

Article

Factors associated with maternal depressive symptoms among low-income, African American smokers enrolled in a secondhand smoke reduction programme

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ABSTRACT

Introduction Maternal depressive symptoms increase the risk of poor maternal and child health outcomes, and are a primary barrier to health behaviour change. Social cognitive theory can guide our understanding of risk factors that may have an impact on maternal depressive symptoms. The aim of this paper was to understand the correlates of maternal depressive symptoms among low-income African American smokers completing a 16-week intervention trial to reduce young children's second-hand smoke exposure (SHSe).

Methods This study presents a secondary analysis of depression symptoms among 227 maternal smokers completing the SHSe-reduction trial. The end-of-treatment Center of Epidemiologic Studies Depression Scale (CES-D) score was used to assess depressive symptoms (dichotomised as 0 = score of < 16 and 1 = score of \geq 16). Multivariate logistic regression analysis was used to test the one-way hypothesis that odds of significant depressive symptoms would be associated with greater total number of household smokers, greater number of

paediatric sick visits, greater daily exposure of child to cigarette smoke by their mother, greater life-event stress, and lower social support, marital status, employment status and level of educational attainment.

Results Number of household smokers (OR = 1.57, $P = 0.049$), social support (OR = 0.88, $P < 0.001$) and life-event stress (OR = 1.04, $P = 0.001$) predicted significant maternal depressive symptoms; all other variables were not significant predictors in the model.

Conclusion Number of household smokers is a novel risk factor for understanding significant maternal depressive symptoms in the context of a childhood SHSe-reduction trial. Improving our understanding of the household-level social milieu in the context of SHSe-reduction interventions will assist in reducing the risk of maternal depressive symptoms.

Keywords: household smokers, maternal depressive symptoms, social cognitive theory

Introduction

Recent evidence suggests that the presence of household smokers may contribute to maternal depressive symptoms through both psychosocial interactions and physiological effects of second-hand smoke exposure (SHSe).¹ Non-smoking mothers have a higher risk of developing depressive symptoms if they live with one or more household smokers, and their risk of developing depressive symptoms increases with every additional smoker in the home.¹ Thus psychosocial risk factors, specifically household smokers, may play an important role in predicting depressive symptoms among maternal smokers who are attempting to reduce household SHSe in order to protect their children.

Studies designed to improve the understanding of factors associated with depressive symptoms in underserved maternal smokers are warranted, given that more severe depressive symptoms increase children's risk for morbidity and mortality, undermine maternal health in general, and may inhibit positive changes in other health behaviour change efforts.²⁻⁴ The presence of depressive symptoms increases the risk of compromised maternal and mental and physical health. This is a particular concern among low-income, urban, African American mothers, a population that is known to bear increased health risks. Many of these mothers live in poverty and are unmarried, unemployed, and more likely to experience depression than other socio-economic groups.⁵ Depression disproportionately affects women (almost twice as much as men),⁶ and African Americans are affected almost twice as much as Caucasians.⁷ As many as 50% of these high-risk women may experience clinically significant depressive symptoms. However, their symptoms are rarely diagnosed or treated properly.^{5,8,9} Therefore identifying maternal depressive symptoms and understanding risk factors that may predict presentation with depressive symptoms in this population remain a top public health priority.⁵

Women who have untreated depressive symptoms are more likely to turn to substance abuse, including nicotine dependence.¹⁰ Maternal depressive symptoms have also been linked to many poor physical and mental health outcomes in the children of these women,¹ which often continue to affect those children throughout the lifespan.^{3,11-18}

Recent studies indicated that depressive symptoms were present in 76% of female smokers compared with 56% of non-smoking women, which included a large proportion of African American women.¹⁹ Conversely, greater than 40% prevalence of smoking among African Americans was reported in those who had significant depressive symp-

toms.²⁰ As a result of the frequent co-occurrence of maternal depressive symptoms and smoking, many urban, low-income, African American children are also disproportionately exposed to second-hand smoke, primarily from maternal smoking.²¹ SHSe is a leading cause of morbidity and mortality in this population,²² and is causally linked to many illnesses.²³⁻³⁵ Consequently, SHSe-reduction intervention programmes for maternal smokers are also a high-priority public health goal.

Both maternal depressive symptoms and maternal SHSe are linked to child health outcomes, with evidence suggesting that maternal depressive symptoms are a barrier to health behaviour change.² Therefore an understanding of the risk factors for maternal depressive symptoms and maternal SHSe is paramount for creating efficacious SHSe-reduction interventions. The SHSe-intervention outcomes will be published elsewhere.³⁶

In the current study, we are interested in identifying risk factors associated with maternal depressive symptoms in a high-risk sample of mothers completing a child SHSe-reduction trial. We framed our understanding of potential factors using a modified model of Atkins' health behaviour intervention and depressive symptoms model,² which uses several constructs of social cognitive theory. By simplifying Atkins' model, we proposed a one-way model to test the individual contributing effects of factors that we considered to be particularly relevant to our target population. We hypothesised that perceived control (influenced by other smokers in the home) was a key factor. Other factors that we postulated would predict depressive symptoms included social support (measured by a social support questionnaire and marital status), self-efficacy (measured by number of paediatric sick visits and child's mean daily exposure to cigarette smoke by their mother), perceived stress and socio-economic status (measured by employment status and educational attainment).

Atkins' model of health promotion for depressed mothers proposes a contributory effect of health promotion activities on depressive symptoms.² In our study, all of the participants enrolled in a programme that aimed to reduce child SHSe and that had an interest in making their homes smoke-free. Participants were randomised to receive one of two SHSe-reduction interventions. One group received an enhanced standard of care that included detailed written health information about second-hand smoke and strategies to promote reduction of child SHSe and a smoke-free home. The other group received intensive behavioural counselling to promote the same outcomes. We shall be including randomisation status as a controlling variable to account for any potential differential effects of programme-related social support.

Social cognitive constructs, risk factors and depressive symptoms

Self-efficacy is a core construct of social cognitive theory (SCT), and refers to an individual's belief in their ability to act or behave in a manner that will produce the desired goals.³⁷ According to SCT, self-efficacy is influenced in four ways, namely gaining mastery over an experience, social modelling, social affirmation and managing reactions to stress.³⁷ In our study we did not measure self-efficacy directly, but our hypotheses represent variables which are thought to relate to self-efficacy. In addition to self-efficacy, social censure of a new behaviour may also present barriers to change, which may lead to depressive symptoms.³⁷ Household smokers may be the leading source of social censure, especially if the new behaviour involves changing the behaviour of all of the smokers in the home and not just that of the mother.

We also postulate that number of paediatric sick visits and total child SHSe by the mother are indirectly associated with depressive symptoms due to their influence on SHSe-reduction self-efficacy. Other studies have shown that child health problems due to SHSe, resulting in increased utilisation of paediatric healthcare services, may increase and exacerbate maternal depressive symptoms.^{38,39} We suspect that paediatric sick visits could contribute to a mother's perception of failure to gain mastery to protect her child from SHSe.

Perceived stress has been associated with depressive symptoms in high-risk African American mothers.⁴⁰⁻⁴³ If maternal smokers do not master the ability to manage physical and psychological reactions to stress, the resulting life stress may be overwhelming and mothers may experience hopelessness, leading to depressive symptoms.^{37,44}

Other social influences have an impact on maternal depression symptoms, the most obvious of these being social support. According to SCT, many health habits are entrenched in social structures.³⁷ Perceived social inefficacy, which may hamper maternal ability to develop supportive relationships that foster positive health behaviour change, may increase vulnerability to developing depressive symptoms through feelings of social isolation.^{2,37}

Socio-economic status and depressive symptoms

The socio-economic status factors of educational attainment and employment status are postulated to affect depressive symptoms by negatively influencing a mother's perceived self-efficacy.² For example, a mother who did not finish high school

may have low self-efficacy with regard to learning new skills that are required for a health behaviour change. As a result, the mother may develop depressive symptoms in anticipation of her failure to learn the new behaviour,⁴⁴ or self-blame for her failure to make a behaviour change.⁴⁵ Employment reduces the risk of depressive symptoms, especially in high-risk women.^{4,46-50} Employment is also strongly related to income, another socio-economic status factor that is linked to depressive symptoms in high-risk women similar to our population.^{8,46} According to SCT, educational attainment is also an important predictor of maternal depressive symptoms, as it influences one's self-efficacy with regard to learning new skills. In addition, there is ample empirical research showing a strong association between level of educational attainment and maternal depressive symptoms.^{51,52}

We postulate that number of household smokers, number of paediatric sick visits, child's average daily exposure to cigarette smoke by the mother, perceived stress, perceived social support, marital status, employment status and level of educational attainment predict depressive symptoms in high-risk, African American maternal smokers at the end of treatment.

Methods

Participants

Maternal smokers were recruited from Women and Infant Care (WIC) offices, paediatric primary care clinics, and newspaper and mass transit advertisements targeting underserved neighbourhoods in Philadelphia, PA. Participants were screened for eligibility if they expressed interest in participating in an intervention study aimed at reducing their child's SHSe. Respondents were eligible for inclusion if they were female, aged ≥ 18 years, smoked ≥ 5 cigarettes per day, and exposed their youngest child (aged < 4 years) to smoke from at least two cigarettes per day. Respondents were excluded if they were currently diagnosed with or in treatment for a DSM-IV-TR⁵³ psychiatric disorder, including major depressive disorder, or were currently pregnant. In total, 227 subjects completed the end-of-study Center of Epidemiologic Studies Depression Scale (CES-D). Three cases with missing data were excluded from the multivariate analysis. This study was approved by the Institutional Review Board at Temple University in Philadelphia, PA. Consent forms were signed by participants prior to collection of the study data.

Procedures and measures

Data for this secondary analysis were taken from the Philadelphia FRESH (Family Rules for Establishing Smoke-free Homes) study, a randomised controlled behavioural counselling trial aimed at reducing SHSe of the participant's youngest child. Interest in smoking cessation was not an inclusion criterion, but interest in reducing child SHSe and creating a smoke-free home was. Subjects recruited to the study were assessed at baseline for demographic variables, smoking history and psychosocial variables. They were then randomised to one of two groups, namely a standard of care control group (SCC) that received a comprehensive self-help manual for second-hand smoke reduction and cessation, or a behavioural counselling group (BC) that received a 16-week behavioural counselling intervention. The BC intervention included two home visits by study counsellors, seven counselling phone calls, and four mailings containing the contents of the educational behavioural change manual that was provided for the SCC group. The SCC group received three retention calls and seven retention postcards over the 16-week period. The primary paper describes the details of the counselling design.³⁶ At the end of the 16-week period, blinded research staff assessed the subjects again for demographic and psychosocial variables. All measures in this study were collected via structured telephone screening and in-person assessment interviews at baseline and at the end of treatment. Some measures were collected only at baseline (marital status, education level, income, employment status and age), and other measures were collected repeatedly at baseline and at the end of treatment (number of household smokers, social support, life-event stress, number of paediatric sick visits, and total child SHSe by the mother). Assessment interviews were conducted either in the participants' homes or in research offices by highly trained staff. All of the study interview questions that were not part of well-established standardised scales were tested for construct validity in a pilot sample similar to the population of the main study.³⁶ Data integrity was ensured by rigorous data-collection and data-entry protocols, which were regularly monitored by the principal investigator and senior research staff.

Outcome variable

End-of-treatment depressive symptoms, which were the primary outcome variable, were measured by the CES-D.⁵⁴ This scale was chosen because of its proven reliability and validity in detecting depressive symptoms in non-clinically depressed populations⁵⁵⁻⁵⁸ and its validity with the low-income, African

American female populations.^{55,59-61} The scale was completed both at baseline and at the end of treatment. Since the outcome of concern was the presence or absence of clinically significant depressive symptoms, the total scores at both the baseline and end-of-treatment time points were dichotomised as 0 = no/low depressive symptoms and 1 = clinically significant depressive symptoms, using the cut-off score of 16 as suggested by the scale's author.⁵⁴

Covariates

Randomisation status

To control for potential effects of the mood management components received exclusively by the BC intervention, randomisation status was also included in the model and coded as 0 = SCC and 1 = BC.

Household smokers

The number of household smokers was obtained during the interview at the end of treatment. The participant was asked 'How many smokers are there living in your home?' This number excluded the participant. It was entered into the model as a continuous variable.

Paediatric clinic sick visits

The number of paediatric sick visits was calculated for the 3 months prior to the end of treatment as the total number of times the child was taken to a paediatric healthcare facility for SHSe-related illnesses, such as ear infections or asthma. Mothers were asked 'How many times did you take [child's name] to the doctor for [SHSe-related illness]?' The mothers were given a list of six different common SHSe-related illnesses. The total number of reported visits was added. Well child visits and sick visits related to injury or elective outpatient procedures were excluded from this calculation. Sick visits were entered into our model as a continuous variable because the distribution of the variable was not highly skewed and there is no clinically meaningful cut-off point for dichotomising sick visits.⁶²

Child SHSe by the mother

The average daily number of cigarettes to which the child was exposed by the study participant at home or in the car at the end of treatment was calculated for this variable. Mothers were told 'I'm going to ask you each questions about *where* you smoked these cigarettes in the last 2 weeks.' Child SHSe was assessed by asking two questions: 'Of the cigarettes

you smoked in a car, how many were smoked while [child's name] was in the car?' and 'Of the cigarettes you smoked at home indoors, how many was [child's name] exposed to?' The answers to both of these questions were added, and the total was then divided by 14 in order to calculate the average daily SHSe of the child by the mother.

Life-event stress

Life-event stress was calculated using a scale created by a panel of intervention and public health experts.³⁸ The scale was tested for content validity in a pilot sample similar to the population of study participants. Suitable internal consistency was shown for this measure ($\alpha = 0.86$). The participants identified stressful life events experienced within the last 3 months along with their perceived level of stress in response to each identified event on a 4-point Likert scale (where 0 = did not occur/not at all stressful and 3 = extremely stressful) in a survey containing 90 items across nine categories of events related to school, work, family and intimate relationships, childcare and caregiving responsibilities, housing and living conditions, crime and legal matters, finances, social activities and health. The final variable was determined by calculating the participants' mean stress rating across all 90 items in the scale. This measure was assessed at baseline and at the end of treatment.

Social support

The Interpersonal Support Evaluation List (ISEL) was used to measure social support.⁶³ Cohen and colleagues⁶⁴ reported good overall test-retest reliability with reliability coefficients ($r = 0.87$) and good internal reliability ($\alpha = 0.90$).⁶⁵ The total ISEL score at the end of treatment was used as a continuous measure in the analysis.

Controlling covariates

Baseline CES-D scores were used to control for variability in depressive symptoms that existed prior to the start of the trial. The scale was dichotomised as 0 = score of < 16 and 1 = score of ≥ 16 .

Education was dummy coded as 0 = less than high-school graduate and 1 = high-school graduate or beyond. Mothers were asked 'What is the highest educational level you have completed?' Responses were selected from 10 possible options ranging from none to graduate degree. Mothers who selected none, elementary school, or some high school were coded 'less than high-school graduate.' The remaining seven options, including General Edu-

cational Development (GED) programme, technical training, high school and above were coded as 'high-school graduate or beyond.'

Marital status was coded as 0 (single) or 1 (married or living with a partner).

Mothers were asked 'What best describes your current marital status?' They were given three possible response options: single, never married; married or living with a partner; widowed, divorced or separated. Mothers who indicated that they were widowed, divorced or separated were considered to be single.

Employment status was coded as 0 (not employed) or 1 (employed). Mothers were asked 'Are you currently employed outside the home?' An affirmative response was coded as employed.

Participant's age was entered into the model as a continuous variable in years. We controlled for maternal age because both younger and older age have been associated with more severe depressive symptoms in this population.^{66,67}

Data analysis

All of the data were verified as being without errors and screened for outliers. All predictor and controlling variables were analysed for potential multicollinearity. Descriptive statistics were calculated for all variables. Direct-entry multivariate logistic regression was used to test our hypothesis. All of the analyses were performed using SPSS v19 software.

Results

Sample characteristics

In total, 57% of the participants reported clinically significant depressive symptoms at the end of treatment, with a mean CES-D score for mothers of 19.41 (SD = 11.5). The mean age of mothers was 30 years (SD = 7.9). They were largely single (82%), unemployed (69%) and had less than a high-school education (59%). Our population is comparable to other low-income communities in the Philadelphia area as well as in other low-income US populations,⁶⁸ and the vulnerable mothers described by Atkins.² Of those randomised to the intervention trial, 125 SSC subjects and 102 BC subjects completed the CES-D at the end of the trial ($P = 0.02$). Only three participants were excluded from the analysis due to incomplete data.

Analysis

Bivariate correlations as well as the variance inflation factors (VIF) test were performed to eliminate potentially collinear covariates. None were present as indicted with all VIF scores < 2 and Pearson correlations < 0.20. Direct-entry logistic regression analyses with all of the postulated covariates and controlling variables resulted in a statistically significant model ($\chi^2 = 95.524$, $P < 0.001$; see Table 1). The model represents a reasonably good fit with a high overall percentage of correctly classified cases (79.5%), and accounts for 46.6% of the variance in depressive symptoms (Nagelkerke $R^2 = 0.466$). The model suggests that when controlling for baseline depressive symptoms (OR = 3.76, $P < 0.001$), end-of-treatment depressive symptoms can be predicted by the number of household smokers (OR = 1.57, $P = 0.049$), stressful life events (OR = 1.044, $P = 0.001$) and decreased social support (OR = 0.88, $P < 0.001$) over the last 3 months. Postulated predictor variables, namely number of paediatric sick visits ($P = 0.677$), marital status ($P = 0.15$), employment status ($P = 0.70$), level of education ($P = 0.40$) and child's average daily exposure to cigarette smoke by the mother ($P = 0.058$), were not significant, although it may be relevant to note that the latter may be an important factor in the model. Among the other controlling variables, randomisation status ($P = 0.68$) and mother's age ($P = 0.50$) were not significant. Participant characteristics are listed by significant depressive symptoms in Table 2.

Discussion

To our knowledge, this is the first study to examine the influence of the number of household smokers and other psychosocial risk factors associated with maternal depressive symptoms during an SHSe-reduction intervention in a sample of high-risk, urban, African American maternal smokers. Number of household smokers, social support and life-event stress were all predictors of maternal depressive symptoms. Other factors known to be associated with maternal depression, including number of paediatric sick visits, child's exposure to cigarette smoke by the mother, level of education, marital status and employment status, did not contribute significantly to the model. Baseline depressive symptoms, which are a controlling variable, accounted for the largest proportion of the variance in end-of-trial maternal depressive symptoms, suggesting that mothers who entered the trial with significant de-

pressive symptoms were likely to have significant symptoms 16 weeks later.

A baseline study of factors related to significant depressive symptoms of maternal smokers entering our SHSe-reduction intervention trial (pre-treatment) provided significant findings suggesting that greater utilisation of paediatric primary care sick visits due to children's SHSe-related illnesses was an important predictor of significant depressive symptoms.³⁸ However, in our current findings, paediatric sick visits were overshadowed by psychosocial variables that may be more relevant to the intervention context. These factors may relate more to mothers' self-perception of SHSe-reduction treatment failure and psychosocial barriers and facilitators to protecting their children from SHSe.

Arguably the most interesting finding of our study suggests an association between household smokers and maternal depressive symptoms. In our population, the odds of developing clinically significant depressive symptoms increased by 61% with each additional household smoker, compared with those mothers with no or clinically non-significant depressive symptoms. Our hypothesis of the effects of social censure based on SCT is supported by this novel finding.³⁷ Household smokers can affect the mother's perceived control over her ability to change her child's environment. As a result, her perceived lack of efficacy may increase her perception of helplessness in attempting to reduce her child's SHSe by changing household smoking rules.

Likewise, social censure and criticism may be driven by social norms, which are known to have a strong influence on smoking outcomes. In this population, it is common to live in multi-generational homes where the mother may not be the matriarch.⁶⁹ If the maternal smoker is not the head of the household, or if smoking behaviour changes are not accepted by other household smokers, the mother may face greater challenges to encourage and enforce home-level changes with regard to second-hand smoke.⁷⁰ Additional research shows that smokers who live in homes with existing smoking bans are more likely to change their smoking behaviour.⁷¹ In our population, child household SHSe prior to the intervention was predicted by the child's daily exposure to cigarette smoke by the mother, and by the presence of more than one smoker in the home.³⁸ This suggests that the mother's efforts to facilitate family-level change, not just her own smoking behaviour, are key to reducing SHSe. Thus, interventions that include the head of the household to develop and build household support for health behaviour change may influence household norms in favour of home smoking bans that may be protective of maternal depressive symptoms.⁷²

Table 1 Direct-entry multivariate logistic regression model of post-intervention depressive symptoms

	Odds ratio (OR)	P-value	95% CI for OR	
			Lower	Upper
<i>Social cognitive factors</i>				
Number of household smokers	1.57	0.049	1.00	2.45
Social support (ISEL)	0.88	< 0.001	0.82	0.93
Life-event stress	1.04	< 0.001	1.02	1.07
<i>Marital status</i>				
(0 = single, 1 = married or living with partner)	0.52	0.148	0.22	1.26
Child's daily mean exposure to cigarette smoke by mother	1.17	0.058	0.99	1.37
Number of paediatric sick visits	1.08	0.677	0.75	1.57
<i>Socio-economic factors</i>				
<i>Employment status</i>				
(0 = unemployed, 1 = employed)	1.16	0.698	0.54	2.49
<i>Level of education</i>				
(0 = less than high school, 1 = at least high school)	0.73	0.398	0.36	1.51
<i>Health promotion activities</i>				
Intervention randomisation status (0 = SCC, 1 = BC)	0.86	0.680	0.42	1.76
<i>Other variables</i>				
<i>Baseline CES-D</i>				
(0-15 = 0, ≥ 16 = 1)	3.76	< 0.001	1.84	7.66
Mother's age	0.99	0.495	0.94	1.03

Statistically significant covariates are shown in bold.

CI, confidence interval; ISEL, Interpersonal Support Evaluation List; CES-D, Center of Epidemiologic Studies Depression Scale; SCC, standard of care control group; BC, behavioural counselling group.

Moreover, research suggests that there is a causal, genetic link between depression and smoking.⁷³ Thus household smokers who are blood relatives may also be predisposed to significant depressive symptoms. This reinforces the importance of SHSe and mood-management interventions at the household level in order to address the shared hereditary predisposition to both conditions.

An appreciation of the housing conditions of low-income African American families and their effect on childhood SHSe is also important for planning household interventions. For instance, most low-income households do not have additional space where smokers can restrict their smoking to isolated, child-free parts of the home.⁷⁰ Understanding the limitations of the physical environment and the constraints that it places on household smoking may also provide insights when designing effective SHSe interventions that also minimise maternal depressive symptoms.

Other factors that were not significant in our model could be considered in future research. For instance, child's SHSe as a result of smoking by the mother did approach the level of statistical significance ($P = 0.058$). Continuing to expose her children to cigarette smoke may result in perceived failure to attain mastery over the new health behaviour. Self-reporting bias may have contributed to an underestimation of children's actual exposure to second-hand smoke, which may have decreased the power to detect significance. A future study that is specifically powered to test for the effects of child's SHSe on maternal depressive symptoms may yield significant findings.

Lack of social support has also been implicated as a key contributor to maternal depressive symptoms in similar populations.^{9,40,74} For instance, African American maternal smokers with friends who smoke are less likely to receive social support for enforcing smoking bans in their homes.⁷⁵ Conversely, being

Table 2 Participant characteristics by end-of-treatment depressive symptoms and all participants

	Significant depressive symptoms present		Significant depressive symptoms absent		All participants	
Mean EOT CES-D score (SD)	27.2	(9)	9.2	(4.1)	19.4	(11.6)
Number of participants (%) with household smokers						
0	3	(2.3%)	13	(13.3%)	16	(7.1%)
1	61	(47.3%)	44	(44.9%)	105	(46.5%)
2	44	(34.1%)	36	(36.7%)	80	(35.4%)
≥ 3	20	(15.6%)	5	(5.1%)	25	(11.1%)
Number (%) of participant paediatric sick visits						
0	86	(66.7%)	76	(77.6%)	162	(71.4%)
1	23	(17.8%)	13	(13.3%)	36	(15.9%)
≥ 2	20	(15.5%)	9	(9.2%)	29	(12.8%)
Average daily child SHSe by mother <i>n</i> (%)						
0	54	(41.9%)	56	(57.1%)	110	(48.5%)
> 0–2	37	(28.5%)	24	(24.5%)	61	(26.9%)
> 2–3	11	(8.6%)	6	(6.1%)	17	(7.5%)
> 3	27	(20.9%)	12	(12.2%)	39	(17.2%)
Social support (ISEL) total score						
Mean (SD)	40	(5.4)	34.3	(5.9)	36.9	(6.3)
Life-event stress total score						
Mean (SD)	11.9	(11.3)	23.5	(19.6)	7	(5.5)
Marital status <i>n</i> (%)						
Married	20	(15.5%)	20	(20)	40	(17.7)
Unmarried	109	(84.5%)	68	(80)	186	(82.3)
Education <i>n</i> (%)						
< High school	81	(63.8%)	60	(61.9%)	141	(62.9%)
≥ High school	46	(36.2%)	37	(38.1%)	83	(37.1%)
Randomisation status <i>n</i> (%)	127	(56.7%)	97	(43.3%)		
Mother's age						
Mean (SD)	29.1	(7.1)	30.9	(9)	29.9	(8)

married may be a source of social support that decreases the likelihood of experiencing depressive symptoms among urban African American women.^{76–78} However, when couples smoke together, this behaviour undermines support for smoking behaviour change and creates potential conflict when one partner changes their smoking behaviour.^{79,80}

The lack of collinearity between social support, marital status and number of household smokers is another interesting finding. It seems to indicate that the source of the participant's perceived social support is not the household members or the participant's partner.

In terms of socio-economic factors, we emphasise that our sample was homogenous with regard to some key demographic characteristics. Most of the mothers were unemployed, single, and had not received a high-school education. The homogeneity may have limited the variability required to show significant differences in the contribution of socio-economic factors to depressive symptoms.

In view of the fact that all of the participants in this study were smokers, it is important to emphasise the well-established reciprocal association between maternal smoking and depressive symptoms. Smokers are known to have higher rates of depress-

ive symptoms than non-smokers,²⁸ and the likelihood of developing depressive symptoms increases after successful smoking cessation.⁸¹ Depressed mood also predicts smoking initiation⁸² and smoking relapse,⁸³ in addition to hindering successful attempts to quit.⁸⁴ Our population of maternal smokers had a 57% prevalence of depressive symptoms which, although lower than the values reported in some previous studies,^{85,86} is still significantly high, given that the prevalence of depressive symptoms in the general (smoking and non-smoking) African American medical population is approximately 14%.²⁸ As depression and smoking are intrinsically linked, by controlling for depressive symptoms at baseline, we are also implicitly controlling for nicotine dependence and its potential confounding of our outcome.

Other limitations of the study

One of the limitations of our study was that we did not ask the participants to elaborate on their relationship to the household smokers. Therefore we do not know whether the household smokers were partners, other relatives, friends or unrelated individuals. In future studies we shall aim to gather this information in order to elucidate where social-support-based interventions should be targeted and why household proximity and marital status show no association with perceived social support.

It is important to note that our cross-sectional data set limits the ability to make inferences about the direction of causality between psychosocial risk factors and maternal depressive symptoms. The generalisability of this study beyond low-income, urban, African American maternal smokers may also be limited.

Our limited sample size was not appropriate for the structural equation modelling techniques that would be necessary for exploring the reciprocal relationships that may exist between variables in our model and as proposed by Atkins.^{2,87} In our model, we tested the one-way hypothesis that maternal depressive symptoms can be predicted from the number of paediatric sick visits, but other studies indicate the reverse relationship, where depressive symptoms predict sick-visit healthcare utilisation.³ A well-powered study would be required to test the possibility that there are reciprocal relationships among our postulated predictors.

Given the high prevalence of depressive symptoms in our population, future research on childhood SHSe interventions should include depressive symptomology as a primary end point, as well as including more intensive mood management and social support interventions. In addition, our study

excluded mothers who self-reported a diagnosis of major depressive disorder (MDD), but did not formally investigate whether participants may have MDD at both baseline and end-of-treatment time points. As a result, we cannot ascertain the prevalence of MDD in our population, but only depressive symptoms as measured by the CES-D.

Significance

Initiating positive health behaviour changes for health promotion may be especially challenging in this population.² Understanding depressive symptoms in the context of applying a health behaviour intervention will ultimately aid the tailoring of health promotion programmes to the specific needs of this complex population. This study provides evidence that SCT may be suitable for understanding maternal depressive symptoms in African American maternal smokers. Our results suggest that future interventions for preventing and reducing maternal depressive symptoms should involve other smokers in the home, target psychosocial factors (e.g. by increasing social support), and encourage the development of coping skills for dealing with stressful life events. Thus we provide further evidence in support of the supposition that maternal smoking behaviour change interventions should be designed at the marital and household level in order to mitigate maternal depressive symptoms.⁸⁸ To date, the majority of the interventions for maternal depressive symptoms and childhood SHSe have been targeted solely at the mother.^{13,89} The addressing of maternal depressive symptoms, especially in the context of smoking behaviour change, could benefit from family-level interventions. Although there are only a few empirically tested family-based interventions, they have been shown to have great promise for reducing the maternal and child health burden of maternal depressive symptoms.^{90,91}

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ETHICAL APPROVAL

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CONFLICTS OF INTEREST

None.

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