ORIGINAL ARTICLE

The Relationship Between Disproportionate Social Support and Metabolic and Inflammatory Markers: Moderating Role of Socioeconomic Context

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ABSTRACT

Objective: The present study examines the association of disproportionate social support (the relative balance of support given versus received) on metabolic and inflammatory outcomes and whether effects vary by socioeconomic context.

Methods: We enrolled a sample of 307 parental caregivers living with a child with a chronic illness. Parents were assessed on four dimensions of social support: emotional support received, instrumental support received, emotional support given, and instrumental support given. Disproportionate social support was calculated as the difference between support received and support given. Participants provided sociodemographic information, were interviewed about financial stress, and were assessed on metabolic (systolic blood pressure, diastolic blood pressure, total cholesterol, body fat percent, and body mass index) and inflammatory (interleukin 6 and C-reactive protein) outcomes.

Results: More disproportionate instrumental and emotional support was associated with higher inflammation (b = 0.10, SE = 0.04, p = .014; b = 0.0.09, SE = 0.05, p = .042, respectively). We observed significant interactions between disproportionate social support and income (b = −0.04, SE = 0.02, p = .021). Parents from lower-income households who gave more emotional support than they received had higher inflammation compared with those from higher-income households. We also observed a significant interaction between disproportionate instrumental support and income (b = 0.04, SE = 0.02, p = .006). Parents from lower-income households who received more instrumental support than they gave had worse metabolic outcomes compared with parents from higher-income households. Parallel interaction patterns were observed using an interview-based measure of financial stress.

Conclusions: These findings show that disproportionate social support has implications for physical health, particularly for caregivers from socioeconomically disadvantaged households.

Key words: social support, health, parents, caregivers, socioeconomic status, inflammation.

INTRODUCTION

One of the most compelling findings in health psychology is the critical role social relationships play in promoting physical health and well-being. In a seminal review published more than 30 years ago, House et al. (1) demonstrated that weaker social ties increased the risk of morbidity and mortality, with the size of these effects comparable in magnitude to smoking, blood pressure, obesity, and physical activity. There is now a large literature documenting an association between social relationships and mortality risk (2). One facet of social relationships, *social support*, has predicted a reduced risk of mortality (3), cardiovascular disease (4), cancer (5), and inflammatory outcomes (6). Most of these studies have focused on social support received (7). However, individuals also actively provide support to others in their daily lives. Hence, researchers have theorized that it may also be important to consider the relative balance of support given and received (8). To date, few studies have examined the relationship between an imbalance between receiving and giving social support and physical health outcomes. The present study sought to examine how imbalances in social support relate to metabolic and inflammatory outcomes in a sample of parental caregivers.

Receiving Social Support and Health

Over the past four decades, studies have linked social support to mental and physical health (9), reduced mortality (1), coronary heart disease (4), breast cancer (10), and preexisting health conditions (11). Evidence also suggests that receiving social support is linked to physiological processes such as immune, neuroendocrine, and cardiovascular functioning (12,13). Accumulating theories suggest that long-term chronic health outcomes are influenced by social support through pathways involving the protective role of social support in physiological responses to chronic stress (12,14).

There is also longitudinal evidence for the health-promoting role of social support. For instance, Yang and colleagues (13) found that social support at baseline was associated with reduced inflammatory biomarkers 10 years later. Longitudinal studies have found evidence linking social support to a reduced risk of mortality

CRP = C-reactive protein, SD = standard deviation, **SES** = socioeconomic status

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(15–17), cardiovascular disease (4), physical inactivity (18), and increased health-related quality of life (19).

Providing Social Support and Health

More recent work has examined the impact of *giving* social support on physical health, with the idea being that giving support to others can be rewarding and stress reducing (20). Indeed, giving social support has been associated with lower mortality rates (21), health-related work absences (22), cardiovascular activity (23), and sympathetic responses to social stress (24). Moreover, studies have demonstrated giving social support to be an independent predictor from receiving social support (21,23).

Other evidence, however, suggests that giving social support can sometimes take a physiological toll, particularly in situations in which one provides high levels of support to others, such as caregiving. One meta-analysis found that caregivers exhibited a slightly greater risk of negative cardiovascular, metabolic, and inflammatory health outcomes compared with noncaregivers (25). Empirical studies have also found associations between caregiving and increased mortality risk (26), reduced immune responses to challenge (27–29), and dysregulation in immune-mediated processes (30). These physiological outcomes are theorized to result from a combination of the demands of caregiving and elevations in chronic stress (31).

One explanation for findings of both benefits and harms of giving support is that the receipt of social support has typically not been taken into account. For example, studies that show a benefit of providing social support may include populations that have a relatively equitable balance of support given and received. Thus, studies that examine the health effects of the relative balance of social support given to support received are an important next step to further understanding the relationship between social support and physical health.

Disproportionate Social Support—Is the Balance of Support Given Versus Received Associated With Health?

Past theories have described imbalances in social support with terms such as reciprocity, social exchange, and relationship equity (32). For example, an early theory of social exchange (8) argued that individuals attempt to balance efforts and rewards in interpersonal relationships, and those who reciprocate support strengthen their social ties. Equity theory suggests those who are more successful in reciprocating support will have a greater well-being and health than will those who lose or profit too much in their exchange (8,33). In general, what these theories have in common is the idea that when there are imbalances in a social relationship, that this can have negative consequences.

Social relationship imbalances can occur across a number of dimensions. For example, with social exchange theory, this refers to an imbalance between effort devoted to a relationship and rewards reaped from that relationship. Others have discussed imbalances in terms of the positive versus negative aspects of social relationships (34). In the present article, we focus on imbalance in terms of the giving versus receiving of social support. We take this approach because the social support literature within health psychology has historically been focused on support received (35), with recent growing interest in the benefits of giving social support (20), but with very little attention having been paid to the joint effects of contrasting aspects of social relationships on health (36). In this article, we conceptualize an individual who gives more support than he/she receives as someone who is doing *disproportionate giving*. Conversely, an individual who receives more support than he/she gives is *disproportionately receiving*. In the present study, we investigate the effects of disproportionate giving or receiving on health.

There is evidence in the mental health domain to support this hypothesis. Both disproportionate giving and disproportionate receiving of social support have been shown to predict worse mental health outcomes across various relationship types (romantic, peers, family, etc.) (37). Disproportionate giving has been shown to elicit feelings of resentment, burden, and dissatisfaction (38,39). Disproportionate receiving has been associated with feelings of indebtedness, guilt, or shame (38–40).

Less is known about the physical health consequences of disproportionate giving or receiving. To our knowledge, two studies provide preliminary evidence that disproportionate giving or receiving would have associations with physical health. In one study, Chandola and colleagues (41) found that nonreciprocity, defined as an imbalance in support in which people give more support than they receive, was associated with elevated odds of poor self-reported health and sleep problems compared with individuals reporting lower nonreciprocity.

Second, Vaananen and colleagues (22) examined the effects of disproportionate giving or receiving of social support on health-related absences from work over a 9-year period. Receiving more support than one gives was associated with more sick absences among women. Contrastingly, among men, giving more support than one receives was associated with more sick days. The authors theorized that gender differences may have been due to different cultural expectations by gender around caretaking. These effects also suggest that the impact of disproportionate social support may not be uniform across groups.

There is also indirect evidence for an effect of disproportionate giving on health from one study that examined the relationship between parental empathy and low-grade inflammation. Manczak and colleagues (42) found that higher levels of empathy were associated with more chronic, low-grade inflammation among parents. These patterns suggest high levels of empathy—which might indicate parents who are disproportionately giving emotional support to their children—have associations with inflammation.

The Role of Socioeconomic Context

The health effects of disproportionate giving or receiving support could also vary by socioeconomic context. Socioeconomic status (SES) is a well-documented and powerful predictor of health and disease, with individuals lower in SES being most vulnerable (43–45). The buffering hypothesis of social support theorizes that psychological benefits of social support result from social support protecting from adverse effects of stress (46). Low SES environments are characterized by various chronic stressors (47,48). Thus, a good balance on social support dimensions may also help buffer the relationship between SES and health. To our knowledge, no prior studies have tested for interactions between SES and disproportionate social support. There is one example that is suggestive of the idea that giving more than one receives could be associated with worse health in lower socioeconomic contexts. That study

found that greater family obligations were associated with higher levels of airway inflammation and poorer asthma control in youth with asthma only if they came from lower-SES households (no associations among those from higher-SES households) (49). Family obligations refer to the behaviors people engage in to provide instrumental help to their families (e.g., caring for family members). Thus, people who are high on family obligations might be disproportionately giving social support. These findings suggest that the disproportionate giving of support could be particularly burdensome to those who are living within socioeconomically disadvantaged contexts.

The Present Study

In the present study, we tested associations between disproportionate social support and metabolic and inflammatory outcomes in a sample of 307 adult parental caregivers living with at least one child with a chronic illness. We tested two hypotheses based on previous literature: a) that disproportionate giving or receiving of social support would be associated with poorer inflammatory and metabolic outcomes and b) that effects would be strongest among individuals from disadvantaged family socioeconomic circumstances.

METHODS

Participants

We enrolled 307 parental caregivers living with a child physician-diagnosed with asthma. Families were recruited from Chicago and surrounding areas between 2014 and 2016. To be included in the study, parents were required to be fluent in English, and only 1% per household was permitted. Parents provided written consent. At a laboratory visit, parents completed a blood draw, provided demographic information, and completed a battery of questionnaires and interviews. This study was approved by Northwestern, NorthShore, and Erie Institutional Review Boards.

Disproportionate Social Support

Disproportionate social support was measured using the 21-item 2-Way Social Support Scale (50). This scale measures four dimensions of social support: emotional support received, instrumental support received (i.e., help with childcare or transportation), emotional support given, and instrumental support given. Items were rated on a 6-point scale ranging from 0 (not at all) to 5 (always). These subscales had good internal reliability: emotional support received (seven items; $\alpha = .92$), instrumental support received (four items; $\alpha = .79$), emotional support given (five items; $\alpha = .86$), and instrumental support given (four items; $\alpha = .70$).

Given the novelty of this study's hypotheses, there is no established, validated method for calculating the disproportionate giving or receiving of social support. We conceptualized disproportionate support as an imbalance, or being higher on one dimension than the contrasting dimension. Thus, we operationalized disproportionate support as a difference score, which is consistent with approaches found in previous social support research outside of health psychology (38,39,51,52).

Because of differences in possible score ranges across the four dimensions, the score for each support dimension was first standardized. Disproportionate support was calculated as the standardized giving score minus the standardized receiving score. Separate scores were calculated for disproportionate instrumental and disproportionate emotional support. Positive values indicate more support given than received (disproportionate giving). Negative values indicate more support received than given (disproportionate receiving).

Health Measures

With healthy samples, meaningful clinical measures of health can be difficult to obtain; thus, many researchers turn to physiological indicators that may be precursors to chronic health conditions. In the present study, we assessed a number of metabolic and inflammatory markers. To reduce the number of analyses conducted, we created a composite metabolic and a composite inflammation score by standardizing and summing measures of each that were collected. This approach parallels operationalizations of allostatic load that have standardized and summed across multiple biological markers in previous studies (53–55). Researchers have argued that such continuous composite measures result in stronger predictors of health outcomes (55). We created separate metabolic and inflammatory composites because of the notion of inflammation as distinct from, and potentially a pathway to, metabolic disease (56).

Metabolic Composite

Five measures relevant to metabolic risk were taken in this study: systolic blood pressure, diastolic blood pressure, total cholesterol, body fat percent, and body mass index. After a 4-minute rest period, resting systolic blood pressure and diastolic blood pressure were measured. Blood pressure was recorded using an automated oscillometer. An initial reading was taken and discarded to allow participants to get used to the cuff. Blood pressure was then recorded a subsequent three times at 2-minute intervals. The average of the final three measurements was calculated and used in analyses. Total cholesterol was measured in nonfasting blood samples (we were not able to obtain more specific lipid profiles because participants were not required to fast). Antecubital blood was collected into an 8.5-mL serum separator tube (Becton-Dickinson, Franklin Lakes, New Jersey). Following the manufacturer's instructions, the tube was centrifuged at 1200*g* for 10 minutes, after which serum was harvested. Total cholesterol was measured on a Roche/Hitachi cobas c701 instrument at the NorthShore University Health System's Core Laboratory. Detection ranges for total cholesterol were 0.1 to 20.7 mmol/L. Body fat percent was calculated via bioelectrical impedance, which uses electrical current resistance to approximate body fat percent (Tanita Model BF-350). Height and weight measurements were obtained using a balance beam scale with stadiometer and were used to calculate body mass index (in kilograms per meter squared). To create a metabolic composite, scores on each of these five measures were standardized and then averaged. Similar types of metabolic composites have been reported in previous literature (57–59), with support for the usage of continuous metabolic composite scores found in previous literature (60).

Low-Grade Inflammation Composite

Low-grade inflammation was assessed from serum samples, including circulating levels of C-reactive protein (CRP) and interleukin 6. CRP was measured in duplicate by high-sensitivity immunoturbidimetric assay on a Roche/Hitachi cobas c502 analyzer. Average intra-assay and interassay coefficients of variation were 2.5% and 5.6%, respectively. The assay's lower limit of detection is 0.2 mg/L. Interleukin 6 was measured in duplicate by electrochemiluminescence on a SECTOR Imager 2400A (MesoScale Discover). The lower limit of detection was 0.19 pg/ml. The median intra-assay coefficient of variation was 2.76%. Raw values of each marker were log transformed to correct for skew. An inflammation composite was computed by averaging standardized log-transformed scores. Similar types of combined inflammation scores have been used in previous research (57,61–63).

Socioeconomic Measures

Income

Parents reported annual gross family income from all sources (i.e., wages, investments, government assistance) and chose a category ranging from 1 to 9, with categories ranging from <$5000 to ≥$200,000.

Financial Stress

As a second measure of families' financial circumstances, parents were interviewed using the UCLA Life Stress Interview (64). This semistructured

interview probes chronic stress over the past 6 months across several domains. Relevant to the present study is the chronic stress section on financial stress. Interviewers asked a series of open-ended questions about finances (e.g., difficulty affording housing, food, transportation, and job instability) and rated the level of chronic, ongoing financial stress on a 5-point scale. Higher numbers reflect more severe and persistent financial difficulties. Interviewer-based ratings allow for a more standardized measure of household financial stress (as opposed to relying on participants' subjective perceptions of financial stress). The validity and reliability of this interview have been shown previously (64). Our research team has conducted this interview for the past 10 years, with interrater reliabilities ranging from 0.88 to 0.94 across subscales (65).

Covariates

A priori, we identified a set of demographic covariates to include in statistical models. Covariates included age, sex, and race/ethnicity (dummy coded for White, Black, and other).

Analytic Method

We conducted a series of multiple hierarchical regression analyses to test if disproportionate social support predicted metabolic and inflammatory outcomes and whether there were interactions with families' socioeconomic conditions. Separate regressions were conducted for metabolic and inflammatory composites. In step 1, metabolic and inflammatory composites were predicted from key covariates (race, age, sex). In step 2, the socioeconomic variable (income or financial stress) and the social support variable (disproportionate instrumental or emotional support) were entered to test main effects. In step 3, we entered an interaction term between social support and the socioeconomic measure. Continuous predictors were mean-centered. Significant interactions were followed by simple slopes tests at ±1 standard deviation (SD) from the social support variable mean.

RESULTS

Preliminary Analyses

Descriptive statistics are presented in Table 1. As expected, income and financial stress were significantly correlated ($r = -0.693$, $p < .001$). Higher income corresponded to less financial stress. The metabolic and inflammation composites were not significantly correlated ($r = 0.100$, $p = .09$).

TABLE 1. Descriptive Statistics ($n = 307$)

	M (SD) or %
Age, y	45 (6.75)
Race/ethnicity	
White	60.10%
Black	21.80%
Other	18.10%
Sex (female)	88.00%
Social support	
Giving instrumental	20.19 (3.68)
Giving emotional	21.66 (3.51)
Receiving instrumental	16.50 (3.76)
Receiving emotional	31.17 (5.47)
Income	
<$75,000	32.6%
$75,000–$149,999	35.8%
>$150,000	31.6%
Financial stress	2.73 (0.94)
Metabolic outcomes	
SBP, mm Hg	119.90 (14.28)
DBP, mm Hg	72.20 (9.97)
Cholesterol, mg/dl	188.53 (32.75)
Body fat, %	36.10 (8.85)
BMI, kg/m^2	29.04 (6.79)
Low-grade inflammation	
IL-6, pg/ml	−0.09 (0.34)
hs-CRP, mg/L	0.17 (0.51)

Financial stress scores ranged from 1 to 5. Higher numbers reflect more severe and persistent financial difficulties. Scores ranged from 0 to 25 for giving emotional and giving instrumental support. Receiving instrumental support scores ranged from 0 to 20. Receiving emotional support scores ranged from 0 to 35. Higher scores indicated more receiving or giving support respectively. Disproportionate social support M and SD values are not shown because values approximated 0 and 1, respectively, after standardization. IL-6 and hs-CRP are log-transformed scores.

M SD = mean; SD = standard deviation; SBP = systolic blood pressure; DBP = diastolic blood pressure; BMI = body mass indexIL-6 = interleukin 6; hs-CRP = high-sensitivity C-reactive protein.

Disproportionate Instrumental Social Support

Analyses With Income

Main Effects

Higher income was associated with lower inflammation ($b = -0.07$, SE = 0.02, $p = .004$) and lower metabolic composite scores ($b = -0.06$, SE = 0.02, $p = .003$; Table 1). More disproportionate instrumental support (giving more than one receives) was related to higher inflammation ($b = 0.10$, SE = 0.04, $p = .014$) but not significantly related to the metabolic composite ($b = 0.05$, SE = 0.03, $p = .15$).

Interaction Effects

Main effects were qualified by a significant interaction between disproportionate instrumental support and income predicting the metabolic composite ($b = 0.04$, SE = 0.02, $p = .006$). Simple slope analyses at ±1 SD of support indicated that the relationship between income and the metabolic composite is strongest when caregivers disproportionately receive instrumental support (Figure 1). Patterns from Figure 1 indicate that, among those who disproportionately receive support, higher metabolic composite scores are found among individuals from a lower-income household (simple main effect of income: $b = -0.10$, SE = 0.03, $p < .001$). To determine effect size, we calculated the standardized β for this simple main effect (which is more interpretable than reporting an effect size for the interaction effect). Effect sizes of 0.1–0.3 are considered small; 0.3–0.5, medium; and ≥0.5, large. The standardized β for this effect was −0.35. When participants reported disproportionately giving support, scores on the metabolic composite were high and not associated with income (simple main effect of income: $b = -0.02$, SE = 0.02, $p = .39$). No significant interaction emerged for inflammation (Table 2).

Analyses With Financial Stress

Main Effects

Higher financial stress predicted higher inflammation ($b = 0.12$, SE = 0.05, $p = .012$) and metabolic composites scores ($b = 0.14$, SE = 0.04, $p < .001$; Table 2). More disproportionate giving of

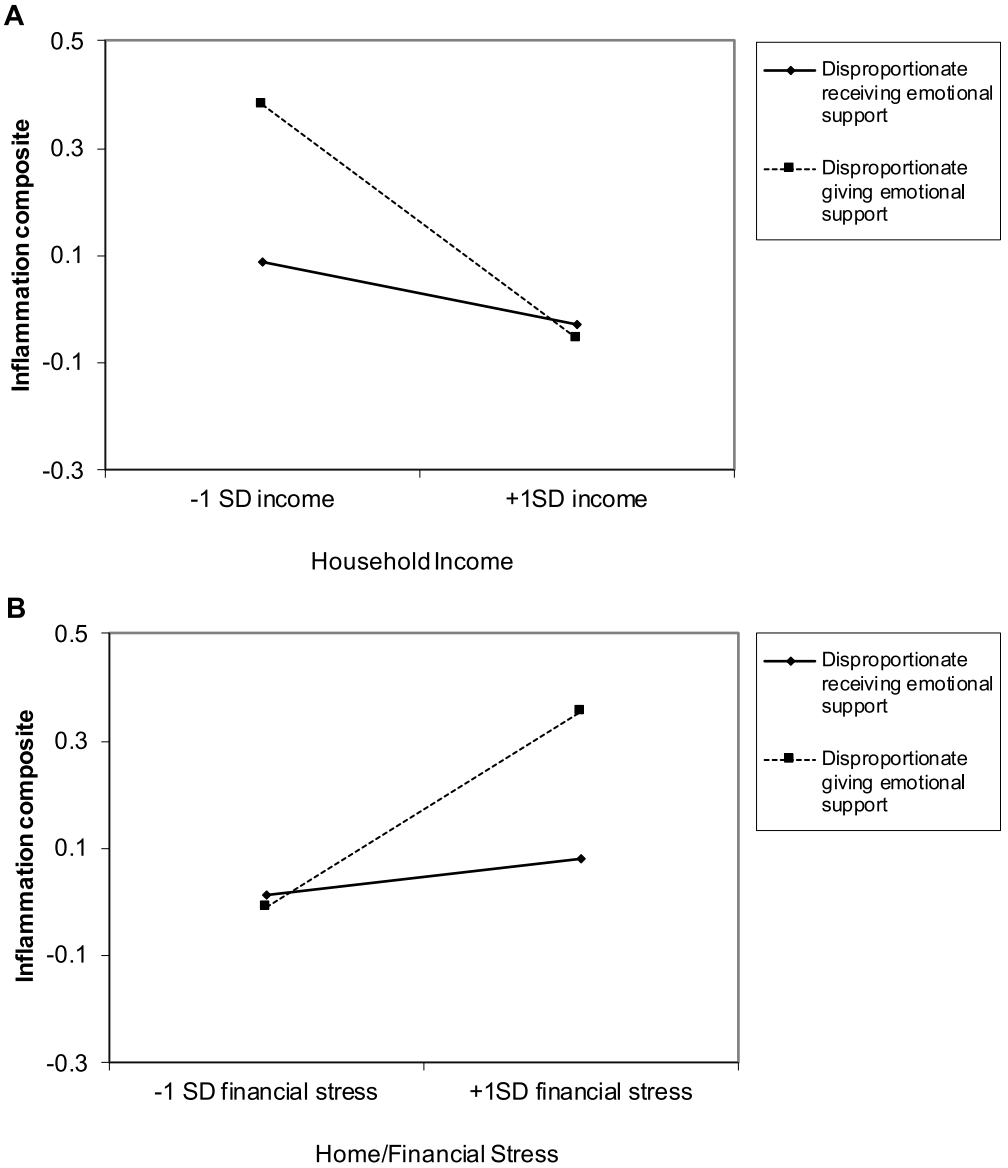

FIGURE 1. The effects of family socioeconomic variables and disproportionate instrumental social support on the metabolic composite. Multiple linear regression analyses that controlled for age, race, and sex were used. The lines represent estimated regression lines for +1 SD instrumental support (representing those who disproportionately give more instrumental support than they receive) and −1 SD instrumental support (representing those who disproportionately receive more instrumental support than they give). The metabolic composite was calculated by averaging standardized systolic blood pressure, diastolic blood pressure, total cholesterol, body fat percentage, and body mass index. The two figures depict significant interaction effects for each family socioeconomic variable: income (A) and an interview-based measure of financial stress (B).

instrumental support was related to higher inflammation ($b = 0.10$, SE = 0.04, $p = .014$) but not significantly related to the metabolic composite ($b = 0.05$, SE = 0.03, $p = .13$).

Interaction Effects

We observed a significant interaction between disproportionate instrumental support and financial stress predicting the metabolic composite ($b = -0.09$, SE = 0.03, $p = .004$). Simple slope analyses at ±1 SD of support indicated that the relationship between financial stress and the metabolic composite score is strongest when caregivers are receiving disproportionate instrumental support (Figure 1). Patterns from Figure 1 indicate that, among those who disproportionately receive support, higher metabolic composite scores are seen among those with high financial stress (simple main effect of financial stress: $b = 0.23$, SE = 0.05, standardized $\beta = 0.35$, $p < .001$) However, when participants reported disproportionate giving of instrumental support, metabolic composite scores are high and not associated with financial stress (simple main effect of financial stress: $b = 0.06$, SE = 0.05, $p = .24$). No significant interaction emerged for inflammation (Table 2).

TABLE 2. Instrumental Social Support and Socioeconomic Variables Predicting Inflammation and Metabolic Composites (n = 307)

	Income	Financial Stress
Inflammatory composite		
Step 1		
Age	0.01 (0.01)	0.01 (0.01)
Race (Black)	0.20 (0.13)	0.20 (0.13)
Race (White)	−0.26 (0.11)***	−0.25 (0.11)***
Sex	−0.28 (0.13)***	−0.28 (0.13)***
Step 2		
SES	−0.07 (0.02)**	0.12 (0.05)***
Disproportionate instrumental support	0.10 (0.04)***	0.10 (0.04)***
Step 3		
Disproportionate instrumental support by SES	−0.01 (0.02)	0.01 (0.04)
Metabolic composite		
Step 1		
Age	0.02 (0.01)**	0.02 (0.01)**
Race (Black)	0.31 (0.10)**	0.32 (0.10)**
Race (White)	−0.15 (0.09)	−0.14 (0.09)
Sex	−0.06 (0.11)	−0.06 (0.11)
Step 2		
SES	−0.06 (0.02)**	0.14 (0.04)*
Disproportionate instrumental support	0.05 (0.03)	0.05 (0.03)
Step 3		
Disproportionate instrumental support by SES	0.04 (0.02)**	−0.09 (0.03)**

Values are reported as b (SE). Top half of the table reports coefficients predicting the inflammatory composite as an outcome; bottom half of the table reports coefficients predicting the metabolic composite as an outcome.

SES = socioeconomic status variable (effect of income in column 1, effect of financial stress in column 2).

* $p < .001$.

** $p < .01$.

*** $p < .05$.

Disproportionate Emotional Social Support

Analyses With Income

Main Effects

Higher income was associated with lower inflammation (b = −0.07, SE = 0.02, p = .003) and metabolic composites scores (b = −0.06, SE = 0.02, p = .002; Table 3). Higher disproportionate giving of emotional support was related to higher inflammation (b = 0.0.09, SE = 0.05, p = .042) but not significantly related to the metabolic composite (b = 0.01, SE = 0.04, p = .77).

Interaction Effects

We observed a significant interaction between disproportionate emotional support and income in predicting the inflammation composite (b = −0.04, SE = 0.02, p = .021). Simple slope analyses at ±1 SD of support indicated that the relationship between income and inflammation is strongest when caregivers are disproportionately giving emotional support (Figure 2). Patterns from Figure 1 indicate that among those who disproportionately give emotional support, higher inflammation is seen if one comes from a lower-income household (simple main effect of income: b = −0.10, SE = 0.03, standardized β = −0.29, $p < .001$) However, when participants reported disproportionate receiving of emotional support, inflammation is lower and not significantly associated with income (simple main effect of income: b = −0.03, SE = 0.03, p = .33).

We note that some participants had high levels of CRP (values >10; n = 16/307). When these cases were removed, the interaction term between disproportionate emotional support and income predicting the inflammation composite remained significant (b = −0.04, SE = 0.02, p = .022). No significant interaction emerged for the metabolic composite (Table 3).

TABLE 3. Emotional Support and Socioeconomic Variables Predicting Inflammation and Metabolic Composites (n = 307)

	Income	Financial Stress
Inflammatory composite		
Step 1		
Age	0.01 (0.01)	0.01 (0.01)
Race (Black)	0.20 (0.13)	0.20 (0.13)
Race (White)	−0.26 (0.11)***	−0.25 (0.11)***
Sex	−0.28 (0.13)***	−0.28 (0.13)***
Step 2		
SES	−0.07 (0.02)**	0.12 (0.05)***
Disproportionate emotional support	0.09 (0.05)***	0.09 (0.05)***
Step 3		
Disproportionate emotional support by SES	−0.04 (0.02)***	0.08 (0.04)***
Metabolic composite		
Step 1		
Age	0.02 (0.01)**	0.02 (0.01)**
Race (Black)	0.31 (0.10)**	0.32 (0.10)**
Race (White)	−0.15 (0.09)	−0.14 (0.09)
Sex	−0.06 (0.11)	−0.06 (0.11)
Step 2		
SES	−0.06 (0.02)**	0.15 (0.04)*
Disproportionate emotional support	0.01 (0.04)	0.00 (0.04)
Step 3		
Disproportionate emotional support by SES	0.00 (0.01)	0.01 (0.03)

Values are reported as b (SE). Top half of the table reports coefficients predicting the inflammatory composite as an outcome; bottom half of the table reports coefficients predicting the metabolic composite as an outcome

SES = socioeconomic status variable (effect of income in column 1, effect of financial stress in column 2).

* $p < .001$.

** $p < .01$.

*** $p < .05$.

Analyses With Financial Stress

Main Effects

Higher financial stress predicted higher inflammation (b = 0.12, SE = 0.05, p = .012) and metabolic composites scores (b = 0.15, SE = 0.04, p < .001; Table 3). More disproportionate emotional support was significantly related to increased inflammation (b = 0.09, SE = 0.05, p = .047) but unrelated to the metabolic composite (b = 0.001, SE = 0.04, p = .88).

Interaction Effects

We observed a significant interaction between disproportionate emotional support and financial stress in predicting inflammation (b = 0.08, SE = 0.04, p = .033). This interaction remained significant when high CRP cases were removed (b = 0.10, SE = 0.04, p = .019). Simple slope analyses at ±1 SD of support indicated that the relationship between financial stress and inflammation composite scores is strongest when caregivers disproportionately give emotional support (Figure 2). Patterns from Figure 2 indicate that, among those who disproportionately give emotional support, higher inflammation is seen when financial stress is high (simple main effect of financial stress: b = 0.20, SE = 0.06, standardized β = 0.24, p = .001). However, when participants disproportionately receive emotional support, inflammation scores are lower and not significantly associated with financial stress (simple main effect of financial stress: b = 0.04, SE = 0.06, p = .57). No significant interactions emerged for the metabolic composite (Table 3).

Potential Confounds

One potential confound of these associations is levels of parental depression. We repeated all analyses controlling for parental depressive symptoms (assessed with the Center for Epidemiologic Studies Depression Scale). All interaction effects reported previously remained significant with this additional covariate including: disproportionate instrumental support by income: b = 0.05, SE = 0.02, p = .002; disproportionate instrumental support by financial stress: b = −0.09, SE = 0.03, p = .002; disproportionate emotional support by income: b = −0.05, SE = 0.02, p = .009; and disproportionate emotional support by financial stress: b = 0.10, SE = 0.04, p = .018.

DISCUSSION

The present study found evidence for main effects of disproportionate social support, as well as interaction effects with family socioeconomic indicators, in predicting metabolic and inflammatory outcomes among parental caregivers. More disproportionate giving of instrumental and emotional support was associated with higher levels of low-grade inflammation. In addition, interaction effects revealed that disproportionate social support is most detrimental to inflammatory and metabolic outcomes for families with greater socioeconomic disadvantage. That is, parents from lower-income households who gave more emotional support than they received had higher low-grade inflammation compared with those from higher-income households. We observed a slightly different pattern with respect to instrumental support, although effects were still largest among those from lower SES households. With respect to disproportionate instrumental support, parental caregivers with lower incomes who received more instrumental support than they gave had higher metabolic composite scores compared with parents from higher-income households. Parallel interaction patterns were observed with a second variable of family socioeconomic circumstances, interviewer-rated chronic financial stress, and effect sizes were in the small to medium range.

We extend prior literature on the physical health effects of giving and receiving social support (21,23) by investigating the physiological effects of their relative balance. We build on two known previous studies on this topic (22,41) by conducting biological assessments of metabolic and inflammatory outcomes that help reduce limitations related to self-reported health and work absence reports in prior studies. This study also extends our understanding of disproportionate social support by demonstrating differential effects by support type (emotional versus instrumental) and by family socioeconomic indicators.

With respect to the findings of the costs of disproportionately giving emotional support, prior literature suggests that giving more support than one receives is associated with a range of negative emotions such as resentment, burden, and dissatisfaction (38,39). These types of emotions have been shown in previous research to be associated with chronic diseases such as cardiovascular disease (66). In addition, parents with lower incomes often have more competing demands combined with fewer economic resources, which may mean fewer opportunities to alleviate stress associated with feelings of resentment, burden, or dissatisfaction. The resulting psychological strain might increase chronic stress particularly for lower-income parents, with downstream implications for physical health. These hypotheses are in line with prior research, which suggests that providing high levels of support such as caregiving can take a physiological toll through elevations in chronic stress (27,28). Contrastingly, parental caregivers with more socioeconomic resources who are giving disproportionate emotional support to others might have financial resources that allow them more opportunities to carve out time for themselves, to relax, and to access support from therapy and mental health services (67). Therefore, they may be able to give more emotional support than they receive without as much physiological cost. Future studies should empirically test these explanations and others to gain a clearer understanding of the underlying psychological and biological pathways.

Our findings suggest that disproportionate giving of instrumental social support is associated with poorer metabolic outcomes for everyone. However, disproportionate receiving of instrumental support was associated with higher metabolic composite scores primarily for lower-income parents. Receiving more support than one gives in a lower-SES context may be particularly harmful because it may signal dependency on others and/or an inability to reciprocate support. Prior literature suggests that receiving more than one gives is associated with feelings of indebtedness, guilt, or shame (38–40). Disproportionate receiving of instrumental support may elicit feelings of guilt and shame particularly for parents who have a lower SES. Feelings of guilt and shame have been shown in prior literature to predict negative physiological outcomes such as increased proinflammatory cytokine activity (68) and cortisol (69). We speculate that, in contrast, parents with higher SES who receive more instrumental support than they give may in part be able to compensate for some of the instrumental help they receive and hence do not have to feel guilty or ashamed for receiving high levels of instrumental support. Future studies should examine these potential explanations to gain a clearer understanding of the patterns observed here.

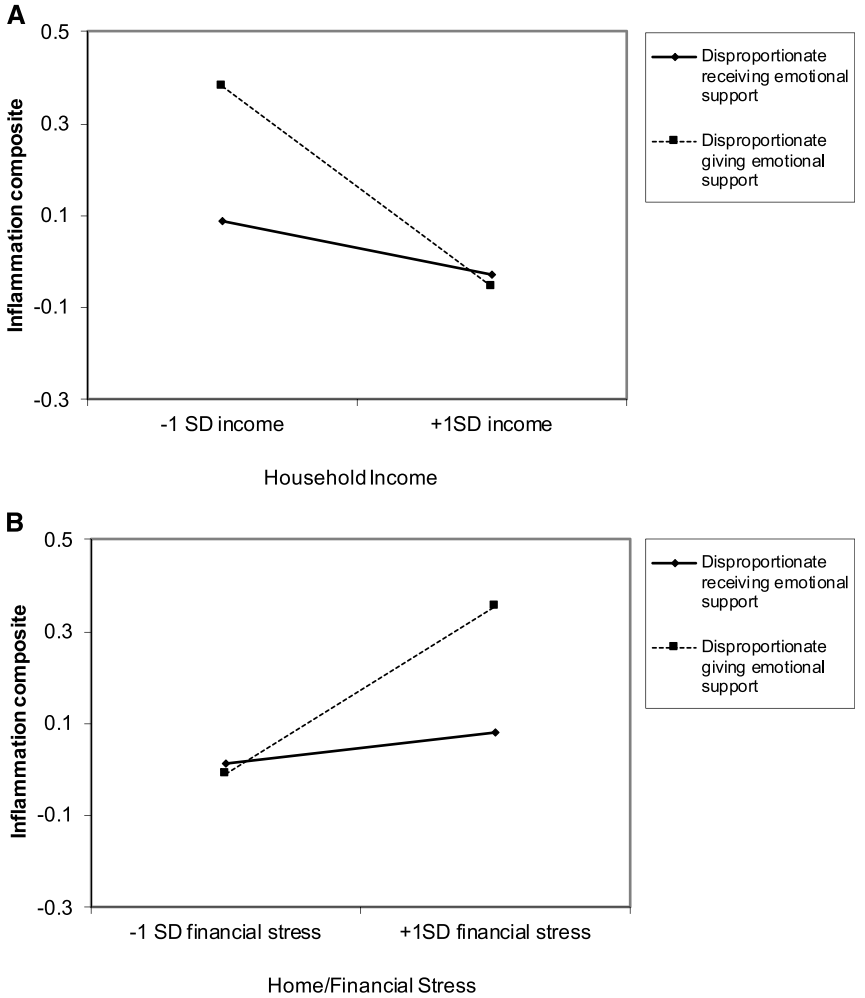

FIGURE 2. The effects of family socioeconomic variables and disproportionate emotional social support on low-grade inflammation. Multiple linear regression analyses that controlled for age, race, and sex were used. The lines represent estimated regression lines for +1 SD emotional support (representing those who disproportionately give more emotional support than they receive) and −1 SD emotional support (representing those who disproportionately receive more emotional support than they give). The inflammation composite was calculated by averaging standardized interleukin 6 and high-sensitivity C-reactive protein (CRP) scores. The two figures depict significant interaction effects for each family socioeconomic variable: income (A) and an interview-based measure of financial stress (B).

Lastly, there was evidence for physiological specificity in the interactive effects. Disproportionate emotional support interactions predicted inflammatory but not metabolic composites. Disproportionate instrumental support interactions predicted metabolic but not inflammation composite scores. The literature on disproportionate social support is relatively small, so to date, no studies have differentiated between social support types when examining the mental and/or physical health outcomes. However, these findings are consistent with broader research documenting the independent effects of emotional and instrumental support on well-being (70). It is plausible that independent mechanisms of emotional versus instrumental support would extend to differences in biological effects by disproportionate support type. Furthermore, although metabolic and inflammatory processes are interrelated with overlapping networks and demonstrated bidirectional effects (71), they also reflect different psychobiological mechanisms that may account for different effects (72). Thus, although speculative, it is possible, for example, that instrumental support is linked to metabolic outcomes because instrumental support is a more physically active type of support (e.g., involving helping others with their responsibilities and helping to take people places or to take care of them when they are sick), which may then be linked to the types of obesity-related measures found in our metabolic composite. In contrast, emotional support may have more direct brain-immune connections, given the more emotion-related pathways. Future research should examine if similar patterning of physiological effects by disproportionate social support type is observed in other samples. If so, one could leverage experimental and/or within-person designs to explore psychosocial and biological mechanisms.

LIMITATIONS

This study had several limitations. First, because of our cross-sectional analyses, we cannot determine causality or directionality of effects. Future research will benefit from prospective longitudinal designs

or experimental manipulations. Second, given the novelty of our study hypotheses, our disproportionate social support scores are not established, validated measures. They are, however, consistent with approaches used in previous literature (38,51). Second, it is important to note that the social support measure used in this study assesses people's perceptions of the support they give and receive, which might or might not correspond to objective reality. Objective measures of social support (apart from network size measures) can be difficult to operationalize, although using other reporters or behavioral observation measures might be beneficial in future studies. In addition, there are multiple ways in which one could conceptualize disproportionate support, and this study reflects merely one approach (contrasting giving versus receiving). There are other approaches—such as contrasting effort versus rewards or contrasting support provision versus one's capacity to provide support—that were not assessed in the present study but that would be informative for future studies to investigate. Furthermore, the social support questionnaire used in this study was designed to probe emotional and instrumental support broadly across all social relationships. However, one limitation of this approach is that, as a result, it is impossible to know which relationships individuals were referring to when answering the items. It may be that disproportionate giving is more normative in some types of relationships (parenting a child) but more burdensome in other types of relationships (romantic partners or friends). Although the approach of assessing social relationships generally (without prompts for specific individuals) is common in established social support measures (e.g., Interpersonal Support Evaluation List (73)), if future studies were to assess giving versus receiving support using separate probes for different sources of social support, one could more precisely determine whether disproportionate giving or receiving in a specific type of relationship is most detrimental to health. In addition, effect sizes in this study were relatively modest, although perhaps normative for behavioral and observational studies where health outcomes are expected to be determined by a multitude of factors. Lastly, our sample consisted of parental caregivers of a child with asthma, was mostly women, and tended to be higher in SES. It remains unclear whether similar patterns arise in other caregivers (i.e., of older adults) and/or in men. Future studies should attempt to replicate these findings across different types of caregiving situations, examine sex differences, and include an emphasis on lower-SES samples.

CONCLUSIONS

The present study examines an understudied support-related predictor: the relative balance of support given versus received. The findings demonstrate that disproportionate support interacts with family socioeconomic variables. Among parental caregivers who give more emotional support than they receive, those with greater socioeconomic disadvantage or difficulties exhibit the greatest inflammation. Second, among those who receive more instrumental support than they give, parents with more socioeconomic disadvantage or difficulties again experience greater costs in terms of higher metabolic scores compared with socioeconomically advantaged parents who also receive disproportionate support. These patterns highlight how the balance of support given versus received is an understudied aspect of social support with implications for physical health. Future research and interventions will need to not only examine if social support balance can causally promote better health but also specify how individuals can achieve an optimal balance of support given and received, with careful attention for how this may differ by type of social support and socioeconomic context.

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REFERENCES

1. House JS, Landis KR, Umberson D. Social relationships and health. Science 1988;24:540–5.
2. Holt-Lunstad J, Uchino BN. Social support and health. Health Behav Theory Res Pract 2015;183–204.
3. Holt-Lunstad J, Smith TB, Layton JB. Social relationships and mortality risk: a meta-analytic review. PLoS Med 2010;7:e1000316.
4. Barth J, Schneider S, von Kanel R. Lack of social support in the etiology and the prognosis of coronary heart disease: a systematic review and meta-analysis. Psychosom Med 2010;72:229–38.
5. Lutgendorf SK, Sood AK, Anderson B, McGinn S, Maiseri H, Dao M, Sorosky JI, de Geest K, Ritchie J, Lubaroff DM. Social support, psychological distress, and natural killer cell activity in ovarian cancer. J Cardiovasc Nurs 2005;20:162–9.
6. Kiecolt-Glaser JK, Loving TJ, Stowell JR, Malarkey WB, Lemeshow S, Dickinson SL, Glaser R. Hostile marital interactions, proinflammatory cytokine production, and wound healing. Arch Gen Psychiatry 2005;62:1377–84.
7. Nurullah AS. Received and provided social support: a review of current evidence and future directions. Am J Health Stud 2012;27:173–88.
8. Walster EG, Walster W, Berscheid E. Equity: Theory and Research. Boston, MA: Allyn and Bacon; 1978.
9. Cohen S, Syme SL. Social Support and Health. New York, NY: Academic Press; 1985.
10. Nausheen B, Gidron Y, Peveler R, Moss-Morris R. Social support and cancer progression: a systematic review. J Psychosom Res 2009;67:403–15.
11. Shor E, Roelfs DJ, Yogev T. The strength of family ties: a meta-analysis and meta-regression of self-reported social support and mortality. Soc Netw 2013; 35:626–38.
12. Uchino BN. Social support and health: a review of physiological processes potentially underlying links to disease outcomes. J Behav Med 2006;29:377–87.
13. Yang YC, Schorpp K, Harris KM. Social support, social strain and inflammation: evidence from a national longitudinal study of U.S. adults. Soc Sci Med 2014; 107:124–35.
14. Miller GE, Cohen S, Ritchey AK. Chronic psychological stress and the regulation of pro-inflammatory cytokines: a glucocorticoid-resistance model. J Health Psychol 2002;21:531–41.
15. Penninx BW, Van Tilburg T, Kriegsman DM, Deeg DJ, Boeke AJ, van Eijk JT. Effects of social support and personal coping resources on mortality in older age: the Longitudinal Aging Study Amsterdam. Am J Epidemiol 1997;146:510–9.
16. Lyyra TM, Heikkinen RL. Perceived social support and mortality in older people. J Gerontol 2006;61:S147–52.
17. Blazer DG. Social support and mortality in an elderly community population. Am J Epidemiol 1982;115:684–94.
18. Scarapicchia TMF, Amireault S, Faulkner G, Sabiston CM. Social support and physical activity participation among healthy adults: a systematic review of prospective studies. Int Rev Sport Exerc Psychol 2017;10:50–83.
19. Leung J, Pachana NA, McLaughlin D. Social support and health-related quality of life in women with breast cancer: a longitudinal study. Psychooncology 2014;23:1014–20.
20. Inagaki TK, Orehek E. On the benefits of giving social support: when, why, and how support providers gain by caring for others. Curr Dir Psychol Sci 2017;26: 109–13.
21. Brown SL, Nesse RM, Vinokur AD, Smith DM. Providing social support may be more beneficial than receiving it: results from a prospective study of mortality. Psychol Sci 2003;14:320–7.
22. Vaananen A, Buunk BP, Kivimaki M, Pentti J, Vahtera J. When it is better to give than to receive: long-term health effects of perceived reciprocity in support exchange. J Pers Soc Psychol 2005;89:176–93.

23. Piferi RL, Lawler KA. Social support and ambulatory blood pressure: an examination of both receiving and giving. Int J Psychophysiol 2006;62:328–36.
24. Inagaki TK, Eisenberger NI. Giving support to others reduces sympathetic nervous system–related responses to stress. Psychophysiology 2016;53:427–35.
25. Vitaliano PP, Zhang J, Scanlan JM. Is caregiving hazardous to one's physical health? A meta-analysis. Psychol Bull 2003;129:946–72.
26. Schulz R, Beach SR. Caregiving as a risk factor for mortality: the Caregiver Health Effects Study. JAMA 1999;282:2215–9.
27. Kiecolt-Glaser JK, Glaser R, Gravenstein S, Malarkey WB, Sheridan J. Chronic stress alters the immune response to influenza virus vaccine in older adults. Proc Natl Acad Sci U S A 1996;93:3043–7.
28. Glaser R, Kiecolt-Glaser JK. Chronic stress modulates the virus-specific immune response to latent herpes simplex virus type 1. Ann Behav Med 1997;19:78–82.
29. Lovell B, Wetherell MA. The cost of caregiving: endocrine and immune implications in elderly and non elderly caregivers. Neurosci Biobehav Rev 2011;35:1342–52.
30. Rohleder N, Marin TJ, Ma R, Miller GE. Biologic cost of caring for a cancer patient: dysregulation of pro- and anti-inflammatory signaling pathways. J Clin Oncol 2009;27:2909–15.
31. Gouin JP, Hantsoo L, Kiecolt-Glaser JK. Immune dysregulation and chronic stress among older adults: a review. Neuroimmunomodulation 2008;15(4–6):251–9.
32. Antonucci TC, Jackson JS. The role of reciprocity in social support. In: Sarason BR, Sarason IG, Pierce GR, editors. Social Support: An Interactional View. New York, NY: John Wiley & Sons; 1990:173–98.
33. Kessler RC, McLeod JD, Wethington E. The costs of caring: a perspective on the relationship between sex and psychological distress. In: Sarason IG, Sarason BR, editors. Social Support: Theory, Research and Applications. Dordrecht: Springer; 1985:491–506.
34. Uchino BN, Bosch JA, Smith TW, Carlisle M, Birmingham W, Bowen KS, Light KC, Heaney J, O'Hartaigh B. Relationships and cardiovascular risk: perceived spousal ambivalence in specific relationship contexts and its links to inflammation. J Health Psychol 2013;32:1067–75.
35. Cohen S. Social relationships and health. Am Psychol 2004;59:676–84.
36. Rook KS. Social networks in later life: weighing positive and negative effects on health and well-being. Curr Dir Psychol Sci 2015;24:45–51.
37. Fyrand L. Reciprocity: a predictor of mental health and continuity in elderly people's relationships? A review. Curr Gerontol Geriatr Res 2010;2010:340161.
38. Ingersoll-Dayton B, Antonucci TC. Reciprocal and nonreciprocal social support: contrasting sides of intimate relationships. J Gerontol 1988;43:S65–73.
39. Jou YH, Fukada H. Stress, health, and reciprocity and sufficiency of social support: the case of university students in Japan. J Soc Psychol 2002;142:353–70.
40. Newsom JT, Schulz R. Caregiving from the recipient's perspective: negative reactions to being helped. Health Psychol 1998;17:172–81.
41. Chandola T, Marmot M, Siegrist J. Failed reciprocity in close social relationships and health: findings from the Whitehall II study. J Psychosom Res 2007;63:403–11.
42. Manczak EM, DeLongis A, Chen E. Does empathy have a cost? Diverging psychological and physiological effects within families. Health Psychol 2016;35:211–8.
43. Braveman PA, Cubbin C, Egerter S, Williams DR, Pamuk E. Socioeconomic disparities in health in the United States: what the patterns tell us. Am J Public Health 2010;100(Suppl 1):S186–96.
44. Adler NE, Boyce T, Chesney MA, Cohen S, Folkman S, Kahn RL, Syme SL. Socioeconomic status and health. The challenge of the gradient. Am Psychol 1994; 49:15–24.
45. Feinstein JS. The relationship between socioeconomic status and health: a review of the literature. Milbank Q 1993;71:279–322.
46. Cohen S, Wills TA. Stress, social support, and the buffering hypothesis. Psychol Bull 1985;98:310–57.
47. Baum A, Garofalo JP, Yali AM. Socioeconomic status and chronic stress: does stress account for SES effects on health? Ann N Y Acad Sci 1999;896:131–44.
48. Santiago CD, Wadsworth ME, Stump J. Socioeconomic status, neighborhood disadvantage, and poverty-related stress: prospective effects on psychological syndromes among diverse low-income families. J Econ Psychol 2011;32:218–30.
49. Lam PH, Levine CS, Chiang JJ, Shalowitz MU, Story RE, Hayen R, Sinard RN, Chen E. Family obligations and asthma in youth: the moderating role of socioeconomic status. Health Psychol 2018;37:968–78.
50. Shakespeare-Finch J, Obst PL. The development of the 2-Way Social Support Scale: a measure of giving and receiving emotional and instrumental support. J Pers Assess 2011;93:483–90.
51. Brown SD, Brady T, Lent RW, Wolfert J, Hall S. Perceived social support among college students: three studies of the psychometric characteristics and counseling uses of the Social Support Inventory. J Couns Psychol 1987;34:337–54.
52. Jou YH, Fukada H. Effects of social support on adjustment of Chinese students in Japan. J Soc Psychol 1995;135:39–47.
53. Levine ME, Crimmins EM. A comparison of methods for assessing mortality risk. Am J Hum Biol 2014;26:768–76.
54. Hawkley LC, Lavelle LA, Berntson GG, Cacioppo JT. Mediators of the relationship between socioeconomic status and allostatic load in the Chicago Health, Aging, and Social Relations Study (CHASRS). Psychophysiology 2011;48:1134–45.
55. Seplaki CL, Goldman N, Glei D, Weinstein M. A comparative analysis of measurement approaches for physiological dysregulation in an older population. Exp Gerontol 2005;40:438–49.
56. Reilly SM, Saltiel AR. Adapting to obesity with adipose tissue inflammation. Nat Rev Endocrinol 2017;13:633–43.
57. Levine CS, Basu D, Chen E. Just world beliefs are associated with lower levels of metabolic risk and inflammation and better sleep after an unfair event. J Pers 2017;85:232–43.
58. Ross K, Martin T, Chen E, Miller GE. Social encounters in daily life and 2-year changes in metabolic risk factors in young women. Dev Psychopathol 2011;23: 897–906.
59. Ekelund U, Brage S, Franks PW, Hennings S, Emms S, Wareham NJ. Physical activity energy expenditure predicts progression toward the metabolic syndrome independently of aerobic fitness in middle-aged healthy Caucasians: the Medical Research Council Ely Study. Diabetes Care 2005;28:1195–200.
60. Eisenmann JC. On the use of a continuous metabolic syndrome score in pediatric research. Cardiovasc Diabetol 2008;7:17.
61. Cho HJ, Kivimäki M, Bower JE, Irwin MR. Association of C-reactive protein and interleukin-6 with new-onset fatigue in the Whitehall II prospective cohort study. Psychol Med 2013;43:1773–83.
62. Harris TB, Ferrucci L, Tracy RP, Corti MC, Wacholder S, Ettinger WH Jr., Heimovitz H, Cohen HJ, Wallace R. Associations of elevated interleukin-6 and C-reactive protein levels with mortality in the elderly. Am J Med 1999;106: 506–12.
63. Giovannini S, Onder G, Liperoti R, Russo A, Carter C, Capoluongo E, Pahor M, Bernabei R, Landi F. Interleukin-6, C-reactive protein, and tumor necrosis factor-alpha as predictors of mortality in frail, community-living elderly individuals. J Am Geriatr Soc 2011;59:1679–85.
64. Hammen C, Marks T, Mayol A, DeMayo R. Depressive self-schemas, life stress, and vulnerability to depression. J Abnorm Psychol 1985;94:308–19.
65. Schreier HM, Chen E. Low-grade inflammation and ambulatory cortisol in adolescents: interaction between interviewer-rated versus self-rated acute stress and chronic stress. Psychosom Med 2017;79:133–42.
66. Kubzansky LD, Kawachi I. Going to the heart of the matter: do negative emotions cause coronary heart disease? J Psychosom Res 2000;48(4–5):323–37.
67. Steele L, Dewa C, Lee K. Socioeconomic status and self-reported barriers to mental health service use. Can J Psychiatry 2007;52:201–6.
68. Kemeny ME, Gruenewald TL, Dickerson SS. Shame as the emotional response to threat to the social self: implications for behavior, physiology, and health. Psychol Inq 2004;15:153–60.
69. Gruenewald TL, Kemeny ME, Aziz N, Fahey JL. Acute threat to the social self: shame, social self-esteem, and cortisol activity. Psychosom Med 2004;66: 915–24.
70. Morelli SA, Lee IA, Arnn ME, Zaki J. Emotional and instrumental support provision interact to predict well-being. Emotion 2015;15:484–93.
71. Raval FM, Nikolajczyk BS. The bidirectional relationship between metabolism and immune responses. Discoveries (Craiova) 2013;1:e6.
72. Palavra F, Reis F, Marado D, Sena A. Biomarkers of Cardiometabolic Risk, Inflammation and Disease. Cham, Switzerland: Springer; 2015.
73. Cohen S, Hoberman H. Positive events and social supports as buffers of life change stress. J Appl Soc Psychol 1983;13:99–12.