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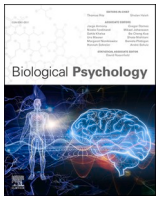

Social strain and cardiovascular reactivity to acute psychological stress: Examining the mediating role of self-esteem

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ABSTRACT

The primary aims of the current study were to (1) examine the association between perceptions of social strain from a spouse/partner, other family members and friends, and cardiovascular reactivity to acute stress, and (2) to identify if the association between perceived social strain and cardiovascular reactivity to acute stress was mediated via self-esteem. A sample of 659 participants completed measures assessing social strain from a spouse/partner, from other family members and from friends, and completed a standardized cardiovascular reactivity protocol consisting of resting baseline and stressor phase (mental arithmetic and Stroop). Systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were monitored throughout the baseline and stressor phases. Greater social strain from family members and from friends were significantly associated with blunted blood pressure reactivity to acute psychological stress. Moreover, diminished self-esteem significantly mediated the association between social strain from all sources and both cardiovascular and psychological responses to acute stress. Results for the association between social strain and cardiovascular reactivity appeared to remain largely robust in follow-up analyses adjusting for perceived social support from various sources. However, after adjusting for depressive symptoms, both the direct and mediating effects of self-esteem on cardiovascular reactivity became non-significant. The direct association between social strain from family members on diminished blood pressure reactivity withstood adjustment for depression. These findings indicate a potential mechanistic pathway that may facilitate the association between social strain and adverse physical health outcomes.

1. Introduction

Over the past several decades, research has continued to accentuate the imperative role of social relationships in influencing both physical (Holt-Lunstad, Smith, & Layton, 2010; Pinquart & Duberstein, 2010; Uchino, 2009), and psychological health (Chang, Chan, & Yip, 2017; Rueger, Malecki, Pyun, Aycock, & Coyle, 2016; Santini, Koyanagi, Tyrovolas, Mason, & Haro, 2015). A substantial body of evidence has specifically examined how social connections influence the pathogenesis and progression of cardiovascular conditions (Compare et al., 2013; Freak-Poli et al., 2021; Teshale et al., 2023). In particular, research has consistently corroborated the protective effect of supportive social relationships, with several prospective studies showing a reduced risk of adverse cardiovascular health outcomes amongst individuals reporting increased levels of social support (Barth, Schneider, & von Känel, 2010; Harding et al., 2022; Wang et al., 2024). The protective effect of social support on physical health is suggested to pertain to the stress buffering hypothesis, which propounds that greater perceptions of social support can act as a coping resource when confronted with psychological stress, thereby attenuating or eliminating the effects of stress on physical health outcomes (Cohen & Wills, 1985).

Social relationships can also serve as a source of stress and, consequently, play a role in undermining health outcomes (Rook, 1990). Birmingham and Holt-Lunstad (2018) propose a social aggravation model as a counterpart to the stress-buffering hypothesis, suggesting that strain arising from social relationships may exacerbate appraisals of existing stressors. In this model, greater social strain is thought to influence the stress appraisal process and contribute to emotionally linked

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physiological stress responses, thereby intensifying, rather than alleviating the impact of stress on health. In support of this social aggravation model, social strain arising from differential sources have been noted to influence health outcomes, such as spousal/partner strain (Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2007; Robles & Kiecolt-Glaser, 2003; Umberson, Williams, Powers, Liu, & Needham, 2006), strain from family members (Chai & Almeida, 2023), as well as wider social networks (Kershaw et al., 2014). In fact, research suggests that social strain from any type of relationship (i.e., partner, children, other family, friends, neighbors) confers a 2–3 fold increased risk for all-cause mortality over an 11-year follow-up (Lund, Christensen, Nilsson, Kriegbaum, & Rod, 2014). Importantly, social strain and social support are not considered opposing ends of the same spectrum, but are instead regarded as independent constructs, each with a distinct implication for prospective health outcomes (Finch, Okun, Barrera, Zautra, & Reich, 1989; Rook, 1990). Evidence suggests that social strain confers a larger negative effect on health in comparison to the beneficial effects of social support (Rouxel, Chandola, Kumari, Seeman, & Benzeval, 2022; Seeman, Gruenewald, Cohen, Williams, & Matthews, 2014).

Several mechanistic pathways have been noted to facilitate the association between social strain and adverse cardiovascular health outcomes (See Birmingham & Holt-Lunstad, 2018; Kiecolt-Glaser & Newton, 2001). One line of research has suggested that greater social strain/relationship conflict may engender maladaptive cardiovascular responses to acute psychological stress, indicating a psychophysiological pathway to disease outcomes (Carlisle et al., 2012; El-Sheikh & Harger, 2001; Holt-Lunstad & Clark, 2014; Smith et al., 2009). This mechanistic underpinning is premised on the cardiovascular reactivity hypothesis (Obrist, 1981), which suggests that exaggerated or prolonged cardiovascular responses to acute stress promotes the development of cardiovascular diseases (Kamarck & Lovallo, 2003; Turner, 1994). Additionally, more recent evidence has also suggested that atypically low (i.e., blunted) cardiovascular stress responses may predict a range of adverse outcomes (O'Riordan, Howard, & Gallagher, 2023; Phillips, Ginty, & Hughes, 2013; Whittaker, Ginty, Hughes, Steptoe, & Lovallo, 2021).

While social support is suggested to exert a positive effect on stress physiology through a stress-buffering mechanism, whereby the perceived availability of others during acute stress exposure can attenuate physiological responses (Cohen & Wills, 1985), the theoretical underpinnings of the association between social strain and maladaptive stress physiology appear to be more nuanced (See Birmingham & Holt-Lunstad, 2018). In particular, the Social Safety Theory suggests that humans have evolved to scan their environments for potential signs of social safety as well as potential social threats (Slavich et al., 2023). The identification or perception of social danger/threats mobilizes a range of physiological systems, including the HPA axis, inflammatory pathways, and the sympathetic nervous system, to prepare the body for physiological responses required for survival. However, consistent overactivation of these systems is proposed to result in allostatic load, leading to maladaptive functioning of stress-related physiological systems (Slavich et al., 2023).

In fact, several studies have reported associations between social strain and cardiovascular reactivity to acute psychological stress (Birmingham, Uchino, Smith, Light, & Sanbonmatsu, 2009; Bloor, Uchino, Hicks, & Smith, 2004; Carlisle et al., 2012; O'Riordan, Howard, Brown, & Gallagher, 2020; Smith et al., 2009). Importantly, these studies have varied in their operationalization of social strain, with most studies to date experimentally manipulating the experience of social strain in laboratory settings. For example prior research has used various experimental manipulations such as having partners engage in conflict (Smith et al., 2009), priming individuals with their self-reported negative social relationships prior to stress exposure (Carlisle et al., 2012), manufacturing a negative social interaction with the experimenter (Birmingham et al., 2009), and recalling adverse social relationships (Bloor et al., 2004). Interestingly, apart from the findings of Birmingham et al. (2009), who demonstrated that men experiencing negative interactions with the experimenter displayed diminished diastolic blood pressure (DBP) reactivity, the manipulation of social strain has been mostly associated with exaggerated cardiovascular stress responses (Bloor et al., 2004; Carlisle et al., 2012; Smith et al., 2009). A parallel line of research has examined strain emerging from ambivalent relationships, rather than explicitly negative interactions, and has similarly found greater cardiovascular stress responses (Holt-Lunstad & Clark, 2014; Holt-Lunstad, Uchino, Smith, & Hicks, 2007; Reblin, Uchino, & Smith, 2010). However, little research has examined how *perceived* strain from one's social network, rather than the manipulation of social strain, predicts stress-induced cardiovascular responses. Given that greater perceptions of social strain represent a chronic form of social stress, in comparison to a singular negative interaction in a laboratory environment, this may engender differential consequences for cardiovascular stress reactivity. In fact, one prior study which examined self-reported perceptions of social strain from one's social network indicated that individuals who report greater perceptions of hostility from others, such as being criticized, yelled at, ridiculed, or argued with, exhibit diminished blood pressure reactivity during a stress-inducing task (O'Riordan et al., 2020). One likely explanation for these blunted cardiovascular responses is that prolonged exposure to social strain may constitute a form of chronic stress that persistently activates physiological responses, leading to allostatic load and impaired functioning of stress response systems (McEwen, 1998).

Self-esteem has been propounded to constitute a fundamental mechanism linking social relationships to physical and mental health outcomes (Thoits, 2011), with several studies accentuating the mediating effects of self-esteem on the association between social relationships, and various measures of physical and psychological wellbeing (Begen & Turner-Cobb, 2012; Bosacki et al., 2007; Chang, Yuan, & Chen, 2018; Kleiman & Riskind, 2013; Schwager et al., 2020; Symister & Friend, 2003). It is posited that social relationships are fundamental in shaping an individual's subjective evaluation of their overall worthiness, and thus, their self-esteem (Brown, Strauman, Barrantes-Vidal, Silvia, & Kwapil, 2011; Harris & Orth, 2020; Kleiman & Riskind, 2013). This is consistent with the *sociometer theory* of self-esteem, which proffers that self-esteem reflects, and is shaped, by the extent to which an individual perceives themselves to be valued, accepted or rejected by others within their social network (Leary, 1999, 2005; Reitz, Motti-Stefanidi, & Asendorpf, 2016). Here, it is suggested that social interactions and relationships serve as indicators of an individual's desirable characteristics, influencing their self-evaluations of competence, attractiveness, and other personal qualities. These self-evaluations, in turn, are posited to contribute to the development of an individual's stable, trait-like self-esteem (Leary, 2012). Thus, the experience of chronic, ongoing forms of social strain from others, rather than a singular negative social interaction, is likely to exert significant effects on long-term levels of global self-esteem. Thus, it is not surprising that individuals who experience ongoing social strain report poorer self-esteem (Borelli & Prinstein, 2006; Juang, Syed, & Cookston, 2012; Smokowski, Bacallao, Cotter, & Evans, 2015; Yu, 2024).

Several studies have highlighted the positive stress-coping strategies employed by individuals with high trait self-esteem when confronted with psychological stress, including greater use of proactive, socio-emotional, and cognitive coping strategies (Furnham, 2024; Li et al., 2023; Lo, 2002; Martyn-Nemeth, Penckofer, Gulanick, Velsor-Friedrich, & Bryant, 2009). Consequently, trait self-esteem is propounded to influence the appraisal of threatening situations, and therefore, contributes to the subjective experience of stress (Pruessner & Baldwin, 2014). Thus, high levels of self-esteem are suggested to confer a stress-buffering effect (Martens, Greenberg, & Allen, 2008). This perspective aligns with the transactional model of stress (Lazarus & Folkman, 1984), which proposes that internal mental characteristics, such as self-esteem, can facilitate effective stress coping (Lazarus & Folkman, 1984). Pruessner and Baldwin (2014) further explain how this positive stress appraisal

among individuals high in self-esteem can, in turn, promote adaptive physiological stress responses. Indeed, prior research has linked self-esteem to cardiovascular reactivity to acute stress exposure. Hughes (2007) reported that diminished self-esteem was associated with greater SBP and DBP reactivity during performance feedback. However, others have linked diminished self-esteem to blunted cardiovascular responses. O'Donnell, Brydon, Wright, and Steptoe (2008) found that lower self-esteem was linked to reduced HRV reactivity during a mental arithmetic task. Similarly, Brown and Creaven (2017) observed that low self-esteem was associated with blunted SBP responses to repeated stress exposure under performance feedback conditions. It has been suggested that a blunted cardiovascular stress response may signal disengagement from the stressor due to a perceived inability to cope with the imposed demands (Hase, Aan Het Rot, de Miranda Azevedo, & Freeman, 2020; Phillips et al., 2013). Thus, the association between diminished self-esteem and blunted cardiovascular reactivity in prior research may reflect a perceived inability to cope and a corresponding motivational disengagement. Despite the association between self-esteem and cardiovascular reactivity, to the best of our knowledge no study to date has examined the potential mediating effects of self-esteem on the association between social strain and cardiovascular reactivity to acute stress.

Although prior research has demonstrated an association between social strain and cardiovascular reactivity (Birmingham et al., 2009; Bloor et al., 2004; Carlisle et al., 2012; Smith et al., 2009), much of the existing work has focused on manipulating social strain in controlled laboratory environments. Consequently, apart from the findings of O'Riordan et al. (2020), there has been limited investigation into perceived social strain arising naturally from one's social network. Moreover, to the best of our knowledge, no study to date has examined the varying effect of social strain on cardiovascular reactivity across different sources, such as spouse/partner, friends, and family members. Finally, although self-esteem has been proposed as a key factor underlying the influence of social relationships on stress and health outcomes (Thoits, 2011), no research has explored the potential mediating role of self-esteem in the relationship between social strain and cardiovascular reactivity.

Considering the above evidence, the primary aims of the current study are to (1) examine the association between perceptions of social strain from a spouse/partner, other family members and friends, and cardiovascular reactivity to acute psychological stress, and (2) to identify if the association between perceived social strain and cardiovascular reactivity to acute stress is mediated via self-esteem. In line with the findings of O'Riordan et al. (2020), who examined perceived social strain from ones social network, rather than the experimental manipulation of social strain, we hypothesize that greater levels of perceived social strain will be associated with increased self-reported stress and diminished cardiovascular reactivity. Furthermore, we expect that self-esteem will mediate this relationship, such that higher perceived social strain will predict lower self-esteem, which in turn will predict diminished cardiovascular responses to acute stress.

2. Methods

2.1. Participants

Data used in the current study was taken from the MIDUS 2 project (Midlife in the United States). A total of 1255 respondents completed the MIDUS 2 biomarker project (Project 4), where cardiovascular responses to acute stress were assessed. The biomarker dataset consisted of two separate cohorts including (1) the longitudinal survey sample ($n = 1054$) and (2) the Milwaukee sample ($n = 201$). Measures of social strain were assessed at the 2004–2006 MIDUS 2 survey project (project 1) and at the MIDUS 2 Milwaukee Survey project. Participants were only included in the current study if they had available data from both the self-reported survey project and biomarker project. In accordance with prior research (Keogh, Howard, & Gallagher, 2022; Keogh, Howard, & Gallagher, 2023), a subsample of participants from the twin's longitudinal study ($n = 388$) was removed due to the potential confound pertaining to the genetic determinants of reactivity (Carmelli, Chesney, Ward, & Rosenman, 1985), and due to the assumption of independence in analyses. Additionally, there were 9 participants who had a pacemaker implanted and were therefore excluded from the current study. Finally, 16 participants completed a longer protocol and were therefore excluded from the analyses. Only participants with complete data on main study variables, except social strain from a spouse or partner, were included in the primary analyses, resulting in 659 participants. This decision was made to ensure the inclusion of strain from other networks for those who did not have a partner/spouse. Given that only participants with a spouse or partner completed questionnaires on spousal/partner strain, analyses including spousal/partner strain were limited to this subsample (n = 435).

2.2. Procedure

The current sample was composed of participants from the longitudinal sample who completed the MIDUS 2 Survey project phone interview/self-administered questionnaire and the biomarker project. Eligible participants attended an overnight stay at one of three General Clinical Research Centers (GCRC) for biomarker data collection. The GCRCs were located at the University of California Los Angeles, the University of Wisconsin, and Georgetown University. Participants provided verbal consent once they agreed to take part in the study, and were then scheduled for a Visit at one of the 3 GCRCs. Before beginning the procedure, participants again provided written consent. The study was approved by the Institutional Review Board at each participating center. Psychometric questionnaires for the assessment of social strain and self-esteem were collected as part of the MIDUS 2 survey project (Project 1) in 2004–2006, and cardiovascular reactivity data was collected as part of the Biomarker project (Project 4) in 2004–2009.

2.3. Measures

2.3.1. Social strain

Social strain from family, from friends, and from a spouse/partner was assessed using three separate subscales. Participants were asked to respond on a Likert scale ranging from 1 (Often) to 4 (Never) to indicate the degree to which they agreed with each statement. Participants were asked to report how often others (i.e., family, friends, spouse/partner) make too many demands of them, criticize them, let them down when they are counting on them, and get on their nerves. For the measure of spouse/partner strain, two additional items were included: how often they argue with you and how often they make you feel tense. The three scales were constructed by calculating the mean of the items in each scale. All items were reverse-coded so that higher scores reflected greater levels of strain. These scales were based on items used in previous research (Grzywacz & Marks, 1999; Schuster, Kessler, & Aseltine, 1990; Walen & Lachman, 2000). These three scales displayed adequate internal consistency with Cronbach's α of.77,.77 and.86 of social strain from friends, family, and spouse/partner respectively. Additionally, these measures have been correlated with diminished perceptions of social support (Schuster et al., 1990) and with increased negative mood and psychopathology (Matthews, Robbins, Preisig, von Känel, & Li, 2021; Xiao & Brown, 2022), indicating both convergent and discriminant validity.

2.3.2. Self-esteem

Self-esteem was assessed using the Rosenberg self-esteem scale (Rosenberg, 1965). Participants responded to each statement using a 7-point Likert scale ranging from 1 (strongly agree) to 7 (strongly disagree), indicating the degree to which they agreed with each statement. Example items include "I take a positive attitude toward myself," "I am able to do things as well as most people," and "On the whole, I am

satisfied with myself." Higher scores are indicative of better self-esteem. This scale demonstrated adequate internal consistency, with a Cronbach's α of.77. Prior research has also reported strong test-retest reliability over a 4-week period, with a reliability estimates of.84 (Martín-Albo, Núñiez, Navarro, & Grijalvo, 2007). Moreover, Robins, Hendin, and Trzesniewski (2001) found that the Rosenberg Self-Esteem Scale demonstrated strong concurrent validity with other validated self-esteem measures (r = .75), as well as strong convergent validity with theoretically expected criteria such as academic competence, self-reported intelligence, social skill, athletic ability, and perceived physical attractiveness.

2.3.3. Self-reported stress

Participants completed measures of self-reported stress both at baseline and after completing the mental arithmetic and Stroop tasks. At each time point, participants were asked to rate their current subjective level of stress on a 10-point Likert scale ranging from 1 (not at all stressed) to 10 (extremely stressed). The self-reported stress ratings following the mental arithmetic and Stroop tasks were averaged to provide a general measure of subjective stress following stress exposure. Changes in self-reported stress from pre-task (i.e., baseline) to post-task were used in manipulation checks to examine whether the stress tasks were perceived as psychologically stressful. In the main analyses, post-task self-reported stress was used as an outcome variable. Adapted versions of this single-item measure have been extensively used in previous cardiovascular reactivity studies (Costello, Creaven, Griffin, & Howard, 2025; Howard, Gallagher, Ginty, & Whittaker, 2023; Keogh, Howard, O'Riordan, & Gallagher, 2021; O'Riordan, Costello, & Hill, 2025; Shier, Keogh, Costello, O. R., & Gallagher, 2021; Tyra, Soto, Young, & Ginty, 2020), and have been associated with state affective responses to acute psychological stress (O'Riordan, Young, Tyra, & Ginty, 2023), as well as physiological stress responses (O'Riordan, Howard, Keogh, & Gallagher, 2023).

2.3.4. Cardiovascular assessment

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were continuously, and non-invasively measured on a beat-to-beat basis using a Finometer. Here, a finger cuff is attached to the participant's finger on their non-dominant arm and an arm cuff is also attached to the participant's upper arm (i.e. at heart level). The arm cuff is used to calibrate reconstructions of the intrabrachial pressure which are estimated using the finger cuff. Additionally, a hydrostatic height correction system is used to correct for differences in height from the hand to the heart. The finometer finger cuff acquires arterial pressure based on the volume clam method (Penaz, 1973). Beat-to-beat electrocardiogram (ECG), was used to assess heart rate (HR). ECG signals were digitized at a sampling rate of 500 Hz. Proprietary event detection software was used to identify R peaks, and research staff inspected R peaks to identify any software errors. Heart rate (i.e., number of beats per minute) was computed using RR intervals. Cardiovascular data was measured in epochs of 300 s, with two epochs measured during the 11-minute resting baseline, and one epoch during each 6-minute psychological stress task. For baseline, Epoch 1 was from 0 to 300 s and Epoch 2 was from 300 to 600 s. For both the Stroop task and mental arithmetic tasks, the epochs were recorded from 0 to 300 s. Physiological calibrations using PhysioCal and self-reported stress measures were conducted during the remaining time of each period.

2.3.5. Stress task

The current study included two psychological stress tasks including a Stroop task and a mental arithmetic task. The Stroop was a modified version, whereby participants were presented with one of the following color name words at a time on a computer screen; blue, red, yellow, green. The color in which the word was typed was either congruent or incongruent with the color name itself. Participants were asked to identify the font color and select the corresponding color key on their keyboard. The speed at which the words were presented varied throughout the task and was dependent on participants' performance. More correct responses resulted in faster stimulus presentation, whereas poorer performance led to slower presentation. The mental arithmetic task was the Morgan and Turner Hewitt (MATH) task (Turner et al., 1986). During the mental arithmetic task, participants responded to a series of addition and subtraction tasks involving two numbers. The task had five levels of difficulty. At the easiest level (i.e., level 1), participants were asked to solve simple problems involving 1-digit + /− 1-digit numbers. At the most difficult level (i.e., level 5), participants were asked to solve problems involving 3-digit + /− 3-digit numbers. All participants began at level 3, and the difficulty level changed based on performance: if a participant performed well, the task increased in difficulty; if performance was poor, the difficulty decreased. The combination of the MATH task and the Stroop task has been extensively employed in prior cardiovascular reactivity studies (Keogh et al., 2022; Keogh et al. 2023; O'Riordan & Costello, 2024; Sommerfeldt, Schaefer, Brauer, Ryff, & Davidson, 2019). The MATH task, and adapted versions of this task have been previously noted to successfully perturb the cardiovascular system (Howard, Hughes, & James, 2011; Hughes, 2007; Lavoie, Miller, Conway, & Fleet, 2001) and display inter-task consistency with other acute stressors (Turner, Sherwood, & Light, 1991; Turner, Sims, Carroll, Morgan, & Hewitt, 1987). Similarly, the Stroop task has also been found to successfully perturb the cardiovascular system (Bibbey, Carroll, Roseboom, Phillips, & de Rooij, 2013), and display strong test-retest reliability over a 3-month period (Sheu, Jennings, & Gianaros, 2012), Moreover, cardiovascular responses to this task have been noted to predict adverse cardiovascular outcomes including intima-media thickness (Ginty et al., 2016), and increases in resting blood pressure (Carroll et al., 2012).

2.4. Covariates

Covariates were selected a priori for adjusted analyses based on previous cardiovascular reactivity research (Gallagher, O'Riordan, McMahon, & Creaven, 2018; Keogh et al., 2023; O'Riordan & Costello, 2025; O'Riordan, Howard, & Gallagher, 2019). Age (years), sex (male = 1, female = 2), smoking status (Smokers = Yes, Non-smokers = 2), prescription medication use (Yes =1, No = 2) resting cardiovascular function (baseline cardiovascular measures) and body mass index (BMI) were included as covariates in models examining cardiovascular reactivity.

2.5. Statistical analyses

Based on the recommendation of prior research (Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991), cardiovascular reactivity scores were computed using the delta method, whereby resting baseline scores were subtracted from stress task scores for each cardiovascular parameter. Aggregated cardiovascular reactivity scores across stress tasks has been found to demonstrate greater ecological validity in terms of predicting cardiovascular responses to real-world stressors experienced in everyday life (Kamarck et al., 2000), and has been shown to increase the reliability of cardiovascular stress reactivity (Kamarck et al., 1992; Kamarck, Jennings, Stewart, & Eddy, 1993). Thus, aggregate cardiovascular reactivity scores were computed across the Stroop and mental arithmetic task. Data was screened for outliers and cardiovascular reactivity scores deviating ±4.00 SD from the mean were removed prior to analyses, resulting in one outlier being removed for SBP reactivity, and two outliers being removed for HR reactivity. To examine if the stress task successfully perturbed the cardiovascular system, a series of paired sample *t*-tests (baseline, task) were conducted on each cardiovascular parameter. Additionally, to examine whether the stress task was perceived as psychologically stressful, a paired-sample *t*-test was used to determine if there was a significant increase in self-reported stress from pre-task to post-task. Pearsons's correlations were used to

examine the association between all continuous variables.

Hierarchical multiple linear regression models were used to examine the association between both social strain and self-esteem with measures of cardiovascular reactivity. Here, potential confounding variables were entered into the models at step 1 (i.e., age, sex, BMI, smoking status, prescription medication use, and baseline cardiovascular measures), with measures of social strain and self-esteem entered at step 2. For self-reported stress, age and sex were entered at step 1, followed by social strain and self-esteem at step 2. Given that only a subsample of participants completed the spousal/partner strain measure, resulting in a smaller sample size (n = 435) than for strain from friends or family members (n = 659), separate hierarchical linear regression models were conducted to examine the associations between social strain from friends, family, and spouse/partner, and measures of cardiovascular reactivity.

Model 4 of Hayes PROCESS macro for SPSS was used to examine the potential mediating effects of self-esteem on the association between social strain and cardiovascular reactivity. 95 % confidence levels for confidence intervals were estimated using bootstrapping samples of 5000. Measures of social strain were entered into separate models as the predictor variable, self-esteem as the mediating variable, and cardiovascular reactivity variables (i.e., SBP, DBP & HR) as the outcome variable. Again, mediation models controlled for the aforementioned confounding variables.

3. Results

3.1. Descriptive statistics manipulation check

Descriptive statistics of demographic and study variables are displayed in Table 1, and correlations between all study variables are displayed in Table S1.

3.2. Manipulation check

A series of paired samples *t*-tests revealed that the stress task successfully perturbed the cardiovascular system for SBP, $t(658) = 26.08$, $p < .001$, $d = 11.80$, DBP, $t(658) = 34.56$, $p < .001$, $d = 4.35$, and HR, $t(658) = 26.91$, $p < .001$, $d = 3.40$. As seen in Table 2, all effects were in the expected direction, with a significant increase from baseline to the stress task for each parameter. Additionally, there was a significant increase in self-reported stress from the baseline to the stressor phase, $t(655) = 35.70$, $p < .001$, $d = 1.81$, indicating that the stressor phase was perceived as psychologically stressful.

Table 1
Means, standard deviations and percentages of demographic and study variables.

Variables	Mean (SD)/N (Percent)	Range
Race *n* (%)		
Black or African American	124 (18.8 %)	-
Asian	3 (.5 %)	-
White	448 (68 %)	-
Multiracial	63 (9.6 %)	-
Native American/Alaska Native	1 (.2 %)	-
Other	18 (2.7 %)	-
Sex (% female)	370 (51.6 %)	-
Age (Years)	55.86 (10.94)	35–84
Social Strain		
Strain from friends	1.86 (.55)	1–4
Strain from family	2.12 (.65)	1–4
Strain from Spouse	2.17 (.64)	1–4
Pre-task Self-reported stress	2.09 (1.60)	1–10
Post-task Self-reported stress	4.62 (1.95)	1–10
SBP Reactivity (mmHg)	11.91 (11.62)	−21.60 – 52.65
DBP Reactivity (mmHg)	5.86 (4.35)	−10.10–21.40
HR reactivity (bpm)	3.51 (3.27)	−6.40 – 16.75

Table 2
Mean and standard deviation values for resting baseline and stress task cardiovascular scores.

	Baseline Mean (SD)	Task Mean (SD)
SBP (mmHg)	125.04 (18.76)	137.02 (22.18)**
DBP (mmHg)	61.80 (12.14)	67.66 (12.59)**
HR (bpm)	72.76 (10.67)	76.33 (11.03)**
Percieved stress	2.09 (1.56)	4.62 (1.95)**

** = Statistically significant difference from respective baseline value at $p < .001$ level.

3.3. Social strain, self-reported stress and cardiovascular reactivity

Statistics for hierarchical multiple linear regression analyses for cardiovascular reactivity and self-reported stress are displayed in Table 3. In regression models examining self-reported stress, potential confounding variables including age and sex were entered into models at step 1, followed by measures of social strain and self-esteem at step 2. While diminished self-esteem was significantly associated with higher self-reported stress, there was no significant association between measures of social strain from a spouse/partner, family or friends and self-reported stress (all p's $\geq .092$).

In analyses controlling for age, sex, BMI, smoking status, prescription medication use and baseline cardiovascular measures, greater social strain from family members was significantly associated with diminished SBP reactivity and DBP reactivity. However, social strain from family was not significantly associated with HR reactivity. Additionally, social strain from friends was significantly associated with lower SBP reactivity. No other significant associations between social strain from friends and cardiovascular reactivity were observed, (all p's $\geq .100$). Additionally, in the subsample of participants with a spouse/partner, there were no significant associations between strain from a spouse/partner and measures of cardiovascular reactivity (all p's $\geq .319$).[3]

Diminished self-esteem was associated with low SBP and DBP reactivity. However, the association between self-esteem and HR reactivity was not significant. See Table 3 for regression analyses.

3.4. Mediation analyses

3.4.1. Self-reported stress

Confounding variables, including age and sex, were entered into the models as covariates. Measures of social strain, including strain from spouse/partner, family, and friends, were entered into the models as predictor variables, with self-esteem as the mediator. Lower self-esteem significantly mediated the association between strain from a spouse/partner, B = .12, 95 % CI [.048,.222], family B = .13, 95 % CI [.063,.215], and friends, B = .09, 95 % CI [.030,.173], and self -reported stress. Greater experiences of social strain from all sources predicted diminished self-esteem, which in turn predicted greater levels of self-reported stress following acute stress exposure.

3.4.2. Cardiovascular reactivity

The aforementioned confounding variables including age, sex, BMI, current smoking status, prescription medication use and baseline cardiovascular measures were entered into models as covariates. Social strain variables were entered into models as the predictor variable, and self-esteem was entered as the mediator. In the subsample of participants who reported having a partner/spouse, there was a significant indirect effect of strain from a partner/spouse on SBP reactivity, $B = -.47$, 95 % CI [-1.030, −.052], and DBP reactivity, $B = -.22$, 95 % CI

[3] A sub-sample of participants who reported having a spouse or partner completed the spousal/partner strain measure. Accordingly, analyses examining spousal/partner strain employed a smaller sample size (n = 435) than those examining strain from friends or family members (n = 659).

Table 3
Regression analyses for the influence of social strain and self-esteem on cardiovascular reactivity and self-reported stress.

	SBP Reactivity			DBP Reactivity			HR Reactivity			Self-Reported Stress		
	β	t	*p*	*β*	t	*p*	*β*	t	*p*	*β*	t	*p*
Step 1												
Age	.23	4.66	< .001	.14	2.76	.006	−.12	−2.47	.014	.11	**2.23**	.026
Sex	−.08	−1.76	.08	.00	0.01	.993	.11	2.24	.025	.09	1.92	.056
Smoking Status	.21	4.37	< .001	.15	3.14	.002	.15	3.19	.002	-	-	-
BMI	−.01	−0.13	.895	−.07	−1.48	.14	−.03	−0.52	.601	-	-	-
Baseline	−.04	−0.92	.359	−.03	−0.69	.49	−.09	−1.86	.064	-	-	-
Prescription medication use	.02	0.35	.728	.02	0.36	.72	.10	2.11	.036			
Step 2 Models adjusted for confounding variables in step 1												
Strain from Spouse	.05	.10	.319	.01	.21	.835	−.04	−.78	.439	.08	1.69	.092
Strain from Family	−.11	−2.83	**.005**	−.12	−3.00	**.003**	−.06	−1.48	.139	.06	1.46	.144
Strain from Friends	−.09	−2.27	**.024**	−.06	−1.63	.104	−.07	−1.65	.100	.04	1.08	.282
Self-esteem	.09	2.40	**.017**	.11	2.98	**.003**	.06	1.62	.105	−.18	−4.65	**< .001**

Significance is highlighted in bold.

[-.426, −.059], via diminished self-esteem.[4] As seen in Fig. 1, greater strain from a partner/spouse was significantly associated with lower levels of self-esteem, which in turn predicted blunted blood pressure reactivity. No significant direct effect emerged in models for either SBP reactivity, $B = 1.29$, t = 1.53, $p = .126$, 95 % CI [-.363, 2.94], or DBP reactivity, $B = .28$, t = .90, $p = .371$, 95 % CI [-.400,.908], suggesting that self-esteem completely mediated the association between strain from a partner/spouse and blood pressure reactivity. There was no significant mediation effect of partner/spouse strain on HR reactivity via self-esteem, B = -.04, 95 % CI [-.171,.080]. See Figure S1 for the mediation path diagram detailing the indirect association between social strain from partner/spouse and DBP reactivity.

Greater social strain from family members was indirectly associated with lower DBP reactivity, B = -.14, 95 % CI [-.303, −.020], via self-esteem (see Fig. 2). Here, greater social strain from family members resulted in diminished self-esteem, $B = -2.67$, t = 5.91, $p < .001$, 95 % CI [-3.558, −1.783], which in turn predicted blunted DBP reactivity, $B = .05$, t = 2.37, $p = .018$, 95 % CI [.009,.099]. There was a direct association between family social strain and DBP reactivity, $B = -.65$, t = 2.40, $p = .017$, 95 % CI [-1.182, −.118], indicating partial mediation. However, there was no significant indirect effect of family strain on either SBP reactivity, B = -.29, 95 % CI [-.676,.040], or HR reactivity, B = -.06, 95 % CI [-.161,.033] via self-esteem.

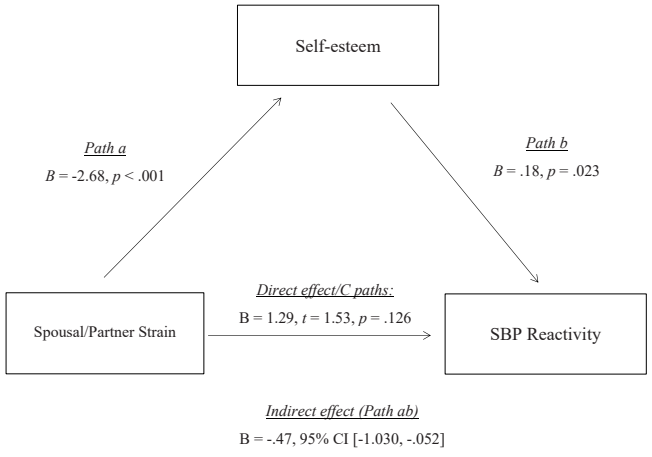

Fig. 1. Mediation path diagram: Indirect effect of spousal/partner strain on SBP reactivity via self-esteem. Analyses are adjusted for potential confounding variables including age, sex, BMI, smoking status, prescription medication use and baseline cardiovascular measures.

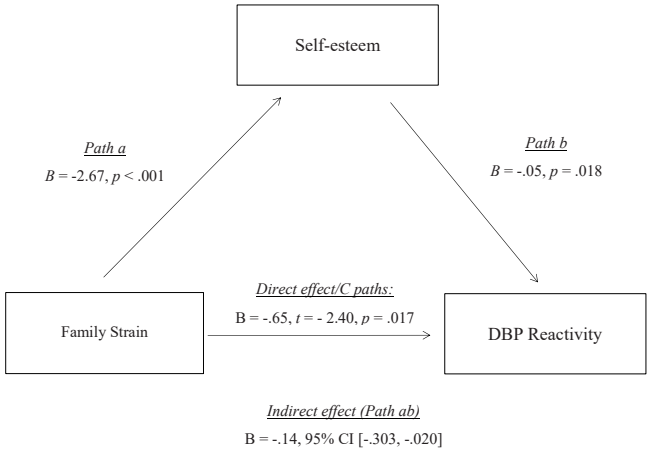

Fig. 2. Mediation path diagram: Indirect effect of Family strain on DBP reactivity via self-esteem. Analyses are adjusted for potential confounding variables including age, sex, BMI, smoking status, prescription medication use and baseline cardiovascular measures.

There was also a significant indirect mediation effect of social strain from friends on both SBP, B = -.22, 95 % CI [-.524, −.007], and DBP reactivity, B = -.11, 95 % CI [-.235, −.021]. As seen in Fig. 3, higher levels of social strain from friends predicted lower levels of self-esteem, which in turn predicted diminished blood pressure reactivity to the acute psychological stress task. There was a significant direct effect of strain from friends on SBP reactivity, B = - 1.59, t = - 1.98, $p = .048$, 95 % CI [- 3.155, −.016], but not DBP reactivity $B = -.39$, t = 1.27, $p = .205$, 95 % CI [-.996,.214], indicating partial mediation for SBP reactivity, and complete mediation for DBP reactivity. No significant mediation effect emerged for the association between social strain from friends and HR reactivity, B = -.04, 95 % CI [-.126,.016]. See Figure S2 for the mediation path diagram detailing the indirect association between social strain from friends and DBP reactivity.

3.5. Sensitivity analyses

Follow-up analyses were conducted to examine whether the significant associations between measures of social strain from a spouse/partner, friends, and family, and cardiovascular reactivity remained significant after adjusting for corresponding measures of perceived

[4] A sub-sample of participants who reported having a spouse or partner completed the spousal/partner strain measure. Accordingly, analyses examining spousal/partner strain employed a smaller sample size (n = 435) than those examining strain from friends or family members (n = 659).

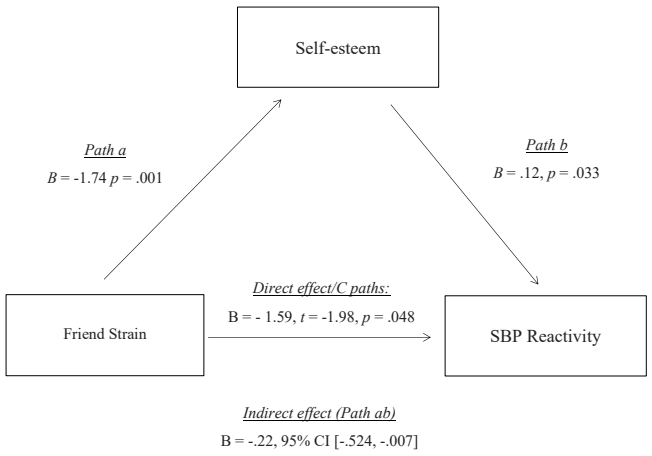

Fig. 3. Mediation path diagram: Indirect effect of strain from friends on SBP reactivity via self-esteem. Analyses are adjusted for potential confounding variables including age, sex, BMI, smoking status, prescription medication use and baseline cardiovascular measures.

social support from these social networks. In these analyses, social support was added into models as a covariate in addition to the aforementioned confounding variables. After controlling for perceived social support from family, social strain from family members remained significantly associated with lower SBP reactivity, β = -.09, t = - 2.11, p = .036, and DBP reactivity, β = -.09, t = -2.19, p = .029. Similarly, after controlling for perceived support from friends, the association between strain from friends and SBP reactivity remained significant, β = -.08, t = - 2.19, p = .029.

Apart from the indirect association between strain from family members and DBP reactivity, B = -.08, 95 % CI [-.196,.002], and social strain from friends and SBP reactivity, B = -.15, 95 % CI [-.420,.042], through lower levels of self-esteem, all other significant mediation effects remained significant in these follow-up analyses controlling for perceived social support. Additionally, the indirect effect of social strain from a spouse/partner, B = .10, 95 % CI [.026,.210], family B = .08, 95 % CI [.029,.157], and friends, B = .08, 95 % CI [.024,.148], on self -reported stress via self-esteem remained significant after adjusting for social support.

Additionally, prior research has consistently reported associations between depressive symptoms and blunted cardiovascular stress responses (Keogh et al., 2021; Liu et al., 2023; O'Riordan, Howard, Keogh, et al., 2023). Thus, additional follow-up analyses were conducted to examine if the observed significant effects withstood adjustment for depressive symptoms using the center of epidemiological studies depression scale (CESD). Here, significant analyses were replicated with depressive symptoms entered into regression models at step one along with the aforementioned confounding variables, and into mediation analyses as an additional covariate.

After adjusting for depressive symptoms, social strain from family members remained significantly associated with lower SBP reactivity, β = -.09, t = - 2.32, p = .021, and DBP reactivity, β = -.10, t = -2.37, p = .018. Interestingly, depressive symptoms were also inversely associated with SBP reactivity, β = -.11, t = - 2.71, p = .007, and DBP reactivity, β = -.14, t = -3.63, $p < .001$, in the same step of these models, indicating that strain from family and depressive symptoms were both independent predictors of blunted cardiovascular reactivity. However, the association between strain from friends and SBP reactivity became non-significant after adjusting for depressive symptoms, β = -.07, t = - 1.78, p = .076.

The association between self-esteem and lower self-reported stress withstood adjustment for depressive symptoms, β = -.13, t = - 2.99, p = .003. However, the association between self-esteem and SBP reactivity, β = .50, t = 1.22, p = .223, and DBP reactivity, β = .06, t = 1.46, p = .144, became non-significant in these adjusted models. For mediation analyses, the indirect effect of social strain from a spouse/partner, B = .10, 95 % CI [.033,.189], and from family, B = .06, 95 % CI [.014,.119], on self -reported stress via self-esteem remained significant after adjusting for depressive symptoms. However, the indirect effects of strain from friends on self-reported stress became non-significant, B = .03, 95 % CI [-.003,.085]. Finally, all mediation indirect effects of social strain on measures of cardiovascular reactivity via diminished self-esteem became non-significant after adjusting for depressive symptoms.

4. Discussion

The primary aims of the current study were to (1) examine the association between perceptions of social strain from a spouse/partner, other family members and friends, and cardiovascular reactivity to acute psychological stress, and (2) to identify if the association between perceived social strain and cardiovascular reactivity to acute stress was mediated via self-esteem. While there was no significant direct association between social strain from a spouse/partner and measures of cardiovascular reactivity, strain from family members predicted diminished SBP and DBP reactivity, while strain from friends predicted diminished SBP reactivity. Diminished self-esteem was associated with higher levels of self-reported stress following the acute stress exposure, and was associated with lower SBP and DBP reactivity. Additionally, lower levels of self-esteem significantly mediated the association between social strain from a spouse/partner, family members, and friends, and diminished cardiovascular stress responses.

The majority of studies to date examining the association between social strain and cardiovascular reactivity to acute psychological stress have experimentally manipulated the experience of strain and conflict in laboratory settings using various manipulations such as engaging partners in conflictual interactions (Smith et al., 2009), priming individuals with names of individuals with whom they have negative relationships prior to acute stress exposure (Carlisle et al., 2012), inducing strain via negative verbal interactions with the experimenter (Birmingham et al., 2009), or having individuals recall their negative social relationships (Bloor et al., 2004). Apart from the findings of Birmingham et al. (2009), who noted lower DBP reactivity amongst men who had a negative interaction with the experimenter prior to acute stress exposure, all other studies have noted that the experimental manipulation of strain and conflict exaggerates cardiovascular stress responses (Bloor et al., 2004; Carlisle et al., 2012; Smith et al., 2009). In contrast, the current findings suggest that those who reported greater perceived strain from their social networks, particularly from friends and from family, exhibit diminished blood pressure reactivity to acute stress. In accordance with our findings, others who have examined self-reported perceived strain arising from various social relationships, such as perceived hostility (i.e., being ridiculed, criticized, consistently experiencing anger, conflict and strain), have also found perceived strain arising from one's social network to predict diminished cardiovascular responses to acute stress (O'Riordan et al., 2020). It is likely that perceived social strain may represent a form of chronic, long-term stress that differs from the short-lasting experimental manipulation of strain and conflict in a laboratory setting, and thus, may engender differential consequences for cardiovascular responses to acute stress. In line with this assertion, it has been suggested that chronic exposure to, or ongoing perceptions of, stress in an individual's life can desensitize the body's stress response systems, resulting in blunted cardiovascular reactivity to acute stress (Carroll, Phillips, Ring, Der, & Hunt, 2005; Ginty & Conklin, 2011). Accordingly, prolonged involvement in aversive social relationships marked by conflict, criticism, or hostility may serve as a persistent stressor that contributes to this dysregulation.

Additionally, diminished self-esteem was also associated with blunted SBP and DBP reactivity. Prior research examining the

association between measures of trait self-esteem and cardiovascular reactivity to acute stress have reported mixed findings. Hughes (2007) reported that diminished self-esteem was associated with greater SBP and DBP reactivity during performance feedback. However, O'Donnell et al. (2008) linked lower self-esteem with reduced heart rate variability reactivity during a mental arithmetic task. Similarly, Brown and Creaven (2017) reported an association between low self-esteem and blunted SBP responses to repeated stress exposure under performance feedback conditions. Finally, it is also important to note that others have reported null-effects (Hughes, 2003). Our results suggest that lower self-esteem is associated with blunted cardiovascular reactivity, adding further support to the notion that low self-esteem may be linked to a diminished physiological stress profile.

Traditionally, diminished or "blunted" cardiovascular responses to acute stress were assumed to be benign, or in some cases protective. However, more recent evidence has continued to show that blunted cardiovascular responses to stress predict an array of negative health outcomes (Carroll, Ginty, Whittaker, Lovallo, & de Rooij, 2017; Phillips et al., 2013). Interestingly, while exaggerated or prolonged cardiovascular responses have been noted to primarily predict an increased risk of adverse cardiovascular disease outcomes (i.e., hypertension, coronary calcification, carotid atherosclerotic plaques) (Chida & Steptoe, 2010), blunted cardiovascular stress responses have been mostly noted to predict an array of non-cardiovascular related health outcomes (O'Riordan et al. 2023). Others have proposed that blunted cardiovascular stress responses may constitute a marker of motivational dysregulation, thereby predisposing individuals to a range of negative psychological outcomes and maladaptive behaviors that rely on behavioral regulation and goal-directed behavior (Carroll et al., 2017; Hase et al., 2020; Whittaker et al., 2021). Consequently, blunted cardiovascular stress responses have been noted to predict an array of cardiovascular disease risk factors (e.g., obesity, smoking status etc.) (Carroll et al., 2008; Ginty et al., 2014; Phillips, Der, Hunt, & Carroll, 2009; Phillips, Roseboom, Carroll, & de Rooij, 2012). Thus, while some research has directly linked blunted cardiovascular reactivity to poor cardiovascular health outcomes (O'Riordan et al. 2023), blunted responses are primarily suggested to constitute an indirect pathway leading to disease via psychological and behavioral mechanisms (Carroll, Lovallo, & Phillips, 2009; Phillips et al., 2013). Thus, these blunted cardiovascular stress responses may signal an indirect pathway that may facilitate the association between social strain and poor cardiovascular health observed in the literature (Chai & Almeida, 2023; Eaker et al., 2007; Kershaw et al., 2014; Lund et al., 2014).

However, it is also imperative to consider that these lower cardiovascular stress responses may not be indicative of a maladaptive stress response, but rather, an acute adaptation to long-term chronic stress exposure. This may be plausible given that no association between social strain and diminished HR reactivity were observed in the current study. A recent review of the literature has noted that blunted HR reactivity is the most robust cardiovascular reactivity predictor of adverse health outcomes (O'Riordan et al. 2023; Turner et al., 2020). In fact, these prior reviews have also revealed that in healthy samples (i.e., free from existing cardiovascular conditions), only blunted HR reactivity has been associated with prospective cardiovascular health outcomes, including greater intima-media thickness of the carotid artery/carotid atherosclerosis (Heponiemi et al., 2007), increases in resting blood pressure (Brody & Rau, 1994) and coronary artery calcification (Matthews, Zhu, Tucker, & Whooley, 2006), with no observed associations for blunted SBP or DBP reactivity. Thus, these lower responses may be indicative of an adaptation to the long-term stress associated with social strain, rather than a maladaptive response. In line with this assertion, others have suggested that frequent exposure to long-term stress may promote a gradual decline in physiological responsiveness to acute stress (Phillips, Carroll, Ring, Sweeting, & West, 2005), whereby blunted physiological reactions may represent a form of adaptation (Boyce & Chesterman, 1990). This process has been referred to as the 'inoculation effect' (Eysenck, 1983). However, given the consistent association between social strain and poorer physical health outcomes observed in the literature (Lund et al., 2014), this point should be taken tentatively.

A further point to consider in the absence of an association between social strain and HR reactivity is the hemodynamic profile that underpins the blood pressure responses exhibited in the current study. Changes in blood pressure may result from an increase in cardiac output (CO; driven by heart rate and stroke volume), known as a myocardial response, or from an increase in total peripheral resistance (TPR), referred to as a vascular response (Gregg, Matyas, & James, 2002; Gregg, Matyas, & James, 2005; James, Gregg, Matyas, Hughes, & Howard, 2012). These distinct physiological patterns are thought to contribute to poor health outcomes via different pathways (Howard, 2023). Thus, the blood pressure responses observed in the current study, particularly in the absence of a corresponding change in HR, may signal a potentially maladaptive vascular cardiovascular response profile among individuals reporting increased social strain. Future research should aim to examine changes in CO and TPR directly, in order to confirm the underlying hemodynamic profile of individuals exposed to increased social strain.

Interestingly, while our findings indicated that social strain from friends and family members was directly associated with blunted cardiovascular stress responses, no significant association between strain from a spouse/partner and cardiovascular reactivity was observed. This is surprising, given that average levels of perceived strain appeared to be higher from a spouse ($M = 2.17$, $SD = .67$), than from friends ($M = 1.86$, $SD = .55$), or family members ($M = 2.12$, $SD = .64$). These non-significant findings may be attributable to the loss of data ($n = 225$ missing values) in analyses examining social strain from a spouse/partner. Specifically, whereas analyses examining strain from family and friends included 659 participants, only a subsample of 435 participants was available for the spousal strain analyses, representing a loss of approximately 34 %. Thus, one potential explanation for the null effect of spousal strain is the reduced statistical power in these analyses relative to those examining strain from friends and family members.

Our mediation analyses indicated that diminished self-esteem acted as a significant indirect pathway in the association between social strain from friends, family and a spouse/partner and blunted blood pressure reactivity to acute psychological stress. Consistent with these findings, self-esteem has been consistently posited as a fundamental mechanism underlying the association between perceptions of social relationships and wellbeing (Thoits, 2011), with numerous studies corroborating its mediating role in the association between perceived social relationships and both physical and psychological health (Begen & Turner-Cobb, 2012; Bosacki et al., 2007; Chang et al., 2018; Kleiman & Riskind, 2013; Schwager et al., 2020; Symister & Friend, 2003). The sociometer theory of self-esteem proffers that self-esteem reflects, and is shaped, by the extent to which an individual perceives themselves to be valued, accepted or rejected by others within their social network (Leary, 1999, 2005; Reitz et al., 2016). Consequently, those who report distress, negativity and strain from their social network often report diminished levels of self-worth and poor self-esteem (Borelli & Prinstein, 2006; Juang et al., 2012; Smokowski et al., 2015; Yu, 2024). The findings of the current study add to this line of research by indicating that this diminished self-esteem plays an important role in the maladaptive stress response or "social aggravation/stress exacerbation" effects of social strain (Birmingham & Holt-Lunstad, 2018; Rook, 1990).

However, it is important to note that in follow-up analyses, the direct effects of self-esteem on stress reactivity, as well as mediation effects of social strain on stress reactivity via increased self-esteem did not withstand adjustment for depressive symptoms. A large body of research to date has consistently reported an association between depressive symptoms and blunted cardiovascular reactivity to acute psychological stress (Keogh et al., 2021; Liu et al., 2023; O'Riordan, Howard, Keogh, et al., 2023). Given that diminished self-esteem is a central component of depression (see Orth & Robins, 2013), these follow-up findings suggest that it is the overlapping variance that self-esteem shares with

depressive symptoms that may account for the diminished reactivity observed. Moreover, it is possible that increased experiences of social strain predict more generalized depressive symptoms and negative affect, rather than merely diminished self-esteem, which in turn may contribute to blunted stress responses. Interestingly, the direct associations between social strain from family and blunted SBP and DBP reactivity remained significant after adjusting for depressive symptoms, with both variables retaining significance in the models concurrently. This pattern of results suggests that although depressive symptoms may account for the direct and mediating effects of self-esteem on cardiovascular stress reactivity, on the other hand, social strain and depressive symptoms appear to independently predict cardiovascular reactivity.

It is also important to acknowledge that alternative interpersonal mechanisms not examined in the current study may account for the observed blunting effect amongst those reporting increased social strain. In particular, social strain may be understood within a broader interpersonal framework, whereby social strain signals a generalized stable relational stance or interpersonal orientation. Specifically, greater social strain may somewhat fit within the framework of attachment theory (Ainsworth, 1979; Bowlby, Ainsworth, & Bretherton, 1992). There may be overlapping variance between increased social strain and insecure attachment bonds, such as anxious attachment (i.e., a strong desire for intimacy and fear of rejection/abandonment) or avoidant attachment (i.e., discomfort with closeness/intimacy and reluctant to trust others due to fear of being hurt) (Ainsworth, Blehar, Waters, & Wall, 2015; Cassidy & Berlin, 1994; Mikulincer, 1995). Moreover, it is posited that these insecure attachment styles promote disease outcomes via dysregulating physiological stress responses systems (Pietromonaco & Beck, 2019), with prior studies linking both avoidance and anxious attachments styles to diminished cardiovascular reactivity (Kim, 2006; McMahon, Creaven, & Gallagher, 2020). Thus, future research should examine if insecure attachment mediates the association between perceptions of social strain and cardiovascular reactivity to stress.

One important strength of the current study pertains to the follow-up analyses adjusting for perceptions of social support from various social networks. This adjustment for social support is imperative for distinguishing the independent predictive utility of social strain, above and beyond the adverse effects typically observed with low social support, and is critical for isolating the specific effects of strain arising from social relationships on stress physiology. Apart from two significant indirect mediation associations for strain from family and friends on blood pressure reactivity, all other main and indirect associations remained statistically significant in analyses adjusting for social support. These findings support the assertion that social strain and social support are not opposing ends of the same spectrum, but are instead independent constructs (Finch et al., 1989; Rook, 1990), each with a distinct implication for cardiovascular stress reactivity.

One limitation of the current study pertains to the inability to definitively determine the directionality of our proposed mediation models. While our analyses and interpretation were premised on prior theory and research (Leary, 1999, 2005; Reitz et al., 2016), cardiovascular stress reactivity was assessed cross-sectionally, which limits conclusions relating to causality. A further limitation of the temporal order of our mediation models relates to the time lag between the assessment of social strain and self-esteem (i.e., project 1; 2004–2006) to the assessment of cardiovascular reactivity (Project 4; 2004–2009). The time lag introduced the possibility that unmeasured confounding factors, such as changes in physical or psychological health resulting from prolonged social strain or reduced self-esteem, may have contributed to the observed mediation effects. A further limitation of this time lag is that the self-reported measures used in the current study may not have fully captured participants' contemporaneous perceptions of their social relationships or self-esteem at the time the stress task was administered. In order to definitively determine the temporal order of the mediation models proposed in the current study, future research should (1) incorporate longitudinal designs to examine prospective changes in social strain, self-esteem, and cardiovascular reactivity over time, and (2) employ experimental designs that manipulate the experience of social stress in a laboratory setting.

Furthermore, more recently it has been suggested that examining cardiovascular responses to repeated stress exposure may be a more appropriate and ecologically valid method of assessing an individual's general stress response tendency, in comparison to examining responses to a singular tress exposure (Hughes, Howard, James, & Higgins, 2011; Hughes, Lü, & Howard, 2018). Thus, we recommend for future research to examine the potential mediating effects of self-esteem on the association between social strain and cardiovascular response adaptation to repeated stress exposure. Finally, given that the mean age of the current sample was 55.68 (SD = 10.94), and that research has consistently indicated that the structure and function of social relationships vary widely across the life span (Sander, Schupp, & Richter, 2017; Wrzus, Hänel, Wagner, & Neyer, 2013), it is possible that our findings may not be generalizable to other cohorts. Thus, we recommend that future research examine the influence of social strain on cardiovascular stress reactivity amongst differential cohorts ranging in age.

The current study findings may have practical implications and inform the development of future interventions aimed at promoting more adaptative psychophysiological responses in individuals experiencing ongoing chronic interpersonal stress. These findings may be particularly relevant for those in high-risk relationships, such as individuals experiencing domestic abuse, intimate partner violence, or family and friendship relationships characterized by ongoing conflict. Indeed, individuals exposed to such chronic interpersonal stressors have been found to be at increased risk of adverse cardiovascular outcomes (Chandan et al., 2020; Cui et al., 2024; El-Serag & Thurston, 2020; Kivimäki et al., 2003), with atypical stress responses proposed as one potential pathway (Cundiff & Smith, 2017). Given that our findings implicate diminished self-esteem in the association between social strain and dysregulated physiological stress responses, interventions aimed at enhancing self-esteem may be beneficial. In particular, cognitive behavioral therapy targeting self-schemas has been noted to be especially effective (Niveau, New, & Beaudoin, 2021), and could be implemented to improve adaptive stress coping and related physiological functioning in this vulnerable group.

In sum, the current study indicated that greater social strain from family members and from friends were significantly associated with blunted blood pressure reactivity to acute psychological stress. Moreover, diminished self-esteem was significantly associated with higher self-reported stress, and was negatively associated with blood pressure reactivity. Finally, diminished self-esteem significantly mediated the association between social strain from all sources and both cardiovascular and psychological responses to acute stress. Greater social strain predicted diminished subjective self-esteem, which in turn was significantly associated with increased levels of self-reported stress and blunted cardiovascular reactivity. However, follow-up sensitivity analyses indicated that both the direct and mediating effects of self-esteem became non-significant after adjusting for depressive symptoms, suggesting that depression may underlie the observed effects for self-esteem. These findings indicate a potential mechanistic pathway that facilitates the association between social strain and adverse physical health outcomes, providing a potential area for targeted intervention for individuals reporting increased strain from their social network.

CRediT authorship contribution statement

O'Riordan Adam: Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization. **Aisling M. Costello:** Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work the author(s) used ChatGPT in order to check grammar and spelling.

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Declaration of Competing Interest

All authors declare no conflict of interest.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.biopsycho.2025.109163.

Data availability

Data will be made available on request.

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