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Self-efficacy as a pathway to long-term smoking cessation among low-income parents in the multilevel Kids Safe and Smokefree intervention

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ABSTRACT

Background: This study investigated the effects of a multi-level smoking intervention on mediators of long-term abstinence in parental smokers, including smoking cessation self-efficacy, smoking urge coping, and perceived support to quit smoking.

Methods: This is a secondary analysis of data from a randomized trial that recruited parental smokers from pediatric clinics in low-income communities (N = 327, 83% women, 83% African American, 79% below poverty level). Following clinical practice guidelines for tobacco intervention ("Ask, Advise, Refer" [AAR]), pediatricians asked all parents about child tobacco smoke exposure (TSE), advised about TSE harms and benefits of reducing TSE, and referred smokers to cessation resources. Eligible parents were then randomized to additional telephone-based smoking behavior counseling (AAR + counseling) or nutrition education (AAR + control). Bioverified 7-day point prevalence smoking abstinence and perceived counselor support were assessed at 12-month follow-up; cessation self-efficacy and urge coping were assessed at 3-month follow-up.

Results: Relative to AAR + control, AAR + counseling was associated with higher self-efficacy, urge coping, and perceived support to quit (all p's < .001). Self-efficacy, but no other mediators, had a significant positive effect on 12-month bioverified smoking abstinence (p < .001). The indirect effect of intervention on 12-month abstinence via self-efficacy suggested mediation via this pathway (p = .002).

Conclusion: Results suggest that all putative treatment pathways were improved more by the multi-level AAR + counseling than the clinic-level AAR + control intervention. Further, self-efficacy at end-of-treatment prospectively predicted long-term cessation, suggesting that building of self-efficacy through treatment may be key to sustained cessation.

1. Introduction

Environmental tobacco smoke exposure (TSE) continues to pose significant public health risks, particularly among children living in low-income and African American communities (Homa et al., 2015). Child TSE often occurs in home settings, so it is critical to address smoking behaviors of parents to protect children from TSE. The Kids Safe and Smokefree (KiSS) intervention study used a multi-level strategy to reduce child TSE and promote smoking cessation among parents from low-income and predominantly African American communities (Lepore et al., 2013). In addition to a brief clinic-level intervention delivered to parents during a pediatric clinic visit with their child, KiSS intervention participants received individualized telephone counseling to modify their smoking behaviors. In this paper, we examine the pathways through which the intervention may have contributed to long-term parental smoking cessation.

Smoking-cessation interventions delivered primarily by telephone have significant reach and potential for helping smokers quit. Free tobacco quitlines are available in all U.S. states (Rudie and Bailey, 2018). There is considerable evidence backing the effectiveness of telephone-based counseling for tobacco smoking cessation (Stead et al., 2007), but little is known about the mechanisms through which these interventions work (Lichtenstein et al., 2010). In particular, we know little about intervention factors that improve long-term smoking outcomes in high-risk, low-income and racial-minority populations because of the dearth of prospective, longitudinal treatment studies with these special populations (Borrelli, 2010). Examining the pathways through which the KiSS intervention influenced long-term smoking cessation outcomes can provide insights into mechanisms of action and inform the next generation of tobacco interventions with underserved populations.

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In the KiSS randomized controlled trial, parents were recruited through pediatric primary care clinics that mostly serve families in low-income and predominantly racial-minority communities. All parents received clinic-level intervention that consisted of pediatric healthcare providers following clinical best practice guidelines (Farber et al., 2015) that included: asking parents if they smoke, advising them about the harms of TSE and the benefits or protecting children from TSE, and referring them to self-help resources (Ask, Advise, Refer; AAR). Eligible, consenting parents were then randomized to one of two conditions: telephone-delivered counseling focused on smoking behaviors (AAR + counseling) or telephone-delivered education focused on family nutrition (AAR + control). Parents' bioverified 7-day point-prevalence smoking abstinence was measured at 12-month follow-up. As previously reported, parents in the AAR + counseling group were 2.47 times more likely to have bioverified smoking abstinence at 12-month follow-up than those in the AAR + control group (Lepore et al., 2018). To minimize ambiguity in the direction of relations in the mediation model investigated in the current paper, putative behavioral mediators were assessed at three months (end-of-treatment), which is nine-months prior to the final abstinence outcome measure.

The conceptual basis for the KISS trial, including the *a priori* identified mediators, has been described elsewhere (Collins et al., 2018; Lepore et al., 2013, 2018). The multilevel KiSS intervention arm combining clinic-level AAR and individual-level, telephone-based behavioral counseling was guided by ecological and associative learning theories, including Social Cognitive Theory (Bandura, 2004) and the Behavioral Ecological Model (Hovell and Hughes, 2009). The manualized telephone counseling intervention applied evidence-based strategies to promote both child protection from TSE and parent smoking cessation. Primary strategies included providing social support, education about and logistical support to access NRT, cognitive-behavioral counseling to develop smoking urge management coping skills, and motivational interviewing techniques to facilitate collaborative, personalized treatment. We theorized that getting parents to first make simple changes in their smoking behavior, such as not smoking near their child and establishing household rules against smoking, would build motivation and self-efficacy to achieve more challenging goals, such as quitting smoking. This behavioral shaping approach was used in our earlier successful intervention promoting child TSE reduction (Collins et al., 2015)

Smoking is a complex behavior influenced by multiple social-environmental, biological, and psychological factors (Borland et al., 2010; Fagerstrom and Fagerström, 2012; Palipudi et al., 2012). Hence, successful interventions are often intensive and target multiple determinants of smoking using evidence-based techniques, such as education, pharmacotherapy, social support, motivational enhancement, and cognitive-behavioral strategies (Fiore et al., 2008; Siu and U.S. Preventive Services Task Force, 2015). The KiSS intervention used this multicomponent approach and focused on three mediators: cessation self-efficacy, use of urge coping skills to control smoking temptations, and social support from the telephone counselor to achieve cessation goals.

The mediator variables were selected based on prior theory and behavioral intervention studies. Self-efficacy is a well-established correlate of abstinence and relapse during smokers' quit attempts (Gwaltney et al., 2009), possibly because it increases efforts to cope with high-risk internal (e.g., negative affect) and external (e.g., visual cues) smoking triggers (Bandura, 2004). For example, in an intervention study designed to promote smoking cessation in a sample of persons living with HIV, participants in the intervention group reported greater increases in cessation self-efficacy than their counterparts in the control group and the change in self-efficacy accounted for 17% of the total beneficial effect of the intervention on abstinence post-intervention (Vidrine et al., 2015). Intentional use of coping strategies during high-risk situations that trigger an urge to smoke also can potentially reduce risks of lapsing during quit attempts. For example, among a sample of smokers attempting to quit, those who applied more coping strategies were better able to resist smoking urges than those who applied relatively few coping strategies (O'Connell et al., 2007). Finally, we identified perceived support from the telephone counselor as a potential mediator because provision of abstinence-specific encouragement, information about smoking and quitting, and emotional support is a hallmark of effective individual, group, and telebased behavioral interventions (Westmaas et al., 2010). There is evidence that when healthcare providers give intratreatment social support, smokers are more likely to be quit than when support is not given (Fiore et al., 2008).

In sum, the present study investigated the effects of the multilevel KiSS intervention on putative mediators of long-term smoking cessation: smoking cessation self-efficacy, urge coping, and counselor support. The longitudinal design helped to clarify directionality between the mediators and the outcome.

1.1. Hypotheses

1 Participants in the AAR + counseling group will report higher cessation self-efficacy and more smoking urge coping behaviors at 3-month follow-up and perceive greater counselor support for quitting at 12-month follow-up than their counterparts in the AAR + control condition (direct effects of intervention on mediators).
2 Cessation self-efficacy and smoking urge coping behaviors at 3-month follow-up, and perceived intra-trial counselor support for quitting at 12-month follow-up, will be positively associated with bioverified smoking abstinence at 12-month follow-up (direct effects of mediators on bioverified quit).
3 There will be significant indirect effects of intervention on bioverified abstinence via the mediators, consistent with mediation.

We also controlled for five potential confounding variables in the mediation model. Four of these–parent depressive symptoms, nicotine dependence, presence of another household smoker, and child age at baseline–were identified *a priori* based on predictors of smoking outcomes found in the child TSE and smoking cessation literature (Lepore et al., 2013). The fifth control variable, self-reported 7-day point prevalence abstinence from smoking at 3-month follow-up, was included based on the observation that not controlling for concurrent quit status can bias estimates of the relation between self-efficacy and subsequent quit status (Gwaltney et al., 2009).

2. Methods

2.1. Procedures

This is a secondary data analysis of a two-group randomized control trial that assessed the efficacy of a multilevel intervention combining pediatric clinic-level intervention (AAR) combined with telephone-based, individual-level counseling to help parents protect their children from TSE and to quit smoking relative to just a pediatric clinic-level intervention (AAR) with attention control consisting of nutrition education. Data were collected either at baseline (Time 1; T1), 3-month follow-up (Time 2; T2), or 12-month follow-up (Time 3; T3). Details on the study protocol, sample and setting are available in previous publications (Collins et al., 2018; Lepore et al., 2013, 2018). The study was conducted in accordance with the Declaration of Helsinki. All procedures were approved by the appropriate Institutional Review Boards, and all participants provided informed consent.

Briefly, parental smokers (N = 327, 83% women, 83% African American; 79% living below the poverty level) were recruited during regular visits at participating pediatrics clinics throughout the city of Philadelphia. The clinics serve neighborhoods predominantly populated by low-income African American families that rent and are at high risk for child TSE (Homa et al., 2015). Eligible participants included

tobacco smoking parents with a child under 11 years old with daily TSE in the home. Additional inclusion criteria included being a daily smoker, > 17 years old, and English speaking. Exclusion criteria included pregnancy, psychiatric diagnosis, non-nicotine drug dependence, and daily consumption of > 2 alcoholic beverages.

During routine pediatric visits, healthcare providers implemented the AAR protocol: parents were asked about child TSE, advised about the harms of TSE and benefits of reducing it, and if they were a smoker they received a referral to cessation resources. Prompts to engage in AAR were embedded in the electronic health record (EHR) system in all participating clinics, so pediatricians would routinely engage in the practice. All parents received the clinic-level intervention independent of their interest in the KiSS trial, as it was part of clinic quality improvement efforts to recommend best care for their patients living with a smoker. In addition, parents received self-help print materials (Environmental Protection Agency's brochure, "Secondhand Smoke and the Health of Your Family") and print resources related to quitting (Pennsylvania Quitline number, information on how to access free NRT).

Following in-clinic AAR intervention, eligible and consented parents completed a baseline assessment survey and were randomized to a 12-week treatment in either: a) an experimental group (AAR + counseling) that received print materials and up to five sessions of telephone counseling for smoking behavior change (M = 3.35 + 1.81 calls completed) or b) an attention control group (AAR + control) that received print materials and telephone nutrition education. The AAR + counseling intervention sessions were delivered by phone by trained and supervised counselors who used cognitive behavioral therapy strategies, including goal setting, self-monitoring, skills training related to coping with smoking urge; social support (provision of encouragement, caring, and information); motivational interviewing techniques to develop a personalized cessation plan; and navigation to acquire and properly use NRT.

2.2. Measures

Treatment group assignment, AAR + control (0) vs. AAR + counseling (1), was the independent variable. The primary outcome was parents' 7-day point prevalence smoking abstinence defined as reporting no cigarettes, not even a puff, in the previous seven days at T3 (0 = smoker; 1 = abstinent). Self-reports were bioverified using saliva cotinine measured by NicAlert™ test strips. Participants were classified as abstinent if their NicAlert™ reading was < 10 ng/ml cotinine (Cooke et al., 2008). We also collected expired carbon monoxide (CO) as a back-up measure (e.g., if a participant was using NRT, which would bias the NicAlert™ reading). Participants were classified as abstinent if their CO was < 10 ppm (Benowitz et al., 2002).

There were three mediators. We used the 12-item Tobacco Urge Management Scale (α = .87) at T2 to measure the extent to which participants applied different coping strategies to avoid smoking temptations (Collins et al., 2018). We used the 12-item self-efficacy for avoiding smoking scale (DiClemente, 1981) at T2 to measure participants' cessation self-efficacy (α = .89). We adapted the 5 positive support items from the Partner Interaction Questionnaire (Cohen and Lichtenstein, 1990) to measure at T3 the extent to which participants perceived that the telephone counselor was supportive of their efforts to abstain from smoking. Items were re-worded to measure positive support from the intervention telephone counselor, not one's partner (e.g., "How often did the phone counselor compliment you on your efforts to not smoke"). We also added two additional items about the counselor's provision of encouragement and help in obtaining nicotine replacement, such as gum or patch. The resulting 7-item measure had good reliability (α = .89).

We measured four control variables at T1: self-reported presence of other smokers in the home (0 = no, 1 = yes), level of nicotine dependence using the Fagerström test (α = .50) (Heatherton et al., 1991), child age, and parents' level of depressive symptoms using the short form of the Center for Epidemiological Studies of Depression measure (α = .78) (Andresen et al., 1994). The fifth control variable, parents' self-reported 7-day point prevalence abstinence (0 = smoker, 1 = nonsmoker), was measured at T2. In an earlier publication (Collins et al., 2018), we showed no differences between the two experimental conditions on a wide range of demographic and smoking-related variables (e.g., education, marital status, parent age, cigarettes smoked per day, number of previous quit attempts, received NRT prescription, used any NRT), so no additional control variables were included in the analyses.

2.3. Analytic approach

All analyses were conducted in R version 3.5.2 (R Core Team, 2018). All variables included in the inferential analysis were normally distributed and no outliers were detected. Analyses using the 'LittleMCAR' function in the *BaylorEdPsych* R package (Beaujean, 2012) showed that data were not missing completely at random (MCAR) ($\chi^2(402) = 503.48, p < .001$). Based on this information and patterns of missing data, the data were assumed to be missing at random (MAR). Because some variables in the inferential analysis were missing > 5% data, due to attrition or non-response, we used multiple imputation with chained equations to generate 40 multiply-imputed datasets using the R package *mice* (Van Buuren and Groothuis-Oudshoorn, 2011). Model results were combined across the 40 multiply-imputed datasets using Rubin's rules (Little and Rubin, 2002)

The study sample was recruited from three hospitals, so Intraclass Correlation Coefficients (ICCs) were calculated to identify significant between-group variability on all outcome variables, which would necessitate a multilevel modeling approach. We calculated ICCs for binary outcome variables using the 'iccbin' function from the R package *ICCbin* (Hossain and Chakraborty, 2017) and for continuous outcome variables using the 'ICCbare' function from the R package *ICC* (Wolak et al., 2012). We used a cut-off value of .05 (i.e., ≥5% between-group variability), and no ICCs surpassed this cut-off (all ICCs were ≤ .02), suggesting that multilevel modeling was not necessary (Peugh, 2010).

For the inferential analysis, we fit a multiple mediation model using the 'sem' function in the R package *lavaan* (Rosseel, 2012) to examine the direct and indirect effects of treatment condition on parent bioverified smoking abstinence at T3. Weighted least squares mean and variance adjusted estimation was used to account for ordinal data. Mediators in the model included cessation self-efficacy and smoking urge coping at T2, and counselor support for quitting at T3. Control variables included T1 measures of parent depressive symptoms, nicotine dependence, presence of another household smoker, and child age, as well as T2 smoking abstinence. To estimate the effects of the mediators ('b' paths) in the model, bioverified abstinence at T3 was regressed onto all variables, including control variables. To estimate the effect of intervention on the mediators, the mediators were regressed onto intervention ('a' paths). Finally, the indirect effect was calculated by multiplying each mediator's 'a' and 'b' path. An alpha level of 0.05 was used for all statistical tests.

3. Results

3.1. Descriptive results

The sample size was 327 (n = 163 AAR + counseling; n = 164 AAR + control). Attrition by 12-month follow-up was modest (12%). Adjusting for clustering within clinics, there was no differential between-group attrition. Table 1 presents the zero-order correlations among major study variables. The three mediating variables were positively and significantly intercorrelated, and self-efficacy was also significantly and positively correlated with bioverified abstinence. Of the four T1 control variables, there were a few small intercorrelations

Table 1
Correlations among study variables (N = 327).

Variable	1	2	3	4	5	6	7	8	9
1. Urge coping T2	–								
2. Self-efficacy T2	.339***	–							
3. Counselor support T3	.400***	.228***	–						
4. Depressive symptoms T1	.104	−.005	.027	–					
5. Nicotine dependence T1	.003	−.142*	.110	.142**	–				
6. Household smokers T1	.026	.041	.120*	.072	.043	–			
7. Child age T1	−.010	−.017	−.026	.107	−.011	−.123*	–		
8. Smoking abstinence T2	.140*	.474***	.160**	.012	−.126*	.077	−.034	–	
9. Bioverified abstinence T3	−.044	.274***	−.013	.008	−.153*	.034	.039	.316***	–

Notes: T1 = pre-intervention baseline; T2 = 3-month end-of-treatment follow-up; T3 = 12-month follow-up. Smoking abstinence T2 (0 = smoker; 1 = abstinent). Bioverified abstinence T3 (0 = smoker; 1 = abstinent).
* p < .05.
** p < .01.
*** p < .001.

with the mediators and bioverified smoking abstinence: greater nicotine dependence was inversely related to self-efficacy and bioverified abstinence and was positively correlated with depressive symptoms; presence of other smokers was positively associated with perceived counselor support. The fifth control variable, T2 self-reported abstinence, was significantly and positively correlated with urge coping, self-efficacy, counselor support, nicotine dependence,and T3 bioverified abstinence.

3.2. Mediation analysis

Fig. 1 shows the results of the mediation model (see Table 2 for full model results including covariates). Neither the direct effect of intervention, nor the total effect on parent bioverified abstinence at T3 (12-month follow-up) was significant. As shown in Fig. 1, there was a significant main effects of treatment condition on all three putative mediators: parents receiving AAR + counseling reported higher cessation self-efficacy at T2, higher use of smoking urge coping skills at T2, and greater perceived counselor support for quitting smoking at T3 than parents receiving just AAR + control (all p's < .001). These findings support the predictions in hypothesis 1. Further, Fig. 1 shows a significant main effect of cessation self-efficacy on bioverified abstinence, such that higher levels of self-efficacy at T2 were associated with a higher likelihood of being abstinent at T3. However, the other two putative mediators were not significantly related to smoking abstinence. These findings partially support the predictions in hypothesis 2. Lastly, there was an indirect effect of intervention on bioverified abstinence through cessation self-efficacy (b = .06, p = .002): receiving AAR + counseling was associated with a higher level of self-efficacy at T2 which in turn was associated with abstinence at T3. There were no indirect effects of the other two putative mediators, so hypothesis 3 was partially supported by the data.

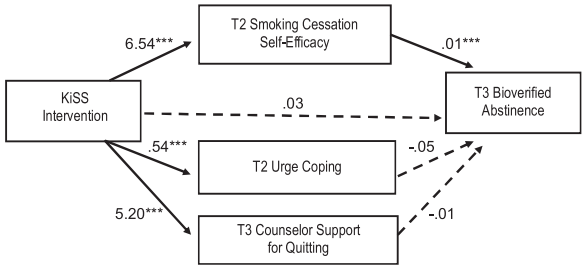

Fig. 1. Hypothesized mediation model testing the direct and indirect effects of KiSS intervention participation on bioverified abstinence at 12-month follow-up (N = 327).
Notes: T2 = 3-month end-of-treatment follow-up; T3 = 12-month follow-up. KiSS intervention: 0 = AAR + control vs. 1 = AAR + counseling. T3 Bioverified abstinence: 0 = smoker; 1 = abstinent. All estimates are unstandardized. Solid lines indicated significant pathways; dashed lines indicate non-significant pathways. *p < .05 **p < .01 ***p < .001.

Table 2
Complete results from path analysis on bioverified abstinence at 12-month follow-up (N = 327).

Path	Estimate (*se*)	*p*-value
Direct effect of intervention on outcome (path c)		
Intervention - > T3 BVA	.03 (*.06*)	.31
Direct effects of intervention on mediators (path a)		
Intervention - > T2 Self-efficacy	6.54 (*1.17*)	< .001*
Intervention - > T2 Urge coping	.54 (*.08*)	< .001*
Intervention - > T3 Counselor support	5.20 (*.65*)	< .001*
Direct effects of mediators on outcome (path b)		
T2 Self-efficacy - > T3 BVA	.01 (*.002*)	< .001*
T2 Urge coping - > T3 BVA	−.05 (*.03*)	.06
T3 Counselor support - > T3 BVA	−.01 (*.004*)	.06
Indirect effects of intervention on outcome (path a x b)		
Intervention * T2 Self-efficacy	.06 (*.02*)	.002*
Intervention * T2 Urge coping	−.03 (*.02*)	.06
Intervention * T3 Counselor support	−.03 (*.02*)	.06
Total effect	.03 (*.04*)	.49
Direct effect of control variables on outcome		
T1 Nicotine dependence - > T3 BVA	−.02 (*.01*)	.03*
T1 Depressive symptoms - > T3 BVA	.001 (*.003*)	.83
T1 Child age - > T3 BVA	.01 (*.04*)	.72
T1 Household smoker - > T3 BVA	.00 (*.001*)	.38
T2 Smoking abstinence - > T3 BVA	.25 (*.07*)	.001*

Notes. T1 = pre-intervention baseline; T2 = 3-month end-of-treatment follow-up; T3 =12-month follow-up. BVA = bioverified 7-day point prevalence abstinence (0 = smoker; 1 = abstinent).
* Denotes significant path.

The mediation analysis also included control variables (not shown in Fig. 1, but see Table 2). The analyses showed a significant effect of two of the five control variables, T1 nicotine dependence (b = -.02, p = .03) and T2 self-reported smoking abstinence (b = .25, p = .001), on T3 bioverified smoking abstinence: persons with higher dependence at baseline were less likely to be abstinent at T3; person who self-reported abstinence at T2 were more likely to be abstinent at T3.

4. Discussion

Findings from the mediation analysis were consistent with the hypothesis of direct effects of the multi-level AAR + counseling intervention on the three putative mediators in a low-income, predominantly African American sample of parental smokers. Relative to persons only receiving AAR + control, persons in the

AAR + counseling group reported greater cessation self-efficacy, greater use of urge coping strategies, and perceived the counselors to be more supportive of their quit efforts. However, self-efficacy was the only significant pathway to 12-month cessation in the model: assignment to the AAR + counseling condition was associated with higher self-efficacy to abstain at 3-month end-of-treatment which, in turn, was associated with a greater likelihood of being abstinent at 12-month follow-up. The time lag between 3-month self-efficacy and the 12-month abstinence, as well as the control for concurrent smoking at end-of-treatment and bioverification of 12-month abstinence, are noteworthy strengths of this finding. Analyses of control variables showed that higher tobacco dependency was inversely related to 12-month abstinence, whereas self-reported abstinence at end-of-treatment was positively associated with bioverified 12-month abstinence.

The evidence suggests that adding intensive telephone counseling to the clinic-level AAR intervention was critical to boosting self-efficacy to quit and this confidence, in turn, sustained long-term smoking abstinence. This is consistent with prior research that suggests higher intervention intensity can improve cessation outcomes (Fiore et al., 2008) and that self-efficacy plays a role in long-term cessation success (Gwaltney et al., 2009; Ockene et al., 2000). A recent study on the effectiveness of the Dutch national quitline, found that self-efficacy to refrain from smoking partially mediated the effects of intervention on self-reported abstinence at 12-month follow-up among parental smokers (Schuck et al., 2014). In the context of the KiSS trial, it is possible that program attempts to shape smoking behavior played a role in increasing self-efficacy. For example, perceived success in achieving short-term goals generated through skills training in the intervention, such as creating household bans on smoking, delaying or reducing amount of cigarette smoking, could enhance self-efficacy. We previously reported that parents in both the AAR + control and AAR + counseling group were able to protect their children from TSE, as evidenced by significant declines in child cotinine from baseline to 12-month follow-up in both groups (Lepore et al., 2018). “Small successes” in changing smoking behaviors could have boosted self-efficacy to quit when combined with the social support (e.g., praise for short-term goal achievement) and behavioral training in smoking urge coping skills in the AAR + counseling condition.

Unexpectedly, level of smoking urge coping at end-of-treatment was unrelated to long-term cessation. These finding partially conflict with those of the Dutch national quitline trial, which found that acceptance of cravings to smoke but not avoidance coping partially mediated the effects of intervention on self-reported abstinence at 12-month follow-up among parental smokers (Schuck et al., 2014). It is possible that differences in our methods of assessing the mediator and outcome accounts for the divergent results. Alternatively, the amount or kind of coping skills training in KiSS may have been insufficient in our target population. We intentionally sampled from communities comprised of low-income and mostly African American residents because these communities have elevated rates of smoking and child TSE. However, these communities also tend to have greater tobacco outlet density and tobacco advertising relative to communities with higher income and lower minority representation (Barbeau et al., 2005; Rodriguez et al., 2013). In our high-risk sample, a combination of many smokers, more tobacco retailers, and more tobacco advertising in the community could have translated into a barrage of daily exposure to smoking cues. Recent research suggests that cigarette smokers who are Black may pay more attention to smoking cues and this can impede cessation attempts, particularly in an environment rich in smoking cues (Robinson et al., 2015). Beyond the cognitive-behavioral strategies implemented in KiSS, novel interventions, such as in situ attentional training using personal mobile devices (Kerst and Waters, 2014), could potentially help to prevent relapse. Booster sessions and reminders about using urge management coping strategies also may be warranted, as well as environmental policies (e.g., smokefree housing, advertising bans) to further support quitters.

Counselor support also did not relate to long-term cessation. This unexpected finding might be attributable to a lack of sensitivity in the support measure. In the overall sample, the mean scale score was near the ceiling: 3.2 out of a maximum possible score of 4.0. In the AAR + control group, the mean score was 2.9, suggesting that control participants were rating the interventionist as being moderately supportive of cessation efforts. However, in the AAR + control condition the telephone counselor only provided education about nutrition and nothing about smoking. It is possible that some AAR + control participants were providing what they thought were the socially desirable responses or were confusing the target of the questions, i.e., confusing the telephone interventionist with the pediatric health care provider who provided messaging and referrals about smoking behaviors to all participants. In future research, it might be advisable to measure perceived counselor support at the end of each counseling session rather than post-treatment.

5. Limitations

Typical of intensive clinic- and community-based smoking interventions (Fiore et al., 2008), the KiSS intervention combined multiple intervention components. Thus, it is not possible to disentangle the specific components of the intervention that most influenced the mediators and outcome. Nor is it possible to identify potential interactive effects of various components, such as the interaction of the clinic-level AAR intervention with the individual-level social support and skills training, because everyone received the AAR. We chose to offer clinic AAR to everyone because it is the emerging standard of care for clinical best practice. Experimental approaches, such as factorial and fractional factorial designs, could be used to isolate intervention components that uniquely and synergistically influence key mediators and smoking outcomes (Baker et al., 2016; Piper et al., 2017; Strecher et al., 2008). In addition, while there was evidence that self-efficacy mediated some of the effects of the multilevel AAR + counseling intervention on long-term cessation, the magnitude of the relation between self-efficacy and long-term smoking abstinence was modest (see Table 1). It is possible that other unmeasured factors were influencing outcomes in the intervention. Another limitation is the small number of follow-up assessments in the trial, which did not allow us to examine whether there might have been some short-term benefits of the mediators on smoking cessation (e.g., 4–6 months out).

6. Conclusions/Implications

Results suggest that all putative treatment pathways were improved more by the multi-level AAR + counseling than the clinic-level AAR + control intervention in a sample of low-income, mostly low-income, African American parental smokers. Further, mediation analyses suggested that the enhancement of self-efficacy by end-of-treatment was linked to long-term cessation, indicating that maintenance of self-efficacy may be key to sustained abstinence. Participants’ level of dependence predicted long-term abstinence independent of intervention condition, with lower quit success among those who were more dependent at baseline. These findings suggest that future interventions with similar high-risk populations need to focus more on addressing dependence. Nicotine replacement therapy and other first-line pharmacological treatments have been shown to effectively address withdrawal symptoms and improve odds of quitting smoking, even among disadvantaged smokers (Hiscock et al., 2012). However, these treatments often are not used or not used effectively among minority and low-income smokers (Burns and Levinson, 2008; Fu et al., 2005; Okuyemi et al., 2010), indicating additional research is needed to understand how to improve uptake and treatment adherence in these special populations. Alternatively, more intensive or highly motivating behavioral interventions, such as contingency management approaches (Donatelle et al., 2000), may prove effective.

Contributors

All authors listed have seen, approved, and contributed to the manuscript. S.J.L. and B.N.C. conceived of the mediation model and overall study design; D.W.S. conducted the primary analyses with input from S.J.L. and B.N.C.; S.J.L. drafted the manuscript, and all authors provided interpretation and editorial comments on the final manuscript. S.J.L. and B.N.C. acquired grant funding for the original trial.

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Declaration of Competing Interest

No conflict declared.

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