Physical Activity Buffers the Effects of Chronic Stress on Adiposity in Youth

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ABSTRACT

Background: *The moderating effect of physical activity (PA) on relations between chronic stress and adiposity is unknown in youth.* ***Purpose:*** *The objective is to assess the mediating effect of PA on relations between stress and adiposity in youth.* ***Methods:*** *Participants were 303 youths (47% Black, 53% White, 50% male, M age = 16.6 years). The Adolescent Resource Challenge Scale assessed personal stress, whereas median rent or mortgage in the neighborhood reflected community stress. Body mass index (BMI) and sum of skinfolds reflected general adiposity, and waist circumference measured central adiposity. Days per week performing PA sufficient to work up a sweat measured PA.* ***Results:*** *Hierarchical regressions predicted each adiposity measure adjusting for age, race, gender, family socioeconomic status, and parental smoking. Independent contributions of personal stress, but not community stress, were found on BMI and sum of skinfolds. A similar model showed that both personal and community stress predicted waist circumference. PA was independently, inversely associated with sum of skinfolds but not BMI or waist circumference. The interaction between PA and personal stress predicted all three adiposity measures. The interaction of PA with community stress predicted BMI.* ***Conclusions:*** *PA appears to buffer the effects of chronic stress on adiposity, providing evidence that PA is a protective factor for health.*

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INTRODUCTION

Over the past 2 decades, American youth became twice as likely to be overweight as increasing trends of sedentariness and consumption of energy-dense foods were observed (1–3). In addition to deleterious effects on physical health, overweight in adolescents also could have long-term social and economic impacts (4).

Evidence from clinical, epidemiological, and basic research accumulated over the past 20 years has clearly demonstrated physical activity (PA) of a moderate intensity can enhance overall health in youth and that increased intensity confers greater benefits (5). The benefits associated with moderate to vigorous PA include reduced risk for cardiovascular (CV) disease and improved glucose metabolism, strength, CV fitness, self-esteem, and body image (5).

In a cross-sectional survey of 9,957 adolescents in Grades 7, 9, and 11, overweight adolescents reported engaging in significantly more unhealthy behaviors (e.g., unhealthy eating habits, watching more TV, and exercising less often) and experiencing greater levels of emotional distress (6). Other researchers also found similar relations between being overweight and problems in unhealthy diet and PA habits, social relations, school experiences, and psychological well-being (7,8).

Stress has been tied to obesity in children and adolescents. Psychological stress has been shown to precede weight gain (9,10). Living in a neighborhood at the lower end of the socioeconomic continuum can cause greater stress due to the increased burdens of coping with limited resources and negative life events for a prolonged period of time. Limited access to health-related resources such as health care and preventive programs can affect residents' health and health behavior (11). Individuals living in a disadvantaged community may have limited access to opportunities for PA and healthy eating (12,13).

The mechanisms through which stress may influence obesity have not been fully elucidated. Behaviorally, stress is thought to contribute to obesity through lifestyle choices while under stress. It has been demonstrated that women tend to prefer high fat or sweet foods when moderately stressed (14) and that the administration of cortisol in healthy men dramatically increased food intake (15). A review of the stress-induced eating literature found a consistent link between stress and overeating in adults, especially in those who are restrained eaters (16). This has been demonstrated recently in children (17) in response to laboratory stressors. The effects of stress on adiposity via such dietary behaviors may be buffered by PA increasing energy expenditure or diminishing negative affect or both. Steptoe, Wardle, Pollard, Canaan, and Davies (18) found that college students in a high stress condition decreased PA compared to nonstressed controls, which lends further support for a direct effect of stress causing inactivity.

Health behaviors tend to cluster, such that individuals who habitually exercise and eat a healthy diet are likely to be nonsmokers and to drink alcohol in moderation; such people tend to be in higher socioeconomic strata (19,20). Exposure to secondary smoke has been associated with health indicators in children, including physical inactivity and higher body mass index (BMI) (21).

Compared to youth with greater resources, risk-moderating effects are most apparent for youth with few resources. A resource is "protective" when it only operates in the presence of a risk and mitigates its negative effect (22). On the other hand, a

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resource can also have a direct effect on the outcome independent of the risk factor. In this case, when the risk level is held constant statistically, high levels of resource lead to more positive outcome (22). Recent work has examined the potential impacts of protective factors on well-being and life adjustment for individuals at risk (23). Individual resources such as social support, self-esteem, and coping skills have been shown to buffer the effects of stress on social and health outcomes in youth who have experienced major life events (24).

There is evidence indicating regular PA can potentially diminish physiological and psychological responses to stress and reduce negative affective states such as depression and anxiety (25). Carmack and colleagues (1999) examined the role of leisure PA and aerobic fitness as potential buffers between minor stressors and resulting distress in a sample of college students. Self-reported leisure PA was found to buffer the effects of minor stress on physical symptoms and anxiety, whereas aerobic fitness showed no moderating effect. It lent support to the distraction hypothesis that PA provides a suspension of thought concerning one's stressors, thus allowing for a brief break from life's daily strain (26). No reports of the potential effect of PA moderating the effect of stress on obesity are available.

This study examined whether PA acts as a protective factor, moderating the effect of stress on adiposity in youth. Following the conceptual model of protective resources (22,23), we tested associations among personal and community stress and adiposity measures after adjusting for demographic and family factors, whether PA was an independent predictor of adiposity, and whether PA moderated the stress–adiposity relation in youth.

METHODS

Participants

Participants for this study were 303 youths (53% White, 47% Black, 50% male, 12–24 years of age). See Table 1 for descriptive information. Participants were in a longitudinal study evaluating the development of CV disease risk factors. Participants were recruited based on a verified family history of CV disease. Details of the study design have been described elsewhere (27). Data collected from 535 participants from 1997 to 1998 as part of an annual laboratory visit were used in this study. Only participants with complete information on the study measures were included in the data analysis. Incomplete data occurred mainly as a result of missing information on personal stress and community stress measures. Adiposity measures were available on all participants. There were no significant differences on the three adiposity measures between participants with and without complete information. The study was approved by the Human Assurance Committee at the Medical College of Georgia.

Measures

Adiposity measures. Anthropometric measures were taken from participants in an examination room during an annual visit. Trained research assistants measured height (via stadiometer, to the nearest 0.1 cm), weight (via a Healthometer scale [Healthometer, Inc., Bridgeview, IL], to the nearest 0.1 kg), and waist circumference (narrowest point at waist, to the nearest 0.1 cm). Tricep, subscapula, and suprailiac skinfold measurements were recorded from the right side of the body (to the nearest 0.1 cm) (28). Sum of skinfolds from the three sites was used as an indicator of general adiposity. BMI was calculated as weight (kilograms)/height (m^2) and served as an indicator of general adiposity. Waist circumference was used as an indicator of central adiposity. Use of three adiposity measures allowed us to evaluate the robustness of relations with stress and PA (29).

Personal and community stress. Personal stress was assessed by the Adolescent Resource Challenge Scale (ARCS). The ARCS is a 35-item scale that assesses whether the respondent has experienced various stressful events during the past 12 months (C. K. Ewart, personal communication). The scale lists stressful life experiences from the neighborhood (e.g., people fighting on my street), family (e.g., family members abused alcohol or drugs), and peer (e.g., my friend used drugs) environment. This scale was chosen because it (a) was designed specifically for use with adolescent populations and (b) assesses the accumulation of life events occurring in multiple environmental settings rather than single discrete stressors, a strategy recommended in stress assessment (30). Total ARCS scores have acceptable test–retest reliability over 4 years (r = .49; C. K. Ewart, personal communication). Construct validity of the scale has been demonstrated through significant correlations between total scores and measures of risk-taking behavior (r = .37), depression (r = .36), general negative affect (r = .44), reports of illness and injury (r = .40), and negative correlations with self-esteem and social support (r = –.25; C. K. Ewart, personal communication). Ewart and Suchday recently found that depression, anger, hostility, and low self-esteem were positively related to stress that was measured using an instrument based on the ARCS (31). Cronbach's alpha was 0.74 for the scale in this study. No significant differences were observed in total score by race, sex, or by race or sex subgroups in this sample (all ps > .05).

Community stress was measured by median monthly rent or mortgage in the community at the 1990 U.S. census block level. This measure has been found to reflect social processes and their associations with individual health status (12,32). For this study, median rent or mortgage was inverted (by subtracting an individual's neighborhood rent or mortgage value from the maximum value for the sample) to produce the community stress variable so that higher values would indicate higher community stress levels.

Physical activity. PA was assessed by the self-reported number of days per week, in or outside of school, during which PA that was sufficient to "work up a sweat" was performed. This measure has been validated previously with more comprehensive self-report measures (e.g., sweat episodes/week and kilocalories/week from the Harvard Activity Survey, r = .62) and with physical measures of sedentary behavior (e.g., higher skinfold measures and higher resting heart rate associated with lower PA) (33,34). A recent study found that it was significantly correlated with energy expenditure (r = .57, p < .05) as measured by the doubly labeled water method (35).

Although more comprehensive measures of PA are available, the sweat index has been frequently used in epidemiological research, where time limitations do not allow the use of time-consuming inventories, such as the 7-day Physical Activity Recall (36). This measure was considered to be the best simple measure of PA at the time these data were collected. Regarding the potential impact of warmer weather on the sweat index, we have previously examined the difference on the sweat index reporting based on the time of year when the participants were tested (unpublished). We did not find the time of year to be associated with a higher level of sweat index reporting.

Demographic and familial factors. Participants self-reported their sex and date of birth. Racial background was based on parental classification as European American or African American according to criteria described previously (37). Parental (father or mother) smoking status was established based on parents' self-report. Fifty percent of participants had at least one parent who reported smoking. Family socioeconomic status (SES) was measured based on father's education and occupation using the Hollingshead Social Status Index (38). Scores ranged from 8 to 66, with higher scores representing greater SES. The sample was approximately normally distributed across social strata with 77% in Strata II–IV, indicating a diverse, middle-class sample.

STATISTICAL ANALYSIS

SPSS 11.0 was used to perform all statistical analysis, with $\alpha = .05$. Distributions of all variables were inspected for normality, and a square root transformation was applied to the index of PA for analysis. Means are reported in untransformed units. Where unequal variances were detected for group comparisons, the Satterthwaite t′ was used with adjusted degrees of freedom. Because exposure to secondary smoke was associated with higher waist circumference, 83 versus 77 cm, $t'(279) = -3.4$, $p < .01$; BMI, 26 versus 23 kg/m^2, $t'(264) = -3.6$, $p < .001$; community stress, 435 versus 380, $t'(301) = -3.9$, $p < .001$; and lower family SES, 31 versus 41, $t'(296) = 6.7$, $p < .001$, the parental smoking variable was included as a control variable. Hierarchical regression was performed separately on BMI, sum of skinfolds, and waist circumference, with age, race, gender, family SES, and parental smoking entered at the first step. Personal and community stress were entered in the second step as risk factors. PA was entered as a protective factor in the third step. Finally, all two-way interaction terms involving PA and the measures of personal stress and community stress were entered at the fourth step. Based on the conceptual model of risk and protective factors, it was expected that there would be a significant increase in R^2 when stress variables and interaction terms were entered in Steps 2 and 4, showing that stress did have a direct effect on adiposity and that PA moderated that effect. A significant increase in R^2 in Step 3 would indicate a significant direct effect of PA on adiposity after controlling the effects of stress. However, this effect need not be significant to show that PA buffers the effect of stress on adiposity.

RESULTS

Table 1 displays the means and standard deviations of the measures used in the study. Correlations of adiposity measures with stress and PA variables were consistent with previous findings but with small magnitudes (see Table 2). Race, sex, and parental smoking status were tested for group differences on all variables. Differences were found for parental smoking, as described previously, and for race and sex. Black adolescents were more likely than White adolescents to have at least one parent who smokes, $\chi^2(1, N = 303) = 12.3$, $p < .001$; greater BMI, 26 versus 24 kg/m^2, $t'(274) = -2.9$, $p < .01$; lower SES, 32 versus 40, $t'(300) = 5.7$, $p < .001$; to be older, 17.0 versus 16.2 years, $t'(301) = -3.1$, $p < .01$; and to experience more community stress, 447 versus 373, $t'(301) = -5.3$, $p < .001$. Girls were more likely than boys to have higher sum of skinfolds, 65 versus 42 mm, $t'(297) = -6.3$, $p < .001$, and BMI, 26 versus 24 kg/m^2, $t'(261) = -2.3$, $p < .05$, and to report less PA, 2.5 versus 3.7 days/week, $t'(292) = 4.6$, $p < .001$.

Hierarchical Regression

Hierarchical regressions were performed for each of the dependent variables: BMI, sum of skinfolds, and waist circumference. Results of these regressions can be seen in Table 3. Figures 1 to 4 are the scatter plots of significant interactions between personal or community stress and PA on adiposity. Regression lines were plotted separately for participants with different levels of PA (0–2 times/week, 3–4 times/week, and 5 times or more/week) to illustrate the nature of the interaction.

General Adiposity: BMI

In the first step, age, race, gender, SES, and parental smoking were entered predicting BMI. In the second step, personal stress, but not community stress, significantly explained variation in BMI ($\Delta R^2 = .03$, $p < .05$). At the third step, PA was not associated with BMI ($\Delta R^2 = .01$, $p > .1$). The interactions of PA with both personal and community stress significantly predicted BMI in the fourth step ($\Delta R^2 = .03$, $p < .05$).

TABLE 1
Descriptive Statistics

	M	*SD*
Age (years)	16.6	2.3
Family socioeconomic status (father's Hollingshead score)	36	14
Personal stress (Adolescent Resource Challenge Scale score)	15	4.8
Community stress (inverted median monthly rent or mortgage)	408	128
Physical activity (sufficient to "work up a sweat," days/week)	3.1	2.1
Sum of skinfolds (cm)	53	34
Waist circumference (cm)	80	16
Body mass index (kg/m^2)	25	6.7

TABLE 2
Correlations Between Variables

	Race	*Sex*	*Parental Smoking*	*SS*	*WC*	*BMI*	*Age*	*SES*	*Personal Stress*	*Comm. Stress*	*Physical Activity*
1. Race[a]	—	–.03	.20**	.04	.10	.17**	.18**	–.31**	–.09	.29**	–.10
2. Sex[a]		—	.00	.34**	–.03	.13*	.00	–.02	.03	.03	–.26**
3. Parental smoking[a]			—	.11	.19**	.20**	.17**	–.36**	.04	.22**	–.02
4. SS[b]				—	.83**	.87**	.18**	–.15**	.15**	.13*	–.25**
5. WC[b]					—	.93**	.21**	–.20**	.15**	.21**	–.12*
6. BMI[b]						—	.24**	–.23**	.15**	.19**	–.15**
7. Age[b]							—	–.08	.04	.11	–.14*
8. SES[b]								—	–.04	–.33**	.12*
9. Personal stress[b]									—	.01	.08
10. Comm. stress[b]										—	–.09
11. Physical activity[b]											—

Note. Race: 1 = White, 2 = Black. Sex: 1 = Male, 2 = Female. Parental smoking: 0 = no, 1 = yes. SS = sum of skinfolds; WC = waist circumference; BMI = body mass index; SES = family socioeconomic status; comm. stress = community stress.
[a]Point biserial correlations. [b]Pearson.
*$p < .05$. **$p < .01$.

TABLE 3
Hierarchical Regression Models Assessing Mediating Effects of PA on Stress–Adiposity Relations

	Waist Circumference			*Body Mass Index*			*Sum of Skin Folds*		
	B	β	*Sig.*	*B*	β	*Sig.*	*B*	β	*Sig.*
Step 1[a]	$R^2 = .09$, $F(5, 297) = 5.7$, $p < .001$			$R^2 = .13$, $F(5, 297) = 9.0$, $p < .001$			$R^2 = .17$, $F(5, 297) = 12.0$, $p < .001$		
Step 2	$\Delta R^2 = .03$, $F(2, 295) = 5.7$, $p < .01$			$\Delta R^2 = .03$, $F(2, 295) = 4.5$, $p < .05$			$\Delta R^2 = .02$, $F(2, 295) = 3.4$, $p < .05$		
Personal stress	.43	.13	< .05	.19	.14	< .05	.86	.12	< .05
Community stress	.02	.14	< .05	.01	.09	> .10	.02	.07	> .20
Step 3	$\Delta R^2 = .01$, $F(1, 294) = 3.0$, $p > .05$			$\Delta R^2 = .01$, $F(1, 294) = 2.1$, $p > .10$			$\Delta R^2 = .02$, $F(1, 294) = 7.0$, $p < .01$		
Days of PA	–2.0	–.10	> .05	–.69	–.08	> .10	–6.1	–.15	< .01
Step 4	$\Delta R^2 = .02$, $F(2, 292) = 3.9$, $p < .05$			$\Delta R^2 = .03$, $F(2, 292) = 4.5$, $p < .05$			$\Delta R^2 = .02$, $F(2, 292) = 3.6$, $p < .05$		
PS × PA interaction	–.50	–.49	< .05	–.21	–.49	< .05	–1.0	–.46	< .05
CS × PA interaction	–.02	–.39	> .05	–.01	–.43	< .05	–.03	–.34	> .05
Model	$R^2 = .15$, $F(10, 292) = 5.3$, $p < .001$			$R^2 = .19$, $F(10, 292) = 6.8$, $p < .001$			$R^2 = .22$, $F(10, 292) = 8.5$, $p < .001$		

Note. PA = physical activity; PS = personal stress; CS = community stress.
[a]Age, race, gender, parental smoking, and family socioeconomic status were entered in first step, $p < .01$ for all three models.

General Adiposity: Sum of Skinfolds

Analogous analyses yielded similar results. In the first step, age, race, gender, SES, and parental smoking were entered predicting sum of skinfolds ($R^2 = .17$, $p < .001$). In the second step, personal stress, but not community stress, explained variation in sum of skinfolds ($\Delta R^2 = .02$, $p < .05$). At the third step, PA was associated with lower sum of skinfolds ($\Delta R^2 = .02$, $p < .01$). The interaction of PA with personal stress significantly predicted sum of skinfolds ($\Delta R^2 = .02$, $p < .05$), whereas the community stress and PA interaction only marginally predicted sum of skinfolds.

Central Adiposity: Waist Circumference

In the first step, age, race, gender, SES, and parental smoking were entered predicting waist circumference ($R^2 = .09$, $p < .001$). At Step 2, personal stress and community stress each independently explained variation in waist circumference ($\Delta R^2 = .03$, $p < .01$). PA was marginally associated with waist circumference at Step 3 ($\Delta R^2 = .01$, $p < .1$). Finally, the interaction of PA with personal stress predicted waist circumference, and the interaction between community stress and PA marginally predicted waist circumference ($\Delta R^2 = .02$, $p < .05$).

DISCUSSION

Major findings of this study indicate that personal stress was consistently associated with adiposity measures, whereas community stress did not show consistent relations with adiposity measures after controlling for family SES. The moderating effect of PA on the stress–adiposity relation was consistently significant in regression models when we examined the Personal Stress × PA interaction. The moderating effect of PA on the community stress–BMI relation was statistically significant,

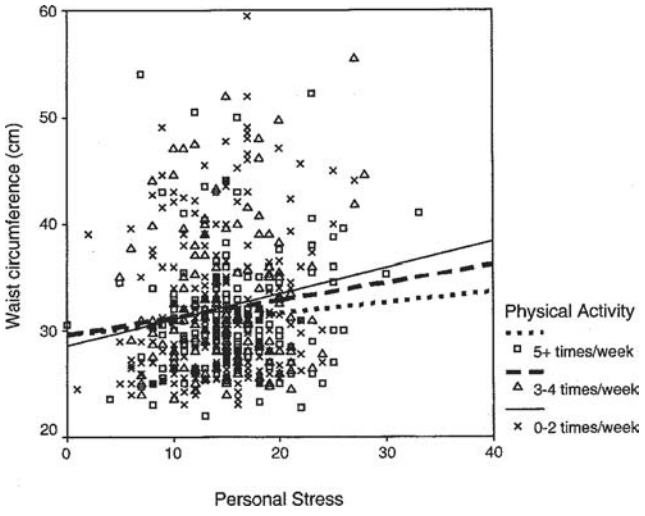

FIGURE 1 Moderating effect of physical activity on the relation between personal stress and waist circumference.

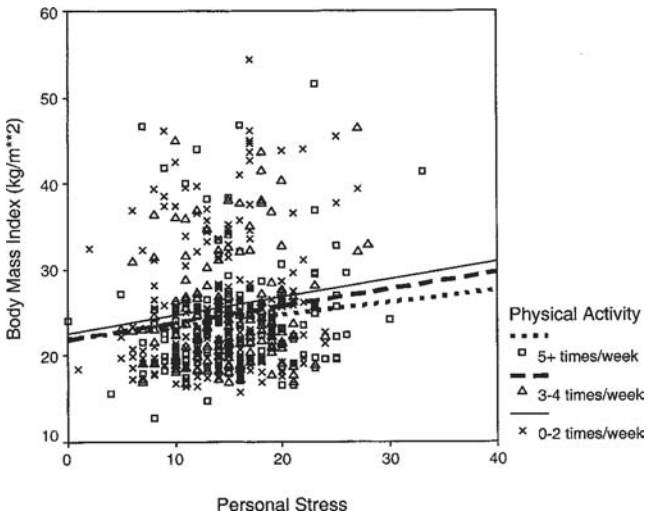

FIGURE 2 Moderating effect of physical activity on the relation between personal stress and body mass index.

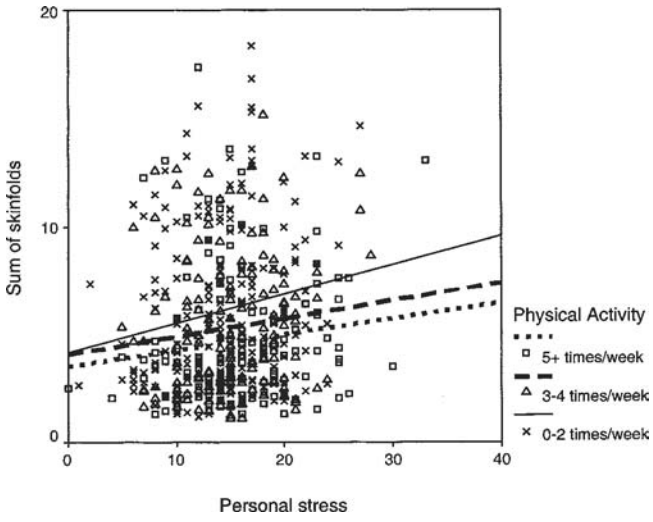

FIGURE 3 Moderating effect of physical activity on the relation between personal stress and sum of skinfolds.

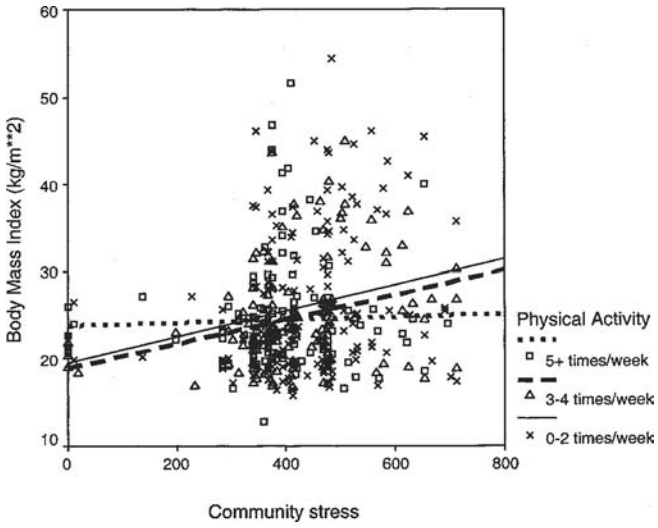

FIGURE 4 Moderating effect of physical activity on the relation between community stress and body mass index.

but only marginal for waist circumference and sum of skinfolds. Figures 1 to 4 provide a clear graphical illustration of the buffering effect of PA on adiposity for those with a high level of stress.

Although the three adiposity indicators are highly correlated (r = .83–.93), use of each is a strength of the study because of recent research findings concerning the differing effects of regional adiposity distribution on disease risk in adults (29,39–41). Although stress is reportedly associated with general adiposity (9,10), it has been hypothesized to be more strongly related to central adiposity (42). The consistency of results across the three models using three adiposity measures (R^2 = .15–.22), adjusting for some powerful predictors of obesity, lends credence to the results. Magnitudes of the correlations between stress and three adiposity measures were similar and thus did not lend support to the suggested unique relation between stress and central adiposity for the youth we studied.

These findings clearly show that personal stress, as measured by experiences of major life events, is associated with increased levels of general and central adiposity in youth. The relation remained even after adjusting for family SES and other background variables. To our knowledge, no study has demonstrated the link between adiposity and personal stress measured by experience of stressful life events in youth. Intervention studies have shown that stress reduction has weight-control benefits in adults (43). These findings are consistent with the hypothesized mechanisms of PA influencing lifestyle choices through improved mood, or directly impacting energy balance by maintaining or increasing energy expenditure (17,18).

The relations between community stress and adiposity measures, however, were not as consistent. A significant relation was found with waist circumference, but no relations were found with general adiposity measures. We assessed the relation of community stress with adiposity variables after adjusting for family SES (which predicted all three adiposity measures) and other demographic variables, resulting in a stringent test of the contribution of community stress. In this way, findings on community stress reflect resources available in the community, apart from assets of the family, which can influence an individual's experience of stress and health outcomes. It is possible that this measure does not capture important aspects of community stress, but is rather a simple SES measure at the neighborhood level. Previous studies have found that community stress is strongly correlated with neighborhood-level SES (24,31). Other means to assess community stress (e.g., community collective efficacy, informant survey, crime statistics) should be explored in future studies.

The robust associations found in this sample between secondary smoke exposure and general and central adiposity, race, and socioeconomic factors are intriguing. These associations may be due to direct physiological effects of secondhand smoke and the association of parental smoking with demographic factors (44). Furthermore, the clustering of lifestyle risk factors (e.g., smoking, excessive alcohol intake, low fruit and vegetable consumption) has been reported in adults (45), and clustering of physiological risk factors for CV disease (e.g., high BMI, lipid disorder, hypertension) have also been reported in adults (46) and more recently in children (47). This clustering of risk factors might further account for the relation between secondary smoke exposure and general and central adiposity, race, and socioeconomic factors in this study. We suggest that as a powerful health risk factor, smoking and secondary smoke exposure be further explored in studies of stress, health behaviors, and health outcomes.

The generalizability of the study may be limited due to several methodological issues. First, because this was an exploratory study, we conducted a cross-sectional analysis to examine the hypothesized moderation effect of PA. This design limits conclusions about cause–effect relations. It is imperative to conduct longitudinal or experimental studies to validate our findings. Second, participants in this study had a family history of early myocardial infarction or hypertension or both, and the average BMI was high. However, these characteristics may be typical for youth in the Southeast region of the United States, also known as the "stroke belt" (48,49), which has historically been characterized by high prevalence of obesity and CV diseases (50). The use of a single-item PA measure is a limitation, although others have found it to be a valid index of activity level in population studies. In this study, its correlations with adiposity measures were significant but small (ranging from –.12 to –.24). Except for the sum of skinfolds model, it was not significantly associated with adiposity after controlling for stress and background variables. Furthermore, the single-item sweat index does not provide information of actual amount of PA and makes it difficult to relate findings from this study to other population studies that used more comprehensive self-report measures such as the 7-day Physical Activity Recall (36). However, sweat episodes may be more memorable and better recalled by children than the number of minutes spent in PA. Future prospective studies using objective measurements of PA and diet will improve understanding of the influence of PA and stress on adiposity.

In sum, the most important finding of this study—PA appeared to buffer the effects of stress on adiposity—provides evidence that PA is a protective resource factor against obesity for youth under stress. Our findings support promotion of regular PA and stress reduction in youth to prevent obesity, a growing epidemic in the United States.

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