ARTICLE

Secondhand Smoke Exposure and Mental Health Among Children and Adolescents

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Objective: To examine a potential association between biologically confirmed secondhand smoke exposure and symptoms of *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*) major depressive disorder, generalized anxiety disorder, panic disorder, attention-deficit/hyperactivity disorder, and conduct disorder using a nationally representative sample of US children and adolescents.

Design: Nationally representative cross-sectional survey of the United States.

Setting: Continental United States.

Participants: Children and adolescents aged 8 to 15 years who participated in the National Health and Nutrition Examination Survey from 2001 to 2004.

Intervention: Measurement of serum cotinine level to assess secondhand smoke exposure among nonsmokers.

Main Outcome Measures: The *DSM-IV* symptoms were derived from selected modules of the National Institute of Mental Health's Diagnostic Interview Schedule for Children Version IV, a structured diagnostic interview administered by trained lay interviewers.

Results: Among nonsmokers, serum cotinine level was positively associated with symptoms of *DSM-IV* major depressive disorder, generalized anxiety disorder, attention-deficit/hyperactivity disorder, and conduct disorder after adjusting for survey design, age, sex, race/ethnicity, poverty, migraine, asthma, hay fever, maternal smoking during pregnancy, and allostatic load. Associations with serum cotinine level were more apparent for boys and for participants of non-Hispanic white race/ethnicity.

Conclusions: Our results are consistent with a growing body of research documenting an association between secondhand smoke exposure and mental health outcomes. Future research is warranted to establish the biological or psychological mechanisms of association.

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THE US SURGEON GENERAL HAS concluded that there is no risk-free level of secondhand smoke (SHS) exposure and estimated that approximately 66% of children aged 3 to 11 years are exposed to SHS.[1] It is well established that SHS exposure causes adverse physical health conditions (eg, respiratory and cardiovascular),[2-4] and there is increasing evidence suggesting that it may also adversely affect mental health. Cross-sectional studies[5,6] show a positive association between SHS exposure and anxiety or depression among adults, and results of a 2010 prospective analysis of a large cohort of adults conducted over more than 6 years suggest that SHS exposure may predict the onset of poor mental health.[6] The effects of SHS exposure on the mental health of children and adolescents are still unclear.

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Because many mental disorders have an onset in youth at a time when SHS exposure is high, it is critical to consider how SHS may be affecting the mental health of children and adolescents so that appropriate preventive measures can be implemented. Despite evidence of an association, the mechanism by which SHS exposure may promote or exacerbate poor mental health is unclear. Secondhand smoke may be a proxy for stressful living conditions, and stress has been associated with poor mental health.[7,8] In response to stress, the hypothalamic-pituitary-adrenal axis and immune, metabolic, autonomic, and cardiovascular systems respond to keep the environment of the body in homeostasis.[9] This balance can be measured by examining allostatic load, which represents the wear and tear of the body's response to prolonged psychological stress[9] and is associated with the onset of physical and mental conditions.[9] Although chronic physical conditions usually manifest in adult-

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hood, there is evidence that prolonged exposure to stress may have an effect on the response of the body to stress and result in poor health even among children.[10-12] Other hypotheses suggest a link between smoking and poor mental health through nicotine and dopamine pathways.[13-15] Smokers who have susceptibility genes to low intrasynaptic dopamine levels have greater smoking-induced dopamine release,[13] which has been associated with higher risk for mental disorders.[16] Secondhand smoke may also affect respiratory conditions, such as asthma,[17] which has been positively associated with mental disorders.[18] Because youth exposure to SHS may come from the mother, another important confounder to consider is maternal smoking during pregnancy, which has been associated with greater risk for mental disorders.[19]

Given the potential mechanisms by which SHS exposure could promote or exacerbate mental health in children and adolescents, it is imperative to conduct further research investigating this association. To date, no studies have examined the effects of SHS exposure on mental health among children and adolescents. Furthermore, 2 previous studies[5,6] in adults were limited because they considered neither a wide range of *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*) symptoms nor how exposure to SHS might differentially affect mental health across at-risk subgroups. It is well established that tobacco use prevalence, patterns, and long-term outcomes differ across sex and race/ethnicity strata,[20] with non-Hispanic blacks carrying a disproportionate burden of tobacco-related morbidity and mortality.[21] In addition, male sex and non-Hispanic black race/ethnicity are associated with slower nicotine metabolism and with higher cotinine cutoff points to differentiate SHS exposure from personal smoking behaviors.[22] Relative to male sex, female sex is associated with a different biological response to stress through the protective effects of estrogen.[9] Given these variations, it is essential to consider how the association between SHS exposure and mental health may differ across subgroups.

This study builds on previous literature to examine the association between biologically confirmed SHS exposure and symptoms of *DSM-IV* major depressive disorder (MDD), generalized anxiety disorder (GAD), panic disorder, attention-deficit/hyperactivity disorder (ADHD), and conduct disorder using a nationally representative sample of US children and adolescents. Analyses are performed among the total population and across sex and race/ethnicity strata to assess differential patterns in the effects of SHS exposure on mental health. It was hypothesized that current SHS exposure is positively associated with *DSM-IV* symptoms and that this association varies across subgroups of sex and race/ethnicity even after adjusting for potential confounders, such as maternal smoking during pregnancy and allostatic load.

METHODS

PARTICIPANTS

Data for this study are from the 2001 to 2004 National Health and Nutrition Examination Survey (NHANES), a nationally representative probability sample of noninstitutionalized US civilians. The response rates for the present study ranged from 79.2% to 92.3% (http://www.cdc.gov/nchs/nhanes/response_rates_cps.htm) depending on the disorder and the source of information (eg, examination vs household interview). The sample for this study included 2901 children and adolescents aged 8 to 15 years. A serum cotinine level of 3 μg/L or higher was used as a cutoff point to distinguish smokers (n = 141) from current nonsmokers (n = 2901) (to convert cotinine level to nanomoles per liter, multiply by 5.675).[22] In analyses stratified by sex, this cutoff point was used for both male and female participants, while cutoff points of 2.77, 2.95, and 1.18 μg/L were used for non-Hispanic blacks, non-Hispanic whites, and Mexican Americans, respectively.[22]

MEASURES

SHS Exposure

Secondhand smoke exposure was measured by level of serum cotinine, a metabolite of nicotine. The serum cotinine level was log transformed, which is the standard method used previously.[5]

DSM-IV Symptoms

Information on mental disorders was derived from the National Institute of Mental Health's Diagnostic Interview Schedule for Children Version IV (DISC-IV), a structured questionnaire administered by lay interviewers to ascertain 12-month diagnostic criteria for *DSM-IV* conditions in children and adolescents.[23,24] Symptoms of *DSM-IV* MDD (child report score range, 0-21), GAD (child report score range, 0-12), panic disorder (child report score range, 0-5), ADHD (parent report score range, 0-23), and conduct disorder (parent report score range, 0-12) were included in the DISC-IV. Furthermore, diagnoses of *DSM-IV* MDD, GAD, panic disorder, ADHD, and conduct disorder were included.

Covariates

Age, sex, and race/ethnicity were self-reported. Poverty was measured by the poverty income ratio, which is the ratio of a family's income to the poverty threshold as determined by federal guidelines. Parent report of migraine, asthma, hay fever, and maternal smoking during pregnancy were considered. An allostatic load index was computed as performed previously in the NHANES.[25] Contingent on data availability, the allostatic load index was composed of C-reactive protein level, systolic blood pressure, diastolic blood pressure, total cholesterol level, and homocysteine level. Based on the distribution of these variables, a cutoff point at the 75th percentile was used for each variable to equal 1; values below the 75th percentile were coded as 0. These values were summed to create the allostatic load index (range, 0-5).[25]

STATISTICAL ANALYSIS

Multiple regression models were used with biologically confirmed SHS exposure to predict *DSM-IV* symptoms among nonsmokers. Three models examined the effects of covariates on the association between SHS exposure and *DSM-IV* symptoms. Model 1 only adjusted for survey design; model 2 further adjusted for age, sex, race/ethnicity, poverty, migraine, asthma, hay fever, and maternal smoking during pregnancy; and model 3 further adjusted for these same variables and allostatic load. To account for the complex survey design of the NHANES, analyses were conducted using statistical software (SAS, version 9.2; SAS Institute, Cary, North Carolina).

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Table 1. Secondhand Smoke Exposure Variables and *DSM-IV* Symptoms Among 2901 Children and Adolescents in the 2001 to 2004 NHANES[a]

Variable	No.[b]	Value
Laboratory Levels, Mean (SE)		
Serum cotinine, μg/L	2637	−2.56 (0.10)
Allostatic load	2578	1.31 (0.03)
C-reactive protein, mg/L	2665	0.12 (0.01)
Total cholesterol, mg/dL	2638	164.33 (0.95)
Blood pressure, mm Hg		
Diastolic	2836	56.82 (0.42)
Systolic	2836	104.26 (0.37)
Homocysteine, μg/L	2681	5.31 (0.03)
Covariates, % (SE)		
Asthma		
Yes	481	15.06 (1.05)
No	2415	84.93 (1.05)
Hay fever		
Yes	351	15.57 (1.22)
No	2537	84.42 (1.22)
Migraine		
Yes	581	18.27 (0.86)
No	2320	81.72 (0.86)
Maternal smoking during pregnancy		
Yes	400	17.08 (1.17)
No	2469	82.91 (1.17)
***DSM-IV* Symptoms, Mean (SE)**		
Major depressive disorder	2901	4.93 (0.22)
Generalized anxiety disorder	2901	2.86 (0.11)
Panic disorder	2901	0.29 (0.02)
Attention-deficit/hyperactivity disorder	2901	3.94 (0.12)
Conduct disorder	2901	1.34 (0.07)
Characteristics, % (SE)		
Age, y		
8-11	1114	48.79 (1.31)
12-15	1787	51.20 (1.31)
Sex		
Male	1412	51.06 (1.01)
Female	1489	48.93 (1.01)
Race/ethnicity		
Non-Hispanic white	829	61.90 (2.57)
Non-Hispanic black	970	14.76 (1.71)
Mexican American	896	12.23 (1.55)
Other	206	11.10 (1.66)
Poverty income ratio		
<1, poor	784	18.30 (1.24)
1-2	766	22.46 (1.03)
>2	1242	56.51 (1.35)
Unknown	109	2.71 (0.49)

Abbreviations: *DSM-IV*, *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition); NHANES, National Health and Nutrition Examination Survey.

SI conversion factors: To convert cotinine level to nanomoles per liter, multiply by 5.675; C-reactive protein level to nanomoles per liter, multiply by 9.524; cholesterol level to millimoles per liter, multiply by 0.0259; and homocysteine level to micromoles per liter, multiply by 7.397.

[a] The percentages do not equal the numerator divided by the denominator because the size of the subsample is not weighted while the percentages are weighted.

[b] Totals vary because of missing data.

RESULTS

CHARACTERISTICS OF THE ELIGIBLE SAMPLE

The total size of the eligible sample was 2901. Sample characteristics are given in **Table 1**. Approximately 51% were male and 49% female. Most of the sample were non-Hispanic white (61.9%), followed by non-Hispanic black (14.8%), Mexican American (12.2%), and other races/ethnicities (11.1%). Respondents reported the highest mean number of *DSM-IV* symptoms for MDD (4.93), followed by ADHD (3.94), GAD (2.86), conduct disorder (1.34), and panic disorder (0.29).

ASSOCIATIONS BETWEEN SHS EXPOSURE AND *DSM-IV* SYMPTOMS

Associations between biologically confirmed SHS exposure and *DSM-IV* symptoms are given in **Table 2**. In all models, serum cotinine level was positively associated with symptoms of MDD, GAD, ADHD, and conduct disorder, while it was unassociated with symptoms of panic disorder. After adjusting for all covariates (model 3), serum cotinine level was most strongly associated with ADHD symptoms (b=.40), such that a 1-U increase in serum cotinine level (log transformed) translates into a 0.40 increase in ADHD symptoms. There was a slightly smaller, although still significant, association of serum cotinine level with symptoms of MDD (b=.22), conduct disorder (b=.18), and GAD (b=.16).

Stratified analyses showed that the association between serum cotinine level and *DSM-IV* symptoms differed across sex. Among male participants, adjusted regression analyses showed a statistically significant association between serum cotinine level and symptoms of MDD (b=.28), GAD (b=.17), ADHD (b=.38), and conduct disorder (b=.31) (**Table 3**). Among female participants, serum cotinine levels remained significantly associated only with symptoms of GAD (b=.17) and ADHD (b=.43).

Stratification by race/ethnicity showed marked differences in the association between serum cotinine level and *DSM-IV* symptoms across racial/ethnic subpopulations. Among non-Hispanic whites, there was a statistically significant association between serum cotinine level and symptoms of MDD (b=.34), GAD (b=.26), and ADHD (b=.51) (**Table 4**). Serum cotinine level was not statistically significant associated with any *DSM-IV* symptoms among non-Hispanic blacks and was associated only with conduct disorder (b=.15) among Mexican Americans.

ASSOCIATIONS BETWEEN SHS EXPOSURE AND DIAGNOSIS OF *DSM-IV* CONDITIONS

Owing to few positive cases, associations between serum cotinine level and diagnoses of *DSM-IV* MDD (n=15), GAD (n=9), and panic disorder (n=11) were not tested. There were sufficient positive cases for ADHD (n=201) and conduct disorder (n=54), although no attempt was made to conduct analyses stratified by sex or by race/ethnicity. In a model only adjusting for survey design, serum cotinine level was positively associated with ADHD (odds ratio, 1.15; 95% confidence interval, 1.03-1.27). However, when further controlling for maternal smoking during pregnancy, serum cotinine level was no longer associated with ADHD (odds ratio, 1.03; 95% confidence interval, 0.92-1.16), while maternal smoking during pregnancy was positively associated with ADHD (odds ratio, 2.62; 95% confidence interval, 1.58-4.33). However, in analyses stratified by ADHD diagnosis, serum co-

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Table 2. Associations Between Secondhand Smoke Exposure and *DSM-IV* Symptoms Among Children and Adolescents in the 2001 to 2004 NHANES

	Major Depressive Disorder			Generalized Anxiety Disorder			Panic Disorder			Attention-Deficit/ Hyperactivity Disorder			Conduct Disorder		
Model[a]	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value
1 (n=2637)	.28	.06	<.001	.16	.05	<.01	.01	.01	.10	.58	.07	<.001	.28	.04	<.001
2 (n=2594)	.21	.08	.01	.16	.06	.01	.01	.01	.38	.39	.08	<.001	.17	.06	<.01
3 (n=2524)	.22	.08	.01	.16	.06	.01	.01	.01	.37	.40	.08	<.001	.18	.06	<.01

Abbreviations: *DSM-IV*, *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition); NHANES, National Health and Nutrition Examination Survey.
[a]Model 1 was adjusted only for survey design. Model 2 was adjusted for survey design, age, sex, race/ethnicity, poverty, migraine, asthma, hay fever, and maternal smoking during pregnancy. Model 3 was adjusted for survey design, age, sex, race/ethnicity, poverty, migraine, asthma, hay fever, maternal smoking during pregnancy, and allostatic load.

Table 3. Sex-Specific Associations Between Secondhand Smoke Exposure and *DSM-IV* Symptoms Among Children and Adolescents in the 2001 to 2004 NHANES[a]

	Major Depressive Disorder			Generalized Anxiety Disorder			Panic Disorder			Attention-Deficit/ Hyperactivity Disorder			Conduct Disorder		
Variable	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value
Male participants (n=1238)	.28	.12	.03	.17	.07	.02	.00	.01	.55	.38	.17	.03	.31	.08	<.01
Female participants (n=1286)	.17	.09	.08	.17	.07	.03	.01	.01	.22	.43	.09	<.001	.05	.05	.30

Abbreviations: *DSM-IV*, *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition); NHANES, National Health and Nutrition Examination Survey.
[a]Sex-stratified models were adjusted for survey design, age, race/ethnicity, poverty, migraine, asthma, hay fever, maternal smoking during pregnancy, and allostatic load.

Table 4. Race/Ethnicity–Specific Associations Between Secondhand Smoke Exposure and *DSM-IV* Symptoms Among Children and Adolescents in the 2001 to 2004 NHANES[a]

	Major Depressive Disorder			Generalized Anxiety Disorder			Panic Disorder			Attention-Deficit/ Hyperactivity Disorder			Conduct Disorder		
Variable	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value	b Level	SE	*P* Value
Non-Hispanic white (n=712)	.34	.11	<.01	.26	.07	<.001	.01	.01	.21	.51	.11	<.001	.13	.07	.07
Non-Hispanic black (n=828)	–.02	.12	.84	.01	.05	.85	.00	.01	.88	.17	.16	.29	.09	.08	.26
Mexican American (n=783)	.00	.19	.96	–.06	.12	.60	.02	.01	.21	.16	.14	.27	.15	.04	<.001

Abbreviations: *DSM-IV*, *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition); NHANES, National Health and Nutrition Examination Survey.
[a]Race/ethnicity–specific models were adjusted for survey design, age, sex, poverty, migraine, asthma, hay fever, maternal smoking during pregnancy, and allostatic load.

tinine level was positively associated with ADHD symptoms among participants without an ADHD diagnosis (b=.46, $P<.001$), while serum cotinine level was not associated with ADHD symptoms among participants with an ADHD diagnosis (b=.33, $P=.11$) after controlling for survey design and maternal smoking during pregnancy. Because a previous study[26] among the same data set correlated SHS exposure with conduct disorder, we did not perform analyses using this variable.

COMMENT

To our knowledge, this is the first study to assess the association between biologically confirmed SHS exposure and mental disorder symptoms in a nationally representative sample of US children and adolescents. Secondhand smoke exposure was positively associated with symptoms of *DSM-IV* MDD, GAD, ADHD, and conduct disorder, but not panic disorder, among the total sample of nonsmokers even after adjusting for age, sex, race/ethnicity, poverty, migraine, asthma, hay fever, maternal smoking during pregnancy, and allostatic load. However, this association differed across sex and race/ethnicity subpopulations, with the most apparent associations noted for male sex and non-Hispanic white race/ethnicity. Serum cotinine level was also positively associated with *DSM-IV* ADHD diagnosis; however, the association between serum cotinine level and ADHD diagnosis was attributed to maternal smoking during pregnancy. Serum cotinine level was associated with ADHD symptoms only among participants without an ADHD diagnosis.

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Both SHS exposure and poor mental health are major public health problems among children and adolescents. Exposure to tobacco smoke among young children has been associated with several short-term and long-term health effects, including sudden infant death syndrome,[27] respiratory complications,[28-30] dental decay,[31] metabolic syndrome,[32] otitis media,[33] and asthma,[33-35] among others. Furthermore, 2 longitudinal studies[36,37] showed that maternal smoking is associated with increased risk of behavioral problems even after adjusting for confounding factors. The findings presented herein provide additional evidence on the harmful effects of SHS exposure on children and adolescents. Our results are consistent with data from previous cross-sectional[5,6] and prospective[6] studies in adults and suggest that exposure to SHS may precipitate the onset of or exacerbate mental disorder symptoms. Data herein further suggest that this association may differ across sex and race/ethnicity subgroups.

There are known variations in the prevalence, patterns of use, and outcomes of smoking across sex and race/ethnicity strata.[38] Individual differences by sex and race/ethnicity in the associations between SHS exposure and *DSM-IV* symptoms may be explained by variations in cotinine metabolism[39,40] or by other biological, social, or environmental factors that vary across sex and race/ethnicity strata and were unaccounted for in the models herein. However, our results also suggest that male sex and non-Hispanic white race/ethnicity may somehow confer greater vulnerability to mental health effects of SHS exposure. Future research is needed to clarify the biological or psychological mechanisms of associations between SHS exposure and mental health, as well as potential reasons for differential associations across sex and race/ethnicity strata.

In the case of ADHD diagnosis, our study findings differ from those of a 2010 study by Xu et al.[41] They also used the 2001 to 2004 NHANES and found that serum cotinine level was positively associated with ADHD diagnosis among Mexican American children but was not associated with ADHD diagnosis among non-Hispanic white and black children even after adjusting for maternal smoking during pregnancy. The study by Xu et al had methodological limitations; ADHD was measured using a single-item question that asked parents whether a health care professional had ever diagnosed their child as having ADHD, and they did not use a cotinine cutoff point to measure SHS exposure. The differences in results between the 2 studies may be related to these measurement variations. Furthermore, current cigarette smokers were more likely to be retained in the analyses by Xu et al. Our findings suggest that the association between SHS exposure and ADHD diagnosis is attributable to in utero tobacco exposure. Recent results from a large longitudinal study by Ekblad et al[42] suggest that in utero tobacco exposure may lead to adverse psychiatric conditions. A recent study by Cho et al[43] among children in 5 Korean cities also found a positive association between SHS exposure and ADHD symptoms. Therefore, the association between SHS exposure and ADHD symptoms may only be significant at the subthreshold level vs at the diagnostic level.

Several limitations to our study must be considered when interpreting the results. These include the cross-sectional design, our inability to control for some potential confounders, and the possibility that intermittent smokers were included in the analyses. There is no established cutoff point for nonsmokers among persons younger than 12 years,[22] and intermittent smokers of any age could have been retained in our analysis if they did not smoke in the 24 to 36 hours before their NHANES blood draw. Some of these participants would not have had detectable serum cotinine levels at the time of their NHANES participation because of the short half-life of this measure.[44] Furthermore, we did not control for alcohol consumption because data on alcohol consumption in participants younger than 12 years were not collected in the NHANES. Another potential confounder that we were unable to control for in our analysis is maternal psychiatric history.[45-47] That is, children with depressed mothers are more likely to have poor mental health.[45-47] In addition, the source of SHS exposure is unknown (eg, mother, father, or other), and illicit drug use was not considered a covariate. Finally, our models included adjustment for allostatic load as a proxy control for psychological stress, which has been shown to be associated with risk of psychiatric symptoms.[7,8] However, controlling for allostatic load did not weaken associations between serum cotinine level and DISC-IV symptoms, so it remains unclear if this measure appropriately controlled for psychological stress.

Despite these limitations, findings in the present study provide critical and much-needed data on associations of biologically confirmed SHS exposure with *DSM-IV* symptoms in a nationally representative sample of US children and adolescents. Our results have important public health implications. Only 26 states in the United States have banned smoking in all public places (such as bars and restaurants), despite evidence that comprehensive public smoking bans lead to reduced incidence of cardiovascular and respiratory conditions.[48] Similar improvements in population-level mental health may be possible.[49] Efforts to ban smoking in public places where children and adolescents are present, including all child care settings and schools, should continue, as well as increased efforts to develop interventions targeted directly at parents and designed to prevent SHS exposure in the homes of children and adolescents.[50,51] Given the critical developmental period of childhood and adolescence, the effects of policy to reduce or ban smoking in public places and in the home may help prevent or reduce the progression of illness in at-risk individuals and alleviate the heavy burden of illness attributable not only to tobacco use but also to mental disorders.

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REFERENCES

1. US Department of Health and Human Services. Children and secondhand smoke exposure. http://www.surgeongeneral.gov/library/smokeexposure/report/fullreport.pdf. Accessed August 4, 2010.
2. Glantz SA, Parmley WW. Passive smoking and heart disease: mechanisms and risk. *JAMA*. 1995;273(13):1047-1053.
3. Lai HK, Ho SY, Wang MP, Lam TH. Secondhand smoke and respiratory symptoms among adolescent current smokers. *Pediatrics*. 2009;124(5):1306-1310.
4. Wang C, Salam MT, Islam T, Wenten M, Gauderman WJ, Gilliland FD. Effects of in utero and childhood tobacco smoke exposure and β2-adrenergic receptor genotype on childhood asthma and wheezing. *Pediatrics*. 2008;122(1):e107-e114. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2748980/?tool=pubmed. Accessed January 24, 2011.
5. Bandiera FC, Arheart KL, Caban-Martinez AJ, et al. Secondhand smoke exposure and depressive symptoms. *Psychosom Med*. 2010;72(1):68-72.
6. Hamer M, Stamatakis E, Batty GD. Objectively assessed secondhand smoke exposure and mental health in adults: cross-sectional and prospective evidence from the Scottish Health Survey. *Arch Gen Psychiatry*. 2010;67(8):850-855.
7. Monroe SM, Harkness K, Simons AD, Thase ME. Life stress and the symptoms of major depression. *J Nerv Ment Dis*. 2001;189(3):168-175.
8. Monroe SM, Simons AD. Diathesis-stress theories in the context of life stress research: implications for the depressive disorders. *Psychol Bull*. 1991;110(3):406-425.
9. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med*. 1998;338(3):171-179.
10. Evans GW. A multimethodological analysis of cumulative risk and allostatic load among rural children. *Dev Psychol*. 2003;39(5):924-933.
11. Evans GW, Kim P, Ting AH, Tesher HB, Shannis D. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Dev Psychol*. 2007;43(2):341-351.
12. Johnston-Brooks CH, Lewis MA, Evans GW, Whalen CK. Chronic stress and illness in children: the role of allostatic load. *Psychosom Med*. 1998;60(5):597-603.
13. Brody AL, Mandelkern MA, Olmstead RE, et al. Gene variants of brain dopamine pathways and smoking-induced dopamine release in the ventral caudate/nucleus accumbens. *Arch Gen Psychiatry*. 2006;63(7):808-816.
14. Caskey NH, Jarvik ME, Wirshing WC. The effects of dopaminergic D_2 stimulation and blockade on smoking behavior. *Exp Clin Psychopharmacol*. 1999;7(1):72-78.
15. Caskey NH, Jarvik ME, Wirshing WC, et al. Modulating tobacco smoking rates by dopaminergic stimulation and blockade. *Nicotine Tob Res*. 2002;4(3):259-266.
16. Brody AL, Olmstead RE, Abrams AL, et al. Effect of a history of major depressive disorder on smoking-induced dopamine release. *Biol Psychiatry*. 2009;66(9):898-901.
17. Siersted HC, Boldsen J, Hansen HS, Mostgaard G, Hyldebrandt N. Population based study of risk factors for underdiagnosis of asthma in adolescence: Odense schoolchild study. *BMJ*. 1998;316(7132):651-656.
18. Goodwin RD, Jacobi F, Thefeld W. Mental disorders and asthma in the community. *Arch Gen Psychiatry*. 2003;60(11):1125-1130.
19. Froehlich TE, Lanphear BP, Auinger P, et al. Association of tobacco and lead exposures with attention-deficit/hyperactivity disorder. *Pediatrics*. 2009;124(6):e1054-e1063. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2853804/?tool=pubmed. Accessed January 25, 2010.
20. US Department of Health and Human Services. *Tobacco Use Among U.S. Racial/Ethnic Minority Groups: African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, Hispanics: A Report of the Surgeon General*. Atlanta, GA: Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention; 1998.
21. Office of the Surgeon General. *The Health Consequences of Smoking: A Report of the Surgeon General*. Atlanta, GA: US Dept of Health and Human Services; May 27, 2004.
22. Benowitz NL, Bernert JT, Caraballo RS, Holiday DB, Wang J. Optimal serum cotinine levels for distinguishing cigarette smokers and nonsmokers within different racial/ethnic groups in the United States between 1999 and 2004. *Am J Epidemiol*. 2009;169(2):236-248.
23. Shaffer D, Fisher P, Dulcan MK, et al. The NIMH Diagnostic Interview Schedule for Children Version 2.3 (DISC-2.3): description, acceptability, prevalence rates, and performance in the MECA Study. Methods for the Epidemiology of Child and Adolescent Mental Disorders Study. *J Am Acad Child Adolesc Psychiatry*. 1996;35(7):865-877.
24. Shaffer D, Fisher P, Lucas CP, Dulcan MK, Schwab-Stone ME. NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): description, differences from previous versions, and reliability of some common diagnoses. *J Am Acad Child Adolesc Psychiatry*. 2000;39(1):28-38.
25. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health*. 2006;96(5):826-833.
26. Braun JM, Froehlich TE, Daniels JL, et al. Association of environmental toxicants and conduct disorder in U.S. children: NHANES 2001-2004. *Environ Health Perspect*. 2008;116(7):956-962.
27. Anderson HR, Cook DG. Passive smoking and sudden infant death syndrome: review of the epidemiological evidence. *Thorax*. 1997;52(11):1003-1009.
28. Anderson LJ, Parker RA, Strikas RA, et al. Day-care center attendance and hospitalization for lower respiratory tract illness. *Pediatrics*. 1988;82(3):300-308.
29. Chen Y, Li W, Yu S. Influence of passive smoking on admissions for respiratory illness in early childhood. *Br Med J (Clin Res Ed)*. 1986;293(6542):303-306.
30. Pattenden S, Antova T, Neuberger M, et al. Parental smoking and children's respiratory health: independent effects of prenatal and postnatal exposure. *Tob Control*. 2006;15(4):294-301.
31. Aligne CA, Moss ME, Auinger P, Weitzman M. Association of pediatric dental caries with passive smoking. *JAMA*. 2003;289(10):1258-1264.
32. Weitzman M, Cook S, Auinger P, et al. Tobacco smoke exposure is associated with the metabolic syndrome in adolescents. *Circulation*. 2005;112(6):862-869.
33. Strachan DP, Cook DG. Health effects of passive smoking. 6. Parental smoking and childhood asthma: longitudinal and case-control studies. *Thorax*. 1998;53(3):204-212.
34. Cook DG, Strachan DP. Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children. *Thorax*. 1997;52(12):1081-1094.

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35. DiFranza JR, Lew RA. Morbidity and mortality in children associated with the use of tobacco products by other people. *Pediatrics*. 1996;97(4):560-568.
36. Fergusson DM, Horwood LJ, Lynskey MT. Maternal smoking before and after pregnancy: effects on behavioral outcomes in middle childhood. *Pediatrics*. 1993; 92(6):815-822.
37. Weitzman M, Gortmaker S, Sobol A. Maternal smoking and behavior problems of children. *Pediatrics*. 1992;90(3):342-349.
38. Johnston LD, O'Malley P, Bachman JG, Schulenberg JE. *Monitoring the Future: National Results on Adolescent Drug Use: Overview of Key Findings, 2009*. Bethesda, MD: National Institute on Drug Abuse; 2010. NIH publication 10-7583.
39. Moolchan ET, Franken FH, Jaszyna-Gasior M. Adolescent nicotine metabolism: ethnoracial differences among dependent smokers. *Ethn Dis*. 2006;16(1):239-243.
40. Benowitz NL, Lessov-Schlaggar CN, Swan GE, Jacob P III. Female sex and oral contraceptive use accelerate nicotine metabolism. *Clin Pharmacol Ther*. 2006; 79(5):480-488.
41. Xu X, Cook RL, Ilacqua VA, Kan H, Talbott EO. Racial differences in the effects of postnatal environmental tobacco smoke on neurodevelopment. *Pediatrics*. 2010; 126(4):705-711.
42. Ekblad M, Gissler M, Lehtonen L, Korkeila J. Prenatal smoking exposure and the risk of psychiatric morbidity into young adulthood. *Arch Gen Psychiatry*. 2010; 67(8):841-849.
43. Cho SC, Kim BN, Hong YC, et al. Effect of environmental exposure to lead and tobacco smoke on inattentive and hyperactive symptoms and neurocognitive performance in children. *J Child Psychol Psychiatry*. 2010;51(9):1050-1057.
44. Benowitz NL. Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiol Rev*. 1996;18(2):188-204.
45. Foster CE, Webster MC, Weissman MM, et al. Remission of maternal depression: relations to family functioning and youth internalizing and externalizing symptoms. *J Clin Child Adolesc Psychol*. 2008;37(4):714-724.
46. Talati A, Wickramaratne PJ, Pilowsky DJ, et al. Remission of maternal depression and child symptoms among single mothers: a STAR*D-Child report [published correction appears in *Soc Psychiatry Psychiatr Epidemiol*. 2007;42(12):971]. *Soc Psychiatry Psychiatr Epidemiol*. 2007;42(12):962-971.
47. Weissman MM, Pilowsky DJ, Wickramaratne PJ, et al; STAR*D-Child Team. Remissions in maternal depression and child psychopathology: a STAR*D-child report [published correction appears in *JAMA*. 2006;296(10):1234]. *JAMA*. 2006;295(12):1389-1398.
48. Herman PM, Walsh ME. Hospital admissions for acute myocardial infarction, angina, stroke, and asthma after implementation of Arizona's comprehensive statewide smoking ban [published online ahead of print May 13, 2010]. *Am J Public Health*. 2011;101(3):491-496. doi:10.2105/AJPH.2009.179572.
49. Bandiera FC, Caban-Martinez AJ, Arheart KL, et al. Secondhand smoke policy and the risk of depression. *Ann Behav Med*. 2010;39(2):198-203.
50. Liles S, Hovell MF, Matt GE, Zakarian JM, Jones JA. Parent quit attempts after counseling to reduce children's secondhand smoke exposure and promote cessation: main and moderating relationships. *Nicotine Tob Res*. 2009;11(12): 1395-1406.
51. Hovell MF, Zakarian JM, Matt GE, et al. Counseling to reduce children's secondhand smoke exposure and help parents quit smoking: a controlled trial. *Nicotine Tob Res*. 2009;11(12):1383-1394.

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