DOES DISSOCIATION OF EMOTIONAL AND PHYSIOLOGICAL REACTIVITY PREDICT BLOOD PRESSURE CHANGE OVER 10 YEARS?

by

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Abstract

One of the major theories of psychosomatic medicine is that pervasive dissociations between physiological reactivity and simultaneous emotion awareness may be an important marker for the long term development of cardiac problems. For example, a defensive coping style is characterized by high physiological reactivity to a stressor, paired with simultaneous low subjective emotional awareness of that stressor. The numerous different factors that have been identified as predictive of later cardiac states have lead to conceptual confusion in the field. Subjective Autonomic Discrepancy (SAD) scores computed by subtracting standardized subjective stress response scores from standardized reactivity scores, are proposed as a simplified method of capturing the dissociation between physiological and emotional reactivity, and thus increasing the explanatory power of predictive models of cardiac health outcomes. Data from a ten-year longitudinal study were used to evaluate the reliability and predictive validity of this phenomenon. It was found that discrepancy scores for blood pressure indices show trait-like stability over a period of 3 years (r = .53, p<.001 for SBP and r = .69, p<.001 for DBP, n=117). However SADS for heart rate showed only moderate 3 year stability (r = .16, p = .06). Encouraged by these results, two different predictor models for blood pressure change were tested. Although linear prediction of systolic blood pressure did come close to traditional definitions of significance, neither a linear nor a quadratic model was found to show significant prospective validity in predicting ambulatory blood pressure change over a 3 or 10 year period. All regression
analyses controlled for initial ambulatory blood pressure means and other relevant control variables. Dissociation between physiological arousal and emotional awareness does not appear to be an important variable in the identification of individuals at risk for later cardiovascular health problems.
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Introduction

The identification of individuals at risk for cardiovascular disease is an important step in the design of effective interventions. Many variables have been identified as predictors of cardiovascular disease. These predictors include family history, cardiovascular reactivity, or the body’s vascular response to stress (Krantz & Manuck, 1984; Linden, 1984; Schwartz et al., 2003); as well as personality factors such as type-D personality (Habra, Linden, Anderson, & Weinberg, 2003; Linden, 2000), and defensiveness (Ketterer et al., 2006; Rutledge & Linden, 2000, 2003; Rutledge, Linden, & Davies, 2000).

There are two main problems associated with prospective research on the multitude of risk factors associated cardiovascular disease: conceptual confusion and low statistical power. Many of the risk factors that have been identified to predict cardiovascular disease are conceptually overlapping and it is unclear what specific variables, or components of variables are the strongest predictors. Multiple regression models quickly lose power when the number of predictor variables is allowed to greatly increase. In consequence because so many different predictors have been identified, much research on predicting cardiovascular disease focuses on pooling inter-related risk factors. Although the pooling of physiological and personality predictors does result in increased prediction of cardiovascular outcomes (Light et al., 1999), these analyses can suffer from low power unless larger and larger samples are used for longitudinal evaluation. Such large-scale prospective research is rare because it is expensive and labor intensive.
The current study investigates the predictive ability of Subjective Autonomic Discrepancy (SAD) scores, a numeric variable that integrates both physiological reactivity and psychological variables into a possibly more parsimonious and more powerful predictor of cardiac outcomes over a 10 year period.

**Reactivity as a Predictor**

Cardiovascular reactivity has long been studied as a predictor of later cardiac disease states. Increased cardiac reactivity following exposure to stressors has been shown to lead to the development of hypertension over extended periods of time (Schwartz et. al., 2003). Blood pressure reactivity to psychological stimuli differentially predicts the development of future hypertension and need for antihypertensive medication at 9 to 12 year follow up (Tuomisto, Majahalme, Kahonen, Fredriksen, & Turjanmaa, 2005). Importantly, in this study and others, reactivity was found to be predictive of future blood pressure elevation over and above traditional clinical risk factors (Moseley & Linden, 2006; Mathews, Salomon, Brady, & Allen, 2003; Stewart, Janicki, Kamarck, 2006; Stewart, & France, 2001; Toumisto et al., 2005). Matthews and colleagues showed that composite measures of reactivity derived from multiple tasks have been related to resting blood pressure at 3-year follow-up in an adolescent sample (SBP $\beta = 0.161$, $p = .009$; DBP $\beta = 0.292$, $p = .003$) (Matthews, Salomon, Brady, & Allen, 2003). The same has been shown to be true at three – year follow-up (42% of variance explained by DBP and 40% of variance explained by SBP), and 10-year follow up in an adult sample (44% of variance explained by DBP and 34% of variance explained by SBP) (Moseley, & Linden, 2006). A review by Treiber and colleagues (2003) also concluded that reactivity significantly predicts preclinical indicators of, and risk for, cardiac health outcomes over
and above baseline blood pressure readings. This was shown to be particularly true for young people.

Laboratory study of cardiac reactivity has used a variety of different types of stressors. Laboratory stressors have been categorized into passive tasks, active or performance based behavioral tasks, and social tasks. Examples of passive tasks are the cold pressor task or watching an unpleasant video, while solving mental arithmetic problems or mirror tracing are both active tasks. An example of a social stressor is having to solve hypothetical problems involving social situations. Researchers have had mixed success eliciting cardiac reactivity using passive tasks alone, while the use of active and social tasks has produced more consistent results (Linden, Rutledge, & Con, 1998; Tuomisto et al., 2005).

The mechanism through which laboratory stressors elicit cardiac reactivity is assumed to be the same as the reactivity mechanism that operates when an individual is faced with a stressor in their day to day life. Because an individual would likely be faced with many different types of stressors in the real world, the use of a variety of different types of stressors (i.e. both active and passive) in a single experimental paradigm represents a more accurate reflection of reactivity in general. Using many different types of stressors in a single paradigm is a way not only to increase reliability of the reactivity response, but also to increase the ecological validity of the experimental task (Tuomisto et al., 2005; Rutledge, Linden, & Paul, 2001). In general, research aggregating physiological responses to tasks of different type has had the most success in predicting cardiac outcomes (Kamarck, 1992; Rutledge et al., 2001).
In general, reactivity has been shown to be a significant predictor of cardiac
disease states. This is especially the case in studies that have utilized a variety of
reactivity paradigms.

**Personality as a Predictor**

In addition to physiological predictors such as reactivity, psychological factors
have also been implicated in the development of cardiac disease states. These include the
personality traits type-D personality, defensiveness, and alexithymia.

Type D personality is characterized by a defensive personality style paired with
high negative affectivity. Type D personality has been linked to the development of
cardiac disease states in general (Denollet, Pedersen, & Vrints, 2006; Habra, Linden,

Factor analytic work has clarified that defensive personality style is composed of
two distinct dimensions: self-deception, and other-deception (Paulhus, 1984). Self-
deceptive behavior tends to remain constant regardless of context, while other-deception
is more closely linked to the concept of impression management, and will change with
changing context (Linden & Rutledge, 2003; Paulhus, 1984). Although overall
defensiveness has been linked to cardiac health outcomes (Ketterer et al., 2006), more in
depth analysis reveals that it is in fact the self-deception factor, and not the other-
deception factor that largely accounts for the variance in this relationship (Rutledge &
Linden, 2000; 2003; Rutledge, Linden, & Davies, 2000). Self-deception has been linked
with a prospective risk for hypertension over a 3 year period, while impression
management scores did not have this predictive value (Rutledge & Linden, 2003;
Rutledge, Linden, & Davies, 2000). As well, self-deception scores in male ischemic heart
disease patients correlated modestly with smaller treatment benefits measured in the reduction of ischemic episodes (Rutledge, Linden, & Davies 2000).

Alexithymia is a trait marked by difficulty identifying and communicating feelings (Labouvie, Lumley, Jain, & Heinze, 2003). This problem is proposed to be due to a cognitive deficit in emotion regulation that leads to difficulty in identifying emotions. During reactivity tasks, alexithymic individuals show normal physiological reactivity but attenuated emotional reactions (Linden, Lenz, & Stossel, 1996) therefore presenting with a desynchronous response. Alexithymia has been linked with preclinical and clinical cardiac disease states (Byrne & Ditto, 2005; Neumann, Sollers, Thayer, & Waldstein, 2004; Todarello, Taylor, Parker, & Fanelli, 1995). Cross sectional study has found that when compared with psychiatric outpatients and normal controls, hypertensive patients show increased rates of alexithymia (Todarello, Taylor, Parker, & Fanelli, 1995). Alexithymia has also been prospectively associated with cardiac morbidity (Neumann, Sollers, Thayer, & Waldstein, 2004).

All these variables conceptually represent the potential for desynchrony between self reported affect and physiological reactivity. However, the many different variables that have been studied, and their often overlapping definitions can lead to confusion in terminology, which interferes with a clear interpretation of the research. In contrast, the SAD variable is posited to be a straightforward operationalization of this concept of desynchrony.

An Alternative to Multiple Overlapping Predictors: The Discrepancy Score

Efforts to study multiple predictors of cardiac health outcomes in an attempt to increase prediction have been met with some success. Light and colleagues found that
later blood pressure prediction from cardiovascular reactivity was modulated by family history of hypertension in at least one parent, and stressful life events (Light et al., 1999). Rutledge and Linden (2003) have also shown that reactivity in the presence of defensive coping style explained long term blood pressure change better than did either predictor alone. However, analyses such as these, testing a large number of hypothesized independent predictors, easily suffer from low power and would require large sample sizes to reach adequate levels of power. Light et al.’s data might have been even more convincing with a larger sample or fewer predictors.

It makes sense to combine affective and physiological responding into a single predictor because these responses are not independent in real-world stressful situations. This rationale for trying to identify and study individuals with desynchronous responding of affect versus physiology had already led to the development of the alexithymia and the repressor concepts discussed above. As well, an easy to compute marker of desynchrony between affect awareness and simultaneous physiological reactivity has the potential to simplify this type of research, and may be an important variable in identifying individuals at later risk for cardiac health problems.

Schwerdtfeger and colleagues (2006) have proposed the use of a single discrepancy score that describes the physiological and stress response components relative to each other. This variable was computed by subtracting standardized changes in negative affect experienced during a well-defined, standardized stressor from standardized changes in heart rate and skin conductance reactivity to the same stressor. In year one of their study, these stressors included a speech stressor, the cold pressor, and a video (viewing of the speech stressor); and at year 2 follow-up the stressors studied were
mental arithmetic, anagrams, and a torture video. These researchers investigated the stability of a single construct that integrates reactivity with some aspects of defensive responding. Schwerdtfeger's work (2006) shows that individual differences in the desynchrony between physiological (reactivity) and negative affect responses to stress are stable over the period of 1 year ($r = .61, p < .001$ for HR and $r = .40, p < .05$ for SCL) and across a variety of different stressor tasks.

The current study extends this work by investigating the ability of the dissociations between reactivity and subjective response to stress, here operationalized as subjective autonomic discrepancy (SAD) scores, to predict changes in cardiac health risk over time. The calculation of scores results in two different types of discrepancy scores. One type is characterized by defensive responding, and consists of a large physiological response to a stressor paired with an attenuated emotional response (resulting in a positive SAD score). In contrast, a negative SAD score reflects a dissociation where a large affective response is paired with an attenuated physiological response (one could also think of this response style as "neurotic"). From this, two different models predicting cardiac health outcomes are possible: a quadratic model and a linear model. Both models predict that positive SAD scores will be related to later cardiac health problems. However, a linear model tests the hypothesis that negative SAD scores will be associated with better cardiac outcomes than both 'balanced' scores and positive SAD scores, which are expected to show the worst outcomes overall. In contrast, a quadratic model tests the hypothesis that both positive and negative SAD scores (or any desynchrony between reactivity scores to laboratory stressors and self report of emotional response)
will be equally associated with later cardiac health problems, and that scores reflecting synchronicity will be associated with the most benign outcomes.

[Fig. 1]

The cardiac reactivity hypothesis (Krantz & Manuck, 1984) is one model that may explain how reactivity paired with defensive response style (as indexed by SADS) can explain disease development—a result that is hypothesized by both the linear and the quadratic model. Linden (1984) and Brownley and colleagues (Brownley, Hurwitz, & Schneiderman, 2000) proposed a pathway such that early elevations in cardiac output (cardiac reactivity to stressors in everyday life) lead to later compensatory elevations in vascular resistance in order to maintain homeostatic blood pressure. If an individual is aware of these elevations in cardiac reactivity, the elevations can be offset by changing behavior. However, if an individual is unaware of these elevations (as in defensive responding), behavior may not be altered. In this case, that person is exposed to more frequent and more pronounced instances of cardiac reactivity, and for more sustained duration. This increased exposure to elevated cardiac response may lead to eventual elevation of tonic blood pressure levels and hypertension. In this way, reactivity to stressors paired with a defensive responding style may eventually lead to negative cardiac health outcomes (Rutledge & Linden, 2003).

A quadratic model of predicting cardiac health outcomes has some intuitive appeal. After all, having any discrepancy between physiological and affective responding to a stressor, whether that discrepancy results from over-responding either affectively or physiologically sounds like it may have a negative impact on health. The conception of synchronous responding as being healthy, and incongruent responding, resulting either
from relative physiological or affective over-responding, as being unhealthy is consistent with lay conceptions of health behaviors as being ‘balanced’ and moderate in nature. In fact, past psychological research has found evidence for curvilinear relationships in predicting blood pressure (Hogan, & Linden, 2005). Hogan and Linden (2005) found evidence for a quadratic relationship between expressed anger and blood pressure in women where women at both extremes of self reported anger expression are found to have lower blood pressure compared to women with moderate levels of self reported anger expression.

Despite the intuitive value of a quadratic model relating SAD to later ambulatory blood pressure, there is evidence to suggest that an exaggerated affective response coupled with a relatively smaller reactivity response to a stressor may in fact serve a protective function, and be associated with lower ambulatory blood pressure levels over extended periods of time. This negative SAD score reflects neurotic responding. A long history of continuing research suggests that contrary to intuition, trait anxiety-neuroticism may have protective properties (Siegman, Anderson, Herbst, Boyle, & Wilkinson, 1991; Siegman, Dembroski, & Ringel, 1987; Watson, & Pennebaker, 1989; Davies, 1970). Neurotic hostility may be protective given that male subjects’ experience of anger-hostility (when provoked) correlated negatively with resting levels of systolic blood pressure, diastolic blood pressure, and heart rate (Siegman et al., 1991). Similarly, it was found in a sample of patients scheduled for coronary angiography that hostility correlated with indices of neuroticism, neurotic hostility, and was thus significantly inversely associated with severity of coronary artery disease (Siegman, Dembroski, & Ringel, 1987). Watson and Pennebaker (1989) found that although negative affectivity was
correlated with health complaints, it was not related to actual long term health status, and Davies (1970) found a negative relationship between blood pressure and history of neurotic traits in childhood, current neurotic symptoms, and the neuroticism scale of the Eysenck Personality Inventory. These results suggest that a negative SAD score may be a marker of a protective function and be associated with more positive cardiac health outcomes compared to a positive SAD score.

Both the linear and quadratic models of SAD proposed in this study hypothesize that positive SAD scores which characterize defensive responding, will lead to worse cardiovascular outcomes. Past work has suggested that dissociations between physiological and affective responding to stressors in either direction (defensive or neurotic) may reflect a tendency for individuals to inadequately process physiological changes related to stress (Schwerdtfeger, Schmukle, & Elgoff, 2006). The current study tests not only whether this is true for response dissociations in the positive direction (a large physiological response paired with a smaller affective response), but also whether it is true for negative SAD characterized by a large affective response paired with a smaller physiological response. If support is found for the quadratic model, than any dissociation between physiological and emotional responding, both defensive and so-called neurotic responding, is related to relatively worse health outcomes. On the other hand, if support is found for the linear model, defensive responding, but not neurotic responding may lead to later negative outcomes.

Both the linear and the quadratic model also propose that synchronous stress responses will be associated with relatively positive outcomes. This is true for both high physiological response paired with high affective response, and for low physiological
response paired with low emotional response. An individual characterized by either of these response styles, when faced with a stressor experiences an emotional response that is proportional to their physiological response. According to the cardiac reactivity hypothesis, elevated resting blood pressure results directly from increase instances of elevated physiological responding to stress. Given this, regardless of whether such an individual exhibits a pairing of a large physiological response and a large emotional response, or a small physiological response paired with a small emotional response, that individual's proportional emotional response is information that may be useful in shaping future behavior to limit their exposure to stressful situations. Therefore it is hypothesized in both the linear and quadratic model that any individual with a synchronous response will have similar and relatively positive cardiac outcomes.

The current study uses three different reactivity paradigms and a longitudinal design to follow a sample of initially normotensive individuals over a period of 10 years. Firstly, it is expected that dissociation between physiological and subjective responding to stressors will show stability over time, thus replicating Schwerdtfeger et al.'s observation. Secondly, if stability of these SAD scores can be shown, it justifies evaluation of the prospective validity of SAD in predicting health outcomes. The predictive ability of two different models will be compared: The linear model predicts that scores reflecting underreporting of subjective emotional response paired with large physiological reactivity (positive SAD scores) will show greater increases in ambulatory blood pressure at long-term follow up compared to both synchronous responders, and with over reporting of emotional response paired with attenuated physiological responding (negative SAD scores); with negative SAD scores being associated with the
lowest levels of ambulatory blood pressure at follow-up. In contrast, the quadratic model predicts that any desynchrony in responding will be associated with greater risk of developing preclinical cardiac states, and cardiovascular disease compared to synchronous responding.

**Method**

**Research Design**

The independent measures used in this study are SAD scores; consisting of the difference between an individual's standardized physiological reactivity to a behavioral stressor, and that individual's standardized report of affective arousal associated with that stimulus. A reactivity score consists of the standardized difference between an index of baseline physiological arousal (for example, systolic blood pressure, diastolic blood pressure, or heart rate) and physiological arousal recorded for the same behavioral stress task. Affective arousal scores were collected in self-report format. The physiological reactivity scores and affective arousal scores used to create SAD scores are (in this study) aggregated over three separate behavioral stress tasks in order to create a more reliable index of reactivity (Kamarck, 1992; Rutledge, Linden, & Paul, 2001).

The dependent variables in this design are 8-10 hour ambulatory systolic and diastolic blood pressure measured at 3-year follow up, and at 24 hour ambulatory monitoring at 10-year follow up.

**Participants**

Data were originally collected for a large scale longitudinal project exploring the relationship of multiple risk factors to the development of hypertension (Moseley & Linden, 2006; Rutledge and Linden, 2003). Participants consisted of university students
and adults from the surrounding community recruited through advertising. In accordance with entrance standards, no participant had a current diagnosis of heart disease or hypertension and revealed normal blood pressure readings at study entry. Complete data were available at intake for 330 participants (157 men and 173 women, average age=26.9 years, SD=11.2). Of these, 124 participants (approximately 38% of the original sample, average age 32.7 years, SD=12.7) returned after 3 years to complete the protocol a second time. At 3 year follow up participants underwent 12 hour ambulatory blood pressure monitoring and replicated the full stress reactivity test package. A further 112 participants (approximately 34% of the original sample, average age 40.3 years, SD=13.3) returned for a 10-year follow to undergo repeated personality testing and 24 hour ambulatory blood pressure monitoring, but not reactivity tests. The primary reason for drop out was relocation as the majority of the original sample consisted of university students who had long since graduated. The ethnic composition of the original sample as well as the sample at both follow up periods was approximately 70% Caucasian and 30% Asian. The original sample had an even split of men and women and this gender balance was preserved for the two follow up tests.

**Measures**

**Physiological Measurement**

Ambulatory blood pressure and heart rate information was collected using SpaceLabs 90207 ambulatory monitors (Spacelabs Medical Inc., WA, USA). These devices weigh approximately 0.7 kg and are worn in a protective pouch. The use and accuracy of these monitors is supported by previous validation work (O’Brien, Mee, Atkins, & O-Malley, 1991). Participants were explicitly instructed to minimize physical
activity during measurement cycles. The monitors sampled 3 times per hour during the
day and once per hour during sleep. Approximately 10% of attempted measurements
were unusable, and these were identified via error codes on the SpaceLabs monitors.
After deletion of all invalid readings, an average of approximately 50 readings per
individual were available for analysis.

Blood pressure and heart rate information collected during the laboratory protocol
was obtained using Dinamap 845 Vital Signs Monitors (Critikon Corporation, Tampa,
FL). Previous validation has shown Dinamap 845 Vital Signs Monitors measurements to
be highly correlated with intra-arterial measurements (Borrow, & Newburger, 1982).

**Behavioral Stress Tasks**

Task instructions were delivered over an intercom system. Participants completed
three 5-minute stress tasks: a handgrip task, a mental arithmetic task, and an anger recall
discussion task. These tasks were completed in counterbalanced order, and a 5-minute
recovery period followed the completion of each task (during which time participants
were instructed to read magazines).

For the isometric handgrip task, participants were instructed to maintain handgrip
tension on a standard dynamometer at 20% maximum for 3 minutes followed by 2
minutes at 30% maximum. For the mental arithmetic task, participants read a set of
incomplete equations aloud from a television and then verbalized their answers.
Arithmetic problems were presented at 5-second intervals and on average participants
answered about 65% of these correctly (thus reflecting a reasonable difficulty level
requiring effort). For the anger-recall discussion task, participants were given two
minutes to recall an anger-provoking situation from their work or personal life, after
which time they discussed the event with a same sex research assistant for 3 minutes. Previous validity work justifies the aggregation of reactivity scores for these three tasks into a single reactivity index (Kamarck, 1996; Moseley & Linden, 2006). Scores aggregated across the handgrip, mental arithmetic, and anger recall tasks show adequate test-retest stability over a 3 year period for systolic blood pressure, diastolic blood pressure, as well as heart rate reactivity indices of reactivity.

**Self Report Affect Measurement**

Collection of self report of affect occurred immediately following exposure to the behavioral tasks via ratings made on Russell's Affect Circumplex (Barrett & Russell, 1998; Russell & Barrett, 1999). This measure conceptualizes affective response as consisting of two separate dimensions: an activation-deactivation dimension, and a pleasant-unpleasant dimension. Any response can be scored as high or low on both of these dimensions. Participants' affective ratings on the pleasant-unpleasant dimension were used for the purposes of this study, as analyses using ratings on the arousal dimension yield weaker results (detailed findings not shown). The measure for this dimension consists of a scale ranging from 1 to 9 where participants indicate their degree of feelings of pleasantness at that time. Factor analytic techniques have been used to demonstrate the validity of this measure for capturing affective response in a self report format (Barrett & Russell, 1998).

**Demographic Information**

Participants answered questions relating to age, sex, tobacco use, exercise frequency, and family history of cardiovascular problems.
Procedure

Participants underwent a similar testing procedure at all three phases of the study. Testing at time one involved a two hour block of laboratory assessment. Participants were encouraged to avoid ingesting alcohol, caffeine, or nicotine or exercising strenuously for the two hours prior to the laboratory session. The procedure and general purpose of the study was outlined for participants before informed consent was given. Participants first completed questionnaire data on demographic and clinical risk factors. Following this, participants sat alone during a 20 minute resting phase while baseline blood pressure and heart rate measurements were taken. Exposure to behavioral stressors commenced at this point. Participants engaged in a counter balanced set of 3 5-minute behavioral stress tasks (handgrip testing, mental arithmetic, and anger recall) during which time reactivity measurements were taken. Following each task, participants were administered questionnaires targeting their subjective report of their affective experience of that task.

Laboratory measurement of resting blood pressure and heart rate was supplemented by ambulatory measurement of blood pressure and heart rate. Ambulatory measurements were taken after the participant completed the protocol in the laboratory. Ambulatory measurement took place over the course of 24 hours at initial testing, at 3-year follow up, and at 10-year follow up. Participants were asked to choose a typical day, free of any specific stressors, for their ambulatory monitoring. Monitors were fitted to participants, pretested on the spot, and returned after monitoring for data analysis. Pretesting was used to determine proper cuff placement and consisted of the first five ambulatory readings taken while in the laboratory. Values were compared with resting blood pressure and heart rate measurements. When pretest values seemed questionable
the cuff was repositioned. Once properly fitted, ambulatory readings commenced and were obtained every 20 minutes.

**Results**

Data analysis was conducted in two parts: a) stability of SAD scores for each cardiovascular index were calculated, and b) SAD scores’ predictive potential for 3-year and 10-year blood pressure levels was investigated for both linear and quadratic predictive models of cardiac health outcomes.

An approximately equal split of men and women participated in the protocol across each testing session; onset of hypertensive medication use was fairly rare across both follow-up samples (5% at 3-year follow up and 12% at 10-year follow up). Resting cardiovascular data were significantly lower at initial data collection compared to 3-year and 10-year follow-up data. As well, the sample at 3 year follow up had a higher ratio of individuals with a family history of hypertension compared to those with no family history of hypertension. Initial resting and ambulatory blood pressure values were significantly lower than 3-year follow up data. However, surprisingly, resting and ambulatory blood pressure values were significantly lower at 10-year follow up compared to 3-year follow up (Moseley & Linden, 2006).

Despite the high drop out rate over the 10 year follow up (62% at 3-year and 65% at 10-year), analysis of reactivity data comparing dropouts to study completers revealed few differences: Study completers participating in the 3-year and 10-year follow ups were (predictably) older and more likely to have a family history of hypertension at baseline compared to drop outs. Participants who completed the 10-year follow up were
also more likely to have higher resting SBP at baseline compared to dropouts (Moseley & Linden, 2006).

Three year stability ratings were calculated for subjective stress ratings, blood pressure, and heart rate responses for an aggregate measure of the three laboratory tasks. One hundred and eleven participants provided complete data for both occasions. Stability analysis revealed that the subjective stress rating pattern was stable over 3 years (r=.46, p<.001), and systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR) residualized change scores were modestly stable with r’s = .29, p=.002; .47, p<.001; and .12, p=.16 respectively. This justified the computation of the stability of discrepancy scores over 3 years; and the resulting scores were r = .38, p<.001 for SBP; r = .52, p<.001 for DBP; and r = .30, p=.002 for HR.

Because the anger recall task is the task most specifically geared toward affect provocation, it was possible that the unpleasantness ratings for the anger recall task (as opposed to an aggregate of the three tasks), may have been a more true to life and valid measure to use in the calculation of SAD scores to predict 10 year cardiovascular outcomes. To guard against this possibility a stability score was also calculated for the subjective stress ratings for the anger recall task alone. These analyses yielded slightly lower stability scores compared to that calculated for an aggregate of the three lab tasks. Therefore, aggregation of reactivity data was used in calculating the stability of discrepancy scores.

The second stage of data analysis tested the validity of SAD for predicting 3 year and 10 year cardiovascular outcomes respectively, for both linear and quadratic predictive models. Two separate stepwise regression models were tested for each follow
up period, one each for the prediction of systolic blood pressure, and one for the prediction of diastolic blood pressure. No regression model was tested for the prediction of heart rate as heart rate did not show adequate stability over a period of 3 years. One hundred and twelve participants provided complete data for lab stress reactivity and ambulatory blood pressure data at intake, then again 10 years later. Relevant control variables as well as participants’ initial daytime ambulatory blood pressure means were entered in the first stage of each model. Relevant control variables were those variables that correlated more than $r = .10$ with the 3 and 10 year blood pressure index, and did not correlate highly with other control characteristics used in the analysis. The control variables that were used were determined by previous research using the same data set (Moseley & Linden, 2006). Because ambulatory blood pressure readings at year 3 were taken over the course of 12 hours, while those at 10 year follow up were taken over the course of 24 hours, and because the control variables that were used were different in the 3 year and 10 year models, direct comparisons can not be made between these two models. However, the goal of the analysis was to create the strongest predictive model possible for each follow up period.

Hierarchical multiple linear regression controlling for initial ambulatory blood pressure means and relevant control variables show that SAD scores came close to traditional definitions of significance in predicting systolic ambulatory blood pressure 3 years later ($\beta = .14$, $p = .056$), but did not predict diastolic ambulatory blood pressure at 3 year follow up ($\beta = .082$, $p = .22$). As well, SAD scores in the linear model were found to be significantly predictive of 3 year blood pressure only very slightly over and above traditional predictors of heart health outcomes controlled for in step one of the analysis.
(change in $R^2 = .016$ for systolic, change in $R^2 = .006$ for diastolic). Tests of the quadratic model predicting ambulatory blood pressure at 3-year follow-up revealed no effect for either systolic or diastolic blood pressure ($\beta = -.75$, $p = .46$ for systolic blood pressure and $\beta = .006$, $p = .93$ for diastolic blood pressure). The quadratic model was found to predict no additional variance in systolic blood pressure at 3-year follow-up compared to the linear model (change in $R^2 = .002$), and no additional variance in diastolic ambulatory blood pressure. Table 2 presents the results of the regression analysis for the 3-year predictions of SBP and DBP.

[Table 1]

Hierarchical multiple linear regression controlling for initial ambulatory blood pressure means and relevant control variables show that SAD scores were not significant predictors of systolic ambulatory blood pressure 10 years later ($\beta = .060$, $p = .49$). As well, in similarly controlled regression, SAD scores did not significantly predict diastolic blood pressure at 10-year follow-up ($\beta = .042$, $p = .58$). SADS were not found to be significantly linearly predictive of 10-year systolic blood pressure over and above traditional predictors of heart health outcomes controlled for in the analysis (change in $R^2 = .003$). Linear prediction of 10-year diastolic blood pressure over and above traditional predictors of heart health entered in step one of the model was also not significant (change in $R^2 = .002$). The quadratic model predicting ambulatory blood pressure at 10-year follow-up did not reach significance for either systolic ($\beta = .004$, $p = .96$) or diastolic blood ($\beta = .036$, $p = .64$). As well, the quadratic model was found to predict no additional variance at 10-year follow-up, in comparison to the linear model, in ambulatory systolic blood pressure, and little additional variance in ambulatory diastolic blood pressure ($R^2 = .001$). Table 3
presents the results of the regression analysis for the 10 year predictions of SBP and DBP.

[Table 2]

Discussion

This paper investigated whether a relationship was present between SAD and later hypertension. The cardiac reactivity hypothesis (Krantz & Manuck, 1984; Linden, 1984) proposes a mechanism that justifies investigation of such a relationship.

Although results supported the stability of SAD scores (replicating and expanding upon work by Schwerdtfeger, Schmukle, & Elgoff, 2006), no support was found to support the relationship between SAD and later increased ambulatory blood pressure. Neither exaggerated physiological responding to a stressor paired with an attenuated emotional response, or attenuated physiological response paired with exaggerated emotional response were associated with more negative cardiac health outcomes. This was true when testing both linear and quadratic regression models. Additional regression analyses also revealed that although physiological reactivity was found to be predictive of later blood pressure indices, neither affective self report alone nor the interaction between reactivity and affect were significant in predicting additional variance in later blood pressure. The consistency of negative results seen here increases belief in their stability, but there is always the chance of alternate explanations.

When faced with a psychological challenge an individual has not only a physiological system response, but an emotional response as well. This study suggested that individuals may be able to use this affective information to shape their behavior in order to limit their exposure to stressful situations within an optimal window, or to
facilitate adaptive coping and recovery. Although the discrepancy between physiological and reactivity and emotion in response to a stressor as operationalized in this study were not found to be predictive of later cardiovascular levels, it may be that this is due to limitations in the way in which the variable was measured, as opposed to flaws in the underlying theory.

Data from this study suggest that affective responses often do not correspond to physiological responses. SAD scores were designed to index this discrepancy between two response modes and thereby reduce the number of needed predictors in statistical tests of prediction models. In this study SAD scores were composed of the difference between a participant's physiological and emotional response to three different stressors. Although physiological reactivity to stress is a widely studied predictor of later cardiovascular levels, less ground work has been done on the role emotional responding to stressful laboratory situations in predicting later physiological cardiovascular outcomes.

A potential limitation of this lies in the single self report measure used to index affect following exposure to stressful stimuli. However, although a single measure of affect was used to assess affective response to each laboratory stressor, measurements of affect were still aggregated across three distinct laboratory tasks for each participant, therefore increasing reliability of affect measurement. The reliability of affect measurement is also suggested by the fact that affect measurement showed the same pattern of stability compared to that of physiological measurement.

Although affect measurement in this study seemed to be reliable, it may be that the measure of emotional response to stress used in the current study was not sufficiently
sensitive to measure affective responding. It may also be that the stressor tasks used in
the current study did not provoke sufficient emotional response from participants to be an
accurate reflection of affective responding to stressors encountered in daily life. It may be
interesting in future work to explore the ability of discrepancy between emotional and
physiological responding to stress to predict cardiac health outcomes making use of more
traditionally trait like indices of emotional responding, such as defensiveness. In future
work it will also be important to continue to explore any differential predictive ability of
positive and negative, as well as synchronous SAD scores.

The current study has several noteworthy strengths. Firstly, the number of
subjects in the study was reasonably large and composed of an even split of men and
women, and an even split of participants with and without a family history of
cardiovascular problems. These subjects were examined both 3 years and 10 years
following their initial participation in the study. Despite the high attrition rate over time,
those who dropped out of the study did not differ critically from those who completed
their final session. Additionally, the reliability of data was enhanced by aggregating
participants' scores across three distinct laboratory stressors. The use of three distinct
stressors represents another strength of this study. SAD scores aggregated across the
three different laboratory stressors were found to be more stable over a three year period
compared to scores for only the anger recall task. As well, the use of three distinct tasks
represents a more ecologically valid reflection of a variety of real life stressful situations
compared to the use of a single task. Perhaps most importantly, combining relevant
predictive variables into a single predictive SAD score ensured that our analyses
possessed more power, as opposed to the use of multiple individual predictors. This study
is, to our knowledge, the first study to investigate the utility of SAD scores in the prediction of blood pressure change over 10 years.

Because the regression analysis used in this study involved the use of multiple predictor variables, there is a possibility that certain predictors act as suppressor variables. Suppressor effects occur when additional predictors entered into the analysis effectively suppress irrelevant variance in the analysis, and may improve the predictive ability of one or more variables (Paulhus, Robins, Trzesniewski, & Tracy, 2004). However, the control variables used in the present analyses were chosen because they correlated with the criterion variable, and had low correlations with other predictor variables, thus effectively protecting against the possibility of the suppression of irrelevant variance.

Although in the current paradigm SAD was not found to be predictive of later ambulatory blood pressure, given different methodology, SAD may yet prove to have some predictive utility in future work. A stronger, but much more logistically difficult test of the predictive utility of the SAD construct would be to tap emotional and physiological reactivity to real world stressors. The creation of SAD scores using these ecologically valid indices may result in increased predictive power of health outcomes. In addition, SAD scores may be predictive of endpoints other than ambulatory blood pressure (as investigated in the current study) such as endocrine or immune dysfunction.
Table 1: Three Year Blood Pressure Prediction

Results of Stepwise Regression Analysis Predicting 3-Year Ambulatory Blood Pressure
From SAD Scores Aggregated Across Task Type, Initial Blood Pressure, and Significant
Control Variables. Results for linear regression can be seen in step 2 of the model
(n=124).

<table>
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<tr>
<th>Variable</th>
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<th>p</th>
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<th>Change in R²</th>
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<td>.48</td>
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<td>SADS (quadratic model)</td>
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<tr>
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**Table 2: Ten Year Blood Pressure Prediction**

Results of Stepwise Regression Analysis Predicting 10-Year Ambulatory Blood Pressure From SAD Scores Aggregated Across Task Type, Initial Blood Pressure, and Significant Control Variables. Results for linear regression can be seen in step 2 of the model (n=112).

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<tr>
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<td>.003</td>
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</table>
Figure 1: Results Expected Based on Predictive Models

Differential outcomes hypothesized by idealized linear and quadratic models of the impact of SAD on ambulatory blood pressure at follow up.
References


Critikon Corporation, Tampa, FL.


Spacelabs Medical Inc., Redmond, WA, USA.


