoms

As part of the adult follow-up questionnaire, participants provided responses to the Center for Epidemiological Studies Depression Scale (CES-D), a widely used measure of recent symptoms of depression. The CES-D assesses the frequency of 20 symptoms in the past week reported on a 4-point Likert scale, ranging from "rarely or none of the time (less than 1 day) to "most or all of the time (5–7 days)" (Radloff, 1977). Consistent with the established measurement of CES-D, scores were summed across the items and subsequently dichotomized using the standard cutpoint into depressive symptoms and no depressive symptoms (CES-D score ≥ 16 and <16, respectively) (Radloff, 1977).

Covariates

We tested whether several early life factors potentially associated with adult depressive symptoms exerted any confounding effect on the associations between early life cigarette smoke exposures and adult depressive symptoms. Indicators of parental socioeconomic status assessed around the time of

participants' birth included maternal education (reported in number of years and dichotomized into less than high school and high school or higher education), annual family income at birth (in categories of less than or equal to $1,300 per capita and greater than $1,300), and parental occupation. In addition to the individual variables, a summary measure of parental socioeconomic factors, developed in the original CPP, was also evaluated (Broman, 1984). Data on parental socioeconomic status along with maternal age at pregnancy were collected through the original CPP at the time of pregnancy and birth. We also examined other covariates for which data were collected as part of the adult follow-up questionnaire and included self-identified race/ethnicity (non-Hispanic white, non-Hispanic African American and Hispanic), and childhood family structure through age 13 (two-parent vs. single-parent or other types of households).

Statistical Analysis

Prevalence of MSP and depressive symptoms in adulthood were examined in relation to other covariates using *t*-tests and chi-square tests. Associations between each exposure variable of cigarette smoke and depressive symptoms were separately analyzed through relative risk regression to produce relevant risk ratios and 95% confidence intervals (CIs). We used log binomial regression because the depression outcome was common in our sample (~25%) (Lumley, Kronmal, & Ma, 2006). We assessed confounding through a minimum of 10% change in the estimate of the associations of MSP and childhood SHS with depression, when the potential confounding variable was added to the model. The variables meeting this confounding criterion included several measures of parental SES, which captured different dimensions of the overall construct and were positively correlated. To reduce multicollinearity, one early life socioeconomic variable, maternal education, which showed the most consistent and/or largest change in the estimate of the association for MSP, childhood SHS and combination of MSP and childhood SHS, was used in multivariable analyses.

We examined the univariable associations of depression separately with each of the early life tobacco exposure variables, followed by modeling the same associations adjusted for maternal education. We further added adult SHS and adult smoking status to each model to examine the degree to which these adult exposures mediated the associations between early life cigarette smoke exposures and adult depression. We also examined the associations between MSP and depression stratified by current active smoking status (current smokers vs. nonsmokers).

RESULTS

Approximately 41% of mothers reported smoking during pregnancy. Participants exposed to MSP were more likely than those not exposed to have younger mothers at pregnancy ($p = .01$), lower family income at birth ($p = .04$) and single-parent or other types of households through age 13 ($p = .006$). Participants exposed to MSP were also more likely to have childhood and adult SHS exposures, use depression medication, and be current smokers (see Table 1). For example, 42% of participants exposed to MSP were current adult smokers compared with 13% of participants who were not exposed to MSP ($p < .0001$).

One quarter of participants had a CES-D score of 16 or higher, suggesting depressive symptomatology. Overall, there were no differences in parental sociodemographic factors between participants with and without depressive symptoms (Table 2). Of the 20 participants (11.7% of the total sample) who currently used medication to treat depressive symptoms, 7 had a CES-D score <16. We repeated our analysis categorizing these 7 participants first as depressed and then as nondepressed. Our results were not different across these two categorizations, and we therefore present our results with participants classified into depressive status based on CES-D score only.

Table 3 shows the results of relative risk regression analyses for the associations between each early life cigarette smoke exposures—that is, MSP, childhood SHS, and the combination of MSP and childhood SHS—and depressive symptoms. The unadjusted risk ratio for the association between MSP and depressive symptoms was 1.73 (95% CI = 1.04, 2.89; Panel 1, Model 1), which remained statistically significant after adjustment for maternal education (*RR* = 1.83, 95% CI = 1.08, 3.09; Panel 1, Model 2). Childhood SHS also had a positive, but statistically nonsignificant, association with depression (*RR* = 1.81, 95% CI = 0.86, 3.77, Panel 2, Model2). Compared to participants with no MSP and no childhood SHS, those exposed to MSP with or without childhood SHS had over two times the risk of depressive symptoms (*RR* = 2.4, 95% CI = 1.07, 5.41; Panel 3, Model 2). The association between exposure to childhood SHS and depressive symptoms was also positive, but did not reach statistical significance (*RR* = 1.54, 95% CI = 0.64, 3.74; Panel 3, Model 2).

Adjusting for adult SHS did not significantly affect the associations between early life cigarette smoke exposures and depression (Panels 1–3, Model 3). The largest effect was observed after adjustment for adult active smoking status, with risk ratios of depression associated with all early life smoke exposures remaining elevated, but no longer statistically significant (Panels 1–3, Model 4). The adjustment for current active smoking reflects a 32% reduction in the parameter estimate (beta coefficient) of the association between MSP and depression. In the fully adjusted models (Panels 1–3, Model 5), the risk of depressive symptoms associated with MSP exposure and with childhood SHS were 1.36 (95% CI = 0.75, 2.45) and 1.40 (95% CI = 0.66, 2.96), respectively. For MSP with or without childhood SHS and for child SHS but no MSP, relative to no MSP and no childhood SHS, the risk ratio of depressive symptoms were 1.69 (95% CI = 0.73, 3.93) and 1.45 (95% CI = 0. 59, 3. 58), respectively (Panels 1–3, Model 5). Finally, we tested for statistical interaction between MSP and adult active smoking and did not find any evidence to suggest that the association between MSP and adult depression varied by active smoking status (data not shown).

DISCUSSION

We considered whether early life exposures to cigarette smoke through MSP and childhood SHS were associated with increased risk of depressive symptoms in mid-adulthood. Our results supported such associations by showing that among adult female offspring, aged 38–44 years, those with exposure to MSP alone or in combination with childhood SHS had a nearly two-fold increased risk of depressive symptoms compared with those without these exposures. To a large extent,

Table 1. Sample Characteristics by Exposure to Maternal Smoking During Pregnancy

	MSP ($N = 72$)	No MSP ($N = 104$)
	$M \pm SD$ or n (%)	$M \pm SD$ or n (%)
Race/ethnicity		
Non-Hispanic African American	23 (32)	34 (33)
Hispanic	23 (32)	44 (42)
Non-Hispanic White	26 (36)	36 (25)
Age at interview (year)	41.1 ± 1.6	41.3 ± 1.5
Early life		
Maternal age at enrollment (year)	25.0 ± 5.5	27.2 ± 6.1**
Years smoked at enrolment	7.6 ± 5.1	0.9 ± 2.2
Maternal education at enrolment		
< High school	26 (36)	51 (50)
≥ High school	46 (64)	52 (50)
Paternal occupation		
Blue collar	61 (90%)	89 (89)
White collar	7 (10)	11 (11)
Annual family income per capita		
≤1,300	43 (63)	48 (47)*
>1,300	25 (37)	54 (53)
Socioeconomic Status Index	54.5 ± 17.5	53.8 ± 18.8
Childhood		
Child household SHS exposure		
Yes	69 (96)	60 (58)***
No	3 (4)	44 (42)
Family structure through age 13		
Single parent	24 (33)	16 (15)**
Both parents	48 (67)	88 (85)
Adulthood		
Adult smoking status		
Current	30 (42)	13 (13)***
Noncurrent	42 (58)	90 (87)
Adult household SHS		
Yes	48 (67)	44 (43)**
No	24 (33)	58 (57)
Depression medication use		
Yes	13 (19)	7 (7)**
No	56 (81)	93 (93)
Depressive symptoms score	12.6 ± 9.4	9.1 ± 7.2

Note. MSP = maternal smoking during pregnancy; SHS = secondhand smoke.
*$p \leq .05$. **$p \leq .01$. ***$p \leq .0001$.

the positive associations between early life exposures to cigarette smoke and adult depressive symptoms were explained by participants' current smoking status. Our prior research in this study population has documented strong positive associations between MSP and participants' smoking status in midlife, after accounting for SHS exposures and a range of sociodemographic characteristics (Tehranifar et al., 2009). Taken together, our results suggest that early life exposures to cigarette smoke, most notably MSP, may have an indirect influence on the risk of depressive symptoms in adulthood that may be mediated through adult active smoking. These findings are compatible with the existing knowledge of adult active smoking as a risk factor for adult depression (Boden et al., 2010; Breslau et al., 1998; Covey, 1998; Fergusson et al., 2003; Flensborg-Madsen, 2011; Glassman et al., 1990; Kendler et al., 1993; Kinnunen et al., 2006; Nakata et al., 2008; Pasco et al., 2008), and the limited prior research suggesting a link between exposure to passive smoking and depression (Bandiera et al., 2010).

Two studies that investigated the relationship between MSP and depression in the offspring have focused on children and young adults—those between 18 and 36 years of age (Fergusson et al., 1998; Weissman et al., 1999). One study reported no association between MSP and *Diagnostic and Statistical Manual* (*DSM*)-III major depressive disorder in a cohort of 77 female offspring who were approximately 27 years of age (Weissman et al., 1999). In another study, which included over 1,000 children followed to age 18, the authors observed a positive association between MSP and *DSM-IV* depressive symptoms; however, these findings were not statistically significant after adjusting for possible confounders and mediators (Fergusson et al., 1998). Both of these studies considered severe depression as the outcomes, and the study by Weissman and colleagues (1999) relied on retrospectively recalled data for MSP. Our analysis extends this limited area of research by assessing MSP through maternal report at the time of their pregnancy with the offspring, further examining exposure to childhood SHS alone and in combination with

Table 2. Sample Characteristics by Depressive Symptoms

	Depressive symptoms ($N = 44$)	No depressive symptoms ($N = 134$)
	$M \pm SD$ or n (%)	$M \pm SD$ or n (%)
Race/ethnicity		
Non-Hispanic African American	20 (45)	48 (36)
Hispanic	12 (27)	45 (34)
Non-Hispanic White	12 (27)	41 (31)
Age at adult follow-up (year)	41.1 ± 1.5	41.3 ± 1.5
Early life		
Maternal age at enrollment (year)	25.2 ± 5.7	26.6 ± 6.0
Years smoked at enrolment	4.8 ± 5.2	3.3 ± 4.8
Maternal education at enrolment		
< High school	19 (44)	58 (43)
≥ High school	24 (56)	76 (57)
Paternal occupation		
Blue collar	40 (95)	111 (87)
White collar	2 (5)	17 (13)
Annual family income per capita		
≤1,300	25 (61)	66 (50)
>1,300	16 (39)	65 (50)
Socioeconomic Status Index	51.5 ± 19.04	55.2 ± 18.02
Childhood		
Child household SHS exposure		
Yes	36 (82)	95 (71)
No	8 (18)	39 (29)
Family structure through age 13		
Single parent	10 (23)	30 (22)
Both parents	34 (77)	104 (78)
Adulthood		
Adult smoking status		
Current	18 (41)	25 (19)**
Noncurrent	26 (59)	108 (81)
Adult household SHS		
Yes	28 (65)	65 (49)
No	15 (35)	68 (51)
Depression medication use		
Yes	12 (29)	8 (6%)***
No	29 (71)	122 (94)

Note. SHS = secondhand smoke.
*$p \le .05$. **$p \le .01$. ***$p \le .0001$.

MSP, and focusing on depression in midlife. We observed a higher risk of depressive symptoms for those exposed to both MSP and childhood SHS suggesting that cumulative exposures may have a larger influence on depression than exposure to only one exposure. Our results also add to the current literature by considering adult exposures to cigarette smoke in the form of passive and active adult tobacco exposures as possible mediators. The potential for mediation of these associations by adult active smoking is further supported by prior studies, including a study conducted in this study population, linking exposure to MSP to increased risk of the offspring smoking in adolescence, as well as in adulthood (Cornelius, Goldschmidt, & Day, 2012; Kandel & Udry, 1999; Rydell, Cnattingius, Granath, Magnusson, & Galanti, 2012; Tehranifar et al., 2009). Furthermore, our findings of early life influences of smoke exposure on adult depression provide insight to clarify the directionality of the association between tobacco smoke exposure and depressive symptoms, which are often examined in cross-sectional studies. If replicated in other larger prospective studies, our results suggest that smoke exposure may lead to depressive symptoms, rather than depressive symptomology contributing to smoking behavior.

The small sample size in our study may have reduced the ability to detect statistically significant associations. Thus, it is important to note that the risk of depressive symptoms associated with early life exposures to tobacco smoke, while statistically nonsignificant in the final models, remained elevated. Another limitation of our study was the lack of data collection on several important factors, including paternal smoking at the time of pregnancy and maternal smoking history prior to pregnancy. Furthermore, data on parental depression experiences and personal past depression experiences were not available. Maternal history of depression may have differed by maternal smoking status during pregnancy and/or childhood, and together with the familial risk of depression may have influenced the observed associations (Kendler, 2006; Kendler, Davis, & Kessler, 1997). Our measure of depression focused on current symptoms and did not capture history of depression; thus, the timing of depression onset in relation to active smoking behavior remains unclear. Given that very

Table 3. Risk Ratio (*RR*) and 95% Confidence Interval (CI) of Depressive Symptoms in Female Adults' Offspring Based on Smoking Exposure Over the Lifecourse

	Depressive symptoms (CES-D score ≥ 16)	No depressive symptoms (CES-D score < 16)	Model 1	Model 2	Model 3	Model 4	Model 5
			Unadjusted (crude) (n = 176)	Adjusted for maternal education (n = 175)	Model 2 + adult SHS exposure (n = 174)	Model 2 + adult active smoking status (n = 172)	Model 2 + adult SHS, adult active smoking status and adult education (n = 172)
	N	*N*	*RR* (95% CI)	*RR* (95% CI)	*RR* (95% CI)	*RR* (95% CI)	*RR* (95% CI)
Panel 1							
MSP							
No	20	84	Reference	Reference	Reference	Reference	Reference
Yes	24	48	1.73 (1.04, 2.89)	1.83 (1.08, 3.09)	1.75 (1.01, 3.02)	1.46 (0.83, 2.54)	1.36 (0.75, 2.45)
Panel 2							
Childhood SHS							
No	8	39	Reference	Reference	Reference	Reference	Reference
Yes	36	95	1.61 (0.81, 3.22)	1.81 (0.86, 3.77)	1.54 (0.72, 3.27)	1.64 (0.79, 3.39)	1.40 (0.66, 2.96)
Panel 3							
MSP and childhood SHS							
No MSP and no child SHS	7	37	Reference	Reference	Reference	Reference	Reference
No MSP with child SHS	13	47	1.36 (0.59, 3.13)	1.54 (0.64, 3.74)	1.32 (0.53, 3.25)	1.66 (0.69, 4.00)	1.45 (0.59, 3.58)
MSP with or without child SHS[a]	24	48	2.10 (0.99, 4.45)	2.40 (1.07, 5.41)	2.09 (0.91, 4.80)	1.96 (0.86, 4.43)	1.69 (0.73, 3.93)

Note. CES-D = Center for Epidemiological Studies Depression Scale; SHS = secondhand smoke; MSP = maternal smoking during pregnancy.
[a]This category is comprised of 21 participants with MSP and childhood SHS and three participants with MSP and no childhood SHS.

few participants with MSP did not have exposure to childhood SHS, we were unable to tease apart the independent effects of these exposures. Our results are limited in their generalizability to men as the sample only included female offspring.

Strengths of the current study include the use of highly valid measure of MSP exposure. Specifically, data for MSP were reported by mothers during pregnancy and not retrospectively, which reduces the potential for recall error. Furthermore, validity concerns regarding social desirability bias of self-reported MSP are minimized given that these data were collected in the late 1950s to early 1960s, when the adverse effects of smoking during pregnancy were not widely known, and this behavior was not highly stigmatized (Martin & Dombrowski, 2008). Data on pregnant mothers' report of their smoking were also confirmed against serum cotinine levels within a sample of the original CPP cohort and found to be highly accurate (kappa = 0.83) (Klebanoff, Levine, Clemens, DerSimonian, & Wilkins, 1998). Our childhood SHS data relied on information retrospectively collected from adult participants. A literature review that evaluated the validity of reports of lifetime SHS exposure in six studies found 80% or higher level of agreement between an individual and their next of kin, including adult offspring reports of childhood SHS exposure compared with parental reports of smoking during their offspring's youth (Barry, 1997). Self-reported active smoking have also been shown to have high accuracy, with self-reported smoking status evaluated against biological markers of cigarette smoke exposure (e.g., cotinine or carbon monoxide levels) showing an average sensitivity and specificity of 88% and 89.2%, respectively (Patrick et al., 1994). The CES-D is an internally reliable measure of depressive symptoms that has been validated in different populations; the Cronbach alpha for CES-D in our sample was high at 0.85 and nearly identical to the same measure in the general population (Radloff, 1977). In addition, the cutoff score of 16 on CES-D for designation of presence of depressive symptoms shows high validity against depression as measured by the *DSM III* (Breslau, 1985).

In conclusion, our results point to a positive association between early life exposures to cigarette smoke through MSP and childhood SHS and depressive symptoms in adulthood. These associations may be largely mediated through adult active smoking. These findings merit further investigations in larger prospective studies, but together with prior research in the area suggest a possible role for targeting smoking prevention across the life course as a target for reducing the burden of depression.

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DECLARATION OF INTERESTS

None declared.

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