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FRONTAL ALPHA AND BETA EEG POWER ASYMMETRY AND

IOWA GAMBLING TASK PERFORMANCE

by

RICHARD TOBY AMOSS

Under the Direction of Michael Owren

ABSTRACT

Frontal electroencephalographic (EEG) alpha (α) asymmetry may index the activation of lateralized affect and motivation systems in humans. Resting EEG activation was measured and its relationship to Iowa gambling task (IGT) performance was evaluated. No effects were found for α power asymmetry. However, beta (β) power asymmetry, an alternative measure of resting EEG activation, was associated with the number of risky decisions made in the early portion of the task. Additionally, IGT deck selection patterns suggest there are at least three distinct performance styles in healthy individuals. Interestingly, β power asymmetry contradicts performance predictions based on accepted frontal asymmetry affect and motivation models.

INDEX WORDS: Electroencephalography, Frontal asymmetry, Emotion, Motivation, Inhibition and approach systems, Iowa gambling task, Performance

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RICHARD TOBY AMOSS

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Master of Arts

in the College of Arts and Sciences

Georgia State University

2009

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2009

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IOWA GAMBLING TASK PERFORMANCE

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August 2009

I dedicate this work to my fellow graduate students, whose friendship and support have been much appreciated during my graduate study. I look forward to a future of professional interaction in our discipline and a lifetime of personal camaraderie. Most of all, I wish you health, happiness, success and fulfillment; however so you please to measure it.

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CHAPTER 1.

INTRODUCTION

The neural systems supporting emotion and decision-making have been under scrutiny for many decades in both human and non-human research. Much work on emotion has focused on subcortical and cortical structures of the limbic system, but research into higher-order control of human emotion is emerging. A growing body of human emotion research has focused on frontal lobe function, more specifically the prefrontal cortex (PFC), and asymmetries in relative levels of hemispheric activity. None would argue the importance of frontal cortex in the utilization of explicit information during mental processing. However, the contributions made implicitly through cortical emotion circuitry are thus far intertwined and inextricable components of decision-making and cognition in general. The present investigation demonstrates an application of asymmetry research to a task that mimics real-world decision making.

Asymmetry between PFC electroencephalographic (EEG) activity in the left and right hemispheres has been found to show differential associations to affective behavior, motivational behavior, and clinical outcomes. Based on such findings, resting frontal asymmetry is thought to index trait-like properties of personality with regard to affect and motivation. In the coming sections, evidence is provided supporting the veracity of separable frontal systems and how they are measured physiologically for study. A diagnostically reliable physiological measure of an individual's propensity for risk of neuropsychological dysfunction in these frontal systems would have great utility in prognoses and treatment interventions. PFC EEG asymmetry may hold value as an indicator of such risk (Coan & Allen, 2003) and would provide a relatively simple and inexpensive diagnostic option. The present investigation adopts the prevailing perspectives of asymmetry research and applies them to a commonly used neuropsychological

task, the Iowa gambling task (IGT) (Bechara, Damasio, Damasio, & Anderson, 1994). A large sample size and preferred methodological procedures were used to evaluate whether the basic tenets underlying the interpretation of frontal EEG asymmetry can be extended to the IGT. Finally, an alternative measure of EEG frontal activation asymmetry was tested in parallel, and task performance is discussed.

Frontal Cortex Asymmetry

Basic human emotional states seem to fall on a continuum from positive to negative, whereby the brain turns one affect off while concurrently turning on the other allowing for varied transitional states. Patients with bipolar disorder may have a dysfunction in the stability of trait-like baselines of emotion sensitivity. They become stuck at different times at one affective pole or the other, usually with a depressive episode following a manic episode. Research points to the left and right hemispheres of the PFC supporting separate systems that underlie risk for affective disorders such as depression and anxiety. Some evidence supports asymmetrical contributions from central locations as well (Davidson et al., 2003) but will not be addressed in the present investigation for consistency.

A positive-negative continuum is not a comprehensive view of human emotion and other dimensions are also connected to EEG asymmetry. In Harmon-Jones' work for instance (Harmon-Jones & Allen, 1998; Harmon-Jones, 2003a, b), anger is conceived to be a negative emotion which can also include an engagement characteristic, approach motivation, normally characteristic of a positive emotion. Additionally, fear, disgust and sadness are negative emotions that differ in disengagement from a stimulus, or withdrawal motivation (Coan & Allen, 2003). Furthermore, other factors playing a moderating role include an individuals' coping ability (Harmon-Jones, 2003a), arousal, affect intensity (Larson & Diener, 1987), and social or

situational influences. Perhaps for simplicity's sake the affect continuum view persists in explorations of psychiatric disorders such as depression, anxiety, and mania because of the difficulty in classifying multidimensional emotions such as anger, aggression, and disgust.

Evidence from patient studies speak to the validity of differential left-right hemisphere involvement in emotion related behavior. Heilman and Valenstein (2003) cite evidence from the Wada test whereby anesthetizing the right hemisphere produced a "euphoric-manic response" (Terzian, 1964; Rossi & Rosadini, 1967). It is thought that releasing the left hemisphere from right hemisphere inhibitory influence may have unveiled this fundamental predilection. Fried, Wilson, MacDonald, and Behnke (1998) reported laughter induced by electrical stimulation in the left superior frontal lobe that was associated with "a sensation of merriment and mirth." The intensity and duration of laughter was dependent on the stimulation current. Only a smile was evoked at low currents, while robust laughter occurred at high currents. Importantly, laughter elicited in the same manner was explained differently each time by the patient. These findings illustrated the importance of arousal and feedback in the production and interpretation of emotion in the frontal lobes. Goldstein (1948) reported that left hemisphere damage often results in anxiety, agitation, and sadness. Heilman and Valenstein (2003) further suggest that left hemisphere disease is associated with depression, particularly where the anterior perisylvian and dorsolateral prefrontal cortex (dlPFC) regions are affected. Moreover, clinical and laboratory observations suggested patients with damage to the left PFC, or left caudate projecting to the left PFC, were more likely to exhibit depression than patients with damage to other regions (Sutton & Davidson, 1997). However, House et al. (1990) did not find differences in depression between right and left anterior lesions resulting from stroke. Approached from a causal perspective, if right and left frontal hemispheres possess separate positive and negative affect systems, then

selective activation of these regions should alter emotional experience accordingly. Indeed, there is evidence that training frontal lobe activity asymmetry using reward and punishment biofeedback produced expected changes in self-report affect and facial electromyographic (EMG) behavior (Allen, Harmon-Jones & Cavender, 2001). Gur, Skolnick and Gur (1994) also found evidence of asymmetrical activation using positron emission tomography (PET). Left frontal activation was found during judgment of happy faces, while right parietal activation was found during judgment of sad faces. Although a physiological reason for the formation of left and right PFC affect systems is unclear, these psychological systems may have emerged as further specializations of evolutionarily founded frontal motivation systems.

Inhibition and Approach Systems

Eysenck (1967) suggested that two basic dimensions of personality, extraversion and emotionality, reflect two functional aspects of the nervous system. Gray (1972, 1981) also proposed two base dimensions of personality, anxiety proneness and impulsivity. Combined, extraversion/impulsivity and emotionality/anxiety proneness form the rudiments of the behavioral activation/approach system (BAS) and behavioral inhibition system (BIS), respectively. Gray (1972, 1987, 1990, 1994) maintains that individuals high in trait behavioral activation actively seek out positive and negative reinforcement. Furthermore, high trait BAS sensitivity was associated with greater general positive affect (Gable, Reis, & Elliot, 2000) and likelihood of aggressive behavior (Wingrove & Bond, 1998). An active BIS on the other hand was believed to lead to inhibited behavior, increased physiological arousal, and more attention (Gray, 1994). BIS was also associated with greater likelihood of general negative affectivity (Gable et al., 2000) and decreased aggression (Wingrove & Bond, 1998).

The opposing constructs, hence termed *BIS/BAS*, were thought to represent sensitivity differences of neural systems to environmental cues (Fowles, 1987, 1993), directing aversive or appetitive motivation. Based on animal studies, the aversive system (BIS) has its neurological underpinnings in the septohippocampal system, afferents from brainstem, and projections from the frontal lobe. This system controls the experience of anxiety (fear, frustration, and sadness), is sensitive to punishment, non-reward, and novelty, and motivates withdrawal. The appetitive system (BAS) has a less well understood neurological substrate but likely involves traditional reward pathways motivating approach. It is sensitive to reward, non-punishment, and escape from punishment and is responsible for positive feelings (elation, happiness, and hope).

Carver and White (1994) believed research on BIS/BAS sensitivity was at that time insufficient and developed new scales to capture and measure the personality dimensions. The four scales consisted of a single BIS scale, having items that aligned with anticipation of punishment, and three BAS subscales. The first subscale, *Drive*, aligned with pursuit of goals. The second subscale, *Fun Seeking*, aligned with desire for new rewards and willingness to approach potential rewards impulsively. And the third subscale, *Reward Responsiveness*, aligned with a positive response to the anticipation of reward. Checking their constructs against the Positive and Negative Affect Schedule (PANAS) of Watson, Clark, and Tellegen (1988), there were the expected relationships to BIS/BAS. *Negative Affectivity* of the PANAS was related to BIS, but not any of the three BAS scales. *Positive Affectivity* was related to all three BAS scales, but not the BIS scale. The development of these scales opened the exploration of BIS/BAS relationships to other personality variables and to physiological measures. For purposes of the present study the BIS/BAS measures were used to evaluate characteristics of our sample and for comparison to normative data.

Lateralized Electrophysiological Frontal Emotion Systems

The case for EEG asymmetry connectedness to affective science has continued to build. Davidson, Taylor, and Saron (1979) may have been the earliest to report that positive affect (e.g., happiness) resulted in significantly higher "frontal ratio" scores with a left-sided reduction in the quantity of the alpha (α) 8-13 Hz bandwidth. Conversely, negative affect (e.g., disgust) was associated with a reversal of the frontal α ratio score. Parietal asymmetry did not differentiate between positive and negative affect. Activity in the α bandwidth was used as an index of inverse cortical activity, which assumes that a brain region producing α rhythms is in a state of cortical idling. Thus, the more α appearing in the EEG of a region the less active or engaged it is. Appendix A provides a detailed explanation of this assumption. In summarizing Davidson's findings, Shaw (2003) notes that responses to emotional experiences such as films associate with frontal rather than posterior α asymmetry, a nod to the functional specificity of α in the PFC.

An alternative perspective regarding EEG indices of activity, and one found in most introductory explanations of typically defined EEG wavebands, is that the beta (β) band (14-30 Hz) serves as the indicator of aroused or activated cortical systems (Niedermeyer, 2005). Evidence has been found for the role of β in attentional mechanisms of humans, in linking spontaneous resting brain oxygen level dependent (BOLD) changes to those in EEG (Laufs et al., 2003), in facilitatory effects (Senkowski, Molholm, Gomez-Ramirez, & Foxe, 2006), and in recovery of attention function after traumatic brain injury (Keller, 2001). Differential relationships between α and β have been found for tasks involving attention, emotion, and cognition (Ray & Cole, 1985); however, these effects were limited to temporal and parietal regions rather than frontal. Nonetheless, the β band has been neglected in frontal asymmetry research and should be investigated alongside inverse the α band.

In attempt to move beyond the self-report method of studying personality dimension structure, Sutton and Davidson (1997) sought a neurobiological manifestation of BIS/BAS motivation dimensions using EEG. Collecting data on BIS/BAS (Carver & White, 1994) and the PANAS (Watson, Clark, & Tellegen, 1988), as well as resting eyes-open (EO) and eyes-closed (EC) EEG, they found that baseline prefrontal EEG asymmetry (frontal ratio) score was correlated with BAS-BIS difference score. Greater right-side activity associated with higher BIS scores, while greater left-side activity associated with higher BAS scores. Additionally, prefrontal asymmetry better predicted the BIS/BAS constructs than those of the PANAS. Harmon-Jones and Allen (1997) also looked at BIS/BAS and α asymmetry and found that greater left frontal activity was associated with BAS, however, support for right hemisphere activity associating with BIS was not found. A question that remained unanswered was whether resting asymmetry can be used to predict how an individual utilizes affect-related motivational information to guide their actions.

Iowa Gambling Task

The IGT has characteristics that should differentially engage lateralized affect and motivation systems. It exploits exploratory learning through delivery of different schedules of reward and punishment of separate card decks in order to approximate a real-life decision-making situation. An individual must evaluate gains and losses experienced while selecting from different decks, and presumably use somatic emotional information to surmise and then maintain an advantageous strategy. The IGT was originally performed manually (Bechara et al., 1994), however, it is currently delivered using computer interface to allow greater experimental control, user consistency, and collection of electrodermal activity (EDA), a measure of autonomic arousal presumed to reflect somatic information. Four decks of cards labeled A, B, C, and D are

shown on-screen during the task, along with the cumulative amount of money loaned to a participant, and the amount of money accumulated by the participant as choices from different decks are made. Each time a participant makes a selection in the task, a varying amount of virtual money in U.S. dollars is immediately awarded, and may be followed by a penalty. Two general types of decks are defined for the task. The first, *bad* (or risky) decks (A and B), have high immediate monetary award (*M* = 125, *SD* = 22.2; range $80-$170) with a higher frequency or weight of subsequent penalty (Deck A: *M* = 187.5, *SD* = 124.4; range $0-$350; Deck B: *M* = 187.5, *SD* = 583.4; range $0-$2500), thus a lower net reward. The second, *good* (safe) decks (C and D), have low immediate award (*M* = 62.5, *SD* = 13.9; range $40-$95) with lower frequency or weight of penalty (Deck D: *M* = 31.3, *SD* = 95.5; range $0-$375; Deck C: *M* = 31.3, *SD* = 25.1, range $0-75), thus higher net reward. The reward and punishment elements of the IGT are designed to drive emotional experience to facilitate decision-making. The positive affect elicited by reward and negative affect due to punishment should act on lateralized frontal affect and motivation systems which are thought to be differentially sensitive to such information.

Over the course of the 100 selections of the IGT, control participants exhibit a shift toward predominantly selecting from the good decks, which represents a rational decision-making progression. However, Bechara and colleagues have found that patients with damage to the ventromedial prefrontal cortex (vmPFC) responded irrationally over the course of the task. They continued to select from bad decks, even when they were able to explicitly state differences in contingences and the most advantageous long-term strategy. Thus, a disconnection exists between explicit knowledge being acquired and overt behavior in these patients. The EDA of control participants in anticipation of selecting from bad decks was greater than that of patients with vmPFC damage, who showed low and equal anticipatory EDA across all decks. This

finding suggested these patients had impairment in the ability to develop a somatic warning signal (Bechara et al., 1994; Damasio, 1996). Furthermore, when control participants were divided into high and low anticipatory EDA groups, low EDA participants picked good decks significantly fewer times, suggesting reduced activity of a somatic warning system may also contribute to poor performance. Patients may not be able to develop implicit expectations for future consequences without the somatic signal and are insensitive to the emotional component of punishment. However, patients did show similar EDA to control participants following reinforcement (awarding of gains), which indicated that reward pathways were likely intact. Thus, an alternative explanation is that vmPFC patients may not be able to override the attractiveness of the higher immediate reward of bad decks regardless of a net loss.

Bechara, Tranel, Damasio, and Damasio (1996) describe that in the beginning period of the IGT participants have no way of knowing the contingencies of each deck and must develop a "hunch" which instructs control participants to start avoiding bad decks after 20-25 selections. The hunch concept led Bechara, Damasio, Tranel, and Damasio (1997) to add descriptive nomenclature to four periods of the IGT. "Pre-punishment" refers to the early period of the task when participants sampled decks without punishment. By the 20th selection, the "pre-hunch" period refers to the point when a few losses have been delivered and control participants began to show anticipatory EDA. The "hunch" period, achieved by the 50th selection, marked participants' suspicion that the bad decks were riskier and control participants showed anticipatory EDA to them. The final period is "conceptual," achieved by selection 80, in which most participants could explain which decks were bad and good and why. In general, control participants who could not explain still performed advantageously. Interestingly, PFC patients that did reach the conceptual level continued to perform disadvantageously and did not develop

anticipatory EDA. While the divisions make intuitive sense, they represent an idealized progression through the IGT that might be expected only of high performing participants. Participants may actually vary considerably in moving from one stage to the next. Nevertheless, they help to conceptualize the decision-making processes in the present investigation.

Key Study

Schutter, de Hann, and van Honk (2004) conducted a study to see if frontal asymmetry correlated to IGT performance. Their result challenged expectations based on the affect and motivation models. Resting activity asymmetry was believed to index trait-like affect and motivation biases of an individual that would be reflected in IGT performance style. Participants with left hemisphere activity dominance should show more risky performance than those with right hemisphere dominance, who should demonstrate safer performance. The authors measured baseline frontal EEG α asymmetry and correlated it to the percentage of risky choices during the IGT. Unexpectedly, they found that participants with dominant right PFC activity made more risky choices, an unsuccessful strategy in the IGT. However, no left dominant participants were reported in the study for comparison. Additional considerations include the small sample size and the use of a single reference electrode behind the right ear that may have contributed to the contrary findings. Because Schutter et al.'s findings oppose the predominant view in the literature, and because α asymmetry findings have likely oversimplified the interpretation of frontal cortex brain system activity, their results beg further controlled validation.

Summary and Goals

The circuitry involved in human emotion is complex and interrelated (Davidson, 2000), and as put forth, two basic frontal circuits are proposed to mediate different colors of emotion. Furthermore, the emotion circuits may overlap two frontal circuits proposed to mediate

motivation. The approach circuit guides appetitive behavior, motivating movement toward a desirable goal and is associated with positive affect, while the withdrawal circuit motivates movement away from a stimulus and is associated with negative affect. Patients with vmPFC damage that disrupts these circuits have impaired decision-making in learning patterns of reward and punishment, as demonstrated by the IGT. The question remains whether this dysfunction is due to an inability to track explicit elements of the task, to reduced attention to covert emotion signals, or to changes in motivation resulting from damage to lateralized systems.

Decision-making relies on a cost-benefit analysis that is associated with expectations about outcomes. The extent to which this cost-benefit analysis is performed consciously, using explicit processes, and through implicit processes is at the heart of an ongoing debate. Damasio's (1996) somatic marker hypothesis (SMH) holds that "gut feeling" biases influence human decision-making implicitly. That is, emotional and visceral associations coupled to a response option, acquired through experience, activate circuitry that covertly motivates a behavioral response. The IGT is proposed to involve emotion-based decision-making. Trait-like levels of activation that index lateralized affect or motivation systems may be obtained from frontal EEG and may reflect the sensitivities participants' have to IGT reward and punishment.

According to the frontal EEG asymmetry models, it would be expected that dominant right frontal cortex activity is associated with negative affect, withdrawal motivation, and punishment information, while dominant left frontal cortex activity is associated with positive affect, approach motivation, and reward information. The reward and punishment aspects of the IGT should act upon these asymmetrical predispositions in a predictable manner. Schutter, et al. (2004) unexpectedly found baseline α asymmetry was related to IGT performance in a manner reverse to predictions, such that right hemisphere activity dominance correlated with risky

performance rather than safe performance. The lack of left hemisphere dominant participants in their sample precludes speculation about left frontal activity and reward sensitivity involvement. Because of these findings and methodological considerations mentioned above, further investigation is required. The present study tests the predictive utility of frontal EEG asymmetry on IGT performance to address whether relatively greater left or right resting frontal activity associates with more risky and disadvantageous decision-making.

Evidence from the research presented thus far supports the idea that resting trait α asymmetry reflects emotional sensitivities to reward and punishment in such a way as to be related to IGT performance. Right hemisphere (relative to left hemisphere) activity dominance should be associated with better IGT performance because right hemisphere dominance is associated with greater withdrawal motivation due to greater punishment sensitivity. In short, task contingencies would be learned more quickly as disadvantageous choices are avoided to a greater extent. Conversely, poorer performance was expected for individuals with relatively greater left hemisphere activity dominance. Left hemisphere dominance should index greater approach propensity, perhaps reinforced by the positive experience of immediate reward, which are higher in the disadvantageous decks. Alternatively, left dominance should indicate less sensitivity to punishment than right dominant individuals. Thus, the first goal of the study was to assess frontal α asymmetry's ability to predict performance on the IGT. This will enable the evaluation of the contradictory findings of Schutter et al. (2004).

The critical quantitative distinction between α and β EEG activity is frequency, while the critical qualitative distinction lay in the characteristics of each waveband regarding what the brain is doing. Fundamentally, it can be argued that the living brain is never in an inactive state, and α activity represents a stage of alert consciousness. This relaxed but wakeful state allows for

information from the environment to pass through thalamic "gates" that help filter unimportant stimuli and maintain vigil for salient stimuli. It is likely that a large contribution from subcortical thalamic nuclei, particularly the nucleus reticularis thalami (nRt), contributes to the high amplitude and the frequency regularity of α in cortical regions (see Appendix A for further discussion). In addition to monitoring the sensory world, α activity may include self-reflective and evaluative processing. However, when processing becomes focused the β state emerges. This is comprised of low amplitude, faster frequency, desynchronized activity appearing when the cortex is released from the idling α state to engage in the task at hand, such as attending to a math problem. Hence β activity is more in line with the idea of "activation" of a brain region than α activity. The α band seems to have enjoyed a boon in research favor over the years because it is easy to identify and track. However, using α to determine baseline activity asymmetry requires inferences be made upon an inverse relationship to behavior. Thus β (as well as higher frequency wavebands) may be more appropriate for study of activation asymmetry.

Because β activity is generally held to be the bandwidth associated with mental engagement (Niedermeyer, 2005), it was examined as a secondary measure of cortical activation. If, as indicated by many asymmetry studies, α power (a measure of the quantity of a waveband) indicates the degree to which neural tissue is in an idling mode, a direct measure of resting system engagement via β power dominance may be more appropriate. Greater β power in the right hemisphere (right hemisphere dominance) should be associated with better performance, and greater β power in the left hemisphere with poorer performance. Consequently, α and β power asymmetry were entered simultaneously into regressions on performance measures in order to refine the interpretation of activity dominance, the second goal of the study.

CHAPTER 2.

METHOD

Participants

Seventy-four participants were non-selectively recruited from Georgia State University's online Sona experiment sign-up system. Nine participants were excluded from the analyses because of tendency toward ambidexterity or left-handedness indicated by an Edinburgh Handedness Quotient (Oldfield, 1971) less than 40. Two more were excluded because of missing data. Eight were excluded due to psychiatric diagnoses of depression, anxiety, or ADHD, most of which was accompanied by pharmacological or psychotherapy intervention. Four of these eight were right-handed. The remaining 59 participants (19 males), 18-40 years old (M_{age} = 20.4, SD = 3.4), were right-handed, free from neurological or psychiatric disorders, and taking no medication known to affect brain activity. The resulting sample consisted of 37% Caucasian, 37% African-American, 14% Asian or Pacific Islander, 7% Hispanic, and 5% other races. Participants received credit toward their class research participation requirement.

Procedure

Participation in the experiment involved a one-time commitment of approximately two hours. The participants were greeted in the lobby of the psychology department and escorted to the testing laboratory by the experimenter. The experimenter gave a brief summary of the experimental session and introduced the participant to the equipment involved. After signing informed consent, participants completed preliminary questionnaires including a neurological and psychiatric history, and the Edinburgh Handedness Inventory. The BIS/BAS and PANAS-X (Watson & Clark, 1999) questionnaires were then be presented for completion in counterbalanced order between participants.

The psychophysiological recording portion of the experiment involved participant testing in a sound attenuated room seated upright in a comfortable chair. A cap containing the EEG electrodes was fitted onto the participant's head and an ear-clip reference electrode was attached to each ear lobe. Due to equipment limitations, linked ears rather than a computed average mastoids reference was employed in the present study similar to Sutton and Davidson (1997). Individual electrodes were placed above and below the right eye to monitor vertical eye movements, and at the outer canthus of each eye to monitor horizontal eye movements. Communication with participants was maintained using a two-way intercom and video monitor. Participants were instructed to sit quietly looking toward a fixation crosshair, but to remain relaxed and awake while resting EEG was obtained. Eyes-open (O) and eyes-closed (C) EEG data were recorded using the counterbalanced sequences of OOCOCOCC or CCOCOCOO as employed in Allen et al. (2001) and Harmon-Jones (2003a), to obtain eight one-minute epochs. Upon completion of the resting measurements, the experimenter entered the testing room and gave instructions for the IGT. Participants were asked to verify that they had not seen the task before and understood what to do in the task. They were not given any instruction beyond that outlined in the standardized IGT instruction set. Participants then completed the computerized version of the IGT while EEG was simultaneously recorded. A modification was made to the default IGT to set a single sound for gains and a single sound for losses rather than multiple sounds for each. This was done to simplify interpretation of brain activity obtained during these periods for future investigation. Once the participant completed the IGT, the EEG cap and other electrodes were removed. Participants were then debriefed, asked not to share information about the experiment with others, and escorted back to the lobby of the psychology department. All

elements of the procedure were conducted in accordance with GSU Institutional Review Board specifications.

Instrumentation

EEG signals were recorded using 10 Biopac (BIOPAC Systems, Inc., Goleta, CA) EEG100C amplifiers (Gain 10,000; High Pass 0.1 Hz, Low Pass 35 Hz) configured for unipolar recording. An Electrocap International (Eaton, OH) EEG cap utilized electrode sites F3, F4 (midfrontal); F7, F8 (lateral-frontal), C3, C4 (central); T3, T4 (anterior-temporal); and Fz, Cz (anterior midline), with a linked ears reference (A1+A2). Impedance values were kept below 10 kΩ and within 5 kΩ between homologous sites. Horizontal and vertical electrooculograms (EOGs) were acquired using 9 mm tin electrodes and two Biopac EOG100C amplifiers (Gain 5,000; High Pass 0.05 Hz). Impedance values were measured using a UFI (Morro Bay, CA) impedance meter. Delivery of the IGT was performed by a dedicated PC (MS Windows XP) with a 17 inch CRT monitor. The selection of cards from the four decks delivered 5 V, 1 ms pulses via transistor-transistor logic (TTL) through a modified parallel cable to digital channels of the Biopac MP150 digitizing unit to be recorded in parallel with the EEG and EOG. All signals were integrated into a single file digitized at 1 kHz using AcqKnowledge (ver. 3.9.1) software on a Mac G4 (OS 10.4) computer.

Signal Processing

The EEG for each of the eight one-minute resting acquisitions was filtered online to remove ambient 60 Hz artifact present in the recording chamber. Filtered waveforms were visually inspected for eye-artifact contamination; and if excessive was corrected using an independent components analysis (ICA) procedure incorporated into the AcqKnowledge software. When movement artifact was evident during acquisition the experimenter collected

more than one minute of resting data, and the contaminated section was manually removed from the EEG record returning the length to one minute.

Electrode sites F3 and F4 were then subjected to separate power spectral density (PSD) procedures for each of the eight minute epochs of baseline resting frontal EEG. PSD values corresponding to the α frequency range (8-13 Hz) were averaged for each minute separately. The values for all eight minutes were then averaged to obtain a stable α power for the site. Finally, the α power values for F3 (left frontal) and F4 (right frontal) were natural log transformed such that an asymmetry score comparing right hemisphere (RH) to left hemisphere (LH) was computed lnALPHA = (ln[RH] - ln[LH]). A positive value indicates relatively greater α in the right hemisphere and thus greater left hemisphere activity. Conversely, a negative value indicates greater activity in the right hemisphere.

In a similar manner, PSD values corresponding to the β frequency range (14-30 Hz) were averaged for each minute separately. The values for all eight minutes from F3 and F4 were averaged and natural log transformed to compute lnBETA = (ln[RH] - ln[LH]). A positive value indicates relatively greater β and greater activation in the right hemisphere. For lnBETA, a negative value indicates greater activity in the left hemisphere.

Analyses

Regression analyses were conducted to assess relationships between lnALPHA and lnBETA to four IGT performance variables. These were net score (money won or lost), percentage of risky deck choices over 100 selections, percentage of risky deck choices during the first 50 selections, and percentage of risky deck choices during the last 50 selections. The 50 selection midpoint was used to allow the pre-punishment to pre-hunch periods to be examined apart from the hunch to conceptual periods of the IGT defined based on Bechara et al. (1997).

Examining the first and last 50 selections separately allows for translation of the present data to the *decision under ambiguity* and *decision under risk* portions of the IGT defined by Brand, Recknor, Grabenhorst, and Bechara (2007). Molecular variation in performance was evaluated using qualitative examination, while correlation and regression were performed on personality and demographic variables to evaluate sample characteristics.

CHAPTER 3.

RESULTS

Regressions of Asymmetries and IGT Performance Measures

The regression result for each of the four IGT performance measures was examined in relation to natural-log-adjusteded α and β power asymmetry scores was examined. Asymmetry scores ranged from -.21 to .25 for lnALPHA (M = .02, SD = .11) and from -.28 to .61 for lnBETA (M = .06, SD = .20). The following analyses unveil whether each type of performance varies as a function of asymmetrical activation. Significant regression Beta coefficients within the four regression models indicate which waveband was an important factor.

Resting "trait" lnALPHA was expected to be predictive of IGT net score based on the affect and motivation models. Additionally, lnBETA was believed to serve as an additional waveband representative of activity that behaves complementarily to lnALPHA. The prediction that right hemisphere dominant asymmetry would be associated with higher net monetary gain than left hemisphere dominant asymmetry scores was not supported by either waveband's asymmetry score. When IGT net score was regressed on frontal lnALPHA and lnBETA scores, no significant association was found (R^2 = .03, F(2, 56) = .73, p = .49). Neither lnALPHA (β = .07, p = .65) nor lnBETA (β = -.17, p = .23) demonstrated significant effects. See Figures 1 and 2.

Regression of percentage of risky choices made during the entire IGT on lnALPHA and lnBETA did not reveal significance (R^2 = .04, F(2, 56) = 1.03, p = .37). Neither lnALPHA (β = -.09, p = .52) nor lnBETA (β = .20, p = .16) asymmetry demonstrated significant associations. See Figures 3 and 4.

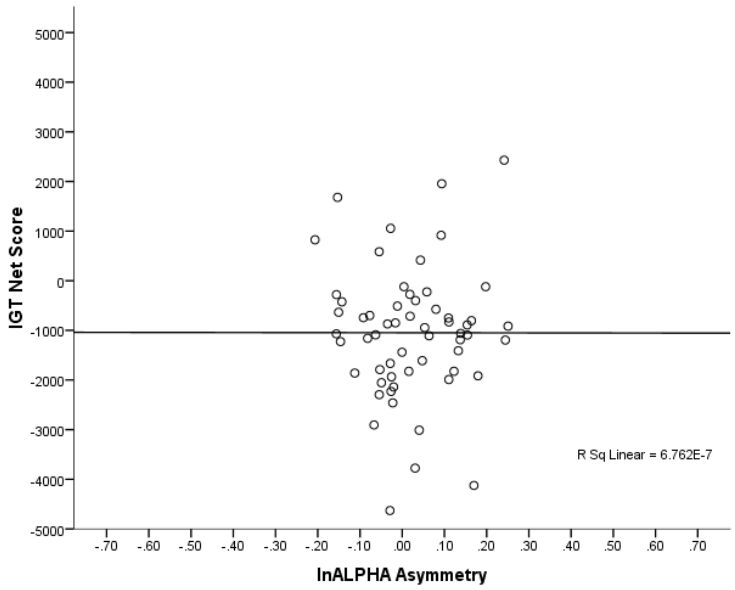

Figure 1. IGT net score was not predicted by lnALPHA

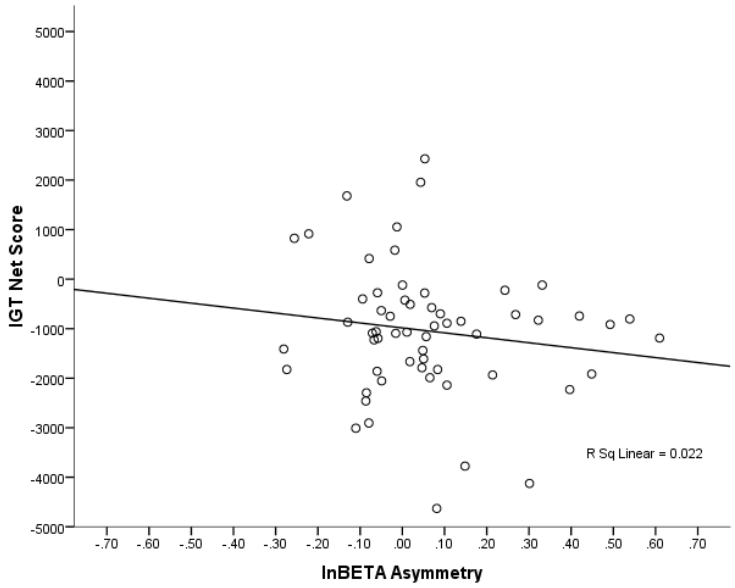

Figure 2. IGT net score was not predicted by lnBETA

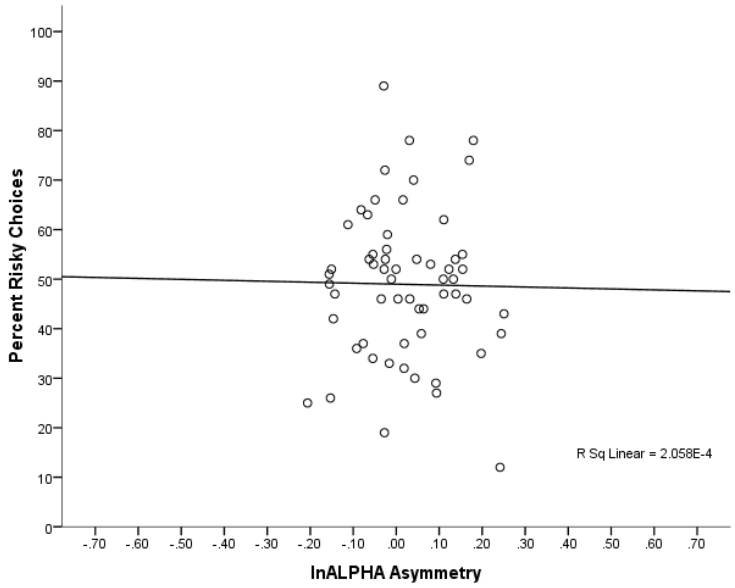

Figure 3. lnALPHA did not predict percentage of risky choices over 100 selections

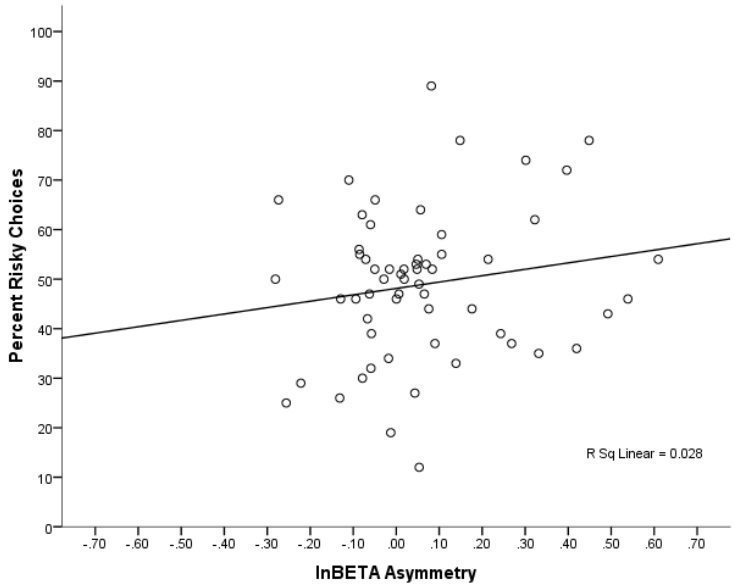

Figure 4. lnBETA did not predict percentage of risky choices over 100 selections

Next, regression examined the relationship of percentage of risky choices made during the first 50 selections to lnALPHA and lnBETA. The regression approached significance (R^2 = .08, $F(2, 56) = 2.55$, $p = .09$). While lnALPHA was not significantly associated (β = -.21, p = .14) with risky choices during early selections, lnBETA did demonstrate a significant

contribution (β = .30, $p < .04$). This suggests that lnBETA right hemisphere dominance is associated with risky choices during decisions under ambiguity. See Figures 5 and 6.

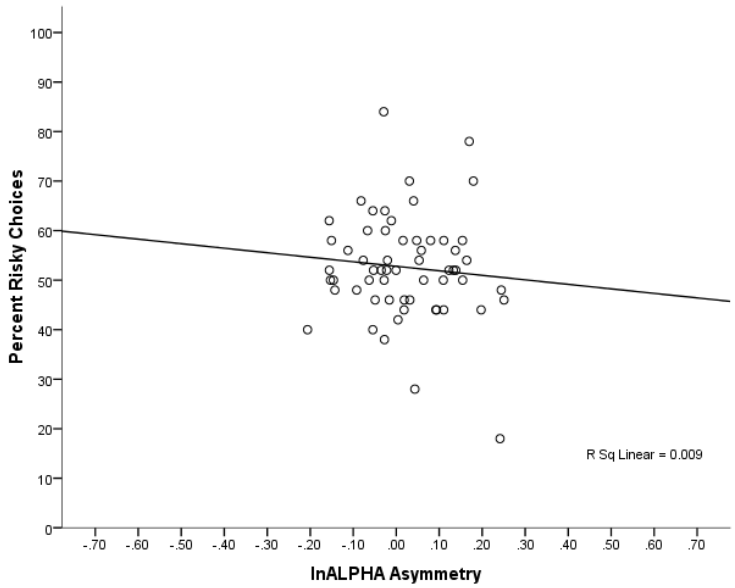

Figure 5. lnALPHA did not predict percentage of risky choices during the first 50 selections

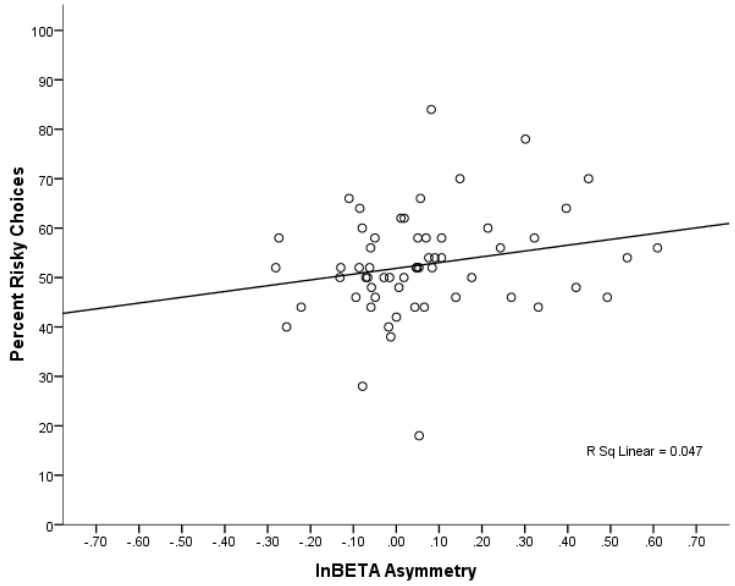

Figure 6. lnBETA contributes significantly to prediction of percentage of risky choices during the first 50 selections

Finally, the percentage of risky choices made during the last 50 selections was regressed on lnALPHA and lnBETA and returned non-significant ($R^2 = .02$, $F(2, 56) = .47$, p = .63).

Neither lnALPHA (β = -.03, *p* = .86) nor lnBETA (β = .14, *p* = .35) asymmetry demonstrated significant associations. See Figures 7 and 8.

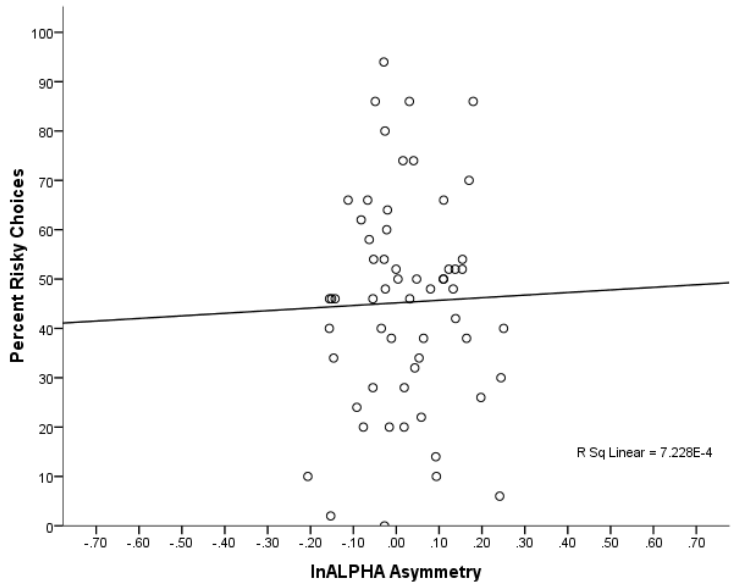

Figure 7. lnALPHA did not predict of percentage of risky choices during the last 50 selections

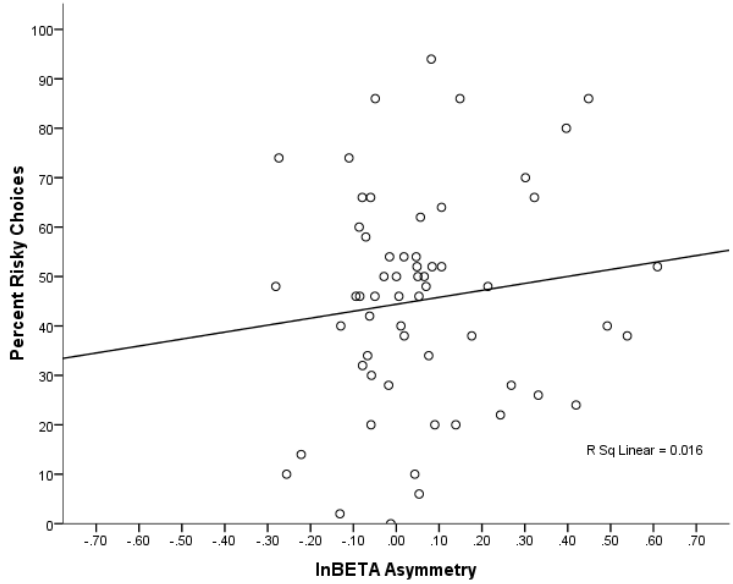

Figure 8. lnBETA did not predict percentage of risky choices during the last 50 selections

IGT Performance Styles

To evaluate and compare the present sample's performance on the IGT with that of previous research some comparable measures were derived. First, the average number of

selections made for each deck over the course of 100 selections is shown in Figure 9 for comparison to Bechara et al. (1994) and Caroselli, Hiscock, Scheibel, and Ingram (2006). Unlike the Bechara findings of a prominence in selections made from both advantageous decks (C and D) during the IGT, the present investigation showed selection preference for an advantageous deck (D) and disadvantageous deck (B). The present sample resembled Caroselli et al., yet did not display definitive evidence of the "special attraction of deck B" or the "prominent deck B phenomenon" reported by Lin, Chiu, Lee, and Hsieh (2007) and Lin, Chiu, Cheng, and Hsieh (2008). Instead, a repeated measures ANOVA revealed an effect for deck preference, Wilks's $\Lambda = .43$, $F(3, 56) = 24.58$, $p < .001$, such that both decks B and D were selected from significantly more times than decks A and C, $F(3, 174) = 15.44$, $p < .001$. Strangely, the pattern of deck preference values was similar to that of cocaine-dependent patients reported in Monterosso, Ehrman, Napier, O'Brien, and Childress (2001). Second, Figure 10 displays the mean difference in the number of advantageous selections and disadvantageous selections (C+D) - (A+B) for five, 20-draw periods during the IGT for comparison to control participant data from Bechara and Damasio (2002), Turnbull, Berry, and Bowman (2003), Bechara (2004), Whitlow et al. (2004), Bowman, Evans, and Turnbull (2005), Caroselli et al. (2006), Brand et al. (2007), and Lawrence, Jollant, O'Daly, Zelaya, and Phillips (2008). Positive numbers indicate preference for safe decks and negative numbers for risky decks. The present sample appears to be in line with the classic view that healthy control participants' learn to shift away from bad decks and predominantly draw from safe decks as the IGT progresses.

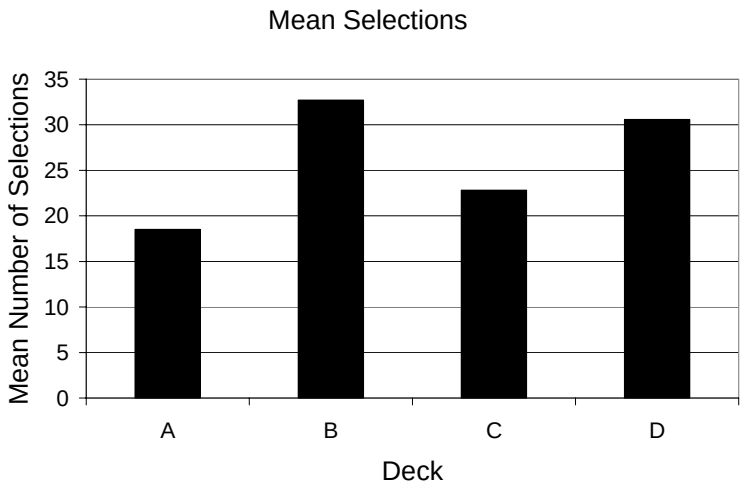

Figure 9. Mean selections made from each deck over the course of the IGT

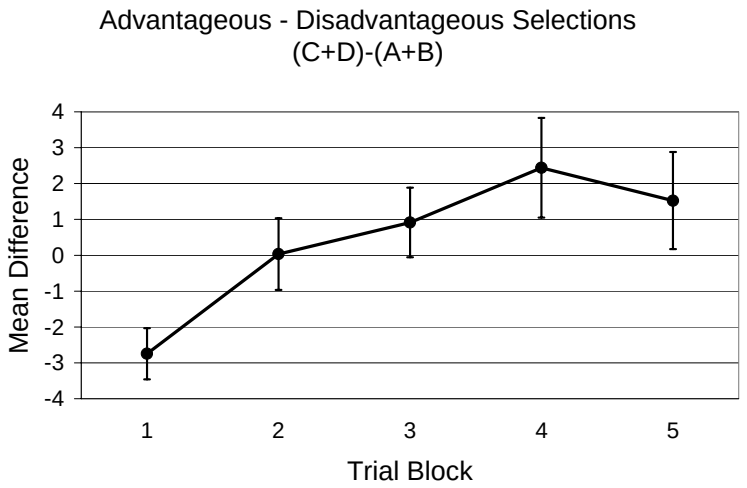

Figure 10. Difference between good and bad deck selections plotted in 5, 20-draw selection bins

Although the average number of safe selections made by participants increased over the course of the task, indicating successful learning of the deck contingencies and a shift toward good decks, it was of interest to explore *how* participants perform. If all participants perform the IGT in the same way, the average learning curve might be expected to have a more dramatic and perpetuated slope toward safe selecting as all enter the hunch period. (Compare Figure 10 to Bowman et al (2005) and Turnbull et al. (2003) to illustrate similarity and dissimilarity of

learning curve.) If differences in perception of the IGT exist, they may manifest in particular styles of responding. In order to statistically examine the responding style of our participants, a median split of the performance variable percent risky choices made during the entire IGT was used to differentiate groups of high and low performers. Conveniently, the median fell at 50% safe choices. Thus, the participants making 50% or more risky choices overall became the low performing group, and those making fewer than 50% risky choices overall became the high performing group.

Using the low performer subgroup, lnALPHA and lnBETA relationships to IGT performance measures (i.e. IGT net score, percent risky choices during the first 50 selections, and during the last 50 selections) was reexamined. No relationship was found for IGT net score or percent risky choices during the last 50 selections. However, lnBETA demonstrated a regression Beta coefficient that approached significance ($\beta = -.36$, $p < .07$) for percent risky choices during the first 50 selections. This suggested that for low performers, right hemisphere activity dominance may be associated with risky selections early in the IGT during the period of decisions under ambiguity.

Using the high performer subgroup, both lnALPHA and lnBETA demonstrated regression Beta coefficients that approached significance ($\beta = -.36$, $p = .08$; $\beta = .37$, $p < .08$) for percent risky choices during the first 50 selections. No relationship was found to percent risky choices during the last 50 selections. Right hemisphere activity dominance measured by α or β bandwidths may contribute to, or reflect equally on the processes of, some mechanism of high performer early-task risk behavior. As was also seen for lnBETA's association with low performers' and all participants' risky choices made during the first 50 selections, the direction of high performers' association suggests right hemisphere dominance yields poorer performance.

These findings are at odds with the hypothesis of the present investigation, but in line with Schutter et al. (2004). Curiously, lnBETA for high performers demonstrated a closer association to IGT net score than in previous regressions ($\beta = -.35$, $p = .10$).

Further exploration of responding style revealed that three groups rather than the two determined by median split may more accurately describe performance data. Fearing averages could obscure dynamics of performance, participants were parceled into low, middle, and high performing response groups. Figure 11 shows the average number of safe selections made during 5-draw selection bins over the course of the task along with possible response strategies. High performers (n = 16), making 61 or more safe selections overall, demonstrated a dramatic increase in safe selecting from beginning to end of the task. Middle performers, making between 40 and 60 safe selections overall, were the largest group (n = 31) and maintained approximate chance level of performance from beginning to end of the task, mirroring the average number of safe selections. Finally, low performers (n = 12), making 39 or fewer safe selections overall, expressed a steady decline in safe selecting over the course of the task. This suggests that there are at least three distinct strategies that might be employed by healthy individuals during the task, one of which is particularly detrimental to success.

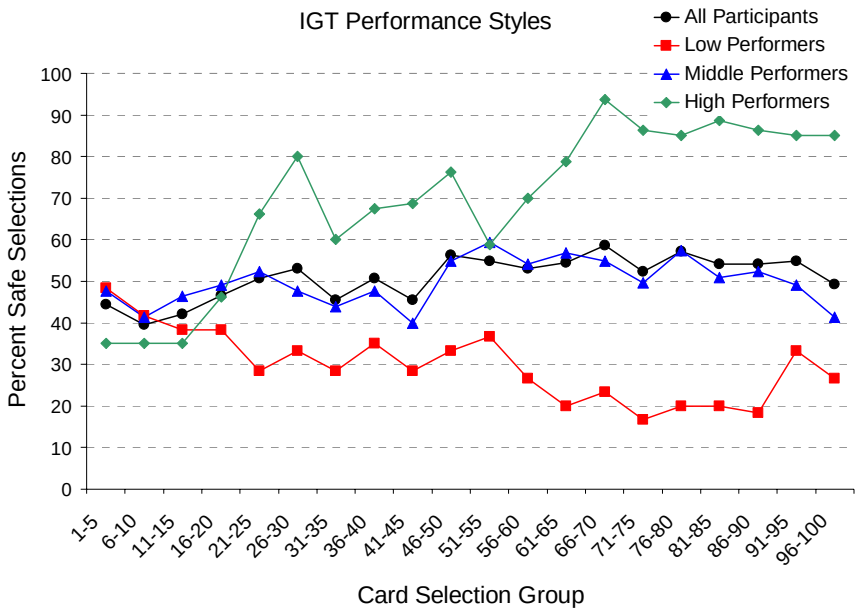

Figure 11. Composite graph of overall average, high, middle and low performing participants' selection styles

Additional Effects and Correlations

Unlike findings of Brand et al. (2007) and Bolla, et al. (2004) who report higher IGT performance in males, sex of participant was not associated with IGT net score, percent risky choices made during the entire task, or during the first or last 50 selections; nor was sex associated with α or β frontal asymmetry. However, sex was significantly correlated with BIS ($r = .27$; $p < .05$), where females reported more ($M = 19.6$; $SD = 3.6$) inhibition than males ($M = 17.2$; $SD = 4.8$). Sex was also significantly correlated with the Shyness subscale of the PANAS-X ($r = -.37$; $p < .01$), indicating females were less shy ($M = 6.4$; $SD = 2.1$) than males ($M = 8.4$; $SD = 2.6$). No other significant correlations to sex were found, however correlations between sex and the *guilt* and *sadness* subscales of the PANAS-X approached significance, with females reporting less of each than males.

The Edinburgh Handedness Quotient computed from the self-report inventory was significantly correlated with lnBETA ($r = .27$; $p < .05$) but not lnALPHA or other study

variables. The degree of right-handedness correlated with greater right-hemisphere lnBETA activity dominance in our group of right-handed individuals. See Figure 12. It should be noted that including ambidextrous and sinistral participants' data into the correlation, removes the significant association. Thus, this effect applies only to dextral individuals. It is possible that this curious finding is the result of dorsolateral premotor and motor system activity during rest and use-dependency effects are a tempting inference. However, verification of this lnBETA lateralization requires further investigation of a large sample of individuals displaying many variations of handedness and physical activity biases.

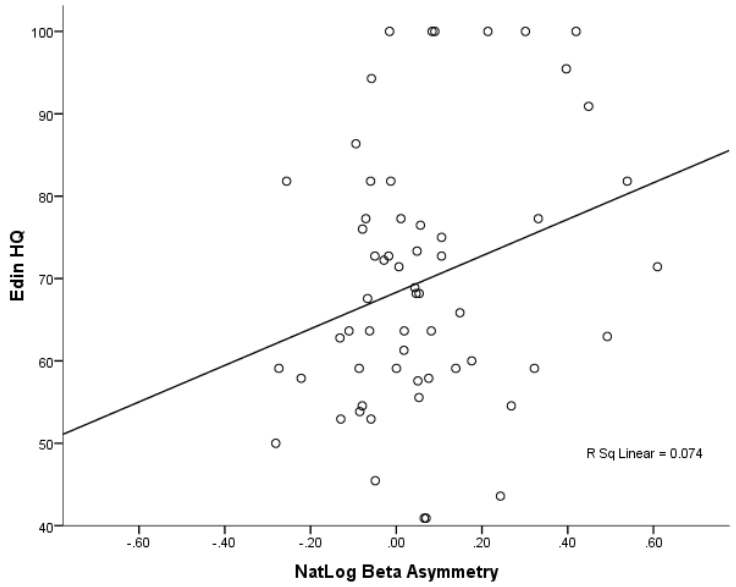

Figure 12. In right-handed participants, lnBETA right hemisphere activity dominance was positively associated with extent of right-handedness

Values obtained for BIS and for the three BAS subscales in the present sample are consistent with those found in the asymmetry study of Harmon-Jones and Allen (1997), which used female control participants, and in the original Carver and White (1994) study, which used male and female control participants. See Appendix B, Table 1. PANAS-X primary positive and negative affect scales were distributed in kind with Watson et al. (1988, 1994) and significantly

correlated with all 11 specific affect subscales in the expected direction. Additionally, *fatigue* and *guilt* subscales also significantly negatively correlated with positive affect. These subscales may be particularly sensitive indicators of an individual's general mood. Further PANAS-X significant intercorrelations are listed in Tables 2 through 5 of Appendix B. The significant correlation between BIS and PANAS negative affect found in Carver and White, then again in Harmon-Jones and Allen's study, was also supported in our data. However, unlike their studies, significant correlations between BAS subscales and PANAS positive affect were not found.

Interestingly, when the primary positive and negative affect scales of the PANAS-X are regressed on lnALPHA and lnBETA, significant regression Beta coefficients suggest lnBETA relates to both affect measures while lnALPHA does not. For positive affect, lnBETA ($\beta = -.32$, $p < .05$) dominance in the right hemisphere associates with lower positive affect and in the left hemisphere with higher positive affect. For negative affect, lnBETA ($\beta = .31$, $p < .05$) dominance in the right hemisphere associates with higher negative affect and in the left hemisphere with lower negative affect. The direction of these associations are in keeping with the original view of frontal activity asymmetry, where the right and left hemispheres regulate negative- and positive-valence emotional circuits, respectively.

CHAPTER 4.

DISCUSSION

Frontal EEG asymmetry has been shown to behave in a trait-like manner with regard to affect and motivation systems in humans. In the present study, inverse α power, commonly used as a measure of resting activity asymmetry, demonstrated no appreciable utility toward predicting IGT performance. However, β power, an alternative measure of resting brain activity asymmetry did display a significant association to risky decisions made during the first 50 selections. When looking at high and low performer subgroups determined using median split of the percent of risky choices on entire IGT, lnBETA approached significance with respect to the decisions under ambiguity (pre-punishment to pre-hunch) period of the IGT. The asymmetry direction indicated that right hemisphere activity dominant individuals make more risky decisions than left hemisphere dominant individuals. This is inconsistent with expectations based on the affect and motivation models and consistent with the finding of Schutter et al. (2004). The results beg the question whether performance in the early portion of the task, as opposed to the late, is somewhat contingent on resting frontal β activity variation, which may also have ties to attentional mechanisms.

Methodological considerations such as linked ears recording reference, the resting condition itself, and specific EEG recording locations are potential sources of complication. For an in depth evaluation of asymmetry methodology refer to Hagemann, Naumann, Becker, Maier, and Barktussek (1998), Coan and Allen (2003, 2004) and Allen, Coan, and Nazarian (2004). Our use of linked ears was less favorable than the recommended computationally averaged ear or mastoids references. However, given that the earliest data on frontal asymmetry effects were found using linked ears, it is not expected to be the critical asymmetry determinant when applied

with diligence. Since the living brain is never in a wholly inactive state, questions remain about the use of resting activity as an appropriate experimental baseline. Despite being asked to relax and refrain from active thought, effects of mental self-projection (Buckner & Carroll, 2006), adaptive downtime, silent speech, and simulation behaviors are likely to occur during rest recordings. Because of such factors the resting state baseline has come under increasing scrutiny in recent years as functional neuroimaging literature booms. It is likely that the utility of frontal activation measures will lie in quantifications made during conditions exploiting careful experimental manipulation rather than through comparisons with, or subtractions from, activation due to unfocussed spontaneous brain activity. With regard to frontal asymmetry recording locations, Davidson (2004) points out that the orbitofrontal cortex (OFC), which is closely associated with emotion, is an area distant from EEG surface electrodes. Furthermore, the dlPFC underlying the electrodes selected for asymmetry computation is primarily associated with cognitive abilities. Hence, the EEG resting state of these functions is more likely to have been what was recorded. Davidson's evaluation makes it seem difficult to imagine the utility of resting frontal EEG asymmetry in characterizing emotion. Yet it also leaves us with a lingering curiosity about what else is going on that could explain previous emotion and motivation findings related to asymmetry. Since β asymmetry shows predictive utility for the early portion of the IGT and its waveband is associated with cortical activation, perhaps its effects have more to do with a participant's task engagement readiness than affective stance. Although task engagement preparedness and motivation are confounded, each involving attention to the task at hand, presumably engagement would be a positive experience motivating approach tendency.

The power values extracted for α and β wavebands which are used in determining the separate asymmetry measures could present a statistical problem known as collinearity in which

overlapping variances may obscure regression model effects. The two wavebands are likely to be somewhat positively correlated because, as mentioned before, they are both states of living brain activity which are also side-by-side in the EEG frequency spectrum. However, they are conceptually independent of each other given their relationships to different types of mental activity. To evaluate collinearity, first, a bivariate correlation was computed between the asymmetry measures (lnALPHA and lnBETA). The asymmetry measures were significantly correlated $r = .38$, $p < .05$ amongst the 59 participants included. This makes sense given that power is derived from the collective amplitude of a particular waveband. Thus, individuals with larger power values in the α range likely have larger power values in other wavebands, simply due to larger overall emittance of voltage through the scalp. Tabachnick and Fidell (2001) recommend considering removal of a collinear variable from a regression if the correlation is around .70. They further that statistical problems arise at correlations of .90 and above. Multicolinearity statistics were computed for α and β power asymmetry values in the regressions as well, which revealed a tolerance value of .86 and a variance inflation factor (VIF) of 1.17. The tolerance measure, which ranges from 0 to 1, indicates the amount of variance that cannot be explained by other regression predictor variables. In the present study, tolerance is well over the .10 limit of concern, thus the majority of variance cannot be explained by another predictor (i.e. asymmetry measure). The safe cutoff for the VIF (1 / tolerance) is 10 (Tabachnick & Fidell, 2001), further reiterating risk of collinearity is within acceptable limits. In all, it seems that collinearity is not obscuring a relationship to either waveband asymmetry measure in the regressions.

In attempt to avert potential collinearity altogether, a new composite measure of asymmetry was derived and tested in simple regressions. To obtain this α to β asymmetry

differential the log transformed α power asymmetry measure (lnALPHA) was divided by the log transformed β power asymmetry measure (lnBETA) to yield an asymmetry metric indicative of an individual's flexibility between wavebands. This measure was regressed on the same performance variables and produced significance levels similar to those of the lnBETA regression beta coefficients reported above. Again, significance was only achieved with regard to performance during the first 50 selections. Thus, in future asymmetry studies it would be interesting to compare measures of flexibility between wavebands of interest rather than relative differences between hemispheres for a single waveband.

Asymmetry scores computed for lnALPHA in the present study were distributed in a manner similar to that found by Sutton and Davidson (1997). Individuals displayed a range of scores (approximately -0.10 to 0.20) indicating both left hemisphere dominant and right hemisphere dominant profiles. This is at odds with the distribution found in Schutter, et al. (2004), which demonstrated a lopsided distribution of dominance (approximately -0.15 to 0) favoring activity in the right hemisphere. It is likely that this difference stemmed primarily from the use of the single reference location behind the right ear. Because the left hemisphere site would have less activity in common with the reference site than the right hemisphere site, a left hemisphere power bias results. By then subtracting left hemisphere α power from a now diminished right hemisphere α power, asymmetry dominance values will favor the left hemisphere. This is in fact what is seen in Schutter et al.; however, the direction of the correlation slope would presumably not be affected by the power bias, so these findings remain intriguing as they stand in stark opposition to the predominant asymmetry models.

A goal for future study should be to understand why effects for the inverse α power were not found during the IGT. That the relationship between α and β asymmetries and the positive

and negative (reward and punishment) aspects of the IGT was not supported should not be taken to mean that they do not exist. Reward and punishment delivery during the IGT effect more than a simple jolt of positive or negative emotional experience to the brain. The complexity of the IGT stems from the mental tracking of monetary relationships and the perception of changing deck contingencies (especially in the ambiguous period) in concert with an individual's mood and arousal level. As mentioned previously, circuits underpinning asymmetry in the PFC may operate in regions too far removed from the dlPFC where the electrodes used to calculate asymmetry lie (Davidson, 2004). The OFC and vmPFC have been the focus of the IGT since its development and research has focused on activity in the lateral and medial aspects of the OFC regarding punishment and reward (O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001). The relationship between the OFC and visceral body information (Fuster, 1997) speaks to the validity of the somatic effects found in vmPFC patients during the IGT supporting the SMH. However, the dlPFC, which is charged with updating information during a task, may require task activation before the expected affect valence and motivation related frontal asymmetries emerge in response to the positive and negative experiences during the deck selections. In other words, resting baseline dlPFC EEG asymmetry may vary critically from that of an engaged dlPFC in the operations that convert ambiguity to risk detection.

The performance of participants on the IGT in this study led to some interesting avenues for further investigation. Qualitative examination of performance revealed that participants displayed three styles of responding high, middle, and low. High performers selected from safe decks the least during the first 15 selections. They then show a dramatic shift toward safe decks during the next 15 selections compared to middle and low performers. Perhaps their selection strategy caused them to encounter more early experience with risky decks (during the pre-hunch

early period) that was punctuated by obvious net monetary loss. Or perhaps these individuals were more sensitive to early performance decrements. Either way early punishments may have informed them of the advantageous strategy more quickly than more variable selection patterns. The majority of participants, however, seem to hover around a chance deck selection probability; presumably not gaining the insight necessary to establish a hunch or conceptual knowledge about deck contingencies. They may "give up" and tend to draw randomly or solely based on their most recent reward and punishment experiences. The below-chance strategy of low performers on the other hand implies that they do not simply pick from decks randomly, but are adopting an opposite strategy to that of high performers. They may possess an inability to acquire insight into bad decks' future consequences and instead perseverate on decks with high immediate rewards. Low memory capacity (or low ability to update memory) for reward and punishment experience, may force these participants to continuously restart their search for the best strategy. In essence, this would keep such individuals in a state of motivation toward larger reward values of bad decks, which may seem like the best way to overcome mounting losses. This eventually results in even greater loss. Because ours were all healthy control participants, individual differences in fluctuations of attention and working memory, influenced by arousal and motivation during the 100 selections of the task, may have much more complicated relationships with the IGT than can be predicted by the affect and motivation models of asymmetry. Comparing performance of the three groups to that of patients with different focal PFC damage may prove more diagnostically useful than frontal EEG asymmetry.

In attempt to evaluate the three performance profiles statistically with respect to asymmetry, regressions were re-run separately for high, middle, and low performer groups. It was revealed that for high performers the regression coefficient of lnBETA approached

significance for predicting IGT net score. Right hemisphere lnBETA activity dominance tended to associate with lower IGT net score, while left hemisphere activity dominance tended to associate with higher IGT net score. Furthermore, the regression coefficient for lnBETA approached significance for predicting high performer risky selection behavior during the entire IGT. Right hemisphere activity dominance tended to associate with more risky selections, while left hemisphere dominance tended to associate with fewer risky selections. These results, though not statistically significant, do lend further credence to the contrary findings of Schutter et al. (2004), at least insofar as efficient IGT task learners are concerned. Inspection of middle and low performing participants revealed no regression coefficients for lnALPHA or lnBETA that approached statistical significance regarding any of the IGT performance measures. Though none of its effects reached statistical significance, lnBETA's regression coefficients consistently outperformed those of lnALPHA on IGT performance measures in these groups. Such findings call into question the ubiquitous use of inverse α power as the sole measure of hemispheric activity dominance.

CHAPTER 5.

CONCLUSIONS

The performance on the IGT was highly variable in this sample of healthy control participants and was loosely tied to resting EEG asymmetry. Although the IGT may possess sufficient sensitivity to distinguish vmPFC patient dysfunction from other PFC patient dysfunction, it may not possess the specificity to link resting brain activity asymmetry to performance in healthy individuals. The findings of this study indicate that if resting asymmetry holds value as a predictor of IGT performance, it may do so only in the early portions of the task in which brain activity has not been altered by the formulation of decision strategies. Additionally, the traditional brain activity asymmetry derived from the α EEG waveband may be too restrictive, and other wavebands may be of interest. Faster wavebands such as β and gamma (30 Hz and higher), which is strongly related to glucose metabolism (Davidson, 2004), may be important estimators of cortical activation. A β-band derived asymmetry measure appears to be a better predictor of the selection strategy adopted by the participants during the first 50 selections than α-band derived asymmetry. Future exploration of the IGT might benefit from fMRI imaging of activity during a simplified IGT. If the contingencies of the task were made more readily apparent in order to make the experience more homogeneous amongst participants, yet maintained a similar long-term deck contingency structure, it could confirm asymmetrical PFC reward and punishment sensitive systems through BOLD signal changes. It is likely that the process of decision-making healthy individuals undergo during the IGT involves complex interactions in the PFC which require further identification and disentanglement from preparatory mechanisms before asymmetrical relationships can be established with confidence.

REFERENCES

Allen, J. J. B., Harmon-Jones, E., & Cavender, J. H. (2001). Manipulation of frontal EEG asymmetry through biofeedback alters self-reported emotional responses and facial EMG. *Psychophysiology, 38*. 685-693.

Allen, J. J. B., Coan, J. A., & Nazarian, M. (2004). Issues and assumptions on the road from raw signals to metrics of frontal EEG asymmetry in emotion. *Biological Psychology, 67*, 183-218.

Andersen, P., & Andersson, S. A. (1968). *Physiological basis of the alpha rhythm.* New York, NY: Century-Crofts.

Baars, B. J. (1997). Some essential differences between consciousness and attention, perception and working memory. *Consciousness and Cognition, 6*, 363-371.

Bechara, A. (2004). The role of emotion in decision-making: Evidence from neurological patients with orbitofrontal damage. *Brain and Cognition, 55*, 30-40.

Bechara, A. & Damasio, H. (2002). Decision-making and addiction (part I). Impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. *Neuropsychologia, 40*, 1675-1689.

Bechara, A., Tranel, D., Damasio, H., & Damasio, A. R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to the prefrontal cortex. *Cerebral Cortex, 6*, 215-225.

Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science, 275*, 1293-1295.

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to the human prefrontal cortex. *Cognition, 50*, 7-15.

Bolla, K. I., Eldreth, D. A., & Cadet, J. L. (2004). Sex-related differences in a Gambling Task and its neurological correlates. *Cerebral Cortex, 14*, 1226-1232.

Bowman, C. H., Evans, C. E. Y., & Turnbull, O. H. (2005). Artificial time constraints on the Iowa Gambling Task: The effects on behavioural performance and subjective experience. *Brain and Cognition, 57*, 21-25.

Brand, M., Recknor, E. C., Grabenhorst, F., & Bechara, A. (2007). Decisions under ambiguity and decisions under risk: Correlations with executive functions and comparisons of two different gambling tasks with implicit and explicit rules. *Journal of Clinical and Experimental Neuropsychology, 29*, 86-99.

Buckner, R. L. & Carroll, D. C. (2006). Self-projection and the brain. *Trends in Cognitive Sciences, 11*, 49-57.

Buzsaki, G., Bickford, R. G., Ponomareff, G., Thal, L. J., Mandel, R., & Gage, F. H. (1988). Nucleus basalis and thalamic control of neocortical activity in the freely moving rat. *Journal of Neuroscience, 8*, 4007-4026.

Caroselli, J. S., Hiscock, M., Scheibel, R. S., & Ingram, F. (2006). The simulated gambling paradigm applied to young adults: An examination of university students' performance. *Applied Neuropsychology, 13*, 203-212.

Carver, C. S. & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology, 67*, 319-333.

Coan, J. A. & Allen, J. J. B. (2003). The state and trait nature of frontal EEG Asymmetry in emotion. In Hugdahl, K. & Davidson, R. J.'s (Eds.) *The Asymmetrical Brain.* Cambridge, MA: The MIT Press.

Coan, J. A. & Allen, J. J. B. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology, 67*, 7-49.

Cotterill, R. (1998). Enhanced Looms. CUP, Cambridge, pp. 508.

Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London: Biological Sciences, 351*, 1413-1420.

Davidson, R. J. (1995). Cerebral asymmetry, emotion, and affective style. In: Davidson, R.J., Hugdahl, K. (Eds.), *Brain Asymmetry*. pp. 361–387. Cambridge, MA: The MIT Press.

Davidson, R. J., Taylor, N., & Saron, C. (1979). Hemisphericity and styles of information processing: Individual differences in EEG asymmetry and their relationship to cognitive performance. In: Abstracts of the Papers Presented at the Eighteenth Annual Meeting of the Society for Psychophysiological Research. *Psychophysiology, 16*, 197.

Davidson, R. J. (1988). EEG measures of cerebral asymmetry: Conceptual and methodological issues. *International Journal of Neuroscience*, *39*, 71–89.

Davidson, R. J. (2000). Affective style, psychopathology and resilience: Brain mechanisms and plasticity. *American Psychologist, 55*, 1196 –1214.

Davidson, R. J. (2004). What doe the prefrontal cortex "do" in affect: Perspectives on frontal EEG asymmetry research. *Biological Psychology, 67*, 219-233.

Davidson, R. J., Kabat-Zinn, J., Schumacher, J., Rosenkranz, M., Muller, D., Santorelli, S. F., Urbanowski, F., Harrington, A., Bonus, K., & Sheridan, J. F. (2003). Alterations in brain and immune function produced by mindfulness meditation. *Psychosomatic Medicine, 65*, 564-570.

Eysenck, H. J. (1967). *The biological basis of personality.* Springfield, IL: Charles C Thomas.

Feige, B., Scheffler, K., Esposito, F., Di Salle, F., Hennig, J., & Seifritz, E. (2005). Cortical and subcortical correlates of electroencephalographic alpha rhythm modulation. *Journal of Neurophysiology, 93*, 2864-2872.

Fowles, D. C. (1987). Application of a behavioral theory of motivation to the concepts of anxiety and impulsivity. *Journal of Research in Personality, 21,* 417-435.

Fowles, D. C. (1993). Biological variables in psychopathology: A psychobiological perspective. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (2nd ed., pp. 57-82). New York: Plenum.

Fried, I., Wilson, C. L., MacDonald, K. A., & Behnke, E. J. (1998). Electric current stimulates laughter. *Nature, 391*, 650.

Fuster, J. M. (1997). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe. 3rd Edition.* Philadelphia, PA: Lippincott-Raven Publishers.

Gable, S. L., Reis, H. T., & Elliot, A. J. (2000). Behavioral activation and inhibition in everyday life. *Journal of Personality and Social Psychology, 78*, 1135-1149.

Goldman, R. I., Stern, J. M., Engle, J., & Cohen, M. S. (2002). Simultaneous EEG and fMRI of the α rhythm. *NeuroReport, 13*, 2487-2492.

Goldstein (1948). *Language and Language disturbances.* New York: Grune and Stratton.

Gonçalves, S. I., de Munck, J. C., Pouwels, P. J. W., Schoonhoven, R., Kuijer, J. P. A., Maurits, N. M., Hoogduin, J. M., Van Someren, E. J. W., Heethaar, R. M., & Lopes da Silva, F. H. (2006). Correlating the alpha rhythm to BOLD using simultaneous EEG/fMRI: Inter-subject variability. *NeuroImage, 30*, 206-213.

Gray, J. A. (1972). The psychophysiological basis of introversion-extraversion: A modification of Eysenck's theory. In V. D. Nebylitsyn & J. A. Gray (Eds.), *The biological bases of individual behaviour* (pp. 182-205). San Diego, CA: Academic Press.

Gray, J. A. (1981). A critique of Eysenck's theory of personality. In H. J. Eysenck (Ed.), *A model for personality* (pp. 246-276). Berlin: Springer-Verlag.

Gray, J. A. (1987). *The psychology of fear and stress* (2nd ed.), Cambridge, England: Cambridge University Press.

Gray, J. A. (1990). Brain systems that mediate both emotion and cognition. *Cognition and Emotion, 4*, 269-288.

Gray, J. A. (1994). Three fundamental emotion systems. In P. Ekman & R. J. Davidson (Eds.), *The nature of emotion* (pp. 243-247). New York: Oxford University Press.

Gur, R. C., Skolnick, B. E., & Gur, R. E. (1994). Effects of emotional discrimination tasks on cerebral blood flow: Regional activation and its relation to performance. *Brain and Cognition,* 25, 271-286.

Hagemann, D, Naumann, E., Becker, G, Maier, S., & Barktussek, D. (1998). Frontal brain asymmetry and affective style: A conceptual replication. *Psychophysiology, 35*, 372-388.

Harmon-Jones, E. (2003a). Anger, coping, and frontal cortical activity: The effect of coping potential on anger-induced left frontal activity. *Cognition and Emotion, 17*, 1-24.

Harmon-Jones, E. (2003b). Clarifying the emotive functions of asymmetrical frontal cortical activity. *Psychophysiology, 40*, 838-848.

Harmon-Jones, E. & Allen, J. J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology, 106*, 159-163.

Harmon-Jones, E. & Allen, J. J. B. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affect valence. *Journal of Personality and Social Psychology, 74*, 1310-1316.

Heilman, K. M. & Valenstein, E. (2003). *Clinical Neuropsychology, 4th Ed.* New York: Oxford University Press.

House, A., Dennis, M., Warlow, C., Hawton, K., & Molyneux, A. (1990). Mood disorders after stroke and their relation to lesion locations. *Brain, 113*, 1113-1129.

Keller, I. (2001). Neurofeedback therapy of atention deficits in patients with traumatic brain injury. *Journal of Neurotherapy, 5*,19-32.

Larson, L. L., Davidson, R. J., Abercrombie, H. C., Ward, R. T., Schaefer, S. M., Jackson, D. C., Holden, J. E., & Perlman, S. B. (1998). Relations between PET-derived measures of thalamic glucose metabolism and EEG alpha power. *Psychophysiology, 35*, 162-169.

Larson, R. J. & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. *Journal of Research in Personality, 21*, 1-39.

Laufs, H., Krakow, K., Sterzer, P., Eger, E., Beyerle, A., Salek-Haddadi, A., & Kleinschmidt, A. (2003). Electroencephalographic signatures of attentional and cognitive default modes in

spontaneous brain activity fluctuations at rest. *Proceedings of the National Academy of Sciences, 100*, 11053–11058.

Lawrence, N. S., Jollant, F., O'Daly, O., Zelaya, F., & Phillips, M. L. (2008). Distinct roles of prefrontal cortical subregions in the Iowa gambling task. *Cerebral Cortex,* Advance Issue September 11, 2008.

Lin, C., Chiu, Y., Cheng, C., & Hsieh, J. (2008). Brain maps of Iowa gambling task. *BioMed Central Neuroscience, 9:72.* http://www.biomedcentral.com/1471-2202/9/72

Lin, C. H., Chiu, Y. C., Lee, P. L., & Hsieh, J. C. (2007). Is deck B a disadvantageous deck in the Iowa Gambling Task? *Behavioral and Brain Functions, 3*, 16-26.

Lindsley, D. B. & Wicke, J. D. (1974). *The electroencephalogram: Autonomous electrical activity in man and animals*. In R. Thompson & M. N. Patterson (Eds.), Bioelectric recording techniques (pp. 3-83). New York, NY: Academic Press.

Monterosso, J., Ehrman, R., Napier, K.L., O'Brien, C.P., & Childress, A.R. (2001). Three decision-making tasks in cocaine-dependent patients: Do they measure the same construct? *Addiction, 96*, 1825–1837.

Morison, R. S., Finley, K. H., & Lothrop, G. N. (1943). Spontaneous electrical activity of the thalamus and other forebrain structures. *Journal of Neurophysiology, 6*, 243-254.

Newman, J. (1995). Thalamic contributions to attention and consciousness. *Consciousness and Cognition, 4*, 172-193.

Newman, J. (1997a). Putting the puzzle together, Part I: Towards a general theory of the neural correlates of consciousness. *Journal of Consciousness Studies, 4*, 47-66.

Newman, J. (1997b). Putting the puzzle together, Part II: Towards a general theory of the neural correlates of consciousness. *Journal of Consciousness Studies, 4*, 100-121.

Niedermeyer, E. (2005). The normal EEG of the waking adult. In Niedermeyer, E. & Lopes Da Silva, F. (Eds.) *Electroencephalography: Basic principles, clinical applications, and related fields. 5th Edition.* Piliadelphia, PA: Lippincott, Williams & Wilkins.

Nunez, P. L. (1995). *Neocortical dynamics and human EEG rhythms*. New York, NY: Oxford.

Oakes, T. R., Pizzagalli, D. A., Hendrick, A. M., Horras, K. A., Larson, C. L., Abercrombie, H. C., Schaefer, S. M., Koger, J. V., & Davidson, R. J. (2004). Functional coupling of simultaneous electrical and metabolic activity in the human brain. *Human Brain Mapping, 21*, 257-270.

O'Doherty, J., Kringelbach, E. T., Rolls, E. T., Hornak, J., & Andrews, C. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience, 4*, 95-102.

Oldfield, R.C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia, 9*, 97-113.

Ray, W. J. & Cole, H. W. (1985). EEG alpha activity reflects attentional demands, and beta activity reflects emotional and cognitive processes. *Science, 228*, 750-752.

Rossi, G. S., & Rosadini, G. (1967). Experimental analysis of cerebral dominance in man. In *Brain Mechanisms Underlying Speech and Language*, C. Millikan and F. L. Darley (eds,). New York: Grune & Stratton.

Schutter, D. J. L. G., de Haan, E. H. F., & van Honk, J. (2004). Anterior asymmetrical alpha activity predicts Iowa gambling performance: Distinctly but reversed. *Neuropsychologia, 42*, 939-943.

Senkowski, D., Molholm, S., Gomez-Ramirez, M., & Foxe, J. J. (2006). Oscillatory Beta Activity Predicts Response Speed during a Multisensory Audiovisual Reaction Time Task: A High-Density Electrical Mapping Study. *Cerebral Cortex, 16*, 1556-1565.

Shaw, J. C. (2003). *The brain's alpha rhythms and the mind: A review of classical and modern studies of the alpha rhythm component of the electroencephalogram with commentaries on associated neuroscience and neuropsychology*. Amsterdam, The Netherlands: Elsevier Science B.V.

Steriade, M., Deschenes, M., Domichm, L., & Mulle, C. (1985). Abolition of spindle oscillation in thalamic neurons disconnected from nucleus reticularis thalami. *Journal of Neurophysiology, 54*, 1473-1497.

Steriade, M. (1994). Sleep oscillations and their blockage by activating systems. *Journal of Psychiatric Neuroscience, 19*, 354-358.

Sutton, S. K. & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8*, 204-210.

Tabachnick, B. G. and Fidell, L. S. (2001). *Using Multivariate Statistics* (4th Ed.) Allyn and Bacon, Needham Heights, MA.

Terzian, H. (1964). Behavioral and EEG effects of intracarotid sodium amytal injections. *Acta Neurochirugica (Vienna), 12*, 230-240.

Turnbull, O. H., Berry, H., & Bowman, C. H. (2003). Direct versus indirect emotional consequences on the Iowa Gambling Task. *Brain and Cognition, 53*, 389-392.

Vogel, F. (2000). *Genetics and the electroencephalogram*. New York: Springer

Watson, D. & Clark, L. A. (1994). *The PANAS-X: Manual for the Positive and Negative Affect Schedule – Expanded Form*. The University of Iowa.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063-1070.

Watson, D. & Clark, L. A. (1999). *The PANAS-X: Manual for the Positive and Negative Affect Schedule - Expanded Form.* Retrieved Sept 28, 2008, from http://www.psychology.uiowa.edu/faculty/Clark/PANAS-X.pdf

Whitlow, C. T., Liguori, A., Livengood, L. B., Hart, S. L., Mussat-Whitlow, B. J., Lamborn, C. M., Laurienti, P. J., & Porrino, L. J. (2004). Long-term heavy marijuana users make costly decisions on a gambling task. *Drug and Alcohol Dependence, 76*, 107–111.

Windgrove, J. & Bond, A. J. (1998). Angry reactions to failure on a cooperative computer game: the effect of trait hostility, behavioural inhibition, and behavioral activation. *Aggressive Behavior, 24*, 27-36.

APPENDIX A: ON ALPHA ACTIVITY

W*hat is Alpha?*

While studies of frontal electroencephalogram (EEG) asymmetry have utilized the alpha (α) waveband to estimate inverse "activity," their aim was not explicitly directed at understanding α, and subsequently, few have provided more than a passing explanation for its validity. In recognition of this oversimplification, a deeper exploration of its utility is warranted to ensure that future studies can be founded in a more concrete understanding. Hans Berger discovered a predominating waveform with a peak frequency at 10 Hz that diminished in amplitude with visual stimulation and mental arithmetic. This was present predominantly over the posterior regions of the brain now associated with the striate and extrastriate regions. The diminution of the waveform is referred to as α blocking, and he found that other stimuli also block α, such as voluntary movement or intention to move (Lindsley & Wicke, 1974). It was inferred that α amplitude was inversely related to attention, and that there "…appears to be an inverse relationship between α and the level of mental activity…" (Shaw, 2003). Lindsley and Wicke say that α and beta (β) waves compose much of the waking state brain activity. However, α lends itself to study because it is prominent and responds to sensory stimulation; furthering that a reduction of α represents a generalized reaction of the brain. Moreover, the onset of α activity presence in monkey and man corresponds to the development of functional activity of cortex and its integration with subcortical centers.

Based on these facts spontaneous EEG in man and animals has served as an index of the state of an organism. EEG, like electrical signals, can index a change when the underlying mechanisms are not fully understood. Some individuals show α in trains of 50 μV to 100 μV, but others may show amplitudes of only 5 μV to 10 μV. Alpha however, has been found to be a stable attribute of an individual over time and between identical twins (Vogel, 2000), thus it is

attractive as a measure of an individual's baseline activity that may index dispositional attributes that can be acted upon by experimental manipulation. The EEG continuum of fast waves to slower waves roughly follows an arousal continuum that fits with experimental findings about the role of the reticular activating system. There are numerous exceptions but the general ideas hold that fast rhythms such as gamma (γ) and β involve high demand mental processing level arousal, followed by α as readiness level arousal, and slow waves such as theta (θ) and deep-sleep delta (δ) involving low arousal consolidation processes possibly linked to long-term potentiation (LTP).

The square of the mean of the α frequency band values (8-13 Hz) extracted by a Fast Fourier Transformation procedure (FFT) indicates the amount of α present in EEG, and can be referred to as power spectral density (PSD). Measuring the power of the α band using a PSD procedure provides a gross estimate of the amount of attention and mental effort exerted by neural tissue in the region underlying an electrode site. This measure can be compared between sites within an individual and statistically across individuals. A caveat to this interpretation is that although α amplitude does seem to relate to the arousal continuum view (disappearing during sleep and at highly aroused states), in the intervening alert state where cognition takes place and uses attention, α does not show a proportional response. Attention is not necessarily consciously controlled (Baars, 1997) and may not be follow a linear function.

Many, but not all individuals, exhibit large amounts of α power while resting with eyes closed. Opening the eyes results in a change from this synchronous state to one consisting of low amplitude desynchrony, often β (13-30 Hz). It is commonly accepted that frontal α is an index of a relaxed state of wakefulness and has been likened to "cortical idling," while β is associated with active thought processing and responding in a mentalizing task. The inverse α assumption

has been adopted in many studies, assuming that if α represents a resting trait state, then inverse α will index the degree to which an individual is *not* in a resting trait state. This stable baseline is then referred to as "activity" for use in determining asymmetry score. For purposes of the present investigation we will maintain that resting "activity" involves a low arousal alert state where tonic levels of α activity will emerge while cognitive capacity is not being taxed or confounded by demanding processes.

What Produces Alpha?

Newman's thalamo-cortical model provides evidence that the nucleus reticularis thalami (nRt) underlies the generation and functional significance of the α rhythm. The nRt is responsible for "the selective gating of information flow to the cortex and the regulating of patterns of EEG activation" (Newman 1995a p182). Shaw (2003) explains that the nRt forms a shell around the lateral nucleus of the thalamus and has many connections to it. It "has no projections to the cortex, but acts as a filter or gating mechanism controlling the flow of information between the thalamus and the cortex." Steriade (1994) found that an "isolated reticular thalamic nucleus generates spindle rhythmicity even after its isolation from other thalamic nuclei and the cerebral cortex." Cotterill (1998) described a schematic function of nRt whereby thalamo-cortical projections pass nRt gates, but can be blocked by inhibitory neurons. The degree of inhibition is modulated by the brain stem reticular formation to determine the level of arousal, a function that is controlled by the parietal cortex. Additionally, selected gates are disinhibited by prefrontal connections. Because the nRt has only inhibitory connections to the thalamic nuclei, Newman (1995) describes its action as a "mosaic of 'gatelets' able to modify patterns of cortical activation in selective ways…." The medial reticular formation (MRF) is mainly excitatory and causes high frequency desynchrony in the EEG. Projections from cortex to

the nRt enable regulation of information flow to the cortex so that the nRt enhances EEG synchrony in the cortex. Active behavior results in asynchronous high frequency EEG activity, while "during behavioral inactivity the nRt channels 'shut down' these high frequency oscillations by generating synchronous activity in the circuits looping between the thalamus and the cortex. The interplay of these excitatory and inhibitory systems produces the complex patterns of activation in the cortex we refer to simply as 'the EEG.' Again, in simplest terms, to 'gate' information flow to a region of the cortex is to generate low frequency, synchronous activity." For a more complete exploration of these ideas, see Newman (1997a & b), and Nunez (1995).

Larson et al. (1998) conducted an early study to look at the relationship between positron emission tomography (PET) thalamic glucose metabolism and EEG α power in humans. They based the experiment on findings from several animal studies. First, thalamic oscillations in the α bandwidth are present in corticate and decorticate cats (Morison, Finley, & Lothrop, 1943). Second, synchronized rhythms in any of the thalamic nuclei could be distributed and imposed on the cortex via thalamocortical cells (Andersen & Andersson, 1968). Third, Steriade et al. (1985) suggest that synchronized rhythms come from nRt specifically. Finally, that selective damage to nRt abolishes rhythmic activity in the thalamus and the cortex of rats (Buzsaki et al., 1988). Larson et al. (1998) found that whole-head α correlated inversely with metabolism and thalamic regional cerebral glucose metabolic rate (rCMRglu) was the same for high (10-13 Hz) and low (8-10 Hz) α. Alpha power to thalamic rCMRglu correlations were the same for both hemispheres and over time, (30 min) greater thalamic metabolism is associated with less α. This first look at thalamic activity and α in humans, however, was correlational and it remained unclear whether or not the thalamus is the generator of the rhythms.

More recent studies have made use of brain imaging techniques that provide greater spatial resolution and event-related paradigms to assess the validity of the α and activity relationship. Oakes et al. (2004) also looked at the association of EEG and PET. EEG was measured concurrently with the uptake of the tracer to be used in the acquisition of the PET image at the end of the session so that both imaging techniques represent the same brain activity period. Findings indicated that lower α band (8.5 -10 Hz) was negatively correlated with PET activation. The lower α band results are in keeping with the widely accepted view that α is "inversely related to brain activation" (Davison, 2000). Goldman et al. (2002) found that as α power increased the fMRI brain oxygen level dependent (BOLD) signal decreased in the occipital, superior temporal, inferior frontal, and the cingulate cortex. They also found that increased α was accompanied by increased BOLD in the thalamus and insula. These results suggest that the poor temporal resolution of the PET scan may illuminate "…trait-like properties of α generation" (Goldman, 2002), while fMRI may reveal "…α modulation on an individual subject level, and thus may highlight the role of the thalamus in moment-to-moment wave generation." Feige et al. (2005) found an inverse relationship between α and the BOLD signal that suggested synchronous activity in the thalamus was associated with decreased metabolism. Gonçalves et al. (2006) looked at individual differences and the correlation of α to the BOLD signal using simultaneous EEG/fMRI during a resting state. They found that α was inversely related to BOLD activation in occipital, inferior temporal gyri, orbitofrontal cortex, pre- and post-central gyrus, superior frontal gyrus, and parietal lobe. However, the inter- and intra-subject variability was wide and some positive correlations were found. Thus, the relationship is not a simple one. The positive correlations they found in the thalamic region could have resulted from the activation of gating mechanisms of the nRt. The BOLD signal increase may have occurred as

populations of thalamic neurons, especially in the reticular nucleus, display α rhythm activity. In general, the results of the aforementioned investigations do lend support to the accepted idea that "activity" is associated with decrease in α power and vice versa.

APPENDIX B: PARTICIPANT SAMPLE DATA

Table 1. Comparison of Current BIS/BAS Means to those of Previous Studies

	Present Study *M (SD)*	Carver & White (1994) *M (SD)*	Harmon-Jones & Allen (1997) *M (SD)*
BIS	18.85 (4.15)	19.99 (3.79)	22.14 (2.85)
BAS-Reward	17.61 (3.05)	17.59 (2.14)	17.89 (2.04)
BAS-Drive	12.24 (2.87)	12.05 (2.36)	12.22 (2.26)
BAS-Fun Seeking	12.54 (2.58)	12.43 (2.26)	12.78 (2.16)

Table 2. BIS/BAS Subscale Intercorrelations

	BAS Fun	BAS Reward
BAS Drive	$r = .531$ $p = .000$	$r = .576$ $p = .000$
BAS Fun	-	$r = .544$ p= .000

Table 3. BIS/BAS and PANAS-X Correlations

	Negative	Fear	Guilt	Sadness	Serenity
BAS Fun	ns	$r = .263$ $p = .044$	ns	ns	ns
BIS	$r = .345$ $p = .007$	$r = .316$ $p = .015$	$r = .263$ $p = .044$	$r = .299$ $p = .022$	$r = –.293$ $p = .024$

* “ns” means non-significant

Table 4. PANAS-X Major Scale to Subscale Correlations

	Fear	Hostility	Guilt	Sadness	Shyness	Fatigue	Joviality	Self-Assured	Attentiveness	Serenity	Surprise
Negative	$r = .811$ $p = .000$	$r = .715$ $p = .000$	$r = .692$ $p = .000$	$r = .582$ $p = .000$	$r = .388$ $p = .002$	$r = .508$ $p = .000$	ns	ns	ns	ns	ns
Positive	ns	ns	$r = –.292$ $p = .025$	ns	ns	$r = –.475$ $p = .000$	$r = .826$ $p = .000$	$r = .808$ $p = .000$	$r = .836$ $p = .000$	$r = .365$ $p = .005$	$r = .629$ $p = .000$

Table 5. PANAS-X Subscale Intercorrelations

	Hostility	Guilt	Sadness	Shyness	Fatigue	Joviality	Self-Assured	Attentiveness	Serenity	Surprise
Fear	$r = .385$ $p = .003$	$r = .520$ $p = .000$	$r = .340$ $p = .008$	$r = .458$ $p = .004$	$r = .383$ $p = .003$	ns		ns	ns	$r = .271$ $p = .038$
Hostility	-	$r = .448$ $p = .000$	$r = .473$ $p = .000$	ns	$r = .345$ $p = .008$	ns		ns	ns	ns
Guilt	-	-	$r = .624$ $p = .000$	$r = .302$ $p = .020$	$r = .450$ $p = .000$	$r = –.275$ $p = .042$		ns	$r = -.325$ $p = .012$	ns
Sadness	-	-	-	$r = .283$ $p = .030$	ns	ns		ns	$r = –.353$ $p = .006$	ns
Shyness	-	-	-	-	$r = .283$ $p = .036$	ns		ns	ns	ns
Fatigue	-	-	-	-	-	$r = -.468$ $p = .000$	$r = –.375$ $p = .003$	$r = –.396$ $p = .002$		
Joviality	-	-	-	-	-	-	ns	$r = .665$ $p = .000$	$r = .556$ $p = .000$	$r = .574$ $p = .000$
Self-Assured	-	-	-	-	-	-	-	$r = .612$ $p = .000$	$r = .389$ $p = .002$	$r = .560$ $p = .000$
Attentiveness	-	-	-	-	-	-	-	-	ns	$r = .455$ $p = .000$
Serenity	-	-	-	-	-	-	-	-	-	$r = .358$ $p = .005$