Philadelphia College of Osteopathic Medicine DigitalCommons@PCOM

PCOM Psychology Dissertations

Student Dissertations, Theses and Papers

2006

Norming the MAD-AS to the STAXI-2 in a Hypertensive Population

Robert J. Liskowicz Philadelphia College of Osteopathic Medicine, robertl@pcom.edu

Follow this and additional works at: http://digitalcommons.pcom.edu/psychology_dissertations
Part of the <u>Clinical Psychology Commons</u>

Recommended Citation

Liskowicz, Robert J., "Norming the MAD-AS to the STAXI-2 in a Hypertensive Population " (2006). *PCOM Psychology Dissertations*. Paper 84.

This Dissertation is brought to you for free and open access by the Student Dissertations, Theses and Papers at DigitalCommons@PCOM. It has been accepted for inclusion in PCOM Psychology Dissertations by an authorized administrator of DigitalCommons@PCOM. For more information, please contact library@pcom.edu.

Philadelphia College of Osteopathic Medicine Department of Psychology

NORMING THE MAD-AS TO THE STAXI-2 IN A HYPERTENSIVE POPULATION

By Robert J. Liskowicz

Submitted in Partial Fulfillment of the Requirements for

the Degree of Doctor of Psychology

November 2006

PHILADELPHIA COLLEGE OF OSTEOPATHIC MEDICINE DEPARTMENT OF PSYCHOLOGY

Dissertation Approval

This is to certify that the thesis presented to us by <u>Robert J. Liskowicz</u> on the 22^{nd} day of <u>May</u>, 2006, in partial fulfillment of the requirements for the degree of Doctor of Psychology, has been examined and is acceptable in both scholarship and literary quality.

Committee Members' Signatures:

Steven Godin, Ph.D., Chairperson

Robert DiTomasso, Ph.D., ABPP

Patrick D. Conaboy, M.D.

Robert DiTomasso, Ph.D., ABPP, Chair, Department of Psychology

Abstract

Development of the Mahan and DiTomasso Anger Scale (MAD-AS) provided a valid and preferable alternative to the existing, lengthy tests of anger which are currently available. However, the MAD-AS was developed on a clinical, psychiatric population, and only one other study to date has attempted to utilize this test on a normal population. With strong links between anger and adverse physical health, and an ongoing controversy over whether anger expression versus anger suppression contributes more highly to the development and maintenance of hypertension, a prospective study measuring anger with established hypertensive subjects is being proposed utilizing both the MAD-AS, as an experimental instrument, and the State-Trait Anger Expression Inventory - 2 (STAXI-2), as an established instrument. This study hopes to lend not only more validity to the MAD-AS with a medical population, but also more evidence to the above controversy.

iii

TABLE OF CONTENTS

		Page			
TABLES					
FIGURES					
CHA	PTER				
1.	INTRODUCTION Anger Defined Anger as Emotion Hypertension Measurement and Assessment of Anger The Controversy Cardiac Reactivity Purpose of Study Research Hypotheses	1 4 8 12 16 22 25 28 28			
2.	METHODOLOGY Participants Measures Completed Procedures Statistical Analysis	31 31 32 33 34			
3.	RESULTS Descriptive Statistics Factor Analysis of the MAD-AS Correlations of the MAD-AS Factors Internal Consistency of the MAD-AS Correlations of the MAD-AS with the STAXI-2 Group Differences - Total Scores Group Differences - Subscale Scores Age as a Confounding Variable	37 38 40 48 49 51 60 61 63			
4.	DISCUSSION Research Hypotheses Demographics MAD-AS Factor Structure Internal Consistency Criterion Validity Group Differences for the MAD-AS and STAXI-2 Age Group Differences Implications and Recommendations Limitations of Study Summary	77 78 81 83 86 87 88 91 94 97 99			

,

REFERENCES

APPENDIX

Letter of Solicitation Demographic Sheet - Subject Demographic Sheet - Clinical Staff Notice to Patients MAD-AS STAXI-2

TABLES

Table				
1.	MAD-AS Factor Loadings	44		
2.	Pearson Inter-Correlations Between MAD-AS Factors	48		
3.	Corrected Item-Subscale Total Score Correlations	51		
4.	Correlations of the Sample Groups	52		
5.	Correlations of the Whole Sample	53		
6.	Correlation of Factor 1	55		
7.	Correlation of Factor 2	56		
8.	Correlation of Factor 3	57		
9.	Correlation of Factor 4	58		
10.	Correlation of Factor 5	59		
11.	MANOVA Results for MAD-AS and STAXI-2 Subscales	63		
12.	Descriptive Statistics for STAXI-2 Total Score	66		
13.	ANOVA Results for STAXI-2 Total Score	67		
14.	Multiple Comparisons for STAXI-2 Total Score	67		
15.	Homogeneous Subsets for STAXI-2 Total Score	68		
16.	Descriptive Statistics for MAD-AS Total Score	69		
17.	ANOVA Results for MAD-AS Total Score	69		
18.	Multiple Comparisons for MAD-AS Total Score	70		
19.	Homogeneous Subsets for MAD-AS Total Score	70		
20.	Descriptive Statistics for Factor 2	71		
21.	ANOVA Results for Factor 2	72		
22.	Multiple Comparisons for Factor 2	72		

vi

Tabl	e	Page
23.	Homogeneous Subsets for Factor 2	73
24.	Descriptive Statistics for Factor 3	74
25.	Descriptive Statistics for Factor 5	75
26.	ANOVA Results for Factor 5	75
27.	Multiple Comparisons for Factor 5	76
28.	Homogeneous Subsets for Factor 5	76

`

FIGURES

Figu	re				Page
1.	Scree	Plot			41

CHAPTER 1

Introduction

Anger is an emotion that is common to every person, and it is nearly a quarantee that an encounter with this emotion will occur regularly either internally or externally. Anger is a frequent and common experience and its universality, as well as its physiological and cognitive components, has long been recognized. According to Kassinove and Sukholdsky (1995), anger plays a significant role in everyday life. These researchers state that although anger may vary in frequency, intensity, and duration, problems with angry feelings and the management of anger are common reasons why people seek professional help. Kassinove (1995) noted that he has long been surprised by the lack of teaching about anger in undergraduate and graduate schools and by the small number of articles that appear in scientific literature, especially compared to the constructs of depression and anxiety. Eckhardt and Deffenbacher (1995) also point out that despite significant advances in the understanding and treatment of mood disorders, psychology seems to have focused overwhelmingly on anxiety and depression over the last century. The importance placed on these two constructs is understandable; however, in comparison, the intensity and power of anger seems to be a largely neglected area of research. Last, an assessment of anger

has yet to result in a diagnostic category with anger as the main feature.

Anger, as part of the fight or flight response, can be an adaptive response to a physical threat. According to Beck (1999), as is true of anxiety, anger is a potentially adaptive response to an appraisal of threat in a social or interpersonal situation. The experience and expression of anger can be a useful and adaptive part of social interaction as we learn to protect ourselves from others, and sometimes, from harmful behaviors. If we perceive that we are somehow being taken advantage of, it may be in our best interest to fight back, usually verbally, especially if our perception is accurate. However, it is the time when that appraisal is too frequent or inaccurate, or our response is exaggerated, or out of proportion to the situation that our behavior is no longer adaptive and becomes problematic.

Problems with anger can be either covert and not expressed at all, or overt and expressed verbally or physically against self, others, or objects. Covert anger, also referred to as suppressed or internalized anger appears to be related to a number of medical conditions including headaches, hypertension, coronary artery disease, and cancer (Kassinove & Sukhodolsky, 1995; Martin, Choi, David, & Wegner, 1999). Overt anger can lead to negative evaluations by others, a negative self-concept, low self-

esteem, interpersonal and family conflict, verbal and physical assault, property destruction, and occupational maladjustment (Deffenbacher, 1992). Many angry individuals are out of touch with their feelings and the way in which they express these feelings. Not only may they be defensive, but they also may genuinely have little selfawareness or insight into how different their emotional experience and expression patterns may be from the norm (Deffenbacher, 1995).

In the area of health, the last decade has seen increased attention to and interest in hostility, anger, and anger expression; this is partially due to the accumulating evidence that implicates anger-related behaviors and moods in the etiology of heart disease and coronary risk factors (Engebretson, Scrota, Nauru, Edward's, & Brown, 1999). Additionally, some researchers have found support for the idea that in patients with established coronary disease hostility appears to predict not only the severity of myocardial ischemia, but also to predict recurrent cardiac events such as myocardial infarction or cardiac death (Helmets, et al., 1995). According to Sharkin (1996), we are just seeing the emergence of quality research on anger, and much of this research is pointing toward adverse health consequences that seem to be associated with chronically experienced, suppressed, or aggressively expressed forms of anger and

hostility.

Additionally, most authors have defined stress in terms of external conditions that presumably produce internal strains that lead to physical disorders. Beck (1976) indicates that internal strains are manifested by states of excitation experienced subjectively as anger, anxiety, or euphoria. These states of excitation, or emotional arousal, are accompanied by increased activity of the autonomic nervous system. One or more physiological systems/organs may be affected by this autonomic arousal (Beck, 1976).

Anger Defined

Anger has been defined in many ways by many different researchers. There are multiple complexities in operationally defining anger. Often concepts of anger, hostility, and aggression are used inconsistently and interchangeably. This difficulty and the resulting problem of reviewing research remains, because a common definition for anger and its various hierarchies has yet to occur. However, it is not impossible to compare, contrast, or analyze the results of these studies. There are many ways in which anger can be conceptualized. One example is the psychometric approach, as developed by Spielberger (1999), to assess reliably the specific areas of the anger

experience and the types of expression that can occur. His approach measures anger both as a state and as a trait, along with its modes of expression, such as suppression, outward expressions, and control. With all of this in mind, it may still be important to review the various perspectives on the construct of anger.

Spielberger, Reheiser, and Sydeman (1995) recognized that the constructs of anger, hostility, and aggression are often linked and that the definitions of these constructs are often ambiguous and inconsistent. They termed the collection of these constructs together as the AHA phenomenon, believing that anger is at its core. Anger, as they pointed out, usually refers to an emotional state that consists of feelings that vary in intensity from mild irritation or annoyance to intense fury and rage. That state could, and usually does, fluctuate as the result of outside influences. As a trait, anger is defined by how prone one is to experience angry feelings over time (Spielberger et al., 1995). An individual's proneness could potentially influence the frequency and intensity of the emotional state of anger (Spielberger et al., 1995). Although hostility usually involves angry feelings, Spielberger et al. (1995) note that this concept has the connotation of a complex set of attitudes that motivate aggressive behaviors directed toward destroying objects or injuring other people. The concept of aggression, on the

other hand, generally implies destructive or punitive behavior directed towards other persons or objects (Spielberger et al., 1995).

According to Kassinove and Sukhodolsky (1995), anger is a negative, internal feeling state associated with specific cognitive and perceptual distortions and deficiencies, such as misappraisals, logical errors, and attributions of blame, injustice, preventability, or intentionality. This feeling state is also associated with subjective labeling, physiological changes, and active tendencies to engage in organized behavioral scripts. It is a combination of uneasiness, discomfort, tenseness, resentment, and frustration. As a multifaceted emotion, it varies in frequency, intensity, duration, in types of expression, and internal experience. Anger then is the sum of the person's thoughts, behaviors, and perceptions, as it relates to being learned through modeling and reinforcement (Kassinove & Sukhodolsky, 1995).

Anger is defined by Tafrate (1995) as the total experience of a short-lived, internal negative feeling state that is associated with physiological reactions, cognitive processes, and subjective labeling. The physiological reactions can include activation of the sympathetic nervous system, release of adrenal hormones, and increased muscle tension. Cognitive processes are defined as inflammatory labeling, imperious attitude, low

frustration tolerance, various cognitive distortions attributions of injustice intentionality, and blameworthiness. Subjective labeling is more an identification of the feeling on a continuum ranging from annoyance and irritation to fury and rage.

The definition of anger, according to Eckhardt and Deffenbacher (1995), refers to an internal, "cognitiveaffective/phenomenological-physiological" condition that can vary in intensity, duration, pervasiveness, and persistence. They see anger as presenting itself on a bell curve, with extreme forms and moderate forms. This definition views each element, cognitive, affective or phenomenological, and physiological, as being related, but independent parts of an overall response system (Eckardt & Deffenbacher, 1995). The cognitive element is seen as being related to information-processing styles and memory. The affective part refers to the subjective experience of feelings, and the physiological area includes autonomic arousal, endocrine changes, and muscle stimulation.

From these perspectives, it may be observed that anger is viewed as a uniting experience, which may include beliefs, behaviors, thoughts, reactions, perceptions, internal states, and history. In all these, the common thread is that anger is multifaceted and exists on multiple continuums.

Anger As Emotion

For some people the thought of anger conjures up an image of a person in a rage. They may have images of slamming doors, shouting, and intimidating communication. Certainly, this can be part of an angry response. However, anger is not one-dimensional; rather, it is multifaceted. It can be found in any temperament; whether a person is shy or is extroverted, perfectionistic or laid-back, he or she can show anger in many ways. Anger is a term that can describe a number of expressions: frustration, irritability, annoyance, blowing off steam, and fretting. The subjective feeling of anger may vary from mild irritation to rage (Beck, 1999). It is a frequent and common emotion that presumably underlies some of society's most serious problems. Anger can also be completely normal and healthy as a human emotion. When anger does get out of control and turns destructive, it can lead to problems, such as crime, domestic violence, abuse, road rage, and substance abuse. When anger is based on honest, realistic convictions, and is expressed assertively and respectfully, then a productive and reasonable outcome may occur. Anger can be used to motivate or to convert stresses to strengths, or it can be used as a weapon to hurt or intimidate. There may be informative value with this emotion. Beck (1999) sees anger as being able to provide a

person with signals that a threat is present or to compel a person to identify the source of the aggravation in order to take corrective action. Anger is also defined as a normal and useful emotion when it is based on honest and realistic convictions. It can be a powerful and unpredictable emotion.

Anger has several components that are important to review, including those that are the physiological, cognitive, and behavioral. These components are not mutually exclusive of each other. As with all other emotions, these components interact and influence each other in a nearly simultaneous manner, its communication consisting of bodily reactions and verbalizations.

The James-Lange theory of emotions (Lange & James, 1922) proposed that the body has specific physiological responses to aversive stimuli, and that feelings are actually perceptions of the body's reaction. From their perspective the physiological reaction, such as increased heart rate, increased perspiration, tightness in the stomach, changes in facial muscles, and so on occurs first; then the person feels angry. The angry feeling follows the specific bodily reaction. Walter Cannon (1929) and Philip Bard (1935) considered this flow of events incorrect. It did not appear likely that the body had physiological reactions specific to each emotion. Increased heart rate and perspiration are common reactions to a number of

feelings. They also questioned the likelihood that there are specific facial muscle changes that occur for each feeling. They proposed that the physiological arousal of the body is general in nature, and that this general arousal and the feeling occur simultaneously. As a result, the James-Lange theory became largely rejected. Recent research, however, has lent evidence to support this theory. For example, a study utilizing anger, sadness, and fear as emotional constructs, found that blood pressure responses were specific to these emotions when produced in imagined situations (Rajita, Lovalo, & Parsons, 1992). Another study (Laird, Cuniff, Sheehan, Shulman, & Strum, 1989) reported that when students were induced either to smile or to frown, they reported developing congruent feelings. The perceived changes in feelings have been explained by changes in cerebral blood flow and cerebral temperature caused by muscle changes in the face, which may have an effect on emotion-linked neurotransmitters (Zajonc, Murphy, & Inglehart, 1989). Despite the controversy, the physiological component of anger generally refers to the changes in autonomic arousal, in adrenal changes, and other endocrine alterations.

The cognitive element of anger refers to encoding and information processing styles, and includes concepts such as attention and scanning, attributions, attitudes, concept accessibility and memory, emotional scripts, self-talk, and

imagery to name a few. Anger is related to a person's enduring cognitive characteristics. Anger is often aroused by challenges to important personal schema, a blameful attack on one's ego identity (Lazarus, 1991), a trespass on a person's own domain (Beck, 1976), violations of personal rules for living and codes of conduct (Ellis, 1977), or frustration of goal-directed behavior.

The behavioral component encompasses overt motor behavior and verbal forms of expression. However, this may be too simplistic. According to Salzinger (1995), behavior can be classified into three general classes: operant, respondent, and hybrid. Operant behavior pertains to behavior that acts on the environment and is controlled by the consequences received as a result of the behavior (Salzinger, 1995). Respondent behavior is related to behavior that is elicited by the environment. Salzinger (1995), however, describes situations in which, both operant and respondent behaviors occur simultaneously or sequentially, leading the last type of behavior to be termed hybrid.

The affective component refers to the internal, subjective experience of specific feelings that one actively labels and identifies (Kassinove & Sukhodolsky, 1995). Anger is aroused by four classes of stimuli: identifiable circumstances (waiting in slow traffic), behavior of others (criticism), objects (a computer that

does not run), and one's own behavior and characteristics (oversleeping) (Deffenbacher, 1999).

Anger can be caused both by external and by internal events. It can be directed toward a specific person or event. It can be caused by worry or ruminations over personal problems. Memories of traumatic events can trigger this emotion, as well.

Hypertension

The most commonly used International Classification of Diseases (ICD-9) code in the United States is hypertension (Messerli, 2003). It is also the most common diseasespecific reason for patients to visit a physician. Its prevalence is so high, that most physicians and health care providers will deal with hypertensive patients almost everyday regardless of the reason. Aside from its prevalence, hypertension has an enormous impact on public health. Hypertensive cardiovascular disease has been identified as the fourth leading cause of disability worldwide, surpassed only by malnutrition, perinatal diseases, and infectious diseases (Messerli, 2003).

In 2006, the estimated direct and indirect cost of cardiovascular disease is \$403.1 billion (American Heart Association, 2006). High blood pressure is estimated to occur in 1 out of every 3 adult Americans, and the

estimated overall cost is \$63.5 billion for 2006 (American Heart Association, 2006). Demographically, hypertension affects a higher percentage of men than of women until age According to the United States Department of Health 45. and Human Services (2004), the prevalence of high blood pressure in Americans by age group reflects a steady incline in percentages among both men and women as age increases. Specifically, 11.1% of men and 5.8% of women in the 20 to 34 year age range have high blood pressure; 21.3% of men and 18.1% of women in the 35 to 44 year age range; 34.1% of men and 34.0% of women in the 45 to 54 year age range; 46.6% of men and 55.5% of women in the 55 to 64 year age range; 60.9% of men and 74.0% of women in the 65 to 74 year age range, and 69.2% of men and 83.4% of women in the 75 years and older age range (U.S. Department of Health and Human Services, 2004). The prevalence of hypertension among blacks in the U.S. is among the highest in the world, and compared with whites, blacks develop hypertension earlier in life, have higher average blood pressures, and are at greater risk for having strokes and heart disease related deaths (American Heart Association, 2006). Compared with white women, black women have a higher prevalence of hypertension, and a higher rate of ambulatory medical care visits for hypertension (American Heart Association, 2006). Hypertension was listed as a primary or contributing cause of death in about 277,000 of over

2,440,000 deaths in the United States in 2003 (American Heart Association, 2006).

It is presently recommended that antihypertensive therapy start in patients who have confirmed hypertension, which is generally defined as a blood pressure exceeding 140/90 mm Hq (Messerli, 2003). One problem with this recommendation, according to Messerli (2003), is that many more patients with blood pressures lower than 140/90 mm Hq have heart attacks, strokes, and other cardiovascular events than do patients who have blood pressure readings above that mark. Also, Messerli (2003) notes that blood pressures should be distinctly lower in treating certain groups of patients, such as those with diabetes, renal failure, and congestive heart failure. Hypertension, therefore, may be most pragmatically defined as a blood pressure level that increases the cardiovascular risk for a given patient, whereas, normotension, or the absence of hypertension, would be defined as a blood pressure level that has no impact on this cardiovascular risk (Messerli, 2003). Essential hypertension is a condition of chronically high blood pressure, in which essential means that the cause is unknown. Secondary hypertension results from a disease specific problem that has as its side effect elevated blood pressure. The American Heart Association (2006) defines high blood pressure as systolic pressure of $140\,$ mm Hg or higher or diastolic pressure of 90 mm Hg or

higher; taking antihypertensive medicine, or being told at least twice by a physician or other health professional that he or she has high blood pressure.

Many patients have higher blood pressure levels when measured by a physician in the office than when measured at This is known as the white coat effect, which is home. very simply a measure of change in blood pressure triggered by the presence of a physician (Messerli, 2003). A distinction can also be made between this phenomenon and what is referred to as white coat hypertension, which is high blood pressure levels solely in the physician's office and normotensive values at home (Messerli, 2003). Thus, the white coat effect is causing the white coat hypertension, but the white coat effect may also be present in patients with established essential hypertension. According to Messerli (2003), the white coat effect is very common in patients with more severe essential hypertension whether treated or untreated, in the elderly, in women, and In patients who have isolated systolic hypertension.

The measurement of blood pressure is likely the clinical procedure of greatest importance, because when measured carefully, it remains one of the most powerful and accurate determinants of cardiovascular status and future cardiovascular events (Messerli, 2003). Although blood pressure is an extremely variable parameter, because it varies from time of day, season of the year, conscious

state and position, it has been well documented that blood pressure taken under standardized conditions in a physician's office is one of the most valuable clinical tools available (Messerli, 2003).

Measurement and Assessment of Anger

In the vast domain of psychological-hypertension literature, a wide range of psychological tools have been utilized to measure anger characteristics. More than a dozen instruments can be identified; however, some of these were not verified by strong psychometric data. Rutledge and Hogan (2002) strongly support the idea that if future research in this area is to be advanced, then established measures with strong psychometric and predictive associations must be applied. The importance of including psychological scales with proven reliability and validity must be highlighted in the investigation of anger and its possible effects on hypertension. Inconsistent results observed across studies may be the result of frequent reliance on psychological assessment tools that do not demonstrate merit. In this vein, it may be of utmost importance to lend increasing amounts of data to experimental devices only when established devices become the comparison.

Although elements of emotional dysregulation and

psychological traits that increase emotional reactivity are important characteristics to cardiac reactivity, the responses of subjects with severe psychopathology symptoms may confound the interpretation of any anger assessment. As a result, screening the subjects for established diagnoses related to a current thought disorder, dementia, paranoid disorder, or traumatic brain injury will be necessary to establish exclusions from the study.

State-Trait Anger Expression Inventory (STAXI-2). The State-Trait Anger Expression Inventory-2 (Spielberger, 1999) is the newest revision of the state-trait inventories. The STAXI-2 assesses state anger, trait anger, and anger expression as do previous ones, but has been revised and expanded from 44 to 57 items. The STAXI-2 was developed to assess components of anger for detailed evaluation both of normal and of abnormal personalities, and to provide a means of measuring the contributions of these components to the development of various medical conditions, particularly hypertension, coronary heart disease, and cancer. It consists of six scales, five subscales, and an Anger Expression Index, which provides an overall measure of the expression and control of anger. In the STAXI-2, three of the five original scales remain unchanged, including Trait Anger, Anger Expression-Out, and Anger Expression-In. The Angry Temperament and Angry Reaction subscales also remain the same. Changes in the

newest version at the scale level involve the Anger Control-Out Scale, which was expanded from seven to eight items, The Anger Control-In Scale, which is entirely new, and the State Anger Scale, which has been expanded from ten to fifteen items.

The STAXI-2 measures the experience of anger, which is composed of two major components, state anger and trait anger, according to Spielberger (1999). State anger is defined as a psychobiological emotional state or condition marked by subjective feelings that vary in intensity from mild irritation or annoyance to intense fury and rage. This type of emotional state is generally accompanied by muscular tension and by arousal of the neuroendocrine and autonomic nervous systems (Spielberger, 1999). Trait anger is defined as the individual differences in the disposition to perceive a wide range of situations as frustrating or annoying and by the tendency to respond to these situations with greater state anger (Spielberger, 1999).

Anger expression and anger control are conceptualized by Spielberger (1999) as having four major components: Anger Expression-Out, Anger Expression-In, Anger Control-Out, and Anger Control-In. Anger Expression-Out refers to the expression of anger toward other persons or objects in the environment. Anger Expression-In is defined as holding in, suppressing angry feelings or directing these feelings inward. Anger Control-Out is based on the control of angry

feelings by preventing expression of anger toward any person or object in the environment. Anger Control-In is based on control of suppressed anger by calming down when angered.

The STAXI-2 was normed, based on the responses of approximately 1,900 subjects from two heterogeneous populations; these included a sample of 1644 normal adults, and a sample of 276 psychiatric inpatients from a dual diagnosis program. The mean age for the total sample was 27 years, with a range of 16 to 63 years. Alpha coefficient measures of internal consistency were uniformly high across all scales and subscales (.84 or higher, median r = .88). One exception to this was for the four item T-Ang/R subscale for normal adults, which was .76 for normal females and .73 for normal males. Spielberger (1999) concludes that the internal consistency reliabilities of the scales and subscales are satisfactory and were not influenced either by gender or by psychopathology.

The STAXI-2 is designed to be brief, easy to administer, easy to score, yet possessing strong psychometric properties. It can be administered both to adolescents and to adults with a sixth-grade reading level. Individuals rate themselves on each item according to a four-point Likert-type scale that assesses either the intensity of their angry feelings at a particular time or how frequently anger is experienced, expressed, suppressed,

or controlled. It is generally completed in 12 to 15 minutes.

Last, the STAXI has been used extensively in research in behavioral medicine and in health psychology, as well as in the effects of anger and its components, as measured by the STAXI, on blood pressure, hypertension, cardiovascular reactivity, and heart disease. The only shortcoming of the STAXI-2 may be the lack of ethnic and racial information on the norming of the samples, because there is a lack of descriptive data concerning the cultural make-up.

The Mahan and DiTomasso Anger Scale (MAD-AS). The MAD-AS (Mahan, 2001) is a 43 item, Guttman style scale used for measuring anger. The items were chosen by an independent review of experts in the field and only those items upon which there was 100% agreement were retained. In his 2001 study, Mahan administered the MAD-AS to 180 participants, broken equally into three groups of 60 to represent an inpatient psychiatric group, a psychotherapy outpatient group, and a control group of subjects not currently in psychotherapy. Factor analysis of the results suggested that the scales measured several components of anger. These include: Anger Dyscontrol (Scale 1), Anger Cognitions (Scale 2), Verbal Anger Expressions (Scale 3), Physiological Arousal (Scale 4), Anger Justification (Scale 5), Externalization (Scale 6), and Anger Resolution (Scale 7). These subscales appear to be homogeneous and stable

over time, with the exception of the Anger Resolution Scale. The instrument was modeled after the Beck inventories, because each item is composed of four sentences that measure the absence or presence of a critical aspect of the construct of anger, including its frequency, intensity, or duration rated on a scale of zero to three. Mahan (2001) also describes similarities between the MAD-AS and the STAXI based on several factors. The Behavioral Dyscontrol factor on the MAD-AS was found to be similar to the AX/Out (Anger Expression-Out) scale on the STAXI because people with high AX/Out scores express anger in aggressive behavior directed toward other persons or objects in the environment. The Verbal Expression of Anger factor on the MAD-AS was also found to be similar to the AX/Out scale, because anger may be expressed verbally in a variety of forms. The Physiological Arousal factor on the MAD-AS compares favorably to the AX/In (Anger Expression-In) scale of the STAXI because people with high AX/In scores experience angry feelings, but tend to suppress them, leading to physiological symptoms. Last, the MAD-AS Externalization of Anger factor displayed a similarity with the T-Anger (Trait Anger) scale on the STAXI because people with high T-Anger often feel they are being treated unfairly by others and are likely to experience a great deal of frustration.

This test appears to represent the development of a

stylistically new and shorter scale for measuring the selfreported physiological, cognitive, and behavioral aspects of anger. Preliminary research supports the construct validity, internal consistency, reliability, and testretest reliability of the scale (Mahan, 2001).

The Controversy

According to the now classic psychosomatic hypothesis by Alexander (1939), the inhibition of angry feelings contributes to the development of hypertension. Reasoning that hostile provocation leads to acute increases in blood pressure in normal persons, Alexander (1939) thought that suppressing one's rage may lead to chronically elevated blood pressure. Six decades later, this proposal continues to motivate research, and although distinct patterns of cardiovascular activation associated with anger have been identified, the role of anger in the development and progression of hypertension is still unclear.

Conceptual distinctions have been offered to refer to different characteristic styles of behavioral response while experiencing anger, and these are the concepts of anger-in and anger-out. Anger-in is typically defined as actively withholding or inhibiting anger expression, whereas anger-out refers to the tendency to respond with verbal or physical aggression (Spielberger et al., 1985). To review the literature on hypertension and anger or hostility is an exercise in discovering numerous conflicts and questionable methodologies. Some studies report that hypertensives bottle up their anger, and others report that hypertensives are more irritable and explosive. Still others find no differences.

A review of the literature by Siegman (1993), reports that five of seven studies that tested the presumed differential relationship between measures of anger-out and anger-in obtained significant positive correlations between anger-out and systolic blood pressure reactivity, and six obtained significant positive correlations between angerout and diastolic blood pressure reactivity. However, there were no significant positive correlations between anger-in and cardiovascular reactivity.

Several studies have examined the influence of suppressed hostility or anger-in on blood pressure, and found that anger-in was positively related to resting blood pressure, prevalent hypertension, atherosclerosis, and adverse lipid profile (Everson, Goldberg, Kaplan, Julkunen, & Salonen, 1998; Eng, Fitzmaurice, Kuzbansky, Rimm, & Kawachi, 2003). Along with these studies, the early psychodynamic research found that hypertensives, including those with borderline hypertension, reported greater intensity of anger and more repressed hostile wishes, or anger-in, than normotensives (Everson et al., 1998).

An idea that is taking hold to bridge this controversy is that expressions of anger or hostility that deviate from the norm in either direction, whether it is withholding or repressing feelings, or outright displays of anger and aggression, may be related to elevated risk of hypertension. A model developed by Linden and Feuerstein (1981), posits the theory that extreme forms of anger responses may be linked with higher blood pressure levels, but a preference for assertive or more temperate responses to angering situations, that is responses that fall between the extremes of anger-in and anger-out, may be associated with lower blood pressure levels. Both anger-out and anger-in can be characterized as resentful styles that serve to prolong feelings of anger and thus sustain elevations in blood pressure (Harburg, Blakelock, & Roper, 1979). Everson et al. (1998), showed a positive relationship between increasing anger scores with both anger expression styles and increasing risk for hypertension over a four year period. A prospective study by Gallagher, Yarnell, Sweetman, Elwood, and Stansfeld (1999), using only male participants, reported similar results; both anger-out and suppressed anger were predictive of incident heart disease.

Studies examining the effects of psychological intervention have provided some important information regarding the relationship between anger and cardiovascular

variables. Therapeutic attempts to decrease verbally and physically aggressive behaviors and to increase constructive, verbal, angry behavior have been successful in the reduction of resting blood pressure in samples of heart disease patients (Davidson, MacGregor, Stuhr, and Gidron, 1999; Linden, Lenz, and Con, 2001). In a recent study of anger coping styles, Eng et al. (2003) concluded that moderate levels of anger expression were protective against the development of cardiovascular disease.

Cardiac Reactivity and Physiology

Research has shown that the full blown expression of anger is associated with heightened cardiovascular reactivity (CVR), and a risk for coronary heart disease (Siegman, 1993). The relevance of the relationship between anger expression styles and CVR is that cardiovascular hyperactivity is thought to be involved in the development of coronary heart disease (Kaplan, Botching, & Maniac, 1993), as well as, in the development of essential hypertension (Fredrikson & Matthew's, 1990).

According to the reactivity hypothesis (Fontana & McLaughlin, 1998), the cumulative effects of excessive cardiovascular reactivity contribute to the development of hypertension and subsequent coronary heart disease. Chronic anger may arouse sympathetic activity and activate

the hypothalamic-pituatary-adrenocortical axis, resulting in elevated levels of serum catecholamines that can adversely affect blood pressure, heart rate, and free fatty acids (Eng et al., 2003). Repeated episodes of anger are believed to cause endothelial damage and promote ateriosclerosis through hemodynamic stress; in addition, intense anger may trigger acute coronary events by initiating vascular and prothrombic changes (Eng et al., 2003).

The impact of psychosocial factors on CVR represents an important line of investigation, because cognitiveemotional responses are important contributors to physiological responses. The role of reactivity in disease pathogenesis remains complicated by the multiple physiological and psychological levels that interact to increase an individual's risk; however, if stress can contribute to the disease process, it becomes possible to argue that more reactive persons, who experience various styles of anger expression, will be more likely to develop cardiovascular disease.

In a review of studies assessing associations between psychological factors and hypertension development, Rutledge and Hogan (2002) calculated a hypertension risk difference of approximately 8% among high psychological distress groups versus low psychological distress groups. Their study also suggests that high standing on anger

(including measures of anger-in, anger-out, and hostility), anxiety, and depression scales is linked to an appreciable increase in prospective risk of hypertension development; this is a level of risk that compares favorably with better established predictors of hypertension, such as obesity and physical inactivity. Another recent study examined the role of anger on cardiovascular activity (Chang, Ford, Meoni, Wang, & Klag, 2002), and discovered that high levels of anger in young men had increased risk and incidence of premature cardiovascular disorders. It was also noted that no specific anger reaction was more or less predictive than another, rather the relationship came out of the highest overall scores of anger with their assessment instrument. Last, there is data that argues for social stressors in the development of hypertension. A study by Gentry, Chesney, Gary, Hall, & Harburg (1982) support earlier observations that persons residing in high socioecological stress areas have more evidence of hypertension and a higher rate of hypertension mortality than do their counterparts who live in low stress areas. They argue that by virtue of living in high stress areas, individuals are predisposed to experiencing a greater number of anger-provoking situations than are persons in low stress areas.

The controversy over suppressed and expressed anger may continue to be debatable; however, there is robust support for the relationship between psychological factors,

such as anger, and hypertension.

Purpose of Study

The purpose of this study is to extend the psychometric evaluation of the MAD-AS to a normal, hypertensive population, and continue to examine the construct validity and reliability of the MAD-AS by comparing it to an established anger assessment instrument, the STAXI-2. Given the great expense in terms both of the human suffering and of the financial burden to society, the link between anger and cardiovascular disorders, such as hypertension, is important to explore. The task of more clearly defining anger and developing better, more accurate measures that will aid in identifying those individuals who have problems with anger becomes crucial.

Research Hypotheses

1) A significant positive correlation is expected between the scores of the STAXI-2 on the Anger Expression Index (AX Index), providing a measure of total anger expression, and the total scores of the MAD-AS both with the control and with hypertensive subjects.

2) Hypertensive subjects will score significantly higher both on the STAXI-2 AX Index and on MAD-AS total score in comparison to the control subjects.

3) A significant and positive correlation is expected between the MAD-AS and STAXI-2 hypertensive groups, and the MAD-AS and STAXI-2 control groups, on the following subscales: Anger Expression-In (STAXI-2) with Physiological Arousal (MAD-AS), Trait Anger (STAXI-2) with Externalization of Anger (MAD-AS), Anger Expression-Out (STAXI-2) with Behavioral Disturbance (MAD-AS), Anger Expression-Out (STAXI-2) with Verbal Expression (MAD-AS). 4) Significant and positive differences are expected on the following subscales when comparing the hypertensive and control groups: Anger Expression-Out, Anger Expression-In, Trait Anger, Behavioral Dyscontrol, Verbal Expression, Physiological Arousal, and Externalization of Anger. 5) Factor structure of the MAD-AS in the current study will correspond to the factors extracted from the original Mahan (2001) study, which is expected to include the following seven factors: (A) Anger Behavioral Dyscontrol, (B) Angry Cognitions, (C) Verbal Expressions of Anger, (D) Physiological Arousal, (E) Anger Justification/Blame, (F)Externalization of Anger, (G)Difficulty with Anger Resolution.

6) The MAD-AS total scores and factor (subscale) scores are expected to demonstrate internal consistency utilizing a summated coefficient alpha of greater than .70.

7) Corrected item-subscale total score correlations will be

positive and significant for the MAD-AS subscales.

CHAPTER 2

Method

Participants

Participants consisted of a sample of 450 patients selected from a general family medicine practice. The 450 patients were split into a control group and a hypertensive group, each consisting of 225 patients. The control group was a set of randomly selected patients identified as having a non-chronic illness, visiting the office for an acute medical problem. The hypertensive group consisted of patients identified as having the diagnosis of hypertension as set forth by the practice, and were making a visit to the office, regardless of purpose of visit. All potential participants were solicited for participation in the study when arriving for their appointments. The age range of the participants was limited to between 18 and 55 years. A review of the consent form was required by the patients before becoming part of the study. They were advised, in writing, about the nature of the study and all participants were informed of their freedom to withdraw from the study at any time. Only age, gender, marital status, and years of education were recorded, and all information remains anonymous. Prospective participants were screened for a Current history of Psychotic Disorder, Paranoid Disorder,

Dementia, and Traumatic Brain Injury; any positive identification of above disorders constituted exclusion from the study in order to maintain a non-psychiatric population from the study.

Measures Completed

The State-Trait Anger Expression Inventory-2(STAXI-2) is a 57 item anger assessment that can be completed by most people with a sixth grade reading level. The STAXI-2 was normed, based on the responses of a heterogeneous sample of 1,900 normal adults and hospitalized psychiatric patients with a mean age of 27 years. Internal consistency alpha coefficients were uniformly high across all scales and subscales; the alpha coefficients for the scales showed a range from .73 to .95, and from .73 to .93 for the subscales. The reported internal consistency for the scales and subscales are satisfactory and not influenced by psychopathology or gender (Spielberger, 1999).

The second measure is The Mahan and DiTomasso Anger Scale (MAD-AS), which is a 43 item anger assessment scale. Factor analysis of the results of the preliminary study suggests that the scale measures several components or subscales of anger that appear to be homogeneous and fairly stable over time. Preliminary research supports the construct validity, internal consistency, reliability, and

test-retest reliability of the scale (Mahan, 2001).

Procedures

Participants included those family practice patients ages 18 to 55 who volunteered for the study. Potential participants were identified by the clinical staff (physicians, physician assistants, and nurse practitioner) of the family practice as their charts were pulled for scheduled appointments or walk-in visits. Although each of the measures is a self-report assessment, the office and clinical staff directly associated with patient care from initiation of visit to conclusion, were trained and familiarized by the author with the letter of solicitation, self-report measures, data collection, and purpose of study. Utilizing the time that patients waited to be seen by clinical staff, the participants were encouraged to complete the STAXI-2, MAD-AS, and demographics, which included age, gender, marital status, and years of education. A collection box was set up at the front desk for deposit by the patient. If the participant, for any reason, could not complete the packet before leaving the office, an addressed, stamped envelope was provided to the person in order to complete the packet and return it to the office. The custom of this family practice is to begin each visit with a weigh-in and blood pressure reading;

these were also obtained and recorded on the packet along with the assessments and demographics. Participants were anonymous, because the assessments were numbered and only general information, as described above, was obtained. The only participants to be excluded were those identified with a current diagnosis of Psychotic Disorder, Paranoid pisorder, Dementia, or Traumatic Brain Trauma. Patients of the practice were informed through postings within the waiting area that the practice was taking part in a study in conjunction with PCOM and that some patients may be asked to participate. Participants were informed of the purpose of the study within the solicitation letter and were allowed to withdraw from the study at any time.

statistical Analysis

A psychometric analysis of the results was conducted utilizing descriptive statistics, factor analysis, Pearson correlations, item reliability analysis, corrected itemsubscale total score correlations, and multivariate analysis of variance. Descriptive statistics included measures of central tendency, standard deviations, and frequency distributions of the demographic and medical information gathered. Analysis of the results was carried out utilizing the Statistical Program for the Social Sciences, version 11.0, for Windows (SPSS) to create a database in which to enter the information. The database was independently entered and verified by the researcher. Verification consisted of the researcher's double-checking data entry for each response for every protocol.

Strategies to test Hypothesis 1. The total score on the MAD-AS for each sample group (hypertensives vs. control) was correlated with total scores on the STAXI-2 for each sample group, utilizing the Pearson Product Moment Coefficient of Correlation.

Strategies to test Hypothesis 2. A multivariate analysis of variance using sample group as the independent variable (hypertensive and control group) and total scores of the MAD-AS and STAXI-2 as dependent variables was calculated.

Strategies to test Hypothesis 3. The MAD-AS subscales for each sample group were also correlated to the STAXI-2 subscales for each sample group using the Pearson Product Moment Coefficient of Correlation, providing specific correlations between the MAD-AS and STAXI-2 subscales.

Strategies to test Hypothesis 4. A multivariate analysis of variance was utilized. The independent variable was the sample group (hypertensive and control group) and the total scores on each individual subscale of the MAD-AS and STAXI-2 listed (dependent variables) were calculated for this evaluation.

Strategies to test Hypothesis 5. In comparing the

factor structures of the MAD-AS in this study and Mahan's study (Hypothesis 3), a principal component, varimax rotated factor analysis of the MAD-AS items was utilized to identify a set of variables or factors. Mahan (2001) utilized a criterion of eigenvalues greater than 1 and extracted seven factors accounting for 62.3% of the variance. Factor loadings criterion for retaining an item on a given factor are noted to be equal to or exceeding .45 according to Mahan (2001). A confirmatory factor analysis was therefore utilized to test the equivalence of factor structures across both Mahan's and the current study's groups.

Strategies to test Hypothesis 6. Cronbach's coefficient alpha reliability was calculated to assess the internal consistency of the total MAD-AS scale as well as for each subscale.

Strategies to test Hypothesis 7. Corrected itemsubscale total score correlations were calculated for the MAD-AS subscales as an additional measure of internal consistency. Corrected item-subscale score correlations are calculated by correlating the score on each item on a given factor (subscale) with the corrected subscale total score. This corrected subscale total score is obtained by summing all the items on a given factor except for the specific item being examined.

CHAPTER 3

Results

Analysis of the results was conducted utilizing the statistical Program for the Social Sciences, version 11.0, for Windows (SPSS). First, descriptive statistics were calculated, including measures of central tendency, standard deviations, and frequency distributions for the demographic data. Descriptive statistics were also outlined for both sample groups, control and experimental, separately, and also for the sample group as a whole. A factor analysis was performed on the MAD-AS scores to determine a factor structure or set of variables. This set of factors was then compared to previous studies' factor structures for corresponding extractions. Several analyses were conducted to test each of the seven hypotheses. То ascertain internal consistency of the MAD-AS, coefficient alpha reliability was calculated for the total MAD-AS scale, as well as for each subscale. Also, corrected itemsubscale total score correlations were computed for the MAD-AS subscales as an additional measure of internal consistency. The criterion validity of the MAD-AS was examined by utilizing Pearson Product Moment Coefficients of Correlation for both total scores of the MAD-AS and

STAXI-2, and also for all relevant subscale scores of the MAD-AS and STAXI-2. Group differences between various anger scores on the MAD-AS and STAXI-2 were examined by conducting a multivariate analyses of variance (MANOVA). Further examination of the group differences was scrutinized for confounding variables, namely age. Using a two-way ANOVA, age was examined for its influence both on the control and on hypertensive group for total scores and several of the subscales with both anger assessment instruments. A statistical significance level, alpha, of .05 was selected for all statistical tests.

Descriptive Statistics

A total of 450 subjects between the ages of 18 and 55 satisfied criteria for inclusion in this study, 225 in the experimental (hypertensive) group and 225 in the control (non-hypertensive) group. Excluded from this study were 57 volunteers, who either did not return packets, complete materials within packets sufficiently, or withdrew from the study before completion of packet.

Of the 450 subjects, 253 (56.2%) were females and 197 (43.8%) were males with a mean age of 38.60 years. There

were 245 (54.4%) married subjects compared to 157 (34.9%) single, 37 (8.2%) divorced, 9 (2.0%) separated, and 2 (0.4%) widowed. The average years of education for the total group was 15.03 (SD=2.82) with a minimum of 7 years and a maximum of 25 years reported. The median for years of education was 16.00 and the mode was noted to be 12.

Experimental Group. The experimental group of hypertensive patients, (n=225) had a mean age of 44.68 (SD=8.22), and consisted of 126 males (56%) and 99 females (44%). Average years of education was observed to be 14.44 (SD=2.85). Blood pressure observations revealed a mean systolic pressure reading of 134.39 (SD=15.01) and a mean diastolic reading of 82.82 (SD=8.42). The average height for this group was 68.08 inches (SD=3.64); this included a minimum height of 55 and a maximum of 77. Height was observed to have a negatively skewed distribution. The average weight was noted to be 197 pounds (SD=44.99) with a minimum weight of 74 and maximum of 375; the median weight was 192 and the mode was 200. Weight displayed a positively skewed distribution. Of this group, there were 151 (67.1%) married subjects, 46 (20.4%) single, 5 (2.2%) separated, 22 (9.8%) divorced, and 1 (0.4%) widowed.

Control Group. The control group (n=225) was composed of 71 males (31.6%) and 154 females (68.4%) of whom the average age was 32.53 (SD=10.19). The marital status of these subjects consisted of 94 (41.8%) married, 111 (49.3%) single, 4 (1.8%) separated, 15 (6.7%) divorced, and 1 (0.4%) widowed. The systolic blood pressure readings conducted for this group showed an average reading of 117.68 (SD=11.63), and the diastolic readings had a mean of 74.52 (SD=8.39). The average height for this group was 66.38 inches (SD=4.18), and the average weight was 162.09 pounds (SD=42.47). A positively skewed distribution was observed both in height and in weight for this group of subjects. The median weight was 150.00 and the mode was 135; the minimum weight registered was 96 and the maximum for these subjects was 350.

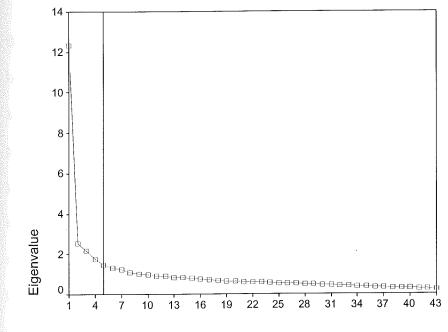
Factor Analysis of the MAD-AS

A principal component, varimax rotated factor analysis was conducted utilizing eigenvalues of greater than 1 to extract nine factors accounting for 57.96% of the variance. Of the nine factors, only five were retained for purposes of further analysis. These five factors presented as the

most reliable and stable after examining a scree plot of the eigenvalues plotted against the factor numbers (see Figure 1), and also, after observing that the other four factors contained three or fewer items each. A factor loading criterion equal to, or greater, than r=.45 was used to determine the items that were retained on a given factor (see Table 1).







Component Number

The factor structures of four previous studies involved in norming the MAD-AS (Beardmore, 2003; D'Andrea, 2004; Mahan, 2001; Martin, 2002) were utilized to compare and confirm the equivalence of this study's factor structure. The purpose of this confirmatory analysis was to remain consistent in the naming of the factors, to remain consistent in comparing MAD-AS factors to STAXI-2 factors, and last, to identify any observable differences across factor structures for the various populations utilized in the previous studies.

Factor 1, Verbal Expression, comprised ten items. These items measured an individual's propensity for becoming angry or annoyed, for exuding anger by being argumentative, critical, or blaming, and for incurring problems socially. Those who score high on this factor are prone to be expressive in their anger, and blame others or external factors for the frequency of angry episodes and resulting problems. This factor showed similarities to the STAXI-2 scale, Trait Anger, which measures how often angry feelings are experienced over time.

Factor 2, Anger Resolution, consisted of eight items. This factor appeared to measure problems with letting go of anger, and holding grudges. High scorers have difficulty

returning to baseline levels of anger, and may have an obsessional quality to their experiences of anger. This type of inward experience of anger appeared to resemble closely the STAXI-2 scale of Anger-In, which measures how often angry feelings are experienced but not expressed.

Factor 3, Behavioral Dyscontrol, was composed of five items. Those who score high on this factor are representative of those individuals who lose control or lose their tempers when angry. This includes overt displays, such as throwing things. High scorers would be more prone to act out aggressively, either verbally or physically, and cause themselves difficulties with others. This factor appears similar to the Anger-Out scale of the STAXI-2, and this scale measures how often angry feelings are expressed in verbally or physically aggressive behavior.

Factor 4, Physiological Arousal, contained four items. The most consistent, individual factor across all studies was this one, which measured the physiological dimension of anger. These items are composed of self-reported symptoms involving increased heart rate, muscle tension, breathing, and restlessness. Those scoring high on this factor are likely to experience physical arousals in relation to their

anger. This factor would appear to be related to the Anger-In scale of the STAXI-2.

Factor 5, Physical Aggression, consists of four items. Items of this factor reflect potential for provocation on a physical level, hitting others, thoughts of hurting others, and threatening others. A high score on this factor indicates a potential for physical violence and otherdirected hostility. This easily parallels the Anger-Out scale of the STAXI-2.

Table 1

MAD-AS Factor Loadings of the Principal Components Varimax Rotated Analysis

Factor 1: Verbal Expression

Eigenvalue=12.31 Variance=28.63% Cumulative Variance=28.63%

Number	Item*	Factor Loading
23. The behavior o	f others causes me to get angry.	.653
32. When people d	isagree with me, I <u> </u>	.627
27. When angry, I	let it show.	.625
16. I blame othe	rs for my anger.	.593
22. My anger has _	_ caused me problems on the job.	.559
20. People inten	d to anger me.	.536
30. I am argume	entative.	.489

31. I tell people when they annoy me.	.485
21. My anger caused me problems in my relationships.	.480
14. I am critical of others when angry.	.472

*Each item includes four Guttman style statements utilizing never, sometimes, often, and always.

Factor 2: Anger Resolution

Eigenvalue=2.54	Variance=5.92%	<i>Cumulative Variance=34.55%</i>

Number	Item* Factor Loa	
7. I_	have trouble letting go of things that have angered me in	the past814
8. I	hold grudges against those who have angered me.	.720
3. I	have trouble letting go of my anger.	.710
1. I	feel a need to get even with those who anger me.	.633
36. Onc	e angered, I get over it quickly.	.522
2. My	anger keeps me up at night.	.486
17. I	think about things that anger me.	.485
4. I	anger more frequently than most people.	.466

*Each item includes four Guttman style statements utilizing never, sometimes, often, and always.

Factor 3: Behavioral Dyscontrol

<u>Eigenvalue=2.17</u>	Variance=5.05%	Cumulative Variance=39.61%
Number	Item*	Factor Loading
9. I lose contro	l when angry.	.738
28. I lose contro	l when angry.	.711
11. I can control	my temper.	.652
10. I throw thing	s when I am angry.	.588
21. My anger ca	used me problems in m	y relationships486

*Each item includes four Guttman style statements utilizing never, sometimes, often, and always.

Factor 4: Physiological Arousal

<u>Eigenvalue=1.76</u>	Variance=4.09%	Cumulative Variance=43.70%
Number	Item*	Factor Loading
38. When angry, I _	feel my heart beatin	g faster804
39. When angry, m	y muscles feel tense	
40. When angry, m	y breathing is rapid.	.769
41. When angry, I	feel restless or agitat	.600

*Each item includes four Guttman style statements utilizing never, sometimes, often, and always.

Factor 5: Physical Aggression

Eigenvalue=1.46 Variance=3.41% Cumulative Variance=47.12%				
Number	Item*	Factor Loading		
34. When provoke	d, I hit people.	.788		
12. I hit those w	.709			
19. When I am ang	ry I have thoughts of h	urting others572		
29. I threaten pe	cople when angry.	.491		

*Each item includes four Guttman style statements utilizing never, sometimes, often, and always.

Correlation of the MAD-AS Factors

The MAD-AS factors were correlated with one another to create an inter-correlation matrix. Pearson Product Moment Coefficients of Correlation was utilized, and all of the correlations were observed to be positive and significant as is shown in Table 2. The correlations ranged from a low of r=+.257, n=450, p<.01, two-tailed, to a high of r=.631, n=450, p<.01, two-tailed.

Table 2

Pearson Inter-correlations Between MAD-AS Factors

	17 . J . J . J	77	Deeter 2		
	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5
	Verbal	Anger	Behavioral	Physiological	Physical
	Expression	Resolution	Dyscontrol	Arousal	Aggression
Factor 1		.551**	.631**	.341**	.457**
Verbal					• • • •
Expression					
Factor 2			.601**	.398**	.495**
Anger					
Resolution					
Factor 3				.305**	.513**
Behavioral					
Dyscontrol					
Factor 4					.257**
Physiological					
Arousal					
Factor 5					
Physical					
Aggression					

Internal Consistency of the MAD-AS

Assessment of the internal consistency of the MAD-AS involved an analysis of the MAD-AS total scale and each of the five factors. By use of Cronbach's coefficient alpha reliability calculations, the coefficient alpha for the entire scale was found to be .93. For each factor, the coefficient alpha was as follows: Factor 1 (Verbal Expression), .75, Factor 2 (Anger Resolution), .77, Factor 3 (Behavioral Dyscontrol), .79, Factor 4 (Physiological Arousal), .81, and Factor 5 (Physical Aggression), .79.

Corrected item-subscale total score correlations were calculated for the five MAD-AS factors as an additional assessment of internal consistency. All correlations were found to be significant at the p<.01 level and positive in their direction (Table 3). Separately, the correlations for each of the items on Factor 1 ranged from r=.45 to r=.59, on Factor 2 from r=.48 to r=.69, on Factor 3 from r=.51 to r=.73, on Factor 4 from r=.50 to r=.68, and on Factor 5 from r=.46 to r=.53. Corrected item-subscale score correlations were calculated by correlating the score on each item on a given factor with the corrected subscale total score. This corrected subscale total score was

obtained by summing all the items on a given factor except for the specific item being examined. For example, Factor 4 contains Items 38, 39, 40, and 41,; utilizing this method, Item 38 would be used to begin examination, and a correlation would then be calculated between the total score for Item 38 and the summed total scores for Items 39, 40, and 41 (corrected total score). This same procedure was then used for each successive item on each of the five factors.

Table 3

Fact Ver Expre	·bal	An	tor 2 ger lution	Beha	tor 3 vioral ontrol	Physio	tor 4 logical usal	Phy.	tor 5 sical ession
Item	<u>r</u>	Item	<u>r</u>	Item	<u>r</u>	Item	<u>r</u>	Item	<u>r</u>
14	.532	1	.605	9	.682	38	.681	12	.530
16	.509	2	.493	10	.512	39	.675	19	.478
20	.461	3	.694	11	.590	40	.660	29	.463
21	.571	4	.567	21	.539	41	.501	34	.528
22	.532	7	.688	28	.730				
23	.587	8	.595						
27	.552	17	.483						
30	.559	36	.529				- Consolition		
31	.459						····		<u> </u>
32	.591								

Corrected Item-Subscale Total Score Correlations

Note: All correlations are significant at the p < .01 level (one-tailed).

Correlations of the MAD-AS with the STAXI-2

A Pearson Product Moment Coefficient of Correlation analysis was performed in several ways to examine criterion validity of the MAD-AS, including correlations of the total scores on the MAD-AS with total scores on the STAXI-2 (STAXI Anger Expression Index) for both the entire sample and for each sample group. Other comparisons involved correlating pairs of subscales from each anger assessment. Each of the five factors of the MAD-AS was paired with one of the three major scales from the STAXI-2 (Trait Anger, Anger Expression In, and Anger Expression Out). The pairings were matched by determining similarities in the type of anger being measured.

The Pearson Product Moment Coefficient of Correlation between the MAD-AS total score and the STAXI-2 total score for the experimental group was r=+.709, n=225, p<.01, onetailed. Very similarly, the correlation for these same total score for the control group was r=+.699, n=225, p<.01, one-tailed (Table 4).

Table 4

Correlations of the Sample Groups

Group Identification			Staxi Anger Expression Index	Madas-total score
experimental group	Staxi Anger	Pearson Correlation	1	.709**
	Expression Index	Sig. (1-tailed)		.000
		Ν	225	225
	Madas-total score	Pearson Correlation	.709**	1
		Sig. (1-tailed)	.000	
		Ν	225	225
control group	Staxi Anger	Pearson Correlation	1	.699**
	Expression Index	Sig. (1-tailed)		.000
		Ν	225	225
	Madas-total score	Pearson Correlation	.699**	1
		Sig. (1-tailed)	.000	
		Ν	225	225

The correlation for the total scores for the whole group (sample groups combined) then resulted in a nearly identical coefficient, r=+.700, n=450, p<.01, one-tailed (Table 5).

Table 5

Correlations of the Whole Sample

		Staxi Anger Expression Index	Madas-total score
Staxi Anger	Pearson Correlation	1	.700**
Expression Inde	ex Sig. (1-tailed)		.000
	N	450	450
Madas-total sco	re Pearson Correlation	.700**	1
	Sig. (1-tailed)	.000	
	N	450	450

**. Correlation is significant at the 0.01 level (1-tailed).

Additionally, the criterion validity was examined by comparing each of the five MAD-AS factors to the chosen factor from the STAXI-2, the measure and description of which was most closely associated with the MAD-AS factor characteristics. As a result, Factor 1 (Verbal Expression) was compared to the STAXI-2 Trait Anger scale, Factor 2 (Anger Resolution) was compared to the STAXI-2 Anger Expression In scale, Factor 3 (Behavioral Dyscontrol) was compared to the Anger Expression Out scale, Factor 4 (Physiological Arousal) was compared to the Anger Expression In scale, and, last, Factor 5 (Physical Aggression) was compared to the Anger Expression Out scale, utilizing both sample groups for each comparison. The results of the Pearson Product Moment Coefficient of Correlation analysis revealed all correlations to be significant at the p<.01 level and positive with a coefficient of correlation ranging from a high of r=.685 to a low of r=.314. The data for each of the correlations are recorded in Tables 6-10. A correlation for Factor 1 (Verbal Expression) and STAXI-2 Trait Anger scale revealed a significant and a strong, positive relationship in the experimental group, r=+.685, n=223, p<.01, one-tailed, with a Coefficient of Determination equal to $r^2=.469$. The correlation for the data in the control group also revealed a significant and a strong, positive relationship, r=+.588, n=225, p<.01, onetailed, with a Coefficient of Determination equal to $r^2=.345$ (Table 6).

Table 6

Correlation of Factor 1

Group Identification			F1	Staxi Trait Anger
experimental group	F1	Pearson Correlation	1	.685*
		Sig. (1-tailed)		.000
		Ν	223	223
	Staxi Trait Anger	Pearson Correlation	.685**	1
		Sig. (1-tailed)	.000	•
		Ν	223	225
control group	F1	Pearson Correlation	1	.588*
		Sig. (1-tailed)		.000
		Ν	225	225
	Staxi Trait Anger	Pearson Correlation	.588**	1
		Sig. (1-tailed)	.000	
		Ν	225	225

A correlation for Factor 2 (Anger Resolution) and the STAXI-2 Anger Expression In scale for the experimental group revealed a significant and a strong, positive relationship, r=+.575, n=224, p<.01, one-tailed, with a Coefficient of Determination equal to r^2 =.330. The data for the control group revealed a significant, but moderate and positive correlation, r=+.456, n=225, p<.01, one-tailed, with a Coefficient of Determination equal to r^2 =.207 (Table 7).

Table 7

Correlation of Factor 2

			Staxi Anger	
Group Identification			Expression In	F2
experimental group	Staxi Anger Expression In	Pearson Correlation	1	.575*
		Sig. (1-tailed)		.000
		Ν	225	224
	F2	Pearson Correlation	.575**	1
		Sig. (1-tailed)	.000	
		Ν	224	224
control group	Staxi Anger Expression In	Pearson Correlation	. 1	.456*
		Sig. (1-tailed)		.000
		Ν	225	225
	F2	Pearson Correlation	.456**	1
		Sig. (1-tailed)	.000	
		Ν	225	225

A correlation for Factor 3 (Behavioral Dyscontrol) and the STAXI-2 Anger Expression Out scale in the experimental group showed a significant, positive, and strong relationship, r=.616, n=225, p<.01, one-tailed, with a Coefficient of Determination equal to r^2 =.379. Data for the control group also shows a significant, positive, and strong correlation, r=+.610, n=225, p<.01, one-tailed, with a Coefficient of Determination equal to r^2 =.372 (Table 8). Table 8

Correlation of Factor 3

	,		Staxi Anger Expression	
Group Identification			Out	F3
experimental group	Staxi Anger	Pearson Correlation	1	.616**
	Expression Out	Sig. (1-tailed)		.000
		Ν	225	225
	F3	Pearson Correlation	.616**	1
		Sig. (1-tailed)	.000	
		Ν	225	225
control group	Staxi Anger Expression Out	Pearson Correlation	1	.610*1
		Sig. (1-tailed)	.	.000
		N	225	225
	F3	Pearson Correlation	.610**	1
		Sig. (1-tailed)	.000	
	ສາກະການສາກັບພາສານປະທານາທາການສາມາດແຮ້ງ ແມ່ນເຊິ່ງ ແມ່ນນີ້ ແມ່ນນີ້ ແມ່ນນີ້ ແມ່ນນີ້ ແມ່ນນີ້ ແມ່ນນີ້ ແມ່ນນີ້ ແມ່ນນີ້	Ν	225	225

A correlation of Factor 4 (Physiological Arousal) and the STAXI-2 Anger Expression In scale for the experimental group revealed a significant and positive relationship, however, weak in its strength, r=+.317, n=225, p<.01, onetailed, with a Coefficient of Determination equal to r^2 =.100. The control group data revealed similar results, a significant, positive, and weak correlation, r=+.314, n=225, p<.01, one-tailed, with a Coefficient of Determination equal to r^2 =.098 (Table 9).

Table 9

Correlation of Factor 4

Group Identification			F4	Staxi Anger Expression In
experimental group	F4	Pearson Correlation	1	.317*
		Sig. (1-tailed)		.000
		Ν	225	225
	Staxi Anger Expression In	Pearson Correlation	.317**	1
		Sig. (1-tailed)	.000	
		Ν	225	225
control group	F4	Pearson Correlation	1	.314**
		Sig. (1-tailed)		.000
		N	225	225
	Staxi Anger Expression In	Pearson Correlation	.314**	1
		Sig. (1-tailed)	.000	
		N	225	225

A correlation for Factor 5 (Physical Aggression) and the STAXI-2 Anger Expression Out scale displayed a significant, positive, and strong relationship for the experimental group, r=+.565, n=225, p<.01, one-tailed, with a Coefficient of Determination equal to r^2 =.319. The data for the control group displayed a significant, positive, and moderate correlation, r=+.443, n=225, p<.01, onetailed, with a Coefficient of Determination equal to r^2 =.196 (Table 10).

Table 10

Correlation of Factor 5

Group Identification			Staxi Anger Expression Out	F5
experimental group	Staxi Anger Expression Out	Pearson Correlation	1	.565*
		Sig. (1-tailed)	. [.000
		Ν	225	225
	F5	Pearson Correlation	.565**	1
		Sig. (1-tailed)	.000	
		Ν	225	225
control group	Staxi Anger Expression Out	Pearson Correlation	1	.443*
		Sig. (1-tailed)		.000
		Ν	225	225
	F5	Pearson Correlation	.443**	1
		Sig. (1-tailed)	.000	
		Ν	225	225

Group Differences between the MAD-AS and the STAXI-2 Total Scores

A multivariate analysis of variance (MANOVA) was conducted on the total scores both for the MAD-AS and for the STAXI-2 to ascertain any significant differences between the hypertensive and the control group. The research hypothesis was that hypertensive subjects would be found to score significantly higher in comparison to the control subjects, based on their total scores on each of the anger assessments. The MANOVA revealed no significant findings with a Wilks' Lambda =.082, F(1, 449) =.001, p>.05. Further data for the STAXI-2 Anger Expression Index (total score) showed a mean score for the experimental group to be 31.69 and for the control group to be 31.73, F(1, 449) = .001, and p>.05. The mean total scores for the MAD-AS experimental group and the control group were, respectively, 32.63 and 34.48, F(1,449)=1.975, and p>.05.

Group Differences between the MAD-AS and the STAXI-2 Subscale Scores

A multivariate analysis of variance (MANOVA) was performed with group identification (Experimental and control) serving as the independent variable, and the subscale scores from the MAD-AS (Factors 1 - 5) and the STAXI-2 (Trait Anger, Anger Expression Out, and Anger Expression In) serving as the dependent variables. An overall Wilks' Lambda =.032, F(8,438)=1676.13, p<.05, revealed significant differences across several of the dependent variables. On the STAXI-2 Anger Expression In scale, control group subjects scored significantly higher than experimental subjects with their means equal to 15.99 and 14.81 respectively, F(1, 446) = 8.321, and p<.01. The MAD-AS Factor 2, Anger Resolution, also revealed significantly higher scores by the control group with a mean of .91 versus the experimental group with a mean of .82, F(1,446)=4.909, and p<.05. On MAD-AS Factor 3, Behavioral Dyscontrol, again, had similar results; the control group mean (.58) was significantly higher than the experimental group mean (.43), F(1,446) = 11.186, and p<.01. The last dependent variable to reveal significant

differences between each group was MAD-AS Factor 5, Physical Aggression, with a control group mean of .20 and an experimental group mean of .14, F(1,446)=4.829, and p<.05. The research hypothesis was an expectation of significant and positive differences on the subscales; the expectation was that the hypertensive group would score higher than the control group. The data reflect a reversed trend from the hypothesis when significant differences occurred. No significant differences were observed in the remaining dependent variables; STAXI-2 Trait Anger, STAXI-2 Anger Expression Out, MAD-AS Factor 1 (Verbal Expression), and MAD-AS Factor 4 (Physiological Arousal). A complete summary of the MANOVA results can be found in Table 11.

Table 11

MANOVA Results for MAD-AS and STAXI-2 Subscales

Dependent Variable	Independent Variable	Mean	F	Significance
Trait Anger	Experimental	16.24	. 436	.509
	Control	16.55	.430	.309
Anger Expression Out	Experimental	14.82	.019	.890
	Control	14.87	.019	.090
Anger Expression In	Experimental	14.81	8.321	0.04
	Control	15.99	0.321	.004
Factor 1 Verbal Expression	Experimental	.9198		.138
	Control	.8658	2.210	
Factor 2 Anger Resolution	Experimental	.8209	4.909	.027
	Control	.9172	4.909	
Factor 3 Behavioral Dyscontrol	Experimental	.4378	11.186	.001
	Control	.5831		
Factor 4 Physiological Arousal	Experimental	1.1892	1.500	221
	Control	1.1144	1.500	.221
Factor 5	Experimental	.1408	4.829	.029
Physical Aggression	Control	.2011	4.029	•029

Age as a Confounding Variable

As stated previously, the data from the previous results section on group differences in the subscales did show several significant differences, but in the opposite direction than expected upon initiation of this study. The literature review clearly indicates a trend in which individuals with cardiovascular heart disease (CHD), including hypertension, score higher than non-hypertensives and those absent of CHD on anger assessments. A further analysis was in order, starting with a t test. On face analysis of the age means for each sample group, the control group had a much lower age than did the experimental group, 32.53 and 44.68 years, respectively. The result of the group age differences t test confirmed that there was a significant difference, t=18.749, p<.01. As a result of this observation, a more detailed analysis was completed.

Recalling that Spielberger (1999) noted a younger normative sample of adults for the development of the STAXI-2 compared to the normative group used in the original STAXI, and that substantial differences were found when this group was assigned to three age groups, he explored the new normative sample in the same manner. However, results from the age groups 30 to 39 years and 40 years and older revealed no significant differences for eleven of the twelve STAXI-2 scales (Spielberger, 1999). Spielberger (1999) then decided to combine those two age groups into one group of 30 years and older. In addition to this, further analysis of the STAXI-2 normative group by

Spielberger (1999) clearly indicated that the frequency with which anger is experienced and expressed declines with age and that anger control increases with age.

Using Spielberger as a model, the chosen age groupings were 18-24 years, 25-34 years, and 35-55 years. A two by three Univariate Analysis of Variance (ANOVA) was performed with the sample groups (control and experimental) serving as one independent variable and age groupings serving as the other; the dependent variables chosen were the total scores of the MAD-AS and STAXI-2, and only those subscales from the MAD-AS and STAXI-2 that showed significant differences in the previous MANOVA.

The ANOVA performed on the STAXI-2 Anger Expression Index (total score) revealed only a significant finding in the main effect for age groups, F(2,449)=3.690, p<.05. There was no significant main effect for group identification and no significant interaction effect between the age and identification. A post-hoc Scheffe' test administered on this significant finding for age groups found that the age group of 35-55 years scored significantly lower than the 18-24 age group. Further analysis, however, reveals that the group sizes are unequal and when the Scheffe' adjusted the subsets to be

homogeneous using the harmonic mean, there was found to be no significant differences among age groups. Tables 12-15 that follow depict the results.

Table 12

Descriptive Statistics for STAXI-2 Total Score

	<u> </u>		Std.	
Group Identification	Age Groups	Mean	Deviation	N
experimental group	ages 18 - 24	41.20	26.790	5
	ages 25 - 34	34.94	14.822	17
	ages 35 - 55	31.19	14.277	203
	Total	31.69	14.676	225
control group	ages 18 - 24	35.19	15.122	64
	ages 25 - 34	32.25	15.568	72
	ages 35 - 55	28.83	13.188	89
	Total	31.73	14.707	225
Total	ages 18 - 24	35.62	16.017	69
	ages 25 - 34	32.76	15.382	89
	ages 35 - 55	30.47	13.974	292
	Total	31.71	14.675	450

Dependent Variable: Staxi Anger Expression Index

ANOVA Results for STAXI-2 Total Score

Dependent Variable: Staxi Anger Expression Index

	Type III Sum				
Source	of Squares	df	Mean Square	F	Sig.
Corrected Model	2215.670 ^a	5	443.134	2.083	.066
Intercept	136131.176	1	136131.176	639.747	.000
GROUP	401.677	1	401.677	1.888	.170
AGEGRPS	1570.511	2	785.255	3.690	.026
GROUP * AGEGRPS	57.764	2	28.882	.136	.873
Error	94478.350	444	212.789		
Total	549275.000	450			
Corrected Total	96694.020	449			

a. R Squared = .023 (Adjusted R Squared = .012)

Table 14

Multiple Comparisons for STAXI-2 Total Score

Dependent Variable: Staxi Anger Expression Index Scheffe

		Mean Difference			95% Confi	dence Interval
(I) Age Groups	(J) Age Groups	(I-J)	Std. Error	Sig.	Lower Bound	Upper Bound
ages 18 - 24	ages 25 - 34	2.86	2.340	.475	-2.89	8.61
	ages 35 - 55	5.15*	1.953	.032	.36	9.95
ages 25 - 34	ages 18 - 24	-2.86	2.340	.475	-8.61	2.89
	ages 35 - 55	2.29	1.766	.431	-2.04	6.63
ages 35 - 55	ages 18 - 24	-5.15*	1.953	.032	-9.95	36
L	ages 25 - 34	-2.29	1.766	.431	-6.63	2.04

Based on observed means.

*. The mean difference is significant at the .05 level.

Homogeneous Subsets for STAXI-2 Total Score

Staxi Anger Expression Index

Scheffe^{a,b,c}

		Subset		
Age Groups	N	1	2	
ages 35 - 55	292	30.47		
ages 25 - 34	89	32.76	32.76	
ages 18 - 24	69		35.62	
Sig.		.530	.373	

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares

The error term is Mean Square(Error) = 212.789.

a. Uses Harmonic Mean Sample Size = 102.904.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

c. Alpha = .05.

The results of the ANOVA for the MAD-AS total score revealed findings similar to those above; there was a significant age group main effect, F(2,449)=8.261, p<.01, but no significant main effect for sample groups or for the interaction effect. The Scheffe' performed on the age group main effect displayed a significant difference between the oldest age group and the youngest, but as homogeneous subsets the differences are no longer significant (Tables 16-19).

Descriptive Statistics for MAD-AS Total Score

Group Identification	Age Groups	Mean	Std. Deviation	N
experimental group	ages 18 - 24	48.00	19.248	5
	ages 25 - 34	37.59	16.681	17
	ages 35 - 55	31.84	14.737	203
	Total	32.63	15.168	225
control group	ages 18 - 24	38.61	13.891	64
	ages 25 - 34	34.90	11.770	72
	ages 35 - 55	31.17	11.485	89
	Total	34.48	12.621	225
Total	ages 18 - 24	39.29	14.373	69
	ages 25 - 34	35.42	12.787	89
	ages 35 - 55	31.63	13.811	292
	Total	33.56	13.968	450

Dependent Variable: Madas-total score

Table 17

ANOVA Results for MAD-AS Total Score

Tests of Between-Subjects Effects

Dependent Variable: Madas-total score

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	4191.332 ^a	5	838.266	4.462	.001
Intercept	162007.625	1	162007.625	862.406	.000
GROUP	533.448	1	533.448	2.840	.093
AGEGRPS	3103.929	2	1551.965	8.261	.000
GROUP * AGEGRPS	350.789	2	175.394	.934	.394
Error	83407.779	444	187.855		
Total	594288.000	450			
Corrected Total	87599.111	449			

a. R Squared = .048 (Adjusted R Squared = .037)

Multiple Comparisons for MAD-AS Total Score

Scheffe						
		Mean Difference			95% Confide	ence Interval
(I) Age Groups	(J) Age Groups	(I-J)	Std. Error	Sig.	Lower Bound	Upper Bound
ages 18 - 24	ages 25 - 34	3.87	2.198	.213	-1.53	9.27
	ages 35 - 55	7.66*	1.835	.000	3.15	12.16
ages 25 - 34	ages 18 - 24	-3.87	2.198	.213	-9.27	1.53
	ages 35 - 55	3.78	1.660	.076	29	7.86
ages 35 - 55	ages 18 - 24	-7.66*	1.835	.000	-12.16	-3.15
<u>k</u>	ages 25 - 34	-3.78	1.660	.076	-7.86	.29

Dependent Variable: Madas-total score

Based on observed means.

*. The mean difference is significant at the .05 level.

Table 19

Homogeneous Subsets for MAD-AS Total Score

Madas-total score

Scheffe^{a,b,c}

		Subset		
Age Groups	N	1	2	
ages 35 - 55	292	31.63		
ages 25 - 34	89	35.42	35.42	
ages 18 - 24	69		39.29	
Sig.		.142	.129	

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares

The error term is Mean Square(Error) = 187.855.

- a. Uses Harmonic Mean Sample Size = 102.904.
- b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

c. Alpha = .05.

The results of the ANOVA for the MAD-AS Factor 2 (Anger Resolution) reflects similar outcome as the previous two outcomes. A significant main effect is found for age groups, F(2,449)=7.117, p<.01, no significant main effect for sample groups, and no significant interaction effect. The Scheffe' again shows significant differences in scores between the oldest and youngest age groups, but no significance is found when homogeneous subsets are created. Complete results for these are found in Tables 20-23. Table 20

Descriptive Statistics for Factor 2

Croup Identification	Age Croupe	Mean	Std. Deviation	Ν
Group Identification	Age Groups			
experimental group	ages 18 - 24	10.6000	6.22896	5
	ages 25 - 34	7.7647	4.84161	17
	ages 35 - 55	6.3571	3.94853	203
	Total	6.5578	4.11565	225
control group	ages 18 - 24	8.0000	3.34759	64
	ages 25 - 34	7.6667	2.87289	72
	ages 35 - 55	6.5955	3.13600	89
	Total	7.3378	3.16392	225
Total	ages 18 - 24	8.1884	3.62295	69
	ages 25 - 34	7.6854	3.30494	89
	ages 35 - 55	6.4298	3.71600	292
	Total	6.9478	3.68740	450

Dependent Variable: FLINT2

ANOVA Results for Factor 2

Tests of Between-Subjects Effects

Dependent Variable: FLINT2							
Source	Type III Sum of Squares	df	Mean Square	F	Sig.		
Corrected Model	267.969 ^a	5	53.594	4.077	.001		
Intercept	7249.596	1	7249.596	551.446	.000		
GROUP	19.869	1	19.869	1.511	.220		
AGEGRPS	187.134	2	93.567	7.117	.001		
GROUP * AGEGRPS	34.976	2	17.488	1.330	.265		
Error	5837.054	444	13.147	ļ			
Total	27827.250	450					
Corrected Total	6105.023	449					

a. R Squared = .044 (Adjusted R Squared = .033)

Table 22

Multiple Comparisons for Factor 2

Dependent Variable: FLINT2

Scheffe

		Mean Difference			95% Confide	ence Interval
(I) Age Groups	(J) Age Groups	(I-J)	Std. Error	Sig.	Lower Bound	Upper Bound
ages 18 - 24	ages 25 - 34	.5030	.58159	.688	9254	1.9314
	ages 35 - 55	1.7586*	.48534	.002	.5666	2.9506
ages 25 - 34	ages 18 - 24	5030	.58159	.688	-1.9314	.9254
	ages 35 - 55	1.2556*	.43902	.017	.1774	2.3338
ages 35 - 55	ages 18 - 24	-1.7586*	.48534	.002	-2.9506	5666
	ages 25 - 34	-1.2556*	.43902	.017	-2.3338	1774

Based on observed means.

*. The mean difference is significant at the .05 level.

Homogeneous Subsets for Factor 2

FLINT2

Scheffe^{a,b,c}

		Subset	
Age Groups	N	1	2
ages 35 - 55	292	6.4298	1
ages 25 - 34	89		7.6854
ages 18 - 24	69		8.1884
Sig.		1.000	.610

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares The error term is Mean Square(Error) = 13.147.

a. Uses Harmonic Mean Sample Size = 102.904.

b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

c. Alpha = .05.

The results of the ANOVA for the MAD-AS Factor 3 (Behavioral Dyscontrol) did not reveal any significant main effects for the sample groups or the age groups, and there were no significant interaction effects between the two As a result, no post-hoc test was necessary. groups. The trend that appeared to remain, in surveying the descriptive statistics, was a decrease in scores as age increased, and although not significantly so, the trend is easily observable (Table 24).

Descriptive Statistics for Factor 3

			Std.	
Group Identification	Age Groups	Mean	Deviation	N
experimental group	ages 18 - 24	3.8000	3.19374	5
	ages 25 - 34	2.8235	2.42990	17
	ages 35 - 55	2.0739	2.16165	203
	Total	2.1689	2.21761	225
control group	ages 18 - 24	3.2969	2.67665	64
	ages 25 - 34	2.7361	2.59465	72
	ages 35 - 55	2.7865	1.87975	89
6 	Total	2.9156	2.36548	225
Total	ages 18 - 24	3.3333	2.69349	69
	ages 25 - 34	2.7528	2.55076	89
	ages 35 - 55	2.2911	2.10241	292
	Total	2.5422	2.32048	450

Dependent Variable: FLINT3

The results of the ANOVA for the MAD-AS Factor 5 (Physical Aggression) revealed a significant age group main effect, F(2,449)=4.608, p<.05, no significant main effect for sample group, and no significant interaction effect between the sample and age groups. The post-hoc Scheffe' showed significant differences between the age group, 18-24, and the age group, 35-55. When these groups were compared as homogeneous subsets, no significant differences were found in the age groups. Results are summarized in Tables 25-28.

Descriptive Statistics for Factor 5

			Std.	
Group Identification	Age Groups	Mean	Deviation	N
experimental group	ages 18 - 24	1.2000	1.30384	5
	ages 25 - 34	1.0000	1.80278	17
	ages 35 - 55	.5123	.99185	203
	Total	.5644	1.08421	225
control group	ages 18 - 24	1.1406	1.57225	64
	ages 25 - 34	.8194	1.19065	72
	ages 35 - 55	.5506	.89203	89
	Total	.8044	1.23092	225
Total	ages 18 - 24	1.1449	1.54611	69
	ages 25 - 34	.8539	1.31901	89
	ages 35 - 55	.5240	.96116	292
	Total	.6844	1.16481	450

Dependent Variable: FLINT5

Table 26

ANOVA Results for Factor 5

Tests of Between-Subjects Effects

Dependent Variable: FLINT5

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	25.262 ^a	5	5.052	3.842	.002
Intercept	89.587	1	89.587	68.119	.000
GROUP	.134	1	.134	.102	.750
AGEGRPS	12.121	2	6.060	4.608	.010
GROUP * AGEGRPS	.553	2	.277	.210	.810
Error	583.929	444	1.315		
Total	820.000	450			
Corrected Total	609.191	449		an and the second s	

a. R Squared = .041 (Adjusted R Squared = .031)

新来したことで

Multiple Comparisons for Factor 5

Dependent Variable: FLINT5

Scheffe

			Mean Difference			95% Confide	ence Interval
	(I) Age Groups	(J) Age Groups	(I-J)	Std. Error	Sig.	Lower Bound	Upper Bound
Γ	ages 18 - 24	ages 25 - 34	.2910	.18395	.287	1608	.7428
		ages 35 - 55	.6210*	.15351	.000	.2439	.9980
	ages 25 - 34	ages 18 - 24	2910	.18395	.287	7428	.1608
		ages 35 - 55	.3300	.13886	.060	0111	.6710
Γ	ages 35 - 55	ages 18 - 24	6210*	.15351	.000	9980	2439
L		ages 25 - 34	3300	.13886	.060	6710	.0111

Based on observed means.

*. The mean difference is significant at the .05 level.

Table 28

Homogeneous Subsets for Factor 5

FLINT5

Scheffe^{a,b,c}

		Subset	
Age Groups	N	1	2
ages 35 - 55	292	.5240	
ages 25 - 34	89	.8539	.8539
ages 18 - 24	69		1.1449
Sig.		.120	.192

Means for groups in homogeneous subsets are displayed. Based on Type III Sum of Squares

The error term is Mean Square(Error) = 1.315.

- a. Uses Harmonic Mean Sample Size = 102.904.
- b. The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

c. Alpha = .05.

CHAPTER 4

Discussion

This is the fifth study that has examined the psychometric properties of the MAD-AS, and its usefulness as a measure of self-reported anger. The first study by Mahan (2001) found strong psychometric properties that appeared to measure successfully the cognitive, behavioral, and physical components of anger; uncovered the multidimensionality of anger, and showed the instrument to be an improvement over many other instruments available. The second study (Martin, 2002) found a similar factor structure using a non-clinical sample, and introduced a parallel version of the instrument using other ratings of anger by a companion called the SO MAD-AS (Significant Other MAD-AS). The next study by Beardmore (2003) utilized an outpatient, clinical population, and again observed a similar and significant factor structure to the original study. D'Andrea (2004), using an outpatient cardiac population that was non-psychiatric, also lent further evidence of the instrument's psychometric soundness. Of the first four studies, two have utilized non-psychiatric or non-clinical samples (Martin, 2002 & D'Andrea, 2004); however, the MAD-AS was developed on a clinical,

psychiatric population. Its usefulness as a brief assessment or screening device in broader populations depends on the results of studies using non-clinical samples, similar to established instruments like the STAXI-2. This study utilized a non-clinical population from which to draw its sample; however, this is the first study to do so in a hypertensive, medical population.

Research results in the present study suggest that the MAD-AS possesses sound psychometric properties, including strong reliability and validity, with favorable comparisons of current factor structure to past ones. The results highlight the instrument's capacity to be sensitive in distinguishing between several dimensions of anger, yet it is brief; an original goal of the Mahan (2001) study was to parallel the MAD-AS to the Beck inventories as a brief screening tool.

Research Hypotheses

The present study proposed seven hypotheses as a basis for exploring specifically the psychometric properties of the MAD-AS, exploring its comparison to the STAXI-2, and

exploring both the similarities and differences of the sample populations.

Hypothesis 1. This study proposed that there would be a significant and positive correlation between the total scores of each assessment instrument (MAD-AS and STAXI-2) both for the control group and for the hypertensive group. The results reflected a strong, positive correlation for each group that was also significant, lending evidence in favor of this hypothesis.

Hypothesis 2. The proposal of this hypothesis was that hypertensive subjects would score significantly higher both on the MAD-AS and on STAXI-2 total scores in comparison to the control subjects. Results did not provide any evidence favoring this hypothesis. No significant differences were found, and comparison of the means also revealed slightly higher total scores for the controls versus the hypertensives for both instruments.

Hypothesis 3. This stated that a significant and positive correlation would occur between the MAD-AS and STAXI-2 hypertensive groups, and also on the MAD-AS and STAXI-2 control groups, on the subscales chosen to be paired, one from each instrument. Results revealed all

correlations to be significant and positive, but with varying strengths of relationships.

Hypothesis 4. Significant and positive differences were expected on the all the subscales of the MAD-AS and three of the subscales of the STAXI-2 when comparing the hypertensive and control group. Of these subscales, only one of the STAXI-2 subscales and three of the five MAD-AS subscales displayed significant differences between groups in their results; however, the results also showed that the control group scored higher than the hypertensive group on all subscales, contrary to expected outcomes.

Hypothesis 5. The factor structure of the MAD-AS in this study was proposed to correspond to the factors extracted from the original Mahan (2001) study, and to include seven factors. Although the results here suggest that only five factors were reliable and stable, these five factors showed favorable equivalence to previous ones.

Hypothesis 6. This study proposed that the MAD-AS total scores and factor scores would demonstrate internal consistency, utilizing a summated coefficient alpha of greater than .70. The coefficient alpha for the entire scale was observed to be .93, and alpha's ranging from a low of .77 to a high of .81 for the five factors.

Hypothesis 7. The last proposal stated that the corrected item-subscale total score correlations would be positive and significant for each of the MAD-AS factors. All corrected item-subscale score correlations, as proposed, were shown to be significant and positive for each of the five factors with strengths varying from moderate to high in their relationships.

Demographics

A total of 507 subjects volunteered to participate in this study. Of these, 450 satisfied criteria for inclusion in this study, 225 in the experimental (hypertensive) group and 225 in the control (non-hypertensive) group. Excluded from the study were 11.24% of the initial total, due to insufficient completion of packet materials. Females represented 56.2% of the 450 subjects and married subjects composed 54.4% of the total group compared to single (34.9%), divorced (8.2%), and widowed (0.4%) individuals. The average number of years of education for the entire sample was 15.03 years. Mean age for this population was 38.60 years, with a significant difference in mean age

between the control group (32.53 years) and the experimental group (44.68 years).

The variety of demographics sought in this study was limited. This was due to concerns of intrusiveness to the volunteers, who were patients coming in for evaluation or for treatment to their family physician's office, likely due to health concerns or illness. Another limitation in conducting a vast demographic screening was essentially Each subject's involvement was predicted to be time. approximately 20-30 minutes in duration with the use of the two anger assessment instruments alone, so an effort was made to remain concise and not prolong the volunteers' time in the office or prolong their own efforts in completing these instruments if they had chosen to complete the packet at home. In surveying the four previous studies, the only potential impact on the dependent measures from the demographics was found to be age (D'Andrea, 2004). D'Andrea (2004) also found other demographics had an effect on the total scores of the MAD-AS, but these were specifically in regard to risk factors, including hypertension, that are associated with the cardiac population utilized.

MAD-AS Factor Structure

The construct validity of an instrument represents the extent to which it accurately measures a specific construct (Kazdin, 1998), and in this study that construct is anger. The factor analysis of the MAD-AS presently supports its construct validity, and follows a pattern similar to previous research that also examined this instrument. The factor analysis in this study extracted nine factors, but under further scrutinization, only five could be sufficiently retained: Verbal Expression, Anger Resolution, Behavioral Dyscontrol, Physiological Arousal, and Physical Aggression.

In the Mahan (2001) study, seven factors were extracted with related items: Anger Dyscontrol, Angry Cognitions, Verbal Expressions of Anger, Physiological Arousal, Anger Justification/Blame, Externalization of Anger, and Difficulty with anger resolution. The Martin (2002) study identified six factors: Difficulty with Anger Resolution, Emotional Dyscontrol, Physiological Arousal, Physical Anger/Aggression, Argumentativeness, and Display of Anger. Beardmore (2003) extracted six factors: Behavioral Dyscontrol, Anger Resolution, Aggression,

Physiological Arousal, Externalization of Anger, and Verbal Expression of Anger. Last, the D'Andrea (2004) study also found six factors: Anger Resolution, Verbal Expression, Behavioral Dyscontrol, Physical Aggression, Physiological Arousal, and Anger Justification. Comparisons of the items in the each of the factors for the above studies revealed frequent similarities. All five studies contain the same four items for the Physiological Arousal factor. There is also an Anger Resolution factor in each of the five studies, but item review reveals only similarities between the last four studies, not with Mahan's. The factor Verbal Expression was represented by items consisting of a vocal expression of anger in all studies, except Martin's, because these items were represented in two separate factors, Argumentativeness and Display Anger. All of the studies found a factor to describe the set of items related to being out of control with angry feelings, Behavioral Dyscontrol; however, this study appears to be most closely associated with the previous three studies, whereas, Mahan's own Behavioral Dyscontrol encompasses this study's Behavioral Dyscontrol and Physical Aggression. Each of the previous studies labeled items describing physical aggressiveness differently compared to this study, with the

exception of D'Andrea's. The factor analysis in this study also revealed six items that did not correlate with any factors. Of these six items, two (numbers 18 and 37) showed no correlations in each of the previous four studies, leading this author to recommend elimination of these two items from the MAD-AS.

In comparing the item groupings on each factor for each study, an obvious similarity is observed especially between this study's structure and D'Andrea's study. This may be notable because both studies utilized medical populations with cardiovascular disease. Although easily observable similarities in the factor structures of the other three studies with this one exist, D'Andrea's reflects the most frequent similarities, but Mahan's appears to reflect the least. This again may be notable, because Mahan utilized an inpatient psychiatric population, but Martin utilized a normal population consisting primarily of students and Beardmore utilized a clinical outpatient population, which may be expected to be less severe in psychopathology than Mahan's population.

Internal Consistency

The internal consistency of an instrument is the degree of consistency or homogeneity of the items within the instrument (Kazdin, 1998). The assessment of this represents the instrument's reliability; the reliability of the MAD-AS was analyzed by the use of Cronbach's coefficient alpha reliability and corrected item-subscale total score correlations.

The coefficient alpha was determined for the entire scale and for each of the five factors. It revealed coefficient alpha's above .70 for all the factors and .93 for the entire scale. These scores reveal a high level of correlation. The more homogeneous a test is found to be and the higher the inter-item consistency, the less likely it is to be influenced by error variance (Anastasi, 1988). Thus this study supports the idea that the MAD-AS does measure the anger constructs it was developed to measure.

Corrected item-subscale total score correlations provide an additional assessment of inter-item consistency. The correlations for the five MAD-AS factors were found to be significant and positive, displaying a range of correlations that were moderate to high in strength. This again supports the proposition that the MAD-AS is consistent in measuring the construct of anger across all items.

Last, to lend more evidence to the internal consistency of the MAD-AS, the five factors were correlated with one another utilizing the Pearson Product Moment Coefficient of Correlation. All of the correlations were significant and positive. The range in strength was broad, from weak to moderately strong. The inter-correlations show that the factors have a close relationship to each other, as they should from the above results, but the strength of the relationships reflect their independence from one another, which is important in lending evidence to the proposition that the MAD-AS is sensitive in assessing the multidimensionality of anger.

Criterion Validity

The criterion validity is a measure of test validity as a correlation with some other criterion with which it should be related, as a way of measuring concurrent or predictive validity. In this study, several of the STAXI-2 subscales were paired with factors of the MAD-AS for

examination along with comparison of the total scores for each instrument. These comparisons were carried out utilizing a Pearson Product Moment Coefficient of Correlation analysis. The results of all these correlations can be viewed in Tables 4-10. In summary, each of the factors and the total score of the MAD-AS correlated both in a positive direction and in a significant manner with the STAXI-2. There was a robust relationship between the total scores of the MAD-AS and STAXI-2 for the entire sample, which remained when the experimental group and the control group were compared. The correlations of the five factors to the predicted subscales of the STAXI-2 showed a broad range of strengths in their relationships; however, their significant correlations, along with the total score results, confirm that the MAD-AS is related to the STAXI-2 in its ability to assess the construct and multidimensionality of anger, including the experience, expression, and control of anger.

Group Differences for the MAD-AS and STAXI-2

Significant differences between the hypertensive group of subjects and the control group were predicted, with

88

higher scores being consistently obtained on the total scores and on all of the subscales both for the MAD-AS and for the STAXI-2 instruments. According to the MANOVA, the results did not reflect any significant differences between the sample groups relative to the total scores for each anger assessment instrument. The findings were not significant for differences between sample groups on their scores for several of the subscales, including the STAXI-2 Trait Anger scale, the STAXI-2 Anger Expression Out scale, the MAD-AS Factor 1 (Verbal Expression), and the MAD-AS Factor 4 (Physiological Arousal). The remaining subscales, the STAXI-2 Anger Expression In scale, the MAD-AS Factor 2 (Anger Resolution), the MAD-AS Factor 3 (Behavioral Dyscontrol), and the MAD-AS Factor 5 (Physical Aggression), did reveal significant differences between the sample group scores, however, not in the direction of the hypotheses. The differences showed that the control group subjects scored significantly higher than their counterparts, the hypertensive subjects.

Several ideas explaining this unexpected result can be posited. The most obvious one is that the control group's mean age was younger than the experimental group's mean age. Spielberger's findings support the idea that the

expression of anger decreases, and the ability to control anger increases with maturity. The possibility exists that significant age differences between the two groups may confound the results, because the younger subjects might endorse more active levels of anger expression or suppression with a poorer sense of control compared to the older subjects. Another idea is that other risk factors for hypertension, such as tobacco use, high cholesterol, physical inactivity, obesity, family history, and diabetes, may be impacting this sample of hypertensives more than the potential psychological risk factors. In this situation, a hypertensive subject may already have moderate levels of anger or an expression style that is not only less extreme overall, but also one that existed prior to the development of hypertension.

As a result, the research hypotheses were not supported, and no evidence could be added to the body of research outside of this study that has shown hypertensives are more expressive or suppressive of anger. Despite this inability, the results here suggest that the MAD-AS may be more sensitive to the multidimensionality of anger, because three of the MAD-AS factors versus only one STAXI-2 subscale displayed significant differences. The MAD-AS

factors that did detect significant differences involved two areas of anger expression, externalized anger and internalized anger. This suggestion has important implications because of the brevity of the MAD-AS. The goal in developing the MAD-AS was to present it as a valid and preferable alternative to the existing lengthy measures of anger, including the current STAXI-2, and, as such, be utilized in similar ways as the Beck inventories.

Age Group Differences

There appeared in the data an easily observable difference in one demographic, namely age. The mean ages for the control group and the experimental group were respectively, 32.53 years and 44.68 years. In one previous study (D'Andrea, 2004), significant differences were found for the total scores of the MAD-AS and STAXI-2, along with several subscales, when age was controlled for. Spielberger's (1999) own research on the adult normative sample for the STAXI-2 also revealed that increases in age resulted in decreased scores, and beyond age thirty there was no need to have separate age groupings, as had been

done prior to finding no significant differences in scores for age groups representing 30-39 and 40-49 years.

Initially a t test was performed on the above means with the sample groups. As suspected, a significant difference between the mean score for the control group and for the experimental group was revealed. Further analysis was completed after splitting the age demographic into groups, similarly to Spielberger (1999). With an age range limited to 18-55 years because of inclusion criteria founded on definitions of essential hypertension, the age groupings decided upon were slightly different from Spielberger's. Also, concerns over potential small cell sizes were considered. The age groupings were then split into 18-24 years, 25-34 years, and 35-55 years. In the experimental group, small cell sizes for the first two age groups were apparent, because only 5 subjects were assigned to the 18-24 age group and 17 subjects to the 25-34 age group. The control group, however, appeared to reflect a more balanced distribution overall, 64 subjects in the 18-24 age group, 72 in the 25-34 age group, and 89 in the 35-55 age group. Following this, a two by three ANOVA was completed, with post-hoc testing, to detect any main effects or interaction effects for the independent

variables of age group and sample group. The dependent variables scrutinized were the total scores for the MAD-AS and STAXI-2, and the MAD-AS Factors 2 (Anger Resolution), 3 (Behavioral Dyscontrol), and 5 (Physical Aggression). Only these three factors had found significant differences between the sample groups on the previous MANOVA, and any significant findings here would have necessitated analyzing the other factors.

The results of the ANOVA reflected no significant main effects for sample group and no significant interaction effects between the sample group and age groups throughout all of the dependent variables. There were significant main effects for age groups on all but Factor 3 (Behavioral Dyscontrol), of the dependent variables; however, the posthoc Scheffe' test revealed only significant differences on each of these main effects between the 18-24 age group and the 35-55 age group. Further analysis by the Scheffe' revealed no significant differences in age groups when a harmonic mean was utilized to create homogeneous subsets because of the unbalanced distributions in size for the age groups. These results display the limitation of comparing small cell sizes with much larger ones. Overall, the results indicate the MAD-AS is sensitive to the same

decreases in anger scores with advancing age; this is similar to the findings for the STAXI-2 normative sample, and that age may be a confounding variable in the study of anger with future research designed to assess its role more specifically.

Implications and Recommendations for Future Research

The usefulness and practicality of using a brief measure of emotion is obvious in terms of time and cost. The MAD-AS appears to be showing itself a sensitive and brief instrument for assessing the multidimensionality of anger. Its sensitivity could provide clinicians with the ability to pinpoint areas of treatment with specific strategies to target these areas for improvement. The use of the MAD-AS in medical settings should not be overlooked. Although age may have confounded some of the results, its utility in assessing anger as a potential psychological factor for treating hypertension may still exist, because it appears to assess the physiological aspects of anger, along with the cognitive and behavioral aspects. The brevity of the MAD-AS seems to be its most important asset. The attraction of having an encompassing and accurate

measurement device that is similarly brief when compared to other anger assessment instruments in a climate of increasing time management, cost effectiveness, and management of care is significant.

A necessary recommendation from this study is that special attention be paid to the demographic of age. One of the difficulties in controlling for this variable in a study utilizing hypertensives is that this population tends to be naturally older. Recalling the demographics from the U.S. Department of Health and Human Services (2004), these reflect dramatic rises in the prevalence of high blood pressure from one age group to the next, as age increases. It is similar for most of the cardiovascular heart diseases; the diagnosis of these problems tends to increase with increasing age. It would appear that another normative population outside of the medical realm be investigated and compared to the non-clinical sample groups utilized in the studies subsequent to the original Mahan study that developed the MAD-AS. However, if another study in the future utilizes a cardiovascular heart disease as a component, then attempts to control for age should be These attempts may include creating age groups undertaken. prior to data collection, balancing distribution between

the age groups, and matching age with other factors to detect any interactions between them. The demographics should also include information on risk factors associated with hypertension. More information may need to be uncovered in relation to how older subjects control their expressions of anger when compared to younger subjects, and whether or not there are any changes in the experience of anger as age increases for individuals. This may assist in creating more sensitive questions for future inclusion into the MAD-AS or in adjusting current questions to reflect advancing age differences.

Various cognitive-behavioral techniques can be valuable in assisting to control anger intensity. Anger can be associated with cognitive distortions, physiological changes, socially and interpersonally reinforced behaviors, and subjective labeling, which are similar to other affective states, such as depression and anxiety. Having a brief and sensitive instrument to detect some of these subtleties can assist in the choice and execution of cognitive behavioral techniques for the clinician.

Limitations of Study

The MAD-AS and the STAXI-2 are both self-report inventories, and although self-report measures are the most commonly used type of measure in the area of psychological research (Kazdin, 1998), this becomes the chief limitation of the study. Self-report measures are characterized by two types of problems, bias on the part of the participant and poor construct validity. In the cases of the MAD-AS and the STAXI-2, subject bias is the problem of greatest concern. The MAD-AS, in keeping with the goal of brevity, cannot utilize fake good or fake bad items; however, it could employ the use of more reverse loaded questions, because currently it only employs three. Future studies may want to consider utilizing an assessment of social desirability in conjunction with the MAD-AS to assist in evaluating participant biases. Also, the MAD-AS does not yet distinguish total scores in terms of clinically relevant ranges, such as low, moderate, or high. The Martin (2002) study is the only one of the five studies to employ conversion of raw scores to z-scores. All future studies should employ z-score conversions, not only for the purpose of plotting an exact location of a raw score within

the sample distribution, but also for initiating the formation of a standardized distribution for direct comparison to other distributions and creating relevant ranges for raw scores. This would assist a clinician in comparing the results of the MAD-AS to other assessments and relevant clinical data to detect biases.

The lack of sensitivity to changes in the experience and expression of anger in older populations by the MAD-AS may hinder the type of subject pool that can be chosen. The use of subjects who have been diagnosed with CHD may include the possibility that the average age of the participant will rise. It is unclear what measures may assist in evaluating possible changes to increase its sensitivity, or whether or not this would be necessary for the MAD-AS overall. The lack of encompassing demographic data is another obstacle not only to determining whether or not the current sample group is representative of the general non-clinical population, but also in not being able to analyze the potential impact these have on the results.

Summary

Finally, this study lends further evidence that the MAD-AS is a valid and structurally consistent instrument for measuring the multiple components of anger. The results reflected robust evidence of internal consistency, construct validity, criterion validity, and stability in its structure. This is now the fifth study to provide similar conclusions, and a broadening normative base. The MAD-AS can be considered a sound instrument that provides an alternative to the lengthy tests currently available, and can have utility both with clinical, and with nonclinical populations.

Alexander, F. (1939). Emotional factors in essential hypertension. Psychosomatic Medicine, 1, 175-179. American Heart Association (2006). Heart disease and

stroke statistics - 2006 Update. Dallas, Texas.

Anastasi, A. (1988). *Psychological testing*. New York: Macmillan.

- Beck, A.T. (1976). Cognitive therapy and the emotional disorders. New York: International Universities Press.
- Beck, A.T. (1999). Prisoners of hate: The cognitive basis of anger, hostility, and violence. New York: Harper Collins.
- Beardmore, R.O. (2003). A normative study of the Mahan and DiTomasso anger scale in an outpatient clinical sample. Unpublished doctoral dissertation, Philadelphia College of Osteopathic Medicine.
- Cannon, W.B. (1929). Bodily changes in pain, hunger, fear, and rage. New York: Branford.
- Chang, P.P., Ford, D.E., Meoni, L.A., Wang, N.Y., & Klag, M.J. (2002). Anger in young men and subsequent premature cardiovascular disease: The precursors study. Archives of Internal Medicine, 162, 8, 901-906.

D'Andrea, K.S. (2004). A normative study of the Mahan and DiTomasso anger scale in an outpatient cardiac population. Unpublished doctoral dissertation, Philadelphia College of Osteopathic Medicine.

Davidson, K., MacGregor, M., Stuhr, J., & Gidron, Y. (1999). Increasing constructive anger behavior decreases resting blood pressure: A secondary analysis of a randomized controlled hostility intervention. International Journal of Behavior Medicine, 6, 268-278.

- Deffenbacher, J.L. (1992). Trait anger: Theory, findings, and implications. In C.D. Spielberger & J.N. Butcher (Eds.), Advances in personality assessment (Vol.9, pp. 177-201). Hillsdale, NJ: Erlbaum.
- Deffenbacher, J.L. (1995). Ideal treatment package for adults with anger disorders. In H. Kassinove (Ed.), Anger disorders: Definitions, diagnosis, and treatment (pp. 151-172). Washington, DC: Taylor & Francis. Deffenbacher, J.L. (1999). Cognitive-behavioral

conceptualization and treatment of anger. Psychotherapy in Practice, 55, 295-309.

Eckhardt, C.I. & Deffenbacher, J.L. (1995). Diagnosis of anger disorders. In H. Kassinove (Ed.), Anger disorders: Definition, diagnosis and treatment (pp. 27-47). Washington DC: Taylor and Francis.

- Ellis, A.E. (1977). Anger: How to live with it and without it. New York: Citadel Press.
- Eng, P.M., Fitzmaurice, G., Kubzansky, L.D., Rimm, E.B., & Kawachi, I. (2003). Anger expression and risk of stroke and coronary heart disease among male health professionals. *Psychosomatic Medicine*, 65, 1, 100-110.
- Engebretson, T.O., Scrota, A.D., Nauru, R.S., Edward's, K., & Brown, W.A. (1999). A simple laboratory method for inducing anger: A preliminary investigation. Journal of Psychosomatic Research, 47, 1, 13-26.
- Everson, S.A., Goldberg, D.E., Kaplan, G.A., Julkunen, J., & Salonen, J.T. (1998). Anger expression and incident hypertension. *Psychosomatic Medicine*, 60,6, 730-735.
- Fontana, A. & McLaughlin, M. (1998). Coping and appraisal
 of daily stressors predict heart rate and blood
 pressure levels in young women. Behavioral Medicine,
 24,1, 5-16.
- Fredrikson, M., & Matthew's, K.A. (1990). Cardiovascular responses to behavioral stress and hypertension: A meta-analytic review. Annals of Behavioral Medicine, 12, 1, 17-39.
- Gallagher, J.E.J., Yarnell, J.W.G., Sweetnam, P.M., Elwood, P.C., & Stansfeld, S.A. (1999). Anger and incident heart disease in the Caerphilly study. Psychosomatic Medicine, 61, 446-453.

Gentry, W.D., Chesney, A.P., Gary, H.E., Hall, R.P., &

Harburg, E. (1982). Habitual anger-coping styles: I. effect on mean blood pressure and risk for essential hypertension. *Psychosomatic Medicine*, 44, 2, 195-202.

- Gravetter, F.J. & Wallnau, L.B. (2000). Statistics for the behavioral sciences. Belmont, CA: Wadsworth.
- Harburg, E., Blakelok, E.H., & Roper, P.J. (1979). Resentful and reflective coping with arbitrary authority and blood pressure: Detroit. Psychosomatic Medicine, 41, 189-202.
- Helmets, K.F., Kranzt, D.S., Merz, C.N.B., Klein, J., & Kop, W.J. (1995). Defensive hostility: Relationship to multiple markers of cardiac ischemia in patients with coronary disease. *Health Psychology*, 14,3, 202-209.
- Jamner L., Shapiro, D., Goldstein, I., & Hug, R. (1991). Ambulatory blood pressure and heart rate in paramedics: effects of cynical hostility and defensiveness. *Psychosomatic Medicine*, 53, 393-406.
- Kaplan, J.R., Botching, M.B., & Maniac, S.B. (1993). Animal models of aggression and cardiovascular disease. In
 A.W. Seigman & T. Smith, (Eds.), Anger, hostility, and the heart. Hillsdale, NJ: Erlbaum.

Kassinove, H. & Sukhodolsky, D.G. (1995). Anger disorders: Basic science and practice issues. In H. Kassinove (Ed.), Anger disorders: Definition, diagnosis, and treatment (1-26). Washington, DC: Taylor & Francis. Kazdin, A.E. (1998). Research design in clinical

and any mode (1990). Rebeaten abbigh in eliniear

psychology. Needham Heights, MA: Allyn & Bacon.

- Laird, J.D., Cuniff, M., Sheehan, K., Shulman, D., & Strum, G. (1989). Emotion specific effects of facial expressions on memory for life events. Journal of Personality and Social Psychology, 4, 87-98.
- Lange, C.G. & James, W. (1922). The emotions. Baltimore, MD: Williams and Wilkins.
- Lazarus, R. (1991). Emotion and adaptation. New York: Oxford.
- Linden, W. & Feurstein, M. (1981). Essential hypertension and social coping behavior. Journal of Human Stress, 7, 28-34.
- Linden, W., Lenz, J.W., & Con, A. (2001). Individualized stress management for essential hypertension: A randomized trial. Archives of Internal Medicine, 161, 1071-1080.

- Martin, D.M. (2002). A comparison of self- and significant other-rated expression of anger using the MAD-AS and the SO-MAD-AS. Unpublished doctoral dissertation, Philadelphia College of Osteopathic Medicine.
- Martin, R., Choi, K.W., David, P., & Wegner, E.L. (1999). Style of anger expression: Relation to expressivity, personality, and health. *Personality and Social Psychology Bulletin*, 25, 10, 1196-1207.
- Messerli, F.H. (2003). Hypertension. In Rakel & Bope
 (Eds.), Conn's Current Therapy 2003 (353-372). New
 York: Elsevier Science.
- Morey, L.C. (1997). Manual for the Personality Assessment Screener: Professional Manual. Odessa, FL: Psychological Assessment Resources.
- Rajita, S., Lovallo, W.R., & Parsons, D.A. (1992). Cardiovascular differentiation of emotions. *Psychosomatic Medicine*, 54, 422-435.

- Rutledge, T. & Hogan, B.E. (2002). A quantitative review of prospective evidence linking psychological factors with hypertension development. *Psychosomatic Medicine*, 64, 5, 758-766.
- Salzinger, K. (1995). A behavior-analytic view of anger and aggression. In H. Kassinove (Ed.), Anger disorders: Definition, diagnosis and treatment (pp. 69-79). Washington, DC: Taylor & Francis.
- Sharkin, B.S. (1996). Understanding anger: Comment on Deffenbacher, Oettling, et al. (1996), Deffenbacher, Lynch et al. (1996), and Kopper and Epperson (1996). Journal of Counseling Psychology, 43, 2, 166-169.
- Siegman, A.W. (1993). Cardiovascular consequences of expressing, experiencing, and repressing anger. *Journal of Behavioral Medicine*, 16, 539-569.
- Spielberger, C.D. (1999). Manual for the State-Trait Anger Expression Inventory-2: Professional Manual. Odessa, FL: Psychological Assessment Resources.
- Spielberger, C.D., Johnson, E.H., Russell, S.F., Crane, R.J., Jacobs, G.A., & Worden, T.J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M.A. Chesney & R.H. Rosenman (Eds.), Anger and hostility in

cardiovascular and behavioral disorders (pp. 5-30). New York: Hemisphere.

- Spielberger, C.D., Reheiser, E.C., & Sydeman, S.J. (1995). Measuring the experience, expression, and control of anger. In H. Kassinove (Ed.), Anger disorders: Definition, diagnosis and treatment (pp. 49-67). Washington, DC: Taylor & Francis.
- Tafrate, R.C. (1995). Evaluation strategies for adult anger disorders. In H. Kassinove (Ed.), Anger disorders: Definition, diagnosis and treatment (pp.109-128). Washington, DC: Taylor & Francis.
- U.S. Department of Health and Human Se rvices. (2004). Hypertension among adults: U.S. 1999-2002 (Source: NHANES). Hyattsville, MD: National Center for Health Statistics.
- Zajonc, R.B., Murphy, S.T., & Ingelhart, M. (1989). Feeling and facial efference: Implications of the vascular theory of emotions. *Psychological Review*, 96, 395-416.

INTRODUCTION

Dear Participant:

We are doing a study on the relationship between feelings and medical problems. If you are a male or female between the ages of 18 and 55, you may be able to take part in this study. Your decision to be in this study is completely voluntary. You may decide not to participate or to discontinue your participation at any time. In no way will your health care be affected whether or not you choose to be in the study. All information will be kept strictly confidential. You will not be asked to provide your name on any material; therefore, no one will be able to identify you. Your doctor and health care workers will not have access to this information.

If you choose to participate, you will be given a packet and asked to fill out three questionnaires that take about 25 minutes of your time. The first questionnaire asks about your age, sex, marital status, and years of education. The other two questionnaires ask questions about your feelings. If you are able to complete this packet while you are waiting to be examined or before you leave the office, a collection box is set up for your convenience. If you can not complete this packet before the end of your visit or before you need to leave, an addressed stamped envelope is provided within the packet for you to take in order to complete the packet at your home and mail back to this office. There will be a number and letter on each form in order to match forms should any pages become separated.

The questionnaires ask about your thoughts, feelings, and behaviors. It is possible that you may learn something about yourself of which you did not know before. In the unlikely event that you become uncomfortable or upset with your answers to any of these questions, please contact Scranton Counseling Center at (570) 348-6100. You may even choose to contact the principal investigator, Steven Godin, Ph.D., MPH, CHES at (570) 422-3562. If you would like a summary of the results of this study, you may contact the co-investigator, Robert Liskowicz, M.A. via email at RobertL.studpoc.Stud@pcom.edu.

Thank you very much for your participation in this investigation!

Robert Liskowicz, M.A., M.S.
Philadelphia College of Osteopathic Medicine (PCOM)
Department of Psychology
4190 City Avenue
Philadelphia, PA 19131 Steven Godin, Ph.D., MPH, CHES Clinical Professor, Dept. of Psychology Philadelphia College of Osteopathic Medicine (PCOM) 4190 City Avenue Philadelphia, PA 19131

DEMOGRAPHICS SHEET - SUBJECT

AGE (in years): _____ GENDER: Male ____ Female ____ (Please check one)

MARITAL STATUS: Married _____ Single _____ Separated ____ Divorced _____ (Please check one)

YEARS OF EDUCATION: _____ *if less than 12 years, GED: Yes ____ No ____ (12 equal to High School Graduate)

DEMOGRAPHICS SHEET – CLINICAL STAFF

BLOOD PRESSURE (in mm Hg): ____/____

HEIGHT (in inches):

WEIGHT (in pounds): _____

NOTICE TO PATIENTS

The Cognetti and Conaboy Family Practice is taking part in a study in conjunction with the Philadelphia College of Osteopathic Medicine (PCOM) in order to investigate the relationship between feelings and medical problems. Some patients may be asked to volunteer to be part of this study.

*Note: This notice will be printed on a 12x16 poster in larger font.

MAD-AS

Marital Status: Age: Sex:

This questionnaire consists of 43 statements or quartets. After reading each group of statements carefully circle the number (0, 1, 2 or 3) next to the **one** statement in each group which best describes the way you have been feeling the past week including today. There are no right or wrong answers. Carefully read each question before answering.

- 1. 0 I never feel a need to get even with those who anger me.
 - 1 I sometimes feel a need to get even with those who anger me.
 - 2 I often feel a need to get even with those who anger me.
 - 3 I always feel a need to get even with those who anger me.
- 2. 0 My anger never keeps me up at night.
 - 1 My anger sometimes keeps me up at night.
 - 2 My anger often keeps me up at night.
 - 3 My anger always keeps me at night.
- 3. 0 I never have trouble letting go of my anger.
 - 1 I sometimes have trouble letting go of my anger.
 - 2 I often have trouble letting go of my anger.
 - 3 I always have trouble letting go of my anger.
- 4. 0 I never anger more frequently than most people.
 - 1 I sometimes anger more frequently than most people.
 - 2 I often anger more frequently than most people.
 - 3 I always anger more frequently than most people.
- 5. 0 I never get angry without reason.
 - 1 I sometimes get angry without reason.
 - 2 I often get angry without reason.
 - 3 I always get angry without reason.
- 6. 0 I am never quick to anger.
 - 1 I am sometimes quick to anger.
 - 2 I am often quick to anger.
 - 3 I am always quick to anger.

- 7. 0 I never have trouble letting go of things that have angered me in the past.
 - 1 I sometimes have trouble letting go of things that have angered me in the past.
 - 2 I often have trouble letting go of things that have angered me in the past.
 - 3 I always have trouble letting go of things that have angered me in the past.
- 8. 0 I never hold grudges against those who have angered me.
 - 1 I sometimes hold grudges against those who have angered me.
 - 2 I often hold grudges against those who have angered me.
 - 3 I always hold grudges against those who have angered me.
- 9. 0 I never lose control when angry.
 - 1 I sometimes lose control when angry.
 - 2 I often lose control when angry.
 - 3 I always lose control when angry.
- 10.0 I never throw things when I am angry.
 - 1 I sometimes throw things when I am angry.
 - 2 I often throw things when I am angry.
 - 3 I always throw things when I am angry.
- 11.0 I can **never** control my temper.
 - 1 I can **sometimes** control my temper.
 - 2 I can often control my temper.
 - 3 I can always control my temper.
- 12. 0 I never hit those who anger me.
 - 1 I sometimes hit those who anger me.
 - 2 I often hit those who anger me.
 - 3 I always hit those who anger me.
- 13.0 I am never a hot head.
 - 1 I am sometimes a hot head.
 - 2 I am often a hot head.
 - 3 I am always a hot head.
- 14.0 I am never critical of others when angry.
 - 1 I am **sometimes** critical of others when angry.
 - 2 I am often critical of others when angry.
 - 3 I am always critical of others when angry.
- 15.0 I never argue with people without reason.
 - 1 I sometimes argue with people without reason.
 - 2 I often argue with people without reason.
 - 3 I always argue with people without reason.

- 16.0 I never blame others for my anger.
 - 1 I sometimes blame others for my anger.
 - 2 I often blame others for my anger.
 - 3 I always blame others for my anger.
- 17.0 I never think about things that anger me.
 - 1 I sometimes think about things that anger me.
 - 2 I often think about things that anger me.
 - 3 I always think about things that anger me.
- 18.0 When I am angry people never fear me.
 - 1 When I am angry people sometimes fear me.
 - 2 When I am angry people often fear me.
 - 3 When I am angry people always fear me.
- 19.0 When I am angry I never have thoughts of hurting others.
 - 1 When I am angry I sometimes have thoughts of hurting others.
 - 2 When I am angry I often have thoughts of hurting others.
 - 3 When I am angry I always have thoughts of hurting others.
- 20. 0 People never intend to anger me.
 - 1 People sometimes intend to anger me.
 - 2 People often intend to anger me.
 - 3 People always intend to anger me.
- 21. 0 My anger never caused me problems in my relationships.
 - 1 My anger sometimes caused me problems in my relationships.
 - 2 My anger often caused me problems in my relationships.
 - 3 My anger always caused me problems in my relationships.
- 22. 0 My anger has **never** caused me problems on the job.
 - 1 My anger has **sometimes** caused me problems on the job.
 - 2 My anger has often caused me problems on the job.
 - 3 My anger has always caused me problems on the job.
- 23. 0 The behavior of others **never** causes me to get angry.
 - 1 The behavior of others sometimes causes me to get angry.
 - 2 The behavior of others often causes me to get angry.
 - 3 The behavior of others always causes me to get angry.

- 24. 0 After expressing my anger I never feel guilty.
 - 1 After expressing my anger I sometimes feel guilty.
 - 2 After expressing my anger I often feel guilty.
 - 3 After expressing my anger I always feel guilty.
- 25. 0 I never tolerate others mistakes.
 - 1 I sometimes tolerate others mistakes.
 - 2 I often tolerate others mistakes.
 - 3 I always tolerate others mistakes.
- 26.0 I never insult people when I am angry.
 - 1 I sometimes insult people when I am angry.
 - 2 I often insult people when I am angry.
 - 3 I always insult people when I am angry.
- 27. 0 When angry, I never let it show.
 - 1 When angry, I sometimes let it show.
 - 2 When angry, I often let it show.
 - 3 When angry, I always let it show.
- 28. 0 I never lose control when angry.
 - 1 I sometimes lose control when angry.
 - 2 I often lose control when angry.
 - 3 I always lose control when angry.
- 29. 0 I never threaten people when angry.
 - 1 I sometimes threaten people when angry.
 - 2 I often threaten people when angry.
 - 3 I always threaten people when angry.
- 30. 0 I am **never** argumentative.
 - 1 I am **sometimes** argumentative.
 - 2 I am often argumentative.
 - 3 I am always argumentative.
- 31. 0 I never tell people when they annoy me.
 - 1 I sometimes tell people when they annoy me.
 - 2 I often tell people when they annoy me.
 - 3 I always tell people when they annoy me.
- 32. 0 When people disagree with me, I never argue.
 - 1 When people disagree with me, I sometimes argue.
 - 2 When people disagree with me, I often argue.
 - 3 When people disagree with me, I always argue.

- 33. 0 I never feel bitter about things.
 - 1 I sometimes feel bitter about things.
 - 2 I often feel bitter about things.
 - 3 I always feel bitter about things.
- 34. 0 When provoked, I never hit people.
 - 1 When provoked, I sometimes hit people.
 - 2 When provoked, I often hit people.
 - 3 When provoked, I always hit people.
- 35. 0 When under stress, I never get angry.
 - 1 When under stress, I sometimes get angry.
 - 2 When under stress, I often get angry.
 - 3 When under stress, I always get angry.
- 36. 0 Once angered, I never get over it quickly.
 - 1 Once angered, I sometimes get over it quickly.
 - 2 Once angered, I often get over it quickly.
 - 3 Once angered, I always get over it quickly.
- 37. 0 I never feel a sense of relief after an angry outburst.
 - 1 I sometimes feel a sense of relief after an angry outburst.
 - 2 I often feel a sense of relief after an angry outburst.
 - 3 I always feel a sense of relief after an angry outburst.
- 38. 0 When angry, I never feel my heart beating faster.
 - 1 When angry, I sometimes feel my heart beating faster.
 - 2 When angry, I often feel my heart beating faster.
 - 3 When angry, I always feel my heart beating faster.
- 39. 0 When angry, my muscles never feel tense.
 - 1 When angry, my muscles **sometimes** feel tense.
 - 2 When angry, my muscles often feel tense.
 - 3 When angry, my muscles always feel tense.
- 40. 0 When angry, my breathing is never rapid.
 - 1 When angry, my breathing is **sometimes** rapid.
 - 2 When angry, my breathing is often rapid.
 - 3 When angry, my breathing is always rapid.

- 41. 0 When angry, I never feel restless or agitated.
 - 1 When angry, I sometimes feel restless or agitated.
 - 2 When angry, I often feel restless or agitated.
 - 3 When angry, I always feel restless or agitated.
- 42. 0 When someone offends me I never retaliate.
 - 1 When someone offends me I sometimes retaliate.
 - 2 When someone offends me I often retaliate.
 - 3 When someone offends me I always retaliate.
- 43. 0 In difficult situations, I never get angry.
 - 1 In difficult situations, I sometimes get angry.
 - 2 In difficult situations, I often get angry.
 - 3 In difficult situations, I always get angry.

Mahan, J.P., & DiTomasso, R. A. (1998) ©, 1998



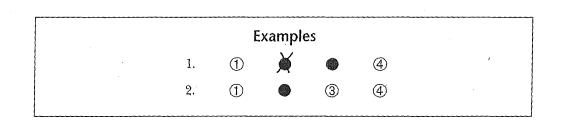
Item Booklet (Form HS)

Instructions

In addition to this Item Booklet you should have a STAXI-2 Rating Sheet. Before beginning, enter your name, gender, and age; today's date; years of education completed, your marital status, and your occupation in the spaces provided at the top of the STAXI-2 Rating Sheet.

This booklet is divided into three Parts. Each Part contains a number of statements that people use to describe their feelings and behavior. Please note that each Part has *different* directions. Carefully read the directions for each Part before recording your responses on the Rating Sheet.

There are no right or wrong answers. In responding to each statement, give the answer that describes you best. DO NOT ERASE! If you need to change your answer, mark an "X" through the incorrect response and then fill in the correct one.



Psychological Assessment Resources, Inc. • 16204 N. Florida Ave., Lutz, FL 33549 • Toll-Free 1.800.331.TEST • www.parinc.com

Reorder #RO-4352

Part 1 Directions

number of statements that people use to describe themselves are given below. Read each statement and then acken the appropriate circle on the Rating Sheet to indicate how you feel *right now*. There are no right or nong answers. Do not spend too much time on any one statement. Mark the answer that *best* describes your *resent feelings*.

Fill in ① for Not at all	Fill in ^② for Somewhat	Fill in 3 for Moderately so	Fill in ④ for Very much so	
How I Feel Right Now				
1.	I am furious	ਖ਼ਜ਼੶ਗ਼੶ਜ਼ਫ਼ਖ਼੶ਫ਼ਖ਼ਖ਼੶ਫ਼ਖ਼੶ਗ਼ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼੶ਫ਼ਖ਼ਖ਼ਖ਼ਫ਼ਖ਼੶ਖ਼ਫ਼ਖ਼ਖ਼ਫ਼ਖ਼ਖ਼ਖ਼੶ਫ਼ਖ਼ਖ਼੶ਫ਼ਖ਼ਖ਼੶ਖ਼ਫ਼ਖ਼੶ਖ਼ਫ਼ਖ਼ਖ਼੶ਖ਼ਖ਼ਖ਼੶ਖ਼ਖ਼ਖ਼੶ਖ਼ਖ਼ਖ਼੶ਖ਼		
2.	I feel irritated			
3.	I feel angry			
4.	I feel like yelling at someby	ody		
5.	I feel like breaking things			
6.	I am mad		د. ۱	
7.	I feel like banging on the t	able		
8.	I feel like hitting someone			
9.	I feel like swearing			
10.	I feel annoyed			
11.	I feel like kicking somebod	у		
12.	I feel like cursing out loud		· · ·	
13.	I feel like screaming			
14.	I feel like pounding somebo	ody		
15.	I feel like shouting out loud	1		

Part 2 Directions

lead each of the following statements that people have used to describe themselves, and then blacken the propriate circle to indicate how you *generally* feel or react. There are no right or wrong answers. Do not spend 10 much time on any one statement. Mark the answer that *best* describes how you *generally* feel or react.

י וויס		
Fill in ① for Almost never Fill in	n ② for Sometimes Fill in ③ for O	Often Fill in ④ for Almost always

How I Generally Feel

- 16. I am quick tempered
- 17. I have a fiery temper
- 18. I am a hotheaded person
- 19. I get angry when I'm slowed down by others' mistakes
- 20. I feel annoyed when I am not given recognition for doing good work
- 21. I fly off the handle
- 22. When I get mad, I say nasty things
- 23. It makes me furious when I am criticized in front of others
- 24. When I get frustrated, I feel like hitting someone
- 25. I feel infuriated when I do a good job and get a poor evaluation

Part 3 Directions

pervone feels angry or furious from time to time, but people differ in the ways that they react when they are pgry. A number of statements are listed below which people use to describe their reactions when they feel angry *furious*. Read each statement and then blacken the appropriate circle to indicate how often you generally react or when the manner described when you are feeling angry or furious. There are no right or wrong answers. to not spend too much time on any one statement.

Fill in ① for Almost never	Fill in ⁽²⁾ for Sometimes Fill in ⁽³⁾ for Often Fill in ⁽⁴⁾ for Almost always
	· · · · · · · · · · · · · · · · · · ·
How I C	Generally React or Behave When Angry or Furious
26	. I control my temper
27.	. I express my anger
28	I take a deep breath and relax
29.	I keep things in
. 30.	I am patient with others
31.	If someone annoys me, I'm apt to tell him or her how I feel
32.	I try to calm myself as soon as possible
33.	I pout or sulk
34.	I control my urge to express my angry feelings
35.	I lose my temper
36.	I try to simmer down
37.	I withdraw from people
38.	I keep my cool
39.	I make sarcastic remarks to others
40.	I try to soothe my angry feelings
41.	I boil inside, but I don't show it
42.	I control my behavior
43.	I do things like slam doors
. 44.	I endeavor to become calm again
45.	I tend to harbor grudges that I don't tell anyone about
46.	I can stop myself from losing my temper
47.	I argue with others
48.	I reduce my anger as soon as possible
49.	I am secretly quite critical of others
50.	I try to be tolerant and understanding
51.	I strike out at whatever infuriates me
52.	I do something relaxing to calm down
53.	I am angrier than I am willing to admit
54.	I control my angry feelings
55.	I say nasty things
56.	I try to relax
57.	I'm irritated a great deal more than people are aware of

onal copies available from: **Psychological Assessment Resources, Inc.** 16204 N. Florida Ave. • Lutz, FL 33549 • 1.800.331.TEST (8378) • www.parinc.com

,