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Loneliness mediates the association between trait social anxiety and cardiovascular reactivity to acute psychological stress

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ABSTRACT

The primary aims of the current study are (1) to examine the association between trait social anxiety and cardiovascular reactivity to acute psychological stress, and (2) to identify if loneliness significantly mediates the association between trait social anxiety and cardiovascular reactivity. A sample of 658 participants completed a cardiovascular reactivity protocol consisting of a resting baseline and stressor phase (mental arithmetic and Stroop), with systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) monitored throughout. Participants also completed self-reported measures assessing social anxiety and loneliness. Social anxiety was associated with increased self-reported stress. However, no significant associations between social anxiety and measures of cardiovascular reactivity were observed in regression analyses. Loneliness was significantly associated with lower SBP and DBP reactivity. Additionally, loneliness significantly mediated the association between trait social anxiety and both SBP reactivity and DBP reactivity. Here, trait social anxiety predicted greater levels of loneliness, which in turn was associated with diminished cardiovascular reactivity. No significant associations emerged for HR reactivity. These blunted blood pressure responses to acute stress may indicate a potential mechanism leading to adverse prospective health outcomes.

1. Introduction

Social anxiety is characterized by consistent and intense feelings of negative social evaluation, embarrassment and social rejection (Morrison and Heimberg, 2013), and encapsulates the behavioral tendency to avoid social interactions and situations due to the perceived risk of potential scrutiny (Alden and Taylor, 2004; Heimberg et al., 2014). Social anxiety symptoms lie on a continuum of severity ranging from mild to debilitating (Davidson et al., 1994; Dell'Osso et al., 2003; Hur et al., 2020), with elevated symptoms associated with impaired quality of life (Khayyam-Nekouei et al., 2013; Wong et al., 2012), increased risk of co-morbidity (Alomari et al., 2022; Koyuncu et al., 2019), and adverse cardiometabolic health outcomes including dyslipidemia (Landén et al., 2004), increased circulating pro-inflammatory cytokines (Carlton et al., 2021), hypertension (Räikkönen et al., 2001; Stein et al., 2014), and myocardial infarction (Shen et al., 2008). Moreover, social anxiety has been found to exacerbate the effects of various other risk factors on a myriad of health outcomes, such as the influence of obesity on inflammation and insulin resistance (Jaremka and Pacanowski, 2019).

One primary focal point of research has examined the association between social anxiety and cardiovascular responses to acute psychological stress (Feldman et al., 2004; Gramer, 2006; Gramer and Sprintschnik, 2008; Ji et al., 2024; Larkin et al., 1998; Liu et al., 2023; Lü et al., 2022; Mauss et al., 2003; Yoon and Quartana, 2012). The majority of studies to date have indicated a relationship between social anxiety and diminished cardiovascular responses to stress. In response to a speech task, Gramer and Sprintschnik (2008) reported an association between social anxiety and blunted SBP, DBP and HR reactivity amongst female students. Similarly, in response to a public speaking task, Lü et al. (2022) reported that both social performance anxiety and social interaction anxiety were significantly associated with diminished SBP, DBP and HR reactivity in a mixed-sex undergraduate sample. In a sample of young junior school students, Ji et al. (2024) also reported that increased levels of social anxiety were significantly associated with lower SBP, DBP and HR reactivity. Additionally, Liu et al. (2023) observed a similar pattern during a mental arithmetic and Stroop task, finding that individuals with higher levels of trait social anxiety exhibited reduced SBP, DBP, and HR reactivity.

In contrast however, Feldman et al. (2004) found that individuals

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who report a tendency to experience anxiousness related to speaking in front of others exhibited greater SBP reactivity during both the preparation period and performance of a stress inducing speech task. Larkin et al. (1998) reported mixed effects, with increased social anxiety associated with lower DBP reactivity to a mental arithmetic task, but larger DBP and HR responses in a subgroup of males who were being observed by females while completing a mental arithmetic and anagram task. Mixed effects have also been noted by Gramer (2006), who found that while social anxiety was associated with greater HR reactivity to a role playing interpersonal task, social anxiety was inversely associated with SBP reactivity to a mental arithmetic stressor. Importantly, others have reported null effects of social anxiety on cardiovascular responses to stress (Mauss et al., 2003; Yoon and Quartana, 2012).

The original cardiovascular reactivity hypothesis posits that persistently exaggerated or prolonged cardiovascular responses to acute psychological stress promotes the development of cardiovascular diseases (Obrist, 1981). However, in line with the majority of studies reporting an association between social anxiety and diminished cardiovascular stress responses (Gramer and Sprintschnik, 2008; Ji et al., 2024; Liu et al., 2023; Lü et al., 2022), more recent evidence has indicated that atypically low or "blunted" cardiovascular reactivity is also predictive of adverse health outcomes (Carroll et al., 2017; Phillips et al., 2013; Whittaker et al., 2021). Importantly, prior evidence has suggested that blunted cardiovascular reactivity does not constitute a uniform construct, as the predictive utility of blunted cardiovascular reactivity for prospective health outcomes varies substantially across cardiovascular parameters (O'Riordan et al., 2023a; Turner et al., 2020). A recent review by O'Riordan et al. (2023a) indicated that blunted HR reactivity was the most common cardiovascular parameter associated with negative outcomes (18 studies, 78.26 %), while only 6 (26.09 %) studies reported a significant effect of blunted SBP reactivity, and 6 (26.09 %) studies reported a significant effect of blunted DBP reactivity. Moreover, it was also noted that HR reactivity was the only cardiovascular reactivity parameter that predicted poor cardiovascular health outcomes in healthy populations, such as greater intima-media thickness of the carotid artery/carotid atherosclerosis (Heponiemi et al., 2007), increased resting blood pressure (Brody and Rau, 1994), and coronary artery calcification (Matthews et al., 2006). In contrast, blunted blood pressure was found to predict adverse cardiovascular outcomes only in those with an existing cardiovascular condition (Herd et al., 2003; Kupper et al., 2015; Sherwood et al., 2017), as well as non-cardiac health outcomes in the general population such as cognitive decline (Yano et al., 2016), self-reported illness frequency/subjective health (Lawler and Schmied, 1992; Phillips et al., 2009a) and addiction relapse (al'Absi et al., 2005). Interestingly, studies reporting an association between social anxiety and blunted reactivity have found effects for SBP, DBP and HR reactivity (Gramer and Sprintschnik, 2008; Ji et al., 2024; Liu et al., 2023; Lü et al., 2022).

It has been suggested that blunted cardiovascular reactivity may constitute a general marker of motivational and behavioral dysregulation, resulting in an increased vulnerability to an array of diverse outcomes (Carroll et al., 2017; Carroll et al., 2009; Whittaker et al., 2021). In line with this assertion, it has been suggested that blunted cardiovascular responses during exposure to psychological stress may be indicative of a motivational disengagement with the psychological stressor (Hase et al., 2020). In fact, the motivational intensity theory suggests that disengagement is predicted when a task appears to be too difficult and/or the necessary effort to succeed is not justified (Brehm and Self, 1989; Silvestrini, 2017; Wright, 1996). Importantly, others have shown affective influences on this type of disengagement (i.e., perception of difficulty and ability to succeed) (Brinkmann and Gendolla, 2008; Gendolla, 2012; Gendolla, 2025; Gendolla and Brinkmann, 2005), which may explain why individuals with social anxiety exhibit blunted reactions. In fact, studies that report blunted cardiovascular responses amongst socially anxious individuals also show increased levels of self-reported stress and negative emotional experiences (Gramer and Sprintschnik, 2008; Lü et al., 2022), suggesting that these blunted responses may be indicative of an increased perception of task difficulty and subsequent task disengagement.

A further theoretical underpinning that may explain the association between social anxiety and atypical cardiovascular stress responses may pertain to the Generalized Unsafety Theory of Stress (See Brosschot et al., 2016). This theoretical perspective suggests that physiological reactions are due to an inability/uncertainty to perceive safety signals from one's environment, rather than generated due to fear and threat (Brosschot et al., 2018). Interestingly, Brosschot et al. (2016) suggests that individuals with anxiety have not learned to identify safety signals across differential contexts, resulting in the perception of generalized unsafety. A further postulate of this theoretical framework that may also pertain to increased levels of social anxiety asserts that individuals with compromised social networks, such as those who report increased loneliness/social isolation, are deprived of their primary source of safety, resulting in feelings of uncertainty and unsafety. This inability to perceive safety can maintain individuals in the "default stress response" (Brosschot et al., 2016, 2018), which may result in allostatic overload and blunted physiological reactions to acute stress (O'Connor et al., 2021).

Indeed, several studies have reported loneliness as a key factor underlying the connection between social anxiety and well-being (Dong et al., 2024; Gallagher et al., 2014; Kealy et al., 2023; Wang and Yao, 2020). It is postulated that socially anxious individuals limit their opportunities to make meaningful social connections, due to persistent negative beliefs about themselves, their negative interpretations of social events and their avoidance of social situations (Stopa and Clark, 2000; Teo et al., 2013). In fact, it is suggested that this type of implicit hypervigilance to social threats, as well as cognitive biases pertaining to perceptions of social dangers during interpersonal interactions increase the likelihood that an individual will behave in ways that push away the very people who may fulfil their social needs (Cacioppo et al., 2014; Cacioppo and Hawkley, 2009; Chen et al., 2020). Thus, it is not surprising that highly socially anxious individuals often report suffering from significant functional impairments pertaining to their social life, friendships and romantic/personal relationships (Aderka et al., 2012; Davidson et al., 1994; Davila and Beck, 2002; Sparrevohn and Rapee, 2009). In fact, others have accentuated the role of social anxiety in the maintenance of subjective loneliness over time, and have suggested that social anxiety may constitute an antecedent to emerging feelings of loneliness (Danneel et al., 2019; Lim et al., 2016), with further evidence indicating that treatment induced reductions in social anxiety predict lower follow-up self-reported loneliness (O'Day et al., 2021). Moreover, loneliness has been consistently found to alter physiological responses to acute psychological stress (See Brown et al., 2018), which has been posited as a potential mechanism linking loneliness to adverse cardiometabolic health outcomes (Alden and Taylor, 2004; Hawkley and Cacioppo, 2010). However, despite the importance of loneliness in understanding the associations between social anxiety and well-being, as well as the consistent association between loneliness and aberrant cardiovascular responses to acute stress, no study to date has examined the mediating effects of loneliness on the association between social anxiety and cardiovascular reactivity to acute psychological stress.

Thus, considering the above evidence, the primary aims of the current study are (1) to examine the association between trait social anxiety and cardiovascular reactivity to acute psychological stress, and (2) to identify if loneliness is a significant mediator of the association between social anxiety and cardiovascular reactivity. In line with the majority of prior studies and evidence to date, we hypothesize that social anxiety will be associated with diminished cardiovascular reactivity to acute psychological stress. Furthermore, we expect that this association will be mediated via self-reported loneliness, whereby high trait social anxiety will predict greater levels of subjective loneliness, which in turn will predict diminished cardiovascular responses.

2. Material and methods

2.1. Participants

Data from the current study was taken from the MIDUS Refresher 1 biomarker project, which was collected between 2012 and 2016, as part of the wider MIDUS Refresher study Survey ($N = 4085$). See Supplementary file 1 for details of the methods for the broader study and protocol. The overall MIDUS Refresher study survey was designed to replenish the original MIDUS 1 baseline cohort. A total of 863 respondents were included in the biomarker project, including 746 individuals from the main sample, and an additional 117 individuals from the Milwaukee sample. A total of 23 participants were excluded from the current study due to having a pacemaker implanted. Moreover, only participants who had complete data for variables used in primary analyses were included, resulting in 658 participants.

2.2. Procedure

Participants were eligible to complete the biomarker project if they (1) completed the MIDUS Refresher Survey Project phone interview and self-administered questionnaire or (2) completed the Milwaukee Refresher Survey Project. The data was collected over a 2 day stay at one of three clinical research units (CRU) including the University of California Los Angeles (UCLA), the University of Wisconsin (UW) or Georgetown University (GU). Verbal consent was obtained prior to attending the CRU when participants were scheduling their visit. Moreover, written informed consent was obtained once participants arrived at the clinic before any of the experimental procedure began. Self-administered questionnaires were completed on day one, and the psychophysiology experimental protocol was completed on day two.

2.3. Measures

2.3.1. Loneliness

Loneliness was assessed using the 7-item UCLA Loneliness Scale (Russell, 1996). Participants were required to answer on a 4-point Likert scale ranging from 1 (never) to 4 (often) the degree to which they agreed with each statement. Example items including "No one really knows me well", "There is no one I can turn to" and "I feel isolated from others." The scale displayed strong internal consistency, with a Cronbach's α of 0.88.

2.3.2. Social anxiety

An adapted version of the Liebowitz Social Anxiety scale was used to assess the severity of social anxiety (Liebowitz, 1987). The original self-reported version of the scale is composed of 24 items that are used to assess both avoidance and feelings of fear in response to various social situations (Fresco et al., 2001). The MIDUS adapted version of this scale included a total of 9 items, whereby participants were required to indicate on a 4-point Likert scale how much fear or anxiety they generally feel during each situation ranging from 1 (none) to 4 (severe). Example items include "Talking to people in authority", "Going to a party" and "Being the center of attention". This 9-item scale was found to have strong internal consistency, with a Cronbach's α of 0.85.

2.3.3. Self-reported stress

At baseline, and following both the mental arithmetic and Stroop task, participants were asked to report their current levels of subjective stress on a 10-point Likert scale ranging from 1 (not at all stressed) to 10 (extremely stressed). Post-task measures of self-reported stress were calculated as the average stress levels across both stress exposures. Changes in self-reported stress (from pre to post task), were used as a manipulation check to examine if the stress task was perceived as psychologically stressful. Post-task measures of self-reported stress were used in main analyses as an outcome variable.

2.3.4. Cardiovascular assessment

Beat-to-beat noninvasive measurement of SBP and DBP were assessed using a Finometer cardiovascular monitor. A finger cuff was placed on participants' middle finger of their non-dominant hand, and an arm cuff was placed on participants' upper arm. Blood pressures was measured at the finger and corrected to the brachial artery standards. Heart rate (HR) was measured using a beat-to-beat electrocardiogram (ECG) and digitized at a sampling rate of 500 Hz. ECG waveforms were submitted to proprietary event detection software to identify R waves, and the RR intervals were used to compute heart rate. Cardiovascular data were analyzed with a specified 300 epoch duration. Two epochs were computed during an 11-min baseline, and one epoch was assessed during both 6-min psychological stress tasks. Baseline was computed as the mean of the two resting 300 epochs, and stress task scores as the mean for the epochs during both tasks.

2.3.5. Stress task

Two stress tasks were used in the current study including The Morgan and Turner Hewitt (MATH) task (Turner et al., 1986), and a Stroop color-word task. The math task was a 6-min computerized mental arithmetic task. Participants were required to respond to addition and subtraction problems of two numbers. The math problems varied across 5 levels of difficulty. At level 1 (i.e., the easiest level), participants responded to problems of 1-digit +/− 1-digit numbers. At level 5 (i.e., most difficult level) participants responded to 3-digit +/− 3-digit numbers. For all participants, the task began at level 3, and the difficulty of the subsequent trial was determined by performance based on the current trial. If participants' responses were correct, the difficulty level on the next trial was increased by one level. However, if the participant was already on level 5, they would remain on this level until they provided an incorrect response. Incorrect responses resulted in a decrease in one level on the next trial. Similarly, if the participant was already on level 1, they remained on this level if they continued to give incorrect responses, and would increase to level 2 once they provided correct responses. During each trial, three elements were presented on the screen in quick succession. Firstly, a math problem was presented on screen for 2 s, followed by the equals sign for 1.5 s and then a solution to the math problem for 1 s. During this one second period when participants were presented with the solution, they were required to press one of two keys on the keypad to indicate if the answer was correct or incorrect. If participants, did not respond within the 1 s period, the response was recorded as incorrect, and the next trial was presented. The ratio of addition to subtraction tasks was 3:7 and the ratio of correct to incorrect answers presented to participants was 1:1. Participants continued to complete trials throughout the 6-min task. Thus, there were no specific number of trials through the task, as the number of trials that a participant completed varied based on their response time.

During a 6-min Stroop color-word task, participants were presented with one of four color words including green, blue, red or yellow on a computer screen. The words were presented in a color font that was either incongruent or congruent with the color name of the word. One of the four color words appeared on screen at a time, and participants had to press one of four keys on a keypad that corresponded with the color of the word (i.e., the color ink of the letters in the word), not the spelling of the word. The rate at which color words were presented to participants varied as a function of performance (i.e., based on correct and incorrect responses) in order to standardize the stressfulness of the task. While greater accuracy resulted in a faster rate of presentation, poor performance resulted in a slower rate of presentation. Thus, the number of trials a participant completed throughout the 6-min task varied depending on accuracy and response rates. Across all participants, the accuracy (i.e., correct responses) was 67 %.

2.4. Open science and transparency

The current study was not pre-registered. However, the data that

support the findings of this study are openly available and can be accessed via the MIDUS Portal (https://midus.colectica.org/). All methodological decisions were made a priori and were based on prior cardiovascular reactivity research, as well as previous studies utilizing the MIDUS dataset. The decision to remove those with an implanted pacemaker was based on studies examining measures of stress psychophysiology from the MIDUS dataset (Keogh et al., 2022, 2023). Additionally, decisions pertaining to the selection of covariates (Creaven et al., 2020; Gallagher et al., 2021; Gallagher et al., 2018; Keogh et al., 2023) as well as the removal of outliers deviating ±4.00 SD from the mean (O'Riordan et al., 2023b; Tyra et al., 2020), were based on similar cardiovascular reactivity research. For the calculation of cardiovascular reactivity, the delta method was employed (stress task – baseline) as this method has been shown to have strong generalizability across stressors (Llabre et al., 1991). Finally, the decision to aggregate cardiovascular reactivity scores was based on prior evidence indicating that aggregated scores across tasks enhances the reliability of stress reactivity (Kamarck et al., 1992; Kamarck et al., 1993) and demonstrates greater ecological validity in terms of predicting cardiovascular responses to real-world stressors (Kamarck et al., 2000).

2.5. Statistical analyses

Cardiovascular reactivity scores were calculated as the arithmetic difference between baseline and task scores for each cardiovascular parameter. Cardiovascular reactivity outliers deviating ±4.00 SD from the mean were removed prior to analyses, resulting in one outlier being removed for SBP reactivity, and one outlier being removed for DBP reactivity. A series of paired sample *t*-tests (baseline, task) were conducted on each cardiovascular parameter in order to examine if the stressor successfully perturbed the cardiovascular system. Additionally, in order to determine if the stress task was perceived as psychologically stressful, a paired sample *t*-test was used to examine if levels of self-reported stress increased from the resting baseline to the stress task period.

Regression analyses were used to examine the association between both social anxiety and loneliness, and measures of cardiovascular reactivity. Potential confounding variables including age, sex, BMI, current smoking status, prescription medication use, and baseline cardiovascular measures were entered into models at step 1, followed by social anxiety and loneliness at step 2. For self-reported stress, age and sex were entered at step 1 followed by social anxiety and loneliness at step 2. Mediation analyses were conducted using model 4 of Hayes PROCESS macro for SPSS, with social anxiety entered into models as the predictor variable, loneliness as the mediator, and measures of cardiovascular reactivity as the outcome variable. 95 % confidence levels for confidence intervals were estimated using bootstrapping samples of 5000.

3. Results

3.1. Descriptive statistics and manipulation check

Participants ranged in age from 26 to 78 (M = 50.74, SD = 12.77 years). A total of 361 (54.9 %) participants were female. Furthermore, 441 (67.2 %) were white, 101 (15.4 %) were African American, 6 (0.9 %) were Native American or Alaska native Aleutian islander/eskimo, 2 (0.3 %) were Native Hawaiian or Pacific Islander, 10 (1.5 %) were Asian, 50 (7.6 %) were multiracial (reported more than one race), and 41 (6.3 %) reported other. Descriptive statistics of demographic and study variables are displayed in Table 1, and correlations between continuous study variables are displayed in Table 2.

A series of paired samples *t*-tests revealed that the stress task successfully perturbed the cardiovascular system for SBP, $t(657) = 22.61$, $p < .001$, $d = 9.72$, DBP, $t(657) = 27.95$, $p < .001$, $d = 4.12$, and HR, $t(657) = 28.42$, $p < .001$, $d = 3.56$. As seen in Table 3, 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 $t(654) = 36.14$, $p < .001$, $d = 1.65$, 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	101 (15.4 %)	–
Asian	10 (1.5 %)	–
White	441 (67.2 %)	–
Multiracial	50 (7.6 %)	–
Native American/Alaska native	6 (0.9 %)	–
Native Hawaiian/Pacific Islander	2 (0.3 %)	
Other	41 (6.3 %)	–
Sex (% female)	361 (54.9 %)	–
Age (Years)	50.74 (12.77)	26–78
Social Anxiety	1.86 (0.55)	1–4
Loneliness	12.71 (4.50)	7–28

3.2. Regression Analyses

In hierarchical multiple linear regression models, age and sex were entered into models at step 1 followed by social anxiety and loneliness at step 2. While there was no significant association between loneliness and self-reported stress, $\beta = 0.002$, $t = 0.04$, $p = .976$, social anxiety was significantly associated with greater levels of self-reported stress, $\beta = -0.20$, $t = 4.74$, $p < .001$.

For regression analyses examining cardiovascular reactivity variables, age, sex, BMI, current smoking status, prescription medication use and baseline cardiovascular measures were entered into models at step 1, followed by loneliness and social anxiety at step 2. Loneliness was significantly associated with lower SBP, $\beta = -0.09$, $t = 2.13$, $p = .033$ and DBP reactivity, $\beta = -0.12$, $t = -3.01$, $p = .003$. However, there was no significant association between loneliness and HR reactivity, $\beta = -0.05$, $t = -1.27$, $p = .205$. Additionally, social anxiety was not significantly associated with SBP, $\beta = 0.04$, $t = 0.99$, $p = .323$, DBP, $\beta = 0.07$ $t = 1.79$, $p = .074$, or HR reactivity, $\beta = 0.03$, $t = 0.77$, $p = .441$ (See Table 4).

3.3. Mediation analyses

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 anxiety was entered into models as the predictor variable, and loneliness was entered as the mediator. As seen in Fig. 1, there was a significant indirect effect of social anxiety on SBP reactivity through loneliness (path ab), $B = -0.57$, 95 % CI [−1.08, −0.11]. Here, social anxiety was significantly associated with increased levels of loneliness (path a), $B = 3.12$, $t = 10.41$, $p < .001$, 95 % CI [2.53, 3.71], with increased levels of loneliness, in turn, associated with lower blood pressure reactivity (path b), $B = -0.18$, t = 2.13, $p = .033$, 95 % CI [−0.35, −0.01].

Similarly, the mediating effect of social anxiety on DBP reactivity via loneliness was also significant (pathway ab), B = − 0.35, 95 % CI [−0.60, −0.13]. Again, increased levels of social anxiety predicted greater levels of loneliness (pathway a), B = 3.18, $t = 10.74$, $p < .001$, 95 % CI [2.60, 3.76], with greater levels of loneliness predicting lower DBP reactivity (pathway b), B = −0.11, t = −3.01, $p = .003$, 95 % CI [−0.18, −0.04] (see Fig. 2).

There was no significant total effect (path c) or direct effect (path c') of social anxiety on either SBP or DBP reactivity, indicating complete mediation. The standardized indirect effects for SBP and DBP reactivity were 0.03, and 0.05 respectively, indicating small effect sizes for our

Table 2
Correlational analyses between social anxiety, loneliness, cardiovascular measures and continuous covariates.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Social Anxiety	–	0.37**	−0.16**	−0.03	−0.03	0.05	0.10**	0.18**	−0.03	0.04	0.10*	0.20**	−0.04	−0.02	0.03
2. Loneliness	–	–	−0.07	0.14**	0.001	0.02	0.03	0.07	−0.05	−0.03	0.01	0.07	−0.12**	−0.14**	−0.06
3. Age	–	–	–	−0.06	0.01	−0.25**	−0.26**	−0.05	0.16**	−0.16**	−0.28**	0.02	0.30**	0.27**	−0.08**
4. BMI	–	–	–	–	0.12**	0.04	0.09*	0.01	0.05	0.00	0.06	−0.01	−0.11**	−0.13**	−0.06
5. Baseline SBP	–	–	–	–	–	0.68**	0.08**	−0.02	0.87**	0.66**	0.10**	−0.03	0.000	0.002	0.08**
6. Baseline DBP	–	–	–	–	–	–	0.23**	−0.02	0.57**	0.94**	0.24**	−0.04	−0.05	−0.08	0.05
7. Baseline HR	–	–	–	–	–	–	–	0.11**	0.05	0.21**	0.94**	0.05	−0.06	−0.05	−0.06
8. Baseline Percieved stress	–	–	–	–	–	–	–	–	0.01	0.00	0.07	0.46**	0.06	0.04	−0.12**
9. Task SBP	–	–	–	–	–	–	–	–	–	0.70**	0.09*	−0.01	0.48*	0.39**	0.14**
10. Task DBP	–	–	–	–	–	–	–	–	–	–	0.25**	−0.03	0.24**	0.27**	0.13**
11. Task HR	–	–	–	–	–	–	–	–	–	–	–	0.06	−0.01	0.03	0.28**
12. Task Perceived stress	–	–	–	–	–	–	–	–	–	–	–	–	0.03	0.03	0.04
13. Reactivity SBP	–	–	–	–	–	–	–	–	–	–	–	–	–	0.81**	0.12**
14. Reactivity DBP	–	–	–	–	–	–	–	–	–	–	–	–	–	–	0.23**
15. Reactivity HR	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

** $p < .01$; * $p < .05$

Table 3
Mean and standard deviation values for resting baseline and stress task cardiovascular scores.

	Baseline Mean (SD)	Task Mean (SD)
SBP (mmHg)	123.10 (17.48)	131.67 (20.00)**
DBP (mmHg)	63.23 (11.40)	67.72 (11.74)**
HR (bpm)	70.55 (10.28)	74.50 (10.70)**
Perceived stress	1.72 (1.19)	4.04 (1.08)**

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

significant mediation effects. Finally, analysis for HR reactivity revealed no significant mediation effect, $B = -0.14$, 95 % CI [−0.37, −0.08].

3.4. Sensitivity and follow-up analyses

Although all models examining cardiovascular reactivity were adjusted for resting cardiovascular function, follow-up replication analyses were conducted on baseline measures. In hierarchical multiple linear regression analyses, age, sex, BMI, current smoking status, and prescription medication use was entered into models at step 1, followed by social anxiety and loneliness at step 2. Neither social anxiety or loneliness were associated with resting SBP, DBP or HR (all p's ≥ 0.282). Additionally, follow-up replication analyses were conducted with outliers included in analyses. These outliers included one value on SBP reactivity deviating 4.13 SD from the mean, and one value on DBP reactivity deviating 5.25 SD from the mean. While the association between loneliness and SBP reactivity became non-significant $\beta = 0.08$, $t = -1.94$, $p = .053$, the association between loneliness and DBP reactivity remained statistically significant $\beta = 0.18$, $t = -2.85$, $p = .005$. Additionally, the indirect mediation effects of social anxiety on SBP reactivity, B = − 0.54, 95 % CI [−0.1.04, −0.06], and DBP reactivity. B = − 0.34, 95 % CI [−0.59, −0.12], remained significant. Finally, supplementary analyses for each stressor individually are presented in Supplementary File 2.

4. Discussion

The primary aims of the current study were (1) to examine the association between social anxiety and cardiovascular reactivity to acute psychological stress and (2) to identify if loneliness significantly mediated the association between social anxiety and cardiovascular reactivity to acute stress. While social anxiety was significantly associated with greater levels of self-reported stress following the stress phase in regression analyses, no significant direct associations emerged for measures of cardiovascular reactivity. In contrast, loneliness was significantly associated with diminished SBP, and DBP reactivity. Moreover, loneliness significantly mediated the association between social anxiety and both SBP and DBP reactivity, indicating an indirect association between trait social anxiety and cardiovascular reactivity.

To date, studies examining the association between social anxiety and cardiovascular reactivity have continued to yield inconsistent findings. While the majority of studies have linked social anxiety to diminished cardiovascular responses to acute stress (Gramer and Sprintschnik, 2008; Ji et al., 2024; Liu et al., 2023; Lü et al., 2022), others have reported antithetical effects, with social anxiety predicting greater cardiovascular stress responses (Feldman et al., 2004). Additionally, some studies have reported mixed (Gramer, 2006; Larkin et al., 1998), as well as null associations (Mauss et al., 2003; Yoon and Quartana, 2012). While our supplementary analyses did show an association between social anxiety and greater SBP reactivity to the mental arithmetic task when examined in isolation, our main analyses employing aggregated cardiovascular response scores across tasks yielded no significant associations between social anxiety and cardiovascular reactivity. One potential explanation for the non-significant direct effect of

Table 4
Regression analyses for the influence of loneliness and social anxiety on measures of cardiovascular reactivity and self-reported stress.

	SBP Reactivity					DBP Reactivity					HR Reactivity					Self-Reported Stress				
	β	t	*p*	Partial	Part	*β*	t	*p*	Partial	Part	*β*	t	*p*	Partial	Part	*β*	t	*p*	Partial	Part
Step 1																				
Age	0.28	7.14	**<0.001**	0.27	0.26	0.26	6.27	**<0.001**	0.24	0.23	−0.12	−2.78	**0.006**	−0.11	−0.11	0.03	0.69	0.489	0.03	0.03
Sex	−0.08	−1.99	**0.047**	−0.08	−0.07	−0.05	−1.23	0.219	−0.05	−0.05	0.02	0.45	0.656	0.02	0.02	0.12	3.01	**0.003**	0.12	0.12
Smoking Status	0.19	5.16	**<0.001**	0.20	0.19	0.12	3.13	**0.002**	0.12	0.12	0.14	3.73	**<0.001**	0.14	0.14	–	–	–	–	–
BMI	−0.08	−2.16	**0.031**	−0.08	−0.08	−0.11	−2.89	**0.004**	−0.11	−0.11	−0.06	−1.57	0.116	−0.06	−0.06	–	–	–	–	–
Medication	−0.01	−0.34	0.737	−0.01	−0.01	0.01	0.17	0.868	0.01	0.01	−0.04	−0.92	0.36	−0.04	−0.04	–	–	–	–	–
Baseline	−0.02	−0.60	0.549	−0.02	−0.02	−0.02	−0.44	0.659	−0.02	−0.02	−0.08	−2.10	**0.036**	−0.08	−0.08	–	–	–	–	–
Step 2																				
Age	0.28	7.08	**<0.001**	0.27	0.26	0.26	6.35	**<0.001**	0.24	0.23	−0.11	−2.68	**0.008**	−0.10	−0.10	0.06	1.50	0.135	0.06	0.06
Sex	−0.08	−2.19	**0.029**	−0.09	−0.08	−0.06	−1.55	0.122	−0.06	−0.06	0.01	0.31	0.757	0.01	0.01	0.11	2.75	**0.006**	0.12	0.12
Smoking Status	0.18	4.77	**<0.001**	0.18	0.17	0.10	2.63	**0.009**	0.10	0.10	0.14	3.48	**<0.001**	0.14	0.13	–	–	–	–	–
BMI	−0.07	−1.75	0.08	−0.07	−0.06	−0.09	−2.30	**0.022**	−0.09	−0.09	−0.05	−1.31	0.19	−0.05	−0.05	–	–	–	–	–
Medication	−0.01	−0.20	0.839	−0.01	−0.01	0.02	0.39	0.694	0.02	0.02	−0.03	−0.81	0.417	−0.03	−0.03	–	–	–	–	–
Baseline	−0.03	−0.65	0.518	−0.03	−0.02	−0.02	−0.50	0.616	−0.02	−0.02	−0.09	−2.11	**0.035**	−0.08	−0.08	–	–	–	–	–
Social Anxiety	0.04	0.99	0.323	0.04	0.04	0.07	1.79	0.074	0.07	0.07	0.03	0.77	0.441	0.03	0.03	0.20	4.74	**<0.001**	0.18	0.18
Loneliness	−0.09	−2.13	**0.033**	−0.08	−0.08	−0.12	−3.01	**0.003**	−0.12	−0.11	−0.05	−1.27	0.205	−0.05	−0.05	0.002	0.04	0.967	0.002	0.002
Overall Model change																				
	ΔR^2	ΔF	*p*	–	–	ΔR^2	ΔF	*p*	–	–	ΔR^2	ΔF	*p*	–	–	ΔR^2	ΔF	*p*	-	-
	0.01	2.29	0.102	–	–	0.01	0.4.75	**0.009**	–	–	0.003	0.85	0.427	–	–	0.04	13.07	**<0.001**	-	-

Significance is highlighted in bold.

social anxiety on measures of cardiovascular reactivity across stressors in the current study may pertain to the social salience of the experimental stressor. The current study employed stressors of relatively low social salience, without any verbal feedback. However, the majority of studies reporting an association between social anxiety and cardiovascular reactivity to date have primarily employed stressors of increased social salience such as various public speaking tasks, or stressors designed to induce feelings of social evaluation such as confederate observations and video recordings (Feldman et al., 2004; Gramer, 2006; Gramer and Sprintschnik, 2008; Ji et al., 2024). In fact, studies examining the influence of social context on the association between trait social anxiety and cardiovascular reactivity have found social anxiety to predict diminished cardiovascular responses to stressors with increased social challenge, but no associations in response to stressors with no social challenges (Lü et al., 2022). Social anxiety is propounded to encapsulate a cognitive bias of interpersonal interpretation of social interactions and situations, resulting in increased negative perceptions in response to social contexts (Chen et al., 2020), accompanied by feelings of negative social evaluation, embarrassment and social rejection (Alden and Taylor, 2004; Heimberg et al., 2014; Morrison and Heimberg, 2013). Therefore, it is likely that stressors of increased social salience may be particularly threatening, and thus, more effective at eliciting physiological responses for highly socially anxious individuals.

Importantly, however, social anxiety was indirectly associated with diminished SBP and DBP reactivity via increased levels of loneliness. It is postulated that socially anxious individuals limit their opportunities to make meaningful social connections, due to negative interpretations of social events and their avoidance of social situations (Stopa and Clark, 2000; Teo et al., 2013). In fact, it is suggested that hypervigilance of social threats and biased perceptions of interpersonal interactions play a fundamental role in pushing away the very people who may fulfil imperative social needs, resulting in increased perceptions of loneliness (Cacioppo et al., 2014; Cacioppo and Hawkley, 2009). Thus, it is not surprising that social anxiety has been noted as an imperative factor in both the development and maintenance of loneliness over time (Danneel et al., 2019; Lim et al., 2016). Moreover, given the influence of loneliness on aberrant physiological responses to acute stress (Brown et al., 2018), adverse prospective health outcomes (O'Súilleabháin et al., 2019; Valtorta et al., 2016), and mortality (Holt-Lunstad et al., 2015), loneliness may play a fundamental role in the association between social anxiety and an array of health outcomes.

One important point of consideration pertains to the direction of our mediation results, with social anxiety indirectly associated with lower cardiovascular responses to the stress via subjective loneliness. While exaggerated cardiovascular reactivity has been predominately associated with prospective cardiovascular health outcomes (Carroll et al., 2012a, 2012b; Chida and Steptoe, 2010), more recent evidence has linked diminished or blunted cardiovascular responses to an array of non-cardiac health related outcomes (Carroll et al., 2017; Phillips et al., 2013; Whittaker et al., 2021). Thus, it is now propounded that bidirectional deviation from normative physiological stress responses may be indicative of a homeostatic dysfunction, and therefore, psychosomatic disease vulnerability (Lovallo, 2011). However, while social anxiety was indirectly associated with blunted SBP and DBP reactivity, no significant indirect associations were observed for HR reactivity. This is an imperative point of consideration given that blunted HR reactivity has been shown to be the most robust cardiovascular reactivity predictor of prospective health outcomes (O'Riordan et al., 2023a; Turner et al., 2020). Moreover, these prior reviews have also revealed that in healthy samples, 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), increased resting blood pressure (Brody and Rau, 1994), and coronary artery calcification (Matthews et al., 2006), with no observed associations for blunted SBP or DBP reactivity. Thus, with the absence of an association between social anxiety and HR reactivity in the

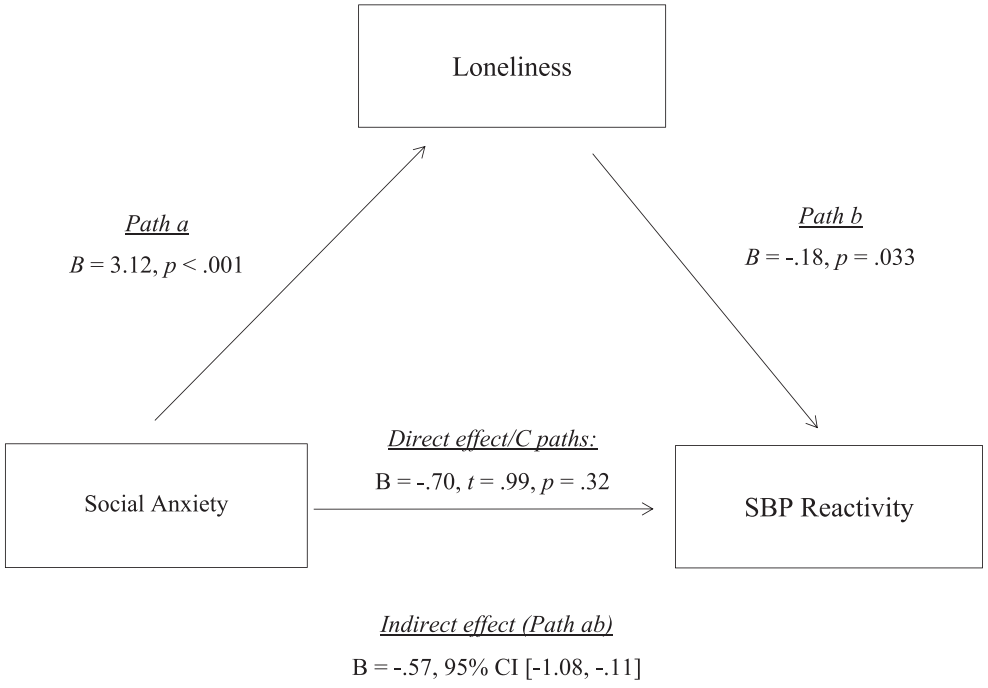

Fig. 1. Mediation path diagram: Indirect effect of social anxiety on SBP reactivity via loneliness. Analyses are adjusted for potential confounding variables including age, sex, BMI, smoking status, prescription medication use and baseline cardiovascular measures.

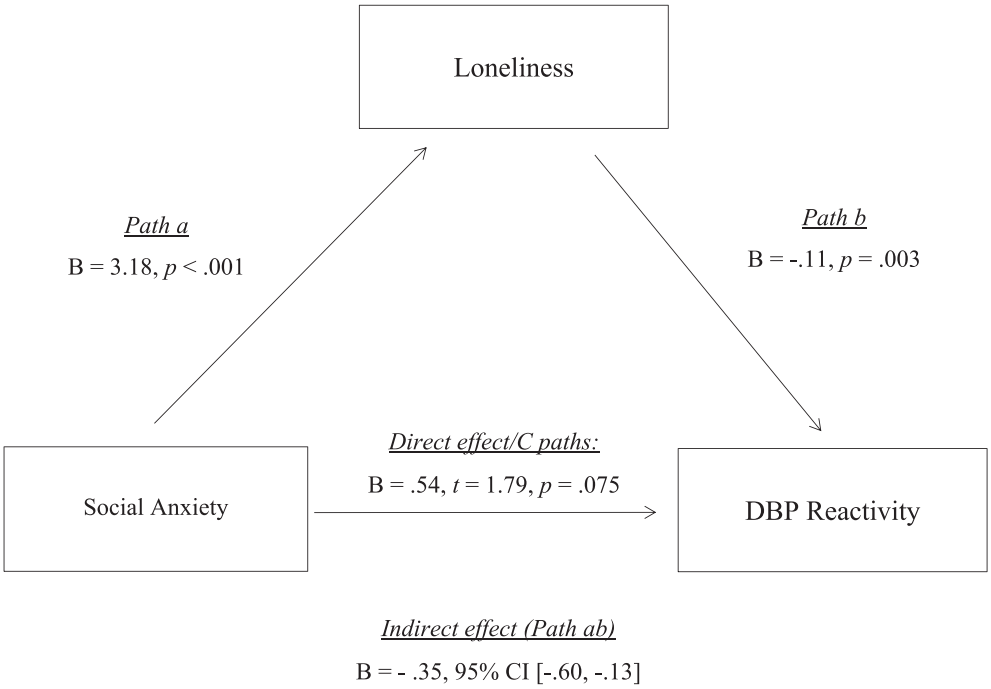

Fig. 2. Mediation path diagram: Indirect effect of social anxiety on DBP reactivity via loneliness. Analyses are adjusted for potential confounding variables including age, sex, BMI, smoking status, prescription medication use and baseline cardiovascular measures.

current study, our findings may not support a direct physiological pathway leading to adverse cardiovascular health for highly socially anxious individuals.

However, blunted SBP and DBP reactivity have been found to predict an array of non-cardiac health-related outcomes both cross-sectionally and prospectively. Cross-sectionally, blunted SBP and DBP reactivity have been associated with smoking status (Ginty et al., 2014; Keogh et al., 2024; Phillips et al., 2009b), obesity (Jones et al., 2012; Singh and Shen, 2013), depressive symptoms (Keogh et al., 2022; Keogh et al., 2021), and poor cognitive function (Ginty et al., 2012), as well as personality traits associated with poorer cardiovascular health outcomes (e.g., Type D personality, neuroticism) (Bibbey et al., 2013; Lü and Yao, 2021; O'Leary et al., 2013). Prospectively, blunted SBP and DBP reactivity have been noted to predict increased self-reported illness frequency over the past 12 months (Lawler and Schmied, 1992), smoking relapse (al'Absi et al., 2005), and a decline in cognitive functioning

(Ginty et al., 2011; Yano et al., 2016). Interestingly many of the aforementioned corollaries and prospective outcomes associated with blunted SBP and DBP reactivity are predictive of cardiovascular disease, and thus, these blunted responses are often posited to constitute an indirect pathway leading to poorer cardiovascular health via behavioral and psychological mechanisms (Phillips et al., 2013; Whittaker et al., 2021). Therefore, blunted SBP and DBP reactivity may be indicative of an indirect pathway, rather than a direct physiological pathway, leading to adverse cardiometabolic health outcomes noted amongst highly socially anxious individuals identified in previous literature (Carlton et al., 2021; Landén et al., 2004; Räikkönen et al., 2001; Shen et al., 2008; Stein et al., 2014).

From a theoretical perspective, blunted cardiovascular responses to acute psychological stress are hypothesized to reflect suboptimal functioning of neuroanatomical areas localizing in the fronto-limbic system, resulting in a motivational and behavioral dysregulation (Carroll et al., 2017; Lovallo, 2011; Phillips et al., 2009a; Phillips et al., 2013). In turn, inadequate functioning of these neuroanatomical areas that regulate motivational and goal-driven behaviors, engenders vulnerability to an array of adverse behavioral and psychological outcomes (Carroll et al., 2017). This motivational basis of blunted cardiovascular reactivity is corroborated by theories of motivational disengagement, whereby blunted cardiovascular responses are suggested to reflect deficient engagement during motivated performance situations (Hase et al., 2020). In fact, the motivational intensity theory suggests that cardiovascular reactions to stress may reflect effort mobilization. However, when the task is perceived as too difficult and/or the necessary effort to succeed is not justified, individuals disengage and display diminished reactivity (Brehm and Self, 1989; Silvestrini, 2017; Wright, 1996). Importantly, prior research has consistently shown affective influences on this type of disengagement (Gendolla, 2012; Gendolla, 2025; Gendolla and Brinkmann, 2005). For example, highly dysphoric individuals exhibit diminished reactions in response to objectively difficult tasks, but not to objectively easy tasks (Brinkmann and Gendolla, 2008). Gendolla (2025) explains how affective states provide information pertaining to the perceived task demands, thereby calibrating effort mobilization, as reflected in cardiovascular responses. Furthermore, in the case of difficult tasks, certain affective states (e.g., sadness) can result in disengagement due to excessive perceptions of subjective demand, resulting in diminished reactivity. In line with the motivational basis of stress reactivity, the blunted blood pressure reactivity associated with social anxiety and loneliness in the current study may reflect disengagement due to feeling overchallenged. This is plausible, given that social anxiety was associated with greater levels of self-reported stress.

Moreover, the indirect association between social anxiety and cardiovascular reactivity through increased levels of loneliness may be further explained by the generalized unsafety theory of stress (See Brosschot et al., 2016). Brosschot et al. (2016) propounds that the stress response is not merely engendered by the perceptions of fear or threat during a particular situation (i.e., not a generated reaction), but rather, the product of an inability/uncertainty to perceive safety signals from one's environment. This ideological perspective posits that the stress response is a default response, that is only "turned off" by an inhibition of prefrontal-subcortical areas once safety signals are perceived (Brosschot et al., 2018). Interestingly, Brosschot et al. (2016) suggests that individuals with anxiety have not learned to identify safety signals across differential contexts, resulting in the perception of generalized unsafety and a disinhibition of the default stress response. It is further propounded that individuals who report increased levels of loneliness are deprived of their primary source of safety (i.e., social network), resulting in feelings of uncertainty, unsafety and chronic disinhibition of the default stress response. Moreover, while the default stress response primarily explains how prolonged stress-related physiological activity can persist across various situations, even without the presence of a stressor (Brosschot et al., 2016, 2018), O'Connor et al. (2021) explains how the default stress response can result in overuse of physiological systems, resulting in allostatic overload and blunted responses to acute stress. Thus, in the context of the current findings, loneliness and social anxiety may be characterized by a reduced ability to identify safety signals, particularly during exposures to acute psychological stress, which may disrupt physiological systems, resulting in blunted reactivity.

A final explanation for the current findings may pertain to the concept of emotional dampening of blood pressure, whereby blood pressure increases may serve as an automatic mechanism to reduce emotional distress via feedback systems (e.g., endogenous opioid and baroreflex systems) (Delgado et al., 2014). Indeed, elevated cardiovascular measures have been associated with a reduced perception of emotional stimuli (McCubbin et al., 2011), and lower emotional arousal (Pury et al., 2004). Extending on this emotional dampening paradigm, Delgado et al. (2014) found that this blood pressure dampening effect not only applies to transient emotional states and reactions, but also to emotional traits such as worry, where higher blood pressure correlates with lower levels of trait worry. Conversely, on the flip side of the emotional dampening paradigm, lower cardiovascular measures may correspond with heightened emotional experiences such as increased levels of anxiety and worry. Thus, while much of the research surrounding the emotional dampening framework has primarily focused on resting cardiovascular function, diminished or blunted cardiovascular responses to acute stress may exacerbate negative emotions such a as anxiety and subjective loneliness.

One limitation of the current study pertains to the cross-sectional design and inability to definitively determine causality, and therefore the direction of the association between variables examined. In fact, it is important to acknowledge there may be alternative explanations relating to the direction of our significant mediation models. In particular, prior research has indicated that blunted SBP reactivity to acute stress predicts anxiety symptoms cross-sectionally, as well as prospectively whilst controlling for initial baseline levels (Sherwood et al., 2017). This alternative explanation of the relationship between anxiety symptoms and cardiovascular reactivity is premised on the perspective that blunted SBP reactivity may be characterized by a deregulation of beta-adrenergic receptors, which may increase susceptibility to anxiety. This proposed mechanistic pathway is corroborated by prior research showing an association between anxiety and sensitivity of beta-adrenergic receptors (Kang and Yu, 2005). Thus, there may be alternative explanations that explain the relationship between social anxiety, loneliness and blood pressure reactivity, than the mediation model proposed here. We recommend that future research employ longitudinal cross-lagged models to determine the directionality of the relationship between social anxiety and cardiovascular reactivity, with both variables assessed concurrently at two separate time points to explore the potential bidirectional relationship over time.

Secondly, the current study examined the influence of trait social anxiety on a continuum of severity within the general population. This approach does add strength to the current study by (1) examining the normal distribution and continuous nature underlying social anxiety and (2) by determining the dimensional prediction of social anxiety that accurately represent the heterogeneity of anxiety symptomology (Cuijpers, 2014; Dell'Osso et al., 2003; Ruscio, 2010). However, given that individuals with a clinical diagnosis of social anxiety disorder may possess a more chronic form of loneliness due to severe social avoidance and isolation (Chou et al., 2011; Davidson et al., 1993; Stangier et al., 2006), the effects reported here may be particularly pronounced amongst those with a clinical diagnosis of social anxiety disorder. Thus, we recommend for future research to examine the potential mediation effects of loneliness amongst clinical samples, and in response to highly social stressors designed to elicit feelings of social threat and evaluation. Thirdly, recent evidence has suggested that examining cardiovascular reactivity to repeated stress exposure may provide a more accurate indication of an individual's general stress response tendency than cardiovascular reactivity to a singular stress exposure (Hughes et al.,

2011; Hughes et al., 2018). Thus, we also recommend for future research to examine the potential mediation effects of loneliness on the association between social anxiety and cardiovascular adaptation to repeated stress exposure. Finally, while the data that support the findings of this study are openly available and can be accessed via the MIDUS Portal, this study was not pre-registered. However, as previously mentioned, decisions pertaining to methodological approaches and analyses were based on previous cardiovascular reactivity research.

In sum, the current study indicates that loneliness significantly mediates the association between trait social anxiety and cardiovascular reactivity to acute psychological stress. In particular, greater levels of trait social anxiety predicted increased levels of loneliness, which in turn was associated with diminished SBP and DBP reactivity to acute stress. These findings indicate a potential mechanistic pathway leading to adverse health outcomes amongst highly socially anxious individuals, and provide a potential area for targeted intervention to promote adaptative physiological stress responses for socially anxious individuals.

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CRediT authorship contribution statement

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

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Declaration of competing interest

All authors declare no conflict of interest.

Data availability

Data will be made available on request.

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