Effect of Anger and Trait Forgiveness on Cardiovascular Risk in Young Adult Females

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High trait anger is linked to adverse cardiovascular outcomes. A potential antidote to the cardiotoxic influence of anger is trait forgiveness (TF), as it has shown associations with improved blood pressure (BP) and cardiovagal tone regulation in cardiac patients. However, it has yet to be determined if anger and forgiveness independently predict cardiovascular parameters. Trait anger (State-Trait Anger Expression Inventory-2) and TF (Tendency to Forgive Scale) were evaluated in 308 (M = 21.11years ± SD = 2.52) healthy female volunteers allocated to 3 related, yet distinct, studies. Hierarchical multiple regressions tested the incremental contribution of TF after accounting for anger. Study 1 assessed autonomic modulation through beat-to-beat BP and spectral analysis to examine sympathovagal balance and baroreflex functioning. Study 2 used tonometry and pulse wave analysis for aortic hemodynamics. Study 3 assessed 24-hour ambulatory BP and ambulatory arterial stiffness index. Hierarchical models demonstrated that anger was significantly associated with increased sympathovagal tone, increased hemodynamic indices, high ambulatory BPs, and attenuated BP variability and baroreflex. In contrast, TF was associated with more favorable hemodynamic effects (i.e., decreased ventricular work and myocardial oxygen consumption). In conclusion, these results demonstrate divergent cardiovascular effects of anger and forgiveness, such that anger is associated with a more cardiotoxic autonomic and hemodynamic profile, whereas TF is associated with a more cardioprotective profile. These findings suggest that interventions aimed at decreasing anger while increasing forgiveness may be clinically relevant. **(Am J Cardiol 2014;114:47−52)**

Considerable attention has been given to the relation between anger and increased cardiac risk,[1] which is relevant to both healthy and cardiac patients. For example, research indicates anger to increase the risk of coronary heart disease among initially healthy patients and to lead to poorer prognosis for patients with heart disease.[2,3] Although the mechanism linking anger to increased cardiovascular risk is not well understood, impaired cardiovascular autonomic modulation and increased ventricular workload may be implicated. A potential antidote to the cardiotoxic influence of anger and hostility may be the cardioprotective properties provided by trait forgiveness (TF). TF has been shown to lower blood pressure (BP) and improve heart rate (HR) variability.[4,5] There is even some evidence that forgiveness predicts mortality,[5] suggesting that failure to forgive unconditionally may be life threatening.

We sought to investigate anger and TF and their potentially divergent relations with cardiovascular risk factors. We employed markers of cardiovascular functioning and tested the relation among these psychological constructs, sympathetic nervous system (SNS) activity, BP control, and noninvasive aortic hemodynamics. We tested the overall hypothesis that anger would predict markers of cardiotoxicity and that TF would be associated with cardioprotection. To this end, we carried out 3 related, yet distinct, studies to test the incremental and unique contribution of TF in comparison with anger in examining the functioning of BP, cardiac autonomic modulation, and aortic hemodynamics. Study 1 assessed autonomic modulation through beat-to-beat BP and power spectral analysis to examine the differential contribution of SNS and parasympathetic nervous system activation on baroreflex sensitivity (BRS) and HR modulation. Study 2 assessed aortic hemodynamics through applanation tonometry and pulse wave analysis (PWA) to allow the measurement of noninvasive surrogates of aortic hemodynamics. Study 3 assessed 24 hour ambulatory BP and ambulatory arterial stiffness.

Methods

A total of 308 healthy young women (M = 21.11 years ± SD = 2.52) participated in this research as approved by the University's intuitional review board. Subjects were allocated to one of the following studies: Study 1—cardiovascular autonomic modulation and baroreflex function; Study 2—aortic hemodynamics; and Study 3—24-hour ambulatory BP. To minimize potential cardiovascular risk confounders, participants were excluded from study participation through an online health screening assessment if they smoked, exercised

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See page 52 for disclosure information.

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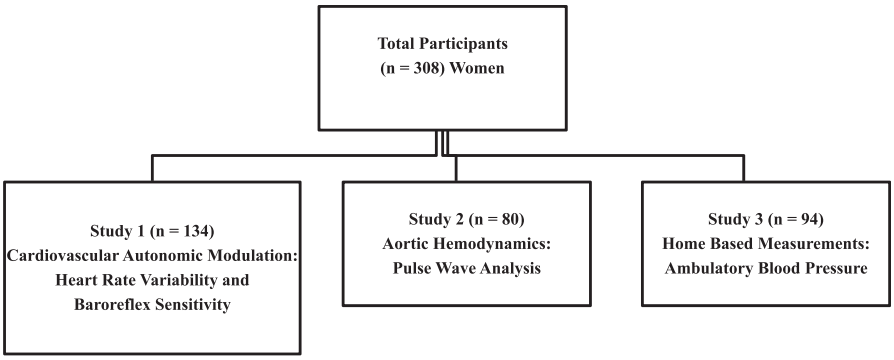

Figure 1. Participant allocation.

regularly as defined as >120 minutes per week in the previous 6 months, were hypertensive as defined as BP ≥140/90 mm Hg, had major chronic diseases, or were taking β blockers, antidepressants, or stimulants. Participants were asked to abstain from caffeine, alcohol, and strenuous physical activity for at least 24 hours before testing and were asked to not eat for 4 hours before testing. Participants were tested in the early follicular phase of the menstrual cycle to avoid potential variations in pressure wave morphology and cardiovascular functioning.

In Study 1, after laboratory familiarization, anthropometrics were measured. Participants then completed a health questionnaire that included a health history form and an anger and TF scale. All data collection was conducted in the afternoon in a quiet, dimly lit, temperature-controlled room (23 ± 1°C) at the same time of the day (±2 hours) to minimize potential diurnal variations in cardiovascular reactivity.[6] After instrument calibration and a 10-minute resting period in a seated position, beat-to-beat finger BP was recorded for 5 minutes.

In Study 2, participants were first introduced to the laboratory setting and familiarized with the study procedures. Body measurements (i.e., height and weight) were taken followed by the completion of a health questionnaire that included a health history form and an anger and TF scale. Data collection was conducted in the afternoon in a quiet, dimly lit, temperature-controlled room (23 ± 1°C) at the same time of the day (±2 hours). Participants were seated and given a 10-minute rest before any measurements were performed. Within 5 minutes after the rest period, measurements for peripheral brachial BP and applanation tonometry of the radial artery for central aortic hemodynamics were taken.

In Study 3, after completing an online health questionnaire, eligible participants were scheduled for a laboratory appointment to complete a 24-hour ambulatory BP assessment. Upon arrival, participants completed an anger scale, a TF scale, health characteristics (height and weight) were measured, and participants were fitted with an ambulatory BP device, which began from 08:00 to 11:00 hours and concluded when the recorder was returned to the laboratory the following day.

The trait subscale of the State-Trait Anger Expression Inventory-2 was used to measure trait anger.[7] Reliability for the sample was $\alpha = 0.87$. TF was measured using the 4-item Tendency to Forgive Scale.[8] Responses were summed into 1 overall score, with a possible range of 4 to 20. Reliability for the sample was $\alpha = 0.81$.

Beat-to-beat BP, HR, systolic BP, and diastolic BP were recorded through finger plethysmography (NIBP-100 Biopac Inc., Goleta, California). This method has been shown to provide accurate measurement of BP changes compared with intra-arterial BP.[9] Mean BP was calculated as systolic BP and diastolic BP, where (1/3) systolic BP + (2/3) diastolic BP = mean BP. The BP peaks were used to calculate the time duration of intervals among heartbeats (R wave to R wave interval, RRI) and were automatically detected using commercially available software (WinCPRS, Turku, Finland). The RRIs were inspected for artifacts, premature beats, and ectopic episodes to calculate heart rate variability (HRV) parameters. The main spectral components of the HRV that we calculated, by means of Fast Fourier transformation, were the low frequency (LF; 0.04 to 0.15 Hz) and the high frequency (HF; 0.15 to 0.4 Hz). The use of absolute units (ms^2) for HF and LF may be obtained in proportion to the total power, which is expressed in normalized units (nu). Normalization is used to exclude the influence of other fractal components.[10] Because there is structural algebraic redundancy inherent in the normalized spectral HRV measures with respect to each other (normalized low frequency [LFnu] = 1 − normalized high frequency [HFnu]) and also with respect to the LF/HF ratio, here we report LFnu to denote cardiac sympathovagal tone.[10,11]

Baroreflex functioning was evaluated through BRS calculated from the electrocardiogram and beat-by-beat BP files with the use of the cross-correlation method,[12] which is a time-domain sequential method for baroreflex function based on spontaneous systolic BP and R-R variability changes.

Indices of vascular function and aortic hemodynamics were obtained using brachial BP and applanation tonometry through PWA, which is defined as the examination of the functioning of the arterial (central) pulse wave, allowing for accurate assessment of central hemodynamic functioning.[13] Brachial BP was recorded using an automated oscillometric device (HEM-705CP; Omron Healthcare, Vernon Hill, Illinois). Brachial systolic and diastolic BP was used to calibrate radial waveforms obtained from a 10-second epoch using a high-fidelity tonometer (SPT-301B; Millar Instruments, Houston). PWA provides a more sensitive marker of cardiovascular function than brachial BP.[14,15] We

Table 1
Summarizes continuous variables, demographics statistics, and cardiovascular parameters for each study

Study	1 (n = 134)	2 (n = 80)	3 (n = 94)
Variable (M ± SD)			
Age (years)	21.28 ± 2.61	21.01 ± 2.44	21.02 ± 2.57
Height (m)	1.67 ± 0.08	1.65 ± 0.07	1.63 ± 0.08
Weight (kg)	68.72 ± 15.85	68.41 ± 9.95	64.00 ± 9.17
Body mass index (kg/m^2)	24.35 ± 4.26	25.10 ± 4.22	24.09 ± 4.17
State-trait anger	15.40 ± 1.59	15.90 ± 1.70	16.68 ± 1.05
Tendency to forgive	12.78 ± 1.52	14.60 ± 2.67	13.77 ± 2.03
Heart rate (bpm)	78.41 ± 9.95	74.53 ± 8.17	—
Mean blood pressure (mm Hg)	92.56 ± 7.28	83.82 ± 6.83	—
Normalized low frequency	0.65 ± 0.09	—	—
Baroflex sensitivity (ms/mm Hg)	16.81 ± 10.94	—	—
Brachial systolic blood pressure (mm Hg)	—	114.38 ± 7.58	—
Brachial diastolic blood pressure (mm Hg)	—	68.86 ± 7.51	—
Aortic systolic blood pressure (mm Hg)	—	97.43 ± 6.40	—
Aortic diastolic blood pressure (mm Hg)	—	69.78 ± 7.62	—
Aortic mean blood pressure (mm Hg)	—	82.29 ± 6.68	—
Systolic time interval (mm Hg/s.min^{-1})	—	1546.90 ± 215.47	—
Diastolic time interval (mm Hg/s.min^{-1})	—	3392.82 ± 288.47	—
Subendocardial viability index (%)	—	223.33 ± 33.45	—
Rate pressure product (bpm × mm Hg × 100)	—	59.93 ± 9.62	—
Ambulatory arterial stiffness index	—	—	0.28 ± 0.16
Ambulatory 24-hour (bpm)	—	—	78.66 ± 8.69
Ambulatory 24-mean BP (mm Hg)	—	—	85.42 ± 6.08
Mean BP_{dp} (%)	—	—	4.94 ± 7.37

measured brachial mean BP, aortic mean BP, systolic pressure time interval (STI; indicator of left ventricular work), diastolic pressure time interval (DTI; coronary perfusion), the ratio of DTI to STI expressed as a percentage (subendocardial viability index [SVI]; surrogate of myocardial perfusion and coronary flow reserve), and rate pressure product (RPP = systolic BP × HR; myocardial oxygen consumption).[16–18] All measurements were obtained in duplicate and averaged. Aortic BP waveforms were derived using a generalized transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). Only high-quality measurements (>80% operator index) were considered for analysis.

Ambulatory BP measurements were collected using validated oscillometric 90217A SpaceLabs (Spacelabs; Wokingham, Berkshire, UK) recorders and calibrated to take 4 measurements per hour for 24 hours. To calculate the ambulatory arterial stiffness index (AASI), the regression slope of ambulatory diastolic BP on ambulatory systolic BP from unedited 24-hour recordings, taken at a rate of 4 per hour, was computed for each participant. The 24-hour mean BP was calculated from the recordings. AASI was defined as 1 minus the regression slope. The stiffer the arterial tree, the closer the regression slope and AASI are to 0 and 1, respectively.[19] The BP dipping was defined as the degree of fall (%) in nocturnal mean arterial pressure relative to the diurnal mean BP: 100 × (1 − [nighttime mean BP ÷ daytime mean BP]).[20]

Pearson correlation coefficients evaluated univariate associations. Hierarchical multiple regression (HMR) analyses were conducted to test the relation between anger and TF with cardiovascular parameters and to demonstrate the incremental contribution of TF from anger in accounting for variance in cardiovascular parameters. A priori alpha level of $p < 0.05$ was considered to be significant. SPSS version 18.0 (SPSS Inc., Chicago, Illinois) was used for all analyses.

Results

Figure 1 shows how the participants were allocated across the 3 studies. Table 1 lists summary statistics for all continuous variables, including demographics, anger, TF, and cardiovascular parameters for Studies 1, 2, and 3.

In Study 1, 134 participants (M_{age} = 21.28 years, SD = 2.61) qualified for study inclusion. Pearson correlations indicated no statistically significant associations ($p < 0.05$) between anger and TF scores with demographic or anthropometric characteristics. HMR analyses were performed to examine the unique relation TF had with each of the beat-to-beat cardiovascular parameters while controlling for the influence of anger. HMR provided an analysis of the incremental contribution of TF scores above and beyond anger scores in accounting for variance in cardiovascular values. For each cardiovascular parameter serving as an outcome, Model 1 of the HMR contained anger as a predictor, whereas Model 2 added TF as a predictor. Model 2 of the HMR analyses (see Table 2) indicated that anger, while controlling for TF, had significant relations with all measured autonomic and cardiovascular outcomes with higher anger scores associated with higher HR, mean BP, LFnu, and lower BRS. Model 2 of the HMR also indicated a significant relation between TF scores and LFnu (but not for HR, mean BP, or BRS) while controlling for anger. The addition of TF in Model 2 of the HMR analyses indicated that TF was able to uniquely predict 7% of the variance in LFnu values; higher TF scores were associated with lower LFnu.

In Study 2, 80 participants (M_{age} = 21.01 years, SD = 2.44) qualified for study inclusion. Inclusion criteria were identical to Study 1. Pearson correlations indicated no statistically significant associations ($p < 0.05$) between anger and TF scores with demographic or anthropometric characteristics. Model 1 of the HMR contained anger as a predictor, whereas Model 2 added TF as a predictor. Model 2 of the HMR analyses (see Table 3) indicated that anger, while controlling for TF, had significant relations with both brachial and aortic hemodynamic pressures, STI, DTI, and RPP, with higher anger scores associated with higher

Table 2
Study 1 (n = 134): hierarchical multiple regression analyses of anger and forgiveness scores on beat-to-beat cardiovascular indices

Variable	Model	Predictors	β	sr	p	Model R^2	Model ΔR^2	Model F
HR (bpm)	1	Anger	0.31	0.31	<0.001	0.10		F(1, 132) = 13.36, p <0.001
	2	Anger	0.33	0.32	<0.001	0.11	0.01	ΔF(1, 131) = 1.53, p = 0.219
		Forgive	−0.11	−0.10	0.219			
Mean BP (mm Hg)	1	Anger	0.23	0.23	0.007	0.05		F(1, 132) = 7.43, p = 0.007
	2	Anger	0.22	0.21	0.015	0.06	0.00	ΔF(1, 131) = 0.54, p = 0.464
		Forgive	−0.06	−0.06	0.464			
LFnu	1	Anger	0.58	0.58	<0.001	0.34		F(1, 132) = 33.89, p <0.001
	2	Anger	0.56	0.58	<0.001	0.41	0.07	ΔF(1, 131) = 22.12, p <0.001
		Forgive	−0.26	−0.31	0.009			
BRS (ms/mm Hg)	1	Anger	−0.18	−0.18	0.041	0.03		F(1, 132) = 4.26, p = 0.041
	2	Anger	−0.18	−0.18	0.045	0.03	0.00	ΔF(1, 131) = 0.01, p = 0.917
		Forgive	0.01	−0.01	0.917			

BP = blood pressure; BRS = baroreflex sensitivity; HR = heart rate; LFnu = normalized low frequency.

Table 3
Study 2 (n = 80): hierarchical multiple regression analyses of anger, forgiveness, and aortic hemodynamic indices

Variable	Model	Predictors	β	sr	p	Model R^2	Model ΔR^2	Model F
HR (bpm)	1	Anger	0.148	0.148	0.229	0.022		F(1, 78) = 1.47, p = 0.229
	2	Anger	0.173	0.173	0.142	0.122	0.100	ΔF(1, 77) = 7.41, p = 0.008
		Forgive	−0.317	−0.316	0.008			
BSBP (mm Hg)	1	Anger	0.488	0.488	<0.001	0.238		F(1, 78) = 20.63, p <0.001
	2	Anger	0.503	0.501	<0.001	0.271	0.033	ΔF(1, 77) = 2.94, p = 0.091
		Forgive	−0.182	−0.182	0.091			
BDBP (mm Hg)	1	Anger	0.693	0.693	<0.001	0.480		F(1, 78) = 61.04, p <0.001
	2	Anger	0.704	0.701	<0.001	0.497	0.017	ΔF(1, 77) = 2.19, p = 0.144
		Forgive	−0.131	−0.130	0.144			
BMAP (mm Hg)	1	Anger	0.690	0.690	<0.001	0.476		F(1, 78) = 59.87, p <0.001
	2	Anger	0.701	0.702	<0.001	0.497	0.021	ΔF(1, 77) = 2.72, p = 0.104
		Forgive	−0.145	−0.200	0.104			
ASBP (mm Hg)	1	Anger	0.613	0.613	<0.001	0.376		F(1, 78) = 39.76, p <0.001
	2	Anger	0.620	0.618	<0.001	0.384	0.008	ΔF(1, 77) = 0.80, p = 0.373
		Forgive	−0.088	−0.087	0.373			
ADBP (mm Hg)	1	Anger	3.18	0.694	<0.001	0.482		F(1, 78) = 61.37, p <0.001
	2	Anger	3.23	0.702	<0.001	0.497	0.015	ΔF(1, 77) = 1.91, p = 0.171
		Forgive	−0.635	−0.122	0.171			
AMAP (mm Hg)	1	Anger	0.690	0.690	<0.001	0.476		F(1, 78) = 59.87, p <0.001
	2	Anger	0.701	0.699	<0.001	0.497	0.021	ΔF(1, 77) = 2.72, p = 0.104
		Forgive	−0.145	−0.145	0.104			
STI (mm Hg/s.min^{-1})	1	Anger	0.513	0.513	<0.001	0.263		F(1, 78) = 23.56, p <0.001
	2	Anger	0.543	0.542	<0.001	0.405	0.142	ΔF(1, 77) = 15.54, p <0.001
		Forgive	−0.378	−0.377	<0.001			
DTI (mm Hg/s.min^{-1})	1	Anger	0.575	0.575	<0.001	0.330		F(1, 78) = 32.56, p <0.001
	2	Anger	0.569	0.567	<0.001	0.335	0.005	ΔF(1, 77) = 0.497, p = 0.483
		Forgive	0.072	0.071	0.483			
SVI (%)	1	Anger	−0.155	−0.155	0.206	0.024		F(1, 78) = 1.63, p = 0.206
	2	Anger	−0.184	−0.183	0.114	0.150	0.126	ΔF(1, 77) = 9.64, p = 0.003
		Forgive	0.356	0.355	0.003			
RPP (bpm × mm Hg)	1	Anger	0.379	0.379	0.001	0.144		F(1, 78) = 11.10, p = 0.001
	2	Anger	0.405	0.403	<0.001	0.242	0.098	ΔF(1, 77) = 8.42, p = 0.005
		Forgive	−0.314	−0.313	0.005			

ADBP = aortic diastolic blood pressure; AMAP = aortic mean arterial pressure; ASBP = aortic systolic blood pressure; BDBP = brachial diastolic blood pressure; BMAP = brachial mean arterial pressure; BSBP = brachial systolic blood pressure; DTI = diastolic time interval; RPP = rate pressure product; sr = semipartial correlation; STI = systolic time interval; SVI = subendocardial viability index.

pressures, STI, DTI, and RPP. Model 2 of the HMR also indicated significant relation (while controlling for anger) between TF scores and HR, STI, SVI, and RPP, with higher TF scores associated with less HR, STI, and RPP but greater SVI. The addition of TF in the Model 2 of the HMR analyses indicated that TF was able to uniquely predict 10% of the variance in HR values, 14.2% of STI values, 12.6% in SVI values, and 9.8% in RPP values.

Table 4
Study 3 (n = 94): hierarchical multiple regression analyses of ambulatory hemodynamic indices

Variable	Model	Predictors	β	sr	p	Model R^2	Model ΔR^2	Model F
24-HR (bpm)	1	Anger	0.12	0.12	0.250	0.01		$F(1, 92) = 1.34$, $p = 0.250$
	2	Anger	0.10	0.10	0.325	0.02	0.01	$\Delta F(1, 91) = 0.86$, $p = 0.357$
		Forgive	−0.10	−0.10	0.357			
24-mean BP (mm Hg)	1	Anger	0.40	0.40	<0.001	0.16		$F(1, 92) = 16.97$, $p < 0.001$
	2	Anger	0.41	0.41	<0.001	0.16	0.01	$\Delta F(1, 91) = 0.94$, $p = 0.335$
		Forgive	−0.09	−0.09	0.335			
MAP_{dp} (%)	1	Anger	−0.39	−0.39	<0.001	0.15		$F(1, 92) = 12.37$, $p < 0.001$
	2	Anger	−0.39	−0.39	<0.001	0.28	0.13	$\Delta F(1, 91) = 11.98$, $p < 0.001$
		Forgive	0.36	0.36	<0.001			
AASI	1	Anger	0.23	0.23	0.032	0.06		$F(1, 92) = 4.76$, $p = 0.041$
	2	Anger	0.24	0.24	0.030	0.06	0.00	$\Delta F(1, 11) = 0.18$, $p = 0.667$
		Forgive	−0.05	−0.05	0.669			

24-HR = 24-hour heart rate; 24-mean BP = 24-hour mean BP; AASI = ambulatory arterial stiffness index; MAP_{dp} = mean arterial pressure dipping percentage.

In Study 3, 94 young participants (M_{age} = 21.02 years, SD = 2.57) qualified for study inclusion. Pearson correlations indicated no statistically significant associations ($p < 0.05$) between anger and TF scores with the demographic or anthropometric characteristics. Model 1 of the HMR contained anger and Model 2 added TF as a predictor. After controlling for anger, the addition of TF in the Model 2 of the HMR analyses (see Table 4) indicated that TF was able to uniquely predict 13% of the variance in 24-hour mean BP dipping scores with higher TF scores associated with greater mean BP dipping. Model 2 of the HMR also indicated that anger remained a significant predictor for 24-hour mean BP, mean BP dipping (mean BP_{dp}), and AASI after controlling for TF scores with higher anger scores associated with higher 24 hours mean BP, higher AASI, and less mean BP dipping.

Discussion

Three studies were conducted to evaluate cardiovascular functioning underlying anger and TF to determine whether TF plays a unique role as a potential protective factor against the development of impaired cardiovascular functioning. The novel findings of the present studies are (1) anger was associated with indices typical of increased hemodynamic and SNS activity, but TF was negatively related to nLF while controlling for anger, (2) the effects of TF are unique from anger and are modulated through decreased ventricular work (STI) and ultimately decreased myocardial oxygen consumption (RPP), and (3) anger remained a significant predictor for 24-hour mean BP, mean BP_{dp}, and AASI after controlling for TF scores, such that higher anger scores predicted higher mean BP, higher AASI, and less mean BP dipping. These results suggest that anger and TF have divergent effects on cardiovascular risk factors. To the best of our knowledge, this is the first research to systemically address the impact of TF and anger on cardioprotection and cardiotoxicity, respectively.

Study 1 demonstrated that TF was associated with decreased sympathovagal tone supporting theoretical models proposed by Thoresen et al[21] and Seybold et al[22] to explain the relation among TF, autonomic nervous system functioning, and health. Conversely anger was associated with cardiovascular autonomic deregulation including decreased BRS. It is worth noting that distention aortic pressure (aortic mean BP) and AASI have been linked to increased arterial stiffness and therefore may explain the anger-induced decreases in BRS.[23] Study 2 assessed aortic hemodynamics and demonstrated decreased ventricular work and decreased myocardial oxygen consumption corresponded to an increase in forgiveness. This constitutes the first empirical investigation into the cardiac mechanisms potentially responsible for the positive cardiovascular health associations with TF. Additionally, anger was associated with increased ventricular work and aortic BP, suggesting that previous studies may have underestimated the impact of this psychological risk factor on cardiovascular function. Finally, Study 3 examined the relation among anger, TF, and cardiovascular functioning over a 24-hour period through ambulatory BP monitoring and found that TF may serve as a protective factor against future cardiovascular disease owing to increased BP dipping. Strikingly, the cardioprotective effect sizes demonstrated by forgiveness in this research are similar to the effect sizes of known β blockers.[24,25]

The novel nature of this research advances the understanding of the physiology underlying both anger and TF with analyses demonstrating that anger and TF are unique, independent predictors of autonomic and cardiovascular parameters. This novelty extends not only to the assessment techniques utilized (i.e., beat-to-beat BP, PWA, ambulatory) but more importantly to the discovery of new mechanisms that might account for the association between TF and health (i.e., cardiac autonomic modulation, ventricular work, myocardial oxygen consumption, nocturnal hemodynamics, and BP dipping). As we view this research as an initial step into the search for physiological mechanisms associated with TF, future studies should emerge that examine stress hormones or blood catecholamine levels as additional mechanisms as they have been linked to SNS activity.[26] At a methodological level, future research utilizing longitudinal designs or manipulations inducing cardiovascular reactivity may replicate and greatly expand our findings. It should be noted, however, that designs examining the role that stress or anger plays in mediating the relation between TF and cardiovascular health suggest that TF alone can uniquely

account for decreased hemodynamic values.[27] Additionally, as there appear to be gender differences in TF[28] it is important for future research to examine the relation between TF and male physiology as the present research is limited to female physiology. Furthermore, as this sample is restricted to young adults devoid of cardiovascular illness and in seemingly good health, the protective effect of TF may have been blunted or restricted.

Disclosures

The authors have no conflicts of interest to disclose.

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