# Predicting Blood Pressure and Heart Rate Change with Cardiovascular Reactivity and Recovery: Results from 3-Year and 10-Year Follow-Up

by

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## Abstract

We examined whether cardiovascular reactivity to and recovery from lab-induced stress is useful in predicting 3-year and/or 10-year changes in ambulatory blood pressure (BP) and heart rate (HR) among initially normotensive, community-dwelling adults and university students. At baseline, participants completed one day of laboratory testing, which included a 20-minute baseline assessment followed by three counterbalanced five-minute laboratory challenges (mental arithmetic, speech, and handgrip). Five-minute recovery periods followed each challenge. Measurements of systolic BP, diastolic BP, and HR were collected throughout this lab protocol. Data were then collected on 10-12 hours of ambulatory monitoring at 3-year follow-up, and on 24 hours of ambulatory monitoring at 10-year follow-up. Regression analyses indicated that after adjustment for initial resting cardiovascular levels along with predictive demographic and risk factor variables, aggregate reactivity scores explained unique and significant variance in ambulatory levels for all of the 3-year indices and two of the three 10-year indices (it did not improve the 10-year HR predictor model). When aggregate recovery data were forced into the models in a later step, it was found to explain unique and significant variance in ambulatory levels above that explained by initial resting cardiovascular levels, predictive demographic and risk factor variables, and aggregate reactivity scores for all of the 3-year indices. Moreover, in these final prediction models, the recovery data appeared to be significantly more predictive than the respective reactivity data. With respect to the 10-year data, aggregate recovery data did not significantly improve prediction models, although it again was found to be more predictive than respective reactivity data in two of the three final 10-year models (the 10-year DBP model did not follow this trend). Family hypertension history data were not found to be significantly associated with reactivity or recovery data, nor was it found to be predictive of longitudinal ambulatory data after adjustment for initial resting cardiovascular levels. In conclusion, our data thoroughly support the utility of reactivity and (particularly) recovery in predicting proximal BP and HR changes, generally support the use of reactivity in long-term BP predictions, and attest to the overall superiority of the DBP model in these predictions.

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## Introduction

Cardiovascular Disease (CVD), defined as disease of the heart and/or blood vessels, is the leading cause of death of Canadians, accounting for 79,389 Canadian deaths in 1998 (the last year for which Stats Canada has data); 35% of all male deaths and 38% of all female deaths (http://ww1.heartandstroke.ca/2/4/2002). It is estimated that 1 in 4 Canadians (i.e., approximately 8 million individuals) have some form of heart disease. Roughly 8 in 10 Canadians have at least one risk factor for CVD, and 11% have three or more risk factors, and there appears to be a growing prevalence of risk factors among young Canadians. The interaction of stress exposure with these individual risk factors is what contributes to the development and progression of this disease (Schwartz et al., 2003). In addition to lives lost, this disease costs the Canadian economy over $18 billion a year, which according to the Canadian Minister of Health, is the largest burden caused by any illness (http://ww1.heartandstroke.ca/3/4/2005). Clearly, there is a need to invest research efforts into examining ways to prevent the development of this disease.

Abnormally high blood pressure (BP), or 'hypertension', represents a pre-clinical phase of CVD (Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). Hypertension is diagnosed when systolic BP is at or exceeds 140mmHg and/or diastolic BP is at or exceeds 90mmHg. In the early stages of hypertension, BP elevation is most likely due to heightened sympathetic nervous system (SNS) arousal and excessive cardiac output (Linden, in press). However, chronic hypertension tends to result from self-perpetuating physiological mechanisms. For example, physiological alterations such as vascular remodelling, endothelial dysfunction, or coronary artery plaque formation can lead to atherosclerosis and poor regulatory vasodilation, which in turn can increase vascular resistance. These permutations eventually sustain increases

in the renal set-point for BP regulation (leading to hypertension) and also feed back to further maintain peripheral alterations (Folkow, 1990).

*Risk Factors - Role of Stress*

One of the main putative risk factors operating to bring about the peripheral alterations implicated in hypertension development is stress. There are various pathways through which stress can exert its effects to propagate hypertension and eventual CVD (see Figure A1 in the Appendix). In general, research has shown that stress is more likely to foster the development of hypertension when it is prolonged (i.e., chronic stress) and/or when it is in combination with a variety of moderating factors. Such factors include certain medical conditions (e.g., diabetes mellitus, hypercholesteremia), demographic characteristics (e.g., older age, male gender), maladaptive health behaviours (e.g., poor diet, smoking, excessive alcohol use, lack of exercise), psychological dispositions (i.e., anxious, hostile, angry, self-deceptive) or a genetic predisposition (i.e., signalled by a family history of hypertension) (Roy, Steptoe, & Kirschbaum, 1998). All of these risk pathways lead to elevated SNS activity (and eventually to those physiological alterations maintaining hypertension) in the face of stress. In particular, heightened activity is observed in the hypothalamic-pituitary-adrenocortical (HPA) axis, as indicated by enhanced levels of epinephrine, norepinephrine, and cortisol (Lovallo & Gerin, 2003).

In terms of research verifying these pathways, animal research has achieved considerable success at showing associations between stress (chronic stress in particular), noted risk factors, and the peripheral alterations characteristic of a hypertensive state. For example, aversive conditioning models have been shown to produce physiological changes over 6-7 months, and intraspecies dominance hierarchies have been shown to yield similar results over only 4 months (Mancia, Ramirez, Bertinieri, Parati, & Zanchetti, 1984). It has been much more difficult,

however, to document direct associations between stress and later concrete physiological changes in humans, and this is in large part due to methodological factors. For example, in humans it is unethical to administer chronic stress, laborious to monitor long-term changes, and extremely difficult to control stress confounds (such as those occurring outside the laboratory). Researchers have therefore directed their efforts at showing relations between cardiovascular changes induced by stressors in the laboratory and future hypertension status, given that such physiological reactions to stress, when substantially elevated, are hypothesized to be early markers of later physiological permutations (Krantz & Manuck, 1984). Such cardiovascular changes include *cardiovascular reactivity* and *cardiovascular recovery*. To the extent that these cardiovascular indices offer unique predictive power in explaining longitudinal BP change, beyond that offered by resting cardiovascular indices and other standard clinical risk factors, they may be considered clinically useful measures for identifying individuals at risk for CVD.

*Cardiovascular Reactivity*

Cardiovascular reactivity ('reactivity') can be defined as the magnitude of increase of an individual's BP or heart rate (HR) to an aversive, challenging, or engaging behavioural lab stressor (Treiber et al., 2003). It is conceptualized as a relatively stable individual trait characteristic, and has been shown to achieve acceptable levels of reliability (surpassing the 0.8 mark) when values are aggregated across multiple lab tasks (Kamarck & Lovallo, 2003). In the realm of the pathway model that links stress to eventual hypertension, heightened reactivity can be considered a visible marker of the heightened SNS activity that arises in the context of those main risk factors previously discussed (Pickering & Gerin, 1990). (See Figure A1 in the Appendix.)

From their recent review of the literature, Treiber et al. (2003) concluded that reasonable

evidence has accumulated over the past several decades to suggest that heightened reactivity may be considered an independent risk factor for hypertension. Research has frequently shown increased reactivity levels to be associated with hypertension status as well as '*at-risk*' status (signalled by a positive family history of hypertension) (Barnes, Treiber, Musante, Turner, Davis, & Strong, 2000; Light et al., 1999). Moreover, a meta-analytic review of the literature by Fredriksen and Matthews (1990) concluded that heightened reactivity to various cognitive, emotional, and physical lab stressors has been related to future hypertension development in longitudinal studies with follow-up intervals of up to 45 years. In addition to these long-term predictions, recent studies have also shown lab-induced reactivity to be capable of predicting more proximal, continuous changes in BP, such as those spanning 3 years (Treiber et al., 2003). The fact that findings linking heightened reactivity with significant increases in longitudinal BP have been replicated across a variety of studies (that display substantial methodological diversity) argues for the credibility of these findings.

It is clear that there are substantial differences in the nature of the cardiovascular responses evoked by different kinds of laboratory stimuli. Heightened reactivity to lab tasks requiring sustained mental effort (i.e., *cognitive* and *psychosocial tasks*) has generally been associated with significant future BP increases, with reactivity usually explaining 4-12% of BP variance after adjustment for standard clinical risk factors (Light, Dolan, Davis, & Sherwood, 1992). Positive associations have also been reported for *active physical tasks*, such as bike exercise and handgrip tasks (Mathews, Woodall, & Allen, 1993). In contrast, *passive physical manipulations,* such as the cold pressor task, have been far less successful at documenting such associations (Carroll et al., 1996), and this extends to findings with *at-risk* individuals (Schneider et al., 2003).

Unfortunately, regardless of the promising associations documented with psychological and active physical lab stressors, there is still considerable doubt in the literature about the predictive ability of cardiovascular reactivity. First, despite the fairly high prevalence of positive associations documented, inconsistencies have still been noted. Contrasting results have generally tended to come from studies having lower statistical power, usually a result of relatively small sample sizes and/or relatively brief follow-up periods (Treiber et al., 2003). In addition, failure to examine interactions between physiological findings and known moderating factors (i.e., obesity, gender, genetic vulnerability, etc.) has likely contributed to the inconsistent findings (Linden, Earle, Gerin, & Christenfeld, 1997). Second, even when positive associations are found in the context of well designed studies, the utility of the reactivity data is still questioned as many researchers feel that these data provide only a minimal increase in predictive power beyond that provided by traditional BP predictors (Carroll, Smith, Shipley, Steptoe, Brunner, & Marmot, 2001). A study by Langewitz, Rueddel, Schaechinger, and Schmieder (1989), for example, found that reactivity to both a math and cold pressor stressor provided no additional predictive power on any cardiovascular index beyond that provided by resting BP values. Additionally, a recent study by Stewart and France (2001) more specifically found that while HR reactivity to a math task predicted an additional 4% of the variance in 3-year follow-up resting SBP, reactivity from neither a math task nor any active or passive physical tasks added significantly to the prediction of 3-year DBP. In a more recent study by these same authors that assessed reactivity following cognitive tasks in an older population, none of the reactivity measures were found to be predictive of 3-year SBP or DBP (Stewart, Janicki, & Kamarck, in press). Lastly, a previous study in our laboratory found that while reactivity from both a math task and a handgrip task failed to explain additional variance in predicting *baseline* ambulatory

BP beyond traditional predictors, reactivity from an emotion-provoking discussion task was successful in this regard (Linden & Con, 1994).

In summary, while there generally seems to be a significant degree of association between lab-induced reactivity to stress (particularly, active stressors) and later BP levels, there is a lack of clarity regarding the degree and significance of the additional predictive variance explained by this cardiovascular index over that explained by standard clinical risk factors.

*Cardiovascular Recovery*

Another aspect of the cardiovascular stress response that is receiving increased attention is cardiovascular recovery ('recovery'), which refers to the process in which cardiovascular parameters return to initial resting levels after termination of stress. Recovery can be measured in terms of the time it takes for cardiovascular levels to return to initial resting levels following termination of stress, or alternatively it can be measured in terms of the degree of elevation still remaining at specific times post stressor termination. In contrast to reactivity, this stress-response index uniquely taps into the *duration* of the stress response. In the realm of the pathway model that links stress to eventual hypertension, delayed recovery can be considered a visible marker of the increased vascular resistance that eventually results from heightened sympathetic activity. (See Figure A1 in the Appendix.)

While this cardiovascular index is far less researched than reactivity, speculation concerning its importance in relation to disease onset has been around for quite some time. Dating back to 1936, Seyle described the stress response as composed of 3 phases, namely *activation* (i.e., activation of the SNS physiological pathway) *resistance* (i.e., continuation of the activation phase), and *exhaustion* (i.e., depletion of the body's resources to be able to respond to stress and continue SNS activation). He hypothesized that activation enduring for an extended

length of time during the resistance stage was a significant contributor to chronic disease development (Seyle, 1936). Recent reports are similarly now suggesting that in the context of CVD development, significant elevation during this recovery period may be just as important, if not more so, as the initial elevation observed during reactivity (Christenfeld, Glynn, & Gerin, 2000; Schwartz et al., 2003). Indeed, it seems valid that individuals whose BP and HR levels remain elevated for long periods of time following stress termination may be at greater health risk (than those who show similar reactivity but recover more quickly), as their delayed ability to recuperate at a biological level effectively exposes their systems to greater amounts of stress. This may be particularly harmful, as chronically elevated stress levels will ultimately increase the allostatic load on such people's bodily systems (i.e., they will experience enhanced physiological wear and tear resulting from repeated efforts by the body to keep itself in balance in the face of external stress; McEwen, 1998). Finally, it is likely that the combination of heightened reactivity with poor recovery may be particularly detrimental, as poor recovery may accelerate those physiological alterations already initiated by heightened sympathetic activity (i.e., elevated reactivity), thus leading to earlier onset of hypertension in people exhibiting vulnerabilities across both stress-response indices (Stewart et al., in press).

Like reactivity, recovery has been shown to achieve acceptable levels of reliability when values are aggregated across multiple tasks (Rutledge, Linden, & Paul, 2000). Moreover, in terms of its ability to mark disease, delayed recovery has been associated with hypertension status (like reactivity) as well as borderline hypertension (particularly DBP recovery), possibly highlighting an early risk marker role for this parameter (Schuler & O'Brien, 1997). Additionally, slow recovery has been linked to familial hypertension, particularly when 'family history' criteria require *both* parents to be hypertensive (Gerin & Pickering, 1995). Furthermore,

several studies have found recovery to be significantly delayed in stressed individuals displaying other cardiovascular risk factors, such as low fitness level (Jamieson & Lavoie, 1987), heightened anger expression (Lai & Linden, 1992), as well as obesity and male gender (Vitaliano, Russo, Paulsen, & Bailey, 1995).

In terms of the specific literature linking poor recovery with disease, positive associations have been documented across a variety of studies that have assessed an array of different types of stressors. Several of these studies, for example, have shown delayed recovery following *physical exercise* to be associated with increased risk of hypertension development for follow-up periods of up to 10 years (Davidoff, Schamroth, Goldman, Diamond, Cilliers, & Myburgh, 1982; Tanji, Champlin, Wong, Lew, Brown, & Amsterdam, 1989). Moreover, a recent study by Nishime, Cole, Blackstone, Pashkow, and Lauer (2000) found that delayed HR recovery from physical exercise was associated with a 4:1 increased risk of *mortality* in a large sample of middle aged patients. Similarly, a study by Cole, Blackstone, Paashkow, Snader, and Lauer (1999) found that delayed HR recovery following physical exercise was found to be predictive of overall mortality six years later, and this relationship was independent of reactivity. Other evidence attesting to the superiority of recovery over reactivity was found in a study by Borghi, Costa, Boschi, Mussi, and Ambrosioni (1986), where DBP recovery following *mental arithmetic* was found to be significantly predictive of 5-year hypertension development among young borderline hypertensives, and to a greater degree than reactivity. Using a more conservative statistical model, a fairly recent study by Stewart and France (2001) found that delayed SBP recovery from physical tasks (but not a math task) predicted higher SBP three years later in a sample of college students, even *after* adjustment for reactivity values. A more recent study by these same authors, which examined reactivity and recovery responses to other cognitive and psychosocial tasks in

normotensive adults, found that delayed SBP recovery was again the most consistent predictor of 3-year increases in both resting SBP and DBP, providing a significant increase in predictive power above that afforded by baseline predictors (Stewart et al., in press). Lastly, a study by Treiber, Musante, Kapuku, Davis, Litaker, and Davis (2001) that assessed adolescents with a family history of CVD, reported that SBP and HR *aggregate* recovery scores (i.e., scores averaged across physical and psychological tasks) were found to predict resting SBP and HR, respectively, at 4-year follow-up, while aggregate DBP recovery was only observed to predict resting DBP at 1-year follow-up.

While there have been a fair amount of findings linking recovery with future BP levels (such as those mentioned above), there remains substantial ambiguity in the literature. Moreover, even in terms of reactivity versus recovery comparisons, while a variety of studies (including those by Borghi et al, 1986; Stewart & France, 2001; Stewart et al., in press; and Rutledge et al., 2000), have shown recovery to display greater predictive power than reactivity, there are other studies, like that by Treiber et al. (2001), where the opposite has been found. Due to this lack of clarity, a recent review of the literature by Schwartz et al. (2003) concluded that there is weak evidence for the predictive power of recovery. They emphasized that future research must utilize longer follow-up times (to allow for sizeable BP changes to occur) and should evaluate special at-risk groups of individuals (to control for potential moderating factors that might otherwise be influencing cardiovascular responses).

In addition to neglecting longer follow-up durations and lacking emphasis on potential moderating factors, another cause of the ambiguous findings has likely been the inability of lab stressors to effectively stress people to the extent that their vulnerable cardiovascular response profiles (particularly related to recovery) are revealed. Indeed, several researchers have criticized

the weak ecological validity of commonly used lab stressors, and in turn the poor generalizability of reactivity and recovery data to real-world cardiovascular functioning (Schwartz et al., 2003). However, there is some speculation in the field that *interpersonal tasks* (particularly those evoking anger and/or hostility) may be able to overcome this weakness. Such tasks tend to be more representative of the chronic life stressors implicated in CVD development (such as job strain, marital stress, and care-giving burden) than the other cognitive and physical tasks commonly used. Interpersonal tasks have been found to yield significantly elevated reactivity patterns and (particularly) delayed recovery patterns across a variety of studies (Engebretson, Matthews, & Scheier, 1989; Fang & Myers, 2001; Lai & Linden, 1992; Linden et al., 1997), with effect sizes generally larger than those observed with non-emotional tasks (Peiper & Brosschot, in press). Additionally, these tasks have shown relatively high power in predicting future BP changes (Ewart & Kolodner, 1993; Linden, Rutledge, & Con, 1998). Findings from our lab have already shown that, compared to other types of tasks, emotion-evoking interpersonal tasks display higher reproducibility across time (Rutledge, Linden, & Paul, 2001) and yield the strongest predictions of baseline ambulatory BP (Linden & Con, 1994). Additionally, we have found that in addition to elevating reactivity and delaying recovery on measures of BP, these tasks produce similar stress-response profiles on measures of cortisol (a stress hormone), attesting to the enhanced stress-inducing abilities of these tasks (i.e., their ecological validity) (Earle, Linden, & Weinberg, 1999).

Another likely factor contributing to the enhanced ecological validity of these interpersonal tasks, and one which explains the longer recovery periods induced by them, is their ability to make people ruminate about the causes and consequences of their emotional distress (Brosschot & Thayer, 2003). By engaging in such rumination, stress levels and physiological

responses are further perpetuated, and thus recovery is prolonged (Schwartz et al., 2003). The fact that this crucial process of rumination is reflected in recovery (rather than reactivity) profiles further argues for the superiority of recovery (over reactivity) in contributing to hypertension and CVD, and thus in being a useful predictor of long-term BP changes.

*Bringing it all together*

In summary, while there are promising findings in the literature linking reactivity and recovery profiles with future BP changes, more research is needed to clarify, in particular, the predictive power of recovery *in light of* the predictive power already offered by standard clinical risk factors and reactivity indices. Given its enhanced ecological validity, research utilizing emotion-provoking interpersonal lab stressors will likely add clarity in this regard.

In regards to *at-risk* status (i.e., genetic vulnerability due to familial hypertension), many studies have shown that both men and women that are *at-risk* for disease tend to exhibit higher resting levels of BP and HR as well as worse cardiovascular response profiles for reactivity and (particularly) recovery. However, there is still some ambiguity in terms of associations between these response profiles and later hypertension status in these individuals. While some studies have observed significantly worse outcomes associated with both stress-response parameters (when significantly elevated), other studies have specifically only found such outcomes associated with poor recovery profiles, and still others have failed to find such associations for either cardiovascular parameter (Anderson, Lane, Taguchi, Williams, & Houseworth, 1989). As roughly 25% of people with one hypertensive parent and 50% of people with two hypertensive parents eventually develop hypertension themselves (Schneider et al., 2003), it would be particularly useful to be able to identify which of these *at-risk* individuals are most likely to develop disease. Clarifying reactivity and recovery relationships in these individuals may

therefore provide crucial information in this regard.

Lastly, it seems that much of the ambiguity in this field stems in large part from the diverse methodologies used. Subject samples tend to vary in terms of age (university students vs. community-dwelling adults) and risk categories (normotensive vs. borderline vs. *at-risk*). Moreover, lab stress packages vary in their incorporation of physical, cognitive, passive, and psychosocial types of tasks. Furthermore, follow-up data vary in terms of resting versus ambulatory measures, and many studies fail to examine interactions between cardiovascular responses and risk factor moderating variables. As not everybody with heightened reactivity and/or delayed recovery develops hypertension, future studies need to focus on evaluating factors that may differentiate those who do from those who don't. By separating out these factors, research contributions will be less ambiguous and therefore more informative.

*Present Study*

Given the importance of needed changes, the present study has incorporated stringent methodological design features in an attempt to achieve less ambiguous findings. While this study shares similarities with the recent studies by Stewart and France (2001)[1] and Stewart et al. (in press)[2] already mentioned, it nevertheless has many additional strengths.

First, this study examines both young and older individuals. This ability to assess a larger age range is important given that discrepant findings have been observed across studies assessing different age cohorts. Second, the present study examines healthy normotensives as well as normotensives *at-risk* for disease (due to familial hypertension). A third advantage of our study is that it evaluates both 3-year and 10-year follow-up data. Evaluating the former range enables

[1] This study looked at 73 normotensive men and women *aged 18-20yrs*, and assessed predictions of 3yr *resting* BP from baseline reactivity and recovery data collected on a series of cognitive, physical, and *passive* lab tasks.
[2] This study looked at 216 normotensive *community-dwelling* men and women, and assessed predictions of 3yr *resting* BP from baseline reactivity and recovery data collected on a series of computerized cognitive lab tasks and a *non-emotion* provoking speech task.

inspection into continuous changes in BP and HR that may highlight unique pathophysiological mechanisms differentiating those who do versus don't go on to develop hypertension. The latter range is useful because it more accurately resembles the duration of time characterizing the development of hypertension and CVD (Kamarck & Lovallo, 2003), thus allowing for concrete changes in study participants' cardiovascular profiles to develop. Another favourable feature of our study is that it utilizes ambulatory monitoring as a way of determining BP and HR endpoints. This form of measurement provides a better understanding of cardiovascular responses to stress, as sampling in the natural environment allows for aggregation of findings across multiple stimuli, thus reducing the influence of unique situational variance (Kamarck & Lovallo, 2003). Moreover, this form of monitoring has been found to be a better long-term predictor of hypertension development than lab resting measures (Perloff, Sokolow, & Cowan, 1991). Finally, in addition to using a standard physical and cognitive task, we also utilized a psychosocial task involving anger provocation to enhance the ecological validity of our research. By using these three theoretically distinct tasks, we hoped to maximize the variability in our lab stress demands and thus best capture real-life variability in stress-response data.

Through incorporating these advantageous features, it is our hope that more conclusive answers to remaining research questions will be achieved, and that eventually this conclusive information will be utilized in clinical practice where it can achieve practical benefits (in terms of providing information about future risk for clinical events). Specifically, the indication of heightened reactivity and/or delayed recovery following lab-induced stress could serve as an early psychophysiological marker for identifying individuals on the road to developing hypertension (perhaps, particularly in the case of those already having a family history of hypertension). Such a screening process could in turn precipitate pre-emptive treatment for these

vulnerable individuals (in the form of improved diet, increased exercise, SNS-lowering drugs, or a variety of stress reduction therapies), which could ultimately revert the course of their diseases entirely.

*Purpose, Objectives & Hypotheses*

The purpose of our study is to examine whether measures of reactivity to, and recovery from, lab-based challenges are clinically useful in predicting 3-year and/or 10-year changes in ambulatory BP and HR among initially normotensive, community-dwelling adults and university students. Our goal is to determine the best predictor model of longitudinal BP and HR in normotensive individuals based on typical presenting information and readily available stress-response data.

The specific objectives of this study are to answer the following questions:

(i) **Is reactivity to lab-induced stress predictive of significant changes in proximal (i.e., 3-year) and long-term (i.e., 10-year) BP and HR, after considering baseline measures and standard risk factors?** Our goal is to replicate previous findings in the literature that have linked heightened reactivity with future BP increases.

(ii) **Is recovery following lab-induced stress predictive of significant changes in proximal and long-term BP and HR, after considering baseline measures, standard risk factors, and reactivity data?** Our goal is to replicate previous findings in the literature that have linked poor recovery with future BP increases.

(iii) **Which cardiovascular response parameter (reactivity vs. recovery) is most useful in predicting proximal and long-term BP and HR changes?** Given that we have speculated recovery to be superior to reactivity, we hypothesize that within *final* predictor models, recovery will yield higher predictive power than reactivity.

(iv) **What is the implication of *at-risk* status (i.e., having a family history of hypertension) in the prediction of proximal and long-term BP and HR changes from reactivity and recovery data?** We hypothesize that associations between

elevated reactivity/recovery and increased longitudinal BP will be significantly stronger in *at-risk* individuals.

(v) **Which lab task (physical, cognitive, anger-evoking psychosocial, or the aggregated composite) yields the highest correlated scores with longitudinal BP and HR, and thus is considered to be the best source of reactivity/recovery data?** We hypothesize that data from the aggregated composite (i.e., three tasks combined) will be most highly correlated with outcome data, and thus will offer the best overall predictive power. We expect this because aggregate data is able to tap into hyper-responding (i.e., elevations across all three tasks), which is more informative and likely more clinically meaningful than elevated responses to any one task. Of the individual tasks, we expect data from the anger-evoking psychosocial task to be most highly correlated with outcome data, particularly in the case of recovery, as this is where rumination related to the task will show its effects.

## Methods

### *Participants*

Three hundred thirty-four adults participated in the first phase of a three-phase investigation, associating laboratory cardiovascular responses to stress with longitudinal ambulatory BP and HR measurements. Participants consisted of students from the University of British Columbia volunteering in return for course credit and adults from the surrounding Vancouver community recruited through local advertising. In accordance with entrance standards, no participant had a concurrent diagnosis of coronary heart disease or hypertension. For reasons related to satisfying statistical assumptions for data analysis (discussed later in more detail), four participants were removed from the study, leaving 330 individuals (157 men and 173 women, average age = 26.9 years) to compose the initial sample. Of these, 125 participants (38% of the original sample; 60 men and 65 women, average age = 32.7 years) returned after 3 years to undergo the protocol a second time, and 117 participants (35% of original sample; 54 men and 63 women, average age = 40.3 years) returned after a further 7 years (i.e., 10 years from

the initial testing date) to undergo further ambulatory cardiovascular testing. The primary reason for dropout was relocation to another city (i.e., the majority of the sample was comprised of university students, many of whom relocated following graduation). The ethnic composition of the original sample was approximately 70% Caucasian and 30% Asian. All participants provided written informed consent to all procedures. Descriptive statistics for demographic and lifestyle information of the samples across the three testing sessions are presented in Table 1 below. [For a more thorough description of these data, see Table A1 in the Appendix.]

***[Insert Table 1]***

*Procedure*

Baseline testing involved a 2-hour block of laboratory assessment. Participants were directed to not ingest alcohol, caffeine, or nicotine, or to exercise strenuously 2 hours prior to the laboratory session. At first, participants completed questionnaire data on demographic and clinical risk factors, and had their body fat levels measured by a female experimenter. Following this, participants sat alone during a 20-minute resting baseline phase while having their BP and HR measured. After this time, stress testing commenced which consisted of alternating task and recovery periods while cardiovascular readings continued to be taken. Specifically, participants performed a counterbalanced set of 5-minute behavioural stress tasks (mental arithmetic, handgrip exercise, and an emotional anger-recall speech task) during which time reactivity measurements were taken. Recovery was assessed during the 5-minute period following each task. This laboratory stress testing protocol was performed at the year-1 testing session (i.e., baseline) as well as at the year-3 follow-up session. [This latter stress testing protocol was performed strictly to enable assessment of test-retest stability correlations for the reactivity and recovery data; data from this session were not used in hierarchical regression analyses for

predicting longitudinal cardiovascular data.]

At year-3 and year-10 follow-up assessment, ambulatory BP and HR readings were collected over the course of 10-12 hours and 24 hours, respectively. For the purposes of ambulatory monitoring, participants were asked to choose a typical day without specific stressors (such as examinations). Monitors were fitted to participants, pretested on the spot, and returned after monitoring for data analysis. Pretesting, which consisted of the first five ambulatory readings, was completed in the laboratory and was used to determine proper placement of the cuff. Whenever pretest values seemed questionable, the cuff was moved to another location, and the values were compared with Dinamap readings. Ambulatory readings were obtained every 20 minutes.

*Physiological Measurement*

Blood pressure and HR information was collected in the natural environment using SpaceLabs model 90207 ambulatory monitors (Spacelabs Medical Inc., Redmond, WA, USA). These devices weigh approximately 0.7 kg and are worn in a protective pouch. Use and accuracy of the SpaceLabs monitor are supported by validation work (O'Brien, Mee, Atkins, & O'Malley, 1991). Participants were explicitly instructed to minimize physical activity during a measurement cycle and to avoid formal exercise while being monitored. Only approximately 10% of attempted measures were unusable. After the deletion of invalid readings, approximately 30 readings per individual were available for analysis. The primary reason for invalid measures was excessive movement. SpaceLabs monitors provide error codes for failed measurement attempts, thereby facilitating identification of the error. Importantly, our analyses of the ambulatory data did not control for the posture of the participant during readings, a factor known to influence the magnitude of the BP response.

Blood pressure and HR information measured during the laboratory session was collected using a Dinamap 845 Vital Signs Monitor (Critkon Corporation, Tampa, FL.). Validation work has shown the Dinamap to provide BP values that are highly correlated with intra-arterial measurements (Borow & Newburger, 1982). Finally, the Dinamap derives BP and HR values based on oscillometric alogorithms. Existing data suggest that oscillometrically-dependent scores may contribute to better reproducibility over time compared to other laboratory techniques (Swain & Suls, 1996).

*Behavioural Lab Tasks*

Each stress testing session commenced with a 20-minute adaptation phase during which time participants sat in a reclining chair alone in a room while BP and HR measurements were taken. Recordings were made at minutes 0, 4, 8, 12, 16, and 20 during this time. After the final baseline reading had been taken, task instructions were given over an intercom system. Participants completed each of the three 5-minute exercises in randomized order, and a 5-minute recovery period followed the completion of each task (during which time participants were instructed to read magazines). A further 15-minute recovery interval commenced after completion of the final task, and further resting measures were taken at minutes 0, 4, 8, 11, and 14 minutes during this time.

For the isometric handgrip task, participants were required to maintain handgrip tension at 20% maximum for 3 minutes followed by 2 minutes at 30% maximum using a standard dynomanometer (Parker et al., 1987). All participants were able to complete this task. For the mental arithmetic task, participants read problems (from a television and VCR unit) aloud and then verbalized their answers. The arithmetic problems were presented at 5-second intervals (Linden, 1991). Performance data were collected, but task difficulty was not varied on this basis.

On average, participants answered approximately 65% of the questions correctly. For the anger-recall discussion, participants were given 2 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 (Linden & Lamensdorf, 1990). Cardiovascular readings were collected at minutes 1.5 and 3.5 during each exercise and recovery period. Tasks of a cognitive, social-emotional, and physical nature were intentionally used to assess a broader domain of reactivity responses. Additional details regarding the measurement, reliability, and predictive value of these tasks are reported in previous articles (Rutledge, Linden, & Paul, 2002).

*Measurement of Coronary Risk Factor Variables*

As already mentioned, data on some specific lifestyle risk factors and demographic characteristics were assessed by basic questionnaire prior to lab stress testing. These data included information on age, gender, cardiovascular medication use, smoking status, weekly alcohol consumption, weekly exercise frequency, family hypertension history, daily and chronic stress, and body fat (mm skinfold). The family history items inquired whether participants had one or two parents with hypertension. [For reason of statistical power, we clustered participants having one or both hypertensive parent(s) into the FH+ group.] For measurements of daily stress, the Daily Stress Inventory (DSI, Brantley, Waggoner, Jones, & Rappaport, 1987) was used, which is a 60-item self-report measure that describes frustrating events, at least some of which are likely to occur in people's lives. It considers (i) whether or not the event occurred within the past 24 hours and (ii) the stress severity of occurring events on a 1-7 Likert-type rating scale. Lastly, we determined body fat levels by means of a six-site skin calliper test, which was repeated twice and averaged for reliability (Nieman, 1986). Although marginally less accurate than hydrostatic body fat measures, skin calliper assessment is vastly more practical and provides

more specific body fat data compared to crude indices such as the body mass index.

It should be noted that, while data on psychological variables (including depression, anger expression, hostility, self-deception, and impression management) were collected at baseline, these data were not analyzed for the current study. These analyses are currently being performed, and findings will be reported elsewhere at a later time.

*Reactivity and Recovery Calculations*

Reactivity and recovery scores were computed for each of our HR, DBP, and SBP indices. For each cardiovascular index, we computed individual task scores by (i) averaging the two readings for each task (taken at minutes 1.5 and 3.5 of the task), and (ii) subtracting this average score from the index baseline mean. The baseline mean was itself calculated by averaging the last two readings taken during the initial adaptation phase of the lab protocol. Following this procedure, nine reactivity and nine recovery scores remained. Heightened reactivity values indicate relatively large cardiovascular elevations during stress, and heightened recovery values indicate relatively delayed return of cardiovascular levels to normal following stress termination.

Aggregate reactivity and recovery values were also calculated, and these were obtained by summing the individual task change scores across each cardiovascular measure. A total of six aggregate scores were obtained: three recovery scores and three reactivity scores. Research by Kamarck (1992) clearly demonstrates that the reliability of stress-reactivity responses is improved with the aggregation of measures across multiple tasks (Kamarck, 1992). Due to this, and reasons discussed later in the Results section, these aggregate values were used in place of individual task reactivity and recovery data in the regression analyses performed to predict longitudinal ambulatory data. Table 2 below displays mean reactivity and recovery scores

obtained at year-1 from both the individual task and aggregate data.

***[Insert Table 2]***

For this study, simple change scores were used in place of residualized change scores, the latter being change scores that have been further regressed on the respective baseline means. Residualized change scores can be statistically superior to simple change scores because they explicitly adjust for pre-existing BP and HR differences in comparison groups. However, correlations between these two types of scores are known to be extremely high (>.9), and previous studies in our lab as well as that of others (that have utilized both types of change scores) have shown that they yield similar findings and conclusions (Swain & Suls, 1996).

In addition to change scores, recovery can be defined in terms of percentages, slopes, absolute time to recovery, area under the curve, and curve-fitting techniques among other measures. In our data, the lack of moment-to-moment recovery readings reduced the conceptual value of the slope, time to recovery, and curve-fitting estimations. Among the measures that remained, we concluded change scores to be the most reliable index.

*Statistical Analyses*

*Tests of stability.* We assessed the reliability across time of the reactivity and recovery data in our sample by computing test-retest Pearson r correlations for participants completing both phases (year-1 and year-3) of the laboratory stress testing protocol.

*Justification of aggregate data.* We used two techniques to warrant using the aggregate reactivity and recovery data over the individual task data. One analysis used Pearson r correlation coefficients to assess the degree of similarity between individual task and aggregate scores. The other technique used Pearson r correlation coefficients to determine the degree of association between the longitudinal cardiovascular data being predicted in the hierarchical

regression models and total area under the curve data for both the individual task and aggregate data. The latter values were calculated by summing the individual reactivity and recovery raw task scores, and then subtracting respective baseline levels from these composite scores. Area under the curve values were utilized because we wanted to specifically focus on the utility of 'aggregate' data versus 'individual task' data while eliminating the effect of 'reactivity' versus 'recovery' on these analyses. As area under the curve values encompass both reactivity and recovery values, we thought this global value of overall stress-response provided an efficient way of achieving this methodological goal.

*Relationships with coronary risk factors.* We used Pearson r correlation coefficients to assess the relationships between baseline aggregate reactivity and recovery scores with baseline and longitudinal coronary risk factor variables. Because of the large number of correlations calculated, we adjusted our criterion for significance to $p < 0.005$.

*Predicting longitudinal cardiovascular data.* We conducted a series of hierarchical regression analyses to determine the unique contribution of cardiovascular reactivity and recovery data to the prediction of year-3 and year-10 follow-up ambulatory BP and HR after adjustment for standard clinical predictors. In an attempt to satisfy the statistical assumptions for multiple regression techniques, scatterplots of predictor and outcome variables were examined. Four participants were consequently removed from the data set due to outlier effects. We then performed separate regression analyses for each of the three cardiovascular indices at each of the two follow-up periods, yielding a total of six regression models. For each one:

(i) **Year-1 resting BP/HR** and **significant control variables** (i.e., coronary risk factor variables) were forced into the model at step 1. Significant *independent* control variables were determined by an initial analysis in which the year-1 resting index was forced into the model in step 1 and all control variables were forced into the model in step 2. Those

variables that were found to explain uniquely significant variance in longitudinal ambulatory data after accounting for that already explained by the year-1 resting index were thus retained for use in future analyses (entry value: $p<.05$). Other variables were excluded to avoid weakening the power of the regression analyses.

(ii) **Aggregate reactivity** was forced into the model at step 2.

(iii) **Aggregate recovery** was forced into the model at step 3.

(iv) **First-order interactions** (i.e., product terms between the aggregate scores and control variables entered in step 1) were forced into the model at step 4.

While our method of calculating recovery values (i.e., subtracting raw scores from the *baseline* average) increased their independence from reactivity values, our recovery values nevertheless remain somewhat dependent on our reactivity values. This is because reactivity scores effectively determine the starting point (i.e., distance away from baseline) from which the recovery process commences. To determine the degree of interdependence between our (aggregate) reactivity and recovery scores, Pearson r correlations between these constructs were calculated, and these are displayed in Table 3 below. The values suggest that while our reactivity and recovery constructs are highly correlated at the .01 level (with correlations ranging from .68 to .71 for SBP, .67 to .74 for DBP, and .58 to .66 for HR), they nevertheless exhibit some independence. Thus, given that these two constructs appear capable of offering unique information, and that the role of this unique information (particularly for recovery) is what is of crucial interest to us in our longitudinal BP and HR predictions, we felt justified in utilizing both these constructs (in separate steps) in our regression models, regardless of some loss of power due to redundancy. This analytical strategy follows previously established traditions for evaluating the independent contributions of reactivity and recovery in predicting longitudinal BP (Stewart et al., in press).

***[Insert Table 3]***

It should also be noted that in all our analyses, reactivity was always entered into the regression models prior to recovery. The reverse sequence was not tested. We thought this was the most practical way to evaluate the data, since we believe reactivity data will always be used whenever recovery data is utilized. We feel this because we know that (i) reactivity data will always be available (whenever recovery data is acquired); and (ii) as noted in the Introduction, reactivity has already been concluded to be an independent risk factor for (and thus valuable predictor of) hypertension development. Thus, the key question appears to be whether recovery data can provide useful information *above and beyond* reactivity data, and this is why recovery data was forced into the regression models in a separate step behind the reactivity data. [For information on the relative superiority of one stress-response parameter over the other, beta weights for reactivity and recovery in the *final* regression models can be compared, and a discussion of such comparisons can be found in the Results section.]

*Statistical power.* We performed power analyses using SPSS 10.1 Sample Power software (www.spss.com). Because of the large sample for the year-1 testing, power levels to detect medium effect sizes met or exceeded 0.9 for tests of interrelationships with risk factor and psychological variables. Alternatively, because of the much smaller sample sizes at the year-3 and year-10 testing dates, and the relative infrequency of high BP among our subjects, power levels approximated 0.7 for detection of medium effect sizes.

## Results

### *Sample Characteristics*

*Demographic and lifestyle control variables.* Table 1 provides data (means, standard deviations, raw totals, and percentages) on demographic and lifestyle control variables for the

samples across the three testing sessions. As can be seen, an approximately equal split of men and women participated in the protocol across each of the three testing sessions. Hypertensive medication use, which was an exclusion criterion at study entry, is rare across the year-3 and year-10 samples, attesting to the low prevalence of high BP development among the subjects across the follow-up periods. Moreover, data on psychological stress indicate that the year-1 sample displayed, on average, a mild level of daily stress, and the year-3 and year-10 samples endorsed a low frequency (8.3% and 11.9%, respectively) of chronic stress secondary to a general medical condition, together indicating that this study sample did not experience a relatively high level of psychological stress.

Chi-square analyses were performed to assess significant differences across the samples on the categorical control variables. Comparisons revealed that the ratio of males to females as well as smokers to non-smokers did not differ significantly across any of the three samples. It does, however, appear that the year-3 sample had a *moderately* ($X^2$=3.084, p=.07) higher ratio of FH+ individuals (i.e., individuals having a family history of hypertension) to FH- individuals (i.e., individuals not having a family history of hypertension). ANOVA was used to assess significant differences across the three samples on all other control variables. Of these, only age was found to show significant differences across the three testing periods (F= 55.4, p<.001). [For a summary of these significance testing results, please see Table A2 in the Appendix.]

*Cardiovascular variables.* Table 1 also displays average resting and ambulatory BP and HR values across the three testing sessions. ANOVA was again used to assess significant differences across the samples (and these findings are displayed in Table A2 of the Appendix). Analyses revealed that the year-1 resting cardiovascular data were significantly different from the year-3 and year-10 resting data across all three indices. However, with respect to year-3 and

year-10 resting data comparisons, only the HR data showed a significant difference. Moreover, while the year-10 DBP and SBP data show increases over the respective year-3 data, the year-10 HR is surprisingly lower than the year-3 HR. More surprisingly, the ambulatory data display declines across all three indices from the year-3 to the year-10 samples, with both the DBP and HR indices showing significant declines ($F=7.4$, $p<.05$ and $F=13.2$, $p<.001$, respectively). These findings suggest that our final year-10 sample was particularly healthy (probably a reflection of our inclusion/exclusion criteria).

*Participant Attrition*

As can be seen from Table 1, there was a high dropout rate from the original sample at the year-3 (62%) and year-10 (65%) testing dates. Thus, analyses were performed to determine whether or not the participants who dropped out of the study significantly differed in certain ways from those remaining in the study (which would have made those participants remaining in the study unrepresentative of the original sample, and thus threatened the generalizability of the study's findings). Numerous independent samples t-tests and Chi-square tests were therefore performed to assess differences in demographic and lifestyle variable *baseline data* between the dropouts and completers associated with each of the year-3 and year-10 testing dates (i.e., those not going vs. going onto these future follow-up sessions). [Results of these significance tests are presented in Table A3 in the Appendix.]

Comparisons revealed that dropouts and completers (across both follow-up sessions) did not differ significantly with respect to gender, body fat levels, exercise frequency, weekly alcohol consumption, tobacco use, or daily stress levels, suggesting that the sample composition across the three testing dates was similar in these respects. Interestingly, a significant difference in average age was found between dropouts and completers associated with both the year-3

(t=4.2, p<.001) and year-10 (t=3.5, p<.001) samples, highlighting the fact that study completers tended to be older than those dropping out. Significant differences were also observed with the family history data associated with both the year-3 and year-10 comparisons ($X^2 = 6.95$, $p<.05$ and $X^2 = 4.05$, $p<.05$, respectively), highlighting the fact that a higher proportion of FH- individuals dropped out of the study relative to FH+ individuals. In terms of the BP and HR data, the only significant difference between completers and dropouts related to SBP data associated with the year-10 sample, where dropouts were shown to have a significantly lower resting SBP than completers (t=1.98, $p <.05$).

The significant differences found with respect to age and resting SBP are likely insignificant at the clinical level given the relatively small size of the respective raw average differences (see Table A1 of the Appendix). Moreover, while the family history data show significant differences between completers and dropouts, this does not transcend into *significant* differences across the three participant samples. Thus, it appears that the high attrition rate did not significantly change the composition of our study sample (and thus did not seriously threaten the generalizability of our study findings).

*Reactivity and Recovery Data*

Table 2 shows means and standard deviations for *baseline* reactivity and recovery data across the three lab tasks as well as for the aggregate data. Again, data are displayed for the total year-1 sample as well as for only those participants remaining at the year-3 and year-10 follow-up periods. Resting cardiovascular data have also been displayed for comparison purposes.

Individual task reactivity data reveal that across all three samples, the discussion task tends to yield the highest SBP and DBP reactivity scores, while the math task tends to yield the highest HR reactivity scores. Individual task recovery data reveal that across all three samples,

the discussion task again tends to yield the highest SBP and DBP scores, while the handgrip task yields the highest HR recovery scores. While some of these differences are significant, others are negligible. [See Table A4 of the Appendix for significance testing results relating to these comparisons.]

Another trend to note is that across all three tasks, the HR index consistently shows smaller reactivity and recovery scores relative to the BP data. This trend is additionally observed with the aggregate data. Moreover, with both the individual task and aggregate data, there is a tendency for significant differences in reactivity scores across tasks to disappear in respective recovery data comparisons. This is particularly noticeable on the SBP and HR indices. One last observation is that the standard deviations across both the individual task and aggregate reactivity and recovery data are fairly large, suggesting that there were considerable individual differences in cardiovascular responses during stress testing.

Independent samples t-tests were again used to compare the (reactivity and recovery) data of sample dropouts and completers. For the individual task data, all comparisons were not significant except for two (specifically, those between year-3 completers and dropouts on both discussion DBP and SBP reactivity scores, highlighting that dropouts had significantly lower scores on these indices.] For the aggregate data, all but one comparison was not significant; the comparison between year-3 completers and dropouts on aggregate SBP reactivity revealed that dropouts again had significantly lower scores. These findings again support the conclusion that the high participant dropout rate did not seriously threaten the generalizability of the study findings. [Table A5 of the Appendix displays the significance testing results in this regard.]

*Stability of Reactivity and Recovery Data*

Table 4 below displays Pearson r correlations representing test-retest stability values for

the individual task and aggregate reactivity and recovery values. Keeping in mind that reactivity and recovery data are not expected to adhere to perfect test-retest reliability due to changes in physiology over time, these stability correlations appear highly credible.

The reactivity data in particular show statistically significant correlations across all individual tasks and indices (ranging from r = .19 to r = .44), with the exception of the handgrip HR reactivity value which falls below significance. The discussion task appears to show the highest DBP and SBP reactivity stability correlations, while the math task shows the highest HR stability correlation. With respect to the aggregate stability scores, the DBP value shows a highly significant stability correlation, while the SBP and HR values fall just below significance.

The recovery stability correlations display a lower level of significance, with only the discussion SBP value and handgrip DBP and SBP values reaching statistical significance. The handgrip task appears to show the highest DBP and HR recovery stability correlations, while the discussion task displays the highest SBP stability correlation. With respect to the aggregate stability scores, the SBP value shows a highly significant stability correlation, while the DBP and HR correlations fall below significance.

***[Insert Table 4]***

*Justification of Aggregate Data Use*

To determine the validity of using the aggregate reactivity and recovery scores in place of the individual task scores for use in the hierarchical regression models, several analyses were performed. First, correlations were computed between the aggregate data and the respective individual task reactivity and recovery scores to determine the extent to which the aggregate data were representative of data from each of the individual tasks. Findings are presented in Table 5 below. For reactivity, data from the discussion task was most highly correlated with the

aggregate data (showing the highest correlations across both BP indices, but not the HR index). However, data from all the individual tasks highly correlated with the aggregate data (on the respective index), with scores ranging from .63 to .86 (all significant at the .01 level). For recovery, data from the handgrip task was most highly correlated with the aggregate data (showing the highest correlations across both BP indices, but again not the HR index). However, again data from all the individual tasks highly correlated with the aggregate data (on the respective index), with scores ranging from .84 to .89 (all significant at the .01 level). Overall, these findings suggest that the aggregate data sufficiently represent the individual task data.

***[Insert Table 5]***

Another analysis that was performed to assess the suitability of the aggregate data examined associations between total area under the curve (AUC) data (for both individual task and aggregate indices) and longitudinal BP and HR data (specifically, the year-10 ambulatory data). Table 6 below displays the findings of these analyses. For SBP, the aggregate data show a statistically significant correlation ($r = .51$, $p<.01$) with the year-10 SBP data that is similar to those correlations displayed by the individual task data (which are all slightly lower, and range from $r = .44$ to $r = .48$, both $p<.01$). For DBP, the aggregate data again show a statistically significant correlation ($r = .55$, $p<.01$) with the year-10 DBP data that is similar to those correlations displayed by the individual task data (which are all slightly lower, and range from $r = .49$ to $r = .52$, both $p<.01$). Lastly, the same trend is observed on the HR index, as the aggregate data again show a statistically significant correlation ($r = .51$, $p<.01$) with the year-10 HR data that is similar to those correlations displayed by the individual task data (which are all slightly lower, and range from $r = .44$ to $r = .50$, $p<.01$).

Thus, both the individual task data and aggregate data appear to be significantly

associated with the follow-up ambulatory data, and therefore both appear to be valid predictors of such. It is interesting to point out, however, that at the *individual* task level, a different task produced the highest (AUC-ambulatory) correlation across each different cardiovascular index (i.e., the discussion task yielding the highest SBP correlation, the handgrip task yielding the highest DBP correlation, and the math task yielding the highest HR correlation). This suggests that no *individual* task was superior (in yielding highly predictive data) across all cardiovascular indices; rather, each task appeared capable of providing superior predictive information in distinct (cardiovascular) realms. This, together with the fact that the aggregate composite yielded the highest correlations with long-term ambulatory data across all three cardiovascular indices, supports the decision to use the aggregate data in place of the individual task data. The additional fact that aggregate data (in general) demonstrate enhanced reliability further justifies this decision (Kamarck, 1992).

***[Insert Table 6]***

*Correlations between Control Variables and Aggregate Reactivity and Recovery Data*

Analyses were also performed to assess Pearson r correlations between baseline reactivity and recovery data and control variables measured across each of the three testing sessions. Due to the large number of correlations computed, significance was set at the more stringent .01 level to adjust for Type 1 error inflation. Table A6 of the Appendix displays these findings.

For the reactivity data, both the SBP and DBP aggregate scores at each of the three testing sessions are highly correlated with sex, with r values ranging from -.24 to -.30 (all $p < .01$). These correlations tend to reflect the strong association between male gender and heightened reactivity scores. Additionally, baseline smoking status was found to correlate significantly with SBP reactivity, with higher reactivity scores surprisingly being associated with

non-smoking status ($r = .15$, $p<.01$). Lastly, baseline age shows a significant correlation ($r = -.16$, $p<.005$) with HR reactivity, highlighting that increasing age was associated with a lower HR reactivity score.

In contrast to the reactivity data, the recovery data show a dearth of significant associations with control variables. The only correlation to note is the association between participant sex and SBP recovery ($r = -.15$, $p<.01$), highlighting that men tended to take longer to recover on this cardiovascular index than women.

*Impact of Family Hypertension History Status*

Given that one of our goals was to examine the impact of *at-risk* status (due to familial hypertension) on predictions of longitudinal BP and HR, we performed additional analyses looking at the contrasting reactivity and recovery profiles between FH+ and FH- individuals. Recall that in the previous section, family history status was not found to be significantly associated with either of these stress-response parameters. Similarly, the graphs displayed in Figure 1 below (and Figure A2 in the Appendix) add further support to this conclusion.

***[Insert Figure 1]***

The graphs in Figure 1 provide a visual representation of the overall stress-response curve on each cardiovascular index, starting with the pre-task baseline resting BP/HR, moving through the two reactivity raw task measurements, and culminating with the two post-task raw recovery measurements. These curves are based on aggregate data that have been presented to depict a typical set of task scores (i.e., scores have been divided by three). As can be seen, both the SBP and DBP curves show that the FH+ individuals are displaying consistently higher BP, starting at baseline and continuing through the four stress-response measurements. Alternatively, the HR curve shows the opposite trend, with the FH- scores being larger than the FH+ scores at

each measurement point.

A further set of graphs displaying total area under the curve data across each of the three cardiovascular indices was produced, and these can be found in Figure A2 of the Appendix. Again, both the SBP and DBP graphs show elevated levels for the FH+ group while the HR graph shows an elevated level for the FH- group. However, across each index these differences appear negligible.

In conclusion, a family history of hypertension does not appear to significantly impact reactivity and recovery levels but does appear to have a strong influence on overall resting BP. This is particularly clear from the area under the curve data, where differences across FH+ and FH- groups (in data that accounts for both reactivity and recovery scores) appear negligible. Moreover, from the graphs in Figure 1, it appears that any differences in raw reactivity and recovery data that do exist (between FH+ and FH- individuals) are in fact simply extensions of those differences already evident in *resting* cardiovascular levels. Thus, it is likely these resting differences that explain the health vulnerability associated with family hypertension history (and thus these are the measures that appear to be most valuable for use in predictions of longitudinal BP and HR).

*Determination of Control Variables to be used in Hierarchical Regression Models*

Simple 2-step regression models were produced to determine which control variables could explain unique variance in longitudinal BP and HR beyond that already explained by resting levels (and thus which ones should be retained in subsequent hierarchical regression analyses). Table 7 below provides a summary of those variables found to be significant. [Tables A7 and A8 in the Appendix provide a more complete summary of these analyses for the year-3 and year-10 predictions, respectively.]

*[Insert Table 7]*

For the year-3 data, age and body fat level were found to explain significant unique variance in both DBP and SBP predictions. Exercise frequency was additionally found to be significant in the DBP model. No control variable was found to explain unique additional variance in the HR model.

For the year-10 data, age was additionally found to explain significant unique variance in the DBP model. While location (i.e., student vs. community) was also found to be significantly predictive in the DBP model, it was found to highly correlate ($r = .74$, $p < .01$) with age, and so to avoid redundancy, it was not retained in subsequent regression models. For the SBP model, both smoking status and exercise frequency were found to be significant, while for the HR model, sex was found to be the lone significant control variable.

Of the control variables found to be significant, a few were noted to show significant correlations with other variables. Age showed a correlation with body fat level of $r = .24$ ($p<.01$), body fat level additionally showed a correlation with exercise frequency of $r = -.14$ ($p<.05$), and exercise frequency additionally showed a correlation with smoking status of $r = .14$ ($p<.01$). Although significant, all these correlations are still below the .25 level. Thus, they were concluded to not be large enough to warrant exclusion in subsequent regression analyses.

*Predictions of Year-3 HR and BP*

Table 8 below presents the results of the hierarchical linear regression analyses for the year-3 predictions of DBP, SBP, and HR. Across each index, the models are highly significant ($p<.001$) in their prediction of year-3 ambulatory levels at each of the three steps of the model. Moreover, across each index, step 1 (resting index plus significant control variables, if any) appears to explain a highly significant proportion of the dependent variable variance. The HR

model shows the largest value for this, with step 1 explaining 36.1% of the year-3 HR variance. The DBP and SBP models display slightly weaker predictions by this step (34.4% and 29.4%, respectively). While resting cardiovascular levels, age, and body fat were found to predict ambulatory levels in the expected direction (i.e., increasing ambulatory scores were predicted by higher resting cardiovascular levels, lower age, and higher body fat), exercise frequency was found to be predictive in the opposite direction from what was expected (i.e., increasing ambulatory scores were surprisingly predicted by higher exercise frequency).

***[Insert Table 8]***

With respect to step 2, significant improvements to all three predictor models were observed following the addition of reactivity. Across each index, this parameter explained significantly unique variance beyond that explained by the traditional predictors, and this was most significant in the SBP model, where reactivity yielded a 10.6% improvement ($F=21.0$, $p<.001$) to the model. However, the DBP and HR models also displayed noteworthy changes, with values of 7.3% ($F=14.9$, $p<.001$) and 4.3% ($F=8.7$, $p<.005$) improvement, respectively. Furthermore, the addition of recovery in step 3 additionally yielded significant improvements in predictions (after considering traditional predictors as well as reactivity data) across all three models. The percentage improvement in this regard was similar across all three models, with values of 2.3% for SBP ($F=4.8$, $p<.05$), 2.2% for DBP ($F=4.7$, $p<.05$), and 2.0% for HR ($F=4.2$, $p<.05$) being observed. Lastly, it can be noted that across all three models, reactivity and recovery predicted ambulatory variance in the expected direction (i.e., increasing ambulatory scores were predicted by higher reactivity and recovery scores).

*Predictions of Year-10 HR and BP*

Table 9 below presents the results of the hierarchical linear regression analyses for the

year-10 predictions of DBP, SBP, and HR. As with the year-3 data, all three models are highly significant ($p<.001$) in their predictions of year-10 ambulatory levels at each of the three steps of the model. Moreover, step 1 again appears to explain a highly significant proportion of the dependent variable variance. In this case, however, the DBP model shows the largest value, with step 1 explaining 38.8% of the 10-year DBP variance. The HR model shows a slightly weaker prediction (35.8%), and the SBP model shows a smaller prediction still (30.9%). While resting cardiovascular levels, age, and exercise were found to predict ambulatory levels in the expected direction (i.e., increasing ambulatory scores were predicted by higher resting cardiovascular levels, lower age, and lower exercise frequency), tobacco use and sex were both found to be predictive in the opposite direction from what was expected (i.e., increasing ambulatory scores were surprisingly predicted by non-smoking status and female gender).

***[Insert Table 9]***

With respect to step 2, only two of the three models show reactivity explaining significantly unique variance in longitudinal data beyond that already explained by the traditional predictors entered in step 1. In the DBP model reactivity yields a 5.0% improvement ($F=9.7$, $p<.01$), while in the SBP model it yields a 2.7% improvement ($F=4.4$, $p<.05$). Alternatively, in the HR model reactivity fails to explain significantly unique variance, as it yields a mere 1% improvement to the predictor model ($F=0.1$, $p=>.05$). Moreover, in contrast to the year-3 data, recovery fails to explain significantly unique variance in long-term SBP, DBP, and HR when entered in step 3. It yields a 1% improvement to the SBP predictor model ($F=1.6$, $p>.05$), a .7% improvement to the DBP predictor model ($F=1.3$, $p=>.05$), and a mere .4% improvement to the HR predictor model ($F=0.7$, $p=>.05$). Lastly, it can be noted that across all three models, reactivity and recovery predicted ambulatory variance in the expected direction (i.e., increasing

ambulatory scores were predicted by higher reactivity and recovery scores).

*Role of Interactions between Stress-Response Variables and Control Variables*

For sake of thoroughness in our attempts to determine the best predictor models, first order interactions between stress-response indices (i.e., reactivity or recovery) and control variables (i.e., demographic and lifestyle variables entered in step 1) were then entered as predictors in a 4th step across all six models. All of these interactions, however, were found to be insignificant in terms of their ability to enhance the predictor models, suggesting that the utility of reactivity and recovery in predicting 3-year and 10-year ambulatory data does not vary on account of such demographic and risk factor information. [For a summary of these analyses, please see Table A9 in the Appendix.] Thus, according to the data we collected, the best predictor models for the year-3 ambulatory data remain to be those displayed in Table 8, while for the year-10 data, it would be those just consisting of steps 1 and 2 for the SBP and DBP data, and that consisting of just step 1 for the HR data.

*Relative Superiority of Reactivity versus Recovery data in Longitudinal Predictions*

Table 10 below displays standardized beta coefficients, t scores, and p-values for all six *final models* (i.e., inclusive of steps 1 through 3). [Note that data pertaining to control variables has been left out of this table, as its primary purpose is to display the relative contributions of BP and HR data; in particular the reactivity versus recovery data.]

***[Insert Table 10]***

One common trend observed across all six models is that the resting BP/HR values entered in step 1 have the largest beta weights, highlighting that they are predicting the largest amount of variance in ambulatory data. Across all models, these resting data are explaining significantly reliable ($p<.001$) variability in the ambulatory data, with beta weights ranging from

.57 for the year-10 SBP prediction to .69 for the year-3 HR prediction.

Another trend observed across five of the six models is that the recovery data show greater predictive power than the reactivity data. Except for in the year-10 DBP model, where the reverse is observed, the recovery beta weights (ranging from .08 in the year-10 HR model to .22 in the year-3 SBP model) are superior to the reactivity beta weights (ranging from -.02 in the year-10 HR model to .18 in the year-3 SBP model). Moreover, in the three models where recovery data were found to be uniquely predictive in the third step (i.e., all the 3-year models), the reactivity data appear to no longer be significantly predictive in the *final* models. Together, these trends highlight the relative superiority of recovery over reactivity in predicting longitudinal cardiovascular data.

## Discussion

While many previous findings in the literature have associated cardiovascular reactivity and recovery profiles with longitudinal BP and HR changes, there have nevertheless been several inconsistencies (likely owing to poor methodology). Additionally, the predictive power of recovery *in light of* reactivity remains unclear. Furthermore, equivocal findings have been found across studies with respect to how these cardiovascular parameters vary as a function of family hypertension status and source of stress (i.e., type of lab stressor).

In response to a pressing need for well designed research in the cardiovascular stress-response literature, we therefore used a combination of well-controlled laboratory and ecologically valid ambulatory measurement strategies to determine the utility of reactivity and recovery data in predicting changes in longitudinal ambulatory BP and HR. Our aim was to determine the best predictor models for longitudinal BP and HR in normotensive individuals based on typical presenting information and readily available stress-response data. To achieve

this, our research questions focused on assessing whether incorporating reactivity and recovery data into predictor models could significantly improve initial predictions based on traditional predictors (i.e., resting cardiovascular levels and demographic and lifestyle risk factors), and if so, to what degree each of these could do this.

*Strengths of our Study*

Our study took advantage of several unique opportunities. First, we prospectively examined cardiovascular predictions over both a 3-year and a 10-year interval, thus providing data on the ability of our baseline variables to predict both proximal and long-term changes in BP and HR levels. Second, we commenced our study with a fairly large sample size and, while there was a high attrition rate at follow-up sessions (relative to the baseline sample), the follow-up samples were also of decent size. Within our large participant sample, two other unique features were that we started with equal amounts of men and women as well as equal numbers of FH+ and FH- individuals, enabling sufficient power for assessing moderator effects. This is the first study of its kind to begin with equal-sized cells on these critical participant characteristics.

Another advantageous feature of our study is that, in addition to utilizing standardized laboratory measurements, we also utilized ambulatory measurement. As indicated earlier, the higher reliability of this method of assessment relative to clinic measures is clear from prior research, as is its superior predictive validity with respect to disease status and mortality. A further advantage of our study is that, at an individual task level, we assessed cardiovascular responding across three theoretically distinct tasks, which included a psychosocial task involving anger provocation in addition to typical cognitive and physical tasks. By doing this, we maximized variability in our lab stress demands (and thus in our stress-response data), thereby better capturing real-life variability and enhancing the ecological validity of our research. We

further enhanced the reliability of our data by aggregating scores across individual lab tasks. This aggregation step was particularly beneficial to our study, given that reactivity and recovery are both expected to change somewhat over time, and thus the (retest) stability of these data is weakened. By utilizing data that maximize reliability in other ways, we improved the validity of our data.

*Our Findings*

Our study found several interesting and exciting findings, most of which confirmed our hypotheses stated in the Introduction.

First, the predictions yielded by the traditional predictors (i.e., the resting cardiovascular data and demographic/lifestyle data entered in step 1 of the regression analyses) across all six predictor models ranged from 29.4% (with the year-3 SBP model) to 38.8% (with the year-10 DBP model). These findings agree with previous research, which has typically found these variables to explain roughly 30% of the variance in longitudinal cardiovascular data.

Second, both reactivity and recovery were found to significantly improve predictor models when added in steps 2 and 3, respectively, of the year-3 prediction models, thus supporting the utility of both reactivity and recovery data in predicting *proximal* BP and HR changes. The degree of model improvement owing to *reactivity* ranged from 4.3% to 10.6%, which generally agrees with conclusions by Mathews et al. (1993) who reported a range of 4-12% improvement based on their review of the literature. As for the *recovery* findings, they are particularly impressive given the conservative nature of our regression models. We looked at the predictive utility of recovery *beyond reactivity*, something that most studies have not done, as we felt this was the most practical way to consider the data (for reasons previously noted). The fact that we still found significant findings for recovery (despite this more conservative approach)

therefore attests to its true predictive utility, and argues against the conclusion by Schwartz et al. (2003) that it is not yet established as a useful predictive tool.

Third, our results *generally* support the utility of reactivity data in predicting *long-term* cardiovascular changes, but do not support the utility of recovery data in this regard. While reactivity was significantly predictive when added in step 2 of the SBP and DBP predictor models (yielding 2.7% and 5.0% improvement, respectively), this was not the case for the HR predictor model, nor was it the case for any of the predictor models when recovery was additionally incorporated (in the third step). This is the first research to assess *long-term* hierarchical predictions of recovery *above and beyond* predictions by reactivity. Thus, while our findings cannot be compared to previous findings, they are informative on their own as they suggest recovery is not a useful predictive tool (alongside traditional predictors and reactivity) in *long-term* cardiovascular predictions. Further research is obviously needed to substantiate these findings. As for the reactivity data, our BP findings are in agreement with conclusions by Treiber et al. (2003) and Fredriksen and Matthews (1990), who noted that significant associations between reactivity and cardiovascular data hold over long periods of time.

[While the HR reactivity findings are in contrast to the conclusions by Fredriksen, Matthews, and Treiber and colleagues, this is not of serious concern given that the validity of HR data in predicting longitudinal hypertension status is questionable as is. For one, HR information is not included in hypertension diagnostic criteria. Moreover, its validity in reflecting psychological stress is questionable, as demonstrated in a study by Shapiro, Jamner, and Goldstein (1997) that found significant positive correlations between daytime stress and ambulatory SBP and DBP in college students but failed to find such a relationship for HR data. Thus, we are not overly concerned or surprised about the weaker HR findings as the significance

of this index in reflecting mental stress and hypertension is unclear. (For these same reasons, we are also not overly concerned about the weaker reactivity and recovery findings displayed by the HR index in the test-retest stability analyses, the correlational analyses between individual task and aggregate data, the correlational analyses between task/aggregate data and long-term ambulatory data, and the regression analyses for the year-3 predictor model.)]

Fourth, several points can be made with respect to the differences between the year-3 and year-10 prediction models. For one, it is interesting that across the SBP and DBP indices, step 1 is more predictive in the year-10 model (rather than the year-3 model), while for the HR index, the year-3 model shows this step to be more predictive. It is also interesting that across the year-3 data, step 1 was found to be most predictive in the HR model, while across the year-10 data it was most predictive in the DBP model. [Note that if we discount these HR findings due to reasons discussed in the previous paragraph, then a similar trend is observed across both sets of data, with the DBP model displaying the largest step 1 prediction.] Greater consistency across the models was observed for steps 2 and 3, as both these steps were found to yield larger degrees of improvement in all three of the year-3 models relative to the respective year-10 models. For the year-3 data, the SBP model yielded the largest degree of improvement following steps 2 and 3. Alternatively, for the year-10 data, the DBP model produced the largest degree of improvement following step 2 while the SBP model (like the year-3 data) produced the largest degree of improvement following step 3. Overall, the DBP model was found to yield the largest final predictions across both the year-3 and year-10 data (both equating to 44% predictive ability). Across the other two indices, the year-3 data displayed higher predictive ability. These findings generally point to the superiority of the year-3 models over the year-10 models, and to the overall superiority of the DBP model over the other two index models.

Fifth, five of the six *final* regression models confirmed our hypothesis regarding the superiority of recovery over reactivity in predicting ambulatory BP and HR (all except the year-10 DBP model). Our findings are consistent with those of Borghi et al. (1986), Stewart and France (2001), Stewart et al. (in press), and Rutledge et al. (2000), all of who have shown recovery to display greater predictive power than reactivity. As suggested earlier, the superiority of recovery over reactivity may lie in its ability to capture the detrimental activity of rumination following stress. Further research in this regard is needed.

Sixth, our findings pertaining to the effects of family hypertension history on reactivity/ recovery predictions were contrary to expected. While we found significant differences in resting cardiovascular levels between FH+ and FH- individuals, we did not find significant differences in their reactivity or recovery scores. Therefore, it appears that the (disease) vulnerability associated with familial hypertension is based in greater resting BP levels. In other words, reactivity and recovery data do not appear to offer crucial additional information that could help in determining which FH+ individuals are most likely to develop hypertension in the future.

Lastly, it should be noted that we did not end up testing the superiority of the interpersonal task predictions over the handgrip and arithmetic task predictions, as the aggregate data was used in place of the individual task data in the predictor models. Nevertheless, certain strengths can be noted with respect to the interpersonal task data. For example, it yielded the highest reactivity and recovery scores on both the SBP and DBP indices, it showed the highest test-retest reliability correlations for SBP and DBP reactivity as well as for SBP recovery, it yielded the highest AUC-10yr ambulatory correlation on the SBP index, and it produced the highest individual task-aggregate composite correlations for SBP and DBP reactivity as well as for HR recovery. Clearly, utilizing this interpersonal task was beneficial in terms of maximizing

the predictive power of the aggregate data. Also, these findings suggest that researchers who (for time and cost reasons) cannot apply a multi-task protocol would get the "most bang for their buck" if they chose an interpersonal stressor.

*How our Study Compares to Previous Similar Studies*

As mentioned earlier in the Introduction, our study shared several similarities with those of Stewart and France (2001) and Stewart et al. (in press), although several methodological distinctions were noted which we felt made our study more reliable, valid, and powerful.

Our findings are similar to those of Stewart and France (2001) in terms of showing the superiority of recovery over reactivity in proximal predictions. However, the fact that we found this trend across all three cardiovascular indices, whereas they only found it across SBP (but not DBP) suggests that our findings are more conclusive. In particular, they failed to observe both reactivity and recovery improve any of their five DBP models (where each model was based on data from different types of individual stress tasks), and their findings were not highly consistent in terms of reactivity and recovery improving the SBP models (as only two of their five models showed significant improvements). These weaknesses may reflect their lower power consequent to them utilizing individual, non-psychological task data (rather than aggregate, mixed task data) and resting (rather than ambulatory) cardiovascular outcome data. While their overall SBP predictions did explain a sizeably larger amount of variance (>60%) compared to our predictions (~40%), it should be pointed out that their step 1 predictions were much higher than ours (~59% compared to our ~30%). It therefore appears that their higher model predictions were, in large part, a reflection of their more favourable sample composition at baseline.

As for how our data compare to those of Stewart et al. (in press), our findings were similar in terms of failing to find any improvements in the models due to interactions between

reactivity/recovery and control variables. Moreover, our studies showed similarities with respect to the degree of improvement yielded by SBP recovery (as they showed .8-2% improvement in their models while we showed 2.3% improvement in our model). Furthermore, both our studies found recovery to be superior to reactivity in 3-year proximal predictions. However, our findings were again more conclusive as we showed this superiority across all three cardiovascular indices, while Stewart et al. (in press) only found this effect with their SBP models. It is possible that their utilization of non-emotional psychological tasks (rather than emotional tasks) and exclusively community-dwelling (average age = 60.1 years) adults (rather than individuals displaying a more variable age range) lowered their power to find such significant relationships.

*Study Limitations*

There are several limitations to our study. One of these is that participant attrition resulted in much smaller follow-up samples than that existing at baseline, which reduced the power in our statistical analyses. Furthermore, with respect to the composition of these samples, it should be pointed out that relatively few of our participants (i.e., about 20%) developed hypertension over the study period. This indicates that the sample was fairly healthy and thus not entirely representative of the general population in this regard. This seems to have resulted from our selection criteria (which led to participants being relatively young and free of any coronary heart disease or hypertension), which appears to have limited the variability in our outcome data (and our power) with respect to linking stress-response data to disease development. Given that several previous studies that have found significant findings (particularly with respect to recovery) utilized borderline hypertensive participants, it is likely that we would have achieved higher predictive power in our regression models (including more significant power over the longer follow-up period) had we assessed such individuals. Caution should therefore be

exercised when generalizing our findings (to older and more at-risk populations).

Additionally, a few limitations can be noted with respect to our study methodology. For one, our failure to control for caffeine intake or body posture during follow-up ambulatory measurements may have added error to our data, as these factors are known to influence the magnitude of BP responses. [Note, however, that this renders our significant findings more conservative and is therefore not a threat here.] Secondly, our use of the handgrip task as our inducer of physical stress may have prevented us from observing more significant relationships, as other longitudinal studies have tended to use more demanding physical tasks (such as bike exercise). Thirdly, while our choice to aggregate tasks did improve the reliability of our data, it may have weakened our recovery protocol. More specifically, as we aggregated across three tasks that tended to yield diverse reactivity/recovery profiles (two of which showed fast recovery while the other showed slow recovery alongside fast reactivity), our aggregate recovery measures may have been significantly diluted. [Note, however, again that this renders our significant findings more conservative.]

Lastly, there is an unavoidable association between reactivity and recovery. This dependence can lead to redundancy in regression models that ultimately prevent the ability to make conclusions about the *total* predictive ability of recovery in determining longitudinal BP and HR. By forcing recovery into the regression models after independently accounting for reactivity, our recovery step (rather) speaks to the *unique additional* explanatory power of recovery beyond reactivity.

In conclusion, it is possible that we may have achieved even stronger predictions in our study, including significant predictions with the year-10 data, had some of these limitations been avoidable (and thus more power to reveal such significant relationships was present).

*Value of Study*

Despite the limitations noted above, our findings are nevertheless informative and valuable. If cardiovascular reactivity and (particularly) recovery can significantly improve prediction models for longitudinal BP and HR levels (as increasing evidence suggests) as well as disease status and mortality, then their utilization by health professionals in standard clinical practice is highly warranted. As mentioned at the outset, CVD is the leading cause of death in Canada, and it is estimated that 1 in 4 Canadians have some form of heart disease and a much larger percentage display at least one risk factor. If there is indeed a way to improve our methods of identifying at-risk individuals, then these improvements should be utilized so that such individuals can be more easily identified and directed towards appropriate treatments that may prevent or revert their diseases, and ultimately save their lives.

Moreover, in addition to being utilized to identify vulnerable persons, reactivity and recovery may also prove beneficial as indicators of progress in psychological treatment programs aimed at improving stress-response styles and overall health. Such programs, particularly those teaching coping skills and distraction techniques, are likely to be effective in improving cardiovascular recovery as they presumably reduce anger, worry, and ultimately rumination (Peiper & Brosschot, in press). Previous research has indeed already documented improved cardiovascular functioning consequent to engaging in emotional expression (as long as not directed at an authority figure) (Hokanson, Burgess, & Cohen, 1963), forgiveness (Lawler et al., 2003), and regular exercise (Malfatto, Branzi, Riva, Sala, Leonetti, & Facchini, 2002; Sharma, Deepak, Bijlani, & Rao, 2004), as well as following listening to music (particularly classical music) (Chafin, Roy, Gerin, & Christenfeld, 2004) and interacting with pets (Allen, Blascovich, & Mendes, 2002). Of course, future research is needed to assess the degree to which these

therapy techniques reliably and validly alleviate mental stress and improve physiological stress-response profiles.

Lastly, reactivity and recovery information is valuable as it extends beyond the cardiovascular system to other bodily systems and chronic diseases. For example, reactivity and recovery data can be extracted from the endocrine and immune systems and can be applied to such things as blood glucose levels, muscle tension, and asthma (Peiper & Brosschot, in press). Thus, in addition to helping target and treat individuals at risk for (or suffering from) the most dangerous disease in our nation, reactivity and recovery data can also be utilized to improve the lives of a much larger population of individuals (afflicted with a variety of other chronic diseases where stress is known to play a role).

*Future Directions*

Given that more and more evidence has accumulated attesting to the utility of reactivity and recovery data, several future research directions are suggested.

First, as already indicated, future studies utilizing similar methodology (to that used in the present study) should examine the ability of cardiovascular reactivity and recovery to improve predictor models of actual disease status (i.e., hypertension status or cardiovascular disease status). While a recent study by Pieper and Brosschot (in press) concluded that there is modest evidence for recovery showing the ability to predict disease, it is possible that different conclusions will be drawn if better methodological design features (such as the use of aggregate data, ambulatory measures, and ecologically valid stressors) are utilized (Peiper & Brosschot, in press).

Another line of research deserving increased attention relates to the examination of psychological mediators of the relationships assessed in this study. In particular, constructs

relating to prolonged negative affect (such as chronic worry, rumination, preservation, helplessness, hopelessness, hostility, depression, defensiveness, anger, etc.) are likely to reveal pathways through which stress operates to negatively impact physiological systems. Such negative moods and dispositions presumably lead to the appraisal of more situations as stressful, thus resulting in longer and more frequent exposure to stress across the lifetime. Several studies have already found certain of these psychological variables to be associated with impaired cardiovascular functioning (Neumann, Waldstein, Sellers, Thayer, & Sorkin, 2004; Vrijkotte, van Doornen, & de Geus, 2000). Phase 2 of the study presented in this manuscript, which aims to assess many of these mediator relationships in the context of cardiovascular reactivity and recovery, is currently underway.

Future research may also want to focus on identifying circumstances in the natural environment that may be particularly influential in triggering stress-response vulnerabilities observed in the laboratory. While work-related anxiety is anticipated to be one of the big stressors in this regard, other sources of anxiety (such as parenting, relationships, social interactions, performance situations, etc.) deserve increased research attention.

Lastly, given that many researchers in this area have emphasized the importance of HPA axis activity in understanding stress-disease links, future research should increase its focus on the interrelations between cardiovascular, neuroendocrine, and immunological systems (Linden et al., 1997). It is possible, for example, that predictor models (of longitudinal cardiovascular data) will be significantly improved if they include measures of reactivity and recovery across a variety of physiological parameters, rather than just cardiovascular indices.

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Table 1
*Demographic, Lifestyle, and Cardiovascular Characteristics of Participants across the Three Measurement Sessions*

| | **Yr-1 Data [N (%)]** | **Yr-3 Data [N (%)]** | **Yr-10 Data [N (%)]** |
|---|---|---|---|
| Total Sample: | 330 (100) | 125 (100) | 117 (100) |
| Sex: | | | |
| Men | 157 (47.6) | 60 (48.0) | 54 (46.2) |
| Women | 173 (52.4) | 65 (52.0) | 63 (53.8) |
| Location at Baseline: | | | |
| Student | 221 (67.0) | 71 (56.8) | 66 (56.4) |
| Community | 109 (33.0) | 54 (43.2) | 51 (43.6) |
| Antiypertensive medication use | 0 (0.0) | 5 (4.2) | 12 (11.2) |
| Current use of tabacco products | 23 (7.0) | 8 (6.4) | 4 (3.6) |
| Family Hypertension History: | | | |
| FH+ | 164 (50.8) | 75 (60.0) | 68 (58.1) |
| FH- | 159 (49.2) | 50 (40.0) | 49 (41.9) |
| Chronic Disease in last 3 years | ---- | 10 (8.3) | 13 (11.9) |
| | **[M (SD)]** | **[M (SD)]** | **[M (SD)]** |
| Age (years) | 26.9 (11.2) | 32.7 (12.7) | 40.3 (13.3) |
| Students | 21.1 (5.3) | 25.2 (6.8) | 31.9 (6.5) |
| Community | 38.7 (10.9) | 42.5 (11.8) | 51.2 (11.9) |
| Body Fat level (mm) | 113.3 (31.4) | 109.5 (28.3) | 64.7 (24.5)[a] |
| Exercise (hours/week) | 2.6 (1.9) | 2.7 (1.9) | 2.3 (1.8) |
| Alcohol Consumption (drinks/week) | 2.2 (3.9) | 2.2 (4.6) | 3.4 (5.5) |
| Daily stress level (DSI) | 44.7 (27.5) | ---- | ---- |
| Resting Cardiovascular Levels: | | | |
| DBP (mmHg) | 67.0 (9.2) | 71.5 (8.2) | 75.3 (10.5) |
| SBP (mmHg) | 114.9 (11.4) | 120.2 (10.0) | 121.0 (23.8) |
| HR (bpm) | 66.3 (10.9) | 78.5 (10.4) | 70.3 (11.3) |
| Ambulatory Cardiovascular Levels: | | ***10-12hr data:*** | ***12hr data[b]:*** |
| DBP (mmHg) | ---- | 80.8 (8.2) | 77.5 (10.4) |
| SBP (mmHg) | ---- | 127.9 (10.6) | 126.1 (13.9) |
| HR (bpm) | ---- | 78.4 (10.5) | 73.6 (10.0) |

[a]Note that the yr-1 and yr-3 body fat values are based on a six-site measurement summation while the yr-10 body fat values are based on a four-site measurement summation. For this reason, the columns were not compared directly.
[b]Although 24hr ambulatory data was collected at the yr-10 follow-up session, only the 12hr daytime ambulatory data from this session has been displayed above (to enable comparison with the yr-3 follow-up data).

Table 2

*Baseline (i.e., Year-1) Resting BP and HR as well as Reactivity and Recovery Scores Across Both the Individual Lab Tasks and Aggregate Composite (Mean [SD])*

| | Yr-1 Sample | Yr-3 Completers Only | Yr-10 Completers Only |
|---|---|---|---|
| **Resting BP & HR** | | | |
| DBP (mmHg) | 67.0 (9.2) | 67.2 (8.6) | 67.9 (10.4) |
| SBP (mmHg) | 114.9 (11.4) | 114.8 (10.2) | 116.6 (12.5) |
| HR (mmHg) | 66.3 (10.9) | 65.6 (11.0) | 64.7 (11.0) |
| **Individual Task Reactivity** | | | |
| *Discussion Task:* | | | |
| DBP (mmHg) | 17.0 (8.2) | 18.1 (7.1) | 17.5 (7.9) |
| SBP (mmHg) | 17.7 (10.1) | 19.2 (9.7) | 17.5 (8.6) |
| HR (bpm) | 8.8 (6.5) | 9.4 (6.5) | 8.4 (6.7) |
| *Math Task:* | | | |
| DBP (mmHg) | 11.8 (7.1) | 12.1 (6.4) | 12.8 (6.9) |
| SBP (mmHg) | 14.0 (9.3) | 14.7 (9.0) | 14.0 (8.2) |
| HR (bpm) | 10.9 (8.4) | 10.4 (7.3) | 10.7 (7.7) |
| *Handgrip Task:* | | | |
| DBP (mmHg) | 5.6 (6.4) | 6.2 (7.1) | 5.7 (6.4) |
| SBP (mmHg) | 6.5 (8.2) | 7.3 (9.6) | 5.9 (8.0) |
| HR (bpm) | 1.8 (4.9) | 1.7 (4.8) | 1.5 (5.3) |
| **Individual Task Recovery** | | | |
| *Discussion Task:* | | | |
| DBP (mmHg) | 3.9 (5.2) | 4.4 (4.8) | 3.9 (4.6) |
| SBP (mmHg) | 5.2 (7.2) | 5.9 (7.7) | 4.7 (6.7) |
| HR (bpm) | 0.0 (4.4) | 0.1 (4.4) | 0.2 (4.2) |
| *Math Task:* | | | |
| DBP (mmHg) | 2.6 (5.1) | 3.0 (4.7) | 2.9 (5.1) |
| SBP (mmHg) | 4.2 (7.4) | 4.5 (7.8) | 3.8 (6.8) |
| HR (bpm) | 0.5 (4.4) | 0.6 (4.2) | 0.3 (4.1) |
| *Handgrip Task:* | | | |
| DBP (mmHg) | 1.3 (4.9) | 1.7 (4.5) | 1.5 (4.7) |
| SBP (mmHg) | 1.7 (7.2) | 2.6 (7.8) | 1.3 (6.2) |
| HR (bpm) | -0.7 (4.6) | -0.9 (5.1) | -0.7 (4.2) |
| **Aggregate Reactivity** | | | |
| DBP (mmHg) | 34.4 (17.2) | 36.4 (16.6) | 36.0 (17.5) |
| SBP (mmHg) | 38.2 (22.2) | 41.3 (23.4) | 37.4 (20.4) |
| HR (bpm) | 21.4 (15.5) | 21.5 (14.9) | 20.7 (15.4) |
| **Aggregate Recovery** | | | |
| DBP (mmHg) | 7.7 (13.1) | 8.9 (12.0) | 8.3 (12.6) |
| SBP (mmHg) | 11.1 (18.8) | 12.9 (21.0) | 9.8 (16.4) |
| HR (bpm) | -0.1 (11.4) | -0.2 (11.0) | -0.2 (10.7) |

Table 3
*Correlations (r) between Baseline Aggregate Reactivity and Recovery Data*

| | For all 330 Participants at Baseline | For 125 Participants Remaining at Year-3 Follow-Up Only *(Data Used in 3-year Regression Models)* | For 117 Participants Remaining at Year-10 Follow-Up Only *(Data Used in 10-year Regression Models)* |
|---|---|---|---|
| **SBP Reactivity & SBP Recovery** | .71** | .69** | .68** |
| **DBP Reactivity & DBP Recovery** | .68** | .67** | .74** |
| **HR Reactivity & HR Recovery** | .58** | .66** | .63** |

** Correlation is significant at the 0.01 level (2-tailed).

Table 4
*Test-Retest Stability Coefficients (r) for Laboratory Reactivity and Recovery Values over 3 Years*

| | REACTIVITY | RECOVERY |
|---|---|---|
| **Individual Task Values** | | |
| *Discussion Task:* | | |
| DBP (mmHg) | .44** | -.03 |
| SBP (mmHg) | .26** | .27** |
| HR (bpm) | .21* | -.09 |
| *Math Task:* | | |
| DBP (mmHg) | .29** | .16 |
| SBP (mmHg) | .23* | .12 |
| HR (bpm) | .24* | .00 |
| *Handgrip Task:* | | |
| DBP (mmHg) | .41** | .20* |
| SBP (mmHg) | .19* | .23* |
| HR (bpm) | .04 | -.13 |
| **Aggregate Values** | | |
| DBP (mmHg) | .44** | .17 |
| SBP (mmHg) | .18 | .25** |
| HR (bpm) | .13 | -.09 |

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 5
*Correlations (r) between Individual Task and Aggregate Composite Reactivity and Recovery Data*

| | Reactivity Aggregates | | | Recovery Aggregates | | |
|---|---|---|---|---|---|---|
| | **SBP** | **DBP** | **HR** | **SBP** | **DBP** | **HR** |
| **Individual Task Reactivity Data** | | | | | | |
| *Discussion:* | | | | | | |
| DBP (mmHg) | .51** | .83** | .19** | ---- | ---- | ---- |
| SBP (mmHg) | .83** | .48** | .12* | ---- | ---- | ---- |
| HR (bpm) | .17** | .23** | .80** | ---- | ---- | ---- |
| *Math:* | | | | | | |
| DBP (mmHg) | .43** | .80** | .32** | ---- | ---- | ---- |
| SBP (mmHg) | .83** | .49** | .33** | ---- | ---- | ---- |
| HR (bpm) | .26** | .30** | .86** | ---- | ---- | ---- |
| *Handgrip:* | | | | | | |
| DBP (mmHg) | .39** | .74** | .21** | ---- | ---- | ---- |
| SBP (mmHg) | .75** | .39** | .14* | ---- | ---- | ---- |
| HR (bpm) | .10 | .13* | .63** | ---- | ---- | ---- |
| **Individual Task Recovery Data** | | | | | | |
| *Discussion:* | | | | | | |
| DBP (mmHg) | ---- | ---- | ---- | .33** | .87** | .10 |
| SBP (mmHg) | ---- | ---- | ---- | .86** | .33** | .05 |
| HR (bpm) | ---- | ---- | ---- | .06 | .14* | .87** |
| *Math:* | | | | | | |
| DBP (mmHg) | ---- | ---- | ---- | .26** | .85** | .13* |
| SBP (mmHg) | ---- | ---- | ---- | .85** | .25** | .06 |
| HR (bpm) | ---- | ---- | ---- | .06 | .14* | .85** |
| *Handgrip:* | | | | | | |
| DBP (mmHg) | ---- | ---- | ---- | .29** | .89** | .20** |
| SBP (mmHg) | ---- | ---- | ---- | .86** | .29** | .07 |
| HR (bpm) | ---- | ---- | ---- | .06 | .14* | .84** |

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 6

*Correlations (r) between Year-10 Longitudinal Ambulatory Cardiovascular Data and Area Under the Curve Data for Individual Task and Aggregate Indices*

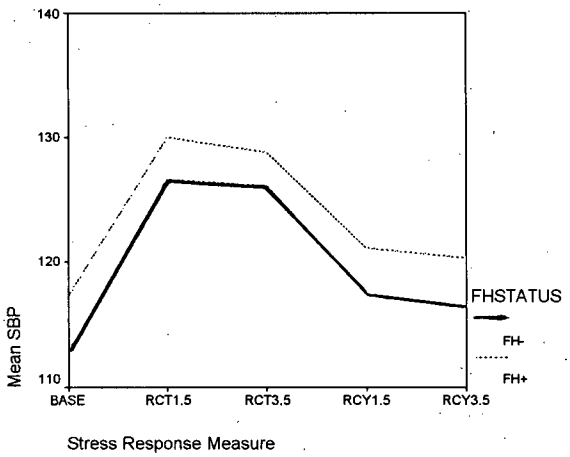
| | Year-10 SBP | Year-10 DBP | Year-10 HR |
|---|---|---|---|
| **Individual Task Data:** | | | |
| *Handgrip Task:* | | | |
| DBP AUC (mmHg) | ---- | .52**[a] | ---- |
| SBP AUC (mmHg) | .47** | ---- | ---- |
| HR AUC (mmHg) | ---- | ---- | .47** |
| *Math Task:* | | | |
| DBP AUC (mmHg) | ---- | .52** | ---- |
| SBP AUC (mmHg) | .44** | ---- | ---- |
| HR AUC (mmHg) | ---- | ---- | .50** |
| *Discussion Task:* | | | |
| DBP AUC (mmHg) | ---- | .49** | ---- |
| SBP AUC (mmHg) | .48** | ---- | ---- |
| HR AUC (mmHg) | ---- | ---- | .44** |
| **Aggregate Composite Data:** | | | |
| DBP AUC (mmHg) | ---- | .55** | ---- |
| SBP AUC (mmHg) | .51** | ---- | ---- |
| HR AUC (mmHg) | ---- | ---- | .51** |

[a] While this handgrip task correlation appears to be equal to the respective math task correlation (displayed in the row directly below), please note that at the 3 decimal level the handgrip task show a superior correlation.

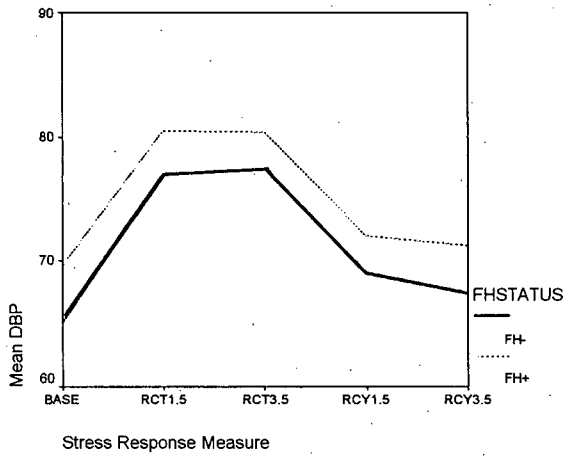
** Correlation is significant at the 0.01 level (2-tailed).

*Figure 1.* Stress Response Curves for Aggregate data in Relation to Family Hypertension Status

**SBP Curve:**




**DBP Curve:**




**HR Curve:**

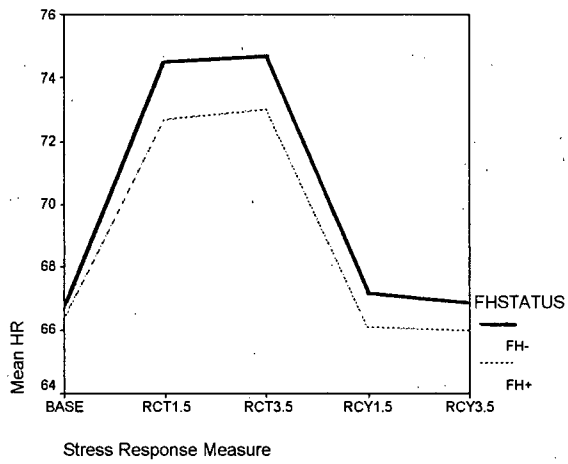


Table 7

*Results of Simple Regression Analyses for Determining Significant Control Variables in Predicting Longitudinal BP and HR*

| Year-3 Data: | | | Year-10 Data: | | |
|---|---|---|---|---|---|
| **Regression Model Variables** | **β[a]** | **T and ρ Values** | **Regression Model Variables** | **β** | **T and ρ Values** |
| **Yr-3 DBP:** | | | **Yr-10 DBP:** | | |
| Sex | .-.05 | -0.6 (.519) | Sex | .01 | 0.1 (.946) |
| FH Status | .03 | 0.4 (.711) | FH Status | -.04 | -0.5 (.651) |
| Location | .18 | 1.7 (.090) | Location | .29 | *2.5* ***(.014)*** |
| Age | -.22 | -2.0 **(.046)** | Age | -.29 | -2.5 **(.015)** |
| Tabacco Use | -.01 | -0.2 (.812) | Tabacco Use | -.09 | 1.1 (.266) |
| Body Fat | .13 | 1.5 (.142) | Body Fat | -.01 | 0.1 (.904) |
| Exercise Freq. | .15 | 2.0 **(.048)** | Exercise Freq. | -.02 | -0.2 (.838) |
| Alcohol Use | .08 | 1.0 (.339) | Alcohol | .01 | -0.1 (.933) |
| Daily Stress | -.00 | -0.0 (.988) | Daily Stress | -.05 | -0.7 (.509) |
| **Yr-3 SBP:** | | | **Yr-10 SBP:** | | |
| Sex | -.06 | -0.6 (.527) | Sex | .01 | 0.1 (.892) |
| FH Status | .12 | 1.4 (.161) | FH Status | -.05 | -0.5 (.594) |
| Location | .17 | 1.5 (.125) | Location | .22 | 1.8 (.074) |
| Age | -.26 | -2.4 **(.020)** | Age | -.04 | -0.3 (.731) |
| Tabacco Use | .11 | 1.3 (.192) | Tabacco Use | .20 | 2.4 **(.018)** |
| Body Fat | .23 | 2.6 **(.009)** | Body Fat | .02 | 0.3 (.793) |
| Exercise Freq. | .04 | 0.5 (.600) | Exercise Freq. | -.18 | -2.1 **(.039)** |
| Alcohol Use | .07 | 0.9 (.389) | Alcohol | .07 | 0.8 (.443) |
| Daily Stress | -.05 | -0.6 (.532) | Daily Stress | -.03 | -0.4 (.704) |
| **Yr-3 HR:** | | | **Yr-10 HR:** | | |
| Sex | .14 | 1.7 (.083) | Sex | .22 | 2.5 **(.013)** |
| FH Status | .04 | 0.5 (.603) | FH Status | .01 | 0.1 (.928) |
| Location | .12 | 1.2 (.247) | Location | .19 | 1.6 (.103) |
| Age | -.16 | -1.6 (.120) | Age | -.16 | -1.3 (.188) |
| Tabacco Use | -.08 | -1.0 (.310) | Tabacco Use | -.02 | -0.2 (.857) |
| Body Fat | .09 | 1.1 (.269) | Body Fat | -.03 | -0.4 (.703) |
| Exercise Freq. | .05 | 0.6 (.564) | Exercise Freq. | .01 | 0.1 (.907) |
| Alcohol | -.01 | -0.1 (.941) | Alcohol | .11 | 1.2 (.221) |
| Daily Stress | .01 | 0.1 (.900) | Daily Stress | -.05 | -0.6 (.553) |

Note. Significant findings (at the .05 level) are indicated in bold font.

[a]β = Standardized Beta Coefficient.

Table 8

*Results of Hierarchical Linear Regression Analyses Predicting Year-3 BP and HR from Baseline Resting Measures, Significant Control Variables, and Aggregate Reactivity and Recovery Data*

| Regression Model | B | β | T and p Values | Multiple $R^2$ | F and ρ Values | Change in $R^2$ | Change in F and ρ |
|---|---|---|---|---|---|---|---|
| **Year-3 DBP Prediction:** | | | | | | | |
| (1) | ---- | --- | ---- | .344 | 21.0 (.000) | .344 | 21.0 (.000) |
| Baseline Resting DBP | .60 | .62 | 7.4 (.000) | ---- | ---- | ----- | ---- |
| Significant Control Variables: | | | | | | | |
| Age | -.04 | -.06 | -0.8 (.445) | ---- | ---- | ---- | ---- |
| Exercise Frequency | .62 | .15 | 2.0 (.046) | ---- | ----- | ---- | ---- |
| (2) Aggregate DBP Reactivity | .14 | .28 | 3.9 (.000) | .417 | 21.3 (.000) | .073 | 14.9 (.000) |
| (3) Aggregate DBP Recovery | .14 | .21 | 2.2 (.032) | .440 | 18.5 (.000) | .022 | 4.7 (.032) |
| **Year-3 SBP Prediction:** | | | | | | | |
| (1) | ---- | ---- | ---- | .294 | 16.6 (.000) | .294 | 16.6 (.000) |
| Baseline Resting SBP | .56 | .54 | 6.7 (.000) | ---- | ---- | ---- | ---- |
| Significant Control Variables: | | | | | | | |
| Age | -.11 | -.13 | -1.5 (.124) | ---- | ---- | ---- | ---- |
| Body Fat | .08 | .23 | 2.8 (.005) | ---- | ---- | ---- | ---- |
| (2) Aggregate SBP Reactivity | .18 | .33 | 4.6 (.000) | .399 | 19.8 (.000) | .106 | 21.0 (.000) |
| (3) Aggregate SBP Recovery | .15 | .22 | 2.2 (.031) | .423 | 17.3 (.000) | .023 | 4.8 (.031) |
| **Year-3 HR Prediction:** | | | | | | | |
| (1) Baseline Resting HR | .57 | .60 | 8.3 (.000) | .361 | 68.9 (.000) | .361 | 68.9 (.000) |
| (2) Aggregate HR Reactivity | .15 | .21 | 2.9 (.004) | .404 | 40.9 (.000) | .043 | 8.7 (.004) |
| (3) Aggregate HR Recovery | .19 | .20 | 2.1 (.043) | .424 | 29.4 (.000) | .020 | 4.2 (.043) |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table 9

*Results of Hierarchical Linear Regression Analyses Predicting Year-10 BP and HR from Baseline Resting Measures, Significant Control Variables, and Aggregate Reactivity and Recovery Data*

| Regression Model | B | β | T and p Values | Multiple $R^2$ | F and p Values | Change in $R^2$ | Change in F and p |
|---|---|---|---|---|---|---|---|
| **Year-10 DBP Prediction:** | | | | | | | |
| (1) | ---- | ---- | ---- | .388 | 34.9 (**.000**) | .388 | 34.9 (**.000**) |
| Baseline Resting DBP | .55 | .65 | 8.1 (**.000**) | ---- | ---- | ---- | ---- |
| Significant Control Variables: | | | | | | | |
| Age | -.06 | -.09 | -1.2 (.252) | ---- | ---- | ---- | ---- |
| (2) Aggregate DBP Reactivity | .11 | .23 | 3.1 (**.002**) | .438 | 28.3 (**.000**) | .050 | 9.7 (**.002**) |
| (3) Aggregate DBP Recovery | .08 | .12 | 1.1 (.261) | .444 | 21.6 (**.000**) | .007 | 1.3 (.261) |
| **Year-10 SBP Prediction:** | | | | | | | |
| (1) | ---- | ---- | ---- | .309 | 16.1 (**.000**) | .309 | 16.1 (**.000**) |
| Baseline Resting SBP | .49 | .51 | 6.3 (**.000**) | ---- | ---- | ---- | ---- |
| Significant Control Variables: | | | | | | | |
| Tobacco Use | 8.30 | .18 | 2.2 (**.030**) | ---- | ---- | ---- | ---- |
| Exercise Frequency | -1.01 | -.18 | -2.2 (**.029**) | ---- | ---- | ---- | ---- |
| (2) Aggregate SBP Reactivity | .09 | .17 | 2.1 (**.039**) | .336 | 13.5 (**.000**) | .027 | 4.4 (**.039**) |
| (3) Aggregate SBP Recovery | .10 | .15 | 1.3 (.207) | .346 | 11.2 (**.000**) | .010 | 1.6 (.207) |
| **Year-10 HR Prediction:** | | | | | | | |
| (1) | ---- | ---- | ---- | .358 | 30.7 (**.000**) | .358 | 30.7 (**.000**) |
| Baseline Resting HR | .51 | .58 | 7.6 (**.000**) | ---- | ---- | ---- | ---- |
| Significant Control Variables: | | | | | | | |
| Sex | 3.38 | .18 | 2.3 (**.023**) | ---- | ---- | ---- | ---- |
| (2) Aggregate HR Reactivity | .02 | .03 | 0.4 (.722) | .359 | 20.3 (**.000**) | .001 | 0.1 (.722) |
| (3) Aggregate HR Recovery | .07 | .08 | 0.8 (.413) | .363 | 15.4 (**.000**) | .004 | 0.7 (.413) |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table 10

*Relative Predictive Power of Resting Cardiovascular Indices, Reactivity Indices, and Recovery Indices Across the Six Regression Models*

| Regression Model | Resting β | Reactivity β | Recovery β | Resting T and p | Reactivity T and p | Recovery T and p |
|---|---|---|---|---|---|---|
| **Year-3 DBP Prediction:** | .69 | .14 | 21 | 8.7 (.000) | 1.5 (.133) | **2.2 (.032)** |
| **Year-3 SBP Prediction:** | .56 | .18 | .22 | 7.5 (.000) | 1.8 (.074) | **2.2 (.031)** |
| **Year-3 HR Prediction:** | .69 | .09 | .20 | 9.3 (.000) | 1.0 (.313) | **2.1 (.043)** |
| **Year-10 DBP Prediction:** | .74 | .15 | .12 | 9.0 (.000) | **1.4 (.168)** | 1.1 (.261) |
| **Year-10 SBP Prediction:** | .57 | .08 | .15 | 6.7 (.000) | 0.7 (.497) | **1.3 (.207)** |
| **Year-10 HR Prediction:** | .60 | -.02 | .08 | 7.4 (.000) | -0.2 (.830) | **0.8 (.413)** |

Note. For the T and p comparisons, the higher predictive power between reactivity and recovery is indicated in bold font.

# APPENDIX

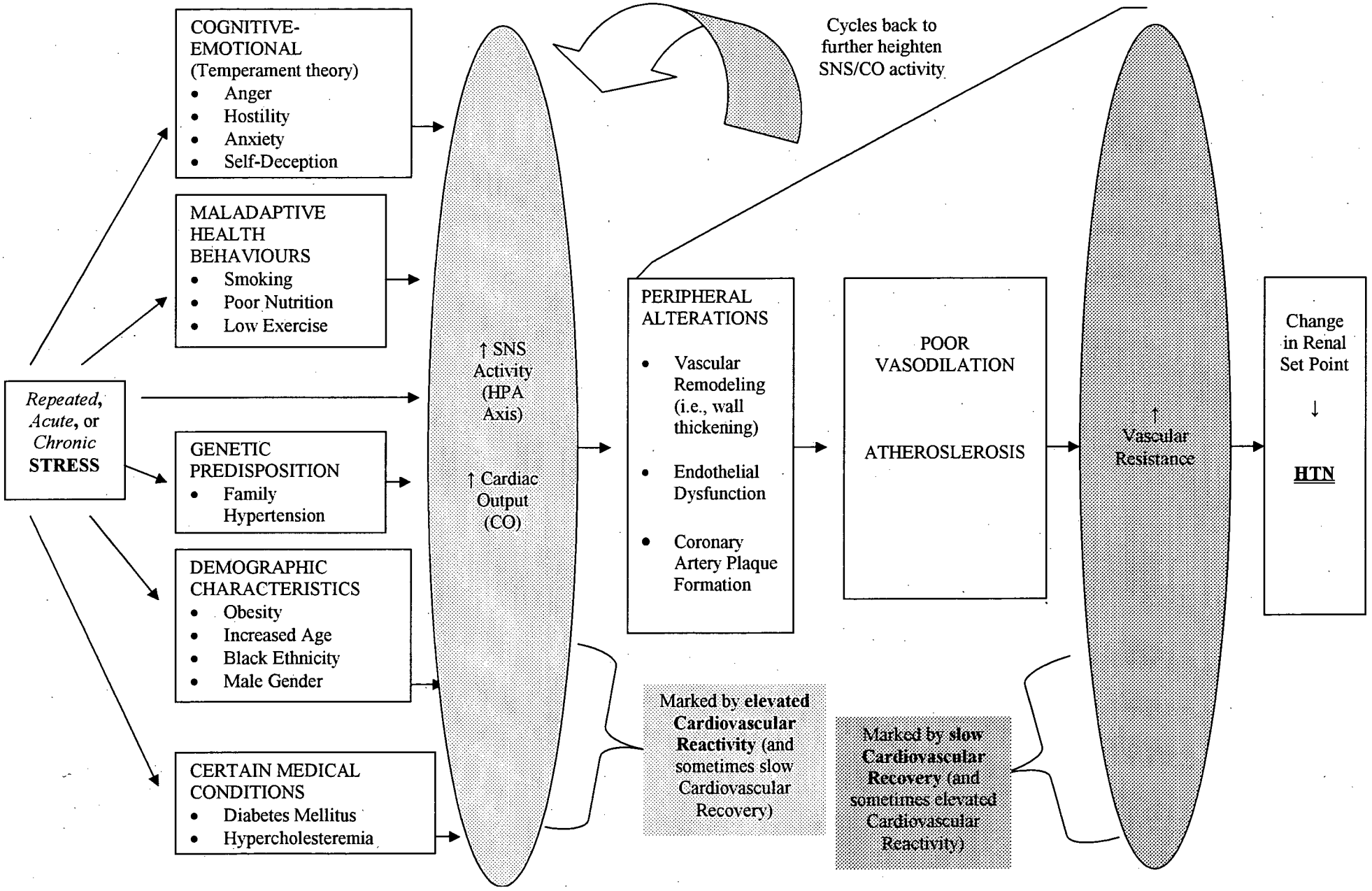



*Figure A1.* Pathways through which Stress Operates to Propagate Hypertension Development

Table A1

*Demographic, Lifestyle, and Cardiovascular Characteristics* [N (%) or M (SD)[a]] *of Participants across the Three Measurement Sessions*

| | Yr-1 Data: | | | Yr-3 Data: | | Yr-10 Data: |
|---|---|---|---|---|---|---|
| | **Original Sample** | **Only Yr-3 Completers** | **Only Yr-10 Completers** | **Yr-3 Sample** | **Only Yr-10 Completers** | **Yr-10 Sample** |
| Total Sample: | 330 (100) | 125(100) | 117(100) | 125 (100) | 117 (100) | 117 (100) |
| Sex: | | | | | | |
| Men | 157 (47.6) | 60 (48.0) | 54 (46.2) | 60 (48.0) | 33 (28.0) | 54 (46.2) |
| Women | 173 (52.4) | 65 (52.0) | 63 (53.8) | 65 (52.0) | 37 (31.4) | 63 (53.8) |
| Location at Baseline: | | | | | | |
| Student | 221 (67.0) | 71 (56.8) | 66 (56.4) | ---- | ---- | ---- |
| Community | 109 (33.0) | 54 (43.2) | 51 (43.6) | ---- | ---- | ---- |
| Antiypertensive medication use | 0 (0) | 0 (0) | 0 (0) | 5 (4.2) | 3 (4.3) | 12 (11.2) |
| Current use of tabacco products | 23 (7.0) | 6 (4.8) | 7 (6.0) | 8 (6.4) | 3 (4.3) | 4 (3.6) |
| Family Hypertension History: | | | | | | |
| FH+ | 164 (50.8) | 75 (60.0) | 68 (58.1) | 75 (60.0) | 44 (62.9) | 68 (58.1) |
| FH- | 159 (49.2) | 50 (40.0) | 49 (41.9) | 50 (40.0) | 26 (37.1) | 49 (41.9) |
| Chronic Disease in last 3 years | ---- | ---- | ---- | 10 (8.3) | 4 (5.7) | 13 (11.9) |
| Age (years) | 26.9 (11.2) | 29.3 (12.6) | 30.0 (13.2) | 32.7 (12.7) | 35.9 (13.9) | 40.3 (13.3) |
| Students | 21.1 (5.3) | 21.8 (6.7) | 21.5 (6.2) | 25.2 (6.8) | 26.2 (8.0) | 31.9 (6.5) |
| Community | 38.7 (10.9) | 39.1 (11.8) | 40.9 (11.7) | 42.5 (11.8) | 44.6 (12.2) | 51.2 (11.9) |
| Body Fat level (mm) | 113.3 (31.4) | 115.7 (29.9) | 116.4 (30.8) | 109.5 (28.3) | 113.9 (29.5) | 64.7 (24.5)[b] |
| Exercise (hours/week) | 2.6 (1.9) | 2.5 (2.0) | 2.4 (2.0) | 2.7 (1.9) | 2.6 (2.0) | 2.3 (1.8) |
| Alcohol Consumption (drinks/week) | 2.2 (3.9) | 1.9 (3.0) | 2.4 (4.3) | 2.2 (4.6) | 2.1 (3.8) | 3.4 (5.5) |
| Daily stress level (DSI) | 44.7 (27.5) | 43.9 (26.1) | 41.7 (21.7) | ---- | ---- | ---- |
| Resting Cardiovascular: | | | | | | |
| DBP (mmHg) | 67.0 (9.2) | 67.2 (8.6) | 67.9 (10.4) | 71.5 (8.2) | 73.0 (7.7) | 75.3 (10.5) |
| SBP (mmHg) | 114.9 (11.4) | 114.8 (10.2) | 116.6 (12.5) | 120.2 (10.0) | 122.3 (8.7) | 121.0 (23.8) |
| HR (bpm) | 66.3 (10.9) | 65.6 (11.0) | 64.7 (11.0) | 78.5 (10.4) | 78.1 (11.4) | 70.3 (11.3) |
| Ambulatory Cardiovascular: | | | | ***10-12hr data:*** | | ***12hr data:*** |
| DBP (mmHg) | ---- | ---- | ---- | 80.8 (8.2) | 81.7 (7.9) | 77.5 (10.4) |
| SBP (mmHg) | ---- | ---- | ---- | 127.9 (10.6) | 129.3 (10.2) | 126.1 (13.9) |
| HR (bpm) | ---- | ---- | ---- | 78.4 (10.5) | 77.9 (11.2) | 73.6 (10.0) |

[a]The top half of the table (from 'Total Sample' down to 'Chronic Disease') refers to N(%) data, while the bottom half of the table (from 'Age' down to 'Ambulatory Cardiovascular' data) refers to M (SD) data.

[b]Yr-1 and Yr-3 body fat level values are based on 6-site measurement summation, while Yr-10 body fat level values are based on 4-site measurement summation. For this reason, the columns should not be compared directly.

Table A2

*Significance Testing Results for Demographic and Resting Cardiovascular Data Across the 3 Measurement Sessions*

| Variable | Overall F and p | P-values for Yr-1 vs. Yr-3 Comparison | P-values for Yr-1 vs. Yr-10 Comparison | P-values for Yr-3 vs. Yr-10 Comparison |
|---|---|---|---|---|
| Age | 55.4 **(<.001)** | **<.001** | **<.001** | **<.001** |
| Body Fat | 1.1 (>.05) | >.05 | ---- | ---- |
| Exercise | 1.3 (>.05) | >.05 | >.05 | >.05 |
| Alcohol | 2.1 (>.05) | >.05 | >.05 | >.05 |
| Smoking | ---------- | >.05 ($X^2$ = .11) | >.05 ($X^2$ = 1.04) | >.05 ($X^2$ = .40) |
| Family Hx | ---------- | *~.07 ($X^2$ = 3.08)* | >.05 ($X^2$ = 1.90) | >.05 ($X^2$ = .07) |
| Sex | ---------- | >.05 ($X^2$ = .04) | >.05 ($X^2$ = .09) | >.05 ($X^2$ = .17) |
| Resting SBP | 9.9 **(<.001)** | **<.005** | **<.005** | >.05 |
| Resting DBP | 29.8 **(<.001)** | **<.001** | **<.001** | >.05 |
| Resting HR | 53.1 **(<.001)** | **<.001** | **<.001** | **<.01** |
| Amb. SBP | 1.2 (>.05) | ---- | ---- | >.05 |
| Amb. DBP | 7.4 **(<.05)** | ---- | ---- | **<.01** |
| Amb. HR | 13.2 **(<.001)** | ---- | ---- | **<.001** |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table A3

*Significance Testing Results for Demographic and Lifestyle Baseline (i.e., Year-1) Data Across Study Completers and Dropouts at Both Follow-Up Sessions*

| Variable | Year-3 Completers vs. Dropouts | Year-10 Completers vs. Dropouts |
|---|---|---|
| Age | T = 4.2, **p <.001** | T = 3.5, **p<.002** |
| Body Fat Level | T = 1.7, p >.05 | T = 1.3, p>.05) |
| Weekly Exercise Frequency | T = -0.6, p>.05 | T = -1.0, p>.05 |
| Weekly Alcohol Consumption | T = 0.6, p>.05 | T = 0.1, p>.05 |
| Daily Stress Inventory | T = -0.8, p>.05 | T = -1.5, p>.05 |
| Sex | $X^2$ = 0.09, p>.05 | $X^2$ = 0.19, p>.05 |
| Smoking Status | $X^2$ = 2.01, p>.05 | $X^2$ = 0.04, p>.05 |
| Family History of Hypertension | $X^2$ = 6.95, **p<.05** | $X^2$ = 4.05, **p<.05** |
| SBP | T = 0.9, p=>.05 | T = 2.0, **p<.05** |
| DBP | T = 1.6, p>.05 | T = 1.2, p>.05 |
| HR | T = -1.1, p>.05 | T = -1.9, p>.05 |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table A4

*Significance Testing Results Pertaining to Comparisons of Reactivity and Recovery Data Across the Three Individual Laboratory Tasks*

| Variable | Overall F and p | P-values for Discussion Task vs. Math Task | P-values for Discussion Task vs. Handgrip Task | P-values for Handgrip Task vs. Math Task |
|---|---|---|---|---|
| **Baseline Sample:** | | | | |
| SBP Reactivity | 122.3 **(<.001)** | **<.001** | **<.001** | **<.001** |
| DBP Reactivity | 198.0 **(<.001)** | **<.001** | **<.001** | **<.001** |
| HR Reactivity | 158.3 **(<.001)** | **<.001** | **<.001** | **<.001** |
| SBP Recovery | 19.3 **(<.001)** | >.05 | **<.001** | **<.001** |
| DBP Recovery | 20.7 **(<.001)** | **<.01** | **<.001** | **<.01** |
| HR Recovery | 6.0 **(<.005)** | >.05 | >.05 | **<.01** |
| **3-YR Completers Only:** | | | | |
| SBP Reactivity | 51.6 **(<.001)** | **<.001** | **<.001** | **<.001** |
| DBP Reactivity | 95.6 **(<.001)** | **<.001** | **<.001** | **<.001** |
| HR Reactivity | 73.3 **(<.001)** | >.05 | **<.001** | **<.001** |
| SBP Recovery | 5.8 **(<.005)** | >.05 | **<.01** | >.05 |
| DBP Recovery | 10.4 **(<.001)** | **<.05** | **<.001** | >.05 |
| HR Recovery | 3.3 **(<.05)** | >.05 | >.05 | **<.05** |
| **10-Yr Completers Only:** | | | | |
| SBP Reactivity | 60.3 **(<.001)** | **<.01** | **<.001** | **<.001** |
| DBP Reactivity | 81.2 **(<.001)** | **<.001** | **<.001** | **<.001** |
| HR Reactivity | 59.6 **(<.001)** | **<.05** | **<.001** | **<.001** |
| SBP Recovery | 8.4 **(<.001)** | >.05 | **<.001** | **<.05** |
| DBP Recovery | 7.9 **(<.001)** | >.05 | **<.001** | >.05 |
| HR Recovery | 1.9 (>.05) | >.05 | >.05 | >.05 |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table A5

*Significance Testing Results for Reactivity & Recovery Baseline (i.e., Year-1) Data Across* Study Completers and Dropouts at Both Follow-Up Sessions

| | Year-3<br>Completers vs. Dropouts | Year-10<br>Completers vs. Dropouts |
|---|---|---|
| **Discussion Task:** | | |
| DBP Reactivity | T = 2.1, **p<.05** | T = 0.8, p>.05 |
| SBP Reactivity | T = 2.2, **p<.05** | T = -0.2, p>.05 |
| HR Reactivity | T = 1.3, p>.05 | T = -0.8, p>.05 |
| DBP Recovery | T = 1.4, p>.05 | T = 0.2, p>.05 |
| SBP Recovery | T = 1.3, p>.05 | T = -0.9, p>.05 |
| HR Recovery | T = 0.3, p>.05 | T = 0.6, p>.05 |
| **Math Task:** | | |
| DBP Reactivity | T = 0.6, p>.05 | T = 2.0, p>.05 |
| SBP Reactivity | T = 1.2, p>.05 | T = 0.1, p>.05 |
| HR Reactivity | T = -0.7, p>.05 | T = -0.2, p>.05 |
| DBP Recovery | T = 1.1, p>.05 | T = 0.8, p>.05 |
| SBP Recovery | T = 0.6, p>.05 | T = -0.7, p>.05 |
| HR Recovery | T = 0.1, p>.05 | T = -0.9, p>.05 |
| **Handgrip Task:** | | |
| DBP Reactivity | T = 1.4, p>.05 | T = 0.3, p>.05 |
| SBP Reactivity | T = 1.5, p>.05 | T = -1.0, p>.05 |
| HR Reactivity | T = -0.2, p>.05 | T = -0.6, p>.05 |
| DBP Recovery | T = 1.2, p>.05 | T = 0.4, p>.05 |
| SBP Recovery | T = 1.7, p>.05 | T = -0.8, p>.05 |
| HR Recovery | T = -0.6, p>.05 | T = 0.0, p>.05 |
| **Aggregate Composite:** | | |
| DBP Reactivity | T = 1.7, p>.05 | T = 1.2, p>.05 |
| SBP Reactivity | T = 2.0, **p<.05** | T = -0.5, p>.05 |
| HR Reactivity | T = 0.1, p>.05 | T = -0.6, p>.05 |
| DBP Recovery | T = 1.3, p>.05 | T = 0.5, p>.05 |
| SBP Recovery | T = 1.4, p>.05 | T = 0.9, p>.05 |
| HR Recovery | T = -0.1, p>.05 | T = -0.1, p>.05 |

Note. Significant findings (at the .05 level) are indicated in bold font.

Table A6

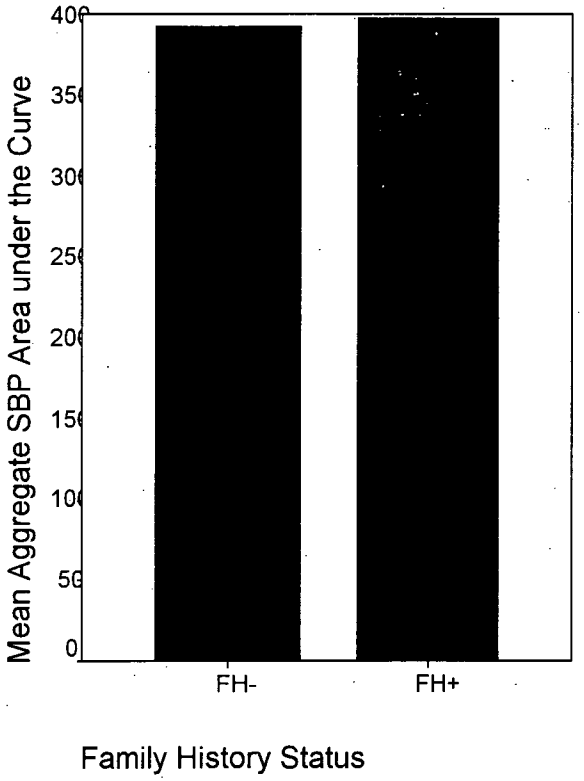
*Correlations (r) between Aggregate (Reactivity and Recovery) Predictors and Demographic/Lifestyle Control Variables*

| | Reactivity Aggregates | | | Recovery Aggregates | | |
|---|---|---|---|---|---|---|
| | **SBP** | **DBP** | **HR** | **SBP** | **DBP** | **HR** |
| **Year-1 Control Variables** | | | | | | |
| Sex | -.27*** | .27*** | -.04 | -.15** | .00 | .05 |
| Location (Student vs. Community) | .06 | -.08 | -.10 | .04 | .03 | -.05 |
| Smoking Status | .15** | .08 | .05 | .12 | .03 | -.02 |
| Family History Status | -.11 | -.10 | -.14 | -.08 | -.12 | -.10 |
| Age | .03 | -.06 | -.16*** | .02 | .01 | -.09 |
| Body Fat Level | -.08 | -.06 | -.03 | -.03 | .06 | .06 |
| Weekly Exercise Frequency | -.02 | -.09 | .09 | .02 | -.08 | .07 |
| Weekly Alcohol consumption | -.02 | -.03 | -.00 | -.06 | -.02 | .08 |
| Daily stress Level | -.11 | -.07 | -.08 | -.03 | -.08 | -.04 |
| **Year-3 Control Variables** | | | | | | |
| Sex | -.27*** | -.30** | .01 | -.19 | -.05 | .11 |
| Smoking Status | .05 | -.05 | .05 | .00 | -.02 | -.03 |
| Family History Status | -.08 | -.11 | -.16 | -.09 | -.19 | -.23 |
| Age | -.01 | -.17 | -.23 | .02 | -.09 | -.18 |
| Body Fat Level | -.07 | -.07 | -.19 | .07 | -.07 | -.13 |
| Weekly Exercise Frequency | .20 | -.19 | .06 | -.14 | -.18 | .02 |
| Weekly Alcohol consumption | -.05 | -.01 | .05 | -.06 | .04 | .01 |
| Antihypertensive Medication Use | -.03 | .03 | .12 | .04 | -.04 | .11 |
| Past Chronic Disease | .00 | .21 | .14 | -.02 | .22 | .17 |
| **Year-10 Control Variables** | | | | | | |
| Sex | -.24** | .27*** | -.04 | -.08 | -.05 | -.00 |
| Smoking Status | .07 | -.13 | -.01 | .13 | -.13 | -.04 |
| Age | .09 | -.03 | -.21 | .07 | .03 | -.15 |
| Body Fat Level | .07 | .11 | -.09 | .12 | .01 | -.04 |
| Weekly Exercise Frequency | .24 | -.21 | -.01 | -.24 | -.06 | .04 |
| Weekly Alcohol consumption | -.04 | .01 | -.05 | -.20 | -.07 | -.07 |
| Antihypertensive Medication Use | -.07 | -.05 | .14 | -.09 | .02 | .17 |
| Past Chronic Disease | .13 | -.02 | .01 | -.09 | -.07 | -.01 |

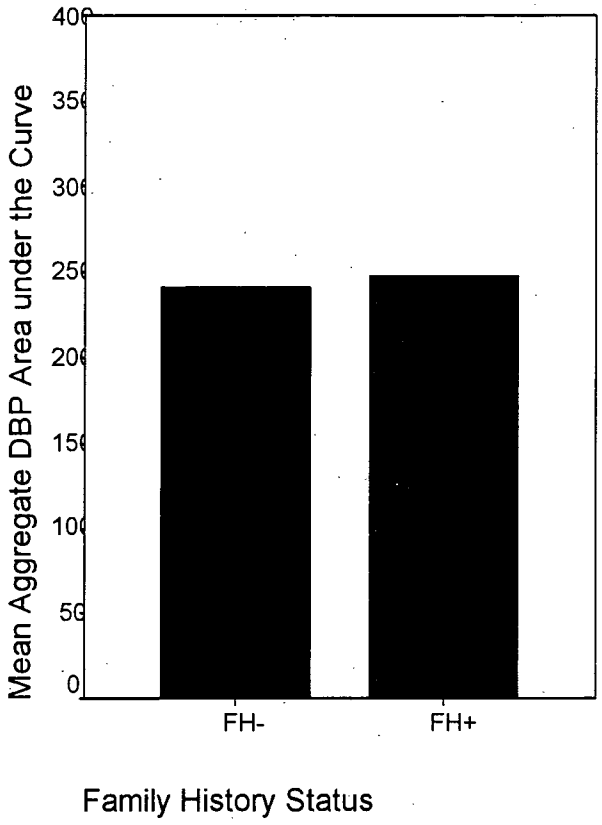
*** Correlation is significant at the 0.005 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

**SBP Data:**




**DBP Data:**




**HR Data:**

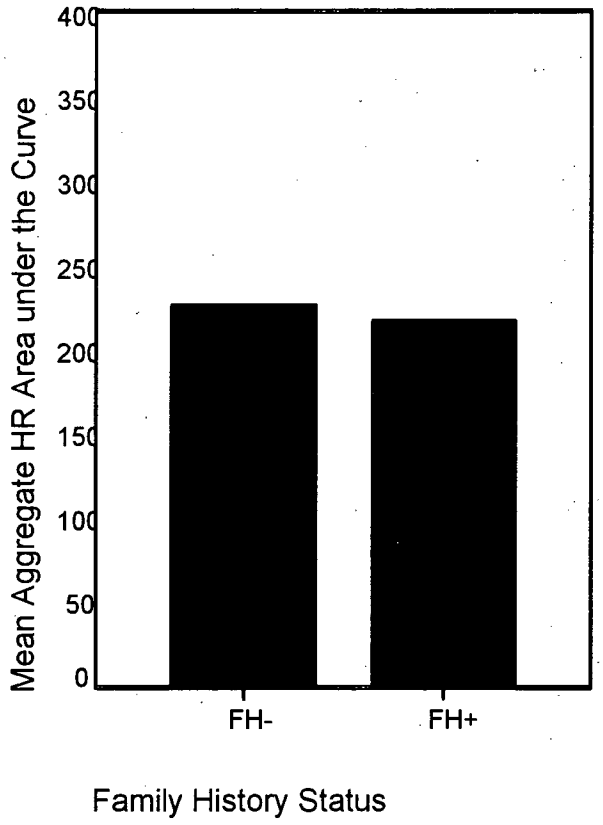



*Figure A2.* Mean Area under the Curve (Aggregate) data in Relation to Family Hypertension Status

Table A7

*Results of Simple Regression Analyses for Determining Significant Control Variables in Predicting Year-3 BP and HR*

| Regression Model | B | β | T and ρ Values | Multiple $R^2$ | F and ρ Values | Change in $R^2$ | Change in F and ρ |
|---|---|---|---|---|---|---|---|
| **Yr-3 DBP Prediction:** | | | | | | | |
| (1) Baseline resting DBP | .54 | .57 | 8.0 **(.000)** | .319 | 57.6 **(.000)** | .319 | 57.6 **(.000)** |
| (2) Control Variables: | ---- | ---- | ---- | .330 | 7.1 **(.000)** | .065 | 1.3 (.223) |
| Sex | -.85 | -.05 | -0.6 (.519) | ---- | ---- | ---- | ---- |
| Family History | .50 | .03 | 0.4 (.711) | ---- | ---- | ---- | ---- |
| Location | 3.00 | .18 | 1.7 (.090) | ---- | ---- | ---- | ---- |
| Age | -.14 | -.22 | -2.0 **(.046)** | ---- | ---- | ---- | ---- |
| Tabacco | -.72 | -.02 | -0.2 (.812) | ---- | ---- | ---- | ---- |
| Body Fat | .03 | .13 | 1.5 (.142) | ---- | ---- | ---- | ---- |
| Exercise | .63 | .15 | 2.0 **(.048)** | ---- | ---- | ---- | ---- |
| Alcohol | .21 | .08 | 1.0 (.339) | ---- | ---- | ---- | ---- |
| Daily Stress | .00 | -.00 | -0.0 (.988) | ---- | ---- | ---- | ---- |
| **Yr-3 SBP Prediction:** | | | | | | | |
| (1) Baseline resting SBP | .51 | .49 | 6.3 **(.000)** | .243 | 39.6 **(.000)** | .243 | 39.6 **(.000)** |
| (2) Control Variables: | ---- | ---- | ---- | .347 | 6.1 **(.000)** | .104 | 2.1 **(.044)** |
| Sex | -1.22 | -.06 | -0.6 (.527) | ---- | ---- | ---- | ---- |
| Family History | 2.49 | .12 | 1.4 (.161) | ---- | ---- | ---- | ---- |
| Location | 3.56 | .17 | 1.5 (.125) | ---- | ---- | ---- | ---- |
| Age | -.22 | -.26 | -2.4 **(.020)** | ---- | ---- | ---- | ---- |
| Tabacco | 5.26 | .11 | 1.3 (.192) | ---- | ---- | ---- | ---- |
| Body Fat | .08 | .23 | 2.6 **(.009)** | ---- | ---- | ---- | ---- |
| Exercise | .22 | .04 | 0.5 (.600) | ---- | ---- | ---- | ---- |
| Alcohol | .25 | .07 | 0.9 (.389) | ---- | ---- | ---- | ---- |
| Daily Stress | -.02 | -.05 | -0.6 (.532) | ---- | ---- | ---- | ---- |
| **Yr-3 HR Prediction:** | | | | | | | |
| (1) Baseline resting HR | .58 | .60 | 8.4 **(.000)** | .364 | 70.3 **(.000)** | .364 | 70.3 **(.000)** |
| (2) Control Variables: | ---- | ---- | ---- | .411 | 8.0 **(.000)** | .047 | 1.0 (.431) |
| Sex | 2.87 | .14 | 1.7 (.083) | ---- | ---- | ---- | ---- |
| Family History | .87 | .04 | 0.5 (.603) | ---- | ---- | ---- | ---- |
| Location | 2.51 | .12 | 1.2 (.247) | ---- | ---- | ---- | ---- |
| Age | -.14 | -.16 | -1.6 (.120) | ---- | ---- | ---- | ---- |
| Tabacco | -3.84 | -.08 | -1.0 (.310) | ---- | ---- | ---- | ---- |
| Body Fat | .03 | .09 | 1.1 (.269) | ---- | ---- | ---- | ---- |
| Exercise | .23 | .05 | 0.6 (.564) | ---- | ---- | ---- | ---- |
| Alcohol | -.02 | -.01 | -0.1 (.941) | ---- | ---- | ---- | ---- |
| Daily Stress | .00 | .01 | 0.1 (.900) | ---- | ---- | ---- | ---- |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table A8

*Results of Simple Regression Analyses for Determining Significant Control Variables in Predicting Year-10 BP and HR*

| Regression Model | B | β | T and ρ Values | Multiple $R^2$ | F and ρ Values | Change in $R^2$ | Change in F and ρ |
|---|---|---|---|---|---|---|---|
| **Yr-10 DBP Prediction:** | | | | | | | |
| (1) Baseline resting DBP | .52 | .62 | 8.2 **(.000)** | .379 | 67.2 **(.000)** | .379 | 67.2 **(.000)** |
| (2) Control Variables: | ---- | ---- | ---- | .429 | 7.6 **(.000)** | .050 | 1.0 (.465) |
| Sex | .10 | .01 | 0.1 (.946) | ---- | ---- | ---- | ---- |
| Family History | -.62 | -.04 | -0.5 (.651) | ---- | ---- | ---- | ---- |
| Location | 4.88 | .29 | 2.5 ***(.014)*** | ---- | ---- | ---- | ---- |
| Age | -.19 | -.29 | -2.5 **(.015)** | ---- | ---- | ---- | ---- |
| Tabacco | 3.01 | -.09 | 1.1 (.266) | ---- | ---- | ---- | ---- |
| Body Fat | .00 | -.01 | 0.1 (.904) | ---- | ---- | ---- | ---- |
| Exercise | -.07 | -.02 | -0.2 (.838) | ---- | ---- | ---- | ---- |
| Alcohol | -.01 | .01 | -0.1 (.933) | ---- | ---- | ---- | ---- |
| Daily Stress | -.02 | -.05 | -0.7 (.509) | ---- | ---- | ---- | ---- |
| **Yr-10 SBP Prediction:** | | | | | | | |
| (1) Baseline resting SBP | .48 | .51 | 6.1 **(.000)** | .255 | 37.6 **(.000)** | .255 | 37.6 **(.000)** |
| (2) Control Variables: | ---- | ---- | ---- | .351 | 5.5 **(.000)** | .096 | 1.7 (.108) |
| Sex | .30 | .01 | 0.1 (.892) | ---- | ---- | ---- | ---- |
| Family History | -1.09 | -.05 | -0.5 (.594) | ---- | ---- | ---- | ---- |
| Location | 5.04 | .22 | 1.8 (.074) | ---- | ---- | ---- | ---- |
| Age | -.04 | -.04 | -0.3 (.731) | ---- | ---- | ---- | ---- |
| Tabacco | 9.42 | .20 | 2.4 **(.018)** | ---- | ---- | ---- | ---- |
| Body Fat | .01 | .02 | 0.3 (.793) | ---- | ---- | ---- | ---- |
| Exercise | -.99 | -.18 | -2.1 **(.039)** | ---- | ---- | ---- | ---- |
| Alcohol | .18 | .07 | 0.8 (.443) | ---- | ---- | ---- | ---- |
| Daily Stress | -.02 | -.03 | -0.4 (.704) | ---- | ---- | ---- | ---- |
| **Yr-10 HR Prediction:** | | | | | | | |
| (1) Baseline resting HR | .50 | .57 | 7.3 **(.000)** | .329 | 54.0 **(.000)** | .329 | 54.0 **(.000)** |
| (2) Control Variables: | ---- | ---- | ---- | .392 | 6.5 **(.000)** | .063 | 1.2 (.324) |
| Sex | 4.16 | .22 | 2.5 **(.013)** | ---- | ---- | ---- | ---- |
| Family History | .15 | .01 | 0.1 (.928) | ---- | ---- | ---- | ---- |
| Location | 3.77 | .19 | 1.6 (.103) | ---- | ---- | ---- | ---- |
| Age | -.12 | -.16 | -1.3 (.188) | ---- | ---- | ---- | ---- |
| Tabacco | -.58 | -.02 | -0.2 (.857) | ---- | ---- | ---- | ---- |
| Body Fat | -.01 | -.03 | -0.4 (.703) | ---- | ---- | ---- | ---- |
| Exercise | .05 | .01 | 0.1 (.907) | ---- | ---- | ---- | ---- |
| Alcohol | .23 | .11 | 1.2 (.221) | ---- | ---- | ---- | ---- |
| Daily Stress | -.02 | -.05 | -0.6 (.553) | ---- | ---- | ---- | ---- |

Note. Significant findings (at the .05 level or smaller) are indicated in bold font.

Table A9

*Hierarchical Linear Regression Model Data Pertaining to Interactions Between Reactivity/Recovery Data and Significant Control Variables (After Being Forced into the Models at Step 4)*

| Regression Model | Change in $R^2$ | Change in F and p | B | β | T and p Values |
|---|---|---|---|---|---|
| **Year-3 DBP Prediction:** | | | | | |
| Interactions: | .013 | 0.7 (.621) | | | |
| Age X DBP Reactivity | | | .00 | .09 | 0.3 (.745) |
| Exercise X DBP Reactivity | | | .03 | .34 | 1.5 (.145) |
| Age X DBP Recovery | | | -.00 | -.07 | -0.3 (.769) |
| Exercise X DBP Recovery | | | -.05 | -.25 | -1.3 (.211) |
| **Predicting Year-3 SBP:** | | | | | |
| Interactions: | .010 | 0.5 (.739) | | | |
| Age X SBP Reactivity | | | .00 | .01 | 0.0 (.981) |
| Body Fat X SBP Reactivity | | | .00 | .39 | 1.1 (.277) |
| Age X SBP Recovery | | | -.00 | -.10 | -0.3 (.738) |
| Body Fat X SBP Recovery | | | -.00 | -.23 | -0.5 (.586) |
| **Predicting Year-10 DBP:** | | | | | |
| Interactions: | .009 | 0.8 (.441) | | | |
| Age X SBP Reactivity | | | .00 | .03 | 0.1 (.918) |
| Age X SBP Recovery | | | .01 | .23 | 0.8 (.400) |
| **Predicting Year-10 SBP:** | | | | | |
| Interactions: | .035 | 1.4 (.231) | | | |
| Tobacco X SBP Reactivity | | | -.03 | -.12 | -0.1 (.950) |
| Exercise X SBP Reactivity | | | .01 | .06 | 0.3 (.788) |
| Tobacco X SBP Recovery | | | .52 | 1.47 | 1.2 (.247) |
| Exercise X SBP Recovery | | | -.05 | -.21 | -1.1 (.281) |
| **Predicting e Year-10 HR:** | | | | | |
| Interactions: | .015 | 1.3 (.273) | | | |
| Sex X HR Reactivity | | | .15 | .42 | 1.2 (.231) |
| Sex X HR Recovery | | | -.28 | -.51 | -1.6 (.113) |