Disinhibition in South African Treatment-naïve Adolescents with Alcohol Use Disorders

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COMPULSORY DECLARATION

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, and has been cited and referenced.

Signature:_____ Date:_____

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Table of Contents

List of Figures

List of Tables

Abstract

The concept of the *disinhibitory complex* refers to a cluster of personality, psychiatric, cognitive, and electrophysiological inhibitory control impairments that have been documented in alcohol use disorders (AUDs). Previous AUD research has focused on treatment-seeking adults with comorbid psychopathology; with regard to the disinhibitory complex, such individuals are likely to differ from younger, treatment-naïve individuals, such as the sample here studied. Further, few studies have examined several domains of disinhibition simultaneously, and so little is known about relationships between the various correlates of disinhibition. This study aimed to (a) examine and characterize the disinhibitory complex in treatment-naïve adolescents with AUDs, (b) investigate sex differences in disinhibition, and (c) determine whether an underlying construct of disinhibition might explain elevated levels of disinhibition in AUDs observed on individual indices. One hundred and sixty-two adolescents: 81 (47 females) with AUDs but without diagnostically significant comorbid psychopathology; 81 (47 females) non- or light-drinking controls were assessed on measures of disinhibited personality (impulsivity, venturesomeness, novelty-seeking, excitement-seeking), externalizing psychopathology (sub-diagnostic threshold symptom counts for conduct disorder and oppositional defiant disorder), executive cognitive disinhibition (number of uncorrected and self-corrected errors on the inhibition task of the Stroop Color-Word test; number of rule violation errors on the Tower of London test); and electrophysiological correlates of disinhibition (amplitudes and latencies of the P300 and F-ERN components). The presence of an AUD was associated with inconsistent evidence of disinhibition; specifically, participants with AUDs were relatively disinhibited on cognitive and on some personality measures, but did not show, relative to controls, evidence of disinhibition on psychiatric, electrophysiological, or other personality measures. Males also showed inconsistent evidence of disinhibition relative to females (viz., they were relatively disinhibited on the psychiatric measure and on a single personality measure, but not on other measures). Further, attempts to model the construct of disinhibition using the selected indices were unsuccessful. These results suggest that disinhibition in AUD populations may be moderated by treatment and/or age factors. In addition, attempts to model the construct of disinhibition using the obtained data and the selected indices may have been hindered by the factor structure of the disinhibitory complex; it is possible that this complex has a hierarchical

structure, and that the manifestation of disinhibition may vary according to the domain examined as well as the method employed.

Keywords: disinhibitory complex; disinhibition; adolescence; alcohol use disorders

Introduction

The focus of this thesis is disinhibition in South African adolescents with alcohol use disorders. The construct of disinhibition refers to various deficits relating to inhibitory control and self-regulation (Iacono, Malone, & McGue, 2003; Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009). Because disinhibition exists as a result of impairments in certain faculties, the discussion of disinhibition presented here is preceded by an introduction to those faculties and to their underlying processes.

Cognitive Control

Cognitive control, a central feature of higher-order cognition, is an executive function responsible for the control of cognitive operations (Posner & DiGirolamo, 1998). Essentially, cognitive control is an attentional process that configures behavior in accordance with task-specific goals (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Wendelken, Ditterich, Bunge, & Carter, 2009). Executive behavioral configuration is essential in everyday functioning because the environments in which we function are complex and changing constantly (MacDonald, Cohen, Stenger, & Carter, 2000). Cognitive control addresses the complexities of everyday living by guiding voluntary and complex elements of cognition and behavior (Luna, Padmanabhan, & O'Hearn, 2010).

The capacity for cognitive control is most essential in difficult, complex, or novel tasks where automatic responses must be overruled (Curtin & Fairchild, 2003). This is because natural and well-trained behaviors are typically rigid responses to sensory stimuli, resisting generalizations to novel situations. Therefore, successful execution of voluntary behavior is contingent on top-down control to override automatic behaviors (Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004).

Mechanisms of cognitive control. The cognitive monitoring hypothesis is one way to understand the mechanisms involved in the implementation of cognitive control. According to this hypothesis, conflicts presenting in the information-processing stream signal an alarm calling for increased engagement of control processes (Botvinick et al., 2001). Processing conflict, or pre-response conflict, refers to conflict that occurs during processing (i.e., before a response is made or an action is carried out) when a stimulus activates more than one response tendency

concurrently (Kerns et al., 2004; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Such a situation may occur when a stimulus primes a prepotent but undesirable response, or when a stimulus primes several responses, none of which is more compelling than the others. Alternatively, conflict may arise following an erroneous response or action, where behavioral outcomes following a response are incongruent with the behavioral goals set out prior to response (Botvinick et al., 2001; Cavanagh, Cohen, & Allen, 2009). This type of conflict is known as response conflict or post-response conflict.

Constituents of the cognitive control system. According to the conflict monitoring hypothesis, the cognitive control system facilitates adaptive, flexible behavior using two constituents: a *supervisory/evaluative constituent* that monitors performance and signals the presence of conflicts indicative of a need for adjustments in control, and a *regulatory constituent* that biases executive control resources towards presenting conflicts to adjust and coordinate goal-directed behavior so that performance is optimized (Botvinick et al., 2001; Carter, Botvinick, & Cohen, 1999; Ridderinkhof, van den Wildenberg et al., 2004; van Veen & Carter, 2006). Although the exact neuroanatomical location of the structures supporting cognitive control is the subject of some debate (Ridderinkhof, Ullsperger et al., 2004), converging neuroimaging evidence points to dual involvement of the anterior cingulate cortex (ACC) and the dorsolateral prefrontal cortex (dlPFC) (Egner, Delano, & Hirsch, 2007; Kerns et al., 2004; MacDonald et al., 2000).

According to the conflict monitoring hypothesis, the ACC comprises the performance monitoring constituent of the cognitive control system. Within the cognitive control system, the primary function of the ACC is to detect pre- and post-response conflict (Carter et al., 1998; Cavanagh et al., 2009). Once conflict is detected, the ACC activates the dlPFC. The latter refers to the regulatory constituent of the cognitive control system; its function is to maintain the requirements of the current task, and to prompt both neural and behavioral adjustments to optimize performance of the task (van Veen & Carter, 2006; Wendelken et al., 2009). The dlPFC performs this role by recruiting additional cognitive resources to address difficulties in processing (i.e., to resolve conflict). Specifically, the dlPFC sends signals to subcortical and posterior cortical brain regions which in turn configure processing in line with current task demands and goals (Ridderinkhof, van den Wildenberg et al., 2004). The recruitment of

additional resources leads to increases in control over cognitive, motor, or emotion systems as necessary to optimize performance (Cavanagh et al., 2009).

The Stroop Color-Word test (SCWT; Stroop, 1935) is used routinely to assess conflict detection and resolution capacities (e.g., Dolan, Bechara, & Nathan, 2008; Kerns et al., 2004). In the inhibition task of the SCWT, participants are presented with a list of the names of three colors (red, green, and blue) printed in either a color congruent with the word (e.g., the word 'blue' printed in blue ink) or a color incongruent with the word (e.g., the word 'green' printed in red ink). They are instructed to say aloud the ink color of each item in succession, and to avoid reading the word. In this task, conflict may both precede (i.e., pre-response conflict) and follow (i.e., post-response conflict) incongruent trials (i.e., those trials where the ink color and word are discrepant). Prior to response on incongruent trials in the inhibition task, high pre-response conflict is created by the simultaneous engagement of competing incompatible responses (i.e., the prepotent response, which is to simply read the word, and the correct response, which is to state the color of the ink) (Yücel & Lubman, 2007). In order to produce the correct response, the participant must comply with task-specific goals by inhibiting the prepotent response, thus resolving the pre-response conflict.

Post-response conflict may arise when pre-response conflict is resolved inadequately and an incorrect response is produced. If ongoing stimulus analysis following provision of an incorrect response reveals the desired response, post-response conflict arises due to interference between the correct (desired) and incorrect (produced) response (Botvinick et al., 2001). Post-response conflict is thus elicited due to a discrepancy between the incorrect, produced response and the correct, desired response.

MacDonald and colleagues (2000) used a modified version of the Stroop paradigm to show that the ACC and dlPFC perform distinct but complementary roles in the neural network supporting cognitive control. By temporally separating the provision of instructions (i.e., to name either the color or name the word) from the response to each respective trial, the authors were able to differentially examine instruction-related strategic processes and response-related evaluative processes. Their findings showed a dissociation between the ACC and dlPFC. The dlPFC was activated selectively during the provision of instructions, and, in particular, when the instruction was to name the color (which is the comparatively difficult task). This observation supports the idea that the dlPFC is employed to engage additional cognitive resources in

anticipation of cognitive challenges. On the other hand, the ACC was engaged selectively around the time of response, when pre- or post-response conflict may occur. Activation of the ACC was relatively increased on incongruent trials in comparison with congruent trials, where pre-response conflict was heightened and post-response conflict (due to error commission) was more likely. This finding illustrates that the ACC is involved in conflict monitoring. Indeed, fMRI studies show that the ACC responds to conflict (Botvinick et al., 2001).

Hence, it appears that the ACC and dlPFC support distinct but complementary processes in the neural network underlying cognitive control. These processes are not, however, linear; instead, they operate in a dynamic fashion in what is termed the 'conflict-control loop', where neither process necessarily precedes the other (Kerns et al., 2004; MacDonald et al., 2000).

Consequences of compromised cognitive control. Successful execution of cognitive control relies on the ability to (a) recognize that competing responses exist (i.e., detect conflict), and (b) bias executive resources toward selection of the appropriate response (i.e., resolve conflict). Following the conflict monitoring hypothesis, impairments in these abilities may manifest in two different ways. At the level of pre-response conflict, individuals with reduced cognitive control capacity are less equipped to detect and to appropriately manage competing response options in the processing stream. As a result, these individuals are less adept at guiding voluntary actions according to task objectives, and are more likely to favor automatic responses (van Veen & Carter, 2006). At the level of post-response conflict, individuals with reduced cognitive control capacity are less adept at detecting when they have made an error or when a given response or behavior does not comply with task objectives; hence, these individuals are less likely to provide a correcting response or behavior (Shuster & Toplak, 2009).

The hallmark of compromised cognitive control is a deficit in self-regulation, which manifests as behavioral and cognitive inhibitory control impairments (Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009). Inhibitory control thus hinges on cognitive control capacity (in particular, the ability to detect and resolve pre-response conflict). Impairments in inhibitory control are commonly described using the term "disinhibition" (Iacono, Malone, & McGue, 2003). A primary feature of disinhibition is the tendency to act upon acute impulses (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2008). These actions are "poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and often result in undesirable outcomes" (Evenden, 1999, p. 1).

The Latent Disinhibitory Complex

Clinical correlates of disinhibition include disinhibited personality traits, substance use disorders (SUDs), externalizing psychopathology (e.g., conduct disorder (CD), oppositional-defiant disorder (ODD)), executive cognitive dysfunction, as well as changes in certain electrophysiological (EEG) components (Begleiter & Porjesz, 1999; Chen et al., 2007; Fein & Chang, 2008; Finn & Hall, 2004; Iacono et al., 2003; Porjesz & Rangaswamy, 2007). Furthermore, a large body of evidence has documented individual associations between the various correlates of disinhibition (e.g., Hopfer, Crowley, & Hewitt, 2003; Kamarajan et al., 2010; Krueger et al., 2002; Slutske et al., 1998; Slutske et al., 2002). In response to this accumulating evidence, contemporary theories suggest that the relationships between these correlates may be better conceptualized in terms of a broad, continuous, and latent dimension of disinhibition (Begleiter & Porjesz, 1999; Finn et al., 2009; Gorenstein & Newman, 1980; Kendler, Prescott, Myers, & Neale, 2003; Krueger et al., 2002; Krueger & Markon, 2006; Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004; Zernicke, Cantrell, Finn, & Lucas, 2010). Notably, Iacono et al. (2003) propose that a common genetic susceptibility to disinhibition underlies the manifestation of disinhibited personality traits, various externalizing and disruptive disorders (including alcohol use disorders (AUDs) and other externalizing psychopathologies[1]), as well as changes in certain EEG components. Further, the proposition is that the common susceptibility to disinhibition also underlies executive cognitive dysfunction observed on neuropsychological assessment (Finn, 2002; Finn & Hall, 2004; Giancola, Martin, Tarter, Pelham, & Moss, 1996).

The notion of a latent disinhibitory complex has two important implications. First, it implies that a spectrum of externalizing pathologies and behaviors, as well as certain cognitive impairments and EEG changes, arise from a latent and generalized complex. With regard to externalizing pathology, comorbidity rates for externalizing disorders (notably CD, ODD, and SUDs) are notoriously high, supporting the notion of a single latent dimension (Armstrong &

[1]According to the DSM-IV-TR (American Psychological Association, 2000) SUDs are nosologically distinct from traditional externalizing psychopathologies such as CD and ODD. However, due to the notion that SUDs and traditional externalizing psychopathologies may arise from a common and latent disinhibitory complex, SUDs are generally considered a form of externalizing psychopathology in the SUD literature (e.g., Krueger et al., 2002; Zernicke et al., 2010). In line with this convention, AUDs are considered a form of externalizing psychopathology for the purposes of this manuscript.

Costello, 2002; Grant et al., 2004; Hedden et al., 2010). In a large-scale meta-analysis of twin studies based on a total sample of approximately 23 000 individuals, Krueger et al. (2002) modeled the covariance among alcohol dependence, drug dependence, and the elements of antisocial personality disorder (CD and antisocial behavior after age 15) using a single latent externalizing factor. Here, the mean level change for the latent factor was larger than for any individual disorder. This finding indicates that comorbidity among externalizing behaviors (including externalizing disorders and SUDs) is likely due to the influence of a single underlying externalizing factor.

In addition, externalizing psychopathology commonly co-occurs with disinhibited personality traits (Krueger et al., 2002), as well as with both executive function impairments and certain EEG characteristics (e.g., Bauer & Hesselbrock, 2003; Finn et al., 2009; Porjesz et al., 2005). Prospective studies have shown that disinhibited personality traits, as well as changes in certain EEG components, predict future onset for a range of externalizing pathologies, including AUDs (Begleiter, Porjesz, Bihari, & Kissin, 1984; Elkins, King, McGue, & Iacono, 2006; Hill, Steinhauer, Locke-Wellman, & Ulrich, 2009; Sher, Bartholow, & Wood, 2000). Taken together with findings supporting the notion of a single underlying externalizing factor, these findings support the notion of a latent and generalized disinhibitory complex, based on the observations that (a) various externalizing behaviors arise from a single latent complex, and (b) disinhibited personality, executive function impairments, and EEG changes commonly co-occur with externalizing psychopathology.

Second, this position proposes that the underlying disinhibitory complex is heritable. Indeed, disinhibition appears to be a core feature and general risk factor for a spectrum of externalizing disorders and disinhibited personality traits (Hopfer et al., 2003; Krueger et al., 2002). For example, alcoholism is well-established as a heritable disorder (Begleiter & Porjesz, 1988; Dick et al., 2010; Knopik et al., 2004). Heritability rates estimate that the offspring of a single alcoholic parent are approximately four times more likely to develop alcoholism than the offspring of non-alcoholic parents (Goodwin, 1979; Knopik et al., 2004). Children at familial risk for developing AUDs (i.e., with a family history of AUDs) consistently demonstrate externalizing tendencies, disinhibited personality traits, and changes in EEG components associated with chronic alcohol use, despite never having consumed alcohol themselves

(Begleiter et al., 1984; Carlson & Iacono, 2008; Finn, Sharkansky, Brandt, & Turcotte, 2000; Hill, Steinhauer, Lowers, & Locke, 1995; Knopik et al., 2004).

In summary, disinhibited personality, externalizing psychopathology, diminished executive cognitive capacity, and changes in certain EEG components co-occur commonly. Studies using at-risk populations have highlighted common genetic links between these phenomena. It therefore appears that these phenomena might constitute different manifestations of a common disinhibitory complex.

Measurement of Disinhibition

Both within and across fields of research, there is a lack of consensus regarding how to define and measure disinhibition (Dick et al., 2010; Yücel, Lubman, & Pantelis, 2004). These inconsistencies in definition and measurement of disinhibition arise for two primary reasons: First, disinhibition may be a latent construct and, therefore, it cannot be measured directly; second, and as mentioned previously, disinhibition may manifest in numerous ways, including disinhibited personality traits, externalizing psychopathology, and executive cognitive dysfunction. Diversity in the various manifestations of disinhibition certainly complicates the issues of definition and measurement. Because disinhibition is probably a *latent, multifactorial* construct, researchers measure the construct using *various indirect* means. Specifically, disinhibition may be measured by examining its clinical correlates, including disinhibited personality traits, externalizing psychopathology, executive cognitive dysfunction, and certain EEG components (Begleiter & Porjesz, 1999; Chen et al., 2007; Fein & Chang, 2008; Finn & Hall, 2004; Iacono et al., 2003; Porjesz & Rangaswamy, 2007).

Furthermore, the correlates of disinhibition may be defined as either phenotypes (i.e., the disorder as it appears) or endophenotypes (i.e., functions that underlie a disorder) of an underlying disinhibitory complex (Goudriaan et al., 2008). A phenotype generally indexes a behavioral manifestation of a disorder. An endophenotype, on the other hand, is an endogenous attribute of an individual that is a direct product of the predisposing genotype (Iacono, Malone, & McGue, 2008). Endophenotypes are useful indices of genetic psychopathological risk, and are thought to supersede clinical diagnosis in estimations of genetic influence (Gottesman & Gould, 2003; Iacono et al., 2003; Porjesz & Rangaswamy, 2007). Nevertheless, it appears that disorders

are optimally characterized using a combination of phenotypical and endophenotypical indicators (Evenden, 1999; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001).

Disinhibition in AUDs

According to the DSM-IV-TR (American Psychological Association, 2000), the AUDs consist of alcohol abuse and alcohol dependence. *Alcohol abuse* is defined as a maladaptive pattern of alcohol use leading to at least one problem with social or occupational functioning within a 12-month period. Examples of problems with functioning include ongoing legal difficulties, consumption of alcohol in hazardous situations, failure in obligations, and ongoing use despite interpersonal or social problems. *Alcohol dependence* is the more severe of the two conditions; such a diagnosis is given when there is evidence of tolerance, withdrawal, or compulsive behavior as a result of alcohol use. This disorder is defined as including problems with functioning in three or more areas of life as a result of ongoing alcohol use. The DSM-IV criteria for AUDs are reliable across different age groups, sexes, and most cultural groups (Schuckit, 2009).

Disinhibition is a core underlying feature of AUDs (Finn, 2002; Gorenstein & Newman, 1980; Porjesz & Rangaswamy, 2007; Zuckerman, 1979). Indeed, individuals with AUDs demonstrate impaired functioning in the regions supporting cognitive control (Beck et al., 2009; Yücel & Lubman, 2007). Alcohol consumption results in acute decreases in inhibitory control (Marczinski, Abroms, Van Selst, & Fillmore, 2005). More importantly, disinhibition may constitute both an antecedent and a consequence of chronic alcohol use (D. B. Clark, Vanyukov, & Cornelius, 2002; Dawes, Tarter, & Kirisci, 1997; Koob & Le Moal, 1997). In other words, (a) elevated levels of disinhibition increase vulnerability for the development of an AUD, and (b) chronic alcohol consumption may induce or magnify levels of disinhibition (Dick et al., 2010; Sher & Trull, 1994; Verdejo-García, Lawrence, & Clark, 2008).

Disinhibited personality in AUDs. Disinhibited personality is a core element of all externalizing disorders, including AUDs (Finn, Mazas, Justus, & Steinmetz, 2002; Masse & Tremblay, 1997). Individuals with AUDs thus exhibit elevated trait levels of disinhibited personality (Tarter et al., 2003). Like AUDs, disinhibited personality is regarded as a phenotype (i.e., a behavioral manifestation) of an underlying disinhibitory complex.

In general terms, the primary features of disinhibited personality include non-planning, liveliness, and risk-taking (Acton, 2003; Finn & Hall, 2004). No single personality trait accounts for all the primary features of disinhibited personality (G. T. Smith et al., 2007; Whiteside & Lynam, 2001). Instead, there are several closely-related forms of disinhibited personality, each of which accounts for individual aspects of this personality type. Variants of disinhibited personality include impulsivity, venturesomeness, novelty-seeking and excitement-seeking, all of which are associated with AUDs and each of which can be measured separately, using self-report questionnaires (Finn & Hall, 2004; Grekin, Sher, & Wood, 2006; Justus, Finn, & Steinmetz, 2001). *Impulsivity* is thought to reflect a problem with inhibitory control, possibly related to increased sensitivity to rewards (Cloninger, 1987a; Finn, Justus, Mazas, & Steinmetz, 1999; Logan, Schachar, & Tannock, 1997). *Venturesomeness* is associated with a tendency toward adventurous or exciting activities (Eysenck & Eysenck, 1978). *Novelty-seeking* represents an inclination toward exploratory activity, and considerable excitement in response to novelty (Cloninger, Svrakic, & Przybeck, 1993; Laucht, Becker, Blomeyer, & Schmidt, 2007). *Excitement-seeking* is associated with pleasure seeking tendencies and difficulty tolerating boredom (Finn et al., 2000).

Elevated levels of disinhibited personality promote alcohol use, prompting the onset of alcohol consumption, and increasing severity of alcohol use problems (Tarter et al., 2004). Disinhibited personality thus predicts increased levels of substance use disorders among adolescents (Laucht et al., 2007; Luengo, Carrillo-de-la-Pena, Otero, & Romero, 1994), young adults (Finn, Bobova, Wehner, Fargo, & Rickert, 2005; Finn et al., 2002; Sher, Wood, Crews, & Vandiver, 1995; Zernicke et al., 2010), and older adults (J. M. Mitchell, Fields, D'Esposito, & Boettiger, 2005).

Externalizing psychopathology in AUDs. AUDs usually occur in the context of other externalizing psychopathology, such as CD, ODD, and attention-deficit/hyperactivity disorder (ADHD) (Armstrong & Costello, 2002; Kuperman et al., 2001; McGue, Slutske, Taylor, & Iacono, 1997; Reebye, Moretti, & Lessard, 1995). Childhood externalizing disorders heighten risk for early-onset SUDs (Hawkins, Catalano, & Miller, 1992; Wilens & Biederman, 1993). In fact, CD mediates the pathway between childhood ADHD and SUD in young adulthood (Biederman et al., 1997; Fergusson, Lynskey, & Horwood, 1993; Lynskey & Fergusson, 1995). Externalizing psychopathology appears to encourage affiliation with deviant peers, thus

increasing risk for SUDs in adolescence (Moss, Lynch, & Hardie, 2003). Furthermore, sub-diagnostic threshold externalizing behaviors, such as social deviance, predict alcohol consumption and AUDs in adolescence (Gerra et al., 2004).

A number of twin studies have pointed to common genetic factors linking AUDs and other externalizing disorders (e.g., Grove et al., 1990; Pickens, Svikis, McGue, & LaBuda, 1995). For instance, based on a sample of 2 682 twin pairs, Slutske et al. (1998) found that heredity accounted for over 70% of the observed association between CD and alcohol dependence. Further, and as mentioned previously, AUDs and certain externalizing psychopathologies are thought to exist as different manifestations of a common disinhibitory complex (Iacono et al., 2008). Externalizing psychopathology is thus another phenotype of the underlying disinhibitory complex,

Cognitive disinhibition in AUDs. Cognitive disinhibition, or cognitive impulsivity, is an endophenotype of the proposed underlying disinhibitory complex (Goudriaan et al., 2008). In other words, inhibitory impairment at a cognitive level underlies the behavioral manifestations (e.g., disinhibited personality, externalizing psychopathology including AUDs) of the underlying disinhibitory complex (Dolan et al., 2008; Reynolds, Ortengren, Richards, & de Wit, 2006). Researchers in this field have employed both neuropsychological and electrophysiological indicators to examine cognitive disinhibition.

Neuropsychological indicators of cognitive disinhibition: Response inhibition. Response inhibition may be defined as "the ability to voluntarily select a task-appropriate, goal-directed response while suppressing a more compelling, but task-inappropriate response" (Luna et al., 2010, p. 1). This capacity is an integral feature of voluntary action: Flexible behavior relies on the ability to inhibit some responses and select others, according to task-specific goals (Luna & Sweeney, 2004; E. K. Miller & Cohen, 2001). For example, response inhibition is required to suppress a competing response plan, one or another simultaneously-activated cognitive process during dual processing, or distractions irrelevant to the task at hand. Behavior control and self-regulation rely on preserved response inhibition capacity (Barkley, 1997; Nigg et al., 2006). Impairments in response inhibition therefore contribute to impulsive, stimulus-driven behavior characteristic of disorders associated with the disinhibitory complex (including CD, ADHD, and AUDs; Barkley, 1997; Dick et al., 2010; Nigg, 2001; Nigg et al., 2006).

Disturbances of the cognitive control system (and specifically of the ACC) may lead to an impaired capacity to (a) engage control in high-conflict situations, and to thus (b) resolve pre- and post-response conflict. These disturbances constitute a form of executive cognitive disinhibition. Indeed, executive cognitive disinhibition has been noted in individuals with AUDs, as well as in children at genetic risk for developing AUDs (Aytaclar, Tarter, Kirisci, & Lu, 1999; Dolan et al., 2008; Giancola et al., 1996; Habeych, Folan, Luna, & Tarter, 2006; Harden & Pihl, 1995; Peterson, Finn, & Pihl, 1992; Silveri, Rogowska, McCaffrey, & Yurgelun-Todd, 2011).

Executive cognitive disinhibition (response inhibition deficits, in particular) may be detected by several neuropsychological tests. Regarding the SCWT, an impaired capacity to resolve pre-response conflict leads to error commission; an impaired capacity to resolve post-response conflict results in a decreased ratio of self-corrected to total errors (Stuss, Floden, Alexander, Levine, & Katz, 2001; van Veen & Carter, 2006; Verdejo-García et al., 2010). When stimulus analysis continues following an erroneous response, a self-correcting response is contingent on adequate management of post-response conflict. If such management is compromised, the individual is inadequately equipped to provide a self-correcting response (Shuster & Toplak, 2009). Nevertheless, both uncorrected and self-corrected errors on the SCWT signify an initial inability to inhibit the prepotent response (i.e., impaired resolution of pre-response conflict). Uncorrected and self-corrected errors on the SCWT thus provide an index of response inhibition (Fillmore, 2004; Fillmore & Vogel-Sprott, 1995).

Like the SCWT, successful performance on the Tower of London test (ToL; Culbertson & Zillmer, 2001) relies on top-down cognitive control to direct responses according to task-specific goals (Jurado & Rosselli 2007). Number of rule violation errors on the ToL is another measure of response inhibition. In the ToL, participants are given examples of rule violation behaviors (i.e., they are told that moving more than one bead at a time, or placing more than the designated number of beads on a particular peg, constitutes a rule violation), and they are instructed to avoid such behaviors (Culbertson & Zillmer, 2001). Avoiding rule violation errors is thus a salient goal on the ToL. Response inhibition impairments are related to difficulties suppressing inappropriate responses (e.g., rule violating behaviors), as well as problems directing behavior according to task-specific goals (e.g., avoiding rule violation errors) (Botvinick et al., 2001; Possin et al., 2009; Wendelken et al., 2009). These difficulties therefore lead to increased frequency of rule violation errors on the ToL. Hence, taken together, errors (uncorrected and

self-corrected) on the SCWT and rule violation errors on the ToL are useful indicators of response inhibition capacity.

In summary, impaired response inhibition is a likely origin of the kinds of dysregulated behavior noted in disorders associated with the disinhibitory complex. Furthermore, response inhibition impairments are observed in individuals at genetic risk for developing AUDs. Response inhibition impairment is thus considered an endophenotype of the underlying disinhibitory complex.

Electrophysiological indicators of cognitive disinhibition: The P300 component. The P3 or P300 component is a long-lasting, positive event-related potential (ERP) peaking between 200 and 600 ms following presentation of a salient stimulus or novel event (Davies, Segalowitz, Dywan, & Pailing, 2001; Ridderinkhof, Ramautar, & Wijnen, 2009). The P300 comprises two related subcomponents, namely the P3a and the P3b. Although the topography of these subcomponents (a) naturally varies among individuals, and (b) appears to be influenced by task-related factors, such as task difficulty, topography is one factor that is used to distinguish the subcomponents of the P300 (Comerchero & Polich, 1998, 1999; Hagen, Gatherwright, Lopez, & Polich, 2006).

The P3a subcomponent has a general frontal distribution, and occurs between 200 and 280 ms following presentation of a novel but *task-irrelevant stimulus* (Hansenne, 2006). For example, the P3a is elicited by infrequent or unpredictable shifts in background stimuli (e.g., auditory tones) while a subject is ignoring the tones and attending to the relevant stimulus (e.g., reading a book). The P3b subcomponent, on the other hand, occurs later (between 280 and 600 ms following stimulus presentation), has a more centro-parietal distribution, and is elicited when an individual attends to a novel and *task-relevant stimulus*. For example, the P3b is elicited in response to infrequently-presented auditory tones, provided the subject is instructed to respond to such stimuli. For the purposes of the current manuscript, the terms ‘P300’ and ‘P300 component’ will be used to refer to the P3a and P3b subcomponents together (and not simply to the P3b subcomponent, as is often the case).

Meaning of the P300. Various cognitive processes related to attention and memory operations have been proposed to explain the origins of the P3a and P3b subcomponents (Desmedt, 1980; Donchin & Coles, 1998; Verleger, 1997). Notably, however, the P300 is thought to signal the activation of neural inhibitory processes over cortical areas (M. E. Smith et

al., 1990; Tomberg & Desmedt, 1998; Verleger, 1988). The inhibition of ongoing, extraneous neural activity is needed to enhance focal attention and to facilitate the transmission of incoming stimulus information (Knight, 1997; Polich, 2007). In other words, the P300 may signify inhibitory processes facilitating selective attention. Preserved P300 represents efficient inhibition of neural processes; relatively attenuated amplitudes and prolonged latencies of the P300 signify impaired neural inhibition.

The association between the P300 and inhibition extends beyond the neural scale. Changes in the P300 have been reported in individuals with elevated levels of disinhibited personality (Ratsma, van der Stelt, Schoffelmeer, Westerveld, & Boudewijn Gunning, 2001), and individuals with externalizing psychopathology (including AUDs) (Carlson, Katsanis, Iacono, & Mertz, 1999; Fein & Chang, 2006; Iacono, Carlson, Malone, & McGue, 2002; Maurage et al., 2007; Porjesz & Begleiter, 1996), as well as in young offspring of alcoholic parents who have never consumed alcohol themselves (Begleiter et al., 1984; Hill, Shen et al., 1999). Furthermore, recent studies have shown both increased levels of impulsivity and reductions in P300 amplitude in individuals with AUDs (Chen et al., 2007; Justus et al., 2001). Mediation analyses have further indicated that the relation between P300 amplitude and AUDs is partially mediated by impulsivity. Reduced frontal cortical inhibition may thus predispose individuals to be impulsive, which, in turn, may be responsible for increasing vulnerability for developing AUDs (Justus et al., 2001).

In summary, the P300 may provide an indication of the integrity of inhibition processes at both neural and behavioral levels. Further, changes in the P300 have been observed in various disorders associated with the disinhibitory complex (including AUDs), as well as among those at genetic risk for developing AUDs. Together, these findings have led to the hypothesis that certain changes in the P300 are indicative of an underlying and heritable disinhibitory complex (Iacono et al., 2002; Porjesz et al., 2005; Tomberg & Desmedt, 1998). In other words, the P300 is believed to be an endophenotype of the proposed underlying disinhibitory complex. Although reduced amplitude and prolonged latency in both the P3a and P3b subcomponents are considered endophenotypical markers for the disinhibitory complex, these changes are observed most consistently for P3b amplitude (Cohen, Wang, Porjesz, & Begleiter, 1995; Maurage et al., 2007).

Electrophysiological indicators of cognitive disinhibition: The F-ERN component. The error-related negativity (ERN) is a negative ERP component with a fronto-central scalp distribution. Traditionally, it is thought to index awareness of error commission (Davies et al., 2001; Taylor, Stern, & Gehring, 2007). When individuals do not require external feedback to know that an error has occurred (e.g., in the SCWT), the ERN arises 50-150 ms following error commission and is called the response-locked error-related negativity (R-ERN) (Dikman & Allen, 2000; Hajcak & Simons, 2002). However, when feedback is required for evaluation of the correctness of a response (e.g., in a gambling-type task where the correctness of an action relies on its outcome), the ERN is elicited 200-300 ms following performance feedback and is known variously as the feedback error-related negativity (F-ERN), feedback-related negativity, medial frontal negativity, or outcome-related negativity (Hajcak, Moser, Holroyd, & Simons, 2006; Onoda, Abe, & Yamaguchi, 2010).

Meaning of the F-ERN. Similar to the P300, various theories have been proposed to explain the mechanisms of the R-ERN and F-ERN. Whereas the conflict monitoring hypothesis proposes that the R-ERN and F-ERN signify post-response conflict arising due to discrepancies between intended and actual outcome, reinforcement feedback hypothesis proposes that these ERPs signify dopaminergic fluctuations arising due to disparities between intended and actual rewards (Botvinick et al., 2001; Carter et al., 1998; de Bruijn, de Lange, von Cramon, & Ullsperger, 2009; Holroyd & Coles, 2002; Houk, Adams, & Barto, 1995; Nieuwenhuis, Holroyd, Mol, & Coles, 2004; Yeung, Botvinick, & Cohen, 2004). The traditional error monitoring hypothesis, and more recent conflict monitoring and reinforcement feedback hypotheses, remain plausible, providing competing explanations of the ERN (Bush, Luu, & Posner, 2000; Emeric et al., 2008). Despite offering different accounts, these theories are consistent in stating that the R-ERN and F-ERN reflect online performance monitoring elicited in response to performance outcomes (errors and negative feedback, respectively).

Efficient processing of performance outcomes is central to adaptive decision-making (Fein & Chang, 2008). Hyposensitivity to negative feedback leads to poor or risky decision-making, which is, in turn, a common feature of disorders associated with disinhibition (L. Clark & Robbins, 2002; T. J. Crowley, Raymond, Mikulich-Gilbertson, Thompson, & Lejuez, 2006; Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2005). Notably, risky decision-making is a

characteristic of AUDs, and is believed to underlie the persistence of harmful behaviors despite mounting negative consequences of the behaviors associated with these disorders.

Recent work suggests that the F-ERN is a measure of sensitivity to negative feedback, with weaker F-ERN responses indicating reduced sensitivity and stronger F-ERN responses indicating increased sensitivity (Fein & Chang, 2008). With clear associations to risky decision-making, it is not surprising that the R-ERN and F-ERN are reduced in AUD populations. With regard to the F-ERN, Kamarajan et al. (2010) found a trend towards reduced amplitudes in males with AUDs relative to healthy controls, while Fein and Chang (2008) reported a negative association between F-ERN amplitude and family history density of alcohol problems (proportion of first-degree relatives with alcohol problems).

In summary, changes in the F-ERN likely index hyposensitivity to negative feedback, a major contributor to risky decision-making observed in disorders associated with disinhibition. Indeed, the association between F-ERN reductions and trait impulsivity has been demonstrated previously (e.g., Kamarajan et al., 2010; Yamaguchi, Onoda, & Abe, 2011). Furthermore, changes in the F-ERN have been observed among those at genetic risk for developing AUDs. The F-ERN (and F-ERN amplitude reductions, in particular) is thus considered an endophenotype of the underlying disinhibitory complex.

Rationale for Research

Scope of Disinhibition Studies

Disinhibition is a multifactorial phenomenon. AUDs constitute only a single manifestation of the disinhibitory complex. Although researchers (e.g., Evenden, 1999; Moeller et al., 2001) have noted that disinhibition in AUDs is best characterized using both phenotypical and endophenotypical indicators of the underlying disinhibitory complex, most studies focus on either disinhibited personality (i.e., personality domain; e.g., Soloff, Lynch, & Moss, 2000), other externalizing psychopathology (i.e., psychiatric domain; e.g., Armstrong & Costello, 2002), neuropsychological indicators of cognitive disinhibition (i.e., executive cognitive domain; e.g., Lawrence et al., 2009), or electrophysiological indicators of cognitive disinhibition (i.e., EEG domain; e.g., Carlson et al., 1999).

Because disinhibition is often examined in limited terms in the AUD literature (and elsewhere), associations between the various indices of disinhibition have not been investigated thoroughly. Although several studies have measured associations or modeled disinhibition using indicators derived from two or three domains of disinhibition (i.e., personality, psychiatric, cognitive, EEG) (e.g., Beck et al., 2009; Bjork, Hommer, Grant, & Danube, 2004; Dolan et al., 2008; Enticott, Ogloff, & Bradshaw, 2006; Finn et al., 2002; Justus et al., 2001; J. M. Mitchell et al., 2005), many of these studies are limited in that they used small sample sizes and focused on male participants only. Furthermore, despite the fact that disinhibition may be measured over several domains, I am unaware of any studies attempting to model disinhibition using disinhibited personality, externalizing psychopathology, executive cognitive disinhibition, and EEG correlates of disinhibition. There is thus a knowledge gap with regard to more multidimensional characterization of disinhibition in AUDs.

Disinhibition in Adolescents

Disinhibition is relatively understudied within adolescent populations (Velanova, Wheeler, & Luna, 2008). This may be because cognitive control capacity has a protracted developmental trajectory extending from childhood to adulthood, where children and adolescents have underdeveloped cognitive control capacities relative to adults (Arbel & Donchin, 2011; Culbertson & Zillmer, 2001; Davies, Segalowitz, & Gavin, 2004; Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010; Levin, Culhane, Hartmann, Evankovich, & Mattson, 1991; Rand,

Wapner, Werner, & McFarland, 1963; Steinberg et al., 2008; Velanova et al., 2008; Yücel & Lubman, 2007). Relative immaturity of cognitive control functions in younger individuals in comparison with adults may be rooted in relative underdevelopment of the various structural, neurochemical, and electrophysiological processes underlying cognitive control (Dahl, 2004; Kalsbeek, Voorn, Buijs, Pool, & Uylings, 1988; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Rosenberg & Lewis, 1995). Synaptic pruning, myelination, and the elaboration of dendritic arborization during adolescence lead to increased specialization and better connectivity among the regions supporting cognitive control (Luna et al., 2010; Luna & Sweeney, 2004). These factors further contribute to cognitive control advances over the period of adolescence.

Individual differences in the maturation rate of cognitive control faculties over the adolescent period may lead to natural variation in these faculties among adolescents. Such variability may hinder efforts to demonstrate impairments in these faculties (as a result of alcohol exposure, for example), thus leading to a relative paucity of data on disinhibition in adolescents.

Furthermore, the substantial neuromaturational processes that occur during the adolescent phase render the adolescent brain (particularly the frontal regions) especially vulnerable to the toxic effects of alcohol (Dahl, 2004). Cognitive control processes may therefore sustain more impairment due to drinking during the adolescent phase in comparison with drinking during adulthood. By virtue of age, however, adolescents generally have shorter drinking histories than adults, and so have had less exposure to alcohol.

Hence, in comparison with adults, (a) adolescents have relatively immature and variable cognitive control functions, and (b) the brain regions supporting cognitive control in adolescents are relatively vulnerable to neurotoxins. On the other hand, however, the brains of adolescents with AUDs have endured comparatively less alcohol exposure. Age is thus an important consideration in studies of disinhibition in AUDs. Because of a paucity of research on disinhibition in adolescent AUDs, little is known regarding the profile of disinhibition in adolescent AUDs.

AUDs in Treatment-naïve Samples

Although alcoholism research focuses largely on individuals seeking treatment for AUDs, at least 75% of individuals with AUDs are untreated and community-dwelling (Dawson et al., 2005; Fein & Landman, 2005). Research has shown that these two populations are distinct:

Treated individuals with AUDs have more severe alcoholism, and more psychiatric and polysubstance comorbidity than their treatment-naïve counterparts (Di Sclafani, Finn, & Fein, 2008; Fein & Landman, 2005; Gazdzinski, Durazzo, Weiner, & Meyerhoff, 2008). In contrast, treatment-naïve individuals with AUDs are, for the most part, high-functioning drug abusers who are able to remain employed or to attend school (Yechiam, Stout, Busemeyer, Rock, & Finn, 2005).

Some studies (e.g., Chao, Meyerhoff, Cardenas, Rothlind, & Weiner, 2003; Fein & Andrew, 2011; Fein, McGillivray, & Finn, 2006; Mazas, Finn, & Steinmetz, 2000; S. Smith & Fein, 2010) have reported reduced evidence of disinhibition in treatment-naïve individuals with AUDs relative to that observed for treated individuals (who display the typical disinhibitory complex) elsewhere (e.g., Beck et al., 2009; Dom, Hulstijn, & Sabbe, 2006; Goldstein et al., 2004; J. M. Mitchell, Fields, D'Esposito, & Boettiger, 2005; Porsjez & Begleiter 1986; Kamarajan et al 2010). It is important to study disinhibition in treatment-naïve individuals with AUDs because (a) these individuals are largely representative of the AUD population, and (b) examination of treatment-naïve samples provides an opportunity to learn about disinhibition related specifically to AUDs (i.e., relatively free from the contamination of other substance and psychiatric comorbidity).

In contrast to samples in the United States, where most previous research in this field has been conducted, adolescents in the Cape Town region prefer alcohol to other drugs, are less likely to engage in polysubstance use, and are less likely to manifest psychiatric comorbidity (Ferrett, Carey, Thomas, Tapert, & Fein, 2010; Ferrett et al., 2011; Flisher, Parry, Evans, Muller, & Lombard, 2003; Parry et al., 2004). The Cape Town region thus provides a unique opportunity to examine community-dwelling adolescents with AUDs in the absence of the confounding effects of psychiatric comorbidity and polysubstance use.

Specific Aims and Hypotheses

The aims of the current study were three-fold. First, I aimed to examine and characterize the disinhibitory complex in treatment-naïve adolescents with AUDs. Second, I aimed to investigate sex differences in disinhibition. Third, I aimed to determine whether an underlying construct of disinhibition might explain elevated levels of disinhibition in AUDs observed on individual indices. These specific hypotheses were tested:

1. In relation to healthy controls, adolescents with AUDs would exhibit elevated disinhibition, as measured using various indices. That is to say, adolescents with AUDs would demonstrate relatively higher levels of disinhibited personality, other externalizing psychopathology, executive cognitive disinhibition, and EEG correlates of disinhibition. As mentioned earlier, some studies (e.g., Chao et al., 2003; Fein & Andrew, 2011; Fein et al., 2006; Mazas et al., 2000; S. Smith & Fein, 2010) suggest that the disinhibitory complex (documented frequently in treated AUD samples) is less evident in treatment-naïve individuals. Treatment-naïve samples, however, are relatively understudied. Because disinhibition in treatment-naïve samples has not been thoroughly investigated, this hypothesis was based on literature using treated samples.
2. In relation to females, males would exhibit elevated levels of disinhibition, as measured using the selected indices. This hypothesis was based on findings indicating that males, independent of AUD status, are more likely to exhibit traits of disinhibition than females (Hicks et al., 2007).
3. Consistent with the notion of a latent construct of disinhibition, the various measures of disinhibition listed above would converge to reveal an underlying construct. This construct would, in turn, explain relatively higher levels of disinhibition in adolescents with AUDs relative to healthy controls.

Methods

Design and Setting

This study was nested within a larger research project whose aim is to examine the effects of heavy alcohol abuse on adolescent brain structure and functioning. The current study used a cross-sectional, quasi-experimental design to (a) compare performance between adolescents with AUDs (Alcohol group) and non-abusing/dependent adolescents (Control group) on various indices of disinhibition, (b) determine the extent to which the selected indices of disinhibition did in fact tap into a latent construct of disinhibition. I used a combination of subjective (self-report personality questionnaires) and objective (psychiatric evaluations, neuropsychological tests, and EEG recordings) measures to evaluate disinhibition. Data collection took place at the Tygerberg medical campus of the University of Stellenbosch. Participants were recruited from primary and secondary schools in accordance with nonrandomized selection criteria.

Participants

Participants (N = 162) were recruited into either an Alcohol or Control group. All participants (a) had English or Afrikaans as their first language, (b) were between the ages of 12 and 18 years, and (c) were attending schools in the northern suburbs of the greater Cape Town region at the time of initial recruitment. The two groups featured equal numbers of males and females.

The exclusion criteria used in the selection process were extensive, such that the results were not confounded by extraneous variables. Exclusion criteria for study participation were as follows: mental retardation, lifetime DSM-IV Axis I diagnoses other than AUD (including psychotic, anxiety, depressive, post-traumatic stress, elimination, eating, tic, attention-deficit/hyperactivity, conduct, and oppositional defiant disorders); signs or symptoms of fetal alcohol syndrome or malnutrition; sensory impairment; history of traumatic brain injury with loss of consciousness exceeding 10 minutes; presence of diseases that may affect the central nervous system (e.g., meningitis, epilepsy, HIV); current use of sedative or psychotropic medication; lifetime dosage exceeding 30 cannabis ‘joints’ or 4 methamphetamine doses; fewer than 6 years of formal education; and lack of proficiency in both English and Afrikaans.

Measures

Group membership. The original English versions of all measures underwent careful translation into Afrikaans (including back-translation) for use with first-language Afrikaans speakers. To elicit alcohol-related data and determine group membership, a revised version of the Timeline Followback (TLFB) procedure (Sobell & Sobell, 1992), which assesses lifetime history of alcohol use and drinking patterns, was used in collaboration with the Schedule for Affective Disorders and Schizophrenia for School Aged Children (6-18 years) Lifetime Version (K-SADS-PL) (Kaufman et al., 1997), which determines the presence or history of psychiatric diagnoses. Both the TLFB and K-SADS-PL are semi-structured, clinician-administered diagnostic tools.

Alcohol group membership was defined by a lifetime dosage in excess of 100 standard drinks[2] plus a DSM-IV diagnosis of alcohol abuse or dependence. The Control group comprised non-drinkers (individuals who had never consumed alcohol) and light drinkers (those with a lifetime dosage not exceeding 60 units of alcohol) with no history of an AUD.

Personality measures. Traits of disinhibited personality were measured using self-report subscales from three major personality inventories. Impulsivity and venturesomeness were measured using the Impulsivity (IVS-Imp) and Venturesomeness (IVS-Ven) subscales of the Impulsivity-Venturesomeness Scale (IVS; Eysenck & Eysenck, 1978). Reliability coefficients estimating internal consistency for these scales lie between 0.71 and 0.78, and the construct validities of these scales are acceptable (Eysenck & Eysenck, 1980).

Novelty-seeking was measured using total score on the Novelty Seeking Scale (NSS) from Cloninger's (1987b) Tridimensional Character Inventory. This scale has well-established psychometric properties (Bagby, Parker, & Joffe, 1992; Gana & Trouillet, 2003).

Excitement-seeking was measured by summing scores from the Disinhibition (SSS-Dis) and Boredom Susceptibility (SSS-BS) subscales of Zuckerman et al.'s (1978) Sensation Seeking Scale (SSS). Reliability coefficients for these subscales lie between 0.56 and 0.76, and they have good construct validity (Zuckerman et al., 1978). Several items in the SSS-Dis subscale refer directly to alcohol and drug use. These items were excluded from the composite tally in order to avoid contamination of personality measures with measures of substance use. Others have

[2]A standard drink was defined as one beer or wine cooler, one glass of wine, or one 1.5-oz shot of liquor (alone or in a mixed drink).

combined the SSS-Dis and SSS-BS subscales in this way, and have further demonstrated that this composite is an indicator of the latent variable of excitement-seeking (Finn & Hall, 2004; Finn et al., 2000; Koopmans, Boomsma, Heath, & van Doornen, 1995).

Although none of the above scales have been used previously with a South African sample beyond the larger project within which this study was nested, these scales have been used in samples of adults and adolescents with AUDs, and so were deemed suitable for use with the current sample (Ferguson & Lynskey, 1995; Finn et al., 2005; Finn et al., 2002; Gerra et al., 2004; Grekin et al., 2006; Müller, Weijers, Böning, & Wiesbeck, 2008; Nagoshi, Walter, Muntaner, & Haertzen, 1992; Zernicke et al., 2010).

Psychiatric measure. Current and past psychiatric symptoms were recorded for DSM-IV Axis I psychiatric disorders during administration of the K-SADS-PL. Although recruitment inclusion criteria saw all participants fall below the diagnostic threshold for psychiatric disorders (except AUDs, in the case of the Alcohol group), subthreshold symptom counts were used to examine sub-diagnostic threshold externalizing psychopathology (aside from AUDs). Specifically, the *externalizing composite* was calculated by summing the number of lifetime symptom counts for CD and ODD. Previous studies (e.g., Di Sclafani et al., 2008; Fein, Di Sclafani, Finn, & Scheiner, 2007; Ferrett et al., 2011) have assessed externalizing psychopathology in AUDs using similar methods. Although ADHD is often included in measures of externalizing psychopathology, symptom counts for this disorder were not included in the current composite based on evidence that the diagnosis does not fall within the classification of disruptive behavioral disorders characterized by social deviance (H. I. Kaplan & Sadock, 1998)

Cognitive measures.

Issues in neuropsychological assessment of cognitive abilities. Neuropsychological test performance is traditionally interpreted using overall achievement scores alone (Lezak, Howieson, & Loring, 2004). Despite the fact that the analysis of intermediate measures, such as number of errors or rule violations committed, provides an indication of the integrity of more circumscribed cognitive processes (e.g., response inhibition processes), these measures are rarely analyzed as independent entities (E. Kaplan, 1988; Possin et al., 2009; Stuss et al., 2001). This practice is surprising because number of errors and number of rule violations have documented

clinical utility over and above overall achievement scores (Carey et al., 2008; Culbertson, Moberg, Duda, Stern, & Weintraub, 2004; Kramer et al., 2003).

Despite the fact that error commission on the SCWT provides a relatively pure estimate of response inhibition, analysis of the number of errors committed on the SCWT is uncommon (Fillmore, 2004; Fillmore & Vogel-Sprott, 1995; Stuss et al., 2001). Instead, response inhibition deficits in AUDs are widely documented using traditional SCWT indices of performance, which include (a) time taken to complete each task, and (b) number of items completed on each task (Dolan et al., 2008; Fernandez-Serrano, Perez-Garcia, Perales, & Verdejo-Garcia, 2010; Golden & Freshwater, 2002; Silveri et al., 2011; Stuss et al., 2001). In some early studies in the field, the number of errors committed was incorporated into total time using various arbitrary methods. For example, some researchers (e.g., Gardner, Holzman, Klein, Linton, & Spence, 1959; Stroop, 1935) added two times the average response time per item for each error to the total time taken to complete the test. In a more recent version of the SCWT, the number of errors committed is disregarded entirely in scoring procedures (Golden & Freshwater, 2002).

SCWT. The inhibition (Color-Word) task from the SCWT (Golden & Freshwater, 2002) was used to measure response inhibition. In the version of the test used here, participants were required to correctly name as many items as possible within 45 seconds. Participants were encouraged to perform this task as quickly as possible, and to self-correct any incorrect responses. This test has been used widely within both AUD and non-AUD child, adolescent, and adult populations (e.g., D. B. Clark, Cornelius, Kirisci, & Tarter, 2005; Dolan et al., 2008; Giancola, Mezzich, & Tarter, 1998; Tapert, Granholm, Leedy, & Brown, 2002), as well as with South African children and adults (e.g., Joska et al., 2011; Savitz & Jansen, 2003).

Traditional indices of SCWT performance have been used to document response inhibition deficits in individuals with AUDs (e.g., Dolan et al., 2008; Fernandez-Serrano et al., 2010; Golden & Freshwater, 2002; Silveri et al., 2011; Stuss et al., 2001). Number of uncorrected errors on the inhibition task is one (infrequently used) measure of response inhibition (Fillmore, 2004; Fillmore & Vogel-Sprott, 1995; Rose & Duka, 2007; E. Strauss, Sherman, & Spreen, 2006; Stuss et al., 2001). I chose to expand this measure by creating a composite score that included both uncorrected and self-corrected errors. This decision was based on the premise that both uncorrected and self-corrected errors signify an incorrect initial response, and thus an inability to inhibit the prepotent response.

Others (e.g., Shuster & Toplak, 2009; Verdejo-García et al., 2010) have used self-corrected errors on the SCWT to examine response inhibition in non-substance-using and substance-using samples, respectively. The *SCWT response inhibition composite* was thus calculated by summing the number of uncorrected and self-corrected errors (i.e., raw scores for uncorrected and self-corrected errors) on the inhibition task. Indeed, previous studies have chosen to analyze raw error scores instead of normed error scores (Rose & Duka, 2007; Shuster & Toplak, 2009).

ToL. In the ToL (Culbertson & Zillmer, 2001), participants move individual beads along a pegboard in an effort to replicate the examiner's arrangements of beads. A primary aim of the task is to replicate the examiner's arrangements using as few moves as possible. The ToL is a well-established test of planning and problem-solving abilities (E. Strauss et al., 2006), and has been used widely to assess these function (e.g., Paraskevaides et al., 2010; Robinson, Goddard, Dritschel, Wisley, & Howlin, 2009). Although less frequently examined than planning ability, response inhibition may be measured using rule violation scores on the ToL. Rule violations occur when participants (a) move more than one bead at a time, or (b) exceed the permissible number of beads per peg. Indeed, ToL rule violation scores have been used elsewhere to examine inhibition in samples of young individuals (e.g., MacAllister et al., 2011; Sarkis, Sarkis, Marshall, & Archer, 2005). In the current study, total rule violation scores on the ToL were used as the second measure of response inhibition.

EEG measures. EEG data were recorded during performance of the (a) visual target detection task, and (b) Balloon Analogue Risk Task (BART; Lejuez et al., 2002). E-Prime software package version 2.0 (Psychology Software Tools Inc., 2007) was used to run these tasks on a Pentium D workstation. The purpose of the E-Prime system was to (a) present stimuli, (b) monitor the accuracy of responses, and (c) send information relating to timing of stimuli and responses using transistor-to-transistor logic (TTL) level signaling. These signals were transmitted over a parallel port to the EEG recorder system, which was a 64-channel Neuroscan SynAmps2 system using Scan acquisition software package version 4.3 (Compumedics Neuroscan Inc., 2008). The Neuroscan system received the TTL-level signals from the E-Prime system and inserted markers in the EEG record to indicate (a) stimulus type and onset time, and (b) response type and timing of response.

For all EEG recordings, the online high-pass filter was set to 0.3 Hz and the online low-pass filter was set to 70 Hz. The amplifier voltage range was 200 mV. During EEG recordings, participants were seated upright, approximately 76 cm away from the computer screen. A cephalic reference electrode (close to Cz) was used for all recordings. Left and right earlobe electrodes were used for obtaining signals for re-referencing of data offline. The ground electrode was placed 8 cm above the nasion. Electrode site impedances were kept below 10 kΩ. A vertical electro-oculogram (EOG) was recorded from bipolar electrodes placed above and below the left eye for use in offline reduction of ocular artifacts. The EEG and EOG channels were sampled at 250 Hz and stored together with stimulus and response markers for offline analysis.

***P300*.** Because the P300 may be elicited through rare, salient events, it is traditionally examined using target detection, or "oddball" tasks (e.g., Cohen et al., 1995; Fein & Andrew, 2011; Rodriguez Holguin, Porjesz, Chorlian, Polich, & Begleiter, 1999). The aim of these tasks is to categorize a series of stimuli presented sequentially and occurring with variable frequency. These stimuli are usually presented to either the visual or auditory modality, and are distinct (i.e., they look or sound different) from one another. The first, and most frequently occurring type of stimulus, is the standard stimulus. Subjects do not respond to these stimuli. Rare non-target, or distracter, stimuli occur relatively infrequently. Because subjects are instructed to ignore rare non-target stimuli, these infrequently-presented stimuli are regarded as "task-irrelevant", and hence produce the P3a on presentation. The third stimulus type is the target stimulus, or "oddball". Target stimuli also appear infrequently. Subjects are, however, instructed to respond to these stimuli, and so target stimuli are "task-relevant", and elicit the P3b on presentation.

In the current study, the visual P300 was measured during administration of a standard three-condition visual target detection task. We presented 280 stimuli over approximately 6.5 minutes in a predetermined, semi-randomized order. The three different stimulus types were: (a) the standard stimulus (a small hollow white square, accounting for 75% of all stimulus presentations); (b) the target stimulus (a small white X, accounting for 12.5% of all stimulus presentations); and (c) the rare non-target stimulus (different shapes of various colors, accounting for 12.5% of all stimulus presentations). Each stimulus appeared on a black screen for 200ms; the inter-stimulus interval was 1000-1100ms. Participants were instructed to press a response box button immediately following each presentation of the target stimulus, and to

ignore any other shapes. Each participant was shown examples of the stimuli and was given a chance to practice the task before data acquisition began. The visual target detection task has been used previously to assess relationships between P300 amplitude, impulsivity, and substance dependence in both adults and adolescents (Carlson et al., 1999; Chen et al., 2007).

F-ERN. The BART is a routine measure of risky decision-making. In this task, the participant is presented with a screen image of an empty balloon. The balloon is inflated with money in discrete increments each time the participant chooses to pump it. Each pump of the balloon, however, carries the risk of it popping, leading to a loss of all money accumulated over previous pumps, and regression to an empty balloon. Furthermore, the probability of the balloon popping increases with each consecutive pump, thereby increasing the risk associated with each consecutive pump. To avoid a loss of rewards, the participant is given the option, following each successful pump, to stop pumping the balloon and bank the money retained inside. However, banking money results in the presentation of a new balloon. A limited number of balloons are presented to the participant over the course of the task. The aim of the task is thus to maximize accumulation of rewards by weighing the value accumulated against the probability of the balloon popping.

In the current study, the F-ERN was measured in response to negative feedback (visual stimuli indicating balloon pops) on a modified version of the BART. Previous studies show that behavioral measures of risky decision-making on the BART are related to real-world risk-taking propensity amongst adolescents, including those with AUDs (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; T. J. Crowley et al., 2006; Lejuez, Aklin, Bornovalova, & Moolchan, 2005).

The original BART underwent several modifications to facilitate ERP analysis (Fein & Chang, 2008). First, the number of trials (new balloon presentations) was raised to 60 to increase the available number of negative feedback epochs for averaging. To restrict the experiment to a reasonable time length, this extension to the task was balanced by limiting the maximum breaking point for each balloon to 20 pumps. Possible presentation of negative feedback was limited to risky decisions by disabling pops resulting from the first pump of an empty balloon (i.e., a non-risky decision). As such, the probability of balloon pops after the second pump was 1/19, after the third pump was 1/18, and so forth, until the 20^{th} pump where a balloon pop was certain. To increase the attraction of accumulating rewards, the amount gained by each successive pump increased by two cents at each pump (i.e., 50 cents were earned on the first

pump, 52 cents on the second pump, 54 cents on the third pump, and so on). The balloons appeared as blue spheres, enlarging in proportion to the amount of money retained in the balloon. Subject response and the provision of feedback were separated by a random delay of 1.0-1.2 seconds. Noise stimuli used in the original implementation of the task to indicate balloon bursts were replaced with a silent balloon burst graphic and text indicating a loss of money. The purpose of this adaptation was to avoid possible unwanted startle responses and muscle artifact. To encourage optimal performance on the BART in the current study, participants were informed before commencement of the experiment that they would receive a percentage of their final winnings in the form of a gift voucher to take home.

Cultural and linguistic considerations. The EEG tasks were presented in a computerized format, with prompts, stimuli, and feedback appearing in English. However, the tasks were relatively free of linguistic bias. With regard to the visual target detection task, stimuli were limited to shapes, and thus independent of linguistic preference. In the BART, although text stimuli were available only in English, the relevant text was simple (e.g., "Pop"), and accompanied by descriptive graphics (e.g., image of popped balloon), thus ensuring that the message was understood. Furthermore, observational accounts of the sample revealed a high level of basic English proficiency amongst first-language Afrikaans speakers, suggesting that task performance for such participants was not compromised due to the English-language materials.

To ensure cultural significance of accumulated rewards on the BART, South African rands and cents (as opposed to American currency as used in the original version) were used to designate rewards. Despite these modifications, the modeling of real-world risk-taking in the original BART (originally described by Lejuez et al., 2002) was maintained in this implementation by adherence to two core principles: (a) the amount to be lost following a balloon pop increased with each successive pump, and (b) the overall gain from each pump decreased after the second pump.

Although the visual target detection task and the BART have not been used previously with South African samples, both have simple designs and there was no reason to expect cultural or socioeconomic effects on performance. In fact, the BART is probably a more ecologically valid method of assessing risky decision-making in the current populations than traditional gambling tasks, in that the BART does not involve gambling *per se*: it is unlikely that

participants were accustomed to gambling practices, but it is likely they were familiar with balloons, and so the BART was a more appropriate task in this case.

Procedure

Preliminary procedure. The study protocol and procedures for the current study adhered strictly to the guidelines contained in the Declaration of Helsinki (2008). All study procedures were approved by the Western Cape Education Department and the Research Ethics Committee of the Stellenbosch University Faculty of Health Sciences. Participants were recruited through oral presentations at schools, as well as via word-of-mouth advertisements. Full written assent and consent (see English version in Appendix A) was obtained from volunteers and parents/guardians respectively. Parents/guardians also completed a parent interview form (see English version in Appendix B). Transport, meals, and refreshments were provided to the participants.

Assessment. Volunteers were screened for eligibility through a detailed medical history, physical and psychiatric examination, as well as urine analysis and breathalyzer testing. Following psychiatric and medical evaluations, eligible participants completed personality questionnaires, and were individually administered neuropsychological and EEG assessments. Completion of personality questionnaires, and of neuropsychological and EEG assessments, were not administered in any particular order. Instead, these assessments were scheduled after psychiatric and medical evaluations according to participant and administrator availability. Full participant assessments (including psychiatric and medical evaluations, completion of personality questionnaires, and neuropsychological and EEG assessments) took place over 2-3 site visits.

Participants each completed a demographics form (see English version in Appendix C). Neuropsychological testing and verbal instructions for EEG tasks were provided in participants' language of choice (English or Afrikaans). Before commencing neuropsychological and EEG assessments, participants were given clear instructions, as well as the opportunity to ask questions if they did not clearly understand the task requirements. Within the EEG assessment, the EEG tasks (visual target detection task and BART) were administered in a random order.

Debriefing. Following assessments, participants were given the opportunity to express any opinions regarding the experience. Following this step, gift vouchers were distributed.

Participants received gift vouchers for each completed assessment (i.e., psychiatric evaluation, neuropsychological assessment, EEG assessment), as well as additional gift vouchers based on total winnings on the BART. Participants were then thanked for their participation and dismissed.

EEG Analysis

Participant eligibility. A subset of the original sample was included for EEG analyses. This subset is hereafter referred to as the "EEG subsample." It was not possible to include all participants in these analyses as EEG recordings for 78 participants took place in excess of 3 months after participants' psychiatric evaluations. In order to maintain validity of information gathered at the psychiatric screen (including alcohol-use information) in relation to psychophysiology, the maximum acceptable delay between psychiatric screen and EEG recording was set at 3 months. Based on this criterion, 84 participants were included in the EEG subsample.

Ocular correction, re-referencing, and data filtering. EEG recordings were processed offline using the Edit program in the Scan software package version 4.3 (Compumedics Neuroscan Inc., 2008). For both the P300 and F-ERN analyses, ocular artifacts were removed using the linear regression procedure implemented in Edit (Semlitsch, Anderer, Schuster, & Presslich, 1986). Data were re-referenced to the right mastoid, and all non-EEG channels were removed from the recordings.

In the P300 analysis, data were band-pass filtered between 0.5 and 15 Hz using a zero-phase lag filter at 48 dB/octave. In the F-ERN analysis, a low-pass filter at 30 Hz was applied to the data using a zero-phase lag filter at 48 dB/octave.

Epoching. In the P300 analysis, stimulus-locked trials were created for all instances where there was a correct behavioral response for the standard, target and rare non-target conditions. In the F-ERN analysis, feedback-locked trials were extracted for positive (balloon enlarging with accumulating rewards) and negative (balloon popping and loss of all rewards in balloon) feedback. In both analyses, epochs comprised 100 ms of data preceding, and 750 ms of data following, stimuli and feedback respectively. Epochs were baseline corrected using the 100 ms pre-stimulus/pre-feedback interval for the P300 and F-ERN analyses, respectively.

Artifact rejection and channel reinterpolation. All channels were then examined for out-of-range voltages (less than -75 mV, or greater than 75 mV). Where the number of artifact-free epochs per condition (standard, target, and rare non-target conditions for the P300 analysis; negative and positive feedback conditions for the F-ERN analysis) on any given channel fell below the designed threshold (20 epochs for the P300 analysis; 10 epochs for the F-ERN analysis), this channel was considered for reinterpolation. This threshold was lower for the F-ERN than P300 analysis as the average number of available epochs was considerably lower in the negative feedback condition (M = 18) for the BART than in each of the target and rare non-target conditions (35 epochs for all participants) in the visual target detection task. Furthermore, although the number of available negative feedback epochs on the BART was subject to participant performance, this was not the case in the visual target detection task. Instead, the number of available epochs on the visual target detection task was constant and independent of subject performance.

In both analyses, reinterpolation was performed as necessary on no more than three electrodes per participant, provided (a) overall data quality was acceptable, and (b) such reinterpolation did not include main channels of interest (midline electrodes for the P300; Fz, FCz, and Cz for the F-ERN). Reinterpolation was conducted on unprocessed, continuous recordings by estimating voltage at the contaminated site using between three and eight (depending on electrode position) equally weighted, neighboring channels. Where reinterpolation took place, all preceding processes were repeated on the reinterpolated data. Finally, recordings were accepted for analysis provided the number of artifact-free epochs was above the relevant threshold on each channel per condition.

Averaging. In both the P300 and F-ERN analyses, ERP waveforms (viz., P3a waveform, P3b waveform, F-ERN waveform) were extracted for each participant by averaging across trials for each condition. In addition to the P300 waveforms, P300 difference waveforms were created by subtracting (a) the standard stimulus-locked waveform from the rare non-target stimulus-locked waveform (viz., P3a difference waveform), and (b) the standard stimulus-locked waveform from the target stimulus-locked waveform (viz., P3b difference waveform). F-ERN difference waveforms were created by subtracting the positive feedback-locked waveform from the negative feedback-locked waveform for each participant (viz. F-ERN difference waveform).

The aim of creating difference waves was to accentuate ERP waveforms, thus aiding between-group ERP peak comparison.

Peak detection. I examined the spatial distributions of the P300 and F-ERN average (ERP and ERP difference) waveforms across participants and found, as expected, a general midline focus for the P300, and a general frontal focus for the F-ERN. In addition, these average waveforms showed some variability across participants with regard to the specific location of the spatial peak. Indeed, others have shown that various characteristics of these components vary naturally across individuals (e.g., Comerchero & Polich, 1998; Comerchero & Polich, 1999; Polich, 1986). Therefore, and in contrast with other studies that have examined maxima for the P300 and F-ERN at predetermined electrode sites (selected on the basis of *a priori* predications regarding the location of the spatial maximum for these components), I chose instead to extract amplitudes and latencies for these components at the location of each individual participant's spatial maximum.

Therefore, and with regard to the P300 analysis, all midline electrodes (Fz, FCz, Cz, CPz, Pz, POz, and Oz) were examined for the maximum positive peak occurring between 300 and 500 ms after relevant stimulus presentation (P3a for the rare non-target condition; P3b for the target condition) in both the P300 and P300 difference waveforms. P3a and P3b amplitude and latencies were extracted at the midline electrode at which the spatial distribution peak was maximal for that participant (for each respective condition). These peaks were identified for both P300 waveforms and P300 difference waveforms.

A similar procedure was used for the F-ERN analysis. In this analysis, the frontal electrodes (Fz, FCz, and Cz) were examined for the maximum negative peak occurring between 200 and 300 ms after presentation of negative feedback in both the F-ERN and F-ERN difference waveforms. F-ERN amplitude and latency was identified at the frontal electrode at which the spatial distribution peak was maximal for that participant. Again, these peaks were extracted for both F-ERN and F-ERN difference waveforms.

Each ERP and ERP difference waveform was visually inspected to confirm that the automated peak detection algorithm had indeed identified an appropriate and clear peak. Peak location was adjusted manually as necessary, based on temporal and spatial distribution of the ERP (sub)component. Where extracted peaks were absent or ambiguous, that (sub)component was scored as missing.

Participant exclusions. Several participants were excluded, for a variety of reasons, from the final analysis of the EEG subsample's data. Of the original group of 84 participants (Alcohol n = 39), 2 (Alcohol n = 1) were excluded from all EEG-related analyses (as well as the sociodemographic analyses) due to poor signal quality.

For the P300 analysis, a single participant in the Alcohol group was excluded due to chance performance on the experimental task. Due to having ambiguous/unclear peaks, the following participants were excluded from the relevant individual analyses: 14 participants (Alcohol n = 6) from the P3a waveform analysis; 9 participants (Alcohol n = 6) from the P3a difference waveform analysis; 2 participants (Alcohol n = 1) from the P3b waveform analysis; and 3 participants (Alcohol n = 2) from the P3b difference waveform analysis.

For the F-ERN analysis, 9 participants (Alcohol n = 5) were excluded due to poor signal quality; 7 participants (Alcohol n = 2) were excluded due to high artifact ratio on channels of interest; and 12 participants (Alcohol n = 6) were excluded due to having too few available negative feedback trials. Due to having ambiguous/unclear peaks, 11 participants (Alcohol n = 9) were excluded from the F-ERN waveform analysis, while 8 participants (Alcohol n = 6) were excluded from the F-ERN difference waveform analysis.

Following all exclusions, the following participants remained for each analysis: 66 participants (Alcohol n = 30) for the P3a waveform analysis; 71 participants (Alcohol n = 30) for the P3a difference waveform analysis; 78 participants (Alcohol n = 35) for the P3b waveform analysis; 77 participants (Alcohol n = 34) for the P3b difference waveform analysis; 42 participants (Alcohol n = 15) for the F-ERN waveform analysis; and 45 participants (Alcohol n = 18) for the F-ERN difference waveform analysis.

Grand average waveforms and topographical maps. Using only participants included for each specific analysis, grand average waveforms for each condition (standard, target, and rare non-target conditions in the P300 analysis; positive and negative feedback conditions for the F-ERN analysis) were produced by averaging across individual participant average waveforms. These grand average waveforms were created separately for the Control and Alcohol groups. Using these grand average waveforms, grand average topographical maps were computed to display the spatial distribution of P300 and F-ERN components. The computation of grand average waveforms and topographical maps was performed on both the ERP and the ERP difference waveforms.

Statistical Analysis

I performed all statistical analyses using SPSS software package version 17.0 (SPSS Inc., 2008). Unless otherwise stated, the threshold for statistical significance was set at $p = .05$.

Preliminary analyses. Before conducting any inferential analyses, I cross-checked data and created frequency tables and box-and-whisker plots so as to better understand the data. I inspected the normality of data both visually and statistically, using histograms and the Shapiro-Wilk test, respectively. I used Levene's test to assess homogeneity of variance. All assumptions for parametric data were upheld, unless otherwise indicated. To confirm that participants for the relevant analyses were drawn from a similar population, I conducted preliminary analyses of sociodemographic measures. These analyses involved 2 (Group) x 2 (Sex) factorial ANOVAs for age and socioeconomic status (SES) data, and chi-square tests for quality of education and language of test administration.

Primary analyses. The primary analyses consisted of two steps. First, I analyzed between-group (Control vs. Alcohol) and between-sex (female vs. male) mean differences on outcome measures (viz., personality, psychiatric, cognitive, EEG). Where assumptions for parametric tests were upheld, I compared means using 2 (Group) x 2 (Sex) factorial ANOVAs. I used separate Mann-Whitney *U*-tests to compare (a) Group and (b) Sex means for non-parametric data. I included sex as a factor for all primary analyses of outcome measures based on some findings indicating that males, independent of AUD status, are more likely to exhibit traits of disinhibition than females (Hicks et al., 2007). Because I used 15 tests to compare means on outcome measures, I applied the Sidak correction for multiple comparisons in order to avoid the likelihood of Type 1 errors. As such, the threshold for statistical significance was set at 0.0034.

Second, I subjected personality, psychiatric, and cognitive measures to exploratory factor analysis. The aim of this analysis was to determine (a) whether these measures converged on an underlying construct of disinhibition, and hence (b) whether group and sex differences found on these measures suggest such differences on a latent construct of disinhibition. I excluded EEG measures from the exploratory factor analysis due to the limited sample for which data on these measures was available.

Results

Sample Sociodemographic Characteristics

All participants were mixed ancestry (colored), and so race was not included as a variable in any of the statistical analyses reported here.

Other studies examining cognitive functions in South African youth (e.g., Jinabhai et al., 2004; Skuy, Schutte, Fridjhon, & O'Carrol, 2001) have highlighted the influence of language of neuropsychological test administration on test performance. For this reason, participants in the Alcohol and Control groups were matched with regard to language of test administration. Similarly, previous researchers (e.g., Manly et al., 1999) have demonstrated the significant influence of quality of education on neuropsychological test performance. Disparities in quality of education in South Africa were entrenched during the Apartheid era, when material and human resources were allocated in favor of schools attended by white children. Although black and colored learners have had better access to educationally advantaged schooling facilities since democratization in 1994, inequalities in quality of education have, to a large degree, remained in place (Grieve & van Eeden, 2010; Shuttleworth-Edwards et al., 2004; van der Berg, Wood, & le Roux, 2002). Here, I followed procedures used in the larger study and determined quality of education based on the Western Cape Education Department's pre-democratization school classification system. Within this classification system, schools previously falling under the Western Cape Education or Cape Education Department categories (previously accessible to only white children) were deemed "advantaged", while schools previously falling under the Department of Education and Training, House of Delegates, or House of Representatives systems (previously reserved for children who were not white) were deemed "disadvantaged" (Western Cape Education Department, 2010).

Regarding SES, I again followed procedures used in the larger study and employed a composite measure to calculate values for that variable. The socioeconomic composite was created using a comprehensive range of indices, based on evidence that SES is a multidimensional phenomenon and best reflected when several indices are taken into account (Bradley & Corwyn, 2002). The SES composite used here comprised familial (family income, highest parental employment category, and highest parental education level), as well as household (total household assets, dwelling type, and bedroom cohabitants) measures. Values for these individual measures were obtained from responses to items on the parent interview and

demographics forms, completed by parents/guardians and participants, respectively. Responses to these items were summed to create the composite score (range: 5 to 41), as follows: (a) family income (range: 1 to 6); (b) highest parental employment category (range 1 to 9; responses were reverse-scored so that higher scores reflected higher occupational status); (c) highest parental education level (range: 1 to 6); (d) total household assets (range: 0 to 7); (e) dwelling type (range: 1 to 6); and (f) bedroom cohabitants (range: 1 to 7), with lower scores indicating lower SES.

Sociodemographic characteristics: Full sample. Table 1 shows descriptive statistics for the full sample's sociodemographic characteristics. As can be seen, there were equal distributions of males and females in the Control and Alcohol groups. Participant age ranged from 12.21 to 16.00 mathematical years (M = 14.84 ± 0.76). Regarding age, there was no significant main effect of Group, $F(1, 158) = 1.919$, $p = .168$, $\eta^2 = .012$, or Sex, $F(1, 158) = 0.480$, $p = .490$, $\eta^2 = .003$. There was also no significant Group x Sex interaction effect, $F(1, 158) = 0.294$, $p = .589$, $\eta^2 = .002$.

Regarding language of test administration, there were no significant between-sex differences, $\chi^2(1) = 2.955$, $p = .086$.

Regarding quality of education, there were no significant between-group or between-sex differences, $\chi^2(1) = 0.492$, $p = .483$, and $\chi^2(1) = 1.073$, $p = .300$, respectively.

Regarding SES, there were non-significant main effects for Group, $F(1, 154) = 1.823$, $p = .179$, $\eta^2 = .012$, and Sex, $F(1, 154) = 0.432$, $p = .512$, $\eta^2 = .003$, as well as a non-significant interaction effect, $F(1, 154) = .109$, $p = .742$, $\eta^2 = .001$.

Table 1
Sociodemographic Characteristics: Full sample (N = 162)

	Group					
	Control			Alcohol		
	Overall	Female	Male	Overall	Female	Male
Outcome variable	(*n* = 81)	(*n* = 47)	(*n* = 34)	(*n* = 81)	(*n* = 47)	(*n* = 34)
Age	14.76 (0.78)	14.75 (0.81)	14.77 (0.74)	14.92 (0.74)	14.85 (0.81)	15.00 (0.63)
Test language[a]						
Afrikaans: English	56:25	30:17	26:8	56:25	30:17	26:8
Quality of education[b]						
Disadvantaged:Advantaged	69:12	43:4	26:8	72:9	41:6	31:3
Socioeconomic status[c]	(*n* = 80)	(*n* = 47)	(*n* = 33)	(*n* = 78)	(*n* = 45)	(*n* = 33)
	28.19 (5.80)	28.57 (6.29)	27.64 (5.07)	26.85 (5.93)	26.98 (6.35)	26.67 (5.38)

Note. For the variables *Age* and *Socioeconomic Status*, means are presented with standard deviations in parentheses. [a]Language of neuropsychological test administration. [b]Based on the Western Cape Education Department's pre-democratization school classification system [c]Estimated using the SES composite score; SES data were missing for 4 participants (Alcohol *n* = 3).

Table 2
Sociodemographic Characteristics: EEG Subsample (N = 82)

	Group					
	Control			Alcohol		
Outcome variable	Overall (*n* = 44)	Female (*n* = 26)	Male (*n* = 18)	Overall (*n* = 38)	Female (*n* = 21)	Male (*n* = 17)
Age	15.22 (1.03)	15.18 (1.11)	15.28 (0.93)	15.49 (1.63)	15.57 (1.56)	15.39 (1.75)
Quality of education[a]						
Disadvantaged:Advantaged	36:8	24:2	12:6	32:6	17:4	15:2
Socioeconomic status[b]	(*n* = 44)	(*n* = 26)	(*n* = 18)	(*n* = 36)	(*n* = 19)	(*n* = 17)
	27.82 (6.22)	27.46 (6.71)	28.33 (5.58)	26.06 (6.60)	26.53 (6.66)	25.53 (6.69)

Note. For the variables *Age* and *Socioeconomic Status*, means are presented with standard deviations in parentheses. [a]Based on the Western Cape Education Department's pre-democratization school classification system [b]Estimated using the SES composite score; SES data were missing for 2 participants in the Alcohol group.

Sociodemographic characteristics: EEG subsample. Table 2 shows descriptive statistics for the EEG subsample's sociodemographic characteristics. Within this subsample, age data were not normally distributed, $W(80) = .913$, $p < .001$, and these data further violated the assumption of homogeneity of variance (as indicated by Levene's test, $F(3,78) = 3.989$, $p = .011$). Transformation of the age data did not resolve these problems, and so Mann-Whitney *U*-tests were used to investigate between-group and between-sex differences on this variable.

In this subsample, participant age ranged from 12.77 to 18.82 years ($M = 15.35 \pm 1.34$). Although all of these participants were drawn from the larger sample described above, several were older at the time of EEG recordings, hence the larger age range. Mann-Whitney *U*-tests indicated no significant between-group, $U(1) = 0.042$, $p = .967$, or between-sex $U(1) = -0.342$, $p = .732$ differences in age.

Concern with language of test administration applied only to analyses of cognitive outcome measures (i.e., those using the entire sample); hence, examination of this variable was not applicable for the EEG subsample.

Regarding quality of education, there were no significant between-group, $\chi^2(1) = 0.082$, $p = .774$, or between-sex, $\chi^2(1) = 1.443$, $p = .230$, differences.

Regarding SES, although these data appeared slightly non-normal, they did not violate the assumption of homogeneity of variance. Furthermore, ANOVA is robust to minor deviations from normality, and so I conducted a 2 (Group) x 2 (Sex) factorial ANOVA on these data. There were no significant main effects of Group, $F(1, 76) = 1.632$, $p = .205$, $\eta^2 = .021$, or Sex, $F(1, 76) = 0.002$, $p = .966$, $\eta^2 < .001$. There was also no significant Group x Sex interaction effect, $F(1, 76) = 0.408$, $p = .525$, $\eta^2 = .005$.

Within the EEG subsample, participants were excluded from each individual EEG analysis for various reasons, as outlined earlier. Hence, each EEG analysis was based on a slightly different group of participants within the EEG subsample. Nevertheless, the sample demographic characteristics for each individual EEG analysis mirrored those of the larger EEG subsample, with no significant between-group or between-sex differences in age, quality of education, or SES (see Appendix D).

Primary Analysis I: Comparison of Performance on Outcome Measures

Table 3 shows descriptive statistics for each of the outcome measures.

Table 3
Descriptive Statistics for Personality, Psychiatric, Cognitive, and EEG Measures

Measure	Group					
	Control			Alcohol		
	Overall	Female	Male	Overall	Female	Male
Personality	(*n* = 81)	(*n* = 47)	(*n* = 34)	(*n* = 81)	(*n* = 47)	(*n* = 34)
IVS-Imp[a]	7.35 (3.88)	7.15 (3.97)	7.62 (3.80)	10.16 (3.21)	10.02 (3.57)	10.35 (2.70)
IVS-Ven[a]	8.30 (2.85)	7.64 (2.90)	9.21 (2.54)	8.22 (2.50)	7.40 (2.55)	9.35 (1.94)
NSS[b]	55.42 (8.29)	53.94 (7.86)	57.47 (8.55)	59.41 (9.89)	59.40 (10.47)	59.41 (9.19)
Excitement-seeking composite[c]	5.01 (2.67)	4.83 (2.73)	5.26 (2.61)	6.57 (2.64)	6.04 (2.40)	7.29 (2.82)
Psychiatric symptom counts	(*n* = 81)	(*n* = 47)	(*n* = 34)	(*n* = 81)	(*n* = 47)	(*n* = 34)
Externalizing composite[d]	0.93 (1.75)	0.38 (1.01)	1.68 (2.24)	2.36 (3.36)	1.51 (2.51)	3.53 (4.02)
Cognition	(*n* = 81)	(*n* = 47)	(*n* = 34)	(*n* = 81)	(*n* = 47)	(*n* = 34)
SCWT response inhibition composite[e]	2.75 (2.03)	2.43 (1.79)	3.21 (2.27)	4.17 (2.45)	4.36 (2.77)	3.91 (1.94)
ToL rule violations	0.17 (0.44)	0.17 (0.48)	0.18 (0.39)	0.75 (1.60)	0.66 (1.20)	0.88 (2.04)
Electrophysiology[f]						
P3a	(*n* = 41)	(*n* = 24)	(*n* = 17)	(*n* = 30)	(*n* = 19)	(*n* = 11)
Latency	401.85 (43.59)	401.50 (44.38)	402.35 (43.80)	412.27 (45.93)	420.21 (42.64)	398.55 (50.19)
Amplitude	11.92 (4.80)	10.82 (4.24)	13.48 (5.23)	12.66 (3.66)	12.34 (3.59)	13.21 (3.88)
P3b	(*n* = 43)	(*n* = 25)	(*n* = 18)	(*n* = 34)	(*n* = 20)	(*n* = 14)
Latency	394.51 (55.25)	386.72 (56.48)	405.33 (53.14)	394.82 (26.77)	380.60 (19.95)	390.86 (34.21)
Amplitude	20.66 (6.99)	20.27 (7.96)	21.21 (5.57)	17.18 (6.13)	17.13 (5.08)	17.25 (7.59)
F-ERN	(*n* = 27)	(*n* = 15)	(*n* = 12)	(*n* = 18)	(*n* = 9)	(*n* = 9)
Latency	249.63 (21.38)	257.87 (22.26)	239.33 (15.52)	243.11 (16.72)	245.33 (9.38)	240.89 (22.25)
Amplitude	-11.79 (7.57)	-9.55 (7.93)	-14.59 (6.33)	-5.93 (8.62)	-4.32 (8.00)	-7.55 (9.37)

Note. Mean scores are provided with standard deviations in parentheses. IVS-Imp = Impulsivity subscale of the Impulsivity-Venturesomeness Scale. IVS-Ven = Venturesomeness subscale of the Impulsivity-Venturesomeness Scale. NSS = Novelty Seeking Scale. SCWT = Stroop Color-Word Test. ToL = Tower of London. [a]From the Impulsivity-Venturesomeness scale. [b]From the Tridimensional Character Inventory. [c]Disinhibition and Boredom Susceptibility subscales of the Sensation Seeking Scale. [d]Total symptoms of conduct disorder and oppositional defiant disorder. [e]Total number of uncorrected and self-corrected errors on the inhibition task of the Stroop Color-Word test. [f]ERP measures refer to those derived from ERP difference waveforms.

Personality measures. Regarding scores on the IVS-Imp, there was a significant[3] main effect of Group, $F(1, 158) = 24.253$, $p < .001$, $\eta^2 = .133$, with participants in the Alcohol group scoring higher than those in the Control group. There was no significant main effect of Sex, $F(1, 158) = .494$, $p = .483$, $\eta^2 = .003$, and no significant Group x Sex interaction effect, $F(1, 158) = 0.014$, $p = .904$, $\eta^2 < .001$.

Regarding scores on the IVS-Ven, there was a different pattern of results, however: There was no significant main effect of Group, $F(1, 158) = 0.012$, $p = 915$, $\eta^2 < .001$, but there was a significant main effect of Sex, $F(1, 158) = 18.828$, $p < .001$, $\eta^2 = .106$, with males scoring higher than females. Again, there was no significant Group x Sex interaction effect, $F(1, 158) = 0.221$, $p = .639$, $\eta^2 = .001$.

On the NSS, there were no significant main effects of Group, $F(1, 158) = 6.541$, $p = .011$, $\eta^2 = .040$, or Sex, $F(1, 158) = 1.495$, $p = .223$, $\eta^2 = .009$, and there was no significant interaction effect, $F(1, 158) = 1.482$, $p = .225$, $\eta^2 = .009$.

On the excitement-seeking composite, there was a significant main effect of Group, $F(1, 158) = 14.972$, $p < .001$, $\eta^2 = .087$, with participants in the Alcohol group scoring higher than those in the Control group. There was no significant main effect of Sex, $F(1, 158) = 4.051$, $p = .046$, $\eta^2 = .025$, and there was no significant Group x Sex interaction effect, $F(1, 158) = 0.950$, $p = .331$, $\eta^2 = .006$.

Psychiatric measure. Scores for the externalizing composite (subthreshold symptom counts for ODD and CD) were severely positively skewed, with more than half of the sample ($n = 97$) having no such symptoms. This skew was expected, given that individuals with threshold psychopathology were excluded from the study, and so the ceiling for psychopathology was low. The assumption of homogeneity of variances was also violated in this case. Various attempts at transforming the data were unsuccessful at rectifying these violations of normality and homogeneity of variance, and so non-parametric tests were used for analyses of these data. Mann-Whitney *U*-tests indicated that sub-diagnostic externalizing symptom counts were equal across groups (Control vs. Alcohol, $U(1) = 8.279$, $p = .004$), and that males had significantly higher symptom counts than females, $U(1) = 17.351$, $p < .001$.

[3]As mentioned previously, the threshold for statistical significance for all between-group comparisons on outcome measures was set at 0.0034 using the Sidak correction for multiple comparisons.

Cognitive measures.

SCWT response inhibition composite. Upon visual inspection, the distribution of these data appeared reasonably normal, but the Shapiro-Wilk test indicated a statistically significant deviation from normality, $W(162) = 0.935$, $p < .001$. These data also violated the assumption of homogeneity of variance (as shown by Levene's test, $F(3, 158) = 2.995$, $p = .033$). A log transformation of the data corrected the latter violation, but did not normalize the distribution, $W(162) = 0.950$, $p < .001$. I considered using the more normally distributed SCWT total score in place of the SCWT response inhibition composite. However, because the former measure carries some interpretive ambiguity (scores may be influenced by both speed and accuracy of performance), I chose to proceed instead with a parametric analysis of the log-transformed SCWT response inhibition composite data. This decision was supported by the fact that ANOVA is robust to minor deviations from normality, and so this violation should not have had a large effect on the results.

Analysis of the SCWT response inhibition composite revealed a significant main effect of Group, $F(1, 158) = 16.250$, $p < .001$, $\eta^2 = .093$, with participants in the Alcohol group making significantly more uncorrected and/or self-corrected errors than those in the Control group. There was no significant main effect of Sex and no significant Group x Sex interaction effect, $F(1, 158) = 0.856$, $p = .356$, $\eta^2 = .005$, and $F(1, 158) = 1.467$, $p = .228$, $\eta^2 = .009$, respectively.

ToL rule violations. These data violated the assumption of homogeneity of variance, and their distribution was severely positively skewed, $W(162) = 0.430$, $p < .001$, with most participants ($n = 123$) making no such errors. Various transformations failed to rectify either violation. Although problematic, there was some variance in the scores for this measure, and so it was not excluded from analysis. Mann-Whitney *U*-tests revealed significant between-group differences, $U(1) = 2.982$, $p = .003$, with participants in the Alcohol group committing more errors than those in the Control group. There were, however, no significant between-sex differences, $U(1) = 0.157$, $p = .876$.

EEG measures. In both the P300 (P3a and P3b) and F-ERN measures, the results obtained for the ERP waveforms were similar to those obtained for the ERP difference waveforms. Hence, only results for the ERP difference waveforms are presented here.

P300.

Spatial distribution and individual maxima. Grand average ERP difference waveforms indicated that the spatial distributions of the P3a (rare non-target condition) and P3b (target condition) were similar in the Control and Alcohol groups. In both groups, the grand average difference waveforms showed that P3a was maximal over the centro-parietal region (CPz); the P3b subcomponent showed a relative posterior spatial distribution at the parietal electrode site (Pz). Figure 1 shows the comparison of (a) grand average difference ERP waveforms (at electrode CPz for the rare non-target condition; at electrode Pz for the target condition) and (b) grand average topographical maps for the Control and Alcohol groups for both the target and rare non-target conditions. Topographical maps, showing spatial distributions across all electrode sites for the P3a and P3b, are displayed at the latency of the respective P300 peak (i.e., P3a or P3b) where the grand average difference waveform was maximal for that condition. In other words, regarding the rare non-target condition, the topographical maps for the Control and Alcohol groups display the spatial distribution of the P3a subcomponent at the latency corresponding to peak amplitude on the grand average difference waveform at electrode CPz; regarding the target condition, the topographical maps for the Control and Alcohol groups display the spatial distribution of the P3b subcomponent at the latency corresponding to peak amplitude on the grand average difference waveform at electrode Pz.

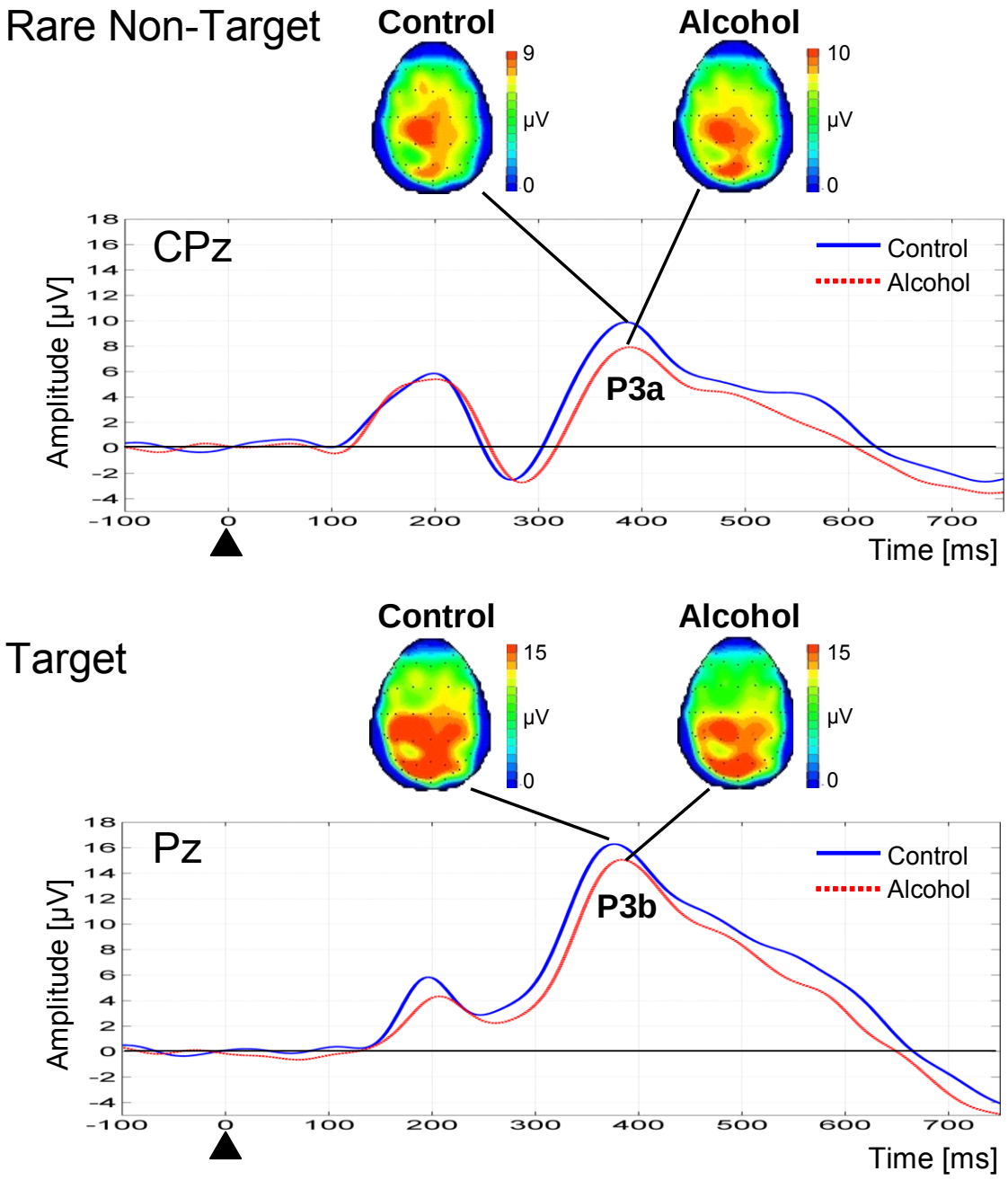

Figure 1. Grand average ERP difference waveforms for the rare non-target condition (rare non-target – standard condition) at electrode site CPz and target condition (target – standard condition) at electrode site Pz, showing comparison of Control and Alcohol groups. Grand average topographical maps are shown for the location of the P300 peaks for both the Control and Alcohol groups.

As expected, there was inter-subject variability in the location of the maximum of the spatial distribution for the P3a and P3b subcomponents across midline electrode sites. Table 4

shows a breakdown by midline electrode site of the location of the P3a and P3b amplitude maximum across subjects. Regarding the P3b, most subjects had a spatial maximum at the parietal electrode site (Pz); however, the P3a showed a bimodal distribution, with an equal number of participants having a maximum over fronto-central (FCz) and parietal (Pz) regions.

Regarding the P3b subcomponent, the majority of individual spatial maxima occurred at Pz, which clearly corresponds to the location of the spatial maximum for the P3b grand average difference waveform (i.e., Pz). Regarding the P3a subcomponent, however, most participants had individual spatial maxima at either FCz or Pz, which does not correspond directly to the location of the spatial maximum for the P3a grand average difference waveform (i.e., CPz). This discrepancy between (a) individual spatial maxima, and (b) the location of the maximum for the P3a grand average difference waveform, suggests that those subjects who showed individual spatial maxima at FCz or Pz had a smaller relative amplitude differences across electrodes than those showing individual spatial maxima at CPz. In other words, relatively large individual amplitude difference at CPz (in comparison with individual amplitude differences at FCz and Pz) lead to a maximum for the grand average difference waveform maximum at the former site, despite the preponderance of individual spatial maxima at FCz and Pz.

Table 4
Breakdown of Spatial Maxima for the P300 ERP Subcomponents and the F-ERN ERP Component Across Electrodes of Interest

	Electrode site						
Component	Fz	FCz	Cz	CPz	Pz	POz	Oz
P3a	2	17	8	11	17	13	3
P3b	3	4	5	10	29	22	4
F-ERN	13	21	11	-	-	-	-

Note. Data represent the number of participants whose spatial maxima occurred at each electrode site.

Topographical distribution of the P3a is associated with perceptions of task difficulty: when individuals find target and standard stimuli distinct in appearance (i.e., easy discrimination task), the P3a has a relative frontal distribution; when individuals find target and standard stimuli similar in appearance (i.e., difficult discrimination task), the P3a has a relative parietal distribution (Comerchero & Polich, 1998, 1999; Hagen et al., 2006). Perception of increased task difficulty may be inferred from prolonged reaction time and decreased accuracy of performance

(Kok, 2001). To determine whether the observed bimodal distribution of the P3a over fronto-central (FCz) and parietal (Pz) regions reflected individual differences in the perceptions of task difficulty, I performed a set of post-hoc analyses examining whether differences in perceptions of task difficulty (i.e., between those who found the task relatively easy and those who found the task relatively difficult) existed between those who showed P3a maxima at FCz, versus those who showed spatial maxima at Pz. These analyses indicated no significant differences in target reaction time (i.e., length of time taken to respond to target stimuli; $t(32) = 0.065$, $p = .949$), target accuracy (i.e., percentage of target trials where there was a behavioral response; $t(32) = 1.195$, $p = .241$), or rare-non target accuracy (i.e., percentage of rare non-target trials where there was no behavioral response; $t(32) = 0.045$, $p = .964$) between those showing fronto-central (FCz) and parietal (Pz) maxima of the P3a subcomponent. Individual differences in P3a spatial distribution thus appear to be unrelated to perceptions of task difficulty.

In addition, there was no difference between Control and Alcohol groups in the location of individual spatial maxima for the P300. In fact, within both groups, (a) the majority of participants showed P3a spatial maxima at FCz and Pz, and the majority of participants showed P3b spatial maxima at Pz, and (b) there was a clear bimodal distribution for the P3a subcomponent (i.e., within the Control group, 10 participants had spatial maxima at FCz and 10 participants had spatial maxima at Pz; within the Alcohol group, 7 participants had spatial maxima at FCz and 7 participants had spatial maxima at Pz). These findings suggest that alcohol use did not influence topography of the P300 component. Instead, the distributions of the P300 component (the bimodal distribution of the P3a subcomponent, in particular) likely represent normal individual variation in P300 topography (Polich & Bloom, 1986).

Group- and sex-related differences. The latencies and amplitudes of the P3a and P3b subcomponents (see Table 3) were extracted from the electrode site at which the spatial maxima occurred for each subject. P3b latency data were non-normally distributed, $W(58) = .849$, $p < .001$, and also violated the assumption of homogeneity of variance (as shown by Levene's test, $F(3, 73) = 2.793$, $p = .046$). Although a log transformation of these data did not correct for the violation of the assumption of normality, this transformation did correct for the violation of homogeneity of variance, and so the log-transformed data were analyzed here.

Regarding P3a and P3b latency, there were no significant main effects of Group, $F(1, 67) = 0.455$, $p = .502$, $\eta^2 = .007$, and $F(1, 73) = 0.635$, $p = .428$, $\eta^2 = .009$, respectively. There were

also no significant main effects of Sex, F (1, 67) = 0.888, p = .349, η^2 = .013, and F(1, 73) = 2.135, p = .148, η^2 = .028, respectively. Similarly, there were no significant Group x Sex interaction effects, F(1, 67) = 1.040, p = .312, η^2 = .015, and F(1, 73) = 0.236, p = .628, η^2 = .003, respectively.

Regarding P3a and P3b amplitude, there were also no significant main effects of Group (F(1, 67) = 0.350, p = .556, η^2 = .005, and F(1, 73) = 5.152, p = .026, η^2 = .066, respectively) or Sex (F(1, 67) = 2.788, p = .100, η^2 = .040, and F(1, 73) = 0.113, p = .738, η^2 = .002, respectively). There were also no significant Group x Sex interaction effects here, F(1, 67) = 0.712, p = .402, η^2 = .011, and F(1, 73) = 0.067, p = .796, η^2 = .001, respectively.

F-ERN.

Spatial distribution and individual maxima. Similar to the P300 data, the grand average ERP difference waveforms showed that the spatial distributions for the F-ERN (negative feedback condition) were similar in the Control and Alcohol groups. In both groups, the F-ERN had its spatial minimum at the fronto-central electrode site (FCz). Figure 2 shows the comparison of (a) grand average difference ERP waveforms at electrode FCz, and (b) grand average topographical maps for the Control and Alcohol groups for the negative feedback condition.

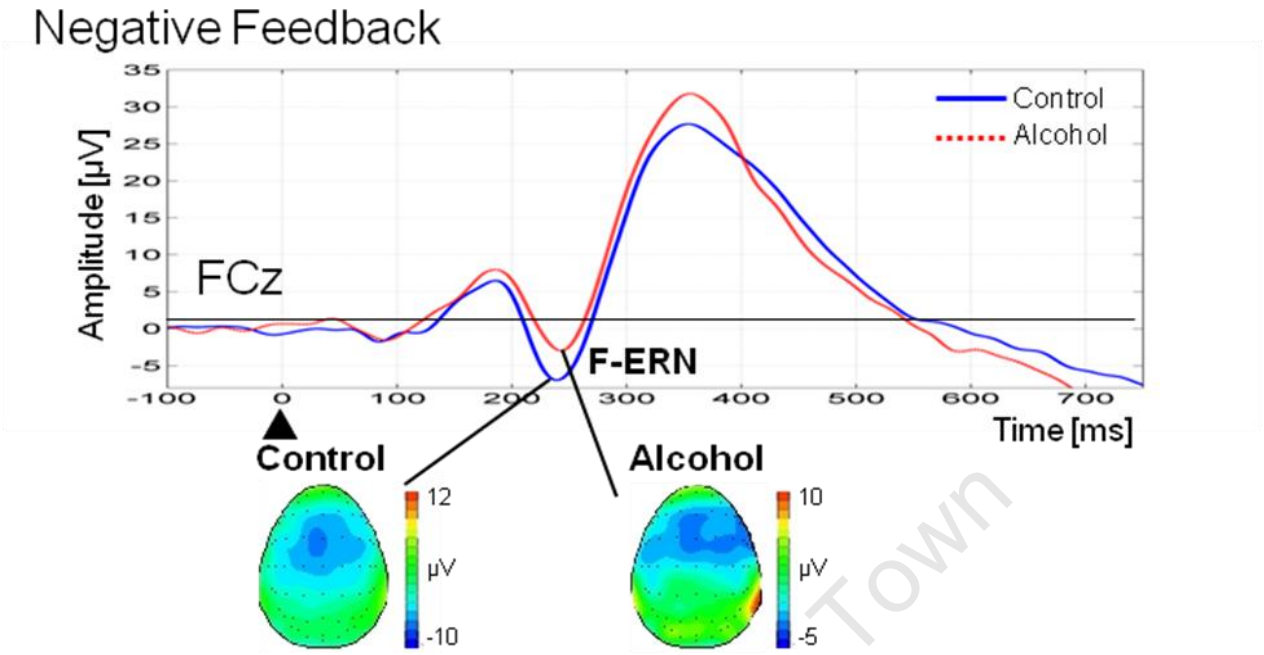

Figure 2. Grand average ERP difference waveform for the negative feedback condition (negative feedback – positive feedback condition) at electrode site FCz, showing comparison of Control and Alcohol groups. Grand average topographical maps are shown for the location of the F-ERN peak for both the Control and Alcohol groups.

In line with the spatial distributions of the grand average topographical maps in Figure 2, most subjects had a spatial minimum of the F-ERN at the fronto-central electrode site (FCz). There was, however, some inter-subject variability in the location of this maximum (see Table 4). Nevertheless, and similar to the findings for the P300, there was no group effect on the location of individual spatial maxima for the F-ERN. Specifically, the majority of participants in each group (i.e., 7 participants in the Alcohol group; 14 participants in the Control group) showed individual spatial maxima at FCz. Again, these results suggest that alcohol use did not influence the spatial distribution of this component.

Group- and sex-related differences. The latencies and amplitudes of the F-ERN (see Table 3) were extracted from the electrode site at which the spatial maxima occurred for each subject. Data for F-ERN latency violated the assumption of homogeneity of variance. A log transformation corrected this violation, and hence these transformed data were analyzed. Regarding F-ERN latency and amplitude, there were no significant main effects of Group ($F(1, 41) = 0.864$, $p = .358$, $\eta^2 = .021$, and $F(1, 41) = 6.543$, $p = .014$, $\eta^2 = .138$, respectively) or Sex ($F(1, 41) = 4.273$, $p = .045$, $\eta^2 = .094$, and $F(1, 41) = 2.973$, $p = .092$, $\eta^2 = .068$, respectively).

There were also no significant Group x Sex interaction effects for either F-ERN latency, $F(1, 41) = 1.269$, $p = .267$, $\eta^2 = .030$, or amplitude, $F(1, 41) = 0.142$, $p = .709$, $\eta^2 = .003$.

Primary Analysis II: Factor Analysis of Personality, Psychiatric, and Cognitive Measures

I conducted exploratory factor analysis using original (i.e., non-transformed) versions of all measures (the four personality measures, the psychiatric measure, and the two cognitive measures). Although log-transformed SCWT response inhibition composite scores had been used in the ANOVA described above, this transformation did not improve the normality of the distribution. Thus, I chose to use the original (non-transformed) SCWT response inhibition composite scores in the factor analysis. Furthermore, factor analysis is robust to deviations from normality and so non-normally distributed measures remain appropriate for this analysis.

Factor extraction and rotation method. I performed a factor analysis using principal axis factoring as the method of extraction and oblique rotation via the direct oblimin method. My decision to use oblique rotation was based on the *a priori* prediction that any emerging subfactors should be correlated. I extracted factors based on Kaiser's criterion for eigenvalues (i.e., eigenvalues above 1), using the scree plot to verify the number of factors.

Initial model. The first attempt at model-fitting was made using all seven measures. The sampling adequacy of the analysis was acceptable according to the Kaiser-Meyer-Olkin measure, KMO = .67, and all individual KMO values were larger than the acceptable level of 0.50. Despite the fact that Bartlett's test of sphericity indicated that the correlations between the measures were significantly large for factor analysis, $\chi^2(21) = 115.586$, $p < .001$, examination of the correlation matrix for the relevant personality, psychiatric, and cognitive measures (see Table 5) gave the first indication that these measures might not have provided the cohesion required for identification of an underlying construct. That is to say, the correlations between these measures were low. Only the associations between the four personality measures fell within appropriate parameters (Pearson's rho between 0.3 and 0.8). Specifically, moderate positive correlations existed between IVS-Imp and NSS ($p < .001$); IVS-Imp and the excitement-seeking composite ($p < .001$); and between NSS and the excitement-seeking composite ($p < .001$). In contrast, the correlation between the two cognitive measures ($p = .145$), as well as correlations across measures in the three categories, were rather low.

Table 5
Correlation Matrix for Personality, Psychiatric, and Cognitive Measures

Measure	1	2	3	4	5	6	7
Personality							
1. IVS-Imp[a]	-	.215	**.507**	**.346**	.171	-.069	.165
2. IVS-Ven[a]	-	-	.239	.206	.167	-.048	.005
3. NSS[b]	-	-	-	**.344**	.151	-.075	.080
4. Excitement-seeking composite[c]	-	-	-	-	.090	.184	.167
Psychiatric symptom counts							
5. Externalizing composite[d]	-	-	-	-	-	-.024	.113
Cognition							
6. SCWT response inhibition composite[e]	-	-	-	-	-	-	.084
7. ToL rule violations	-	-	-	-	-	-	-

Note. Data presented are Pearson's correlations. Moderate correlations (Pearson's *rho* between 0.3 and 0.8) appear in boldface. IVS-Imp = Impulsivity subscale of the Impulsivity-Venturesomeness Scale. IVS-Ven = Venturesomeness subscale of the Impulsivity-Venturesomeness Scale. NSS = Novelty Seeking Scale. SCWT = Stroop Color-Word Test. ToL = Tower of London. [a]From the Impulsivity-Venturesomeness scale. [b]From the Tridimensional Character Inventory. [c]Disinhibition and Boredom Susceptibility subscales of the Sensation Seeking Scale. [d]Total symptoms of conduct disorder and oppositional defiant disorder. [e]Total number of uncorrected and self-corrected errors on the inhibition task of the Stroop Color-Word test.

After factor extraction and rotation, the initial model suggested a two-factor solution. However, this solution did not provide a clear factor structure, as indicated in the pattern matrix (see Table 6) and confirmed using the structure matrix; additionally, the scree plot did not confirm that this initial two-factor solution was ideal. In this solution, the first factor (eigenvalue = 2.089) explained 21% of the variance, while the second factor (eigenvalue = 1.177) explained 7% of the variance. Thus, the initial two factors accounted for only 28% of the variance within the model (well below the acceptable level of 50%) (Kline, 2005).

Table 6
Pattern Matrix Factor Structure For Two-Factor Solution Using Seven Outcome Measures

Measure	Factor 1	Factor 2
Personality		
IVS-Imp[a]	**.708**	-.017
IVS-Ven[a]	.356	-.024
NSS[b]	**.703**	-.051
Excitement-seeking composite[c]	.445	.425
Psychiatric symptom counts		
Externalizing composite[d]	.255	-.014
Cognition		
SCWT response inhibition composite[e]	-.168	**.540**
ToL rule violations	.150	.187

Note. Data presented are factor loadings. Factor loadings larger than 0.50 appear in boldface. IVS-Imp = Impulsivity subscale of the Impulsivity-Venturesomeness Scale. IVS-Ven = Venturesomeness subscale of the Impulsivity-Venturesomeness Scale. NSS = Novelty Seeking Scale. SCWT = Stroop Color-Word Test. ToL = Tower of London. [a]From the Impulsivity-Venturesomeness scale. [b]From the Tridimensional Character Inventory. [c]Disinhibition and Boredom Susceptibility subscales of the Sensation Seeking Scale. [d]Total symptoms of conduct disorder and oppositional defiant disorder. [e]Total number of uncorrected and self-corrected errors on the inhibition task of the Stroop Color-Word test.

Subsequent models. Following this unsatisfactory model, I attempted to create several other models. I attempted each of these models based on the findings from previous models, and with the aim of providing a clear factor solution that accounted for at least 50% of the variance in the model. Measure inclusion in subsequent models was based on factor loadings for each preceding model, where measures with the lowest/most ambiguous loadings were eliminated in succession.

Based on this criterion, measures were eliminated from the model in the following order: the excitement-seeking composite was first to be removed, followed by the SCWT response inhibition composite, and then the externalizing composite. Following successive exclusion of measures, a two-factor structure was maintained for each model; however, none of these models satisfied the model criteria (i.e., clear factor solution with at least 50% explained variance) outlined above. After removing the externalizing composite, the factor structure of the resulting model indicated that a successful solution was unlikely. At this point, the indicator with the most ambiguous factor loading (-.001 on factor 1; .358 on factor 2) was the ToL rule violation score. If this indicator was removed from the model, a single-factor solution with three indicators (i.e., IVS-Imp, IVS-Ven, and NSS) remained. This solution fell short of the statistical requirements for a plausible model: the factor loading for IVS-Ven was .319, which is below the recommended threshold of .40 (Stevens, 1992); and the model explained only 37% of the variance, which is below the recommended threshold of 50% (Kline, 2005). More importantly, it is conceptually problematic that all remaining indicators were derived from self-report personality measures. In contrast with the aim of the factor analysis, which was to uncover a latent construct of disinhibition using a broad array of indicators, this model solution likely reflected methodological similarity between the personality measures. Consequently, at this point I abandoned attempts to create the model.

In addition to exploratory factor analyses using data for all participants, I attempted factor analysis, using similar methods, on data from the Alcohol group alone. I attempted the latter factor analysis to investigate whether higher average scores on several outcome measures (in the Alcohol group relative to the Alcohol and Control groups combined) would improve correlations between outcome measures, and consequently improve model fit. However, the overall pattern of correlations observed using data from all participants (i.e., Alcohol and Control groups combined) remained largely unchanged using scores from the Alcohol group alone. Consequently, and expectedly, data form the Alcohol group alone produced unsatisfactory factor solutions, much like those produced using data from all participants.

Discussion

Disinhibition is a core feature of alcohol use disorders (AUDs; Finn, 2002; Gorenstein & Newman, 1980; Porjesz & Rangaswamy, 2007; Zuckerman, 1979), and manifests in various ways in individuals with AUDs. A large number of studies report that such individuals display elevated levels of disinhibited personality traits (Justus et al., 2001; Masse & Tremblay, 1997; Sher et al., 2000), externalizing psychopathology (e.g., CD, ODD; Fein et al., 2007; Gorenstein & Newman, 1980; Krueger et al., 2002), executive cognitive disinhibition (Dolan et al., 2008; Sarkis et al., 2005; Tapert et al., 2002), as well as evidence of EEG correlates of disinhibition (Chen et al., 2007; Fein & Chang, 2008; Hansenne, 2006).

The purpose of the current study was to investigate the disinhibitory complex (i.e., the latent and generalized spectrum of externalizing pathologies and behaviors, as well as certain cognitive impairments and electrophysiological (EEG) changes) in a South African sample of treatment-naïve adolescents with AUDs. This is a particularly interesting and worthwhile research objective because (a) few studies have employed a broad range of indicators to examine the disinhibitory complex, (b) relative to research on adult AUDs, little is known about the relationship between disinhibition and AUDs during adolescence, and (c) treatment-naïve individuals with AUDs (who make up the largest proportion of the AUD population) are distinct from treated individuals, and may differ in the extent to which they exhibit the disinhibitory complex. The specific aims of the study were as follows: First, I aimed to examine and characterize the disinhibitory complex (using personality, psychiatric, cognitive, and EEG indices) in treatment-naïve adolescents with AUDs; second, I aimed to investigate sex differences in performance on these measures; third, I aimed to determine whether an underlying construct of disinhibition might explain elevated levels of disinhibition in AUDs observed on individual indices.

With regard to those specific aims, the central findings were: First, Alcohol-group participants showed some evidence of disinhibition relative to Control-group participants; however, the former were not consistently disinhibited across personality, psychiatric, cognitive, and EEG domains. Specifically, Alcohol-group participants were comparatively disinhibited on some personality measures (i.e., they scored higher on the Impulsivity subscale of the Impulsivity-Venturesomeness Scale (IVS-Imp) and the excitement-seeking composite, but not on the Venturesomeness subscale of the Impulsivity-Venturesomeness Scale (IVS-Ven) or on the

Novelty Seeking Scale (NSS)), as well as on both cognitive measures (i.e., they scored higher on the Stroop Color-Word test (SCWT) response inhibition composite and Tower of London (ToL) rule violations measures). However, those in the Alcohol group did not demonstrate elevated levels of externalizing psychopathology (as measured on the externalizing composite), and they showed no EEG correlates of disinhibition (as measured using the P300 and F-ERN components).

Second, males showed inconsistent evidence of disinhibition relative to females. Specifically, males were relatively disinhibited on a single personality measure (i.e., they scored higher on the IVS-Ven), and they showed relatively elevated levels of externalizing psychopathology (as measured on the externalizing composite), but they did not show relatively increased levels of disinhibition on any other measures.

Third, attempts to model the latent construct of disinhibition, using the selected indices and the obtained dataset, were unsuccessful.

The following sections will seek to interpret these findings in terms of the disinhibitory complex (of which AUDs are one manifestation), and will seek to relate these findings specifically to treatment-naïve individuals with AUDs. With regard to the first central finding, I begin with a discussion of domains where AUD-related disinhibition emerged on outcome measures (i.e., personality and cognitive domains), and then proceed to discuss possible reasons for inconsistent AUD-related disinhibition across domains of measurement. Then, I discuss sex-related differences in disinhibition. Finally, and with regard to the third central finding, I provide explanations for the unsuccessful modeling of the latent construct of disinhibition.

AUD-related Disinhibition on Some Personality and Cognitive Measures

Disinhibited personality.

Impulsivity and excitement-seeking. In the current study, the Alcohol-group participants demonstrated elevated levels of impulsive and excitement-seeking traits relative to the Control-group participants. The implication of these between-group differences is that the behavior of the current sample of adolescents with AUDs appears to be characterized by difficulties with inhibitory control (i.e., elevated impulsive personality traits), and that these adolescents tend to become bored easily and to engage in appetitive behaviors to experience positive affect (i.e., elevated excitement-seeking personality traits) (Finn, 2002). These features of behavior among

Alcohol-group participants were expected because AUDs are associated with compromised inhibitory control (Finn, 2002; Gorenstein & Newman, 1980; Porjesz & Rangaswamy, 2007; Zuckerman, 1979), and alcohol consumption itself may be considered an appetitive behavior (Zuckerman, 1994).

Venturesomeness and novelty-seeking. In contrast with findings for impulsivity and excitement-seeking, there were no between-group differences in venturesomeness or novelty-seeking traits. I propose separate reasons to account for the absence of between-group differences in (a) venturesomeness and (b) novelty-seeking traits. Regarding the IVS-Ven, items on this subscale largely concern the appeal of various expensive outdoor activities (e.g., scuba diving, caving, water skiing, parachute jumping). Although responses to these items do not necessarily rely on prior participation in such activities, the current sample was drawn largely from socioeconomically disadvantaged households, and so it is quite likely that these participants had never engaged in these activities, and that they were, in fact, also unfamiliar with them. If participants were indeed unfamiliar with the content of items included in the IVS-Ven, the validity of this subscale may have been compromised. Because the IVS has not been used previously with a South African population beyond the larger project within which this study was nested, there is no information available regarding cultural/socioeconomic limitations of the IVS-Ven for use within this population.

A second possible reason for the absence of a between-group difference on the IVS-Ven concerns the construct validity of this subscale. The IVS-Ven is used considerably less often than the corresponding IVS-Imp subscale. In fact, use of the IVS in AUDs is often limited to the IVS-Imp (e.g., Finn et al., 2005; Finn et al., 2002; Zernicke et al., 2010). This relatively frequent use of the IVS-Imp may be attributed to the observation that, of all the forms of disinhibited personality, impulsivity is the most direct measurement of behavioral impairments in inhibitory control, and thus most consistently related to AUDs (Finn, 2002; Verdejo-García et al., 2008). The IVS-Ven, on the other hand, is not only a relatively underused subscale of the IVS, but is also used to examine disinhibited personality considerably less often than either the NSS or the Sensation Seeking Scale (from which the excitement-seeking composite was created). Taken together, (a) relatively infrequent usage of the IVS-Ven measure elsewhere, and (b) the absence of a between-group difference on this measure here, may provide an indication that this subscale

is a less effective index of disinhibited personality. In other words, the IVS-Ven measure may have relatively poor construct validity.

With regard to relative preserved construct validity on the other measures of disinhibited personality used here (i.e., IVS-Imp, NSS, and excitement-seeking), items on the IVS-Ven have a particular focus on the appeal of activities, whereas items on the other measures of disinhibited personality focus less on particular activities and more on general tendencies and behaviors (e.g., "Do you generally do and say things without stopping to think?"; "I like it when people can do whatever they want, without strict rules and regulations"). The broader focus of items included in the IVS-Imp, NSS, and excitement-seeking composite may have led to relatively preserved construct validity of these measures.

In summary, possible compromised validity of the IVS-Ven for use within the current population as well as possible questions concerning construct validity (i.e., the degree to which the IVS-Ven indexes disinhibited personality) of this subscale are potential reasons for the lack of between-group effects observed here.

Regarding novelty-seeking, non-significant between-group differences on this measure were unexpected because novelty-seeking and impulsivity measures assess essentially the same trait (Finn et al., 2002). Because both measures appear to index similar impulsive, under-controlled behaviors (Cloninger, Sigvardsson, & Bohman, 1988; Masse & Tremblay, 1997), one would expect observed between-group differences in impulsivity to extend to novelty-seeking. Nevertheless, the test for between-group differences in novelty-seeking ($p = .011$) would have produced a significant result if a more lenient threshold for statistical significance (e.g., the conventional $p = 0.05$) had been applied. Instead, I applied the Sidak correction for multiple comparisons, resulting in a conservative threshold of $p = 0.0034$. Although falling short of the modified threshold for statistical significance, the result of the test for between-group differences in novelty-seeking indicates, at least, a trend toward the expected result.

In summary, between-group differences were not apparent for all forms of disinhibited personality assessed here; nevertheless, these results indicate some elevation in disinhibited personality in individuals with AUDs. Such an elevation is observed frequently in previous work (e.g., Ball, 1996; Finn et al., 2002; von Knorring, von Knorring, Smigan, Lindberg, & Edholm, 1987).

Cognitive disinhibition.

Response inhibition on the SCWT. Comparatively higher scores on the SCWT response inhibition composite by Alcohol-group participants are indicative of relative difficulties inhibiting the prepotent response (i.e., selecting an appropriate response over a competing, inappropriate response) (Luna et al., 2010). Response inhibition impairments are, in turn, typically rooted in a compromised ability to detect and resolve pre-response conflict (van Veen & Carter, 2006).

Engagement on the SCWT has been associated with activity of the anterior cingulate cortex (ACC), one of the primary brain structures implicated in cognitive control (Botvinick et al., 2001; Carter et al., 1998). Within the cognitive control system, the ACC is responsible for detecting both pre- and post-response conflict (Carter et al., 1998; Cavanagh et al., 2009; Kerns et al., 2004; van Veen & Carter, 2002). If the ACC does not adequately detect the presence of competing response options, executive resources are not allocated to resolve such interference, and an automatic (i.e., incorrect) response is produced on incongruent trials. Following error commission, impaired ability to detect post-response conflict in turn reduces capacity to provide a self-correcting response following error commission (Shuster & Toplak, 2009). In this case, if the ACC does not detect a discrepancy between the produced and desired responses (i.e., that an error occurred), a self-correcting response is not produced. Thus, difficulties detecting pre-response conflict lead to *increased* error rate, while difficulties detecting post-response conflict lead to *decreased* ratio of self-corrected errors to uncorrected errors. In other words, ability to detect pre-response conflict is negatively associated with error rate, while ability to detect post-response conflict is positively associated with self-corrected error rate.

In the current study, the SCWT response inhibition composite was calculated by summing together the number of uncorrected and number of self-corrected errors on the inhibition task of the SCWT. It may appear problematic to combine uncorrected and self-corrected error scores in this way because uncorrected errors signify *impaired* ability to detect and resolve *pre*-response conflict, while self-corrected errors (as a proportion of total errors) signify *preserved* ability to detect and resolve *post*-response conflict. Despite these disparities, both uncorrected and self-corrected errors signify error commission, i.e., an impaired ability to initially detect and resolve pre-response conflict. Higher scores on the SCWT response inhibition

composite thus indicate impairments in response inhibition attributable to difficulty detecting and resolving pre-response conflict.

In everyday life, impaired response inhibition, as observed among Alcohol-group participants, leads to a tendency to favor automatic responses, even when this tendency results in undesirable outcomes. Response inhibition impairments manifest as generalized disinhibited behavior, which is a hallmark of diminished cognitive control in AUDs (Lawrence et al., 2009; G. P. Strauss, Allen, Jorgensen, & Cramer, 2005).

***Response inhibition on the ToL task*.** Alcohol-group participants committed more rule violations than Control-group participants on this task. Similar to number of errors (uncorrected and self-corrected) on the SCWT, increased number of rule violations on the ToL is indicative of difficulties with response inhibition and self-monitoring, as well as an associated preference for automatic responses (Culbertson & Zillmer, 2001). An increased number of rule violations on the ToL may also, however, have an affective component. For instance, individuals with oppositional tendencies may intentionally commit more rule violations in accordance with an overall non-compliant behavioral style (Giancola et al., 1998). Indeed, AUDs are consistently associated with a range of oppositional and non-compliant behaviors (Iacono et al., 2002; H. I. Kaplan & Sadock, 1998). Furthermore, individuals may commit rule violations by disinhibiting in response to frustration arising from difficulties with the task (Culbertson & Zillmer, 2001). Although it is not possible within the limits of the current study's design to disentangle the relative contributions to rule violation commission of (a) response inhibition impairments, (b) oppositional and non-compliant tendencies, and (c) disinhibition in response to frustration, it is worth noting that all three possible explanations are consistent with an underlying disinhibitory complex in individuals with AUDs (Lawrence et al., 2009; G. P. Strauss et al., 2005).

Inconsistent AUD-related Disinhibition Across Measurement Domains

Measurement scope of disinhibition. As mentioned previously, other studies have consistently documented AUD-related disinhibition in personality, psychiatric, cognitive, and EEG domains. However, most previously-published studies have focused on a single domain; as Reynolds et al. (2006) note, few have, in their investigations of disinhibition of AUDs, considered these domains simultaneously. Of those that have investigated disinhibition across two or more domains, some have reported consistent evidence of disinhibition in individuals

with AUDs, while others have not. For example, Lawrence et al. (2009) and Dolan et al. (2008) each examined two domains of disinhibition and found, respectively, that individuals with AUDs had relatively impaired response inhibition and increased trait impulsivity, and relatively increased trait impulsivity and disinhibited performance on a gambling task. Similarly, Kamarajan et al. (2010) examined three domains of disinhibition (i.e., personality, cognitive, and EEG domains) and found significantly elevated levels of trait impulsivity and attenuated P300 amplitudes in individuals with AUDs. Within the same sample, however, individuals with AUDs performed comparably to healthy controls on a measure of executive cognitive disinhibition, and did not show significant reductions in F-ERN amplitude.

The scope of the Kamarajan et al. study (i.e., personality, cognitive, and EEG domains) was slightly more limited than that of the current study, which also included a psychiatric composite measure (i.e., externalizing composite). Nevertheless, the findings presented in the former study indicate that disinhibition in AUDs may be observed inconsistently both across and within domains (viz., between-group differences in the personality domain, but not in the cognitive domain; in the EEG domain, between-group differences in P300 amplitude, but not in F-ERN amplitude). This observation mirrors that of the current study, where those in the Alcohol group showed inconsistent evidence of disinhibition both across and within domains (viz., between-group differences in the cognitive domain, but not in the psychiatric or EEG domains; in the personality domain, between-group differences in scores on the IVS-Imp and the excitement-seeking composite, but not on the IVS-Ven or NSS).

Of note, however, is that Kamarajan et al. used a sample of treated adults with AUDs, whereas this study used a sample of treatment-naïve adolescents with AUDs. Although Kamarajan et al. showed that disinhibition may present inconsistently in treated adults with AUDs, the use of treatment-naïve adolescents in the current study might have contributed further to the inconsistent profile of disinhibition observed here. To explain this point, I present in the next section a brief discussion of the possible influences of using (a) treatment-naïve rather than treated samples, and (b) adolescents rather than adults. Although some reasons for individual non-significant group differences were presented previously, the following factors apply over and above preceding arguments.

Treatment-naïve versus treated AUD samples. Previous research (e.g., Beck et al., 2009; Dom, Hulstijn, & Sabbe, 2006; Goldstein et al., 2004; J. M. Mitchell et al., 2005) on disinhibition in AUD samples has, by and large, used participants from treatment-seeking or treated populations. Participants in this study, however, had never undergone treatment for AUDs. Importantly, this characteristic set the current sample apart from most AUD samples because treatment-naïve individuals with AUDs tend to have less severe alcoholism, and less psychiatric and polysubstance comorbidity than their treatment-seeking counterparts (Di Sclafani et al., 2008; Fein & Landman, 2005; Gazdzinski et al., 2008). The distinctions between treatment-naïve and treated AUD samples imply that the usual profile of disinhibition in AUD (based on findings in treated samples) is not necessarily applicable in samples of treatment-naïve individuals with AUDs. In addition to the noted distinctions between treatment-naïve and treated samples, any significant substance (other than alcohol) use and diagnostically significant psychopathology (other than AUDs) warranted exclusion from study participation. In this way, the current study's eligibility criteria further restricted levels of polysubstance use and other psychopathology within this treatment-naïve AUD group.

In each section below, I present evidence to explain inconsistencies in disinhibition with regard to (a) reduced severity of alcoholism, (b) less psychiatric comorbidity, and (c) less polysubstance comorbidity in the current treatment-naïve sample relative to treated samples studied elsewhere. These explanations focus on domains where Alcohol-group participants showed inconsistent or non-significant evidence of disinhibition (i.e., those findings that did not conform to the *a priori* predictions of the current study).

Disinhibited personality in treatment-naïve AUDs. Alcohol exposure is associated with elevated disinhibited personality traits (Bjork et al., 2004; J. M. Mitchell et al., 2005; Whiteside & Lynam, 2003). Specifically, cumulative alcohol exposure may promote the development of these traits, and may also exacerbate traits that were present previously (Verdejo-García et al., 2008; Zernicke et al., 2010). Disinhibited personality and externalizing psychopathology (including AUDs) are believed to exist on a continuum of externalizing behavior (Krueger et al., 2002); this proposal is based on extensively documented correlations between the two (e.g., Howard, Kivlahan, & Walker, 1997; Sher & Trull, 1994). Others have reported, however, that associations between disinhibited personality and AUDs no longer apply when polysubstance comorbidity is taken into account (McGue, Iacono, & Slutske, 1999). For these reasons,

comparatively lower levels of (a) alcohol exposure, (b) other externalizing psychopathology (i.e., aside from AUDs), and (c) polysubstance use in treatment-naïve versus treated individuals with AUDs might have constrained levels of disinhibited personality in the current sample of alcohol-using participants, thus leading to inconsistent between-group differences in disinhibited personality (i.e., significant between-group differences in impulsivity and excitement-seeking; non-significant between-group differences in venturesomeness and novelty-seeking).

Externalizing psychopathology in treatment-naïve AUDs. Although there was a trend (p = .004) towards elevated sub-diagnostic threshold externalizing psychopathology (as measured using the externalizing composite, i.e., aside from AUDs) among Alcohol-group participants relative to Control-group participants, this difference was non-significant at the modified threshold for significance (i.e., p = 0.0034). As mentioned previously, treatment-naïve individuals with AUDs generally have less psychiatric comorbidity than their treatment-seeking counterparts (Di Sclafani et al., 2008). In addition, this study's eligibility criteria specified that any diagnostically significant externalizing psychopathology (other than AUDs) warranted exclusion from study participation. The current Alcohol-group participants thus had less externalizing psychopathology (i.e., strictly sub-diagnostic threshold psychopathology) than that observed in most samples studied elsewhere (e.g., Carlson et al., 1999; Iacono et al., 2002; Zernicke et al., 2010). Taken together, the treatment-naive status of the current sample and the study's eligibility criteria are two plausible explanations for the absence of a significant group effect on the externalizing composite measure.

EEG correlates of disinhibition. Before proceeding with a discussion of the influence of the treatment-naïve status of the current AUD group on EEG correlates of disinhibition, I first discuss some trends that emerged in these data, and then discuss the implications of non-significant between-group differences in these measures.

Isolated trends toward between-group differences. There were no statistically significant between-group differences in EEG correlates of disinhibition in the current study. However, the grand average difference waveforms for both the P3b subcomponent of the P300 (Figure 1) and the F-ERN component (Figure 2) suggest amplitude reductions among Alcohol-group participants relative to Control-group participants. Although these differences were not large enough to produce statistically significant results using the modified threshold for statistical significance, tests for between-group differences in (a) P3b amplitude (p = .026) and (b) F-ERN

amplitude (p = .014) would have produced significant results if a more lenient threshold for statistical significance was applied.

In contrast with trends toward between-group differences in amplitudes of the P3b subcomponent and F-ERN component, there were no such trends toward either attenuated amplitude of the P3a subcomponent, or prolonged latencies of the P3a, P3b, and the F-ERN. Specifically, tests for between-group differences in (a) P3a amplitude (p = .556), and (b) latencies of the P3a, P3b and the F-ERN (p = .502; p = .428; p = .358, respectively) were non-significant and would remain so even if a more lenient threshold for statistical significance were to be applied. Indeed, other studies have reported similar results; specifically, these studies have found an absence of trends toward between-group differences in these particular EEG parameters (i.e., P3a amplitude, and latencies of the P3a, P3b and the F-ERN) alongside clear trends toward between-group differences in amplitudes of the P3b and F-ERN (Cohen et al., 1995; Maurage et al., 2007; Porjesz, Begleiter, Bihari, & Kissin, 1987).

Studies that have found between-group differences in P3a amplitude and F-ERN latency between individuals with AUDs and healthy controls have generally reported relatively attenuated P3a amplitudes and prolonged F-ERN latencies in AUDs (Rodriguez Holguin et al., 1999; Yeung & Cohen, 2006). The observed (non-significant) differences in P3a amplitude and F-ERN latency in the current study, however, were in the opposite direction to that expected based on those studies. Specifically, the P3a subcomponent was slightly smaller and the F-ERN was slightly later among Control-group participants relative to Alcohol-group participants. Latencies of the P3a and P3b subcomponents, on the other hand, were slightly prolonged among Alcohol-group participants relative to Control-group participants (i.e., a non-significant difference, but in the direction expected based on studies that have found such differences) (Pfefferbaum, Horvath, Roth, & Kopell, 1979; Whipple, Berman, & Noble, 1991). Regarding P3a amplitude and F-ERN latency, it is unclear why these differences were in an opposite direction to that expected; it is worth noting, however, that these differences were non-significant, and, further, that between-group differences in these EEG parameters are reported inconsistently in the literature.

Despite the noted trends toward between–group differences in some EEG parameters (i.e., P3b and F-ERN amplitude), overall non-significant results across all EEG parameters suggest reasonably preserved P300 and F-ERN among Alcohol-group participants. I will now

demonstrate the implications of these overall non-significant findings by pointing out the functional significance of the P300 and F-ERN.

Implication of non-significant differences in the P300 and F-ERN. Preserved P300 represents efficient neural inhibition processes; changes in the P300 (reduced P3b amplitude in particular) are thought to represent impaired neural inhibition (Knight, 1997; Polich, 2007). The F-ERN, on the other hand, represents sensitivity to negative feedback (Fein & Chang, 2008). Preserved F-ERN is an indication of adaptive levels of sensitivity to negative feedback; changes in the F-ERN (reduced amplitude in particular) may indicate hyposensitivity to negative feedback.

The absence of between-group differences on the P300 and F-ERN measures suggests, therefore, that AUDs were not related to impaired neural inhibition or hyposensitivity to negative feedback, respectively. In other words, and with regard to preserved P300, these results indicate that Alcohol-group participants were able to successfully inhibit extraneous neural activity, thereby contributing to intact selective attention faculties.

With regard to preserved F-ERN, these results suggest that the Alcohol-group participants maintained adaptive levels of sensitivity to negative feedback. Hyposensitivity to negative feedback in other AUD samples is believed to prompt poor or risky decision-making (i.e., the persistence of harmful behaviors, such as drinking behaviors, despite mounting negative consequences of the behaviors), which is, in turn, one of the central features of AUDs (L. Clark & Robbins, 2002; T. J. Crowley et al., 2006; Goudriaan et al., 2005). There seems to be an incongruency here, then: Participants in the current Alcohol group endorsed diagnostically significant levels of AUDs (which are usually characterized by poor decision-making) alongside apparently adaptive levels of sensitivity to negative feedback (i.e., preserved F-ERN, which is indicative of adaptive decision-making capacity). This apparent incongruency suggests, therefore, that either (a) poor decision-making among Alcohol-group participants is rooted in an alternate mechanism not examined here (e.g., hypersensitivity to reward), (b) the F-ERN is not an accurate measure of hyposensitivity to punishment, or (c) the F-ERN was moderated by sample characteristics here (such as the treatment-naïve status of the sample).

It is not possible within the limits of this study's design to directly examine the relevance of the two former proposed reasons for non-significant differences in the F-ERN; however, given the probable impact of the treatment-naïve status of the sample on other indices of disinhibition

examined here, I now discuss the possible influence of this characteristic on the observed non-significant between-group differences in EEG correlates.

EEG correlates of disinhibition in treatment-naïve AUDs. Changes in the P300 and the ERN are not exclusive to AUDs. Various externalizing disorders and other SUDs have documented reductions in the P300 (e.g., Iacono, Carlson, Taylor, Elkins, & McGue, 1999; Porjesz & Rangaswamy, 2007; Porjesz et al., 2005) and ERN (e.g., de Bruijn et al., 2006; Forman et al., 2004; Franken, van Strien, Franzek, & van de Wetering, 2007; Ruchsow et al., 2006; van Meel, Heslenfeld, Oosterlaan, & Sergeant, 2007). In fact, alcohol exposure, comorbid psychopathology, and other substance use may contribute independently to AUD-related ERP reductions noted elsewhere. For example, animal studies have found that even relatively brief alcohol exposure in adolescence is sufficient to result in P300 changes that endure into adulthood (Criado, Wills, Walker, & Ehlers, 2008; Ehlers & Criado, 2010); others have suggested that P300 and ERN reductions are no longer applicable when psychiatric comorbidity is taken into account (Hill, Locke, & Steinhauer, 1999; Kessler, 2004). Because EEG changes are not exclusive to AUDs, comparatively lower levels of alcohol exposure, psychiatric comorbidity, and polysubstance use in the current treatment-naïve sample are likely reasons for an absence of statistically significant P300 and F-ERN amplitude reductions among Alcohol-group participants. Indeed, others have noted dramatically smaller reductions in P300 amplitudes in treatment-naïve samples relative to treated samples (Fein & Andrew, 2011).

In contrast with the P300, the F-ERN is relatively understudied. Furthermore, I am not aware of a comparable study that measured the F-ERN in a similar sample (i.e., treatment-naïve adolescents with AUDs), using an equivalent task (i.e., the Balloon Analogue Risk Task (BART)). With regard to methodology and use of particular experimental paradigms, although some researchers (e.g., Kamarajan et al., 2010) have used traditional gambling tasks to document a trend towards F-ERN reductions in (treatment-seeking) individuals with AUDs, EEG activity during administration of the BART is virtually untapped. Only two previous studies (M. J. Crowley et al., 2009; Fein & Chang, 2008) have examined the F-ERN during administration of the BART. Neither study, however, used a sample similar to that used here; the former examined F-ERN in adolescents with prenatal exposure to drugs (including cocaine), whereas the latter focused on treatment-naïve adults with AUDs. Furthermore, neither study used a control group:

Crowley et al. examined the F-ERN as a function of risk-taking propensity, whereas Fein and Chang examined the F-ERN as a function of genetic vulnerability to AUDs.

In summary, differences in sample characteristics (i.e., age, treatment status, and history of drug exposure), differences in methodology and experimental paradigms, as well as inconsistent use of control groups in previous studies limit (a) possible inferences about the relationship between AUDs and the F-ERN, as well as (b) possible comparisons between the current findings and those reported elsewhere. Nevertheless, these studies have shown that (a) the F-ERN is influenced by genetic vulnerability to AUDs, and (b) the F-ERN is (non-significantly) reduced in both treatment-naïve (as in the current study) and treated (as shown elsewhere) individuals with AUDs (Fein & Chang, 2008; Kamarajan et al., 2010). Whereas treatment-naïve individuals with AUDs appear to be spared from changes in the P300, these findings suggest that the F-ERN might be an endophenotype of AUDs, and, further, that the F-ERN is changed (although not substantially, it seems) in both treated and treatment-naïve individuals with AUDs .

Adolescents versus adults with AUDs. Studies of disinhibition in AUDs focus primarily on adult samples (Velanova et al., 2008). Those studies that have investigated AUDs in adolescents (e.g., Andrucci, Archer, Pancoast, & Gordon, 1989; Bauer & Hesselbrock, 2003; Carlson et al., 1999) have rarely controlled for extraneous influences on disinhibition, such as childhood externalizing diagnoses and other substance involvement. Therefore, because (a) the adolescent phase is relatively understudied, and (b) many studies that focus on adolescence are confounded by extraneous issues, relatively little is known about the profile of disinhibition in adolescent AUDs.

The study of AUD-related disinhibition specific to the adolescent phase is warranted because disinhibition in adolescents with AUDs is not the same as disinhibition in adults with AUDs. The reason for this is four-fold. First, adolescence is a critical phase of brain development, characterized by significant neural maturation, dendritic pruning, and increased myelination (Gogtay et al., 2004; Sowell, Thompson, & Toga, 2004). Due to ongoing maturation during this phase, adolescents have relatively immature cognitive control functions in relation to adults (Arbel & Donchin, 2011; Culbertson & Zillmer, 2001; Davies et al., 2004; Geier et al., 2010; Levin et al., 1991; Rand et al., 1963; Steinberg et al., 2008; Velanova et al., 2008; Yücel & Lubman, 2007). Second, maturation during adolescence may render the adolescent brain

particularly vulnerable to the neurotoxic effects of alcohol, leading to increased susceptibility to alcohol-related cognitive and EEG changes (Brown & Tapert, 2004; Dahl, 2004; Hill, Yuan, & Locke, 1999; Polich, Pollock, & Bloom, 1994; Rodriguez Holguin et al., 1999; Tapert & Brown, 1999). Third, adolescents usually have less protracted drinking histories than adults, simply because the latter are likely to have been drinking for much longer. The brains of adolescents with AUDs have thus endured relatively less exposure to the neurotoxic effects of alcohol. Chronic and heavy alcohol consumption can lead to homeostatic dysregulation, thus weakening overall self-regulation (Koob & Le Moal, 1997). Relatively less cumulative alcohol exposure may therefore limit manifestations of disinhibition in adolescents with AUDs.

Despite the relative vulnerability of the maturing adolescent brain, the existing literature suggests that the adverse effects of AUDs in adolescents tend to be more subtle than those exhibited by adults with AUDs (Moss, Kirisci, Gordon, & Tarter, 1994). Indeed, this notion is consistent with the findings presented here. It is possible, therefore, that relatively short drinking histories in the current sample of adolescents with AUDs (compared to adult samples studied elsewhere) contributed to inconsistent evidence of disinhibition in this group. Nevertheless, because AUDs are relatively understudied in both adolescent and treatment-naïve groups, it is not possible to say whether age or treatment-naïve status made a larger contribution to the inconsistent profile observed here.

Fourth, individual differences in the maturation rate of cognitive control faculties over the adolescent period may lead to relatively variable cognitive control faculties among adolescents relative to adults. Possible increased variability in these faculties within the current adolescent sample may thus have obscured further possible group differences in disinhibition, thus contributing to the inconsistent picture of disinhibition observed here.

Cognitive control in socioeconomically disadvantaged samples. Although this investigation does not attempt to compare the findings presented here to those presented elsewhere using comparatively privileged or first-world samples, it is important to consider the influence of the background of the current sample on the findings. As mentioned previously, the current sample was drawn largely from socioeconomically disadvantaged households. It is well-established that socioeconomic deprivation has a negative impact on overall cognitive development (Bergen, 2008; Kaplan et al,, 2001 Lee, Kawachi, Berkman, & Grodstein, 2003). Although the Alcohol-group participants did demonstrate relative evidence of disinhibition on

the cognitive measures, one may speculate that overall socioeconomic deprivation in the sample (including both Control-group and Alcohol-group participants) may have had a substantial influence on overall performance, thus obscuring further AUD-related differences in disinhibition.

Of course, and as mentioned in previous instances, it is also possible that the stringent threshold for statistical significance applied in this study lead to fewer significant findings than those reported elsewhere. Some other studies examining multi-method indices of disinhibition did not make similar adjustments for the number of between-group comparisons (Dolan et al., 2008; Kamarajan et al., 2010; Lawrence et al., 2009). Therefore, the ratio of statistically significant between-group differences to non-significant between-group differences may have been inflated in those studies. This possible inflated ratio of statistically significant results may have contributed to relatively consistent profiles of disinhibition reported in those papers. Further, given differences in selection of the threshold for statistical significance, it is possible that the pattern of results observed here (i.e., inconsistent evidence of disinhibition across and within domains) is at least partially a reflection of the way in which disinhibition truly manifests, and not purely a result of the current sample characteristics (i.e., treatment-naïve adolescents). I briefly address this issue at a later stage in this manuscript.

Sex Differences in Disinhibition

There was only partial evidence of sex differences on the various outcome measures. Specifically, males exhibited elevated venturesomeness traits and externalizing psychopathology (as measured using the externalizing composite) relative to females, but there were no sex differences in the cognitive or EEG domains. In the following sections, I discuss the reasons for (a) significant sex differences in venturesomeness and externalizing psychopathology, and (b) the absence of sex differences in cognitive and EEG domains of disinhibition.

Some sex differences in disinhibited personality and externalizing psychopathology.

The observed sex differences in venturesomeness traits and externalizing psychopathology were expected because males generally exhibit more evidence of disinhibited personality traits and of externalizing psychopathology than females (Arnett, 1996; Finn & Hall, 2004; Hicks et al., 2007; Iacono et al., 2003; Laucht et al., 2007; Loeber, Burke, Lahey, Winters, & Zera, 2000). In addition, these sex differences may be more pronounced during the adolescent

phase due to sex-specific maturation rates of the functions supporting cognitive control. As mentioned previously, cognitive control faculties have a protracted developmental trajectory, extending from childhood to adulthood (Arbel & Donchin, 2011; Culbertson & Zillmer, 2001; Davies et al., 2004; Geier et al., 2010; Levin et al., 1991; Rand et al., 1963; Steinberg et al., 2008; Velanova et al., 2008; Yücel & Lubman, 2007). Some suggest that a slower rate of development of the functions supporting cognitive control in young males relative to females may accentuate sex-related differences in disinhibition during early adolescence (Hill & Steinhauer, 1993; Steinhauer & Hill, 1993).

With regard to the venturesomeness measure (IVS-Ven), it is not immediately clear why the observed sex difference on this measure did not generalize to other measures of disinhibited personality. However, and as noted earlier, items on the IVS-Ven largely concern the appeal of outdoor activities that include a significant risk component, such as scuba diving, caving, water skiing, and parachute jumping. Other research has shown that boys have an increased propensity for risk-taking than girls, and that this relatively increased propensity is independent of AUD status (e.g., Arnett, 1996; Laucht et al., 2007). In contrast to the IVS-Ven measure, the other measures of disinhibited personality (viz., IVS-Imp, NSS and excitement-seeking) examined here do not focus specifically on risky outdoor activities. Taken together, the facts that (a) boys are more inclined to participate in risky activities, and (b) items on the IVS-Ven focus on the appeal of risky activities (while the other measures of disinhibited personality do not share this focus), may explain the observed sex differences on the venturesomeness measure and absence of such differences on the other measures of disinhibited personality.

Absence of sex differences in cognitive and EEG domains. Although many researchers propose that males, independent of AUD status, are more likely to exhibit evidence of disinhibition than females (Hicks et al., 2007), mixed reports exist in the AUD literature with regard to sex differences in cognitive and EEG domains of disinhibition (e.g., M. J. Crowley et al., 2009; Fein & Andrew, 2011; Hoffman & Polich, 1999; J. M. Mitchell et al., 2005; Nelson, Patrick, Collins, Lang, & Bernat, 2011; S. Smith & Fein, 2010; Stout, Rock, Campbell, Busemeyer, & Finn, 2005). For instance, and with regard to the cognitive domain, Mitchell et al. (2005) and Smith and Fein (2010) reported comparable performance between males and females on a delay discounting task and on a composite measure including indices of response inhibition. Stout et al. (2005) also reported similar performance between males and females on a measure of

risky decision-making; however, separate analyses by gender indicated that males with AUDs performed more poorly than those without, and that this AUD-related deficit was not observed among female participants. Thus, it appears that sex-related differences in disinhibition may present inconsistently in AUD populations, and, indeed, that sex and AUD status may interact in the manifestation of disinhibition.

Compared to the other domains of disinhibition, females with AUDs are particularly understudied with regard to the EEG domain. Nevertheless, the few studies that have considered alcohol-exposed female populations have uncovered inconsistent evidence of sex-related differences within this domain. For instance, and with regard to the F-ERN, Crowley at al. (2009) examined a sample of individuals prenatally exposed to cocaine and other drugs and found attenuated difference F-ERN waveforms for females relative to males (i.e., males showed more variability in their responses to the presentation of positive and negative feedback). These sex-related differences may suggest relatively increased disinhibition among females. However, in another study that reported F-ERN decrements following acute exposure to alcohol in a sample of non-substance-abusing individuals, females did not show reduced F-ERN amplitudes compared to males (Curtin & Fairchild, 2003). Although the samples used in these studies differ from the current sample (and so the results of these studies cannot be compared directly to those found here), these contrasting findings suggest, at least, that sex-related differences in the F-ERN among individuals exposed to alcohol are inconsistent.

With regard to the P300, many studies focusing on males with AUDs have detected decrements in the amplitude of this component (Cohen et al., 1995; Emmerson, Diustman, Shearer, & Chamberlin, 1987; Porjesz et al., 1987). A study focusing on females, on the other hand, found no differences in the P300 between those with AUDs and those without (Parsons, Sinha, & Williams, 1990). These findings appear to suggest that females with AUDs are less susceptible to P300 decrements than males with AUDs. However, studies examining male and female alcohol-exposed individuals simultaneously indicate that this may not be the case. For instance, Nelson et al. (2011) and Fein and Andrew (2011) failed to find overall sex differences in the P300 either following acute administration of alcohol in a sample of non-substance-abusing individuals, or within a sample of treatment-naïve individuals with AUDs and matched controls, respectively. Again, although these findings are not directly comparable with the

current findings, they do indicate inconsistencies in sex-related differences in the P300 among individuals exposed to alcohol.

In summary, it is difficult to contextualize the absence of sex differences on cognitive and EEG measures within the current study because (a) sex differences have been reported inconsistently in the literature, and (b) many studies examining the influence of sex on cognitive and EEG correlates of disinhibition in alcohol-exposed individuals used individuals dissimilar to those used here (e.g., individuals acutely exposed to alcohol; individuals prenatally exposed to alcohol), or focus exclusively on male participants. Hence, further work is needed to clarify the influence of sex on these domains of disinhibition.

Unsuccessful Modeling of the Latent Disinhibitory Complex

A large body of research supports the notion that disinhibition is a latent, generalized complex with various behavioral, cognitive, and electrophysiological dimensions (Begleiter & Porjesz, 1999; Finn et al., 2009; Gorenstein & Newman, 1980; Kendler et al., 2003; Krueger et al., 2002; Krueger & Markon, 2006; Tarter et al., 2004; Zernicke et al., 2010). The original objective of the factor analysis conducted here was thus to uncover a single latent dimension of disinhibition, using a hybrid of indices measuring disinhibited personality, externalizing psychopathology[4], and executive cognitive disinhibition. The secondary aim of this procedure was to use the derived factor scores to predict performance on EEG measures.

Despite evidence of disinhibition in personality and cognitive domains in the current data, attempts to model the construct of disinhibition using personality, psychiatric, and cognitive indices were unsuccessful. Possible explanations for the unsuccessful model include overall low correlations between the two cognitive measures, and similarly low correlations across the domains of measurement. These low correlations may have arisen due to restricted range or methodological issues. Another explanation for the unsuccessful model concerns the factor structure of disinhibition. The following sections discuss, in turn, each of those possible explanations for unsuccessful modeling of disinhibition.

[4]Here, and in all subsequent instances, the use of the term *externalizing psychopathology* with regard to the current study refers to the externalizing composite (i.e., sub-diagnostic threshold symptom counts for CD and ODD), and does not include symptom counts for AUDs.

Outcome measure correlations.

***Low correlations due to range restriction in psychiatric and cognitive measures*.** Unlike overall moderate and statistically significant correlations within the personality domain,[5] associations (a) between the two cognitive measures (SCWT response inhibition composite and ToL rule violations), and (b) across the personality, psychiatric, and cognitive domains, were generally small and non-significant. Notably, data for the psychiatric (externalizing composite) and one of the cognitive (ToL rule violations) measures were severely skewed (see Appendix E). Examination of the distributions of the externalizing composite and ToL rule violations scores indicate that a floor effect was present in these data. This floor effect arose because the range of scores for these measures was restricted, with most participants having no sub-diagnostic externalizing psychopathology (i.e., they had scores of zero on the externalizing composite), and most participants committing no rule violations on the ToL. In fact, the modal score for both these measures was zero.

[5]There were several moderately sized and statistically significant correlations within the personality domain measures. These correlations (specifically between scores on measures of impulsivity and novelty-seeking; scores on measures of impulsivity and excitement-seeking; and scores on measures of novelty-seeking and excitement-seeking) were expected because impulsivity, novelty-seeking, and excitement-seeking are closely related aspects of disinhibited personality (Finn et al., 2002; Justus et al., 2001; Masse & Tremblay, 1997). The measure for venturesomeness, on the other hand and as mentioned previously, may be a less valid measure for use with the current population; further, this subscale may have compromised construct validity as a measure of disinhibited personality. These issues concerning the validity of this subscale may thus have prevented significant correlations relating to this measure.

It is possible, however, that significant correlations between personality measures (observed both in the current study and elsewhere) are inflated as these measures are derived using the same method (i.e., self-report personality questionnaires). Common method variance (CMV; Campbell & Fiske, 1959) refers to the proportion of variance in measurement attributable to the method used. Because the method variance component of total variance is common across measures assessed using the same method, the relationships between these measures may be inflated. Consequently, associations between measures derived using the same method may be larger than those observed due to the underlying construct alone (Spector, 2006). However, the influence of CMV varies greatly according to constructs assessed and methods used, and CMV often has very little influence on correlations between measures (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). Unfortunately, it was not possible within the limits of this study's design to use the multimethod-multitrait procedure to parse the influence of CMV from true variance between traits (Campbell & Fiske, 1959). It is therefore difficult to comment conclusively on the relative contributions on CMV and true variance to the correlations observed here. However, the contrast between moderate correlations within the personality domain, and overall low correlations across domains, suggests that CMV did contribute to the correlations observed here.

Nevertheless, impulsivity, novelty-seeking, and excitement-seeking remain overlapping forms of disinhibited personality, and so there are strong conceptual grounds (independent of CMV) for the observed associations between these measures (Finn & Hall, 2004; Grekin et al., 2006; Justus et al., 2001). It appears, then, that CMV and true variance contributed independently to the correlations observed here, where the associations attributable to true variance were likely inflated due to CMV.

With regard to the externalizing composite, I attribute the floor effect (and consequent skewness of the distribution) to the study's eligibility criteria. Because only individuals with sub-diagnostic symptom counts were eligible for study participation, the possible number of endorsed externalizing symptoms was limited.

With regard to ToL rule violations, previous use of this test has shown that, even in adolescent and child populations, rule violations do not occur frequently (Culbertson & Zillmer, 1998). In fact, individuals over 9 years of age rarely commit more than three rule violations in total over 10 trials. Although the number of rule violations committed on the ToL remains a useful index of response inhibition, the range for this measure is inherently restricted in grossly neurologically intact[6] populations, such as the one studied here (Carey et al., 2008; Culbertson et al., 2004).

Range restriction for the externalizing composite and ToL rule violations leads to limited variance in these measures (Howell, 2004). Variance in scores is necessary in order to observe correlations. However, restricted range (i.e., limited variance) of scores for the externalizing composite and ToL rule violations attenuated associations between each of these measures and the remaining personality and cognitive measures. In summary, range restriction in these measures (i.e., in the externalizing composite and ToL rule violation measures) is thus probably responsible for the overall low correlations observed here.

Low correlations due to methodological issues.

Multi-method measurement. Although restriction of range in some measures in the current study might, indeed, have diminished observed correlations across the personality, psychiatric, and cognitive domains of measurement, some research suggests a lack of association across these domains even in the absence of such restrictions. For example, several studies report associations between some personality and cognitive measures of disinhibition, but not between other such measures (Dolan et al., 2008; Shuster & Toplak, 2009). In contrast, other studies fail to find *any* significant associations across personality and cognitive domains (Crean, de Wit, &

[6]Participants in the current Alcohol-group were both treatment-naïve and high-functioning (i.e., able to remain at school despite alcohol-use problems). Although some neurological deficits may be expected in individuals with SUDs, separate lines of research provide evidence that (a) treatment-naïve and (b) high-functioning drug users have relatively preserved cognitive functioning compared with treated or low functioning drug abusers, and further, that treatment-naïve, high-functioning drug users do not show gross cognitive deficits (Chao et al., 2003; Horner, Waid, Johnson, Latham, & Anton, 1999; S. Smith & Fein, 2010).

Richards, 2000; Dom, De Wilde, Hulstijn, & Sabbe, 2007; S. H. Mitchell, 1999; Reynolds, Richards, Horn, & Karraker, 2004; White et al., 1994).

Kamarajan et al. (2010) reported associations between EEG correlates of disinhibition (measured using the P300 and F-ERN) and measures of disinhibited personality, but failed to find similar associations between EEG correlates of disinhibition and measures of executive cognitive disinhibition. Other studies (Helmers et al. (1995) and Reynolds et al. (2006)) reported moderate to large correlations between several indices of disinhibited personality, but failed to find significant associations between personality and cognitive measures of disinhibition.

With regard to cognitive measures, the two latter studies used a relatively large group of cognitive indices (four in each case). Furthermore, the cognitive measures in those studies were less restricted than those in the current study as they were not limited to error scores (which, as noted above, are often marked by range restrictions). The distribution of data on each outcome measure is not mentioned by Helmers et al., while it appears that scores for most measures were normally distributed in the Reynolds et al. study as scores for only one cognitive measure (out of a total of seven outcome measures) were transformed to improve normality. Regarding that latter study, at least, it is reasonable to assume that normality was relatively preserved, and hence that the absence of significant associations between personality and cognitive measures of disinhibition was not a result of range restriction.

These findings suggest, then, that low correlations between self-report personality questionnaires and cognitive measures of disinhibition might arise due to methodological differences between these measures. Indeed, personality and cognitive measures differ in several fundamental ways (Reynolds et al., 2006). Personality measures are a reflection of individual differences in ways of perceiving the world and responding to it (Dick et al., 2010). In addition, self-report questionnaires rely on an individual's ability to recognize and report on their behavioral tendencies in a range of contexts, often from an allocentric perspective. These self-perceptions are an approximation of behavior, and may be subject to various biases. For example, individuals may respond to particular self-report measures in a manner that maintains consistency among attitudes, perceptions, and attributions (Staw, 1975). In summary, personality measures likely reflect both cognitive and affective processes relating to perceptions of the self and the world that may be contaminated by bias (Dick et al., 2010).

Cognitive measures, on the other hand, are generally considered relatively objective (i.e., relatively bias-free) means of assessing a construct (Reynolds et al., 2006). However, cognitive measures typically index relatively specific cognitive processes (such as response inhibition, as in the current study), and these circumscribed processes may not necessarily generalize to the broader behavioral contexts indexed by personality measures.

Hence, because personality measures index disinhibition in broad, subjective terms, whereas cognitive measures assess more circumscribed abilities in more objective terms, it makes sense that these measures might not relate strongly (Dick et al., 2010). Nevertheless, several studies have successfully modeled disinhibition using two- or three-way combinations of disinhibited personality, externalizing psychopathology, and executive cognitive disinhibition (Enticott et al., 2006; Kirby, Petry, & Bickel, 1999; Logan et al., 1997; Richards, Zhang, Mitchell, & de Wit, 1999; Swann, Bjork, Moeller, & Dougherty, 2002). These factor analyses thus suggest that, at least in certain cases, multi-method indices may successfully index disinhibition, despite methodological differences in the acquisition of these data.

Constitution of the SCWT response inhibition composite. Examination of Table 5 (the correlation matrix for personality, psychiatric, and cognitive measures) revealed an unexpected (non-significant) negative association between the SCWT response inhibition composite and (a) personality and (b) psychiatric measures. Negative associations between these measures imply that poor response inhibition is associated with less disinhibited personality traits and less externalizing psychopathology. These associations are in an opposite direction to those expected according to predictions centered around the notion of a disinhibitory complex (Iacono et al., 2003). The negative correlations thus compromised attempts to model the latent construct of disinhibition following this notion.

The finding of such negative associations is not without precedent in the literature, however. For instance, Shuster and Toplak (2009) examined the relationships between performance on laboratory tasks of cognitive disinhibition (including both the Stroop Color-Word test and a traditional gambling task) and self-reported measures of behavioral disinhibition (specifically, impulsivity and hyperactivity) in a sample of non-substance-using young adults. Despite the fact that this study did not include individuals with AUDs, Shuster and Toplak noted similar negative associations between SCWT interference scores and self-reported measures of behavioral disinhibition. They explored these unexpected findings by examining individual

associations between measures of behavioral disinhibition and (a) uncorrected errors on the SCWT and (b) self-corrected errors on the SCWT. These analyses revealed that uncorrected errors shared non-significant correlations with behavioral measures of disinhibition (direction of association not specified), whereas self-corrected errors were negatively correlated with behavioral disinhibition. According to the authors, these differences suggest that uncorrected errors signify impaired response inhibition, whereas self-corrected errors signify good cognitive performance (i.e., preserved metacognitive awareness of error commission).

As mentioned previously, the different mechanisms employed in the commission of uncorrected and self-corrected errors may imply that these scores should, ideally, not be combined into a single composite score. Nevertheless, these measures were combined in the current study following the premise that both types of errors signify an inability to resolve pre-response conflict.

To test whether the premise for combining uncorrected and self-corrected errors was flawed, and further, whether the current data support the associations reported by Shuster and Toplak, I performed a similar set of post-hoc correlation analyses using data from the current study. The findings of these analyses did not support the hypothesis proposed by Shuster and Toplak. Specifically, and with regard to uncorrected errors, negative (non-significant) associations existed for venturesomeness and novelty-seeking. With regard to self-corrected errors, negative (non-significant) associations existed for impulsivity, novelty-seeking, and the externalizing composite. Hence, it appears that negative associations remained largely intact when assessing individual correlations between the constituents of the SCWT response inhibition composite and other outcome measures. These findings suggest that uncorrected and self-corrected errors do indeed have overlapping mechanisms (i.e., inability to detect/resolve pre-response conflict), and therefore do not reflect opposing processes, as suggested elsewhere.

It remains unclear, therefore, why the SCWT response inhibition composite was negatively associated with both personality and psychiatric measures in the current study. One may speculate that the negative direction of associations was spurious, arising due to limited variance in the scores for the SCWT response inhibition composite (as mentioned previously). It is worth noting, however, that because these negative associations were both extremely small and statistically non-significant, there is relatively little statistical basis for concern.

Nevertheless, and in general terms, future work should attempt to better characterize how the various domains of disinhibition relate to one another.

Disinhibition as a multifactorial construct. Within the theoretical framework that proposes the notion of a generalized disinhibitory complex, disinhibition is generally believed to represent a spectrum of externalizing behaviors (Armstrong & Costello, 2002; Krueger & Markon, 2006). Emerging findings suggest, however, that disinhibition may in fact comprise several factors. For example, a factor analysis of executive cognitive disinhibition in children with behavioral problems suggested two factors: a cognitive inhibitory control factor and a motivational inhibitory control factor (Kindlon, Mezzacappa, & Earls, 1995). Using a more comprehensive set of indices (including disinhibited personality and externalizing psychopathology), Bogg and Finn (2010) uncovered a hierarchical four-factor structure of disinhibition (comprising three subfactors and one superfactor).

In the current study, the indicators of disinhibited personality loaded on a separate subfactor to the indicators of executive cognitive disinhibition. Taken together, these findings suggest that (a) disinhibition is not necessarily a unitary construct, and may instead have a hierarchical structure; (b) the number of emerging factors of disinhibition may vary according to the number of domains examined (i.e., two factors emerged using indicators from a single domain in the Kindlon et al. study; four factors emerged using indicators from two domains in the Bogg and Finn study); and (c) each domain of disinhibition may constitute a discrete factor (each with possible associated subfactors).

In the current study, seven indicators relating to three domains of disinhibition were used to model disinhibition. The reason for using such a broad range of indicators was to attempt to characterize the complexity of the disinhibition construct as comprehensively as possible. However, if each domain of disinhibition indeed constitutes a discrete factor, then disinhibition in the current study would be represented, in the simplest possible form, by a three-factor solution. Such a model was not possible in the current study because at least three indicators are required to represent each factor in a structural equation model based on a small sample size (N = 162) (Kline, 2005). The ratio of seven possible indicators to three domains of disinhibition clearly falls short of this requirement. It is thus possible that the current model of disinhibition was unsuccessful because the underlying factor structure was too complex in relation to the study

parameters. In other words, a successful, complex model of disinhibition may have emerged if more indicators were employed to represent each domain.

In summary, disinhibition is a complex, multifactorial construct, with a possible hierarchical factor structure. It remains unclear, still, which combination of measures are best suited to its measurement, and indeed how many factors underlie this construct. The findings of the current study, along with those presented in other previously published work (e.g., Bogg & Finn, 2010; Kindlon et al., 1995), suggest, however, that each domain of measurement may represent a discrete factor. Unfortunately, the current model of disinhibition was limited by the available number of indicators, and so this hypothesis could not be tested.

In addition, correlations between indicators were limited in the current study. If each domain of disinhibition indeed constitutes a discrete factor, a low correlation between the cognitive measures (due to range restriction) meant that these measures were ill-suited to represent a single executive cognitive disinhibition factor. With regard to measures in general, correlations between measures were constrained due to relatively lower scores in treatment-naïve adolescents with AUDs compared to treated adults used elsewhere (Di Sclafani et al., 2008; Fein & Andrew, 2011; Fein & Landman, 2005; Gazdzinski et al., 2008; Moss et al., 1994). Larger correlations both within the cognitive domain and between domains would have increased the likelihood of creating a successful model.

I acknowledge that alternative statistical methods may have been applicable to the investigations conducted here. Specifically, these data may have been analyzed using multivariate or multiple regression techniques. The analyses conducted here, however, were exploratory and hypothesis-driven. The primary hypothesis (which informed the selection of statistical methods) was the idea that the relevant measures tap into a latent construct of disinhibition. Had the current investigations been based on previous findings (i.e., not been exploratory in nature), alternative statistical methods may have been more appropriate.

I therefore attribute the unsuccessful attempt to model disinhibition using personality, psychiatric, and cognitive measures to (a) a limited selection of indicators of disinhibition, and (b) overall low correlations between outcome measures due, in large part, to characteristics of the measures (i.e., the restricted range problem) and of the sample (i.e., treatment-naïve adolescents rather than treated adults) . Further research using more comprehensive selections of indicators representing each domain of disinhibition (i.e., personality, psychopathology, cognition, EEG),

and each demonstrating reasonable variance, is thus needed to characterize this complex construct.

Summary and Conclusions

AUDs in adolescents are pervasive problems, with widespread economic and social ramifications (Flisher, Ziervogel, Chalton, Leger, & Robertson, 1996; T. R. Miller, Lesting, & Smith, 2001; Stoelb & Chiriboga, 1998; Sutherland & Shepherd, 2001; Zhang & Wieczorek, 1997). Disinhibition relating to AUDs is, in turn, is a complex issue with the capacity to affect people's lives in numerous ways. Research relating to AUDs in treatment-naïve adolescent populations is underdeveloped. Because most individuals with AUDs do not seek formal treatment, it is important that disinhibition relating to treatment-naïve populations (and adolescents, in particular) is better understood, so as to facilitate appropriate interventions.

The aims of the current study were to (a) examine and characterize the disinhibitory complex in treatment-naïve adolescents with AUDs, (b) investigate sex differences in disinhibition, and (c) determine whether an underlying construct of disinhibition might explain elevated levels of disinhibition in AUDs observed on individual indices. Analysis of the current data yielded several interesting results. Specifically, the findings presented here indicate that treatment-naïve adolescents with AUDs exhibit some evidence of disinhibition relative to healthy controls. Importantly, however, disinhibition manifested inconsistently in the current AUD sample. Although these findings for treatment-naïve adolescents with AUDs were not directly compared to those for treated adults with AUDs, the inconsistent profile of disinhibition found here suggests that the current sample of treatment-naïve adolescents demonstrated disinhibition to a lesser extent than treated adults studied elsewhere. Further, the arguments presented here illustrate that treatment-naïve status and age (relating specifically to (a) possible vulnerability of the structures supporting cognitive control, (b) immaturity of cognitive control functions, (c) relatively short drinking histories, and (d) possible increased variability in cognitive control functions in adolescents versus adults) are important considerations in the relationship between AUDs and disinhibition. Future research should acknowledge the potential influence of these factors on findings relating to disinhibition, and investigate the mechanisms of these factors further.

It is unclear why individuals with AUDs in the current study demonstrated disinhibition in particular domains (i.e., personality, cognition), but not others (i.e., psychiatric, EEG). It is possible that certain domains of inhibitory control are more susceptible to AUD-related disinhibitory effects than others. Counter to this argument, however, is the observation that other studies reporting inconsistent evidence of disinhibition across domains have found different patterns of inconsistencies to those found here. For instance, Kamarajan et al. (2010) found at least partial evidence of EEG correlates of disinhibition co-occuring with elevated disinhibited personality traits and an absence of executive cognitive disinhibition in individuals with AUDs. Evidence of AUD-related disinhibition in the current study, however, manifested in cognitive and personality domains, and spared psychiatric and EEG domains. Although the individuals with AUDs used in the Kamarajan et al. study differed from the current Alcohol-group participants (i.e., on the basis of age and treatment status), these findings suggest that the inconsistent evidence of disinhibition found in the current study did not arise due to differential susceptibility to AUD-related disinhibitory effects in some domains relative to others.

The current study contributes to the literature because the observed pattern of results is not consistent with distinctions along phenotypical/endophenotypical lines. In other words, among Alcohol-group participants, relative disinhibition was evident on both phenotypical and endophenotypical measures (i.e., some personality measures and both cognitive measures, respectively), while intact inhibitory control was demonstrated on other phenotypical and endophenotypical measures (i.e., some personality measures and all EEG measures). Future work should aim to define the reasons for increased susceptibility to disinhibition in particular domains.

Second, males showed inconsistent evidence of disinhibition relative to females. Specifically, males were relatively disinhibited on a single measure of disinhibited personality and showed elevated levels of externalizing psychopathology in relation to females. In contrast, however, there were no sex differences in cognitive or EEG domains. Nevertheless, an overall pattern of inconsistent sex-related differences in disinhibition, such as that observed here, has been noted previously in the literature (e.g., M. J. Crowley et al., 2009; Fein & Andrew, 2011; Hoffman & Polich, 1999; J. M. Mitchell et al., 2005; Nelson et al., 2011; S. Smith & Fein, 2010; Stout et al., 2005). Because many previous studies have focused exclusively on male

participants, future work should include female participants in assessments of disinhibition in order to better examine the influence of sex on disinhibition.

Lastly, unsuccessful attempts to model the construct of disinhibition in the current study support the possibility that disinhibition is a complex construct, comprising several domains, each of which may constitute a discrete factor. Because investigation of disinhibition using simultaneous examination of a broad range of domains is rarely attempted, future research should endeavor to use multi-method measurement to better characterize the complexity of disinhibition.

References

Acton, G. S. (2003). Measurement of impulsivity in a hierarchical model of personality traits: Implications for substance use. *Substance Use & Misuse, 38*(1), 67-83.

Aklin, W. M., Lejuez, C. W., Zvolensky, M. J., Kahler, C. W., & Gwadz, M. (2005). Evaluation of behavioral measures of risk taking propensity with inner city adolescents. *Behavior Research and Therapy, 43*(2), 215-228.

American Psychological Association. (2000). *DSM-IV-TR: Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Publishing.

Andrucci, G. L., Archer, R. P., Pancoast, D. L., & Gordon, R. A. (1989). The relationship of MMPI and Sensation Seeking Scales to adolescent drug use. *Journal of Personality Assessment, 53*(2), 253-266.

Arbel, Y., & Donchin, E. (2011). When a child errs: The ERN and the Pe complex in children. *Psychophysiology, 48*(1), 55-63.

Armstrong, T. D., & Costello, E. J. (2002). Community studies on adolescent substance use, abuse, or dependence and psychiatric comorbidity. *Journal of Consulting and Clinical Psychology, 70*(6), 1224–1239.

Arnett, J. J. (1996). Sensation seeking, aggressiveness, and adolescent reckless behavior. *Personality and Individual Differences, 20*(6), 693-702.

Aytaclar, S., Tarter, R. E., Kirisci, L., & Lu, S. (1999). Association between hyperactivity and executive cognitive functioning in childhood and substance use disorder in early adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*(2), 172–178.

Bagby, R. M., Parker, J. D. A., & Joffe, R. T. (1992). Confirmatory factor analysis of the Tridimensional Personality Questionnaire. *Personality and Individual Differences, 13*(11), 1245-1246.

Ball, S. A. (1996). Type Aand Type B alcoholism: Applicability across sub-populations and treatment settings. *Alcohol Health & Research World, 20*(1), 30-35.

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121*(1), 65-94.

Bauer, L. O., & Hesselbrock, V. M. (2003). Brain maturation and subtypes of conduct disorder: Interactive effects on P300 amplitude and topography in male adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(1), 106-115.

Beck, A., Schlagenhauf, F., Wustenberg, T., Hein, J., Kienast, T., Kahnt, T., et al. (2009). Ventral striatal activation during reward anticipation correlates with impulsivity in alcoholics. *Biological Psychiatry, 66*(8), 734-742.

Begleiter, H., & Porjesz, B. (1988). Potential biological markers in individuals at high risk for developing alcoholism. *Alcoholism: Clinical and Experimental Research, 12*(4), 488-493.

Begleiter, H., & Porjesz, B. (1999). What is inherited in the predisposition toward alcoholism? A proposed model. *Alcoholism: Clinical and Experimental Research, 23*(7), 1125-1135.

Begleiter, H., Porjesz, B., Bihari, B., & Kissin, B. (1984). Event-related brain potentials in boys at risk for alcoholism. *Science, 225*(4669), 1493-1496.

Bergen, D. C. (2008). Effects of poverty on cognitive function: A hidden neurologic epidemic. *Neurology, 71*(6), 447-451.

Biederman, J., Wilens, T., Mick, E., Faraone, S. V., Weber, W., Curtis, S., et al. (1997). Is ADHD a risk factor for psychoactive substance use disorders? Findings from a four-year prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(1), 21-29.

Bjork, J. M., Hommer, D. W., Grant, S. J., & Danube, C. (2004). Impulsivity in abstinent alcohol-dependent patients: Relation to control subjects and type 1–/type 2–like traits. *Alcohol, 34*(2–3), 133-150.

Bogg, T., & Finn, P. R. (2010). A self-regulatory model of behavioral disinhibition in late adolescence: Integrating personality traits, externalizing psychopathology, and cognitive capacity. *Journal of Personality, 78*(2), 441-470.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*(3), 624-652.

Bradley, R. H., & Corwyn, R. F. (2002). Socioeconomic status and child development. *Annual Review of Psychology, 53*, 371-399.

Brown, S. A., & Tapert, S. F. (2004). Adolescence and the trajectory of alcohol use: Basic to clinical studies. *Annals New York Academy of Science, 1021*, 234-244.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215-222.

Campbell, D. T., & Fiske, D. W. (1959). Convergent and discriminant validation by the multitrait-multimethod matrix. *Psychological Bulletin, 56*(2), 81-105.

Carey, C. L., Woods, S. P., Damon, J., Halabi, C., Dean, D., Delis, D. C., et al. (2008). Discriminant validity and neuroanatomical correlates of rule monitoring in frontotemporal dementia and Alzheimer's disease. *Neuropsychologia, 46*(4), 1081–1087.

Carlson, S. R., & Iacono, W. G. (2008). Deviant P300 amplitude development in males is associated with paternal externalizing psychopathology. *Journal of Abnormal Psychology, 117*(4), 910-923.

Carlson, S. R., Katsanis, J., Iacono, W. G., & Mertz, A. K. (1999). Substance dependence and externalizing psychopathology in adolescent boys with small, average, or large P300 event-related potential amplitude. *Psychophysiology, 36*(5), 583-590.

Carter, C. S., Botvinick, M. M., & Cohen, J. D. (1999). The contribution of the anterior cingulate cortex to executive processes in cognition. *Reviews in the Neurosciences, 10*(1), 49-57.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science, 280*(5364), 747-749.

Cavanagh, J. F., Cohen, M. X., & Allen, J. J. (2009). Prelude to and resolution of an error: EEG phase synchrony reveals cognitive control dynamics during action monitoring. *The Journal of Neuroscience, 29*(1), 98-105.

Chao, L. L., Meyerhoff, D. J., Cardenas, V. A., Rothlind, J. C., & Weiner, M. W. (2003). Abnormal CNV in chronic heavy drinkers. *Clinical Neurophysiology, 114*(11), 2081-2095.

Chen, A. C., Porjesz, B., Rangaswamy, M., Kamarajan, C., Tang, Y., Jones, K. A., et al. (2007). Reduced frontal lobe activity in subjects with high impulsivity and alcoholism. *Alcoholism: Clinical and Experimental Research, 31*(1), 156-165.

Clark, D. B., Cornelius, J. R., Kirisci, L., & Tarter, R. E. (2005). Childhood risk categories for adolescent substance involvement: A general liability typology. *Drug and Alcohol Dependence, 77*(1), 13-21.

Clark, D. B., Vanyukov, M., & Cornelius, J. (2002). Childhood antisocial behavior and adolescent alcohol use disorders. *Alcohol Research and Health, 26*(2), 109-115.

Clark, L., & Robbins, T. (2002). Decision-making deficits in drug addiction. *Trends in Cognitive Sciences, 6*(9), 361-363.

Cloninger, C. R. (1987a). Neurogenetic adaptive mechanisms in alcoholism. *Science, 236*(4800), 410-416.

Cloninger, C. R. (1987b). A systematic method for clinical description and classification of personality variants. A proposal. *Archives of General Psychiatry, 44*(6), 573-588.

Cloninger, C. R., Sigvardsson, S., & Bohman, M. (1988). Childhood personality predicts alcohol abuse in young adults. *Alcoholism: Clinical and Experimental Research, 12*(4), 494-505.

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry, 50*(12), 975-990.

Cohen, H. L., Wang, W., Porjesz, B., & Begleiter, H. (1995). Auditory P300 in young alcoholics: Regional response characteristics. *Alcoholism: Clinical and Experimental Research, 19*(2), 469-475.

Comerchero, M. D., & Polich, J. (1998). P3a, perceptual distinctiveness, and stimulus modality. *Cognitive Brain Research, 7*(1), 41-48.

Comerchero, M. D., & Polich, J. (1999). P3a and P3b from typical auditory and visual stimuli. *Clinical Neurophysiology, 110*(1), 24-30.

Compumedics Neuroscan Inc. (2008). Neuroscan SCAN (Version 4.4) [Computer software]. El Paso, TX: Compumedics Neuroscan, Inc.

Crean, J. P., de Wit, H., & Richards, J. B. (2000). Reward discounting as a measure of impulsive behavior in a psychiatric outpatient population. *Experimental and Clinical Psychopharmacology, 8*(2), 155–162.

Criado, J. R., Wills, D. N., Walker, B. M., & Ehlers, C. L. (2008). Electrophysiological effects of dizocilpine (MK-801) in adult rats exposed to ethanol during adolescence. *Alcoholism: Clinical and Experimental Research, 32*(10), 1752-1762.

Crowley, M. J., Wu, J., Crutcher, C., Bailey, C. A., Lejuez, C. W., & Mayes, L. C. (2009). Risk-taking and the feedback negativity response to loss among at-risk adolescents. *Developmental Neuroscience, 31*(1-2), 137-148.

Crowley, T. J., Raymond, K. M., Mikulich-Gilbertson, S. K., Thompson, L. L., & Lejuez, C. W. (2006). A risk-taking "set" in a novel task among adolescents with serious conduct and substance problems. *Journal of the American Academy of Child and Adolescent Psychiatry, 45*(2), 175-183.

Culbertson, W. C., Moberg, P. J., Duda, J. E., Stern, M. B., & Weintraub, D. (2004). Assessing the executive function deficits of patients with Parkinson's disease: Utility of the Tower of London-Drexel. *Assessment, 11*(1), 27-39.

Culbertson, W. C., & Zillmer, E. A. (1998). The Tower of LondonDX: A standardized approach to assessing executive functioning in children. *Archives of Clinical Neuropsychology, 13*(3), 285-301.

Culbertson, W. C., & Zillmer, E. A. (2001). *Tower of London: Technical manual*. New York, NY: Multi-Health Systems Inc.

Curtin, J. J., & Fairchild, B. A. (2003). Alcohol and cognitive control: Implications for regulation of behavior during response conflict. *Journal of Abnormal Psychology, 112*(3), 424-436.

Dahl, R. E. (2004). Adolescent brain development: A period of vulnerabilities and opportunities. *Annals of the New York Academy of Sciences, 1021*(1), 1-22.

Davies, P. L., Segalowitz, S. J., Dywan, J., & Pailing, P. E. (2001). Error-negativity and positivity as they relate to other ERP indices of attentional control and stimulus processing. *Biological Psychology, 56*(3), 191-206.

Davies, P. L., Segalowitz, S. J., & Gavin, W. J. (2004). Development of error-monitoring event-related potentials in adolescents. *Annals of the New York Academy of Sciences, 1021*(1), 324-328.

Dawes, M. A., Tarter, R. E., & Kirisci, L. (1997). Behavioral self-regulation: Correlates and 2 year follow-ups for boys at risk for substance abuse. *Drug and Alcohol Dependence, 45*(3), 165-176.

Dawson, D. A., Grant, B. F., Stinson, F. S., Chou, P. S., Huang, B., & Ruan, W. J. (2005). Recovery from DSM-IV alcohol dependence: United States, 2001-2002. *Addiction, 100*(3), 281-292.

de Bruijn, E. R. A., de Lange, F. P., von Cramon, D. Y., & Ullsperger, M. (2009). When errors are rewarding. *The Journal of Neuroscience, 29*(39), 12183-12186.

de Bruijn, E. R. A., Grootens, K. P., Verkes, R. J., Buchholz, V., Hummelen, J. W., & Hulstijn, W. (2006). Neural correlates of impulsive responding in borderline personality disorder: ERP evidence for reduced action monitoring. *Journal of Psychiatric Research, 40*(5), 428-437.

Desmedt, J. E. (1980). P300 in serial tasks: An essential post-decision closure mechanism. *Progress in Brain Research, 54*, 682-686.

Di Sclafani, V., Finn, P., & Fein, G. (2008). Treatment-naïve active alcoholics have greater psychiatric comorbidity than normal controls but less than treated abstinent alcoholics. *Drug and Alcohol Dependence, 98*(1-2), 115-122.

Dick, D. M., Smith, G., Olausson, P., Mitchell, S. H., Leeman, R. F., O'Malley, S. S., et al. (2010). Understanding the construct of impulsivity and its relationship to alcohol use disorders. *Addiction Biology, 15*(2), 217-226.

Dikman, Z. V., & Allen, J. J. (2000). Error monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology, 37*(1), 43-54.

Dolan, S. L., Bechara, A., & Nathan, P. E. (2008). Executive dysfunction as a risk marker for substance abuse: The role of impulsive personality traits. *Behavioral Sciences & the Law, 26*(6), 799-822.

Dom, G., De Wilde, B., Hulstijn, W., & Sabbe, B. (2007). Dimensions of impulsive behaviour in abstinent alcoholics. *Personality and Individual Differences, 42*(3), 465–476.

Dom, G., Hulstijn, W., & Sabbe, B. (2006). Differences in impulsivity and sensation seeking between early- and late-onset alcoholics. *Addictive Behaviors, 31*(2), 298-308.

Donchin, E., & Coles, M. G. H. (1998). Context updating and the P300. *Behavioral and Brain Sciences, 21*(01), 152-154.

Egner, T., Delano, M., & Hirsch, J. (2007). Separate conflict-specific cognitive control mechanisms in the human brain. *NeuroImage, 35*(2), 940-948.

Ehlers, C. L., & Criado, J. R. (2010). Adolescent ethanol exposure: Does it produce long-lasting electrophysiological effects? *Alcohol, 44*(1), 27-37.

Elkins, I. J., King, S. M., McGue, M., & Iacono, W. G. (2006). Personality traits and the development of nicotine, alcohol, and illicit drug disorders: Prospective links from adolescence to young adulthood. *Journal of Abnormal Psychology, 115*(1), 26-39.

Emeric, E. E., Brown, J. W., Leslie, M., Pouget, P., Stuphorn, V., & Schall, J. D. (2008). Performance monitoring local field potentials in the medial frontal cortex of primates: Anterior cingulate cortex. *Journal of Neurophysiology, 99*(2), 759-772.

Emmerson, R. Y., Diustman, R. E., Shearer, D. E., & Chamberlin, H. M. (1987). EEG, visually evoked and event related potentials in young abstinent alcoholics. *Alcohol, 4*(4), 241-248.

Enticott, P. G., Ogloff, J. R. P., & Bradshaw, J. L. (2006). Associations between laboratory measures of executive inhibitory control and self-reported impulsivity. *Personality and Individual Differences, 41*(2), 285–294.

Evenden, J. L. (1999). Varieties of impulsivity. *Psychopharmacology, 146*(4), 348-361.

Eysenck, S. B. G., & Eysenck, H. J. (1978). Impulsiveness and venturesomeness: Their position in a dimensional system of personality description. *Psychological Reports, 43*(3 Pt 2), 1247-1255.

Eysenck, S. B. G., & Eysenck, H. J. (1980). Impulsiveness and venturesomeness in children. *Personality and Individual Differences, 1*(1), 73-78.

Fein, G., & Andrew, C. (2011). Event-related potentials during visual target detection in treatment-naïve active alcoholics. *Alcoholism: Clinical and Experimental Research, 35*(6), 1171–1179.

Fein, G., & Chang, M. (2006). Visual P300s in long-term abstinent chronic alcoholics. *Alcoholism: Clinical and Experimental Research, 30*(12), 2000-2007.

Fein, G., & Chang, M. (2008). Smaller feedback ERN amplitudes during the BART are associated with a greater family history density of alcohol problems in treatment-naïve alcoholics. *Drug and Alcohol Dependence, 92*(1-3), 141-148.

Fein, G., Di Sclafani, V., Finn, P., & Scheiner, D. L. (2007). Sub-diagnostic psychiatric comorbidity in alcoholics. *Drug and Alcohol Dependence, 87*(2-3), 139-145.

Fein, G., & Landman, B. (2005). Treated and treatment-naïve alcoholics come from different populations. *Alcohol, 36*(2), 19-26.

Fein, G., McGillivray, S., & Finn, P. (2006). Normal performance on a simulated gambling task in treatment-naïve alcohol-dependent individuals. *Alcoholism: Clinical and Experimental Research, 30*(6), 959-966.

Ferguson, D. M., & Lynskey, M. T. (1995). Alcohol misuse and adolescent sexual behaviors and risk taking. *Pediatrics, 98*(1), 91-96.

Fergusson, D. M., Lynskey, M. T., & Horwood, L. J. (1993). Conduct problems and attention deficit behaviour in middle childhood and cannabis use by age 15. *The Australian and New Zealand Journal of Psychiatry, 27*(4), 673-682.

Fernandez-Serrano, M. J., Perez-Garcia, M., Perales, J. C., & Verdejo-Garcia, A. (2010). Prevalence of executive dysfunction in cocaine, heroin and alcohol users enrolled in therapeutic communities. *European Journal of Pharmacology, 626*(1), 104-112.

Ferrett, H. L., Carey, P. D., Thomas, K. G. F., Tapert, S. F., & Fein, G. (2010). Neuropsychological performance of South African treatment-naïve adolescents with alcohol dependence. *Drug and Alcohol Dependence, 110*(1-2), 8-14.

Ferrett, H. L., Cuzen, N. L., Thomas, K. G. F., Carey, P. D., Stein, D. J., Finn, P. R., et al. (2011). Characterization of South African adolescents with alcohol use disorders but without psychiatric or polysubstance comorbidity. *Alcoholism: Clinical and Experimental Research, 35*(9), 1705-1715.

Fillmore, M. T. (2004). Environmental dependence of behavioral control mechanisms: Effects of alcohol and information processing demands. *Experimental and Clinical Psychopharmacology, 12*(3), 216-223.

Fillmore, M. T., & Vogel-Sprott, M. (1995). Expectancies about alcohol-induced motor impairment predict individual differences in responses to alcohol and placebo. *Journal of Studies on Alcohol, 56*(1), 90-98.

Finn, P. R. (2002). Motivation, working memory, and decision making: A cognitive-motivational theory of personality vulnerability to alcoholism. *Behavioral and Cognitive Neuroscience Reviews, 1*(3), 183-205.

Finn, P. R., Bobova, L., Wehner, E., Fargo, S., & Rickert, M. E. (2005). Alcohol expectancies, conduct disorder and early-onset alcoholism: Negative alcohol expectancies are associated with less drinking in non-impulsive versus impulsive subjects. *Addiction, 100*(7), 953-962.

Finn, P. R., & Hall, J. (2004). Cognitive ability and risk for alcoholism: Short-term memory capacity and intelligence moderate personality risk for alcohol problems. *Journal of Abnormal Psychology, 113*(4), 569-581.

Finn, P. R., Justus, A., Mazas, C., & Steinmetz, J. E. (1999). Working memory, executive processes and the effects of alcohol on Go/No-Go learning: Testing a model of behavioral regulation and impulsivity. *Psychopharmacology, 146*(4), 465-472.

Finn, P. R., Mazas, C. A., Justus, A. N., & Steinmetz, J. (2002). Early-onset alcoholism with conduct disorder: Go/no go learning deficits, working memory capacity, and personality. *Alcoholism: Clinical and Experimental Research, 26*(2), 186-206.

Finn, P. R., Rickert, M. E., Miller, M. A., Lucas, J., Bogg, T., Bobova, L., et al. (2009). Reduced cognitive ability in alcohol dependence: Examining the role of covarying externalizing psychopathology. *Journal of Abnormal Psychology, 118*(1), 100-116.

Finn, P. R., Sharkansky, E. J., Brandt, K. M., & Turcotte, N. (2000). The effects of familial risk, personality, and expectancies on alcohol use and abuse. *Journal of Abnormal Psychology, 109*(1), 122-133.

Flisher, A. J., Parry, C. D., Evans, J., Muller, M., & Lombard, C. (2003). Substance use by adolescents in Cape Town: Prevalence and correlates. *Journal of Adolescent Health, 32*(1), 58-65.

Flisher, A. J., Ziervogel, C. F., Chalton, D. O., Leger, P. H., & Robertson, B. A. (1996). Risk-taking behaviour of Cape Peninsula high-school students. Part X. Multivariate relationships among behaviours. *South African Medical Journal, 86*(9), 1094-1098.

Forman, S. D., Dougherty, G. G., Casey, B. J., Siegle, G. J., Braver, T. S., Barch, D. M., et al. (2004). Opiate addicts lack error-dependent activation of rostral anterior cingulate. *Biological Psychiatry, 55*(5), 531-537.

Franken, I. H., van Strien, J. W., Franzek, E. J., & van de Wetering, B. J. (2007). Error-processing deficits in patients with cocaine dependence. *Biological Psychology, 75*(1), 45-51.

Gana, K., & Trouillet, R. (2003). Structure invariance of the Temperament and Character Inventory (TCI). *Personality and Individual Differences, 35*(7), 1485-1495.

Gardner, R., Holzman, P. S., Klein, G., S., Linton, H., & Spence, D. P. (1959). Cognitive control: A study of individual consistencies in cognitive behavior. *Psychological Issues, 1*(4), 1-186.

Gazdzinski, S., Durazzo, T. C., Weiner, M. W., & Meyerhoff, D. J. (2008). Are treated alcoholics representative of the entire population with alcohol use disorders? A magnetic resonance study of brain injury. *Alcohol, 42*(2), 67-76.

Geier, C. F., Terwilliger, R., Teslovich, T., Velanova, K., & Luna, B. (2010). Immaturities in reward processing and its influence on inhibitory control in adolescence. *Cerebral Cortex, 20*(7), 1613-1629.

Gerra, G., Angioni, L., Zaimovic, A., Moi, G., Bussandri, M., Bertacca, S., et al. (2004). Substance use among high-school students: Relationships with temperament, personality traits, and parental care perception. *Substance Use & Misuse, 39*(2), 345-367.

Giancola, P. R., Martin, C. S., Tarter, R. E., Pelham, W. E., & Moss, H. B. (1996). Executive cognitive functioning and aggressive behavior in preadolescent boys at high risk for substance abuse/dependence. *Journal of Studies on Alcohol, 57*(4), 352-359.

Giancola, P. R., Mezzich, A. C., & Tarter, R. E. (1998). Disruptive, delinquent and aggressive behavior in female adolescents with a psychoactive substance use disorder: Relation to executive cognitive functioning. *Journal of Studies on Alcohol and Drugs, 59*(5), 560-567.

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., et al. (2004, May). *Dynamic mapping of human cortical development during childhood through early adulthood.* Paper presented at the meeting of the National Academy of Sciences of the United States of America.

Golden, C. J., & Freshwater, S. M. (2002). *Stroop Color and Word test: A manual for clinical and experimental uses*. Los Angeles, CA: Stoelting Co.

Goldstein, R. Z., Leskovjan, A. C., Hoff, A. L., Hitzemann, R., Bashan, F., Khalsa, S. S., et al. (2004). Severity of neuropsychological impairment in cocaine and alcohol addiction: Association with metabolism in the prefrontal cortex. *Neuropsychologia, 42*(11), 1447-1458.

Goodwin, D. S. (1979). Alcoholism and heredity: A review and hypothesis. *Archives of General Psychiatry, 36*(1), 57-61.

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review, 87*(3), 301-315.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry, 160*(4), 636-645.

Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & van den Brink, W. (2005). Decision making in pathological gambling: A comparison between pathological gamblers, alcohol dependents, persons with Tourette syndrome, and normal controls. *Cognitive Brain Research, 23*(1), 137-151.

Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & van den Brink, W. (2008). The role of self-reported impulsivity and reward sensitivity versus neurocognitive measures of disinhibition and decision-making in the prediction of relapse in pathological gamblers. *Psychological Medicine, 38*(1), 41-50.

Grant, B. F., Stinson, F. S., Dawson, D. A., Chou, S. P., Dufour, M. C., Compton, W., et al. (2004). Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Archives of General Psychiatry, 61*(8), 807-816.

Grekin, E. R., Sher, K. J., & Wood, P. K. (2006). Personality and substance dependence symptoms: Modeling substance-specific traits. *Psychology of Addictive Behaviors, 20*(4), 415-424.

Grieve, K. W., & van Eeden, R. (2010). A preliminary investigation of the suitability of the WAIS-III for Afrikaans-speaking South Africans. *South African Journal of Psychology, 40*(3), 262-271.

Grove, W. M., Eckert, E. D., Heston, L., Bouchard, T. J., Jr., Segal, N., & Lykken, D. T. (1990). Heritability of substance abuse and antisocial behavior: A study of monozygotic twins reared apart. *Biological Psychiatry, 27*(12), 1293-1304.

Habeych, M. E., Folan, M. M., Luna, B., & Tarter, R. E. (2006). Impaired oculomotor response inhibition in children of alcoholics: The role of attention deficit hyperactivity disorder. *Drug and Alcohol Dependence, 82*(1), 11-17.

Hagen, G. F., Gatherwright, J. R., Lopez, B. A., & Polich, J. (2006). P3a from visual stimuli: Task difficulty effects. *International Journal of Psychophysiology, 59*(1), 8-14.

Hajcak, G., Moser, J. S., Holroyd, C. B., & Simons, R. F. (2006). The feedback-related negativity reflects the binary evaluation of good versus bad outcomes. *Biological Psychology, 71*(2), 148-154.

Hajcak, G., & Simons, R. F. (2002). Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Research, 110*(1), 63-72.

Hansenne, M. (2006). Event-related brain potentials in psychopathology: Clinical and cognitive perspectives. *Psychologica Belgica, 46*(1-2), 5-36.

Harden, P. W., & Pihl, R. O. (1995). Cognitive function, cardiovascular reactivity, and behavior in boys at high risk for alcoholism. *Journal of Abnormal Psychology, 104*(1), 94-103.

Hawkins, J. D., Catalano, R. F., & Miller, J. Y. (1992). Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention. *Psychological Bulletin, 112*(1), 64-105.

Hedden, S. L., Martins, S. S., Malcolm, R. J., Floyd, L., Cavanaugh, C. E., & Latimer, W. W. (2010). Patterns of illegal drug use among an adult alcohol dependent population: Results from the National Survey on Drug Use and Health. *Drug and Alcohol Dependence, 106*(2-3), 119-136.

Helmers, K. F., Young, S. N., & Pihl, R. O. (1995). Assessment of measures of impulsivity in healthy male volunteers. *Personality and Individual Differences, 19*(6), 927–935.

Hicks, B. M., Blonigen, D. M., Kramer, M. D., Krueger, R. F., Patrick, C. J., Iacono, W. G., et al. (2007). Gender differences and developmental change in externalizing disorders from late adolescence to early adulthood: A longitudinal twin study. *Journal of Abnormal Psychology, 116*(3), 433-447.

Hill, S. Y., Locke, J., & Steinhauer, S. R. (1999). Absence of visual and auditory P300 reduction in nondepressed male and female alcoholics. *Biological Psychiatry, 46*(7), 982-989.

Hill, S. Y., Shen, S., Locke, J., Steinhauer, S. R., Konicky, C., Lowers, L., et al. (1999). Developmental delay in P300 production in children at high risk for developing alcohol-related disorders. *Biological Psychiatry, 46*(7), 970-981.

Hill, S. Y., Steinhauer, S., Lowers, L., & Locke, J. (1995). Eight-year longitudinal follow-up of P300 and clinical outcome in children from high-risk for alcoholism families. *Biological Psychiatry, 37*(11), 823-827.

Hill, S. Y., & Steinhauer, S. R. (1993). Assessment of prepubertal and postpubertal boys and girls at risk for developing alcoholism with P300 from a visual discrimination task. *Journal of Studies on Alcohol, 54*(3), 350-358.

Hill, S. Y., Steinhauer, S. R., Locke-Wellman, J., & Ulrich, R. (2009). Childhood risk factors for young adult substance dependence outcome in offspring from multiplex alcohol dependence families: A prospective study. *Biological Psychiatry, 66*(8), 750-757.

Hill, S. Y., Yuan, H., & Locke, J. (1999). Path analysis of P300 amplitude of individuals from families at high and low risk for developing alcoholism. *Biological Psychiatry, 45*(3), 346-359.

Hoffman, L. D., & Polich, J. (1999). P300, handedness, and corpus callosal size: Gender, modality, and task. *International Journal of Psychophysiology, 31*(2), 163-174.

Holroyd, C. B., & Coles, M. G. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679-709.

Hopfer, C. J., Crowley, T. J., & Hewitt, J. K. (2003). Review of twin and adoption studies of adolescent substance use. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(6), 710-719.

Horner, M. D., Waid, L. R., Johnson, D. E., Latham, P. K., & Anton, R. F. (1999). The relationship of cognitive functioning to amount of recent and lifetime alcohol consumption in outpatient alcoholics. *Addictive Behaviors, 24*(3), 449-453.

Houk, J. C., Adams, J. L., & Barto, A. G. (1995). A model of how the basal ganglia generate and use neural signals that predict reinforcement. In J. Houk, J. Davis & D. Beiser (Eds.), *Models of information processing in the basal ganglia* (pp. 249-270). Cambridge, MA: MIT Press.

Howard, M. O., Kivlahan, D., & Walker, R. D. (1997). Cloninger's tridimensional theory of personality and psychopathology: Applications to substance use disorders. *Journal of Studies on Alcohol, 58*(1), 48–66.

Howell, D. C. (2004). *Fundemental statistics for the behavioral sciences* (5th ed.). Belmont, CA: Thomson Wadsworth.

Iacono, W. G., Carlson, S. R., Malone, S. M., & McGue, M. (2002). P3 event-related potential amplitude and the risk for disinhibitory disorders in adolescent boys. *Archives of General Psychiatry, 59*(8), 750-757.

Iacono, W. G., Carlson, S. R., Taylor, J., Elkins, I. J., & McGue, M. (1999). Behavioral disinhibition and the development of substance-use disorders: Findings from the Minnesota Twin Family Study. *Developmental Psychopathology, 11*(4), 869-900.

Iacono, W. G., Malone, S. M., & McGue, M. (2003). Substance use disorders, externalizing psychopathology, and P300 event-related potential amplitude. *International Journal of Psychophysiology, 48*(2), 147-178.

Iacono, W. G., Malone, S. M., & McGue, M. (2008). Behavioral disinhibition and the development of early-onset addiction: Common and specific influences. *Annual Review of Clinical Psychology, 4*, 325-348.

Jinabhai, C. C., Taylor, M., Rangongo, M. F., Mkhize, N. J., Anderson, S., Pillay, B. J., et al. (2004). Investigating the mental abilities of rural Zulu primary school children in South Africa. *Ethnicity and Health, 9*(1), 17-36.

Joska, J., Westgarth-Taylor, J., Myer, L., Hoare, J., Thomas, K., Combrinck, M., et al. (2011). Characterization of HIV-associated neurocognitive disorders among individuals starting antiretroviral therapy in South Africa. *AIDS and Behavior, 15*(6), 1197-1203.

Jurado, M. B., & Rosselli , M. (2007). The elusive nature of executive functions: A review of our current understanding. *Neuropsychology Review, 17*(3), 213–233.

Justus, A. N., Finn, P. R., & Steinmetz, J. E. (2001). P300, disinhibited personality, and early-onset alcohol problems. *Alcoholism: Clinical and Experimental Research, 25*(10), 1457-1466.

Kalsbeek, A., Voorn, P., Buijs, R. M., Pool, C. W., & Uylings, H. B. (1988). Development of the dopaminergic innervation in the prefrontal cortex of the rat. *The Journal of Comparative Neurology, 269*(1), 58-72.

Kamarajan, C., Rangaswamy, M., Tang, Y., Chorlian, D. B., Pandey, A. K., Roopesh, B. N., et al. (2010). Dysfunctional reward processing in male alcoholics: An ERP study during a gambling task. *Journal of Psychiatric Research, 44*(9), 576-590.

Kaplan, E. (1988). A process approach to neuropsychological assessment. In T. Boll & B. Bryant (Eds.), *Clinical neuropsychology and brain function: Research, measurement, and practice* (pp. 129–167). Washington, DC: American Psychological Association.

Kaplan, H. I., & Sadock, B. J. (1998). *Synopsis of psychiatry*. Baltimore, MD: Williams and Wilkins.

Kaplan, G. A., Turrell, G., Lynch, J. W., Everson, S. A., Helkala, E. L., & Salonen, J. T. (2001). Childhood socioeconomic position and cognitive function in adulthood. *International Journal of Epidemiology, 30*(2), 256-263.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., et al. (1997). The Schedule for Affective Disorders and Schizophrenia for School-Aged Children- Present and Lifetime Version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(7), 980-988.

Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry, 60*(9), 929-937.

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science, 303*(5660), 1023-1026.

Kessler, R. C. (2004). The epidemiology of dual diagnosis. *Biological Psychiatry, 56*(10), 730-737.

Kindlon, D., Mezzacappa, E., & Earls, F. (1995). Psychometric properties of impulsivity measures: Temporal stability, validity and factor structure. *Journal of Child Psychology and Psychiatry, 36*(4), 645-661.

Kirby, K. N., Petry, N. M., & Bickel, W. K. (1999). Heroin addicts have higher discount rates for delayed rewards than non-drug-using controls. *Journal of Experimental Psychology-General, 128*(1), 78–87.

Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York, NY: The Guilford Press.

Knight, R. T. (1997). Distributed cortical network for visual attention. *Journal of Cognitive Neurosciences, 9*(1), 75-91.

Knopik, V. S., Heath, A. C., Madden, P. A. A., Bucholz, K. K., Slutske, W. S., Nelson, E. C., et al. (2004). Genetic effects on alcohol dependence risk: Re-evaluating the importance of psychiatric and other heritable risk factors. *Psychological Medicine, 34*(8), 1519-1530.

Kok, A. (2001). On the utility of P3 amplitude as a measure of processing capacity. *Psychophysiology, 38*(3), 557–577.

Koob, G. F., & Le Moal, M. (1997). Drug abuse: Hedonic homeostatic dysregulation. *Science, 278*(5335), 52-58.

Koopmans, J. R., Boomsma, D. I., Heath, A. C., & van Doornen, L. J. (1995). A multivariate genetic analysis of sensation seeking. *Behavior Genetics, 25*(4), 349-356.

Kramer, J. H., Jurik, J., Sha, S. J., Rankin, K. P., Rosen, H. J., Johnson, J. K., et al. (2003). Distinctive neuropsychological patterns in frontotemporal dementia, semantic dementia, and Alzheimer disease. *Cognitive and Behavioral Neurology, 16*(4), 211-218.

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111*(3), 411-424.

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2*, 111-133.

Kuperman, S., Schlosser, S. S., Kramer, J. R., Bucholz, K., Hesselbrock, V., Reich, T., et al. (2001). Developmental sequence from disruptive behavior diagnosis to adolescent alcohol dependence. *The American Journal of Psychiatry, 158*(12), 2022-2026.

Laucht, M., Becker, K., Blomeyer, D., & Schmidt, M. H. (2007). Novelty seeking involved in mediating the association between the dopamine D4 receptor gene exon III polymorphism and heavy drinking in male adolescents: Results from a high-risk community sample. *Biological Psychiatry, 61*(1), 87-92.

Lawrence, A. J., Luty, J., Bogdan, N. A., Sahakian, B. J., & Clark, L. (2009). Impulsivity and response inhibition in alcohol dependence and problem gambling. *Psychopharmacology, 207*(1), 163-172.

Lee, S., Kawachi, I., Berkman, L. F., & Grodstein, F. (2003). Education, other socioeconomic indicators, and cognitive function. *American Journal of Epidemiology, 157*(8), 712-720.

Lejuez, C. W., Aklin, W., Bornovalova, M., & Moolchan, E. T. (2005). Differences in risk-taking propensity across inner-city adolescent ever- and never-smokers. *Nicotine and Tobacco Research, 7*(1), 71-79.

Lejuez, C. W., Read, J. P., Kahler, C. W., Richards, J. B., Ramsey, S. E., Stuart, G. L., et al. (2002). Evaluation of a behavioral measure of risk taking: The Balloon Analogue Risk Task (BART). *Journal of Experimental Psychology: Applied, 8*(2), 75-84.

Levin, H. S., Culhane, K. A., Hartmann, J., Evankovich, K., & Mattson, A. J. (1991). Developmental changes in performance on tests of purported frontal lobe functioning. *Developmental Neuropsychology, 7*(3), 377-395.

Lezak, M., Howieson, D., & Loring, D. (2004). *Neuropsychological assessment*. New York: Oxford University Press.

Loeber, R., Burke, J. D., Lahey, B. B., Winters, A., & Zera, M. (2000). Oppositional defiant and conduct disorder: A review of the past 10 years, Part I. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*(12), 1468-1484.

Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science, 8*(1), 60-64.

Luengo, M. A., Carrillo-de-la-Pena, M. T., Otero, J. M., & Romero, E. (1994). A short-term longitudinal study of impulsivity and antisocial behavior. *Journal of Personality and Social Psychology, 66*(3), 542-548.

Luna, B., Padmanabhan, A., & O'Hearn, K. (2010). What has fMRI told us about the development of cognitive control through adolescence? *Brain and Cognition, 72*(1), 101-113.

Luna, B., & Sweeney, J. A. (2004). The emergence of collaborative brain function: fMRI studies of the development of response inhibition. *Annals of the New York Academy of Sciences, 1021*, 296-309.

Lynskey, M. T., & Fergusson, D. M. (1995). Childhood conduct problems, attention deficit behaviors, and adolescent alcohol, tobacco, and illicit drug use. *Journal of Abnormal Child Psychology, 23*(3), 281-302.

MacAllister, W. S., Bender, H. A., Whitman, L., Welsh, A., Keller, S., Granader, Y., et al. (2011). Assessment of executive functioning in childhood epilepsy: The Tower of London and BRIEF. *Child Neuropsychology, 1*(1), 1-12.

MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science, 288*(5472), 1835-1838.

Manly, J. J., Jacobs, D. M., Sano, M., Bell, K., Merchant, C. A., Small, S. A., et al. (1999). Effect of literacy on neuropsychological test performance in nondemented, education-matched elders. *Journal of the International Neuropsychological Society, 5*(3), 191-202.

Marczinski, C. A., Abroms, B. D., Van Selst, M., & Fillmore, M. T. (2005). Alcohol-induced impairment of behavioral control: Differential effects on engaging vs. disengaging responses. *Psychopharmacology, 182*(3), 452-459.

Masse, L. C., & Tremblay, R. E. (1997). Behavior of boys in kindergarten and the onset of substance use during adolescence. *Archives of General Psychiatry, 54*(1), 62-68.

Maurage, P., Philippot, P., Verbanck, P., Noel, X., Kornreich, C., Hanak, C., et al. (2007). Is the P300 deficit in alcoholism associated with early visual impairments (P100, N170)? An oddball paradigm. *Clinical Neurophysiology, 118*(3), 633-644.

Mazas, C. A., Finn, P. R., & Steinmetz, J. E. (2000). Decision-making biases, antisocial personality, and early-onset alcoholism. *Alcoholism: Clinical and Experimental Research, 24*(7), 1036-1040.

McGue, M., Iacono, W. G., & Slutske, W. (1999). Personality and substance use disorders: II. Alcoholism versus drug use disorders. *Journal of Consulting and Clinical Psychology, 67*(3), 393–404.

McGue, M., Slutske, W., Taylor, J., & Iacono, W. G. (1997). Personality and substance use disorders: I. Effects of gender and alcoholism subtype. *Alcoholism: Clinical and Experimental Research, 21*(3), 513–520.

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24*(1), 167-202.

Miller, T. R., Lesting, D. C., & Smith, G. S. (2001). Injury risk among medically identified alcohol and drug abusers. *Alcoholism: Clinical and Experimental Research, 25*(1), 54–59.

Mitchell, J. M., Fields, H. L., D'Esposito, M., & Boettiger, C. A. (2005). Impulsive responding in alcoholics. *Alcoholism: Clinical and Experimental Research, 29*(12), 2158-2169.

Mitchell, S. H. (1999). Measures of impulsivity in cigarette smokers and non-smokers. *Psychopharmacology, 146*(4), 455-464.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry, 158*(11), 1783-1793.

Moss, H. B., Kirisci, L., Gordon, H., & Tarter, R. E. (1994). A neuropsychologic profile of adolescent alcoholics. *Alcoholism: Clinical and Experimental Research, 18*(1), 159-163.

Moss, H. B., Lynch, K. G., & Hardie, T. L. (2003). Affiliation with deviant peers among children of substance dependent fathers from pre-adolescence into adolescence: Associations with problem behaviors. *Drug and Alcohol Dependence, 71*(2), 117-125.

Müller, S. E., Weijers, H.-G., Böning, J., & Wiesbeck, G. A. (2008). Personality traits predict treatment outcome in alcohol-dependent patients. *Neuropsychobiology, 57*(4), 159-164.

Nagoshi, C. T., Walter, D., Muntaner, C., & Haertzen, C. A. (1992). Validation of the Tridimensional Personality Questionnaire in a sample of male drug users. *Personality and Individual Differences, 13*(4), 401-409.

Nelson, L. D., Patrick, C. J., Collins, P., Lang, A. R., & Bernat, E. M. (2011). Alcohol impairs brain reactivity to explicit loss feedback. *Psychopharmacology, 218*(2), 419–428.

Nieuwenhuis, S., Holroyd, C. B., Mol, N., & Coles, M. G. (2004). Reinforcement-related brain potentials from medial frontal cortex: Origins and functional significance. *Neuroscience and Biobehavioral Reviews, 28*(4), 441-448.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin, 127*(5), 571-598.

Nigg, J. T., Wong, M. M., Martel, M. M., Jester, J. M., Puttler, L. I., Glass, J. M., et al. (2006). Poor response inhibition as a predictor of problem drinking and illicit drug use in adolescents at risk for alcoholism and other substance use disorders. *Journal of the American Academy of Child and AdolescentPsychiatry, 45*(4), 468-475.

Onoda, K., Abe, S., & Yamaguchi, S. (2010). Feedback-related negativity is correlated with unplanned impulsivity. *Cognitive Neuroscience and Neuropsychology, 21*(10), 736-739.

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing: On the functional significance of the Pe vis-à-vis the ERN/Ne. *Journal of Psychophysiology, 19*(4), 319-329.

Paraskevaides, T., Morgan, C., Leitz, J., Bisby, J., Rendell, P., & Curran, H. (2010). Drinking and future thinking: Acute effects of alcohol on prospective memory and future simulation. *Psychopharmacology, 208*(2), 301-308.

Parry, C. D., Myers, B., Morojele, N. K., Flisher, A. J., Bhana, A., Donson, H., et al. (2004). Trends in adolescent alcohol and other drug use: Findings from three sentinel sites in South Africa (1997-2001). *Journal of Adolescence, 27*(4), 429-440.

Parsons, O. A., Sinha, R., & Williams, H. L. (1990). Relationships between neuropsychological test performance and event-related potentials in alcoholic and nonalcoholic samples. *Alcoholism: Clinical and Experimental Research, 14*(5), 746-755.

Peterson, J. B., Finn, P. R., & Pihl, R. O. (1992). Cognitive dysfunction and the inherited predisposition to alcoholism. *Journal of Studies on Alcohol, 53*(2), 154-160.

Pfefferbaum, A., Horvath, T. B., Roth, W. T., & Kopell, B. S. (1979). Event-related potential changes in chronic alcoholics. *Electroencephalography and Clinical Neurophysiology, 47*(6), 637-647.

Pickens, R. W., Svikis, D. S., McGue, M., & LaBuda, M. C. (1995). Common genetic mechanisms in alcohol, drug, and mental disorder comorbidity. *Drug and Alcohol Dependence, 39*(2), 129–138.

Podsakoff, P. M., MacKenzie, S. B., Lee, J. Y., & Podsakoff, N. P. (2003). Common method biases in behavioral research: A critical review of the literature and recommended remedies. *Journal of Applied Psychology, 88*(5), 879-903.

Polich, J. (1986). Normal variation of P300 from auditory stimuli. *Electroencephalography and Clinical Neurophysiology, 65*(3), 236-240.

Polich, J. (2007). Updating P300: An integrative theory of P3a and P3b. *Clinical Neurophysiology, 118*(10), 2128-2148.

Polich, J., & Bloom, F. E. (1986). P300 and alcohol consumption in normals and individuals at risk for alcoholism. A preliminary report. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 10*(2), 201-210.

Polich, J., Pollock, V. E., & Bloom, F. E. (1994). Meta-analysis of P300 amplitude from males at risk for alcoholism. *Psychological Bulletin, 115*(1), 55-73.

Porjesz, B., & Begleiter, H. (1996). Effects of alcohol on electrophysiological activity of the brain. In H. Begleiter & B. Kissin (Eds.), *The pharmacology of alcohol and alcohol dependence* (pp. 207-247). New York, NY: Oxford University.

Porjesz, B., Begleiter, H., Bihari, B., & Kissin, B. (1987). Event-related brain potentials to high incentive stimuli in abstinent alcoholics. *Alcohol, 4*(4), 283-287.

Porjesz, B., & Rangaswamy, M. (2007). Neurophysiological endophenotypes, CNS disinhibition, and risk for alcohol dependence and related disorders. *The Scientific World Journal, 7*(2), 131-141.

Porjesz, B., Rangaswamy, M., Kamarajan, C., Jones, K. A., Padmanabhapillai, A., & Begleiter, H. (2005). The utility of neurophysiological markers in the study of alcoholism. *Clinical Neurophysiology, 116*(5), 993-1018.

Posner, M. I., & DiGirolamo, G. J. (1998). Executive attention: Conflict, target detection, and cognitive control. In R. Parasuraman (Ed.), *The attentive brain* (pp. 401-423). Cambridge, MA: MIT Press.

Possin, K. L., Brambati, S. M., Rosen, H. J., Johnson, J. K., Pa, J., Weiner, M. W., et al. (2009). Rule violation errors are associated with right lateral prefrontal cortex atrophy in neurodegenerative disease. *Journal of the International Neuropsychological Society, 15*(3), 354-364.

Psychology Software Tools Inc. (2007). E-Prime (Version 2.0) [Computer Software]. Pittsburgh, PA: Psychology Software Tools, Inc.

Rand, G., Wapner, S., Werner, H., & McFarland, J. H. (1963). Age differences in performance on the Stroop Color-Word test. *Journal of Personality, 31*(4), 534-558.

Ratsma, J. E., van der Stelt, O., Schoffelmeer, A. N., Westerveld, & Boudewijn Gunning, W. (2001). P3 event-related potential, dopamine D2 receptor A1 allele, and sensation-seeking in adult children of alcoholics. *Alcoholism: Clinical and Experimental Research, 25*(7), 960-967.

Reebye, P., Moretti, M. M., & Lessard, J. C. (1995). Conduct disorder and substance use disorder: Comorbidity in a clinical sample of preadolescents and adolescents. *Canadian Journal of Psychiatry, 40*(6), 313-319.

Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences, 40*(2), 305-315.

Reynolds, B., Richards, J. B., Horn, K., & Karraker, K. (2004). Delay discounting and probability discounting as related to cigarette smoking status in adults. *Behavioural Processes, 65*(1), 35–42.

Richards, J. B., Zhang, L., Mitchell, S., & de Wit, H. (1999). Delay or probability discounting in a model of impulsive behavior: Effect of alcohol. *Journal of the Experimental Analysis of Behavior, 71*(2), 121-143.

Ridderinkhof, K. R., Ramautar, J. R., & Wijnen, J. G. (2009). To Pe or not to Pe: A P3-like ERP component reflecting the processing of response errors. *Psychophysiology, 46*(3), 531-538.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science, 306*(5695), 443-447.

Ridderinkhof, K. R., van den Wildenberg, W. P., Segalowitz, S. J., & Carter, C. S. (2004). Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition, 56*(2), 129-140.

Robinson, S., Goddard, L., Dritschel, B., Wisley, M., & Howlin, P. (2009). Executive functions in children with Autism Spectrum Disorders. *Brain and Cognition, 71*(3), 362-368.

Rodriguez Holguin, S., Porjesz, B., Chorlian, D. B., Polich, J., & Begleiter, H. (1999). Visual P3a in male alcoholics and controls. *Alcoholism: Clinical and Experimental Research, 23*(4), 582-591.

Rose, A. K., & Duka, T. (2007). The influence of alcohol on basic motoric and cognitive disinhibition. *Alcohol and Alcoholism, 42*(6), 544-551.

Rosenberg, D. R., & Lewis, D. A. (1995). Postnatal maturation of the dopaminergic innervation of monkey prefrontal and motor cortices: A tyrosine hydroxylase immunohistochemical analysis. *The Journal of Comparative Neurology, 358*(3), 383-400.

Ruchsow, M., Walter, H., Buchheim, A., Martius, P., Spitzer, M., Kachele, H., et al. (2006). Electrophysiological correlates of error processing in borderline personality disorder. *Biological Psychology, 72*(2), 133-140.

Sarkis, S. M., Sarkis, E. H., Marshall, D., & Archer, J. (2005). Self-regulation and inhibition in comorbid ADHD children: An evaluation of executive functions. *Journal of Attention Disorders, 8*(3), 96-108.

Savitz, J. B., & Jansen, P. (2003). The Stroop Color-Word interference test as an indicator of ADHD in poor readers. *Journal of Genetic Psychology, 164*(3), 319-333.

Schuckit, M. A. (2009). Alcohol-use disorders. *The Lancet, 373*(9662), 492-501.

Semlitsch, H. V., Anderer, P., Schuster, P., & Presslich, O. (1986). A solution for reliable and valid reduction of ocular artifacts, applied to the P300 ERP. *Psychophysiology, 23*(6), 695-703.

Sher, K. J., Bartholow, B. D., & Wood, M. D. (2000). Personality and substance use disorders: A prospective study. *Journal of Consulting and Clinical Psychology, 68*(5), 818-829.

Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology, 103*(1), 92-102.

Sher, K. J., Wood, M. D., Crews, T. M., & Vandiver, P. A. (1995). The Tridimensional Personality Questionnaire: Reliability and validity studies and derivation of a short form. *Psychological Assessment, 7*(2), 195-208.

Shuster, J., & Toplak, M. E. (2009). Executive and motivational inhibition: Associations with self-report measures related to inhibition. *Consciousness and Cognition, 18*(2), 471-480.

Shuttleworth-Edwards, A. B., Kemp, R. D., Rust, A. L., Muirhead, J. G., Hartman, N. P., & Radloff, S. E. (2004). Cross-cultural effects on IQ test performance: a review and preliminary normative indications on WAIS-III test performance. *Journal of Clinical and Experimental Neuropsychology, 26*(7), 903-920.

Silveri, M. M., Rogowska, J., McCaffrey, A., & Yurgelun-Todd, D. A. (2011). Adolescents at risk for alcohol abuse demonstrate altered frontal lobe activation during Stroop performance. *Alcoholism: Clinical and Experimental Research, 35*(2), 218-228.

Skuy, M., Schutte, E., Fridjhon, P., & O'Carrol, S. (2001). Suitability of published neuropsychological test norms for urban African secondary school students in South Africa. *Personality and Individual Differences, 30*(8), 1413-1425.

Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A., Bucholz, K. K., Dunne, M. P., et al. (1998). Common genetic risk factors for conduct disorder and alcohol dependence. *Journal of Abnormal Psychology, 107*(3), 363-374.

Slutske, W. S., Heath, A. C., Madden, P. A., Bucholz, K. K., Statham, D. J., & Martin, N. G. (2002). Personality and the genetic risk for alcohol dependence. *Journal of Abnormal Psychology, 111*(1), 124-133.

Smith, G. T., Fischer, S., Cyders, M. A., Annus, A. M., Spillane, N. S., & McCarthy, D. M. (2007). On the validity and utility of discriminating among impulsivity-like traits. *Assessment, 14*(2), 155-170.

Smith, M. E., Halgren, E., Sokolik, M., Baudena, P., Musolino, A., Liegeois-Chauvel, C., et al. (1990). The intracranial topography of the P3 event-related potential elicited during auditory oddball. *Electroencephalography and Clinical Neurophysiology, 76*(3), 235-248.

Smith, S., & Fein, G. (2010). Cognitive performance in treatment-naive active alcoholics. *Alcoholism: Clinical and Experimental Research, 34*(12), 2097-2105.

Sobell, L. C., & Sobell, M. B. (1992). Timeline follow-back: A technique for assessing self reported alcohol consumption. In A. Litten (Ed.), *Measuring alcohol consumption: Psychosocial and biochemical methods* (pp. 41–72). Totowa, N.J: Humana Press.

Soloff, P. H., Lynch, K. G., & Moss, H. B. (2000). Serotonin, impulsivity, and alcohol use disorders in the older adolescent: A psychobiological study. *Alcoholism: Clinical and Experimental Research, 24*(11), 1609-1619.

Sowell, E. R., Thompson, P. M., & Toga, A. W. (2004). Mapping changes in the human cortex throughout the span of life. *Neuroscientist, 10*(4), 372-392.

Spector, P. E. (2006). Method variance in organizational research: Truth or urban legend? *Organizational Research Methods, 9*(2), 221-232.

SPSS Inc. (2008). SPSS Statistics (Version 17.0) [Computer software]. Chicago, IL: SPSS Inc.

Staw, B. M. (1975). Attribution of the "causes" of performance: A new alternative interpretation of cross-sectional research on organizations. *Organizational Behavior and Human Performance, 13*, 414-432.

Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., & Woolard, J. (2008). Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: Evidence for a dual systems model. *Developmental Psychology, 44*(6), 1764-1778.

Steinhauer, S. R., & Hill, S. Y. (1993). Auditory event-related potentials in children at high risk for alcoholism. *Journal of Studies on Alcohol, 54*(4), 408-421.

Stevens, J. P. (1992). *Applied multivariate statistics for the social sciences* (2nd ed.). Erlbaum, NJ: Hillside.

Stoelb, M., & Chiriboga, J. (1998). A process model for assessing adolescent risk for suicide. *Journal of Adolescence, 21*(4), 359-370.

Stout, J. C., Rock, S. L., Campbell, M. C., Busemeyer, J. R., & Finn, P. R. (2005). Psychological processes underlying risky decisions in drug abusers. *Psychology of Addictive Behaviors, 19*(2), 148-157.

Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). Oxford/New York: Oxford University Press.

Strauss, G. P., Allen, D. N., Jorgensen, M. L., & Cramer, S. L. (2005). Test–retest reliability of standard and emotional Stroop tasks. *Assessment, 12*, 330–337.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*(6), 643-662.

Stuss, D. T., Floden, D., Alexander, M. P., Levine, B., & Katz, D. (2001). Stroop performance in focal lesion patients: Dissociation of processes and frontal lobe lesion location. *Neuropsychologia, 39*(8), 771-786.

Sutherland, I., & Shepherd, J. P. (2001). Social dimensions of adolescent substance use. *Addiction, 96*(3), 445-458.

Swann, A. C., Bjork, J. M., Moeller, F. G., & Dougherty, D. M. (2002). Two models of impulsivity: Relationship to personality traits and psychopathology. *Biological Psychiatry, 51*(12), 988–994.

Tapert, S. F., & Brown, S. A. (1999). Neuropsychological correlates of adolescent substance abuse: Four-year outcomes. *Journal of the International Neuropsychological Society, 5*(6), 481-493.

Tapert, S. F., Granholm, E., Leedy, N. G., & Brown, S. A. (2002). Substance use and withdrawal: Neuropsychological functioning over 8 years in youth. *Journal of the International Neuropsychological Society, 8*(7), 873-883.

Tarter, R. E., Kirisci, L., Habeych, M., Reynolds, M., & Vanyukov, M. (2004). Neurobehavior disinhibition in childhood predisposes boys to substance use disorder by young adulthood: Direct and mediated etiologic pathways. *Drug and Alcohol Dependence, 73*(2), 121-132.

Tarter, R. E., Kirisci, L., Mezzich, A., Cornelius, J. R., Pajer, K., Vanyukov, M., et al. (2003). Neurobehavioral disinhibition in childhood predicts early age at onset of substance use disorder. *American Journal of Psychiatry, 160*(6), 1078-1085.

Taylor, S. F., Stern, E. R., & Gehring, W. J. (2007). Neural systems for error monitoring: Recent findings and theoretical perspectives. *The Neuroscientist, 13*(2), 160-172.

Tomberg, C., & Desmedt, J. E. (1998). Human perceptual processing: Inhibition of transient prefrontal-parietal 40 Hz binding at P300 onset documented in non-averaged cognitive brain potentials. *Neuroscience Letters, 255*(3), 163-166.

van der Berg, S., Wood, L., & le Roux, N. (2002). Differentiation in black education. *Development South Africa, 19*(2), 289-306.

van Meel, C. S., Heslenfeld, D. J., Oosterlaan, J., & Sergeant, J. A. (2007). Adaptive control deficits in attention-deficit/hyperactivity disorder (ADHD): The role of error processing. *Psychiatry Research, 151*(3), 211-220.

van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14*(4), 593-602.

van Veen, V., & Carter, C. S. (2006). Conflict and cognitive control in the brain. *Current Directions in Psychological Science, 15*(5), 237-240.

Velanova, K., Wheeler, M. E., & Luna, B. (2008). Maturational changes in anterior cingulate and frontoparietal recruitment support the development of error processing and inhibitory control. *Cerebral Cortex, 18*(11), 2505-2522.

Verdejo-García, A., del Mar Sánchez-Fernández, M., Alonso-Maroto, L., Fernández-Calderón, F., Perales, J., Lozano, Ó., et al. (2010). Impulsivity and executive functions in polysubstance-using rave attenders. *Psychopharmacology, 210*(3), 377-392.

Verdejo-García, A., Lawrence, A. J., & Clark, L. (2008). Impulsivity as a vulnerability marker for substance-use disorders: Review of findings from high-risk research, problem gamblers and genetic association studies. *Neuroscience & Biobehavioral Reviews, 32*(4), 777-810.

Verleger, R. (1988). Event-related potentials and cognition: A critique of the context updating hypothesis and an alternative interpretation of P3. *Behavioral and Brain Sciences, 11*(3), 343-427.

Verleger, R. (1997). On the utility of P3 latency as an index of mental chronometry. *Psychophysiology, 34*(2), 131-156.

von Knorring, L., von Knorring, A. L., Smigan, L., Lindberg, U., & Edholm, M. (1987). Personality traits in subtypes of alcoholics. *Journal of Studies on Alcohol, 48*(6), 523-527.

Wendelken, C., Ditterich, J., Bunge, S. A., & Carter, C. S. (2009). Stimulus and response conflict processing during perceptual decision making. *Cognitive, Affective and Behavioral Neuroscience, 9*(4), 434-447.

Western Cape Education Department. (2010). Western Cape Education Department Homepage. Retrieved September 26, 2010, from http://www.wced.wcape.gov.sa

Whipple, S. C., Berman, S. M., & Noble, E. P. (1991). Event-related potentials in alcoholic fathers and their sons. *Alcohol, 8*(4), 321-327.

White, J. L., Moffitt, T. E., Caspi, A., Bartusch, D. J., Needles, D. J., & Stouthamer-Loeber, M. (1994). Measuring impulsivity and examining its relationship to delinquency. *Journal of Abnormal Psychology, 103*(2), 192-205.

Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*(4), 669–689.

Whiteside, S. P., & Lynam, D. R. (2003). Understanding the role of impulsivity and externalizing psychopathology in alcohol abuse: Application of the UPPS impulsive behavior scale. *Experimental and Clinical Psychopharmacology, 11*(3), 210–217.

Wilens, T. E., & Biederman, J. (1993). Psychopathology in preadolescent children at high risk for substance abuse: A review of the literature. *Harvard Review of Psychiatry, 1*(4), 207-218.

World Medical Association. (2008). Declaration of Helsinki. Ethical principles for medical research involving human subjects, *59th WMA General Assembly*. Seoul, South Korea.

Yamaguchi, S., Onoda, K., & Abe, S. (2011). *Association of impulsivity with feedback-related negativity during gambling task*. Paper presented at the 2011 IEEE/ICME International Conference on Complex Medical Engineering, Harbin, China.

Yechiam, E., Stout, J. C., Busemeyer, J. R., Rock, S. L., & Finn, P. R. (2005). Individual differences in the response to forgone payoffs: An examination of high functioning drug abusers. *Journal of Behavioral Decision Making, 18*(2), 97–110.

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*(4), 931-959.

Yeung, N., & Cohen, J. D. (2006). The impact of cognitive deficits on conflict monitoring. *Psychological Science, 17*(2), 164-171.

Yücel, M., & Lubman, D. I. (2007). Neurocognitive and neuroimaging evidence of behavioural dysregulation in human drug addiction: Implications for diagnosis, treatment and prevention. *Drug and Alcohol Review, 26*(1), 33-39.

Yücel, M., Lubman, D. I., & Pantelis, C. (2004). Addiction, disinhibition, impulsivity, compulsivity: What's the difference, why does it matter and what is the role of context? *Addiction, 99*(12), 1506-1507.

Zernicke, K. A., Cantrell, H., Finn, P. R., & Lucas, J. (2010). The association between earlier age of first drink, disinhibited personality, and externalizing psychopathology in young adults. *Addictive Behaviors, 35*(5), 414-418.

Zhang, L., & Wieczorek, W. (1997). The impact of age of onset of substance use on delinquency. *Journal of Research in Crime and Delinquency, 34*(2), 253–270.

Zuckerman, M. (1979). Sensation seeking and risk taking. In C. E. Izard (Ed.), *Emotions in personality and psychopathology* (pp. 163–197). New York, NY: Plenum Press.

Zuckerman, M. (1994). *Behavioral expressions and biosocial bases of sensation seeking.* Cambridge: Cambridge University Press.

Zuckerman, M., Eysenck, S., & Eysenck, H. J. (1978). Sensation seeking in England and America: Cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology, 46*(1), 139-149.

Appendix A
Assent and Consent Form (English version)

PATIENT INFORMATION AND CONSENT LEAFLET

Effects of Heavy Alcohol Abuse on Adolescent Brain Structure and Function

Investigators: P Carey, H Ferrett, C Naude, JP Fouche, B Alexander, M Morris

Dear Volunteer:

DESCRIPTION AND PURPOSE OF THE STUDY

You/your child are/is being invited to take part in a study carried out by the Anxiety and Stress Disorders Research Unit in the Department of Psychiatry at the University of Stellenbosch. Please take some time to read the information presented here, which will explain the details of this project. Please ask the study staff or doctor any questions about any part of this project that you do not fully understand. It is very important that you are fully satisfied that you clearly understand what this research entails and how your child could be involved. Also, your child's participation is **entirely voluntary** and you are free to decline to participate. If you say no, this will not affect you or your child negatively in any way whatsoever. You are also free to withdraw him/her from the study at any point, even if you do initially agree to let him/her take part. The study has been approved by the Committee for Human Research of the University of Stellenbosch. It will be conducted according to Medical Research Council guidelines on good clinical practice (2003) as well as the Declaration of Helsinki Guidelines (Edinburgh, 2000), which provide detailed guidelines that relate to the ethical conduct of studies involving human subjects.

Why are we doing this research?

Previous studies have shown that when alcohol is used heavily, it can have damaging effects on the brain. We are however unsure as to how serious these effects may be in young people. This study will try and answer some of these questions by studying the effects of heavy alcohol use in young people (adolescents) during this time of important brain growth and development. It may be that you have been requested to participate as a suitable candidate, or as someone who matches other young people for age and education, but who does not use or uses only a very limited amount of alcohol – i.e. a normal control.

We will be asking young people between the ages of 12 and 18 years who are heavy users of alcohol and a similar control group who do not use/use only limited amounts of alcohol, to participate. We plan to enroll a total of 160 people in this study which will be conducted at Tygerberg Hospital and in the MRC Unit on Anxiety Disorders of the University of Stellenbosch.

If you decide to take part in this study and you are using/abusing alcohol, you will be asked not to drink alcohol before each of the study sessions.

STUDY PROCEDURES

Your involvement in the study will require you to visit the study doctor/team on four occasions. At a screening visit with the study doctor or psychologist we will interview you, much like a normal visit to your family doctor to assess whether you are eligible for our study. This visit will include questions on your emotional and physical health as well as your school and home environment. If, following this initial examination you appear to be suitable for the study, we will invite you to go through this information and consent form to ensure that you understand all of what the study will involve. Once we have addressed any questions you may have and you and your parent/guardian provide written consent (permission) to your participation, we will proceed with the study. We will then ask you to fill in a number of questionnaires that will ask you about your alcohol use, how you perform at school, and use your free time as well as some other general questions. Finally, we will conduct physical examination. As part of this examination, we will require a urine sample to exclude recent use of any other prohibited drugs/medications and will take a small blood sample of about four table spoons of blood to check for any problems in your kidney and liver function, potential infections with hepatitis B and C as well as looking at lead levels and other markers of your nutritional status. If you are a female, a pregnancy test will be done at this stage whether there is a risk of you being pregnant or not, ie whether you are sexually active or not. This is required for safety purposes. Once all these procedures have been completed, we will schedule the second visit within days of the screening visit and also a session with your parent/guardian. We expect this visit to take about 1½ hours.

At all of the subsequent visits we will perform a breathalyzer test on you at the beginning of each session.

At the second visit you will undergo a series of tests called a neuropsychological evaluation. This study visit with the psychologist will take the form of a number of pencil and paper tests which will involved some writing and drawing as we test your memory, concentration and planning abilities (deleted mental flexibility). Many of these are like a normal IQ test that you may have done at school before. All of these tests are important and will help us determine if alcohol has any effects on these aspects of your brain's functioning. This will take about 2-2 ½ hours.

The third study visit will involve you having a test of your brain waves/activity also known as an EEG or Electro-encephalogram. For this test you will sit upright in a chair. A study assistant will then carefully place a number of sticky discs (electrodes) on your scalp (head). Each of these electrodes will then be connected by wires to a box that will measure the electrical activity of your brain. During this process you will be asked to respond to a number of questions that will appear on a television screen in front of you. It will be important to sit still for this examination and should take about 30 minutes. You will be able to stop and rest at any time if you feel you need to. As we are simply measuring the activity in your brain, there is no risk associated with doing and EEG. In total this visit should take about 1 hour.

During the fourth and final session you will have a type of brain scan, called an MRI (magnetic resonance imaging) scan. The scan will require you to lie on your back on a table that will move into the scanning machine for the 30 minutes it will take for the scan. During this time you will

be able to close your eyes and rest. You will also be able to talk to the study doctor/assistant at all times during the scan if you should experience any discomfort. The scan is a safe procedure if you have been screened correctly for the presence of any magnetic material on or inside you such as pace-makers, surgical clips and metal objects in the eyes. A formal screen for this will be done at the screening visit by a member of the study team. When the magnet in the machine is switched on, it will make some loud banging noises, but you will be clearly warned when this will take place. At this time you will feel nothing and the noise is not harmful to you in any way. To minimise the possible discomfort associated with this, we will give you some soft earplugs to put in and will also put earphones on so that you can listen to music if you so choose.

DISCOMFORT ASSOCIATED WITH THE STUDY

There are only low or minimal risks associated with your participation in this study. If you feel tired at any point in any of the visits, you should please ask your study doctor/psychologist for a rest. If for some reason you are unable to complete a visit on a particular day we may reschedule to complete the assessments at another time.

POTENTIAL BENEFITS

There may be no direct benefits to you for participating in this study. However, you will be making an important contribution to this research that may benefit others in the future. We expect that the results of this study will help us understand the effects of heavy alcohol use on brain development in young people.

COMPENSATION FOR STUDY PARTICIPATION

While you will not be paid to take part in this study, all evaluations will be provided at no cost to you or your medical aid. We will however pay you for any cost incurred in attending the prescribed study visits.

CONFIDENTIALITY

Your participation is regarded as strictly confidential. The results of the study will be published in the professional literature and made available to of the Committee for Human Research of Subcommittee C at the University of Stellenbosch, but your identity will not be revealed at any time to people outside of the study team.

THE RIGHT TO ASK QUESTIONS/WITHDRAW FROM THE STUDY

You have the right to ask questions at any time about any aspect of the study. If you have any queries, you can contact Barenise Alexander at 073 548 3928. These will also be the 24 hour contact details should you need to contact us in the event off an emergency.

Your participation in the study is entirely voluntary. You have the right to withdraw at any time. If you decide to withdraw from the study, it will not jeopardize you or any future treatment you may require in any way.

You are entitled to a signed copy of this document.

If you agree to take part, please complete the following section:

Assent of minor

I (*Name of Child/Minor*) **.....** have been invited to take part in the above research project entitled**: Effects of Heavy Alcohol Abuse on Adolescent Brain Structure and Function.**

The study doctor/nurse and my parents have explained the details of the study to me and I understand what they have said to me.

- They have also explained that this study will involve 4 assessments which include interviews, filling questionnaires, a physical examination including a blood test, an EEG and a brain scan.
- I also know that I am free to withdraw from the study at any time if I am unhappy.
- By writing my name below, I voluntarily agree to take part in this research project. I confirm that I have not been forced either by my parents or doctor to take part.

.....

Name of child **(To be written by the child if possible)**

Declaration by parent/legal guardian

By signing below, I *(name of parent/legal guardian)* **.....** agree to allow my child (*name of child*) **.....** who is **.....** years old, to take part in a research study entitled: **Effects of Heavy Alcohol Abuse on Adolescent Brain Structure and Function**

I declare that:

- I have read or had read to me this information and consent form and that it is written in a language with which I am fluent and comfortable.
- If my child is older then 7 years, he/she must agree to take part in the study and his/her ASSENT must be recorded on this form.
- I have had a chance to ask questions and all my questions have been adequately answered.
- I understand that taking part in this study is **voluntary** and I have not been pressurised to let my child take part.
- I may choose to withdraw my child from the study at any time and my child will not be penalised or prejudiced in any way.
- My child may be asked to leave the study before it has finished if the study doctor or researcher feels it is in my child's best interests, or if my child do not follow the study plan as agreed to.

Signed at (*place*) **.....** on (*date*) **.....**20**.....**

.....

Signature of parent/legal guardian

Declaration by investigator
I *(name)* **.....** declare that:

- I explained the information in this document to (*name of child)* **.....**
  I encouraged him/her to ask questions and took adequate time to answer them.
- I am satisfied that he/she adequately understand all aspects of the research, as discussed above.
- I did/did not use an interpreter (*if an interpreter is used, then the interpreter must sign the declaration below).*

Signed at (*place*) **.....** on (*date*) **.....**20**.....**

.....
Signature of investigator

Declaration by interpreter
I *(name)***.....** declare that:

- I assisted the investigator (*name*) **.....**

  to explain the information in this document to

  (*name of parent/legal guardian*) **.....**

  using the language medium of Afrikaans/Xhosa.

- We encouraged him/her to ask questions and took adequate time to answer them.
- I conveyed a factually correct version of what was related to me.
- I am satisfied that the parent/legal guardian fully understands the content of this informed consent document and has had all his/her questions satisfactorily answered.

Signed at (*place*) **.....** on (*date*) **.....**20**.....**

.....
Signature of interpreter

Appendix B
Parent Interview Form

PAR – PARENT INTERVIEW (CLINICIAN ADMINISTERED or SELF-REPORT)

PARENT INFORMATION:

Full name:			
Relationship to child:	1. Mother 2. Father 3. Grandmother 4. Grandfather 5. Guardian 6. Other (specify):		
Contact numbers:	Home:	Work:	Cel:
Marital status:	1. married 2. co-habiting 3. widowed 4. divorced & living apart 5. divorced & living together 6. separated 7. remarried 8. other (specify):		
Combined household income (before tax deductions) **PER YEAR**	1. Less than R10 000 2. R10 000 – 20 000 3. R20 000 – 40 000 4. R40 000 – 60 000 5. R60 000 – R100 000 6. More than R100 000		

PARENTAL EMPLOYMENT:

What do you do for a living? (e.g. teacher, professor, unemployed, student)	
What does your child's other parent / caregiver do for a living?	

DEVELOPMENTAL MILESTONES (CHILD)

How old was your child when they did the following tasks for the first time?		
sitting	5 – 8 months	older than 9 months
crawling	7 – 9 months	older than 10 months
walking	11 – 15 months	older than 16 months
first words spoken	10 – 15 months	older than 16 months
speaking in short sentences	18 – 24 months	older than 2 years
speaking in full sentences	3 – 4 years	older than 4 years

PARENTAL EDUCATION:

Highest level of education reached?	Mother	Father	Guardian
Mark one response for each person as follows: 1. 0 years (No Grades / Standards) = No formal education (never went to school) 2. 1-6 years (Grades 1-6 / Sub A-Std 4) = Less than primary education (didn't complete primary school) 3. 7 years (Grade 7 / Std 5) = Primary education (completed primary school) 4. 8-11 years (Grades 8-11 / Stds 6-9) = Some secondary education (didn't complete high school) 5. 12 years (Grade 12 / Std 10) = Secondary education (completed senior school) 6. 13+ years = Tertiary education (completed university / technikon / college) 7. Don't know	1. 2. 3. 4. 5. 6. 7.	1. 2. 3. 4. 5. 6. 7.	1. 2. 3. 4. 5. 6. 7.

Appendix C
Demographics Form

DEM – DEMOGRAPHIC QUESTIONNAIRE (participant self-report)

GENERAL INFORMATION:

Full name:				
How would you describe your race?	1. Black	2. Coloured		3. White
	4. Asian	5. Other(specify):		6. Refuse to answer
Contact numbers:	Person	Home	Work	Cel
	Self			
	Mother			
	Father			
	(Guardian)			
Residential Address:				

EDUCATION:

Name and area of Current School:	School: Suburb / area:

RESIDENTIAL INFORMATION:

How long have you lived at your current address?	
How would you describe your dwelling?	1. Shack 2. Wendy house or backyard dwelling 3. Tent or traditional dwelling 4. Flat / apartment 5. Town house / semi-detached house 6. Freestanding brick house 7. Other (specify):
Which of these items do you have in your home? (mark as many as necessary)	A. Tap water B. Flush toilet inside home C. Electricity D. Telephone (landline) E. Television F. Computer G. Car
How many people sleep in the same room with you at night when you are at home?	1. one 2. two 3. three 4. four 5. five 6. more than five 7. none

FAMILIAL INFORMATION:

Who is your primary care-giver? (Describe the relationship, e.g. mother, father, uncle etc.)	
What is your relationship with your BIOLOGICAL MOTHER?	1. Unknown 2.Known, but irregular contact 3. Known and regular contact 4. Living with child 5. Deceased
How old is she? (If deceased, specify age and reason of death)	
What is your relationship with your BIOLOGICAL FATHER?	1. Unknown 2.Known, but irregular contact 3. Known and regular contact 4. Living with child 5. Deceased
How old is he? (If deceased, specify age and reason of death)	

What is your parents' marital status?	1. married 2. co-habiting 3. widowed 4. divorced & living apart 5. divorced & living together 6. separated 7. remarried 8. other (specify):

Do you live with anyone that has a current alcohol problem or uses drugs?	1. No	2. Yes
	Specify relationship	Specify substance/s abused

Do you live with anyone that used to have an alcohol problem or used drugs in the past?	1. No	2. Yes
	Specify relationship	Specify substance/s abused

SEXUAL HISTORY:

GIRLS ONLY:	
How old were you when you had your first period?	
Have you ever been pregnant?	Yes No
Have you ever terminated a pregnancy?	Yes No
Have you had a child / children?	Yes No
BOYS ONLY:	
How old were you when your voice broke?	
Have you ever made someone pregnant?	Yes Don't know No
BOYS AND GIRLS:	
Have you ever had sex?	Yes No

Have you ever had sex without using a condom?	Yes No
Do you think it is important to use a condom when you have sex?	Yes No
Do you use any form of contraception?	1. None 2. Condom 3. Pill 4. Injection 5. Morning-after pill 6. Other (specify):
Are you waiting to have sex until you are older?	Yes No
Have you ever had any kind of sexual contact with anyone?	Yes No
Did you have sex before your 15th birthday?	Yes No
Have you ever been forced or pressured into having sex when you didn't want to?	Yes No
Have you ever been high on drugs or alcohol when you had sex with someone?	Yes No
During the last 12 months, have you had a discharge from, or sores on your private parts?	Yes No
Have you ever had sex with someone you know or suspect has HIV or AIDS?	Yes No
Have you ever been tested for HIV?	Yes No
When were you last tested for HIV and what were the results?	Date: Result: Positive Negative
Have you had sex with two or more people in the past 3 months?	Yes No
Have you ever had anal sex (this means the penis enters the anus)?	Yes No
Did you use a condom the last time you had sex?	Yes No
When you have sex, do you talk to your partner about using a condom?	Yes No
Have you ever been sexually involved with someone who is more than 5 years older than you?	Yes No
Have you ever had sex with someone who is the same gender as you?	Yes No
Have you ever had sex with someone who has sex with both males and females?	Yes No
Have any of your closest friends had sex?	Yes No

Appendix D
Sociodemographic Characteristics: Individual Samples for EEG Analyses

Table D1

	Group					
	Control			Alcohol		
Outcome variable	Overall	Female	Male	Overall	Female	Male
P3a analysis	(*n* = 41)	(*n* = 24)	(*n* = 17)	(*n* = 30)	(*n* = 19)	(*n* = 11)
Age	15.20 (1.05)	15.15 (1.14)	15.27 (0.95)	15.77 (1.67)	15.70 (1.49)	15.90 (2.01)
Quality of education[a]	33:8	22:2	11:6	25:5	16:3	9:2
Socioeconomic status[b]	(*n* = 41)	(*n* = 24)	(*n* = 17)	(*n* = 28)	(*n* = 17)	(*n* = 11)
	27.59 (6.30)	27.00 (6.72)	28.41 (5.75)	26.46 (6.37)	26.65 (6.20)	26.18 (6.93)
P3b analysis	(*n* = 43)	(*n* = 25)	(*n* = 18)	(*n* = 34)	(*n* = 20)	(*n* = 14)
Age	15.23 (1.04)	15.19 (1.13)	15.28 (0.93)	15.54 (1.67)	15.50 (1.56)	15.60 (1.86)
Quality of education[a]	35:8	23:2	12:6	28:6	16:4	12:2
Socioeconomic status[b]	(*n* = 43)	(*n* = 25)	(*n* = 18)	(*n* = 32)	(*n* = 18)	(*n* = 14)
	27.91 (6.26)	27.60 (6.81)	28.33 (5.58)	26.63 (6.75)	26.89 (6.66)	26.29 (7.11)
F-ERN analysis	(*n* = 27)	(*n* = 15)	(*n* = 12)	(*n* = 18)	(*n* = 9)	(*n* = 9)
Age	15.24 (1.14)	15.10 (1.19)	15.41 (1.10)	15.59 (1.72)	15.45 (1.46)	15.73 (2.02)
Quality of education[a]	21:6	14:1	7:5	16:2	7:2	9:0
Socioeconomic status[b]	(*n* = 27)	(*n* = 15)	(*n* = 12)	(*n* = 18)	(*n* = 9)	(*n* = 9)
	29.11 (6.57)	28.67 (7.16)	29.67 (6.02)	26.72 (7.25)	28.22 (6.82)	25.22 (7.76)

Note. Demographic characteristics appear as relevant to ERP difference waveforms. For the variable *Quality of Education*, the ratio presented is Disadvantaged:Advantaged. For the variables *Age* and *Socioeconomic Status*, values given are means, with standard deviations in parentheses. [a]Based on the Western Cape Education Department's pre-democratization school classification system. [b]Estimated using the SES composite score; SES data were missing for 2 participants in the Alcohol group.

Appendix E
Distributions of the Externalizing Composite and Tower of London Rule Violation Measures

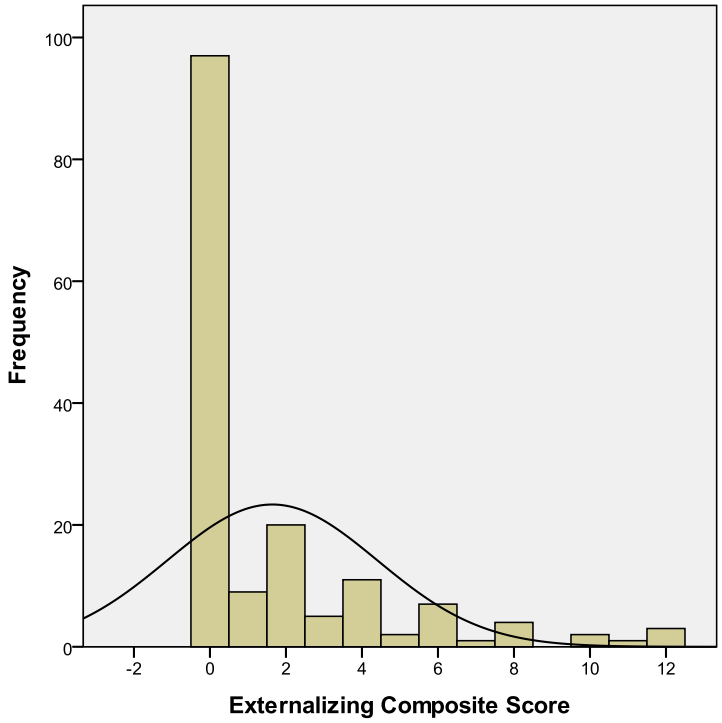

Figure E1. Distribution of scores for the externalizing composite. Each participant's score for this measure was calculated by summing the number of lifetime symptoms for conduct disorder and oppositional defiant disorder for that particular participant.

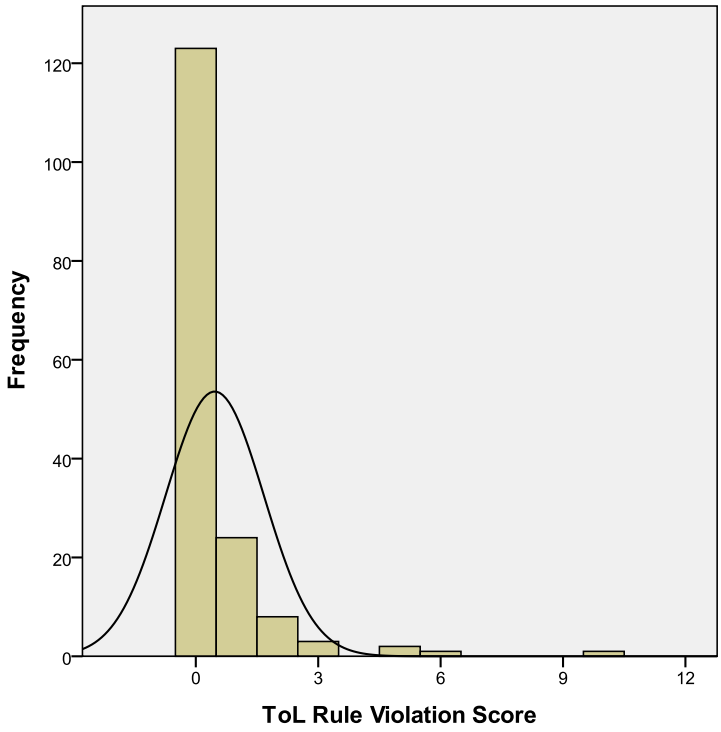

Figure E2. Distribution of scores for Tower of London rule violations.