

# Frontal Regulation of Blood Glucose Levels as a Function of Hostility

by

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## Abstract

From a neuropsychological perspective, hostile men have displayed dysregulation of right cerebral systems as evidenced through an exaggerated sympathetic stress response, with cardiovascular reactivity for blood pressure and heart rate. Altered right cerebral functioning, with hostility and anger, has been demonstrated within functional cerebral systems to include auditory (Demaree & Harrison, 1997), visual (Harrison & Gorelczenko, 1990; Herridge, Harrison, Mollet, & Shenal, 2003), somatosensory (Herridge, Harrison, & Demaree, 1997; Rhodes, Harrison, & Demaree, 2002), motor (Demaree, Higgins, Williamson, & Harrison, 2002) and premotor systems (Williamson & Harrison, 2003). Each of these studies has demonstrated cardiovascular reactivity (blood pressure and heart rates measures) concurrently with altered sensory or motor functional correlates of the right hemisphere. However, the neuropsychological mechanisms and functional regulation for the mobilization of glucose have not been examined.

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## Frontal Regulation of Blood Glucose Levels as a Function of Hostility

Cardiovascular disease is the number one killer of men and women in the United States. Averaging more than one death per minute or 2,600 deaths a day, cardiovascular disease was responsible for 1,415,000 deaths in the year 2000. Of those with cardiovascular disease, 54% have coronary artery disease (CAD), a narrowing of the arteries that results in both myocardial infarction (heart attack) and angina pectoris (chest pain). In 2000, coronary artery disease, or CAD, was responsible for the deaths of 515,204 people (American Heart Association, 2003). Currently 12,900,000 men and women are living with CAD (American Heart Association, 2003).

The American Heart Association (2003) reports specific risk factors that increase the likelihood of heart disease, including tobacco smoke, high blood cholesterol, lipids, physical inactivity, obesity, diabetes, as well as *the metabolic syndrome*. While the American Heart Association analysis (2003) is extensive and summarizes findings from a multitude of research projects, it remains incomplete with regard to a number of key factors. Perhaps foremost among the risk factors is hostility. Recent research has posited that hostility is a key predictor in the development of cardiovascular disease (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Guijarro, Hallet, Miller, Smith, & Turner, 1996; Helmer, Ragland, & Syme, 1991; Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996; Scherwitz et al., 1992). These recent findings on hostility allow for a greater understanding of cardiovascular disease. However, the construct of hostility has proven to be multifaceted and necessitates continued research (Rhodes & Harrison, 2002).

The existing literature on hostility is robust and remains at the forefront of the scientific literature in the field of Health Psychology and more recently, Neuropsychology.

Both areas of research have demonstrated associations between hostility and the stress response. Research within the area of Health Psychology has found hostile individuals to have increased lipids levels (Vogel, 1997), both increases and decreased cholesterol levels (Chen, Lu, Wu, & Chang, 2001; Finney, Stoney, & Engebretson, 2002), and decreased glucose levels (Donhoe & Benton, 1999; McCrimmon, Ewing, Frier, & Deary 1999; Surwitt et al., 2002). The mobilization of these stress related resources within the body is crucial for the body's ability to ready itself for action (Carlson, 2001; Selye, 1976). Lability, or over responding, within the stress response system has been shown to have implications for cardiovascular disease. Dysregulation in the mobilization of these resources is potentially disruptive as evidenced by glucose irregularities, to include episodes of hyperglycemia and hypoglycemia. Variant levels of mobilized glucose may yield deprivation states with the resultant consequence of increased hostility and aggression (ADA; Benton, Kumari, & Brain, 1992; McCrimmon et al., 1999; Surwit et al., 2002; Virkunen, 1992).

From a neuropsychological perspective, hostile men have displayed dysregulation of right cerebral systems as evidenced through an exaggerated sympathetic stress response, with cardiovascular reactivity for blood pressure and heart rate. Altered right cerebral functioning, with hostility and anger, has been demonstrated within functional cerebral systems to include auditory (Demaree & Harrison, 1997), visual (Harrison & Gorelczenko, 1990; Herridge, Harrison, Mollet, & Shenal, 2003), somatosensory (Herridge, Harrison, & Demaree, 1997; Rhodes, Harrison, & Demaree, 2002), motor (Demaree, Higgins, Williamson, & Harrison, 2002) and premotor systems (Williamson & Harrison, 2003). Each of these studies has demonstrated cardiovascular reactivity (blood pressure and heart rates measures) concurrently with altered sensory or motor functional correlates of the right hemisphere.

However, the neuropsychological mechanisms and functional regulation for the mobilization of glucose has not been examined.

There is an emerging need to more broadly investigate the physiological reactivity of hostile and angry men. Previous research has demonstrated that dysfunction in the stress response in the form of glucose mobilization, or glucose uptake, results in heightened hostility. A logical extension of this research is to investigate additional components of the stress response in hostile men from a neuropsychological or functional cerebral systems perspective, specifically the impact of stress on glucose mobilization.

#### *Type A, Hostility, and CAD*

Historically, hostility has been associated with Type A Behavior Pattern (TABP) and was originally thought to be merely a component of TABP (Friedman & Rosenman, 1974). Freeman (1986) defines Type A behavior as an individual who struggles to reach poorly defined goals, in the shortest time possible, in a hostile and aggressive fashion. In a review of the previous literature on TABP, Haynes, Levine, Feinlab, and Kannal (1980) demonstrated significant associations among TABP, hostility, and heart disease. This relationship between heart disease and Type A behavior has been longstanding (Dembroski & Costa, 1987; Dembroski, MacDougall, Lushene, 1979; Haynes et al., 1978; Koskenvuo et al., 1988; Lee & Cameron, 1986; Stevens, Turner, Rhodewalt, & Talbot, 1984; Tennant & Langeldudecke, 1985; Williams et al., 1980). The relationship between TABP and heart disease may be exacerbated by the demonstrated increases in cardiovascular responses in those with TABP (Dembroski et al., 1978).

Attempting to understand TABP further, Dembroski & Costa (1987) reviewed the potential risk factors of CAD, as well as their relationship to TABP. The review suggests that

TABP is a multidimensional construct, containing several risk factors to include hostility. Dembroski and Costa (1987) conclude that further examination and research of hostility is needed. Such research may ultimately provide further insights into CAD. Hostility, as depicted by these researchers, was demonstrated to be a multifaceted construct with significant associations with characteristics such as antagonism, uncooperativeness, rudeness, and disagreeableness among others.

In a more recent review of TABP, Williams, Nieto, Sanford, & Tyroler (2001) examined the components of TABP in the Atherosclerosis Risk in Communities Study (ARIC) which involved 12,900 middle-aged men and women to explore the relationship between Type A and CHD. Anger and hostility were identified as valid indicators of CHD. Results indicated that a strong, angry temperament, as opposed to anger in reaction to criticism or frustration, was a predictor of CHD. Here, the researchers define angry temperament as individuals who “experience anger longer, more frequently, more intensely, and in a broader range of situations and express it more quickly, needing little or no provocation” (Williams et al. 2001, p. 156), whereas anger in reaction is dependent on provocation. Williams et al. (2001) posit that an angry temperament may be mediated by sympathetic nervous system arousal, resulting in cardiovascular hyper-reactivity as well as increased levels of catecholamines, ultimately leading to atherosclerosis. Williams et al. (2001) found hostility and TABP were independently related to CAD with hostility more strongly correlated to atherosclerosis. Thus, these researchers suggest that hostility is accountable for atherosclerosis above and beyond TABP. This key finding provides further insight into hostility’s role in the development of atherosclerosis.



There is a long standing association between hostility (Dembroski & Costa 1987; Freeman, 1986), and heart disease. Eckhardt, Norlander, & Deffenbacher (2004) posit that hostility and anger have been interchanged in an inappropriate fashion, while in fact these are two distinct multidimensional constructs. Citing Buss (1961) as one of the original researchers of hostility, "...who regarded the construct as an attitude that involves the dislike and negative evaluation of others." The authors describe findings pertaining to the risk behaviors of hostility and their relationship to CHD. The researchers conclude that hostility is a multifaceted construct that involves cognitive variables of cynicism, mistrust, denigration, as well as negative affect and expression. These researchers depict anger initially in terms of a label placed on physiological arousal, as well as its involvement in hostility. This connection between anger and hostility yields mixed findings, requiring further research to unravel the two constructs.

With more recent research focusing exclusively on hostility, rather than TABP, there remain discrepancies concerning the definition of hostility. Christense, Wiebe, & Lawton (1997) interpret hostility to be an interpersonal attitude marked by suspiciousness, mistrust, disparaging views of others, and liability for anger. While a substantial portion of hostility research has focused on interpersonal styles, other research has examined cerebral arousal and autonomic nervous system activation. Smith (1994) describes hostility as "...a devaluation of the worth and motives of others, an expectation that others are likely sources of wrong doing, a relational view of being in opposition toward others, and a desire to inflict harm or see others harmed" (p.26). Within Smith's analysis of hostility, he incorporates a "psychophysiological reactivity" component in which hostility is linked with cardiovascular disease. Smith's rationale is that hostile individuals have increased and exaggerated

physiological responses to their environment, ultimately resulting in damage to their cardiovascular system. This physiological aspect of hostility adds another component to the definition, which seems to motivate a reexamination of the construct (Rhodes et al., 2002). For the purposes of this paper, hostility will be conceptualized in accord with Smith's definition.

It should be noted that the association between hostility and CAD has not been totally corroborated resulting in mixed findings. Helmer et al. (1991) administered the Cook Medley Hostility Scale as well as Behavior Pattern Hostility Index to 118 men and 40 women undergoing coronary angiography. No significant relationships were found between hostility and CAD. It was noted that poor health factors might mediate the relationship between hostility and CAD. Freeman (1986) found no link between TABP and CAD, although he did find an association between hostility and CAD. Freeman did note that other psychosocial causes, to include hostility and inwardly directed anger, might influence the development of CAD.

### *Hostility and Health Risks*

With a strong connection between hostility and heart disease, many researchers have attempted to identify potential mediating factors between these two constructs. Scherwitz et al. (1992) collected data on hostility, using the Cook Medley Hostility Scale in 5,115 younger (18 – 30 years) adults. Hostility scores were strongly associated with increased tobacco and marijuana smoking, alcohol consumption, and caloric intake. Scherwitz et al. (1992) concluded that hostility is indirectly linked to the development of CAD, as those with High Hostile scores are more likely to engage in behavior that is detrimental to health, and which increase the risk for CAD. Kawachi et al. (1996) support a

similar finding between hostility and poor health behaviors. Their research suggests that individuals with high levels of hostility also have increased tendencies for poor health behaviors to include smoking, drinking of alcohol, increased body weight, and cardiovascular disease.

Niaura et al. (2002) focused on potential mediating factors in the *metabolic syndrome*, which includes visceral obesity, insulin resistance, hyperglycemia, dislipidemia, and hypertension in 774 unmedicated, cardiovascular disease free men. Hostility levels were taken from the Cook-Medley Hostility Scale. Other measures included anthropometric data, serum lipids, fasting insulin concentrations, blood pressure, cigarette smoking, alcohol consumption, and total dietary calories all of which were used to predict CHD. Results indicate that hostility was negatively correlated with education and was positively related to caloric intake. Overall, Niaura et al. (2002) found hostility to predict CHD above the other measures including traditional CHD risk factors, blood lipid profiles, sociodemographic characteristics, alcohol consumption, and cigarette use.

From these findings, it can be concluded that hostility is a multifaceted construct containing several potential definitions each with slightly different interests. Hostility, originally thought to be a mere component of Type A personality, has been shown to be associated with the development of CAD and may be exacerbated by poor health behaviors to include cigarette smoking, consumption of alcohol, and increased caloric intake. However, researchers have not taken into account neuropsychological or physiological factors with regard to cerebral laterality effects, or components of arousal activation including cholesterol, lipid, and sugar mobilization.

### *The Stress Response*

Selye's stress model (Selye, 1976) asserts that when the human body prepares itself for stress, arousal levels increase and the flight or fight response is activated. One energy source for this arousal mechanism is the metabolism of glucose. The breakdown of sugar supplies the body with the extra energy it requires as the body readies itself for action. The sympathetic nervous system is responsible for activation of arousal systems to include resultant increases in heart rate, blood pressure, and the breakdown of cholesterol, lipids, and sugar subsequent to a stressful event. Dysfunction within the cerebral systems of processing or regulating negative emotional stimuli may impact the body's ability to ready itself for action, which may be peripherally manifested in any particular aspect of the arousal system to include sugar regulation.

Carlson (2001) has focused on behavioral, autonomic, and endocrine systems during stress responses. Active in the arousal process, the autonomic system, composed of both the sympathetic and parasympathetic branches, employs several neurotransmitters to increase heart rate and blood pressure to include epinephrine and norepinephrine. Carlson posits the result of the long-term release of these chemicals is heart disease. The second focus of Carlson's review of the stress response is the endocrine system, which produces cortisol, a glucocorticoid. Cortisol breaks down carbohydrates and protein into sugar in times of stress. Here, increased arousal and stress, requires additional energy supplied in the form of glucose.

Other researchers have examined other arousal mechanisms to include lipids and cholesterol. Vogele (1997) found High Hostile men to have increased lipid levels, specifically increased triglyceride levels when compared to Low Hostile men. After a

stressful condition, in this case task performance (mental arithmetic and mirror star tracing), High Hostile men had increased levels of anger, frustration, anxiety, blood pressure, and heart rate. Voegle concludes that High Hostiles have increased lipid levels for two reasons, the first being an increase in sympathetic tone which leads to higher circulating catecholamines that mobilize fatty tissue. The second reason is that High Hostiles are more likely to drink alcohol and to smoke cigarettes. It is from this standpoint that Voegle attempts to explain the link between hostility and cardiovascular disease. Subsequent research has supported the concept that hostile men have increased lipid levels when compared to Low Hostile men (Chen et al., 2001; Finney, et al. 2002).

Concerning arousal, cholesterol has proven to be more controversial with numerous contradictory results. Richards, Hof, & Alvarenga (2000) found those with elevated scores on hostility and aggression measures also had increased cholesterol levels. Others have documented increased hostility only when cholesterol levels are low in a population of men who previously abused cocaine (Buydens-Branchey, Branchey, Hudson, & Ferguson, 2000). In a sample of hospitalized men with a history of violent behavior, cholesterol levels were found to be lower than the general population (Hillbrand, Spitz, & Foster, 1995). These researchers suggest a curvilinear relationship between aggression and cholesterol with the most frequent acts of aggression associated with low levels of cholesterol. Despite previous associations between cholesterol and hostility, Fowkes et al. (1992) demonstrated no relationship between the two.

#### *Glucose Regulation and Hostility*

The current experiment focused on the role of glucose in the arousal system, primarily in men who have a documented emotional dysfunction, specifically hostility. As a basic

requirement for normal human functioning, glucose is essential to the arousal process. Those experiencing glucose abnormalities (hyperglycemia or hypoglycemia) have demonstrated tendencies for increased aggression.

There are two primary types of sugar dysfunction to include diabetes and hypoglycemia. There are three forms of diabetes consisting of Type 1 (immune-mediated or insulin dependent), Type 2 (insulin-resistant), and gestational diabetes (occurs during pregnancy) (American Diabetes Association Complete Guide to Diabetes, 1999). The ADA defines diabetes as a condition that occurs when the body cannot produce or use insulin properly and it is characterized by high blood sugar, around 120 milligrams of glucose/ deciliter of blood (mg/dl).

In contrast to hyperglycemia (diabetes), the ADA defines hypoglycemia as low blood sugar which results from too much insulin, too little carbohydrate, too little food, too much exercise, a delayed meal, and/ or alcohol on an empty stomach. Although the ADA, and the Centers for Disease Control (CDC) do not give specific cut-offs for a hypoglycemic episode, low blood sugar is thought to be in the range of 50-70 mg/dl. The ADA asserts that a key group of indicators in an individual having a hypoglycemic episode are the “autonomic symptoms” (American Diabetes Association Complete Guide to Diabetes, 1999). These include the opening of blood vessels, increased blood pressure, increased heart rate, as well as fluctuations in emotional states to include increased anger. The ADA also affirms that prolonged attacks of hypoglycemia have been associated with heart disease, because individuals with hypoglycemia have increased heart rates for extended periods. Aside from these autonomic symptoms, the ADA describes the effect of low blood sugar on the brain to include anger, lack of coordination, confusion, personality change, and unconsciousness,

among others (p.161). It is interesting to note that many individuals experiencing low blood sugar are completely unaware of their condition, a similar characteristic to those endorsing high levels of hostility (Demaree & Harrison, 1997).

Previous researchers have documented the association between hostile behavior and poor glucose regulation. Virkkunen (1982) examined the role of hypoglycemia in violent offenders using a glucose tolerance test, finding habitually violent offenders to have hypoglycemic tendencies when compared to non-violent offenders. In a related study of men that did not have a history of aggression or abnormal glucose metabolism, Benton et al. (1982) documented increased aggression after an induced hypoglycemic episode. After fasting for nearly 12 hours, the men had increased levels of aggression as measured by multiple aggression measures including the CMHS.

Hostility and blood glucose were found to have a positive relationship after participants received the hyperinsulinaemic glucose clamp technique used by McCrimmon et al. (1999). This glucose clamp technique systematically injects insulin intravenously over an extended period of time. Once a specified level is reached, the researchers are able to “clamp” and maintain the level of glucose in the blood. This procedure was employed because it provides the ability to examine glucose levels for an extended period of time. Using both nondiabetic men and women, McCrimmon et al. (1999) had multiple groups of participants, one that fasted over night and another that underwent the hyperinsulinaemic clamp technique to induce the hypoglycemic episode. As indicated by the State-Trait Anger Expression Inventory (STAXI), participants had increased levels of anger and frustration after the hypoglycemic episode.

Donhoe & Benton (1999) found similar results with nondiabetic women. After having participants fast overnight, the researchers administered an oral glucose tolerance test. Subjects consumed a drink containing 50g of sugar and were then observed for several hours. Blood glucose levels were measured with a finger prick glucometer (ExacTech, Medisense Britian). Participants were then given the Rosenzweig Picture-Frustration Study, a semi-projective aggression measure. Results indicated that lower blood glucose levels were associated with increased scores of aggression and frustration.

Employing nondiabetic African-American men, Surwitt et al. (2002) demonstrated a relationship among elevated hostility scores on the Cook Medley Hostility scale, fasting insulin, and insulin sensitivity. The researchers posit that race also seems to play a role in glucose metabolism and hostility, as African-American men had a significantly stronger correlation with glucose dysfunction and hostility than Caucasian men. Surwitt et al. (2002) suggest that hostility is an independent risk factor for diabetes in African-American men as the current findings match previous research indicating men with high average blood glucose (measured by HbA<sub>1c</sub>) are 2.5 times more likely to suffer from CAD.

The mobilization of sugar is a key element in the arousal process. Irregularities in sugar activation, primarily in the form of diabetes (blood glucose over 120 mg/dl) and hypoglycemia (blood glucose under 70 mg/dl), have a strong potential to result in increased hostile and aggressive behaviors. While this association between low glucose levels and hostility has been found in diabetes research, there has been no known research examining the role of glucose and hostility from a neuropsychological perspective.



*Neuropsychology and Emotion*

Providing foundational research to the field of neuropsychology, Alexandr Luria (*The Working Brain*, 1973) theorized that the brain was organized into specific zones working together in a concerted fashion. Luria's model incorporates the notions of localization and equipotentialism into a functional cerebral systems approach that claims each section of the brain works together to produce behavior. In his functional systems approach, Luria indicates three basic functional units to include the unit for regulating tone or arousal (the brainstem's reticular activating system), the unit for receiving, analyzing, and storing sensory information (posterior cerebral regions), and the unit for programming, regulation, and verification of activity (frontal lobes). Luria's model posits that arousal initially originates at the brainstem level with recruitment affecting the intensity of sensory systems projecting to the posterior cortical areas. The frontal lobes connect with many posterior cerebral systems and with the mesencephalon for the regulation and inhibition of these regions.

Subsequent to Luria's research, several prominent findings concerning arousal and the autonomic nervous system were revealed. Zimmerman et al. (1990) documented an increase in heart rate and blood pressure when a barbiturate was injected into the left carotid artery, thereby, putting the left hemisphere and purportedly the parasympathetic nervous system to sleep. Opposite results with sympathetic activation were found when a barbiturate was injected into the right carotid artery. Oppenheimer, Gleb, Girvin, & Hachinski (1992) found similar results when stimulation of the right posterior region resulted in increased heart rate and blood pressure, whereas stimulation of the left posterior region was associated with decreased heart rate and blood pressure.

The landmark research of Luria, accompanied by that of Zimmerman and Oppenheimer, provide a framework for more recent developments in neuropsychology. Luria demonstrated specific zones within the brain that work together to form a functional cerebral system. Zimmerman and Oppenheimer were able to demonstrate, specifically, that both increments and decrements in heart rate are controlled by specific cerebral zones, which operate collectively as a functional cerebral system. These relative changes in heart rate and blood pressure lend themselves well to emotion research because positively and negatively valanced affective events yield corresponding changes in autonomic functioning. More specifically, negatively valanced emotional processing of anger or hostility has resulted in heightened blood pressure and heart rate (Harrison & Gorelczenko, 1990; Herridge & Harrison, 1996) whereas processing of positive emotions has frequently resulted in stable or lowered blood pressure and heart rate.

Previous emotion research implicated subcortical systems, as autonomic changes would accompany shifts in affect from positive to negative. Additionally, the limbic system was thought to mediate changes in emotion (Heilman, Bowers, & Valenstein, 1985). However, more recent research indicates that broad cerebral systems play a substantial role in emotion (Davidson 1993). Heilman et al. (1993) posit the right cerebral cortex and subcortical systems are differentially responsible for emotion and arousal mechanisms essential for negative affective processing. Using Galvanic Skin Response (GSR) and lesion research, these researchers have provided evidence that the right cerebrum not only is responsible for emotional behaviors, but that negative emotions are often associated with heightened levels of arousal and sympathetic tone. The right hemisphere was further differentiated among anterior and posterior systems as Heilman, Bowers, & Valenstein (1985) posit that right

posterior cerebral systems are involved in the reception and comprehension of emotion, whereas right anterior cerebral regions are involved in the expression and regulation of emotion.

While the right hemisphere has long been implicated in the processing of emotion (Bowers et al., 1987; Heilman et al., 1975; Heilman et al., 1984), Tucker (1981) postulated that interhemispheric mechanisms involving contralateral inhibition across interhemispheric commissures was responsible for emotion. Relative activation of the right hemisphere was predicated with negative emotion and relative activation of the left hemisphere with positive emotion (Tucker & Williamson, 1984). More recent research by Tucker has implicated both the role of cortical and subcortical systems in emotion processing. Tucker, Derryberry, & Luu (2000) address brainstem and other subcortical functions in emotion as they provide a vertical integration model emphasizing the interaction between cortical and subcortical structures. Tucker et al. (2000) describe emotion in an evolutionary fashion that is organized and then reorganized at each level of the nervous system. These researchers posit that brainstem systems are capable of processing emotion, as are cortical systems. The distinction between the two systems, as described by these authors involves planning and future events. While subcortical systems are capable of producing sympathetic and parasympathetic changes with aggression, as well as many other responses, the cortical systems allow for planning.

Similar to Tucker's earlier work, Davidson (Davidson, 1995; Davidson & Irwin, 1999) proposes a valence (pleasure vs. displeasure) model of emotion and posits that the right hemisphere is specialized for the processing of negative emotion and withdrawal behaviors, whereas the left hemisphere is specialized for the processing of positive emotions and

approach behaviors. While Heilman and Tucker provide the foundations for affective research, our laboratory has provided evidence of four primary quadrants (anterior-posterior and left-right cerebral hemispheres) contributing to emotional processing. Superficially, Lee, Meador, Loring, & Bradley (2002) refer to this type of model as a “valence model” of emotion.

Research in our laboratory has demonstrated that the right hemisphere is differentially responsible for the reception and expression of both positive and negative emotion, however it is predominately involved in negative emotion. In contrast to the right hemisphere, the left hemisphere is largely responsible for the expression of positive emotion.

#### *Neuropsychology and Hostility*

Recently, research on emotion has focused on the negative emotions including hostility from a functional cerebral systems perspective to determine neuropsychological correlates and laterality effects. Shapiro et al. (2000) used single-photon emission computed tomography (SPECT) to measure cerebral blood flow following administration of a stressor (mental arithmetic) to hostile men. These researchers demonstrated that the stressor decreased prefrontal blood flow in the left frontal-temporal regions in the High Hostile group. The High Hostile group also showed marginal increases in heart rate during the stressor. The authors did not appreciate the relative right cerebral activation associated with increased heart rate. Shapiro et al. (2000) conclude that the prefrontal regions may regulate cardiac changes perhaps leading to CHD. Moreover, hostility may exacerbate these conditions with characteristic features of dysregulation and reactivity to stress.

In efforts to establish the cerebral mechanisms responsible for hostility, Louis et al. (1992) administered PET scans to 10 normal adult men (mean age= 25). The PET scans were

purported to assess the ongoing metabolic processes and to provide a more direct means of localizing cerebral metabolic glucose rates. After the infusion of D-[F] deoxyglucose (FDG), participants reported their thoughts, feelings, and free associations, which were blindly scored using the Gottschalk-Gleser Anxiety and Hostility Scale with 90 words or more being the criterion for a reliable sample. Examination of the white matter revealed significant positive correlations between hostility and glucose metabolic rates at the right superior frontal, the right superior parietal, and the right occipital lobes. Heightened metabolic rates provided supportive evidence of right cerebral activation with hostility.

Using a systematic functional cerebral systems approach, we have investigated the sensory, motor, and premotor correlates of hostility by measuring the relative activation states among brain regions. For example, our laboratory experimentally compared the appraisal and the expression of emotion within the left and the right hemisphere for visual, auditory, somatosensory, motor, and premotor regions. Hostility, considered a negative emotion, is correlated with sympathetic arousal systems including increased heart rate and blood pressure.

Harrison & Gorelczenko (1990) assessed cerebral asymmetry in the visual perception of affect for hostile men and women. Employing a tachistoscope, participants were instructed to identify angry, happy, or neutral faces in either the left or right visual field. Hostile participants showed faster affect perception and a negative perceptual bias restricted to the left visual field. Herridge, Harrison, Mollet, and Shenal (2004) replicated and extended this research using perceptual accuracy measures within the visual modality while adding a stress component, specifically a cold pressor. Hostile men demonstrated decreased accuracy in the

recognition of emotional faces within the left visual field, whereas women demonstrated symmetry across both visual fields.

Continuing the systematic line of research, Demaree & Harrison (1997) examined the auditory systems in hostile men. Using high and Low Hostile men, as indicated by the Cook-Medley Hostility Scale (CMHS), arousal levels were tested with physiological, behavioral, and laterality measures. Participants were administered an auditory dichotic listening test and then underwent a provocative pain stressor, specifically, the application of a cold pressor stimulus. The results indicate that High Hostile men had increased blood pressure, heart rate, and correctly identified more word sounds (phonemes) at the left ear following the stressor. Relative increased levels of arousal, as well as the heightened left ear advantage, are indicative of increased right cerebral activation for High Hostile men when compared to Low Hostile men. This increase in right cerebral activation in High Hostile men occurred with a corresponding increase in sympathetic tone using cardiovascular measures. Diametrically opposite results were found in the Low Hostiles who showed heightened left cerebral activation as evidenced through a dynamic increase in right ear word sound identification and lowered heart rate and blood pressure to the stressor.

Hostile men have demonstrated asymmetry for skin conductance as a primary measure of sympathetic arousal (Herridge et al., 1997). High Hostiles have evidenced increased skin conductance at the left hemibody, as well as a reduced habituation rate at the left hemibody when compared to the right hemibody subsequent to posing facial configurations consistent with anger (corrugator muscle contraction). Low Hostiles evidenced prolonged habituation rates at the right hemibody suggestive of relative left cerebral activation in this group.

Extending this line of research for the motor systems, Demaree et al. (2002) examined grip strength in right handed High and Low Hostile men. Each group was equivalent on handedness scores with a right hemibody preference across sensory and motor domains. Hand dynamometer measurements were used to assess grip strength, which is a measure of hemispheric motor functioning. It was expected that High Hostile men would demonstrate increased “antigravity” strength as measured using handgrip strength due to right frontal lobe dysfunction. Specifically, the prediction that upper motor neuron deficits at the right frontal region would result in heightened flexor “grip” at the left hand was confirmed.

Recently, Williamson & Harrison (2003) investigated the left and right prefrontal regions in this group evaluating cardiovascular reactivity to lateralized prefrontal stressors. The Controlled Oral Word Association Test (COWAT) and Ruff Figural Fluency Test (RFFT) were used as verbal and nonverbal frontal lobe stressor respectively. Previous research has demonstrated the COWAT to be sensitive to left frontal functioning (Benton & de Hamsher, 1976), whereas the RFFT is sensitive to right frontal functioning (Demakis & Harrison, 1997; Foster, Williamson, & Harrison, 2004). High and Low Hostile men completed both frontal stressors. The results indicate that the verbal and nonverbal stressor tests produced diametrically opposite effects on systolic blood pressure in high-hostile males. Specifically, systolic blood pressure increased subsequent to the nonverbal stressor (RFFT), whereas systolic blood pressure decreased subsequent to the verbal stressor (COWAT). For the Low Hostile group, the verbal stressor (COWAT) stressor increased systolic blood pressure, whereas the nonverbal stressor (RFFT) yielded no significant changes for systolic blood pressure. This research has implications for the anterior and posterior control for cardiovascular regulation in hostile men, as Williamson & Harrison (2003) conclude that the

frontal regions were unable to regulate blood pressure with the concurrent demand of the stressor task citing a capacity model. This research is in accord and extends previous research on the anterior-posterior model of hostility, specifically supporting relative right posterior activation and relative right frontal deactivation for High Hostile men.

Our laboratory has demonstrated asymmetry for a population with emotional dysregulation, primarily hostility. Research indicates hostility is associated with a relative increased activation at the right posterior cerebral regions as previously evidenced by skin conductance, behavioral laterality measures, heart rate, and blood pressure. More recent research has employed frontal lobe tasks with resultant changes in cardiovascular reactivity as a function of hostility.

#### *Rationale*

The purpose of the present experiment is to unite two existing literatures concerning hostility. Research within Health Psychology has demonstrated increased levels of hostility subsequent to a deprivation of a component in the arousal process, specifically sugar. Employing a systematic functional cerebral systems approach, hostile individuals have demonstrated a relative activation for right posterior brain regions, specifically for vision, audition, and somatosensory regions, whereas there has been a relative decreased activation for motor and premotor regions or right frontal lobe regulating capacity. Employing an anterior-posterior model of hostility, this systematic line of research has demonstrated increased right posterior activation and relatively decreased capacity for right frontal regions in hostile men.

Hostility, considered a negative emotion, is correlated with sympathetic arousal systems as manifested in increased heart rate and blood pressure. This body of research primarily



measures cardiovascular responses subsequent to a cold pressor (stressor); more recent research has employed frontal lobe tasks (fluency tasks) as stressors. These stress conditions have examined the relative increases in heart rate and blood pressure; however they have not investigated other arousal mechanisms including glucose mobilization. Glucose levels in hostile men have yet to be examined and are thought to play a key role in arousal as the body readies itself for an upcoming challenge. Unlike other arousal mechanisms, there is a finite amount of glucose in the body, and the brain requires specific levels to function at an optimal level (see diabetes and hypoglycemia ranges). Under stress, glucose levels should increase dramatically as arousal mechanisms are activated. Subsequent to the initial activation, glucose levels should drop as the finite sugar levels are metabolized. Moreover, hostile men are predicted to show altered regulatory control over glucose mobilization as a result of deficient right frontal capacity for regulatory control over the arousal response.

### *Phase I Hypothesis*

1. High Hostile men will have increased physiological arousal, as measured by glucose, SBP, and HR as a function of the right frontal stressor (nonverbal stressor) and will have decreased responses to the left frontal stressor (verbal stressor).
2. Group selection will impact blood sugar regulation, as High Hostiles will evidence greater increases in blood glucose levels to the right frontal stressor (nonverbal stressor) when compared to the Low Hostiles.
3. Group selection (High or Low Hostile) will affect performance on fluency measures, where High Hostiles will have lower scores on the right frontal stressor (nonverbal stressor), and higher scores on the left frontal stressor (verbal stressor)

whereas Low Hostiles will evidence higher scores on the right frontal stressor (nonverbal stressor).

### *Phase II Hypotheses*

1. The High Hostile group will experience increased reactivity to the cold pressor as evidenced by increased glucose levels, SBP, DBP, and HR when compared to the Low Hostile condition.
2. Recovery time will be a function of group selection, as the High Hostile group will require increased time to habituate when compared to the Low Hostile group.

## Method

### *Phase One*

#### *Participants*

A total of 151 participants completed the online screening in return for extra credit in their undergraduate psychology courses. From this initial screening, a total of 34 right handed men were selected, and agreed to participate in the experiment. Women participants were not be used in order to maintain a more homogenous and lateralized sample. Previous research on the processing of emotion has demonstrated heightened levels of symmetry for women (Harrison & Gorelczenko, 1990; Harrison, Gorelczenko, & Cook 1990; Higgins & Harrison 1999; Synder, Harrison & Gorman 1995). Participants reported no previous history of diagnosed developmental problems, hypoglycemia, hyperglycemia, hypertension, hypotension, or hyperthyroidism. Participants reported no history of head injury, loss of consciousness for more than five minutes, neurological, psychiatric, or psychiatric diagnosis, heart disease, or pancreatic disease. Participants were currently not taking prescribed,

unprescribed, allergy, or “illegal” medications. Participants were excluded if they were smokers of tobacco products or if they consumed three or more drinks of alcohol more than twice a week (see Medical History Appendix C). Participants were initially screened online using the Cook-Medley Hostility Scale (CHMO) (Appendix B). High Hostile participants were defined as those scoring 28 or above on the CHMO (maximum score = 50). Low Hostile participants were defined as those scoring 19 or below on the CHMO. These cut-off scores represent the upper and lower thirds of the CHMO distribution and are consistent with previous research on hostility (Demaree & Harrison, 1997; Demaree, et al., 2002; Harrison & Gorelczenko, 1990). Those individuals meeting these criteria were invited to participate in the experiment.

A total of 151 participants completed the online screening in return for extra credit in their undergraduate psychology courses. From this initial screening, a total of 34 right handed men were selected, and agreed to participate in the experiment. This sample size was further reduced by a total of 10 participants. Two participants were excluded based on their responses on the Medical History Questionnaire. Four participants were excluded as a result of scores on their second completion of the CMHO, which occurred in the laboratory and was given to ensure stable hostility levels. One participant’s score on the CMHO changed by 10 points, resulting in a reversal of his group inclusion from Low Hostile to High Hostile. The 3 other participants scores regressed towards the mean and did not meet criteria for either the Low or the High Hostile group. Finally, 2 participants from each group (High and Low) were excluded due to extreme scores on the second measurement of the CMHO. Subsequent to the elimination of the appropriate participants, a total of 24 right handed, non smoking, men with no significant medical history participated in the experiment.

### *Self-Report Measures*

The 50-item Cook-Medley Hostility Scale (CMHO) has been frequently used as a valid predictor of hostility (Helmer, et al., 1991; Larkin, Martin, & McClain, 2002; Scherwitz et al., 1992). Originally based on portions of the Minnesota Multiphasic Personality Inventory (Surwit et al., 2002), the CMHS is the most common hostility measure and is a valid predictor of medical, psychological, and interpersonal outcomes of trait based hostility (Contrada, 1992). According to Christensen et al. (1997) the CMHO has proven to have reliable internal consistency (coefficient alpha  $r = .86$ ). Test-retest consistency confirmation is also reliable ( $r=.84$ ).

### *Physiological*

Heart rate (HR) in beats per minute (BPM) and blood pressure in millimeters of mercury (mmHg), to include both systolic blood pressure (SBP) and diastolic blood pressure (DBP), were recorded as measures of autonomic nervous system functioning. Measurements were taken using the Takeda Medical Digital Blood Pressure Meter (model UA-751).

### *Blood Glucose Measurement*

The current research on glucose measurement indicates marked benefits from obtaining glucose from the forearm (Lee, Weinart, Miller, 2002; Pfutzner et al., 2003; Tieszen & New, 2003). The Therasense Freestyle Glucometer is a leading device for forearm testing (Demers, Kane, Bakst, Busch, & Hamilton, 2003). In comparison to the One Touch, Ultra Blood Glucose Monitoring System, The Freestyle meter maintains greater accuracy, demonstrates more clinically acceptable readings when compared to intravenous blood samples, and requires fewer sticks (White, Braco, & Malone, 2002).

There is much controversy concerning the at-home, self-test measurement of blood glucose levels. The ADA (American Diabetes Association Complete Guide to Diabetes, 1999) recommends daily testing of glucose levels in those with sugar dysregulation. Yet of the glucometers assessed to date, all have failed to meet the 95% accuracy rating standard set by the ADA (Brunner et al., 1998; Nichols, et al., 1995; Rheney & Kirk, 2000). Glucometers are considered imprecise for the diagnosis of hypoglycemia or hyperglycemia (Sacks et al., 2002; Tranjanoski, Brunner, Gfrerer, Wach, & Wieber, 1996). Despite failing to meet the standard for diagnostic purposes, the ADA continues to recommend the use of glucometers as they allow for the monitoring of blood glucose levels (American Diabetes Association Complete Guide to Diabetes, 1999). In the present experiment, the glucometer is useful for measurement rather than for diagnostic purposes. It was used in a fashion resembling the procedures for the daily monitoring of glucose levels established by the ADA.

Historically, manufacturers of home glucose monitoring devices have recommended obtaining blood samples from the fingertips to assess blood sugar levels. With the advancement of glucometers, blood samples can now be drawn from alternative sites to include the forearm region to assess blood glucose levels. However, there is some controversy over the accuracy of forearm testing particularly concerning the difference between forearm and finger sites when glucose levels are rapidly ascending or descending. Peled, Wong, & Gwalani (2002) had participants sugar load and found the forearm testing to be less accurate at detecting the swift change in glucose levels when compared to the finger tip sites, yet found the forearm to be reliable, otherwise. Lee et al. (2002) demonstrated a few significant differences in the level of accuracy after employing the two methods for 190 diabetics over the course of the day. Other researchers have found no difference between

finger-prick testing and forearm measurements (Pfutzner et al., 2002), even with rapid changes in participants' glucose levels (Jungheim & Koschinsky, 2002). Regardless of the controversies, a marked benefit in forearm testing has been the ease of obtaining a blood sample and the noteworthy decrease in pain (Lee et al., 2002; Pfutzner et al., 2003; Tieszen & New, 2003). Forearm testing further increases the readiness for testing, particularly when frequent blood samples are attained (Pfutzner et al., 2002).

### *Behavioral Measures*

#### *Verbal Stressor (Verbal Fluency)*

The Controlled Oral Word Association Test (COWAT) is a measure of verbal fluency (Benton & de Hamsher, 1976). Individuals with left frontal lobe deficits often have lower scores on this verbal fluency test when compared to a normal population (Johnstone, Holland, & Larimone, 2000; Ruff, Light, Parker, & Levin, 1997). The COWAT consists of three one-minute trials in which participants are instructed either to write or to say as many words that begin with a specific letter (F, S, or T) as possible. Proper names, numbers, and the same word with a different suffix do not qualify and are not scored. In accordance with Everhart (1997) and Williamson & Harrison (2003), the letters F, S, and T were used based on the tendency for the normal population to produce nearly equal words for each letter (10-12 per minute).

#### *Nonverbal Stressor (Nonverbal Fluency)*

The Ruff Figural Fluency Test (RFFT) is a paper and pencil test consisting of five sections and a measure of nonverbal fluency. Within each section there are 35 dot matrices arranged in a 5" x 7" pattern. The participants were given one minute to connect three or more dots, making as many unique patterns as possible in the time allotted. Scoring consists

of counting the number of patterns minus the number of perseverative errors (repetition of previously drawn pattern) for each trial. The total score is considered the total number of patterns minus the number of perseverative errors. A perseverative error is defined as any repetition in design by the participant. In accordance with Williamson & Harrison (2003), as well as Everhart (1997), three sheets containing the 35 dot matrices instead of five sheets were used to maintain uniformity with the COWAT.

### *Procedure*

After completion of the CHMO, participants were contacted by email to participate in the experiment if the scores on the hostility measure meet the criteria for either low (19 or below) or high (28 and above) hostility. Subsequent to review of the online completion of Medical History questionnaire, Laterality measure (see Appendix D), and the Alcohol measure (Appendix E), participants were given a brief outline of the experiment and informed that a forearm prick will be administered. Before entering the laboratory, participants were requested to abstain from caffeine, tobacco, and alcohol and to also eat a small meal or snack.

Upon arrival to the laboratory, the Informed Consent form was reviewed and signed by the participant. The CMHO was completed again by the participant to ensure stability of hostility scores. Once the participant completed the appropriate forms, he was seated in the chair and fitted for the blood pressure cuff and heart rate monitor. Once fitted for the blood pressure and heart rate monitor, the researcher left the room and repeated the following instructions: "Please take about one minute to become accustomed to your surroundings. Please sit still in the chair and face forward." After a 90-second adaptation period, the experimenter reentered the room and recorded baseline measures of glucose, SBP, DBP, and

HR (see Appendix A for blood glucose procedures). BP and HR readings were initially taken twice to ensure accuracy. A third BP and HR was taken if either SBP or DBP differed by 20 mm/Hg or if HR differed by 8 bpm. Glucose was taken after the left forearm was cleaned with an alcohol swab and then quickly lanced the participant's left forearm for the baseline blood glucose level (see Appendix A for glucose procedures). For each glucose measurement, the experimenter wore gloves. Used tests strips were placed in a certified biohazard box and disposed of appropriately. Participants with high (approaching or over 200 mg/dl) or low blood glucose (below 60-70 mg/dl) were informed of the atypical blood glucose reading and recommendations for further testing by a physician were made.

Immediately after baseline levels of glucose, SBP, DBP, and HR were recorded, participants were instructed that they would complete either the verbal or the nonverbal fluency measure, the order of which was counterbalanced. The instructions for the fluency measure were then read and the task was completed. After completion of the task, glucose levels, SBP, DBP, and HR were assessed. Following another 90-second adaptation period, glucose, SBP, DBP, and HR were measured and the second task was administered. Glucose, SBP, DBP, and HR were recorded immediately after the second task.

### *Phase II*

Phase II employed a cold pressor stressor and began 90 seconds after both counterbalanced fluency design tests were completed in Phase I. The participants in both phases were the same and the physiological measurements of heart rate, blood pressure, and glucose levels were taken with the same apparatus and procedures.



### *Cold Pressor*

In accord with Demaree & Harrison (1997) and Rhodes & Harrison (2002) a cold pressor was employed as a stressor. A cooler housed both ice and water, which was kept at the constant temperature of 0 degrees Celsius (range to + 3 degrees). The water temperature was measured using a standard mercury thermometer.

### *Procedures*

A baseline measure of glucose, SBP, DBP, and HR was taken before the participant placed his left hand in the ice water. After baseline measures were recorded, the participants were given the following instructions:

“When you are instructed, please place your left hand in the water to a point about one inch above your wrist. You will be asked to keep your hand in the water for 45 seconds. You have the option to withdraw your hand at any time, but we prefer that you do not. Although this may be difficult, please try your hardest to keep your hand in the water until instructed to take it out. Do you have any questions? Ready, begin.”

After 45 seconds, the participants were instructed to remove their hand from the water and SBP, DPB, and HR levels were taken followed by the measurement of glucose.

After 10 minutes (Rhodes & Harrison, 2002), glucose, SBP, DBP, and HR levels were recorded.

## Results

### *Phase 1 Results*

Separate 3 way mixed design Analyses of Variance (ANOVAs) were performed on the physiological variables including glucose levels (mg/dl), systolic blood pressure

(mm/Hg), diastolic blood pressure (mm/Hg), and heart rate (BPM) with the fixed factor of Group (High Hostile and Low Hostile ) and with repeated measures for Stress Condition (verbal and nonverbal stressor ) and Trial (pre and post stress). For each ANOVA, post hoc comparisons were made using Tukey's LSD (Winer, 1971). An a priori level of significance was set at  $p \leq .05$ .

It was hypothesized that High Hostile men would have increased physiological arousal, as measured by glucose, SBP, and HR as a function of the nonverbal stressor and decreased physiological arousal to the verbal stressor. Support for this predication was found only for the variable of glucose. Here, a significant interaction was found for Group x Condition,  $F(1,22) = 4.16, p \leq .05$ . For the High Hostile group, glucose levels (mg/dl) were significantly higher for the nonverbal stressor ( $M = 101.37, SD = 13.75$ ) when compared to the verbal stressor ( $M = 95.79, SD = 11.20$ ). For the Low Hostile group, glucose levels (mg/dl) remained stable, or unchanged, as a function of the nonverbal stressor ( $M = 95.63, SD = 22.04$ ) and as a function of the verbal stressor ( $M = 96, SD = 21.34$ ). Group differences in mean glucose levels as a function of Condition (verbal and nonverbal stressors) can be seen in Figure 1. The interaction effect of Group x Condition x Trial for glucose was not significant,  $F(1,22) = .53, p > .47$ . Means and standard deviations for this nonsignificant interaction are depicted in Table 1. The ANOVA summary table for the analysis of the glucose measures is shown in Table 2.

The remaining 3 way mixed design ANOVAs for SBP, DBP, and HR yielded no significant interactions among the factors of Group, Condition, or Trial. In Tables 3-5, the physiological variables and each of the potential interactions are listed with the  $F$  values, degrees of freedom ( $df$ ), and  $p$  values.

For the behavioral measures, total fluency scores and total error scores were analyzed using separate 2 way mixed design ANOVAs with the fixed effects of Group (High Hostile and Low Hostile) and with the repeated measures of Condition (verbal and nonverbal stressors). It was predicted that group selection (High or Low Hostile) would affect performance on the verbal and nonverbal stressors (fluency measures). Specifically, it was predicted that the High Hostiles would have lower scores on the nonverbal stressor (RFFT) than on the verbal stressor (COWAT) in both within group and between group comparisons. There was partial support for this prediction as a significant interaction effect was found for Group x Condition,  $F(1,22) = 4.90, p > .03$ . The High Hostile men made significantly more errors on the nonverbal stressor ( $M = 17.18, SD = 19.88$ ) when compared to the Low Hostile men ( $M = 5.81, SD = 4.33$ ). On the verbal stressor, the High Hostiles made significantly less errors ( $M = .04, SD = 0.66$ ) when compared to the Low Hostile group ( $M = 2.08, SD = 2.93$ ). These values are represented in Table 6, and graphically shown in Figure 2. It should be noted that due to the extreme variability in error scores ( $range = 53, SD = 14.05, Variance = 197.56, CV = 176.92$ ) the outliers (1) in each group were excluded.

However, no significant interactions were found for the additional analyses of the behavioral measures to include the variable of total fluency score. The interaction of Group x Condition,  $F(1,22) = .59, p \leq .59$  for total fluency score was not reliable and reflected no difference among groups for the number of correct items on the verbal and the nonverbal stressors.

### *Phase II Results*

Phase II employed 2 factor mixed design ANOVAs with the fixed effect of Group (High and Low Hostile) and with the repeated measure of Trial (pre, post1, post2). The

primary prediction that the High Hostile group would experience increased reactivity to the cold pressor as evidenced by increased glucose levels, SBP, DBP, and HR when compared to the Low Hostile group was not supported. There were no significant interaction effects for these variables.

Secondly, it was hypothesized that recovery time to the stressor would vary as a function of group with the High Hostile group requiring increased time to habituate when compared to the Low Hostile group. This was not supported by statistical analysis. Tables 7-10 display the ANOVA summary table for each dependent variable (glucose, SBP, DBP, and HR). Table 11 displays mean glucose levels and standard deviations for the Low Hostile and the High Hostile group across trials.

### Discussion

The literature on hostility is robust with findings from multiple areas within psychology, which reflect both the complexity and the evolution of the construct over time (Dembroski and Costa, 1987; Eckhardt, Norlander, & Deffenbacher, 2004). In accord with previous research on hostility, the present experiment employed lateralized stressors to a population with a diminished capacity for negative emotional regulation, specifically hostility, using the physiological measures of blood pressure and heart rate (Williamson & Harrison, 2003). However, the current experiment extended this research by measuring blood glucose mobilization as a function of concurrent left and right frontal lobe stressors in High and Low Hostile men.

There are two primary findings from the current experiment that add to the existing hostility literature. The first is that High and Low Hostiles metabolize glucose at different rates as a function of lateralized stressors. Specifically, High Hostiles mobilize heightened

levels of glucose to nonverbal stress when compared to both verbal stress and to Low Hostiles. Further, the glucose levels of the Low Hostiles remain stable despite the completion of the left and the right frontal stressors. The second major finding is that High Hostiles make more errors on a design fluency task, when compared to the verbal fluency task and to the errors made by the Low Hostile group. This finding indicates that High Hostiles have difficulty manipulating spatial arrangements under a time constraint. Moreover, the results support a limited capacity interpretation where High Hostiles dysregulate glucose when confronted with a dual task challenge for the right frontal regions. The results support increased frontal regulatory capacity among Low Hostiles where there is glucose stability with verbal or nonverbal stressors and performance on these measures is superior to the High Hostile group.

The findings from this experiment support a right hemispheric model of hostility. Here, hostility has been previously associated with increased activation for auditory (Demaree & Harrison, 1997), visual (Harrison & Gorelczenko, 1990; Herridge, Harrison, Mollet, & Shenal, 2003), and somatosensory modalities (Herridge, Harrison, & Demaree, 1997; Rhodes, Harrison, & Demaree, 2002). Diminished regulatory capacity of the right frontal regions has received support for motor (Demaree, Higgins, Williamson, & Harrison, 2002) and premotor systems (Williamson & Harrison, 2003). Within this model of hostility, it is postulated that High Hostiles have a diminished capacity for concurrently completing a frontal lobe stressor while inhibiting or regulating arousal systems, resulting in the increased activation and exaggerated responses for cardiovascular systems (Williamson & Harrison, 2003) and altered sensory and perceptual appraisal of emotional stimuli across modalities.

The data from the present experiment add to this right hemispheric model of hostility by demonstrating an exaggerated stress response in High Hostile men. However, beyond previous research on hostility, the present experiment demonstrated glucose dysregulation in High Hostiles and specifically to a stress challenge for right frontal systems. High Hostile men demonstrated significant increases in glucose when completing a nonverbal fluency stressor and had significant difficulty manipulating spatial arrangements under a time constraint when compared to the Low Hostile men. Previous research has demonstrated that individuals with right frontal lobe strokes or brain injuries have significantly lower scores, or increased error ratios on nonverbal fluency tasks, compared to those without right frontal lobe deficits (Ruff, Allen, Farrow, Niemann, & Wylie, 1994). More recently, Foster, Williamson, & Harrison (2005) demonstrated the significant relationship between performance on the RFFT and right frontal capacity. More specifically, the low design fluency group evidenced increased delta magnitude over the right frontal region using quantitative electroencephalography.

As noted by the significant difference in the total number of error scores on the nonverbal task, it is clear that the poor performance of High Hostiles on this measure reflects a potentially diminished capacity for the right frontal region. Taken together, High Hostile men make significantly more errors on the nonverbal fluency test and demonstrate significant increases in glucose while subjected to this type of stressor. Two potential explanations are offered below in the interpretation of these results.

Initially, the High Hostiles' increase in glucose levels to the nonverbal stressor provides evidence for a faulty system with a diminished capacity for regulation of the appropriate glucose levels. In accord with Kinsbourne's Functional Cerebral Space Model

(Kinsbourne, 1980; Reuter-Lorenz, Kinsbourne, & Moscovitch, 1990), the completion of the nonverbal stress by the High Hostiles produced an interference effect with the regulation of the sympathetic nervous system (Williamson & Harrison, 2005). This interference effect is also evident in the increased mobilization of glucose as the High Hostiles have a diminished capacity to concurrently regulate both systems. It follows that the over-appraisal of negative affect within the those that have emotional lability for anger would occur with physiological responses in preparing for “fight” or “flight” to include glucose mobilization, as demonstrated here, and potentially cholesterol and other substances which negatively impact long term health and cardiovascular disease.

A second interpretation of these findings may support Selye’s (1976) model of stress whereby the stress that is applied to a system causes a response to remove the stress and to return to ‘pre-stress’ levels. Applying this concept to the current experiment provides evidence for the diminished capacity of the High Hostiles in regulating their stress response albeit a stress response regulated by right frontal systems that play a role in anger modulation and mobilization for the sympathetic nervous system.

It may also be the case that the High Hostiles require this influx of fuel to cope with stress, or in the case of this experiment, to complete the right frontal stressor. The High Hostiles may potentially be metabolizing glucose at a greater rate to compensate for regions with diminished capacity, specifically the right anterior cingulate. Support for this finding is found by Dwyer (2002) who reports that glucose is the primary fuel for the brain and that this substance is involved in nearly all of the brain’s activities to include all cognitive abilities and nearly all cellular processes. Dwyer further notes that the regulation of glucose is not fully understood, especially at a global level, however, glucose dysregulation has been

associated with depression (Jacobson, Samson, Weinger & Ryan, 2002) and Schizophrenia (Henderson & Ettinger, 2002) as well as diabetic and hypoglycemic conditions that underlie cardiovascular disease.

It should be noted that more global changes are evident in those with diabetes. In a review of the literature on glucose, McCall (2002) finds that those with diabetes have between a two-and a six-fold risk of experiencing a stroke. Further, those surviving a stroke will have greater difficulty with recovery, as neurotransmitter metabolism is altered. Interestingly, McCall sites Woo et al. (1988) who reports that the increased risk of stroke in diabetics may be the result of the stress response. Specifically, when diabetics are experiencing a hyperglycemic episode, they continue to mobilize glucose in response to stress, thereby further increasing their glucose levels, and eventually resulting in a vascular accident. It appears as if glucose regulatory dysfunction often leads to heightened dysfunction subsequent to stress. Taken from a functional cerebral systems view, it may be the case that the right frontal region in particular is unable to inhibit a reflex glucose release, which results in a continued, and unrestricted, mobilization of glucose instability in the associated affective systems, sympathetic system, and for cognitive processing.

Although there may be additional mechanisms linking glucose dysfunction and hostility, it appears as if both variables are involved in the stress response. Unfortunately, the long-term consequences of an exaggerated stress response, as experienced by those with heightened levels of hostility, can be deadly. Previously, hostility has been linked to heart disease, cardiovascular disease, hardening of the arteries (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989; Guijarro, Hallet, Miller, Smith, & Turner, 1996; Helmer, Ragland, & Syme, 1991; Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996; Scherwitz et



al., 1992) altered cholesterol levels (Chen, Lu, Wu, & Chang, 2001; Finney, Stoney, & Engebretson, 2002) lipid dysregulation (Vogel, 1997), and most notably glucose dysregulation (Donhoe & Benton, 1999; McCrimmon, Ewing, Frier, & Deary 1999; Surwitt et al., 2002). Interestingly, as part of the recently published Atherosclerosis Risk in Communities (ARIC) study researchers examined over 6,000 participants and reported that glucose and heart function are strongly intertwined. The presence of one of these factors increases the likelihood of the other. The report ultimately states that the researchers are unsure how glucose and heart function are related, but that those individuals with cardiac problems have a much grimmer prognosis if glucose dysregulation is present (Schroeder, Chambles, Liao, Prineas, Evans, Rosamond, & Heiss, 2005). As noted by these researchers, these variables, along with cholesterol and lipid regulation, warrant further investigation.

It should be noted that there are several methodological limitations in the present experiment, which may explain the lack of significant results for the cardiovascular measures. After the experiment began, it is likely that the participants were reacting to glucose readings taken from their right forearms. Here, the glucose levels were taken after the participant's forearm was lanced and the glucose strip was placed directly on the drop of blood. To minimize the participant's reaction, they were asked to not watch and to focus on the wall directly in front of them. At no time during the experiment did a participant complain of pain or of having his blood drawn. After the completion of the experiment, the participant was asked if the glucose readings were painful. One hundred percent of participants responded with a "no" to this question.

It should be noted that SBP, DBP, and HR were taken at the left arm at the same time that the glucose levels were being assessed at the right forearm. This procedure was followed to

reduce potential time delays in reactivity to the stressors. As noted in the results section, there were no significant interactions for SBP, DBP, and HR. It is possible that the glucose reading may have been confounded by the evaluation of SBP, DBP, and HR as an intrusive measure. As noted earlier, recent research has linked these two processes which require further research (Schroeder et al., 2005).

The other major limitation in the present experiment was that the measurement of glucose provided only one reading in a system that is always changing. Clarke & Sokoloff (1999) report that the brain requires a constant supply of glucose to supply the neurons and glia. This supply is further complicated by the constant shift in glucose levels and glucose requirements of both the central and peripheral nervous systems. The results of the current experiment indicate that right frontal stressors increased glucose levels in the High Hostile group. Unfortunately, the experiment did not measure how quickly glucose was being metabolized, or for how long. In response to stress, it may be the case that glucose spikes rapidly, has a delayed metabolic rate, or has a prolonged breakdown. These factors were not taken into account in this experiment. An additional limitation in this domain is that the researchers did not know if glucose levels were rising or lowering.

To side step these difficulties, other researchers have developed protocols that require subjects undergoing glucose manipulations to fast the night before the experiment and to then measure glucose several times over the course of several hours (Ellison et al., 2002).

Additionally, researchers, under the guidance of a licensed physician, employ a hypoglycemic clamp in which insulin is infused intravenously until a specified glucose level is reached (Fruehwald-Schultes, Born, Kern, Peters, & Fehm, 2000). A continuation of the present research could potentially measure glucose multiple times before and after stress to

develop a better understanding of glucose's time course. Given that the current experiment did not employ these precautions to ensure glucose stability, the argument can be made that the significant relationships that were found for Phase I are quite robust. For Phase II, or the cold pressor paradigm, the large variation in scores for glucose may have been lessened by having the participants fast the night before the experiment and employing the hypoglycemic clamp.

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Table 1

*Mean glucose values ( mg/dl) as a function of the nonsignificant interactions of Group x Condition x Trial.*

Group	Condition	Trial	Mean	SD
HH	1	1	92.00	9.09
HH	1	2	99.58	12.18
HH	2	1	100.50	11.57
HH	2	2	102.25	15.79
LH	1	1	93.25	19.66
LH	1	2	98.75	23.43
LH	2	1	94.00	23.08
LH	2	2	97.25	21.85

*Note. HH=High Hostile , LH=Low Hostile , Condition 1=verbal stressor , Condition 2 = nonverbal stressor, Trial 1=pre stress, Trial 2=post stress.*

Table 2

*ANOVA summary table for the Glucose comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	0.16	1	0.69
Condition	3.18	1	0.08
Group x Condition	4.16	1	0.05*
Trial	15.98	1	.0006*
Group x Trial	0.02	1	0.89
Condition x Trial	2.70	1	0.11
Group x Condition x Trial	0.53	1	0.47

*Probability Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 3

*ANOVA summary table for the Systolic Blood Pressure (mmHg) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	0.45	1	0.51
Condition	0.00	1	0.95
Group x Condition	2.51	1	0.12
Trial	12.11	1	.0021*
Group x Trial	0.01	1	0.93
Condition x Trial	0.71	1	0.40
Group x Condition x Trial	1.88	1	.1846

*Probability Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 4

*ANOVA summary table for the Diastolic Blood Pressure (mmHg) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>P</u>
Group	2.47	1	0.13
Condition	1.08	1	0.30
Group x Condition	0.48	1	0.49
Trial	8.70	1	.0074*
Group x Trial	1.83	1	0.18
Condition x Trial	1.70	1	0.20
Group x Condition x Trial	0.63	1	0.43

*Probability Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 5

*ANOVA summary table for the Heart Rate (bpm) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	0.79	1	0.38
Condition	2.56	1	0.12
Group x Condition	0.01	1	0.91
Trial	2.49	1	0.12
Group x Trial	0.06	1	0.81
Condition x Trial	0.68	1	0.41
Group x Condition x Trial	0.61	1	0.44

*Probability Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 6

*High Hostile s Evidence Significantly More Error Scores for Nonverbal stressor.*

Group	Condition	N	Mean Score	SD
HH	1	12	0.41	0.66
HH	2	12	19.50	21.43
LH	1	12	2.08	2.93
LH	2	12	8.33	9.64

*Note. HH=High Hostile , LH=Low Hostile , Condition 1=verbal stressor , Condition 2= nonverbal stressor.*



Table 7

*Phase II ANOVA summary table for the Glucose (mg/dl) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	0.17	1	0.68
Trial	1.37	2	0.26
Group x Trial	0.57	2	0.57

Note. Variables noted with \* reflect significance of  $p \leq .05$ .

Table 8

*Phase II ANOVA summary table for the Systolic Blood Pressure (mmHg) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	0.16	1	0.69
Trial	1.83	2	0.17
Group x Trial	0.89	2	0.41

*Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 9

*Phase II ANOVA summary table for the Diastolic Blood Pressure (mmHg) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	1.14	1	0.29
Trial	1.58	2	0.21
Group x Trial	0.34	2	0.71

*Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 10

*Phase II ANOVA summary table for the Heart Rate (bpm) comparisons.*

Source	<u>F</u>	<u>df</u>	<u>p</u>
Group	0.07	1	0.79
Trial	6.36	2	.0037*
Group x Trial	0.33	2	0.72

*Note. Variables noted with \* reflect significance of  $p \leq .05$ .*

Table 11

*Mean glucose levels (mg/dl) as a function of Group and Trial.*

Group	Trial	N	Mean	SD
HH	1	12	103.33	13.08
HH	2	12	101.58	14.73
HH	3	12	103.33	12.49
LL	1	12	98.75	22.14
LL	2	12	98.83	23.02
LL	3	12	101.83	20.64

*Note. HH=High Hostile , LH=Low Hostile , Trial 1=pre stress, Trail 2=post stress, Trial 3=10 minutes after stress*

Figure 1

Group differences in Glucose levels (mg/dl) as a function of Condition (verbal or nonverbal stressor).

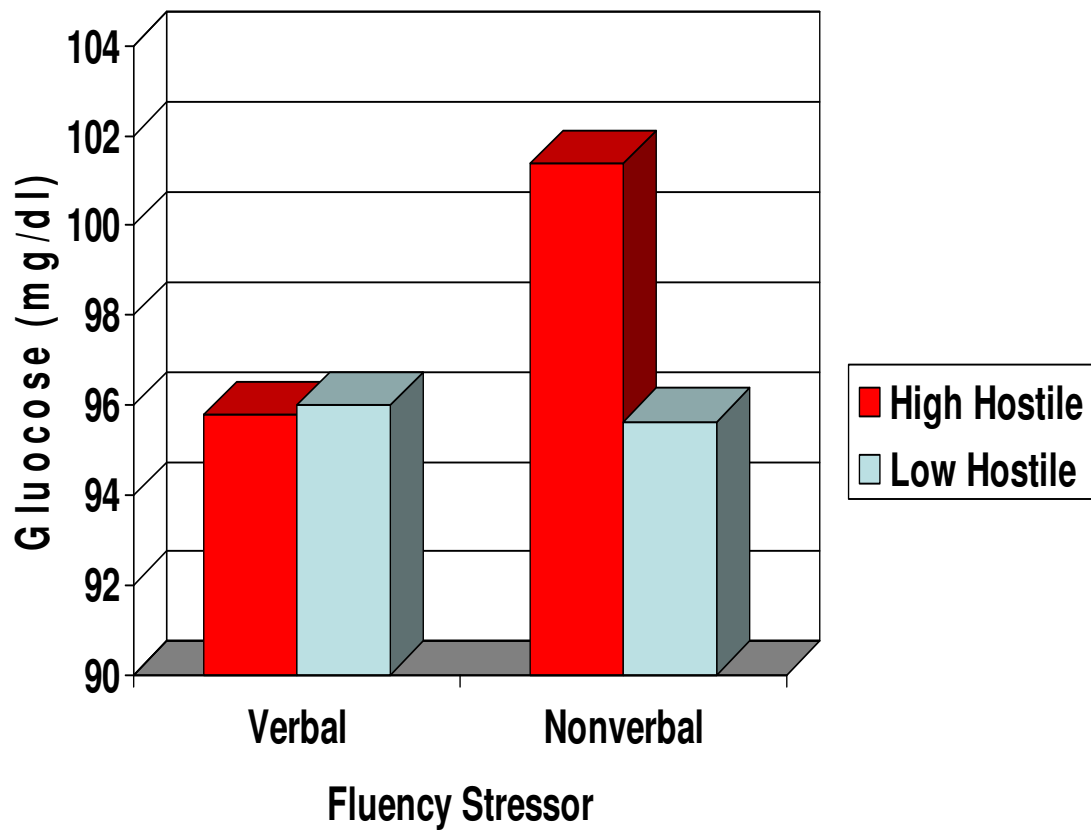
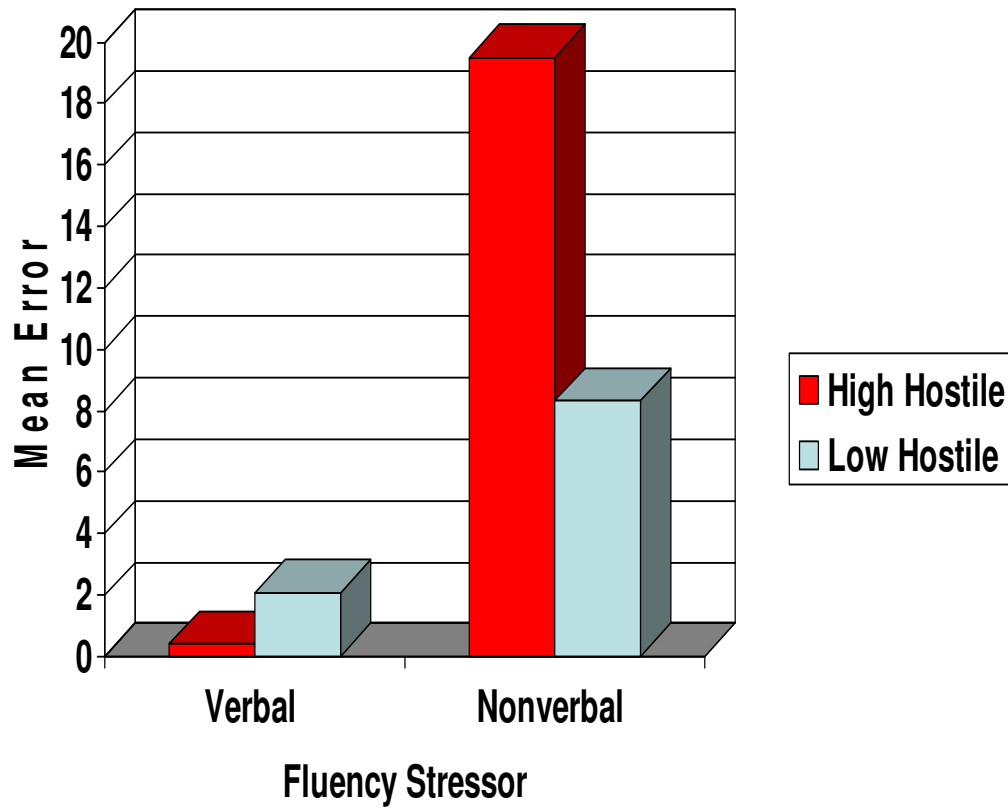


Figure 2

Mean error scores as a function of Group and Condition (verbal or nonverbal stressor).



## Appendix A Glucose Measurement

1. Before beginning be sure to wear latex gloves to prevent the spread the blood-borne pathogens.
2. Use isopropyl alcohol swab to clean the outside of the lancing device
3. Remove the cap of the lancing device. Snap off the clear cap on the lancing device at an angle
4. Insert the lancet firmly into the white lancet holder cup. (Pushing the lancet into the cup may cock the device; this is okay).
5. Twist off the rounded top of the lancet by holding the lancet firmly in place with one hand; use the other hand to twist off the rounded top
6. Replace the cap until it snaps or clicks into place. Be careful not to touch the exposed needle on the lancet
7. Set the desired lancing level by moving the dial to the appropriate level. Level 1 is the shallowest depth, level 5 is the deepest depth, and level 3 is recommended.
8. Pull the dark blue cocking handle out until it clicks.
9. To bring fresh blood to the surface of the forearm, rub the test site vigorously for a few seconds until you feel it getting warm. Hold the clear cap down against the top of the forearm. Depress the release button. Do not lift up.
10. Continue to hold the lancing device and gradually increase pressure for a few seconds.
11. While holding the lancing device, look at the arm, look through the clear cap; the blood sample should be about the size of a pinhead.
12. Lift the lancing device straight up; be careful not to smear the blood sample.
13. Make sure the test strip is in the meter and the meter is turned on.
14. Bring the test strip to the blood sample at a slight angle.
15. Do not remove the test strip until the meter makes a beep or the screen begins moving clockwise.
16. Testing is complete when the meter makes two beeps and the blood glucose level is displayed on the screen.
17. When testing is finished, snap off the clear cap from the lancing device.
18. Hold the lancet over a sharps container or a puncture proof container and lid
19. Pinch the white clip that hold the lancet until the lancet falls out



Appendix B  
Cook Medley Hostility Scale

Direction: If a statement is true or mostly true, as pertaining to you, circle the letter T.  
If a statement is false, or usually not true about you, circle the letter F.  
Try to give a response to every statement.

1. When I take a new job, I like to be tipped off on who should be gotten next to.	T	F
2. When someone does me wrong, I feel I should pay him back if I can, just for the principle of the thing.	T	F
3. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.	T	F
4. I often had to take orders from someone who did not know as much as I did.	T	F
5. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.	T	F
6. It takes a lot of argument to convince most people of the truth.	T	F
7. I think most people lie to get ahead.	T	F
8. Someone has it in for me.	T	F
9. Most people are honest chiefly through the fear of getting caught.	T	F
10. Most people will use somewhat unfair means to gain profit or an advantage, rather than lose it.	T	F
11. I commonly wonder what hidden reason another person may have for doing something nice for me.	T	F
12. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.	T	F
13. I feel that I have often been punished without cause.	T	F
14. I am against giving money to beggars.	T	F
15. Some of my family have habits that bother me very much.	T	F
16. My relatives are nearly all in sympathy with me.	T	F
17. My way of doing things is apt to be misunderstood by others.	T	F
18. I don't blame anyone for trying to grab everything they can get in this world.	T	F
19. No one cares what happens to you.	T	F
20. I can be friendly with people who do things I consider wrong.	T	F
21. It is safer to trust nobody.	T	F
22. I do not blame a person for taking advantage of someone who lays himself open to it.	T	F
23. I have often felt that strangers were looking at me critically.	T	F
24. Most people make friends because friends are likely to be useful to them.	T	F
25. I am sure that I am being talked about.	T	F
26. I am likely not to speak to people until they speak to me.	T	F
27. Most people inwardly dislike putting themselves out to help other people.	T	F

28. I tend to be on guard with people who are somewhat more friendly than I had expected.	T	F
29. I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.	T	F
30. People often disappoint me.	T	F
31. I like to keep people guessing what I'm going to do next.	T	F
32. I frequently ask people for advice.	T	F
33. I am not easily angered.	T	F
34. I have often met people who are supposed to be experts who were no better than I.	T	F
35. It makes me think of failure when I hear of the success of someone I know well.	T	F
36. I would certainly enjoy beating a crook at his own game.	T	F
37. I have at times had to be rough with people who were rude or annoying.	T	F
38. People generally demand more respect for their own rights than they are willing to allow for others.	T	F
39. There are certain people whom I dislike so much I am inwardly pleased when they are catching it for something they have done.	T	F
40. I am often inclined to go out of my way to win a point with someone who has opposed me.	T	F
41. I am quite often not in on the gossip and talk of the group I belong to.	T	F
42. The man who had the most to do with me when I was a child (such as my father, step- father, etc.) was very strict with me.	T	F
43. I have often found people jealous of my good ideas just because they had not thought of them first.	T	F
44. When a man is with a woman, he is usually thinking of things related to her sex.	T	F
45. I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.	T	F
46. I have frequently worked under people who seem to have things arranged so that they get credit for good work, but are able to pass off mistakes to those under them.	T	F
47. I strongly defend my own opinions as a rule.	T	F
48. People can pretty easily change me even though I thought that my mind was made up on a subject.	T	F
49. Sometimes I am sure that other people can tell what I'm thinking.	T	F
50. A large number of people are guilty of bad sexual conduct.	T	F

Appendix C  
Medical History Questionnaire

1	Do you have any history of congenital or developmental problems ?	Yes	No
2	Do you have any history of learning disabilities or special education?	Yes	No
3	Do you have any history of hypoglycemia (low blood glucose)?	Yes	No
4	Do you have any history of hyperglycemia (diabetes)?	Yes	No
5	Are you experiencing blood glucose problems at present?	Yes	No
6	Do you have any history of hypertension? (high blood pressure)	Yes	No
7	Do you have any history of hypotension? (low blood pressure)	Yes	No
8	Do you have any history of hyperthyroidism?	Yes	No
9	Do you have any history of hypothyroidism?	Yes	No
10	Have you ever suffered a head injury resulting in a hospital stay longer than 24 hours?	Yes	No
11	Have you ever been knocked out or rendered unconscious (more than 5 minutes)?	Yes	No
12	Have you ever suffered "black-out" or fainting spells?	Yes	No
13	Do you have a history of other neurological disorders (e.g. stroke or brain tumor)?	Yes	No
14	Have you ever received psychiatric/psychological care or counseling?	Yes	No
15	Have you ever been hospitalized in a psychiatric facility/hospital?	Yes	No
16	Have you ever been diagnosed with a psychiatric/psychological disorder?	Yes	No
17	Have you ever been administered any (neuro)psychological tests or measures?	Yes	No
18	Do you have a history of substance abuse or alcohol abuse?	Yes	No
19	Do you have any history of heart disease?	Yes	No
20	Do you have any history of pancreatic disease?	Yes	No
21	Are you currently taking any prescription blood-thinning medications?	Yes	No
22	Do you have a history of high blood pressure?	Yes	No
23	Do you have any uncorrected visual or hearing impairments?	Yes	No
24	Are you able to read, write, and speak English effectively?	Yes	No
25	Do you consume three or more alcoholic more than two nights a week?	Yes	No
26	Have you ever experienced a medical or psychiatric condition that could potentially affect cognitive functioning, such as stroke, electroconvulsive treatment, epilepsy, brain surgery, encephalitis, meningitis, multiple sclerosis, Parkinson's Disease, Huntington's Chorea, Alzheimer's dementia, Schizophrenia, Bipolar Disorder ?	Yes	No
27	Have you ever used smoked or used tobacco products?	Yes	No
28	Do you use any unprescribed or "illegal/street" drugs?	Yes	No
29	Are you taking any of the following medications: antidepressant, antianxiety, antipsychotic?	Yes	No
30	Are you taking any allergy or cold medication?	Yes	No

If you answered “yes” to any of the above please explain fully:

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Appendix D  
Laterality Questionnaire

Participant #: \_\_\_\_\_

Circle the appropriate number after each item.

With which hand would you throw a ball to hit a target?	1	-1	0
With which hand do you draw?	1	-1	0
With which hand do you use an eraser on paper?	1	-1	0
With which hand do you remove the top card when dealing?	1	-1	0
With which foot do you kick a ball?	1	-1	0
If you had to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up on a chair, which foot would you place on the chair first?	1	-1	0
Which eye would you use to peep through a keyhole?	1	-1	0
If you had to look into a dark bottle to see how full it was which eye would you use?	1	-1	9
Which eye would you use to sight down a rifle?	1	-1	0
If you wanted to listen to a conversation going on behind a closed door, which ear would you place against the door?	1	-1	0
If you wanted to listen to someone's heartbeat, which ear would you place against his or her chest?	1	-1	0
Into which ear would you place your earphone of a transistor radio?	1	-1	0

# of Right    +    # of Left    =    Total Score  
 \_\_\_\_\_    +    \_\_\_\_\_    =    \_\_\_\_\_

Is mother right or left hand dominant? \_\_\_\_\_

Is father right or left hand dominant? \_\_\_\_\_

Appendix E  
Alcohol Use Questionnaire

This information will be used confidentially with only your subject number. We wish to determine if you drink alcohol or not, as it might affect your EEG.

1. **Since January 2002**, on the average how many drinks do you have when you drink alcohol? (one drink equals 12 oz beer, 4 oz wine, or 1 - 1 1/2 oz of hard liquor)

- 1. I did not drink alcohol during this time period
- 2. 1-2 drinks
- 3. 3-4 drinks
- 4. 5-6 drinks
- 5. 7-8 drinks
- 6. 9-12 drinks
- 7. 13-16 drinks
- 8. 17 or more

2. **OVER THE PAST TWO WEEKS**, on the average how many drinks per day do you have when you drink alcohol?  
(one drink equals 12 oz beer, 4 oz wine, or 1 - 1 1/2 oz of hard liquor)

- 1. I did not drink alcohol during this time period
- 2. 1-2 drinks
- 3. 3-4 drinks
- 4. 5-6 drinks
- 5. 7-8 drinks
- 6. 9-12 drinks
- 7. 13-16 drinks
- 8. 17 or more

3. On how many occasions **OVER THE LAST TWO WEEKS** did you consume five or more drinks at one sitting?  
(one drink = 12 oz beer, 4 oz wine; 1 - 1 1/2 oz of hard liquor)

- 1. I did not drink alcohol during this time period
- 2. 1-2 occasions
- 3. 3-4 occasions
- 4. 5-6 occasions
- 5. 7-8 occasions
- 6. 9-12 occasions
- 7. 13-16 occasions
- 8. 17 or more
- 9. I drank alcohol, but always less than five drinks at one sitting

4. When was the last time you drank alcohol and how much did you drink?

Appendix F  
Nicotine Dependence Questionnaire

Fagerström Test for Nicotine Dependence                      Subject No. \_\_\_\_\_  
 Heatherton, T.F., Kozlowski, L.T.,                              Age \_\_\_\_\_  
 Frecker, R.C., & Fagerström, K-O (1991)                      Sex                      M \_\_\_ F \_\_\_

The Fagerström Test for Nicotine Dependence:  
 A revision of the Fagerström Tolerance Questionnaire.  
 Britist Journal of Addiction, 86, 1119-1127.

Type of Cigarette Smoked: \_\_\_\_\_

1. How soon after you wake up do you smoke your first cigarette?

Within 5 minutes (3 points)    \_\_\_                      \_\_\_\_\_  
 6 – 30 minutes (2 points)        \_\_\_  
 31-60 minutes (1 point)        \_\_\_  
 After 60 minutes (0 points)    \_\_\_

2. Do you find it difficult to refrain from smoking in places where  
 It is forbidden e.g. in church, at library, cinema, etc?

Yes (1 point)                      \_\_\_                      \_\_\_\_\_  
 No (0 point)                        \_\_\_

3. Which cigarette would you hate most to give up?

The first one in the morning (1 point) \_\_\_                      \_\_\_\_\_  
 All others (0)                        \_\_\_

4. How many cigarettes/day do you smoke?

10 or less (0 points)    \_\_\_                      \_\_\_\_\_  
 11-20 (1 point)            \_\_\_  
 21-30 (2 points)         \_\_\_  
 31 or more (3 points)    \_\_\_

5. Do you smoke more frequently during the first hours after waking  
 than during the rest of the day?

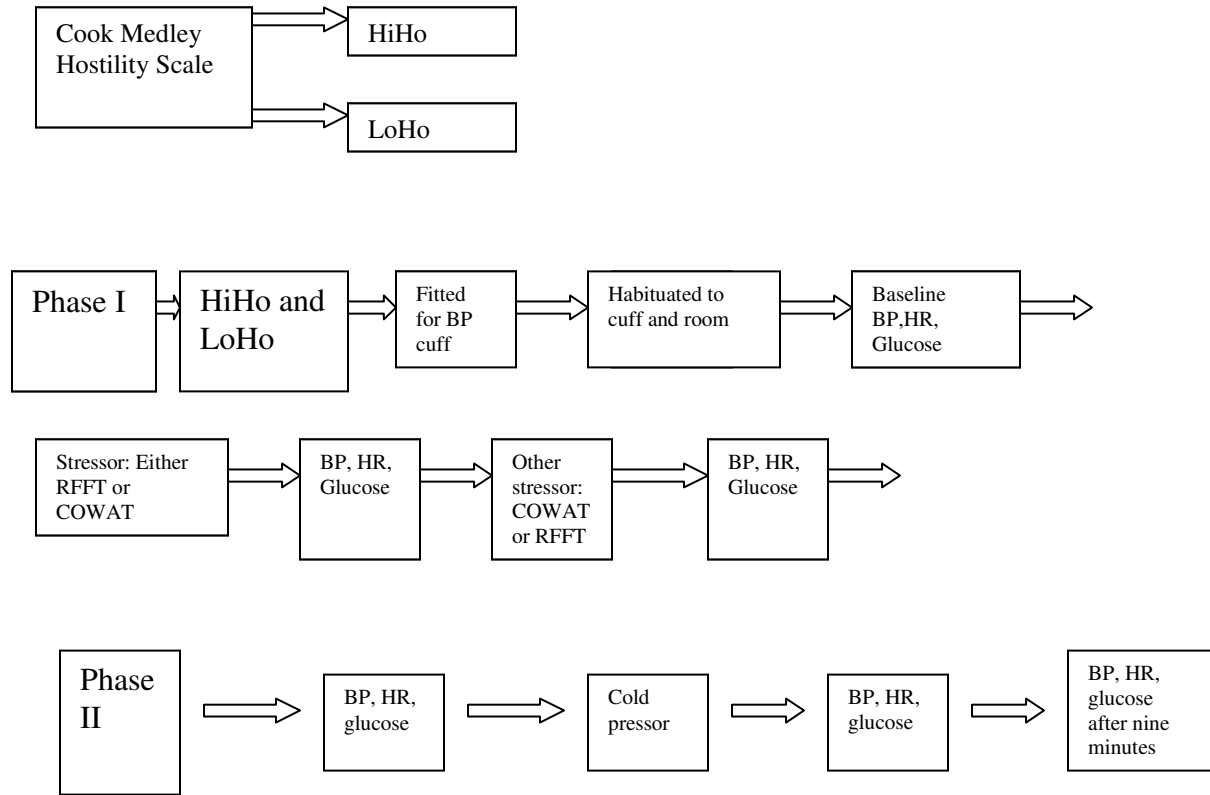
Yes (1 point)                      \_\_\_                      \_\_\_\_\_  
 No (0 pint)                        \_\_\_

6. Do you smoke if you are so ill that you are in bed most of the day?

Yes (1 point)                      \_\_\_                      \_\_\_\_\_  
 No (0 pint)                        \_\_\_

TOTAL:                                \_\_\_\_\_

Appendix G  
Flow Chart for Participants





Appendix H  
Phase I Data for Physiological Variables

Subject	CMHO1	CMHO2	MedHx	Handed	Smoke	Snack	Caff	Condition	Trial	Glucose	SBP	DBP	HR
10	9	8	2	13	0	1	0	1	1	96	117	69	66
10	9	8	2	13	0	1	0	1	2	96	119	70	64
10	9	8	2	13	0	1	0	2	1	84	126	72	63
10	9	8	2	13	0	1	0	2	2	91	115	72	67
4	13	12	1	5	0	1	0	1	1	81	131	68	68
4	13	12	1	5	0	1	0	1	2	89	135	76	64
4	13	12	1	5	0	1	0	2	1	86	125	74	67
4	13	12	1	5	0	1	0	2	2	91	138	68	65
27	13	12	1	9	0	0	0	1	1	106	129	64	86
27	13	12	1	9	0	0	0	1	2	106	135	104	78
27	13	12	1	9	0	0	0	2	1	108	127	61	80
27	13	12	1	9	0	0	0	2	2	104	142	72	73
8	14	13	1	13	0	.	.	1	1	85	118	54	60
8	14	13	1	13	0	.	.	1	2	101	117	56	58
8	14	13	1	13	0	.	.	2	1	103	119	62	60
8	14	13	1	13	0	.	.	2	2	110	118	72	58
21	13	13	3	2	0	0	0	1	1	95	118	69	76
21	13	13	3	2	0	0	0	1	2	96	123	80	68
21	13	13	3	2	0	0	0	2	1	92	122	70	72
21	13	13	3	2	0	0	0	2	2	96	122	68	68
18	16	14	1	9	0	0	0	1	1	76	113	77	48
18	16	14	1	9	0	0	0	1	2	78	121	69	48
18	16	14	1	9	0	0	0	2	1	69	115	72	54
18	16	14	1	9	0	0	0	2	2	72	112	75	60
30	7	15	1	5	0	0	0	1	1	84	114	68	89
30	7	15	1	5	0	0	0	1	2	82	126	80	72
30	7	15	1	5	0	0	0	2	1	84	124	75	86
30	7	15	1	5	0	0	0	2	2	83	130	72	71
5	18	17	3	7	0	0	0	1	1	81	101	67	66
5	18	17	3	7	0	0	0	1	2	86	101	76	71
5	18	17	3	7	0	0	0	2	1	89	113	69	71
5	18	17	3	7	0	0	0	2	2	86	102	71	72
15	15	17	0	11	0	1	0	1	1	145	124	70	80
15	15	17	0	11	0	1	0	1	2	167	132	80	91
15	15	17	0	11	0	1	0	2	1	160	124	75	92
15	15	17	0	11	0	1	0	2	2	158	131	76	92
19	16	17	3	13	0	1	0	1	1	83	103	62	71
19	16	17	3	13	0	1	0	1	2	99	121	64	73
19	16	17	3	13	0	1	0	2	1	87	118	56	67
19	16	17	3	13	0	1	0	2	2	88	111	60	71
7	17	18	2	11	0	0	0	1	1	77	133	81	56
7	17	18	2	11	0	0	0	1	2	81	143	77	51
7	17	18	2	11	0	0	0	2	1	80	132	80	56
7	17	18	2	11	0	0	0	2	2	84	140	85	56
16	19	18	5	7	0	1	0	1	1	110	115	64	66
16	19	18	5	7	0	1	0	1	2	104	125	76	71
16	19	18	5	7	0	1	0	2	1	86	126	75	83
16	19	18	5	7	0	1	0	2	2	104	125	79	76
23	19	19	3	9	0	1	0	1	1	99	112	65	66
23	19	19	3	9	0	1	0	1	2	104	120	70	65
23	19	19	3	9	0	1	0	2	1	101	112	61	73
23	19	19	3	9	0	1	0	2	2	103	116	68	63
32	16	19	1	6	0	0	0	1	1	127	138	65	69
32	16	19	1	6	0	0	0	1	2	125	125	70	65
32	16	19	1	6	0	0	0	2	1	122	122	68	72
32	16	19	1	6	0	0	0	2	2	125	128	64	67
13	19	20	2	12	0	0	0	1	1	108	117	68	84
13	19	20	2	12	0	0	0	1	2	107	127	69	77
13	19	20	2	12	0	0	0	2	1	115	112	74	88

13	19	20	2	12	0	0	0	2	2	102	122	73	77
3	18	23	1	11	0	1	0	1	1	106	129	68	87
3	18	23	1	11	0	1	0	1	2	97	140	70	84
3	18	23	1	11	0	1	0	2	1	97	134	67	96
3	18	23	1	11	0	1	0	2	2	98	124	71	95
12	20	23	2	8	0	1	0	1	1	99	102	56	88
12	20	23	2	8	0	1	0	1	2	111	103	77	86
12	20	23	2	8	0	1	0	2	1	90	109	68	96
12	20	23	2	8	0	1	0	2	2	96	105	68	84
11	33	28	2	8	0	0	0	1	1	79	124	80	71
11	33	28	2	8	0	0	0	1	2	77	140	84	64
11	33	28	2	8	0	0	0	2	1	84	139	80	69
11	33	28	2	8	0	0	0	2	2	81	139	77	64
17	29	28	2	7	0	.	.	1	1	78	108	67	76
17	29	28	2	7	0	.	.	1	2	92	104	69	82
17	29	28	2	7	0	.	.	2	1	98	102	61	78
17	29	28	2	7	0	.	.	2	2	95	112	72	70
9	33	29	3	12	0	1	0	1	1	87	139	73	65
9	33	29	3	12	0	1	0	1	2	87	132	68	61
9	33	29	3	12	0	1	0	2	1	93	99	80	57
9	33	29	3	12	0	1	0	2	2	83	132	72	75
25	30	32	1	10	0	1	0	1	1	99	124	72	82
25	30	32	1	10	0	1	0	1	2	93	131	84	82
25	30	32	1	10	0	1	0	2	1	89	132	74	91
25	30	32	1	10	0	1	0	2	2	101	137	75	80
26	37	33	4	10	0	0	0	1	1	105	108	81	66
26	37	33	4	10	0	0	0	1	2	105	112	66	64
26	37	33	4	10	0	0	0	2	1	91	112	57	67
26	37	33	4	10	0	0	0	2	2	104	116	74	65
31	30	33	2	5	0	1	0	1	1	99	129	60	63
31	30	33	2	5	0	1	0	1	2	107	119	62	65
31	30	33	2	5	0	1	0	2	1	105	123	52	69
31	30	33	2	5	0	1	0	2	2	105	116	61	71
1	33	34	1	10	0	0	0	1	1	95	118	67	65
1	33	34	1	10	0	0	0	1	2	98	137	75	47
1	33	34	1	10	0	0	0	2	1	100	121	76	67
1	33	34	1	10	0	0	0	2	2	98	131	76	70
2	29	34	3	4	0	1	0	1	1	96	121	56	52
2	29	34	3	4	0	1	0	1	2	111	116	66	53
2	29	34	3	4	0	1	0	2	1	109	114	57	65
2	29	34	3	4	0	1	0	2	2	123	114	58	60
6	35	34	3	7	1	1	1	1	1	93	120	69	76
6	35	34	3	7	1	1	1	1	2	112	125	60	70
6	35	34	3	7	1	1	1	2	1	116	116	64	68
6	35	34	3	7	1	1	1	2	2	132	121	62	68
28	31	35	2	9	0	1	0	1	1	87	100	54	62
28	31	35	2	9	0	1	0	1	2	106	112	67	56
28	31	35	2	9	0	1	0	2	1	102	103	64	60
28	31	35	2	9	0	1	0	2	2	99	102	54	64
29	35	36	0	9	0	0	0	1	1	83	116	64	47
29	35	36	0	9	0	0	0	1	2	89	119	67	48
29	35	36	0	9	0	0	0	2	1	95	109	60	46
29	35	36	0	9	0	0	0	2	2	87	112	56	48
14	41	38	1	7	0	1	0	1	1	103	120	62	68
14	41	38	1	7	0	1	0	1	2	118	123	68	67
14	41	38	1	7	0	1	0	2	1	124	126	63	61
14	41	38	1	7	0	1	0	2	2	119	123	66	64
24	39	39	1	13	0	1	0	1	1	112	121	72	64
24	39	39	1	13	0	1	0	1	2	104	129	70	62
24	39	39	1	13	0	1	0	2	1	99	122	72	57
24	39	39	1	13	0	1	0	2	2	107	125	66	59
34	39	39	5	12	0	0	0	1	1	96	144	75	76
34	39	39	5	12	0	0	0	1	2	102	144	79	70
34	39	39	5	12	0	0	0	2	1	103	145	84	65
34	39	39	5	12	0	0	0	2	2	94	145	83	81

Appendix I  
Phase I Data for Behavioral Measures

Subject	CMHO1	CMHO2	MedHx	Handed	Smoke	Snack	Caff	Condition	Score	Error
10	9	8	2	13	0	1	0	1	42	0
10	9	8	2	13	0	1	0	2	41	8
4	13	12	1	11	0	1	0	1	41	0
4	13	12	1	11	0	1	0	2	69	10
27	13	12	1	9	0	0	0	1	40	8
27	13	12	1	9	0	0	0	2	51	2
8	14	13	1	13	0	.	.	1	28	0
8	14	13	1	13	0	.	.	2	37	2
21	13	13	3	2	0	0	0	1	47	0
21	13	13	3	2	0	0	0	2	66	7
18	16	14	1	9	0	0	0	1	47	1
18	16	14	1	9	0	0	0	2	53	15
30	7	15	1	5	0	0	0	1	41	1
30	7	15	1	5	0	0	0	2	44	7
5	18	17	3	7	0	0	0	1	55	4
5	18	17	3	7	0	0	0	2	60	0
15	15	17	0	11	0	1	0	1	68	0
15	15	17	0	11	0	1	0	2	64	5
19	16	17	3	13	0	1	0	1	36	0
19	16	17	3	13	0	1	0	2	45	36
7	17	18	2	11	0	0	0	1	43	4
7	17	18	2	11	0	0	0	2	47	2
16	19	18	5	7	0	1	0	1	47	7
16	19	18	5	7	0	1	0	2	62	6
23	19	19	3	9	0	1	0	1	35	5
23	19	19	3	9	0	1	0	2	53	1
32	16	19	1	6	0	0	0	1	22	2
32	16	19	1	6	0	0	0	2	44	18
13	19	20	2	12	0	0	0	1	37	3
13	19	20	2	12	0	0	0	2	39	46
3	18	23	1	11	0	1	0	1	53	0
3	18	23	1	11	0	1	0	2	55	5
12	20	23	2	8	0	1	0	1	55	1
12	20	23	2	8	0	1	0	2	65	10
11	33	28	2	8	0	0	0	1	26	0
11	33	28	2	8	0	0	0	2	45	1
17	29	28	2	7	0	.	.	1	40	0
17	29	28	2	7	0	.	.	2	72	14
9	33	29	3	12	0	1	0	1	20	0
9	33	29	3	12	0	1	0	2	67	14
25	30	32	1	10	0	1	0	1	56	1
25	30	32	1	10	0	1	0	2	46	43
26	37	33	4	10	0	0	0	1	40	0
26	37	33	4	10	0	0	0	2	47	4
31	30	33	2	5	0	1	0	1	41	0
31	30	33	2	5	0	1	0	2	43	0
1	33	34	1	10	0	0	0	1	37	1
1	33	34	1	10	0	0	0	2	52	53
2	29	34	3	4	0	1	0	1	45	2
2	29	34	3	4	0	1	0	2	53	47
6	35	34	3	7	1	1	1	1	47	0
6	35	34	3	7	1	1	1	2	40	5
28	31	35	2	9	0	1	0	1	40	0
28	31	35	2	9	0	1	0	2	57	48
29	35	36	0	9	0	0	0	1	31	1
29	35	36	0	9	0	0	0	2	58	2
14	41	38	1	7	0	1	0	1	36	0
14	41	38	1	7	0	1	0	2	58	3
24	39	39	1	13	0	1	0	1	51	1

24	39	39	1	13	0	1	0	2	72	9
34	39	39	5	12	0	0	0	1	37	1
34	39	39	5	12	0	0	0	2	41	6

Appendix J  
Phase II Data for Cold Pressor

Subject	CMHO1	CMHO2	MedHx	Handed	Smoke	Snack	Caff	Trial	Glucose	SBP	DBP	HR
10	9	8	2	13	0	1	0	1	98	120	64	64
10	9	8	2	13	0	1	0	2	100	110	55	57
10	9	8	2	13	0	1	0	3	105	112	74	62
4	13	12	1	5	0	1	0	1	89	120	68	63
4	13	12	1	5	0	1	0	2	89	138	84	63
4	13	12	1	5	0	1	0	3	89	128	75	64
27	13	12	1	9	0	0	0	1	101	130	64	78
27	13	12	1	9	0	0	0	2	98	94	79	73
27	13	12	1	9	0	0	0	3	114	141	57	77
8	14	13	1	13	0	.	.	1	114	106	67	58
8	14	13	1	13	0	.	.	2	117	126	68	61
8	14	13	1	13	0	.	.	3	130	118	76	57
21	13	13	3	2	0	0	0	1	97	115	69	72
21	13	13	3	2	0	0	0	2	101	123	76	64
21	13	13	3	2	0	0	0	3	94	122	68	66
18	16	14	1	9	0	0	0	1	80	121	72	53
18	16	14	1	9	0	0	0	2	81	112	68	45
18	16	14	1	9	0	0	0	3	81	116	80	52
30	7	15	1	5	0	0	0	1	85	117	76	70
30	7	15	1	5	0	0	0	2	85	124	76	75
30	7	15	1	5	0	0	0	3	87	111	68	70
5	18	17	3	7	0	0	0	1	89	128	72	64
5	18	17	3	7	0	0	0	2	87	105	64	60
5	18	17	3	7	0	0	0	3	89	112	72	57
15	15	17	0	11	0	1	0	1	162	127	69	89
15	15	17	0	11	0	1	0	2	164	130	76	76
15	15	17	0	11	0	1	0	3	148	120	72	85
19	16	17	3	13	0	1	0	1	93	110	64	69
19	16	17	3	13	0	1	0	2	93	124	61	48
19	16	17	3	13	0	1	0	3	110	116	64	61
7	17	18	2	11	0	0	0	1	79	132	89	52
7	17	18	2	11	0	0	0	2	78	141	80	49
7	17	18	2	11	0	0	0	3	81	128	83	52
16	19	18	5	7	0	1	0	1	98	124	69	77
16	19	18	5	7	0	1	0	2	93	140	85	68
16	19	18	5	7	0	1	0	3	94	122	69	70
23	19	19	3	9	0	1	0	1	99	115	70	62
23	19	19	3	9	0	1	0	2	94	112	69	55
23	19	19	3	9	0	1	0	3	116	116	68	66
32	16	19	1	6	0	0	0	1	124	124	53	75
32	16	19	1	6	0	0	0	2	123	139	89	65
32	16	19	1	6	0	0	0	3	122	140	68	73
13	19	20	2	12	0	0	0	1	107	120	69	60
13	19	20	2	12	0	0	0	2	108	123	69	68
13	19	20	2	12	0	0	0	3	119	121	72	86
3	18	23	1	11	0	1	0	1	96	126	64	68
3	18	23	1	11	0	1	0	2	98	127	73	68
3	18	23	1	11	0	1	0	3	99	124	63	76
12	20	23	2	8	0	1	0	1	117	107	64	96
12	20	23	2	8	0	1	0	2	122	104	69	84
12	20	23	2	8	0	1	0	3	131	104	68	75
11	33	28	2	8	0	0	0	1	83	129	78	68
11	33	28	2	8	0	0	0	2	84	132	84	71
11	33	28	2	8	0	0	0	3	85	135	68	64
17	29	28	2	7	0	.	.	1	99	104	63	84
17	29	28	2	7	0	.	.	2	100	115	65	49
17	29	28	2	7	0	.	.	3	109	105	64	69
9	33	29	3	12	0	1	0	1	89	127	80	80
9	33	29	3	12	0	1	0	2	84	119	69	62

9	33	29	3	12	0	1	0	3	94	138	88	66
25	30	32	1	10	0	1	0	1	103	128	70	82
25	30	32	1	10	0	1	0	2	105	140	98	70
25	30	32	1	10	0	1	0	3	106	123	76	88
26	37	33	4	10	0	0	0	1	103	108	72	60
26	37	33	4	10	0	0	0	2	98	123	84	74
26	37	33	4	10	0	0	0	3	98	108	72	66
31	30	33	2	5	0	1	0	1	101	119	48	67
31	30	33	2	5	0	1	0	2	97	108	36	44
31	30	33	2	5	0	1	0	3	101	112	58	64
1	33	34	1	10	0	0	0	1	103	123	71	68
1	33	34	1	10	0	0	0	2	98	142	86	67
1	33	34	1	10	0	0	0	3	98	142	72	72
2	29	34	3	4	0	1	0	1	117	113	52	54
2	29	34	3	4	0	1	0	2	128	112	50	52
2	29	34	3	4	0	1	0	3	116	114	68	54
6	35	34	3	7	1	1	1	1	128	113	65	58
6	35	34	3	7	1	1	1	2	111	136	87	66
6	35	34	3	7	1	1	1	3	117	124	61	68
28	31	35	2	9	0	1	0	1	101	98	56	61
28	31	35	2	9	0	1	0	2	102	103	60	53
28	31	35	2	9	0	1	0	3	110	96	59	66
29	35	36	0	9	0	0	0	1	92	110	70	46
29	35	36	0	9	0	0	0	2	85	120	68	46
29	35	36	0	9	0	0	0	3	83	114	60	43
14	41	38	1	7	0	1	0	1	121	117	63	62
14	41	38	1	7	0	1	0	2	127	139	67	60
14	41	38	1	7	0	1	0	3	123	120	52	60
24	39	39	1	13	0	1	0	1	101	123	60	52
24	39	39	1	13	0	1	0	2	99	131	73	58
24	39	39	1	13	0	1	0	3	104	122	65	60
34	39	39	5	12	0	0	0	1	94	145	72	64
34	39	39	5	12	0	0	0	2	95	144	84	61
34	39	39	5	12	0	0	0	3	99	140	79	64

## Curriculum Vitae

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### Education

H.S. Kecoughtan High School, Hampton, Va, June 1998  
 B.A. Honors Scholar from Virginia Polytechnic and State University, May 2002

Major Psychology  
 Minor Communication Studies  
 Maters (anticipated) Virginia Polytechnic Institute and State University, Spring 2005  
 Ph.D.(anticipated) Virginia Polytechnic Institute and State University, Spring 2007

### Honors and Awards

- Advanced degree awarded 2002, *In Honors Baccalaureate*
- Research Excellence Award Recipient, 2002
- Induction into four honor societies in recognition of academic success
  - University Honors Program, 1998-2002
  - University Honors Associates, 1998-2000
  - National Society of Collegiate Scholars, 1998-2002
  - Phi Eta Sigma Honor Society, 1998-2002

### Grant Recipient Awards

- Graduate Research Development Project Grant for 2004-2005
  - Funding for Master's Thesis provided by Virginia Tech Graduate School

## NEUROPSYCHOLOGY

### Neuropsychological Assessment Team

- Community participation (Spring 2004)
  - Participation in community services board planning team for patient's living, work, and social environments subsequent to completion of neuropsychological assessment for patient (referred by community services board)
- Supervisor of Neuropsychological Assessment Team (Spring 2004)
  - Provided team instruction on syndrome analysis for identification of neuropsychological symptoms of brain disorders

- Lead team didactic of patient cases
  - Reviewed and assisted newer members of team with assessment report writing
  - Demonstrated and supervised administration and interpretation of neuropsychological tests to include the Trail Making Test Part A and B, the Rey Auditory Verbal Learning and Rey Recognition test (RAVLT), the Rey Copying a Complex Figure Test, the Ruff Figural Fluency Test, the Beck Depression Inventory (BDI), the State Trait Anxiety Inventory (STAXI- 2, Form HS), the Luria's Circle, Square, Triangle test, the Luria's M and N's test, the Zung Anxiety Inventory, Draw a House test, Draw a Clock test, Controlled Oral Word Association Task (COWAT), and the Wechsler Memory Scale, 3<sup>rd</sup> Edition (WMS-III)
  - Instruction provided to newer members of team on the administration and analysis of Quantitative Electroencephalogram (QEEG)
- Member of the Neuropsychological Assessment Team (Fall 2003, Spring 2004)
    - Completed six assessments for the Fall 2003 and Spring 2004 semesters
      - Assessment for head injury, developmental disabilities, mental retardation, chronic pain, and memory deficits
    - Development of syndrome analysis skills to identify clusters of neuropsychological symptoms reflecting specific types of impairment
    - Developed administration and interpretation of neuropsychological test skills
      - Completed a total of six assessments for Fall 2003 and Spring 2004 semesters (includes interview, testing, scoring, analysis, report writing, and feedback)
    - Completed three QEEGs for patients as part of neuropsychological assessment
- Attendance on the Neuropsychological Assessment Team (Fall 2001 - Spring 2003)
    - Exposure to syndrome analysis
    - Exposure to QEEG
    - Exposure to and experience acquired for the administration of neuropsychological tests

### **Neuropsychological Technician**

- Employed in Neuropsychological Private Practice (1 year) as Neuropsychological Technician
- Employed at Lewis Gale Medical Center (1 year) as Neuropsychological Technician
  - Experience in scheduling outpatient appointments as well as administration and scoring of neuropsychological tests
- Assisted with neuropsychological assessments to include aspects of syndrome analysis and standardized testing for Head Injury, Dementia, Stroke, Neoplasm, Concussion, Alzheimer's Disease, Herpetic Encephalopathy, Viral Encephalopathy, Hostility, Expressive Aphasia, Receptive Aphasia, Visual Hallucinations, Auditory Hallucinations, Cortical Blindness, among others
- Administration of neuropsychological assessment techniques and instruments to include Hand Dynamometer, Finger Tapping, Sensory Screening, Rapid Alternating



Movements, Finger to Nose, Frontal Eye Fields, Geographical Awareness, Rey Complex Figure, among others

- Administration of standardized neuropsychological assessments to include The Denman Neuropsychological Battery, Dementia Rating Scale, Trail Making A and B, Wechsler Memory Scale (III), Rey Auditory Verbal Learning Test, Beck Depression Inventory, Geriatric Depression Inventory, Zung Anxiety Scale.
- Administration and analysis of numerous Quantitative Electroencephalograms (QEEG)

## CLINICAL PSYCHOLOGY

### **Clinical Experiences**

- Participation on clinical practicum teams (8 semesters)
  - Volunteered for additional clinical practicum for Fall 2003 and Spring 2004
- Logged a over 108 therapy contact hours with clients in session (not including assessment time, practicum time, or report writing)
- Acquired the ability to develop case diagnosis, conceptualization, and carry out treatment strategies
- Experience with diagnosis and treatment of Depression, Pedophilia, Attention Deficit Disorder, Explosive Anger Disorder, Paranoid Personality Disorder, Dependent Personality Disorder, and Generalized Anxiety Disorder
- Participation in both long term therapy (one year) and more intense short term therapy (several weeks)

### **Psychological Assessment**

- Employed through the Psychological Services Center to administer Psychoeducational assessments (Spring 2004)
- Three assessments completed for Attention Deficit Disorder, Attention Deficit/Hyperactivity Disorder, Reality Testing, and Learning Disability for Reading and Mathematics
- Administration, scoring, and interpretation of Conner's Adult ADHD Rating Scale (Self), Conner's Adult ADHD Rating Scale (Other), Conner's Continuous Performance Test, Second Version, Conner's Retrospective Structured Clinical Interview, Paced Auditory Serial Attention Test, Symptom Checklist-90-Revised, Wechsler Adult Intelligence Scale, 3<sup>rd</sup> Edition, Wechsler Memory Scale, 3<sup>rd</sup> Edition, Wender Parents' Rating Scale, Wender Utah Rating Scale, and Wechsler Individual Achievement Test, 2<sup>nd</sup> Edition

### **Summer Practicum**

- Employed through the Psychological Services Center for summer clinical therapy position
- Completed 32 client contact hours (not including report writing, test administration, and preparation)
- Participation in weekly practicum team to assist and participate in other team member's diagnosis and treatment of clients

## Teaching

- Graduate Teaching Assistant (15 hours) during Fall 2004 for Lab in Physiological Psychology
- Graduate Teaching Assistant (15 hours) during Spring 2004 for Lab in Physiological Psychology
  - Awarded highest ranking provided by students for the categories of ‘overall rating of instructor’ and ‘concern of students’
- Graduate Teaching Assistant (10 hours) during Fall 2003 for Lab in Physiological Psychology
- Graduate Teaching Assistant (15 hours) during Spring 2003 for Introductory to Psychology Recitation.
- Graduate Teaching Assistant (15 hours) during Fall 2002 for Introductory to Psychology Recitation.

## RESEARCH

### **Undergraduate Honors Independent Study**

- Title: *An Examination of Posttraumatic Stress Disorder Regarding Manmade and Natural Disasters Concerning Children*
  - Research on Post Traumatic Stress Syndrome with relation to both natural and man made disasters
  - Topics included the role of social support and coping strategies subsequent to child exposure and experience of Hurricane Andrew, industrial fires, and single family residential fires.

### **Undergraduate Honors Thesis**

- Title: *Visual Hallucinations: An Analysis of Affective Valence*
- Archival data used from a Rehabilitation Unit in a Tertiary Care Medical Center
- Review of data revealed an association between location of visual formaesthesia and associated affective valence.
- Thesis presented to Dr. David Harrison (advisor), Dr. Helen Crawford, and Dr. Danny Axom May, 2002.

### **Masters Thesis**

- Titled: *Frontal Factors in Glucose Metabolism: A Function of Hostility*
- Proposed: November 12, 2003 to Dr. David W. Harrison (chair), Dr. W. David Crews, and Dr. Helen J. Crawford.
- Graduate Research Development Project Grant for 2004-2005

- This project entails stressing hostile individuals and examining their physiological reactivity (heart rate, blood pressure, and glucose) before and after stress

### Research in Progress

- Master's thesis titled: *Frontal Factors in Glucose Metabolism: A Function of Hostility*
- Research project titled: *Eye Tracking: An Analysis of Smooth Pursuit and the Frontal Eye Fields*
  - Using electro-oculography (EOG), smooth pursuit as well as other eye movements will be examined in both men and women, and those with elevated scores for hostility and depression
- Research project titled: *The Neurophysiology of Emotional Memories*
  - The happy and sad memories of men and women are examined through the QEEG data
  - The participant's performance on standard neuropsychological tests will be examined by the data from the QEEG

## PUBLICATIONS

- Foster, P.S., Mollet, G.A., Walters, R.P., & Harrison, D.W. (2004). Quantitative electroencephalographic evidence for the double dissociation of the trail making test. *Archives of Clinical Neuropsychology, 19*, 903.
- Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Performance on the figure trail making test as a function of right frontal lobe delta activity. *Archives of Clinical Neuropsychology, 19*, 903-904.
- Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Hostility as a function of right frontal lobe slow wave activity. *Archives of Clinical Neuropsychology, 19*, 941-942.
- Mollet, G.A., Harrison, D.W., Walters, R.P., & Foster, P.S. (2004). Thalamic syndrome: Lateralized emotional multimodal hallucinations. *Archives of Clinical Neuropsychology, 19*, 937.
- Harrison, D.W., Beck, A. L., Vendemia, J. M., & Walters R. P. (2002). Ambient Sensory Conditions: Modification of Receptive Speech Deficits in Left Side Stroke Patients Using Bight Light. *Perceptual and Motor Skills*.
- Harrison, D.W., Walters, R.P., Williamson, J.A, Foster P. (2002). Lateralized Visual Hallucinations: Analysis of Affective Valence. [Abstract]. *Archives of Clinical Neuropsychology, 17*, 752.
- Walters, R.P. (2003). Introduction to Biological Basis of Behavior. In Lehman, Dual, Finney (eds.), *Introductory Psychology Recitation Reader* (pp 23-25). Boston: McGraw Hill.
- Walters, R.P., & Harrison, D.W. (2001). *Lateralized Visual Hallucinations: Analysis of Affective Valence*. Honors Thesis, Virginia Polytechnic Institute and State University.
- Walters, R.P., Beck, A.L., & Harrison, D.W. (2002) An Analysis of Tactile Deficits: In Patients Reporting Visual Formaesthesias. [Abstract]. *Archives of Clinical Neuropsychology, 17*, 763.

## Poster Presentations

- Beck, A.L., Mollet, G.A., Foster, P.S., Walters, R.P., & Harrison, D.W. (2003). Thalamic syndrome: Lateralized multimodal hallucinations. Poster presented at the 19<sup>th</sup> Annual Virginia Tech Graduate Research Conference.
- Foster, P.S., Mollet, G.A., Walters, R.P., & Harrison, D.W. (2004). Quantitative electroencephalographic evidence for the double dissociation of the trail making test. Poster presented at the 24<sup>th</sup> Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.
- Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Hostility as a function of right frontal lobe slow wave activity. Poster presented at the 24<sup>th</sup> Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.
- Foster, P.S., Walters, R.P., Mollet, G.A., & Harrison, D.W. (2004). Performance on the figure trail making test as a function of right frontal lobe delta activity. Poster presented at the 24<sup>th</sup> Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.
- Harrison, D.W., Walters, R.P., Williamson, J.A, Foster P., (2002, October). *Lateralized Visual Hallucinations: Analysis of Affective Valence*. Poster session presented at National Academy of Neuropsychology, Miami, Florida.
- Mollet, G.A., Harrison, D.W., Fosters, P.S., and Walters, R.P. (2004, November). *Thalamic Syndrome: Lateralized Emotional Multimodal Hallucinations*. Poster session presented at National Academy of Neuropsychology, Seattle, Washington.
- Mollet, G.A., Walters, R.P., & Harrison, D.W. (2004). Alexia without agraphia: Connecting the disconnection. Poster presented at the 20<sup>th</sup> Annual Virginia Tech Graduate Research Conference.
- Mollet, G.A., Harrison, D.W., Walters, R.P., & Foster, P.S. (2004). Thalamic syndrome: Lateralized emotional multimodal hallucinations. Poster presented at the 24<sup>th</sup> Annual Conference of the National Academy of Neuropsychology, Seattle, WA. November, 2004.
- Walters, R.P., Beck, A.L., & Harrison, D.W. (2002, October) Beck *An Analysis of Tactile Deficits: In Patients Reporting Visual Formaesthesias*. Poster session presented at National Academy of Neuropsychology, Miami, Florida.
- Walters, R.P., Beck, A.L., Harrison, D.W., & Mollet, G.A. (2003). A lack of relationship between auditory and visual hallucinations in a rehabilitative population. Poster presented at the 19<sup>th</sup> Annual Virginia Tech Graduate Research Conference.