2011

Smoking Cessation and Relapse Among Pregnant African-American Smokers in Washington, DC

Ayman El-Mohandes
CUNY School of Public Health

M. Nabil El-Khorazaty
RTI International

Michele Kieley
CUNY School of Public Health

Marie G. Gantz
RTI International

Matern Child Health J (2011) 15:S96–S105
DOI 10.1007/s10995-011-0825-6

Smoking Cessation and Relapse Among Pregnant African-American Smokers in Washington, DC

Ayman A. E. El-Mohandes · M. Nabil El-Khorazaty · Michele Kiely · Marie G. Gantz

Published online: 8 June 2011

Abstract Smoking is the single most preventable cause of perinatal morbidity. This study examines smoking behaviors during pregnancy in a high risk population of African Americans. The study also examines risk factors associated with smoking behaviors and cessation in response to a cognitive behavioral therapy (CBT) intervention. This study is a secondary analysis of data from a randomized controlled trial addressing multiple risks during pregnancy. Five hundred African-American Washington, DC residents who reported smoking in the 6 months preceding pregnancy were randomized to a CBT intervention. Psycho-social and behavioral data were collected. Self-reported smoking and salivary cotinine levels were measured prenatally and postpartum to assess changes in smoking behavior. Comparisons were made between active smokers and those abstaining at baseline and follow-up in pregnancy and postpartum. Sixty percent of participants reported quitting spontaneously during pregnancy. In regression models, smoking at baseline was associated with older age, <a high school education and illicit drug use. At follow-up closest to delivery, smoking was associated with lower education, smoking and cotinine level at baseline and depression. At postpartum, there was a relapse of 34%. Smokers postpartum were significantly more likely to smoke at baseline and use illicit drugs in pregnancy. Mothers in the CBT intervention were less likely to relapse. African-American women had a high spontaneous quit rate and no response to a CBT intervention during pregnancy. Postpartum mothers' resolve to maintain a quit status seems to wane despite their prolonged period of cessation. CBT reduced postpartum relapse rates.

Keywords Smoking · Pregnancy · African-Americans · Washington, DC

M. Nabil El-Khorazaty: Deceased.

Clinical trial registration: ClinicalTrials.gov, www.clinicaltrials.gov, NCT00381823.

A. A. E. El-Mohandes
College of Public Health, University of Nebraska Medical Center, Omaha, NE, USA

M. N. El-Khorazaty · M. G. Gantz
RTI International (RTI International is a trade name of Research Triangle Institute), Rockville, MD, USA

M. Kiely
National Institutes of Health, Bethesda, MD, USA

M. Kiely (✉)
Epidemiology Branch, Division of Epidemiology, Statistics and Prevention Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, 6100 Executive Blvd, Rm. 7B-05, Rockville, MD 20852-7510, USA
e-mail: kielym@nih.gov

Introduction

Smoking during pregnancy is associated with problems of placentation [1], low birthweight [2–8], prematurity [3, 9, 10], sudden infant death [10–12], infant mortality [12], and later physical [13], developmental [13] and behavioral [10, 14, 15] problems. A significant percentage of smokers continue to smoke during pregnancy and are unable to quit on their own despite their knowledge of the risks involved. Existing behavioral interventions are only modestly successful, having an attributable benefit of no more than 10% above spontaneous quit rates among pregnant women [16].

Success in smoking cessation during pregnancy may be different among different ethnic groups. In one study, Mexican–Americans had three times higher cessation rates than non-Hispanic whites [17]. Only a few studies in the literature describe African-American smoking behaviors during pregnancy and postpartum, and even fewer tested the efficacy of smoking cessation interventions programs in that population. Although smoking rates during pregnancy are lower among African Americans, genetically mediated differences in nicotine metabolism are associated with higher nicotine levels among African Americans compared to whites [18].

High rates of low birthweight and prematurity in African-Americans may be partly attributable to smoking in pregnancy [19], either independently or as a complicating factor for other medical risks including chronic hypertension. Although contested by some authors in the literature [20], the effect of smoking on poor birth outcomes has been estimated as high as 14.4% among black births [21]. To improve birth outcomes among African-Americans, there is a need to better understand their smoking behaviors in pregnancy and postpartum, including spontaneous cessation and relapse rates, associated variables impacting on these rates, and responses to behavioral interventions.

This study is a secondary analysis of a larger randomized controlled trial (RCT) addressing multiple risks during pregnancy. The main results of the RCT have been published elsewhere [22, 23]. This paper investigates smoking cessation and relapse rates among African American women reporting smoking in the 6 months preceding pregnancy in Washington, DC Women were recruited during pregnancy and followed through the postpartum period and randomized to an integrated cognitive behavioral intervention addressing smoking, environmental tobacco smoke exposure (ETSE), depression, and intimate partner violence (IPV).

Method

Study Population

The population recruited to this study was part of a larger cohort recruited to the District of Columbia Healthy Outcomes of Pregnancy Education (DC-HOPE), under the umbrella of the National Institutes of Health-District of Columbia Initiative to Reduce Infant Mortality in Minority Populations. DC-HOPE was a randomized controlled trial evaluating the efficacy of an integrated cognitive behavioral intervention targeting cigarette smoking, environmental tobacco smoke exposure (ETSE), intimate partner violence (IPV) and depression during pregnancy. Mothers were eligible if they were 18 years or older, English-speaking, less than 29 weeks gestation and Washington, DC residents. Women were recruited from six prenatal care sites and were screened using an audio-computer assisted self-interview (A-CASI) (For details see El-Khorazaty et al. [24]). Recruitment occurred between July 2001 and October 2003 and followed through July 2004. Baseline interviews for eligible women occurred on average 9 days after screening. IRB approval was obtained from all participating institutions.

There were 2,913 women screened and 1,515 were ineligible. Of the 1,398 eligible women, 1,070 enrolled as eligible minority participants. (See Fig. 1) 1,044 women were included in these analyses; they self-identified as African-American. Eligible women consented for randomization into the intervention or usual care group. Permuted block randomization was site- and risk-specific. The field staff were blinded with respect to the block size. Eight women (6 intervention and 2 usual care) were identified as suicidal during intervention or data collection and were referred immediately to mental health care and excluded from further participation. Five hundred women were screened into the study as having smoked a puff of a cigarette or more in the 6 months preceding pregnancy. This level was chosen to be as inclusive as possible because these women were at risk for continuing to smoke or relapsing if they had quit early in pregnancy.

Data and Saliva Sample Collection

Data on sociodemographic and behavioral risk were collected during a baseline telephone interview, on average within 9 days of screening. Follow-up telephone interviews were conducted during the second and third trimesters (22–26 weeks and 30–34 weeks, respectively) and 8–10 weeks postpartum to evaluate changes in the psychobehavioral risks. Interviewers were blinded to whether women were in the intervention or usual care group. Smoking risks during pregnancy and postpartum were measured based on self-report. Saliva samples were collected at the prenatal care site on average within 19 days following the baseline interview, within a week from the follow-up telephone interview and 23 days following the postpartum interview. Salivary cotinine was measured by a radio-immune assay using gas chromatography-mass spectrometry (GC/MS) with lower detection limits of 10 ng/ml. IPV was measured using the Revised Conflict Tactics Scale physical assault and sexual coercion subscales [25]. Depression was measured using the 20-item Hopkins Symptom Checklist-Depression Scale [26].

Intervention

Of the 500 women included in these analyses, 262 were randomized to the intervention group and 238 were

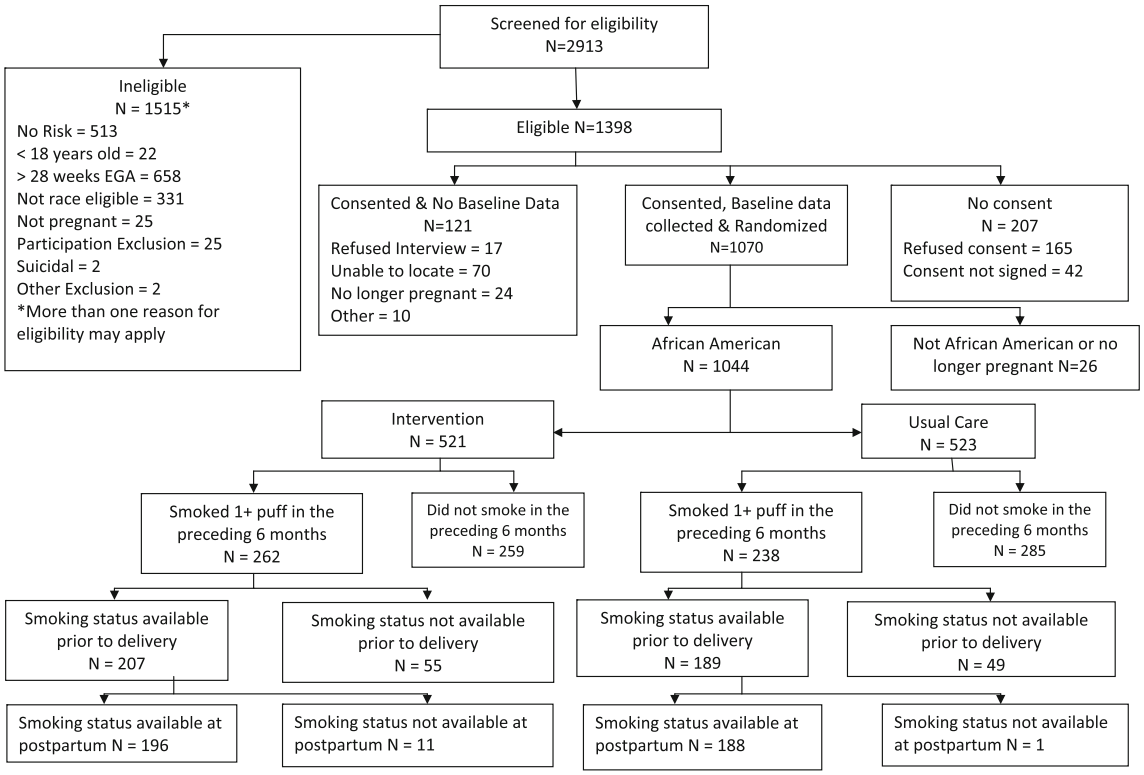

Fig. 1 Profile of project DC-HOPE randomized controlled trial

randomized to usual care. This integrated intervention was based on a conceptual framework of overlapping and interactive behavioral risks. Such risk factors are known to co-occur within a population of urban African-Americans living in communities with high poverty rates. The risks selected are all associated with poor pregnancy outcomes [27]. The smoking intervention was delivered to women who self-reported as smokers and not on a cutoff cotinine level since randomization was based on the initial response to the A-CASI.

The 10-session intervention was delivered during prenatal (8 sessions) and postpartum (2 booster sessions) care visits. Four prenatal sessions were considered minimal adherence. The session duration was approximately 35 min. The smoking intervention was consistent with the Smoking Cessation or Reduction in Pregnancy Trial (SCRIPT) and the Counseling and Behavioral Interventions Work Group of the United States Preventive Services Task Force recommendations, a five-step behavioral counseling approach [28, 29]. The intervention was tailored to the woman's stage of change. Women were encouraged to avoid triggers and to use alternative coping and behavioral change strategies. The intervention included content to address both active smoking and ETSE, whether or not they met criteria for ETSE.

The intervention sessions also addressed the other associated risks. For depression, the intervention focused on secondary prevention of symptoms in pregnancy and extended into the postpartum period. Cognitive behavioral therapy strategies for mood management, increasing positive social interactions, and pleasurable activities were emphasized. The IPV interventions used the Parker's model to address the role of a negative partner support [30]. Danger assessment to identify risks for harm and prevention options were considered along with the development of a safety plan. (For more details see Katz et al. [27]). All measures were based on validated questionnaires.

Statistical Analysis

Women who were screened as having smoked a puff of a cigarette or more in the 6 months preceding pregnancy were compared according to their self-reported smoking status at baseline interview, last follow-up interview prior to delivery, and postpartum interview conducted 8–10 weeks after delivery. Comparisons were conducted by means of Chi-square tests for binary variables and *t* tests for continuous variables.

Based on the results of these bivariate comparisons, we used logistic regression procedures to model the probability of cigarette smoking at each of the three time points controlling for covariates with P value <0.10 in the bivariate analyses. For control variables with a strong colinearity, we selected the variables with the highest level

of significance or the greatest biological plausibility. Control variables included alcohol and illicit drug use during pregnancy, depression, IPV, prior smoking status, cotinine levels, and the intervention. Variables descriptive of demographic and socioeconomic status (maternal age, education level, and Medicaid enrollment) were included as covariates so their cumulative effects could be accounted for in the final logistic model. We used the LOGISTIC procedure in SAS version 9.1.3 (SAS Institute, Cary, NC) to conduct the analysis.

Results

Of the 500 mothers reporting cigarette smoking at screening, data were available on 396 at a follow-up interview prior to delivery and 384 mothers were interviewed in the postpartum period. No significant differences between the 500 and the 396 or 384 were seen in any sociodemographic or behavioral characteristics at baseline. (Data not shown) No significant differences were noted between the intervention and usual care groups regarding sociodemographic or behavioral characteristics at baseline. Among the 500 women who reported smoking at A-CASI screening and were included in these analyses, 39% reported active smoking at baseline. An earlier paper from our study showed that women who reported smoking at A-CASI screening were significantly less likely to resolve risk (smoking, ETSE, depression and IPV) during pregnancy [22].

A significant difference was noted in salivary cotinine levels collected at baseline between mothers who self-identified as smokers and non-smokers (179 ± 156 ng/ml vs. 32 ± 59 ng/ml, $P < 0.001$). At baseline 86.4% of women who reported themselves as non-smokers had a salivary cotinine level <50 ng/ml and 90.3% <100 ng/ml.

In a logistic regression model, the factors that remain significantly associated with smoking at baseline are reviewed in Table 4A. Older maternal age, education at less than high school level and illicit drug use as reported by mothers at baseline were the factors significantly associated with smoking at baseline (Table 1).

At follow-up prior to delivery, 34% of mothers reported smoking. A significant difference was noted in salivary cotinine levels collected at that time between those reporting smoking or not (135 ± 145 ng/ml vs. 28 ± 57 ng/ml, $P < 0.001$). 83.5% of the women who reported themselves as non-smokers had a salivary cotinine level <50 ng/ml and 88.5% had a level <100 ng/ml. Of the women reporting smoking during the follow-up interview, 13.0% had not been smoking at baseline and represented a relapse. Similarly, 12.8% of non-smokers had smoked at baseline but quit at a later stage of pregnancy. There was no significant interventional effect on smoking behavior as reported in the follow-up interviews during pregnancy. Women who continued to smoke during pregnancy were significantly older, had a lower level of education attainment, and had higher rates of enrollment in Medicaid. These women were also more likely to have reported alcohol and illicit drug use during the baseline interview and higher baseline cotinine levels. Depression at baseline was a predictor of smoking at follow-up, while IPV was not. Depression and IPV confirmed during the follow-up interview were associated with smoking. No significant differences were seen between the characteristics of smokers randomized to the intervention group and those in usual care (Table 2).

In a logistic regression model (Table 4B), the factors that retained significant association with continued smoking at follow-up were active smoking at baseline and salivary cotinine levels at baseline. Depression at the follow-up period preceding delivery was also predictive of active smoking during the same time period.

In the postpartum period, 50% of participants self-reported as actively smoking. Salivary cotinine levels were significantly higher in women reporting active smoking (249 ± 176 ng/ml vs. 109 ± 149 ng/ml, $P < 0.001$). Only 60.4% of the women who reported themselves as non-smokers had a salivary cotinine level <50 ng/ml, and 64.4% had cotinine levels <100 ng/ml. The intensity of

Table 1 Women screening positive for smoking before pregnancy: baseline assessment

Characteristic	Smoking (n = 195)	Not smoking (n = 305)	*P* value
Maternal age (mean ± SD)	26.9 ± 6.3	23.6 ± 4.5	<0.001
Pregnancies (incl. current) (mean ± SD)	4.8 ± 2.9	3.5 ± 2.2	<0.001
Previous live births (mean ± SD)	2.2 ± 1.9	1.2 ± 1.4	<0.001
Relationship status:			0.770
Single/separated/ widowed/divorced	152 (78.0%)	241 (79.0%)	
Married/living with partner	43 (22.0%)	64 (21.0%)	
Education level:			0.002
<High school	91 (46.7%)	97 (31.8%)	
High school/GED	79 (40.5%)	147 (48.2%)	
Some college or more	25 (12.8%)	61 (20%)	
Medicaid recipient	174 (89.2%)	241 (79.3%)	0.004
Alcohol use	68 (34.9%)	72 (23.7%)	0.007
Illicit drug use	54 (27.7%)	47 (15.4%)	<0.001
Depression	102 (52.3%)	129 (42.3%)	0.029
Intimate partner violence	73 (37.4%)	89 (29.2%)	0.054

Table 2 Women screening positive for smoking before pregnancy: baseline smokers randomized to intervention versus usual care

Characteristic	Intervention (n = 105)	Usual care (n = 90)	*P* value
Maternal age (mean ± SD)	26.9 ± 6.5	26.8 ± 6.1	0.930
Pregnancies (incl. current) (mean ± SD)	4.8 ± 3.1	4.7 ± 2.6	0.948
Previous live births (mean ± SD)	2.2 ± 2.1	2.2 ± 1.7	0.991
Relationship status:			0.522
Single/separated/widowed/divorced	80 (76.2%)	72 (80.0%)	
Married/living with partner	25 (23.8%)	18 (20.0%)	
Education level:			0.765
<High school	51 (48.6%)	40 (44.4%)	
High school/GED	42 (40.0%)	37 (41.1%)	
Some college or more	12 (11.4%)	13 (14.4%)	
Medicaid recipient	95 (90.5%)	79 (87.8%)	0.545
Alcohol use	39 (37.1%)	29 (32.2%)	0.472
Illicit drug use	28 (26.7%)	26 (28.9%)	0.730
Active smoking at follow-up	62 (74.7%)	54 (78.3%)	0.607
Active smoking at postpartum	64 (83.1%)	67 (91.8%)	0.111
Cotinine level at baseline (mean ± SD)	192.9 ± 165.0	162.4 ± 144.6	0.216
Cotinine level at follow-up (mean ± SD)	146.0 ± 139.4	131.9 ± 117.6	0.528
Cotinine level at postpartum (mean ± SD)	290.8 ± 182.7	236.1 ± 162.2	0.103
ETSE at baseline	89 (87.3%)	75 (84.3%)	0.555
ETSE at follow-up	66 (80.5%)	52 (75.4%)	0.448
ETSE at postpartum	58 (74.4%)	56 (78.9%)	0.516
Depression at baseline	57 (54.3%)	45 (50.0%)	0.550
Depression at follow-up	39 (47.0%)	34 (49.3%)	0.779
Depression at follow-up postpartum	23 (29.9%)	23 (31.5%)	0.828
Intimate partner violence at baseline	38 (36.2%)	35 (38.9%)	0.698
Intimate partner violence at follow-up	8 (9.8%)	6 (8.7%)	0.823
Intimate partner violence at postpartum	6 (7.8%)	7 (9.6%)	0.696

smoking in the postpartum was significantly higher than during the two preceding time points as confirmed by cotinine levels. Among postpartum smokers salivary cotinine levels were significantly higher than the baseline levels ($P < 0.001$) or the levels at follow-up closest to delivery ($P < 0.001$). Of the women reporting smoking postpartum, 34% had not reported smoking during the follow-up interview during pregnancy. This group was considered postpartum relapsers. A much smaller percentage (7.4%) of women not reporting smoking postpartum had reported smoking during pregnancy. A higher likelihood of smoking postpartum was associated in bivariate analysis with older age, higher gravidity and parity, lower educational attainment, higher Medicaid enrollment, other substance use, active smoking at baseline and follow-up (Table 3). Depression documented at baseline, during follow-up interviews or in the postpartum was significantly associated with active smoking. IPV did not show a similar association at any of the three time points. The intervention for the first time showed an association with reported smoking abstinence in the postpartum period, at a *P* value of 0.053.

In a logistic regression model, factors that increased the likelihood of reported smoking in the postpartum were active smoking as reported by mothers and cotinine levels at baseline and illicit drug use during pregnancy. The intervention had a significant protective effect against smoking in the postpartum period (Table 4C).

Discussion

The results of this study confirm the difficulty pregnant mothers who smoke have in quitting during pregnancy. Mothers included in our study that were less educated, depressed or using illicit substances were least likely to quit. The literature emphasizes the underlying demographic and psychosocial factors that impact smoking behaviors among African-American women [31]. In spite of findings that African-Americans were significantly more likely than

Table 3 Women screening positive for smoking before pregnancy: postpartum assessment

Characteristic	Smoking (n = 191)	Not smoking (n = 193)	*P* value
Maternal age (mean ± SD)	25.9 ± 6.0	24.0 ± 4.9	<0.001
Pregnancies (incl. current) (mean ± SD)	4.5 ± 2.6	3.3 ± 2.2	<0.001
Previous live births (mean ± SD)	2.0 ± 1.9	1.2 ± 1.4	<0.001
Relationship status:			0.957
Single/separated/widowed/divorced	149 (78.0%)	151 (78.2%)	
Married/living with partner	42 (22.0%)	42 (21.8%)	
Education level:			<0.001
<High school	89 (46.6%)	55 (28.5%)	
High school/GED	79 (41.4%)	96 (49.7%)	
Some college or more	23 (12.0%)	42 (21.8%)	
Medicaid recipient	172 (90.1%)	149 (77.6%)	<0.001
Alcohol use	67 (35.1%)	43 (22.4%)	0.006
Illicit drug use	57 (29.8%)	23(11.9%)	<0.001
Active smoking at baseline	131 (68.6%)	19 (9.8%)	<0.001
Active smoking at follow-up	100 (64.5%)	8 (4.7%)	<0.001
ETSE at baseline	159 (85.0%)	134 (70.5%)	<0.001
ETSE at follow-up	115 (74.7%)	95 (55.6%)	<0.001
ETSE at postpartum	147 (79.0%)	97 (51.1%)	<0.001
Cotinine level at baseline (mean ± SD)	143.3 ± 155.4	36.3 ± 73.1	<0.001
Cotinine level at follow-up (mean ± SD)	122.3 ± 128.6	33.7 ± 75.0	<0.001
Cotinine level at postpartum (mean ± SD)	248.8 ± 176.0	109.3 ± 149.2	<0.001
Depression at baseline	98 (51.3%)	82 (42.5%)	0.008
Depression at follow-up	78 (50.3%)	59 (34.3%)	0.003
Depression at postpartum	62 (32.6%)	42 (21.8%)	0.017
Intimate partner violence at baseline	73 (38.2%)	57 (29.5%)	0.072
Intimate partner violence at follow-up	16 (10.3%)	11 (6.4%)	0.200
Intimate partner violence at postpartum	22 (11.6%)	15 (7.8%)	0.210
Intervention group	88 (46.1%)	108 (56.0%)	0.053

whites to express a desire to quit smoking [32] it is not clear what barriers prevent them from quitting. None of the studies meeting the guidelines for inclusion in the Public Health Service Report [33] specified abstinence rates by racial/ethnic group [34]. The only studies that report on the results of smoking cessation interventions during pregnancy by race come to opposite conclusions [35, 36]. Studies examining smoking cessation interventions during pregnancy published since the Public Health Service Report did not report their spontaneous cessation and relapse rates in pregnancy and postpartum by race/ethnicity, or found no differences in rates by race [37–41].

Our results agree with previous authors showing a significant spontaneous cessation rate among smokers who become pregnant [42, 43]. The quit rate of more than 60% in our population of urban African-American pregnant women experiencing other socioeconomic and psychological stressors is encouraging. Notably, amongst this population of smokers women who reported quitting during pregnancy experienced a high rate of depression (42%) and IPV (29%) throughout the pregnancy. Almost one-third of these women who reported quitting on their own had not completed high school, and the majority were Medicaid enrollees. It is hard to determine whether the social desirability of quitting during pregnancy within this community, and/or the knowledge of the detrimental effects of smoking on the fetus could have been the main driving force.

The underlying depressive symptoms in our study population may have interfered with their ability to control their smoking. There is a growing awareness of the prevalence of depressive symptoms within the smoking population, with a range of 22–61% amongst those entering smoking cessation programs [44–46]. The literature is mixed regarding the effect of depression on the success of smoking cessation. One may infer that depression interferes with short term quit rates and not long-term success in smoking cessation [47, 48]. In the logistic analyses we conducted at three time points, depression was predictive

Table 4 Logistic regression models to predict active smoking among pregnant women at baseline, follow-up, and postpartum

Characteristic	Odds ratio	95% Confidence interval
(A) Active smoking at baseline[a]		
Maternal age	1.14	1.10, 1.18
Education level:		
<High school	2.43	1.30, 4.54
Completed high school or GED	1.36	0.75, 2.47
At least some college (reference)	1.00	–
Illicit drug use	2.09	1.27, 3.44
(B) Active smoking at follow-up[b]		
Active smoking at baseline	18.54	8.63, 39.84
Cotinine level at baseline (10 ng/ml)	1.09	1.05, 1.13
Depression at follow-up prior to delivery	2.69	1.27, 5.68
(C) Active smoking at postpartum[c]		
Active smoking at baseline	10.89	5.28, 22.47
Cotinine level at baseline (10 ng/ml)	1.04	1.01, 1.08
Illicit drug use	2.38	1.11, 5.12
Intervention group	0.45	0.25, 0.80

[a] This model also controlled for Medicaid status, alcohol use during pregnancy, depression at baseline and IPV at baseline

[b] This model also controlled for maternal age, education, Medicaid enrollment status, alcohol and illicit drug use during pregnancy and IPV at follow-up

[c] This model also controlled for maternal age, education, Medicaid enrollment status, alcohol, depression at postpartum and IPV at baseline

of smoking during the follow-up period during pregnancy but neither at baseline nor postpartum.

Other studies have confirmed the effect of psychosocial challenges as a mediator of smoking in pregnancy [49, 50]. Alcohol and illicit drug use in the Washington, DC residents we recruited may have also interfered with their ability to quit. The relationship between alcohol use and smoking during pregnancy has been previously confirmed, although not in an exclusively African-American population [51]. Studies have shown smoking cessation in alcohol drinkers to be more difficult due to reactivity between alcohol and nicotine withdrawal [52, 53]. In our logistic models, alcohol effect was not seen to be significantly associated with smoking at any of the three time points. Illicit drug use significantly increased the chances of smoking at baseline and during the postpartum period. Illicit drug use may have served as a surrogate for the severity of addiction to nicotine, a reliable marker for maintenance of smoking and failure of cessation attempts among African-Americans [54]. This is confirmed by our findings that active smoking at baseline and cotinine level at baseline, markers for intensity of smoking, were both predictors of smoking during the follow-up interview closest to delivery and postpartum. Other studies show similar results using reported number of cigarettes smoked early in pregnancy as a marker for intensity of smoking [40].

The literature shows an association between poverty and smoking during pregnancy and postpartum [51, 55, 56]. In this study we used Medicaid enrollment as a marker for poverty. In bivariate analyses, Medicaid was a significant predictor of smoking during the three time points. In the logistic models, Medicaid lost its significance and a low level of education was only significantly associated with smoking at baseline. However, other studies have shown that education is negatively associated with smoking during pregnancy and with relapse after delivery [57]. It is plausible that level of education may influence the knowledge base mothers draw upon in their decision making during pregnancy. A more compelling argument would be a high resilience in mothers attaining higher educational levels under challenging living conditions in environments of urban poverty. Such women may also possess a higher level of self-efficacy proven to impact significantly on successful smoking cessation [40]. Women with higher levels of education may be products of a more supportive social environment, which is known to influence successful quitting during pregnancy as well [39].

Some women may quit early in pregnancy due to physical aversion to tobacco smoke during the first trimester [58]; these pregnant mothers may then be susceptible to relapse at a later stage. Our results show relapse during pregnancy as reported by the population we studied (13%) to be lower than previously reported in the Canadian study (21%) [43]. In fact, the reported smoking rates in our population declined from 39% at baseline to 33% during follow-up.

The postpartum period represents a different challenge, where a high percentage of women resume smoking after a prolonged period of cessation. The literature cites mediators to resumed smoking such as postpartum depression and concerns related to weight gain [59, 60]. Although this has not been studied in populations that are predominately

black, in a study based on the Pregnancy Risk Assessment Monitoring system, postpartum relapse was significantly more likely among black mothers [59]. It is also possible mothers are less aware of the harms of ETSE to infants as compared to in utero exposure. The added stress of parenting a newly born infant, especially among mainly single mothers with limited social network and community support, may trigger the need for stress relief associated with smoking [49]. Sixty-five percent of women who quit during pregnancy will relapse by 3 months and an additional 10% by 6 months postpartum [61]. Other studies show that of those who quit smoking during pregnancy, half relapse at 2–6 months [62] and 60–70% relapse within 1 year [63] after delivery. In our study, women who reported actively smoking increased from 33 to 50% during our follow-up period of 10 weeks postpartum.

Few studies address the efficacy of interventions targeting reduction of postpartum relapse [64, 65]. We were encouraged this integrated intervention did impact on relapse rates reported postpartum. This could be explained by a longer exposure to the intervention, but also the emphasis on ETSE as a significant risk to the newborn infant, which may have encouraged mothers to maintain their quit status. In addition, emphasis on mood regulation could have assisted mothers in dealing with postpartum depression and the stress associated with caring for a newborn. Previous studies showing similar success postpartum emphasized interventions including partners and close friends, and encouraging the social networks to support the mother in her decision [66, 67]. Although our intervention did not address either of these strategies directly, it encouraged women to establish a supportive social network, and as such may have had similar effects. Furthermore, studies emphasize the postpartum success of interventions if they start earlier in pregnancy [67], which was our case.

The strength and limitation of our study is that it was conducted with high-risk African American women. The results cannot be generalizable to other populations without corroboration. Although the intervention did not influence smoking behavior significantly during the pregnancy, it had a protective effect against relapse during the postpartum. This study also confirmed the importance of associated addictions to illicit drugs and co-occurring depression as important associations with smoking during pregnancy in this population. More qualitative research to examine why African American women may or may not be inclined to stop smoking in pregnancy may inform research in the future in the design of appropriate interventions with efficacy in this population.

The results of this study support the importance of screening early in pregnancy and providing mothers with opportunities for behavioral modification through culturally informed interventions. Behavioral interventions for smoking should be available but cannot be relied upon alone as the intervention of choice for mothers who continue to smoke during pregnancy. More research is needed to test efficacy and safety of pharmacological therapy with proven efficacy in non-pregnant populations. Studies such as ours emphasize the importance of expanding prenatal care beyond the medical model in order to respond to the complex health risks of minority populations during pregnancy.

Acknowledgments The authors wish to thank the field work staff, the interviewers, and data management staff. We wish to thank the participants who welcomed us into their lives in hopes of helping themselves and their children. This work was supported by grants no. 3U18HD030445; 3U18HD030447; 5U18HD31206; 3U18HD031919; 5U18HD036104, *Eunice Kennedy Shriver* National Institute of Child Health and Human Development and the National Center on Minority Health and Health Disparities, National Institutes of Health, Department of Health and Human Services. These analyses were supported, in part, by the intramural program of the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development.

Conflict of interest None of the authors have any conflict of interests to declare.

References

1. Zdravkovic, T., Genbacev, O., McMaster, M. T., & Fisher, S. J. (2005). The adverse effects of maternal smoking on the human placenta: A review. *Placenta, 26*(Suppl A), S81–S86.
2. Bernstein, I. M., Mongeon, J. A., Badger, G. J., Solomon, L., Heil, S. H., & Higgins, S. T. (2005). Maternal smoking and its association with birth weight. *Obstetrics and Gynecology, 106*(5), 986–991.
3. Jaddoe, V. W., Troe, E. J., Hofman, A., Mackenbach, J. P., Moll, H. A., Steegers, E. A., et al. (2008). Active and passive maternal smoking during pregnancy and the risks of low birth weight and preterm: The generation R study. *Paediatric and Perinatal Epidemiology, 22*, 162–171.
4. Li, C. Q., Windsor, R. A., Perkins, L., Goldenberg, R. L., & Lowe, J. B. (1993). The impact on infant birth weight and gestational age of cotinine-validated smoking reduction during pregnancy. *JAMA, 269*, 1519–1524.
5. Magee, B. D., Hattis, D., & Kivel, N. M. (2004). Role of smoking in low birth weight. *Journal of Reproductive Medicine, 49*, 23–27.
6. Peacock, J. L., Cook, D. G., Carey, I. M., Jarvis, M. J., Bryant, A. E., Anderson, H. R., et al. (1998). Maternal cotinine level during pregnancy and birth weight for gestational age. *International Journal of Epidemiology, 27*, 647–656.
7. Secker-Walker, R. H., Vacek, P. M., Flynn, B. S., & Mead, P. B. (1997). Smoking in pregnancy, exhaled carbon monoxide, and birth weight. *Obstetrics and Gynecology, 89*, 648–653.
8. Wang, X., Tager, I. B., Van Vunakis, H., Speizer, F. E., & Harahan, J. P. (1997). Maternal smoking during pregnancy, urine cotinine concentrations, and birth outcomes. A prospective cohort study. *International Journal of Epidemiology, 26*, 978–988.
9. Burns, L., Mattick, R. P., & Wallace, C. (2008). Smoking patterns and outcomes in a population of pregnant women with other

substance use disorders. *Nicotine & Tobacco Research, 10*, 969–974.
10. Shea, A. K., & Steiner, M. (2008). Cigarette smoking during pregnancy. *Nicotine & Tobacco Research, 10*, 267–278.
11. Hunt, C. E., & Hauck, F. R. (2006). Sudden infant death syndrome. *CMAJ, 174*(13), 1861–1869.
12. Salihu, H. M., & Wilson, R. E. (2007). Epidemiology of prenatal smoking and perinatal outcomes. *Early Human Development, 83*, 713–720.
13. Jaakkola, J. J. K., & Gissler, M. (2004). Maternal smoking in pregnancy, fetal development, and childhood asthma. *American Journal of Public Health, 94*(1), 136–140.
14. Day, N. L., Richardson, G. A., Goldschmidt, L., & Cornelius, M. D. (2000). Effects of prenatal tobacco exposure on preschoolers' behavior. *Journal of Developmental and Behavioral Pediatrics, 21*, 180–188.
15. Wakschlag, L. S., & Han, S. L. (2002). Maternal smoking during pregnancy and conduct problems in high-risk youth: A developmental framework. *Development and Psychopathology, 14*, 351–369.
16. Lumley, J., Chamberlain, C., Dowswell, T., Oliver, S. S., Oakley, L., Watson, L. (2009). Interventions for promoting smoking cessation during pregnancy. *Cochrane Database Systems Review, 8*(3), CD001055.
17. Camilli, A. E., McElroy, L. F., & Reed, K. L. (1994). Smoking and pregnancy: A comparison of Mexican-American and non-Hispanic white women. *Obstetrics and Gynecology, 84*, 1033–1037.
18. Wang, X., Zuckerman, B., Pearson, C., Kaufman, G., Chen, C., Wang, G., et al. (2002). Maternal cigarette smoking, metabolic gene polymorphism, and infant birth weight. *JAMA, 287*, 195–202.
19. Moore, M. L., & Zaccaro, D. J. (2000). Cigarette smoking, low birth weight, and preterm births in low-income African American women. *Journal of Perinatology, 3*, 176–180.
20. Mathews, T. J., & MacDorman, M. F. (2006). Infant mortality statistics from the 2003 period linked birth/infant death data set. *National Vital Statistics Reports, 54*, 1–29.
21. Barnett, E. (1995). Race differences in the proportion of low birth weight attributable to maternal cigarette smoking in a low-income population. *American Journal of Health Promotion, 10*(2), 105–110.
22. Joseph, J. G., El-Mohandes, A. A. E., Kiely, M., El-Khorazaty, M. N., Gantz, M. G., Johnson, A. A., et al. (2009). Reducing psychosocial and behavioral pregnancy risk factors: Results of a randomized clinical trial among high-risk pregnant African American women. *American Journal of Public Health, 99*(6), 1053–1061.
23. El-Mohandes, A. A., Kiely, M., Joseph, J. G., Subramanian, S., Johnson, A. A., Blake, S. M., et al. (2008). An intervention to improve postpartum outcomes in African-American mothers: A randomized controlled trial. *Obstetrics and Gynecology, 112*(3), 611–620.
24. El-Khorazaty, M. N., Johnson, A. A., Kiely, M., El-Mohandes, A. A. E., Subramanian, S., Laryea, H. A., et al. (2007). Recruitment and retention of low-income minority women in a behavioral intervention to reduce smoking, depression, and intimate partner violence during pregnancy. *BMC Public Health, 7*, 233.
25. Straus, M. A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The Revised Conflict Tactics Scale (CTS2): Development and preliminary psychometric data. *Journal of Family Issues, 17*, 283–316.
26. Derogatis, L. R., Lipman, R. S., Rickels, K., Uhlenhuth, E. H., & Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A self-report symptom inventory. *Behavioral Science, 19*, 1–15.
27. Katz, K. S., Blake, S. M., Milligan, R. A., Sharps, P. W., White, D. B., Rodan, M. F., et al. (2008). The design, implementation and acceptability of an integrated intervention to address multiple behavioral and psychosocial risk factors among pregnant African American women. *BMC Pregnancy Childbirth, 8*, 22.
28. Windsor, R. A. (2000). Counseling smokers in Medicaid maternity care: The SCRIPT project. *Tobacco Control, 9*(Suppl 1), i62.
29. Whitlock, E. P., Orleans, C. T., Pender, N., & Allan, J. (2000). Evaluating primary care behavioral counseling interventions: An evidence-based approach. *American Journal of Preventive Medicine, 22*(4), 267–284.
30. Parker, B., McFarlane, J., Soeken, K., Silva, C., & Reel, S. (1999). Testing an intervention to prevent further abuse to pregnant women. *Research in Nursing and Health, 22*, 59–66.
31. Webb, M. S., & Carey, M. P. (2008). Tobacco smoking among low-income Black women: Demographic and psychosocial correlates in a community sample. *Nicotine & Tobacco Research, 10*, 219–229.
32. Royce, J. M., Hymowitz, N., Corbett, K., Hartwell, T. D., & Orlandi, M. A. (1993). Smoking cessation factors among African-Americans and whites. COMMIT Research Group. *American Journal of Public Health, 83*, 220–226.
33. Fiore, M. C., Bailey, W. C., Cohen, S. J., Dorfman, S. F., Goldstein, M. G., Gritz, E. R., et al. (2000). *Treating tobacco use and dependence. AHRQ publication no. 00–0032*. Washington DC: US Department of Health and Human Services.
34. Piper, M. E., Fox, B. J., Welsch, S. K., Fiore, M. C., & Baker, T. B. (2001). Gender and racial/ethnic differences in tobacco-dependence treatment: A commentary and research recommendations. *Nicotine & Tobacco Research, 3*, 291–297.
35. Gebauer, C., Kwo, C. Y., Haynes, E., & Wewers, M. (1998). A nurse-managed smoking cessation intervention during pregnancy. *Journal of Obstetric, Gynecologic, and Neonatal Nursing, 27*(1), 47–53.
36. Windsor, R. A., Lowe, J., Perkins, L., Smith-Yoder, D., Artz, L., Crawford, M., et al. (1993). Health education for pregnant smokers: Its behavioral impact and cost benefit. *American Journal of Public Health, 83*, 201–206.
37. Händel, G., Hannöver, W., Röske, K., Thyrian, J. R., Rumpf, H. J., John, U., et al. (2009). Naturalistic changes in the readiness of postpartum women to quit smoking. *Drug and Alcohol Dependence, 101*(3), 196–201.
38. Higgins, S. T., Heil, S. H., Badger, G. J., Skelly, J. M., Solomon, L. J., & Bernstein, I. M. (2009). Educational disadvantage and cigarette smoking during pregnancy. *Drug and Alcohol Dependence, 104*(Suppl 1), S100–S105.
39. Ma, Y., Goins, K. V., Pbert, L., & Ockene, J. K. (2005). Predictors of smoking cessation in pregnancy and maintenance postpartum in low-income women. *Maternal and Child Health Journal, 9*, 393–402.
40. Morasco, B. J., Dornelas, E. A., Fischer, E. H., Oncken, C. A., & Lando, H. A. (2006). Spontaneous smoking cessation during pregnancy among ethnic minority women: A preliminary investigation. *Addictive Behaviors, 31*, 203–210.
41. Ruger, J. P., Weinstein, M. C., Hammond, S. K., Kearney, M. H., & Emmons, K. M. (2008). Cost-effectiveness of motivational interviewing for smoking cessation and relapse prevention among low-income pregnant women: A randomized controlled trial. *Value Health, 11*(2), 191–198.
42. Canadian Task Force on the Periodic Health Examination. (1994). *The Canadian guide to clinical preventive health care*. Ottawa: Canada Communication Group—Publishing, p. 28.
43. Kirkland, S. A., Dodds, L. A., & Brosky, G. (2000). The natural history of smoking during pregnancy among women in Nova Scotia. *CMAJ, 163*, 281–282.

44. Hall, S. M., Muñoz, R. F., & Reus, V. I. (1994). Cognitive-behavioral intervention increases abstinence rates for depressive-history smokers. *Journal of Consulting and Clinical Psychology, 62*, 141–146.
45. Hall, S. M., Muñoz, R. F., Reus, V. I., Sees, K. L., Duncan, C., Humfleet, G. L., et al. (1996). Mood management and nicotine gum in smoking treatment: A therapeutic contact and placebo-controlled study. *Journal of Consulting and Clinical Psychology, 64*, 1003–1009.
46. Kinnunen, T., Doherty, K., Militello, F. S., & Garvey, A. J. (1996). Depression and smoking cessation: Characteristics of depressed smokers and effects of nicotine dependence. *Journal of Consulting and Clinical Psychology, 64*, 791–798.
47. Glassman, A. H., Covey, L. S., Dalack, G. W., Stetner, F., Rivelli, S. K., Fleiss, J. F., et al. (1993). Smoking cessation, clonidine, and vulnerability to nicotine among dependent smokers. *Clinical Pharmacology and Therapeutics, 54*, 670–679.
48. Ginsberg, D., Hall, S. M., Reus, V. I., & Muñoz, R. F. (1995). Mood and depression diagnosis in smoking cessation. *Experimental and Clinical Psychopharmacology, 3*, 389–395.
49. Goedhart, G., van der Wal, M. F., Cuijpers, P., & Bonsel, G. J. (2009). Psychosocial problems and continued smoking during pregnancy. *Addictive Behaviors, 34*, 403–406.
50. al'Absi, M., Carr, S. B., & Bongard, S. (2007). Anger and psychobiological changes during smoking abstinence and in response to acute stress: Prediction of smoking relapse. *International Journal of Psychophysiology, 66*, 109–115.
51. Martin, L. T., McNamara, M., Milot, A., Bloch, M., Hair, E. C., & Halle, T. (2008). Correlates of smoking before, during, and after pregnancy. *American Journal of Health Behavior, 32*, 272–282.
52. McKee, S. A., Krishnan-Sarin, S., Shi, J., Mase, T., & O'Malley, S. S. (2006). Modeling the effect of alcohol on smoking lapse behavior. *Psychopharmacology (Berl), 189*, 201–210.
53. Zimmerman, R. S., Warheit, G. J., & Ulbrich, P. M. (1990). The relationship between alcohol use and attempts and success at smoking cessation. *Addictive Behaviors, 15*(3), 197–207.
54. Choi, W. S., Okuyemi, K. S., Kaur, H., & Ahluwalia, J. S. (2004). Comparison of smoking relapse curves among African-American smokers. *Addictive Behaviors, 29*, 1679–1683.
55. Jun, H.-J., & Acevedo-Garcia, D. (2007). The effect of single motherhood on smoking by socioeconomic status and race/ethnicity. *Social Science and Medicine, 65*, 653–666.
56. Everett-Murphy, K., Steyn, K., Mathews, C., Petersen, Z., Odendaal, H., Gwebushe, N., et al. (2010). The effectiveness of adapted, best practice guidelines for smoking cessation counseling with disadvantaged, pregnant smokers attending public sector antenatal clinics in Cape Town, South Africa. *Acta Obstetricia et Gynecologica Scandinavica, 89*, 478–489.
57. Kahn, R. S., Certain, L., & Whittaker, R. C. (2002). A reexamination of smoking before, during, and after pregnancy. *American Journal of Public Health, 92*, 1801–1808.
58. Pletsch, P. K., Pollak, K. I., Peterson, B. L., Park, J., Oncken, C. A., Swamy, G. K., et al. (2008). Olfactory and gustatory sensory changes to tobacco smoke in pregnant smokers. *Research in Nursing and Health, 31*, 31–41.
59. Allen, A. M., Prince, C. B., & Dietz, P. M. (2009). Postpartum depressive symptoms and smoking relapse. *American Journal of Preventive Medicine, 36*, 9–12.
60. Quinn, G., Ellison, B. B., Meade, C., Roach, C. N., Lopez, E., Albrecht, T., et al. (2006). Adapting smoking relapse–prevention materials for pregnant and postpartum women: Formative research. *Maternal and Child Health Journal, 10*, 235–245.
61. US Department of Health and Human Services. (1990). *The Health Benefits of Smoking Cessation*. US Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Chronic Disease prevention and Health Promotion, Office on Smoking and Health. DHHS Publication No. (CDC) 90-8416.
62. Carmaichael, S. L., & Ahluwalia, I. B. (2000). Correlates of postpartum smoking relapse: Results from Pregnancy Risk Assessment Monitoring System (PRAMS). *American Journal of Preventive Medicine, 19*, 193–196.
63. Severson, H. H., Andrews, J. A., Lichtenstein, E., Wall, M., & Akers, L. (1997). Reducing maternal smoking and relapse: Long-term evaluation of a pediatric intervention. *Preventive Medicine, 26*, 120–130.
64. Fang, W. L., Goldstein, A. O., Butzen, A. Y., Hartsock, S. A., Hartmann, K. E., Helton, M., et al. (2004). Smoking cessation in pregnancy: A review of postpartum relapse prevention strategies. *The Journal of the American Board of Family Practice, 17*(4), 264–275.
65. Reitzel, L. R., Vidrine, J. I., Businelle, M. S., Kendzor, D. E., Costello, T. J., Li, Y., et al. (2101). Preventing postpartum smoking relapse among diverse low-income women: A randomized clinical trial. *Nicotine & Tobacco Research, 12*, 326–335.
66. Gielen, A. C., Windsor, R. A., Faden, R., O'Campo, P., Repke, J., & Davis, M. (1997). Evaluation of smoking cessation intervention for pregnant women in an urban prenatal clinic. *Health Education Research, 12*, 247–254.
67. Mullen, P. D., Richardson, M. A., Quinn, V. P., & Ershoff, D. H. (1997). Postpartum return to smoking: Who is at risk and when. *American Journal of Health Promotion, 11*, 323–330.