

Psychophysiological characteristics of narcissism during active and passive coping

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Abstract

This study provides the first psychophysiological analysis of narcissism by measuring autonomic responses during active and passive anticipatory coping in 40 undergraduate men who scored high or low on the Narcissistic Personality Inventory (NPI). Compared to the low NPI group, the high NPI group showed greater preejection period (PEP) shortening, cardiac deceleration, and skin conductance response (SCR) habituation during anticipation of an aversive stimulus ($p < .02$). As expected, SCR and PEP reactivity were greater during active than passive coping. In the case of PEP, this effect emerged only in the low NPI group; the high NPI group showed the greatest PEP reactivity during the first task, regardless of coping demands. These data support hypothesized relationships among narcissism, psychopathy, and psychological predictors of cardiovascular disease, and suggest that a psychobiological dimension may underlie important features of narcissism.

Descriptors: Narcissism, Skin conductance response, Cardiovascular reactivity, Psychopathy, Personality, Impedance cardiography

Despite 100 years of theoretical and clinical work on narcissism (see Akhtar & Thomson, 1982), most of the quantitative empirical work on this personality construct has been conducted within the past 20 years. This empirical work stems primarily from two developments: (a) the inclusion of narcissistic personality disorder in recent editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III, DSM-IV; American Psychiatric Association (APA), 1980, 1994) and (b) the development of psychometrically sound instruments for assessing narcissism in normal and clinical populations (Emmons, 1987; Raskin & Terry, 1988). Most of the empirical work on narcissism has involved validation of these instruments by correlating them with various self-report measures or behavioral ratings (Emmons, 1987; Raskin & Terry, 1988; Rhodewalt & Morf, 1995). However, some experimental studies of narcissism have emerged recently (e.g., Bushman & Baumeister, 1998; Kernis & Sun, 1994; Morf & Rhodewalt, 1993; Rhodewalt & Morf, 1998).

Narcissism has been conceptualized as a cognitive and affective preoccupation with the self (Westen, 1990), characterized by egocentricity, grandiosity, arrogance, envy, a need for admiration, and a lack of empathy (Akhtar & Thomson, 1982; APA, 1994). Nar-

cissists have fragile or unstable self-esteem, a sense of entitlement and unreasonable expectations of special treatment, and an interpersonal style that is exploitative, insensitive, competitive, dominant, and aggressive (APA, 1994; Raskin & Terry, 1988; Rhodewalt, Madrian, & Cheney, 1998). Threats to self-esteem, involving criticism, defeat, or the failure of others to meet narcissistic expectations of entitlement and special treatment, tend to provoke hostility, contempt, shame, and rage (APA, 1994; Bushman & Baumeister, 1998; Kernis & Sun, 1994; Morf & Rhodewalt, 1993; Rhodewalt & Morf, 1998). Indeed, many features of narcissism, such as grandiosity and dominance, seem to serve a defensive function to manage negative affect and maintain self-esteem (Raskin, Novacek, & Hogan, 1991; Raskin & Terry, 1988; Rhodewalt & Morf, 1995).

The most widely used research instrument for assessing narcissism is the Narcissistic Personality Inventory (NPI; Raskin & Terry, 1988). The NPI is a 40-item forced-choice questionnaire based on DSM-III (APA, 1980) criteria for narcissistic personality disorder. The NPI provides a measure of individual differences in narcissism, with extreme elevations reflecting pathological narcissism and less extreme scores reflecting variations in narcissistic personality traits. Thus the NPI is appropriate for use with clinical or nonclinical populations. The NPI has good internal consistency and retest stability, and there is considerable evidence of its construct validity (see Emmons, 1987; Raskin & Hall, 1981; Raskin & Terry, 1988). Scores on the NPI correlate positively with self-report and behavioral measures of egocentricity (Emmons, 1987; Raskin & Terry, 1988), grandiosity (Raskin et al., 1991; Raskin & Novacek, 1991; Rhodewalt & Morf, 1995), hostility and antago-

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nism (Bradlee & Emmons, 1992; Fukunishi, Hattori, Nakamura, & Nakagawa, 1995; Raskin & Terry, 1988; Raskin et al., 1991; Rhodewalt & Morf, 1995), dominance (Bradlee & Emmons, 1992; Emmons, 1984; Raskin & Terry, 1988; Raskin et al., 1991), aggression (Bushman & Baumeister, 1998; Raskin & Terry, 1988), self-esteem (Emmons, 1984; Kernis & Sun, 1994; Raskin & Terry, 1988; Raskin et al., 1991; Rhodewalt & Morf, 1995, 1998; Rhodewalt et al., 1998), self-esteem instability and affective lability (Emmons, 1987; Rhodewalt et al., 1998), competitiveness, assertiveness, and leadership (Raskin & Terry, 1988), as well as extraversion, exhibitionism, impulsivity, sensation seeking, and impatience (Bradlee & Emmons, 1992; Emmons, 1984; Raskin & Hall, 1981; Raskin & Terry, 1988). Scores on the NPI correlate negatively with empathy (Watson, Grisham, Trotter, & Biderman, 1984) and anxiety (Emmons, 1984; Raskin & Novacek, 1989).

In addition to these correlates, recent studies have linked narcissism to psychopathy (Blackburn & Coid, 1998; Harpur, Hare, & Hakstian, 1989; Zagon & Jackson, 1994) and Type A behavior (Fukunishi et al., 1995, 1996), two areas in which a psychophysiological analysis has been useful and heuristic. Although most experimental research on narcissism has focused on psychosocial concepts such as threats to self-esteem and self-esteem management (e.g., Bushman & Baumeister, 1998; Kernis & Sun, 1994; Morf & Rhodewalt, 1993; Rhodewalt & Morf, 1998), the association of narcissism with psychopathy and Type A behavior raises the possibility that a fundamental psychobiological dimension may be involved (Cloninger, 1987; Fowles, 1994; McBurnett, 1992). Accordingly, a psychophysiological analysis of narcissism may clarify or reveal important attentional, motivational, affective, and self-regulatory processes involved in narcissism, psychopathy, and cardiovascular reactivity and disease.

Narcissism, Psychopathy, and Psychophysiological Reactivity to Aversive Events

Kernberg (1989) has suggested that narcissism is the foundation or core of psychopathy and antisocial personality disorder. Indeed, many characteristics of narcissism are features of psychopathy and antisocial personality disorder, including egocentricity, grandiosity, low empathy, low anxiety, a sense of entitlement, interpersonal exploitativeness, dominance and aggression, hostility and antagonism, and impulsivity and sensation seeking (Cleckley, 1976; Harpur et al., 1989; Patrick, 1994; Zagon & Jackson, 1994). Moreover, both self-report and observer ratings indicate that there are significant relationships between narcissism and psychopathy (Blackburn & Coid, 1998; Harpur et al., 1989; Zagon & Jackson, 1994). Thus, well-defined psychophysiological characteristics of psychopathy and antisocial personality disorder, such as diminished skin conductance responding in anticipation of aversive events (Fowles, 1980; Hare, 1978, 1982; Hare & Craigen, 1974; Hare, Frazelle, & Cox, 1978; Lykken, 1995; Ogloff & Wong, 1990; Tharp, Maltzman, Sydulko, & Ziskind, 1980) and diminished skin conductance orienting responses (Hare & Craigen, 1974; Raine & Venables, 1984; Raine, Venables, & Williams, 1995), may also characterize narcissism and indeed stem from a common narcissistic core.

Psychophysiological research has consistently linked psychopathy to diminished electrodermal reactivity in anticipation of punishment or aversive events (Fowles, 1980; Hare, 1978, 1982; Hare & Craigen, 1974; Hare et al., 1978; Ogloff & Wong, 1990; Tharp et al., 1980), regardless of whether those events are unavoidable or avoidable (Hare, 1982; Ogloff & Wong, 1990). The data linking psychopathy to heart rate reactivity are less consistent. Several findings suggest that psychopaths show enhanced heart rate accel-

eration in anticipation of aversive events (Hare, 1978, 1982; Hare & Craigen, 1974; Hare et al., 1978; Ogloff & Wong, 1990). On the basis of such findings and theoretical concepts of sensory rejection (Lacey, 1967) and active coping (Obrist, 1981), Hare (1978, 1998) has hypothesized that psychopaths may possess a particularly efficient active coping mechanism for reducing the impact of aversive threat. However, a closer examination of these studies suggests a more complicated picture, with nearly as many instances of enhanced heart rate deceleration as acceleration in psychopaths (e.g., Hare, 1982; Ogloff & Wong, 1990), as well as cases in which both cardiac acceleration and deceleration are stronger in psychopaths than in nonpsychopaths (Hare & Craigen, 1974; Hare et al., 1978). Given the moderate magnitude and phasic pattern of heart rate reactivity in these studies, it seems reasonable to suggest as an alternative hypothesis that psychopaths may exhibit enhanced parasympathetic modulatory control or tuning of heart rate, resulting in heightened parasympathetically mediated cardiac acceleration or deceleration depending on the attentional, motor, or coping demands at hand.

Fowles (1980, 1988, 1994) has used Gray's (1987) motivational theory to develop a theoretical analysis of the electrodermal and heart-rate findings in the psychopathy literature. According to Fowles, variations in heart rate reflect the activity of a behavioral activation system (BAS), an appetitive motivational system that initiates behavior in response to stimuli associated with reward, approach, or active avoidance. These stimuli elicit increases in heart rate, but have little or no impact on electrodermal responding. In contrast, variations in electrodermal responding reflect the activity of a behavioral inhibition system (BIS), an aversive motivational system that inhibits behavior in response to stimuli associated with punishment, nonreward, or passive avoidance. Such aversive stimuli elicit electrodermal responding, but have no consistent effects on heart rate. These two motivational systems presumably interact in a reciprocal, antagonistic manner. Fowles argues that the BIS of psychopaths is deficient, resulting in diminished electrodermal reactivity and a lack of anxiety in anticipation of aversive events, as well as poor inhibition of impulsivity and aggression. In contrast, the BAS of psychopaths is presumably either normal or disinhibited due to the BIS deficiency, resulting in either normal or exaggerated heart-rate reactivity during anticipatory stress.

Although psychopaths are low in anticipatory anxiety, there is some evidence that they may be prone to heightened somatic anxiety (Schalling, 1978), including symptoms of cardiovascular hyperreactivity and an exaggerated fight/flight response to aversive stimuli (Fowles, 1994). Thus, despite diminished sympathetic effects on the electrodermal system, sympathetic adrenomedullary effects on the cardiovascular system may be enhanced in psychopathy. An enhanced fight/flight response might contribute to the characteristic impulsivity and aggression associated with psychopathy (Fowles, 1994), as well as to the link between risk factors for antisocial behavior and those for cardiovascular disease (Pine et al., 1996). A similar combination of deficient behavioral inhibition, normal or disinhibited behavioral activation, and heightened mobilization for fight or flight might characterize narcissism, if it indeed forms the core of psychopathy (Kernberg, 1989).

Narcissism, Psychological Predictors of Heart Disease, and Cardiovascular Reactivity to Stress

Narcissism correlates positively with hostility and antagonism, dominance and competitiveness, and Type A behavior, all of which are related in turn to cardiovascular disease (Booth-Kewley & Friedman, 1987; Houston, Babyak, Chesney, Black, & Ragland, 1997;

Kaplan, Manuck, Clarkson, & Prichard, 1985; Miller, Smith, Turner, Guijarro, & Hallet, 1996). The pathophysiological link between these psychological traits and cardiovascular disease may involve excessive sympathetically mediated cardiovascular reactivity to psychological stress (Blascovich & Katkin, 1993; Kaplan et al., 1985; Krantz & Manuck, 1984; Miller et al., 1996; Obrist, 1981; Wright, Contrada, & Glass, 1985).

Several studies have demonstrated that sympathetic cardiovascular reactivity to stress is positively related to hostility and Type A behavior (e.g., Burns, Friedman, & Katkin, 1992; Contrada et al., 1982; Harbin, 1989; Larson & Langer, 1997; Lyness, 1993; Miller, Dolgoy, Friese, & Sita, 1998; Suls & Wan, 1993; Wright et al., 1985). These effects tend to be strongest for those measures that reflect β -adrenergic sympathetic effects on the heart, such as pre-ejection period, cardiac output, and systolic blood pressure. Moreover, research with humans (Smith, Limon, Gallo, & Ngu, 1996) and monkeys (Kaplan et al., 1985) has demonstrated that cardiovascular reactivity to stress is positively related to dominance. Finally, Rasmussen, Willingham, and Glover (1996) found that unstable self-esteem predicted heart rate and systolic blood pressure reactivity, and that cynical hostility predicted systolic and diastolic blood pressure reactivity during mental stress in men; further, the effects of cynical hostility on systolic blood pressure reactivity were mediated by unstable self-esteem. The relevance of the latter finding to narcissism stems from two lines of evidence: (a) narcissists tend to have unstable self-esteem (Rhodewalt et al., 1998), and threats to self-esteem engender hostility, anger, and aggression in narcissists (Bushman & Baumeister, 1998; Rhodewalt & Morf, 1998); and (b) narcissists use grandiosity and dominance to manage hostility and maintain self-esteem (Raskin et al., 1991). Thus, narcissism may be a fundamental personality dimension that accounts for relationships among self-esteem instability, hostility, dominance, and cardiovascular reactivity. Accordingly, it seems reasonable to predict a positive relationship between narcissism and sympathetic cardiovascular reactivity to stress.

Psychophysiological Analysis of Narcissism

The present experiment drew on these two separate lines of research to provide an initial psychophysiological analysis of narcissism. Several assumptions underlie this analysis. First, it assumes that normal personality and abnormal personality vary quantitatively along continuous dimensions, with personality disorders appearing at the extremes of those dimensions, so that differences between normal and abnormal personality variants are primarily quantitative rather than qualitative in nature (Cloninger, 1987; Cloninger, Svrakic, & Przybeck, 1993; Livesley, Jang, Jackson, & Vernon, 1993). Second, it assumes that many personality dimensions involve neurobiological components, and that normal and abnormal personality variants share these components (Cloninger, 1987; Cloninger et al., 1993; Fowles, 1980, 1988, 1994; Livesley et al., 1993; McBurnett, 1992; Siever & Davis, 1991). Third, it assumes that certain psychophysiological measures tap the neurobiological components of personality, either directly or indirectly (Fowles, 1980, 1988; Siever & Davis, 1991). Although there is a substantial genetic basis for many personality dimensions and relevant neurobiological systems and psychophysiological responses (Cloninger, 1987; Cloninger et al., 1993; Ditto, 1993; Fowles, 1994; Livesley et al., 1993; Lykken, Iacono, Haroian, McGue, & Bouchard, 1988; Siever & Davis, 1991), these assumptions do not preclude the role of environmental influences in nurturing and shaping neurobiological and psychophysiological features of personality (cf. Fowles, 1988; Livesley et al., 1993).

We exposed participants with high or low scores on the NPI to counterbalanced presentations of passive and active anticipatory coping tasks similar to those used in research on psychopathy (Hare, 1982; Hare et al., 1978; Ogloff & Wong, 1990; Tharp et al., 1980). In the passive coping task, the occurrence of an aversive stimulus at the end of a countdown period was unavoidable, whereas in the active coping task it was avoidable by a simple motor response. These active and passive conditions represent opposite poles of Obrist's (1981) active-passive coping dimension, which has figured so prominently in research on cardiovascular reactivity to stress. According to Obrist (1981), β -adrenergic sympathetic effects on the heart should predominate during active rather than passive coping, particularly when environmental uncertainty and behavioral uncertainty are high (Bongard, 1995; Brener, 1987; Contrada et al., 1982; Kelsey, 1991; Kelsey et al., 1999; Light & Obrist, 1980; Sherwood, Allen, Obrist, & Langer, 1986). Therefore, we expected greater β -adrenergic cardiac reactivity during active than passive coping, especially when the active coping task occurred first.

We measured skin conductance response (SCR) frequency, heart period (HP), and pre-ejection period (PEP) during the anticipatory countdown periods of each task, as well as state anxiety immediately after each task. SCR frequency is a sensitive measure of the sympathetic nervous system response to novel or threatening stimuli (Fowles, 1980; Katkin, 1965; Kelsey, 1991). Although SCR has been used in many studies of psychopathy, it has seldom been used in research on cardiovascular disease. HP, an inverse index of heart rate, reflects both sympathetic and parasympathetic nervous system influences on the heart, whereas PEP, an inverse index of myocardial contractile force, reflects primarily β -adrenergic sympathetic influences on the myocardium (Berntson et al., 1994; Cacioppo et al., 1994; Kelsey, 1991; Sherwood et al., 1986, 1990; Siegel et al., 1970). PEP has not been measured in studies of psychopathy, and has seen surprisingly little use in studies of cardiovascular disease, although it is widely accepted as one of the best indexes of sympathetic effects on the heart. Moreover, PEP is highly sensitive to circulating epinephrine (McCubbin, Richardson, Langer, Kizer, & Obrist, 1983; Mezzacappa, Kelsey, & Katkin, 1999), so it provides an excellent index of the sympathetic adrenomedullary activation that characterizes the fight/flight response.

Based on the psychopathy literature, we expected lower state anxiety and lower SCR reactivity during passive and active anticipatory coping in participants scoring high as compared to low on narcissism. Based on Hare's (1978, 1998) sensory rejection/active coping hypothesis of heart-rate reactivity in psychopathy, we would expect greater HP shortening (i.e., heart rate acceleration) in those scoring high as compared to low on narcissism. On the other hand, based on the proposed alternative hypothesis, we would expect greater parasympathetically mediated HP modulation, resulting perhaps in greater HP lengthening (i.e., heart rate deceleration) during anticipatory coping (cf. Brunia, 1993; Obrist, 1981), in those scoring high as compared to low on narcissism. The literatures on psychopathy and cardiovascular reactivity to stress suggest divergent predictions about the relationship between narcissism and PEP reactivity. Based on the electrodermal deficits associated with psychopathy and the assumption of a unified sympathetic response to stress, we would expect diminished PEP reactivity, as well as diminished SCR reactivity, during active and passive coping in those scoring high as compared to low on narcissism; that is, we would expect a generalized sympathetic hyporeactivity to stress in narcissism. In contrast, based on the cardiovascular reactivity lit-

erature and the possibility of a fractionated sympathetic response to stress (Kelsey, 1991; Kelsey et al., 1999; Wallin, 1981), we would expect heightened PEP reactivity in those scoring high as compared to low on narcissism, especially when the task was novel or required active coping. This latter prediction is also consistent with the notion of heightened somatic anxiety and exaggerated fight/flight responding in psychopathy (Fowles, 1994; Schalling, 1978).

Method

Participants

Seventy-seven undergraduate men (ages 18 to 33 years) in psychology classes at a southern public university completed the 40-item NPI (Raskin & Terry, 1988) for extra course credit. Subsequently, we recruited 20 men with NPI scores in the upper third (range from 21 to 32, $M = 25.0$, $SD = 3.7$) and 20 men with NPI scores in the lower third (range from 1 to 14, $M = 9.2$, $SD = 3.7$) of this initial sample distribution to participate individually in a psychophysiological study 4 to 10 weeks later. These participants received additional credit toward a course grade. We randomly assigned equal numbers of participants with high and low NPI scores to receive either the passive or active task first, thereby counterbalancing the order of task presentation.

Setting and Instrumentation

The experiment took place in a sound-attenuated, temperature-controlled, electrically shielded recording room measuring approximately $2.5 \times 3.5 \times 2.5$ m. The room contained an unobtrusive surveillance camera, a computer CRT monitor and numeric keypad, amplified audio speakers, and an intercom for communication and monitoring. Participants sat upright in a comfortable, upholstered chair throughout the experiment. An adjoining room housed all remaining equipment.

A laboratory computer controlled the pace of the experiment and the presentation of the aversive stimulus. In both tasks, the aversive stimulus was a 1 s, 100 dB(A), 1,000 Hz tone produced by a function generator (Wavetek Model 20) connected to an amplified speaker located directly in front of the participant. We verified the intensity of the tone with a sound level meter (Realistic Model 33-2050) before each experimental session. In the active coping task, the participant could prevent the occurrence of the aversive tone by pressing a key on a numeric keypad connected to the laboratory computer.

We measured HP and PEP from impedance cardiographic (ICG) and electrocardiographic (ECG) signals recorded continuously on a polygraph (Grass Model 7D) connected to an MS-DOS micro-computer system (see Kelsey & Guethlein, 1990; Kelsey et al., 1998). A Minnesota Impedance Cardiograph (Instrumentation for Medicine Model 304B) recorded basal transthoracic impedance (Z_0) and the first derivative of pulsatile changes in transthoracic impedance (dZ/dt) from a set of four disposable aluminum/mylar tape electrodes. A 4-mA, 100-kHz alternating current was passed through an outer pair of electrodes around the upper neck and abdomen, and the Z_0 and dZ/dt signals were recorded from an inner pair of electrodes around the base of the neck and the thoracic xiphisternal junction. The outer current electrodes were separated from the respective inner voltage electrodes by at least 3 cm (Sherwood et al., 1990). Given that the measurement of PEP is unaffected by ICG electrode configuration (Boomsma, de Vries, & Orlebeke, 1989; Sherwood, Royal, Hutcheson, & Turner, 1992), we minimized participant discomfort by placing the four elec-

trodes on the left side of the body between the front and back midline surfaces, rather than completely encircling the body. The resulting signals from these "half-band" electrodes were indistinguishable from those recorded with traditional "full-band" electrodes. We obtained the ECG from the internal signal provided by the Minnesota ICG (Kelsey et al., 1998, 1999). We measured SCRs from the thenar and hypothenar eminences of the nonpreferred hand via two Ag-AgCl electrodes attached with appropriate adhesive collars (0.8 cm^2 recording surface area) and 0.05 molar NaCl electrolyte conducting medium (Fowles et al., 1981). A constant 0.5 V signal was applied across the two electrodes, and SCRs were recorded at a sensitivity of $0.05 \mu\text{S}/\text{mm}$ using a high-gain preamplifier (Grass Model 7P1) with a time constant of 0.8 s.

We readministered the NPI at the beginning of the laboratory session to confirm the stability of the initial narcissism assessment. We also administered the state form of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970) at the beginning of the laboratory session and after each task. We modified the instructions for the posttask administrations, so that participants answered the questionnaire based on how they felt during the task they had just completed (Kelsey, 1991; Spielberger et al., 1970).

Procedure

A female experimenter met each participant at the laboratory, and provided general information about the nature of the experimental apparatus and procedures. She informed the participant that physiological responses would be measured both at rest and during perceptual-motor tasks, and that he could withdraw at any time without penalty. After giving informed consent, the participant washed his hands and completed a second administration of the NPI. The experimenter seated the participant in the recording room and attached the physiological recording electrodes to him. The experimenter left the recording room for approximately 10 min to adjust and calibrate the physiological equipment in an adjoining room while the participant completed the first administration of the STAI. After retrieving the completed STAI, the experimenter instructed the participant to rest quietly, move as little as possible, and await further instructions. The participant sat alone during the remaining experimental procedures, and received all ensuing instructions via audiotape.

The experiment consisted of an initial 10-min pretask baseline period and two counterbalanced task periods separated by a 5-min intertask baseline period. All participants received instructions to rest quietly and relax for each of the baseline periods. Each task period consisted of 5 trials that involved a 27-s visual countdown from 9 to 1, with each numeral appearing for 3 s in the center of the CRT display in front of the participant. In the passive coping task, the unavoidable aversive tone occurred 1 s after the last numeral disappeared from the display. In the active coping task, the participant could avoid the tone by pressing the center key on the numeric keypad during the 1-s delay after the last countdown numeral. An intertrial interval, varying randomly between 36 and 39 s, followed the occurrence or successful avoidance of each tone. The experimenter briefly reentered the recording room to administer the STAI immediately after each task period. After the last task period, the experimenter removed all recording devices from the participant and debriefed him.

Dependent Measures

The synchronized ECG, dZ/dt , and Z_0 recordings were saved directly to magnetic disk, and scored subsequently using an inter-

active MS-DOS microcomputer system described in detail by Kelsey and Guethlein (1990) and Kelsey et al. (1998). A trained operator scored the digitized ECG and ICG signals on a beat-to-beat basis, and then ensemble averaged them with reference to the ECG R-wave over consecutive 9-s sampling intervals (Kelsey & Guethlein, 1990; Kelsey et al., 1998; Muzi et al., 1985) for the first 27 s of each of the last 2 min of the pretask and intertask baseline periods, and for the 27-s anticipatory countdown phase of each trial of the two task periods. Partial cardiac cycles were excluded from all averages, and occasional missing data were replaced by interpolation. We derived averaged beat-to-beat values for HP (ms), defined as the interval between the peaks of successive ECG R-waves, and ensemble averaged values for PEP (ms), defined as the interval between the peak of the ECG R-wave and the corresponding dZ/dt B-wave (Kelsey, 1991; Kelsey & Guethlein, 1990; Kelsey et al., 1998, 1999; Sherwood et al., 1990; Siegel et al., 1970). A rater blind to group status manually scored the number of nonspecific SCRs exceeding a criterion of $0.05 \mu\text{S}$ during the same sampling intervals of the last 2 min of the baseline periods and each trial of the task periods (Kelsey, 1991).

Results

Self-Report Measures

The NPI scores from the second test administration at the laboratory session ranged from 13 to 34 for the high NPI group ($M = 22.3$, $SD = 5.4$) and from 1 to 18 for the low NPI group ($M = 7.8$, $SD = 3.9$). The score of one participant in the high NPI group dropped into the low range at the second test administration; however, excluding this participant had no impact on the results, so he was retained in the high NPI group. A 2 (NPI Groups) \times 2 (Task Orders) \times 2 (Test Administrations) MANOVA confirmed that NPI scores were consistently higher overall in the high NPI group than in the low NPI group, $F(1,36) = 145.20$, $p < .0005$. The groups showed an equivalent decline in NPI scores across test administrations, $F(1,36) = 17.85$, $p < .0005$, perhaps due to changes over time or differences in setting (group versus individual administration) between testing sessions. There were no significant effects of task order on NPI scores (all $p > .30$), indicating successful random assignment of participants to counterbalanced task orders. Finally, the retest reliability of the NPI was excellent: The random effects intraclass correlation coefficient (Shrout & Fleiss, 1979), which takes into account the discrepancy between repeated measures as well as the rank order of scores, was .91. These results support the reliability of the original assignment of participants into high and low NPI groups.

A 2 (NPI Groups) \times 2 (Task Orders) \times 3 (Administrations) MANOVA of STAI scores revealed that state anxiety was significantly lower overall in the high NPI group ($M = 29.2$) than in the low NPI group ($M = 34.7$), $F(1,36) = 5.91$, $MSE = 151.8$, $p < .02$, $r = .38$. The mean of the latter group was comparable to that of normative college samples (Spielberger et al., 1970). There were no other significant effects for state anxiety.

Physiological Measures

Equipment failure resulted in lost physiological data for one participant in the high NPI group and one participant in the low NPI group, and poor dZ/dt signal quality precluded accurate scoring of PEP for another participant in the high NPI group. To equalize the cell sizes for all physiological analyses, we randomly deleted one participant from the low NPI group, leaving a final sample of 36 participants. Preliminary analyses indicated no significant group

differences in physiological activity over 9-s sampling intervals, so we averaged these intervals for each baseline minute and task trial.

Baseline activity. A series of 2 (NPI Groups) \times 2 (Task Orders) \times 2 (Periods) \times 2 (Minutes) ANOVAs of physiological activity during the last 2 min of the baseline periods revealed no significant effects (all $p > .09$). Thus baseline physiological activity was equivalent among groups and stable over time (see Table 1).

Task reactivity. We assessed physiological reactivity to the tasks for each measure by subtracting average values for the last minute of each baseline period from average values for each trial of the corresponding task period. Given the time-ordered nature of the experimental design, we used polynomial trend analyses, rather than general repeated measures analyses, to evaluate physiological reactivity over tasks and trials. We used a weighted Bonferroni adjustment procedure (Rosenthal & Rubin, 1983) to preserve a familywise error rate of 5% for tests of the polynomial contrasts constituting the main and interaction effects involving trials, weighting the polynomial trend components in reverse order to give lower order trends priority over higher order trends (Kelsey, 1991; Kelsey et al., 1999). Thus, the linear trend over trials was assigned a weight of four and tested at $\alpha = .02$, the quadratic trend over trials was assigned a weight of three and tested at $\alpha = .015$, and so forth.

A 2 (NPI Groups) \times 2 (Task Orders) \times 2 (Tasks) \times 5 (Trials) univariate trend analysis revealed a tendency toward less overall SCR reactivity in the high NPI group ($M = 0.6$) than in the low NPI group ($M = 0.8$), $F(1,32) = 3.41$, $MSE = 1.4$, $p < .075$, $r = .31$. As Figure 1 indicates, however, this tendency varied as a function of task order, $F(1,32) = 9.90$, $p < .004$, $r = .49$, with significant group differences emerging only when the active task occurred first. Likewise, although active coping elicited greater SCR reactivity than passive coping, $F(1,32) = 12.34$, $MSE = 2.2$, $p < .001$, $r = .53$, this effect varied with task order, $F(1,32) = 11.69$, $p < .002$, $r = .52$, arising primarily when the active task occurred first. These task effects did not vary as a function of narcissism. There were significant linear and quadratic trends in SCR reactivity over trials ($p < .005$), and the overall linear trend varied as a function of narcissism, $F(1,32) = 6.01$, $MSE = 0.8$, $p < .02$, $r = .40$. As Figure 2 shows, SCR reactivity habituated more

Table 1. Means (SDs) of Physiological Measures During Baseline Periods in Groups with High or Low Scores on the Narcissistic Personality Inventory (NPI)

Measure	Low NPI (n = 18)		High NPI (n = 18)	
	Pre-Passive	Pre-Active	Pre-Passive	Pre-Active
SCR (freq)	0.48 (0.70)	0.33 (0.50)	0.56 (0.84)	0.48 (0.73)
PEP (ms)	84.4 (14.0)	84.8 (13.2)	87.6 (10.5)	87.9 (11.3)
HP (ms)	869.2 (96.7)	846.9 (94.1)	809.5 (125.7)	818.0 (115.4)

Note: The values represent the last minute of the respective pretask baseline periods. SCR = skin conductance response frequency; PEP = prejection period; HP = heart period.

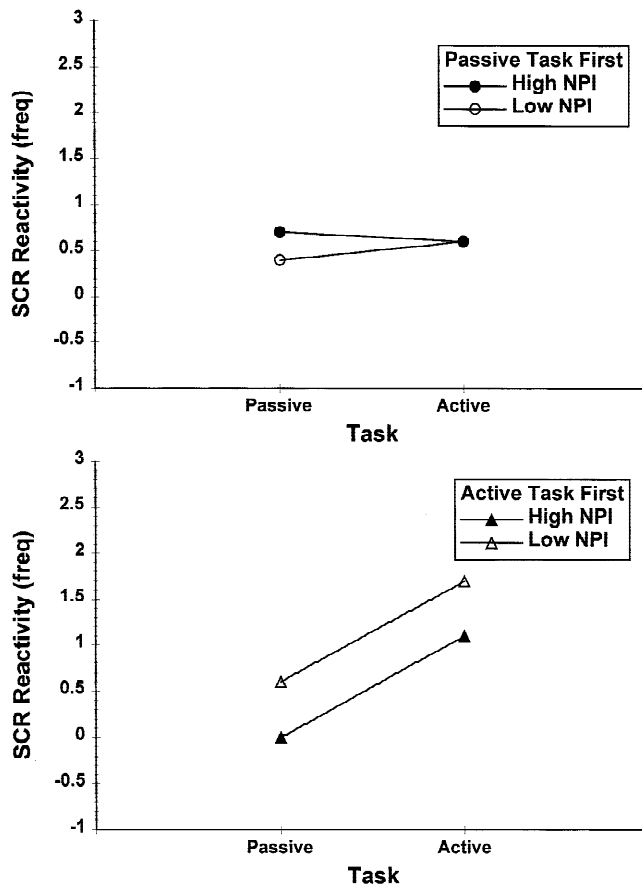


Figure 1. Mean skin conductance response (SCR) reactivity (changes from baseline) during passive and active coping tasks as a function of task order in groups with high or low scores on the Narcissistic Personality Inventory (NPI).

rapidly in the high NPI group than in the low NPI group, regardless of coping condition.

As PEP and HP reflect interrelated aspects of the cardiovascular system, we analyzed these measures together via MANOVA, using Wilks' Λ criterion for multivariate significance testing. We assessed the relative contribution of each cardiac measure to a significant multivariate effect with univariate F tests and F -to-remove tests (Huberty & Morris, 1989), using modified Bonferroni-adjusted probability levels to assess statistical significance in each case (Simes, 1986). A 2 (NPI Groups) \times 2 (Task Orders) \times 2 (Tasks) \times 5 (Trials) multivariate trend analysis revealed a significant effect of narcissism on overall cardiac reactivity to the tasks, Wilks' $\Lambda = .711$, $F(2,31) = 6.29$, $p < .005$. Overall, PEP shortened significantly more in the high NPI group ($M = -1.8$ ms) than in the low NPI group ($M = 0.1$ ms), $F(1,32) = 6.67$, $MSE = 45.2$, $p < .015$, $r = .42$, and HP lengthened significantly more in the high NPI group ($M = 30.5$ ms) than in the low NPI group ($M = -5.4$ ms), $F(1,32) = 8.38$, $MSE = 13,857$, $p < .007$, $r = .46$. F -to-remove tests indicated that HP contributed primarily to this effect, $F(1,31) = 5.07$, $p < .04$, with a marginal residual contribution from PEP, $F(1,31) = 3.53$, $p < .07$.

The active coping task elicited greater cardiac reactivity than the passive coping task, Wilks' $\Lambda = .722$, $F(2,31) = 5.97$, $p < .006$, due to significantly greater PEP shortening during active than passive coping, $F(1,32) = 12.32$, $MSE = 35.6$, $p < .001$, $r = .53$.

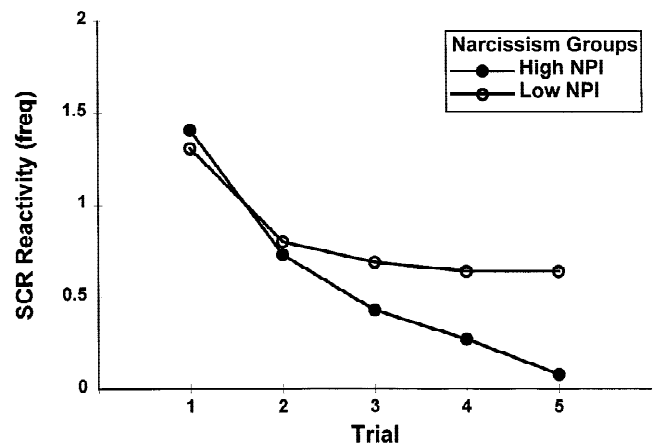


Figure 2. Mean skin conductance response (SCR) reactivity (changes from baseline) over task trials in groups with high or low scores on the Narcissistic Personality Inventory (NPI).

However, a significant Order \times Task interaction (Wilks' $\Lambda = .778$, $F(2,31) = 4.42$, $p < .02$) indicated that this effect on PEP emerged primarily when the active task occurred first, $F(1,32) = 9.09$, $p < .005$, $r = .47$. More important, there was a significant Narcissism \times Order \times Task interaction, Wilks' $\Lambda = .789$, $F(2,31) = 4.15$, $p < .025$. As Figure 3 shows, the differential impact of active versus passive coping on PEP reactivity occurred only in the low NPI group; the high NPI group showed the greatest PEP shortening during the first task presentation, regardless of coping condition, $F(1,32) = 7.02$, $p < .012$, $r = .42$. There were no significant differences in HP reactivity as a function of active versus passive coping (all $p > .25$). F -to-remove tests confirmed the unique contribution of PEP to these task effects, all $F(1,31) > 6.85$, $p < .015$.

There were significant linear and quadratic trends in cardiac reactivity over trials ($p < .001$), regardless of coping condition. However, only the quadratic trend varied as a function of narcissism, Wilks' $\Lambda = .647$, $F(2,31) = 8.47$, $p < .001$. Both PEP and HP contributed significantly to this multivariate effect, $F(1,32) = 7.97$ and 7.79 , $MSE = 3.3$ and $1,554$, respectively, both $p < .009$, both $r > .44$. F -to-remove tests confirmed the independent contributions of PEP and HP to this effect, $F(1,31) = 7.55$ and 7.38 , respectively, $p < .015$. As Figure 4 shows, the attenuation of PEP reactivity over trials was slower and the quadratic trend in HP reactivity was stronger in the high NPI group than in the low NPI group.

Discussion

These data link narcissism to a complex psychophysiological profile involving diminished SCR reactivity, heightened PEP reactivity, and enhanced cardiac deceleration in anticipation of an aversive stimulus. For all three measures, both the overall magnitude and the pattern of psychophysiological reactivity over trials varied as a function of narcissism. This psychophysiological profile resembles certain psychophysiological characteristics of psychopathy and psychological predictors of heart disease, thus providing empirical support for theoretical links between narcissism and each of these areas.

Active Versus Passive Coping

Consistent with Obrist's (1981) concept of active versus passive coping and previous research (Bongard, 1995; Contrada et al.,

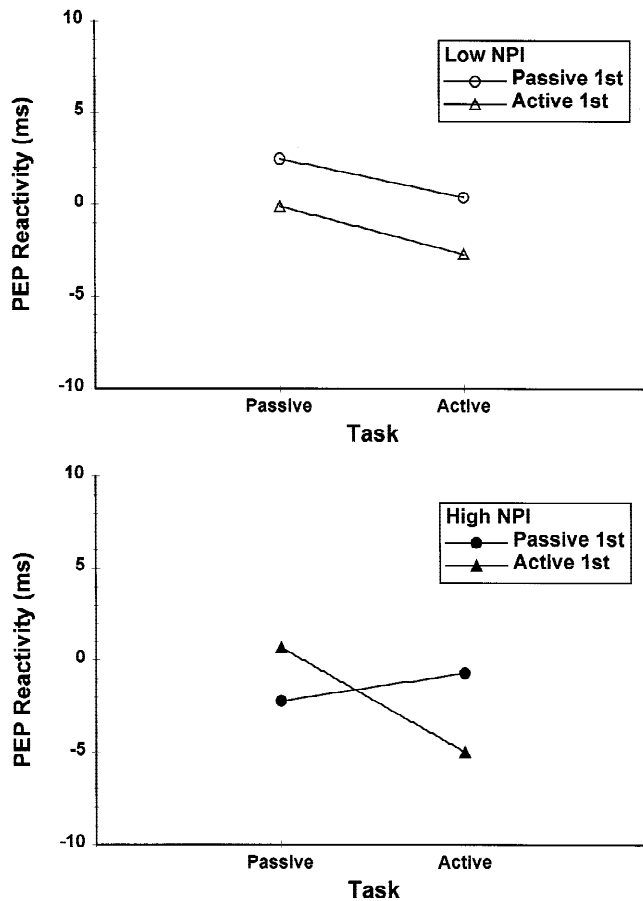


Figure 3. Mean prejection period (PEP) reactivity (changes from baseline) during passive and active coping tasks as a function of task order in groups with high or low scores on the Narcissistic Personality Inventory (NPI).

1982; Kelsey, 1991; Light & Obrist, 1980; Obrist, 1981), the active coping task elicited greater anticipatory shortening of PEP than did the passive coping task, indicating that β -adrenergic sympathetic effects on the heart were greater during active than passive coping. As in some prior studies (Hare, 1982; Kelsey, 1991), the active task also elicited greater SCR reactivity than did the passive task, suggesting a general tendency toward greater sympathetic reactivity during active than passive coping. No such effects emerged for HP, which lengthened similarly during both tasks, suggesting parasympathetic rather than sympathetic mediation of anticipatory heart rate reactivity (Obrist, 1981). As expected, however, the differential impact of active versus passive coping on PEP and SCR reactivity emerged mainly when the active task occurred first. Thus, the most pronounced sympathetic effects on the heart and sweat glands occurred during active coping when the environment and aversive stimulus were relatively novel (Kelsey, 1991; Kelsey et al., 1999; Light & Obrist, 1980; Obrist, 1981).

Diminished Anxiety and Electrodermal Reactivity

Consistent with predictions from the psychopathy literature, narcissism was associated with low state anxiety and diminished SCR reactivity in anticipation of an aversive stimulus. Anticipatory SCR reactivity was lower overall in the high NPI group than in the low NPI group when the active coping task occurred first. Further-

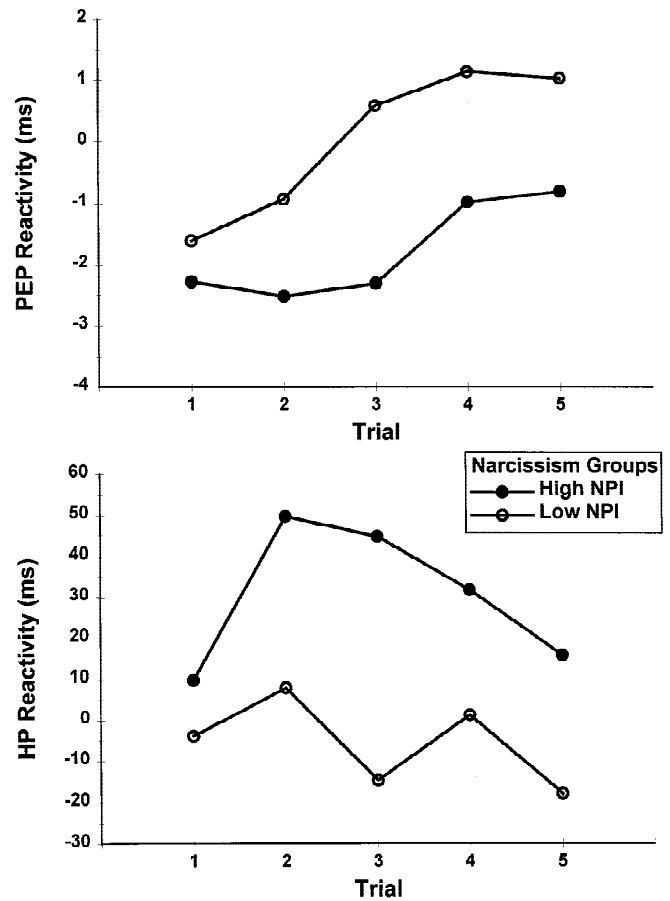


Figure 4. Mean prejection period (PEP) and heart period (HP) reactivity (changes from baseline) over task trials in groups with high or low scores on the Narcissistic Personality Inventory (NPI).

more, although the two narcissism groups showed similar SCR reactivity during the early trials of both tasks, the high NPI group exhibited faster habituation of SCR reactivity during later trials. These deficits in SCR reactivity and state anxiety suggest that narcissism may involve an impairment in the processing of information about aversive events, perhaps because of a deficit in central systems that mediate anticipatory anxiety and behavioral inhibition (Cloninger, 1987; Depue & Spont, 1986; Fowles, 1980, 1988, 1994; Graeff, Guimaraes, de Andrade, & Deakin, 1996; Gray, 1987; Hare, 1978, 1998; McBurnett, 1992). Diminished activity in such systems could account for several characteristics of narcissism besides low anxiety, including impulsivity, impatience, sensation seeking, and aggression. Moreover, deficits in the processing of aversive or distressing information may lead to a failure to recognize and respond appropriately to distress in others, thereby contributing to the characteristic lack of empathy in narcissism. Although the SCR deficits associated with narcissism were less pronounced than those generally associated with psychopathy (cf. Fowles, 1980; Hare, 1978), this discrepancy is actually consistent with Kernberg's (1989) conceptualization of a narcissistic continuum. According to Kernberg, psychopathy emanates from extreme or pathological narcissism, which was, we hope, underrepresented in our college sample. It may be that the profound SCR deficits commonly found in psychopathy and antisocial personality disorder are actually extreme manifestations of pathological narcissism.

Enhanced Cardiac Deceleration and Vigilant Readiness

Narcissism was associated with enhanced heart-rate deceleration in anticipation of an aversive stimulus. The high NPI group exhibited greater anticipatory lengthening of HP as compared to the low NPI group, especially during the middle and later trials of the two tasks. This sort of anticipatory cardiac deceleration is generally mediated by the parasympathetic nervous system (Obrist, 1981), and typically occurs during selective attention and motor preparation (Brunia, 1993). Thus, these results suggest greater parasympathetic modulation of heart rate, as well as greater motor preparation, selective attention, and vigilance, in those scoring high as compared to low on narcissism. These results contrast with several findings of enhanced cardiac acceleration in psychopaths (Hare, 1978; Hare & Craigen, 1974; Hare et al., 1978), as well as with Hare's (1978) sensory rejection/active coping hypothesis of heart rate reactivity in psychopathy. Directional differences in anticipatory heart rate reactivity and associated differences in attention, information processing, and motor preparation may distinguish narcissists from psychopaths. On the other hand, the cardiac deceleration associated with narcissism is consistent with some findings from the psychopathy literature (Hare, 1982; Ogloff & Wong, 1990), as well as with the proposed alternative hypothesis of enhanced parasympathetic modulatory control of heart-rate reactivity in psychopaths.

Enhanced Myocardial Reactivity and Mobilization of Effort

Narcissism was characterized by enhanced PEP reactivity in anticipation of an aversive stimulus. The anticipatory shortening of PEP was greater in the high NPI group than in the low NPI group, particularly during the initial task exposure. Although the expected differential effects of active versus passive coping on PEP reactivity occurred in the low NPI group, the high NPI group showed the greatest PEP reactivity during the first task exposure, regardless of coping condition. Again, group differences in reactivity varied over trials. The two narcissism groups showed similar PEP reactivity during the initial trials of the two tasks, but the high NPI group exhibited slower habituation of PEP reactivity during subsequent trials. These results point to a positive relationship between β -adrenergic cardiac reactivity to stress and narcissism, and suggest a stronger fight/flight response (Fowles, 1994; Schalling, 1978) and a greater mobilization of effort in preparation for action in those scoring high as compared to low on narcissism (Kelsey, 1991; Kelsey et al., 1999; Obrist, 1981; Pribram & McGuinness, 1975; Sherwood et al., 1986). Moreover, these results suggest that narcissism may involve an indiscriminate and sustained mobilization of effort under conditions of environmental uncertainty and threat, regardless of actual coping demands or the availability of an effective coping response. Enhanced fight/flight reactivity and mobilization of effort could contribute to several characteristics of narcissism, including dominance, competitiveness, hostility, antagonism, impulsivity, and aggression.

Although our findings for PEP have no counterpart in the psychopathy literature, they are consistent with the notion of heightened somatic anxiety and exaggerated fight/flight responding in psychopathy (Fowles, 1994; Schalling, 1978). Moreover, the PEP results are generally consistent with associations between β -adrenergic cardiovascular hyperreactivity to stress and various psychological predictors of heart disease that are characteristic of narcissism (e.g., dominance, hostility, and Type A behavior), suggesting that narcissism may be an essential dimension underlying these traits. Taken together, the SCR and PEP findings argue against the necessity of a unified sympathetic response to stress (cf. Kelsey,

1991; Wallin, 1981) or a generalized deficit in sympathetic reactivity in narcissism.

Autonomic Influences in Narcissism

Although the group differences in SCR reactivity and cardiac deceleration are readily interpretable in terms of autonomic influences, the group differences in PEP reactivity might reflect autoregulatory influences involving preload (left ventricular filling) and afterload (aortic diastolic pressure) rather than sympathetic-adrenomedullary influences. The effects of narcissism on PEP reactivity to the initial task exposure and PEP reactivity over trials remained significant after controlling for HP reactivity in *F*-to-remove tests, so it is unlikely that these effects were attributable to preload and the Frank-Starling mechanism (cf. Kelsey et al., 1999; Obrist, Light, James, & Strogatz, 1987; Sherwood et al., 1990). Even the main effect of narcissism on overall PEP reactivity remained marginally significant after controlling for overall HP reactivity, suggesting some residual sympathetic influence. We cannot be certain about the effects of afterload on PEP reactivity because blood pressure measurements were not available in this study (Kelsey et al., 1999; Obrist et al., 1987; Sherwood et al., 1990). However, a contribution of afterload to the group differences in PEP reactivity would require a drop in blood pressure in the high NPI group during anticipatory stress, which seems unlikely given the aversive nature of the tasks used in this study (Obrist, 1981). Although it might be tempting to suggest that a hypotensive response accompanied the SCR hyporeactivity associated with narcissism, the best available evidence indicates that there is little or no covariation in the sympathetic control of blood pressure and electrodermal activity (Wallin, 1981). Furthermore, research indicates that preload and afterload exert negligible effects on PEP when posture remains constant (Berntson et al., 1994; Cacioppo et al., 1994; Obrist et al., 1987), as in the present experiment. Therefore, it seems more likely that β -adrenergic sympathetic effects on the heart, rather than preload or afterload, were primarily responsible for most of the group differences in PEP reactivity during anticipatory stress.

The convergent pattern of SCR and PEP reactivity during active versus passive coping suggests a unified sympathetic nervous system response to stress, whereas the divergent pattern of SCR hyporeactivity and PEP hyperreactivity associated with narcissism suggests a fractionated sympathetic response (Kelsey, 1991; Obrist, 1981; Wallin, 1981). This sympathetic response fractionation occurred in the context of apparent parasympathetic influences on HP, suggesting that narcissism may involve coactivation of autonomic influences on the heart (Berntson et al., 1994; Cacioppo et al., 1994). Nevertheless, describing this complex psychophysiological pattern in terms of sympathetic response fractionation and autonomic coactivation does little to elucidate its connections with the psychological and behavioral characteristics of narcissism. Fortunately, there are several theoretical models that may explain these connections.

Theoretical Models

Based on Gray's motivational theory (Gray, 1987), Fowles (1980, 1988, 1994) has proposed that low anxiety and diminished SCR reactivity in anticipation of aversive events arise from a deficiency in the processing of aversive information by the BIS. Accordingly, our data on state anxiety and SCR reactivity suggest that narcissism may involve deficiencies in behavioral inhibition and aversive information processing similar to those ascribed to psychopathy. Unfortunately, Fowles' model cannot account for our cardiovas-

cular data. His model gives no consideration to PEP, but proposes that heart rate (and its reciprocal, heart period) reflects the processing of appetitive information by the BAS. As the appetitive BAS presumably interacts with the aversive BIS in a reciprocal, antagonistic manner, a deficient BIS may release activity in the BAS, resulting in cardiac acceleration (Fowles, 1980, 1988). However, cardiac deceleration, rather than acceleration, accompanied the SCR hyporeactivity that we observed in narcissism. Although PEP shortening might indicate an increase in BAS activity, it seems more likely that it reflects activity in the unconditioned fight/flight system described by Gray (1987), especially because stimulus and task novelty were more important than coping requirements in determining PEP reactivity in narcissists.

The Pribram and McGuinness (1975) model of arousal, activation, and effort appears to provide a better explanation of the psychophysiological response pattern that emerged for narcissism. According to this model, an arousal system regulates externally triggered, reflexive responses to environmental stimuli, including aversive stimuli. The orienting response is the fundamental psychophysiological reflex of the arousal system. This system includes the same neural circuitry as the BIS described by Gray (1987; Fowles, 1994; McBurnett, 1992). Thus the SCR hyporeactivity that we observed in narcissism may reflect a deficit in arousal and reflexive reactivity to external stimulation. In contrast, an activation system regulates internally generated selective attention, vigilance, and tonic motor readiness. Pribram and McGuinness (1975) suggested that tonic heart-rate deceleration, which is typically observed during selective attention and motor preparation (Brunia, 1993; Obrist, 1981), is the fundamental psychophysiological characteristic of the activation system. Thus the enhanced cardiac deceleration that we observed in narcissism may reflect heightened activation and internally generated attention. Finally, an effort system regulates the arousal and activation systems by coordinating and integrating information about environmental stimuli and behavioral responses. Consequently, this system is particularly responsive to environmental and task novelty (Kelsey et al., 1999). The fight/flight response is the fundamental reflex associated with the effort system. Thus, the PEP hyperreactivity that we observed in narcissism may reflect heightened effort. In sum, the Pribram and McGuinness model suggests that the SCR hyporeactivity, enhanced cardiac deceleration, and PEP hyperreactivity that we found in narcissism reflect respectively hypoarousal, hyperactivation, and heightened effort during the processing of aversive information.

Although the psychophysiological characteristics of narcissism may arise from multiple neurobehavioral systems, a deficit in central serotonergic inhibitory control may form the cornerstone of the narcissistic psychobiological profile. Several psychobiological theories of personality and psychopathology emphasize the importance of a central serotonergic system in the inhibitory control of behavior (Cloninger, 1987; Depue & Spoont, 1986; Fowles, 1994; Graeff et al., 1996; Gray, 1987; McBurnett, 1992; Pribram & McGuinness, 1975). Notwithstanding minor differences in terminology and emphasis, these theories generally postulate that a central serotonergic system promotes anticipatory anxiety and aversive conditioning while exerting inhibitory control over two other systems: (a) a central dopaminergic system that promotes behavioral activation (Cloninger, 1987; Cloninger et al., 1993; Depue & Spoont, 1986; Fowles, 1994; Gray, 1987) and tonic motor readiness (Pribram & McGuinness, 1975), and (b) a central neurohormonal system that controls the fight/flight response (Fowles, 1994; Graeff et al., 1996; McBurnett, 1992; Pribram & McGuinness, 1975). In

terms of the two major models described above, these three central neural systems correspond respectively to Gray's behavioral inhibition, behavioral activation, and fight/flight systems (Fowles, 1994; Gray, 1987; McBurnett, 1992), and to Pribram's arousal, activation, and effort systems (Pribram & McGuinness, 1975).

Several lines of evidence support the relevance of this general model to the psychophysiological characteristics of narcissism. First, Graeff et al. (1996) demonstrated that pharmacological manipulations of central serotonergic function in rats and humans altered anxiety and SCR reactivity in anticipation of aversive events, as well as aspects of the fight/flight response. Although they did not evaluate cardiovascular function, their theoretical framework suggests that impairments of serotonergic function should enhance PEP reactivity by disinhibiting the fight/flight response. Second, Quirk and Sourkes (1977) demonstrated that a decrease in central serotonergic neurotransmission coupled with an increase in central dopaminergic neurotransmission promoted adrenomedullary synthesis of epinephrine, the essential hormone of the fight/flight response and a major determinant of PEP reactivity (McCubbin et al., 1983; Mezzacappa et al., 1999). Third, recent studies have linked aggression in monkeys (Kyes, Botchin, Kaplan, Manuck, & Mann, 1995) and aggression, impulsivity, and hostility in men (Manuck et al., 1998) to reduced central serotonergic function, as indexed indirectly by low prolactin responses to fenfluramine challenge. Thus, converging lines of evidence suggest that diminished central serotonergic function may contribute fundamentally to important psychophysiological and behavioral characteristics of narcissism and psychopathy. Moreover, the impact of reduced central serotonergic function may extend to cardiovascular disease as well, given recent evidence of an inverse relationship between the prolactin response to fenfluramine challenge and resting blood pressure in humans (Muldoon et al., 1998).

Conclusions

Consistent with factor analyses of self-report data (Emmons, 1984; Raskin & Terry, 1988), our psychophysiological data indicate that narcissism is a multidimensional construct. These dimensions may be grounded in a limited set of interacting neurobehavioral systems (Cloninger, 1987; Cloninger et al., 1993; Fowles, 1994; Gray, 1987; McBurnett, 1992; Pribram & McGuinness, 1975). Indeed, many of the psychophysiological and behavioral characteristics of narcissism may hinge on a fundamental deficit in central serotonergic function. Although recent experimental work on narcissism has concentrated on demonstrating psychosocial and behavioral manifestations of narcissistic traits within interpersonal contexts involving threats to self-esteem (Bushman & Baumeister, 1998; Kernis & Sun, 1994; Morf & Rhodewalt, 1993; Rhodewalt & Morf, 1998), our data indicate that psychophysiological manifestations of narcissistic traits are apparent even in a relatively simple personal context involving the threat of an impending aversive event.

Our data provide psychophysiological support for theoretical links between narcissism and psychopathy, and between narcissism and psychological predictors of heart disease. Future research on psychopathy and antisocial personality disorder may benefit from a functional analysis of the role of narcissism in these disorders (Harpur et al., 1989; Patrick, 1994; Zagon & Jackson, 1994), as well as from the addition of PEP as an index of sympathetic cardiac reactivity to aversive stimuli. Moreover, in line with recent recommendations (Lenfant, 1998), future cardiovascular research may benefit by considering narcissism as an underlying personality dimension that may account for the aggregation of various

psychosocial risk factors for cardiovascular disease. Bushman and Baumeister (1998) and Rhodewalt and Morf (1998) have demonstrated that experimental manipulations involving threats to self-esteem evoke hostility, anger, and aggression in narcissists. These experimental manipulations are similar to many of the harassment manipulations used in research on cardiovascular reactivity to stress. Moreover, Raskin et al. (1991) have demonstrated that narcissistic grandiosity and dominance moderate hostility and bolster self-esteem. Thus, narcissism may provide a functional context for

understanding the role of factors such as hostility and dominance in the development of cardiovascular disease. Finally, recent research by Pine et al. (1996) links risk factors for antisocial behavior and delinquency to those for cardiovascular disease. Our data suggest that narcissism may provide a foundation for this linkage. Thus, research on narcissism may elucidate important similarities and differences in the pathophysiological processes and clinical outcomes associated with psychopathy and psychological predictors of cardiovascular disease.

REFERENCES

- Akhtar, S., & Thompson, J. A. (1982). Overview: Narcissistic personality disorder. *American Journal of Psychiatry*, *139*, 12–20.
- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Berntson, G. G., Cacioppo, J. T., Binkley, P. F., Uchino, B. N., Quigley, K. S., & Fieldstone, A. (1994). Autonomic cardiac control. III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. *Psychophysiology*, *31*, 599–608.
- Blackburn, R., & Coid, J. W. (1998). Psychopathy and the dimensions of personality disorder in violent offenders. *Personality and Individual Differences*, *25*, 129–145.
- Blascovich, J., & Katkin, E. S. (1993). *Cardiovascular reactivity to psychological stress and disease*. Washington, DC: American Psychological Association.
- Bongard, S. (1995). Mental effort during active and passive coping: A dual task analysis. *Psychophysiology*, *32*, 242–248.
- Boomsma, D. I., de Vries, J., & Orlebeke, J. F. (1989). Comparison of spot and band impedance cardiogram electrodes across different tasks. *Psychophysiology*, *26*, 695–699.
- Booth-Kewley, S., & Friedman, H. S. (1987). Psychological predictors of heart disease: A quantitative review. *Psychological Bulletin*, *101*, 343–362.
- Bradlee, P. M., & Emmons, R. A. (1992). Locating narcissism within the interpersonal circumplex and the five factor model. *Personality and Individual Differences*, *13*, 821–830.
- Brener, J. (1987). Behavioural energetics: Some effects of uncertainty on the mobilization and distribution of energy. *Psychophysiology*, *24*, 499–512.
- Brunia, C. H. M. (1993). Waiting in readiness: Gating in attention and motor preparation. *Psychophysiology*, *30*, 327–339.
- Burns, J. W., Friedman, R., & Katkin, E. S. (1992). Anger expression, hostility, anxiety, and patterns of cardiac reactivity to stress. *Behavioral Medicine*, *18*, 71–78.
- Bushman, B. J., & Baumeister, R. F. (1998). Threatened egotism, narcissism, self-esteem, and direct and displaced aggression: Does self-love or self-hate lead to violence? *Journal of Personality and Social Psychology*, *75*, 219–229.
- Cacioppo, J. T., Berntson, G. G., Binkley, P. F., Uchino, B. N., Quigley, K. S., & Fieldstone, A. (1994). Autonomic cardiac control. II. Non-invasive indices and basal response as revealed by autonomic blockades. *Psychophysiology*, *31*, 586–598.
- Cleckley, H. (1976). *The mask of sanity* (5th ed.). St. Louis, MO: Mosby.
- Cloninger, C. R. (1987). A systematic method for the clinical description and classification of personality variants. *Archives of General Psychiatry*, *44*, 573–588.
- Cloninger, R. C., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry*, *50*, 975–990.
- Contrada, R. J., Glass, D. C., Krakoff, L. R., Krantz, D. S., Kehoe, K., Isecke, W., Collins, C., & Elting, E. (1982). Effects of control over aversive stimulation and Type A behavior on cardiovascular and plasma catecholamine responses. *Psychophysiology*, *19*, 408–419.
- Depue, R. A., & Spoont, M. R. (1986). Conceptualizing a serotonin trait: A behavioral dimension of constraint. *Annals of the New York Academy of Sciences*, *487*, 47–62.
- Ditto, B. (1993). Familial influences on heart rate, blood pressure, and self-report anxiety responses to stress: Results from 100 twin pairs. *Psychophysiology*, *30*, 635–645.
- Emmons, R. A. (1984). Factor analysis and construct validity of the Narcissistic Personality Inventory. *Journal of Personality Assessment*, *48*, 291–300.
- Emmons, R. A. (1987). Narcissism: Theory and measurement. *Journal of Personality and Social Psychology*, *52*, 11–17.
- Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology*, *17*, 87–104.
- Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. *Psychophysiology*, *25*, 373–391.
- Fowles, D. C. (1994). A motivational theory of psychopathology. In W. D. Spaulding (Ed.), *Nebraska symposium on motivation: Integrated views of motivation and emotion* (Vol. 41, pp. 181–238). Lincoln, NE: University of Nebraska Press.
- Fowles, D. C., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology*, *18*, 232–239.
- Fukunishi, I., Hattori, M., Nakamura, H., Li, K., Hua, Z. Q., & Kratz, T. S. (1996). Relationships between Type A behavior, narcissism, and maternal closeness for college students in Japan, the United States of America, and the People's Republic of China. *Psychological Reports*, *78*, 939–944.
- Graeff, F. G., Guimaraes, F. S., de Andrade, T. G. C. S., & Deakin, J. F. W. (1996). Role of 5-HT in stress, anxiety, and depression. *Pharmacology, Biochemistry and Behavior*, *54*, 129–141.
- Gray, J. A. (1987). Perspectives on anxiety and impulsivity: A commentary. *Journal of Research in Personality*, *21*, 493–509.
- Harbin, T. J. (1989). The relationship between the Type A behavior pattern and physiological responsivity: A quantitative review. *Psychophysiology*, *26*, 110–119.
- Hare, R. D. (1978). Electrodermal and cardiovascular correlates of psychopathy. In R. D. Hare & D. Schalling (Eds.), *Psychopathic behavior: Approaches to research* (pp. 107–144). Chichester, England: Wiley.
- Hare, R. D. (1982). Psychopathy and physiological activity during anticipation of an aversive stimulus in a distraction paradigm. *Psychophysiology*, *19*, 266–271.
- Hare, R. D. (1998). Psychopathy, affect and behavior. In D. J. Cooke, R. D. Hare, & A. E. Forth (Eds.), *Psychopathy: Theory, research and implications for society* (pp. 105–137). Dordrecht, The Netherlands: Kluwer Academic Publishers.
- Hare, R. D., & Craigen, D. (1974). Psychopathy and physiological activity in a mixed-motive game situation. *Psychophysiology*, *11*, 197–206.
- Hare, R. D., Frazelle, J., & Cox, D. N. (1978). Psychopathy and physiological responses to threat of an aversive stimulus. *Psychophysiology*, *15*, 165–172.
- Harpur, T. J., Hare, R. D., & Hakstian, A. R. (1989). Two-factor conceptualization of psychopathy: Construct validity and assessment implications. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, *1*, 6–17.
- Houston, B. K., Babyak, M. A., Chesney, M. A., Black, G., & Ragland, D. R. (1997). Social dominance and 22-year all-cause mortality in men. *Psychosomatic Medicine*, *59*, 5–12.
- Huberty, C. J., & Morris, J. D. (1989). Multivariate analysis versus multiple univariate analyses. *Psychological Bulletin*, *105*, 302–308.
- Kaplan, J. R., Manuck, S. B., Clarkson, T. B., & Prichard, R. W. (1985). Animal models of behavioral influences on atherogenesis. In E. S.

- Katkin & S. B. Manuck (Eds.), *Advances in behavioral medicine* (Vol. 1, pp. 115–163). Greenwich, CT: JAI Press.
- Katkin, E. S. (1965). Relationship between manifest anxiety and two indices of autonomic response to stress. *Journal of Personality and Social Psychology*, 2, 324–333.
- Kelsey, R. M. (1991). Electrodermal lability and myocardial reactivity to stress. *Psychophysiology*, 28, 619–631.
- Kelsey, R. M., Blascovich, J., Tomaka, J., Leitten, C. L., Schneider, T. S., & Wiens, S. (1999). Cardiovascular reactivity and adaptation to recurrent psychological stress: Effects of prior task exposure. *Psychophysiology*, 36, 818–831.
- Kelsey, R. M., & Guethlein, W. (1990). An evaluation of the ensemble averaged impedance cardiogram. *Psychophysiology*, 27, 24–33.
- Kelsey, R. M., Reiff, S., Wiens, S., Schneider, T. R., Mezzacappa, E. S., & Guethlein, W. (1998). The ensemble averaged impedance cardiogram: An evaluation of scoring methods and interrater reliability. *Psychophysiology*, 35, 337–340.
- Kernberg, O. F. (1989). The narcissistic personality disorder and the differential diagnosis of antisocial behavior. *Psychiatric Clinics of North America*, 12, 553–570.
- Kernis, M. H., & Sun, C. (1994). Narcissism and reactions to interpersonal feedback. *Journal of Research in Personality*, 28, 4–13.
- Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiological reactivity and risk for cardiovascular disease: A review and methodologic critique. *Psychological Bulletin*, 96, 435–464.
- Kyes, R. C., Botchin, M. B., Kaplan, J. R., Manuck, S. B., & Mann, J. J. (1995). Aggression and brain serotonergic responsivity: Response to slides in male macaques. *Physiology & Behavior*, 57, 205–208.
- Lacey, J. I. (1967). Somatic response patterning and stress: Some revisions of activation theory. In M. H. Appley & R. Trumbull (Eds.), *Psychological stress: Issues in research* (pp. 14–37). Englewood Cliffs, NJ: Prentice-Hall.
- Larson, M. R., & Langer, A. W. (1997). Defensive hostility and anger expression: Relationship to additional heart rate reactivity during active coping. *Psychophysiology*, 34, 177–184.
- Lenfant, C. (1998). Task force on behavioral research in cardiovascular, lung, and blood health and disease. *Circulation*, 98, 281–286.
- Light, K. C., & Obrist, P. A. (1980). Cardiovascular response to stress: Effects of opportunity to avoid, shock experience, and performance feedback. *Psychophysiology*, 17, 243–252.
- Livesley, W. J., Jang, K. L., Jackson, D. N., & Vernon, P. A. (1993). Genetic and environmental contributions to dimensions of personality disorder. *American Journal of Psychiatry*, 150, 1826–1831.
- Lykken, D. T. (1995). *The antisocial personalities*. Hillsdale, NJ: Lawrence Erlbaum.
- Lykken, D. T., Iacono, W. G., Haroian, K., McGue, M., & Bouchard, T. J. (1988). Habituation of the skin conductance response to strong stimuli: A twin study. *Psychophysiology*, 25, 4–15.
- Lyness, S. A. (1993). Predictors of differences between Type A and B individuals in heart rate and blood pressure reactivity. *Psychological Bulletin*, 114, 266–295.
- Manuck, S. B., Flory, J. D., McCaffery, J. M., Matthews, K. A., Mann, J. J., & Muldoon, M. F. (1998). Aggression, impulsivity, and central nervous system serotonergic responsivity in a nonpatient sample. *Neuropsychopharmacology*, 19, 287–299.
- McBurnett, K. (1992). Psychobiological approaches to personality and their applications to child psychopathology. In B. B. Lahey & A. E. Kazdin (Eds.), *Advances in clinical child psychology* (Vol. 14, pp. 107–164). New York: Plenum Press.
- McCubbin, J. A., Richardson, J. E., Langer, A. W., Kizer, J. S., & Obrist, P. (1983). Sympathetic neuronal function and left ventricular performance during behavioral stress in humans: The relationship between plasma catecholamines and systolic time intervals. *Psychophysiology*, 20, 102–110.
- Mezzacappa, E. S., Kelsey, R. M., & Katkin, E. S. (1999). The effects of epinephrine administration on impedance cardiographic measures of cardiovascular function. *International Journal of Psychophysiology*, 31, 189–196.
- Miller, S. B., Dolgoy, L., Friese, M., & Sita, A. (1998). Parental history of hypertension and hostility moderate cardiovascular responses to interpersonal conflict. *International Journal of Psychophysiology*, 28, 193–206.
- Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin*, 119, 322–348.
- Morf, C. C., & Rhodewalt, F. (1993). Narcissism and self-evaluation maintenance: Explorations in object relations. *Personality and Social Psychology Bulletin*, 19, 668–676.
- Muldoon, M. F., Sved, A. F., Flory, J. D., Perel, J. M., Matthews, K. A., & Manuck, S. B. (1998). Inverse relationship between fenfluramine-induced prolactin release and blood pressure in humans. *Hypertension*, 32, 972–975.
- Muzi, M., Elbert, T. J., Tristani, F. E., Jeutter, D. C., Barney, J. A., & Smith, J. J. (1985). Determination of cardiac output using ensemble-averaged impedance cardiograms. *Journal of Applied Physiology*, 58, 200–205.
- Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective*. New York: Plenum Press.
- Obrist, P. A., Light, K. C., James, S. A., & Strogatz, D. S. (1987). Cardiovascular responses to stress: I. Measures of myocardial response and relationships to high resting systolic pressure and parental hypertension. *Psychophysiology*, 24, 65–78.
- Ogloff, J. R. P., & Wong, S. (1990). Electrodermal and cardiovascular evidence of a coping response in psychopaths. *Criminal Justice and Behavior*, 17, 231–245.
- Patrick, C. J. (1994). Emotion and psychopathy: Startling new insights. *Psychophysiology*, 31, 319–330.
- Pine, D. S., Wasserman, G., Coplan, J., Staghezza-Jaramillo, B., Davies, M., Fried, J. E., Greenhill, L., & Shaffer, D. (1996). Cardiac profile and disruptive behavior in boys at risk for delinquency. *Psychosomatic Medicine*, 58, 342–353.
- Pribram, K., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychological Review*, 82, 116–149.
- Quirk, M., & Sourkes, T. (1977). Central dopaminergic and serotonergic systems in the regulation of adrenal tyrosine hydroxylase. *Journal of Neurochemistry*, 28, 137–147.
- Raine, A., & Venables, P. H. (1984). Electrodermal non-responding, schizoid tendencies, and antisocial behavior in adolescents. *Psychophysiology*, 21, 424–433.
- Raine, A., Venables, P. H., & Williams, M. (1995). High autonomic arousal and electrodermal orienting at age 15 as protective factors against criminal behavior at age 29 years. *American Journal of Psychiatry*, 152, 1595–1600.
- Raskin, R., & Hall, C. (1981). The Narcissistic Personality Inventory: Alternate form reliability and further evidence of construct validity. *Journal of Personality Assessment*, 45, 159–162.
- Raskin, R., & Novacek, J. (1989). An MMPI description of the narcissistic personality. *Journal of Personality Assessment*, 53, 66–80.
- Raskin, R., & Novacek, J. (1991). Narcissism and the use of fantasy. *Journal of Clinical Psychology*, 47, 490–499.
- Raskin, R., Novacek, J., & Hogan, R. (1991). Narcissistic self-esteem management. *Journal of Personality and Social Psychology*, 60, 911–918.
- Raskin, R., & Terry, H. (1988). A principal-components analysis of the Narcissistic Personality Inventory and further evidence of its construct validity. *Journal of Personality and Social Psychology*, 54, 890–902.
- Rasmussen, P. R., Willingham, J. K., & Glover, T. L. (1996). Self-esteem stability, cynical hostility, and cardiovascular reactivity to challenge. *Personality and Individual Differences*, 21, 711–718.
- Rhodewalt, F., Madrian, J. C., & Cheney, S. (1998). Narcissism, self-knowledge organization, and emotional reactivity: The effect of daily experiences on self-esteem and affect. *Personality and Social Psychology Bulletin*, 24, 75–87.
- Rhodewalt, F., & Morf, C. C. (1995). Self and interpersonal correlates of the Narcissistic Personality Inventory: A review and new findings. *Journal of Research in Personality*, 29, 1–23.
- Rhodewalt, F., & Morf, C. C. (1998). On self-aggrandizement and anger: A temporal analysis of narcissism and affective reactions to success and failure. *Journal of Personality and Social Psychology*, 74, 672–685.
- Rosenthal, R., & Rubin, D. B. (1983). Ensemble-adjusted *p* values. *Psychological Bulletin*, 94, 540–541.
- Schalling, D. (1978). Psychopathy-related personality variables and the psychophysiology of socialization. In R. D. Hare & D. Schalling (Eds.), *Psychopathic behavior: Approaches to research* (pp. 85–106). Chichester, England: Wiley.
- Sherwood, A., Allen, M. T., Fahrenberg, J., Kelsey, R. M., Lovallo, W. R., & van Doornen, L. J. P. (1990). Committee report: Methodological guidelines for impedance cardiography. *Psychophysiology*, 27, 1–23.
- Sherwood, A., Allen, M. T., Obrist, P. A., & Langer, A. W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. *Psychophysiology*, 23, 89–104.

- Sherwood, A., Royal, S. A., Hutcheson, J. S., & Turner, J. R. (1992). Comparison of impedance cardiographic measurements using band and spot electrodes. *Psychophysiology*, *29*, 734–741.
- Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin*, *86*, 420–428.
- Siegel, J. H., Fabian, M., Lankau, C., Levine, M., Cole, A., & Nahmad, M. (1970). Clinical and experimental use of thoracic impedance plethysmography in quantifying myocardial contractility. *Surgery*, *67*, 907–917.
- Siever, L. J., & Davis, K. L. (1991). A psychobiological perspective on the personality disorders. *American Journal of Psychiatry*, *148*, 1647–1658.
- Simes, R. J. (1986). An improved Bonferroni procedure for multiple tests of significance. *Biometrika*, *73*, 751–754.
- Smith, T. W., Limon, J. P., Gallo, L. C., & Ngu, L. Q. (1996). Interpersonal control and cardiovascular reactivity: Goals, behavioral expression, and the moderating effects of sex. *Journal of Personality and Social Psychology*, *70*, 1012–1024.
- Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Suls, J., & Wan, C. K. (1993). The relationship between trait hostility and cardiovascular reactivity: A quantitative review and analysis. *Psychophysiology*, *30*, 615–626.
- Tharp, V. K., Maltzman, I., Sydulko, K., & Ziskind, E. (1980). Autonomic activity during anticipation of an aversive tone in noninstitutionalized sociopaths. *Psychophysiology*, *17*, 123–128.
- Wallin, B. G. (1981). Sympathetic nerve activity underlying electrodermal and cardiovascular reactions in man. *Psychophysiology*, *18*, 470–476.
- Watson, P. J., Grisham, S. O., Trotter, M. V., & Biderman, M. D. (1984). Narcissism and empathy: Validity evidence for the Narcissistic Personality Inventory. *Journal of Personality Assessment*, *48*, 301–304.
- Westen, D. (1990). The relations among narcissism, egocentrism, self-concept, and self-esteem: Experimental, clinical, and theoretical considerations. *Psychoanalysis and Contemporary Thought*, *13*, 183–239.
- Wright, R. A., Contrada, R. J., & Glass, D. C. (1985). Psychophysiological correlates of Type A behavior. In E. S. Katkin & S. B. Manuck (Eds.), *Advances in behavioral medicine* (Vol. 1, pp. 39–88). Greenwich, CT: JAI Press.
- Zagon, I. K., & Jackson, H. J. (1994). Construct validity of a psychopathy measure. *Personality and Individual Differences*, *17*, 125–135.

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