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High social strain and physical health: Examining the roles of anxious arousal, body mass index, and inflammation

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ABSTRACT

Social relationships have powerful effects on physical health. Indeed, high social strain (i.e., frequent negative interactions with friends, family, or one's partner) increases risk of morbidity and mortality. Frequent social strain leads to anxious arousal and an increased body mass index (BMI), both of which may be underlying mechanisms for the association between social strain and health given that persistent anxious arousal promotes damaging biological and behavioral conditions contributing to increased inflammation. When chronic, heightened inflammation results in the deterioration of overall health. The purpose of the current study was to investigate anxious arousal, BMI, and inflammation as underlying mechanisms of the association between social strain and health. A sample of 763 middle aged adults participating in the Midlife in the United States (MIDUS 2) study completed self-report measures of social strain, anxious arousal, and physical health. Blood collection and a physical examination were completed to measure BMI and inflammation. Using 5000 bootstrap samples, results indicated that greater social strain was associated with poorer self-reported health (SRH) due to the serial pathway from high anxious arousal to BMI and inflammation.

1. Introduction

Interpersonal relationships have powerful effects on health and well-being. In fact, the impact of negative interpersonal relationships on risk of mortality are comparable to other risk factors such as smoking and obesity (Holt-Lunstad et al., 2010). Much attention has been given to the advantageous health outcomes associated with social support, though there is a tendency to erroneously equate frequent social interaction with high social support (Rook, 1984). This is problematic given that the harmful effects on health from frequent negative social interactions may be enough to outweigh the protective health effects of being around other individuals (Yang et al., 2014).

Social strain refers to the frequency and degree to which an individual experiences negative interactions with friends, family members, or significant others. Negative social interactions have consistently been found to be associated with poor health outcomes (Lincoln, 2000; Rook, 1984; Sneed and Cohen, 2014; Yang et al., 2014). Studies have linked higher social strain with poorer self-reported health, greater functional limitations, and a higher number of health conditions including hypertension and poor glycemic control in individuals with type 1 diabetes (Newsom et al., 2008; Helgeson et al., 2009; Sneed and Cohen, 2014). Frequent negative interactions with close others may lead to deleterious health outcomes because negative interactions are highly salient and evoke a stress response; an individual who experiences frequent strenuous interactions with family members, friends, or significant others may ruminate on the events, sustaining physiological activation that may negatively impact health (Umberson et al., 2006).

Because forming interpersonal attachments is a fundamental human motivation (Baumeister and Leary, 1995), conflict and strain in social relationships may be particularly upsetting. Several studies have found that negative social interactions are associated with poorer psychological wellbeing and increased symptoms of anxiety (Lincoln, 2000; Rook, 1984; Newsom et al., 2005; Walen and Lachman, 2000; Abbey et al., 2010). Empirical evidence also suggests that non-supportive social interactions are associated with enhanced hypothalamic pituitary adrenal (HPA) and sympathetic nervous system (SNS) activation (Seeman and McEwen, 1996), perhaps stemming from a threat to one's sense of social belonging (Baumeister and Leary, 1995). These physiological systems underlie the experience of anxious arousal, a subset of somatic anxiety symptoms such as racing heart, upset stomach, and sweating. Somatic symptoms of anxiety are more strongly linked with levels of inflammation than cognitive symptoms of anxiety such as worry (Duivis et al., 2013). This evidence provides support for the idea that anxious arousal specifically, rather than general anxiety and distress, may be a pathway linking social strain with poor physical health.

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Accordingly, frequently negative social interactions may promote increased anxious arousal over time and contribute to poorer health.

Physiological stress promoted by conflict with close others impacts inflammatory cytokine production through activation of nuclear factor-Kappa B (NF-κB) (Bierhaus et al., 2003). Indeed, in a study examining perceived social strain in adults, higher social strain was associated with higher interleukin-6 (IL-6), C-reactive protein (CRP), fibrinogen, E-selectin, and intracellular adhesion molecular-1 (ICAM-1) (Yang et al., 2014). Because anxious arousal is also associated with heightened inflammation (Duivis et al., 2013), the previously established association between high social strain and inflammation may be due to increased anxious arousal.

Persistent anxious arousal may promote damaging biological and behavioral conditions that are associated with higher body mass index (BMI), a common indicator of overweight and obesity. For example, obesity and anxiety disorders are closely related due to the dysregulated biological pathways they share (Petry et al., 2008). Adipocytes in white adipose tissue secrete inflammatory cytokines such as IL-6 and tumor necrosis factor alpha (TNF-α), putting those with higher BMI at risk of increased inflammation (Shelton and Miller, 2010). Unhealthy dietary and sedentary behaviors may also be shared among those with heightened anxious arousal and BMI (Lopresti and Drummond, 2013). Accordingly, higher BMI may also contribute to increased inflammation (Lopresti and Drummond, 2013). Heightened inflammation over time can result in increased incidence of disease and mortality (Ershler and Keller, 2000; Kiecolt-Glaser et al., 2010).

The consequences of high social strain on the body and mind are undoubtedly harmful. Considering the known associations between interpersonal conflict, anxiety, body weight, and inflammation, (Kiecolt-Glaser et al., 2010; Rook, 1984), we hypothesized that individuals who reported more strenuous social relationships would also report poorer physical health. We predicted that the association between social strain and physical health would be explained by anxious arousal, BMI, and inflammation.

2. Material and methods

2.1. Sample

Data were obtained from the Midlife in the United States (MIDUS 2) study from 2004 to 2006, a follow-up to the original MIDUS 1 study in 1995-1996. The MIDUS study examines behavioral, psychological, and social factors in a nationally representative sample of middle aged adults. The baseline sample of participants were recruited through random digit dialing, and wave 2 participants were recruited from the original MIDUS 1 study between 7.8 and 10.4 years after wave 1. Complete data were obtained from 763 individuals aged 35 to 86 (M_{age} = 55.52 SD = 11.61). Participants in the MIDUS 2 were more likely to be female and Caucasian than participants from the original MIDUS 1 sample (Chirinos et al., 2017).

2.2. Measures

Participants mailed in self-administered questionnaires assessing social strain at two time points as part of the MIDUS 1 and MIDUS 2 projects. A subset of participants attended a subsequent 24 h stay at one of three General Clinical Research Centers (UCLA, University of Wisconsin, and Georgetown University), depending on geographic location of the participant, as part of the in-person Biomarker session of the MIDUS study (i.e., Project 4). Participants who took part in Project 4 completed the in-person session an average of 25.32 months (SD = 14.22) after Project 1 self-assessed questionnaires. SRH and anxious arousal symptoms were assessed with self-administered questionnaires the night of Day 1 of the in-person visit. Fasting blood samples were collected from each participant before breakfast on the morning of Day 2 to assess inflammation. Height and weight measurements from a physical examination on Day 2 were used to compute BMI. Additional details on the biomarker session protocol have been described elsewhere (Dienberg Love et al., 2010). All participants provided informed consent and the study was approved by the Institutional Review Boards at each site.

2.2.1. Social strain

Social strain was measured using four indices of negative social interaction with spouse/partner, friends, and family members. All items were answered on a 4-point scale ranging from 1 (often) to 4 (never). Items included, “How often do they criticize you?”, “How often do they make too many demands on you?”, “How often do they let you down when you are counting on them?”, “How often do they get on your nerves?” Two additional items were included to assess social strain from partner/spouse: “How often does he or she (i.e., partner/spouse) argue with you?” and “How often does he or she (i.e., partner/spouse) make you feel tense?” Strain scores from family members, friends, and spouse/partner were averaged to form a total social strain variable, where higher scores indicated higher total social strain. Internal consistency for social strain in the present study was good (α = .85). Social strain was measured at two time points: first as part of the MIDUS 1 study, and again 9 years later as part of the MIDUS 2 follow up. In ancillary analyses, a composite score of strain at both time points was created as an indicator of chronicity.

2.2.2. Anxious arousal

Anxious arousal was measured using the 62-item Mood and Anxiety Symptom Questionnaire (MASQ), designed to assess symptoms of general distress (Clark and Watson, 1991). The anxious arousal subscale included 17 items where participants were asked to report the degree to which they experienced certain feelings or sensations (e.g., pain in chest), in the previous week on a 5-point scale ranging from 1 (very slightly or not at all) to 5 (extremely). Other items included in the MASQ measure symptoms of general distress and symptoms of depression and were therefore not included. Internal consistency for the anxious arousal scale was acceptable (α = .76).

2.2.3. Inflammation

Serum interleukin-6 (IL-6) was measured using high-sensitivity enzyme-linked immunosorbent assay (ELISA) (Quantikine, R&D Systems, Minneapolis, MN). To measure C-reactive protein (CRP) and fibrinogen, a particle enhanced immunonepholometric assay was utilized (BNII nephelometer, Dade Behring Inc., Deerfield, IL). E-selectin (Parameter Human sE-Selectin Immunoassay; R&D Systems, Minneapolis, MN) and soluble intracellular adhesion molecule-1 (ICAM-1; Parameter Human sICAM-1 Immunoassay; R&D Systems, Minneapolis, MN) were measured using ELISA kits. All intra- and inter-assay coefficients of variance were < 10%, which is within the acceptable range. As is common when analyzing biomarkers, distributions for each marker of inflammation were skewed. As a result, log-transformations were utilized to normalize each distribution. Following transformation, markers were z-standardized and combined to form a composite indicator of inflammation.

2.2.4. Physical health

Self-reported physical health (SRH) was measured via a single item from the 36-item Medical Outcomes Study Short Form (SF-36) answered on a 5-point scale ranging from 1 (excellent) to 5 (poor): “In general, would you say your PHYSICAL HEALTH is excellent, very good, good, fair, or poor?” This self-reported physical health item is widely used in both research and clinical settings due to its strong associations with clinically relevant markers of health status (Meng et al., 2014). The item has been found to be reliably comparable to longer instruments assessing physical health and predicting morbidity and mortality (DeSalvo et al., 2005). Additionally, findings are consistent between the single-item and multi-item scales for measuring self-

reported health (Murdock et al., 2016).

2.3. Covariates

Participant age, gender, race/ethnicity, and use of nonsteroidal anti-inflammatory drugs (NSAIDs) were assessed with self-administered questionnaires and included as covariates in the mediation analyses. Social support was added as a covariate in ancillary analyses to determine whether the relationship between social strain and health goes above and beyond the effects of social support. Social support was self-assessed with a mail-in questionnaire as part of the MIDUS 2 Project 1. All items were answered on a 4-point scale ranging from 1 (a lot) to 4 (not at all). Items included, "How much do members of your family really care about you?", "How much do they understand the way you feel about things?", "How much can you rely on them for help if you have a serious problem?", and "How much can you open up to them if you need to talk about your worries?" Support scores from family members, friends, and spouse/partner were averaged to form a total social support variable, where lower scores indicated higher total social support. Use of alcoholic beverages was also included in ancillary analyses. Participants were asked to rate the extent to which they drank alcoholic beverages within the past month on a scale ranging from 1 (everyday) to 6 (never drinks). Study participants were also asked to rate whether or not they were a current smoker, which was utilized as a covariate in ancillary analyses.

2.4. Data analysis

SPSS statistical software (IBM, 2012) was used for all data analyses. The model presented in Fig. 1 was tested using the PROCESS macro for SPSS (Hayes, 2013) to evaluate serial mediation. 5000 bootstrap samples were used to analyze indirect effects. Age, gender, race/ethnicity, and NSAID use were included as covariates in the mediation analyses. These variables have been shown to be associated with inflammation and self-reported health (Brüünsgaard and Pedersen, 2003; Klein and Flanagan, 2016; Cheng et al., 2013; Schmeer and Tarrence, 2018). Social support, use of alcoholic beverages, and current smoking status were included as covariates in ancillary analyses.

3. Results

Means and standard deviations for study variables are presented in Table 1. Using zero-order correlations (see Table 2) higher social strain was associated with greater anxious arousal, poorer self-reported health, and younger age. Greater anxious arousal was associated with higher BMI and inflammation, poorer SRH, non-white race/ethnicity, and female gender. Higher BMI was associated with greater inflammation, poorer self-reported health, non-white race/ethnicity, male gender, and use of NSAIDs. Inflammation was associated with

Table 1
Descriptive statistics for study variables.

Measure	Mean (SD) or %
Social strain	5.94 (1.24)
Anxious arousal	21.39 (4.49)
Body mass index	29.09 (5.71)
Interleukin-6 (pg/ml)	
Raw	2.74 (2.90)
Transformed	.30 (.32)
C-reactive protein (ug/mL)	
Raw	2.63 (3.89)
Transformed	.14 (.50)
Fibrinogen (mg/dL)	
Raw	337.90 (84.52)
Transformed	2.52 (.12)
E-selectin (ng/mL)	
Raw	41.94 (21.26)
Transformed	1.57 (.23)
ICAM-1 (ng/mL)	
Raw	288.12 (100.17)
Transformed	244 (.14)
Self-reported health	2.34 (0.90)
Age	55.52 (11.61)
Race/ethnicity	
White	96.3%
Non-white	3.7%
Gender	
Female	49.5%
Male	50.5%
Use of NSAIDs	
Yes	2.3%
No	97.7%

Note. ICAM = intracellular adhesion molecule; NSAID = non-steroidal anti-inflammatory drugs.

Table 2
Pearson correlations between study variables.

Variable	1	2	3	4	5	6	7	8	9
1. Social strain	–								
2. Anxious arousal	.23*	–							
3. Body mass index	.06	.10*	–						
4. Inflammation	.02	.10*	.44*	–					
5. Self-reported health	.13*	.35*	.26*	.23*	–				
6. Age	- .22*	.03	- .01	.19*	.04	–			
7. Race/ethnicity	- .03	- .08*	- .08*	- .01	- .07	.04	–		
8. Gender	.07	.09*	- .08*	.03	- .03	- .14*	.01	–	
9. NSAID use	.04	.08*	.11*	.10*	.16*	.09*	- .01	- .01	–

Note.
* $p < .05$; Gender coded as 1 = male and 2 = female. Race/ethnicity coded as 0 = non-white and 1 = white. NSAID = non-steroidal anti-inflammatory drug.

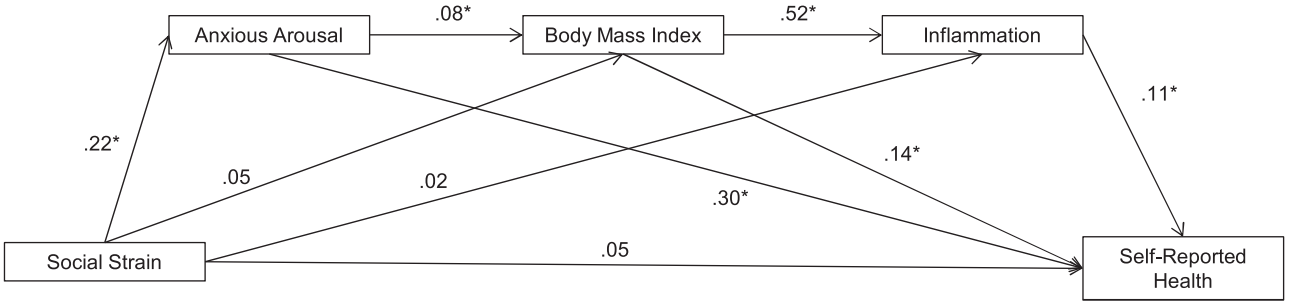

Fig. 1. A mediation model of associations between social strain, anxious arousal, body mass index, inflammation, and self-reported health. Standardized regression coefficients are presented. Indirect effects using 5000 bootstrap samples: anxious arousal (.066 *SE* = .013, 95% *CI* = .041, .093), body mass index (.007 *SE* = .005, 95% *CI* = - .002, .019), inflammation (.002, *SE* = .004, 95% *CI* = - .007, .011), and serial mediation (i.e., mediation in sequence; .003, *SE* = .001, 95% *CI* = .001, .002). Control variables included participant age, gender, race/ethnicity, and use of non-steroidal anti-inflammatory drugs. * $p <$ .05.

poorer SRH, older age, and use of NSAIDS. SRH was also associated with use of NSAIDs.

When testing the model presented in Fig. 1, higher social strain was associated with greater anxious arousal, but was not directly associated with BMI, inflammation, or SRH. Increased anxious arousal was associated with higher BMI and poorer SRH. Greater inflammation was also associated with poorer SRH. When examining indirect effects, the pathway from social strain to SRH via anxious arousal was significant given that zero was not included in the 95% confidence interval generated using 5000 bootstrap samples. The pathway from social strain to SRH via BMI and inflammation was not significant as zero was included in the confidence interval (.007, SE = .005, 95% CI = - .002, .019). Furthermore, the serial pathway from social strain to SRH via anxious arousal, BMI, and inflammation was significant, as expected (.003, *SE* = .001, 95% *CI* = .001, .002). In ancillary analyses, we tested whether or not the chronicity of social strain would be important by combining self-reports of social strain from multiple time points. In these analyses, all significant associations and indirect effects remained.

Given the cross-sectional nature of the data, caution is warranted when hypothesizing directionality of effects; however, we tested a reverse serial mediation model to determine if there was a significant indirect effect of SRH on social strain via inflammation, BMI, and anxious arousal. In the model (see Fig. 2), poorer SRH was associated with higher inflammation, BMI, and anxious arousal. Inflammation and BMI were no longer associated with higher anxious arousal. Anxious arousal remained significantly associated with social strain; however, inflammation, BMI, and SRH were not associated with social strain. Bootstrap analyses yielded a significant indirect effect of SRH on social strain via anxious arousal (.072, *SE* = .016, 95% *CI* = .042, .105); however, all other indirect effects were non-significant.

In ancillary analyses, we included social support as an additional covariate when testing the model represented in Fig. 1. These analyses yielded a non-significant association between anxious arousal and BMI (β = .07, *p* = .09), indicating that social support may reduce the strength of the association between anxious arousal and BMI. Although the reduction in the strength of this association was small, it led to a non-significant indirect effect for the serial pathway from social strain to SRH through anxious arousal, BMI, and inflammation (.001, *SE* = .001, 95% *CI* = .000, .002). Use of alcoholic beverages and smoking status were also examined as covariates to the model tested in Fig. 1. Unfortunately, data for use of alcoholic beverages was unavailable for 230 participants. All significant associations remained, but the serial mediation pathway was no longer significant (.002, *SE* = .001, 95% *CI* = .000, .004). Likewise, data for smoking status was unavailable for 436 participants. Including smoking status as an additional covariate yielded a non-significant association between anxious arousal and BMI (β = .02, *p* = .65). All other associations remained significant outside of the serial mediation pathway (.001, *SE* = .001, 95% *CI* = - .001, .003).

4. Discussion

Our findings indicated that those with greater social strain from family, friends, and significant other exhibited poorer self-reported health due to heightened anxious arousal, higher BMI, and increased inflammation. As outlined in Table 2, social strain and SRH were significantly associated (r = .13, *p* < .05); however, when including health-related factors (i.e., anxious arousal, BMI, and inflammation) in the regression model, the direct pathway was no longer significant, suggesting that those variables account for a significant portion of the association. Moreover, support exists for the relevance of measuring mediators linking the variables in the absence of a direct effect when other variables are included in regression analyses (Hayes, 2009). High social strain has been linked with poor psychological outcomes, including anxiety (Abbey et al., 2010). Anxiety is associated with higher BMI (Lopresti and Drummond, 2013). Additionally, symptoms of anxiety, especially somatic symptoms, are linked with elevated inflammation (Duivis et al., 2013). Accordingly, the study findings provide a potential pathway to explain the relationship between high social strain and poor physical health. Importantly, findings remained when including a host of covariates in the analyses (i.e., participant age, race/ethnicity, gender, use of NSAIDS, smoking status, and use of alcohol); however, the association between anxious arousal and BMI was reduced when including social support as a covariate in ancillary analyses. Such findings are consistent with research indicating that social support can benefit mental and physical health, even in the face of social strain. Indeed, having more positive interactions than social strain related interactions with close others is associated with better physical health, even if social strain is relatively high (e.g., Haase et al., 2016).

In order to determine if social strain over time contributed to self-assessment of physical health, we included a composite measure of social strain at two time points (i.e., 9 years). Findings were identical, likely due to a strong association between social strain at time 1 and time 2 (r = .89). The experience of social strain over an extended period of time may have contributed to the observed finding of poorer health; however, it will be necessary to measure other variables included in the model at more than one-time point in order to determine longitudinal effects. For example, because inflammation was only assessed at one-time point, it is impossible to completely establish temporal precedence. Analyses also showed a significant indirect effect of SRH on social strain due to anxious arousal. This may suggest that poor perceived health may evoke anxiety that in turn contributes to strained social relationships. Longitudinal study of these variables will be necessary to determine which direction of the association is more informative, or whether bi-directional associations develop over time.

The identification of anxious arousal and inflammation as mechanisms mediating the association between social strain and physical health creates opportunities for intervention. Although social strain cannot always be avoided in individuals' lives, interventions that target

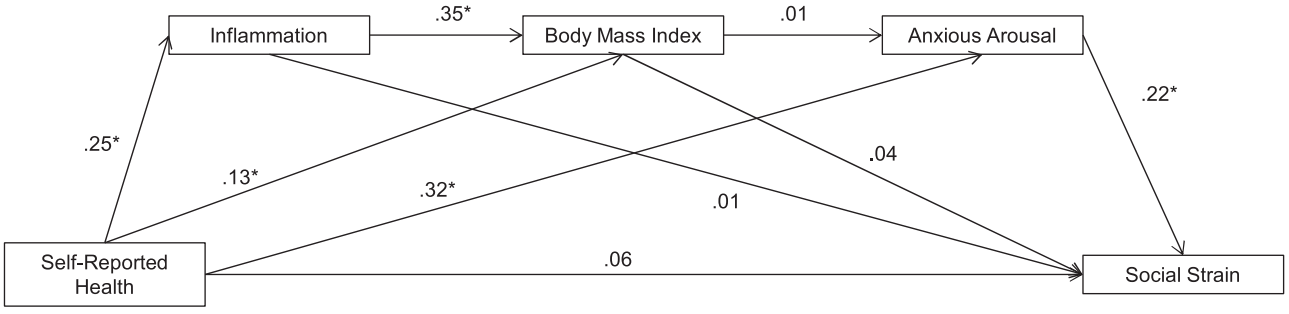

Fig. 2. A mediation model of associations between social strain, anxious arousal, body mass index, inflammation, and self-reported health. Standardized regression coefficients are presented. Indirect effects using 5000 bootstrap samples: anxious arousal (.072 *SE* = .016, 95% *CI* = .042, .105), body mass index (.005 *SE* = .006, 95% *CI* = - .005, .017), inflammation (.002, *SE* = .010, 95% *CI* = - .016, .021), and serial mediation (i.e., mediation in sequence; .001, *SE* = .001, 95% *CI* = - .001, .002). Control variables included participant age, gender, race/ethnicity, and use of non-steroidal anti-inflammatory drugs. * *p* < .05.

anxious arousal and associated inflammation could help to limit potential damage to mental and physical health due to strenuous relationships. Evidence supports the utility of Acceptance and Commitment Therapy (ACT) in improving clinical anxiety outcomes (Swain et al., 2013). Mindfulness-based techniques such as meditation have also been found to have beneficial effects on symptoms of anxiety. Meditation involves the intentional and repeated practice of activating the body's parasympathetic nervous system and has been shown to improve one's ability to manage anxiety (Hoge et al., 2013). Research has demonstrated the efficacy of mindfulness-based techniques in clinical settings, due to their association with lower levels of psychological distress, anxiety, and worry (Edenfield and Saeed, 2012). By curbing physiological activation, mindfulness meditation may be beneficial in reducing anxious arousal levels and precluding a subsequent increase in inflammation. Additionally, one study showed that a mindfulness meditation training intervention reduced IL-6 due to changes in functional connectivity in the brain (Creswell et al., 2016). By indirectly influencing inflammation, mindfulness meditation may be particularly useful for individuals who experience high social strain.

Chronic interpersonal conflict is associated with increased expression of inflammatory signaling molecules and creates a potential for hyper-inflammatory responses, aggravating inflammation-related disease pathogenesis (Miller et al., 2009). Individuals who have experienced chronic stress, such as those with strenuous relationships, may be primed for heightened inflammatory responses. In a study examining individuals with depression, those with more depressive symptoms produced more IL-6 in response to a laboratory stressor (Fagundes et al., 2013). This effect may explain why those with high social strain demonstrate increased inflammation. Worry and apprehension of conflict in social relationships may also be significant contributors to the association between social strain and physical health. Worry and anticipatory stress prolongs physiological activation, increasing cardiovascular, endocrinological, immunological, and neurovisceral activity (Brosschot et al., 2006). When an individual has experienced strain in their relationships, they may experience heightened reactivity in regards to anxious arousal in comparison to those with less social strain. Future work should include these potential factors to examine how they contribute to anxious arousal and inflammation.

Indirect mechanisms such as maladaptive health behaviors may also explain the association between high social strain and poor physical health, especially as they relate to BMI. High perceived stress, a condition likely experienced by an individual with strenuous relationships, is associated with a higher fat diet, less frequent exercise, and cigarette smoking (Ng and Jeffrey, 2003). Daily stressors have also been found to be associated with maladaptive metabolic and inflammatory responses to high-fat meals (Kiecolt-Glaser et al., 2015). Individuals who take part in more frequent physical activity tend to have lower BMI and lower inflammatory biomarker concentrations (Beavers et al., 2010). Accordingly, modifiable health behaviors such as physical activity and diet may be imperative for individuals who suffer from high social strain, and should be explored in future work.

The present study is limited by its cross-section design. Although the reverse model tested provides initial evidence of directionality, future studies should evaluate social strain, anxious arousal, BMI, inflammation, and physical health over time to determine directionality of the effect. This study is also limited in its measurement of the social strain construct. Due to the cross-sectional format of the measure and the use of few items to operationalize strain, we cannot be sure whether the nature of the strenuous relationships or frequent conflict was promoting the observed effects. Examining the duration and intensity of strain in close relationships, in addition to instances of conflict in those relationships in a daily diary format, would provide insight on the link between conflict and social strain. The majority of the sample examined in this study was white (i.e., 96.3%), and therefore, generalizations about the overall population cannot be inferred.

5. Conclusion

Current findings add to the literature by identifying anxious arousal, BMI, and inflammation as mechanisms underlying the association between high social strain and poor health. The study demonstrates how social strain in interpersonal relationships can lead to both psychological and physiological outcomes that increase one's likelihood of poor health. Identification of this serial pathway is significant for the development of targeted interventions for those at risk of poor health outcomes.

Conflict of interest

The authors of this manuscript have no conflict of interest to disclose.

Declarations of interest

None.

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