

## ABSTRACT

### The Relationship between Measures of Executive Cognitive Function and P3 Amplitude and Latency in the Auditory Perseveration Task

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This study examined the relationship between measures of executive cognitive function (ECF) and P3 amplitude and latency elicited by Dr. Lance Bauer's Auditory Perseveration (AP) Task. Participants were 66 adults (12 men, 54 women) who were undergraduate students at Baylor University. Participants completed the study in two sessions: (1) a 2.5 hour researcher-administered interview to collect demographic, psychological and executive cognitive functioning data and (2) a 1.5 hour EEG session completed within seven days of the interview. The purpose of the current study was to clarify if the P3 elicited by the AP task should be used as an indicator of general impulsivity, or if it is actually a more finely-tuned neurophysiological indicator of deficits in specific executive function abilities. Employing topographical analysis, the study examined whether the P3 ERP elicited by the AP task was a more frontal P3a or a more parietal P3b ERP. The most significant finding of the study was that the AP task elicited a fronto-central P3a ERP. Significant correlations suggested that that more attenuated P3a amplitude was correlated with ECF measures indicative of perseveration caused by problems with the allocation of attentional resources. Longer P3a latency was

correlated with ECF scores indicating errors of omission (or failing to respond). Shorter P3a latency was correlated with ECF scores indicative of perseverative response patterns. The main findings from this study reaffirm the usefulness of the P3a as a marker for deficits of executive cognitive function.

The Relationship between Measures of Executive Cognitive Function and P3  
Amplitude and Latency in the Auditory Perseveration Task

by

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A Dissertation

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## DEDICATION

To my family

## CHAPTER ONE

### Introduction

In 1929, Berger established that electrical activity in the brain could be recorded by placing electrodes on the scalp. Since that time, researchers have employed this recording tool (the electroencephalogram, or EEG) to study the relationships between cortical electrical activity and psychological phenomena. In the last thirty years, EEG research has focused on electrical potentials that are locked to specific events in time (event-related potentials, or ERPs; Fabiani, Gratton, & Coles, 2000). The P3, a type of ERP, is a positive peak that occurs approximately 300 milliseconds after a stimulus is presented (Polich et al., 1997). The P3 is elicited by a simple discrimination task (an “oddball” paradigm) in which stimuli are presented in random order, and one stimulus has a greater probability of occurring than another (Polich et al., 1997; Verleger & Berg, 1991).

The *amplitude* of a P3 response is thought to reflect the allocation of attentional resources toward processing the rare event, such that a diminished amplitude would signify a deficit in resource allocation (Houston, Bauer, & Hesselbrock, 2004; Humphreys & Kramer, 1994). The P3 *latency* is thought to be an indication of stimulus evaluation time, equal to the time it takes to evaluate the stimulus and take appropriate action, with longer latencies coinciding with longer stimulus-to-response time measurements (Donchin & Coles, 1988; Duncan et al., 1994; Hillyard & Kutas, 1983; Kutas, McCarthy, & Donchin, 1977; Polich, 1987; Polich et al., 1997). In this way, the P3 has been shown to be useful as a gauge of working memory (Donchin, Karis, Bashore,

Coles & Gratton, 1986). The P3 is typically employed to study component scalp distribution differences that could reflect size and/or orientation differences of underlying neural generators (Johnson, 1989, 1993; Polich & Squire, 1993; Verleger, Heide, Butt, & Kompf, 1994). The P3 is useful in that it is an “involuntary index of recognition” in behavioral research, and in one study, was successfully used to detect malingering (an increased P3 in malingering condition vs. an amnesia condition; Ellwanger, Rosenfeld, Hankin, & Sweet, 1999).

There has been a great deal of debate over the cognitive model of the main components of the P3 wave, the fronto-central (maximally located at Fz and Cz) “P3a” and the parietal (maximally located at Pz) “P3b” waveforms. Almost all reliable reports of anterior P3a ERPs involve the use of novel stimuli that elicit an early, fast-habituating, fronto-central alerting response that likely comes from neural loci (predominantly in the frontal lobe) related to fast attentional allocation which does not require deep processing (i.e., regardless of whether the stimulus is attended to or ignored; Posner, 1992; Posner & Petersen, 1990). P3a ERPs, rarely observed unless the stimuli are surprising and unrelated to task performance, are thought to reflect a passive, involuntary cortical response to stimuli. When more memory processes are required to store stimulus information for later use in the task (the stimuli are attended to), the later parietal P3b is elicited (Knight, 1996; Polich & Squire, 1993). This later P3b response is related to stimulus discrimination, and therefore, is dependent on the attention that the individual allocates to the information that is to be coded into working memory (Courchesne, Hillard, & Galambos, 1975). In patients with unilateral frontal lesions (interruption of the limbic-prefrontal circuitry which is necessary to process novel information), Knight

(1984) demonstrated that the P3a had a more posterior location, maintained this location over time, and resembled the P3b when compared with non-lesioned controls. P3b ERPs did not differ between controls and frontal lesion patients. Knight concluded from this that the P3a “early orienting response” was modulated, although not primarily generated, by frontal brain structures, while the P3b ERP was relatively independent of these frontal brain areas. This finding illustrates that there is a likely functional/structural difference between the P3a and P3b ERP components. The P3b most likely represents the active, voluntary updating of task-relevant categorization of the task stimuli (Donchin & Coles, 1988) and has been shown to be especially sensitive to stimuli that are especially novel within their context (more physically or categorically deviant stimuli elicit the largest P3b ERPs; Squires, Squires, & Hillyard, 1975; Nasman & Rosenfeld, 1990). However, it is possible that neural structures responsible for generating the P3a may also be at least partly responsible for the generation of the P3b (hence, the debate over the classification and origins of these two P3 components; Knight, 1984). Detection of the deviation of contextual stimuli has been shown to exert an independent and additive effect on P3b amplitude (Nasman & Rosenfeld, 1990). In other words, during task-related target detection, the P3b component reflects the special attention that is allocated to explicitly non-task related stimulus characteristics. Luria (1973) coined the term “involuntary orienting” to describe the basis for the prefrontal-limbic role in generating the P3b.

### *Psychopathology and the P3*

Amplitude and latency of the P3 have been shown to vary as a function of the level of psychopathology present in the individual (and in some cases, psychopathology present in the individuals’ first-degree relatives). P3 amplitude/latency variance has been

demonstrated in individuals with disruptive behavior disorders, substance-use disorders, Cluster B personality disorders, impulsive aggression, and other psychopathology, such as depression, Bipolar Disorder, Schizophrenia, and PTSD. Most of these disorders/conditions have behavioral disinhibition as a core facet to their etiologies.

### *Disruptive Behavior Disorders*

Patients with unmedicated Attention-Deficit/Hyperactivity Disorder (AD/HD) have been shown to have significantly more single-trial variability in the “P3 processing window” (250-500ms after the onset of the stimulus). This variability was reduced significantly with the application of stimulant medication (Lazzaro, Anderson, Gordon, Clarke, Leong, & Meares, 1997). McPherson and Salamat (2004) found a greater separation between the P3a and the P3b (reduced amplitudes and longer latencies) in individuals with AD/HD as compared to controls, suggesting a processing lag associated with the disorder. Individuals with AD/HD also displayed a significantly higher false alarm rate, longer reaction time, and a higher rate of “guessing” than controls. Gonzalez (1996) found that P3 amplitude could be used to distinguish between AD/HD subtypes (AD/HD: Combined Type and AD/HD: Predominately Inattentive Type; amplitude was significantly lower at the Cz site in the Inattentive subtype) and between individuals with AD/HD, regardless of subtype, and normal controls (AD/HD groups had significantly lower P3 amplitudes at the Pz site). These findings suggest that a parietally-reduced P3 could be used as an indicator of AD/HD and related deficits, and a centrally-reduced P3 in individuals with diagnosed AD/HD can be used to differentiate between the two AD/HD subtypes.

Conduct Disorder (CD) is a persistent, repetitive pattern of behavior that is associated with aggression to people or animals, destruction of property, theft, lying, and serious violations of rules (running away from home or skipping school) that affect the individual's performance in society, at school, or at work. Conduct Disorder is diagnosed during childhood, and the subtypes (Childhood-Onset Type [before age 10], Adolescent Type [after age 10], and Unspecified Onset [age not known]) are differentiated by the age of the individual at the onset of symptomology (American Psychiatric Association, 2000). Boys who engaged in rules violations, a subtype of the behavioral problems associated with Conduct Disorder, did not evidence a normal P3 amplitude increase with maturation (this effect was not demonstrated with any other specific CD behaviors, i.e., destructiveness, theft, or lying). Additionally, this maturational deficit was localized to P3 generators within the frontal lobe. Parietal P3 generators matured normally regardless of the presence of rules violations (Bauer & Hesselbrock, 2003). Bauer (1997) found that the frontal P3 decrement was significantly related to the number of childhood Conduct Disorder symptoms, but not with the presence/absence of a family history of alcoholism. Also, this P3 decrement alone correctly identified 70.6% of patients who later relapsed, and 53.3% of patients who did not, in a residential drug treatment program. In 2001, Bauer and Hesselbrock found that the prefrontal areas of the brain underlying Conduct Disorder problem behaviors probably caused affected adolescents to be less able to engage in the cognitive "error-checking" process that occurs before decisions about differences in events or stimuli.



### *Substance Use Disorders*

Reduction in P3 amplitude has long been considered to be an ERP marker for those individuals who are at risk for developing substance-use disorders (Hesselbrock, Begleiter, Porjesz, O'Connor, & Bauer, 2001; Steinhauer, Hill, & Zubin, 1987). P3 amplitude has also been associated with addictive behaviors, and has been found to be predictive of resumption of drinking in recovering alcoholics. These findings suggest the utility of conceptualizing substance-use disorders in terms of an organic mental disorder because of the localizability of the P3 ERP component in individuals evidencing these addictive behaviors (Parsons, 1994).

Substance Abuse is related to other comorbid psychopathology and this relationship may be mediated by impulsivity (Swann, Dougherty, & Pazzaglia, 2004). Children who are at high risk for substance use disorder have been shown to exhibit deviations in temperament, an attenuated amplitude on the P3, and heightened aggressivity compared with control groups (Giancola & Tarter, 1999). Biggins, MacKay, Poole, and Fein (1995) found prolonged P3a latency in elderly chronic alcoholics as compared to elderly controls, and posited that P3a latency could be used as an objective measure of the long-term effects of chronic alcohol abuse on the cortex. In a predictive study, lower P3 amplitudes were observed in 10-12 year-old boys who later developed a substance use disorder *before* age 19 (Habeych, Charles, Sciabassi, Kirisci, & Tarter, 2005). Individuals with earlier initial use of psychoactive substances showed a significantly reduced P3 amplitude (Iacono & McGue, 2006). On a broader scale, Carlson, McLarnon, and Iacono (2007) found significantly more externalizing disorders and a reduced P3 amplitude in individuals with early-onset substance use disorders

(before age 20) as compared to individuals with late-onset substance use disorders or individuals with no substance-use disorders. The P3 amplitude reduction was still present in individuals with externalizing disorders and *no* substance-use disorders, indicating that the P3 could be used as an index for externalizing behavior in addition to assessing the risk for developing substance use disorders.

### *Cluster B Personality Disorders*

Antisocial, Borderline, and Narcissistic Personality Disorders often co-occur with Substance Abuse (Putt, Dowd, & McCormick, 2001). Cocaine-abusing individuals with a diagnosis of Antisocial Personality Disorder (ASPD) showed reduced P3a amplitudes at frontal electrode sites during a visual selective attention task (measuring impulsivity) as compared to individuals without ASPD or in control groups (Bauer, 1997). Bauer (1994) also showed a frontal reduction in P3 amplitude (anterior brain dysfunction) in individuals with ASPD *and* a family history of alcoholism as compared with individuals with only ASPD and no family history of alcoholism (1<sup>st</sup> and 2<sup>nd</sup> degree relatives). Costa et al. (2000) reported a reduced anterior P3 amplitude in participants with ASPD. Borderline Personality Disorder (BPD) is characterized by disinhibited behavior, fluctuating affect, interpersonal problems, and impulsivity (DSM-IV-TR; APA, 2000). An attenuated P3 amplitude and prolonged P3 latency has also been observed in participants with BPD as compared with controls (Blackwood, Sharp, Walker, & Goody, 1996; Drake, McLoughlin, Pepper, & Minkoff, 1991; Kutcher et al., 1987, 1989). Houston, Ceballos, Hesselbrock, and Bauer (2005) posited that there may be abnormal maturational processes at work in the brains of adolescent girls who evidence BPD symptomology.

### *Impulsive Aggression*

Impulsive aggression is defined as “a hair trigger aggressive response to provocation with loss of behavioral control. This loss of control is not secondary to any medical or psychiatric disorder and, by virtue of the spontaneity of the act, is not planned” (Barratt, 1991). The P3 has also been employed as a marker for psychopathology involving heightened impulsivity and aggression. P3 deficits are associated with heightened levels of impulsiveness, indicating the value of the P3 as a neurophysiological marker of impulsivity (Barratt, Pritchard, Faulk, & Brandt, 1987; Carrillo-de-la-Pena, 1992). Impulsive aggressive individuals showed a significantly reduced P3 amplitude compared to nonaggressive controls (Gerstle, Mathias, & Stanford, 1998) and a prolonged latency in response to target stimuli (Mathias & Stanford, 1999). In a double-blind, placebo-controlled crossover design, Barratt, Stanford, Felthous, and Kent (1997) found that P3 amplitudes were increased significantly by application of phenytoin (an anti-epileptic drug) in impulsive aggressive individuals, but not in premeditated (nonimpulsive) aggressive individuals or in controls.

### *Other Psychopathology*

The P3 has also been shown to correlate with memory problems associated with depression. However, depression history as well as task difficulty may influence the utility of the P3 in documenting the neurophysiological aspects of the disorder (Houston et al., 2004). Auditory P3 amplitudes have been shown to be significantly smaller in individuals with melancholic, unipolar depression, and were normalized after a depression-alleviating treatment was administered (Gangadhar, Ancy, Janakiramaiah, & Umopathy, 1993). Melancholic individuals with hallucinations and/or delusions have

been shown to exhibit reduced P3 amplitudes during an auditory “oddball” paradigm (Santosh, Malhotra, Raghunathan, & Mehra, 1994). It has been suggested that, in addition to indicating cognitive functioning, the P3 amplitude may reflect the participant’s degree of emotional involvement in a task, a supposition that is strengthened by the fact that depressed individuals typically display significantly smaller P3 amplitude, but no difference in P3 latency (Diner, Holcomb, & Dykman, 1985). Giving motivating instructions prior to a task in order to increase involvement has been shown to increase P3 amplitude (Carrillo-de-la-Pena & Cadaviera, 2000). P3 amplitude was found to be significantly reduced in depressed patients who had attempted suicide as compared to depressed patients who had not (Hansenne, Pitchot, Moreno, Torrecilas, Mirel, & Ansseau, 1994). Two studies have demonstrated that higher levels of impulsive aggression predict suicidal behavior and an earlier age of onset of mood disorder (Brent et al., 2003; Melhem et al., 2007). This indicates that impulsive aggression may predispose individuals to certain life events that probably give rise to depression, or may be an atypical/incomplete form or a precursor of depression.

There is also evidence of P3 variability in PTSD, Bipolar Disorder, and Schizophrenia. Kaufman, Kimble, McTeague, Kaloupek, Forti, and Keane (2002) demonstrated that individuals diagnosed with Posttraumatic Stress Disorder (PTSD) who scored high on dissociative experiences (employing the Dissociative Experiences Scale) evidenced significantly reduced P3 amplitudes in response to distracting novel sounds and significantly longer latencies (processing times) in response to both distracting and target stimuli in a novelty P3 paradigm (as compared to individuals with PTSD who scored lower on dissociative experiences). Souza et al. (1995) also found that auditory

P3 latency was significantly longer in patients with Bipolar Disorder and in patients with Schizophrenia, but only patients with Schizophrenia showed an attenuated P3 amplitude.

### *Behavioral Disinhibition and Externalizing Disorders*

Behavioral disinhibition is an over-arching concept that involves the inability to inhibit socially forbidden/undesirable behaviors. Often observed in childhood, characteristic behaviors in AD/HD, CD, and oppositional defiant disorder (ODD) range from impulsivity and attention problems (AD/HD) to rule breaking and defiant, antisocial behaviors (CD and ODD). A feature of ASPD is that CD was present during childhood, but that the antisocial behavior persisted into adolescence and adulthood. Substance use disorders are also associated with behavioral disinhibition, especially when the substance ingestion takes place early in life (i.e., before it is legal for the person to purchase alcohol or cigarettes). All childhood antisocial, disruptive, and substance use diagnoses are collectively termed ‘externalizing disorders.’ In the absence of such diagnoses, disinhibited individuals will still likely evidence behaviors such as trouble with law enforcement, sexual deviance and experimentation at a young age, early-onset drug abuse, and defiant/rule-breaking behaviors (Iacono, 1998; Iacono, Malone, & McGue, 2003). Behavioral disinhibition may be a heritable/learned common factor which, when present in the father, predisposes the child to a greater risk of developing externalizing psychopathology. For example, Malone, Iacono, and McGue (2002) found that alcoholic fathers were significantly more likely to have an 11-year-old child with CD or ODD than would nonalcoholic fathers. The result for AD/HD risk was similar, but not statistically significant. These children were also more likely to have begun smoking cigarettes, to have been drunk, and to have at least one symptom of substance dependence. This risk

grew significantly in the 17-year-old cohort that was tested in the Malone et al. study. Similarly, the children of fathers who have been diagnosed with Adult Antisocial Behavior (AAB; no CD diagnosis in their childhood) or ASPD were equally likely to have CD, and more than twice as likely to have it as compared to fathers without ASPD (Elkins, Iacono, & Doyle, 1997). Twin studies investigating the genetic contribution to the development of externalizing disorders have demonstrated a genetic influence on CD and ODD (Levy & Holzman, 1997; Nadder, Silberg, Eaves, Maes, & Meyer, 1998), smoking (Kendler, Thornton, & Pedersen, 2000), alcohol abuse (Prescott & Kendler, 1999), other substance use disorders (Grove et al., 1990; Kendler & Prescott, 1998; Van den Bree, Johnson, Neale, & Pickens, 1998) and antisocial behavior (Grove et al., 1990; Jacobson, Prescott, & Kendler, 2000). Fathers with antisocial or substance use disorders (as well as alcoholic fathers) typically have sons who display similar magnitude reduction in P3 amplitude (Iacono et al., 2003). Some studies have shown that measures of temperament related to behavioral disinhibition predict later antisocial behavior and substance abuse, and suggest that the level of constraint may be a mediating temperament factor that influences the development of externalizing psychopathology (Caspi et al., 1997; Cloninger, Sigvardsson, & Bohman, 1988; Masse & Tremblay, 1997; Tarter & Vanyukov, 1994). It is likely that P3 amplitude reduction associated with oddball tasks (like the AP task) could be used as an index for externalizing behaviors characterized by behavioral disinhibition (Iacono et al., 2003). Thus, behavioral disinhibition (with heightened impulsivity as a core feature) is probably a significant source of the P3 amplitude and latency variability that has been demonstrated in all of the aforementioned psychopathologies.

### *Executive Function*

Executive function refers to the set of broad regulatory processes that modulate cognition and response in a wide variety of contexts, and are especially critical when novel responses must be carried out in the presence of more habitual dominant responses, and when task goals have to be actively maintained in the face of potent internal and external distracters. These “oddball” tasks could range from simple discrimination tasks to more complex social interaction contexts. Recent research posits that executive function may actually be a conglomeration of interrelated functions rather than a single function (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000), and several studies suggested that various executive functions are also related to more maladaptive personality constructs, such as those that occur in personality disorders. In particular, the inability to retain pertinent information in the working memory system and to prevent distraction because of response inhibition are likely to be affected. Therefore, the ability to control thought and behavior as measured by executive function tasks should be related to a variety of personality disorders. This relationship is not particularly surprising, because, according to the DSM-IV-TR (APA, 2000), poor impulse control, as well as difficulties in emotion regulation and cognitive processing, typify most personality disorders (Dolan & Park, 2002; Matheson & Langdon, 2008). Executive cognitive functioning (ECF) in the prefrontal cortex plays a major role in the regulation of goal-directed behavior and is involved in attentional control, strategic goal planning, organization, and cognitive flexibility (Deckel, Bauer, & Hesselbrock, 1995). Neuropsychological testing, such as the Wisconsin Card Sort Task (WCST) and the Delis- Kaplan Executive Function System (DKEFS) test are used to identify and quantify

ECF deficits. The DKEFS Card Sorting task was based on the WCST, and has been shown to reliably measure set shifting and distinguish the different types of perseveration deficits (failure to change a given response pattern, even though it is no longer useful to the individual) present in the participant (Parsons, 1994). Set shifting (changing the required rule paradigm governing the correct response to a task) is the major executive cognitive skill that is tested using the WCST. Failure to set shift is often an indicator of perseveration. A greater level of perseveration is present in individuals with heightened levels of impulsivity, lower executive cognitive functioning, and frontal lobe damage (Deckel, Hesselbrock, & Bauer, 1996). Response inhibition occurs when an individual is able to successfully set-shift in a task, and switches to a new set of rules for responding, thus inhibiting the response that would have been necessary for the previous rule set. This inhibition is modulated by the individual's speed of cognitive processing, which, in turn, is dependent on axonal myelination and synaptogenesis (Travis, 1997).

Specifying perseveration type is useful in order to elucidate whether memory and/or ECF deficits are present in the individual. There are three generally-recognized subtypes of perseveration: continuous, stuck-in-set, and recurrent perseveration. Continuous perseveration deficits (the inappropriate repetition without interruption of a current behavior) were only associated with ECF deficits, but not memory problems, indicating this type of perseveration indexes problems with motor output. Stuck-in-set perseveration is associated with both ECF and memory functioning. Recurrent perseveration is viewed as distinct from ECF and memory abilities, and is not associated with tests of cognitive function (Possin, Filoteo, Reosch, Zizak, Rilling, & Davis, 2005). Recurrent perseveration increases with age. However, continuous perseveration does not



vary with age. This suggests multiple simultaneous processing skills, and more demand placed on cognitive resources in elderly individuals. Continuous perseveration differences could distinguish between a cognitive and memory dysfunction. In perseveration research, differences were found that suggest that individuals may employ different cognitive resources as they get older, which may be indicative of a maturational decline in frontal brain volume or a change in neural mechanisms with increasing age (Foldi, Helm-Estabrooks, Redfield, & Nickel, 2003). ECF has been found to be partly modulated by the maturation of the frontal lobe system (Travis, 1997).

### *Disruptive Behavior Disorders*

Executive cognitive function (ECF) deficits have been proposed as an important neuropsychological correlate of AD/HD (Pennington & Ozonoff, 1996). Neuroimaging studies have indicated that there are small neurobiological abnormalities in the prefrontal cortex of children with AD/HD (Castellanos et al., 1994; Tannock, 1998). Antisocial adults often evidenced anterior brain dysfunction, as measured by neuropsychological test performance (Deckel et al., 1996; Gillen & Hesselbrock, 1992). Wild-Wall, Oades, Schmidt-Wessels, Christiansen, and Falkenstein (2009) found that children with AD/HD had greater impairment in controlled than in automatic processing and inhibition, and this was likely due to a deficit in inhibitory processing, as evidenced by a reduced P3a in a Go/No-Go task. Children with CD have been shown to have deficits in executive cognitive functioning and in verbal abilities (Giancola, Mezzich, & Tarter, 1998). Frontal lobe dysfunction has also been associated with impulsivity, heightened risk for substance abuse/dependence, problems with executive cognitive function, and behavioral issues, such as Conduct Disorder in adults (Deckel, et al., 1995). However, Clark, Prior,

and Kinsella (2000) found that executive function deficits were specific to AD/HD, as those individuals evidencing comorbid AD/HD and Oppositional Defiant/Conduct Disorder (ODD/CD) evidenced significantly more problems with strategic planning and personal behavioral monitoring as compared to a control group with only ODD/CD. Similarly, Fairchild et al. (2009), in a study that employed the Risky Choice Task and the Wisconsin Card Sort Task (WCST) to assess ECF, suggested that impairments in ECF do not underlie decision-making deficits that are associated with CD, but rather, a faulty, more risk-prone reward system is present, especially in early-onset CD.

### *Substance Use Disorders*

Individuals who abuse drugs often display neurocognitive problems in making decisions. Their choice of action is often based on the greatest and quickest reward, rather than on thoughts toward a positive future outcome. Psychopathic heroin addicts displayed significant deficits in decision making, and made many more disadvantageous decisions than did non-psychopathic heroin addicts (as measured by the PCL-R (Psychopathy Checklist-Revised), suggesting that if psychopathy is present decision-making deficits may be made worse in these addicts (Vassileva et al., 2006). In a study of adolescents with psychopathic characteristics in a substance abusing cohort, O'Neill, Lidz, & Heilbrun (2003) found that psychopathic characteristics were negatively related to variables dealing with treatment process and outcome, such as substance abuse, participation in treatment programs, attrition rates, and clinical improvement. However, the presence of psychopathic characteristics was positively correlated with the number of times the individual was arrested in the year following release from the treatment program. In another study, violent mentally ill prisoners were shown to have a higher

level of polysubstance abuse and more incidences of psychopathic traits than non-violent ones (Sreenivasan, Kirkish, Shoptaw, Welsh, & Ling, 2000). Substance abuse is often comorbid with externalizing behaviors (Holdcraft, Iacono, & McGue, 1998). In addition, fewer neurons have been found in the frontal lobes of alcoholics in post-mortem studies (Giancola & Tarter, 1999).

### *Cluster B Personality Disorders*

Antisocial Personality Disorder (ASPD) is characterized by a pattern of irresponsible, delinquent, and criminal behavior that begins in childhood and persists into adulthood (APA, 2000), is associated with high rates of substance abuse in the general population, and has been found to be related to poorer alcohol and drug abuse treatment outcomes. Further, the comorbidity of severe mental illness (SMI), ASPD, and Substance Use Disorder (SUD) is associated with victimization, aggression, legal problems, and poor social functioning. Antisocial adults often evidence anterior brain dysfunction, as measured by neuropsychological test performance (Deckel et al., 1996; Gillen & Hesselbrock, 1992). The number of ASPD symptoms has been found to predict Substance Abuse in individuals with severe mental illness (Crocker et al., 2005). In Antisocial Personality Disorder (ASPD), it was found that cocaine dependent subjects with ASPD were more impulsive and aggressive than were controls (Moeller, Dougherty, & Barratt, 2002). The aggression and antisocial behavior exhibited in ASPD has been associated empirically with poor executive functioning (Giancola, 2004; Morgan & Lilienfeld, 2000). Borderline Personality Disorder (BPD) has also been shown to be associated with poor performance on ECF tasks, but in the case of BPD, this affective

arousal and motivation may modulate these ECF deficits (Fertuck, Lenzenweger, Clarkin, Hoermann, & Stanley, 2006).

### *Impulsive Aggression*

Deficits in cognitive set shifting are linked to impulse control problems and reduced P3 amplitudes. Physiological underarousal and increased hostility and Lifetime History of Aggression scale scores have been demonstrated in more impulsive individuals. Such dispositional impulsivity consists of novelty thrill seeking, behavioral disinhibition, and nonplanning. Buss Perry Aggression Questionnaire total score is correlated with nonplanning and disinhibited behavior. One major difficulty in impulsivity research is the daunting task of teasing apart the neurophysiological correlates of impulsivity and aggression, because of the often overlapping symptomology (Houston & Stanford, 2005). Trait impulsiveness is also associated with executive cognitive functioning (ECF). Individuals demonstrating heightened levels of trait impulsivity also show deficits in key factors associated with ECF, such as goal-directed planning and motor deficits (Pietrzak, Sprague, & Snyder, 2008). The Lifetime History of Impulsive Behavior scale is constructed around two key subtypes: functional and dysfunctional impulsivity. Functional impulsivity is defined as a failure of impulse control in situations in which impulsive behavior could be beneficial to the individual. Dysfunctional impulsivity is a lack of impulse control that is detrimental to the individual (Schmidt, Fallon, & Coccaro, 2004). The P3 ERP has been demonstrated to be a better measure of mental speed, attention, and allocation than earlier potentials, such as the N1 and P2 (Verma, Twitty, & Fuerst, 1993). Whipple and Noble (1991) found a correlation between P3 amplitude and visuospatial neuropsychological deficits. Villemarette-

Pittman, Stanford, and Greve (2001) found that individuals who displayed impulsive aggressive outbursts showed deficits on complex verbal tasks of executive cognitive function that required spontaneous organization, but not on tasks only requiring limited verbal output. This result suggests that inefficient executive function, not language ability, is responsible for the variability in verbal task performance in impulsive aggressive individuals.

### *The AP Task*

The Auditory Perseveration (AP) task is used to evoke a P3 ERP. The P3 can be obtained with any discrimination task in which one stimulus has a higher probability than another. The AP task involves two tones, a low pitch tone (500Hz) and a high pitch tone (1000Hz). The participant is given directions about pushing specified buttons with the thumbs when hearing certain tones. When a “white noise” burst sounds, it signals to the participant that it is time to switch, and to push the opposite button from what they were just pressing for the same tone. The P3 is evoked after the white noise burst. Tests similar to the AP task (like the Wisconsin Card Sort task) are used to look at perseverative errors indicative of impulsivity (Almasy et al., 1999; Gevins & Cuttillo, 1986; Hillyard & Kutas, 1983; Houston, et al., 2004; Polich, 1998; Polich et al., 1997). The AP task has been used to elicit the P3 in several different contexts. Adolescents with CD and adults with ASPD show no maturational change in P3 activity associated with the AP Task. Discrimination studies have demonstrated that the P3 amplitude elicited by the AP task could be used as a predictor of those individuals that would successfully complete a residential drug treatment programs, and also of those individuals who would likely relapse (Bauer & Hesselbrock, 2003). Thus, the P3 waveform could possibly be

used as a physiological marker of possible future relapse in individuals seeking treatment for drug dependence.

Recent research (which employed the AP task) found that the P3 amplitude could be used to successfully discriminate between individuals who completed a residential drug treatment program and those who did not. Interestingly, the Auditory Perseveration task elicited both P3a and P3b waveforms (described earlier). For non-completers, P3 amplitudes were most attenuated in the Pz (parietal) location. P3 amplitudes for treatment program completers followed this general trend (except for a slight, non-significant increase in amplitude from sites Fz to Cz, then decrease to Pz), but overall, the amplitudes in the completers were significantly elevated compared to non-completers. Following the logic of current theories as to the nature of P3a and P3b amplitudes, the data lead to the conclusion that the treatment program non-completers had less attentional resources to allocate to the AP task (reduced frontal/central amplitude as compared to completers). When the task merited the use of memory storage mechanisms in order to save stimulus information for later use (such as remembering which button to push for the low/high tone after the white noise burst), non-completers showed marked deficits in memory coding as compared to participants who completed the drug treatment program (reduced P3b amplitude at P3). Similarly, P3 latencies are smallest at frontal and central sites for treatment program non-completers, indicating more impulsive button-pushing and less time used in the evaluation of the stimulus. Not surprisingly, the longest latency (paired with the lowest amplitude) in the non-completer group was for the parietal midline electrode (Pz) because individuals who have difficulty in coding memory for storage would most likely also take longer to attempt to code that information. The

longest latency for the treatment program completers was, surprisingly, in the Fz electrode. This might suggest that treatment program completers were just more thorough in their evaluation of the target stimulus presentation. The shortest latency for treatment program completers was at the Pz electrode (paired with a higher amplitude than that found in the non-completer group). These data suggested that completers not only allocate more neural mechanisms for memory storage, but they also employ these memory storage mechanisms faster than treatment program non-completers. These deficits in storage capability and capacity (measured by the P3b), paired with basic deficits in executive cognitive functioning and the reduced ability to effectively allocate attentional resources (measured by the P3a), appeared to be the principle features that distinguished between the completer and non-completer drug treatment program groups in the study (Baldrige & Stanford, 2007). This research coincided with a recent pilot study that indicated that drug offenders who exhibited greater executive cognitive functioning deficits (as measured by lower scores on assessments such as the Wisconsin Card Sort Task and STROOP Color/Word Task) also had a more attenuated P3 waveform across midline sites (Dolan, Baldrige, & Stanford, 2008). This indicates that the P3 is probably more of a direct measure of executive cognitive functioning, as the Auditory Perseveration task (that was used to elicit the P3 in both studies) was modeled after the Wisconsin Card Sort Task by its creator, Dr. Lance Bauer.

The purpose of the current study was to run a comprehensive battery of executive cognitive functioning measures in conjunction with the AP task. This study served to clarify if the P3 elicited by the AP task could be employed as a generalized predictor of impulsivity, or if it is actually a more finely-tuned neurophysiological indicator of

deficits in specific executive function abilities. This research is novel in that no study has determined the precise neuropsychological correlates of changes in P3 amplitude associated with the Auditory Perseveration task.



## CHAPTER TWO

### Method

#### *Procedure*

Participants completed the testing in two sessions. The first session (2.5 hours in duration) consisted of researcher-administered, interview-style psychological tests selected to collect demographic and background information, assess depression, anxiety, impulsiveness, and executive cognitive functioning. During the second session (1.5 hours in duration), an electroencephalogram (EEG) was administered and the participant completed the Auditory Perseveration task. The combined testing sessions were approximately four hours long.

#### *Participants and Recruitment*

Participants were 100 undergraduate volunteers recruited from Baylor University in Waco, Texas who were between 18 and 25 years of age at the time of testing. Participants were recruited online, and volunteered for the study via the SONA research site (available through Baylor University). Participants were compensated with four research participation credit hours (through the SONA site) and a \$20 Walmart giftcard for their participation in the study.

#### *Power Analysis*

The G3 Power 3.1.2 program (Faul, Erdfelder, Buchner & Lang, 2009) was used for the power analysis. A medium effect size (0.25) was used to determine the number of participants necessary for the study. It was estimated that a total sample size of 62

participants would be sufficient to maintain power at 0.8 with  $\alpha = 0.05$ . In order to assure that this sample size would be reached after the exclusionary criteria for the study were applied and certain participants were excluded from the analysis, a sample size of 100 participants were initially interviewed for the study.

### *Exclusionary Criteria*

Participants who evidenced an overall IQ of less than 80, a head injury resulting in unconsciousness or hospitalization, and bipolar or psychotic disorders were excluded from the study. Also, individuals with a medical and/or neurological illness (i.e., seizures or brain surgery), those who were currently taking psychoactive medication/s, and individuals who evidenced current substance abuse were also excluded from the study.

### *Background and Exclusionary Measures*

#### *Demographic and Background Questionnaire*

The demographic/background questionnaire provided information about the participants' age, employment history, education, ethnicity, and medical history. The medical history information (e.g., psychological diagnoses, history of head injury, medication history) were used to determine participants' eligibility for the study.

#### *SCID-I Screen Patient Questionnaire-Extended (SSPQ-X)*

The SCID-I (SSPQ-X; First, Gibbon, Spitzer, Williams, & Benjamin, 1991; Zanarini, 2000) is a computer-administered, 589 question diagnostic tool that covers the major areas of DSM-IV Axis I including Mood Disorders, Psychotic Symptoms, Anxiety Disorders, Substance Use Disorders, Eating Disorders, and Somatoform Disorders.

Although kappa values (a statistic that corrects for chance agreement) for the SCID-I range widely (from .57 to 1.0), all indicate a fair to good level of agreement. Also, because of the uniform computer administration style of the questionnaire, inter-rater reliability problems associated with administering the questionnaire as a clinical interview were avoided during this study. The SCID-I was employed to determine if Bipolar Disorder, psychotic symptoms, or substance abuse were present that would cause the participant to be excluded from the study.

#### *Personality Assessment Inventory (PAI)*

The PAI (Morey, 2007) is a 344 item self-report questionnaire that evaluates constructs relevant to psychopathology and personality assessment in adults aged 18 to 89 (i.e., anxiety, depression, aggression). The PAI consists of 22 non-overlapping scales and provides a comprehensive screening of psychopathology: four validity scales, 11 clinical scales, five treatment scales, and two interpersonal scales. Reliability studies indicate that the PAI maintains a high degree of internal consistency across samples (median alpha and test retest correlations exceed .80 for the 22 scales). Validity studies show convergent and discriminant validity with more than 50 other measures of psychopathology (Morey, 2007). The PAI was employed to determine if Bipolar Disorder, depression, psychotic symptoms, or substance abuse were present that would cause the participant to be excluded from the study.

#### *Personality Diagnostic Questionnaire (PDQ-4)*

The PDQ-4 (Tye & Mullen, 2006) is a 99 question, True/False, computer-administered assessment recommended as a screen for personality disorders and provides

possible DSM-IV diagnoses useful in assisting an actual diagnosis by a clinician (Tye & Mullen, 2006; Hyler, 1997). Validity studies support that the results obtained from the PDQ-4 are consistent with the diagnoses of trained mental health care workers (Hyler & Lyons, 1988; Maffei et al., 1995). The PDQ-4 also contains a validity scale as a measure of invalid responses (Hyler, 1997). The PDQ-4 was used to specify the personality disorder/s present in the participant.

#### *Wechsler Abbreviated Scale of Intelligence (WASI)*

The WASI (Wechsler, 1999) is a shortened test used to assess intellectual abilities and functioning in adults and children. Two of the four subtests (Vocabulary as a measure of Verbal Scale IQ, and Matrix Reasoning as a measure of Performance Scale IQ) were administered in an interview style in order to generate a Full-2 IQ score. Concurrent validity studies using the WASI show high correlations between the WASI and other widely-validated tests of ability, such as the WAIS-III, Wechsler Individual Achievement Test (WIAT), and the Kaufman Brief Intelligence Test (Axelrod, 2002; The Psychological Corporation, 1999; Zhu & Tulskey, 1999). Participants who attained an IQ score under 80 were excluded from the study.

#### *Self-Report Measures*

##### *Barratt Impulsiveness Scale (BIS-11)*

The BIS-11 (Patton, Stanford, & Barratt, 1995) is a 30 item self-report questionnaire that evaluates general impulsiveness while recognizing that impulsivity is likely a multi-factorial construct. The scale produces six first order factor scores and three second-order factor scores. A total general impulsiveness score is attained by

summing the factor scores, and higher scores indicate a greater level of impulsiveness. The items were scored on a four-point scale (Rarely/Never [1], Occasionally [2], Often [3], Almost Always/Always [4]). Reliability coefficients published for the BIS-11 (Cronbach's alpha) range from .79 to .83 (Patton et al., 1995). The BIS-11 was used as both a multi-factor and a total index of impulsiveness present in the participant.

#### *Buss-Perry Aggression Questionnaire (BPAQ)*

The BPAQ (Buss & Perry, 1992) self-report questionnaire contains 29 items and assesses level of aggression. Four factors make up this scale: 1) physical aggression (frequency of acting aggressively), 2) verbal aggression (frequency of speaking aggressively), 3) anger (emotional component of aggression) and 4) hostility (cognitive component of aggression). Items are scored on a five-point scale: 1=Extremely uncharacteristic of me, to 5=Extremely characteristic of me. Cronbach's alpha reliability coefficient for the BPAQ ranges from .72 to .89 (Buss & Perry, 1992). The BPAQ was employed as a measure of the various forms of aggression, and a total BPAQ score was calculated to indicate the overall level of aggression present in the participant.

#### *Executive Cognitive Functioning Assessment*

##### *Iowa Gambling Task (IGT)*

The IGT (Bechara, Damasio, Damasio, & Anderson, 1994) is a computer-administered psychological task which has been shown to simulate real-life decision making. In the task, participants are shown four decks of virtual cards. They are instructed that each time they click on a card they will be awarded some virtual money, but that choosing some cards will cause the participant to be deducted some money. The

point of the game is to win as much money as possible. The decks differ from each other in the number of trials it takes for the participant to incur a loss. Hence, some of the decks are “good decks” because they lead to overall gains, and some decks are considered “bad decks” because they lead to overall losses. The IGT has been demonstrated to be an ecologically valid and insightful test used to detect decision-making problems in several neurologic and psychiatric samples (Bechara, 2004; Brand, Grabenhorst, Starcke, Vandekerckhove, & Markowitsch, 2007). The IGT was employed to indicate if problems in decision making indicative of perseveration were present in the participant.

#### *Wisconsin Card Sort Task (WCST)*

The WCST (Eling, Derckx, & Maes, 2008; Grant & Berg, 1948) was created in 1948 by Grant and Berg to measure concept formation, abstract reasoning, and response strategies to changing contextual information. It is employed as a neuropsychological assessment of the ability of the participant to set-shift (i.e., to maintain cognitive flexibility in the face of changing response paradigms), and has been shown to assess executive dysfunction in the frontal lobe. The WCST has been adapted for computer administration (Psychological Assessment Resources, 2003). During testing, the participant is presented with four “key cards” on the screen. The shapes on the cards differ in quantity, color, and design. The participant is then instructed to match cards from a pile to each one of the key cards, thus forming separate piles for each key card. The participant is not told how to match the cards, but is informed after each match whether the match was correct or incorrect. During the test, the matching rules are changed (based on quantity, color, or design). The mistakes made during testing and the

time required to learn the new rules are analyzed to compute scores including: categories achieved, trials, errors, and perseverative errors. The WCST was employed as a measure of the participants' ability to modulate impulsive responding.

#### *Delis-Kaplan Executive Function System (D-KEFS)*

The D-KEFS (Delis, Kaplan, & Kramer, 2001) is composed of a battery of 9 stand-alone researcher-administered tests that measure cognitive flexibility and factors related to higher level executive function of the frontal lobe. The test has been normed for individuals aged 8 to 89 years. The DKEFS has test-retest correlations of .43 to .73 and moderate internal consistency reliability coefficients (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). Because of time constraints present in the study, seven of the nine D-KEFS tests were chosen to elucidate the ECF deficits present in the participant.

*Trail Making Test.* The Trail Making subtest (involving the participant drawing a line to connect different letter/number sequences) uses a process approach that elucidates what errors are associated with letter sequencing, number sequencing, visual scanning deficits, motor speed discrepancies, and cognitive flexibility.

*Verbal Fluency Test.* This test is composed of three conditions. 1. Letter Fluency: The examinee says words beginning with a specified letter as quickly as possible; 2. Category Fluency: The examinee is asked to say words belonging to a designated semantic category; 3. Category Switching: The examinee must alternate between saying words from two different semantic categories.

*Design Fluency Test.* This test measures ability to generate novel designs as quickly as possible, response inhibition, and cognitive flexibility. The test is composed of three conditions. In each condition, the participant is presented rows of boxes containing dots that the examinee must connect, with four lines only, to make different designs. (Conditions: 1. Draw as many designs as possible. 2. Connect only unfilled dots, leaving filled dots blank. 3. Alternate connections between filled and unfilled dots.)

*Color/Word Interference Test.* The Color/Word Interference subtest uses completion times as the primary performance measures. There are two baseline conditions (Color Naming and Word Reading) and two higher-level conditions (Inhibition and Inhibition/Switching). Participants must inhibit reading words denoting colors while naming the colors themselves in the inhibition condition. The higher-level conditions provide a normed indication of cognitive flexibility.

*Sorting Test.* In the Sorting subtest, participants must sort sets of six cards into as many different categorization rules as possible (Free Sorting Confirmed Correct), and then must describe the concepts that the examiner used to generate each sort (Sort Recognition Description). This subtest measures cognitive flexibility and verbal/non-verbal concept formation abilities.

*Twenty Questions Test.* This test assesses the ability to identify the various categories and subcategories represented. The participant is presented with a stimulus page depicting pictures of 30 common objects, and tries to ask the fewest number of yes/no questions in order to identify the unknown target object. Several process measures



are provided to assist the clinician in determining the neurocognitive mechanisms responsible for poor performance on this test.

*Word Context Test.* Examinees must discover the meaning of a made-up word based on its use in five sentences. The Word Context subtest measures processing efficiency, cognitive flexibility, and response initiation.

### *Physiological Measurements*

Psychophysiological EEG measures were recorded between the hours of 9 am and 2 pm to control for diurnal variations in EEG scalp patterns. Participants were seated in a comfortable chair in a chamber designed to block incoming sound and light. The scalp and mastoid areas were prepared by a vigorous but painless scrubbing with alcohol to remove scalp oils, and a mildly abrasive substance (NuPrep) to increase reading sensitivity by increasing blood flow to the scalp. The participant's head was then fitted with an Electro-Cap (Neuroscan) consisting of 64 electrodes, arranged according to the International 10-20 system with standard and intermediate positions. Electrodes were referenced to an average reference, and four electrooculogram (EOG) electrodes were affixed near the eyes in order to record horizontal and vertical eye movements that may contaminate the EEG data. An ocular artifact reduction technique was applied to the data offline in order to remove any eye movement data contamination. An "ocular artifact" was defined as an eye blink artifact (measured at the VEO electrode site) which had a voltage threshold of less than  $-200 \mu\text{V}$  and a refractory period of 600 milliseconds. The "ocular artifact reduction technique" involved: (1) identifying all ocular artifacts with a numerical marker in Neuroscan; (2) identifying the EEG section that occurred temporally

200 ms before the ocular artifact marker to 600 ms after the ocular artifact marker; (3) deleting that temporal section of EEG material. The EEG was recorded continuously at a sampling rate of 1,000 samples per second and was amplified by SYNAMPS<sup>2</sup> amplifiers (Neuroscan). Filter bandpass was set at 0.1 Hz to 35 Hz. Impedance for each electrode was maintained at less than 5 k $\Omega$ .

#### *Auditory Perseveration Task*

The Auditory Perseveration (AP) task was used to elicit the P3 ERP component. A P3 wave can be obtained with any discrimination task in which one stimulus has a higher probability of occurring than another. The AP task involves two tones, a low pitch tone (500 Hz) and a high pitch tone (1000 Hz). The participant is given directions about pushing specified buttons with the thumbs when certain tones sound. When a “white noise” burst sounds, it signals to the participant that it is time to switch, and to push the opposite button from what they were just pressing for the same tone. All stimuli last 1500 ms and there is a 2500 ms interval between stimuli. The low and high pitched tones are presented an equal number of times in a random order. The occurrence of the white noise burst cannot be predicted by the participant, because it occurs much less frequently than the low or high tones, and is interspersed randomly between the low and high tones. The P3 is elicited after the white noise burst. Tests similar to the AP task (like the Wisconsin Card Sort task) have been used to investigate perseverative errors indicative of impulsivity (Hillyard & Kutas, 1983; Polich, 1998; Gevins & Cutillo, 1986).

### *Statistical Analysis*

Hierarchical linear regression and simple correlational analysis were used to determine the relationship between executive cognitive measures and the P3 amplitude and latency.

### *Hypotheses*

- I. The specific components of ECF (problem solving, planning, concept formation, impulse control, perseverative errors, cognitive flexibility, processing efficiency, and response initiation/inhibition) that are associated with P3 amplitude and latency were elucidated while controlling for DSM-IV Axis I and Axis II (personality disorder) psychopathology that have been shown to affect the variance of the P3 ERP component.
  - a. It was hypothesized that participants who make a greater number of perseverative errors, those who are less cognitively flexible, and those with faster response initiation (on ECF tests) would display a more attenuated P3 amplitude elicited by the AP task.
- II. The AP task's ability to elicit a more fast, frontal (P3a) and/or a later, more parietal (P3b) P3 component will be assessed.
  - a. It was hypothesized that the AP task would generate both P3a and P3b components, as fast attentional allocation (P3a) and contextual updating (P3b) are both involved in completing the AP task.

## CHAPTER THREE

### Results

#### *Demographic Information*

The sample consisted of 66 participants, 12 men and 54 women. All participants were enrolled as undergraduate students at Baylor University at the time of testing. Approximately 53% of the participants were classified as White/Non-Hispanic ( $n = 35$ ), 15% were Hispanic ( $n = 10$ ), 15% were Asian ( $n = 10$ ), 9% were Black ( $n = 6$ ), 2% were Black/Hispanic ( $n = 1$ ), 2% were Native American ( $n = 1$ ), and 5% were classified as “Other” ( $n = 3$ ). As participants were offered research participation credit as one incentive to participate in the study (from classes most often attended by underclassmen), the sample distribution was approximately 55% Freshman ( $n = 36$ ), 27% Sophomore ( $n = 18$ ), 11% Junior ( $n = 7$ ) and 8% Senior ( $n = 5$ ). Sample means and standard deviations for age, years of education, and Full-Scale IQ are presented in Table 1.

Table 1. Demographic Information

Variable	<i>M</i>	<i>SD</i>
Age	19.14	0.99
Years of Education	13.71	0.94
FSIQ (WASI)	108.79	15.10

#### *Exclusionary Criteria*

One-hundred participants were originally scheduled for the study. Thirty-four participants were disqualified after meeting a variety of exclusionary criteria. In all, ten were disqualified for a likely diagnosis of Schizoid and/or Schizotypal personality

disorder indicated by PDQ-4 results. Three individuals were disqualified for evidencing depressive symptomology employing the PDQ-4 and PAI. Four were disqualified for failing to complete the test battery in the time allotted (D-KEFS, BIS or PDQ-4 data were missing). Four individuals either failed to return for the EEG portion of the test or missed the initial interview/testing portion. In three cases, the texture of the participants' hair did not permit for an accurate EEG administration (i.e., cornrows or excessive height of the hair itself). Three individuals evidenced alcohol abuse, and two individuals admitted to excessively abusing substances other than alcohol. One individual was disqualified for a history of seizures. One participant was disqualified for a traumatic brain injury resulting in unconsciousness for more than five minutes. Two individuals had a prior diagnosis of Depression and Bipolar disorder, respectively. One individual had severe eyesight problems that negatively affected her performance on many of the D-KEFS tasks (the participant squinted and indicated she could not see the ink on the pages). These disqualified participants were still compensated with a \$20 Wal-Mart gift card and either full or partial research participation credit as long as the two scheduled testing sessions were attended.

### *Topographical Analysis*

Amplitude of the P3 component of the event-related potential (ERP) at midline electrodes (Fz, Cz, Pz) was analyzed using a one-way repeated-measures ANOVA. Results showed a significant effect for electrode site (Wilks's  $\Lambda = .60$ ,  $F(2,64) = 21.10$ ,  $p < 0.01$ , multivariate  $\eta^2 = .40$ ). Follow-up contrasts showed significant differences ( $p < 0.05$ ) in P3 amplitude between sites Fz ( $M = 19.46$ ,  $SE = 0.84$ ) and Cz ( $M = 20.39$ ,  $SE = 0.97$ ), between sites ( $p < 0.01$ ) Cz ( $M = 20.39$ ,  $SE = 0.97$ ) and Pz ( $M = 18.23$ ,  $SE =$

0.87), and between sites ( $p < 0.05$ ) Fz ( $M = 19.46$ ,  $SE = 0.84$ ) and Pz ( $M = 18.23$ ,  $SE = 0.87$ ).

Latency of the P3 component of the event-related potential (ERP) at midline electrodes (Fz, Cz, Pz) was also analyzed using a one-way repeated-measures ANOVA. Results showed a significant effect for electrode site (Wilks's  $\Lambda = .90$ ,  $F(2,64) = 3.66$ ,  $p < 0.05$ , multivariate  $\eta^2 = .10$ ). Follow-up contrasts showed significant differences ( $p < 0.05$ ) in P3 latency between sites Fz ( $M = 330.80$ ,  $SE = 3.26$ ) and Pz ( $M = 336.00$ ,  $SE = 3.46$ ), and between sites ( $p < 0.01$ ) Cz ( $M = 330.79$ ,  $SE = 3.36$ ) and Pz ( $M = 336.00$ ,  $SE = 3.46$ ). Amplitude and latency measures for Fz, Cz, and Pz are presented in Table 2. P3 waveforms at electrode sites Fz, Cz, and Pz are presented in Figure 1, and a topographical representation of the locations of these electrodes is shown in the Appendix.

Table 2. P3 Amplitude and Latency Measures in Fz, Cz and Pz

Measure/Site	Amplitude ( $\mu\text{V}$ )		Latency (ms)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Midline Electrodes</i>				
Fz	19.46	6.84	330.80	26.46
Cz	20.39	7.85	330.79	27.29
Pz	18.23	7.03	336.00	28.08

Note.  $\mu\text{V}$  = microvolts from baseline, ms = milliseconds from the onset of the stimulus

#### *Correlational Analysis*

Correlation coefficients were computed between both averaged P3 amplitudes and averaged latencies for frontal (averaged F3, Fz and F4) and central (averaged C3, Cz and C4) electrode sites, primary measures, personality self-report scales, and behavioral data. P3 amplitude and latency correlations with the Buss-Perry Aggression Questionnaire subscales and total score and the Barratt Impulsiveness Scale-11 subscales and total score

are presented in Table 3. P3 amplitude and latency correlations with the Wisconsin Card Sort Task and Auditory Perseveration Task are presented in Table 4. P3 amplitude and latency correlations with the Delis-Kaplan Executive Function System are presented in Table 5. In general, while only a limited number of variables were found to be significantly correlated, the results suggest that P3 amplitude was inversely related to executive function measures that assess the ability to change response patterns. P3 latency was negatively correlated with variables that measure response accuracy and perseveration and highly correlated with variables that by their nature, indicate that more mistakes were made on a given task.

Table 3. P3 Amplitude and Latency Correlations with the Buss-Perry Aggression Questionnaire and Barratt Impulsiveness Scale-11

Measure	<u>Frontal Correlations</u>		<u>Central Correlations</u>	
	Amplitude	Latency	Amplitude	Latency
<b>Buss-Perry Aggression Questionnaire</b>				
Physical Aggression	-.16	-.05	-.10	.01
Verbal Aggression	-.21	*.26	-.14	.23
Anger	-.15	.07	-.10	.09
Hostility	-.07	-.08	-.03	.01
Total Score	-.18	.04	-.11	.09
<b>Barratt Impulsiveness Scale-11</b>				
Attention	-.12	-.01	-.15	-.02
Motor	-.05	.16	-.02	.17
Self-Control	-.21	.21	-.20	.22
Cognitive Complexity	.03	.15	.01	.17
Perseverance	-.14	-.04	-.12	-.02
Cognitive Instability	-.08	.19	-.10	.16
Attentional Impulsiveness	-.12	.09	-.15	.07
Motor Impulsiveness	-.09	.11	-.06	.13
Non-planning Impulsiveness	-.13	.22	-.14	.24
Total Score	-.14	.18	-.14	.18

\*=Correlation is significant at  $p < 0.05$ ; \*\* = Correlation is significant at  $p < 0.01$

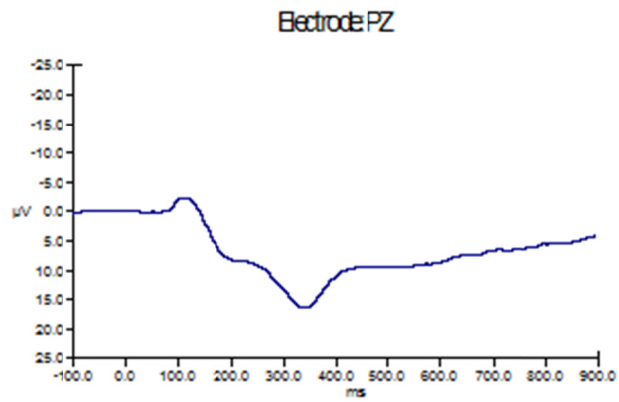
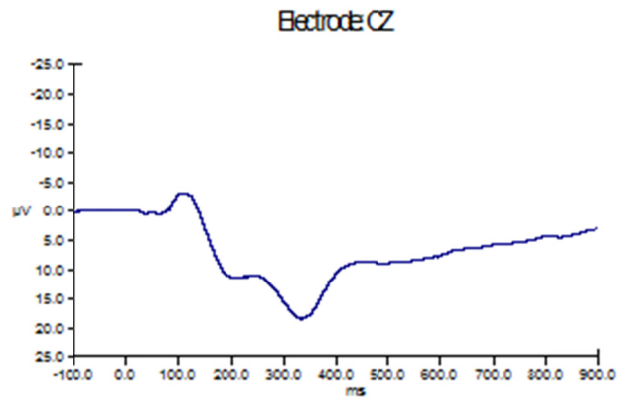
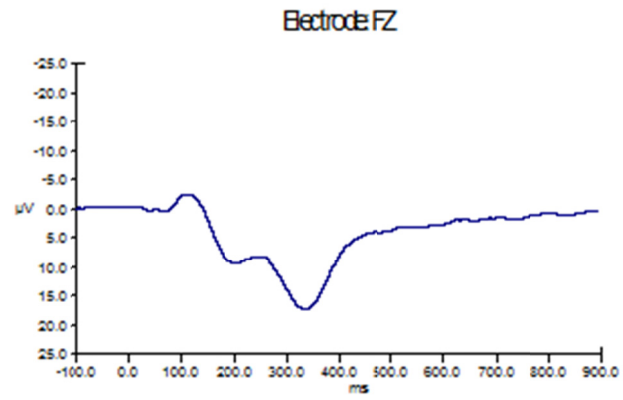


Figure 1. P3 ERP waveforms at electrode sites Fz, Cz, and Pz.



Table 4. P3 Amplitude and Latency Correlations with the Wisconsin Card Sort and Auditory Perseveration Tasks

Measure	Frontal Average		Central Average	
	Amplitude	Latency	Amplitude	Latency
<b>Wisconsin Card Sort Task</b>				
Total Errors T Score	-.08	-.04	-.08	-.01
Perseverative Responses T Score	-.09	.03	-.12	.05
Perseverative Errors T Score	-.09	.02	-.12	.04
Nonperseverative Errors T Score	.05	-.12	.09	-.08
Conceptual Level Responses T Score	.02	-.04	.03	-.01
Categories Completed	.03	-.03	.05	.01
Trials to Complete 1 <sup>st</sup> Category	-.07	.20	-.11	.22
Failure to Maintain Set	.03	-.12	.03	*-.25
Learning to Learn	.07	.20	.08	*.25
<b>Auditory Perseveration Task</b>				
Total # of Perseverative Errors	.23	*-.29	.23	**-.36
Total # of Omissions	-.15	*.29	-.14	**-.41
Total # of Hits	-.18	.09	-.22	.05

\*=Correlation is significant at  $p < 0.05$ ; \*\* = Correlation is significant at  $p < .01$

Table 5. P3 Amplitude and Latency Correlations with the Delis-Kaplan Executive Function System

Measure	Frontal Average		Central Average	
	Amplitude	Latency	Amplitude	Latency
<b>Delis-Kaplan Executive Function System: Trails</b>				
Visual Scanning Scaled Score	.12	.06	.17	.12
Number Sequencing Scaled Score	-.17	.04	-.12	.08
Letter Sequencing Scaled Score	.01	.11	-.01	.09
N/L Sequencing Scaled Score	-.13	.09	-.15	.10
Motor Speed Scaled Score	-.05	.03	-.05	.03
All Error Types: N/L Scaled Score	.04	*-.30	.03	**-.33
<b>Delis-Kaplan Executive Function System: Verbal Fluency</b>				
Letter Fluency Total Correct	-.02	-.12	-.03	-.03
Category Fluency Total Correct	.13	-.01	.14	.08
Cat. Switch. Tot. Correct Responses	-.13	.22	-.20	.18
Cat. Switch. Tot. Switch. Accuracy	-.17	.18	*-.25	.15
Set-Loss Errors	-.11	-.09	-.15	-.05
Repetition Errors	.13	.02	.08	-.08
Percent Set-Loss Errors	-.10	-.10	-.15	-.07
Percent Repetition Errors	.09	-.05	.04	-.12
Cat. Switch. % Switching Accuracy	-.22	-.19	-.25	-.20
<b>Delis-Kaplan Executive Function System: Design Fluency</b>				
Filled Dots: Total Correct	.10	-.02	.13	.06
Empty Dots: Total Correct	-.10	-.13	-.07	-.06
Switching: Total Correct	-.06	.09	.01	.13
Total Correct	.01	-.03	.05	.04
Tot. Set-Loss Designs	-.01	-.12	.07	-.11
Tot. Repeated Designs	.02	-.14	-.01	-.12

Table 5 Continued. P3 Amplitude and Latency Correlations with the Delis-Kaplan Executive Function System

Measure	Frontal Average		Central Average	
	Amplitude	Latency	Amplitude	Latency
Tot. Attempted Designs	-.02	.04	.01	.12
Percent Design Accuracy	.09	-.21	.13	-.18
Delis-Kaplan Executive Function System: Color-Word				
Color Naming	.16	.05	.14	.10
Word Reading	.14	.02	.13	.09
Inhibition	.14	.08	.09	.08
Inhibition/Switching	.09	-.21	.13	-.20
Error: Inhibition	-.12	-.04	-.10	.03
Error: Inhibition/Switching	-.22	-.22	-.12	-.19
Delis-Kaplan Executive Function System: Sorting				
Free Sorting Confirmed Correct Sorts	-.10	-.02	-.09	.14
Free Sorting Description Score	-.07	-.11	-.07	.03
Sort Recognition Description Score	.16	-.20	.21	-.04
Combined Description Score	.06	-.16	.10	.01
Delis-Kaplan Executive Function System: Twenty Questions				
Initial Abstraction Score	*-.26	.07	-.14	.05
Total Questions Asked	-.10	-.13	-.08	-.04
Total Weighted Achievement Score	*-.25	-.13	-.23	-.03
Delis-Kaplan Executive Function System: Word Context				
Total Consecutively Correct	-.13	-.07	-.10	-.06
Consistently Correct Ratio	.06	-.08	.08	-.04
Repeated Incorrect Responses	*-.28	-.05	*-.29	-.07

\*=Correlation is significant at  $p < 0.05$ ; \*\* = Correlation is significant at  $p < 0.01$

*Regression Analysis to Predict Frontal and Central Averaged P3 Amplitude and Latency*

A series of four stepwise multiple regression analyses were conducted to predict both frontal and central averaged P3 amplitude and latency from a combination of measures of executive cognitive function found in the Wisconsin Card Sort Task and the Delis-Kaplan Executive Function System. The first regression analysis was conducted to predict P3 averaged frontal amplitude. The analysis included age, years of education, WASI FSIQ, D-KEFS Twenty Questions Initial Abstraction Score and D-KEFS Total Weighted Achievement Score as predictors. These variables were included in the regression analysis because the scores were found to be significantly correlated with P3 averaged frontal amplitude. The linear combination of demographic and executive

cognitive function (ECF) measures was significantly related to P3 averaged frontal amplitude,  $F(5,60) = 3.36, p = .01$ . The sample multiple correlation coefficient was .47, indicating that approximately 22% of the variance of the P3 averaged frontal amplitude in the sample can be accounted for by the linear combination of demographic and ECF measures. In Table 6, indices are presented to indicate the relative strength of the individual predictors. Three of the bivariate correlations between the predictors and P3 frontal amplitude were negative, as expected, and two of the five indices were statistically significant ( $p < .05$ ). The partial correlations between Age, Years of Education, and Twenty Questions Initial Abstraction Score were significant. On the basis of these correlational analyses, it could be concluded that the only useful predictor of P3 averaged frontal amplitude is the Twenty Questions Initial Abstraction Score. It alone accounted for 7% ( $-.26^2 = .07$ ) of the variance in the P3 averaged frontal amplitude, while the other variables contributed only an additional 15% ( $22\% - 7\% = 15\%$ ). However, it is difficult to make judgments about the relative importance of these predictors because age and years of education are correlated at .82.

Table 6. The Bivariate and Partial Correlations of the Predictors with P3 Averaged Frontal Amplitude

	Correlation between each predictor and P3 Frontal Amplitude	Correlation between each predictor and P3 Frontal Amplitude controlling for all other predictors
Age	.16	*.32
Years of Education	-.04	*-.28
WASI IQ Score	.05	.18
20Q: Initial Abstraction Score	*-.26	*-.26
20Q: Total Weighted Achievement	*-.25	-.18

\*=Correlation is significant at  $p < 0.05$ ; \*\* = Correlation is significant at  $p < 0.01$

The second regression analysis was conducted to predict P3 averaged frontal latency. The analysis included age, years of education, overall IQ, and D-KEFS Trails All Error Types: N/L Scaled Score as predictors. The linear combination of predictor variables was not found to be significantly related to P3 averaged frontal latency,  $F(4,61) = 1.75, p = .15$ . The sample multiple correlation coefficient was .32, indicating that approximately 10% of the variance of the P3 averaged frontal latency in the sample can be accounted for by the linear combination of demographic and D-KEFS measures. In Table 7, indices are presented to indicate the relative strength of the individual predictors. Two of the bivariate correlations between the predictors and P3 frontal latency were positive, and one of the four indices was statistically significant ( $p = .01$ ). Only the partial correlation between P3 averaged frontal latency and D-KEFS Trails All Error Types: N/L Scaled Score was found to be significant. On the basis of these correlational analyses, it could be concluded that the only useful predictor of P3 averaged frontal latency is the D-KEFS Trails All Error Types: N/L Scaled Score. It alone accounted for 9% ( $-.30^2 = .09$ ) of the variance in the P3 averaged frontal latency, while the other variables accounted for only an additional 1% ( $10\% - 9\% = 1\%$ ).

Table 7. The Bivariate and Partial Correlations of the Predictors with P3 Averaged Frontal Latency

	Correlation between each predictor and P3 Frontal Latency	Correlation between each predictor and P3 Frontal Latency controlling for all other predictors
Age	.06	.02
Years of Education	.05	.05
WASI IQ Score	-.05	-.03
T: All Err. Types: N/L Scaled Score **	-.30	**-.31

\*=Correlation is significant at  $p < 0.05$ ; \*\* = Correlation is significant at  $p < 0.01$

The third regression analysis was conducted to predict P3 averaged central amplitude. The analysis included age, years of education, overall IQ, and the D-KEFS Verbal Fluency Category Switching Total Accuracy Score as predictors. The linear combination of the demographic and executive cognitive function (ECF) measures was significantly related to P3 averaged central amplitude,  $F(4,61) = 4.47, p < .01$ . The sample multiple correlation coefficient was .48, indicating that approximately 23% of the variance of the P3 averaged frontal amplitude in the sample can be accounted for by the linear combination of demographic and ECF measures. In Table 8, indices are presented to indicate the relative strength of the individual predictors. Two of the bivariate correlations between the predictors and P3 central amplitude were negative, as expected, and one of the four indices was statistically significant ( $p < .05$ ). The partial correlations between P3 averaged central amplitude and Age, Years of Education, and D-KEFS Verbal Fluency Category Switching Total Switching Accuracy were significant ( $p < .01$ ). On the basis of these correlational analyses, it could be concluded that the only useful predictor of P3 averaged central amplitude are age, years of education, and the D-KEFS Verbal Fluency Category Switching Total Switching Accuracy score. These three variables accounted for 14% ( $.38^2 = .14$ ), 13% ( $-.36^2 = .13$ ) and 11% ( $-.33^2 = .11$ ) of the variance in the P3 averaged frontal amplitude, respectively, while the other variables contributed only an additional 16% ( $38\% - 22\% = 16\%$ ). However, it is difficult to make judgments about the relative importance of these predictors because age and years of education are correlated at .82.

Table 8. The Bivariate and Partial Correlations of the Predictors with P3 Averaged Central Amplitude

	Correlation between each predictor and P3 Central Amplitude	Correlation between each predictor and P3 Central Amplitude controlling for all other predictors
Age	.11	** .38
Years of Education	-.10	** -.36
WASI IQ Score	.08	.20
VF: Cat. Sw. Total Sw. Accuracy	* -.25	** -.33

\*=Correlation is significant at  $p < 0.05$ ; \*\* = Correlation is significant at  $p < 0.01$

The fourth regression analysis was conducted to predict P3 averaged central latency. The analysis included age, years of education, overall IQ, Wisconsin Card Sorting Task Learning to Learn score and Wisconsin Card Sorting Task Failure to Maintain Set score as predictors. The linear combination of predictor variables was not found to be significantly related to P3 averaged central latency,  $F(5,59) = 1.97, p = .10$ . The sample multiple correlation coefficient was .14, indicating that approximately 2% of the variance of the P3 averaged central latency in the sample can be accounted for by the linear combination of the five demographic and ECF measures. In Table 9, indices are presented to indicate the relative strength of the individual predictors. Two of the bivariate correlations between the predictors and P3 central latency were negative, and two of the five indices were statistically significant (two at  $p < .05$ ). The two partial correlations between P3 averaged central latency and Wisconsin Card Sort Task Learning to Learn and Wisconsin Card Sort Task Failure to Maintain Set were significant. On the basis of these correlational analyses, it could be concluded that the only useful predictors of P3 averaged central latency are the Wisconsin Card Sort Task Learning to Learn and the Wisconsin Card Sort Task Failure to Maintain Set scores.

Table 9. The Bivariate and Partial Correlations of the Predictors with P3 Averaged Central Latency

	Correlation between each predictor and P3 Central Latency	Correlation between each predictor and P3 Central Latency controlling for all other predictors
Age	.04	-.12
Years of Education	.04	.08
WASI IQ Score	-.01	-.08
WCST: Learning to Learn	*.25	*.29
WCST: Failure to Maintain Set	*-.24	*-.29

\*=Correlation is significant at  $p < 0.05$

## CHAPTER FOUR

### Discussion

The main finding of the current study was that the Auditory Perseveration Task elicited a fronto-central P3a waveform. In addition, significant correlations suggest that more attenuated P3a amplitude was correlated with executive cognitive function (ECF) measures indicative of perseveration caused by problems with the allocation of attentional resources. Longer P3a latency was correlated with ECF scores indicating errors of omission (or failing to respond). Shorter P3a latency was correlated with ECF scores indicative of perseverative response patterns. It should be noted that the BPAQ subscales and the BIS were not included in the regression analysis due to a lack of correlation with the P3.

#### *P3a and P3b*

The P3 is elicited by so-called “oddball tasks” in which stimuli are presented in random order, and one stimulus occurs less often than another (Verleger & Berg, 1991). In light of some interesting patterns that emerged during analysis of the P3 midline amplitude and latency site averages for the Auditory Perseveration task, a more in-depth exploration as to the loci of the “true” P3 wave that was elicited by this “oddball” task was seemingly merited. There has been much debate over the cognitive model of the main accepted components of the P3 wave, the fronto-central (Fz and Cz) “P3a” and the parietal (Pz) “P3b”. In this study, the Auditory Perseveration Task was found to elicit a fronto-central P3a waveform, as evidenced by peak amplitude at Cz and significantly



earlier latency at Fz (frontal) and Cz (central) compared to the Pz (parietal) electrode site. Almost all reliable reports of P3a waveforms involve the use of novel stimuli (like the white-noise burst in the AP task) that elicit an early, fast-habituating, fronto-central alerting response that most likely comes from neural loci (predominantly in the frontal lobe) that are related to fast attentional allocation which does not require deep processing (Posner, 1992; Posner & Petersen, 1990). When more memory processes are employed to store stimulus information for later use in the task, the later parietal P3b is elicited and is associated with greater target categorization (Knight, 1996; Polich & Squire, 1993).

P3 amplitude reflects the successful (or unsuccessful, if attenuated) apportioning of attentional resources to the task (Humphreys & Kramer, 1994). Bauer (1997), employing the Auditory Perseveration Task to elicit the P3 ERP, found that anterior P3a amplitude was a reliable predictor of relapse in individuals with cocaine dependence. A recent study by Anderson, Baldridge, and Stanford (2011) found that P3a amplitude was also predictive of substance abuse treatment program completion in a substance-dependent population. Examining current research, a strong case can be made that P3a amplitude could be interpreted as an index of executive function. For example, a relationship was found between P3a amplitude and thickness of the cortex that was found to be predictive of executive function (Fjell, Walhovd, Fischl, & Reinvang (2007). Decreased grey matter in the prefrontal cortex is often observed in psychopathology with executive dysfunction as a core feature. This reduction cortical thickness has been found in major depression (Vasic, Walter, Hose, & Wolf, 2008), antisocial behavior (Raine, Lencz, Bihrlé, LaCasse, & Colletti, 2000), schizophrenia (Kawada et al., 2009), substance abuse disorders (DeBellis et al., 2005), and impulsivity (Boes et al, 2009).

There are strong indications of a relationship between substance abuse and deficits in executive cognitive function (Dolan, Bechara, & Nathan, 2008), and it may be one of the key factors that lead to patients failing to complete treatment programs for substance abuse (McKellar, Kelly, Harris, & Moos, 2006). Fjell et al. (2007) found that cognitive function and age share a large portion of variance in accounting for P3a amplitudes, but thickness of the cortex still accounted for significant variation in P3a amplitudes while controlling for age. Bauer and Hesselbrock (2003) have posited that those at risk for Antisocial Personality Disorder and substance dependence have impaired development in areas of the frontal cortex as indicated by the absence of the predictable age-related increases that are normally seen in P3a amplitude through adolescence. Some studies have found that maturational fluctuations in frontal P3a characteristics that are observed in adulthood are accompanied by a decrement in executive function in older adults (Friedman, Nessler, Johnson, Ritter, & Bersick, 2008; West, Schwarb, & Johnson, 2010). It has been shown that the novelty of the stimulus is an important factor in generating P3a potentials, while P3b appears to indicate the involvement of memory systems in stimulus categorization (Polich, 2007). Fjell et al. (2007) constructed path models describing the relationships between cognitive function and the P3a and P3b ERPs and concluded that P3a is a more suitable indicator of executive function, whereas P3b is a more indicative of fluid function. Bauer (1997, 2001) found that differences in amplitude and source analyses of P3a indicate problems in prefrontal processes linked to behavioral inhibition in drug abusing individuals who are more likely to relapse. Moreover, in adolescents at high risk for ASPD and substance dependence, age-related deficits appear to be confined to the frontal cortex, whereas the parietal generators of P3b seem to mature at a normal

rate (Bauer and Hesselbrock, 2003). Anderson et al. (2011) suggest that it is reasonable to infer that a relationship between P3a amplitude and successful completion of a drug abuse treatment program is dependent on the utility of P3a amplitude as a neurological marker for deficits in executive processes in the frontal lobe, which result from inherited age-related deficits that may also influence cortical gray matter volume in this brain region critical for planning and behavioral control.

### *P3a Amplitude*

In the current study, more attenuated P3a (frontal averaged and central averaged) amplitude was found to be significantly correlated with a greater number of D-KEFS Word Context Repeated Incorrect Responses. In other words, smaller P3a amplitudes were found in individuals who continued to respond the same way to a question, even when the individual knew that the response was incorrect. These individuals, therefore, evidenced perseveration in the Word Context subtest by failing to change this response, though the “rule set” provided by the task merited a change in response. This is consistent with Bauer’s (1997, 2001) conceptualization that more attenuated P3a waveform could be used as a marker in individuals who are likely to evidence behavioral disinhibition and relapse. More impulsive responding (as evidenced by smaller P3a amplitudes) seemed to actually be beneficial on some tests of the D-KEFS. Through regression modeling, D-KEFS Twenty Questions Initial Abstraction Score was found to be a useful predictor of P3a averaged frontal amplitude. Individuals with smaller average P3a frontal amplitudes eliminated a greater number of potential choices by their first question asked on the Twenty Questions Task. Age, years of education, and D-KEFS Verbal Fluency Category Switching Total Switching Accuracy Score were found to be

useful predictors of averaged central amplitude. Higher Total Switching Accuracy scores were found to be correlated with smaller P3a amplitudes.

### *P3a Latency*

P3 latency is thought to be equal to the time it takes to evaluate the stimulus and take appropriate action (Kutas et al., 1977; Polich, 1987). It was found that longer P3a latencies result in more errors of omission (failing to make a response or not making a decision) while shorter P3a latencies result in more perseverative responses (staying with the previous response paradigm or failing to switch when another response rule is necessary; Bauer, 1997). The current study found similar results. Shorter central averaged latency was found to be significantly correlated with WCST Failure to Maintain Set (an indicator of a deficit in working memory). Predictably, shorter frontal and central averaged latency were also associated with a greater number of Perseverative Errors on the Auditory Perseveration Task. In addition, and notably, shorter frontal and central averaged latency was associated with a higher D-KEFS Trails All Error Types: N/L Scaled Score, indicating, again, that more perseverative responding is associated with shorter P3a latency.

Longer central averaged latency was significantly correlated with the WCST Learning to Learn score, indicating more efficient processing of the rules of the WCST in individuals who take longer to respond. Also similar to Bauer's 1997 findings, longer frontal and central averaged latencies were found to be significantly correlated with a greater Total Number of Omissions in the AP Task. In other words, the longer these individuals waited to respond, the greater the likelihood that these individual would in fact, fail to respond or not respond at all.

Examining regression models, D-KEFS Trails All Error Types N/L Scaled score was found to be a useful predictor of P3 averaged frontal latency, indicating that individuals making more perseverative errors evidenced a significantly shorter frontal latency. Understandably, faster, more impulsive responding was found to be linked with a failure to switch to a different response rule, when necessary. Conversely, Wisconsin Card Sort Task Learning to Learn scores were found to be useful predictors of longer averaged central latency. This demonstrates that individuals who took longer to evaluate the tasks and make a decision actually processed and learned the response rules of these tasks more effectively than those with shorter evaluation times.

#### *Limitations*

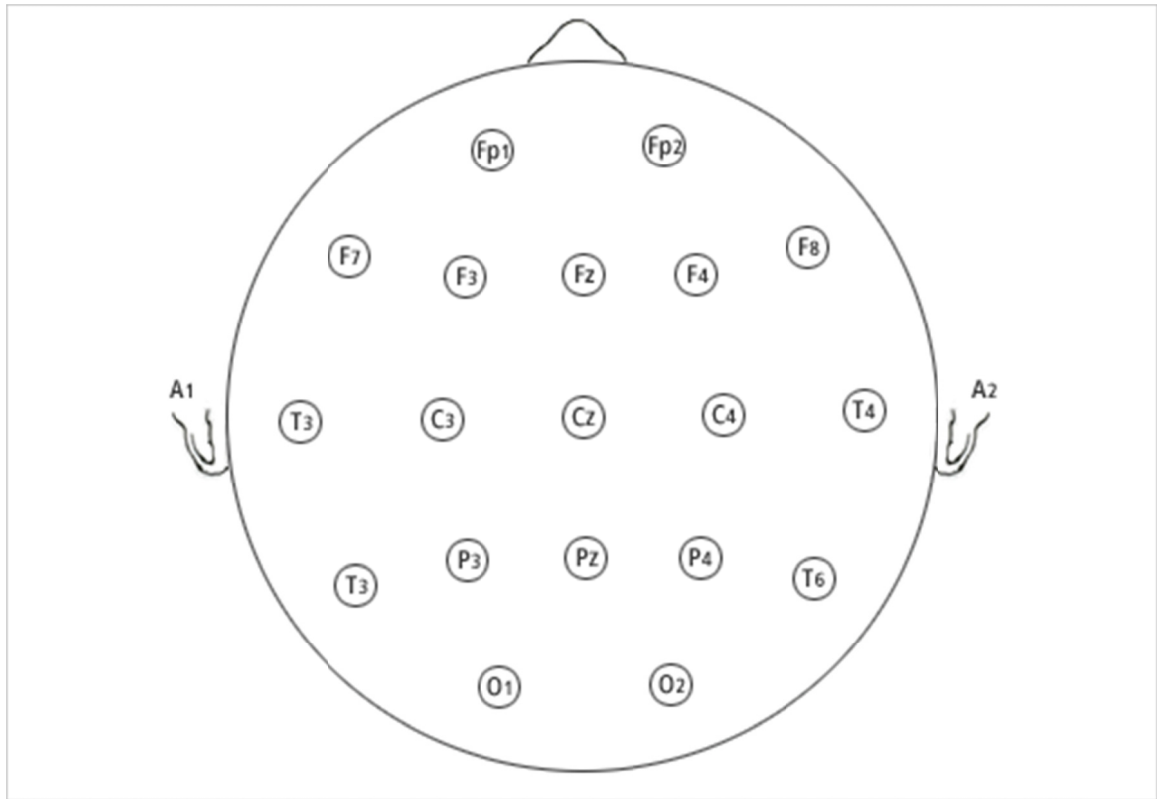
The current study encountered several limitations. The stringent exclusionary criteria limited the size of the sample to  $N = 66$ . Thirty-four of the original 100 participants were excluded from the study for reasons ranging from the likely presence of psychopathology to simple failure to report for the study (reasons for exclusion are delineated in the Results section). Findings would likely be more robust with a larger sample size, but due to time and budgetary constraints of the study, this was not possible. Also, because convenience sampling was used, the sample consisted mostly of Freshmen and Sophomore undergraduate students, as these students enrolled more often than Juniors and Seniors in the classes that offered research participation credit for the study. A more representative sample of equal numbers of Freshmen, Sophomores, Juniors, and Seniors would be preferable to be able to reasonably generalize to a larger college population.

### *Conclusions*

In conclusion, the main findings from this study reaffirm the usefulness of the P3a as a marker for deficits of executive cognitive function. Individual neuropsychological subtests were found to be useful in predicting P3a frontal and central averaged amplitude and latency. Bauer (1997) reported that anterior P3a amplitude was a reliable predictor of relapse in cocaine-dependent individuals and Anderson, Baldrige, and Stanford (2011) found that P3a amplitude was also predictive of substance abuse treatment program completion in a substance-dependent population. The results from this study indicate that employing neuropsychological tests of perseveration and omission as a pre-screening measure in drug abuse treatment programs might be a useful and more cost-effective way to divide the residents into groups based on presence or absence of executive cognitive dysfunction. After this distinction is made, drug treatment regimens could be more effectively tailored for the two groups.

## APPENDIX

### International 10-20 System Electrode Positioning



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