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Pathways Linking Socioeconomic Status and Postpartum Smoking Relapse

Michael S. Businelle^a, Darla E. Kendzor^a, Lorraine R. Reitzel^b, Jennifer Irvin Vidrine^b, Yessenia Castro^b, Patricia Dolan Mullen^c, Mary M. Velasquez^d, Ludmila Cofta-Woerpel^e, Paul M. Cinciripini^e, Anthony J. Greisinger^f, and David W. Wetter^b

^aDivision of Health Promotion and Behavioral Sciences, University of Texas School of Public Health, Dallas, TX, USA

^bDepartment of Health Disparities Research, University of Texas M.D. Anderson Cancer Center, Houston, TX, USA

^cDivision of Health Promotion and Behavioral Sciences, University of Texas School of Public Health, Houston, TX, USA

^dCenter for Social Work Research, University of Texas at Austin, Austin, TX, USA

^eDepartment of Behavioral Science, University of Texas M.D. Anderson Cancer Center, Houston, TX, USA

^fKelsey Research Foundation, Houston, TX, USA

Abstract

Background—Low socioeconomic status (SES) exacerbates the high rate of smoking relapse in women following childbirth.

Purpose—This study examined multiple models of potential mechanisms linking SES and postpartum smoking relapse among women who quit smoking due to pregnancy.

Methods—Participants were 251 women enrolled in a randomized clinical trial of a new postpartum smoking relapse prevention intervention. Four models of the prepartum mechanisms linking SES and postpartum smoking relapse were evaluated using a latent variable modeling approach.

Results—Each of the hypothesized models were a good fit for the data. As hypothesized, SES indirectly influenced postpartum smoking relapse through increased prepartum negative affect/ stress, reduced sense of agency, and increased craving for cigarettes. However, the model that included craving as the sole final pathway between SES and relapse demonstrated superior fit when compared with all other models.

Conclusions—Findings have implications for future interventions that aim to reduce postpartum relapse.

Keywords

Smoking; Postpartum; Structural Equation Modeling; Relapse; Socioeconomic Status

Corresponding Author: Michael S. Businelle, Ph.D., University of Texas School of Public Health, Dallas Regional Campus, 6011 Harry Hines Blvd., V8.112, Dallas, TX, 75390-9128, michael.businelle@utsouthwestern.edu Phone: 214-648-1070. **Conflict of Interest Statement:** The authors have no conflict of interest to disclose.

Introduction

Although the number of women who smoke cigarettes in the United States has declined over the past half century [1], approximately 22% of all women of child bearing age continue to smoke [2] and 10–12% of women report smoking during their pregnancy [3]. Although half of all women who smoke quit immediately prior to or during their pregnancy [4, 5], up to 80% of those women will return to smoking within one year postpartum [e.g., 6]. Unfortunately, many studies have shown that children exposed to secondhand smoke have higher mortality rates and are at greater risk for numerous health problems including respiratory diseases and ear infections [e.g., 7, 8, 9]. In order improve the health of women and their children, studies are needed to illuminate the reasons why women return to smoking during the postpartum period.

Low socioeconomic status (SES) has been consistently linked with higher rates of smoking in women before and during pregnancy [e.g., 10, 11-15] and is also a risk factor for relapse to smoking following childbirth [10, 13, 16]. Despite widespread knowledge of the relations between SES, smoking, and smoking relapse, few studies have examined the mechanisms linking SES to postpartum smoking relapse in women who quit due to pregnancy. However, women of lower SES experience higher levels of perceived stress and negative affect during pregnancy compared to their higher SES counterparts [17, 18]. Higher perceived stress and negative affect are related to lower self-efficacy for abstinence [17] and a higher likelihood of postpartum relapse [e.g., 16, 19, 20]. Finally, abstinent pregnant women who report lower agency (e.g., self-efficacy/confidence) for maintaining abstinence are more likely to relapse postpartum [21, 22]. Thus, increased negative affect and perceived stress, and lower selfefficacy for smoking cessation may tax the already limited resources of socioeconomically disadvantaged pregnant women and may trigger greater craving for cigarettes and postpartum smoking relapse. The purpose of this study was to investigate prepartum negative affect/stress, self-efficacy, and craving as potential mechanisms linking SES and postpartum smoking relapse.

Over the past decade, a number of conceptual models have been developed that link SES to specific health behaviors and overall health. Notable models include those developed by Gallo and Mathews [23] and Adler and Ostrove [24]. Both of these models hypothesize that SES is linked to health and health behavior through environmental and psychosocial variables. More specifically, these models hypothesize that individuals of lower SES may exhibit more negative health behaviors than individuals of higher SES due to increased exposure to stressors and fewer interpersonal and intrapersonal resources that can be used to effectively cope with perceived stressors. Recent research has provided support for these models by indicating that perceived stress, negative emotions (e.g., depression, anxiety), social support, and other interpersonal and intrapersonal resources and constraints do mediate the relation between SES and several health behaviors [e.g., 25, 26]. These mediating variables may be particularly relevant for pregnant women, given the stress associated with pregnancy and caring for a newborn (e.g., physical discomfort, sleep deprivation, decreased availability of time to care for self and others), pregnancy-related and postpartum negative affect, and the increased need for social support and other inter- and intra-personal resources in the pre- and postpartum periods (e.g., assistance with prenatal care, postpartum care of the newborn, effective coping skills)[e.g., 17, 18, 27]. Perceived stress and negative emotions may be exacerbated among socioeconomically disadvantaged pregnant women who have fewer financial and social resources, and this may increase the likelihood of postpartum relapse.

In the area of substance abuse, the Witkiewitz and Marlatt [28] cognitive-behavioral model also hypothesizes mediational pathways through which distal variables like SES can affect

substance use and relapse. More specifically, variables such as negative affect, perceived stress, cognitions, social support, agency (e.g., self-efficacy for quitting), and craving are hypothesized to play key roles in the relation between SES and substance use. Notably, Witkiewitz and Marlatt [28] hypothesized reciprocal relationships among cognitive variables (e.g., agency, expectancies, craving, motivation), affective variables, and stress-related variables. In other words, craving for nicotine may increase negative affect and vice versa, with either direction of causality affecting the likelihood of relapse. These relationships remain to be evaluated among socioeconomically disadvantaged pregnant women who may experience both elevated negative affect and craving.

Based on the work of Adler and Ostrove [24], Gallo and Mathews [23], and Witkiewitz and Marlatt [28], Businelle and colleagues [29] developed and tested a conceptual model evaluating the hypothesized mediational pathways linking SES to smoking cessation in a racially/ethnically diverse sample of men and women seeking smoking cessation treatment. Results indicated that low SES increased the likelihood of relapse through its effects on perceived neighborhood disadvantage, social support, negative affect/stress, and agency for quitting. Additionally, results showed that craving indirectly increased the likelihood of relapse by increasing negative affect and perceived stress, and by reducing agency.

Importantly, many of the significant pathways described in Businelle et al. [29] have yet to be tested in women who quit smoking due to pregnancy. For instance, it is unclear whether prepartum craving for cigarettes predicts postpartum relapse among women who have been abstinent for an extended period of time, although previous studies have indicated that craving significantly predicts relapse among ex-smokers even after extended periods of abstinence [e.g., 30, 31]. Additionally, it is unclear if the pattern of relationships between negative affect, perceived stress, agency, and craving in women who quit smoking due to pregnancy mirrors the pattern seen in the sample described in Businelle et al. [29].

The primary purpose of this study was to evaluate conceptual models of the pathways linking SES and postpartum smoking relapse in a sample of women who quit smoking due to pregnancy. Key potential mediators were previously selected based on relevant theoretical models [e.g., 24, 28, 32] and hypothesized models were developed in which the configuration of several key latent variables and the direction of pathways between latent variables were based on the model described in Businelle et al. [29; see Figure 1, Model 1]. Specifically, low SES was hypothesized to be related to high negative affect/stress, which in turn was hypothesized to result in lower agency, which was hypothesized to increase the likelihood of postpartum smoking relapse. In addition, this model included hypothesized indirect pathways linking craving to smoking relapse through high negative affect and low agency. Because the model described in Businelle et al. [29] was developed in a substantially different sample of smokers (i.e., a racially/ethnically diverse sample of current smokers attempting to quit); a secondary purpose of this study was to determine if alternate configurations of the hypothesized mediating variables and the inclusion of additional pathways between the mediators and subsequent relapse would significantly improve the model fit for this unique sample. Specifically, we proposed three alternate models to: determine whether craving for cigarettes was a more proximal predictor for postpartum relapse as compared to agency (see Figure 1, Model 2); test whether including direct pathways from negative affect/stress and craving to relapse would improve the fit of Model 1 (see Figure 1, Model 3); and determine if adding direct pathways from negative affect/ stress and agency to relapse would improve the fit of Model 2 (see Figure 1, Model 4).

Methods

Participants

Data for the current study were collected as part of a randomized clinical trial designed to evaluate a Motivation And Problem Solving (MAPS) counseling approach to prevent postpartum relapse among women who quit smoking due to pregnancy [33]. Participants were recruited through the local health care system and via newspaper, radio, bus, and clinic advertisements. Participants were eligible to participate if they: 1) were at least 18 years of age, 2) were fluent in English, 3) were between 30 and 33 weeks pregnant, and 4) quit smoking during their current pregnancy or within 2 months prior to becoming pregnant.

Procedures

Potential participants were screened over the telephone to determine eligibility. Those who met the study inclusion criteria were invited to enroll in the study over the phone and were scheduled for the baseline session. Informed consent was completed at the baseline session. Participants were randomly assigned to receive usual care (n = 115) or one of two experimental MAPS treatments (n = 136). Those assigned to the usual care group received self-help materials and brief relapse prevention advice in the clinic after they completed baseline questionnaires. Materials used for usual care were based on the Treating Tobacco Use and Dependence Clinical Practice Guideline [34]. Participants assigned to the MAPS groups received usual care and either six MAPS telephone counseling sessions or six MAPS telephone counseling sessions and two in-person MAPS sessions [see 33 for further details about the MAPS treatment sessions]. All demographic and psychosocial measures described below were collected during the baseline visit which occurred between 30 to 33 weeks of pregnancy. Participants completed follow-up assessments at 8 and 26 weeks postpartum. Participants were compensated with \$40.00 gift cards and other small items (e.g., diaper bags) at each assessment visit. Participant enrollment and flow through the study have been reported elsewhere [see 33]. The study was approved by the Institutional Review Board of the University of Texas MD Anderson Cancer Center.

Measures

Demographics—Demographic information including age, annual household income, total years of education completed, number of prior pregnancies, partner status (i.e., married or living with significant other = 1; single = 0), race/ethnicity (i.e., three dummy coded variables representing African American, Latino, and Other Race were created with White as the comparison group), and employment status (i.e., employed or on maternity leave from a full- or part-time job = 1; homemaker, student, or unemployed = 0) were collected at the baseline visit.

Tobacco use—Information related to tobacco use was collected at the baseline visit and included years smoked and pre-pregnancy smoking rate.

Negative Affect/stress—Negative affect/stress was assessed using three separate measures collected at the prepartum baseline visit. Variables were chosen and combined based on the findings of previous research [e.g., 29]. The *Positive and Negative Affect Scale* [PANAS; 35] consists of Positive Affect and Negative Affect subscales, each consisting of 10 affect related adjectives. Scores range from 10 to 50 and higher scores indicate higher levels of positive and negative affect. Only the Negative Affect subscale was used in the current study. The *Center for Epidemiologic Studies Depression Scale* [CES-D; 36] consists of 20-items designed to assess depressive symptoms in community populations. Scores range from 0 to 60, and scores 16 and higher indicate clinically significant distress. The

Perceived Stress Scale [37] consists of four items designed to assess the degree to which an individual believes his/her life is stressful. Scores range from 0 to 16 and higher scores indicate greater levels of perceived stress.

Agency—Two measures of prepartum agency were administered during the baseline visit in the current study. The *Self-Efficacy/Confidence Scale* [SECS; 38] was developed to assess an individual's confidence in their ability to refrain from smoking in three types of situations: positive affect/social situations, negative affect situations, and habit/craving situations. Each subscale consists of three items and higher scores indicate higher confidence in the ability to refrain from smoking. The *Affective Information Processing Questionnaire* [AIPQ; 39] assesses an individual's belief in his/her ability to control their mood without smoking in 10 negative affect laden hypothetical situations. Higher scores indicate greater belief in the ability to successfully control mood without smoking.

Craving—Craving for cigarettes was assessed at the baseline visit using the four item Craving subscale from the *Wisconsin Smoking Withdrawal Scale* [WSWS; 40]. Each item is rated on a 5-point Likert-type scale, with higher scores indicating higher levels of craving.

Smoking Status—Self-reported postpartum smoking abstinence (i.e., continuously abstinent = 0; smoking at least one puff by follow-up = 1) was assessed at the week 8 and week 26 follow-up sessions and was confirmed via carbon monoxide (CO) readings or by salivary cotinine in cases where we could not obtain CO readings. CO readings greater than or equal to 10 parts per million were considered indicative of relapse as were salivary cotinine readings greater than or equal to 20 ng/ml.

Analytic Plan

The Mplus software package [41] was used to fit the data to the four hypothesized structural equation models. Potential mediator variables were selected based upon previous research and hypothesized links between SES and smoking relapse [e.g., 25, 29, 42]. Latent variables were created for SES, Negative Affect/Stress, Agency, and Craving. The factors used to construct these latent variables were identical to those used in Businelle et al. [29]. Specifically, the SES latent variable included three indicators: education, income, and employment status. The Negative Affect/Stress latent variable included the Positive and Negative Affect Scale negative affect scale, the Perceived Stress Scale, and the Center for Epidemiologic Studies Depression Scale. The Agency latent variable included the three Self-Efficacy/Confidence Scale subscales and the Affective Information Processing Questionnaire. The Craving latent variable was made up of the four-item Craving subscale from the Wisconsin Smoking Withdrawal Scale. Age, partner status, race/ethnicity, and number of previous births, were included as covariates in all models. In order to control for the effect of treatment on relapse status, a treatment group variable (i.e., Motivation And Problem Solving [MAPS] vs. usual care) was included in all models. The primary outcome variable was whether or not participants relapsed by the 8 week postpartum follow-up. In cases where abstinence status could not be determined due to missing data, participants were considered relapsed consistent with the "intent-to-treat" approach commonly used in other smoking cessation studies [e.g., 43, 44]. However, analyses were also conducted after coding those who did not attend the 8 week follow-up session as missing.

Employment status, partner status, race/ethnicity, and relapse status were coded as categorical variables. All other variables were coded as continuous. The outcome variable for all models was categorical. As such, weighted least squares with robust standard errors (WLSMV) parameter estimation was used to estimate free parameters. The following goodness of fit indices were used to determine how well the observed data fit the

hypothesized models [see 45, 46–48]: 1) chi-square goodness of fit index [e.g., 46, 49], 2) comparative fit index [CFI; 45], 3) Tucker-Lewis Index [TLI; 50], 4) root mean square error of approximation [RMSEA; 51], and 5) weighted root mean square residual (WRMR). Recommended cutoff points for these indices are as follows: CFI .95, TLI .95, RMSEA

.05, and WRMR 1.0. Significance of indirect pathways between SES and relapse status were tested using the "Model Indirect" command in Mplus.

As previously noted, four structural equation models were developed and tested to determine: 1) if the current data were a good fit for the model described in Businelle et al. [29], and 2) if alternate models of the relation between SES, psychosocial mediators, and postpartum smoking relapse would fit the current data better than the Businelle et al. model. Although all of the tested models included the same observed and latent variables, the linkages of the latent variables differed across the models (see Figure 1). Model 1 was most closely related to the previously tested model [29]. Alternate models were compared to Model 1 to determine the best fitting model for the current data.

Because there are no empirical tests of the superiority of non-nested structural equation models using the WLSMV estimator, models were re-estimated using the maximum likelihood estimator in order to obtain the Bayesian Information Criterion [BIC; 52] for each model. The BIC is a likelihood measure that can be used to determine if one non-nested structural equation model is superior to another [52–54]. Specifically, BIC differences of greater than 10 points may be interpreted as "very strong" evidence that the model with the smaller BIC is superior to the model with the larger BIC [53, 54]. BIC differences of 6–10 points indicate "strong" evidence that the model with the smaller BIC is superior to the model with the larger BIC [53, 54].

Results

Participant Characteristics

A total of 251 women enrolled in the study (32% African American, 30% Latino, 36% White). On average, participants were 24.6 (\pm 5.3) years old and reported 12.9 (\pm 2.0) years of education. Overall, participants reported 0.88 previous births (\pm 1.14) and 62.8% of the sample reported that they were married or living with their significant other. Median household income was \$20,000 – \$29,999 and 41% percent of the sample reported that they were currently working or on maternity leave from a full or part time job. Participants reported smoking 10.2 (\pm 7.6) cigarettes per day prior to becoming pregnant and had been smoking for 6.8 (\pm 4.3) years. Consistent with recent abstinence from smoking, expired carbon monoxide levels were 2.1 (\pm 1.6) parts per million on average at the baseline visit. See Table 1 for additional participant characteristics.

Attrition

A total of 200 (79.7%) participants completed assessments at the 8-week follow-up and 197 (78.5%) participants completed the 26-week follow-up. Participants who did not complete follow-up assessments were coded as relapsed. A total of 64.5% and 80.1% of the sample were classified as relapsed by the 8-week and 26-week follow-ups, respectively.

Associations among Observed Variables

Zero order correlations among all observed variables are listed in Table 2. As expected, variables within the same latent construct had the highest correlations. Notably, week 8 relapse status was significantly correlated with all but two of the indicator variables (i.e., income and PANAS negative affect) that were included in the models tested in this study.

Structural Equation Modeling

Four models were developed to describe possible relations between SES and week 8 relapse after controlling for age, partner status, race/ethnicity, and number of previous births. Results indicated that Model 1 was a good fit for the observed data: χ^2 (43, N = 251) = 54.59, p = .11; CFI = .959; TLI = .967; RMSEA = .033; WRMR = .832 and examination of the LaGrange modification indices resulted in no changes to the model. Results indicated that Model 2 [χ^2 (45, N = 251) = 53.90, p = .17; CFI = .969; TLI = .975; RMSEA = .028; WRMR = .799], Model 3 [χ^2 (43, N = 251) = 49.59, p = .23; CFI = .977; TLI = .981; RMSEA = .025; WRMR = .785], and Model 4 [χ^2 (44, N = 251) = 53.08, p = .16; CFI = . 968; TLI = .974; RMSEA = .029; WRMR = .794] were also an adequate fit for the observed data. It is notable that each of the hypothesized direct pathways in Models 1 and 2 were significant. However, the direct pathways linking the Negative affect/Stress and Agency latent variables to relapse were not significant in Models 3 and 4 (i.e., only the direct pathway from Craving to relapse was significant). Each of these models was re-run after coding women who did not attend the week 8 follow up as missing. Results were virtually identical to the intent to treat analyses.

In order to determine if any of the alternate models were superior to Model 1 [from 29], Bayesian Information Criterion (BIC) values for each model were obtained by refitting the data using maximum likelihood estimation. Results revealed that the BIC for Model 1 was 15520.94, the BIC for Model 2 was 15509.92, the BIC for Model 3 was 15518.26, and the BIC for Model 4 was 15518.63. Thus, based on the Raftery [54] criteria, these findings indicate that there is "very strong" evidence that Model 2 is superior to Model 1 and "strong" evidence that Model 2 is superior to Models 3 and 4. Since Models 3 and 4 were not significantly different from Model 1, they will not be discussed further. The standardized path coefficients and factor loadings for Models 1 and 2 are displayed in Figures 2 and 3 respectively. BIC values were also obtained by re-running these maximum likelihood models after coding women who did not attend the week 8 follow up as missing (i.e., completer only analyses). Results indicated that there is "strong" evidence that Model 2 is superior to Models 1, 3, and 4.

SES directly and indirectly influenced week 8 postpartum relapse status. Specifically, low SES was significantly related to greater risk of relapse by the 8 week follow-up assessment across all models. The indirect pathways between SES and week 8 relapse status were examined using the "Model Indirect" function in Mplus. Results indicated that the indirect pathway linking SES to relapse in Model 1 (i.e., SES \rightarrow Negative Affect/Stress \rightarrow Agency \rightarrow Week 8 Postpartum Relapse) approached significance (standardized structural coefficient = -0.024, *p* = .057). In Model 2, both of the indirect paths linking SES to week 8 smoking relapse were significant. Specifically, the path SES \rightarrow Negative Affect/Stress \rightarrow Agency \rightarrow Craving \rightarrow Week 8 Postpartum Relapse was significant (standardized structural coefficient = -.018; *p* = .02) and the path SES \rightarrow Negative Affect/Stress \rightarrow Craving \rightarrow Week 8 Postpartum Relapse was also significant (standardized structural coefficient = -.018; *p* = .02) and the path SES \rightarrow Negative Affect/Stress \rightarrow Craving \rightarrow Week 8 Postpartum Relapse was also significant (standardized structural coefficient = -.023; *p* = . 03). Importantly, negative affect/stress, craving, and agency also had significant direct/ indirect effects on relapse status in both Model 1 and Model 2. A decomposition of all the indirect and total effects on relapse status for Models 1 and 2 for each of the latent variables is depicted in Table 3.

Models were rerun using the Week 26 data. Each of the models were a good fit for the Week 26 relapse data, and there were only minor differences between the Week 8 and Week 26 models. For example, with respect to our final model, the only difference between the Week 8 and the Week 26 model was that the pathway between treatment group and relapse status became nonsignificant in the Week 26 model.

Other Issues

Most previous studies have shown that nicotine dependence is related to SES [e.g., 55, 56] and smoking cessation [e.g., 16, 57, 58], although several recent studies have indicated that level of nicotine dependence may not predict smoking relapse [e.g., 29, 59]. In addition, studies have found that living with a smoker may predict smoking relapse in women who quit smoking due to pregnancy [e.g., 22, 60, 61]. In the current study, multiple measures of nicotine dependence (i.e., number of cigarettes smoked per day prior to pregnancy, time to first cigarette after waking prior to pregnancy) and living with a smoker were related to SES indicators, but were not related to postpartum smoking relapse. Attempts to add these variables into the tested models resulted in reductions in model fit. Therefore, these variables were not included in final models.

Discussion

The current study is among the first to examine mediational pathways between SES and postpartum smoking relapse in women who quit smoking due to pregnancy. As most postpartum smoking relapse occurs among socioeconomically disadvantaged women [4], understanding the mechanisms that underlie this process may inform relapse prevention interventions that aim to reduce relapse rates within vulnerable postpartum populations. Multiple mediator variables (i.e., negative affect/stress, craving, and agency) were examined and four plausible models were tested. Analyses indicated that a model that included craving as the sole final pathway between SES and relapse (Model 2) was the best fit for the data. As shown in Figure 3, low SES both directly and indirectly increased the likelihood of relapse in women who quit smoking due to pregnancy. Specifically, findings indicated that women of lower SES reported higher levels of negative affect/stress during the latter part of their pregnancy (i.e., 30–33 weeks into pregnancy) and both high negative affect/stress and low agency increased the likelihood of postpartum relapse by increasing craving to smoke. These findings illuminate mechanisms through which SES impacts postpartum smoking relapse and highlight potential targets for future tailored treatments to prevent postpartum relapse.

The superiority of Model 2 over the other three models that were tested in the current study highlights the important role that prepartum craving plays as a mediator of the SESpostpartum relapse relationship. To the best of our knowledge, this is the first study to show that prepartum craving predicts postpartum relapse. However, some previous work has indicated that craving for cigarettes can predict smoking relapse, long after cessation [30, 31]. The weight of the relationship between craving and postpartum relapse becomes more evident when the results of Models 3 and 4 are taken into consideration. In Models 3 and 4, direct pathways linking each of the cognitive variables (i.e., negative affect/stress, agency, and craving) to smoking relapse were simultaneously tested. Results indicated that the paths between craving and relapse maintained significance, while pathways between the other cognitive variables and relapse were not significant. These findings suggest that prepartum negative affect/stress and agency primarily have an indirect impact on postpartum smoking relapse through their effects on prepartum craving. Although smoking cessation treatments most definitely have a major component focused on coping with craving, there are actually very few relapse prevention interventions that are implemented outside the context of achieving initial cessation. Perhaps even more importantly, coping with craving is not a primary topic of focus for smokers who have been abstinent for extended periods of time as are women who quit smoking immediately prior to or during pregnancy [see 62 for a review on pre and postpartum smoking interventions]. Therefore, the fact that prepartum craving for cigarettes is the final common pathway linking SES to smoking relapse suggests that interventions may need to have a specific focus on preparing women, and low SES women in particular, to cope with cravings for cigarettes, and that high levels of prepartum craving might signal a need for intervention or additional intervention. In addition, low SES women

may benefit from additional training in learning how to diffuse negative affect/stress and low self-efficacy for quitting before they can lead to greater cravings for cigarettes and eventual relapse.

The results of this study support findings from previous research that have shown the relation between SES and smoking relapse is mediated by negative affect/stress [29, 63], craving [29], and agency [29, 63]. However, this is the first study to simultaneously examine multiple mediational pathways between SES and postpartum smoking relapse. It is important to note that the models tested in the current study examined how affective and cognitive variables may interact to effect the relation between SES and postpartum relapse, whereas the Adler and Ostrove (1999), Gallo and Matthews (2003), and Witkiewitz and Marlatt (2004) models are more general frameworks of the relation between SES and health/ health behavior. Thus, this study has begun to clarify how SES interacts with these important mechanisms to increase the likelihood of postpartum relapse. Future research should examine whether these models can be improved by adding other potential mediators of the SES-relapse relationship (e.g., health concern, knowledge of the negative health effects of cigarette smoke on children, biological measures of stress, neighborhood disadvantage, infant temperament, sleep quantity/quality).

It is notable that a previous study found that agency was the best fitting final common pathway between SES and smoking relapse and craving only influenced relapse indirectly through increased negative affect/stress and reduced agency [29]. The finding that agency was not the best fitting final common pathway in the current study was not completely unexpected considering the significant differences between the samples described in Businelle et al. [29] and the current study. For instance, the Businelle et al. [29] sample consisted of men and women who were undergoing a quit attempt and the negative affect/ stress, agency, and craving measures were all collected on the quit date. In this study, these variables were collected 30-33 weeks into pregnancy, months after subjects had already quit smoking. Taken together, these findings suggest that post-quit cravings measured shortly before childbirth have a more direct impact on postpartum relapse than negative affect/stress and agency, while quit date measures of agency have a more direct impact on relapse in men and women during the early stages of a quit attempt. One potential explanation is that craving experienced months after quitting serves as a critical marker of relapse vulnerability. For example, craving may reflect a host of potential influences (e.g., dependence, comorbidity, chronic stress, environmental factors). Conversely, among individuals who are in their first days of abstinence, craving is a much more typical experience and may be less predictive than one's confidence that they can maintain abstinence [e.g., see 29].

The current study has a number of strengths. First, the models tested in the current study were based on a smoking relapse model that was developed in a completely different sample of diverse smokers seeking cessation treatment [see 29]. Thus, there is evidence that this general framework may be applied to diverse groups of smokers/former smokers seeking treatment. Second, the current sample was comprised of a diverse group of women (e.g., nearly identical numbers of African American, Latino, and White women). Thus, study findings may be generalizable across several racial/ethnic groups. A third strength of the current study is that self-reported abstinence was confirmed via biochemical means at both postpartum time points. A final strength is that multiple models were tested and compared to determine the best fitting model for the current data. Future research should further test these models to determine if Model 2 is in fact superior to the other three models of the relation between SES and postpartum smoking relapse.

The current study also has a number of limitations. First, although each of the four tested models were an adequate fit for the observed data and validate portions of a previously

tested model [29], other plausible models may have been an equal or better fit for the data. Thus, additional evaluation of these models using larger sample sizes is needed for further model validation [64]. A second limitation is that the mediating variables (i.e., craving, negative affect/stress, agency) were all collected at the same point in time. Future research studies should collect longitudinal data on each hypothesized mediator variable in order to obtain a more accurate indication of the temporal interactions between each of the hypothesized mediator variables. The timing of assessments has important implications for the results of the current study. Assessments of negative affect/stress, agency, and craving were conducted at 30-33 weeks into pregnancy, while relapse was measured at 8 and 26 weeks postpartum. Future studies should build upon this model by adding postpartum assessments of these key mechanisms. Postpartum assessments could be used in conjunction with prepartum assessments to identify whether the trajectories of these variables predict relapse. A third limitation of the current study is that negative affect, depression, and perceived stress were all placed into one latent construct. This was done because these variables were highly correlated (range of correlation: .518 to .655) and each of them loaded highly onto the Negative Affect/Stress latent variable (loadings ranged from .682 to .819). Future research in this area may benefit from collecting larger samples sizes that will allow for the parsing of the roles of perceived stress, negative affect, and depression as mediators of the SES-smoking relapse relation.

The findings of this study highlight potential avenues through which the SES-postpartum smoking relapse gradient may be reduced. Specifically, prepartum interventions for socioeconomically disadvantaged women who quit smoking due to pregnancy may be more effective if they target variables that mediate the SES-relapse relationship (e.g., negative affect/stress, agency for maintaining abstinence, and craving for smoking). Such interventions might be more effective than standard cessation interventions because they would specifically address and ameliorate the varying stressors and circumstances that contribute to postpartum relapse. Although the appropriate timing of such tailored treatments (i.e., prepartum, postpartum, or both) is unclear, previous researchers have suggested that relapse prevention treatments may be most beneficial if they are offered through the prenatal and postpartum periods [e.g., 6, 65]. Future research studies should further examine how specific sources of perceived stress and negative affect (e.g., financial strain, temperament of the child, lack of transportation, neighborhood disadvantage) impact smoking craving and subsequent relapse in low SES women. Clarification of how specific pre- and post-partum stressors impact craving and relapse may lead to the development of more effective postpartum relapse prevention treatments. In addition, postpartum pharmacotherapy may be considered as a way to reduce craving and negative mood for low SES women who quit smoking due to pregnancy.

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Model 4





Model 2

Structural equation models of the relation between SES and relapse status.

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Figure 2.

Standardized path coefficients and factor loadings for Model 1. SES = Socioeconomic Status; PANAS NA = Positive and Negative Affect Scale-Negative Affect; PSS = Perceived Stress Scale; CES-D = Center of Epidemiologic Studies Depression Scale; AIPQ = Affective Information Processing Questionnaire. 0 = continuous abstinence, 1 = relapsed; 0 = usual care, 1 = MAPS.

Solid paths are significant at the p < .05 level, dotted paths are not significant.

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Figure 3.

Standardized path coefficients and factor loadings for Model 2. SES = Socioeconomic Status; PANAS NA = Positive and Negative Affect Scale-Negative Affect; PSS = Perceived Stress Scale; CES-D = Center of Epidemiologic Studies Depression Scale; AIPQ = Affective Information Processing Questionnaire.

0 =continuous abstinence, 1 =relapsed; 0 =usual care, 1 =MAPS.

Solid paths are significant at the p < .05 level, dotted paths are not significant.

Table 1

Participant Characteristics

Variable	Mean (SD)
Positive and Negative Affect Scale-Negative Affect	21.71 (8.33)
Perceived Stress Scale	6.47 (3.14)
Center of Epidemiologic Studies Depression Scale	18.23 (11.33)
Self-Efficacy/Confidence Scale	
Positive Affect	3.74 (0.98)
Negative Affect	3.38 (1.19)
Habit/Craving	4.18 (0.84)
Affective Information Processing Questionnaire	5.05 (1.33)
Wisconsin Smoking Withdrawal Scale	
Urge	1.39 (1.29)
Thoughts	1.32 (1.24)
Desire	1.47 (1.31)
Focus	0.83 (1.02)

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Table 2

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Zero Urder Cori	elations													
	1	7	3	4	S	9	7	8	6	10	11	12	13	14
SES														
1. Education	ı													
2. Employment ^I	.401 ^{***}	ı												
3. Income	.520***	.343***	ı											
Affect/Stress														
4. PANAS NA	021	013	059	ı										
5. PSS	154*	094	182**	.655***	·									
6. CES-D	209***	114	234	.579***	.518***	ı								
Agency														
7. Positive affect	078	076	066	293***	213***	205***	I							
8. Negative affect	039	.058	.013	314***	239***	241	.743***	ı						
9. Habit/craving	.071	.062	.125	347***	269***	298***	.762***	.725***	I					
10. AIPQ	.060	.020	.064	270***	259***	233	.420***	.449***	.505***					
Craving														
11. Urges	051	059	039	.355***	.363***	.229***	362***	326 ^{***}	360 ^{***}	208***				
12. Thoughts	061	038	013	.273***	.305***	.188**	346***	286***	364***	253***	.683	,		
13. Desire	016	.007	.063	.237***	.270***	.137*	317***	301***	328***	205***	.617 ^{***}	.634 ^{***}		
14. Focus	072	038	012	.305***	.325***	.219***	420 ^{***}	380***	434 ^{***}	262***	.527 ^{***}	.675***	.542***	ı
Relapse Status														
15. Week 8 ²	148*	129*	128	.066	.145*	.129*	192**	240***	191	134*	.203 ^{***}	.306***	.196**	.276 ^{***}
* <i>p</i> <.05;														
$_{p < .01;}^{**}$														
$^{***}_{p < .001};$														

 $^{I}0$ = unemployed, 1 = employed;

 $^20 =$ continuous abstinence, 1 = relapsed.

SES = Socioeconomic Status; PANAS NA = Positive and Negative Affect Scale-Negative Affect; PSS = Perceived Stress Scale; CES-D = Center of Epidemiologic Studies Depression Scale; AIPQ = Affective Information Processing Questionnaire.

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Table 3

Standardized Indirect and Total Effects of Predictors of Smoking Relapse at 8 Weeks Postpartum

	Mode	Model 1 Model 2		el 2
Variable	Indirect	Total	Indirect	Total
Socioeconomic Status	-0.02^{*}	-0.24	-0.04	-0.26
Negative Affect/Stress	0.09	0.09	0.19	0.19
Agency	-	-0.41	-0.20	-0.20
Craving	0.25	0.25	-	0.43

Note: All indirect and total effects are significant at the p < .05 level;

* p = .057

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