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EEG Features of Explore-Exploit Decision-Making in Alcohol Use Disorder

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ABSTRACT

Little research has assessed explore-exploit behavior in addiction using drug cues and even fewer studies have measured the neural activity underlying these behaviors. The present study aims to explore brain mechanisms of disordered decision-making in alcohol use disorder (AUD) through electroencephalography (EEG) during performance of a novelty bandit task with alcohol imagery and using a validated computational model of explore-exploit dynamics. Individuals with AUD (n = 28) and age and sex-matched controls (n = 27) showed differences in choice behavior and showed differences in EEG activity as a function of exploratory behavior, chosen stimulus type, and explore-exploit computational parameters. Individuals with AUD also showed a relationship between self-reported symptom severity and exploratory behavior as well as EEG activity and chosen stimulus type. These findings indicate that AUD may be characterized by aberrant exploratory behavior that relates to markers of functional cortical dynamics.

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INTRODUCTION

Alcohol use disorder (AUD) is a substance use disorder (SUD) characterized by an impaired ability to stop or decrease alcohol use despite adverse consequences (NIAAA, 2017) and is one of the most common and debilitating psychological disorders in the world (Grant et al., 2015). The *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5; American Psychiatric Association, 2013) defines AUD as meeting at least two of eleven criteria during a 12-month period. Criteria for AUD listed in the DSM-5 include the inability to control drinking, craving, tolerance, withdrawal, risky behavior, and adverse social, occupational, and health consequences. AUD can be classified as mild, moderate, or severe depending on how many diagnostic criteria are endorsed.

It's estimated that roughly one third of all adults in the United States will meet criteria for AUD at some point in their lives (Grant et al., 2015), while the consequences of alcohol use go beyond the diagnosis itself. With an average of over 90,000 deaths per year, alcohol ranks as the third leading preventable cause of death in the United States (Esser et al., 2020) and results in considerable impairment in physical health, mental health, and social functioning (Rehm, 2011; Ugochukwu et al., 2013). Adding to the disease burden and in part reflecting an inadequate understanding of the disorder, only 7.3% of those diagnosed with AUD receive treatment within a year (SAMHSA, 2019). AUD correlates with a number of putative risk factors such as genetic polymorphisms, neuroticism, impulsivity, parental loss, peer alcohol use, and the prices of alcoholic beverages (Kendler, 2012). The disorder also shares significant psychiatric comorbidity with other SUDs, personality disorders, anxiety disorders, major depressive disorder, attention-deficit hyperactivity disorder, and post-traumatic stress disorder (Castillo-Carniglia, Keyes, Hasin, & Cerdá, 2019).

Though risk factors and comorbidities have been identified, the etiology of AUD is still a matter of ongoing research (Gowin, Sloan, Stangl, Vatsalya & Ramchandani, 2017). While continuous exploitation of alcohol's rewarding properties is clearly at play in AUD, there is a period of time in the development of the disorder where exploration must take place when alcohol is still novel and its reward value unknown (Bidwell et al., 2015), while exploration must also happen when those with AUD have to explore rewarding alternatives to unhealthy drinking behavior. The tradeoff between this kind of exploitation and exploration is a hallmark computational problem in reinforcement learning (RL) that is increasingly relevant to psychiatric research (Addicott, Pearson, Sweitzer, Barack, & Platt, 2017). The explore-exploit tradeoff is only just beginning to be studied in the context of SUDs (Aloi et al., 2021), and measures of this decision-making property may help to improve the diagnosis, treatment, and assessment of AUD.

Computational neuroscience has emerged as a leading framework for leveraging mathematical models of normative brain function to better understand the neural underpinnings of decision-making problems like the explore-exploit tradeoff. Computational approaches can uncover pathological operations underlying motivated decision-making revealing what specific deficits drive maladaptive decisions to consume substances despite adverse consequences. Better characterization of how SUDs like AUD exhibit irregular neuronal computations could contribute to refinement of existing substandard methods of classifying and diagnosing AUDs, predicting AUD risk, and for establishing sensitive measures of treatment-induced change.

Though progress has been made in the identification and treatment of AUD, questions remain as to optimal methods for screening and classifying alcohol use behavior as

pathological and assessing where individuals with an AUD lie along the spectrum of severity (Leggio, Kenna, Fenton, Bonenfant, & Swift, 2009; Hagman, 2017; Campbell and Strickland, 2019). While used as the gold standard for self-reported severity of AUD, questionnaires like the Alcohol Use Disorders Identification Test (AUDIT) (Bush et al., 1998) have demonstrated variable success as screening tools (Lange, Shield, Monteiro, & Rehm, 2019; Moehring et al., 2019). Further complicating the picture, there is evidence that AUD is heterogeneous and can be categorized into subtypes (Müller et al., 2020). The assessment of AUD fundamentally relies on self-reported symptomatology and is thus subject to response bias. Unlike other medical diagnoses, AUD and other psychological disorders are outcome-based rather than process-based (Kwako et al., 2016) and there has been a concerted effort to use neuroscience to improve SUD nosology for over two decades (Charney et al., 2002).

Neural Correlates of Alcohol Use Disorder

In order to best understand how AUD relates to neural signatures of pathological decision-making, it's important to understand how alcohol affects the brain. Ethanol, the chemical responsible for the psychoactive effects of alcoholic beverages, interacts with a number of ligand-gated ion channels and its potentiation of the gamma-aminobutyric acid (GABA)-A receptor is particularly important for the sedative-hypnotic effects that are typical of acute alcohol exposure (Koob, 2003). Alcohol affects a variety of neurotransmitters both directly and indirectly and meta-analyses show that alcohol users have reduced striatal D2/D3 dopamine receptor availability (Kamp et al., 2019). Receptor features of AUD may play a role in potential alterations of explore-exploit behavior which is also affected by

alterations in dopaminergic functioning (Frank, Doll, Oas-Terpstra, & Moreno, 2009; Costa, Tran, Turchi, & Averbeck, 2014; Chakroun, Mathar, Wiehler, Ganzer, & Ganzar, 2020).

Koob and Volkow (2016) proposed an influential three-stage recurring model of addiction that links a series of overlapping and interacting brain circuits that are the targets of acute and chronic effects of addictive drugs such as alcohol. During the binge/intoxication stage, neutral stimuli gain incentive salience associated with drug availability fostering habit formation and drug seeking with associated changes in the basal ganglia. The withdrawal/negative affect stage consists of opponent "anti-reward" effects that occur after the drug disappears via neuroadaptation in circuits like the extended amygdala. The stage of preoccupation/anticipation involves heightened drug cue salience against a backdrop of augmented reward thresholds and sensitized stress neurocircuitry which can lead to relapse from deficits in executive function as mediated by the prefrontal cortex. Models of explore-exploit behavior probe neural computations involved in both habit formation and executive functions like cognitive flexibility, key processes in Koob and Volkow's (2016) three-stage model.

Studies using functional magnetic resonance imaging (fMRI) have been able to parse altered neural activity including reliably enhanced activation of ventral striatum, anterior cingulate, and ventromedial prefrontal cortex (vmPFC) after exposure to alcohol cues in individuals with AUD (Schacht, Anton, & Myrick, 2013). Among those regions that respond to alcohol cues, ventral striatal activity is particularly predictive of relapse behavior (Courtney et al., 2016). Related reward circuitry is active when comparing heavy to light drinkers in areas like the anterior cingulate and insular cortices in response to alcohol cues (Ihssen, Cox, Wiggett, Fadardi, & Linden, 2011). Even prior to diagnosis, individuals at-risk

for AUD have shown enhanced activation of the nucleus accumbens in response to monetary reward cues compared to HCs (Crane et al., 2017).

Altogether, the fMRI research on AUD has highlighted aberrant activity in a network of brain regions implicated in reward responsivity and executive function. Task-related fMRI research in AUD has demonstrated altered fronto-striatal connectivity in transitions from goal-directed to habitual action that may represent a candidate biomarker for impaired decision-making in AUD (Galandra, Basso, Cappa, & Canessa, 2018). However, despite a significant literature characterizing brain networks that are relevant to AUD symptomatology, fMRI is inadequate to fully characterize the disorder's neuropathology because it is an indirect measure of brain activity. fMRI can't capture certain neurophysiological properties and lacks the temporal resolution at which the brain operates during a variety of processes which are relevant to neural activity underlying aberrant decision-making.

Electroencephalography (EEG) Studies of Alcohol Use Disorder

Electroencephalography (EEG) is a neuroimaging tool well suited to capture unique neurophysiological properties of AUD. Because neurocognitive processes occur over very short periods of time spanning from tens of milliseconds (ms) to several seconds, EEG's high temporal resolution allows it to capture the brain's rapid dynamics (Cohen, 2014). In contrast to fMRI, which primarily measure changes in brain hemodynamics, EEG is a direct measure of neuroelectric activity and can reveal more of the brain's distinct physical properties (Pang & Robinson, 2018). The brain is an immensely complex system which transfers information multidimensionally and EEG data has the added benefit of being at least four-dimensional yielding information about time, space, frequency, power (the strength of frequency-specific

based activity), and phase (the timing of frequency-specific activity) (Cohen, 2014). The event-related potential (ERP) represents a simple and precise measure of EEG signal in the time domain with decades of research including in the study of alcohol addiction (Pfefferbaum, Horvath, Roth, & Kopell, 1979).

The P300 (P3) component of the ERP has been specifically implicated in the etiology of AUD since it appears to be attenuated in pre-adolescent sons of those with the disorder (Begleiter, Porjesz, Bihari, & Kissin, 1984). The P3 family has two subcomponents which reflect differentiable psychological processes. The P3a has a mid-frontal topographical distribution, responds and habituates rapidly to novel stimuli, and is thought to reflect a top-down central orienting response (Polich, 2007). The P3b has a more posterior-parietal distribution and is thought to relate to context updating (Donchin, 1981) and accumulation of evidence leading to a decision (Cavanagh, 2015; Twomey, Murphy, Kelly, & O'Connell, 2015; Rac-Lubashevsky & Kessler, 2019).

Both P3 subcomponents play an important role in decision-making suggesting their potential role as distinct computational biomarkers in AUD. In a recent meta-analysis on the P3 in AUD, Hamidovic & Wang (2019) found that the P3b subcomponent is reduced for visual stimuli and auditory stimuli to a lesser degree while findings were mixed for the P3a. The authors suggest that the P3a could be representative of alcohol's neurotoxic effects while the P3b corresponds to AUD's heritability. This assessment comes from findings showing that attenuation of the P3a is specifically associated with frontocortical and hippocampal damage in AUD (Knight, 1984; 1996) while the P3b relates to AUD's heritability (Holguin, Corral, & Cadaveira, 1998). Because individuals with AUD recover from some of alcohol's neurotoxic effects during abstinence (Nixon & Crews, 2004), the P3 subcomponents could

play a role in indexing AUD's dynamic course and phenotypic variation. In line with the notion that P3b reflects AUD heritability, attenuated P3b amplitude during a visual oddball task at age 14 predicted drinking behavior at age 17 (Harper, Malone, & Iacono, 2021).

EEG research has also made great progress in characterizing the brain's reward signal (Glazer, Kelley, Pornpattananangkul, Mittal, & Nusslock, 2018). There have been mixed findings as to whether AUD is generally associated with blunted (Koob, 2011; Aloi et al., 2020) or enhanced reward responsivity (Bjork, Smith, & Hommer, 2008; van Holst, Clark, Veltman, van den Brink, & Goudriaan, 2014; Hixson, Burkhouse, Gorka, & Klumpp, 2019). Despite this ambiguity, little research has been done probing the relationship between AUD and the reward positivity (RewP), an ERP component elicited by rewards and enhanced during better-than-expected feedback (Cavanagh, 2015; Holroyd, Pakzad-Vaezi, & Krigolson, 2008). One preliminary study found that AUD seemed to normalize the RewP of individuals with internalizing psychopathologies (depressive and anxiety disorders) since those without a comorbid AUD showed a blunted RewP compared to those with a comorbid AUD (Hixson et al., 2019). Sehrig, Odenwald, & Rockstroh (2021) found that inducing craving with visual and olfactory alcohol cues accentuated the RewP and variable decision-making in a risk-taking task among high-craving participants. Probing the role of the RewP in AUD will be crucial for fully characterizing its neuropathology as research has increasingly highlighted the role of the RewP in predicting substance use problems (Joyner et al., 2019).

Time-frequency (TF) analysis of EEG captures important multidimensional information about the brain which isn't captured by ERPs. For example, TF data can be interpreted in terms of neural oscillations which are a fundamental organizing mechanism

from synapses to brain networks across multiple spatial and temporal scales (Varela, Lachaux, Rodriguez, & Martinerie, 2001; Cohen, 2014). Some research during resting EEG (when participants are not engaged in an experimental task) demonstrates that elevated beta band activity (13-30 Hz) may be a risk marker for AUD (Coutin-Churchman, Moreno, Añez, & Vergara, 2006; Rangaswamy et al., 2004). One study found that patients with severe AUD show attenuated delta (1-4 Hz) and theta band (4-8 Hz) activity at rest that's associated with cortical atrophy measured by structural MRI (Coutin-Churchman et al., 2006). Another found that AUD patients have decreased delta band activity at rest over frontopolar regions compared to controls and that delta power is modulated by whether patients abstained from or relapsed back into alcohol use during treatment (Saletu-Zyhlarz et al., 2004).

Task-related spectral activity maps onto a number of cognitive domains relevant to AUD. During a Go/NoGo task individuals with AUD have shown diminished delta, theta, and slow alpha band (8-9.5 Hz) activity during both response execution and inhibition as well as diminished fast alpha band (10-12.5 Hz) activity during response inhibition which suggests compromised early attentional processing (Pandey et al., 2016). Theta activity along the midline frontal cortex in particular has been suggested as an important marker of cognitive control (Cavanagh & Frank, 2014) and research suggests that it relates to AUD severity during flanker task performance suggesting weakened response conflict resolution (Harper, Malone, & Iacono, 2018).

Based on over half a century of research it is clear that AUD is associated with measurable alterations in brain activity. In spite of this, while research in cancer and heart disease has uncovered biomarkers that have been used effectively in screening, diagnosis, prognosis, monitoring and treatment selection over the past 20 years, psychiatry has yet to

uncover remotely comparable indicators of dysfunction for AUD and other psychological disorders (Ludwig & Weinstein, 2005; Jaffe, Babuin, & Apple, 2006; Jollans & Whelan, 2018). This is in large part due to the complexity and heterogeneity of symptom presentation in AUD which cannot be captured by standardized self-report measures. By improving our understanding of the psychological dimensions of AUD such as complex decision-making, we can get closer to a better understanding of the brain mechanisms underlying AUD symptomatology.

Decision-Making in Alcohol Use Disorder

Many consequences of SUDs result from their deleterious acute and long-term effects on cognition and behavior (Kwako, Momenan, Litten, Koob, and Goldman, 2016). In AUD, there are associated deficits in response inhibition (Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009), delay discounting (Petry, 2001), episodic memory (Noël et al., 2012), working memory (Kopera et al., 2012), and cognitive control (Wilcox et al., 2014). Impairments in other executive functions are also a feature of AUD and have been found in reward-guided decision-making (Beylergil et al., 2017), cue reactivity (DePalma, Ceballos, & Graham, 2017), attentional bias (Zetteler, Stollery, Weinstein, & Lingford-Hughes, 2006), social cognition, and emotional processing (Bora and Zorlu, 2017; Le Berre, 2019).

The field of computational psychiatry, defined broadly as a mathematical approach to assess latent drivers of behavior in psychopathology, has recently made important strides in better understanding the precise decision-making aberrations and their neural correlates in addiction (Gueguen, Schweitzer, & Konova, 2021). In simple RL paradigms there appear to be minimal differences between those with SUDs and HCs (Park et al., 2010; Myers et al., 2016) and mixed evidence has been found for reduced dopaminergic encoding of reward

prediction error (RPE; the difference between expected and received reward) (Chiu, Lohrenz, & Montague, 2008; Deserno et al., 2015).

To better understand how addiction might be characterized by a shift to habitual compulsion from goal-directed behavior, ‘model-free’ and ‘model-based’ algorithms have been used to map these distinct mechanisms. An imbalance between these systems has been demonstrated in AUD (Huys, Deserno, Obermayer, Schlagenhauf, & Heinz, 2016) and has been shown to increase with shorter durations of abstinence (Doñamayor, Strelchuk, Baek, Banca, & Voon, 2018), though it may only be present after chronic use (Nebe et al., 2018). Hogarth (2020) contends that much of the work on habitual behavior in rodent models of addiction occurs in decision environments which don’t reflect the complexity present in human addiction and contrary to habit theories of addiction, the imbalance between habitual and goal-directed behavior in addiction appears to be driven by reduced model-based RL rather than resulting from overreliance on model-free decision-making (Sebold et al., 2014; Reiter et al., 2016).

Gueguen, Schweitzer, & Konova (2021) propose a multidimensional and temporally dynamic model of computational decision-making which accounts for loss aversion, risk tolerance, learning rate, ambiguity tolerance, and model-based/mode-free imbalance. They argue that broadening the parameter space of computational decision-making could increase fidelity with regard to assessing different addictive substances, stages in the ‘addiction cycle’, and clinical subtypes given the heterogeneity problem in psychiatric nosology (Konova et al., 2020; Feczko et al., 2019). Moreover, a lack of exploration of potentially rewarding activities which serve as an alternative to drinking may be important for AUD. Given the multitude of decision-making parameters that could be relevant to AUD, the

explore-exploit dilemma represents an understudied dimension of decision-making that deserves expanded investigation.

The Explore-Exploit Tradeoff

The explore-exploit tradeoff represents a crucial component of reward-guided decision-making, wherein individuals choose between exploring options with uncertain outcomes or exploiting known outcomes (Addicott et al., 2017). While exploitation may maximize near-term reward, the information that is learned during exploration can be used later to maximize rewards in the long-term so that a deliberate balance between the two strategies is required to optimize performance (Barack & Gold, 2016). The restless multi-armed bandit task captures this tradeoff and can model flexible, adaptive behavior in response to changing stimulus probabilities that ties to motivational neurocircuits (Cavanagh, 2015; Ebitz, Albarran, & Moore, 2018; Costa, Mitz, & Averbeck, 2019). Importantly, the task also taps into model-based directed exploration rather than random exploration driven by decision noise (Zajkowski, Kossut, & Wilson, 2017).

Using Partially Observable Markov Decision Processes (POMDPs), which are useful in tasks where the future depends on present choices, has allowed an optimal characterization of normative explore-exploit decision-making (Averbeck, 2015). Importantly, the models yielded by these frameworks can be parameterized to fit individual behavior (Furl & Averbeck, 2011) and can suggest biases or deficits that help specify pathological decision-making (Averbeck et al., 2013). POMDP modeling provides measures of trial-by-trial changes in choice behavior in response to dynamic presentation of information in ways which may better capture latent computations of real-world decision-making. Importantly, POMDP modeling of bandit task choice behavior has shown preference over alternative RL

models in predicting animal behavior (Costa, Mitz, & Averbeck, 2019). There is a body of literature assessing explore-exploit processes in addiction. This includes studies of cigarette smokers (Addicott et al., 2013), methamphetamine users (Harle et al., 2015), cocaine users (Wang et al., 2019), AUD compared to binge eating disorder (Morris et al., 2016), individuals with more than one SUD (Smith et al., 2020), and adolescents with AUD or cannabis use disorder (Aloi et al., 2021). However, few of these studies have used drug cues, few have measured the neural activity of substance users, and none have used predictive computational modeling approaches.

The present study aims to investigate the relationship between neural activity and explore-exploit decision-making in response to alcohol cues in AUD vs. controls and along the spectrum of severity within AUD. To measure categorical neurobehavioral features of AUD, between-group analyses will measure brain responses during explore-exploit behavior as a function of whether or not someone has an AUD or is a healthy control using the whole sample. Variance along the AUD spectrum will be measured via brain responses during bandit performance as a function of AUDIT scores only among individuals who have an AUD.

METHODS

Inclusion and Exclusion Criteria

All participants had to be between the ages of 18 and 55 and fluent in English with no history of epilepsy or seizure, no neurological impairment or learning disorder, no current use of psychoactive medication, and no history of head trauma resulting in loss of consciousness for over 5 minutes. Control participants also had to have an AUDIT score of 3 or below at the time of their participation. AUD participants had to be recruited from

ABQDRINQ (NIH #R01AA025762), a separate study conducted by investigators from the UNM Center on Alcoholism, Substance Abuse, and Addictions (CASAA) and the Mind Research Network (MRN) to assess neurocognitive patterns associated with changes in the drinking behavior of moderate to heavy drinkers. Inclusion criteria for ABQDRINQ included self-identity as a heavy/binge/weekly drinker, being right-handed, having used alcohol during the 30 days prior to study admission, having an AUDIT score > 8 for men and > 7 for women, and explicitly seeking help for their drinking. Additional exclusion criteria for ABQDRINQ included being in treatment for AUD during the 6 months prior to admission, history of major alcohol withdrawal, and past-year substance dependence other than nicotine or cannabis.

Participants

30 participants (17 female) were recruited from the ABQDRINQ study for an AUD sample. 28 controls (16 female) were recruited from other studies in the Cavanagh lab as well as from the community. One control participant was removed due to having an AUDIT score that was too high and preliminary data preprocessing revealed two participants whose EEG data were compromised, resulting in a final $n = 28$ (16 female) in the AUD group and $n = 27$ (16 female) controls. Table 1 includes descriptive statistics for demographic information between groups. Chi-square tests of independence revealed no statistically significant differences between groups for sex ($p = 0.874$), ethnicity ($p = 0.933$), or race ($p = 0.231$). Independent samples t-tests revealed no statistically significant differences between groups for age ($p = 0.741$) or years of education ($p = 0.151$). Welch's unequal variances t-tests showed statistically significant group differences for AUDIT ($p < .001$) and Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988) scores ($p = 0.023$). Across the whole sample,

none of these variables were intercorrelated, but within AUD, AUDIT scores correlated with BDI (Spearman's rho = 0.403, $p = 0.034$) and years of education (Spearman's rho = 0.454, $p = 0.015$). Though both groups differed by BDI, it was not included in analyses as depressive symptoms are a characteristic feature of AUD (Li et al., 2020) and thus shouldn't be included as a covariate (Dennis et al., 2009).

Table 1

Group Demographics

		Control	**AUD**
N (female)		27 (16)	28 (16)
Age (SD)		37.7 (10.7)	38.6 (9.43)
Years of education (SD)		16.1 (2.96)	15 (2.3)
AUDIT (SD)		1.33 (0.92)	10.5 (5.57)
BDI (SD)		4.63 (7.69)	11 (12.1)
Ethnicity	Hispanic:	9	10
	Non-Hispanic:	17	18
Race	Asian:	3	0
	African American:	0	0
	Caucasian:	19	17
	Native American/Alaskan:	2	5
	Native Hawaiian/Pacific Islander:	0	0
	Mixed race:	0	1
	Other:	2	3

Procedure

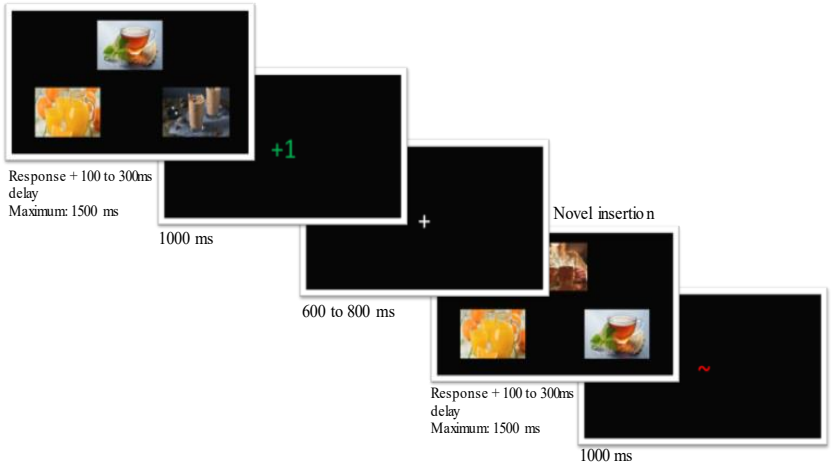
For recruitment of some controls, the AUDIT was completed over the phone in addition to in lab before participation to ensure individuals' scores didn't meet the AUDIT exclusion criterion, but in lab AUDIT scores were used for statistical analyses. After arriving at the lab and going over and signing a consent form, individuals completed a series of paper questionnaires including the AUDIT and BDI. EEG was recorded on a 64-channel Brain Vision system (Brain Products GmbH, Munich, Germany) between .01-100 Hz at a sampling rate of 500 Hz. Vertical electrooculogram (VEOG) and electrocardiogram (EKG) were used to capture ocular and cardiac artifacts and linked mastoid electrodes were used for baseline referencing with CPz as an online reference.

Task

Participants completed a series of computerized tasks including the three-armed bandit task with alcohol and non-alcohol beverage stimuli (Figure 1). The task consisted of 350 trials in which participants chose between three images that were probabilistically associated with a reward. Fixation crosses were displayed for a variable length of time between 600 and 800 ms between trials. Trials consisted of three peripheral choice targets being presented

Figure 1

Three-Armed Bandit Task Schematic



in the upper, left, or right area pseudo-randomly assigned on each trial (Figure 1). Participants had 1500 ms to select one of the three images by pressing one of three face buttons on a handheld controller corresponding to the locations of the images. If the participant failed to respond in time, a null signal ("No Response Detected") would be displayed. After their response and a delay between 100 and 300 ms, feedback was provided with either a green +1 or a red ~ for 1000 ms. During the experiment, 50 images (split evenly between alcohol and non-alcohol stimuli) were introduced which randomly replaced one of the existing options with a minimum of 5 trials and a maximum of 9 trials between novel insertions (mean number of trials between novel insertions = 6.86, SD = 1.63). At the start of the experiment, the three initial choices were randomly assigned a reward probability of 0.2, 0.5, or 0.8. Novel choice options were also randomly assigned one of these reward probabilities when introduced. No more than two of the three options could be assigned the same reward probability at a time. Participants were instructed to win as many points as possible by choosing the image that rewarded them most often. They were also told about the probabilistic nature of the rewards and that the images' positions did not affect the probability of receiving a reward.

Data Analysis

To test hypotheses about the neural substrates of explore-exploit behavior in AUD these data were modeled as a function of both i) discrete explore-exploit decision events surrounding novel stimulus insertions, and ii) continuous changes in latent value parameters associated with alcohol and non-alcohol choice options using the well-validated POMDP model of explore-exploit behavior (Averbeck, 2015).

Computational Modeling

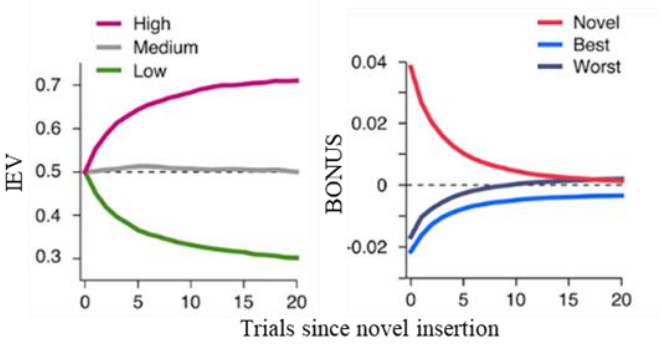
Optimal explore-exploit decision making was modeled based on estimates of state and action values derived from a POMDP. Each trial represents a decision state where state value is the value of the option with the highest action value among the three choices and the action value is the sum of its immediate expected value (IEV) and future expected value (FEV). IEV is an estimate of the likelihood that a given option will be rewarded based on prior outcomes, whereas FEV is the sum of potential future rewards. An additional parameter, referred to as the exploration BONUS, reflects trial-to-trial changes in exploration based on the difference in the FEV of an individual option relative to the average FEV of all available options. Figure 2 shows how the POMDP parameters change across trials since novel stimuli are introduced to model explore-exploit behavior. IEV increases as the stimulus with the highest reward probability is sampled. The BONUS parameter diminishes as novel stimuli are explored. POMDP-derived explore-exploit parameters have already demonstrated predictive validity for modeling behavior in nonhuman primates (Costa et al., 2019) and in humans (Hogeveen et al., 2021).

EEG Data Preprocessing and Analysis

All EEG preprocessing was carried out in MATLAB and made use of EEGlab (Delorme & Makeig, 2004). EEG data were epoched at -500 to 1000 ms relative to stimulus presentation for cue-locked data and at -500 to 1000 ms relative to feedback stimulus presentation.

Figure 2

POMDP Parameters



Note. Reproduced with permission from Costa et al. (2019)

Data were high-pass filtered at 0.1 Hz and independent components analysis was used to remove eyeblinks. Bad electrodes and epochs were removed after visual inspection of the data and data were averaged referenced. EEG epochs were separated into three categories based around the insertion of novel stimuli with pre-insertion, insertion, and post-insertion trials being delineated for different analyses. TF measures were derived from custom-written MATLAB functions (Cavanagh, Cohen, & Allen, 2009). The fast Fourier transformed (FFT) power spectrum of single trial EEG data was multiplied with the FFT spectrum of complex Morlet wavelets with the final result representing time on the x-axis (in ms), frequency on the y-axis (in Hz), and power represented by a color scale (in decibels) for TF analysis.

Regions of interest in ERP were selected from electrodes which were closest to maximal activation along the midline during conditions or behaviorally relevant contrasts between conditions i.e. alcohol minus non-alcohol insertion trials. P3a was selected from midfrontal electrodes and P3b was selected from posterior-parietal electrodes and time windows for P3 subcomponents were based on the peak of maximum activation between 250 and 500 ms (Johnson, 1993; Polich, 2007). The RewP was defined as maximal activation appearing at around 250 ms over fronto-central sites following rewarding feedback (Cavanagh, 2015). The frontal theta region of interest was selected based on maximal activation within the approximate theta band range in the time window following cue presentation across conditions (Cavanagh & Frank, 2014). Feedback-related TF activity was selected based on condition wherein reward-locked TF activity showed the largest amount of activity in the delta range and non-reward-locked activity showed the largest amount of activity in the theta range, both reflecting prior research on feedback-related oscillatory dynamics (Cohen, Elger, & Ranganath, 2007; Knyazev, 2007).

Hypotheses

In the context of a bandit task, the P3a is elicited by novel salient stimuli (Polich, 2007; Cavanagh, 2015) such as novel insertions. The P3b is elicited by cues with enhanced motivational significance (Nieuwenhuis et al., 2005; Cavanagh, 2015) such as cues with higher reward probabilities. The RewP is elicited by surprising rewards during feedback after participants have made bandit selections (Cavanagh, 2015). Robust linear mixed effects models (RLMMS) were used for analysis with predictor variables of interest as fixed effects and participant ID as a random effect and were generated using the R package robustlmm. An RLMM approach was chosen because it does not make parametric assumptions about data structure (besides that model parameters are estimable) and has demonstrated efficiency in limiting outlier influence (Koller, 2016).

Discrete explore-exploit behaviors were measured via participant's selection of the novel option (i.e. exploration), the best alternative (i.e. exploiting the non-novel option with highest reward probability), or the worst alternative (i.e. non-novel option with lowest reward probability). On the first two trials after a novel insertion, participants are expected to be more likely to explore the novel stimulus than to exploit the best alternative option based on prior data collected in the Hogeveen lab (Hogeveen et al., 2021). This demonstrates that participants learn to explore novel stimuli rather than engaging in random exploration which often occurs in bandit tasks that lack novel stimuli (Daw, O'Doherty, Dayan, Seymour, & Dolan, 2006; Speekenbrink & Konstantinidis, 2015; Averbeck, 2015).

It's hypothesized that those with AUD will demonstrate higher selection of novel and best alcohol stimuli than controls and that AUD severity will scale positively with selection of best and novel alcohol stimuli within AUD. To test these hypotheses, RLMMs will be run

predicting choice probabilities with group for the between groups analysis and AUDIT for the within group analysis and chosen cue type as fixed effects. Following main effects analyses, interactions between group and cue type and AUDIT and cue type will be run to evaluate the potential influence of AUD and severity on choice behavior toward alcohol stimuli.

EEG features are hypothesized to be affected by choice behavior in response to novel alcohol cues between groups. The P3a, P3b, and frontal theta activity are hypothesized to be increased in AUD in response to alcohol cues relative to controls. Individual RLMMs will be run predicting P3a amplitude, P3b amplitude, and frontal theta power, respectively, by an interaction between cue type, group, and probability of choosing the novel stimulus. EEG features are also hypothesized to increase with selection of novel alcohol cues within AUD as a function of AUDIT. To test these hypotheses, individual RLMMS will be run predicting P3a amplitude, P3b amplitude, and frontal theta power, respectively, by an interaction between cue type, AUDIT, and probability of choosing the novel stimulus.

Since the BONUS parameter represents a potentially more sensitive measure of exploratory behavior, it is also hypothesized to increase and scale positively with EEG features toward alcohol cues in AUD relative to. Individual RLMMs will be run predicting P3a amplitude, P3b amplitude, and frontal theta power, respectively, with an interaction between group, cue type, BONUS and IEV to distinguish explore and exploit decision-making. Similarly, the BONUS parameter is hypothesized increase in response to alcohol cues in conjunction with AUDIT as a function of increased activity of EEG features within AUD. Individual RLMMs will be run predicting P3a amplitude, P3b amplitude, and frontal theta power by an interaction between AUDIT, cue type, BONUS and IEV.

Because aberrant reward sensitivity is a feature of AUD (Hixson et al., 2019), feedback-locked activity is also hypothesized to be affected by choice behavior between groups. Despite prior evidence showing the RewP doesn't predict explore-exploit behavior (Cavanagh, 2015), the present study aims to investigate the degree to which the RewP and feedback-locked delta activity may relate to the potentially more sensitive POMDP parameters given the increasing importance of the RewP in SUD research (Joyner et al., 2019). It's hypothesized that the RewP and related delta activity are augmented in AUD in as a function of BONUS for alcohol cues relative to controls. Individual RLMMs will be run predicting RewP amplitude and reward-locked delta activity, respectively, by an interaction between group, cue type, and BONUS or IEV. It's also hypothesized that the RewP and reward-locked delta activity will increase with BONUS toward alcohol cues along the AUDIT spectrum within AUD. Individual RLMMs will be run predicting RewP and reward-locked delta activity, respsectively, by an interaction between AUDIT, cue type, and BONUS or IEV. Similar models will be run on non-reward-locked activity to differentiate specificity to the reward signal.

RESULTS

Behavioral Data

On the Alcohol Three-Armed Bandit Task, periodic novel stimulus insertions explicitly forced participants to make explore-exploit choices (cf., Costa et al., 2019). Therefore, to directly quantify explore-exploit behavior, we computed the choice probability of selecting the novel stimulus (exploration) versus the best available alternative (exploitation) on the first two trials post-insertion. Participants tended to choose the best alternative more often than the novel stimulus (MD = 0.064; $t(54) = 2.73$, $p = 0.008$) and

tended to choose the novel stimulus more often than the worst stimulus (MD = 0.066; t(54) = 3.78, $p < .001$).

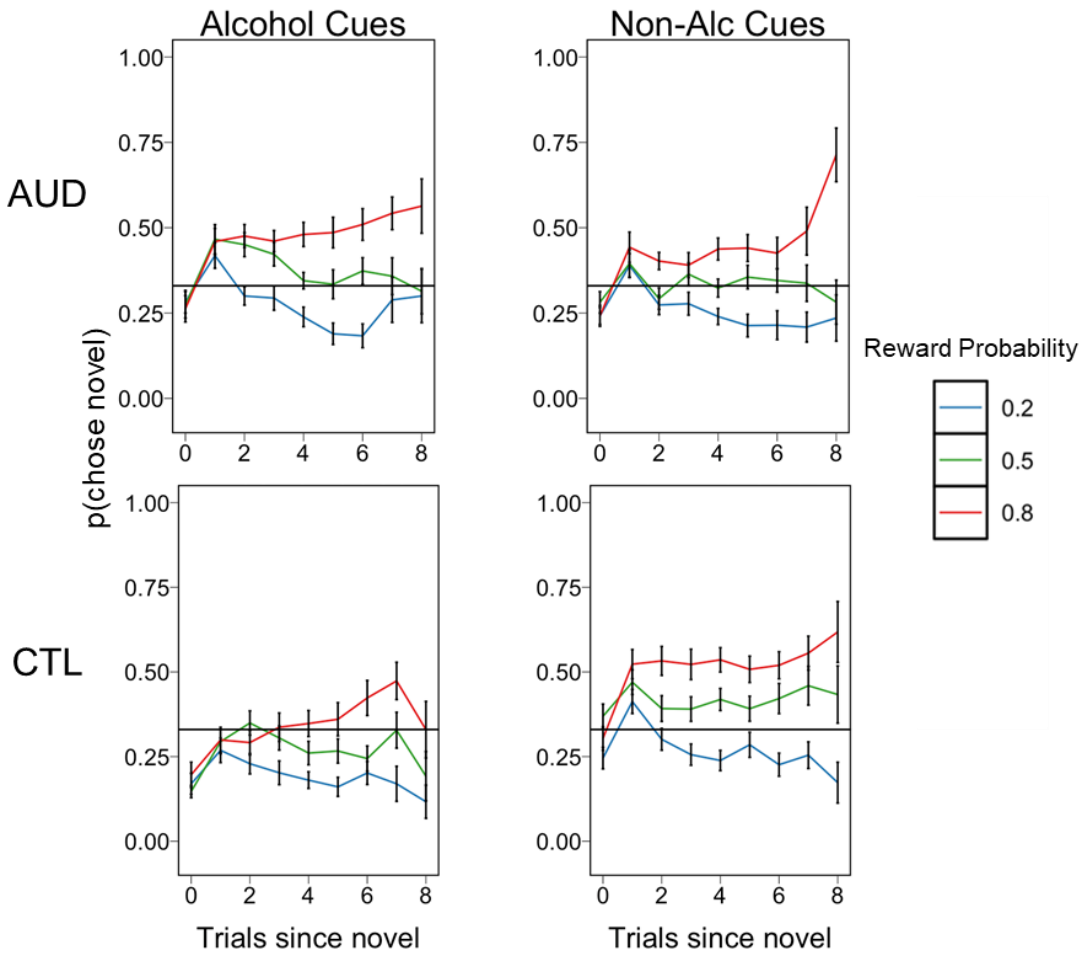
Figure 3 displays the probability of each group selecting a novel stimulus between alcohol and non-alcohol stimulus types at each probability of reward across trials since a novel insertion. Figure 4 displays probabilities of selecting the novel, best, or worst stimulus for each group across trials since a novel insertion. Participants chose the best stimulus more often as trials since a novel insertion increased (beta = 0.013, $p < .001$; Figure 4) demonstrating that they learned to exploit the stimulus most likely to offer a reward over time. Participants also chose the novel stimulus more often as a function of reward probability and trials since a novel insertion (beta = 0.017, $p = 0.005$; Figure 3), demonstrating that they learned to explore rewarding novel stimuli over time. Across groups, participants showed a preference for exploring novel non-alcohol stimuli (M = 0.355, SD = 0.138) over alcohol stimuli (M = 0.289, SD = 0.141); t(109) = 3.373, $p = 0.001$. Figure 5 shows response times across trials since a novel insertion between groups and cue types.

Importantly, the probability of exploring the novel stimulus was predicted by an interaction between group and cue type (Table 2; beta = 0.202, t = 5.594, $p < .001$). Specifically, those with AUD selected novel alcohol stimuli more often than controls (beta = 0.136, z = 3.689, $p < .001$) and controls selected non-alcohol stimuli more often than alcohol stimuli (beta = 0.168, z = 6.524, $p < .001$) (Figure 6). No statistically significant relationships were found for probability of selecting the best or worst stimulus between groups. For the within AUD analysis, the probability of selecting the best stimulus was positively predicted by AUDIT scores (Figure 7; Table 3; beta = 0.008, t = 2.436, $p = 0.002$), but did not vary as a function of cue type, indicating a generalized association between AUD severity and sticking

with the best available option. This effect remained after controlling for years of education as a fixed effect (beta = 0.009, t = -0.834, *p* = 0.016), which was correlated with AUDIT within AUD (Figure 9). Figure 8 shows the relationship between AUDIT and BDI scores.

Figure 3

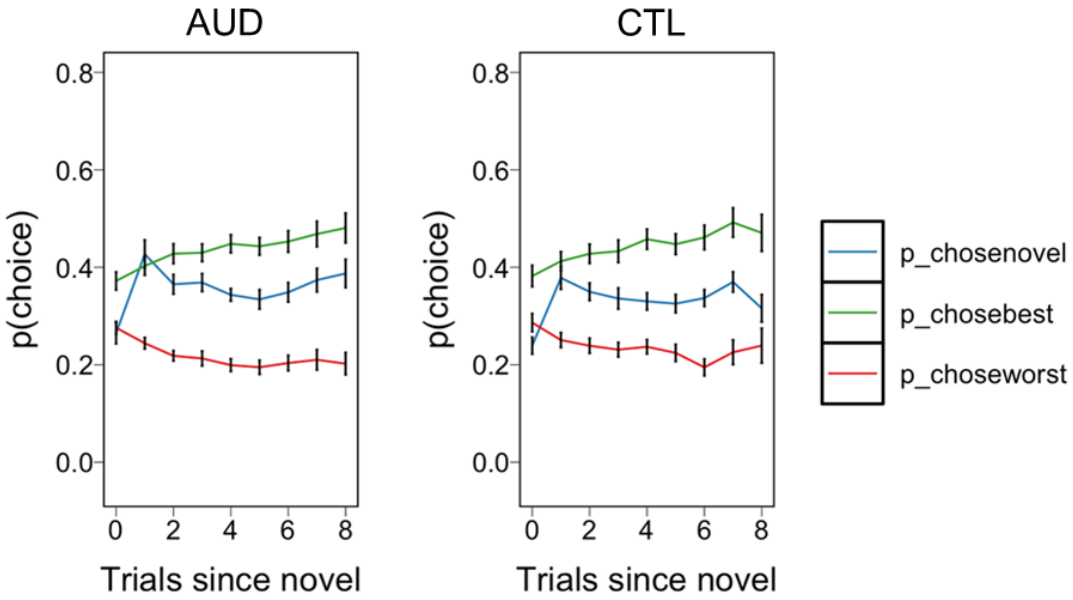
Novel Choice Behavior by Group and Stimulus Type



Note. The probability of choosing the novel stimulus at each level of reward probability

Figure 4

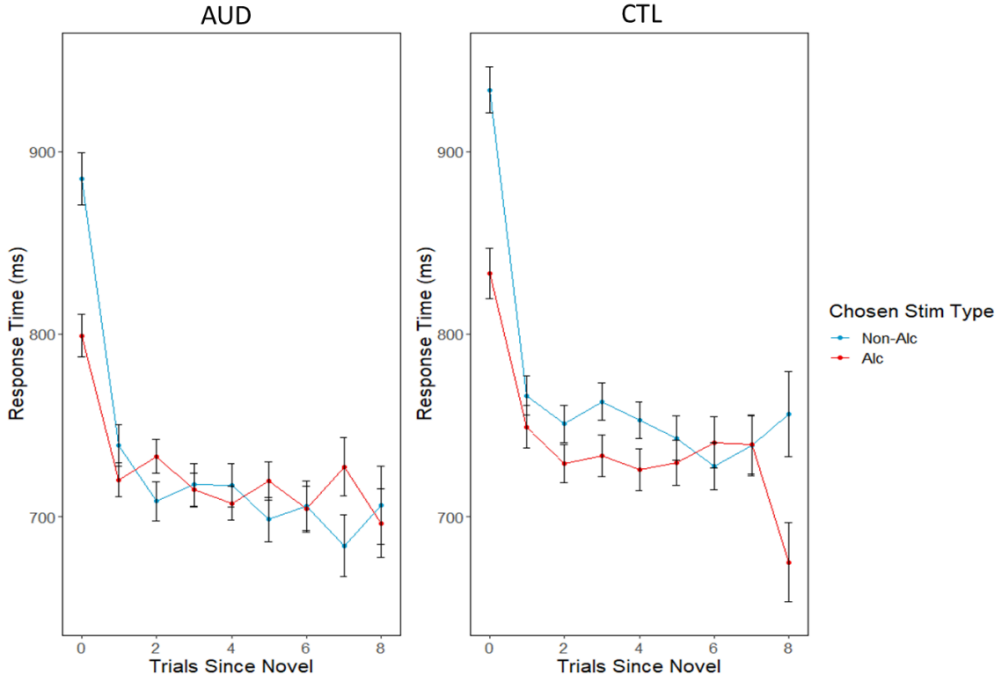
Explore-Exploit Choice Probabilities Between Groups



Note. Probability of selecting the novel option, the best alternative, or the worst alternative (i.e. the non-novel option with the lowest reward probability).

Figure 5

Response Time by Group and Chosen Cue Type



Note. Response time in milliseconds over trials since a novel insertion

Table 2

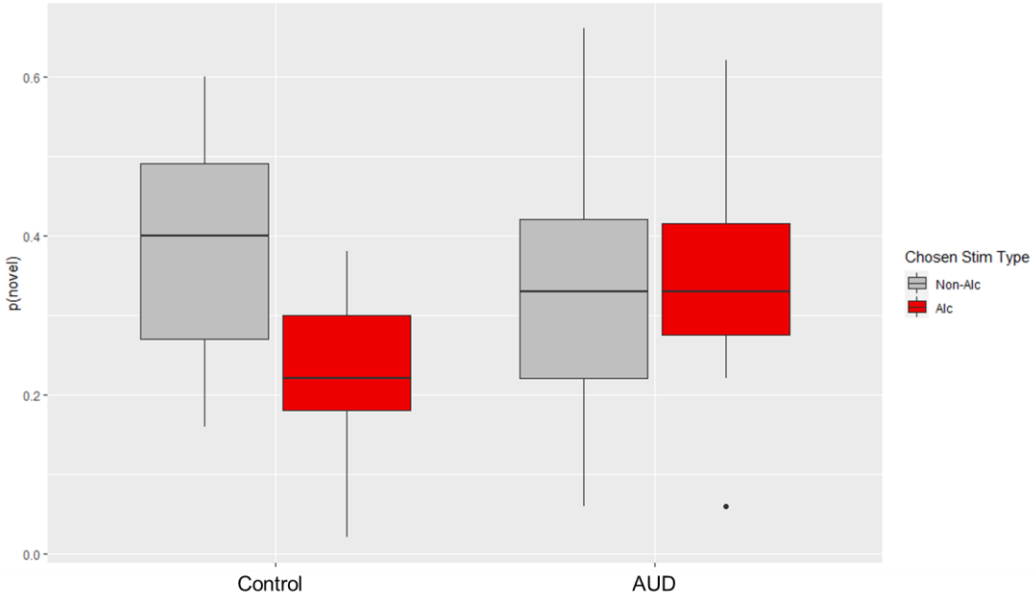
Probability of Choosing the Novel Stimulus Between Groups and Within AUD

**Main effects and interactions modelled separately*

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	0.336	0.024	<.001
	Group	0.034	0.029	0.243
	Cue type	**-0.071**	**0.026**	**0.009**
Interaction				
	Group*cue type	**0.202**	**0.036**	**<.001**
Within AUD				
Main effects				
	Intercept	0.26	0.055	<.001
	AUDIT	0.006	0.005	0.173
	Cue type	0.036	0.023	0.125
Interaction				
	AUDIT*cue type	-0.001	0.004	0.865

Figure 6

Boxplot of Probability of Choosing the Novel Stimulus by Group and Cue Type



Note. Probability of choosing the novel stimulus on insertion and one trial after

Table 3

Probability of Choosing the Best Stimulus Between Groups and Within AUD

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	0.385	0.023	<.001
	Group	-0.001	0.029	0.975
	Cue type	-0.002	0.02	0.927
Interaction				
	Group*cue type	-0.02	0.035	0.574
Within AUD				
Main effects				
	Intercept	0.301	0.042	<.001
	AUDIT	**0.008**	**0.003**	**0.022**
	Cue type	-0.011	0.025	0.658
Interaction				
	AUDIT*cue type	0.005	0.004	0.303

Figure 7

Within AUD p(best) by AUDIT with Model Fit

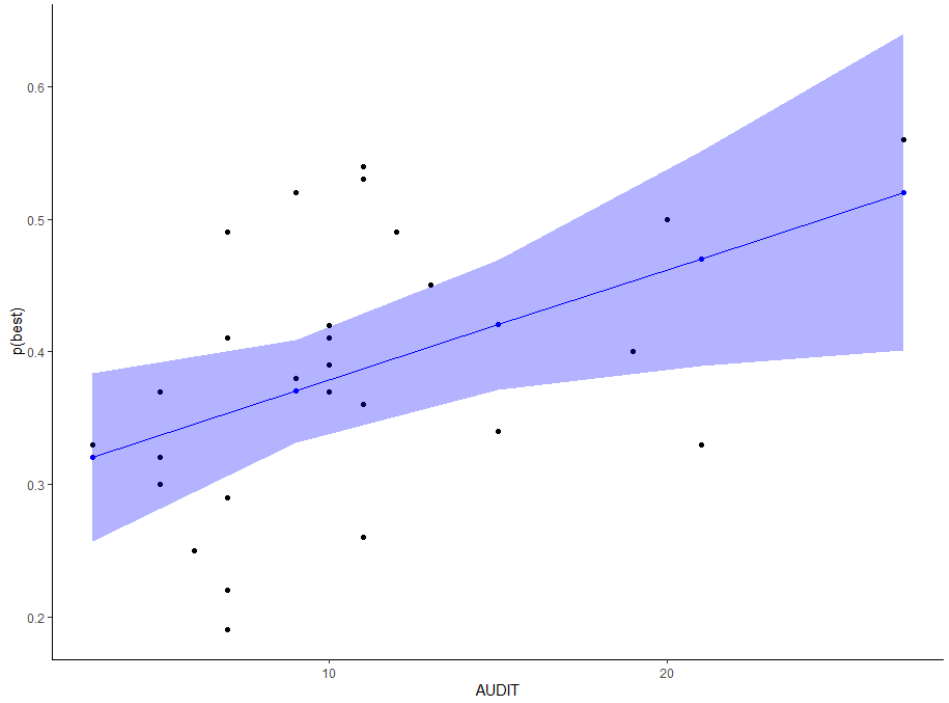


Figure 8

Relationship Between BDI and AUDIT within AUD

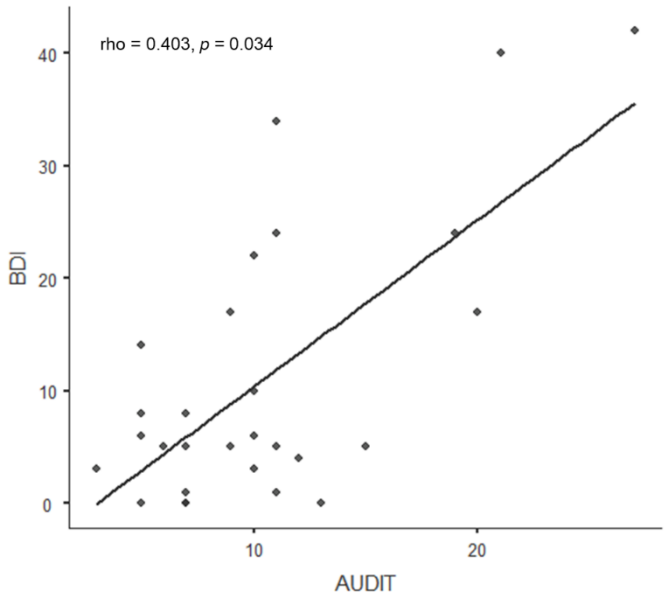
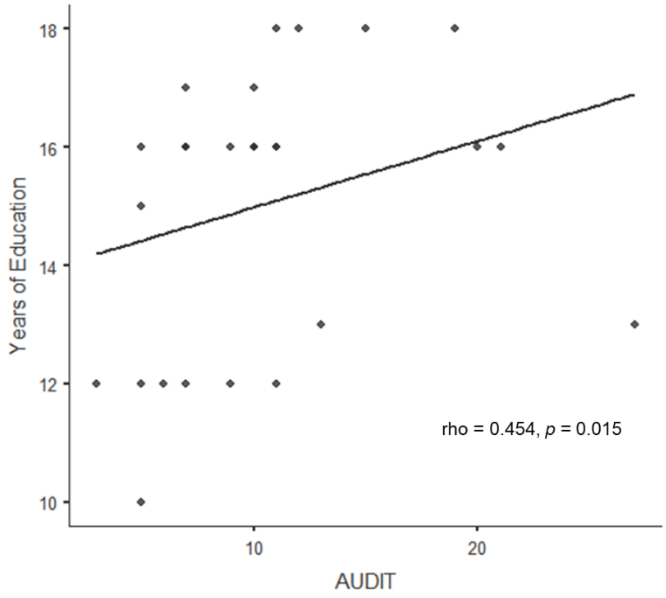


Figure 9

Relationship Between Years of Education and AUDIT within AUD



POMDP Data

For analyses of subject-level POMDP parameter estimates, one control participant was removed as an outlier for having a BONUS score that was 8.113 standard deviations below the mean. Between groups, BONUS was predicted by an interaction between group and cue type (Table 4; beta = 0.126, t = 2.189, p = 0.033). Specifically, AUD had higher BONUS values for alcohol stimuli than controls (beta = 0.155, z = 2.285, p = 0.022), suggesting that they showed a stronger preference for novel alcohol stimuli, and AUD had higher BONUS values for alcohol stimuli than for non-alcohol stimuli (beta = 0.1, z = 2.5, p = 0.012), suggesting they showed a preference for alcohol stimuli over non-alcohol stimuli as they sampled novel cues (Figure 10).

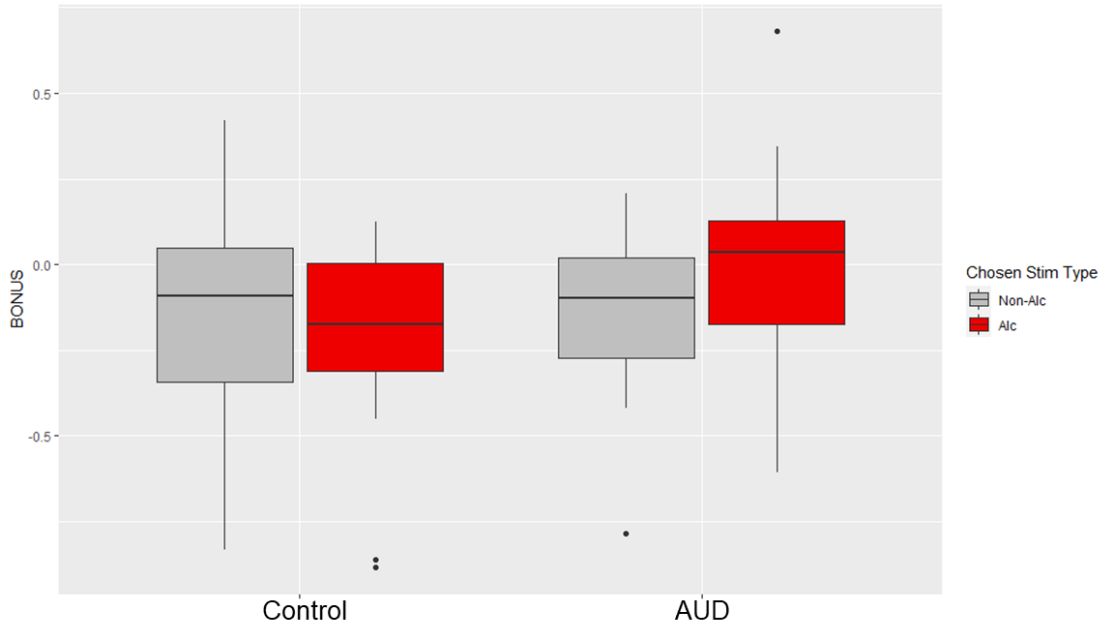
Table 4

BONUS Between Groups and Within AUD
Main effects and interactions modelled separately

Between groups				
Main effects				
	Effect	Estimate	*SE*	*p*
	Intercept	-0.184	0.048	<.001
	Group	0.095	0.063	0.134
	Cue type	0.045	0.03	0.141
Interaction				
	Group*cue type	**0.126**	**0.058**	**0.033**
Within AUD				
Main effects				
	Intercept	0.301	0.042	0.01
	AUDIT	0.012	0.007	0.005
	Cue type	**0.1**	**0.032**	**0.005**
Interaction				
	AUDIT*cue type	0.006	0.006	0.323

Figure 10

Boxplot of BONUS by Group and Cue Type



EEG Data

Wilcoxon signed-rank tests revealed no significant differences in P3a amplitude between pre-insertion and insertion trials (p = 0.747), insertion and post-insertion trials (p = 0.811), or pre-insertion and post-insertion trials (p = 0.779). P3b amplitude did not correlate with probability of choosing the best stimulus (rho = -0.153, p = 0.112) or with IEV (rho = -0.071, p = 0.46). Rewarding feedback elicited a RewP based on a paired samples t-test between feedback amplitude following reward (M = 2.08, SD = 1.07) versus non-reward (M = 1.88, SD = 1.14); t(54) = 2.32, p = 0.024.

Figures 11-17 show ERP and TF plots with marked regions of interest as well as topographical plots. During the first two trials post-insertion, P3a amplitude was predicted by a three-way interaction between group, cue type, and probability of choosing the novel stimulus (Table 5; beta = 10.814, t = 2.511, p = 0.015). This relationship is driven by the

difference between stimulus types in AUD (beta = 5.71, z = 2.371, p = 0.018) with a positive relationship between P3a and probability of selecting the novel stimulus for alcohol stimuli (beta = 4.715, p = 0.047) (Figure 18). Within AUD frontal theta power at feedback during non-reward trials was predicted by an interaction between cue type and AUDIT scores (Table 5; beta = 0.036, t = 2.493, p = 0.019).

Figure 11

P3a Region of Interest Across Conditions

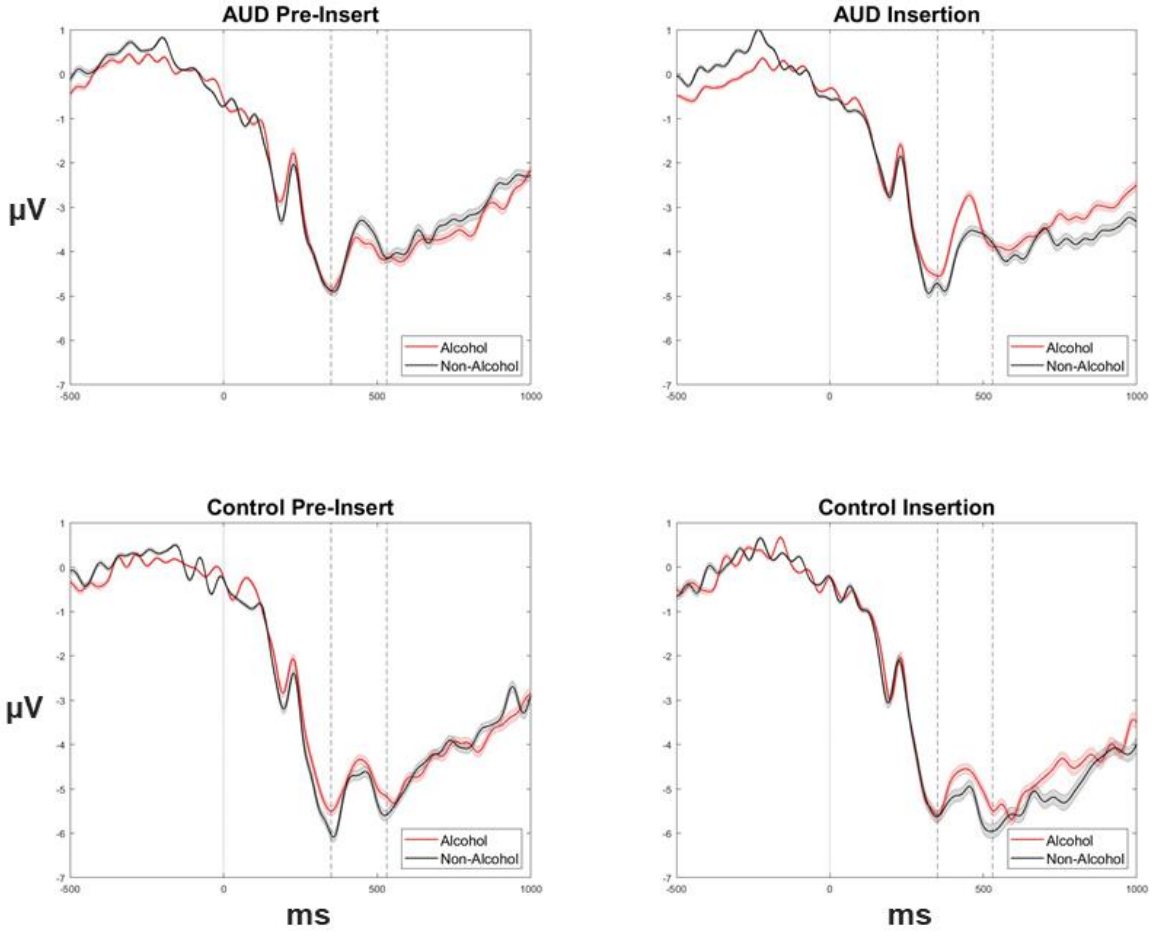


Figure 12

P3b Region of Interest Across Conditions

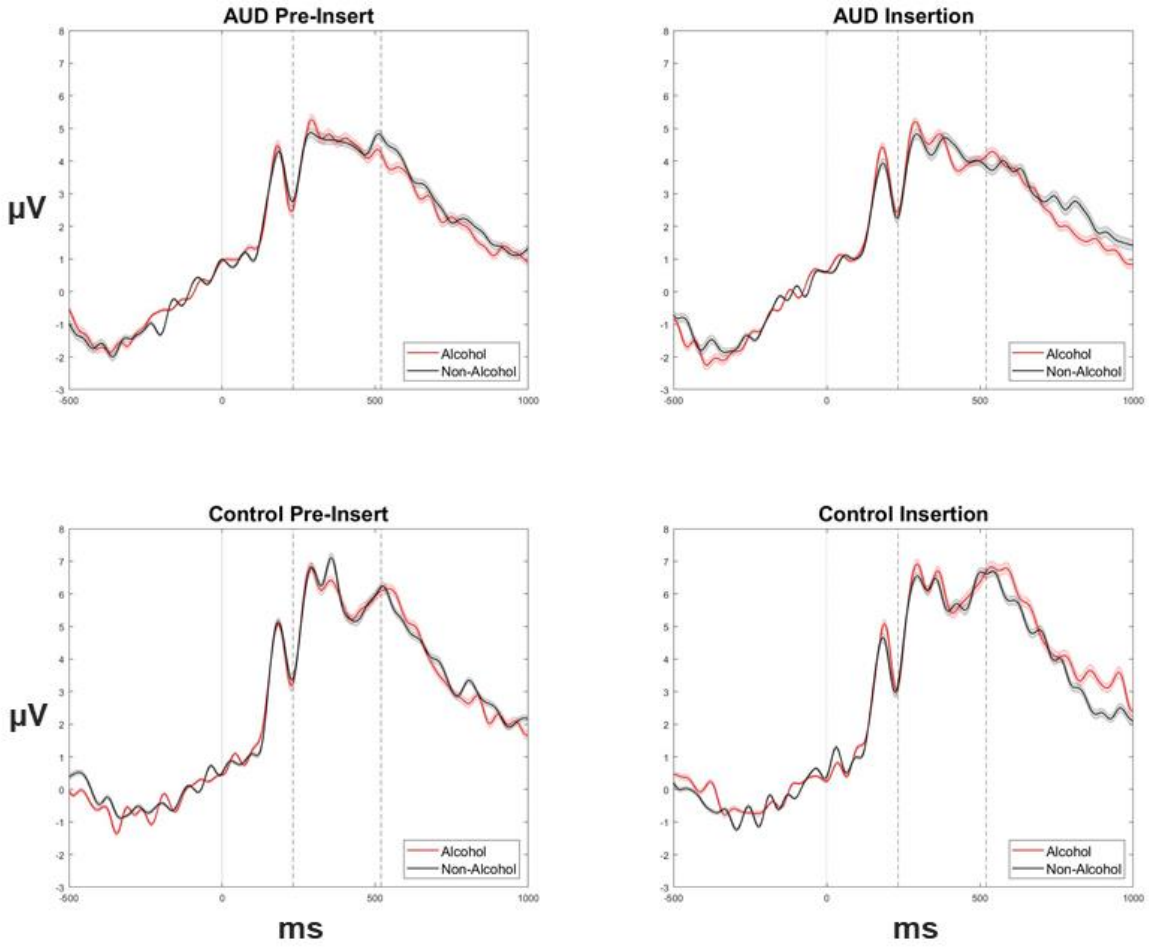
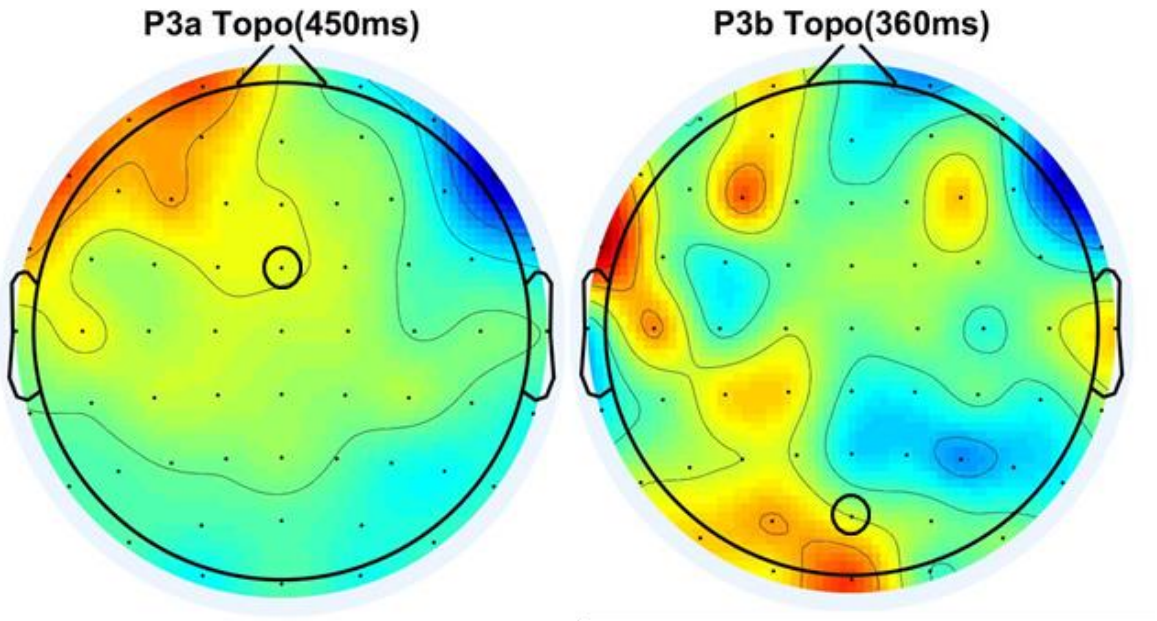


Figure 13

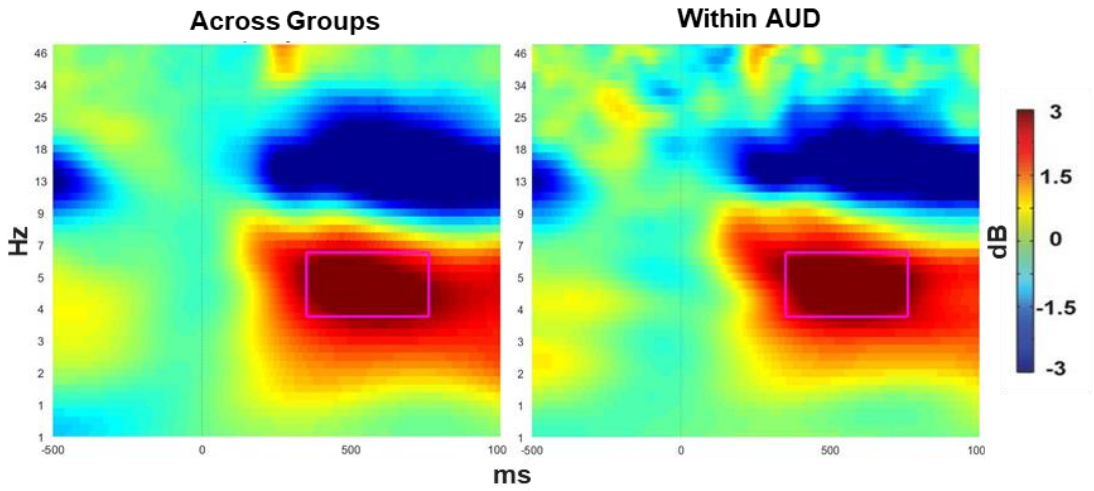
P3a and P3b Topographical Plots



Note. The P3a topographical plot is a contrast of alcohol minus non-alcohol insertion trials at 450ms post-insertion. Electrode FCz is circled. The P3b topographical is a contrast of alcohol minus non-alcohol insertion trials at 360ms post-insertion. Electrode POz is circled.

Figure 14

Cue-Locked TF Region of Interest



Note. TF Plot at electrode FCz. Hz = hertz, ms = milliseconds, dB = decibels

Figure 15

Feedback-Locked ERP Region of Interest

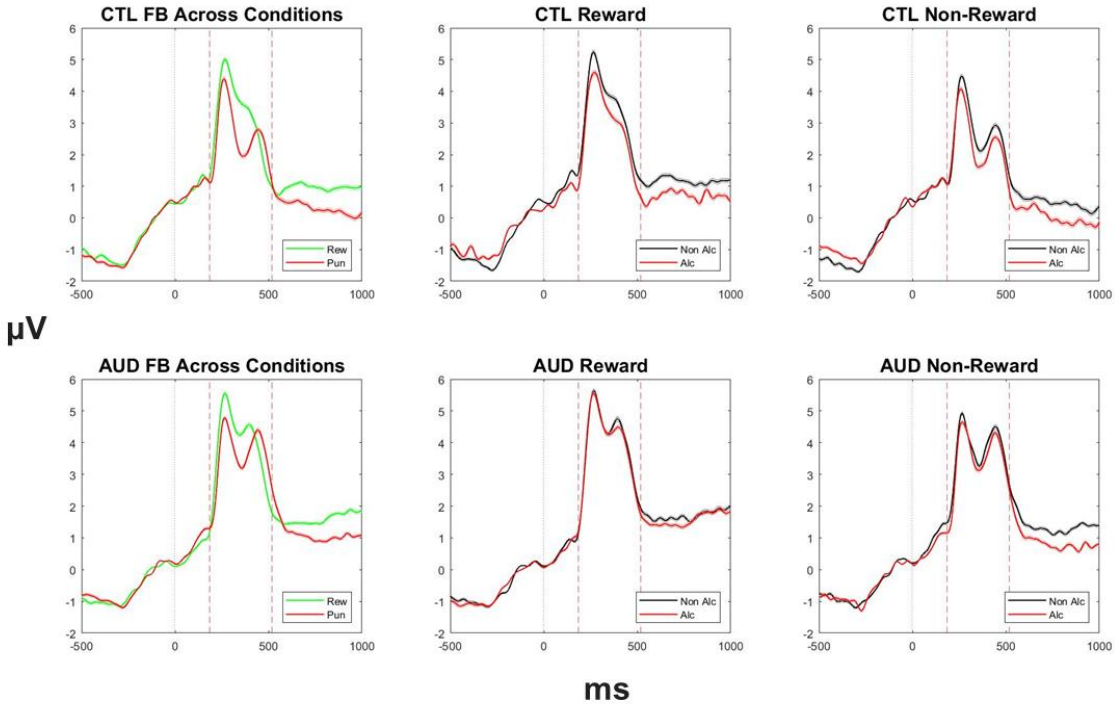
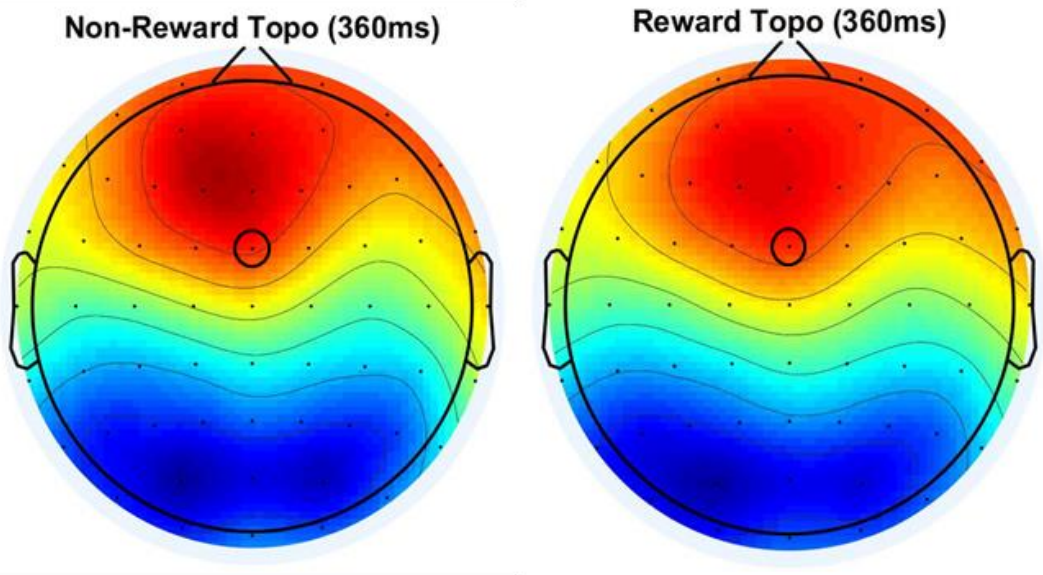


Figure 16

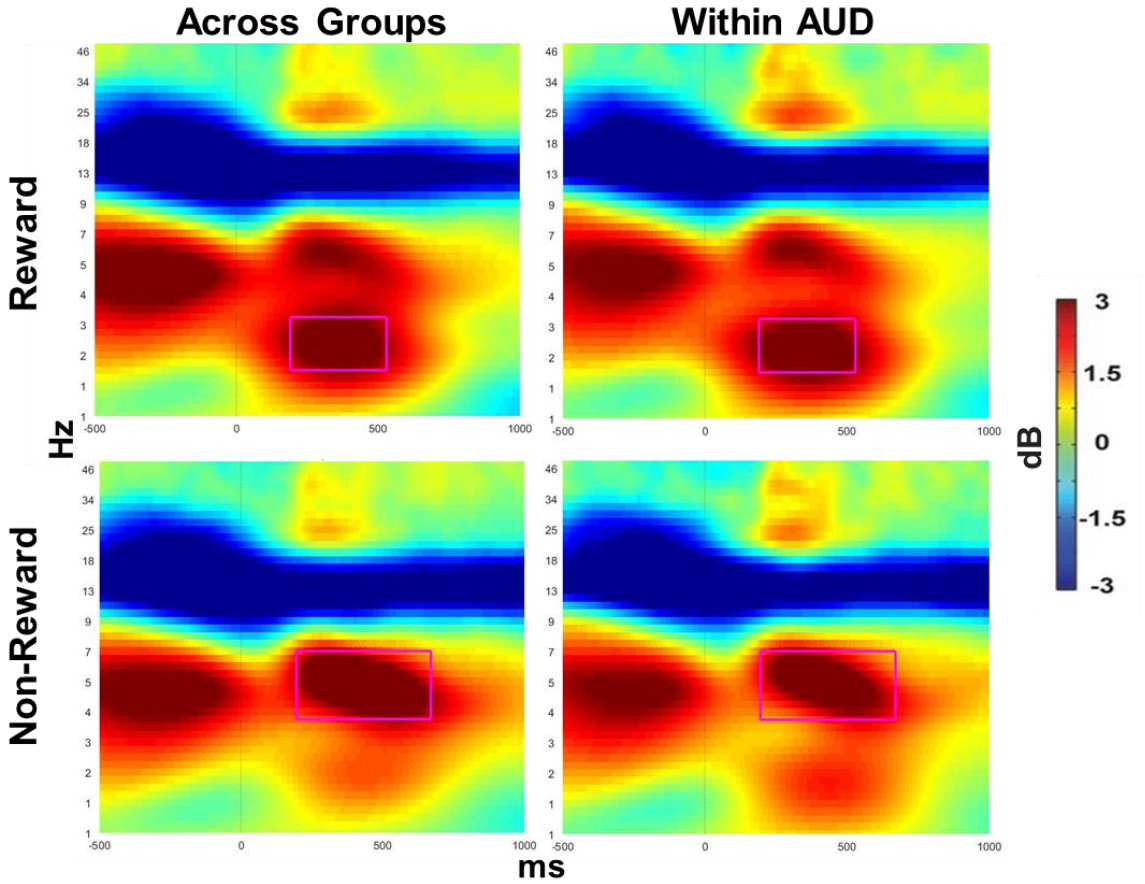
Topographical Plots at Feedback



Note. Both topos taken from the trough of ERP N2 component at 360ms. Electrode FCz circled.

Figure 17

TF Regions of Interest at Feedback



Note. TF Plot at electrode FCz. Hz = hertz, ms = milliseconds, dB = decibels

Table 5

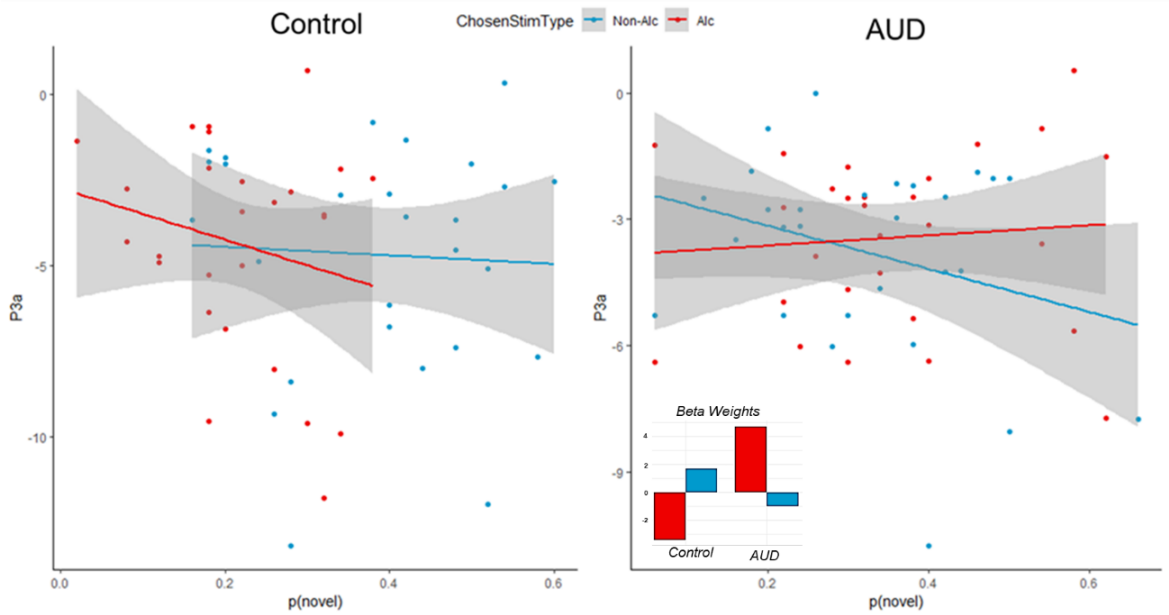
P3a Amplitude by Group or AUDIT, Chosen Cue Type, and Probability of Choosing the Novel Stimulus

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	-4.44	0.662	<.001
	Group	0.519	0.679	0.448
	Cue type	0.204	0.243	0.404
	p(chose novel)	0.747	1.303	0.568
Interactions				
	Group*cue type	**-3.219**	**1.359**	**0.022**
	Cue type*p(chose novel)	-5.106	3.572	0.158
	Group*p(chose novel)	-2.685	3.521	0.448
	Group*cue type*p(chose novel)	**10.81**	**4.306**	**0.015**
Within AUD				
Main effects				
	Intercept	-4.468	0.96	<.001
	AUDIT	0.055	0.068	0.431
	Cue type	0.104	0.303	0.734
	p(chose novel)	0.92	1.952	0.639
Interactions				
	AUDIT*cue type	0.248	0.173	0.166
	Cue type*p(chose novel)	**15.09**	**6.031**	**0.02**
	AUDIT*p(chose novel)	0.411	0.462	0.378
	AUDIT*cue type*p(chose novel)	-0.725	0.449	0.121

Figure 18

*P3a Amplitude by Group*Cue Type*p(novel) Scatter Plot*



Note. Fit lines are schematic and don't reflect model estimates shown as beta weights on the inlaid bar plot

EEG and POMDP Data

P3a amplitude was predicted by a three-way interaction between group, cue type, and IEV (Table 6; beta = -3.792, t = -2.404, p = 0.019). This relationship was driven by the contrast between stimulus types in AUD (beta = 2.52, z = 2.184, p = 0.029), the contrast between groups for non-alcohol stimuli (beta = 0.261, z = 0.219, p = 0.017), and a negative relationship between IEV and P3a amplitude in controls for non-alcohol stimuli (beta = -1.927, p = 0.037) (Figure 19). Across groups, cue-locked frontal theta power scaled positively with the BONUS parameter (Figure 20; Table 7; beta = 0.661, t = 2.122, p = 0.037). Other models stipulated in the introduction without statistically significant findings or non-hypothesized incidental findings can be found in the appendices.

Table 6

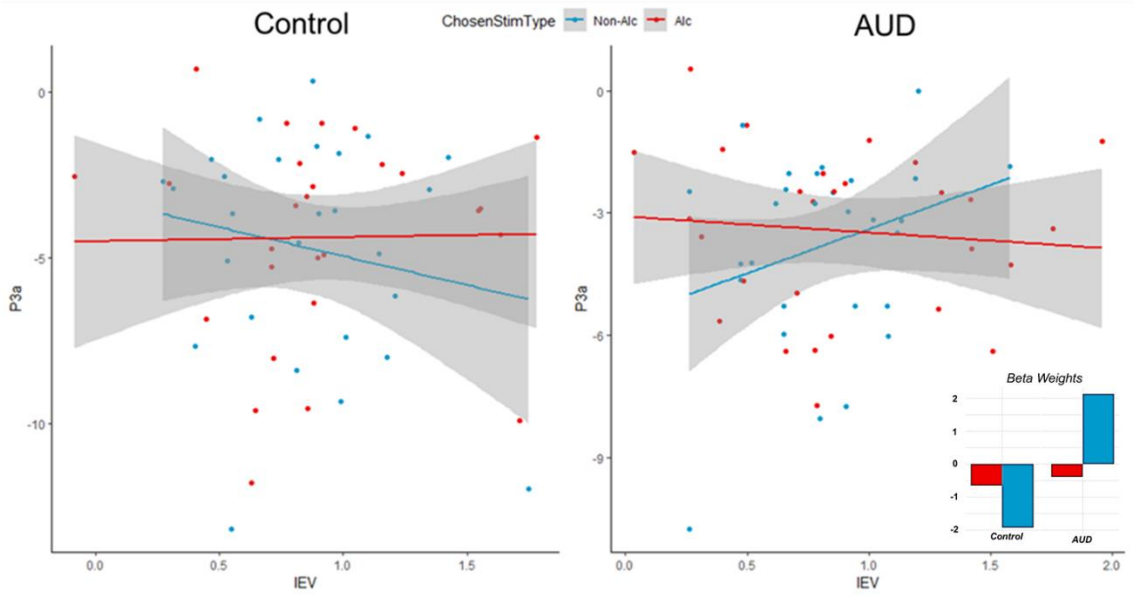
P3a Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	-3.58	0.665	<.001
	Group	0.643	0.704	0.365
	Cue type	0.149	0.216	0.495
	BONUS	-0.276	0.709	0.698
	IEV	-0.864	0.483	0.077
Interactions				
	Group*cue type	**3.645**	**1.405**	**0.012**
	Cue type*BONUS	-0.465	1.402	0.741
	Group*BONUS	-0.753	2.083	0.719
	Cue type*IEV	1.275	1.077	0.241
	Group*IEV	**4.054**	**1.7**	**0.02**
	Group*cue type*BONUS	0.644	2.196	0.771
	Group*cue type*IEV	**-3.792**	**1.577**	**0.019**
Within AUD				
Main effects				
	Intercept	-4.046	0.95	<.001
	AUDIT	0.07	0.07	0.329
	Cue type	0.245	0.321	0.45
	BONUS	-0.956	1.074	0.378
	IEV	-0.495	0.735	0.504
Interactions				
	AUDIT*cue type	0.269	0.33	0.421
	Cue type*BONUS	-2.188	4.323	0.617
	AUDIT*BONUS	-0.021	0.376	0.955
	Cue type*IEV	-0.797	3.365	0.814
	AUDIT*IEV	0.408	0.374	0.281
	AUDIT*cue type*BONUS	0.176	0.356	0.626
	AUDIT*cue type*IEV	-0.243	0.334	0.473

Figure 19

*P3a Amplitude Predicted by IEV*Group*Cue Type Scatter Plot*



Note. Fit lines are schematic and don't reflect model estimates shown as beta weights in inlaid bar plot

Table 7

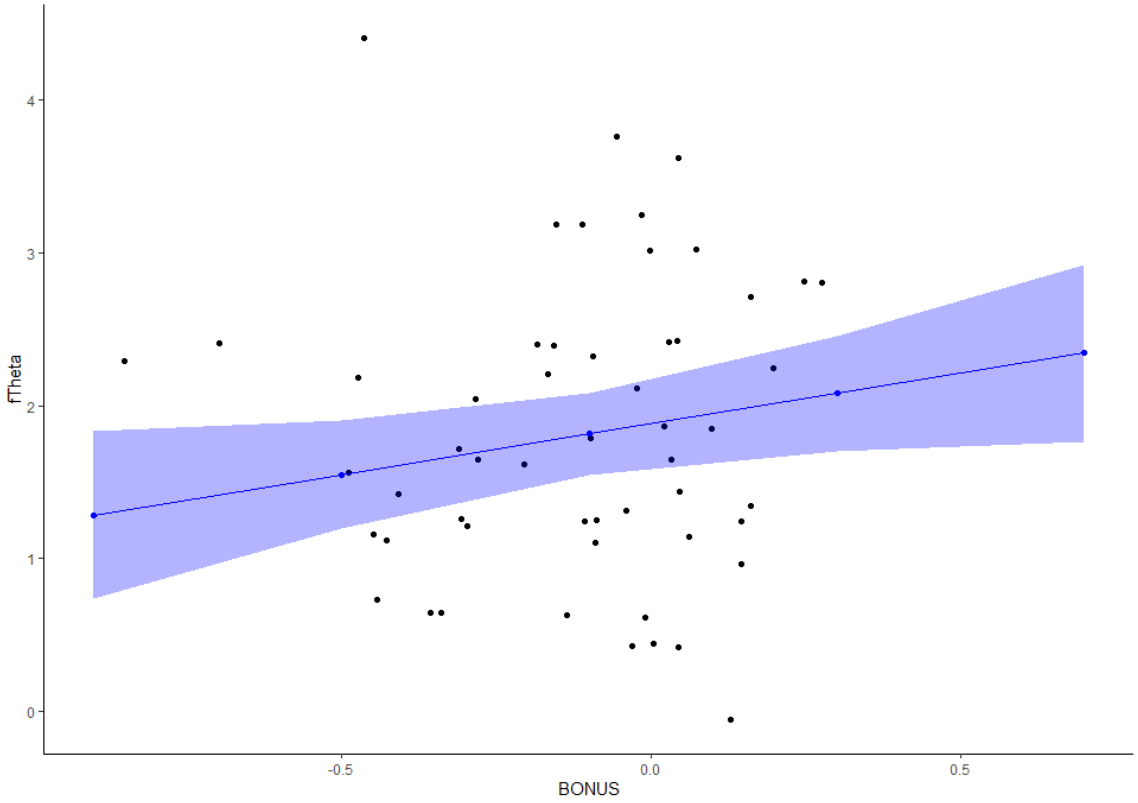
Cue-Locked Frontal Theta Power by Group or AUDIT, Chosen Cue Type, and POMDP

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.919	0.273	<.001
	Group	-0.274	0.274	0.323
	Cue type	-0.165	0.098	0.1
	BONUS	**0.661**	**0.311**	**0.037**
	IEV	0.215	0.212	0.314
Interactions				
	Group*cue type	-0.248	0.619	0.69
	Cue type*BONUS	0.636	0.62	0.31
	Group*BONUS	0.084	0.901	0.926
	Cue type*IEV	-0.246	0.475	0.606
	Group*IEV	-0.522	0.727	0.475
	Group*cue type*BONUS	-0.579	0.971	0.554
	Group*cue type*IEV	0.03	0.693	0.966
Within AUD				
Main effects				
	Intercept	0.775	0.269	0.007
	AUDIT	0.02	0.02	0.32
	Cue type	-0.09	0.093	0.342
	BONUS	-0.047	0.309	0.88
	IEV	0.126	0.211	0.551
Interactions				
	AUDIT*cue type	0.112	0.089	0.222
	Cue type*BONUS	0.313	1.168	0.791
	AUDIT*BONUS	0.024	0.102	0.813
	Cue type*IEV	1.023	0.91	0.272
	AUDIT*IEV	0.155	0.101	0.133
	AUDIT*cue type*BONUS	-0.018	0.096	0.851
	AUDIT*cue type*IEV	-0.116	0.09	0.211

Figure 20

Cue-Locked Frontal Theta by BONUS with Model Fit



Note. Scatter plot with model fit from table 8

DISCUSSION

The present study sought to assess the relationship between explore-exploit behavior in AUD vs. controls and within AUD according to self-reported severity. As hypothesized, group and cue type interacted to predict probability of choosing the novel stimulus. Increased selection of novel alcohol stimuli in AUD demonstrates a bias for exploration of alcohol cues relative to controls. No group or cue effects on selection of the best stimulus suggests that individuals with AUD may over-rely on exploration rather than exploitation of alcohol-related stimuli.

Within AUD, AUDIT scores predicted probability of selecting the best stimulus. This contradicts a previous finding on AUDIT scores and selection of the best stimulus in the novelty bandit (Aloi et al., 2021), though the present study focuses on adults rather than adolescents and did not offer a monetary reward. This relationship may also be due to the appetitive quality of all stimuli on the task i.e. images of beverages, driving their selection in those with more severe AUD symptoms. It also may be related to "stimulus stickiness", which assays choice repetition regardless of reinforcement history (Kanen et al., 2021). This may relate to an incidental finding wherein AUDIT scaled with theta power following non-rewarding feedback (Appendix K).

P3a amplitude did not differ as a function of whether it occurred on pre-insertion, insertion, or post-insertion trials. This may be due to the fact that P3a had a more complex relationship with novel stimuli. Between groups, the hypothesis that P3a amplitude would increase as a function of probability of selecting the novel stimulus in AUD to alcohol stimuli was confirmed. When comparing controls to AUD, exploration of cue types was differentiated in AUD with a positive relationship between the P3a and probability of

selecting the novel stimulus for alcohol cues. No other group-cue type estimate was statistically significant. This finding suggests that the P3a plays a role in executive function during exploration of alcohol cues in AUD, capturing attention and signaling the need for cognitive control. This relates to findings from the fMRI literature wherein alcohol imagery elicits increased vmPFC activation in AUD (Schacht, Anton, & Myrick, 2013) and decision-making relates to altered vmPFC-striatal connectivity (Galandra, Basso, Cappa, & Canessa, 2018).

The BONUS parameter was predicted by an interaction between group and cue type which was driven by the difference between cue exploration within AUD. Like the probability of choosing novel stimuli, BONUS values for alcohol stimuli were higher for AUD than for controls, suggesting that the BONUS parameter accords with measures of exploratory behavior in reflecting group and cue interactions. Although P3a amplitude did not relate to the BONUS parameter as previously hypothesized, P3a amplitude was predicted by an interaction between IEV, group, and cue type as an incidental finding. In AUD relative to controls, IEV scaled positively with P3a amplitude for non-alcohol stimuli. Future analyses will look at the relationship between liking of these same stimuli measured in another task of the experiment to evaluate whether non-alcohol stimuli were particularly well-liked and better correlate with IEV. BONUS did positively predict cue-locked frontal theta power across groups, though this was not explicitly hypothesized and is thus a post-hoc finding. It suggests that cue-locked frontal theta may play a general role in regulating exploratory behavior and accords with previous work on its role in novelty detection (Cavanagh & Frank, 2014) and metacognitive decision-making in the face of subjective uncertainty (Soutschek, Moisa, Ruff, & Tobler, 2021).

Future Directions

Important signal in brain activity and behavior may be lost by averaging across particular trials (Stokes & Spaak, 2016) and trial-level analysis is an important potential future direction in the analysis of explore-exploit behavior. Instead of modeling subject-level POMDP parameters, trial-by-trial estimates could be used to better capture variance which takes place after novel insertions. Additionally, by covarying for trends in stimulus liking, analyses may yield more specific coordination between brain activity and choice behavior. Future iterations of the present study may use neutral cues in addition to non-alcohol beverages to contrast potential liking effects. Follow-up analyses may also model stimulus stickiness to assess the behavioral pattern underlying the relationship between AUDIT scores and probability of selecting the best stimulus (Robbins & Cardinal, 2019).

In addition to cue liking, other sources of data are available for follow-up analysis on the present dataset. In particular, information from the ABQDRINQ study indicates whether or not participants went forward with treatment. This could represent an important individual difference for neurobehavioral characteristics since treatment-seeking status can produce clinically significant effects (Prisciandaro et al., 2016). Additionally, because present AUDIT measurements were taken at one timepoint, they are potentially less sensitive to overall AUD severity during the 12-month period that is stipulated for diagnosis of an AUD. The ABQDRINQ study followed participants for a year prior to their participation in the present study and recorded AUDIT scores at various timepoints. Cumulative AUDIT scores and variability over time could be an improved marker of within AUD severity. Correlations between BDI and years of education represent potential sources of heterogeneity and although including years of education didn’t change the AUDIT-behavior relationship,

follow-up research may need to account for these within-group correlations in more complex analyses.

Conclusion

A clear difference in performance on the novelty bandit task was demonstrated between those with AUD and controls for alcohol vs. non-alcohol stimuli both in terms of raw behavior and POMDP model parameters of behavior. This alone represents an important contribution to our understanding of explore-exploit behavior in addiction by showing the influence of substance-related cues in driving exploration rather than habitual exploitation as has been the primary finding of the literature on explore-exploit process in addiction thus far. Several EEG features were predicted from group, severity, choice behavior, and cue type in ways which accord with prior research and in ways which suggest a more nuanced relationship between brain dynamics in explore-exploit in AUD and the need for follow-up analyses. Further spectral decomposition and phase synchronization analyses of these findings could better explain these neural dynamics and their mechanistic role in altering decision-making in AUD. This study indicates the need for further evaluation of potential EEG biomarkers of AUD which are sensitive to the aberrations in dynamic exploration that characterize the disorder.

APPENDICES

A. P3 Subcomponents by Group or AUDIT and Chosen Cue Type

B. Cue-Locked Frontal Theta by Group or AUDIT and Chosen Cue Type

C. P3b Amplitude by Group or AUDIT, Chosen Cue Type, and Probability of Selecting the Novel Stimulus

D. Cue-Locked Frontal Theta by Group or AUDIT, Chosen Cue Type, and Probability of Selecting the Novel Stimulus

E. Feedback-Locked ERPs by Group or AUDIT and Chosen Cue Type

F. Feedback-Locked TF Power by Group or AUDIT and Chosen Cue Type

G. P3b Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

H. Reward Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

I. Non-Reward Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

J. Reward TF Power by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

K. Non-Reward TF Power by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

APPENDIX A

P3 Subcomponents by Group or AUDIT and Chosen Cue Type

P3a Amplitude by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

Between groups				
Main effects				
	Effect	Estimate	*SE*	*p*
	Intercept	-4.19	0.494	<.001
	Group	0.533	0.674	0.432
	Cue type	0.147	0.225	0.517
Interaction				
	Group*cue type	0.065	0.454	0.886
Within AUD				
Main effects				
	Intercept	-4.286	0.816	<.001
	AUDIT	0.067	0.068	0.3218
	Cue type	0.139	0.282	0.627
Interaction				
	AUDIT*cue type	0.02	0.052	0.705

P3b Amplitude by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

Between groups				
Main effects				
	Effect	Estimate	*SE*	*p*
	Intercept	3.875	0.453	<.001
	Group	-0.882	0.622	0.162
	Cue type	0.056	0.182	0.762
Interaction				
	Group*cue type	-0.156	0.367	0.672
Within AUD				
Main effects				
	Intercept	3.716	0.854	<.001
	AUDIT	-0.069	0.072	0.345
	Cue type	-0.024	0.225	0.915
Interaction				
	AUDIT*cue type	-0.071	0.038	0.075

APPENDIX B

Cue-Locked Frontal Theta by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.902	0.21	<.001
	Group	-0.15	0.286	0.602
	Cue type	-0.105	0.098	0.289
Interaction				
	Group*cue type	-0.075	0.197	0.706
Within AUD				
Main effects				
	Intercept	1.01	0.372	0.011
	AUDIT	0.076	0.269	0.021
	Cue type	-0.134	0.125	0.012
Interaction				
	AUDIT*cue type	-0.028	0.023	0.224

APPENDIX C

P3b Amplitude by Group or AUDIT, Chosen Cue Type, and Probability of Choosing the Novel Stimulus

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	3.755	0.581	<.001
	Group	-0.89	0.624	0.16
	Cue type	0.079	0.198	0.692
	p(chose novel)	0.355	1.082	0.743
Interactions				
	Group*cue type	0.207	1.155	0.859
	Cue type*p(chose novel)	-2.869	3.039	0.349
	Group*p(chose novel)	3.596	3.024	0.238
	Group*cue type*p(chose novel)	-0.7	3.662	0.849
Within AUD				
Main effects				
	Intercept	3.108	0.975	0.003
	AUDIT	-0.08	0.073	0.28
	Cue type	-0.097	0.248	0.7
	p(chose novel)	2.321	1.753	0.192
Interactions				
	AUDIT*cue type	0.009	0.121	0.939
	Cue type*p(chose novel)	-1.299	4.218	0.761
	AUDIT*p(chose novel)	0.123	0.369	0.742
	AUDIT*cue type*p(chose novel)	-0.15	0.314	0.636

APPENDIX D

Cue-Locked Frontal Theta Power by Group or AUDIT, Chosen Cue Type, and Probability of Choosing the Novel Stimulus

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.723	0.281	<.001
	Group	-0.168	0.286	0.56
	Cue type	-0.075	0.105	0.475
	p(chose novel)	0.531	0.56	0.346
Interactions				
	Group*cue type	0.111	0.618	0.858
	Cue type*p(chose novel)	2.592	1.621	0.115
	Group*p(chose novel)	-0.938	1.572	0.553
	Group*cue type*p(chose novel)	-1.834	1.956	0.353
Within AUD				
Main effects				
	Intercept	1.013	0.438	0.027
	AUDIT	0.075	0.032	0.025
	Cue type	-0.134	0.129	0.307
	p(chose novel)	-0.004	0.86	0.996
Interactions				
	AUDIT*cue type	-0.056	0.071	0.433
	Cue type*p(chose novel)	0.44	2.463	0.86
	AUDIT*p(chose novel)	0.153	0.2	0.451
	AUDIT*cue type*p(chose novel)	0.049	0.183	0.79

APPENDIX E

Feedback-Locked ERPs by Group or AUDIT and Chosen Cue Type

Reward Amplitude by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

Between groups

Main effects

	Effect	Estimate	*SE*	*p*
	Intercept	1.928	0.211	<.001
	Group	0.283	0.29	0.334
	Cue type	-0.097	0.087	0.268
Interaction				
	Group*cue type	0.187	0.18	0.305
Within AUD				
Main effects				
	Intercept	2.191	0.427	<.001
	AUDIT	-0.002	0.036	0.96
	Cue type	-0.019	0.095	0.847
Interaction				
	AUDIT*cue type	0.005	0.018	0.771

Non-Reward Amplitude by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

Between groups

Main effects

	Effect	Estimate	*SE*	*p*
	Intercept	1.646	0.236	<.001
	Group	0.509	0.325	0.124
	Cue type	-0.163	0.091	0.078
Interaction				
	Group*cue type	-0.033	0.182	0.855
Within AUD				
Main effects				
	Intercept	1.858	0.47	<.001
	AUDIT	0.029	0.04	0.467
	Cue type	-0.179	0.11	0.116
Interaction				
	AUDIT*cue type	-0.008	0.021	0.712

APPENDIX F

Feedback-Locked TF Power by Group or AUDIT and Chosen Cue Type

Reward TF Power by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.479	0.254	<.001
	Group	0.031	0.352	0.929
	Cue type	0.072	0.073	0.33
Interaction				
	Group*cue type	-0.238	0.142	0.098
Within AUD				
Main effects				
	Intercept	1.465	0.506	0.008
	AUDIT	0.009	0.043	0.84
	Cue type	-0.039	0.087	0.661
Interaction				
	AUDIT*cue type	0.017	0.015	0.273

Non-Reward TF Power by Group or AUDIT and Chosen Cue Type

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.664	0.185	<.001
	Group	-0.329	0.255	0.203
	Cue type	0.055	0.062	0.381
Interaction				
	Group*cue type	-0.089	0.122	0.473
Within AUD				
Main effects				
	Intercept	0.512	0.303	0.103
	AUDIT	**0.082**	**0.025**	**0.003**
	Cue type	0.012	0.079	0.88
Interaction				
	AUDIT*cue type	**0.029**	**0.013**	**0.038**

APPENDIX G

P3b Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	3.69	0.583	<.001
	Group	-0.778	-0.623	0.217
	Cue type	0.004	0.186	0.985
	BONUS	0.088	0.614	0.886
	IEV	0.125	0.419	0.766
Interactions				
	Group*cue type	-1.514	1.141	0.19
	Cue type*BONUS	-0.038	1.137	0.974
	Group*BONUS	1.233	1.704	0.472
	Cue type*IEV	-1.297	0.874	0.144
	Group*IEV	-0.654	1.402	0.642
	Group*cue type*BONUS	-1.93	1.781	0.284
	Group*cue type*IEV	1.397	1.282	0.281
Within AUD				
Main effects				
	Intercept	3.416	0.998	0.002
	AUDIT	-0.074	0.077	0.342
	Cue type	-0.099	0.26	0.705
	BONUS	0.219	0.917	0.812
	IEV	0.482	0.65	0.463
Interactions				
	AUDIT*cue type	0.145	0.219	0.512
	Cue type*BONUS	0.322	2.754	0.908
	AUDIT*BONUS	0.165	0.283	0.561
	Cue type*IEV	2.856	2.206	0.208
	AUDIT*IEV	0.331	0.275	0.236
	AUDIT*cue type*BONUS	-0.112	0.227	0.626
	AUDIT*cue type*IEV	-0.242	0.222	0.286

APPENDIX H

Reward Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	2.02	0.269	<.001
	Group	0.189	0.284	0.51
	Cue type	-0.078	0.087	0.376
	BONUS	0.054	0.287	0.85
	IEV	0.003	0.195	0.987
Interactions				
	Group*cue type	0.749	0.544	0.175
	Cue type*BONUS	**-1.149**	**0.543**	**0.04**
	Group*BONUS	0.132	0.811	0.871
	Cue type*IEV	0.306	0.417	0.466
	Group*IEV	-0.507	0.666	0.448
	Group*cue type*BONUS	1.581	0.85	0.687
	Group*cue type*IEV	-0.342	0.612	0.578
Within AUD				
Main effects				
	Intercept	2.466	0.479	<.001
	AUDIT	-0.001	0.037	0.979
	Cue type	-0.031	0.111	0.783
	BONUS	0.316	0.399	0.433
	IEV	-0.286	0.286	0.323
Interactions				
	AUDIT*cue type	0.019	0.114	0.868
	Cue type*BONUS	-0.74	1.426	0.608
	AUDIT*BONUS	0.007	0.149	0.962
	Cue type*IEV	0.084	1.145	0.942
	AUDIT*IEV	0.065	0.144	0.655
	AUDIT*cue type*BONUS	0.104	0.117	0.386
	AUDIT*cue type*IEV	-0.021	0.115	0.854

APPENDIX I

Non-Reward Amplitude by Group or AUDIT, Chosen Cue Type, and POMDP Parameters

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.587	0.295	<.001
	Group	0.456	0.316	0.155
	Cue type	-0.175	0.093	0.068
	BONUS	-0.151	0.309	0.625
	IEV	0.12	0.21	0.569
Interactions				
	Group*cue type	0.645	0.585	0.276
	Cue type*BONUS	-0.034	0.583	0.953
	Group*BONUS	-0.593	0.874	0.5
	Cue type*IEV	0.807	0.448	0.078
	Group*IEV	0.214	0.719	0.767
	Group*cue type*BONUS	-0.122	0.914	0.895
	Group*cue type*IEV	-0.667	0.658	0.315
Within AUD				
Main effects				
	Intercept	1.741	0.501	0.001
	AUDIT	0.034	0.039	0.389
	Cue type	-0.113	0.13	0.393
	BONUS	-0.581	0.46	0.215
	IEV	-0.015	0.326	0.962
Interactions				
	AUDIT*cue type	-0.023	0.134	0.867
	Cue type*BONUS	1.19	1.7	0.491
	AUDIT*BONUS	0.132	0.168	0.437
	Cue type*IEV	0.195	1.351	0.887
	AUDIT*IEV	-0.079	0.165	0.632
	AUDIT*cue type*BONUS	-0.115	0.14	0.423
	AUDIT*cue type*IEV	0.018	0.136	0.896

APPENDIX J

Reward TF Power by Group or AUDIT, Chosen Cue Type, and POMDP

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.491	0.292	<.001
	Group	-0.018	0.347	0.958
	Cue type	0.062	0.071	0.389
	BONUS	-0.257	0.248	0.303
	IEV	0.034	0.169	0.839
Interactions				
	Group*cue type	0.133	0.418	0.751
	Cue type*BONUS	0.122	0.413	0.769
	Group*BONUS	0.047	0.644	0.943
	Cue type*IEV	0.306	0.319	0.342
	Group*IEV	-0.069	0.552	0.901
	Group*cue type*BONUS	-0.883	0.647	0.179
	Group*cue type*IEV	-0.402	0.473	0.399
Within AUD				
Main effects				
	Intercept	1.462	0.545	0.012
	AUDIT	0.016	0.04	0.722
	Cue type	0.034	0.102	0.742
	BONUS	-0.495	0.374	0.195
	IEV	-0.149	0.273	0.588
Interactions				
	AUDIT*cue type	0.051	0.087	0.56
	Cue type*BONUS	-0.728	1.045	0.495
	AUDIT*BONUS	0.001	0.123	0.992
	Cue type*IEV	-0.046	0.862	0.958
	AUDIT*IEV	0.074	0.116	0.532
	AUDIT*cue type*BONUS	-0.025	0.086	0.775
	AUDIT*cue type*IEV	-0.023	0.088	0.795

APPENDIX K

Non-Reward TF Power by Group or AUDIT, Chosen Cue Type, and POMDP

Main effects and interactions modelled separately

	Effect	Estimate	*SE*	*p*
Between groups				
Main effects				
	Intercept	1.7	0.222	<.001
	Group	-0.387	0.25	0.128
	Cue type	0.037	0.063	0.557
	BONUS	0.044	0.214	0.837
	IEV	0.044	0.147	0.765
Interactions				
	Group*cue type	-0.333	0.366	0.366
	Cue type*BONUS	-0.32	0.362	0.381
	Group*BONUS	0.516	0.558	0.359
	Cue type*IEV	-0.251	0.279	0.373
	Group*IEV	0.165	0.471	0.723
	Group*cue type*BONUS	0.719	0.568	0.212
	Group*cue type*IEV	0.32	0.412	0.442
Within AUD				
Main effects				
	Intercept	0.457	0.353	0.204
	AUDIT	**0.074**	**0.028**	**0.011**
	Cue type	-0.067	0.087	0.451
	BONUS	0.527	0.31	0.099
	IEV	0.257	0.221	0.252
Interactions				
	AUDIT*cue type	0.002	0.086	0.984
	Cue type*BONUS	-0.066	1.081	0.952
	AUDIT*BONUS	0.024	0.111	0.832
	Cue type*IEV	-0.298	0.866	0.812
	AUDIT*IEV	-0.035	0.108	0.749
	AUDIT*cue type*BONUS	0.019	0.089	0.829
	AUDIT*cue type*IEV	0.023	0.087	0.783

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