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Early Childhood Family Intervention and Long-term Obesity Prevention Among High-risk Minority Youth

AUTHORS: Laurie Miller Brotman, PhD,[a] Spring Dawson-McClure, PhD,[a] Keng-Yen Huang, PhD,[a] Rachelle Theise, PsyD,[a] Dimitra Kamboukos, PhD,[a] Jing Wang, MA,[a] Eva Petkova, PhD,[a] and Gbenga Ogedegbe, MD[b]

[a]*New York University Child Study Center, Department of Child and Adolescent Psychiatry, and* [b]*Division of General Internal Medicine, Department of Medicine, New York University School of Medicine, New York, New York*

WHAT'S KNOWN ON THIS SUBJECT: The evidence base for obesity prevention is extremely limited. Although minority youth are at higher risk of obesity, and early childhood is a critical period for prevention, only 1 program has demonstrated sustained effects on obesity in young minority children.

WHAT THIS STUDY ADDS: Among youth at high risk for obesity based on income, minority status, and child behavior problems, early intervention that promotes effective parenting led to meaningful differences in obesity in preadolescence. Early family intervention is an innovative and promising approach.

abstract

OBJECTIVES: To test the hypothesis that family intervention to promote effective parenting in early childhood affects obesity in preadolescence.

METHODS: Participants were 186 minority youth at risk for behavior problems who enrolled in long-term follow-up studies after random assignment to family intervention or control condition at age 4. Follow-up Study 1 included 40 girls at familial risk for behavior problems; Follow-up Study 2 included 146 boys and girls at risk for behavior problems based on teacher ratings. Family intervention aimed to promote effective parenting and prevent behavior problems during early childhood; it did not focus on physical health. BMI and health behaviors were measured an average of 5 years after intervention in Study 1 and 3 years after intervention in Study 2.

RESULTS: Youth randomized to intervention had significantly lower BMI at follow-up relative to controls (Study 1 P = .05; Study 2 P = .006). Clinical impact is evidenced by lower rates of obesity (BMI ≥95th percentile) among intervention girls and boys relative to controls (Study 2: 24% vs 54%, P = .002). There were significant intervention-control group differences on physical and sedentary activity, blood pressure, and diet.

CONCLUSIONS: Two long-term follow-up studies of randomized trials show that relative to controls, youth at risk for behavior problems who received family intervention at age 4 had lower BMI and improved health behaviors as they approached adolescence. Efforts to promote effective parenting and prevent behavior problems early in life may contribute to the reduction of obesity and health disparities. *Pediatrics* 2012;129:1–8

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Childhood obesity is a costly public health problem.[1,2] The growing epidemic of obesity is associated with increasing prevalence of hypertension and diabetes,[2–5] particularly in low-income and minority children.[6] Rates of overweight (BMI ≥ 85th percentile) have doubled among 2- to 5-year-olds over the past 3 decades; overweight preschool-aged children are 5 times more likely to be obese (BMI ≥ 95th percentile) at age 12 than nonoverweight children.[7] Early childhood has been identified as a critical period for obesity prevention based on developmental patterns related to "adiposity rebound,"[8–11] stabilization of health behaviors that contribute to obesity,[12–14] and the growing influence of environmental factors (eg, portion size, prompts from parents) on children's regulation of eating in response to satiety cues.[15]

Evidence from a randomized controlled trial (RCT) of nutrition education and physical activity suggests the potential of obesity prevention during early childhood. Specifically, intervention resulted in a significant reduction in BMI z scores over 2 years among low-income African American children;[16] there was, however, no effect on BMI z scores in a second cohort of Latino children from similar backgrounds.[17] No other interventions have demonstrated sustained obesity prevention effects in young minority children. The limited impact of existing interventions may be, in part, because of the failure to promote key aspects of the early family environment that influence children's health behaviors and risk for obesity.

Two fundamental aspects of effective parenting influence a broad range of developmental outcomes including obesity: responsiveness (ie, warmth, sensitivity, involvement) and control (ie, expectations for self-control by child, parental discipline).[18–25] Preschool-aged children of parents who exhibited low responsiveness and high control were 5 times more likely to be overweight than children of parents with appropriate levels of responsiveness and control.[23]

Furthermore, children with behavior problems, such as impulsivity, or a family history of behavior problems are at increased risk for poor health, including obesity.[26–28] A 21-year prospective study of more than 2200 children demonstrated that behavior problems at age 5 more than doubled the odds of adult obesity, independent of childhood overweight, diet, family meals, television, and exercise.[29] Given that ineffective parenting increases the risk of both child behavior problems and obesity, strengthening parenting for children at risk for behavior problems may result in lower rates of obesity.

We conducted 2 RCTs of family interventions aimed at promoting effective parenting for low-income, minority children at risk for behavior problems, and documented improvements in parental responsiveness and control.[30–35] We expected that study participants were also at risk for obesity and hypothesized that family intervention in early childhood would mitigate this risk, resulting in lower rates of obesity. The current study takes advantage of 2 RCTs with prospective follow-up studies of behavioral outcomes to examine intervention effects on obesity and related health behaviors as high-risk youth approach adolescence.[19]

METHODS

Overview of Study Design

We report findings from long-term follow-up assessments of children enrolled in 1 of 2 RCTs ($n = 99$; $n = 496$) aimed at the promotion of effective parenting and child behavioral regulation. Neither trial targeted obesity, but the study populations are at high risk for obesity based on income, minority status, and behavior problems. Behavioral family intervention took place during early childhood (ages 3–5 years)

and included a series of weekly 2-hour parent and child groups over a 6-month period. The interventions did not address nutrition, activity, or weight. Descriptions of the interventions and positive effects on parenting (eg, responsiveness, control) and child behavior (eg, aggression, social competence, stress response) have been reported.[30–35]

Follow-up Study 1 Overview and Participants

The RCT was designed to evaluate the efficacy of family intervention among children at very high risk for behavior problems.[36] The trial enrolled 99 younger siblings of adjudicated youth (48% of eligible) in 5 cohorts from 1997 to 2001; after enrollment of each cohort, families were randomized to intervention (an adapted version of the *Incredible Years Series;* 22 2-hour parent and child groups[30,37]) and to a no-intervention control condition. Children were followed prospectively over a 6-year period, from ages 4 to 10 years. During the trial, children were assessed 4 times, every 8 months; during the follow-up phase, children were evaluated 4 times, every 12 months, with the last evaluation occurring between 2003 and 2007. For the current follow-up study, the health assessment was conducted in 2006 for all participants, an average of 5 years after intervention (ranging from 4 to 8 years for the last and first cohorts, respectively). Families who had participated in the trial were approached for follow-up health assessments by telephone and informed consent and assent were obtained at the assessment. The follow-up study was approved by the University Institutional Review Board.

Although we attempted to evaluate boys and girls at the follow-up health assessment, there was an underrepresentation of boys from the control condition, with none from the earlier cohorts. This resulted in narrow overlap in the distributions of age (intervention: range = 9.58–13.25, mean = 11.02, SD = 1.24;

control: range = 7.75–11.58, mean = 9.52, SD = 1.25), such that control boys were significantly younger than intervention boys (1.5 years; $t = -2.73$, $P = .01$). The follow-up sample of boys did not permit unbiased estimates of intervention effects and therefore was not included in the current study. The Study 1 sample included 40 girls.

Follow-up Study 2 Overview and Participants

This cluster (school) RCT was designed to evaluate the efficacy of family intervention (and a parallel school component). Ten elementary schools were randomly assigned to intervention (*ParentCorps*[34]) or a no-intervention control condition in 2005. Seventy-seven percent of children who were in Pre-Kindergarten enrolled in the RCT in 4 cohorts (from 2005 to 2008). Children were followed prospectively over a 4-year period, from ages 4 to 8 years. During the trial, children were assessed at the beginning and end of Pre-Kindergarten and Kindergarten (4 times); during follow-up, children were evaluated yearly at the end of first and second grade. For the current follow-up study, the health assessment was conducted in second grade (~age 8), which occurred in 2009 and 2010 for the first 2 cohorts (of 4; $n = 496$ of 1050), 3 years after intervention. Informed consent for the health assessment in second grade was obtained from parents in writing when children were in first grade. The follow-up study was approved by the institutional review boards of the University and City Department of Education. To evaluate health outcomes in an at-risk subgroup parallel to Study 1, we consider health assessment data from children with elevated behavior problems at age 4 ($n = 146$; ~25% with T scores >50 on the aggression subscale of the Behavioral Assessment Scale for Children[38] or any physical aggression on the New York Teacher Rating Scale[39]. Prospective longitudinal analysis of children in the control condition support our

focus on this subgroup of children; 54% of children with elevated behavior problems at age 4 were obese (BMI >95th percentile) at age 8, relative to only 19% of children without elevated behavior problems at age 4.

Obesity: BMI and Body Size Ratings

The primary outcome measure was BMI calculated from height (by using a stadiometer) and weight (by using an electronic scale) assessed during a physical exam by trained medical students or research assistants masked to intervention assignment. BMI was standardized by age and sex according to Centers for Disease Control and Prevention growth charts and BMI z scores were analyzed. A dichotomous indicator of obesity (BMI ≥95th percentile) was used to evaluate clinical impact.

The measurement of height and weight and the calculation of BMI z scores were introduced in both follow-up studies at the final health assessment. To address the absence of a measure of overweight at baseline or during early follow-up assessments, we rated body size from archived videotapes of child behavior in Study 1, and during school assessments of child behavior in Study 2. The systematic and prospective rating of body size in Study 2 was based on the emerging findings from Study 1 demonstrating the strong predictive relation between body size ratings and BMI in preadolescence (noted later in this article).

Research assistants rated body size on a 9-point standardized Figure Rating Scale[40] from videotapes at baseline and 3 follow-up time points in Study 1 and during in-school assessments at baseline and 3 follow-up time points in Study 2. By using the Body Size Rating system (BSR),[41] raters were trained to achieve >90% agreement with "gold standard" body size ratings from videotapes of ethnically diverse children.[34]

Inter-rater reliability and stability of BSR were high. In both studies, body size ratings were also obtained at the follow-up when height and weight were assessed to allow for thorough psychometric evaluation. Concurrent BSR (by raters unaware of height, weight, or BMI) was highly related to BMI at follow-up. Importantly, BSR in early childhood was predictive of BMI at follow-up (r values > 0.60). Sensitivity and specificity of BSR were maximized at BSR ≥6 for obesity (BMI ≥95th percentile).

Health Behaviors: Activity and Nutrition

In both studies, parents reported their children's sedentary activity on 2 items assessing the amount of time children spent watching television and other screen time (eg, video games, computer or Internet) on a typical day. In Study 1, youth reported on their physical activity in the past week on the Block Kids Physical Activity Screener,[42] which assesses the frequency of 9 moderate-to-vigorous activities. In Study 2, parents reported the frequency with which their children engaged in 5 types of moderate-to-vigorous physical activities in the past week. Nutritional intake was measured in Study 1 only. Youth reported on their nutritional intake in the past week on the Block Kids Food Frequency Questionnaire,[43] which includes 77 food items based on the NHANES 1999–2000 dietary recall data and questions about portion size with pictures to enhance accuracy. This measure yields summary scores for total calories and the percentage of calories from carbohydrates, protein, and fat.

Blood Pressure

Blood pressure was measured in Study 1 only. Trained staff used a validated oscillometric automated blood pressure monitor (Welch Allyn, Skaneateles Falls, NY) following standard guidelines.[44] Three consecutive measurements were

taken, an average was calculated separately for systolic and diastolic blood pressure, and values were standardized by age, sex, and height.[45]

Statistical Analyses

Multiple Imputation for Missing Follow-up Data

Multiple imputation for BMI and BSR was carried out for youth with BSR measured at previous follow-up times; imputation was warranted given high serial autocorrelations and strong associations between BMI and BSR. Fifty imputations were generated, separately for the control and intervention conditions, and by gender.[46] Health behavior measures were not imputed because these behaviors were not assessed at previous times and none of the measures available at earlier times, such as BSR, were associated with them. Imputation for BMI and BSR was done with Proc MI and analyzed with Proc MIANALYZE in SAS (SAS Institute, Cary, NC).

Although girls who completed the Study 1 assessment ($n = 29$) were younger than those who did not ($t = -2.11$, $P = .04$), attrition did not result in differences between intervention and control groups with regard to age, ethnicity, or BSR (P values = .15–.87), allowing for unbiased estimates of intervention effects. Imputation yielded a final sample of 40 girls (77% of the randomized sample); there were no differences between these 40 cases and the other 12 cases for which data were not imputed (since recent BSR data were not available). For Study 2 girls and boys, there were no differences between those who completed the follow-up ($n = 60$) and those who did not with respect to age, ethnicity, and BSR (girls: P values = .12–.51; boys: P values = .29–.69). Study 2 assessments were conducted with 98% of students who remained in the 10 study schools in second grade. Imputation resulted in a final Study 2 sample of 66 girls and 80 boys (100% of the first 2

cohorts of the randomized sample with behavior problems at baseline).

Approach to Analyses of Group Differences

Intervention-control condition differences on BMI and BSR were assessed with linear and logistic regression analyses by using intent-to-treat principles. Given previous findings of differential intervention effects on obesity for boys and girls in the literature,[47–52] intervention-by-gender interactions were assessed (in Study 2). To evaluate group differences in the trajectory of BSR, the generalized estimating equations approach was used[53,54] and postintervention outcomes were regressed on intervention condition, age, and the intervention-by-age interaction term to test whether the magnitude of the intervention effect changes over time. In Study 1, blood pressure and nutrition indices were evaluated as multivariate constructs, by using a multivariate analysis of variance–type mixed effects model[55] with outcomes modeled as a function of domain, intervention, and the domain-by-intervention interaction; significant interactions were followed by estimation of domain-specific intervention effects. For Study 2, the effect of clustering of children within schools was taken into account by allowing for correlation between outcomes of children from the same school. All models were fit by using Proc GENMOD in SAS.

RESULTS

Sample Characteristics and Baseline Equivalence

Mean age at baseline was 4.12 (SD = 0.69) for Study 1 and 4.38 (SD = 0.31) for Study 2; mean age at follow-up was 10.88 years (SD = 1.51) for Study 1 and 7.77 years (SD = 0.31) for Study 2. In Study 1, 55% of participants were black, 32.5% were Latino, and 12.5% were of other/mixed ethnicity. In Study 2, 87% were black, 8.9% were Latino, and 4.1%

were of other/mixed ethnicity. In Study 1, the average family size was 6.2 (SD = 2.4); 72.5% of parents did not have more than a high school education; 57.5% had annual household incomes below $15 000; and 30% were single parents. In Study 2, the average family size was 4.5 (SD = 1.8); 48% of parents did not have more than high school education; 16.4% had annual household incomes below $15 000 (44% lived below federal poverty guidelines); and 51% were single parents. The intervention and control conditions were not significantly different on any of these characteristics (all P values > .05). Because there was a pattern (albeit not significant) for baseline BSR to be lower in the intervention condition relative to the control condition in both trials, intervention differences on BMI and BSR at follow-up are presented with and without adjustment for baseline BSR.

Intervention Effects on BMI and BSR

In both Study 1 ($n = 40$) and Study 2 ($n = 146$), BMI z and BSR at follow-up were significantly lower in the intervention condition relative to the control condition (Table 1). In Study 1, although the intervention effect was no longer statistically significant after adjusting for baseline BSR, the magnitude of the group difference remained clinically important. In Study 2, even after adjustment for baseline BSR, the intervention-control difference remained both statistically significant and clinically important (24% vs 54% obese, $P = .002$). Significant and robust intervention effects on obesity (BMI ≥95th percentile) were found for both genders, with substantially lower odds of obesity for both intervention girls and boys relative to controls (girls: 19% vs 54% obese, odds ratio = 0.20, confidence interval (0.05, 0.81), $P = .02$; boys: 24% vs 50% obese, odds ratio = 0.32, confidence interval (0.11, 0.92), $P = .03$). Baseline BSR did not significantly moderate the intervention effect on

TABLE 1 Intervention Effects on BMI and BSR

	Study 1 (*n* = 40)						Study 2 (*n* = 146)					
	Mean (SD) or %[b]		Model-based inference[a]				Mean (SD) or %[b]		Model-based inference[a]			
			Not adjusted for baseline BSR		Adjusted for baseline BSR				Not adjusted for baseline BSR		Adjusted for baseline BSR	
	Intrv N = 19	Ctrl N = 21	Effect[c] (95%CI)	*P*	Effect[a] (95%CI)	*P*	Intrv N = 106	Ctrl N = 40	Effect[c] (95%CI)	*P*	Effect[a] (95%CI)	*P*
BMI *z*	0.61 (1.23)	1.34 (1.12)	−0.79 (−1.57, 0.003)	.05	−0.47 (−1.22, 0.28)	.22	0.66 (0.98)	1.17 (1.04)	−0.53 (−0.91, −0.16)	.006	−0.31 (−0.62, −0.0007)	.05
BSR	4.68 (2.02)	6.05 (1.64)	−1.42 (−2.64, −0.21)	.02	−0.95 (−2.13, 0.23)	.11	4.55 (0.97)	5.30 (1.12)	−0.75 (−1.16, −.34)	<.001	−0.56 (−0.93, −0.19)	.003
BMI ≥95	21%	39%	0.40 (0.08, 2.01)	.27	0.74 (0.10, 5.26)	.76	24%	54%	0.27 (0.11, 0.63)	.002	0.34 (0.11, 1.04)	.06
BSR ≥6	22%	54%	0.24 (0.05, 1.15)	.07	0.35 (0.06, 1.88)	.22	15%	49%	0.18 (0.07, 0.45)	<.001	0.19 (0.07, 0.53)	.002

Baseline values for BSR were as follows: Study 1: intervention M = 4.83 (0.74), control M = 5.35 (1.20); Study 2: intervention M = 4.37 (1.23), control M = 4.86 (0.85). Baseline values for BSR ≥6 were as follows: Study 1: 16.7% of intervention, 38.1% of control; Study 2: 13.0% of intervention, 13.2% of control. P values for all contrasts >.05. CI, confidence interval; Ctrl, control; Intrv, intervention.

[a] Analyses in Study 1 controlled for pubertal development and child age at follow-up (due to the broad range) and analyses in Study 2 took into account the hierarchical nature of the data (ie, participants were nested within schools). In Study 2, the intervention-by-gender interaction was nonsignificant for BMI and BSR (*P*s > .50).

[b] Means and SDs for the continuous variables (BMI and BSR); percent obese for the indicator variables (BMI ≥95% and BSR ≥6).
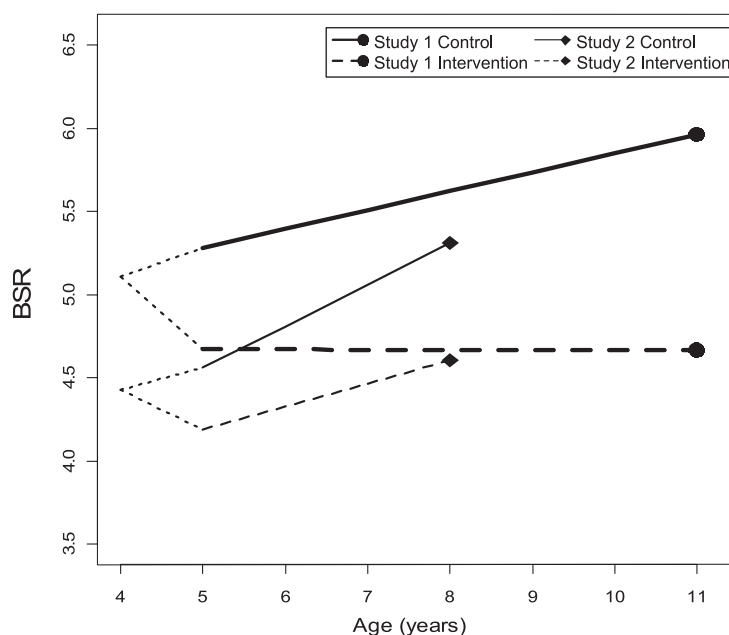
[c] For the continuous variables, intervention effect is the difference between the means for the 2 groups (intervention minus control); for the indicator variables, intervention effect is the odds ratio for obesity (intervention versus control).

BMI *z* or BMI ≥95th percentile at follow-up.

Analysis of BSR developmental trajectories from early childhood through the follow-up period demonstrates highly similar patterns across samples and for boys and girls. Group differences emerged early (by age 6 in both samples) and were maintained over time (Fig 1). Baseline BSR did not moderate the intervention effect on the BSR trajectory in Study 1, but it was a significant moderator in Study 2 (intervention-by-age-by-BSR interaction Estimate (SE) = −0.21 [0.09], *P* = .02), such that group differences were greater over time for children with higher BSR at baseline.

Intervention Effects on Health Behaviors and Blood Pressure

In both trials, there were significant intervention-control group differences in health behaviors in all domains assessed at follow-up (Table 2). In Study 1, relative to controls, intervention girls spent more time engaged in moderate-to-vigorous physical activity and less "screen time." Similarly, in Study 2 intervention girls engaged in less "screen time" and intervention boys were more physically active than same gender peers in the control group. Although there were no intervention-control group differences in total calories consumed (*z* = 1.46, *P* = .14; measured only in Study 1), there were differences in the proportion of calories consumed from

FIGURE 1

BSR trajectories from early childhood through preadolescence. In Study 1, the intervention effect on body size was observable at age 6 (*d* = 0.71; difference intervention − control at age 6 [SE] = −0.73 [0.43], *P* = .09) and was maintained to age 11, with a trend for the effect to increase over time (*d* = 0.93; intervention-by-age interaction [SE] = −0.11 [06], *P* = .07). In Study 2, the intervention effect was also observable at age 6 (*d* = 0.41; difference intervention − control at age 6 = −0.48 [0.15], *P* = .001), was maintained over time (intervention-by-age interaction [SE] = −0.11 [0.13], *P* = .38), and was not moderated by gender (intervention-by-gender interaction [SE] = −0.54 [0.44], *P* = .22), with positive intervention effects for both girls and boys.

TABLE 2 Intervention Effects on Health Behaviors and Blood Pressure

	Study 1				Study 2							
	Girls				Girls				Boys			
	Intervention	Control	z	P	Intervention	Control	z	P	Intervention	Control	z	P
Sedentary activity, mean (SD)	2.69 (1.22)	4.00 (1.64)	−2.39	.02	1.76 (0.66)	2.17 (0.93)	−1.69	0.09	1.56 (0.64)	1.78 (0.80)	−0.26	.79
Physical activity, mean (SD)	2.18 (1.48)	1.04 (0.87)	2.72	.01	2.41 (0.89)	2.13 (0.78)	0.80	0.42	2.95 (0.96)	1.90 (1.27)	1.88	.06
Nutritional intake[a]												
% from carbohydrates	52.49	62.02	−4.15	<.001	$\chi^2_{Intervention*Domain}$=11.44, P = .003							
% from protein	13.31	10.94	3.22	.001								
% from fat	36.46	29.07	3.46	.001								
Blood pressure[a]												
Systolic z, mean (SD)	−0.73 (0.85)	0.29 (0.80)	−2.69	.01	$\chi^2_{Intervention*Domain}$=4.75, P = .03							
Diastolic z, mean (SD)	−0.10 (0.61)	0.33 (0.50)	−1.71	.09								

Nonimputed data were analyzed; Study 1 N = 24–27 girls; Study 2 N = 55–58 (Girls N = 29–31; Boys N = 26–27), controlling for child age at follow-up in Study 1 and adjusting for the hierarchical nature of the data in Study 2.
[a] Not assessed in Study 2.

carbohydrates, protein, and fat; compared with controls, intervention girls consumed fewer of their calories from carbohydrates. Finally, relative to control girls, intervention girls had significantly lower blood pressure (measured only in Study 1).

DISCUSSION

Among youth at high risk for obesity based on income, minority status, and child behavior problems, early intervention that promotes effective parenting (eg, responsiveness, control) led to meaningful differences in BMI more than 3 years later in preadolescence. Intervention-control group differences on BSR trajectories in both high-risk samples and differences on health behaviors that are putative causes of obesity (eg, diet, physical and sedentary activity) corroborate this pattern of results.

Overall, findings of long-term group differences on obesity, health behaviors and blood pressure reinforce earlier demonstrations of the broad range of benefits from behavioral family intervention for children with or at risk for behavior problems[30–35,56–60] and are consistent with theoretical models that implicate parenting in the promotion of healthy physical development. Reductions in BMI among overweight Australian children whose parents participated in a treatment program to improve parenting (eg, responsiveness) also provide support for this approach.[52] Obesity interventions that are narrowly focused on eating and activity without changing fundamental aspects of the early family environment are likely to be insufficient, especially for children at high risk.[17,61,62]

The demonstration of obesity prevention effects in 2 independent high-risk samples is highly encouraging but findings must be interpreted in light of limitations. Most importantly, the RCTs were not designed for the purpose of evaluating physical health outcomes, rates of attrition were relatively high, and baseline measures of BMI and health behaviors were not available in either study. However, we were able to use BSR measures that were available at baseline and throughout the follow-up studies to address many of these limitations. BSR measures in early childhood were strong predictors of BMI at follow-up; BSR was used to establish equivalence on obesity at baseline and allowed for imputation of BMI data for the original randomized samples (100% in Study 2). We conservatively adjusted for baseline BSR when examining intervention-control group differences on BMI at follow-up, and in the larger Study 2 sample, the difference on BMI remained significant and meaningful. Because health behaviors were not predicted by BSR, we were unable to adjust for baseline and it remains possible that the differences in health behaviors observed at follow-up were not a result of intervention.

This study evaluated long-term obesity prevention in 2 samples of children at high risk for behavior problems, and suggests several avenues for further inquiry. Future research should examine the level of risk for obesity conferred by child behavior problems and associated parenting practices, and explore shared risk factors and common pathways for behavioral health and physical health outcomes. Behavioral family intervention may be a promising strategy for obesity prevention for broadly defined populations, or it may be appropriate only for children with familial or individual risk factors for behavior problems. Findings from this study should not be generalized to other populations without additional investigations.

CONCLUSIONS

The public health impact of successful obesity prevention among high-risk minority youth would be great given the disproportionately high rates of obesity-related morbidity and mortality

in minority populations. Early intervention that promotes effective parenting in children at high risk is an innovative and promising approach to obesity prevention that deserves further inquiry.

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