



Research report

Secondhand smoke exposure across the life course and the risk of adult-onset depression and anxiety disorder

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ABSTRACT

Introduction: The aim of this paper was to investigate the association between childhood and adulthood exposure to secondhand smoke (SHS) and depression, panic attack, and generalized anxiety disorder among adults in the United States over a 10-year period.

Methods: Data were drawn from the Midlife Development in the United States (MIDUS) Waves 1 and 2 ($N=2053$). Self-reported childhood and adulthood SHS exposure at Wave 1 (1994) was examined in relation to incident depression, panic attack, and generalized anxiety disorder 10 years later at Wave 2 (2005).

Results: Childhood SHS alone was not associated with mood and anxiety disorders in adulthood. Exposure to SHS in both childhood and adulthood was associated with increased depression and panic attack in adulthood. These associations did not appear to be due to confounding.

Limitations: SHS exposure was measured via self-report; biological data confirming exposure were not collected. More objective measures of SHS exposure are needed in future studies.

Conclusions: In summary, persistent exposure to SHS across the life course may be associated with increased risk of depression and panic attacks. Our results are consistent with prior findings and extend earlier results by showing a relationship between SHS exposure and mental health problems over time. Replication with biological measures of SHS over time is a necessary next step toward better understanding the pathways explaining these relationships.

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1. Introduction

Over the past decade, several states in the US have implemented a number of tobacco control policies in an effort to protect non-smokers from secondhand smoke (SHS¹) exposure (Callinan et al., 2010). Although regulations typically extend to a variety of enclosed and open public places including restaurants, bars, workplaces, and even to university campuses, policies that regulate smoking behavior in private residences are rare and difficult to enforce. Some research has shown that states that have enacted strict smoking bans may actually stimulate similar clean-air environments in homes and in vehicles (Naiman et al., 2011), particularly when children are present (Zhang et al., 2012). Despite that, smoking in homes leaves non-smokers unprotected from SHS exposure (Shields, 2007) and evidence suggests that Hispanic, African American, and non-Hispanic white households with higher levels of adult smoking, older children, and lower levels of education may be

disproportionately affected (Mills et al., 2011). Moreover, young people now have the highest levels of exposure to SHS as smoking continues to be concentrated in the home (Hawkins et al., 2012; Shields, 2007). Approximately 35% of all children in the US live in a household with one or more regular smokers (King et al., 2009; Schuster et al., 2002) (≥ 1 d/wk). More than four million youth ages 12–17 in the US lives with at least one adult who smokes in the home.

Although evidence of an association between SHS exposure and various physical health risks (e.g., asthma, cancer, and cardiovascular disease) is well documented, (Faught et al., 2009; Winickoff et al., 2010) new lines of research are investigating the link between SHS exposure and mental health (Bandiera et al., 2010, 2011; Hamer et al., 2010). Psychiatric disorders, particularly depression and anxiety disorders, have tremendous public health implications. Major depression is a leading burden of disease worldwide and will be the second leading cause of disability by 2020 (Murray and Lopez, 1996). Anxiety disorders are the most common mental health problems affecting the population (Kessler et al., 2005) and are associated with substantial morbidity, distress, work loss, and psychiatric, substance use and physical comorbidity (World Health Organization, 2010). While both genetic and environmental risk factors have been identified for

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depression and anxiety disorders, the etiology of these disorders still remains poorly understood.

One recent study has found strong associations between SHS exposure and increased depression among adults (Bandiera et al., 2010). These associations persist longitudinally and after adjusting for a range of confounding demographic and social factors. Despite the apparent public health implications of smoking and SHS exposure on adults, research in the past 25 years has attended to the critical acute and long-term ramifications of smoking and SHS exposure on children (Weitzman and Gittelman, 2011). Since several psychiatric disorders have an onset during childhood, (Merikangas et al., 2010a; Patel et al., 2007; Weitzman and Gittelman, 2011) it is crucial to evaluate the relationship between SHS exposure and mental health, particularly because of the high levels of SHS exposure among youth.

Two studies have examined the impact of SHS exposure on the risk of conduct disorder (CD) in youth (Braun et al., 2008; Fergusson et al., 1993). Both studies, one of which used parent-reported exposure and the other serum cotinine, found an association between SHS exposure and increased risk of CD in children. Another study reported a link between biologically confirmed SHS exposure and symptoms of major depressive disorder (MDD), generalized anxiety disorder (GAD), attention-deficit hyperactivity disorder (ADHD), and CD among children and adolescents (Bandiera et al., 2011). The association remained significant even after controlling for several demographic factors including race/ethnicity, gender, age, and physical health factors (e.g., migraine, asthma, hay fever, and maternal smoking during pregnancy). While several studies have adjusted for a variety of demographic, social, and physical health factors, no study to our knowledge has controlled for physical abuse or drug or alcohol use problems. Further, no study to date has separated out the relations between SHS exposure in childhood vs. adulthood, nor examined the dose–response relationship between exposure to SHS in childhood and adulthood on depression and anxiety disorders in adulthood over a 10-year span.

Thus, the current study will attempt to address some of the limitations of past research by employing a longitudinal design spanning over 10 years to examine the relationship between childhood and adulthood SHS exposure and the risk of incident depression and anxiety disorders in adulthood among a nationally representative sample of adults in the United States. The study will also examine the potential role of common causal risk factors including child abuse, smoking, and drug and alcohol abuse in these relationships.

2. Methods

2.1. Sample

Data were drawn from the two waves of the Midlife Development in the United States Survey (MIDUS) (Brim et al., 2010). The MacArthur Midlife Research Network collected Wave 1 data from 1994 to 1995 and Wave 2 data from 2004 to 2006; a national survey of Americans in adulthood that investigated behavioral, psychological, and social factors related to physical and mental health. Wave 1 consisted of a nationally representative multistage probability sample (main sample) of community-dwelling English speakers in the continental United States ($n=3032$). Participants who completed the telephone interview were mailed a self-administered questionnaire. The response rate from the mailed questionnaire was 86.6%, yielding an overall response rate of 61% ($0.70 \times 0.87 = 0.61$). Approximately 70% of Wave 1 participants took part in the Wave 2 survey collected by the Institute on Aging at the University of Wisconsin–Madison and supported by the National

Institute on Aging (2004–2006). Wave 2 participants completed a 30-min telephone interview and a self-administered questionnaire was mailed to them. Of the 3032 participants from Wave 1, 2101 completed the Wave 2 telephone surveys (response rate of 69.5%). For this study, we analyzed only data from those who participated in the Wave 1 main sample who completed both the phone and mail-in surveys, participated in the Wave 2 survey, and had complete information for Wave 2 outcome variables.

2.2. Measures

2.2.1. Secondhand smoke exposure

For childhood SHS exposure, at Wave 1, all participants were asked, “When you were growing up, that is during your first 16 years, did you live with anyone in your household who smoked cigarettes or other tobacco products (other than yourself)?” Possible responses included *no*, *father*, *mother*, and *someone else*. Those who responded affirmatively to living with anyone who smoked in the household were considered exposed to SHS as a child. For adulthood SHS exposure, at Wave 1, all participants were asked, “At the current time, does anyone regularly smoke cigarettes or other tobacco products INSIDE your home [other than yourself]?” Those who responded in the affirmative were considered to have SHS exposure in adulthood.

2.2.2. Cigarette smoking

At each Wave (1 and 2), all participants were asked if they had ever smoked a cigarette. Those who responded affirmatively were asked whether they currently “smoked regularly—that is at least a few cigarettes a day.” Participants who responded affirmatively were included as those with a history of daily smoking.

2.2.3. Depression and anxiety disorders

The MIDUS psychiatric diagnoses were based on the Composite International Diagnostic Interview Short Form scales, a series of diagnostic-specific scales that were developed from item level analyses of the Composite International Diagnostic Interview questions in the National Comorbidity Survey (Costa and McCrae, 1988; Kessler et al., 1994). The Composite International Diagnostic Interview Short Form scales were designed to reproduce the full Composite International Diagnoses as exactly as possible, with only a small subset of the original questions. Composite International Diagnostic Interview Short Form diagnoses at 12 months included in the MIDUS were MDD, panic attacks (PA), and GAD. These measures were used in Wave 1 past-12 month disorders.

2.2.4. Child maltreatment

Physical abuse categories were modeled after the Conflict Tactics Scale (Straus, 1979) using 15 different item measures from the MIDUS self-administered questionnaire. Respondents were asked how frequently their mother or father smashed or kicked something in anger; pushed, grabbed, or shoved them; slapped them; threw something at them; kicked, bit, or hit them with a fist; hit or tried to hit them with something; beat them up; choked them; burned or scalded them. Since physical response categories ranged in frequency from *never* to *often* for each (*mother and father*), respondents who reported experiencing abuse as *sometimes* or *often* were coded as 1 and those who reported *never* or *rare* were coded as 0.

2.2.5. Statistical analyses

Data were managed and analyzed with SPSS version 21.0 (IBM Corp., 2012). Descriptive statistics for the variables of interest as well as for potential confounders were generated. We used cross-tabulation to

determine their distribution according to SHS category (e.g., No SHS, Child SHS Only, Adult SHS Only, and Child and Adult SHS). Multinomial logistic regression analyses were then used to calculate odds ratios (with 95% confidence intervals) estimating the associations between childhood SHS as the predictor variable and odds of lifetime MDD, GAD, and PA as the outcome variables. The same procedure was repeated in a separate model to determine the strength of the association between adult SHS and odds of lifetime MDD, GAD, and PA. Finally, another model was generated to assess the strength of the association between SHS exposure in both childhood and in adulthood and odds of lifetime MDD, GAD, and PA. All analyses were adjusted for demographics—including age, gender, education level, and total household income from wages, pensions, social security, and government assistance—lifetime daily smoking, physical abuse, and substance use in Wave 1.

3. Results

3.1. Demographic characteristics associated with SHS exposure

Prior to conducting the analyses, descriptive statistics were obtained and characteristics were broken down by level of SHS exposure (see Table 1). Participants in the entire sample ranged in age from 20 to 74 years, with a mean age of 43.46 years (SD=12.29) and mean reported household income of \$69,037.02 (SD=\$59696.87). A majority of the sample was comprised of Caucasian respondents (80.3%), 6.8% were Black/African American, 0.6% were Native American/Alaskan, 0.8% were Asian, 1.6% were Native Hawaiian/Pacific, and 0.6% classified themselves as Other; 49.3% were male and 50.8% were female; 68.2% were married, 2.4% were divorced, 15.0% were never married, 6.1% were widowed, and 8.3% were separated; 14.0% reported having at least some grade school education up to GED, 31.8% were high school graduates,

27.7% had some college education, 15.6% reported having a bachelor's degree, and 11.0% reported having some graduate school training.

3.2. Childhood SHS exposure and depression/anxiety disorders in adulthood

The associations between childhood SHS only and MDD, GAD, and PA were not statistically significant (see Table 2).

3.3. Adult SHS exposure and depression/anxiety disorders in adulthood

Exposure to SHS during adulthood only was associated with significantly increased odds of PA but this association was no longer statistically significant after adjusting for demographics, lifetime daily smoking, or substance use disorder at Wave 1 (see Table 3). The associations between adult SHS and MDD and GAD were not statistically significant.

3.4. Exposure to SHS in childhood and adulthood and depression/anxiety disorders in adulthood

Exposure to SHS during both childhood and adulthood was associated with increased odds of MDD and PA. With the exception of PA after adjusting for lifetime daily smoking, these associations were not due to confounding variables. The link between GAD and exposure to SHS during both childhood and adulthood was not, however, statistically significant (see Table 4).

Table 1
Demographics associated with child and adult SHS exposure.

Demographic characteristics	No SHS exposure n=240	Child SHS only n=541	Adult SHS only n=282	Child + adult SHS n=990
*Age (M, SD)	41.05 (11.8)	40.98 (11.7)	46.14 (13.1)	44.64 (12.2)
*Gender (N, %)				
Male	96 (40.0%)	231 (42.7%)	151 (53.5%)	534 (53.9%)
Female	144 (60.0%)	310 (57.3%)	131 (46.5%)	457 (46.1%)
Race (N, %)				
White	193 (80.4%)	431 (79.7%)	220(78.0%)	805 (81.3%)
Black/African American	16 (6.7%)	34 (6.3%)	29 (10.3%)	59 (6.0%)
Native American/Alaskan	1 (0.4%)	7 (1.3%)	0 (0%)	5 (0.5%)
Asian	5 (2.1%)	7 (1.3%)	2 (0.7%)	3 (0.3%)
Native Hawaiian/Pacific Islander	4 (1.7%)	5 (0.9%)	8 (2.8%)	17 (1.7%)
Other	2 (0.8%)	4 (0.7%)	0 (0%)	7 (0.7%)
Marital status (N, %)				
Married	166 (69.2%)	365 (67.5%)	211 (74.8%)	658 (66.5%)
Separated	0 (0%)	19 (3.5%)	7 (2.5%)	24 (2.4%)
Divorced	38 (15.8%)	83 (15.3%)	24 (8.5%)	162 (16.4%)
Widowed	16 (6.7%)	24 (4.4%)	24 (8.5%)	61 (6.2%)
Never married	19 (7.9%)	51 (9.4%)	17 (6.0%)	84 (8.5%)
*Education (N, %)				
Grade school up to GED	14 (5.8%)	48 (8.9%)	34 (12.1%)	191 (19.3%)
High school graduate	54 (22.5%)	139 (25.7%)	107 (37.9%)	353 (35.7%)
Some college	59 (24.6%)	167 (30.9%)	75 (26.6%)	267 (27.0%)
Bachelors degree	62 (25.8%)	104 (19.2%)	44 (15.6%)	111 (11.2%)
Any graduate school	51 (21.3%)	83 (15.3%)	23 (8.2%)	68 (10.8%)
*Annual household income (M, SD)	83,158 (68,043)	80,741 (66,530)	60,312 (49,491)	62,137 (54,878)
*Lifetime daily smoking (N, %)	81 (33.8%)	234 (43.3%)	153 (54.3%)	698 (70.5%)
*Any physical abuse (N, %)	45 (18.8%)	103 (19.0%)	71 (25.2%)	310 (31.3%)
*Substance use disorder, W1 (N, %)	2 (0.8%)	11 (2.0%)	3 (1.1%)	42 (4.2%)
*MDD (N, %)	42 (17.5%)	93 (17.2%)	49 (17.4%)	273 (27.6%)
*GAD (N, %)	13 (5.4%)	18 (3.3%)	12 (4.3%)	79 (8.0%)
*PA (N, %)	20 (8.3%)	41 (7.6%)	38 (13.5%)	155 (15.7%)

* p < 0.05.

Table 2
Exposure to SHS in childhood only and depression/anxiety disorders in adulthood.

	No SHS exposure <i>n</i> =240	Child SHS only <i>n</i> =541	OR (95% CI)	AOR ¹ (95% CI)	AOR ² (95% CI)	AOR ³ (95% CI)	AOR ⁴ (95% CI)
MDD	42 (17.5%)	93 (17.2%)	0.9 (0.7, 1.5)	1.3 (0.8, 2.2)	0.8 (0.5, 1.3)	1.0 (0.6, 1.6)	0.9 (0.6, 1.4)
GAD	13 (5.4%)	18 (3.3%)	0.6 (0.3, 1.3)	0.8 (0.3, 2.3)	0.4 (0.2, 0.9)	0.8 (0.3, 2.0)	0.8 (0.3, 1.7)
PA	20 (8.3%)	41 (7.6%)	0.9 (0.5, 1.6)	0.9 (0.5, 1.8)	0.5 (0.3, 0.9)	0.9 (0.5, 1.6)	0.8 (0.4, 1.4)

AOR¹ = Adjusted for age, sex, education level, and income.

AOR² = Adjusted for lifetime daily smoking.

AOR³ = Adjusted for any physical abuse.

AOR⁴ = Adjusted for any substance use disorder, Wave 1.

Table 3
Exposure to SHS in adulthood only and depression/anxiety disorders in adulthood.

	No SHS exposure <i>n</i> =240	Adult SHS only <i>n</i> =282	OR (95% CI)	AOR ¹ (95% CI)	AOR ² (95% CI)	AOR ³ (95% CI)	AOR ⁴ (95% CI)
MDD	42 (17.5%)	49 (17.4%)	1.0 (0.6, 1.6)	1.6 (0.8, 2.9)	0.8 (0.4, 1.3)	1.1 (0.7, 1.9)	1.1 (0.7, 1.7)
GAD	13 (5.4%)	12 (4.3%)	0.8 (0.3, 1.7)	1.1 (0.3, 3.5)	0.3 (0.1, 0.8)	1.2 (0.5, 3.2)	1.0 (0.5, 2.5)
PA	20 (8.3%)	38 (13.5%)	1.7 (1.0, 3.0)	1.5 (0.7, 3.2)	1.0 (0.5, 1.9)	1.9 (1.0, 3.6)	1.7 (0.9, 2.9)

AOR¹ = Adjusted for age, sex, education level, and income.

AOR² = Adjusted for lifetime daily smoking.

AOR³ = Adjusted for any physical abuse.

AOR⁴ = Adjusted for any substance use disorder, Wave 1.

Bold=*p* < 0.05.

Table 4
Exposure to SHS in childhood and adulthood and mental disorders in adulthood.

	No SHS exposure <i>n</i> =240	Child & Adult SHS exposure <i>n</i> =990	OR (95% CI)	AOR ¹ (95% CI)	AOR ² (95% CI)	AOR ³ (95% CI)	AOR ⁴ (95% CI)
MDD	42 (17.5%)	273 (27.6%)	1.8 (1.3, 2.6)	2.2 (1.4, 3.7)	1.5 (1.0, 2.3)	1.7 (1.1, 2.5)	1.8 (1.2, 2.6)
GAD	13 (5.4%)	79 (8.0%)	1.5 (0.8, 2.8)	2.1 (0.8, 5.1)	0.8 (0.4, 1.5)	1.7 (0.8, 3.8)	1.7 (0.9, 3.4)
PA	20 (8.3%)	155 (15.6%)	2.0 (1.3, 3.3)	1.8 (1.0, 3.3)	1.1 (0.6, 1.9)	1.9 (1.1, 3.3)	1.8 (1.1, 2.9)

AOR¹ = Adjusted for age, sex, education level, and income.

AOR² = Adjusted for lifetime daily smoking.

AOR³ = Adjusted for any physical abuse.

AOR⁴ = Adjusted for any substance use disorder, Wave 1.

Bold=*p* < 0.05.

4. Discussion

In spite of the decrease in SHS exposure in the United States over the past 20 years (Centers for Disease Control and Prevention, 2011; Centers for Disease Control and Prevention National Center for Chronic Disease Prevention and Health Promotion Office on Smoking and Health, 2012), due in large part to the implementation of smoking bans and the decline in youth and adult smoking, an estimated 88 million nonsmokers had measurable levels of cotinine in their systems in 2007–2008 according to the Centers for Disease Control and Prevention (Office on Smoking and Health, 2013). Despite smoke-free zones on university campuses, work places, restaurants, and other public environments, cigarette smoking in private residences, cars, and in other enclosed spaces continues to expose children and non-smoking adults to SHS (Centers for Disease Control and Prevention Coordinating Center for Health Promotion National Center for Chronic Disease Prevention and Health Promotion Office on Smoking and Health, 2006; Warren et al., 2006). Thus, our finding that individuals who are exposed to SHS in both childhood and in adulthood are at greater risk of developing MDD and PA has potential public health implications.

The association between SHS and MDD, in particular, was not explained by demographics, substance use, physical abuse, or smoking, consistent with the findings of Bandiera et al. (2010). The relationship between SHS and PA, however, may be partially explained by one's own smoking. This result is not surprising given that several epidemiological studies have documented prospective evidence that cigarette smoking increases the risk of

panic disorder (Breslau et al., 2004; Merikangas et al., 2010b; Moylan et al., 2012). Our findings also support an increased risk of PA among individuals that have been exposed to SHS exclusively in adulthood, however this association seems to be due to demographics, daily smoking, and/or substance use. Thus, it appears from these data that the greatest risk of developing MDD and/or PA is associated with SHS exposure in both childhood and in adulthood and not in one or the other in isolation, suggesting a potential dose–response relationship.

Although these data cannot provide evidence of a causal link between SHS exposure and PA and MDD, several possible mechanisms may account for our findings. Increased SHS exposure in childhood and in adulthood may be indicative of overall lower socioeconomic status, as SHS exposure tends to be higher for individuals living below the poverty level, (Office on Smoking and Health, 2013) and financial disparity is a known risk factor of depression and anxiety (Almeida et al., 2012; Murali and Oyeboode, 2004). However our findings suggest that the relationship between exposure to persistent SHS and PA/MDD is not explained by household income. Another possible explanation is that individuals exposed to higher levels of SHS are typically in environments with higher levels of stress. Previous studies suggest that stress is strongly linked to smoking, (Kassel et al., 2003; Tsourtos et al., 2008) as well as to depressive and anxiety symptoms (Hicks et al., 2009; Kessing et al., 2003; Nugent et al., 2011). Thus, the SHS–MDD–PA link may be a result of personal vulnerability to the effects of stress and exposure to stressful environments (Patten, 2013).

A number of limitations should be considered when interpreting our results. First, we relied on participants' retrospective reports of childhood and adulthood SHS exposure. Unfortunately, biological data confirming SHS exposure, such as serum cotinine levels, were not collected. Employing objective measures of SHS exposure in future research is certainly warranted. Second, childhood SHS exposure was assessed by asking participants whether or not they lived with a smoker, which is a relatively indirect indicator of SHS exposure. Thus, the current sample may have inadvertently included participants who lived with a regular smoker but were not regularly exposed to SHS in the home. However, previous research suggests that if a child lives with a regular smoker, one of the most dominant sites of SHS exposure is indeed the home (Klepeis, 1999; Mannino et al., 2001). Third, there are a number of potential confounders, such as childhood socioeconomic status, parental depression, and family functioning, that we were unable to control, which have been associated with SHS exposure (Galobardes et al., 2004; Mannino et al., 2001) as well as with depression (Gilman et al., 2002; Maughan et al., 2013; Raudino et al., 2013) and anxiety (Gallo and Matthews, 2003; Hamer et al., 2010; Letourneau et al., 2013). Future studies are needed to examine these variables.

In summary, although the extant literature on the association between SHS and mental health outcomes is somewhat mixed (Bandiera et al., 2010, 2011; Bot et al., 2013; Lam et al., 2013), our data reveal findings consistent with positive associations between SHS exposure and increased risk of MDD and PA. While the mechanisms underlying such associations are not yet known, expanding this area of research is imperative in informing and driving the development of policies that can potentially prevent and/or ban SHS in a variety of settings that are currently outside of the purview of present smoking laws.

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Conflict of interest

The authors have no conflicts of interest.

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