Physiology & Behavior 287 (2024) 114709

Contents lists available at ScienceDirect

Physiology & Behavior

journal homepage: www.elsevier.com/locate/physbeh

Examining the moderating effects of anger expression style on the association between facets of trait anger and cardiovascular responses to acute psychological stress

Adam O'Riordan [a,*], Aisling M. Costello [b]

[a] Department of Psychology, College for Health, Community and Policy, The University of Texas at San Antonio, San Antonio, TX, United States
[b] Department of Psychology, Trinity University, San Antonio, TX, United States

ARTICLE INFO

Keywords:
Trait anger
Anger expression
Suppression
Cardiovascular reactivity
Stress

ABSTRACT

Objective: The current study aims to (1) examine the association between measures of trait anger (i.e., anger temperament and anger reaction) and cardiovascular reactivity to acute psychological stress, and (2) to identify if anger expression styles moderate the association between trait anger and cardiovascular reactivity.
Methods: A sample of 669 participants completed a standardized cardiovascular reactivity protocol consisting of 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 measures of trait anger including anger temperament and anger reaction, as well as measures assessing anger expression styles including anger-in, anger-out and anger control.
Results: Anger temperament was significantly associated with blunted cardiovascular reactivity, as well as increased levels of subjective stress. Moreover, the association between anger temperament and cardiovascular reactivity was significantly moderated by anger-in, with associations observed only amongst those who reported an increased tendency to suppress their anger. The association between anger reaction and cardiovascular reactivity was moderated by both anger-out and anger control.
Conclusion: While blunted cardiovascular responses may be a mechanism linking facets of trait anger to adverse health outcomes, the current findings accentuate the importance of considering expression styles when examining the association between anger experience and cardiovascular reactivity.

1. Introduction

Cardiovascular diseases are currently the leading cause of global mortality, with an estimated 17.9 million cardiovascular related deaths each year, representing 32 % of all global deaths [1]. In addition to well established traditional risk factors including obesity, diabetes, physical inactivity and smoking status [2-4], a further line of research has continued to accentuate the role of psychological factors in the etiology and progression of cardiovascular diseases [5-7]. Early investigations into the role of psychological factors in cardiovascular morbidity and mortality predominantly focused on the Type A behavioral patten, characterized by aggressiveness, competitiveness, short temper, anger, heightened time consciousness, continual preoccupation with deadlines, and an inability to relax [8-10]. While a series of earlier studies linked the Type A behavioral pattern to adverse cardiovascular health outcomes/cardiovascular risk factors [11-13], later studies identified that anger/hostility was the "toxic" component promoting poorer cardiovascular health outcomes [14-16].

Moreover, a meta-analysis of 44 studies linked anger/hostility to an increased risk of experiencing an array of prospective coronary heart disease outcomes in both healthy populations, and populations with existing cardiovascular conditions [17]. More recent evidence has continued to show associations between anger and a range of adverse cardiovascular health outcomes including atherosclerosis, hypertension, myocardial infarction, stroke and cardiac/all-cause mortality [18-23]. In fact, Anger is now identified as a psychological risk factor for cardiovascular disease by the European Cardiovascular Disease Task Force [24].

The multidimensional nature of trait anger has received considerable attention in the literature, and several underlying factor structures have

* Corresponding author.
E-mail address: Adam.ORiordan@UTSA.Edu (A. O'Riordan).

https://doi.org/10.1016/j.physbeh.2024.114709
Received 5 July 2024; Received in revised form 19 September 2024; Accepted 2 October 2024
Available online 5 October 2024

been proposed [25,26]. One proposed underlying factor structure differentiates between two sub facets of trait anger including trait anger temperament and trait anger reaction using the Trait Anger Expression Inventory [27]. Conceptually, these facets of trait anger vary in terms of the degrees by which individuals experience anger across situations and environments. Anger temperament is characterized by the predisposition toward quick, unprovoked/minimally provoked anger, that occurs across a broad range of situations. In contrast, anger reaction refers to the tendency to experience anger aroused in response to frustration, criticism, unfair treatment, or environmental triggers [27,28]. Further, several studies employing factor analyses have confirmed the unique structure of the anger temperament and anger reaction facets, supporting the empirical distinction between scales [29-32]. Moreover, studies have corroborated the unique and independent prediction of these individual facets for adverse cardiometabolic health outcomes. For example, trait anger temperament has been associated with an increased risk of coronary heart disease outcomes (e.g., myocardial infarction) and cardiovascular death [33], an increased risk of developing diabetes [34], and increased atherosclerosis of the carotid artery [35]. Others have reported that increased levels of trait anger reaction are associated with greater systemic inflammation [36], increased arterial stiffness [37], and greater intima-media thickness of the carotid artery [38]. Thus, in addition to conceptual and empirical distinctions, these individual facets have unique and independent predictive utility for cardiometabolic health outcomes.

A further line of research has accentuated the influence of anger expression styles on cardiovascular health outcomes. A particular focus has been placed on anger in, which is characterized by internalizing and suppressing anger, and directing that anger inward toward oneself [39,40]. Anger in has been linked to several health outcomes including hypertension [41], as well as an increased risk of myocardial infarction and cardiac death [42,43]. However, overtly and externally expressed anger towards others/environment (i.e., anger out) has also been associated with a range of adverse outcomes including greater intima-media thickness [35], an increased risk of stroke [44] and cardiovascular mortality [43]. Thus, it has been posited that increased levels of anger in and anger out are representative of bidirectional extremes in anger expression, and therefore may predispose individuals to poor cardiovascular health [45]. In contrast, the ability to successfully and healthy control the expression of anger (i.e., anger control) has been associated with a reduced risk of cardiovascular disease and ischemic heart disease [46].

Aberrant physiological responses to acute psychological stress have been posited as a potential mechanism facilitating the association between trait anger/anger expression and cardiovascular health [47,48]. This mechanism is premised on the cardiovascular reactivity hypothesis, which posits that exaggerated or prolonged cardiovascular responses to psychological stress promotes the development of cardiovascular diseases [49]. A more recent extension of this hypothesis has also indicated that atypically low or "blunted" cardiovascular responses may also signal psychosomatic disease vulnerability [50,51]. Interestingly, a large number of studies have indicated that the influence of trait anger and hostility on physiological responses to acute stress varies depending on expression tendencies of individuals [52-56]. In particular, several studies have indicated that increased levels of anger/hostility paired with the tendency to suppress anger (i.e., high anger-in), promotes larger cardiovascular responses [52,53,56]. Burns [52] reported interaction effects of anger expression and trait anger on measures of cardiovascular reactivity, whereby high trait anger was associated with greater SBP, DBP and HR responses to a harassment reaction time stressor only amongst men who suppressed their anger. Similarly, Burns, Friedman [53] noted moderating effects of anger-in on the relationship between hostility and measures of cardiac reactivity (i.e., pre-ejection period and inter-beat interval) during exposure to a mental arithmetic stress task, with greater cardiac reactivity exhibited amongst individuals reporting both increased hostility and anger-in. A similar interactional effect was noted by Vella and Friedman [56], whereby high levels of hostility were associated with greater DBP reactivity in response to a mental arithmetic task only amongst individuals who scored high on anger-in.

In contrast, while Bongard, al'Absi [55] revealed no significant interactional effects with anger-in, anger-out significantly moderated the association between high levels of hostility and cardiovascular reactivity, with highly hostile individuals displaying low SBP, DBP and HR reactivity only when scoring high on anger-out expression. While, Burns and Katkin [54] also reported a similar interaction effect between anger-out and hostility, high levels of hostility were found to be associated with greater SBP, DBP and HR reactivity amongst individuals who outwardly expressed their anger. While these prior studies have accentuated the importance of considering anger expression styles when examining the association between trait anger and cardiovascular reactivity, these studies have yielded inconsistent findings. Additionally, the moderating role of anger control (i.e., successful and healthy control of anger expression) on the association between trait anger and cardiovascular reactivity remains unclear. Moreover, while sub facets of trait anger including anger reaction and anger temperament have been shown to independently predict adverse cardiometabolic health outcomes [33-38], research has not elucidated the association between these individual facets and cardiovascular reactivity to stress.

Considering the above evidence, the primary aims of the current study are (1) to examine the association between facets of trait anger (i.e., anger temperament and anger reaction), and anger expression styles (i.e., anger-in, anger-out, and anger control) and cardiovascular reactivity to acute psychological stress, and (2) to identify if the association between facets of trait anger and cardiovascular reactivity are moderated by anger expression styles. Given that anger reaction encapsulates the propensity to experience anger in response to frustration, criticism, unfair treatment, or environmental triggers [27,28], we hypothesize that effects will be pronounced for anger reaction in comparison to anger temperament. Moreover, it is expected that the association between facets of trait anger and cardiovascular reactivity will be pronounced amongst those who report lower control of emotional expression, increased anger-in (i.e., suppression), as well as those who report increased anger-out.

2. Material and methods

2.1. Participants

Data from the current study was taken from the MIDUS 2 biomarker project, which was collected between 2004 and 2009. Biomarker data was collected at three General Clinical Research Centers (GCRC) (University of California Los Angeles, University of Wisconsin, and Georgetown University). The biomarker projects consisted of 1255 respondents. A total of 1054 respondents were individuals from the longitudinal study and took part in the baseline MIDUS 1 project. The MIDUS 2 project also included an additional sample from Milwaukee, which was included to examine health issues in minority populations. The Milwaukee area was stratified according to the proportion of the population that were African American. Those areas with high concentrations were sampled at higher rates than areas with lower concentrations. An additional 201 participants were also included in the biomarker project from the Milwaukee sample. As per previous research [57,58] a sample of 388 participants from the twins longitudinal study was removed due to the potential confound pertaining to the genetic determinants of reactivity [59], and due to the assumption of independence in analyses. An additional 16 participants who completed a longer version of the protocol were also excluded from the current study. Finally, a further 9 participants were excluded due to having a pacemaker implanted. Only participants who had complete data for variables used in primary analyses were included, resulting in 669 participants.

2.2. Procedure

MIDUS participants were eligible to participate in the biomarker projects if they completed the MIDUS 2 Survey project phone interview and self-administered questionnaire, or completed the Milwaukee 1 survey project. Eligible participants were asked to attend an overnight stay at a GCRC, at one of three sites (University of California Los Angeles, University of Wisconsin, or Georgetown University Site 3). Verbal consent was obtained once participants agreed to take part in the study and then participants were scheduled for a GCRC visit. Additionally, written consent was obtained from participants before beginning the study procedure. Self-administered questionnaires were completed on day one, and the psychophysiology experimental protocol was completed on day two.

2.3. Measures

2.3.1. Trait anger and anger expression

Measures of trait anger and anger expression were assessed using the State-Trait Anger Expression Inventory [40]. Anger temperament and anger reaction were assessed using 4 items each from the trait anger scale. Examples of items measuring anger temperament include "*I have a fiery temper*" and "*I am a hotheaded person*". Examples of items measuring anger reaction include "*It makes me furious when I am criticized in front of others*" and "*I feel infuriated when I do a good job and get a poor evaluation*". Participants responded to each item on a 4-point Likert scale ranging from 1 (Almost never) to 4 (Almost always) in correspondence to how they feel each statement describes themselves. Both facets of trait anger were shown to have adequate internal consistency with a Cronbach's α of 0.82 and 0.74 for anger temperament and anger reaction respectively.

The anger expression in and anger expression out subscales were both assessed using 8-items each. Examples of items measuring anger-in include *"I boil inside, but don't show it"* and *"I keep things in"*. Examples of anger-out items include *"I express my anger"* and *"I strike out at whatever infuriates me"*. Finally, four items were used to measure the control of anger expression. Examples include *"I control my temper"* and *"I calm down faster"*. Again, responses to anger expression variables were answered on 4-point Likert scale ranging from 1 (Almost never) to 4 (Almost always). Again, these scales displayed adequate internal consistency with Cronbach's α of 0.80, 0.77 and 0.67 for anger-in, anger-out and anger control scales respectively.

2.3.2. Self-reported stress

Participants were asked to report their current levels of stress before and after each stressor on a 10-point Likert scale ranging from 1 (not at all stressed) to 10 (extremely stressed). For the purpose of the current study, self-reported stress was calculated as the average stress levels across both stress exposures. Changes in self-reported stress (from pre to post), were used as a manipulation check to examine if the stress task was perceived as psychologically stressful. As per previous research [60], post-task measures of self-reported stress were used in main analyses as an outcome variable.

2.3.3. Cardiovascular assessment

Continuous beat-beat monitoring of SBP and DBP were assessed using a Finometer cardiovascular monitor. Noninvasive measures are taken from one's finger arterial pressure using the volume clamp method [61]. A finger cuff is attached to the participant's middle finger on their non-dominant arm, and an arm cuff is attached to the participants upper arm at heart level. The arm cuff is used to accurately calibrate reconstructions of the intrabrachial pressure derived from the finger cuff. The Finometer also uses a hydrostatic height correction system to correct participant's hand height to heart level. Heart rate (HR) was measured using a beat-to-beat electrocardiogram (ECG). Beat to beat ECG signals were collected and digitized at a sampling rate of 500 Hz. R waves were detected using proprietary event detection software. The ECG waveforms were inspected by research staff to identify errors in R wave detection due to software errors. The series of normal RR intervals were then used to compute HR (i.e., beats per minute). 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-minute psychological stress tasks. Baseline values were computed as the average of the two epochs taken during the baseline period, and stress task values were computed as the mean of the epoch taken during each stressor. If only one epoch was available for baseline/task periods, this value was used in place of mean values.

2.3.4. Stress task

The stress tasks included a mental arithmetic task and a Stroop task. The Morgan and Turner Hewitt (MATH) task was used for the mantal arithmetic [62]. During this computer-administered task, participants are presented with math problems whereby they respond to a series of addition or subtractions by indicating if the response presented is correct or incorrect. The difficulty of the task can range across 5 levels from math problems involving two 1-digit numbers (level 1) to problems involving two 3-digit numbers (level 5). The difficulty was modified throughout the task depending on participants performance. During the modified Stroop color-word task, participants were presented with one of four color name works (i.e., red, green, yellow, blue) on a computer screen in a font color that is either congruent or incongruent with the name. Participants were required to select the font color (i.e., ink color) of each word on a keypad. The speed at which words were presented varied with performance, with more correct responses resulting in faster presentation, and incorrect responses resulting in slower presentation.

2.4. Statistical analyses

Cardiovascular reactivity scores were computed using the delta method, whereby baseline scores were subtracted from respective stress task scores for each cardiovascular parameter (i.e., stress task—baseline), producing cardiovascular reactivity scores for SBP, DBP and HR. Preliminary analyses were conducted to identify outliers on cardiovascular reactivity scores deviating ±4.00 SD from the mean. This resulted in one outlier being removed for SBP reactivity, and 2 outliers being removed for HR reactivity.

In order to examine if the stressor successfully perturbed the cardiovascular system, a series of paired samples *t*-tests (baseline, task) were conducted on each cardiovascular parameter. 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 measures of trait anger and cardiovascular reactivity. Potential confounding variables including age, sex, BMI, current smoking status and baseline cardiovascular measures were entered into models at step 1, followed by anger temperament and anger reaction at step 2. For self-reported stress, age and sex were entered at step 1 followed by trait anger variables at step 2. Similarly, in order to examine the association between anger expression and cardiovascular reactivity, potential confounding variables were entered at step 1, followed by anger-in, anger-out and anger control at step 2. A preliminary collinearity diagnostic revealed that there was no indication of multicollinearity between trait anger variables, or between anger expression variables with all VIF < 10 (Largest $= 1.17$) and all tolerance statistics >0.1 (Lowest $= 0.85$).

Moderation analyses were conducted using model 1 of Hayes PROCESS macro for SPSS. Here, trait anger variables (i.e., anger temperament, anger reaction) were entered into models separately as predictor variables, and anger expression variables (anger in, anger out, anger control) were entered into models as moderating variables. 95 % confidence levels for confidence intervals were estimated using bootstrapping samples of 5000. Significant interaction effects were followed-up to examine the conditional effects of trait anger on cardiovascular

reactivity at low (−1 SD), intermediate (mean) and high (+1 SD) levels of anger expression. Again, all moderation analyses were adjusted for aforementioned confounding variables including age, sex, BMI, smoking status and baseline cardiovascular variables.

3. Results

3.1. Descriptive statistics and manipulation check

Participants ranged in age from 35 to 85 ($M = 55.96$, SD = 11.02 years). A total of 373 (55.8 %) participants were female. Furthermore, 450 (67.3 %) were white, 131 (19.6 %) were African American, 1 (0.1 %) was Native American or Alaska native Aleutian islander/eskimo, 3 (0.4 %) were Asian, and 65 (9.7 %) were multiracial (reported more than one race), and 17 (2.5 %) reported other. Descriptive statistics of demographic and study variables are displayed in Table 1.

A series of paired samples *t*-tests revealed that the stress task successfully perturbed the cardiovascular system for SBP, $t(668) = 26.32$, $p < 0.001$, $d = 11.77$, DBP, $t(668) = 34.94$, $p < 0.001$, $d = 4.34$, and HR, $t(668) = 27.01$, $p < 0.001$, $d = 3.39$. 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 $t(665) = 35.94$, $p < 0.001$, $d = 1.81$, indicating that the stressor phase was perceived as psychologically stressful.

3.2. Trait anger, anger expression and self-reported stress

In hierarchical multiple linear regression models, age and sex were entered into models at step 1, followed by anger temperament an anger reaction at step 2. Both anger temperament, $\beta = 0.08$, $t = 2.02$, $p = 0.044$, and anger reaction, $\beta = 0.10$, $t = 2.41$, $p = 0.016$, were both independently associated with greater levels of self-reported stress during exposure to the stressor. Similar analyses for anger expression variables revealed that anger-in was associated with greater levels of self-reported stress, $\beta = 0.14$, $t = 3.49$, $p < 0.001$. Additionally, greater levels of control of anger expression were associated with lower self-reported stress, $\beta = -0.10$, $t = -2.51$, $p = 0.012$. Anger-out was not significantly associated with perceived stress, $\beta = 0.02, 0.44$, $p = 0.664$.

3.3. Trait anger, anger expression and cardiovascular reactivity

In analyses controlling for age, sex, BMI, smoking status and baseline cardiovascular measures, anger temperament was associated with lower DBP, $\beta = -0.10$, $t = -2.68$, $p = 0.008$ and HR reactivity, $\beta = -0.08$, $t = -1.97$, $p = 0.049$. Although anger temperament was also associated with lower SBP reactivity, this association did not reach statistical significance, $\beta = -0.07$, $t = -1.76$, $p = 0.080$. For anger expression variables, greater anger control was significantly associated with increased SBP, $\beta = 0.10$, $t = 2.51$ $p = 0.012$, DBP, $\beta = 0.09$, $t = 2.42$, $p = 0.016$, and HR reactivity, $\beta = 0.11$, $t = 2.79$, $p = 0.005$. As seen in table 3, no significant associations emerged for anger reaction, or for either anger-in or anger-out.

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

Variables	Mean (SD)/N (Percent)	Range
Race *n* (%)		
Black or African American	131 (19.6 %)	–
Asian	3 (0.4 %)	–
White	450 (67.3 %)	–
Multiracial	65 (9.7 %)	–
Native American/Alaska native	1 (0.1 %)	–
Other	17 (2.5 %)	–
Sex (% female)	373 (55.8 %)	–
Age (Years)	55.96 (11.02)	35–85
Trait Anger		
Anger Temperament	5.22 (1.77)	4.00–16.00
Anger reaction	7.63 (2.43)	4.00–16.00
Anger Expression		
Anger In	14.76 (4.11)	8.00–30.00
Anger Out	13.14 (3.43)	8.00–29.00
Anger Control	9.77 (2.30)	4.00–13.00
SBP Reactivity (mmHg)	11.89 (11.59)	−21.60–52.65
DBP Reactivity (mmHg)	5.86 (4.34)	−10.10–21.40
HR reactivity (bpm)	3.49 (3.26)	−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)	124.93 (18.83)	136.91 (22.20)**
DBP (mmHg)	61.85 (12.05)	67.71 (12.51)**
HR (bpm)	72.75 (10.64)	76.29 (11.02)**
Percieved stress	2.08 (1.54)	4.60 (1.94)**

** =Statistically significant difference from respective baseline value at $p < 0.001$ level.

3.4. Moderation analysis

3.4.1. Trait anger temperament and anger expression

In moderation analyses, trait anger temperament was entered into models as the predictor, and anger expression variables were entered into separate models as the moderating variable. All models adjusted for the aforementioned confounding variables including age, sex, BMI, smoking status and baseline cardiovascular measures. Anger in (i.e., the suppression of anger) moderated the association between anger temperament and SBP Reactivity, $\beta = -0.11$, 95 % CI [−0.20, −0.01], $t = -2.26$, $p = 0.024$. Anger temperament was associated with lower SBP reactivity amongst individuals who scored high on anger-in (+1 SD), $\beta = -0.62$, 95 % CI [−1.14, −0.11], $t = -2.38$, $p = 0.018$. However, no effects were observed amongst those who reported low (−1 SD), or intermediate levels of anger-in. Similarly, there was also a significant anger temperament × anger-in interaction for DBP reactivity, $\beta = -0.04$, 95 % CI [−0.07, −0.001], $t = 2.04$, $p = 0.042$. Again, anger temperament was associated with blunted DBP reactivity solely amongst individuals who reported increased (+1SD) suppression of anger (i.e., high anger in), $\beta = -0.31$, 95 % CI [−0.51, −0.12], $t = 3.14$, $p = 0.002$. No association between anger temperament and DBP reactivity was observed amongst those scoring low or intermediate on anger-in. However, anger-in did not significantly moderate the association between anger temperament and HR reactivity, $\beta = -0.11$, 95 % CI [−0.02, 0.04], $t = 0.77$, $p = 0.439$. Neither the outward expression of anger (all p's ≥ 0.253), or the control of anger expression (all p's ≥ 0.479), moderated the association between anger temperament and measures of cardiovascular reactivity. Significant interactional effects for anger temperament are displayed in Fig. 1.

3.4.2. Trait anger reaction and anger expression

Trait anger reaction was entered into models as the predictor, and anger expression variables were entered into separate models as the moderating variable. All models adjusted for the aforementioned confounding variables including age, sex, BMI, smoking status and baseline cardiovascular measures.

The association between anger reaction and SBP reactivity was significantly moderated by anger-out, $\beta = -0.17$, 95 % CI [−0.27, −0.07], $t = -3.28$, $p = 0.011$. Higher levels of anger reaction was associated with diminished SBP reactivity amongst individuals who reported greater levels of the outward expression of anger, $\beta = -0.52$, 95 % CI [−0.99 −0.04], $t = -2.13$, $p = 0.034$. In contrast, anger reaction was associated with greater SBP reactivity amongst those who reported low (−1 SD) levels of anger out expression, $\beta = 0.63$, 95 % CI [−0.11,

Table 3
Regression analyses for the influence of trait anger and anger expression 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	0.23	6.07	**<0.001**	0.18	4.71	**<0.001**	−0.14	−3.65	**<0.001**	0.07	1.91	0.057
Sex	−0.11	−2.97	**0.003**	−0.04	−0.92	0.356	0.07	1.83	0.068	0.04	1.15	0.250
Smoking Status	0.22	5.91	**<0.001**	0.17	4.50	**<0.001**	0.13	3.22	**0.001**	–	–	–
BMI	−0.06	−1.69	0.091	−0.09	−2.37	**0.018**	−0.07	−1.77	0.078	–	–	–
Baseline	−0.05	−1.44	0.150	−0.03	−0.86	0.389	−0.08	−2.12	**0.034**	–	–	–
Step 2: Trait Anger												
Age	0.22	5.85	**<0.001**	0.17	4.36	**<0.001**	−0.15	−3.91	**<0.001**	0.10	2.46	**0.014**
Sex	−0.11	−2.98	**0.003**	−0.04	−0.95	0.342	0.07	1.85	0.065	0.04	1.14	0.254
Smoking Status	0.22	5.82	**<0.001**	0.17	4.38	**<0.001**	0.12	3.09	**0.002**	–	–	–
BMI	−0.06	−1.61	0.107	−0.08	−2.20	**0.028**	−0.06	−1.57	0.118	–	–	–
Baseline	−0.05	−1.43	0.155	−0.04	−0.99	0.324	−0.09	−2.25	**0.025**	–	–	–
Anger Temperament	−0.07	−1.76	0.080	−0.10	−2.68	**0.008**	−0.08	−1.97	**0.049**	0.08	2.02	**0.044**
Anger Reaction	0.01	0.354	0.723	0.01	0.17	0.867	−0.02	−0.46	0.643	0.10	2.41	**0.016**
Step 2: Anger Expression												
Age	0.22	5.73	**<0.001**	0.17	4.19	**<0.001**	−0.15	−3.74	**<0.001**	0.12	3.00	**0.003**
Sex	−0.10	−2.70	**0.007**	−0.03	−0.68	0.498	0.08	2.08	**0.038**	0.04	1.12	0.263
Smoking Status	0.21	5.60	**<0.001**	0.16	4.13	**<0.001**	0.11	2.85	**0.004**	–	–	–
BMI	−0.05	−1.41	0.158	−0.08	−1.99	**0.048**	−0.06	−1.49	0.138	–	–	–
Baseline	−0.05	−1.42	0.155	−0.03	−0.84	0.402	−0.08	−2.15	**0.032**	–	–	–
Anger In	−0.001	−0.02	0.981	−0.02	−0.40	0.687	−0.02	−0.55	0.582	0.14	3.49	**<0.001**
Anger Out	0.01	0.16	0.877	−0.03	−0.75	0.452	0.02	0.43	0.668	0.02	0.44	0.664
Anger Control	0.10	2.51	**0.012**	0.09	2.42	**0.016**	0.11	2.79	**0.005**	−0.10	−2.51	**0.012**

Significance is highlighted in bold.

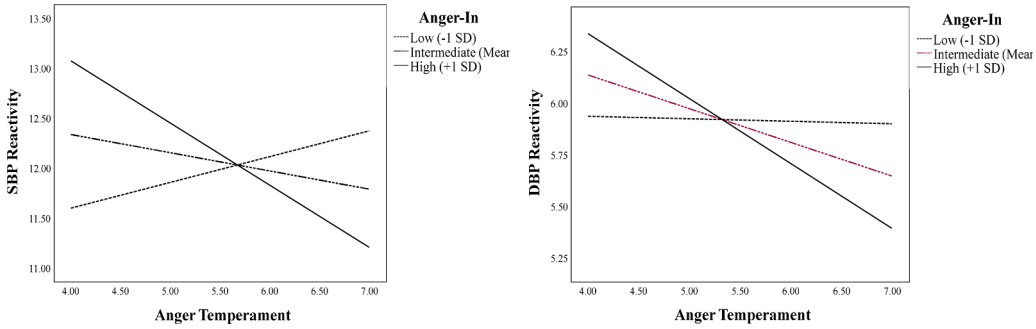

Fig. 1. Significant interactional effects between anger temperament and anger-in on SBP and DBP reactivity.

1.16], $t = 2.38$, $p = 0.017$. Similarly, a significant anger reaction × anger out expression interaction for DBP reactivity, $\beta = -0.07$, 95 % CI [−0.10, −0.03], $t = 3.33$, $p = 0.001$, revealed that anger reaction was associated with lower DBP reactivity amongst individuals reporting high levels (+1SD) of anger-out expression, $\beta = -0.21$, 95 % CI [−0.39, −0.03], $t = -2.26$, $p = 0.024$, but greater reactivity amongst those reporting low anger-out expression (−1 SD), $\beta = 0.24$, 95 % CI [.04, 0.44], $t = 2.33$, $p = 0.020$. For HR reactivity, there was also a significant anger reaction × anger out expression interaction, $\beta = -0.03$, 95 % CI [−0.06, −0.004], $t = -2.21$, $p = 0.027$. Here, greater levels of anger reaction was associated with blunted HR reactivity amongst individuals who reported high levels of anger out expression, $\beta = -0.16$, 95 % CI [−0.30, −0.02], $t = -2.18$, $p = 0.029$. Anger reaction was not related to HR reactivity amongst those who reported lower (−1 SD) or intermediate levels of anger out expression (see Fig. 2).

As seen in Fig. 3, the control of anger expression also moderated the association between anger reaction and HR reactivity, $\beta = 0.55$, 95 % CI [.01, 0.10], $t = 2.43$, $p = 0.015$. Here, greater anger reaction was associated with diminished HR reactivity amongst individuals reporting lower levels of control of anger expression, $\beta = -0.16$, 95 % CI [−3.00, −0.02], $t = -2.24$, $p = 0.025$. No associations were observed amongst individuals reporting intermediate or high levels of control of anger expression. A similar interactional effect between anger reaction and the control of anger expression was noted for DBP reactivity, $\beta = 0.06$, 95 % CI [.01, 0.12], $t = 2.17$, $p = 0.030$. While anger expression was primarily associated with diminished DBP reactivity amongst those reporting low levels of control of anger expression, this conditional effect did not meet statistical significance, $\beta = -0.16$, 95 % CI [−0.34, 0.02], $t = 1.73$, $p = 0.080$. Again, no significant associations were observed for those reporting intermediate ($p = 0.84$) or higher ($p = 0.18$) levels of control of anger expression. No moderating effect of anger in (all p's ≥ 0.102) was found for the association between anger reaction and cardiovascular reactivity.

3.5. Sensitivity analyses

Although there was no evidence of multicollinearity, follow-up regression analyses were conducted on significant associations with other anger variables removed as predictor variables. All significant associations remained significant when other anger predictor variables were removed from models.

Additionally, given that 468 (70 %) of participants from our final sample reported taking prescription medication, follow-up analyses controlling for prescription medication use were conducted on significant effects observed for cardiovascular reactivity. Apart from the association between anger temperament and HR reactivity, $\beta = -0.08$, $t = -1.90$, $p = 0.058$, all follow-up regression analyses remained significant. Additionally, all significant interactional and conditional effects in

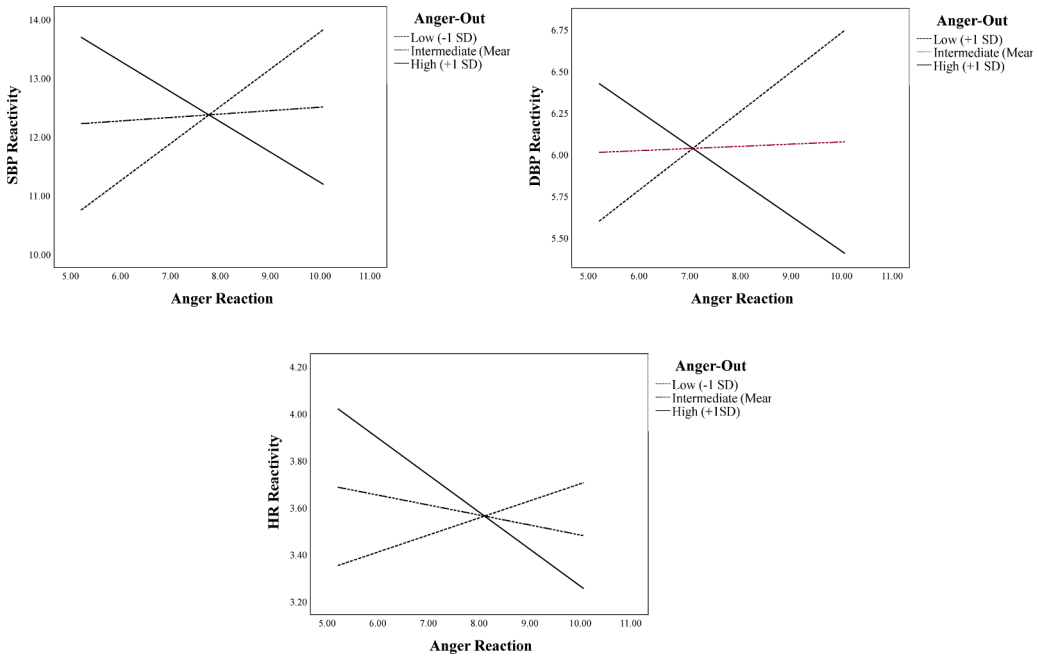

Fig. 2. Significant interactional effects between anger reaction and anger-out on SBP, DBP and HR reactivity.

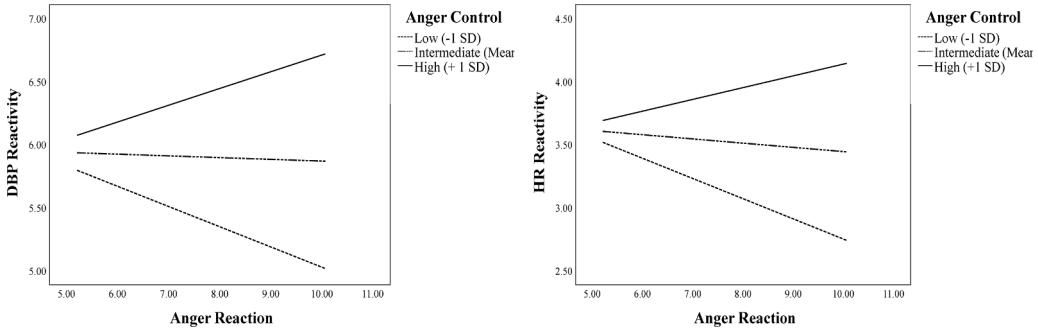

Fig. 3. Significant interactional effects between anger reaction and anger control on DBP and HR reactivity.

moderation analyses remained significant.

4. Discussion

The current study aimed to examine the association between facets of trait anger (i.e., anger temperament and anger reactions), anger expression styles (anger-in, anger-out and anger control) and cardiovascular reactivity to acute psychological stress. Secondly, the current study aimed to examine if anger expression styles moderated the association between facets of trait anger and cardiovascular reactivity to stress. Trait anger temperament was associated with greater self-reported stress following the stress exposure, as well as lower cardiovascular reactivity. While anger reaction was also associated with increased subjective stress, no significant associations were observed with measures of cardiovascular reactivity. Neither anger-in nor anger-out expression was associated with cardiovascular reactivity. However, the control of anger expression was associated with greater SBP, DBP and HR reactivity, as well as lower levels of subjective stress. While anger-in moderated the association between anger temperament and cardiovascular reactivity, both anger-out and anger-control moderated the association between anger reaction and cardiovascular reactivity.

To date several studies have corroborated an association between trait anger and cardiovascular reactivity to stress [63]. In fact, aberrant physiological stress responses have been posited as a potential pathway facilitating the association between subjective anger and adverse cardiometabolic health outcomes [47,48]. A meta-analysis including 281 studies revealed that cognate psychological factors including anger/-hostility, aggression, or Type-A behavior were associated with greater cardiovascular responses to stress across all studies [63]. In contrast, our findings suggest that the anger temperament facet is associated with lower cardiovascular reactivity to acute stress. Importantly however, several other studies have also noted similar relationships between measures of trait anger and analogous constructs, and blunted cardiovascular reactivity to acute stress [64-66]. One potential explanation for these antithetical findings may pertain to the specific measure of trait anger employed in the current study. While some prior studies linking trait anger to exaggerated cardiovascular reactivity have employed global measures of trait anger and hostility [67,68], the current study is the first to examine the sub facets of trait anger temperament and trait anger reaction. In fact, similar associations with diminished cardiovascular responses have also been previously noted when examining particular sub facets of anger and hostility. For example, studies examining certain subcomponents such as somatic anger [69], as well as cynical hostility have noted associations with blunted cardiovascular reactivity [65,66]. Thus, the varying associations between trait anger and related constructs and cardiovascular reactivity may be owing to differential constituent facets examined in the literature.

However, no significant associations between trait anger reaction

and cardiovascular reactivity were observed in the current study. This is somewhat surprising considering that anger reaction encapsulates the tendency to experience anger in response to frustration, criticism, unfair treatment, or environmental triggers [27,28]. One potential explanation may pertain to more recent research relating to atypically low or "blunted" cardiovascular responses [50,51]. It is suggested that blunted cardiovascular responses may represent stable underlying behavioral and psychological phenotypes, including personality temperaments [51]. Thus, blunted cardiovascular reactivity may signal the general tendency to experience anger across an array of situations and environments (i.e., anger temperament) rather than anger experienced in response to particular situations and triggers (i.e., anger reaction). Additionally, the current study employed a computerized mental arithmetic and Stroop stress task, without any verbal feedback. These particular stressors may have been sufficiently provocative to elicit differential responses for individuals varying on the anger temperament dimension, who experience anger across a broad range of situations and environments. However, given that anger reaction encapsulated the tendency to experience anger only in response to anger provoking triggers such as criticism and unfair treatment [27], more provocative stressors may be required to elicit cardiovascular responses for those scoring high on anger reaction, such as tasks involving harassment, interpersonal conflict, and negative verbal feedback [52,54,67].

Prior research examining the association between anger expression and cardiovascular reactivity has shown varying effects. Anger-in has been associated with both greater [70] and lower [71] cardiovascular stress responses. Similarly, mixed effects have also been noted for anger-out, with studies showing both positive [72] and negative associations [73]. However, our findings revealed no significant main effects for the association between anger-in or anger-out and measures of cardiovascular reactivity. However, consistent with prior research [71], anger control was associated with greater cardiovascular reactivity as well as lower levels of subjective stress. Taking into account recent research on blunted cardiovascular reactivity and its negative future health implications [74,75], these heightened responses among individuals with increased control of emotional expression might be seen as adaptive and potentially beneficial for promoting cardiometabolic health, as noted in prior research [46,76]. This is plausible given that the greater cardiovascular responses amongst those scoring high on anger control were paired with reduced levels of subjective stress.

Consistent with prior research [52-56], our moderation analyses revealed that the influence of trait anger on measures of cardiovascular reactivity varies depending on the tendency by which individuals expressed their emotions. Prior research has predominately indicated that high trait anger/hostility paired with the tendency to suppress anger is associated with greater cardiovascular responses to stress [52,53,56]. In contrast, our moderation analyses indicated that anger temperament was associated with diminished responses amongst individuals who suppressed their anger. However, in line with our findings, more recent evidence has indicated that individuals who engage in emotional suppression during anger recall tasks (i.e., aimed at recalling and reexperiencing anger) exhibit blunted blood pressure and heart rate responses [77]. Additionally, one parallel line of research has indicated that individuals who experience high levels of negative emotions, whilst simultaneously inhibiting the expression of these emotions (i.e., Type D personality) exhibit diminished cardiovascular responses to acute stress [78]. While exaggerated cardiovascular reactivity has been predominately associated with prospective cardiovascular health outcomes [79-81], diminished or blunted cardiovascular responses have been primarily associated with an array of non-cardiac health related outcomes [50,51,82,83]. Moreover, it has been posited that blunted cardiovascular reactivity to acute stress may serve as a marker of deficits in motivation and behavioral regulation [51]. Consequently, blunted cardiovascular responses have been associated with several cardiovascular disease risk factors (e.g., smoking status, obesity) [84-87]. Therefore, while some studies have reported a direct association between blunted cardiovascular reactivity and cardiovascular health outcomes [74], blunted reactivity is primarily posited to constitute an indirect pathway leading to poorer cardiovascular health via behavioral and psychological mechanisms [50,88]. In fact, prior research has suggested that blunted cardiovascular reactivity may indicate an indirect behavioral pathway to disease amongst highly hostile individuals [65].

Both anger-out and anger control moderated the association between anger reaction and cardiovascular reactivity. Here, increased anger reaction was associated with diminished cardiovascular reactivity amongst individuals who reported a lack of control over the expression of their anger, and those who reported overtly and externally expressing anger towards others/environment. Interesting, antithetical associations were observed for low anger-out expression, with anger reaction associated with greater blood pressure. Given more recent research relating to blunted cardiovascular reactivity and health [51], these conditional effects may be viewed as adaptive. Anger reaction refers the propensity to become angry when provoked by specific events such as personal criticism, rejection or neglect [27,28]. Thus, the outward expression of anger (e.g., striking out at perceived triggers), as well as a lack of control of anger expression (e.g., inability to control temper) may be particularly harmful for individuals who are highly anger reactive, particularly during anger provoking situations.

Prior research has continued to show that both anger temperament and anger reaction are associated with an array of adverse cardiometabolic health outcomes. For example, trait anger temperament has been associated with an increased risk of coronary heart disease outcomes, as well as cardiac mortality [33], an increased risk of developing diabetes [34], and greater atherosclerosis of the carotid artery [35]. Additionally, trait anger reaction has been linked to greater systemic inflammation [36], increased arterial stiffness [37], and greater intima-media thickness of the carotid artery [38]. Given recent research accentuating the pathogenic effects of blunted cardiovascular responses to acute stress on psychological, behavioral and physical health outcomes [74,75], the current study suggests that these responses may indicate a mechanistic pathway facilitating the association between trait anger and poorer cardiovascular health outcomes.

One limitation of the current study pertains to the psychological stressors employed. While the current study examined cardiovascular reactions to conventional psychological stressors including a mental arithmetic and Stroop task, highly anger provoking stressors such as harassment and interpersonal conflict tasks may be particular toxic for individuals who are classified as anger reactive, and who externally express their anger [52,54,67]. Such anger provoking stressors are likely to evoke deleterious psychological and physiological reactions amongst those scoring high on trait anger, and may constitute a more ecologically valid representation of stress responses exhibited in real life settings. In fact, a meta-analyses of studies examining the association between anger/hostility and cardiovascular reactivity has suggested that the stressor provocation moderates the association between anger/hostility and cardiovascular reactivity, with greater responses exhibited by high trait anger/hostile individuals in response to provocative stressors [89]. Additionally, more 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 [90,91]. Thus, we recommend future research to examine the influence of trait anger and anger expression styles on cardiovascular response habituation to recurring stress exposure designed to specifically elicit anger. The experimental manipulation of expressive suppression has been shown to perturb the cardiovascular system during exposure to psychological stress [92]. Thus, future research should extend on the current findings by examining if the instructional engagement in anger expression/suppression in the current moment moderates the association between facets of trait anger and cardiovascular stress reactivity. Furthermore, future research should examine interventions relating to the control of emotional expression (e.g., ability to control temper and calm down

faster) for promoting more healthful cardiovascular response for those scoring high on measures of trait anger, particularly trait anger reaction. Additionally, several other underlying factor structures of trait anger have been proposed in the literature [25,26]. Thus, while the current study examined the sub facets of anger temperament and anger reaction, future research should extend on the current findings by examining other proposed dimensions of trait anger, such as representing angry emotions, aggressive behaviors, and cynicism [26].

5. Conclusion

In sum, trait anger temperament was associated with blunted DBP reactivity to acute psychological stress, as well as increased levels of self-reported stress. The control of anger expression was associated with greater cardiovascular responses, and decreased levels of self-reported stress. Anger temperament was associated with diminished cardiovascular reactivity only amongst those who suppressed their anger. In contrast, anger reaction was associated with diminished cardiovascular reactions amongst those who reported lower control of anger expression, as well as amongst those who reported expressing their anger overtly and externally. These findings accentuate the importance of considering anger expression styles when examining the association between trait anger and cardiovascular stress responses.

Funding

Publicly available data from the MIDUS study was used for this research. Since 1995 the MIDUS study has been funded by the following: John D. and Catherine T. MacArthur Foundation Research Network National Institute on Aging (P01-AG020166), National Institute on Aging (U19-AG051426). Biomarker data collection was further supported by the NIH National Center for Advancing Translational Sciences (NCATS), Clinical and Translational Science Award (CTSA) program as follows: UL1TR001409 (Georgetown) UL1TR001881 (UCLA) 1UL1RR025011 (UW).

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.

Declaration of competing interest

The authors declare that they have no conflict of interest.

Data availability

The data that support the findings of this study are openly available and can be accessed via the MIDUS Portal (https://midus.colectica.org/).

References

[1] World Health Organization. Cardiovascular diseases (CVDs) 2021.
[2] H.B. Hubert, M. Feinleib, P.M. McNamara, W.P. Castelli, Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study, Circulation 67 (1983) 968–977.
[3] J. Stamler, O. Vaccaro, J.D. Neaton, D. Wentworth, Diabetes, other risk factors, and 12-yr cardiovascular mortality for men screened in the multiple risk factor intervention trial, Diabetes Care 16 (1993) 434–444.
[4] M. Helfand, D.I. Buckley, M. Freeman, R. Fu, K. Rogers, C. Fleming, et al., Emerging risk factors for coronary heart disease: a summary of systematic reviews conducted for the U.S. preventive services task force, Ann. Intern. Med. 151 (2009) 496–507.
[5] K.M. MacMahon, G.Y. Lip, Psychological factors in heart failure: a review of the literature, Arch. Intern. Med. 162 (2002) 509–516.
[6] Z. Khayyam-Nekouei, H. Neshatdoost, A. Yousefy, M. Sadeghi, G. Manshaee, Psychological factors and coronary heart disease, ARYA Atheroscler. 9 (2013) 102–111.
[7] C. Albus, C. Waller, K. Fritzsche, H. Gunold, M. Haass, B. Hamann, et al., Significance of psychosocial factors in cardiology: update 2018: position paper of the German cardiac society, Clin. Res. Cardiol. 108 (2019) 1175–1196.
[8] M. Friedman, R.H. Rosenman, Overt behavior pattern in coronary disease. Detection of overt behavior pattern A in patients with coronary disease by a new psychophysiological procedure, JAMA 173 (1960) 1320–1325.
[9] S. Sahoo, S.K. Padhy, B. Padhee, N. Singla, S. Sarkar, Role of personality in cardiovascular diseases: an issue that needs to be focused too!, Indian Heart J. 70 (Suppl 3) (2018) S471–S4s7.
[10] A. Steptoe, G.J. Molloy, Personality and heart disease, Heart. 93 (2007) 783–784.
[11] M. Friedman, R.H. Rosenman, Association of specific overt behavior pattern with blood and cardiovascular findings; blood cholesterol level, blood clotting time, incidence of arcus senilis, and clinical coronary artery disease, J. Am. Med. Assoc. 169 (1959) 1286–1296.
[12] M. Friedman, R.H. Rosenman, R. Straus, M. Wurm, R. Kositchek, The relationship of behavior pattern A to the state of the coronary vasculature. A study of fifty-one autopsy subjects, Am. J. Med. 44 (1968) 525–537.
[13] M. Friedman, S.O. Byers, R.H. Roseman, F.R. Elevitch, Coronary-prone individuals (type A behavior pattern. Some biochemical characteristics, JAMA 212 (1970) 1030–1037.
[14] A. Rozanski, J.A. Blumenthal, J. Kaplan, Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy, Circulation 99 (1999) 2192–2217.
[15] M. Myrtek, Meta-analyses of prospective studies on coronary heart disease, type A personality, and hostility, Int. J. Cardiol. 79 (2001) 245–251.
[16] R.B. Williams Jr., Refining the type A hypothesis: emergence of the hostility complex, Am. J. Cardiol. 60 (1987) 27j–32j.
[17] Y. Chida, A. Steptoe, The association of anger and hostility with future coronary heart disease: a meta-analytic review of prospective evidence, J. Am. Coll. Cardiol. 53 (2009) 936–946.
[18] O.E. Titova, J.A. Baron, K. Michaëlsson, S.C. Larsson, Anger frequency and risk of cardiovascular morbidity and mortality, Eur. Heart J. Open 2 (2022) oeac050.
[19] K. Tezuka, Y. Kubota, T. Ohira, Y. Shimizu, K. Yamagishi, M. Umesawa, et al., Anger expression and the risk of cardiovascular disease among urban and rural Japanese residents: the circulatory risk in communities study, Psychosom. Med. 82 (2020) 215–223.
[20] C. Trudel-Fitzgerald, L.R. Reduron, I. Kawachi, L.D. Kubzansky, Specificity in associations of anger frequency and expression with different causes of mortality over 20 years, Psychosom. Med. 83 (2021) 402–409.
[21] K. Tezuka, Y. Kubota, T. Ohira, I. Muraki, M. Hayama-Terada, Y. Shimizu, et al., Retirement status after the age of 60 years modifies the association between anger expression and the risk of cardiovascular disease: the circulatory risk in communities study, Geriatr. Gerontol. Int. 24 (2024) 385–389.
[22] K. Tezuka, Y. Kubota, T. Ohira, I. Muraki, M. Hayama-Terada, Y. Shimizu, et al., Impact of perceived social support on the association between anger expression and the risk of stroke: the circulatory risk in communities study (CIRCS), J. Epidemiol. 33 (2023) 159–164.
[23] W. Eckerle, A. Koldhekar, M. Muldoon, J. Stewart, T. Kamarck, Independent associations between trait-anger, depressive symptoms and preclinical atherosclerotic Progression, Ann. Behav. Med. 57 (2023) 409–417.
[24] M.F. Piepoli, A.W. Hoes, S. Agewall, C. Albus, C. Brotons, A.L. Catapano, et al., Guidelines: editor's choice: 2016 European guidelines on cardiovascular disease prevention in clinical practice: the sixth joint task force of the European society of cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of 10 societies and by invited experts) developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR), Eur. Heart J. 37 (2016) 2315.
[25] W.T. Riley, F.A. Treiber, The validity of multidimensional self-report anger and hostility measures, J. Clin. Psychol. 45 (1989) 397–404.
[26] R. Martin, D. Watson, C.K. Wan, A three-factor model of trait anger: dimensions of affect, behavior, and cognition, J. Pers. 68 (2000) 869–897.
[27] C.D. Spielberger, G. Jacobs, S. Russell, R.S. Crane, Assessment of anger: the state-trait anger scale. Advances in Personality Assessment, Routledge, 1983, pp. 161–189.
[28] H. Kassinove, D.G. Sukhodolsky, C.I. Eckhardt, S.V. Tsytsarev, Development of a Russian state-trait anger expression inventory, J. Clin. Psychol. 53 (1997) 543–557.
[29] D.G. Forgays, D.K. Forgays, C.D. Spielberger, Factor structure of the state-trait anger expression inventory, J. Pers. Assess. 69 (1997) 497–507.
[30] D.K. Forgays, C.D. Spielberger, S.A. Ottaway, D.G. Forgays, Factor structure of the state-trait anger expression Inventory for middle-aged men and women, Assessment 5 (1998) 141–155.
[31] V. del Barrio, A. Aluja, C. Spielberger, Anger assessment with the STAXI-CA: psychometric properties of a new instrument for children and adolescents, Pers. Individ. Difer. 37 (2004) 227–244.
[32] Alcázar Olán, R.J., Deffenbacher, J.L., Byrne, Z. Assessing the factor structure of the anger expression inventory (ML-STAXI) in a Mexican sample. 2015.
[33] J.E. Williams, F.J. Nieto, C.P. Sanford, H.A. Tyroler, Effects of an angry temperament on coronary heart disease risk: the atherosclerosis risk in communities study, Am. J. Epidemiol. 154 (2001) 230–235.
[34] S.H. Golden, J.E. Williams, D.E. Ford, H.C. Yeh, C.P. Sanford, F.J. Nieto, et al., Anger temperament is modestly associated with the risk of type 2 diabetes mellitus:

the atheroslcerosis risk in communities study, Psychoneuroendocrinology 31 (2006) 325–332.
[35] M.E. Bleil, J.M. McCaffery, M.F. Muldoon, K. Sutton-Tyrrell, S.B. Manuck, Anger-related personality traits and carotid artery atherosclerosis in untreated hypertensive men, Psychosom. Med. 66 (2004) 633–639.
[36] S. Shivpuri, L.C. Gallo, P.J. Mills, K.A. Matthews, J.P. Elder, G.A. Talavera, Trait anger, cynical hostility and inflammation in Latinas: variations by anger type? Brain Behav. Immun. 25 (2011) 1256–1263.
[37] J.E. Williams, R. Din-Dzietham, M. Szklo, Trait anger and arterial stiffness: results from the atherosclerosis risk in communities (ARIC) study, Prev. Cardiol. 9 (2006) 14–20.
[38] K. Räikkönen, K.A. Matthews, K. Sutton-Tyrrell, L.H. Kuller, Trait anger and the metabolic syndrome predict progression of carotid atherosclerosis in healthy middle-aged women, Psychosom. Med. 66 (2004) 903–908.
[39] C.D. Spielberger, E.C. Reheiser, S.J. Sydeman, Measuring the experience, expression, and control of anger, Issues Compr. Pediatr. Nurs. 18 (1995) 207–232.
[40] Spielberger, C.D. State-Trait Anger Expression Inventory: Professional Manual. Odessa, FL1996.
[41] S.A. Everson, D.E. Goldberg, G.A. Kaplan, J. Julkunen, J.T. Salonen, Anger expression and incident hypertension, Psychosom. Med. 60 (1998) 730–735.
[42] J. Denollet, Y. Gidron, C.J. Vrints, V.M. Conraads, Anger, suppressed anger, and risk of adverse events in patients with coronary artery disease, Am. J. Cardiol. 105 (2010) 1555–1560.
[43] S. Assari, Hostility, anger, and cardiovascular mortality among Blacks and Whites, Res. Cardiovasc. Med. 6 (2017) 1–9.
[44] S.A. Everson, G.A. Kaplan, D.E. Goldberg, T.A. Lakka, J. Sivenius, J.T. Salonen, Anger expression and incident stroke: prospective evidence from the Kuopio ischemic heart disease study, Stroke 30 (1999) 523–528.
[45] L.D. Kubzansky, I. Kawachi, Going to the heart of the matter: do negative emotions cause coronary heart disease? J. Psychosom. Res. 48 (2000) 323–337.
[46] A. Haukkala, H. Konttinen, T. Laatikainen, I. Kawachi, A. Uutela, Hostility, anger control, and anger expression as predictors of cardiovascular disease, Psychosom. Med. 72 (2010) 556–562.
[47] M. al'Absi, S. Bongard, Neuroendocrine and behavioral mechanisms mediating the relationship between anger expression and cardiovascular risk: assessment considerations and improvements, J. Behav. Med. 29 (2006) 573–591.
[48] J. Suls, Anger and the heart: perspectives on cardiac risk, mechanisms and interventions, Prog. Cardiovasc. Dis. 55 (2013) 538–547.
[49] P.A. Obrist, Cardiovascular Psychophysiology: A Perspective, Plenum Press, 1981.
[50] A.C. Phillips, A.T. Ginty, B.M. Hughes, The other side of the coin: blunted cardiovascular and cortisol reactivity are associated with negative health outcomes, Int. J. Psychophysiol. 90 (2013) 1–7.
[51] D. Carroll, A.T. Ginty, A.C. Whittaker, W.R. Lovallo, S.R. de Rooij, The behavioural, cognitive, and neural corollaries of blunted cardiovascular and cortisol reactions to acute psychological stress, Neurosci. Biobehav. Rev. 77 (2017) 74–86.
[52] J.W. Burns, Interactive effects of traits, states, and gender on cardiovascular reactivity during different situations, J. Behav. Med. 18 (1995) 279–303.
[53] J.W. Burns, R. Friedman, E.S. Katkin, Anger expression, hostility, anxiety, and patterns of cardiac reactivity to stress, Behav. Med. 18 (1992) 71–78.
[54] J.W. Burns, E.S. Katkin, Psychological, situational, and gender predictors of cardiovascular reactivity to stress: a multivariate approach, J. Behav. Med. 16 (1993) 445–465.
[55] S. Bongard, M. al'Absi, W.R. Lovallo, Interactive effects of trait hostility and anger expression on cardiovascular reactivity in young men, Int. J. Psychophysiol. 28 (1998) 181–191.
[56] E.J. Vella, B.H. Friedman, Hostility and anger in: cardiovascular reactivity and recovery to mental arithmetic stress, Int. J. Psychophysiol. 72 (2009) 253–259.
[57] T.M. Keogh, S. Howard, S. Gallagher, Early life adversity and blunted cardiovascular reactivity to acute psychological stress: the role of current depressive symptoms, Psychosom. Med. 84 (2022) 170–178.
[58] T.M. Keogh, S. Howard, S. Gallagher, Behavioural clusters characteristic of cardiovascular reactivity profiles relate to poorer health outcomes, Br. J. Health Psychol. 28 (2023) 513–531.
[59] D. Carmelli, M.A. Chesney, M.M. Ward, R.H. Rosenman, Twin similarity in cardiovascular stress response, Health Psychol. 4 (1985) 413–423.
[60] S. Gallagher, A. O'Riordan, G. McMahon, A.M. Creaven, Evaluating personality as a moderator of the association between life events stress and cardiovascular reactivity to acute stress, Int. J. Psychophysiol. 126 (2018) 52–59.
[61] Penaz, J. Photoelectric measurement of blood pressure, volume and flow in the finger. Digest of the 10th International Conference on Medical and Biological Engineering-Dresden, 19731973.
[62] J.R. Turner, J.K. Hewitt, R.K. Morgan, J. Sims, D. Carroll, K.A. Kelly, Graded mental arithmetic as an active psychological challenge, Int. J. Psychophysiol. 3 (1986) 307–309.
[63] Y. Chida, M. Hamer, Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: a quantitative review of 30 years of investigations, Psychol. Bull. 134 (2008) 829–885.
[64] S.A. Neumann, K.J. Maier, J.P. Brown, P.P. Giggey, D.C. Cooper, S.J. Synowski, et al., Cardiovascular and psychological reactivity and recovery from harassment in a biracial sample of high and low hostile men and women, Int. J. Behav. Med. 18 (2011) 52–64.
[65] A.T. Tyra, R.C. Brindle, B.M. Hughes, A.T. Ginty, Cynical hostility relates to a lack of habituation of the cardiovascular response to repeated acute stress, Psychophysiology 57 (2020) e13681.
[66] D. Carroll, G. Davey Smith, D. Sheffield, M.J. Shipley, M.G. Marmot, The relationship between socioeconomic status, hostility, and blood pressure reactions to mental stress in men: data from the Whitehall II study, Health Psychol. 16 (1997) 131–136.
[67] G.D. Bishop, G. Robinson, Anger, harassment, and cardiovascular reactivity among Chinese and Indian men in Singapore, Psychosom. Med. 62 (2000) 684–692.
[68] E.C. Suarez, C.M. Kuhn, S.M. Schanberg, R.B. Williams Jr., E.A Zimmermann, Neuroendocrine, cardiovascular, and emotional responses of hostile men: the role of interpersonal challenge, Psychosom. Med. 60 (1998) 78–88.
[69] L.A. Durel, C.S. Carver, S.B. Spitzer, M.M. Llabre, J.K. Weintraub, P.G. Saab, et al., Associations of blood pressure with self-report measures of anger and hostility among black and white men and women, Health Psychol. 8 (1989) 557–575.
[70] P.J. Mills, J.E. Dimsdale, Anger suppression: its relationship to beta-adrenergic receptor sensitivity and stress-induced changes in blood pressure, Psychol. Med. 23 (1993) 673–678.
[71] P.P. Vitaliano, J. Russo, S.L. Bailey, H.M. Young, B.S. McCann, Psychosocial factors associated with cardiovascular reactivity in older adults, Psychosom. Med. 55 (1993) 164–177.
[72] S. Suchday, K.T. Larkin, Biobehavioral responses to interpersonal conflict during anger expression among anger-in and anger-out men, Ann. Behav. Med. 23 (2001) 282–290.
[73] P.J. Mills, R.H. Schneider, J.E. Dimsdale, Anger assessment and reactivity to stress, J. Psychosom. Res. 33 (1989) 379–382.
[74] A. O'Riordan, S. Howard, S. Gallagher, Blunted cardiovascular reactivity to psychological stress and prospective health: a systematic review, Health Psychol. Rev. 17 (2023) 121–147.
[75] A.I. Turner, N. Smyth, S.J. Hall, S.J. Torres, M. Hussein, S.U. Jayasinghe, et al., Psychological stress reactivity and future health and disease outcomes: a systematic review of prospective evidence, Psychoneuroendocrinology 114 (2020) 104599.
[76] J.M. Boylan, C.D. Ryff, Varieties of anger and the inverse link between education and inflammation: toward an integrative framework, Psychosom. Med. 75 (2013) 566–574.
[77] J.M. Cundiff, J.R. Jennings, K.A. Matthews, Social stratification and risk for cardiovascular disease: examination of emotional suppression as a pathway to risk, Pers. Soc. Psychol. Bull. 45 (2019) 1202–1215.
[78] A. O'Riordan, S. Gallagher, S. Howard, Type D personality and cardiovascular reactivity to acute psychological stress: a systematic review and meta-analysis, Health Psychol. 42 (2023) 628–641.
[79] Y. Chida, A. Steptoe, Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: a meta-analysis of prospective evidence, Hypertension 55 (2010) 1026–1032.
[80] D. Carroll, A.T. Ginty, R.C. Painter, T.J. Roseboom, A.C. Phillips, S.R de Rooij, Systolic blood pressure reactions to acute stress are associated with future hypertension status in the Dutch famine birth cohort study, Int. J. Psychophysiol. 85 (2012) 270–273.
[81] D. Carroll, A.T. Ginty, G. Der, K. Hunt, M. Benzeval, A.C Phillips, Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality, Psychophysiology 49 (2012) 1444–1448.
[82] A.C. Phillips, Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health, Biol. Psychol. 86 (2011) 106–113.
[83] A.C. Whittaker, A. Ginty, B.M. Hughes, A. Steptoe, W.R. Lovallo, Cardiovascular stress reactivity and health: recent questions and future directions, Psychosom. Med. 83 (2021) 756–766.
[84] D. Carroll, A.C. Phillips, G. Der, Body mass index, abdominal adiposity, obesity, and cardiovascular reactions to psychological stress in a large community sample, Psychosom. Med. 70 (2008) 653–660.
[85] A.C. Phillips, T.J. Roseboom, D. Carroll, S.R. de Rooij, Cardiovascular and cortisol reactions to acute psychological stress and adiposity: cross-sectional and prospective associations in the Dutch famine birth cohort study, Psychosom. Med. 74 (2012) 699–710.
[86] A.T. Ginty, A. Jones, D. Carroll, T.J. Roseboom, A.C. Phillips, R. Painter, et al., Neuroendocrine and cardiovascular reactions to acute psychological stress are attenuated in smokers, Psychoneuroendocrinology 48 (2014) 87–97.
[87] A.C. Phillips, G. Der, K. Hunt, D. Carroll, Haemodynamic reactions to acute psychological stress and smoking status in a large community sample, Int. J. Psychophysiol. 73 (2009) 273–278.
[88] D. Carroll, W.R. Lovallo, A.C. Phillips, Are large physiological reactions to acute psychological stress always bad for health? Soc. Personal. Psychol. Compass. 3 (2009) 725–743.
[89] J. Suls, C.K. Wan, The relationship between trait hostility and cardiovascular reactivity: a quantitative review and analysis, Psychophysiology 30 (1993) 615–626.
[90] B.M. Hughes, S. Howard, J.E. James, N.M. Higgins, Individual differences in adaptation of cardiovascular responses to stress, Biol. Psychol. 86 (2011) 129–136.
[91] B.M. Hughes, W. Lü, S. Howard, Cardiovascular stress-response adaptation: conceptual basis, empirical findings, and implications for disease processes, Int. J. Psychophysiol. 131 (2018) 4–12.
[92] A.T. Tyra, T.A. Fergus, A.T. Ginty, Emotion suppression and acute physiological responses to stress in healthy populations: a quantitative review of experimental and correlational investigations, Health Psychol. Rev. 18 (2024) 396–420.