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PEDIATRIC ORIGINAL ARTICLE

Unsupportive parenting moderates the effects of family psychosocial intervention on metabolic syndrome in African American youth

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BACKGROUND/OBJECTIVE: Family relationships have been linked to obesity and related disorders in youth, but few studies have provided causal evidence of this association. This study tested the impact of a family psychosocial intervention on components of metabolic syndrome—a condition driven largely by abdominal obesity—in African American youth. In particular, the study tested whether effects were strongest among those who started at highest risk, that is, with high levels of unsupportive parenting at baseline.

SUBJECTS/METHODS: Randomized clinical trial of a community sample of 391 African American youth (mean age = 11.2 years) conducted in 2001–2002, with follow-up metabolic syndrome assessment in 2014–2015. Participants were assigned either to receive a weekly family intervention or to a control group. The primary study outcome was the number of components of metabolic syndrome that were clinically elevated at age 25, including central adiposity, blood pressure, triglycerides, glucose and low high-density lipoproteins. Unsupportive parenting was measured by questionnaires at baseline.

RESULTS: Significant interaction effects were found between group assignment and baseline unsupportive parenting on counts of metabolic syndrome components in youth (beta = − 0.17, $P = 0.03$). Among those who started with higher levels of unsupportive parenting at age 11, participation in the family intervention reduced the number of clinically elevated components of the metabolic syndrome at age 25 relative to the control group. No such effect was seen among those who started with good parenting. Mediation analyses suggested that changes in the psychosocial targets of the parenting intervention partially accounted for the effects amongst those high in unsupportive parenting at baseline (effect size = − 0.350, s.e. = 0.178).

CONCLUSIONS: These findings suggest that efforts to improve family relationships may be able to ameliorate the detrimental effects that harsh and unsupportive parenting have on obesity-related outcomes such as metabolic syndrome in youth.

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INTRODUCTION

Metabolic syndrome is a cluster of risk factors largely driven by abdominal obesity, but also including high blood pressure, impaired glucose control and lipid dysregulation. It is detectable in childhood and adolescence,[1] and is a precursor to a number of diseases later in life, including diabetes, heart disease and stroke.[2] Metabolic syndrome impairs quality of life and generates annual financial costs of nearly $250 billion.[3]

Difficult family relationships have been associated with a variety of poor health outcomes across the lifespan,[4–6] including ones related to metabolic syndrome. For example, adolescent girls who reported unsupportive parenting had greater metabolic risk,[7] and among adolescents with diabetes, lower levels of parental acceptance were associated with poorer metabolic control via poor treatment adherence.[8]

Conversely, close, positive relationships with parents appear to buffer children from the effects of adversity on obesity-related outcomes. For example, maternal responsiveness buffers children who experience life stressors from elevations in allostatic load (a multisystem indicator of physiological risk, including obesity).[9] High levels of maternal warmth in childhood also buffers adults exposed to childhood adversity from metabolic syndrome, allostatic load and inflammatory activity in adulthood.[10–12]

If positive parental relationships that naturally occur can serve this protective role, then interventions designed to improve parenting may also provide similar health benefits, as well as provide causal evidence for the benefits of positive family relationships on obesity-related outcomes. Previous intervention research has documented that a family system intervention improves long-term glucose levels in youth with diabetes who have poor metabolic control.[13,14] Parenting interventions also produce greater declines in children's cortisol levels compared with a control group,[15] and lower levels of inflammation in youth 8 years later compared with a control group.[16]

In the present study, we sought to conduct the first test of which we are aware of the impact of a parenting intervention on the prevention of a clinical outcome related to obesity—metabolic syndrome—in youth. Consistent with previous research that has documented larger intervention effects for those most in need,[17,18] we hypothesized that a family intervention would produce the biggest benefits on metabolic syndrome among those youth who started out at baseline with high levels of unsupportive parenting. Or stated a different way, we

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hypothesized that we would see the expected association between unsupportive parenting and risk for metabolic syndrome among youth in a control group, but that a family intervention would disrupt, and potentially eliminate, this association. We also tested whether changes to the psychosocial targets of our parenting intervention would explain intervention group differences in metabolic syndrome.

SUBJECTS AND METHODS

See Online Supplementary for additional details.

The Strong African American Families (SAAF) program is a family-centered prevention program designed to prevent risk behaviors in youth by enhancing parental warmth, involvement and communication. Details of the original SAAF prevention trial, sample and recruitment are provided in the Online Supplementary and as well, are reported elsewhere.[19–22] Briefly, from 2001 to 2002, when youth were age 11, families were randomly assigned to either the SAAF intervention or a control condition. From 2014 to 2015, when youth were age 25, they were assessed for metabolic syndrome (trial registration number: NCT03139214).

Participants

Participants in the SAAF trial included 667 African American families who resided in nine rural counties in Georgia. One youth from each family (mean age at baseline = 11.2, s.d. = 0.34) and a parent participated. At baseline, 46.3% lived below federal poverty standards. From the original 667, 500 were randomly selected due to funding constraints to participate in a collection of biological data collection at age 19. At age 25, participants were recontacted for another assessment, with 391 agreeing to a blood draw for metabolic syndrome; this constituted the sample in the present study. Written informed consent/assent was obtained from caregivers and youth. Each family was paid $100 for the baseline assessment, and $160 for the assessment (including questionnaires) and blood draw at age 25. The University of Georgia's Institutional Review Board reviewed and approved all study procedures.

Intervention

The SAAF prevention program consisted of seven consecutive, 2 h weekly meetings held at community facilities, with separate parent and youth skill-building curricula and a family curriculum (see refs 19–22 for a complete description, including a summary of efficacy findings). Parents in the intervention condition were taught how to be involved parents, how to engage in high levels of monitoring, how to have effective control and communication, nurturant parenting techniques, the consistent provision of instrumental and emotional support, adaptive racial socialization strategies and methods for communicating with youth about avoidance of sex and alcohol use. Youth learned about the importance of having and abiding by household rules, adaptive behaviors to use when encountering racism, the importance of forming goals for the future and making plans to attain them, and strategies for resisting alcohol use. The control group received three leaflets about child development, stress management and exercise.

Procedures

Questionnaire measures were collected at baseline in participants' homes. Following the assessment, those families randomized to the intervention condition participated in SAAF. When youth were age 25, a field researcher who was also a certified phlebotomist went to each participant's home to collect questionnaire data and to draw a blood sample for metabolic syndrome assessment.

Measures

Unsupportive parenting. Unsupportive parenting was assessed at baseline from questionnaire measures of harsh parenting,[23] lack of supportive communication[24,25] and lack of parental support[26] that were given to youth and parents, and combined into a single composite score. See Online Supplementary for details. Higher values indicated more unsupportive parenting.

Metabolic syndrome. When each youth was 25 years old, a certified phlebotomist went to the participant's home in the morning to draw a fasting blood sample. Blood was drawn into Serum Separator Tubes (Becton-Dickinson, Franklin Lakes, NJ, USA), centrifuged on site and serum was collected and frozen immediately on dry ice. At the end of the study, glucose was measured photometrically using a UV test on a Roche/Hitachi cobas c502 analyzer (Roche Diagnostics, Indianapolis, IN, USA). High-density lipoproteins and triglycerides were measured on a Roche/Hitachi cobas c701 analyzer. Resting blood pressure was monitored three times with a Critikon Dinamap Pro 100 (Critikon; Tampa, FL, USA) while youth sat reading quietly. The field researcher recorded the participant's waist circumference at the midpoint of the upper iliac crest and lower costal margin, at the midaxillary line.

The presence of adult metabolic syndrome was defined by the International Diabetes Federation guidelines.[2] Metabolic syndrome components included the following: (a) central adiposity, defined by ethnic and sex-specific cutoffs for waist circumference (for individuals of African descent, cutoffs are $\geqslant 94$ and $\geqslant 80$ cm for men and women, respectively); (b) high blood pressure (systolic pressure $\geqslant 130$ or diastolic pressure $\geqslant 85$); (c) high triglyceride levels ($\geqslant 150$ mg dl^{-1}); (d) high fasting glucose levels ($\geqslant 100$ mg dl^{-1}); and (e) low high-density lipoprotein levels (< 40 mg dl^{-1} in men and < 50 mg dl^{-1} in women). Two outcomes variables were calculated. Given concerns that have been raised about the classification and diagnosis of metabolic syndrome as a disease,[27,28] our primary outcome was an ordinal measure of the number of metabolic syndrome components for which the participant met clinical cutoff criteria; these could range from 0 to 5 ($M = 1.57$, s.d. = 1.14). As a secondary outcome, we calculated metabolic syndrome diagnosis, which included the presence of central adiposity plus at least two of the four additional components described above.[2] Of 391 participants, 67 (17.1%) were classified as having metabolic syndrome at age 25, a rate comparable to other national studies.[29]

Targets of the parenting intervention. The parenting intervention targeted parental involvement, family rules, parent norms about the avoidance of risky behaviors and parental warmth. These constructs were assessed at baseline, and then at ages 12, 13, 16 and 17 as indicators of long-term changes in the targets of the parenting intervention. Measures were combined into a single score at each time point. Scores across the follow-up periods were averaged. See Online Supplementary for details. Higher values indicated better parenting behaviors.

Covariates. Demographic and potential psychosocial confounders were assessed and statistically controlled in data analyses. These variables included gender and family socioeconomic disadvantage, as well as depressive symptoms, life stress and unhealthy behaviors assessed at age 25. Youth race and age were not included as covariates as they were the same for all participants.

Six dichotomous variables were summed to form a family socioeconomic disadvantage index at baseline. A score of 1 was assigned to each of the following: family poverty based on federal guidelines; primary caregiver unemployment; receipt of temporary assistance for needy families; primary caregiver single parenthood; primary caregiver education level less than high school graduation; and caregiver-reported inadequacy of family income. This procedure has been used in previous studies.[30,31]

Because it is possible that unsupportive parenting might simply be serving as a proxy for other psychosocial variables that affect metabolic syndrome, we assessed a number of potential alternative explanations for results, including depressive symptoms (Center for Epidemiological Studies Depression),[32] life stress[33] and unhealthy behaviors (Youth Risk Behavior Survey)[34] at age 25, and also included these as covariates in analyses. See Online Supplementary for details.

Analytic approach

Analyses were conducted using logistic and linear regression models that included demographic covariates (gender and family socioeconomic status), main effects of unsupportive parenting and intervention status, and a multiplicative interaction term between unsupportive parenting and intervention status predicting counts of metabolic syndrome components (linear regression), as well as metabolic syndrome diagnosis (logistic regression). Intervention status and gender were dummy coded: SAAF participants were coded 1 and control participants were coded 0; male participants were coded 1 and female participants were coded 0. Follow-up analyses were then conducted in which the psychosocial variables of depression, life stress and unhealthy behaviors were added to the models as covariates.

To test whether intervention effects could be due to improvements in the targets of the parenting intervention among those who started out high in unsupportive parenting, we estimated a mediation model with latent difference scores in those who were in the top 35% of scores on the unsupportive parenting measure.[35] We calculated a latent difference score that reflected the degree to which the targets of the parenting intervention improved from before to after the SAAF intervention. Next, we estimated structural coefficients reflecting the association between intervention group and parenting (path A), and parenting and metabolic syndrome components (path B). Then we quantified the indirect or mediating effect of improved parenting as the product of these two regression coefficients (A × B). Nonparametric bootstrapping (1000 times) was used to obtain the bias-corrected and accelerated confidence intervals of the indirect effect.[36] Youth gender, family socioeconimic status, depression, life stress and unhealthy behaviors were controlled in the model.

RESULTS

Preliminary analyses

A two-factor multivariate analysis of variance was conducted to evaluate the equivalence of study variables for participants who did and did not provide blood samples at age 25 by intervention group. No significant main effects or interaction effects emerged for any study variable (Online Supplementary Table S1). Table 1 presents descriptive statistics and correlations among study variables for the control group and the SAAF group.

Primary analyses: counts of metabolic syndrome components

Linear regression analyses revealed a main effect for unsupportive parenting as well as a significant interaction between unsupportive parenting and intervention condition in predicting counts of metabolic syndrome components (Table 2, model 1, $\beta = -0.170$, $P = 0.031$; $\Delta F(1385) = 4.669$, $\Delta R^2 = 0.011$). The main effect was such that more unsupportive parenting at age 11 was associated with a greater number of clinically elevated metabolic syndrome components at age 25. The interaction effect was such that the association between unsupportive parenting and counts of metabolic syndrome components was present in the control group (simple slope = 0.087, s.e. = 0.039, 95% confidence interval (CI; 0.012, 0.165), $P = 0.039$), but not in the intervention group (simple slope = −0.017, s.e. = 0.030, 95% CI (−0.075, 0.042), $P = 0.578$). Another way of testing the effects is to conduct regions of significance testing to determine at what values of unsupportive parenting the intervention and control groups differ. Johnson–Neyman regions of significance testing revealed that among those who started above 1.26 s.d.'s in unsupportive parenting, the intervention group had a smaller average number of metabolic syndrome components on which youth were clinically elevated relative to the control group. The effect size was −0.359 (s.e. = 0.182), meaning that for those high in unsupportive parenting, the intervention group had a 0.36 lower metabolic syndrome component score than the control group.

When we included the psychosocial variables of depressive symptoms, life stress and unhealthy behaviors as covariates in the analyses, we found that both the main effect of unsupportive parenting and the interaction effect between unsupportive parenting and intervention condition remained significant in predicting counts of metabolic syndrome components (Table 2, model 2, $\beta = -0.170$, $P = 0.031$; $\Delta F(1382) = 4.697$, $\Delta R^2 = 0.011$).

To depict the interaction graphically, we plotted estimated counts of metabolic syndrome components at low (2 s.d.'s below the mean; −2 s.d.) and high (3 s.d.'s above the mean; +3 s.d.) levels of unsupportive parenting according to intervention status. The results are illustrated in Figure 1. Unsupportive parenting when youth were 11 was significantly associated with counts of metabolic syndrome components at age 25 among those randomly assigned to the control group (simple slope = 0.096, s.e. = 0.039, 95% CI (0.019, 0.173), $P = 0.014$). However, unsupportive parenting was not associated with metabolic syndrome among youth randomly assigned to the intervention group (simple slope = −0.010, s.e. = 0.030, 95% CI (−0.069, 0.050), $P = 0.751$). Or stated another way, regions of significance testing revealed that among those who started above 1.20 s.d.'s in unsupportive parenting, the intervention group had a smaller average number of metabolic syndrome components on which youth were clinically elevated relative to the control group. The effect size was −0.350 (s.e. = 0.178), meaning that for those high in unsupportive parenting, the intervention group had a 0.35 lower metabolic syndrome component score than the control group (Figure 1).

Secondary analyses: metabolic syndrome diagnosis

Logistic regression analyses revealed a marginally significant interaction between unsupportive parenting and intervention condition in predicting metabolic syndrome diagnosis (Table 3, model 1, odds ratio = 0.807, Wald (1) = 3.007, $P = 0.083$). No main effects of either unsupportive parenting or intervention condition emerged. When the psychosocial variables of depressive symptoms, life stress and unhealthy behaviors were included in the model as covariates, the interaction effect remained marginally significant (Table 3, model 2, odds ratio = 0.812, Wald (1) = 2.814, $P = 0.093$).

Table 1. Descriptive statistics and correlations among study variables for control and SAAF groups

Variables	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*
M	0.19	1.55	0.39	2.53	0.14	1.14	12.52	0.04
S.d.	0.39	1.13	0.49	1.42	2.39	1.63	7.86	3.42
1. Metabolic syndrome diagnosis status (age 25)	—	0.752***	−0.179**	0.137*	−0.025	−0.028	−0.003	0.015
2. Counts of metabolic syndrome components (age 25)	0.731***	—	−0.358***	0.146*	−0.017	−0.076	−0.032	−0.109
3. Gender, male	−0.036	−0.167*	—	−0.075	−0.004	0.014	−0.010	0.022
4. Family SES disadvantage (age 11)	0.168*	0.195*	0.065	—	0.115	0.019	0.027	−0.066
5. Unsupportive parenting (age 11)	0.150	0.181*	0.052	0.166*	—	0.085	0.129	0.113
6. Life stress (age 25)	−0.040	−0.026	0.043	0.176*	0.055	—	0.152*	−0.029
7. Depressive symptoms (age 25)	0.013	0.062	−0.147	0.142	0.102	0.300***	—	0.164*
8. Unhealthy behaviors (age 25)	0.115	−0.064	0.104	0.001	0.132	0.023	0.073	—
M	0.15	1.53	0.42	2.24	−0.20	1.14	12.09	0.06
S.d.	0.36	1.15	0.50	1.47	2.19	1.40	7.78	3.62

Abbreviations: SAAF, Strong African American Families; SES, socioeconomic status. Upper diagonal: descriptive statistics and correlations for SAAF group ($n = 228$); lower diagonal: descriptive statistics and correlations for control group ($n = 163$). *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

Table 2. Unsupportive parenting and intervention status as predictors of counts of metabolic syndrome components ($N = 391$)

	Counts of metabolic syndrome components (age 25)					
	Model 1			*Model 2*		
	B	*S.e.*	β	B	*S.e.*	β
Gender, male	−0.643	0.111	−0.279***	−0.631	0.111	−0.273***
Family SES disadvantage (age 11)	0.120	0.038	0.154**	0.121	0.038	0.154**
Unsupportive parenting (age 11)	0.089	0.039	0.181*	0.096	0.039	0.196*
Intervention (SAAF)	−0.053	0.111	−0.023	−0.055	0.111	−0.024
Unsupportive parenting × SAAF	−0.105	0.049	−0.170*	−0.105	0.049	−0.170*
Life stress (age 25)	—	—	—	−0.047	0.036	−0.064
Depressive symptoms (age 25)	—	—	—	−0.001	0.007	−0.006
Unhealthy behaviors (age 25)	—	—	—	−0.025	0.016	−0.077

Abbreviations: SAAF, Strong African American Families; SES, socioeconomic status. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

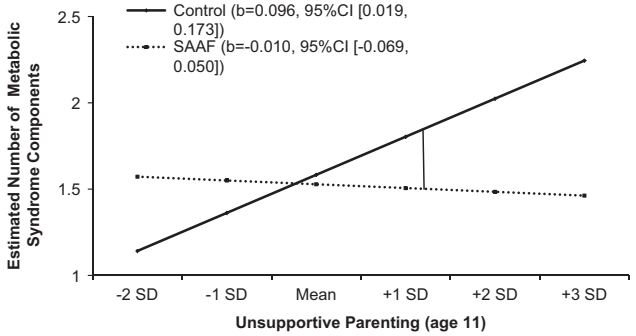

Figure 1. The effect of unsupportive parenting at age 11 on youths' counts of metabolic syndrome components at age 25 by intervention status. Numbers in parentheses refer to simple slopes for the control group and the intervention (SAAF) group. Regions of significance analyses indicate that the differences between the intervention and control groups are significant above 1.2 s.d.'s of unsupportive parenting.

Mediation analyses

We tested whether improvements in the psychosocial targets of the parenting intervention could account for the intervention group differences in metabolic syndrome components, among those who started out high in unsupportive parenting (top 35%, $n = 137$). Figure 2 depicts the results of the mediation analyses. These results suggest that the reduced number of metabolic syndrome components in the intervention group is partially attributable to improvements in parenting (among those who start out high in unsupportive parenting). The positive coefficient for path A indicates that being in the intervention group was associated with statistically significant long-term improvements in parenting. The negative coefficient for path B indicates that the more parenting improved, the fewer metabolic syndrome components youth were elevated on. Multiplying these coefficients yielded an indirect effect of −0.051 with a bootstrapped 95% CI of −0.150, −0.003. Thus, the indirect pathway from intervention to improved parenting to fewer metabolic syndrome components was statistically significant. Nonetheless, intervention group status remained associated with metabolic syndrome components, even after accounting for parenting (path C′), thus suggesting that there are other additional pathways through which the intervention works. Overall model fit was good, with $\chi^2(4) = 6.430$, $P = 0.169$, comparative fit index = 0.955 and root mean square error of approximation = 0.067 (95% CI = 0, 0.157).

DISCUSSION

These results support the hypothesis that participation in a family-centered intervention program designed for African American families can ameliorate the association between unsupportive parenting and counts of metabolic syndrome components—a cluster of risk factors, including abdominal obesity—in young adults. Among youth in the control group, higher levels of unsupportive parenting at age 11 prospectively predicted a greater number of clinically elevated metabolic syndrome components at age 25. In contrast, among youth who participated in the SAAF family intervention, there was no relationship between unsupportive parenting assessed pre-intervention (at age 11) and components of metabolic syndrome at age 25, suggesting that the intervention mitigated the effects that unsupportive parenting can have on youth metabolic syndrome. Or stated another way, the intervention and control groups differed on metabolic syndrome components only at higher levels (>1.2 s.d.) of unsupportive parenting. Effects on metabolic syndrome were not due to potential alternative explanations such as youth depressive symptoms, unhealthy behaviors or the occurrence of life stressors. Mediation analyses were consistent with an explanation in which the SAAF intervention reduced metabolic syndrome components in part by improving the targets of the parenting intervention (for example, parental involvement, establishment of family rules and positive parent–child interactions) among those who started out high in unsupportive parenting. These findings are also noteworthy because the study was conducted with a sample of African Americans from low-income backgrounds in the rural southern United States, a region with some of the highest rates of metabolic syndrome in the country.[37]

Prevention researchers have previously demonstrated a form of moderation in which intervention effects are stronger for individuals who are at highest risk at program entry.[20] This is consistent with the patterns from the present study in that the SAAF intervention appeared to have the strongest effects on metabolic syndrome components for those who scored highest on unsupportive parenting at baseline. Furthermore, these results are consistent with previous research that has documented that parenting interventions eliminated the effects of unsupportive parenting on youth catecholamine levels[17] and on youth telomere length.[18] These patterns may also explain why there were no overall main effects of intervention group status on metabolic syndrome. It may be because an effective parenting intervention operates by reducing the negative health impacts primarily in higher-risk groups where there are difficult family environments to begin with.

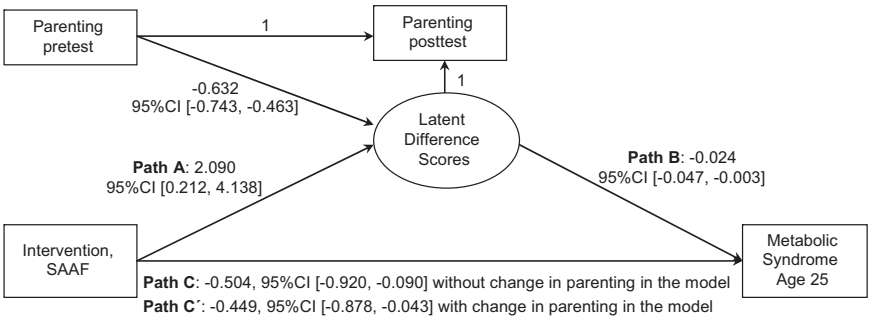

Figure 2. A mediation model of intervention status, changes in the targets of the parenting intervention and metabolic syndrome at age 25 among those who were high in unsupportive parenting (top 35%, $N = 137$, 86 intervention, 51 control) at baseline. Family socioeconomic status, youth gender, youth depressive symptoms, life stressors and unhealthy behaviors at age 25 were controlled (not shown). Pretest represents baseline values, and post test represents values averaged post intervention across ages 12, 13, 16 and 17. Unstandardized coefficients with bias-corrected and accelerated 95% CIs are presented. Indirect effect: −0.051, 95% CI (−0.150, −0.003).

Table 3. Unsupportive parenting and intervention status as predictors of metabolic syndrome diagnosis ($N = 391$)

	Metabolic syndrome diagnosis (age 25)					
	Model 1			*Model 2*		
	B	*S.e.*	*Exp*(B)	B	*S.e.*	*Exp*(B)
Gender, male	−0.726	0.302	0.484*	−0.744	0.305	0.475*
Family SES disadvantage (age 11)	0.267	0.097	1.307**	0.286	0.098	1.330**
Unsupportive parenting (age 11)	0.171	0.100	1.186	0.160	0.100	1.174
Intervention (SAAF)	0.232	0.290	1.262	0.230	0.291	1.258
Unsupportive parenting × SAAF	−0.214	0.123	0.807^{+}	−0.208	0.124	0.812^{+}
Life stress (age 25)	—	—	—	−0.005	0.018	0.995
Depressive symptoms (age 25)	—	—	—	−0.063	0.101	0.939
Unhealthy behaviors (age 25)	—	—	—	0.057	0.041	1.058

Abbreviations: SAAF, Strong African American Families; SES, socioeconomic status. $^{+}P < 0.10$, $*P < 0.05$, $**P < 0.01$.

Interestingly, SAAF was originally designed to prevent and reduce rates of youth substance use by enhancing protective caregiving practices and youth self-regulatory competence.[19] Evaluations of the SAAF program confirmed its efficacy in preventing the initiation and escalation of alcohol and drug use and conduct problems across several years,[20,38,39] enhancing protective parenting practices[22] and increasing youth self-regulatory capabilities.[19,22] Additional analyses revealed that the SAAF program was efficacious when the primary caregiver presented clinical levels of depressive symptoms[40] and when the primary caregiver reported economic hardship.[41] More recently, interest has turned to understanding the ways in which these types of family interventions might also influence health.[13,14] We previously documented that the SAAF intervention reduced levels of pro-inflammatory cytokines in youth at age 19 compared with a control group.[16] We also previously documented intervention × parenting interactions on youth catecholamine levels at age 20. Among youth in the control group, unsupportive parenting at age 11 predicted elevated epinephrine and norepinephrine at age 20, whereas intervention eliminated the association between parenting and catecholamines.[17] In the present study, we extended these findings by re-assessing youth at age 25, this time for an outcome directly relevant to diabetes—metabolic syndrome, and found a parallel interaction effect.

It is possible that harsh and unsupportive parenting triggers hormonal and inflammatory responses that, accumulating over time, have implications for a number of obesity-related chronic diseases.[4,42,43] Low-grade inflammation and exposure to high levels of hormones are known to facilitate the development of the components of metabolic syndrome.[42,44,45] In this study, we considered the possibility that SAAF effects on reductions in catecholamines and cytokines served as mediators for intervention effects among participants who were high in unsupportive parenting. In separate models, we included inflammatory markers and hormone output as mediators of the SAAF × parenting interaction on metabolic syndrome. However, the mediating pathways did not reach statistical significance (data not presented). The relatively low percentage of participants with metabolic syndrome at this young age probably contributed to a lack of power to detect these effects. These processes will continue to be examined in subsequent waves of data collection, when the number of participants displaying metabolic syndrome is expected to increase.

Psychologically, the mediation analyses suggested that more warm and nurturant parents may be better able to establish rules and routines that help their children learn emotion regulation strategies for coping with daily life stressors, which in turn may reduce the physiological effects of stress.[6,46,47] Nurturant parents may also be more involved and provide emotional and instrumental social support to youth that mitigate the effects of life stressors on their physiological systems.[10,48] If these adaptive psychological strategies and supports are maintained over the long term (as suggested by the long-term follow-up measures of the targets of the parenting intervention), this may lead to impacts over time such as the prevention of obesity and other related diseases that are at least in part behaviorally determined.

Limitations of the present study include the fact that the original trial was not designed with metabolic syndrome as an end

point, and hence we do not have baseline measures of metabolic syndrome. In addition, the sample is one of rural, African American families, and it is unknown whether these findings would generalize to urban African American families or to members of other racial or ethnic groups. Finally, longer-term follow-up assessments as youth progress into middle and older adulthood would allow us to track the development of other obesity-related clinical outcomes such as types 2 diabetes.

In sum, the present study documented that a family intervention ameliorated the associations of unsupportive parenting with counts of metabolic syndrome components in youth. These findings suggest that teaching families effective parenting strategies may be one way to combat the detrimental effects that harsh and unsupportive parenting have been found to have on obesity-related outcomes across the lifespan.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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