A family-oriented psychosocial intervention reduces inflammation in low-SES African American youth

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Children of low socioeconomic status (SES) are at elevated risk for health problems across the lifespan. Observational studies suggest that nurturant parenting might offset some of these health risks, but their design precludes inferences about causal direction and clinical utility. Here we ask whether a psychosocial intervention, focused on improving parenting, strengthening family relationships, and building youth competencies, can reduce inflammation in low-SES, African Americans from the rural South. The trial involved 272 mothers and their 11-y-old children from rural Georgia, half of whose annual household incomes were below the federal poverty line. Families were randomly assigned to a 7-wk psychosocial intervention or to a control condition. When youth reached age 19, peripheral blood was collected to quantify six cytokines that orchestrate inflammation, the dysregulation of which contributes to many of the health problems known to pattern by SES. Youth who participated in the intervention had significantly less inflammation on all six indicators relative to controls (all P values < 0.001; effect sizes in Cohen’s d units ranged from −0.69 to −0.91). Mediation analyses suggested that improved parenting was partially responsible for the intervention’s benefits. Inflammation was lowest among youth who received more nurturant-involved parenting, and less harsh-inconsistent parenting, as a consequence of the intervention. These findings have theoretical implications for research on resilience to adversity and the early origins of disease. If substantiated, they may also highlight a strategy for practitioners and policymakers to use in ameliorating social and racial health disparities.

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Significance

Children from families of low socioeconomic status (SES) are vulnerable to a variety of health problems. These risks begin in early childhood and persist across the lifespan. Studies hint that nurturant parenting may offset these health risks, but it remains unclear whether these findings reflect a causal process and have clinical utility. Here we describe a randomized controlled trial, which sought to improve parenting and build youth competencies in low-SES African American families. The endpoint was low-grade inflammation, a process that underlies many health problems to which low-SES youth are vulnerable. Eight years after the intervention, youth who participated had significantly less inflammation than controls. If substantiated, these findings may provide a strategy for narrowing some of America’s social and racial disparities in health.
predisease states, and it also facilitates their progression into clinical entities such as diabetes, coronary heart disease, and stroke (23, 24), which are highly prevalent among African Americans residing in the Stroke Belt that stretches across the Southeastern United States, where our study takes place. Accordingly, we collected blood from youth whose families took part in the trial, and examined whether participating in this family-oriented intervention reduced expression of cytokines that orchestrate low-grade inflammation.

**Results**

The Strong African American Families (SAAF) program was designed to enhance parenting, strengthen family relationships, and foster youth competencies. The data presented here come from SAAF’s original clinical trial (25), which included 667 African American mothers and their 11- to 19-year-old children, all of whom resided in rural Georgia. At study entry, 46% of the families lived below federal poverty standards, and 90% fell into the federal low-income category. Of the families, 372 were randomly assigned to SAAF and 295 were randomly assigned to an assessment-only control condition. SAAF comprised seven weekly group meetings with separate activities for parents and children, as well as a family curriculum.

Eight years after the trial ended, we measured low-grade inflammation in 272 youth, who were 19 years of age at the time. These youth constitute the analytic sample here. At study entry, they were identical to the broader sample in terms of demographics, parenting quality, and mental health (Table S1; all P values > 0.15). However, these youth did participate more actively in SAAF, attending a mean of 5.11 sessions vs. 4.45 for those subjects without inflammation data, and their mothers showed greater improvements in parenting across the trial (Table S1; all P values > 0.02). Of the youth in the analytic sample, 173 had participated in SAAF and the other 99 had been assigned to the control group. Comparisons of these groups suggested that, despite attrition, the effects of random assignment were still evident. Specifically, SAAF and control subjects with inflammation data were identical in terms of sex, family and neighborhood SES, parenting quality, externalizing behavior, and cigarette smoking (all t values > 0.90, P values > 0.36; Table 1). Nevertheless, to minimize residual confounding, we adjusted for sex and family SES in all analyses presented.

Peripheral blood was collected from these youth and, using a multiplex assay from MesoScale Discovery, we measured circulating levels of six cytokines that orchestrate inflammation.

The cytokines were interleukins (IL)-1β, -6, -8, and -10, plus tumor necrosis factor α and IFN-γ. Correlations showed that concentrations of these molecules were strongly interrelated. The average intercytokine correlation was r = 0.75 and the range was r = 0.53–0.95. Because of this clustering, we formed an inflammation composite for each subject by summing the six z-scored cytokine values. The composite showed high levels of internal consistency (Cronbach’s α = 0.93) and functioned as the primary endpoint in the analyses below.

Our initial analysis was designed to determine whether SAAF affected inflammation. Adjusting for sex and family SES, subjects who participated in SAAF displayed significantly lower scores on the inflammation composite than did controls, F(1,268) = 51.63, P < 0.0001. Fig. 1 depicts this finding. To gauge the magnitude of this difference, we also computed the effect size indicator Cohen’s d. This value gives the difference between groups in pooled SD units; in the behavioral sciences, d values of 0.10, 0.30, and 0.50 (absolute values) are considered small, medium, and large-sized disparities, respectively (26). The d for this comparison was −0.90, indicating that, among SAAF subjects, inflammation composite values were almost 1 SD lower than those of controls.

Next, we disaggregated the composite to determine whether SAAF’s effects varied across cytokines. As Table 2 shows, they did not. Adjusting for sex and family SES, SAAF youth displayed significantly lower concentrations of all six cytokines measured, Fs (1,268) = 28.97–51.04, P values < 0.001, compared with control subjects. The d values for these comparisons ranged from −0.69 to −0.91, with an average of −0.79. Fig. 2 depicts these comparisons graphically.

SAAF is a multifaceted intervention that could have resulted in lower inflammation through any of several pathways. Based on the findings described in the Introduction, we hypothesized that this effect was attributable to improved parenting. To test this hypothesis, we estimated a mediation model with latent difference scores (27). First, we calculated a latent difference score that reflected the degree to which parenting improved from before to after SAAF implementation. Second, we calculated regression coefficients reflecting the associations between SAAF status and improved parenting (Path A) and improved parenting and the inflammation composite (Path B). Third, the indirect or mediating effect of parenting was quantified as the product of these two regression coefficients (A × B). Nonparametric bootstrapping was used to obtain the bias-corrected and accelerated confidence intervals of the indirect effect (28). The indirect effect was calculated 5,000 times using random sampling with replacement to build a sampling distribution. Sex and family SES were statistically controlled in the models.

The results of this analysis suggested that lower inflammation among SAAF participants was partially attributable to improved parenting. Fig. 3 depicts these findings. The positive coefficient for Path A indicates that participation in SAAF was associated with statistically significant improvements in parenting from pre- to posttrial. The negative coefficient for Path B indicates that, with inflammation even after accounting for parenting, as the significant Path C coefficient indicates. This result suggests that SAAF reduced inflammation through a combination of improved parenting and additional pathways. Overall model fit was good (29), with χ²(4) = 4.04, P = 0.40, comparative fit index = 1.00, and root mean square error of approximation = 0.006 (CI = 0.09).

To evaluate these patterns’ consistency, we reestimated mediation models separately for each cytokine. Table 3 displays the results. In each case, analyses supported a scenario wherein SAAF related to lower inflammation via improved parenting, as well as other pathways.

The questionnaire used in mediation analyses captures both nurturant-involved and harsh-inconsistent parenting (30). To clarify the aspects of parenting that were responsible for SAAF’s effects, we re-estimated the mediation models after separating these dimensions. However, the results suggested that uni-dimensional improvements were insufficient to modify

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**Table 1. Sample characteristics at study entry**

<table>
<thead>
<tr>
<th></th>
<th>SAAF (n = 173)</th>
<th>Control (n = 99)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
</tr>
<tr>
<td>Sex, male</td>
<td>0.43</td>
<td>0.50</td>
</tr>
<tr>
<td>Parenting quality</td>
<td>41.93</td>
<td>4.73</td>
</tr>
<tr>
<td>Family SES risk</td>
<td>2.42</td>
<td>1.45</td>
</tr>
<tr>
<td>Neighborhood SES risk</td>
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<td>2.48</td>
</tr>
<tr>
<td>Externalizing problems</td>
<td>11.23</td>
<td>7.78</td>
</tr>
<tr>
<td>Smoking frequency</td>
<td>0.05</td>
<td>0.22</td>
</tr>
</tbody>
</table>

P values are based on independent-groups t tests.
inflammation: for nurturant-involved parenting, indirect effect = −0.11, 95% CI [−0.39, 0.01]; for harsh-inconsistent parenting, indirect effect = −0.10, 95% CI [−0.38, 0.00]. Thus, SAAF was associated with the lowest inflammation levels in youth when it both increased the frequency of nurturant-involved parenting and decreased the frequency of harsh-inconsistent parenting.

To clarify further SAAF’s actions, we considered the possibility that it reduced youth smoking or adiposity, both of which are potent inflammatory stimuli (31). In separate models, we included variables reflecting smoking (frequency of cigarette use) and adiposity (body mass index). These variables were treated as mediators that linked changes in parenting quality with the inflammation composite. However, these indirect/mediating pathways did not reach statistical significance in either circumstance: for smoking, indirect effect = 0.01, 95% CI [−0.01, 0.04]; for adiposity, indirect effect = 0.00, 95% CI [−0.01, 0.03]. We also tested models where SAAF worked through smoking and obesity to reduce inflammation, regardless of parenting changes. Again, there was no evidence to support such a scenario: for smoking, indirect effect = −0.03, 95% CI [−0.26, 0.08]; for adiposity, indirect effect = 0.03, 95% CI [−0.03, 0.33].

Last, we considered whether SAAF’s effects might vary depending on SES. Families who live in more disadvantaged circumstances face greater material, psychosocial, and medical challenges. As a result, they might derive larger benefits from the intervention. To test this hypothesis, we added a neighborhood SES × SAAF interaction term to the latent mediation model described earlier, and tested for mediated moderation (32). Overall model fit was good, with χ²(8) = 6.99, P = 0.54, comparative fit index = 1.00, and root mean square error of approximation = 0.00 (CI = 0.07). The indirect pathway linking the interaction term, changes in parenting, and inflammation scores was statistically significant (coefficient = −0.046; 95% CI [−0.113, −0.009] with 5,000 bootstraps). These findings indicate that the more disadvantaged a family was, the more SAAF improved parenting quality (coefficient = 0.33, P < 0.01), and the lower youth inflammation scores were at age 19 (coefficient = −0.14, P < 0.05).

Discussion

Children from low-SES families are at heightened risk for health problems across the lifespan (1, 2, 4, 11). Although recent studies suggest that nurturant parenting might offset some of these health risks (13, 14, 18), they were not designed to permit inferences about causality or clinical utility. We conducted secondary analyses of data from a family-oriented intervention to determine whether it related to inflammation among low-SES, African American youth from the rural South. The results indicated that youth who participated in SAAF had significantly less inflammation 8 y after the intervention than did controls. These benefits were evident across six different inflammatory cytokines, and were most pronounced for the families living in the most disadvantaged circumstances. Mediation analyses were consistent with a scenario in which SAAF reduced inflammation, in part, by improving the quality of parenting these youth received.

These results may have implications for both research and practice. Conceptually, they build upon the observational studies described in the Introduction (13, 14, 18) and suggest that the buffering influences of enhanced parenting on inflammatory and cardiometabolic outcomes are likely to be causal in nature. In that regard, our results converge with the “parental effects” commonly observed in animal models, wherein maternal caregiving tendencies exert lasting influences on offspring physiology, especially in the brain, endocrine, and immune systems (33–35). Clinically, the findings suggest that SAAF, and perhaps other interventions focused on strengthening parenting and families, could play a role in forestalling or ameliorating some of the health problems for which low-SES youth are at risk. Because we did not assess actual health outcomes, further studies are needed to determine SAAF’s relevance for practice. Low-grade inflammation contributes to multiple health problems that pattern by SES during childhood and adolescence, including obesity, insulin resistance, high blood pressure, the early stages of coronary heart disease, and psychiatric conditions like depression, posttraumatic stress disorder, and substance abuse (15, 16, 36–39). A trial with endpoints like these would help clarify the clinical significance of our findings. Studies that track endpoints like cardiovascular morbidity would also be informative, as inflammation plays a key pathogenic role in atherosclerosis (15). However, the lengthy follow-up period required would make such work logistically challenging.

How might participating in SAAF have led youth to show less inflammation 8 y later? Mediation analyses suggested that the intervention’s benefits were partially attributable to improved parenting, but that additional mechanisms of action were also responsible. We considered the possibility that SAAF rendered youth less vulnerable to initiating smoking or developing obesity, both of which are potent inflammatory stimuli. Although findings were inconsistent with this scenario, it will be important in follow-up studies to evaluate more thoroughly the role of lifestyle. We did not collect pretrial measures of adiposity, and smoking rates in our subjects were negligible before and after SAAF. Therefore, changes in these candidate mediators could not be evaluated in relation to the intervention or inflammation. Also of interest would be SAAF’s effects on other lifestyle factors, like dietary composition and physical activity, as well as deep abdominal fat, which is a major reservoir of inflammatory activity (24, 37, 40). Future research should also consider the hypothesis that SAAF helped to ameliorate the impact of stressors common in this population such as deprivation, conflict, and

**Table 2. SAAF effects on inflammation, separately by cytokine**

<table>
<thead>
<tr>
<th>Cytokine</th>
<th>SAAF (n = 173)</th>
<th>Control (n = 99)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SEM</td>
</tr>
<tr>
<td>IL-8</td>
<td>2.76</td>
<td>0.05</td>
</tr>
<tr>
<td>TNF-α</td>
<td>0.83</td>
<td>0.04</td>
</tr>
<tr>
<td>IFN-γ</td>
<td>0.84</td>
<td>0.06</td>
</tr>
<tr>
<td>IL-6</td>
<td>0.83</td>
<td>0.07</td>
</tr>
<tr>
<td>IL-10</td>
<td>0.74</td>
<td>0.03</td>
</tr>
<tr>
<td>IL-1β</td>
<td>0.08</td>
<td>0.06</td>
</tr>
<tr>
<td>IL-8</td>
<td>0.83</td>
<td>0.04</td>
</tr>
<tr>
<td>IFN-γ</td>
<td>0.64</td>
<td>0.03</td>
</tr>
<tr>
<td>Composite</td>
<td>−1.60</td>
<td>0.37</td>
</tr>
</tbody>
</table>

*F values are from between-group ANOVAs comparing SAAF and Control subjects, with Sex and Family SES as covariates. Cohen’s d is a measure of effect size, expressed in SD units. ***P < 0.001.*
violence, and discrimination. Indeed, SAAF may have instilled a “shift and persist” style in youth. This style entails a combination of shifting (accepting life for what it is and adapting oneself to it) and persisting (enduring life with strength by holding on to meaning and optimism), which together mitigate the health impact of stressors that many low-SES youth face. Indeed, research shows that low-SES youth who display shift and persist traits have inflammation profiles and health outcomes similar to their high-SES peers (41, 42). Consistent with our findings of partial mediation, SAAF could have instilled shift and persist tendencies via its emphasis on nurturant-involved parenting and/or its efforts to directly instill competencies in youth. Finally, SAAF’s parenting components may have reduced the frequency of conflict, violence, and neglect in the home, any of which can activate stress-related autonomic and endocrine pathways in youth, with downstream consequences of inflammation (43–49). In future intervention research, it will be important to obtain deeper and broader coverage of parenting behaviors, life stress, and strategies like shift and persist, so these mediator hypotheses can be tested formally.

Several limitations of this study must be noted. First, the SAAF trial was not designed with inflammation as an endpoint. As a result, we did not collect pretrial blood samples that could be used to determine whether the intervention and control groups’ inflammation profiles changed differentially over time. At study entry, the SAAF and control groups were similar in terms of SES, parenting quality, mental health, and lifestyle. These findings are consistent with the possibility that the groups began the trial with similar inflammation profiles. Nevertheless, until prepost data are available, conclusions about SAAF’s capacity to bring about changes in inflammation must be viewed as tentative. Second, we obtained blood for cytokine assessment from only a subset of participants. Before SAAF, these families were identical to the broader trial population in terms of SES, parenting quality, mental health, and lifestyle. With that said, these families were more actively engaged in the intervention, and perhaps as a consequence showed greater improvements in parenting. As a result, the disparities in inflammation we detected here may be larger in magnitude than would have been observed in the full sample. Lastly, the study did not assess SAAF’s effects on formal health outcomes, so the clinical relevance of the inflammation findings remains uncertain. All of these limitations can be addressed in a follow-up trial that is designed and executed with an a priori focus on assessing inflammation and its downstream clinical repercussions.

Despite these limitations, the study provides initial evidence suggesting that a family-oriented intervention can reduce inflammation in youth, in part by improving the quality of the parenting they receive. To the extent that they are substantiated in future research, these results may provide a strategy for narrowing some of the racial and social disparities in health apparent in the United States (1, 50), particularly those conditions originating in childhood (51, 52). Indeed, rates of childhood poverty have risen steadily in the United States in recent years, and this trend has been especially pronounced in rural, African American communities (53). When layered on top of existing social and racial disparities, these childhood poverty trends have the potential to worsen American’s population health in the coming decades (54) and to limit the country’s ability to develop its human capital (55). Thus, interventions with the capacity to

Fig. 2. SAAF’s effects were consistent across the six cytokines measured. In each figure, dots represent individual data points, the wide horizontal bar is the group mean, and the error bars reflect 95% confidence intervals.

Fig. 3. Results are consistent with the hypothesis that SAAF’s ability to reduce inflammation is partially attributable to improved parenting. The figure shows results from a mediation model with latent difference scores. Solid and dashed lines reflect significant and nonsignificant paths, respectively. Unstandardized coefficients are shown. *P < 0.05; **P < 0.01; ***P < 0.001.
Table 3. Results of mediation models with latent difference scores, separately by cytokine

<table>
<thead>
<tr>
<th>Parameter</th>
<th>IFN-γ</th>
<th>IL-10</th>
<th>IL-1β</th>
<th>IL-6</th>
<th>IL-8</th>
<th>TNF-α</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main effect: SAAF → Cytokine</td>
<td>-0.35***</td>
<td>-0.27***</td>
<td>-0.65***</td>
<td>-0.76***</td>
<td>-0.48***</td>
<td>-0.45***</td>
</tr>
<tr>
<td>SAAF → Changes in parenting</td>
<td>1.18*</td>
<td>1.18*</td>
<td>1.18*</td>
<td>1.19*</td>
<td>1.18*</td>
<td>1.18*</td>
</tr>
<tr>
<td>Changes in parenting → Cytokine</td>
<td>-0.01*</td>
<td>-0.01*</td>
<td>-0.02*</td>
<td>-0.03**</td>
<td>-0.02*</td>
<td>-0.02*</td>
</tr>
<tr>
<td>Direct effect: SAAF → Cytokine</td>
<td>-0.24***</td>
<td>-0.25***</td>
<td>-0.63***</td>
<td>-0.73***</td>
<td>-0.46***</td>
<td>-0.43***</td>
</tr>
<tr>
<td>Indirect (mediated) effect SAAF →</td>
<td>-0.01</td>
<td>-0.02</td>
<td>-0.02</td>
<td>-0.04</td>
<td>-0.02</td>
<td>-0.02</td>
</tr>
<tr>
<td>Parenting → Cytokine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>95% CI for indirect (mediated) effect with 5,000 bootstraps</td>
<td>[-0.039, -0.001]</td>
<td>[-0.039, -0.002]</td>
<td>[-0.071, -0.002]</td>
<td>[-0.104, -0.005]</td>
<td>[-0.060, 0]</td>
<td>[-0.053, -0.002]</td>
</tr>
</tbody>
</table>

Values are unstandardized coefficients derived from latent difference score mediation models. ***P < 0.001; **P < 0.01.


