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Objectively Measured Secondhand Smoke Exposure and Mental Health in Children

Evidence From the Scottish Health Survey

Mark Hamer, PhD; Tamsin Ford, PhD; Emmanuel Stamatakis, PhD; Samantha Dockray, PhD; G. David Batty, PhD

Objective: To examine the association between objectively assessed secondhand smoke (SHS) exposure and mental health in a representative sample of British children.

Design: Cross-sectional study.

Setting: Community-based population sample from the 2003 Scottish Health Survey.

Participants: Nine hundred one nonsmoking children (mean [SD] age, 8.3 [2.5] years).

Main Exposure: Exposure to SHS was determined from salivary cotinine level and self-report.

Main Outcome Measure: Psychological distress assessed using the Strengths and Difficulties Questionnaire (SDQ).

Results: Forty percent of the sample demonstrated high SHS exposure (cotinine level >0.70 ng/mL). Children with higher cotinine levels were more likely to live in areas of greater socioeconomic deprivation. Participants in the highest cotinine quartile (>0.70 ng/mL) had significantly higher total SDQ scores compared with those in the lowest quartile (age- and sex-adjusted mean difference = 2.8; 95% confidence interval, 1.6 to 3.9). There was evidence of a dose-response effect across the cotinine group (*P* trend = .001). Of the SDQ subscales, the strongest associations with cotinine levels emerged for hyperactivity and conduct disorder. These associations remained statistically significant after adjustment for possible confounders including social deprivation, single-parent status, body mass index, chronic illness, and physical activity.

Conclusion: Objectively assessed SHS exposure was associated with poorer mental health among children.

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Author Affiliations: Department of Epidemiology and Public Health, University College London, London (Drs Hamer, Stamatakis, Dockray, and Batty), and Institute of Health Services Research, Peninsula College of Medicine and Dentistry, Plymouth (Dr Ford), England; and Medical Research Council Social and Public Health Sciences Unit, Glasgow, and Centre for Cognitive Ageing and Cognitive Epidemiology, University of Edinburgh, Edinburgh, Scotland (Dr Batty).

APPROXIMATELY 2 MILLION children in Britain currently live in a household where they are exposed to secondhand smoke (SHS)[1,2] and approximately 66% of children aged 3 to 11 years are exposed to SHS in the United States.[3] In children, exposure to SHS is related to elevated rates of mortality and respiratory illnesses, such as lower respiratory tract infections, wheezing, middle ear infection, and asthma.[1,4] However, to date, less attention has been paid to the impact, if any, of SHS exposure on their mental health. Mental illness is increasingly being recognized as the most significant health concern for children and adolescents in developed countries, with an estimated prevalence of 8% to 23% of the child and adolescent population in European countries.[5]

There are good reasons to anticipate an influence of SHS exposure on mental health in children. First, in animal studies, tobacco smoke can induce negative mood and a depression-like state manifested in decreased sensitivity to natural reward and enhanced sensitivity to stress and anxiety-eliciting situations later in life.[6] Second, in humans, data from the United States have shown that prenatal and postnatal tobacco exposure are associated with higher risks of conduct disorders.[7] Nevertheless, in another study, high cotinine levels were not associated with depression in nonsmoking adolescents, but there was an inverse association between cotinine level and depression among the smokers.[8] Given the paucity of evidence in this area, it is difficult to draw any firm conclusions from the existing data. The aim of the present study was therefore to examine the association between objectively assessed SHS exposure and mental health in a representative sample of British children.

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Table 1. Association Between Salivary Cotinine Level and Other Characteristics in 901 Children

Characteristic	Cotinine Category, ng/mL ≤0.05 (n=101)	0.06-0.30 (n=280)	0.31-0.70 (n=159)	0.71-14.99 (n=361)	*P* Trend
Age, y, mean (SD)	8.4 (2.5)	8.5 (2.5)	8.2 (2.5)	8.2 (2.4)	.61
Boys, %	52.5	46.1	48.4	49.3	.71
Most deprived, %	5.0	7.5	8.8	28.5	<.001
Sports and active play, frequency/wk, mean (SD)	7.4 (3.7)	7.7 (3.5)	7.9 (3.4)	7.8 (3.7)	.75
Single-parent status, %	17.8	16.4	10.1	32.4	<.001
Chronic condition, %	21.8	17.5	22.0	21.6	.54
Asthma	5.9	7.1	6.9	9.7	.47
Body mass index, mean (SD)[a]	17.9 (2.9)	18.2 (5.3)	17.8 (2.9)	18.5 (3.3)	.23

[a]Calculated as weight in kilograms divided by height in meters squared.

METHODS

STUDY DESIGN AND PARTICIPANTS

The present study used data from the 2003 Scottish Health Survey that consisted of a nationally representative household-based sample of the general population.[9] Seventy-seven percent of eligible households with children took part in the survey. The parents of the children (aged 4-12 years; mean [SD], 8.3 [2.5] years) gave full informed consent to participate in the study and ethical approval was obtained from the London Research Ethics Council. Trained interviewers visited households and interviewing was conducted using computer-assisted personal interviewing.

MENTAL HEALTH

Psychological distress was assessed using the parental version of the Strengths and Difficulties Questionnaire (SDQ), which has demonstrated good reliability and validity.[10,11] The main outcome was total difficulties score (ranging from 0-40), with a higher score representing worse mental health. This was calculated by adding the scores from the SDQ subscales of hyperactivity, emotional symptoms, conduct problems, and peer problems.

ASSESSMENT OF SHS EXPOSURE

In all children, objective exposure to SHS was assessed using salivary cotinine level, which is a reliable and valid circulating biochemical marker of nicotine exposure.[12,13] A dental roll saturated with the child's saliva was placed in a tube and later analyzed with a rapid liquid chromatography technique using a Hewlett Packard (Palo Alto, California) hp5890 gas chromatograph. The technique had a coefficient of variation less than 7%. All valid cotinine values were obtained from saliva samples with sufficient volume to undertake the assay and with no evidence of contamination. Data on self-reported smoking and exposure to SHS were only collected from children 8 years and older since self-reported measures are considered unreliable in younger children. None of these children reported smoking in the previous week. Questions about SHS exposure first inquired about whether the children felt that they were exposed to SHS and if so, where (at home, other people's homes, or public transportation). Children reporting nonsmoking status but with a salivary cotinine level more than 15 ng/mL were removed from all analyses (<1% of the sample) because such values may be indicative of recent smoking.

COVARIATE DATA

Interviewers measured height and weight of the children for the calculation of body mass index. Parents reported the weekly frequency of sports and active play (for at least 15 minutes) of their children and any chronic health conditions. Demographic data were also recorded including single-parent status and questions on deprivation. We used The Scottish Index of Multiple Deprivation, which was assessed from 31 indicators in 6 individual domains including parents' employment, current income, housing, health, education, and skills and training.[9]

STATISTICAL ANALYSIS

Based on previously used cutoffs,[14,15] salivary cotinine level was categorized into quartiles: low SHS exposure (salivary cotinine level ≤0.05 ng/mL or below the detectable limit [the referent group]), low-moderate SHS exposure (0.06-0.30 ng/mL), moderate SHS exposure (0.31-0.70 ng/mL), and high SHS exposure (0.71-14.99 ng/mL). The SDQ score was normally distributed and we therefore used general linear models to examine associations with cotinine categories. The models were adjusted for age, sex, quintile of deprivation index, single-parent status, body mass index, chronic illness, and physical activity (number of weekly episodes of sports and active play lasting at least 15 minutes). A dose-response association was examined using a test for linear trend across groups. There were no clear differences in our results between boys and girls, so the data were pooled and adjusted by sex. All analyses were conducted using SPSS (version 14; SPSS Inc, Chicago, Illinois).

RESULTS

The initial sample with available cotinine data consisted of 1015 children, although after excluding those with missing SDQ scores (n=34) and further demographic information (n=80), the analytical sample was based on 901 children (mean [SD] age, 8.3 [2.5] years). The excluded children did not differ in age (8.4 vs 8.3 years) compared with those included, although fewer excluded children were from the least deprived quintile (12.3% vs 25.0%; *P*=.007). The mean value for salivary cotinine level was 1.29 ng/mL (95% confidence interval [CI], 1.16 to 1.42).

The relation between children's salivary cotinine levels and their other characteristics in shown in **Table 1**.

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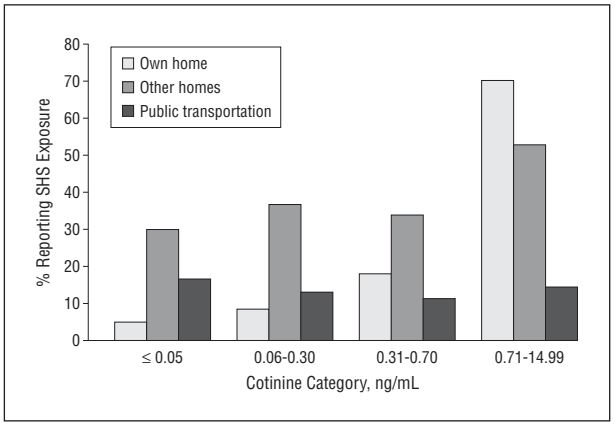

Figure. The association between objectively assessed and self-reported exposure to secondhand smoke (SHS) in various places among children aged 8 to 12 years (n=524).

Children with higher cotinine levels were more likely to live in areas of greater socioeconomic deprivation, be resident in a single-parent household, and report asthma. Among the older children who provided information on self-reported SHS exposure (n=524), cotinine values were most strongly related to exposure at home (**Figure**), suggesting that exposure to parental smoke is the most important contributor to SHS exposure in children.

Using existing bandings,[10] an abnormally high SDQ total difficulties score (≥20) was found in 3.0% and 2.7% of children aged 4 to 8 years and 9 to 12 years, respectively. The proportion of children with borderline scores (SDQ score of 16-19) was 5.0% and 6.6% in 4- to 8-year olds and 9- to 12-year olds, respectively. These proportions are largely comparable with the normative data for British children.[16] Independent predictors of SDQ total difficulties score included sex (girls had lower SDQ scores than boys, regression coefficient $\beta = -0.78$ SDQ units; 95% CI, –1.33 to –0.22 SDQ units), age (12-year-olds had lower scores than 4-year-olds, $\beta = -1.61$; 95% CI, –2.70 to –0.52), socioeconomic deprivation (more highly deprived children had higher scores, $\beta = 3.2$; 95% CI, 2.0 to 4.4), single-parent status (children of single-parent families had higher scores, $\beta = 1.7$; 95% CI, 0.9 to 2.5), medical conditions (children with existing medical conditions had higher scores, $\beta = 1.8$; 95% CI, 1.0 to 2.7), and physical activity (least physically active tertile had higher scores, $\beta = 1.2$; 95% CI, 0.4 to 2.1).

The relation between cotinine values and SDQ scores is shown in **Table 2**. Participants in the highest cotinine quartile (>0.70 ng/mL) had significantly higher total SDQ scores compared with those in the lowest quartile (age- and sex-adjusted mean difference=2.8; 95% CI, 1.6 to 3.9) and there was evidence of a dose-response effect across the cotinine group (*P* trend=.001). After possible confounding factors were added to the multivariable model, there was some evidence of attenuation (mainly through socioeconomic factors) but the SHS exposure–SDQ association remained statistically significant (Table 2). Of the SDQ subscales, the strongest associations with cotinine levels emerged for hyperactivity and conduct disorder; again, there was some evidence of attenuation of these gradients in the multiply adjusted models.

In the subsample of older children (8- to 12-year-olds) with available data on self-reported SHS exposure, there was an association between self-reported exposure in the home and SDQ score that was similar in magnitude to that observed in the highest cotinine exposure category (**Table 3**).

COMMENT

Results from this representative sample of British children suggest an association between increased SHS exposure and worse mental health, as reflected by higher scores on the SDQ questionnaire. Although smoke-free legislation has contributed to a successful secular decline in SHS exposure among children, the evidence suggests that those children living with smokers and in disadvantaged households continue to experience substantial contact with SHS.[1] Our data (collected before smoke-free legislation) confirm that objectively assessed SHS exposure is most closely associated with reported exposure in the home, which is consistent with previous reports.[17] Indeed, self-reported exposure to SHS in the home was also robustly associated with SDQ scores in the older children. Previous reports have suggested that the accuracy of parental and youth reporting of SHS exposure might vary considerably according to the reporting source and age and sex of youth respondent.[18,19]

The financial burden of passive smoking in children is estimated to be at least £9.7 million (US $15.2 million) each year in UK primary care visits and asthma costs and £13.6 million (US $21.4 million) in UK hospital admissions.[1] These figures are based entirely on physical illness since, as described, the effects of objective SHS exposure on mental health have not been well examined. Previous studies have produced conflicting evidence. Data from the National Health and Nutrition Examination Survey have shown that prenatal and postnatal tobacco exposure are associated with higher risks of conduct disorders.[7] Nevertheless, in another study, high cotinine levels were not associated with depression in nonsmoking adolescents, but there was an inverse association between cotinine level and depression among the smokers.[8] In a small cohort of adolescents, active smoking was associated with a higher risk of depressive episodes over 5 years' follow-up, which was partly explained by dysfunction of the hypothalamic-pituitary-adrenal axis.[20] Other mechanisms might involve the dopaminergic system. Smokers who are genetically predisposed to a low resting intrasynaptic dopamine level have heightened smoking-induced dopamine release,[21] which has been associated with greater depression and anxiety[22] that are often comorbid with conduct disorder in childhood.[23] Thus, this genetic predisposition may also operate in relation to SHS exposure, especially in children of smoking parents who might be more likely to possess this genetic vulnerability. Since it has been reported that more than 80% of children are aware that passive smoking is harmful,[1] an involuntary exposure to a known harmful effect might also adversely affect mental health. We did not obtain data on prenatal smoking. However, since smoking is a lifelong habit often taken up in adoles-

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Table 2. Association Between Salivary Cotinine Level and SDQ Score in 901 Children

Cotinine Group	SDQ Score, Mean (SD)	β (95% CI)		
		Age-Adjusted	Model 1[a]	Model 2[b]
Total score				
≤0.05 ng/mL	6.4 (4.7)	1 [Reference]	1 [Reference]	1 [Reference]
0.06-0.30 ng/mL	6.5 (4.7)	0.1 (−1.1 to 1.3)	0.2 (−1.0 to 1.3)	0.2 (−1.0 to 1.3)
0.31-0.70 ng/mL	7.2 (5.5)	0.8 (−0.6 to 2.1)	0.6 (−0.7 to 1.8)	0.6 (−0.7 to 1.8)
>0.70 ng/mL	9.2 (5.6)	2.8 (1.6 to 3.9)	1.6 (0.4 to 2.8)	1.6 (0.5 to 2.8)
P trend		.001	.003	.003
Emotional symptoms				
<0.05 ng/mL	1.4 (1.6)	1 [Reference]	1 [Reference]	1 [Reference]
0.06-0.30 ng/mL	1.5 (1.7)	0.1 (−0.3 to 0.5)	0.03 (−0.4 to 0.4)	0.1 (−0.3 to 0.4)
0.31-0.70 ng/mL	1.9 (1.8)	0.5 (0.03 to 0.9)	0.4 (0.01 to 0.9)	0.5 (0.01 to 0.9)
>0.70 ng/mL	1.8 (1.9)	0.4 (−0.01 to 0.8)	0.1 (−0.3 to 0.6)	0.1 (−0.3 to 0.6)
P trend		.02	.11	.10
Hyperactivity				
<0.05 ng/mL	2.8 (2.3)	1 [Reference]	1 [Reference]	1 [Reference]
0.06-0.30 ng/mL	2.8 (2.4)	−0.1 (−0.6 to 0.5)	−0.1 (−0.6 to 0.5)	−0.01 (−0.5 to 0.5)
0.31-0.70 ng/mL	2.9 (2.4)	−0.01 (−0.6 to 0.6)	−0.03 (−0.6 to 0.6)	−0.01 (−0.6 to 0.6)
>0.70 ng/mL	3.9 (2.4)	1.0 (0.5 to 1.6)	0.7 (0.2 to 1.30)	0.8 (0.2 to 1.30)
P trend		.001	.001	.001
Conduct disorder				
<0.05 ng/mL	1.2 (1.3)	1 [Reference]	1 [Reference]	1 [Reference]
0.06-0.30 ng/mL	1.2 (1.3)	0.01 (−0.3 to 0.3)	−0.03 (−0.4 to 0.3)	−0.01 (−0.3 to 0.3)
0.31-0.70 ng/mL	1.3 (1.6)	0.1 (−0.2 to 0.5)	0.04 (−0.3 to 0.4)	0.05 (−0.3 to 0.4)
>0.70 ng/mL	1.9 (1.7)	0.7 (0.4 to 1.0)	0.4 (0.02 to 0.7)	0.4 (0.03 to 0.7)
P trend		.001	.01	.01
Peer problems				
<0.05 ng/mL	1.0 (1.6)	1 [Reference]	1 [Reference]	1 [Reference]
0.06-0.30 ng/mL	1.2 (1.5)	0.1 (−0.2 to 0.5)	0.1 (−0.3 to 0.4)	0.1 (−0.2 to 0.5)
0.31-0.70 ng/mL	1.2 (1.5)	0.1 (−0.3 to 0.5)	0.1 (−0.3 to 0.4)	0.1 (−0.3 to 0.5)
>0.70 ng/mL	1.7 (1.7)	0.7 (0.3 to 1.0)	0.3 (−0.03 to 0.7)	0.3 (−0.02 to 0.7)
P trend		.001	.14	.16

Abbreviations: CI, confidence interval; SDQ, Strengths and Difficulties Questionnaire.
[a] Model 1 contains adjustment for age, sex, deprivation index, and single-parent status.
[b] Model 2 has additional adjustment for body mass index, chronic conditions, and frequency of sports and active play.

Table 3. Association Between Self-reported SHS Exposure and Total SDQ Score in 524 Children Aged 8 to 12 Years

Exposure	Sample Size	SDQ Score, Mean (SD)	β (95% CI)		
			Age-Adjusted	Model 1[a]	Model 2[b]
Exposed in own home					
No	345	6.5 (4.7)	1 [Reference]	1 [Reference]	1 [Reference]
Yes	179	9.5 (5.6)	3.0 (2.06 to 4.0)	1.8 (0.7 to 2.8)	1.6 (0.6 to 2.6)
Exposed in other homes					
No	305	6.7 (4.4)	1 [Reference]	1 [Reference]	1 [Reference]
Yes	219	8.8 (5.1)	2.1 (1.2 to 3.1)	1.4 (0.5 to 2.4)	1.3 (0.3 to 2.2)
Exposed in public places					
No	452	7.6 (4.5)	1 [Reference]	1 [Reference]	1 [Reference]
Yes	72	7.3 (4.7)	−0.3 (−1.7 to 1.1)	−0.9 (−2.2 to 0.4)	−1.0 (−2.3 to 0.4)

Abbreviations: CI, confidence interval; SDQ, Strengths and Difficulties Questionnaire; SHS, secondhand smoke.
[a] Model 1 contains adjustment for age, sex, deprivation index, and single-parent status.
[b] Model 2 has additional adjustment for body mass index, chronic conditions, frequency of sports and active play, and mutual adjustment for SHS exposure in different places.

cence, it is possible that young children with smoking parents were also exposed to tobacco during the prenatal period. Low birth weight and decreased in utero brain growth are 2 of multiple potential etiologic pathways proposed as mediating the effects of prenatal tobacco smoke exposure on child neurodevelopment,[24] which is likely to influence mental health.

The strengths of the study include the sampling of a relatively large, representative general population-based group of children, the objective measurement of SHS exposure, and the well-characterized study members, which facilitates insights into the role of potential confounding factors. The limitations of the present study should also be recognized. Given the cross-sectional na-

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ture of this study, we cannot determine the causal nature of the association between SHS exposure and mental health, although it is unlikely that reverse causation is operating. The possibility remains that the results could be explained by residual confounding. We did, however, adjust our analyses for several important confounders, including a strong measure of deprivation that was assessed from 31 indicators in 6 individual domains. The data on mental health are based on parental reports; however, the SDQ is a highly validated instrument designed for this purpose and parental or teacher reports are more accurate in predicting psychiatric disorder than self-report.[25] Further, although self-report measures of internalizing emotional problems may be more valid than parent reports, the SDQ has been shown to have high concordance between child and parent for all scales.[26] Smokers are more likely to have common mental disorders, such as depression,[27] so this might have biased parental reports, although we did not have information about the mental health of the parents. The children excluded from our analyses were more likely to be from more deprived homes and thus probably exposed to greater SHS and more likely to have poorer mental health,[28] which may have led to an underestimation of the strength of the SHS exposure–mental health association herein. Lastly, although the SDQ reliably identifies children and adolescents with psychopathology, and increasing total difficulties scores are associated with an increasing likelihood of psychiatric disorder, it is a dimensional rather than a diagnostic measure.[29] Nevertheless, the SDQ has been shown to have clinical relevance, since in a previous study in the community, the questionnaire identified more than 70% of children with conduct, hyperactivity, depressive, and some anxiety disorders with a specificity of 94.6% and a sensitivity of 63.3%.[25]

In conclusion, we found an association between SHS exposure and poor mental health, as reflected by higher scores on the SDQ. This study provides additional reasons to continue to educate parents about the multiple health dangers of exposing their children to tobacco smoke, particularly within the home.

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Correspondence: Mark Hamer, PhD, Department of Epidemiology and Public Health, 1-19 Torrington Pl, University College London, London WC1E 6BT, England (m.hamer@ucl.ac.uk).
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Announcement

Trial Registration Required. In concert with the International Committee of Medical Journal Editors (ICMJE), *Archives of Pediatrics and Adolescent Medicine* will require, as a condition of consideration for publication, registration of all trials in a public trials registry (such as http://ClinicalTrials.gov). Trials must be registered at or before the onset of patient enrollment. This policy applies to any clinical trial starting enrollment after July 1, 2005. The trial registration number should be supplied at the time of submission.

For details about this new policy, and for information on how the ICMJE defines a clinical trial, see the editorials by DeAngelis et al in the September 8, 2004 (2004;292:1363-1364) and June 15, 2005 (2005;293:2927-2929) issues of *JAMA*. Also see the Instructions to Authors on our Web site: www.archpediatrics.com.

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