PSYCHOLOGICAL RESPONSE STYLES AND CARDIOVASCULAR HEALTH: CONFOUND OR INDEPENDENT RISK FACTOR?

by

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B.A., University of Alaska Anchorage, 1994
M.A., University of British Columbia, 1996

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

in

THE FACULTY OF GRADUATE STUDIES

(Department of Psychology)

We accept this thesis as conforming
to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

August, 1998

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Date August 13, 1998

DE-6 (2/88)

Abstract

We used the results from two large scale cardiovascular investigations as a platform for examining ways in which psychological response style measures could improve the prediction of cardiovascular health outcomes. Of particular focus was the long-standing conceptual controversy over whether response styles are better treated as confounds to the self-report of stress-related personality characteristics or as separate personality traits. Study 1 consisted of a 3-year prospective study of ambulatory blood pressure levels in a healthy adult sample of males and females (N=125). Study 2 comprised a pharmacological treatment study among ischemic heart disease patients (N=95). Questionnaire batteries completed in each study included self-report measures of depression, anger expression, daily stress, and hostility, along with self-deception and impression management response style scales. In each study, we investigated direct relationships between the response style measures and cardiovascular outcomes, moderator relationships between response style x psychological risk factor interactions and cardiovascular endpoints, and finally between the psychological risk factors and cardiovascular measures after statistically extracting response style variance from the psychological risk factor scores.

Results most strongly supported the main effects model. Higher self-deception scores predicted elevated 3-year diastolic and systolic blood pressure means in study 1, and poorer treatment outcomes in study 2. In both investigations these relationships proved stable after controlling for baseline cardiovascular standing. Importantly, efforts to statistically control for response style effects within the psychological risk factors did not improve predictive power with these measures. The above findings favor efforts to treat response styles as potentially independent psychological contributors to cardiovascular health outcomes, and support ongoing attempts to identify biobehavioral mechanisms through which personality dispositions may impact the appearance or progression of disease.

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Acknowledgement

Please note: The use of the term "we" in the ensuing text is meant to acknowledge the efforts of those without whom this thesis would not have been possible. This study was given life by the existence of two large scale data files diligently conceptualized and collected over the course of nearly a decade. It would be an error of gross proportions on my part to claim or even imply sole responsibility for this work. Many hands helped set the scene for the story told in the following pages, among them: Andrea Con, M.A., Carmen Stossel, B.A., Joseph Lenz, Ph.D., Richard Davies, M.D., Jeffery Maurice, B.A., and especially the enterprising vision of my supervisor Dr. Wolfgang Linden. My apologies to those I've overlooked.

Thomas Rutledge

PSYCHOLOGICAL RESPONSE STYLES AND CARDIOVASCULAR HEALTH: CONFOUND OR INDEPENDENT RISK FACTOR?

Individuals are often less than completely honest when providing personal information to others. Consistent with the Heisenberg Principle, it seems the very act of measurement carries the risk of altering the characteristic(s) under investigation. Because of our dependence on criteria that are frequently subjective by definition, this is a methodological issue of particular concern for behavioral scientists (Rosenthal & Rosnow, 1992).

Given the seriousness of the problem, it should come as no surprise to learn that factors affecting the validity of self-report descriptions have a long history of research in psychology, dating at the very minimum to Hathaway and McKinley's development of the Minnesota Multiphasic Personality Inventory (MMPI; McKinley, Hathaway, & Meehl, 1948; Meehl & Hathaway, 1946), and arguably even to Freud's repression phenomenon (e.g., Freud, 1957). The aim of this thesis is to provide the reader with a substantive chronicle of two modern approaches for treating what will be termed response style tendencies, and to describe the results of an empirical assessment of these strategies with respect to cardiovascular health.

A brief overview of response styles in psychological assessment.

Under normal circumstances, the assessment environment can affect an individual's responding in a number of ways. Interview participants almost universally show a tendency to exaggerate so-called desirable behaviors - book reading, condom use, church attendance, and the like - and to downplay undesirable behaviors (Crowne & Marlowe, 1964). This pattern is commonly referred to as socially desirable responding (Edwards, 1957). Similarly, many individuals are motivated to present themselves in a favorable or unfavorable light in order to achieve (or avoid) some perceived end, or in other cases may

simply be unwilling to admit even minor personal faults. These behaviors can range from outright efforts to deceive or malinger to more subtle attempts to deny, repress, or avoid negative information (Graham, 1993).

Whereas all of the above examples can be characterized as attempts to mislead others, a related and perhaps even more invasive pattern appears in which the individual seems to engage in a kind of motivated unawareness of personal difficulties, a behavior usually termed self-deception (Sackeim & Gur, 1978). This construct differs from other response styles in that self-deceptive tendencies have been found to be relatively unaffected by demand characteristics of the testing situation (Linden, Paulhus, & Dobson, 1986). Partly for this reason, many researchers believe that self-deception is the primary source of response style distortions acting on self-report instruments (Paulhus, 1984; Weinberger, Schwartz, & Davidson, 1979). A final form of response distortion also possesses a motivational link, in this case a lack of interest in the assessment procedure. In these cases, typically referred to as response sets (Cronbach, 1950; Goldberg, Rorer, & Greene, 1970), individuals strive to complete the assessment quickly, and may answer questions randomly or based on some easily applied algorithm (e.g., answering false to all questions).

Response style tendencies in the psychological literature have been measured using a number of self-report instruments, although the Marlowe-Crowne Social Desirability Scale (Crowne & Marlowe, 1964), the Self and Other-Deception Scales (Sackeim & Gur, 1978), the Balanced Inventory of Desirable Responding (Paulhus, 1984) and Byrne's Repression-Sensitization Scale (Byrne, Steinberg, & Schwartz, 1968) are probably the most well known of these tools. Interestingly, results from a series of factor analytic studies (Paulhus, 1984; Sackeim, 1983) indicate that the content of the above scales can be described in terms of two dimensions. Lie scales and other measures of defensiveness

were found to load highly on one factor, labelled impression management, whereas the Self-Deception and Repression-Sensitization scale items loaded on a distinct second factor called self-deception. The Marlowe-Crowne showed loadings on both factors, suggesting that items from this measure assess both impression management and self-deception tendencies. The value of these findings is that they allow us to reduce a diverse response style vocabulary including denial, lying, malingering, defensiveness, repression, self- and other-deception, social desirability, and suppression, among others, to two quantitatively distinct entities.

Time has shown us that few methods of data collection with human participants are immune to response style influences, certainly not the interviews, behavioral observation techniques, and self-report instruments so commonly employed in psychological research (Wiggins, 1973). Although the pervasiveness of these influences has long been acknowledged, their proper conceptualization remains a point of contention (McCrae & Costa, 1983).

Two traditions of response style measurement.

An ever present goal in psychological research is to maximize the extent to which one is measuring the construct or constructs of interest, and not some other variable (Kazdin, 1992). Because response styles represent a potential threat to the aim of measurement purity, it was critical that researchers develop strategies for evaluating these influences. But how might this be done in practice? Perhaps the most obvious method for testing the validity of assessment data is to compare self-report information to that from an objective source. In typical clinical cases, unfortunately, this is a difficult or even impossible task. Instead, researchers were often forced to infer testing biases through the individuals' self-report patterns, most commonly by incorporating additional questionnaires or subscales into the assessment procedure that were designed to tap response style

tendencies. Individuals whose responses indicated an unrealistically moral standard of behavior (e.g., always telling the truth or never listening to gossip), or a lack of what are believed to be nearly universal shortcomings (e.g., littering), were judged to be high in social desirability or defensiveness (Crowne & Marlowe, 1964).

With a means of accurate measurement in place, the development of algorithms meant to correct for these influences was a logical next step. An illustration of this method in practice can be found in Hathaway and McKinley's use of the K-correction procedure in the MMPI (Graham, 1993). Adherents to this perspective treated response style effects as confounds to the validity of assessment instruments, and sought to develop quantitative methods for identifying and correcting for these biases. Additional examples of this strategy are seen in a number of other mainstream personality and psychopathology scales, including the California Psychological Inventory (Gough, 1950) and Millon Clinical Multiaxial Inventory (Millon, 1987).

Explicit correction or validation methods such as those employed in the aforementioned scales are certainly the most common tactic of this paradigm, but they are not the only method developed to control for response style biases over the years. Edwards (1970), for example, took a somewhat different approach to this end in his Personal Preference Scale by developing items judged to be of similar desirability. Although clever, this method did not ultimately have a lasting influence on the assessment field due to the considerable time and research involved in developing items equivalent in social desirability. Other relevant strategies for detecting or countering response style effects, given only brief mention here for sake of brevity, include the frequent use of reverse ordered questionnaire items and the bogus pipeline technique (Arkin & Lake, 1983). The description of these tactics is hardly exhaustive, but serves here to highlight

the theme that response styles are commonly perceived by many researchers to be a type of artifact whose net effect is to cloud the interpretability of assessment data.

A second group of researchers took a rather different angle on the issue. As implied above, evidence suggests that individuals reporting low scores on a variety of psychological symptom inventories can be categorized into at least two groups; those who are actually low on the reported characteristic(s), and those who score low as a result of a defensive or repressive reporting style (Weinberger, et al., 1979; It should be noted that the use of repression in this literature bears only a superficial resemblance to the same term used by psychoanalytic theorists, and is treated in this paper as synonomous with self-deception. Specifically, rather than representing a forceful ejection of painful memories or experiences from awareness in the classic Freudian sense, repression here refers to an at least partly conscious coping style that functions through intentional suppression or inhibition of distressing cognitive material to minimize perceived threat). Instead of treating response styles as a kind of demand characteristic produced by assessment pressures, response styles from this second perspective were viewed as trait-like features of personality that imparted a tendency to minimize the expression of distressing or threatening information from the self and others (Gur & Sackheim, 1979). Individuals high on this trait were expected to score consistently higher on measures of adjustment, honesty, and other socially positive behaviors, and lower on measures of anxiety, anger, depression, and similar scales that might prove threatening to confess.

Up to this point the two perspectives appear to share a similar conceptualization of response styles, in that each position sees these effects as at least partly willful attempts to filter the expression of personal information in a pattern consistent with some internal or external motivation. Pragmatically speaking, however, followers of the second tradition make a significant detour with the role that response styles play in their investigations.

These researchers operate under the position that stylistic effects revealed through written or verbal assessments are representative of the individual's characteristic manner of processing emotional information. Response styles are, therefore, expected to generalize across situations involving self-disclosure of a potentially stressful nature. Furthermore, because evidence exists suggesting that the chronic act of avoiding, denying, or repressing distressing self-references may have negative health implications (Alexander, 1939; Esterling, Antoni, Mahendra, & Schneiderman, 1990; Gross & Levenson, 1997; Pennebaker & O'Heeron, 1984) efforts are encouraged by these researchers to examine response style effects as independent personality dispositions or as moderators of other psychological traits.

Response styles and personality variables.

Briefly stated, these two perspectives boil down to a style versus substance debate (McCrae & Costa, 1983). Are response style effects better conceptualized as a validity confound affecting the accuracy of self-reports, or as separate personality dimensions capable of meaningful associations with other constructs? Fortunately, this question is far from rhetorical. A considerable body of research exists on this topic in the personality field, and it builds a surprisingly strong case for the treatment of response styles as substantive traits rather than stylistic artifacts. A number of individual studies show that attempts to correct scores for social desirability effects usually do not improve validity coefficients (e.g., Arkin & Lake, 1983; Dicken, 1963; Eysenck, 1962), and in some cases may even reduce zero order relationships (Linehan & Nielsen, 1981). Wiggins (1973), in his review of the personality assessment field, also came to the conclusion that efforts to control for response style effects were largely unwarranted. In light of observations that measures of self-deception, defensiveness, and social desirability can account for up to 30-50% of the variance in many psychological inventories, including those designed to be free

of traditional response biases (Linden, Paulhus, & Dobson, 1986; McCrae & Costa, 1983), some researchers contend that these correlations represent substantive covariation between response styles and psychological variables.

Correcting for response style effects, however, remains a common practice in spite of this research. Users of the MMPI and MMPI-2, for example, almost universally employ the K-correction procedure, in spite of studies showing that correction steps typically do not improve predictions with test subjects (Clopton, Shanks, & Preng, 1987; McCrae, et al., 1989). One study in this area even showed that K-corrected scores lead to decreased accuracy with normal samples (Hsu, 1986).

This raises an important question. Why, in the face of strong evidence to the contrary, might researchers continue to see response style influences as an important source of concern? Answers to this question are not difficult to find, although some are less defensible than others. McCrae and Costa (1983) argue that with many personality scales, it may be little more than habit on the part of researchers to perceive response style correlations in anything but a negative light, what could be called methodological inertia. With the MMPI-2, on the other hand, continued reliance on the K-correction procedure allows the user to make normative reference to the very sizable literature created with the original MMPI (Graham, 1993). Finally, in circles outside of the personality field the persistent treatment of response styles as measurement confounds may be perfectly justified on the basis of construct validity or related arguments, but this view requires research that directly tests the utility of response style measures as artifacts versus predictive personality measures in order to garner support. One important area in which the latter statement applies is the behavioral medicine field. Response style effects are seen as troublesome here because they may result in a failure to identify salient relationships between psychological and health variables in research, or to recognize the

presence of psychological or physical symptoms in patients that could affect the course of treatment (Jorgensen, Johnson, Kolodziej, & Scheer, 1996; Linden, 1988). It is to a brief survey of this literature that we now turn.

Response styles and physical health.

Research indicates that depression levels are among the strongest predictors of survival in the year following a heart attack (e.g., Frasure-Smith, Lesperance, & Talajic, 1993). Similarly, meta-analytic reviews show that distress symptoms including hostility, depression, life stress, and anger expression are consistently related to both the development of cardiovascular illness and the prognosis for treatment (Booth-Kewley & Friedman, 1987; Jorgensen, et al., 1996). Perhaps most critical, existing research suggests that the identification and treatment of these characteristics can lower the risk of disease, positively impact immune system functioning, and increase survival time for victims of cancer, AIDS, and heart disease (Frasure-Smith, 1991; Linden, Stossel, & Maurice, 1996; McEwan, 1998). Each of these findings demonstrate the importance of accurately measuring these variables.

Interestingly, there is now evidence that the link between psychological factors and disease may be more than correlational. Recent investigations by Ironson et al., (1992), and Gullette et al., (1997) demonstrated that the experience of anger (and perhaps other negative emotional states as well) can lower the blood pumping efficiency of the heart and trigger myocardial ischemic events in individuals with heart disease. Coupled with evidence indicating that negative psychological states can also cause platelet activation and coronary vasoconstriction in coronary patients (Grignani, et al., 1991; Yeung, et al., 1991), it appears that we are beginning to advance beyond mere relational discussions of psychological and physical variables to identifying specific physiological processes through which these factors may be associated.

Because the above disease conditions collectively kill well over a thousand people a day in North America alone (Booth-Kewley & Friedman, 1987), the impact of psychological risk factors on disease endpoints is not to be taken lightly. In addition to increasing survival time and improving quality of life for disease victims, the identification and treatment of contributing psychological factors has the potential to save our health care system millions of dollars (Oldridge, Furlong, & Feeny, 1993). The stakes in this field are clearly very large.

But with the implications of this field so serious, and the benefits so apparent, measurement precision and construct validity issues have become increasingly salient. In the previous section it was observed that response style measures often account for a significant portion of the variance in personality scales (McCrae & Costa, 1983). Almost identical findings are reported with respect to response style correlations with psychological and physical symptoms (Linden, Paulhus, & Dobson, 1986). Thus, the tendency to minimize negative personal qualities or behaviors appears to extend equally well to the reporting of traditional psychological risk factors and physical disease symptoms. Even more compelling is that this finding applies even in cases of individuals with significant heart disease, where the failure to detect or report symptoms such as acute angina can have fatal consequences (Davies, et al., 1993).

The latter instance is particularly noteworthy here because it implies that the repression or avoidance of symptoms can, in at least some situations, have deleterious effects on health (Nolan & Wielgosz, 1991). This position is in contrast with Taylor's (e.g., Taylor & Brown, 1988) positive illusions research in which individual's under-reporting signs of psychological distress is usually viewed in a favorable light (also see Lazarus, 1983; and Lockard & Paulhus, 1980). On the other hand, the position is consistent with research both past and present that suggests a repressive coping style can

have a negative impact on physical health (Denollet, 1991, 1993; Pennebaker & Susman, 1988). For example, self-deceptive tendencies have been linked to an inhibited immune response (Esterling, Antoni, Kumar, & Schneiderman, 1990), increased cancer risk (Cull, 1990; Greer & Morris, 1975), and to health behaviors such as lower levels of social support and the tendency to dismiss somatic symptoms that could contribute to disease prognosis (Denollet, 1993; Greene, Moss, & Goldstein, 1973; Shaw & Cohen, 1985). Resolving the apparent discrepancy between these two treatments of symptom suppression will clearly require additional research.

Summary.

The forthcoming sections examine in some detail the research implicating response styles as potential risk factors with respect to two specific health conditions: hypertension and coronary heart disease (CHD). Prior to this discussion, however, several summary comments are deserving of mention with regards to the current discourse. First of all, the behavioral medicine field presents an important area in which the conceptualization of response styles remains unresolved. Many researchers believe the relationships observed between anger, depression, hostility, and disease incidence would be more consistent, and perhaps even more substantive, were measurement tools cleansed of response style effects (e.g., King, Reis, Porter, & Norsen, 1993; Matthews, Siegel, Kuller, Thompson, & Varat, 1983; Shaw & Cohen, 1985). Consistent with this view, the presence of response style distortions may prevent researchers or practitioners from making accurate statements with respect to an individual's status on established psychological risk factors, and could even lead intervention efforts astray by making it difficult to identify those at increased risk (Dimsdale & Hackett, 1982). Parallel with arguments introduced earlier, the merits of this perspective can only be assessed through attempts to statistically correct for response style

effects. But in spite of the potential impact of this finding, it currently remains unexplored in the cardiovascular literature.

Once again, however, the alternative to this position is that response styles function as independent personality traits. The overlapping variance between psychological symptom inventories and response style measures may represent a common pathway between personality and disease. Arguably, an undetermined portion of the psychological factor-disease relationships attributed to anger, depression, hostility and other scales may interact or even be mediated by response style characteristics. This would imply that efforts to control or remove these effects could actually reduce observed correlations in some contexts. Recall that in the MMPI literature predictions among normal test participants were impaired by employment of the K-correction procedure (Hsu, 1986).

Prior research has also tacitly assumed that response style effects must be linear. Because current evidence exists to suggest that relations between blood pressure and psychological risk factors such as anger or hostility may be curvilinear in form (Linden & Lamensdorf, 1990), future investigations should acknowledge the possibility of nonlinear effects among response style measures as well. Finally, it is even possible that repressive symptom reporting could be a by-product of disease rather than a contributor. The knowledge of one's disease status may affect the way in which we perceive or report physical and emotional symptoms (Irvine, Garner, Olmsted, & Logan, 1989). The observation that individuals commonly experience a period of denial in the aftermath of a traumatic event such as the diagnosis of serious illness may be an example of this process (Kleinke, 1991). These issues will be explored in subsequent sections.

In closure of this section, current behavioral medicine research raises an old methodological issue. The difference here, however, is that the life and death issues

present in behavioral medicine research make the stakes markedly higher than those found in the personality field. As indicated, the crux of the current thesis is to report an empirical investigation of these perspectives, but we will first explore two specific disease literatures in which existing evidence shows that the response style versus substance debate is most salient.

Response styles and hypertension.

A compelling body of research indicates that individuals diagnosed with essential hypertension have difficulties expressing or communicating negative emotions (Roter & Ewart, 1992). Separate studies have documented this finding using a variety of measures, showing that hypertensives exhibit lower levels of assertion, poor social skills, and a characteristic suppression or denial of negative emotions (Diamond, 1982; Hahn, Brooks, & Hartsough, 1993; Jorgensen & Houston, 1986; Linden & Feuerstein, 1983). The implication of these findings is that emotional suppression may be a personality trait that reliably distinguishes hypertensive from normotensive populations, or even that the chronic avoidance or repression of emotional information could promote increases in blood pressure (Alexander, 1939). Other research on this issue, in contrast, suggests that the repressive tendencies shown by hypertensive groups may be a reaction to their condition and not a cause of the disease itself (Irvine, et al., 1989). Knowledge of one's hypertensive status may, therefore, moderate the relationship between blood pressure and emotional coping.

In order to definitively resolve this debate, it would be necessary to prospectively evaluate the relationship of emotional repression with blood pressure levels in a sample of healthy adults. Indications that response style measures could predict blood pressure changes over time would be a strong indictment of these tendencies, whereas the lack of temporal relations would be consistent with Irvine's (Irvine, et al., 1989) finding that

emotional repression may instead be a style some individuals adopt in response to learning of their hypertensive status. The present thesis is perhaps the first to report findings from such an investigation. Previous research in this area, due largely to the prohibitive cost of prospective data collection, relied largely on methodologies that compared physiological responding between self-report defined repressive and nonrepressive participants on a diverse set of experimental tasks. This research is briefly summarized below. Generally stated, to the extent that self-deceptive or defensive participants demonstrated physiological responding patterns that indicate increased arousal, this evidence is interpreted as consistent with the hypothesis that emotional inhibition could promote blood pressure increases. Thus, although this research does little to directly clarify the cause versus consequence debate in the hypertension literature, it can potentially identify mechanisms through which response style characteristics and blood pressure may be associated.

The evidence on the effects of emotional expression on blood pressure can be categorized into three paradigms: a) studies such as those cited at the outset of this section that compare hypertensive and nonhypertensive participants on emotional coping measures; b) a small and relatively recent set of controlled laboratory investigations in which participants have voluntarily inhibited affective expression; and c) a larger and entirely correlational collection of studies which document response style and blood pressure associations using participants categorized according to their scores on response style instruments. Because our major theme at this stage is to survey possible connections between response styles and temporal blood pressure changes - and not to merely describe differences between existing hypertensive and normotensive groups - only the latter two domains are given further treatment here.

If one considers emotional suppression a characteristic reaction to hypertension rather than a potential risk factor for the condition, then it seems only logical to assume that individuals employ this form of coping in order to minimize personal distress. But do these efforts to decrease emotional responding actually pay off? Ironically, although there is some evidence to suggest that emotional inhibition can increase pain tolerance and lower skin conductance levels prior to a painful medical intervention (e.g., Colby, Lanzetta, & Kleck, 1977), more recent work indicates that suppression is more likely to result in increased rather than decreased arousal. Gross and Levenson (1993; 1997), for example, found that efforts to inhibit positive and negative emotions in reaction to a film led to significant increases in skin conductance, cardiovascular activity, and respiratory rates in participants relative to those not assigned to the suppression condition.

Clearly, there are too few studies of this nature to draw firm conclusions regarding suppression effects. But in spite of this shortcoming, the findings do imply that emotional inhibition can produce at least transient increases in cardiovascular activity, among other physiological indices, and that these effects may occur despite actually lower metabolic demands resulting from the decrease in emotionally expressive behaviors (Gross & Levenson, 1997). These findings are also noteworthy because they concur with conclusions drawn from correlational studies in this area.

Research investigating cardiovascular responding patterns among participants classified as either high or low repressors on the basis on response style scores show that high repression status is associated with increased cardiovascular reactivity in response to a variety of laboratory stress tasks and difficult interpersonal scenarios (Davidson, 1996; Hahn, Brooks, & Hartsough, 1993; Jorgensen & Houston, 1986; King, Taylor, Albright, & Haskell, 1990; Shapiro, Goldstein, & Jamner, 1995), and with higher resting blood pressure levels among males (Jamner, Shapiro, Goldstein, & Hug, 1991; King, et al.,

1990; Linden, Chambers, Lenz, & Maurice, 1993). In addition, similar differences were observed when low and high repressors were compared on physiological (heart rate, skin conductance, and muscle tension) and behavioral (reaction time and verbal interference) measures of anxiety during a laboratory stress task, suggesting that despite their lower self-report scores on psychological distress scales repressors were actually as or more anxious than those endorsing higher levels of distress (Weinberger, et al., 1979). Notably, with the exception of the Hahn, Brooks,and Hartsough (1993) study, all of the above findings were based on normotensive samples.

This evidence is generally consistent with the view that emotional inhibition can contribute to the development of hypertension. That is, although the correlational design of these studies prevents etiological references, the observed pattern of effects is what would be anticipated were emotional inhibition a causal agent for hypertension. What is perhaps less apparent is that this evidence can also be interpreted to support the treatment of response styles as measurement confounds. Specifically, these results demonstrate that participants' scores on self-report instruments cannot be taken at face value, and illustrate the need to routinely assess response style effects when documenting associations between psychological and physical health variables. Efforts to identify stable relations between traditional psychological risk factors and cardiovascular outcomes could be impaired by the increased error variance produced from response style influences. Alternatively, response style status could moderate relationships between psychological and cardiovascular variables. These prospects suggest that the often inconsistent findings reported in health psychology journals could be improved were response style effects removed, or were participants classified on the basis of emotional coping status prior to examining other relationships.

Response styles and cardiovascular disease.

Biobehavioral research shows that up to half of all new coronary heart disease (CHD) cases cannot be accounted for using standard medical risk factors, a fact which gives strong endorsement to the continuing efforts by investigators to identify salient personality and behavioral contributions (Denollet, 1993). Whereas person variables such as depression, anger, hostility, and stress levels remain the key players in this research (Booth-Kewley & Friedman, 1987), there is also data to suggest that emotional repression, as measured by response style instruments, may be linked to the development and progression of CHD. Analyses drawn from the Framingham Study and the Multiple Risk Factor Intervention Trial each indicated that the inhibition or nonexpression or emotions - particularly anger and hostility - was related to CHD incidence (Dembroski, MacDougall, Costa, & Grandits, 1989; Haynes, Feinleib, & Kannel, 1980). Denollet (1995) further showed that a distressed personality, consisting of high levels of experienced stress and low levels of emotional expression, was associated with a five-fold mortality rate in men following myocardial infarction. Emotional expression in these studies was measured using Spielberger's Anger-In Scale (Spielberger, et al., 1985) and the Marlowe-Crowne Social Desirability Scale.

Furthermore, these findings are buffered by evidence associating self-deceptive qualities with coronary prone behaviors (Denollet, 1991; 1993; Emmons, 1992). This research proposes that repressive tendencies may affect coronary status by disposing behavioral tendencies, such as a habitual denial of physical symptoms and reduced help-seeking, that could influence the course of disease. Thus, although lacking the sheer volume of findings evident with more established psychological risk factors, the handful of studies to date which have examined self-deceptive qualities in cardiovascular research

have produced results consistent with the hypothesis that this characteristic could be independently related to CHD progression and mortality.

Additional research regarding these associations is clearly needed. Because of the well established relations between traditional psychological risk factors and coronary disease, however, response style effects should not be measured in isolation. As discussed by Denollet (1995), response styles may be most useful in the context of cardiovascular disease when combined with other measures of psychological functioning. Such findings support efforts to examine response styles in a moderating role as well as in direct relational models with CHD. Finally, due to the known suppressive effects of response styles on the reporting of psychological distress, attempts to partial or otherwise correct for these effects may likewise provide an improved prediction of coronary status.

Summary and current objectives.

Physicist John Platt (1964) argued that science could be most rapidly advanced by devising studies in which two or more incompatible theories were contested. He called this process strong inference, a label made in explicit reference to the usual slow pace of scientific progress. Despite very different circumstances, a similar form of theory testing is supported in the current paper. Specifically, the aim of the present investigation is to explore the utility of two contrasting conceptualizations of response styles in cardiovascular research. Preceding sections contained an overview of these perspectives, showing that there is now considerable evidence to question a long tradition of viewing response style characteristics solely as methodological artifacts. Findings from the personality literature gave early indication that response styles such as self-deception and social desirability could be more accurately treated as substantive traits rather than stylistic confounds (McCrae & Costa, 1983), and more recent data from a varied collection of studies supports this view in the cardiovascular field as well.

However, the belief that response styles represent measurement impurities has not gone away in the face of this evidence, nor do I argue that it should. A large and impressive body of research supports associations between psychological risk factors such as anger, hostility, life stress, and depression, and coronary disease indicators. Recent investigations have even begun to document physiological processes that may substantiate these relations (e.g., Ironson, et al., 1992). Because of our heavy reliance on self-report based assessments of psychological risk factors, and the high overlap these variables commonly show with response style measures, we cannot be sure what ingredients in these risk factors are most relevant to disease.

Furthermore, this description is not intended to suggest that these two views of response styles are entirely contradictory. Explicit efforts to maintain anonymity are often used to prevent or minimize defensive responding in research, and there is no evidence to suggest this is unwise (Linden, et al., 1986). Similarly, no one doubts the utility of practices designed to reduce demand characteristics from laboratory protocols. Instead, it is argued that response style scores may reflect more than artifactual variance in many scenarios, and in addition capture a stable information processing style with possible health implications.

As is well known, the typical effect of response styles is to reduce reports of psychological distress, whereas most associations between psychological factors and coronary disease are positive. Were response style influences standard across individuals, such that their scores on psychological distress measures were uniformly depressed, then the presence of these effects would be tantamount to a linear transformation, and would not affect statistical relations (Gordon & Gross, 1978). But this is not how response styles behave. Rather, individuals differ in the extent to which their reports reflect response style influences, particularly those resulting from self-deceptive qualities, and this

raises the possibility that these effects could be masking the extent to which psychological risk factors predict disease. In addition, because knowledge of an individual's absolute standing on psychological risk factors such as depression or stress is an important consideration for determining their eligibility for psychosocial interventions following a cardiac event, differential distortions resulting from response style effects may impair diagnostic validity and even misdirect treatment. In support of this statement, the diagnosis of clinical depression has proven to be a strong predictor of post-cardiac event mortality, whereas subclinical levels show less consistent relations (Frasure-Smith, et al., 1993). Response style effects may limit the accuracy of these diagnoses.

Study description & hypotheses.

We pursued a test of these two response style treatments using data from two recent large scale cardiovascular investigations. In the first, termed the REACT study, blood pressure changes were tracked over a three year course in a sample of normotensive adults. Participants completed an intensive protocol at the beginning and end of the three year interval that included 8-12-hour ambulatory blood pressure monitoring, the collection of physiological measurements in response to a set of laboratory stress tasks, an assessment of physical and lifestyle factors with established relations to blood pressure - including exercise patterns, smoking habits, and body fat levels - and the completion of a questionnaire battery. Because ambulatory means are recognized as the gold standard among blood pressure measurement techniques (Kamarck, et al., 1992), we gave primary focus to these measures. Self-report measures of depression, anger expression, self-deception and impression management, hostility, and daily stress were included as part of the battery at each interval.

The second investigation, termed the Canadian Atenolol/Amlodipine Silent Ischemia Study (CASIS), comprised a pharmacological treatment study for patients with

ischemic heart disease. Participants completed a twelve week treatment protocol consisting of a drug free baseline period, assignment to one of two anti-ischemic medications, and finally to a combined pharmacological treatment regimen. On four dates across the course of treatment patients completed a thorough physical assessment - including 48-hour Holter monitoring of ischemic episodes, treadmill testing, and blood pressure measurements - and a psychological test battery containing measures of hostility, anger expression, depression, type A behavior, daily stress, and self-deception and impression management.

Based on previous findings and the design of the current pair of investigations, we investigated the utility of response styles as predictors of disease by testing the following hypotheses:

Main effects predictions.

1) Within the REACT study, we expected baseline measures of self-deception and impression management to show positive associations with systolic and diastolic blood pressure levels at the three year testing interval. Gender differences in these predictions were also explored.

2) In the context of the ischemic heart disease trial (CASIS), we predicted that high baseline levels of self-deception and impression management would be associated with more severe cardiac disease status as measured by frequency or duration of ischemia, exercise functioning, and the presence of angina. Main effect predictions would also be supported by associations between high baseline response style scores and reduced treatment effects on ischemia, exercise performance, or the presence of angina following medication.

Moderator effects predictions.

1) Among REACT participants, we expected psychological risk factor x response style interaction terms to demonstrate independent predictive potential for three year ambulatory blood pressure means after controlling for main effect associations. Linear as well as nonlinear interaction terms were examined.

2) Moderator effects in the CASIS study were assessed by using psychological risk factor x response style interaction terms to predict baseline disease severity and pharmacological treatment effects. Linear and nonlinear interaction effects were each examined after controlling for main effect associations.

Confound effects predictions.

1) Within the REACT study, we projected that correlations between depression, anger, hostility, and three year ambulatory blood pressure levels would increase following the removal of response style effects via part correlations.

2) The prediction of disease severity among CASIS patients via self-report measures of depression, anger, and hostility were expected to improve following correction for response style effects. Similarly, the potential for these psychological risk factors to predict pharmacological treatment effects was also expected to be enhanced following the removal of response style influences.

Method

Study 1 (REACT)

Participants.

Three hundred and twenty nine male and female adults (157 males, 172 females, average age 27.1) participated in the first component of a two phase investigation associating laboratory and ambulatory blood pressure measurements with psychological predictors. 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 either coronary heart disease or hypertension. One hundred and twenty six participants (63 males, 63 females, average age 32.8) subsequently returned for the second test date following a three year interval. The ethnic composition of both sample groups was approximately 70% Caucasian and 30% Asian.

Design.

Participants completed an identical protocol at each testing date. The testing procedure involved a laboratory assessment during which participants performed a counterbalanced set of stress tasks while having their blood pressure monitored, and an ambulatory assessment in which they wore an ambulatory blood pressure monitor over the course of an 8-12 hour work day. Ambulatory assessment always preceeded laboratory testing, and was conducted on a separate day. Participants completed a psychological test battery at the beginning of the laboratory session.

Instrumentation.

Blood pressure and heart rate information were collected from participants during the laboratory sessions using a Dinamap 845 Vital Signs Monitor (Critikon Corporation, Tampa/Florida). Validation work has shown the Dinamap to provide blood pressure values

that directly correspond with intra-arterial measurements (Borow & Newburger, 1982). Cardiovascular activity in the natural environment was assessed with Spacelabs Model 90207 ambulatory monitors. These devices weigh about 1.5 pounds and are worn in a protective pouch. Use of the Spacelabs monitor is supported by validation work (O'Brien, Mee, Atkins, & O'Mally, 1991).

Psychological measurement.

(a) Laboratory stress tasks: Participants completed a series of three 5-minute stress tasks during laboratory testing, including 1) An isometric handgrip test (duplicating the procedure used by Parker et al. [1987]) requiring 3 minutes at 20% of maximum contraction and 2 minutes at 30% of maximum; 2) a mental arithmetic task in which videotaped math equations were presented at 10 second intervals with vocal response delivery (Linden, 1991); and 3) a discussion task in which the participant discussed a recent anger provoking event with a same sex experimenter (Linden & Lamensdorf, 1990). Blood pressure measurements were taken at minutes 1.5 and 3.5 of each task.

(b) Self-report instruments: Prior to the stress tasks, participants completed a battery of psychological instruments, including measures of depression, hostility, daily stress, type A behavior, anger expression, and self-deception and impression management. The questionnaires used and scoring methods for these instruments are described in Table 1. Research supports the reliability and validity of each instrument (see references in table 1).

Insert Table 1 about here

Procedure.

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, pre-tested on the spot, and returned 8-12 hours later for data analysis. Pre-testing, consisting of the first five ambulatory readings, were taken in the lab, and were used to determine proper cuff placement. Whenever pre-test values appeared uncredible, the cuff placement was changed and comparisons with Dinamap readings were obtained. Ambulatory readings were taken every 20 minutes. Participants were explicitly instructed to minimize physical activity during a measurement cycle, and only approximately 10% of attempted measures were unusable. The Spacelabs monitors provide error codes for failed measurement attempts, thereby facilitating error identification.

Participants were directed not to ingest alcohol, caffeine, or nicotine, or to exercise strenuously 2 hours prior to the lab component of the study. Completion of a demographic questionnaire assessing exercise, smoking, and drinking habits preceeded lab testing, and participant's body fat levels were measured by a female experimenter using a 6-site protocol (Nieman, 1986). Each lab session commenced with a 20 minute adaptation phase during which subjects completed questionnaires. Participants were seated in a reclining chair and were alone in the room for this time period. After the final baseline reading had been taken at minute 20, task instructions were given over an intercom system. A 5 minute recovery period followed the completion of each stress task, and a further 15 minute recovery interval commenced at the completion of the final task. A total of five cardiovascular readings were collected during the baseline and recovery periods (min's 0, 4, 8, 12, 16, & 20 of the baseline interval and 0, 4, 8, 11, & 14 of the recovery phase).

Statistical analyses.

Descriptive statistics were used to present salient physical and psychological characteristics of the sample at the two testing intervals. We employed dependent t-tests to explore possible changes in these characteristics over the three year period. Primary analyses, consisting of a test of the relationship(s) between response style and blood pressure, moderator effects, and of the associations between psychological risk factors and blood pressure following the correction for response styles are described below:

(a) We assessed the main effects hypotheses between self- and other-deception measures and blood pressure levels using zero order correlations and hierachical regression. Initially, we performed correlations in order to assess the presence of relationships. These values were collected for the complete sample and separately for males and females. In cases in which gender differences appeared, subsequent analyses were also conducted separately. Where self-deception or impression management showed significant associations with ambulatory means, we used regression methods to determine the stability of the observed relationship after controlling for standard physical risk factors. At step one of each regression equation, we entered baseline measures of smoking status, exercise frequency, and body fat levels. Forced entrance of the baseline self- and/or other-deception scores followed at step two. This established a more conservative field for assessing response style contributions. Diastolic and systolic ambulatory means collected at the end of the three year study served as dependent measures. This strategy permitted a prediction of temporal blood pressure levels after controlling for established risk factors.

(b) Moderator effects were examined in accordance to methods proposed by Baron and Kenny (1986). This strategy permits an assessment of linear as well as nonlinear (quadratic) interactions. First of all, we created psychological risk factor x response style interaction terms (ΨxR) by taking the product of each psychological risk factor with self-

deception and impression management. This resulted in two interaction terms for each of the five psychological risk factors (depression, anger-in, anger-out, hostility, and daily stress), one across self-deception and the other across impression management. Each of the ΨxR terms denote linear interaction effects. These variables were tested through hierachical regression by forcing them into the equation in the step following the psychological risk factor and response style measures. Results from this methods are equivalent to those produced by standard analysis of variance (ANOVA) methods.

Finally, quadratic effects (the simplest type of nonlinear model) were also assessed via hierachical regression, this time by first generating a square of each of the two reponse style terms (R^2) and a ΨxR^2 term. As described by Cohen and Cohen (1983), quadratic terms are assessed by regressing the dependent measure (blood pressure means here) on the two predictors (response style and psychological risk factor), followed by the linear interaction term (Ψ x R), the square of the moderator (R^2) and finally the independent variable x squared moderator term (Ψ x R^2). Quadratic effects are supported in cases in which the latter term is significant.

Notably, blocking and ANCOVA procedures are inappropriate for assessing moderator effects in the present scenario. The latter methods are designed for cases in which a third variable shows a moderate to high correlation with the outcome measure, such as with pre-test scores (Rosenthal & Rosnow, 1992). In the current scenario, the concern was instead with the moderate correlations response styles (our third variable) expressed with regards to the psychological predictors.

(c) We tested the confounding effects model using part correlations between psychological risk factors and ambulatory blood pressure means, with the former corrected for self-deception and impression management influences. Although blood pressure means, like questionnaire data, arguably contain some degree of error, there is no

reason to believe these errors share a common source. The only variance common to the psychological risk factors and blood pressure scores should be reflective of substantive covariation. Consequently, the statistical removal of response style effects from the self-report inventories should improve the prediction of the ambulatory means through a reduction in error variance. This is equivalent to stating that "true scores" on the psychological risk factors (corrected scores) should be better predictors of blood pressure values than the simple observed scores, and is the same logic employed by users of the MMPI when attempting to correct for K-scale influences.

In practice, this resulted in a simple two-step analysis. Initially, we calculated zero-order correlations between baseline measures of depression, hostility, daily stress, anger expression (anger-in and anger-out), and ambulatory blood pressure means collected at the end of the three year study. These values served as a comparative standard for effects achieved following the removal of response style effects from the psychological measures. We calculated tests of correlation coefficients (Steiger, 1980) in order to assess potential differences among the Pearson r values. As with the main effects predictions, all results were presented in the form of effect size indicators (e.g., Pearson r's or beta weights) in order to standardize the interpretation of results and to allow us to generate conclusions that were based on more than probabilistic criteria. Finally, to provide a more systematic assessment of the confound model, we completed the same pair of analytic steps with regards to correlations between our psychological risk factors and time 1 blood presure means.

Study 2 (CASIS)

Participants.

One hundred and eighty five patients (male = 155, female = 30, average age = 60.6 years) with previous electrocardiographically positive exercise treadmill tests were studied after anti-ischemic drug therapy had been withdrawn for a minimum of 5 half lives. All patients had coronary disease established earlier by coronary arteriography, or by the presence of thallium-201 perfusion abnormalities. Patients were excluded if they required digitalis or if they had ECG abnormalities that would render the ST segment unreadable, such as a left bundle branch block, atrial fibrillation, or atrial flutter. Furthermore, patients went on to complete the treatment study only if they evidenced ischemic episodes during the initial 48 hour holter monitoring. Approximately 95 patients met this criteria. Ten Canadian academic centers, each enrolling approximately 10 patients, participated in the completion of the study. Furthermore, a total of 80 patients completed all four phases of treatment. The study received ethics committee approval at all participating centers, and all patients provided written informed consent.

Design.

The study consisted of a randomized, double-blind, parallel design trial, conducted uniformly at each of the 10 participating centers. Following a baseline evaluation without anti-ischemic medication, patients were randomized to either an amlodipine treatment group or an atenolol group. Patients received the active drug or placebo in counterbalanced sequence. Subsequently, all patients were evaluated on amlodipine plus atenolol in combination. This created a total of four testing dates for patients; the first consisting of a medication free evaluation, followed by placebo and active treatment periods, and finally a combination treatment interval. Each treatment phases was three

weeks in duration, with endpoints (exercise treadmill test, Holter monitoring, and psychological tests, each described below) assessed at the end of each treatment period.

Exercise testing.

Symptom limited exercise treadmill testing was completed using the Bruce protocol. Twelve lead ECG's were recorded at baseline, at the end of each 3-minute stage of the protocol, at the onset of 1.0 mm ST segment depression, at the occurrence of anginal symptoms, and at peak exercise. ST segment depression was evaluated at 0.06-0.08 seconds after the J point, and determined as 1 mm or great ST depression from the patient's own resting baseline. Exercise was limited by moderate to severe symptoms of angina, breathlessness or exhaustion, or by the occurrence of hypotension or more than 3 beats of ventricular tachycardia.

Ambulatory ECG monitoring.

Patients underwent 48 hour ambulatory ECG monitoring on two successive days. Three channel recordings were obtained on magnetic tape using either Marquette model 8005 or Delmar Avionics model 459 amplitude modulated recorders. Both met American Heart Association criteria for adequate frequency response to record the ST segment. After careful skin preparation, pre-gelled electrodes were applied to monitor leads V5, II, and AVF. After calibration, baseline recordings were obtained in the left and right lateral decubitus, supine, prone, and standing position, and after 1 minute of hyperventilation in the standing position.

Tapes were analyzed by a skilled technologist using a Marquette Laser Holter Scanner. All episodes were over-read by a physician. The ST segment was measured at 0.08 seconds after the J point. An ischemic episode was defined as at least 1 mm of planar or downsloping ST depression from baseline lasting for at least 1 minute and separated from an adjacent episode by at least 1 minute. The onset, maximal ST deviation, and

offset of each episode were printed on paper at 25 mm per second, and the ST segment trend data for the entire recording period were saved for later referral. For each patient, the number of episodes in 48 hours, the total duration of ischemia, and the integral of ST depression over time were recorded.

Psychological testing.

Self-report questionnaires were administered on the day following exercise treadmill testing, and included measures of hostility, daily stress, depression, type A behavior, and self-deception and impression management. Table 1 provides an overview of the content and scoring methods for these instruments.

Statistical analyses.

Simple descriptive statistics were employed to assess patients' pre-treatment and post-treatment demographic, psychological, and cardiac characteristics. Cardiac measures used in the analyses included ischemic frequency, treadmill exercise time, resting blood pressure values, and the presence of angina.

(a) In the main effects model we assessed the relationship between response styles and cardiac disease severity using correlational and regression procedures. There were too few females among the patient sample to split the analyses across gender. We also assessed main effect relations by using baseline response style scores to predict treatment effectiveness in the form of reductions in the number of events and total duration of ischemia, improvements in exercise performance, and the presence of angina at the conclusion of the combined treatment phase. The treatment effectiveness measures consisted of change scores calculated as the total improvement occuring over the baseline to post-treatment periods.

(b) Moderator effects were addressed in a manner parallel to that introduced with the REACT study. Again, we initially created ΨxR interaction terms to assess linear effects

and R^2 and $\Psi x R^2$ terms to assess quadratic effects. Because of the large number of tests this exercise generated, and the consequent elevation of our type I error rate, we used an alpha level of .01 as the criteria for declaring statistical significance. All tests were addressed via hierachical regression methods.

(c) Tests of the confounding effects model in this study also paralled those used in the prospective blood pressure investigation. Specifically, we initially calculated zero-order correlations between the cardiac variables, treatment effectiveness, and psychological risk factor measures, and then compared these numbers to the same pattern of relationships observed following the removal of response style effects via part correlations. Finally, we employed tests of correlation coefficients in order to establish differences between corrected and uncorrected relationships.

Results

Assumptions & outlier testing.

Tests of statistical assumptions for forthcoming correlation and regression results were conducted in accordance to guidelines provided by Stevens (1992). We assessed the linearity and homoscedasticity assumptions by examining plots comparing standardized residuals and predicted values. Similarly, we assessed the normality and independence assumptions by checking for outlying values within histograms of standardized error scores.

Outliers can significantly impact the accuracy of correlational and regression analyses, particularly in cases where sample sizes are small to moderate (Wilcox, 1998). We followed a two step procedure in examining outliers: first assessing the presence of outliers among the outcome and predictor variables, and second examining the influence of identified outliers on regression models. The presence of outliers among outcome variables was determined using Weisberg's t statistic (Stevens, 1992), whereas Mahalanobis distance (D) terms were generated to detect outlying data points for the predictor variables. Finally, we computed Cook's distance values for all outlying points to quantify their impact. Outliers with Cook's distance values < 1.0 are not deemed to be significantly influential and were allowed to remain in the original equations. On the other hand, we carefully scrutinized outliers with Cook's distance values > 1.0 to determine possible sources of variability (e.g., coding errors, missing values, etc.), and removed values believed to be highly unrepresentative. This resulted in the removal of a single male case from the REACT file whose three year ambulatory means were extremely high (154 diastolic and 180 systolic reading) and did not meet criteria for the study.

Study 1 (REACT).

Descriptives.

Table 2 provides means and standard deviations for the test sample on relevant demographic, physical, and psychological variables at each of the two time points. These numbers are presented separately for male and female participants. The time 2 sample was appreciably smaller, but also evenly balanced across genders. The numbers from Table 2 also suggest that older participants were more likely to return at time 2.

Insert Table 2 About Here

Main effect predictions.

Correlations between baseline impression management and self-deception scores and three year ambulatory diastolic and systolic blood pressure means are presented in Table 3. Values are shown for the complete sample, as well as broken down across male and female subgroups. Consistent with predictions, baseline self-deception scores were associated with higher diastolic and systolic blood pressure means at the three year follow-up date, r's (125) = .20, .21 for diastolic and systolic means respectively, p's<.05. However, these relationships proved to be strongly moderated by gender status. When examined across separate gender groups, self-deception scores were a highly significant predictor of higher diastolic and systolic means among the male group, r's (62) = .41, .41, p's=.001, whereas these same relationship were nonsignificant among females, r's (63) = .01, .03 for diastolic and systolic means respectively, p's>.05. Tests for comparing independent correlation coefficients revealed that the associations between self-deception scores and blood pressure were significantly higher for males relative to females, Z's (124) = 2.03, 1.99 for diastolic and systolic means respectively, p's<.05. Impression

management scores failed to correlate with either blood pressure index (r's<.1), although the relationships were again somewhat stronger (albeit in this case nonsignificantly) among the male subgroup.

Insert Table 3 About Here

In light of the robust associations observed between self-deception scores and ambulatory blood pressure means among the male group, we were further interested in assessing the stability of this predictor after controlling for a set of standard physical risk factors. This step provided a means of determining the practical value of self-deception measurements with regards to well-established blood pressure predictors. The results of hierachical regressions conducted separately for the prediction of diastolic and systolic blood pressure means are summarized in Table 4. In the first step of each analysis, body fat levels, smoking status (yes or no), and exercise duration were forced into the equation followed by baseline self-deception scores. As expected, physical risk factors (body fat levels in particular) were significantly associated with higher three year blood pressure levels, F's (3,57) = 3.1, 4.1 for diastolic and systolic means respectively, p's<.05. In each case, however, self-deception scores contributed significant predictive power, accounting for an additional 14% of the variance with diastolic means (F[4,56] = 12.2, p= .001) and an additional 15% of the variance with systolic means (F[4,56] = 12.5, p<.001).

Insert Table 4 About Here

Moderator effect predictions.

Due to the large number of tests in this model, we adopted an alpha level of .01 for the interpretation of all moderator analyses. In order to assess linear psychological risk factor x response style interaction effects, we completed a series of hierarchical regression analyses in which the individual risk factor and response style score were entered at step one followed by the respective interaction term (Baron & Kenny, 1986).

Results from these analyses, consisting of zero order relationships between linear interaction terms and prospective blood pressure levels as well as standardized beta values following the entrance of the main effect terms are summarized in Table 5. Beta values obtained via this method are equivalent to part correlations in which the main effect variance has been removed from the interaction term. Male and female outcome data are presented separately. Parallel with earlier main effect findings, there were no significant associations between interaction terms and blood pressure means among the female subgroup (F's <1). Among males, several reliable zero order interaction effects were present, with the self-deception x anger-in and self-deception x anger-out terms evidencing significant associations with higher three year diastolic means (r's .32 & .34 for the self-deception x anger-out and self-deception x anger-in terms respectively, p's<.01) and trends toward the same pattern with systolic levels. However, none of these relationships remained significant after controlling for main effects. Notably, the self-deception by hostility interaction effect became reliable (at the .05 level) after entering main effect terms (r[62]=-.30), but the relationship was in a direction opposite of that predicted.

Insert Table 5 About Here

Table 6 shows the same pattern of findings for quadratic relationships. Based on these numbers, there was little evidence to suggest that relationships between blood pressure means and interaction terms were quadratic in nature. As seen with the linear interaction terms, statistically reliable zero order relationships were present in some cases. However, these associations did not hold up after extracting linear main effect and interaction terms.

Insert Table 6 About Here

Further investigation of the interaction effects revealed that among the linear and quadratic interaction terms which reliably predicted blood pressure means, each was found to be highly correlated with self-deception scores (r's>.7). Because self-deception scores were themselves significantly associated with diastolic and systolic blood pressure levels, this pattern of intercorrelations provides an account for why particular interaction terms evidenced predictive relationships (i.e., because they were primarily reflecting self-deception variance) and for why they failed to remain significant after controlling for self-deception. Consistent with this interpretation, re-running the linear and quadratic regressions without including self-deception scores in a previous step showed that interaction terms significant at the zero order level generally remained reliable after controlling for psychological risk factors.

Confound effect predictions.

The confounding effects model proposed that the removal of response style effects would improve blood pressure predictions based on our self-report measures of anger expression, depression, daily stress, and hostility. As a type of validity check for these tests, however, we first wished to establish the pattern of intercorrelations between the

reponse style and psychological risk factor instruments employed in this study. These values are shown in Table 7. Consistent with expectations, impression management and self-deception values were negatively correlated with all five risk factor measures (p's [318] <.05). In addition, self-deception was a significantly stronger suppressor of depression scores relative to impression management (r's -.36 versus -11, Z = 3.2, p'<.05).

Insert Table 7 About Here

Table 8 provides zero order and part correlation values between baseline measures of depression, anger-in, anger-out, hostility, daily stress, and three year ambulatory diastolic and systolic means. Part correlation values represent associations in which the potentially confounding influence of self-deception and impression management tendencies observed in Table 7 have been removed from the psychological risk factor variables. These results are again presented separately for male and female subgroups.

Insert Table 8 About Here

Pearson r values from Table 8 fail to support the prediction that the statistical removal of response style tendencies would improve relationships between psychological risk factors and prospective blood pressure means. Zero order correlations were nonsignificant for all predictors in the male and female samples, and were by and large unchanged after controlling for self-deception and impression management effects.

In order to present a more comprehensive test of this model, we further conducted zero order and part correlations between the above psychological measures and blood pressure means collected at the first time point. These numbers therefore quantify

concurrent rather than predictive relationships, but hold the considerable advantage of representing a much larger pool of participants (334 versus 127). Table 9 shows the results from these analyses separately for gender subgroups.

Insert Table 9 About Here

Even adopting a liberal .05 alpha level, however, our set of psychological risk factors again proved to be unrelated to ambulatory blood pressure means both prior to and following the statistical extraction of response style influences. Gender differences were similarly absent.

Discussion

The results from study 1 provide strong support for using response styles as independent predictors of blood pressure levels. Self-deception scores reliably predicted higher diastolic and systolic levels across a three year interval for the male subgroup, and these associations were robust even after controlling for a set of physical risk factors. No such pattern of relationships was present within the female group, however, nor did impression management scores demonstrate associations with any blood pressure index.

As discussed at the outset of this paper, the most original contribution of study 1 was to provide a forum for investigating response style-blood pressure relationships across a longitudinal time frame. The positive findings illustrated here add to an already compelling literature in suggesting response styles tendencies are linked in some way to the regulation of blood pressure levels among men. Even more impressively, the results shown here were recently replicated in an independent three-year study of blood pressure among young adults (MacGregor, Davidson, & Prkachin, 1998). After controlling for baseline blood pressure levels and a set of traditional hypertensive risk factors, these

researchers observed a significant correlation between a measure of self-deception/defensiveness taken at baseline and higher three year diastolic means collected in the laboratory. Taken collectively, these outcomes build a powerful case for using response style measures in behavioral studies of blood pressure. Furthermore, because of the differences in the way response styles and blood pressure were measured in the MacGregor et al (1998) investigation, we also contend that their results may be interpreted as yielding some degree of external validation to the findings reported here.

On a broader level, the main effect hypotheses supported in this study are consistent with previous research documenting relationships between self-deception and elevated reactivity and resting blood pressure levels among males in previous laboratory experiments (e.g., Linden, et al., 1993; King, et al, 1990; Weinberger, et al., 1979). However, the current results also extend these findings by substantiating self-deception and blood pressure associations across a longitudinal design, after controlling for standard physical risk factors, and by demonstrating their stability with ambulatory blood pressure measures which are increasingly recognized as the gold standard in the field for the assessment of blood pressure.

In contrast to the strong main effect relationships, we found little evidence for using response styles in a moderating role, or as statistical confounds to psychological risk factors. Although a number of linear and nonlinear self-deception x psychological risk factor interactions did show reliable associations with blood pressure means at the zero order level, only one of the effects proved even marginally significant after controlling for main effect terms - and this single correlation actually predicted lower blood pressure values. Furthermore, the high intercorrelations observed between significant interaction terms and self-deception scores suggested that the moderator effects were largely a product of self-deception variance, and therefore represented redundant information.

The extraction of response style influences from the psychological risk factors via part correlations had no apparent effect on the relationships observed between blood pressure means and self-report measures of depression, anger expression, daily stress, and hostility. Because none of the psychological risk factors expressed significant correlations with blood pressure levels even prior to controlling for response style effects, we felt the reliance on three year ambulatory means as the sole outcome criteria might provide an inadequate test of the confounding effects model. Our efforts to enhance the scope of these findings by assessing correlations between psychological risk factors and baseline ambulatory values generated similar results, however, suggesting that the presence of response style influences had little impact on the validity of the self-report instruments with respect to blood pressure. This finding is consistent with results cited by McCrae (1983) and others in the personality field who have also cited nonsignificant changes in validity coefficients following the statistical removal of response style effects. To the author's knowledge this study is the first to directly report on validity effects in the cardiovascular literature.

In closure of this theme, although our findings provide no direct support for the confounding effects model as expressed here, in the absence of statistically reliable results between our set of psychological risk factors and blood pressure we do not believe our results afford the only test of this strategy. A second case would be the scenario in which risk factor variables generated initial predictive ability, at which time we could assess changes resulting from the extraction of response style variance. Fortunately for us, we observed just such a situation in the analysis of the CASIS outcomes.

Study 2 (CASIS).

Descriptives.

Table 10 provides means, standard deviations, and statistical test results for ischemic heart disease patients across the baseline and combined treatment phases on relevant demographic and cardiac variables. Results from separate repeated measure ANOVA's revealed highly significant improvements across all cardiac measures as a result of the combined treatment program (all p's $<.01$), including reduced incidence and duration of ischemia, improved exercise performance, and fewer reports of angina (F's [1,74] 57.8, 36.0, 64.0, & 9.0 for ischemic frequency, ischemic duration, exercise time, and angina incidence respectively).

Insert Table 10 About Here

Main effect predictions.

Main effect analyses included tests for relationships between response styles and baseline disease severity as well as using response styles to predict treatment outcomes. Zero order correlations between self-deception, impression management, measures of pre-treatment disease severity, and treatment changes are shown in Table 11. Neither impression management nor self-deception scores were associated with any of the four baseline cardiac disease measures (p's $> .2$). Impression management items also failed to predict treatment changes.

Insert Table 11 About Here

Self-deception values, however, did show one-tailed predictive potential with respect to ischemic treatment outcomes (p's <.05). Specifically, baseline self-deception scores were linked to smaller improvements in the reduction of ischemic episodes and 48 hour ischemic duration resulting from the combination pharmacological treatment (r's [80] = -.19 & -.2 with ischemic duration and number of episodes, respectively). After controlling for baseline disease severity with the two ischemic variables, self-deception scores reliably accounted for additional variance in each case, F's (1, 78) =5.3, 4.0, p's<.05 (two-tailed). This outcome is summarized in Table 12.

Insert Table 12 About Here

Moderator effect predictions.

We followed a two-step procedure in assessing moderator effects. Initially, we examined zero order relationships between interaction terms formed between the psychological risk factors and response style indices, and measures of baseline disease severity and treatment effectiveness. Resulting values for linear interaction terms are shown in Table 13. We employed an alpha level of .01 in the interpretation of these tests in order to minimize type I error inflation. Where zero order correlations proved reliable, we followed up by submitting the predictive interaction terms to hierarchical regression analyses in which the psychological risk factor and response style main effects had been previously forced into the equation. The same set of steps were followed in the examination of nonlinear moderator effects.

Insert Table 13 About Here

Results from these analyses were in accordance with those observed earlier among REACT participants. A number of linear interaction terms proved to be reliable predictors at the zero order level. The self-deception x hostility term was associated with smaller treatment benefits in the reduction of ischemic episodes, $r(80) = -.30$, $p<.01$. Additionally, the self-deception x daily stress score predicted poorer treatment outcomes in terms of smaller improvements in exercise performance ($r[76] = -.31$, $p<.01$). As shown in Table 13, however, neither of these terms held up in subsequent regressions. Because each of the significant interaction terms was highly correlated with main effect predictors, they were unable to account for additional variance after controlling for self-deception, hostility, and daily stress relationships. In fact, due to the multicollinearity effects created by including the highly intercorrelated interaction terms in the regression analyses, beta weights for these predictors were reduced to nonsignificance in most cases.

As seen in Table 14, our data failed to support the prediction that nonlinear interaction terms would improve our model of CHD. Again, although several quadratic effect terms evidenced reliable correlations with treatment outcomes at the zero order level, none of the interaction terms created for these analyses met our criteria for significance following steps to control for main effects (Cohen & Cohen, 1983).

Insert Table 14 About Here

Confound effect predictions.

As was the case with the REACT study, we began an assessment of confound effects by exploring the size of relationships between response style scores and psychological risk factor measures. Table 15 showcases these values. Correlations between impression management, self-deception, and psychological risk factors were

universally negative, but nonsignificant in the majority of cases. Impression management demonstrated reliable associations with anger-out and daily stress (r's[95] = -.24, -.23, with anger-in and daily stress respectively, p's<.05), whereas self-deception was related to lower anger-in and hostility scores (r's [95] = -.31, -.32, with anger-out and hostility respectively, p's<.01). Neither response style scale evidenced an association with Beck Depression Inventory values.

Insert Table 15 About Here

Zero order correlations between baseline measures of anger-in, anger-out, depression, daily stress, and hostility, measures of pre-treatment disease severity, and pharmacological treatment changes are presented in Table 16. We observed no significant relationships between the five psychological risk factors and initial disease severity as measured by the number of ischemic episodes and total ischemic duration. High levels of pre-treatment depression, however, were related to poor exercise performance (r[96]=-.20, p=.04). Baseline daily stress scores were likewise associated with an increased presence of angina at the pre-treatment period (r [92] = .23, p = .025). In addition, daily stress levels exhibited reliable associations with several indices of treatment effectiveness, including smaller reductions in the number and total duration of ambulatory ischemia, a higher incidence of angina at post-treatment, and smaller improvements on exercise testing (r's [75] =.20, .23, .26, & .19, p's<.05 [one-tailed] for ischemic duration, number of ischemic episodes, exercise performance, and the presence of angina at post-treatment, respectively).

Insert Table 16 About Here

Table 17 provides the same pattern of relationships between psychological risk factors and cardiac measures in which response style influences have been removed from the psychological variables via part correlations. The pattern of findings largely mirrors those observed prior to controlling for response style tendencies, however the absolute effect sizes (pearson r's) actually decreased in nearly every case. The previously reliable associations between depression and pre-treatment exercise performance, for example, and between daily stress levels and changes in the number of ischemic episodes and total duration, were no longer significant at the two tailed .05 level. Relationships between daily stress, the presence of angina, and changes in exercise performance also decreased in size following the removal of response style effects, but nonetheless remained statistically reliable.

Insert Table 17 About Here

Discussion

The results described in Study 2 again provide the clearest support for using response styles as main effect predictors. However, this conclusion was not as sweeping as noted in the context of the REACT investigation. Among a predominantly male sample of ischemic heart disease patients, self-deception scores collected prior to a potent pharmacological treatment program were associated with smaller treatment benefits as measured in the form of reductions in the number of episodes and total duration of ischemia. Patients with stronger self-deceptive tendencies, therefore, showed less

improvement on these measures relative to those with lower self-deception scores. These relationships also proved to be quite robust, remaining significant even after controlling for pre-treatment disease severity, and are in line with those observed between self-deception and cardiovascular health in previous studies (Denollet, 1991; Denollet, et al., 1995; Emmons, 1992; Shaw & Cohen, 1985) . Nonetheless, response styles overall showed only modest potential as predictors of cardiovascular disease variables. Self-deception did not exhibit significant relationships with any measure of baseline disease severity, and likewise failed to reliably predict either post-treatment angina or improvements in exercise performance. Impression management scores, finally, did not correlate with any of our cardiac measures.

Moderator effect terms once again offered little or no improvement to our predictor models after controlling for main effect relationships. Where significant zero order relations were observed between linear or quadratic interaction terms and treatment outcome variables, subsequent hierarchical regressions revealed that they in every case disappeared after first entering the main effect terms into the equation.

The most compelling set of findings from our perspective concerned the analyses of the confounding effects model. The aim in this circumstance was to assess the impact of response styles on a set of established psychological risk factors for cardiac disease, and we explored this issue by examining the predictive abilities of the risk factors before and after statistically controlling for response style variance with part correlations. Several of the risk factor variables - anger-in and daily stress in particular - evidenced consistent negative relationships with measures of pre-treatment disease severity and treatment outcomes. This furnished a credible stage for examining response style effects.

In contrast to hypotheses posed by the confounding effects model, however, the removal of response style influences actually lowered the predictive power of the

psychological risk factors in six of eight cases. Although none of the six affected relationships was reduced in a statistically significant sense, in three cases the correlations were no longer reliable. This outcome suggests that controlling for response style variance would have changed the conclusions drawn from this study by indicating fewer significant findings.

In two cases, correlations either did not change or increased after extracting response style variance. The negative relationship between depression and pre-treatment exercise performance was unchanged moving from zero order to part correlations, and an examination of Table 15 suggests an explanation for this stability. Specifically, whereas measures of anger expression, daily stress, and hostility each correlated at a reliable level with either self-deception or impression management scores, we did not observe a relationship between self-reported depression and the two response style dimensions. As a result, the part correlation for depression was largely unaffected.

The final significant relationship, observed between hostility and reductions in the number of ischemic episodes, does not lend itself to a ready explanation. The latter association became significant only after removing response style influences. Although this finding is entirely consistent with the confounding effects model, its anomolous status relative to changes seen among other psychological risk factors serves to complicate interpretations. On one hand, it could be that hostility is somehow affected differently by response styles, or that hostility and response styles even work together to promote behaviors that could affect treatment outcomes. Previous studies have shown, for example, that hostility may interact with defensive characteristics to predict higher blood pressure levels (Jamner, Shapiro, Goldstein, & Hug, 1991). Similarly, perhaps only hostility, due to its very negative connotations and publically regarded connection to heart disease (e.g., type A personality), promotes a high level of internal and external censorship

among this population. Finally, in light of the fact that only self-deception correlated with hostility, it appears that the removal of self-deceptive versus impression management variance has different effects.

Neither of these interpretations fits with the data observed here, however. The response styles scores collected in this study correlated as strongly with anger expression and daily stress reports as with hostility. Furthermore, interaction terms failed to account for added predictive power after controlling for main effects, suggesting that observed relationships were not a result of synergistic influences. Lastly, although it was true that only self-deception showed a suppressive relationship with hostility, the same could be said of anger-in scores. The difference is that anger-in relationships decreased rather than increased after extracting self-deception variance. Thus, although the improvement in predictive power with the hostility index was consistent with predictions, the overall pattern of findings runs in the opposite direction. The more general conclusion is that researchers should be cautious about efforts to "trim the response style fat" from other psychological measures. Instead of creating purified constructs that are more capable of forming meaningful associations with other variables, the evidence reviewed here suggests one may lose critical information. Coupled with the earlier finding that self-deception scores were associated with treatment outcomes, this may indicate the presence of a more general distress factor relevant to cardiovascular disease that is captured mutually by psychological risk factors as well as by self-deception measures.

General Discussion

This investigation examined the role(s) of response styles in the context of cardiovascular research. The results of the separately described REACT and CASIS studies raised a number of issues germane to the premise of this paper. First of all, we observed consistent evidence for conceptualizing response styles as meaningful personality constructs, and little or no evidence for the longstanding position that response styles function primarily to distort observable links between health status and the reporting of physical and psychological symptoms. Although we witnessed the expected negative correlations between response style scores and psychological risk factors in both studies, statistical extraction of the response style influences proved to have little impact on the predictive potential of the risk factor measures. In fact, the removal of response style variance actually worsened predictions of treatment outcomes among CASIS patients. This finding supports the need to identify plausible mechanisms through which response styles may themselves be linked to cardiovascular health. Researchers have speculated upon several such models in recent years, and these accounts are briefly examined here.

A second issue of note is that the connection(s) between response styles and cardiovascular health appear to be rather unidimensional. That is, only self-deception scores showed an ability to predict blood pressure and changes in ischemic heart disease variables. In no case did impression management scores evidence utility in these regards. This suggests something unique regarding the information captured by the self-deception measures, and we will touch upon the literature discriminating between the two response style dimensions.

Third and finally, our results also indicated that the self-deception-cardiovascular health relationship is relevant only to men. Although some studies have documented relationships between response styles and blood pressure among women (King, et al,

1990), the existing literature is largely in agreement with our own findings in suggesting a more consistent association among men (Jamner, et al., 1991; Linden, et al, 1993; Weinberger, et al., 1979). If true, however, why or how might response style effects differ for male populations? Several explanations are offered on this issue.

Biobehavioral mechanisms linking response styles and cardiovascular health.

A question of critical importance to the interpretation of our findings concerns the mechanisms through which response style characteristics may impact cardiovascular health. In order to argue that response style relationships represent more than correlational descriptors, one must advance explanatory models that are both consistent with existing knowledge and amenable to scientific testing.

One plausible account suggests that the repression of negative emotions can actually promote a heightened physiological response to stressful events (Gross & Levenson, 1997), possibly leading to elevated resting blood pressure levels over time. Alexander (1939) first advanced the notion that the development of hypertension and cardiovascular disease could be related to an inability to express negative emotions. Sometimes regarded as the psychosomatic model of hypertension, Alexander's research primarily centered upon repressed hostility, but the concept may be applied here more generally to negative affect states. The numerous studies showing relationships between higher defensiveness or self-deception scores and increased physiological reactivity (e.g., King et al., 1990; Shapiro et al., 1995; Weinberger et al., 1979) are consistent with this developmental model, as are results indicating associations between response style status and higher resting blood pressure levels (e.g., Jamner et al., 1991; Linden, et al., 1993). The prospective relationships observed here and elsewhere (MacGregor et al., 1998), buffer this position still further. Thus, self-deceptive characteristics could accentuate cardiovascular changes by disposing a coping style that exacerbates sympathetic nervous

system activity under stress. Whether changes result from the increased intensity of the stress response or from a prolonged duration of sympathetic activity, however, is not entirely clear.

A separate difficulty with the psychosomatic theory is that it does not clearly differentiate between the importance of the stress response and the frequency with which stress is experienced. Ironson's results (Ironson, et al., 1992) suggest, for example, that long-term cardiovascular changes may be more strongly affected by how often an individual experiences negative emotions as opposed to how the affect is expressed. Taken from this perspective, self-deceptive characteristics may impact cardiovascular functioning by disposing an increase in the number or duration of stressful encounters. Although there is little direct evidence on this issue, we earlier discussed findings that hypertensive individuals frequently demonstrate poor social skills and lower levels of assertiveness, each qualities that could increase the frequency of negative social interactions (Linden & Feuerstein, 1981). Jamner (Jamner, et al., 1991) describes a closely related process in which a type of approach-avoidance conflict may be present among self-deceptive individuals in which they repress their experience of negative emotions in order to be liked by others. In light of the recent attention given to the role of interpersonal stressors in cardiovascular disease (Linden & Rutledge, 1996), this could be a valuable area for future inquiry.

Preliminary findings from recent cardiovascular investigations also indicate that self-deception relates to a number of overt behavioral patterns that could contribute to the development or progression of disease. In particular, self-deceptive individuals may show a tendency to deny illness symptoms (Denollet, 1993), to gain less information relevant to treatment (Shaw & Cohen, 1985), receive lower levels of social support (Emmons, 1992), and to be less likely to adhere to treatment (King, et al., 1990). These behaviors have

clear ramifications for patients coping with existing disease, but could also have pathogenic consequences in some scenarios. For example, the failure to notice indications of elevated sympathetic arousal could prevent one from departing stressful encounters, or from seeing the impact a demanding job, relationship, or lifestyle is having on their health. Preventive behaviors may also suffer, resulting in a possible avoidance of screening assessments, genetic testing, routine medical checkups, and dietary or exercise habits - among a host of other factors - that impact the manifestation of disease.

A final implication of the main effect findings is that it may not be necessary to distinguish between "true" and "repressed" anxiety (if such a distinction actually exists) in order to show relationships between response styles and physical functioning. Weinberger's (Weinberger, et al., 1979) results, although also indicating associations between response styles and elevated arousal levels, differed on this point in that it was believed to be the masked emotional material and not the generalized response style pattern itself that contributed to stress reactivity. Our main effect results indicate nothing about participant's "true" standing on measures of psychological risk factors, but instead demonstrate independent predictive value.

Self-deception versus impression management.

Why might self-deception scores prove useful in the prediction of cardiovascular outcomes whereas impression management scores do not? Impression management measures, after all, share a conceptual similarity with self-deception in that both act to suppress negative self-relevant information from others. Similarly, our results, as well as those from previous research (Linden, et al., 1986), indicate that impression management and self-deception scales correlate to a similar extent with measures of psychopathology.

Interestingly, however, the value of self-deception in cardiovascular trials may have less to do with an individual's style of reporting psychological symptoms and more to

do with how they process and respond to physical signs of stress or disease. As discussed earlier (Denollet, 1991), research suggests that high self-deceivers are less likely to notice indications of physical duress, and subsequently less likely to seek out social support or medical assistance. In addition, studies show that self-deception, but not impression management, is correlated with the reporting of physical symptoms (Linden, et al., 1986). This combination of findings suggests that the self-deception dimension of response styles makes a relatively unique contribution to physical health. The cognitive aspects of self-deception leave one poorly equipped to monitor physiological arousal levels, which may affect underlying feedback systems and promote a tendency to dismiss warning signs, whereas the behavioral products of self-deception could delay help-seeking responses, act to maintain involvement in stressful encounters, or negatively affect adherance to treatment following a medical event.

Such an account is consistent with our present understanding of self-deception mechanisms. It must be emphasized, however, that the currently very small body of research on the topic makes this or any other explanation preliminary. The task of establishing behavioral concomitants of psychological risk factors remains an important objective for cardiovascular researchers at this time. Although our results indicate that self-deception may deserve inclusion among this set of health-relevant person variables, we are unable to offer definitive evidence regarding causal mechanisms for our findings. As is so often recounted, such an account requires much additional research.

An account of gender differences.

The simplest explanation for the absence of self-deception relationships among the females group is that self-deception may be a predominantly male characteristic. Recent work by Paulhus (personal communication, May 20, 1998), for example, showed that self-deception overlaps meaningfully with narcissistic personality traits, which are believed to

be more prevalent among males (DSM-IV, 1994). Perhaps a relative floor effect exists among females with respect to self-deception, an effect that would limit our ability to find correlational relationships.

An examination of the male and female subgroups from the REACT study does not support this explanation, however. Men's self-deception scores (N=151, mean = 8.19, sd = 3.11) were very similar to those from the female subgroup (N=165, mean = 8.41, sd = 3.29). Women, therefore, appear to be at least as self-deceptive as men in our sample.

The more complex interpretation is that the biobehavioral pathways linking personality features, behavioral mediators, and cardiovascular activity differ between men and women. The results from many previous studies suggest that certain personality characteristics such as hostility are more salient to males (Dembroski, et al., 1989; Linden, et al., 1993), whereas factors such as the availability of social support may be more for women (e.g., Gerin, et al., 1992). From this perspective, the relative strength of self-deception relationships among men relative to women is consistent with existing knowledge.

Unfortunately, the comfort provided by the latter interpretation is offset by the fact that it fails to bring us any closer to an explanation for the phenomena. If, as our numbers indicate, men are not simply more self-deceptive, we must look deeper for a connecting mechanism. Consistent with Alexander (1939), self-deceptive traits may represent a coping method relied upon by some men to process stressful interpersonal or life events, and the chronic internalization of the negative emotions resulting from these events could influence resting baroreceptor activity. Importantly, this view considers self-deception an agent that is activated under stressful circumstances. Given the lower inclination shown by men in some studies to engage in healthier coping methods such as social support seeking (Rutledge, et al., 1996), this may suggest that - despite similar self-deceptive

means across gender groups - men rely to a greater extent upon repressive or self-deceptive coping styles with respect to physical health. Such a model remains consistent with Gur and Sackeim's (1979) definition of self-deception as a personality feature that promotes the tendency to minimize the conscious experience of negative self-relevant information, but further add that self-deceptive qualities could affect some forms of self-relevant informations (i.e. health related) more than other. In summary, gender status may moderate the expression of self-deception characteristics. In light of the robust relationships documented here between self-deception and higher blood pressure levels among men, their consistency with previous laboratory findings, and the many as yet unresolved questions, this would appear to be a fruitful avenue for future research.

Conclusion.

Where does this collection of findings leave us? The primary objective of this thesis was to examine response styles in a forum that allowed us to ascertain their value in several different roles. Our results are among the first to tackle this issue with cardiovascular data, but they are clearly in line with conclusions drawn previously by personality researchers in suggesting response styles can be better described as personality traits. Like other person variables, response style chararacteristics vary across individuals and appear to possess both cognitive and behavioral manifestations.

Gur and Sackeim (1979) and Costa and McCrae (1983) are among those who have encouraged researchers to treat response styles as more than suppressing agents. Their arguments were based largely upon studies in the personality literature showing that the extraction of response style effects generally failed to improve predictions. Our numbers support a similar conclusion, but go well beyond the bounds of previous investigations by demonstrating response style effects in an area in which accurate predictions can have serious health implications.

In closure of this paper, it is a worthwhile exercise to briefly integrate our findings with the methodologies used in previous cardiovascular investigations incorporating response style variables. Three types of relationships are often presented with regards to personality or behavioral variables in health psychology research. Unfortunately, because most person variables relevant to health outcomes are either not subject to manipulation (e.g., gender, personality traits) or difficult to change (e.g., aggressive behavior, smoking), the great majority of these relationships (including our own) are correlational in nature. This weakness limits the interval validity of research findings, and typically requires multimodel approaches to establishing causality.

The simplest and most common form of behavioral medicine research attempts to identify concurrent relationships between person variables and health processes. Studies such as those presented earlier illustrating associations between response style scores and physiological reactivity (e.g., Shapiro, et al., 1995) are an example of this methodology. While such research is vital to our progress as a science, the results suffer from an intractable chicken and egg dilemma. An advantage of the current investigation is that we were able to go beyond this type of methodology with many of our analyses.

A second and typically stronger form of research involves relationships observed in the context of treatment outcome predictions. Treatment adherance as a predictor of post-MI mortality is a commonly employed example of this type of association in behavioral medicine research. The CASIS trial presented data of this sort, using response styles and psychological risk factors to predict the effects of a pharmacological treatment program among ischemic heart disease patients. A key benefit of this type of design is that we were able to control for at least some alternative explanations such as baseline disease severity. The finding that self-deception was able to reliably predict ischemic

improvements even after ruling out initial disease status provides a stronger endorsement of the variable.

The third and most valuable form of behavioral medicine research is also the least practical to conduct. This methodology is prospective in scope, using time 1 variables to predict changes in health status across an extended time period. Lacking the ability to cleanly manipulate person variables, prospective designs potentially offer the most robust relationships. Because they possess all the advantages of treatment outcome relationships, with the additional benefit of minimizing chicken and egg explanations (i.e., the disease causing changes in personality or behavior), prospective relationships are highly valued but rarely acquired due to the costs involved in recruiting and tracking participants for lengthy time periods. This makes the results of the REACT study particularly valuable. The associations between self-deception and higher 3-year diastolic and systolic blood pressure means held up even after controlling for physical risk factors and time 1 blood pressure levels. In combination with the many concurrent response style-blood pressure relationships discussed in previous sections this builds a compelling case for incorporating response style measures in research aiming to explicate the factors contributing to cardiovascular illness.

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TABLES 1-17

Table 1.
A description of psychological measures used at baseline testing.

Factor	Test used	Description	Scoring
Hostility	Cook-Medley scale of Minnesota Multiphasic Personality Inventory (MMPI) (Cook and Medley, 1954)	50 self-descriptive statements referring to feelings of distrust towards others.	Patient rates each statment on a 1-5 scale with high total scores meaning high hostility
Depression	Beck's Depression Inventory (Beck, 1978)	22 self-descriptive items referring to depression-related thoughts and behaviors	Items are rated on a 0-3 scale with high scores indicating depression. Possible range range of scores is 0-66.
Daily stress	Daily Stress Inventory (Brantley et al., 1987)	60 items describing frustrating events that may occur during a typical day.	Patient endorses events which occurred during past 24 hours, and rates resulting stress on a 1-7 scale. Scores (items endorsed x rating) ranges from 0-420.
Anger-In/Anger-Out	Spielberger Anger Expression Scale (Spielberger, 1985)	24 items assessing response patterns when angry	Items are rated on a 1-4 scale. The 24 items are balanced so that eight tap anger-in, anger-out, and anger-control. Range is 8-32.
Self-deception/ Impression management	Balanced Inventory of Defensive Responding (Paulhus, 1984)	40 items tap admissions to minor personal flaws & moral transgressions	Patient endorses each item on a 1-7 scale, with high scores indicating high deception. 20 items assess each dimension. Scores range from 0-20.

Table 2.

Means and standard deviations for the REACT sample on relevant demographic, physical, and psychological characteristics.

	Males(N=150, 62)		Females(N=165, 63)	
	Time 1	Time 2	Time 1	Time 2
Variable				
Age	25.9	31.3(12.4)	28.2	34.2(13.0)
Exercise(hours)	2.5(1.9)	------	2.7(1.9)	------
Body fat	106.9(27.8)	103.3(30.8)	124.2(29.0)	111.7(30.1)
Diastolic BP	81.5(12.9)	82.3(10.0)	79.6(8.1)	80.1(8.2)
Systolic BP	133.9(10.9)	131.2(11.6)	125.0(9.9)	125.4(10.8)
Smokers (yes/no)	14/146	5/60	10/154	3/48
Depression	6.4(4.7)	5.1(4.6)	8.6(6.9)	8.7(7.6)
Daily stress	42.6(24.3)	------	46.5(30.0)	------
Anger-in	17.1(3.9)	16.3(3.4)	17.2(3.9)	17.2(3.9)
Anger-out	15.0(3.7)	14.6(2.7)	15.0(3.6)	14.4(3.5)
Hostility	20.1(7.8)	17.2(8.0)	18.1(7.9)	16.4(9.5)

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* $p<.05$ ** $p<.01$

Table 3.

Male, female, and overall sample intercorrelations between baseline impression management and self-deception scores with 3-year ambulatory diastolic and systolic blood pressure levels.

	Overall sample (N=125)		Male(N=62)		Female(N=63)	
	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic
Impression Mgmnt.	.04	−.02	.19	.16	−.06	−.09
Self-deception	.20*	.21*	.41**	.41**	.01	.03

*<.05 **<.01

Table 4.

Hierarchical regression results for the prediction of 3-year ambulatory diastolic and systolic blood pressure means among men (N=63).

		Ambulatory diastolic means					
	Beta	F.	df	P-level	F-change	P-Fchange	% Variance
Step 1	------	3.1	3,55	.04	------	------	6
Physical variables*							
Step 2	.37	7.2	4,54	.002	12.2	.001	20
Self-deception							
		Ambulatory systolic means					
	Beta	F.	df	P-level	F-change	P-Fchange	% Variance
Step 1	------	4.6	3,55	.01	------	------	8
Physical variables*							
Step 2	.35	9.0	4,54	<.001	12.5	<.001	23
Self-deception							

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* smoking status, body fat levels, and exercise duration

Table 5.

Zero order and standardized beta coefficients between linear psychological risk factor x response style interaction terms and 3-year ambulatory diastolic and systolic blood pressure means.

	Males (N=62)		Females (N=63)	
	Diastolic	Systolic	Diastolic	Systolic
Self-deception x Depression	.14/.03	.00/.00	−.09/−.07	−.06/−.02
Self-deception x Anger-in	.34**/−.04	.29/−.19	−.05/−.08	.01/−.07
Self-deception x Anger-out	.32**/−.05	.23/−.15	.01/.01	.04/−.09
Self-deception x Daily stress	−.01/−.14	.00/−.04	.08/.10	.08/.10
Self-deception x Hostility	.22/−.20	.12/−.30*	.01/.03	−.04/−.08
Impression mgmt x Depression	.10/.20	−.02/.07	−.1/−.05	−.11/−.11
Impression mgmt x Anger-in	.23/.14	.16/.04	−.11/−.13	−.12/−.17
Impression mgmt x Anger-out	.18/.10	.06/.02	−.05/.04	−.07/−.04
Impression mgmt x Daily stress	−.05/−.08	−.09/−.08	.00/.04	−.05/.00
Impression mgmt x Hostility	.16/.07	.02/−.04	−.07/−.06	−.19/−.19

** $p<.01$ * $p<.05$

Table 6.

Zero order (left) and part correlation (right) coefficients between quadratic psychological risk factor x response style interaction terms and 3-year ambulatory diastolic and systolic blood pressure means.

	Males (N=62)		Females (N=63)	
	Diastolic	Systolic	Diastolic	Systolic
Self-deception x Depression	.23/.04	.17/.04	−.12/−.12	−.09/−.09
Self-deception x Anger-In	.35**/−.05	.31/−.19	−.03/−.09	.02/−.07
Self-deception x Anger-Out	.34**/−.05	.28/−.14	.02/.04	.05/−.03
Self-deception x Daily stress	.10/−.14	.14/−.02	.07/.07	.07/.05
Self-deception x Hostility	.27/−.18	.19/−.30*	.02/.03	−.03/−.08
Impression mgmt x Depression	15/.18	.05/.08	−.09/−.06	−.07/.01
Impression mgmt x Anger-In	.21/.09	.18/.03	−.08/−.10	−.08/−.12
Impression mgmt x Anger-Out	.19/.09	.11/.06	−.03/.13	−.04/.10
Impression mgmt x Daily stress	.01/−.07	−.01/−.07	.00/.05	−.03/.06
Impression mgmt x Hostility	.18//.06	.09/−.02	−.04/.01	−.14/−.08

** $p<.01$ * $p<.05$

Table 7.

Intercorrelations between self-report measures of anger-in, anger-out, depression, daily stress, hostility, and response style measures of self-deception and impression management (N=318).

	Self-Deception	Impression Management
Anger-In	−.33***	−.19**
Anger-Out	−.22***	−.26***
Daily Stress	−24***	−.16**
Depression	−.36***	−.11*
Hostility	−.28***	−.28***

* p<.05 ** p<.01 *** p<.001

Table 8.

Correlation coefficients between baseline measures of depression, hostility, anger-in, anger-out, and daily stress with 3 year ambulatory blood pressure means before/after controlling for response style effects.

	Males(N=62)		Females(N=63)	
	Diastolic	Systolic	Diastolic	Systolic
Anger-In	.04/.08	.01/.04	−.05/.01	.01/.01
Anger-Out	.00/.04	−.16/−.13	−.01/.05	.08/−.04
Daily Stress	−.18/−.12	−.2/−.14	.04/.04	.02/.02
Depression	−.07/.07	−.14/−.01	−.06/−.07	−.07/−.07
Hostility	−.01/.01	−.14/−.14	−.00/−.02	−.04/−.06

Table 9.

Correlation coefficients between baseline measures of depression, hostility, anger-in, anger-out, and daily stress with time 1 ambulatory blood pressure means before/after controlling for response style effects.

	Males(N=145)		Females(N=159)	
	Diastolic	Systolic	Diastolic	Systolic
Anger-In	.13/.14	.01/.07	−.02/−.01	−.04/−.05
Anger-Out	−.02/−.01	−.06/−.01	−.10/−.06	−.02/−.01
Daily Stress	−.08/−.10	−.10/−.09	.01/.02	.01/.01
Depression	−.04/−.04	−.15/−.10	.08/.09	−.02/−.04
Hostility	.03/.04	−.05/−.02	−.07/−.05	−.11/−.12

Table 10.

Means, standard deviations, and statistical test results for important demographic and cardiac disease variables among CASIS patients at baseline and following combination atenolol/amlodipine treatment (N=75).

Variable	Mean (Standard deviation)-Baseline	Mean(Standard deviation)-Post-treatment	F-value/p-level
Age	60.6(8.2)	------	------
Ischemic episodes	5.3(7.0)	3.1(5.3)	57.8/<.001
Ischemic duration(min)	29.8(49.1)	14.3(30.5)	36.0/<.001
Exercise time(secs)	398.5(144.7)	476.8(149.7)	64.0/<.001
Report of angina(yes)	52%	39%	9.0/<.001
Daily stress	8.3(6.7)	6.3(7.2)	------
Depression	7.9(5.3)	5.8(4.9)	------
Anger-In	17.0(4.1)	15.5(3.8)	------
Anger-Out	14.5(4.1)	13.5(3.2)	------
Hostility	119.8(12.6)	116.6(13.2)	------

Table 11.

Zero order correlations between baseline self-deception and impression management scores with measures of pre-treatment disease severity (N= 95) and treatment changes (N=80).

	Self-deception	Impression management
Pre-treatment		
Report of angina	.07	.09
Ischemic episodes	−.05	.05
Ischemic duration	.04	.05
Exercise time	.08	.03
Post-treatment		
Report of angina	.04	.09
Reductions in ischemic episodes	−.21*	−.04
Reductions in ischemic duration	−.19*	−.02
Improvements in exercise time	−.14	.10

* $p<.05$ (one-tailed)

Table 12.

Results of hierarchical regression analyses predicting reductions in the number of ischemic episodes and total ischemic duration using baseline self-deception scores.

Number of Ischemic Episodes

	Beta	F.	df	P-level	F-change	P-Fchange	% Variance
Step 1	.64	56.0	1,78	<.001	------	------	42%
Baseline severity							
Step 2	.21	21.0	2,77	<.001	4.1	.04	45%
Self-deception							

Total Duration of Ischemia

	Beta	F.	df	P-level	F-change	P-Fchange	% Variance
Step 1	.85	199.6	1,78	<.001	------	------	72%
Baseline severity							
Step 2	.16	71.7	2,77	<.001	5.3	.02	74%
Self-deception							

Table 13.

Relationship between linear response style x psychological risk factor interaction terms, baseline disease severity, and pharmacological treatment changes before (after) controlling for main effects .

	pre-treatment(N=95)				post-treatment changes(N=80)			
	Report of angina	Ischemic events	Ischemic duration	Exercise time	Report of Angina	Ischemic events	Ischemic duration	Exercise improvements
SDxA-IN	−.11	.03	−.06	.05	−.12	−.16	−.20	−.17
SDxA-OUT	.08	.01	−.02	.10	.04	−.27	−.23	−.12
SDxBDI	−.06	−.04	−.07	−.11	−.10	−.19	−.11	−.08
SDxCMHQ	.04	.00	−.06	.04	−.02	−.30*(−.03)	−.21	−.08
SDxDSI	−.17	−.02	−.11	.07	−.24	−.26	−.23	−.31*(−.14)
ODxA-IN	−.05	.07	.03	−.01	−.06	−.09	.02	.06
ODxA-OUT	.11	.04	.04	.08	.15	.06	.09	.06
ODxBDI	−.01	−.02	.01	−.15	−.05	.01	−.02	.07
ODxCMHQ	.06	.00	.02	.01	.05	.04	.03	.16
ODxDSI	−.14	−.04	−.12	.07	−.22	.21	.21	−.27

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* $p < .01$

SD - Self-Deception OD - Impression Management A-IN - Anger-In A-OUT - Anger-Out

BDI - Beck Depression Inventory CMHQ - Cook-Medley Hostility Scale DSI - Daily Stress Inventory

Table 14.

Relationship between nonlinear response style x psychological risk factor interaction terms, baseline disease severity, and pharmacological treatment changes before (after) controlling for main effects .

	pre-treatment(N=95)				post-treatment changes(N=80)			
	Report of angina	Ischemic events	Ischemic duration	Exercise time	Report of Angina	Ischemic events	Ischemic duration	Exercise improvements
SDxA-IN	−.02	.04	−.05	.07	−.04	−.22	−.21	−.18
SDxA-OUT	.10	.03	−.03	.09	.03	−.29*(.02)	−.23	−.14
SDxBDI	−.02	−.01	−.06	−.05	−.07	−.26	−.17	−.09
SDxCMHQ	.06	.02	−.05	.05	.01	−.29*(−.00)	−.19	−.11
SDxDSI	−.10	.00	−.08	.10	−.15	−.28	−.24	−.33*(−.16)
ODxA-IN	.03	.09	.03	−.01	−.01	−.07	.02	.07
ODxA-OUT	.13	.07	.04	.04	.12	.03	.06	.09
ODxBDI	.06	−.00	.02	−.08	−.01	−.02	−.07	.13
ODxCMHQ	.10	.03	.03	.01	.05	.02	.00	.14
ODxDSI	−.05	−.02	−.11	.12	−.13	−.18	−.21	−.26

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* $p < .01$

SD - Self-Deception OD - Impression Management A-IN - Anger-In A-OUT - Anger-Out

BDI - Beck Depression Inventory CMHQ - Cook-Medley Hostility Scale DSI - Daily Stress Inventory

Table 15.

Intercorrelations between self-report measures of anger-in, anger-out, depression, daily stress, hostility, and response style measures of self-deception and impression management (N=95)

	Self-Deception	Impression Management
Anger-In	−.31**	−.15
Anger-Out	−.17	−.24*
Daily Stress	−.08	−.23*
Depression	−.15	−.11
Hostility	−.32**	−.17

* $p<.05$ ** $p<.01$

Table 16.

Zero order correlations between psychological risk factors, measures of pre-treatment disease severity, and pharmacological treatment changes.

	Anger-In	Anger-Out	Depression	Daily Stress	Hostility
Pre-treatment(N=95)					
Report of angina	−.28*	−.04	−.09	−.23*	−.05
Ischemic episodes	.10	−.05	−.05	−.06	.08
Ischemic duration	-.05	−.03	−.04	−.13	−.06
Exercise time	−.10	.05	−.20*	.01	−.12
Post-treatment(N=80)					
Report of angina	−.21*	.03	−.11	−.32**	−.19
Reductions in ischemic episodes	−.09	.08	.06	−.23*	−.12
Reductions in ischemic duration	.07	.13	.02	−.20	−.08
Improvements in exercise time	.01	−.00	−.02	−.26*	.15

* $p<.05$ ** $p<.01$ (one-tailed)

Table 17.

Part correlations between psychological risk factors, measures of pre-treatment disease severity, and pharmacological treatment changes.

	Anger-In	Anger-Out	Depression	Daily Stress	Hostility
Pre-treatment(N=95)					
Report of angina	−.24*	.00	−.08	.18	−.03
Ischemic episodes	.08	−.06	.05	−.05	−.15
Ischemic duration	−.05	−.03	−.05	−.12	−.08
Exercise time	−.07	−.07	−.20*	.02	−.11
Post-treatment(N=80)					
Report of angina	−.18	.06	−.07	−.19	−.16
Reductions in ischemic episodes	−.05	.12	.09	−.22*	−.22*
Reductions in ischemic duration	.11	−.03	.05	−.18	−.15
Improvements in exercise time	−.01	−.01	−.03	−.24*	.11

* $p<.05$ (one-tailed)