预防干预可以减少儿时家庭风险与随后的交感神经系统活动水平关系的证据。研究目的：本文采用准实验设计，提出两个假设，其中一个假设是预防干预可能会降低交感神经系统活动水平。方法：对学龄前的非洲裔美国儿童，通过随机分配到强族裔非洲裔美国家庭（SAAF）项目或对照条件。当这些儿童11岁时（M = 11.2岁），主要照顾者提供了关于他们自己的压力性症状和自尊，以及他们接收到的不支持的养育。当孩子们20岁时（M = 20.1岁），指示器交感神经系统活动，交感胺失活和去甲肾上腺素，被从其夜尿中检测。结果：压力的育儿环境被预测为之后的交感神经系统活动水平的有通过。结论：预防项目可以诱导交感胺失活水平是重要从理论和实际关注的，因为这证实了家庭风险因素对交感神经系统活动的升高是不一的。
Youths assigned randomly to the control condition who, at age 11, were living with a primary caregiver who reported psychological dysfunction—defined as relatively high levels of depressive symptoms and low self-esteem—or provided nonsupportive parenting would evidence relatively high catecholamine levels at age 20. This study was also designed to test the hypothesis that, among youths exposed to nonsupportive parenting or low caregiver functioning, random assignment to the Strong African American Families (SAAF) program (Brody et al., 2004) would be associated with relatively low catecholamine levels at age 20. SAAF is a family-centered prevention program designed to prevent risk behaviors by enhancing parental warmth, involvement, and communication. In observational studies, these practices have buffered youths from the effects of stress on biological regulatory systems (Chen & Grange, 2012). Evaluations of SAAF confirmed its efficacy in enhancing these protective parenting practices (Brody, Kogan, & Grange, 2012).

**Method**

**Overview**

A pretest assessment, which included the collection of data on low parental support and parental psychological dysfunction, was conducted when the youths were 11 years of age (M = 11.2, SD = 0.34). Participation in the SAAF trial began when the youths were 11.5 years of age. The data on the catecholamines epinephrine and norepinephrine were obtained from overnight urine voids when the youths were 20 years of age (M = 20.4, SD = 0.61).

**Participants**

Participants in the SAAF trial included 667 African American families who resided in rural Georgia. At pretest, although the primary caregivers in the sample worked an average of 39.4 hr per week, 46.3% lived below federal poverty standards; the proportion was 53.7% at the age-20 assessment. From a sample of 561 at the age-18 data collection (a retention rate of 84%), 500 emerging adults were selected randomly to take part in the assessment of catecholamines at age 20; the selection of a random subsample was made necessary by financial constraints associated with the costs of assaying the catecholamines. Of this subsample, 476 agreed to participate; they constituted the sample in the present study. At age 11, 277 of these participants had been assigned randomly to the SAAF condition and 199 had been assigned randomly to the control condition. The original random assignment oversampled participants into the SAAF condition; this accounts for the greater number of 20-year-olds in the SAAF group. Written informed consent was provided. Each family was paid $100 after the assessment. Comparisons of pretest data collected at age 11 and data collected at age 18, using independent t tests and chi-square tests, of the youths who provided catecholamine data at age 20 with those who did not provide them did not reveal any differences on any demographic or study variables.

**Intervention Implementation**

The SAAF prevention program consisted of seven consecutive weekly meetings held at community facilities, with separate parent and youth skill-building curricula and a family curriculum (see Brody et al., 2012, for a complete description). Each meeting included separate, concurrent training sessions for parents and youths, followed by a joint parent–youth session during which the families practiced the skills they learned in the separate sessions. Concurrent and family sessions each lasted 1 hr; thus, parents and youths received 14 hr of prevention programming. During the weeks when the intervention families participated in the prevention sessions, the control families received three leaflets via postal mail describing early adolescent development, stress management, and the benefits of exercise. Parents in the prevention condition were taught the consistent provision of instrumental and emotional support, high levels of monitoring and control, adaptive racial socialization strategies, and the establishment of clear norms for the use of alcohol and other drugs. Youths were taught adaptive behaviors to use when encountering racism, the importance of forming goals for the future and making plans to attain them, and resistance efficacy skills. Coverage of the curriculum components (fidelity) exceeded .80 for the primary caregiver and target sessions. To preserve the random nature of the group assignment, the analyses reported here included all families who completed the pretest regardless of the number of prevention sessions that they actually attended (an intent-to-treat analysis). These families were retained in the analysis to preclude the introduction of self-selection bias into the findings.

**Data Collection Procedures**

Data on parenting, parental psychological adjustment, and demographic characteristics were collected in participants’ homes using a standardized protocol at pretest. Two African American field researchers worked separately with the primary caregiver and the target child. Interviews were conducted privately, with no other family members present or able to overhear the conversation. Catecholamines, alcohol use, smoking, exercise, BMI, and general health problems were assessed when youths were 20 years of age.

**Measures**

**Socioeconomic risk index.** Six dichotomous variables formed a socioeconomic risk index that was used as a control in the data analyses. 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. The scores were summed to form the index.

**Alcohol use, smoking, exercise, BMI, and health problems.** At age 20, youths reported their alcohol use, cigarette smoking, exercise, BMI, and health problems. Youths reported past-month alcohol use on a 6-point scale ranging from none to 20 or more days and smoking on a 7-point scale ranging from not at all to about two packs a day. Because the distributions of both smoking and alcohol use were skewed, we applied a log transformation to normalize the ratings. Youths also indicated the number of days during the past week on which they were physically active for a
This quasi-experimental study tested hypotheses about the effects of prevention at age 11 on catecholamines at age 20.

Results

Plan of Analysis

Two regression models were executed, with nonsupportive parenting or parental dysfunction as predictors, to test the first study hypothesis. These models included controls for gender, socioeconomic risk at age 11, and alcohol use, smoking, exercise, BMI, and general health problems at age 20. Two additional regression models were executed to test the second study hypothesis. These models tested the contributions to catecholamine levels of nonsupportive parenting or parental psychological dysfunction, assignment to SAAF or the control group, and the interaction of nonsupportive parenting/parental psychological dysfunction with prevention status. Interactions were interpreted through the plotting of estimated levels of catecholamines at low (1 standard deviation below the mean; −1 SD) and high (1 standard deviation above the mean; +1 SD) levels of either nonsupportive parenting or parental psychological dysfunction according to prevention status.

Tests of Hypotheses

Family risks at age 11 and catecholamine levels at age 20.

Supplemental Table 1, available online, presents descriptive statistics along with bivariate correlations. Tests of the first study hypothesis indicated that, for youths assigned randomly to the control group, both nonsupportive parenting, β = −.175, p < .05; ΔF(1, 190) = 6.111, p < .02, ΔR² = .030, and parental psychological dysfunction, β = .203, p < .01; ΔF(1, 190) = 7.198, p < .01, ΔR² = .035, at age 11 forecast elevated catecholamine levels 9 years later, with gender, SES risk, alcohol use, smoking, exercise, BMI, and general health problems controlled (see Supplemental Table 2, online). The results did not change when epinephrine and norepinephrine were analyzed separately.

Prevention programming and risk for elevated catecholamines. The second hypothesis proposed interaction effects between each of the hypothesized risk factors and prevention program participation status on catecholamine levels at age 20. Participants assigned randomly to SAAF were assigned a code of 1, and those assigned randomly to the control condition were assigned a code of 0. The analysis for nonsupportive parenting revealed an interaction with prevention status, β = −.195, p < .05; ΔF(1, 465) = 6.007, p < .02, ΔR² = .012. A significant interaction also emerged for parental psychological dysfunction, β = −.143, p < .05; ΔF(1, 465) = 4.078, p < .05, ΔR² = .008. (See Models 1 and 2 in Supplemental Table 3, online, for additional information about these analyses.) These interactions are illustrated in Figures 1a and 1b. Nonsupportive parenting, simple-slope = 0.176, SE = 0.062, p < .01, and parental psychological dysfunction, simple-slope = 0.217, SE = 0.068, p < .01, when youths were 11 were significantly associated with youth catecholamine levels at age 20 among those randomly assigned to the control group. These risk factors were not associated with catecholamine levels among youths randomly assigned to the prevention group, simple-slope = −0.010, SE = 0.044, ns for nonsupportive parenting; simple-slope = 0.042, SE = 0.058, ns for parental psychological dysfunction. When the analyses were executed separately for norepinephrine and epinephrine, the prevention buffering effect was more apparent for norepinephrine (both ps < .05) than for epinephrine (both ps < .10).

Discussion

This quasi-experimental study tested hypotheses about the effects of prevention at age 11 on catecholamines at age 20.
Young adults who had experienced high parental psychological dysfunction or nonsupportive parenting at age 11 and were assigned to the prevention condition evinced lower catecholamines than did their control group counterparts. From a public health perspective, these results suggest that developmentally appropriate interventions designed to enhance supportive parenting practices may buffer the effects of family risks on later SNS activity, particularly for families evincing high levels of risk.

A comment is warranted about intermediate or mediating processes that may contribute to a refined understanding of the obtained protective effects. Participation in SAAF, but not assignment to the control condition, was associated with a decrease in nonsupportive parenting from pretest to posttest; see Supplemental Table 4, online. This suggests that, among youths exposed to high family-risk levels, those assigned to SAAF may have had caregivers whose parenting skills enhanced youth self-regulation and emotion-regulation abilities. These abilities would help youths to avoid a stress-generation loop in which low self-regulation and emotion-regulation levels lead to heightened experiences with life stress that, in turn, would intensify SNS activity. Because examination of the effect of SAAF on SNS activity was not anticipated when the prevention trial began, no measures were included to index these potential mediating processes. This conjecture awaits further research.

Several limitations of the present study should be addressed in future research. First, it is not known whether the results of this study would generalize to urban African Americans or to families of other ethnicities. Second, because catecholamines were not one of the outcomes of interest when the study started, no baseline assessment for them is available. Hence, we cannot...
conclude that the intervention changed catecholamine levels. Third, the assay of catecholamines did not indicate how much of the variance is tonic versus phasic and did not indicate where the catecholamines were released (e.g., lungs, heart, or muscle). Future research should determine how exposure to family risks affects the nature of catecholamine release. Nevertheless, this analysis is one of the first to examine the ways in which family risk factors “get under the skin” to increase risk for heightened SNS activity and to suggest that family-centered preventive intervention can interrupt this process.

References


