Potential Side Effects of Unhealthy Lifestyle Choices and Health Risks on Basal and Reactive Heart Rate Variability in College Drinkers

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ABSTRACT. Objective: Emerging adults often begin making independent lifestyle choices during college, yet the association of these choices with fundamental indicators of health and adaptability is unclear. The present study examined the relationship between health risks and neurocardiac function in college drinkers. **Method:** Heart rate variability (HRV) was assessed at baseline and in reaction to a paced breathing challenge in 212 college drinkers (53.8% women). Basal HRV served as a general indicator of health. Reactive HRV (during paced breathing) was used as a marker of an individual's adaptability to challenge. The relationship of HRV to alcohol use, cigarette use, exercise, sleep, and body mass index (BMI) was assessed. **Results:** Greater alcohol use and less exercise were associated with lower basal HRV. BMI was unrelated to basal HRV but was negatively associated with reactive HRV during the

breathing challenge. **Conclusions:** High levels of alcohol use and lack of exercise are negative correlates of cardiovascular and general health, even in apparently healthy college drinkers. The negative relationship between BMI and reactive HRV suggests that overweight individuals have reduced ability to psychophysiologicaly adapt to challenges; understanding the temporal course of this relationship is needed. This study highlights the importance of examining HRV at baseline and in response to a challenge to capture the active neurocardiac processes that contribute to health and adaptive responding. The suppressive effects of health risks on HRV are modifiable; thus, HRV may be useful in evaluating the health benefits of lifestyle change and in promoting change behaviors in college drinkers. (*J. Stud. Alcohol Drugs*, 74, 787–796, 2013)

HEAVY DRINKING IS ONE OF THE MOST highly prevalent health risk behaviors in college students (O'Malley and Johnston, 2002). College students who engage in frequent heavy drinking also often report other health risks, such as smoking cigarettes, carrying excess weight, getting inadequate sleep, and showing reduced engagement in physical activity (Fromme et al., 2008; Laska et al., 2009). Although many college drinkers mature out of hazardous alcohol use behaviors (Dawson et al., 2004; Kerr et al., 2009), these individuals may nonetheless establish unhealthy lifestyle habits during college that resonate across the lifespan (Nelson et al., 2008). Considerable evidence supports the notion that the maintenance of negative health factors places individuals at risk for developing serious, yet preventable, health problems. However, little is known about the mechanisms that support this relationship. In fact, little research has examined the association of unhealthy lifestyles during college with the current health and well-being of

young, otherwise healthy college students. One exception is a study reporting that heavy alcohol consumption, smoking, lack of cardiovascular exercise, and eating a high saturated-fat diet statistically predicted undesirable cholesterol levels and blood pressure during college (Spencer, 2002).

The present study focused on understanding the relationship between health risks and heart rate variability (HRV) in college drinkers. HRV, the variability in the time interval between heartbeats, supports physical and mental health (Cohen and Taylor, 2002) and can provide a snapshot of how current lifestyle factors affect current health and adaptive capacity. HRV can be measured at rest or during a low-demand task (i.e., basal HRV) to serve as a sensitive index of cardiovascular function and provide a gauge of general health state (Abboud, 2010; Goldstein, 2001). In adults, low basal HRV has been linked to poor cardiovascular health (Schmidt et al., 2005; Stein et al., 2006) and a variety of physical and psychological disorders, such as asthma (Kazuma et al., 1997), diabetes (Vinik et al., 2003), major depression (Licht et al., 2008; Nahshoni et al., 2004), anxiety disorders (Cohen and Benjamin, 2006; Friedman and Thayer, 1998), and posttraumatic stress disorder (Blechert et al., 2007). HRV also has been negatively associated with unhealthy lifestyle choices and health risks, such as chronic heavy alcohol use (Ingjaldsson et al., 2003; Thayer et al., 2006), heavy cigarette use (Barutcu et al., 2005; Cagirci et al., 2009), sleep deprivation (Spiegel et al., 2004; Stein and Pu, 2012), physical

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inactivity (Hautala et al., 2003; Iwasaki et al., 2003; Okazaki et al., 2005; Sloan et al., 2009), and greater body mass index (BMI; Molino et al., 2009; Quilliot et al., 2008).

Change in HRV from the basal state can also be measured during high-demand challenges (i.e., reactive HRV) to serve as a dynamic barometer of how the heart and brain are communicating in the moment (Bates et al., 2011; Benarroch, 1997, 2008) and to measure an individual's ability to flexibly and adaptively respond to an acute stressor (reviewed in Appelhans and Luecken, 2006; Giardino et al., 2000; Lehrer et al., 2000). Recently, reactive HRV has been linked to attention-deficit/hyperactivity disorder (Negrao et al., 2011), antisocial behavior (De Vries-Bouw et al., 2011), depression and anxiety (Shinba et al., 2008; Tucker et al., 2012), acute alcohol intoxication (Vaschillo et al., 2008), and response to emotional and alcohol-related visual cues (Rajan et al., 1998; Udo et al., 2009; Vaschillo et al., 2008).

The present study examined the extent to which lifestyle choices and health risks are associated with basal and reactive HRV in college students who drink alcohol. Through the identification of lifestyle choices and related health risks that are linked to HRV, the present study aimed to provide an initial empirical basis for a mechanistic model of how neurocardiac processes may participate in the maintenance of overall health. Based on the results of studies of adults, it was hypothesized that basal HRV would be negatively associated with alcohol involvement, BMI, and cigarette use but positively associated with exercise intensity and hours of sleep. To assess reactive HRV, we further measured HRV in response to a slow-paced breathing challenge, during which participants completed six breaths per minute (compared with a normal breathing rate of 12–20 breaths per minute). No one has yet evaluated how a combination of lifestyle factors is associated with reactive HRV. Examining the relationship between reactive HRV during the paced breathing challenge and lifestyle choices and health risk thus was exploratory, but we expected reactive HRV to show negative associations with risky health behaviors and positive associations with protective health behaviors. This preliminary hypothesis was based on the idea that reactive HRV during the paced breathing challenge can provide insight into the adaptive capacity of an individual, which is likely to be reduced by drinking, smoking, weight gain, and/or a lack of exercise.

Method

Participants

The sample comprised 212 emerging adults (53.8% women; $M_{\text{age}} = 21.3$ years, $SD = 1.2$) who reported using alcohol during the past year and participated in one of two larger studies that examined individual differences in physiological reactivity to emotional and alcohol-related stimuli. Both

parent studies were performed during the same timeframe in our laboratory. A total of 176 participants were recruited as college drinkers through university and community bulletin boards and newspaper advertisements. This parent study assessed factors that affect physiological reactivity to, and memory for, emotionally arousing and appetitive visual cues. It included the administration of a beverage (alcohol, placebo, or mixed juice control) after completion of the baseline assessment. Because of the alcohol beverage administration, students were excluded from the parent study if they did not consume at least four drinks (three drinks for women) at least twice per month in the past year; were more than 20% over- or under-weight from the ideal for gender, height, and body frame based on the Metropolitan Life Height–Weight Table (Metropolitan Life Insurance Company, 1983); were regular current users of psychoactive drugs; or were pregnant based on a urinalysis.

In addition, 16 college students who had previously been mandated to a brief intervention for violating university policies about on-campus alcohol and other drug use, and 20 college student athletes were included from a parallel study on physiological reactivity in high-risk samples. Mandated students and student athletes are at heightened risk for alcohol-related problems (Barnett and Read, 2005; Yusko et al., 2008). The mandated sample has been previously described (Buckman et al., 2010). The athlete sample comprised collegiate golfers and swimmers who volunteered after a team meeting information session in which the study was described. No beverage was administered in this parent study.

Exclusion criteria for both parent studies included a past-year history of any psychiatric diagnosis, a lifetime history of a psychotic disorder or substance dependence, current psychoactive medication use, any medical condition that might interfere with physiological recording, or any abnormal cardiovascular or respiratory function detected in the laboratory. Of the participants, 65.5% were non-Hispanic White, 18.2% were Asian, and 8.6% were non-Hispanic African American; the remaining participants (7.7%) identified themselves as “mixed or other.” They averaged 15 years of education ($SD = 1.5$), and 83.6% reported a family income of more than \$41,000.

Procedures

All studies were approved by the university Institutional Review Board for the Protection of Human Subjects Involved in Research. All participants provided written informed consent and were reimbursed for their time. Each laboratory session lasted approximately 3–4 hours and included completing questionnaires, being exposed to stimulus cues, and completing memory tasks (data not presented). Participants individually completed the laboratory session between 9:30 A.M. and 3:30 P.M. to minimize biological cir-

cadian variations in psychophysiology. They were asked to refrain from alcohol or other drug use (except caffeine and cigarettes) for the 24 hours before the laboratory session. A blood alcohol concentration of zero was confirmed through breath analysis.

Baseline assessment. The participant performed a standardized low cognitive demand “plain vanilla task” (Jennings et al., 1992) for 5 minutes. During this task, the participant sequentially viewed colored rectangles at the rate of one rectangle per 10 seconds on a computer screen and silently counted the number of blue rectangles. Compared with a “resting” baseline, the use of a standardized, minimally demanding cognitive task was shown to produce a baseline HRV value with better between- and within-subject stability and generalizability across sessions (Jennings et al., 1992).

Paced breathing. A visual breathing pacer (E-Z Air, Thought Technology Ltd., Plattsburgh, NY) was presented on the screen, and participants were instructed to inhale when the pacer went up and to exhale when the pacer went down during a 5-minute period. The pacer was set at a rate of one complete inhalation/exhalation breathing cycle every 10 seconds (i.e., 0.1-Hz frequency, six breaths per minute). To avoid hyperventilation, participants were asked to breathe slowly, but not too deeply, as demonstrated during a brief practice of the task.

Measures

Heart rate variability and respiration. The electrocardiogram (ECG) record and respiration frequency were collected at a rate of 1,000 samples per second by a Powerlab Acquisition system (ADInstruments, Colorado Springs, CO). Recorded data were exported to a WinCPRS software program (Absolute Aliens Oy, Turku, Finland) for analyses. The program measured beat-to-beat heart intervals as periods between R-to-R waves of the ECG (RRI) and segmented the succession of RRIs into 5-minute blocks. RRI spectra were calculated through Fourier analysis (Cooke et al., 1999; Taylor et al., 1998). Cubic interpolation of the non-equidistant waveform of the RRI sequence was completed after artifacts and irregular beats were edited. The following three HRV indices were analyzed: standard deviation of normal-to-normal intervals (SDNN), the power of the RRI spectrum in the high-frequency range (HF HRV; 0.15–0.4 Hz), and the power of the RRI spectrum in the low-frequency range (LF HRV; 0.05–0.15 Hz). SDNN include all physiologically relevant heart rate oscillatory frequency components and reflect the general activity of cardiac autonomic regulation (Task Force of the European Society of Cardiology and the American Society of Pacing and Electrophysiology, 1996). HF HRV reflects cardiac activity controlled by the vagus nerve, as well as phase variation in vagal effects on the heart associated with respiration (Berntson et al., 1997; Task Force, 1996). LF HRV strongly

correlates with cardiac baroreflex activity (Berntson et al., 1997), a mechanism that contributes to the integration of the autonomic nervous system and central nervous system processes (Hempel et al., 2007). Because respiration influences heart rate (Berntson et al., 1997), to account for its effects, mean respiration frequency (Hz) and mean respiration tidal volume were calculated for each task.

Lifestyle factors. Participants’ sociodemographic information, alcohol and other drug use, and exercise habits were assessed with self-report questionnaires. An updated version of the Rutgers Health and Human Development Project Alcohol and Drug Use Questionnaire (Pandina et al., 1984) was used to assess quantity and frequency of alcohol use and frequency of cigarette use in the past 30 days. An alcohol quantity–frequency index (QFI) was calculated by multiplying quantity and frequency of alcohol use in the past 30 days.

In line with Berg and colleagues’ (2010) definition of smokers (≥ 25 of past 30 days), participants were defined as regular cigarette users if they selected response choices of cigarette use about four to six times per week, every day, or more than once a day during the past 30 days (1 = yes; 0 = no). To measure exercise activity, we asked, “In the past year, how often have you usually exercised or participated in sports for at least a total of 30 minutes a day or more (e.g., basketball, running, jogging, swimming laps, tennis, bicycling, or similar aerobic activities)?” and “How long is each exercise session on average?” An exercise index was calculated by multiplying the average frequency of exercise per week in the past year by the average duration of each exercise session in minutes. Participants also reported the average number of hours that they slept each night in the past month. Weight and height were measured at the laboratory. BMI was calculated using the following formula: weight (kg) / height (m²) (Garrow and Webster, 1985).

Depression and anxiety symptoms. Depression and anxiety symptoms were measured as covariates because of their potential associations with HRV (e.g., Cohen et al., 2000; Friedman and Thayer, 1998; Galetta et al., 2012; Hughes and Stoney, 2000; Koschke et al., 2009; Nahshoni et al., 2004), using the Beck Depression Inventory–II (BDI-II; Beck et al., 1996) and the Beck Anxiety Inventory (BAI; Beck and Steer, 1993), respectively.

Analysis

Multiple linear regression analysis was conducted separately for each HRV index assessed during baseline and paced breathing tasks to examine whether the following measures were associated with resting and reactive HRV: BMI, exercise pattern, the average number of hours of sleep in the past 30 days, and alcohol and cigarette use in the past 30 days. To correct skewness and kurtosis, a natural logarithm transformation was applied to the alcohol QFI and the

TABLE 1. Participants' lifestyle choices and health risks

Lifestyle factors	%	<i>M (SD)</i>	Min.	Max.
BMI, kg/m ²		24.0 (3.5)	16.1	36.6
% overweight/obese individuals ^a	35.0			
Alcohol use in the past 30 days				
Quantity, no. of drinks per occasion ^b		4.8 (2.2)	0.01	13.0
Frequency, no. of occasions per week		1.6 (1.1)	0.01	5.5
% regular heavy episodic drinking ^c	62.3			
Cigarette use				
Any use in the past 30 days, %	31.9			
≥4 days a week, %	12.7			
Exercise pattern				
Frequency in the past year, days per week		2.8 (1.9)	0	7.0
Average duration, minutes		56.1 (24.1)	10.0	150.0
Sleep pattern				
Average no. of hours of sleep, last year		7.1 (1.0)	4.0	10.0
% reporting trouble sleeping	18.9			
BDI-II ^d		4.0 (4.5)	0	26
BAI ^e		3.4 (4.0)	0	18

Notes: Min. = minimum; max. = maximum; BMI = body mass index; BDI-II = Beck Depression Inventory–II (Beck et al., 1996); BAI = Beck Anxiety Inventory (Beck and Steer, 1993); no. = number. ^aBMI ≥ 25 kg/m²; note that this index is different from the weight classification used in the Metropolitan Life Height–Weight Table (1983). ^bAll participants in this study reported drinking in the past year; however, five individuals did not report any drinking in the past 30 days. ^cHeavy episodic drinking was defined as five or more standard drinks per occasion for men and four or more standard drinks per occasion for women (Wechsler et al., 1995); standard drinks were defined as 12 oz. of regular (5%) beer or malt beverage, 1.5 oz. of 80 proof (~40%) distilled spirits, and 5 oz. of regular (12%–17%) wine or champagne. ^dBDI-II score range: 0–63; 0–13 = minimal depression, 14–19 = mild depression, 20–28 = moderate depression, and 29–63 = severe depression. ^eBAI score range: 0–63; 0–7 = minimal anxiety, 8–15 = mild anxiety, 16–25 = moderate anxiety, and 25–63 = severe anxiety.

exercise index, as well as to all HRV measures. BDI-II and BAI scores, age, race, income, and gender were included as covariates to account for their potential relationships to health risks and HRV indices. For the analysis of basal HRV, mean respiration frequency and mean respiration tidal volume were included as covariates to adjust for the effects of respiration on HRV. For the analysis of reactive HRV, regression analyses were conducted while controlling for the respective baseline HRV index in each model. Moreover, in these latter models, the mean tidal volume was included in the analysis, but respiration frequency was not because frequency was controlled by the experimental protocol (i.e., there was no variability in the instructed breathing frequency of six breaths per minute). HF HRV during the paced breath-

ing task was not analyzed because breathing in the low frequency range (six breaths per minute) may increase HF HRV, but not because of an increase in vagal traffic (Song and Lehrer, 2003). Thus, a total of five regression models were conducted.

For any participant who had data only from an alcohol administration session as a part of the parent study ($n = 25$), baseline HRV data (collected before alcohol consumption) but not reactive HRV data (collected after alcohol consumption) were used because acute alcohol consumption influences HRV (Vaschillo et al., 2008). No significant differences in basal HRV were found between any of the beverage groups, nor were there differences in reactive HRV between the placebo and control groups (all $ps > .05$).

TABLE 2. Zero-order correlations

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
1. Basal SDNN	—									
2. Basal LF HRV	.85**	—								
3. Basal HF HRV	.87**	.69**	—							
4. 6 BPM SDNN	.58**	.46**	.49**	—						
5. 6 BPM LF HRV	.48**	.35**	.40**	.96**	—					
6. BMI	.07	.12	.02	-.12	-.17*	—				
7. Alcohol QFI	-.08	-.02	-.17*	-.01	-.01	.12	—			
8. Cigarette use	-.01	.00	-.01	.08	.07	.03	.22**	—		
9. Exercise pattern	.25**	.19**	.18**	.18**	.14*	.19**	-.04	-.21**	—	
10. Hours of sleep	.05	.06	.02	.08	.09	-.09	.07	.02	.01	—

Notes: SDNN = standard deviation of normal-to-normal intervals; LF HRV = low-frequency heart rate variability; HF HRV = high-frequency heart rate variability; 6 BPM = breathing paced at 6 breaths per minute; BMI = body mass index; alcohol QFI = alcohol quantity–frequency index (log-transformed). Cigarette use in the past 30 days was defined as use at least four times per week for the past 30 days (1 = yes; 0 = no). All HRV indices are log-transformed. * $p < .05$; ** $p < .01$.

TABLE 3. Multiple linear regression analysis predicting heart rate variability (HRV) indices during baseline from cardiovascular disease risk factors

Variables	<i>B</i> (<i>SE</i>)	β	UV %	<i>t</i>	<i>F</i> (13, 197)	<i>p</i>	<i>R</i> ²
SDNN					2.89	<.01	.16
BMI	0.00 (0.01)	.03	0.0	0.36			
Alcohol QFI, log	-0.07 (0.04)	-.12	1.2	-1.64			
Regular cigarette use	0.04 (0.09)	.03	0.1	0.49			
Exercise index, log	0.06 (0.02)	.19	2.8	2.55*			
No. of hours of sleep	0.01 (0.03)	.02	0.0	0.23			
LF HRV					3.80	<.01	.20
BMI	0.02 (0.02)	.06	0.4	0.94			
Alcohol QFI, log	-0.17 (0.09)	-.13	1.3	-1.78			
Regular cigarette use	0.10 (0.21)	.05	0.1	0.50			
Exercise index, log	0.06 (0.06)	.08	0.5	1.09			
No. of hours of sleep	0.03 (0.07)	.03	0.0	0.49			
HF HRV					2.41	<.01	.14
BMI	0.00 (0.02)	.01	0.0	0.11			
Alcohol QFI, log	-0.23 (0.10)	-.18	2.2	-2.21*			
Regular cigarette use	0.13 (0.23)	.04	0.2	0.59			
Exercise index, log	0.12 (0.06)	.14	1.6	1.92			
No. of hours of sleep	-0.01 (0.08)	-.01	0.0	-0.12			

Notes: *B* = unstandardized; β = standardized; UV = unique variance; SDNN = standard deviation of normal-to-normal intervals; BMI = body mass index; alcohol QFI = alcohol quantity–frequency index (log-transformed); no. = number; LF HRV = low-frequency (0.05–0.15 Hz) heart rate variability; HF HRV = high-frequency (0.15–0.4 Hz) heart rate variability. Regular cigarette use was defined as reporting cigarette use at least four times per week for the past 30 days (1 = yes, 0 = no). All models included the following covariates: age, gender (women = 1, men = 0), race (White = 1, non-White = 0), mean respiration frequency at baseline, and mean tidal volume at baseline. Mean respiration frequency was a significant covariate for all models (for SDNN: *B* [*SE*] = -1.48 [0.63], β = -.17, UV = 3.0%, *t* = -2.36; for LF HRV: *B* [*SE*] = -4.01 [1.49], β = -.19, UV = 2.9%, *t* = -2.70; for HF HRV: *B* [*SE*] = -4.19 [1.63], β = -.19, UV = 2.4%, *t* = -2.57). Gender (*B* [*SE*] = -0.47 [0.15], β = -.24, UV = 4.2%, *t* = -3.20) and race (*B* [*SE*] = 0.28 [0.14], β = -.13, UV = 1.6%, *t* = 1.98) were also significant covariates for LF HRV. Alcohol, cigarette, and marijuana use were assessed for the past 30 days.

**p* < .05.

Results

Table 1 shows participant characteristics, including BMI, lifestyle choices, and number of depression and anxiety symptoms. Table 2 shows zero-order correlations between study variables; HRV indices were significantly correlated with each other. Exercise was positively associated with all HRV indices at baseline and during the paced breathing task. Greater BMI was correlated with lower LF HRV during the paced breathing task (absolute level, not reactivity score). Cigarette use was negatively correlated with exercise levels. Greater alcohol use was negatively correlated with HF HRV at baseline and positively correlated with more cigarette use in the past 30 days.

The results of the basal HRV regression analyses showed that, adjusting for sociodemographic factors and depression and anxiety symptoms, alcohol use was significantly negatively associated with HF HRV at baseline (Table 3). Exercise was significantly positively associated with SDNN. BMI, cigarette use, and hours of sleep were not significantly associated with HRV indices at baseline. The tolerance index (Schroeder, 1990) indicated that multicollinearity among the regressors (covariates) was equivalently low in all models (tolerance index: BMI = 0.86, alcohol use QFI [log] = 0.77, cigarette use = 0.85, exercise index [log] = 0.80, and hours of sleep = 0.86).

Results of the reactive HRV regression analyses showed that, adjusting for baseline HRV indices and sociodemographic factors, BMI was significantly related to SDNN and LF HRV (Table 4). There was equivalent low multicollinearity among the regressors in both models (tolerance index: BMI = 0.85, alcohol use QFI [log] = 0.75, cigarette use = 0.85, exercise index [log] = 0.79, and hours of sleep = 0.89).

Discussion

Parents and caretakers shape much of the environment during childhood and adolescence; but for many individuals, health-related lifestyle factors change considerably during emerging adulthood (i.e., ages 18–25 years; Arnett, 2000). These changes can serve as the foundation for health-related behaviors that are maintained throughout life (Nelson et al., 2008). As such, college is a unique developmental stage in which many emerging adults begin to make their own lifestyle choices. An important yet unanswered question is whether unhealthy choices, such as heavy episodic drinking and inadequate physical exercise, during the college years exert only temporary, circumscribed negative consequences, or whether they have more lasting psychophysiological ramifications.

This study provided a first step in addressing this question by examining the cross-sectional relations between HRV and multiple health risks often found in otherwise healthy

TABLE 4. Multiple linear regression analysis predicting heart rate variability (HRV) indices during the paced breathing task from cardiovascular disease risk factors

Variables	<i>B</i> (<i>SE</i>)	β	UV (%)	<i>t</i>	<i>F</i> (13, 171)	<i>p</i>	<i>R</i> ²
SDNN					13.01	<.01	.50
BMI	-0.02 (0.01)	-.17	2.6	-2.95*			
Alcohol QFI, log	-0.03 (0.03)	-.07	0.4	-1.10			
Regular cigarette use	0.12 (0.06)	.11	1.1	1.90			
Exercise index, log	0.01 (0.02)	.02	0.0	0.33			
No. of hours of sleep	-0.03 (0.02)	-.08	0.6	-1.37			
LF HRV					5.02	<.01	.28
BMI	-0.05 (0.02)	-.22	3.9	-3.01*			
Alcohol QFI, log	-0.08 (0.08)	-.08	0.5	-1.06			
Regular cigarette use	0.22 (0.16)	.09	0.8	1.34			
Exercise index, log	0.05 (0.05)	.08	0.5	1.12			
No. of hours of sleep	-0.03 (0.06)	-.04	0.2	-0.62			

Notes: *B* = unstandardized; β = standardized; UV = unique variance; SDNN = standard deviation of normal-to-normal intervals; BMI = body mass index; alcohol QFI = alcohol quantity–frequency index (log-transformed); no. = number; LF HRV = low-frequency (0.05–0.15 Hz). Participants who were assigned to the alcohol condition (*n* = 25) were excluded from the analysis as acute alcohol consumption can influence HRV (Vaschillo et al., 2008) (*N* = 187). Regular cigarette use was defined as reporting cigarette use at least four times per week for the past 30 days (1 = yes, 0 = no). All models included the following covariates: age, gender (women = 1, men = 0), race (White = 1, non-White = 0), and mean respiration volume during the paced breathing task. Baseline HRV indices were a significant covariate (for SDNN: *B* [*SE*] = 0.56 [0.05], β = .65, UV = 35.9%, *t* = 11.05; for LF HRV: *B* [*SE*] = 0.34 [0.06], β = .43, UV = 15.4%, *t* = 6.03). Alcohol and cigarette use were assessed for the past 30 days.

**p* < .05.

college students. Some, but not all, of the hypotheses were supported. As hypothesized, a negative relationship between the intensity of alcohol use and basal HRV in college drinkers was identified. The higher an individual's alcohol use, the lower was his/her basal HF HRV, potentially indicating less optimal vagal functioning. This observation is compelling in light of previously observed reductions in HRV in chronic, heavy drinking adults (Ingjaldsson et al., 2003; Thayer et al., 2006). The present finding suggests that relatively heavier drinking during emerging adulthood is also linked to suboptimal neurocardiac functioning, even in the absence of alcohol dependence. It will be important for future prospective studies to determine whether alcohol use among college students has direct health implications beyond the more visible alcohol-related negative consequences, such as accidental injuries, getting into fights, unwanted sex, and experiencing hangovers.

Furthermore, similar to the positive associations between physical activity and HRV indices found in healthy adults and seniors (Hautala et al., 2003; Iwasaki et al., 2003; Okazaki et al., 2005; Sloan et al., 2009), physical activity was positively associated with basal SDNN among this sample of college drinkers. Thus, across the lifespan, it appears that higher levels of physical activity are associated with relatively better cardiac health. It will be important to determine the temporal course of this relationship and to evaluate also whether increased HRV may mediate some of the benefit of physical activity on cardiac health (Deligiannis et al., 1999; Kouidi et al., 2013).

This study also found that BMI was negatively associated with reactive HRV measured during an acute breathing

challenge that taxed the cardiorespiratory system. HRV during 0.1-Hz paced breathing is thought to provide a measure of the maximal capacity of the cardiovascular system for change and flexibility (Lehrer et al., 2000; Vaschillo et al., 2006) by synchronizing cardiorespiratory and neurocardiac processes (Vaschillo et al., 2006), which are two main influences on cardiac variability. Breathing at 0.1 Hz provokes high-amplitude oscillations primarily in the LF portion of the RRI spectrum. The observation that reactive LF HRV was lower in healthy college students with higher BMI suggests the possibility of early-stage difficulties in a neurocardiac mechanism that regulates adaptability. This adds to the already considerable public health concerns related to the increasing prevalence of obesity, which has been linked with various chronic conditions in adults, including cardiovascular diseases, type 2 diabetes, and certain cancers (Ogden et al., 2007).

Overall, the present results suggest that heavy alcohol consumption, less physical activity, and unhealthy weight are associated with subtly diminished neurocardiac function in college drinkers. Reduced neurocardiac signaling may have an important role in the daily functioning of college students. For example, adequate HRV may be important for college students to constructively cope with the daily stressors common during emerging adulthood (Fabes and Eisenberg, 1997). Resilience to stress from changes in the environment, which can be gauged from reactive HRV measures, is essential in negotiating successful transitions into adulthood (Masten et al., 2004; Rudolph et al., 2001). Moreover, identifying subtle neurocardiac disruptions well before the onset of physical or mental disorders, regardless of whether

they are the cause and/or consequence of malleable health risk factors, may have considerable value in public health. Cardiovascular disease is a leading cause of morbidity and mortality in the United States, accounting for 1 in every 2.8 deaths (Roger et al., 2011). The risk for the first cardiovascular event increases dramatically after age 35 in men and age 45 in women (American Heart Association, 2010). Yet, the atherosclerotic processes that underlie myocardial infarction and stroke often start in childhood and are influenced across the lifespan by potentially modifiable lifestyle factors (Hayman and Reineke, 2003; Hayman et al., 2007). The present study provides support for examining both basal and reactive HRV in young, apparently healthy individuals to capture a more complete picture of health.

The current findings did not support all hypothesized relationships, which should be interpreted in the context of several unique aspects of the present study. First, the sample was limited by the strict inclusion/exclusion criteria of the parent studies that restricted the ranges of health issues and behaviors of the present sample, compared with those in the general population of emerging adults attending college. Without this restriction, the overall variance explained by the models, as well as the unique variance explained by each lifestyle factor, might have been much greater. Despite the restriction in range, the total variance explained ranged from 14% to 20% for basal HRV and from 28% to 50% for reactive HRV. Note that the relatively modest unique contributions by individual lifestyle factors reflect that many of these lifestyle factors are found together in individuals, and their shared influence on HRV is collectively removed when unique contributions are examined. The findings may not generalize to college students who do not drink, who drink lightly, or who are alcohol dependent.

Second, the list of health risks and lifestyle choices assessed in this study was not comprehensive. Future studies should include additional factors, such as diet and cholesterol levels. In addition, our assessment of smoking might not have been sufficiently fine grained compared with other studies in the literature. Thus, contrary to prediction, the present study did not find a significant relationship between cigarette use and HRV. Our operationalization of smokers was based on frequency but not quantity of smoking, with daily or nearly daily smoking serving as a proxy measure for heavy smoking. This might have contributed to the inconsistent results between the current study and previous studies of heavy adult smokers (Barutcu et al., 2005; Cagirci et al., 2009). In addition, students who regularly used drugs other than cigarettes were underrepresented in this sample. The lack of association between sleep and HRV in the present study may indicate that sleep quality, rather than sleep duration, may be more strongly related to HRV (Brandenberger et al., 2003).

Finally, because of the cross-sectional nature of the data, it was not possible to examine the temporal relationship

between lifestyle factors and cardiovascular function. It is conceivable that health risks negatively affect neurocardiac dynamics and that relative decrements in neurocardiac dynamics could give rise to tendencies to avoid physical exercise, engage in drinking to cope with stress, and/or to have a higher BMI. The cross-sectional associations found in the present study suggest that it is important for future research to address the direction of causal links between the various lifestyle factors and HRV.

Despite some of the limitations noted above, the present study has a number of implications for public health, which warrant further experimental and prospective study. For example, an increase in LF HRV during the paced breathing task used in this study is thought to be driven by the activation of the baroreflex that amplifies heart rate oscillation in the LF of the spectral density at 0.1 Hz (Lehrer et al., 2000; Song and Lehrer, 2003). The baroreflex is a key mechanism that supports bidirectional communication between the heart and the brain to ensure stabilization of blood pressure (Cevese et al., 2001) and coordinated regulation of stress and emotion by the central nervous system and autonomic nervous system (Appelhans and Luecken, 2006; Thayer and Lane, 2000; Vaschillo et al., 2008). The present findings suggest that HRV, as an active neurocardiac signaling process, may serve as a mechanistic link between unhealthy lifestyle choices and suboptimal cardiovascular control in emerging adulthood, reduced resilience to stress and emotional challenge, and potentially, increased risk for cardiovascular problems in the future. Suppression of HRV by health risk behaviors is modifiable, and basal HRV often rebounds on cessation of these behaviors (e.g., Castello et al., 2011; de Jonge et al., 2010; Lewis et al., 2010; Mouridsen et al., 2012; Munjal et al., 2009; Sjoberg et al., 2011; Sridhar et al., 2010; Weise et al., 1986). Thus, HRV may also be useful as a tool to objectively evaluate the positive effects of changes in lifestyle.

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References

- Abboud, F. M. (2010). The Walter B. Cannon Memorial Award Lecture, 2009. Physiology in perspective: The wisdom of the body. In search of autonomic balance: The good, the bad, and the ugly. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology*, 298, R1449–R1467.
- American Heart Association. (2010). *Heart disease and stroke statistics—2010 update*. Dallas, TX: Author.
- Appelhans, B. M., & Luecken, L. J. (2006). Heart rate variability as an index of regulated emotional responding. *Review of General Psychology*, 10, 229–240.
- Arnett, J. J. (2000). Emerging adulthood. A theory of development from

- the late teens through the twenties. *The American Psychologist*, 55, 469–480.
- Barnett, N. P., & Read, J. P. (2005). Mandatory alcohol intervention for alcohol-abusing college students: A systematic review. *Journal of Substance Abuse Treatment*, 29, 147–158.
- Barutcu, I., Esen, A. M., Kaya, D., Turkmen, M., Karakaya, O., Melek, M., . . . Basaran, Y. (2005). Cigarette smoking and heart rate variability: Dynamic influence of parasympathetic and sympathetic maneuvers. *Annals of Noninvasive Electrocardiology*, 10, 324–329.
- Bates, M. E., Buckman, J. F., Vaschillo, E. G., Fonoberov, V. A., Fonoberova, M., Vaschillo, B., . . . Mezi, I. (2011). The redistribution of power: Neurocardiac signaling, alcohol and gender. *PLoS ONE*, 6(12), e28281.
- Beck, A. T., & Steer, R. A. (1993). *Beck Anxiety Inventory Manual*. San Antonio, TX: Harcourt Brace Jovanovich.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory* (2nd ed.). San Antonio, TX: Psychological Corp.
- Benarroch, E. E. (1997). The central autonomic network. In P. A. Low (Ed.), *Clinical autonomic disorders* (2nd ed., pp. 17–23). Philadelphia, PA: Lippincott-Raven.
- Benarroch, E. E. (2008). The arterial baroreflex: Functional organization and involvement in neurologic disease. *Neurology*, 71, 1733–1738.
- Berg, C. J., Parelkar, P. P., Lessard, L., Escoffery, C., Kegler, M. C., Sterling, K. L., & Ahluwalia, J. S. (2010). Defining “smoker”: College student attitudes and related smoking characteristics. *Nicotine & Tobacco Research*, 12, 963–969.
- Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., . . . van der Molen, M. W. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, 34, 623–648.
- Bleichert, J., Michael, T., Grossman, P., Lajtman, M., & Wilhelm, F. H. (2007). Autonomic and respiratory characteristics of posttraumatic stress disorder and panic disorder. *Psychosomatic Medicine*, 69, 935–943.
- Brandenberger, G., Viola, A. U., Ehrhart, J., Charloux, A., Geny, B., Piquard, F., & Simon, C. (2003). Age-related changes in cardiac autonomic control during sleep. *Journal of Sleep Research*, 12, 173–180.
- Buckman, J. F., White, H. R., & Bates, M. E. (2010). Psychophysiological reactivity to emotional picture cues two years after college students were mandated for alcohol interventions. *Addictive Behaviors*, 35, 786–790.
- Cagirci, G., Cay, S., Karakurt, O., Eryasar, N., Kaya, V., Canga, A., . . . Akdemir, R. (2009). Influence of heavy cigarette smoking on heart rate variability and heart rate turbulence parameters. *Annals of Noninvasive Electrocardiology*, 14, 327–332.
- Castello, V., Simões, R. P., Bassi, D., Catai, A. M., Arena, R., & Borghi-Silva, A. (2011). Impact of aerobic exercise training on heart rate variability and functional capacity in obese women after gastric bypass surgery. *Obesity Surgery*, 21, 1739–1749.
- Cevese, A., Gulli, G., Polati, E., Gottin, L., & Grasso, R. (2001). Baroreflex and oscillation of heart period at 0.1 Hz studied by alpha-blockade and cross-spectral analysis in healthy humans. *The Journal of Physiology*, 531, 235–244.
- Cohen, H., & Benjamin, J. (2006). Power spectrum analysis and cardiovascular morbidity in anxiety disorders. *Autonomic Neuroscience: Basic & Clinical*, 128, 1–8.
- Cohen, H., Benjamin, J., Geva, A. B., Matar, M. A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. *Psychiatry Research*, 96, 1–13.
- Cohen, M. A., & Taylor, J. A. (2002). Short-term cardiovascular oscillations in man: Measuring and modelling the physiologies. *The Journal of Physiology*, 542, 669–683.
- Cooke, W. H., Hoag, J. B., Crossman, A. A., Kuusela, T. A., Tahvanainen, K. U., & Eckberg, D. L. (1999). Human responses to upright tilt: A window on central autonomic integration. *The Journal of Physiology*, 517, 617–628.
- Dawson, D. A., Grant, B. F., Stinson, F. S., & Chou, P. S. (2004). Another look at heavy episodic drinking and alcohol use disorders among college and noncollege youth. *Journal of Studies on Alcohol*, 65, 477–488.
- de Jonge, L., Moreira, E. A. M., Martin, C. K., Ravussin, E., & the Pennington CALERIE Team. (2010). Impact of 6-month caloric restriction on autonomic nervous system activity in healthy, overweight, individuals. *Obesity*, 18, 414–416.
- Deligiannis, A., Kouidi, E., & Tourkantonis, A. (1999). Effects of physical training on heart rate variability in patients on hemodialysis. *American Journal of Cardiology*, 84, 197–202.
- De Vries-Bouw, M., Popma, A., Vermeiren, R., Doreleijers, T. A., Van De Ven, P. M., & Jansen, L. M. (2011). The predictive value of low heart rate and heart rate variability during stress for reoffending in delinquent male adolescents. *Psychophysiology*, 48, 1597–1604.
- Fabes, R. A., & Eisenberg, N. (1997). Regulatory control and adults’ stress-related responses to daily life events. *Journal of Personality and Social Psychology*, 73, 1107–1117.
- Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, 44, 133–151.
- Fromme, K., Corbin, W. R., & Kruse, M. I. (2008). Behavioral risks during the transition from high school to college. *Developmental Psychology*, 44, 1497–1504.
- Galetta, F., Carpi, A., Abraham, N., Guidotti, E., Russo, M. A., Camici, M., . . . Santoro, G. (2012). Age related cardiovascular dysfunction and effects of physical activity. *Frontiers in Bioscience (Elite edition)*, 4, 2617–2637.
- Garrow, J. S., & Webster, J. (1985). Quetelet’s index (W/H²) as a measure of fatness. *International Journal of Obesity*, 9, 147–153.
- Giardino, N., Lehrer, P. M., & Feldman, J. (2000). The role of oscillations in self-regulation: Their contribution to homeostasis. In D. Kenney & F. J. McGuigan (Eds.), *Stress and health: Research and clinical applications* (pp. 27–52). London, England: Harwood.
- Goldstein, D. S. (2001). *The autonomic nervous system in health and disease*. New York, NY: Marcel Dekker.
- Hautala, A. J., Mäkikallio, T. H., Kiviniemi, A., Laukkanen, R. T., Nissilä, S., Huikuri, H. V., & Tulppo, M. P. (2003). Cardiovascular autonomic function correlates with the response to aerobic training in healthy sedentary subjects. *American Journal of Physiology: Heart and Circulatory Physiology*, 285, H1747–H1752.
- Hayman, L. L., Meininger, J. C., Daniels, S. R., McCrindle, B. W., Helden, L., Ross, J., . . . Williams, C. L. (2007). Primary prevention of cardiovascular disease in nursing practice: Focus on children and youth: A scientific statement from the American Heart Association Committee on Atherosclerosis, Hypertension, and Obesity in Youth of the Council on Cardiovascular Disease in the Young, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council on Nutrition, Physical Activity, and Metabolism. *Circulation*, 116, 344–357.
- Hayman, L. L., & Reineke, P. R. (2003). Preventing coronary heart disease: The implementation of healthy lifestyle strategies for children and adolescents. *Journal of Cardiovascular Nursing*, 18, 294–301.
- Hempel, R. J., Tulen, J. H., van Beveren, N. J., Mulder, P. G., & Hengeveld, M. W. (2007). Subjective and physiological responses to emotion-eliciting pictures in male schizophrenic patients. *International Journal of Psychophysiology*, 64, 174–183.
- Hughes, J. W., & Stoney, C. M. (2000). Depressed mood is related to high-frequency heart rate variability during stressors. *Psychosomatic Medicine*, 62, 796–803.
- Ingjaldsson, J. T., Laberg, J. C., & Thayer, J. F. (2003). Reduced heart rate variability in chronic alcohol abuse: Relationship with negative mood, chronic thought suppression, and compulsive drinking. *Biological Psychiatry*, 54, 1427–1436.

- Iwasaki, K., Zhang, R., Zuckerman, J. H., & Levine, B. D. (2003). Dose-response relationship of the cardiovascular adaptation to endurance training in healthy adults: How much training for what benefit? *Journal of Applied Physiology*, 95, 1575–1583.
- Jennings, J. R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, P. (1992). Alternate cardiovascular baseline assessment techniques: Vanilla or resting baseline. *Psychophysiology*, 29, 742–750.
- Kazuma, N., Otsuka, K., Matsuoka, I., & Murata, M. (1997). Heart rate variability during 24 hours in asthmatic children. *Chronobiology International*, 14, 597–606.
- Kerr, W. C., Greenfield, T. K., Bond, J., Ye, Y., & Rehm, J. (2009). Age-period-cohort modelling of alcohol volume and heavy drinking days in the US National Alcohol Surveys: Divergence in younger and older adult trends. *Addiction*, 104, 27–37.
- Koschke, M., Boettger, M. K., Schulz, S., Berger, S., Terhaar, J., Voss, A., . . . Bär, K.-J. (2009). Autonomy of autonomic dysfunction in major depression. *Psychosomatic Medicine*, 71, 852–860.
- Kouidi, E., Vergoulas, G., Anifanti, M., & Deligiannis, A. (2013). A randomized controlled trial of exercise training on cardiovascular and autonomic function among renal transplant recipients. *Nephrology, Dialysis, Transplantation*, 28, 1294–1305.
- Laska, M. N., Pasch, K. E., Lust, K., Story, M., & Ehlinger, E. (2009). Latent class analysis of lifestyle characteristics and health risk behaviors among college youth. *Prevention Science*, 10, 376–386.
- Lehrer, P. M., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: Rationale and manual for training. *Applied Psychophysiology and Biofeedback*, 25, 177–191.
- Lewis, M. J., Balaji, G., Dixon, H., Syed, Y., & Lewis, K. E. (2010). Influence of smoking abstinence and nicotine replacement therapy on heart rate and QT time-series. *Clinical Physiology and Functional Imaging*, 30, 43–50.
- Licht, C. M., de Geus, E. J., Zitman, F. G., Hoogendijk, W. J., van Dyck, R., & Penninx, B. W. (2008). Association between major depressive disorder and heart rate variability in the Netherlands Study of Depression and Anxiety (NESDA). *Archives of General Psychiatry*, 65, 1358–1367.
- Masten, A. S., Burt, K. B., Roisman, G. I., Obradović, J., Long, J. D., & Tellegen, A. (2004). Resources and resilience in the transition to adulthood: Continuity and change. *Development and Psychopathology*, 16, 1071–1094.
- Metropolitan Life Insurance Company (1983). The Metropolitan Life Height–Weight Table. *Statistical Bulletin*, 64, 2–9.
- Molfino, A., Fiorentini, A., Tubani, L., Martuscelli, M., Rossi Fanelli, F., & Laviano, A. (2009). Body mass index is related to autonomic nervous system activity as measured by heart rate variability. *European Journal of Clinical Nutrition*, 63, 1263–1265.
- Mouridsen, M. R., Bendsen, N. T., Astrup, A., Haugaard, S. B., Binici, Z., & Sajadieh, A. (2012). Modest weight loss in moderately overweight postmenopausal women improves heart rate variability. *European Journal of Preventive Cardiology*. Advance online publication. doi: 10.1177/2047487312444367
- Munjal, S., Koval, T., Muhammad, R., Jin, Y., Demmel, V., Roethig, H. J., . . . Unverdorben, M. (2009). Heart rate variability increases with reductions in cigarette smoke exposure after 3 days. *Journal of Cardiovascular Pharmacology and Therapeutics*, 14, 192–198.
- Nahshoni, E., Aravot, D., Aizenberg, D., Sigler, M., Zalsman, G., Strasberg, B., . . . Weizman, A. (2004). Heart rate variability in patients with major depression. *Psychosomatics*, 45, 129–134.
- Negrão, B. L., Bipath, P., van der Westhuizen, D., & Viljoen, M. (2011). Autonomic correlates at rest and during evoked attention in children with attention-deficit/hyperactivity disorder and effects of methylphenidate. *Neuropsychobiology*, 63, 82–91.
- Nelson, M. C., Story, M., Larson, N. I., Neumark-Sztainer, D., & Lytle, L. A. (2008). Emerging adulthood and college-aged youth: An overlooked age for weight-related behavior change. *Obesity*, 16, 2205–2211.
- Ogden, C. L., Yanovski, S. Z., Carroll, M. D., & Flegal, K. M. (2007). The epidemiology of obesity. *Gastroenterology*, 132, 2087–2102.
- Okazaki, K., Iwasaki, K.-I., Prasad, A., Palmer, M. D., Martini, E. R., Fu, Q., . . . Levine, B. D. (2005). Dose–response relationship of endurance training for autonomic circulatory control in healthy seniors. *Journal of Applied Physiology*, 99, 1041–1049.
- O'Malley, P. M., & Johnston, L. D. (2002). Epidemiology of alcohol and other drug use among American college students. *Journal of Studies on Alcohol, Supplement 14*, 23–39.
- Pandina, R. J., Labouvie, E. W., & White, H. R. (1984). Potential contributions of the life span development approach to the study of adolescent alcohol and drug use: The Rutgers Health and Human Development Project, a working model. *Journal of Drug Issues*, 14, 253–268.
- Quilliot, D., Bohme, P., Zannad, F., & Ziegler, O. (2008). Sympathetic-leptin relationship in obesity: Effect of weight loss. *Metabolism*, 57, 555–562.
- Rajan, I., Murthy, P. J. N. V., Ramakrishnan, A. G., Gangadhar, B. N., & Janakiramaiah, N. (1998). Heart rate variability as an index of cue reactivity in alcoholics. *Biological Psychiatry*, 43, 544–546.
- Roger, V. L., Go, A. S., Lloyd-Jones, D. M., Adams, R. J., Berry, J. D., Brown, T. M., . . . Wylie-Rosett, J., & the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2011). Heart disease and stroke statistics—2011 update: A report from the American Heart Association. *Circulation*, 123, e18–e209.
- Rudolph, K. D., Lambert, S. F., Clark, A. G., & Kurlakowsky, K. D. (2001). Negotiating the transition to middle school: The role of self-regulatory processes. *Child Development*, 72, 929–946.
- Schmidt, H., Müller-Werdan, U., Hoffmann, T., Francis, D. P., Piepoli, M. F., Rauchhaus, M., . . . Werdan, K. (2005). Autonomic dysfunction predicts mortality in patients with multiple organ dysfunction syndrome of different age groups. *Critical Care Medicine*, 33, 1994–2002.
- Schroeder, M. A. (1990). Diagnosing and dealing with multicollinearity. *Western Journal of Nursing Research*, 12, 175–184.
- Shinba, T., Kariya, N., Matsui, Y., Ozawa, N., Matsuda, Y., & Yamamoto, K. (2008). Decrease in heart rate variability response to task is related to anxiety and depressiveness in normal subjects. *Psychiatry and Clinical Neurosciences*, 62, 603–609.
- Sjoberg, N., Brinkworth, G. D., Wyckley, T. P., Noakes, M., & Saint, D. A. (2011). Moderate weight loss improves heart rate variability in overweight and obese adults with type 2 diabetes. *Journal of Applied Physiology*, 110, 1060–1064.
- Sloan, R. P., Shapiro, P. A., DeMeersman, R. E., Bagiella, E., Brondolo, E. N., McKinley, P. S., . . . Myers, M. M. (2009). The effect of aerobic training and cardiac autonomic regulation in young adults. *American Journal of Public Health*, 99, 921–928.
- Song, H. S., & Lehrer, P. M. (2003). The effects of specific respiratory rates on heart rate and heart rate variability. *Applied Psychophysiology and Biofeedback*, 28, 13–23.
- Spencer, L. (2002). Results of a heart disease risk-factor screening among traditional college students. *Journal of American College Health*, 50, 291–296.
- Spiegel, K., Leproult, R., L'Hermite-Balériaux, M., Copinschi, G., Penev, P. D., & Van Cauter, E. (2004). Leptin levels are dependent on sleep duration: Relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. *Journal of Clinical Endocrinology & Metabolism*, 89, 5762–5771.
- Sridhar, B., Haleagrahara, N., Bhat, R., Kulur, A. B., Avabratha, S., & Adhikary, P. (2010). Increase in the heart rate variability with deep breathing in diabetic patients after 12-month exercise training. *Tohoku Journal of Experimental Medicine*, 220, 107–113.
- Stein, P. K., Barzilay, J., Domitrovich, P. P., Chaves, P. H. M., Gottdiener, J., Rich, M. W., . . . Kronmal, R. (2006). Abnormal heart rate variability is a powerful independent predictor of long-term cardiovascular mortality in the elderly: The cardiovascular health study [Abstract 992-136]. *Journal of the American College of Cardiology*, 47(4), Supplement 1, 28A–29A.

- Stein, P. K., & Pu, Y. (2012). Heart rate variability, sleep and sleep disorders. *Sleep Medicine Reviews*, 16, 47–66.
- Task Force of the European Society of Cardiology and the American Society of Pacing and Electrophysiology (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation*, 93, 1043–1065.
- Taylor, J. A., Carr, D. L., Myers, C. W., & Eckberg, D. L. (1998). Mechanisms underlying very-low-frequency RR-interval oscillations in humans. *Circulation*, 98, 547–555.
- Thayer, J. F., Hall, M., Sollers, J. J., III, & Fischer, J. E. (2006). Alcohol use, urinary cortisol, and heart rate variability in apparently healthy men: Evidence for impaired inhibitory control of the HPA axis in heavy drinkers. *International Journal of Psychophysiology*, 59, 244–250.
- Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, 61, 201–216.
- Tucker, P., Pfefferbaum, B., Jeon-Slaughter, H., Khan, Q., & Garton, T. (2012). Emotional stress and heart rate variability measures associated with cardiovascular risk in relocated Katrina survivors. *Psychosomatic Medicine*, 74, 160–168.
- Udo, T., Bates, M. E., Mun, E. Y., Vaschillo, E. G., Vaschillo, B., Lehrer, P., & Ray, S. (2009). Gender differences in acute alcohol effects on self-regulation of arousal in response to emotional and alcohol-related picture cues. *Psychology of Addictive Behaviors*, 23, 196–204.
- Vaschillo, E. G., Bates, M. E., Vaschillo, B., Lehrer, P., Udo, T., Mun, E. Y., & Ray, S. (2008). Heart rate variability response to alcohol, placebo, and emotional picture cue challenges: Effects of 0.1-Hz stimulation. *Psychophysiology*, 45, 847–858.
- Vaschillo, E. G., Vaschillo, B., & Lehrer, P. M. (2006). Characteristics of resonance in heart rate variability stimulated by biofeedback. *Applied Psychophysiology and Biofeedback*, 31, 129–142.
- Vinik, A. I., Maser, R. E., Mitchell, B. D., & Freeman, R. (2003). Diabetic autonomic neuropathy. *Diabetes Care*, 26, 1553–1579.
- Wechsler, H., Dowdall, G. W., Davenport, A., & Rimm, E. B. (1995). A gender-specific measure of binge drinking among college students. *American Journal of Public Health*, 85, 982–985.
- Weise, F., Müller, D., Krell, D., Kielstein, V., & Koch, R. D. (1986). Heart rate variability in chronic alcoholics: A follow-up study. *Drug and Alcohol Dependence*, 17, 365–368.
- Yusko, D. A., Buckman, J. F., White, H. R., & Pandina, R. J. (2008). Risk for excessive alcohol use and drinking-related problems in college student athletes. *Addictive Behaviors*, 33, 1546–1556.