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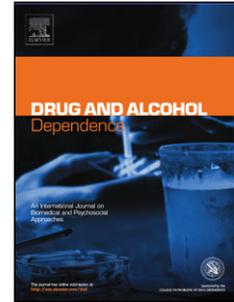
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Aspects of alcohol use disorder affecting social cognition as assessed using the Mini Social and Emotional Assessment (mini-SEA)

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Highlights

- Alcohol use disordered (AUD) individuals perform poorly in social processing tasks.

- Years of drinking is single strongest predictor of problems reading social situations.

- Emphasizes need for early intervention to stop or reduce drinking in AUD.

- Relevance to clinical/treatment challenges of working with AUD individuals.

Abstract

Background: Alcohol Use Disorder (AUD) is associated with problems with processing complex social scenarios. Little is known about the relationship between distinct AUD-related factors (e.g., years of problematic drinking), aspects of cognitive function and dysfunction in individuals diagnosed with AUD, and the relative impact these may have on social cognition.

Aims: To explore differences in social cognition between a group of participants diagnosed with AUD and controls, using a clinical measure, the Mini Social and Emotional Assessment (mini-SEA). The mini-SEA was used to evaluate social and emotional understanding through a facial emotional recognition task and by utilising a series of social scenes some of which contain a faux pas (social error).

Methods: Eighty-four participants (individuals with AUD and controls) completed demographic and a general cognitive and social cognitive test battery over three consecutive days.

Results: Between group analyses revealed that the participants with AUD performed less well on the faux pas test, and differences were also revealed in the emotional facial recognition task. Years of problematic alcohol consumption was the strongest predictor of poor ToM reasoning.

Conclusion: These results suggest a strong link between AUD chronicity and social cognition, though the direction of this relationship needs further elucidation. This may be of clinical relevance to abstinence and relapse management, as basic social cognitive skills and ability to maintain interpersonal relationships are likely to be crucial to recovery.

1. Introduction

Alcohol Use Disorder (AUD) is described as a chronic relapsing condition with definitive behavioural markers (Diagnostic and Statistics Manual, 2013). Recognised clinically as affecting decision making, relationships, and, in severe cases, neurological function, the severity of clinical presentation is associated with poorer treatment outcomes (Booth et al., 1991; Boschloo et al., 2012). Of particular relevance to rates of long-term abstinence and relapse are social skills, social support, and interpersonal relationships (Kornreich et al., 2001). Problems with emotional understanding, empathy, apathy, and social inhibition may all reflect the cumulative neurotoxic effects of abusive drinking patterns impacted by a confluence of psychiatric comorbidity, lifestyle circumstances, and poly-drug use (Foisy et al., 2005; Kornreich et al., 2001; Oscar-Berman and Marinkovic, 2007).

A growing number of social processing paradigms have been developed which show that AUD is associated with errors in the decoding of other's emotional expressions (Clark et al., 2007; Kornreich et al., 2013; Maurage et al., 2008; Philippot et al., 1999) and differences in automatic perspective taking (Cox et al., 2016) in clinical cohorts following detoxification. Problems are also evidenced in more complex social processing tasks such as humour processing and the detection of irony (Amenta et al., 2013; Uekermann et al., 2007). Similarly, a growing body of work reports that theory of mind (ToM), the ability to infer what others think, believe, know or feel, is also impaired in AUD (Bosco et al., 2014; Maurage et al., 2016; Thoma et al., 2013). In particular, affective aspects of ToM, such as the ability to decode others' feelings (Maurage et al., 2016) and empathy, the ability to experience other's feelings, appear specifically impaired (Bosco et al., 2014; Dethier and Blairy, 2012; Thoma et al., 2013).

AUD-related brain pathology and the impact on core cognitive functioning (Oscar-Berman and Marinković, 2007) is likely to significantly limit normal psychological

processing and engagement in the social world, including help-seeking and responsivity to support. However, more specific assessment of the impact of AUD on social cognition is somewhat lacking. Social cognition deficits could be related to the extensive and often transient range of neurological deficits caused by distinct AUD-related factors such as years of illness (alcoholism), alcohol craving, and units consumed (Maurage et al., 2015). These factors, in addition to the age of onset and lifestyle, could have a critical influence on cognitive difficulties, but their specific relationships with social cognitive performance remain poorly investigated. Between 50% and 80% of problem drinkers show evidence of cognitive impairments (Wadd et al., 2013), and such impairments, particularly social cognition deficits, have a negative effect on rates of recovery (Kornreich et al., 2001).

Social cognition deficits are not routinely screened for in AUD. Issues with emotional communication (Kornreich et al., 1992; Monti et al., 1990), negative affect (Marlatt, 1979), and empathy (Bosco et al., 2014) may relate to poor engagement in treatment, drop-out, and relapse (Hunter-Reel et al., 2009). Particularly important is the ability and willingness to experience empathy and understanding in treatment. Both of these are linked to prosocial behaviour, and their absence is related to hostility (Marshall and Marshall, 2011). While researchers continue to document social cognition problems in participants with AUD, very little research has investigated which social cognitive processes are affected. This raises the question of whether poor social cognition is related to or predicted by general poor cognitive functioning (e.g., executive functions) or to AUD related behaviours (e.g., years of drinking, average units consumed and age started drinking problematically).

The current study employed the Mini Social Cognition and Emotional Assessment task (mini-SEA; Bertoux et al., 2012), a clinical measure of social cognition used widely with dementia patients, to explore its clinical utility in highlighting differences between an AUD cohort and a comparable adult control group. The mini-SEA evaluates ToM by testing a

participant's capacity to detect, explain, and make inferences about intentions, belief and feelings of other's. Thoma et al. (2013) have used a similar method to highlight differences between healthy controls and alcohol dependent participants, with the latter showing reduced faux pas scores as evidenced by poor faux pas understanding and empathy scores. The method used here, though, differs: the mini-SEA is significantly briefer, making it ideal for clinical application. In addition, Thoma et al. (2013) only partially delineated some of the different sub-components of ToM and did not explore all dimensions measured by the faux pas procedure. Because ToM is not a monolithic function but a multi-faceted complex process, we delineated ToM into several dimensions (detection, identification, understanding/knowledge of faux pas, attribution of intention, attribution of belief, and empathy) in order to better understand it and its interaction with other cognitive and AUD variables. Past research has shown that while alcohol dependence can significantly impact some social processing skills e.g., decoding of negative emotional faces, while other skills e.g., decoding positive emotional faces, remain spared (Kornreich et al., 2013).

A further difference between the current study and Thoma's are the clinical considerations of this task. In order to reduce the possible confounding factors of problems with working memory and language/semantic deficits, this version of the mini-SEA provides visual contextual information. Though widely used in stroke, dementia, and traumatic brain injury, this is the first time this task has been used in the field of substance misuse.

A final difference to note is that in the emotional facial decoding task the mini-SEA uses full emotional facial expressions. Thoma et al. presented participants with eye regions only. It may be argued that this method lacks ecological validity, as it is artificial compared to everyday processing of emotional expressions.

The aims of the current study are (1) to deconstruct the ToM subcomponents and examine the extent to which these scores were predicted by cognitive ability and AUD-

related behaviours and (2) to explore the clinical utility of the mini-SEA to assess social cognition in AUD compared with age/gender matched control group. To the authors' knowledge, this is the first study to investigate social cognition in AUD using the mini-SEA. Our hypotheses are that (1) individuals with AUD have clear deficits in social processing, aspects of ToM, and emotion recognition compared to age- and education-matched controls, and (2) AUD-related behaviours and general cognitive functioning both significantly affect social cognition abilities in participants with AUD.

2. Methods

2.1 Participants

Ethical approval was granted by London Metropolitan University (where the work was carried out). All participants provided written informed consent. Individuals with AUD were assured that taking part was voluntary and did not form part of their treatment. Participants with AUD were recruited from a set of provincial outpatient service centres in the UK. All clinical participants met the DSM-V (2013) criteria for AUD as assessed by a qualified health practitioner. All patients were required to be alcohol-free at the time of visiting the respective centre for therapy (as measured by breathalyser tests). In total 45 participants completed the test battery and all self-reported at least 3 weeks of abstinence (see Table 1 for demographics).

Participants were excluded if they reported current or former poly-drug use or if there was any history of neurological impairment or current psychiatric and mental health diagnosis (this was assessed by the lead Psychiatrist and available medical records). Participants were excluded if they were currently being prescribed medication for assisted detoxification. Table 1 presents data on recent and historical detoxification (for historical, specific timelines could not be recalled).

Forty control participants were drawn from a larger sample of non-clinical staff and

students from the University and the treatment centre and matched (by age and gender only) to the participants in the AUD cohort. Controls reported no history of alcohol or other drug abuse, though all but one participant consumed alcohol on a weekly or monthly basis (see Table 1). Two participants reported being prescribed selective serotonin reuptake inhibitors (SSRIs) for depressive illness in the past (>3 months). Smoking was more common in the AUD group (though given several temporarily sustained quit attempts, very few participants with AUD could estimate number of years using tobacco), but participants with AUD reported smoking fewer cigarettes per day than the control group. Participants with AUD scored higher on the measures of anxiety and depression and for units of alcohol consumed (UC) per week (currently for the controls and prior to treatment for participants with AUD).

2.2 Assessment of AUD-related behaviours

AUD-related behaviours were measured by a self-report questionnaire and through clinical assessment data collected by the treatment centres. Participants were asked to indicate the average number of units of alcohol consumed per week prior to treatment (UC), years of problem drinking (YoD) (measured from time alcohol drinking behaviour had been highlighted by a medical professional), self-reported age at which alcohol use became a problem (AoPD), and the estimated age started drinking alcohol (ASD). We also sought to examine how many years of treatment participants had received, but very few participants could report a clear indication of this. The study also captured data on total/lifetime previous clinical detoxifications (a factor which may affect cognition; Duka et al., 2003). Only 5 participants could provide accurate information, and therefore this was not included in the main analysis. Craving was measured using the Alcohol Craving Questionnaire (ACQ Short-form-revised; Singleton et al., 1994). Although not exclusively related to AUD, assessments for clinical depression using the BDI (Beck, 1961) and clinical levels of anxiety (STAI; Speilberger, 1983) were included given the high comorbidity rating between these diagnoses

and AUD and the well-documented relationship between these two clinical conditions and the processing of emotional stimuli (Driessen et al., 2001).

2.2.1 Cognitive assessment. Participants completed a general cognitive assessment test battery. The colour naming Stroop task (ST) (Stroop, 1935) (50 congruent/50 incongruent randomised trials) was included as a measure of response inhibition, with scores calculated by subtracting the number of accurate congruent trials from incongruent trials. The following sub-tests from the Weschler Adults Intelligence Scale (WAIS-IV: Weschler, 2008) were also administered: Similarities (SIM) and Vocabulary (VB) (classical measures of Verbal comprehension), Sequencing (SQ) and Block Design (BD) (measures of Perceptual Reasoning), and Digit Span Forwards (DSF) and backwards (DSB) (assessing working memory).

2.2.2 Social cognition assessment. The mini-SEA (Bertoux et al., 2012) is a clinically validated test (see Bertoux, 2014) to assess social cognition. The mini-SEA relies on two well-validated tests, the faux pas test (Stone et al., 1998) and the Ekman's faces test (Ekman et al., 1977), that have been translated and validated in many languages— including English, the language in which both tests were created and originally validated— and in many different clinical and non-clinical populations (including severe depression and dementias; Bertoux et al, 2012)

The mini-SEA allows the computation of two scores (ToM and emotion recognition) and a general composite score. The modified and reduced version of the faux pas test is composed of 10 short verbal and visual stories (plus one example) presenting a social interaction with 2 or more characters; 5 of these stories contain a social faux pas committed by one character and 5 were control stories (without any faux pas). ToM is evaluated by testing a participant's ability to detect and explain faux pas as well as to make inferences about a character's intentions, beliefs and feelings. Thus, the task offers a detailed insight into

mental state reasoning through the division of 6 separate ToM sub-scores (detection (DET), identification (ID), knowledge of faux pas (KNOW), attribution of intention (INT), attribution of belief (BEL), and empathy (EMP)) and two control questions assessing general comprehension of the story (see Bertoux et al., 2012, for further details). The current study used the latest (2014) version of the mini-SEA which is supplemented with visual aids which aim to alleviate the working memory.

The second subtest of the mini-SEA is a facial emotion recognition test which requires participants to identify emotional expressions depicted in a series of photographs. It comprises 35 faces selected from the larger emotion face set developed by Ekman. The participant can choose between 6 emotions for each face (happiness, surprise, sadness, fear, disgust and anger) or a neutral expression. Each was presented 5 times for Caucasian male and female faces.

2.3 Procedure

Participants completed the test battery over 3 consecutive days, and the maximum testing time in any one day was 1 hour. Testing was counterbalanced over the 3 days. There were some differences in smoking behaviour between the two groups (see table 1), but smokers were not asked to abstain before testing periods to maintain relative ecological validity, and because nicotine withdrawal may negatively impact on testing (mood, irritability) and be a disincentive for participants taking part.

3. Results

3.1 Data analysis

To ascertain differences between the control and the participants with AUD on the mini-SEA, an independent samples t-test was run using the mini-SEA composite score as the dependent variable.

To highlight any specific social cognition differences that may exist between the

groups, two separate MANCOVAs (controlling for STAI and BDI scores) were conducted: 1) Group x ToM sub-scores (DET, ID, KNOW, INT, BEL, EMP) and 2) group x emotional recognition (neutral, happiness, surprise, sadness, anger, fear and disgust).

After observing group differences on the test battery, separate multiple linear regression analyses were conducted for participants with AUD *only,* in order to explore which AUD-related behaviours and cognitive factors were the best predictors of ToM sub-scores.

3.1.1 Between Subject Effects. There was a significant difference between the control and participants with AUD on the mini-SEA composite score $t(83) = 6.62$, $p<.001$ CI (5.58 – 10.37).

STAI and BDI scores negatively correlated with facial emotional recognition scores for the AUD group only ($p=.021$ and $p <.001$ respectively). Only depression scores within the AUD (and not the control group) significantly and negatively correlated with faux pas sub-scores ($p<.001$). Therefore, the BDI and STAI scores formed covariates in the MANCOVA.

Table 2 presents the MANCOVA for ToM sub-scores (derived from the faux pas task). There were significant group differences for all of the ToM sub-scores. For the facial emotion recognition task, there was a significant overall difference in performance between the AUD and control groups, $F (7, 75) = 4.26$, $p = .001$ $\eta_p^2 .285,$ driven by poorer accuracy in the individuals with AUD for recognition of fear, disgust, anger, and for the neutral emotion condition (scores by emotional valence are shown in Figure 1). Bonferroni adjustments were employed for these multiple comparisons.

3.1.2 Regression analysis. Table 3 presents the results of multiple linear regressions analyses. Given the high smoking prevalence in our AUD group, cigarettes per day was included as an AUD-related factor. In relation to specific ToM sub-scores, overall YoD was the strongest predictive factor of ToM variance. YoD was negatively correlated with

detection and knowledge. Belief was not predicted by any cognitive or AUD-related factor. However, intention was predicted by both anxiety (STAI) and YoD, and performance on the empathy subscale was negatively predicted by both YoD and the Stroop task (response inhibition).

4. Discussion

This study utilized a clinically validated measure, the mini-SEA, to assess social cognition in individuals with AUD compared to an age and gender matched control group. Additionally, the study aimed to deconstruct ToM subcomponents from the mini-SEA and examine the extent to which these scores were predicted by cognitive ability and AUD-related behaviors. Firstly, the data supported the prediction that AUD is strongly associated with social processing differences compared to a control group and demonstrated that the AUD cohort showed a greater number of errors in the ToM subcomponents of the mini-SEA measure and facial emotional recognition compared to controls.

As hypothesized, participants in the AUD group performed poorly on the faux pas task compared to controls, confirming earlier results by Thoma et al. (2013). Given the dominance of YoD in predicting/influencing poor performance in the range of tasks, this result supports the need for early intervention (i.e., opportunistic, educational-based and in primary care settings). Earlier detection, intervention, and treatment of alcohol related problems will likely lessen the social, psychological and both transient and long-term neurological impact of alcohol use.

Secondly, the specific nature of deficits and their relationship to AUD-related factors and cognitive skills in the AUD group were explored by deconstructing the faux pas task into ToM sub-scores. Linear regression, aiming to specifically investigate the influence of the cognitive and AUD variables to each of the ToM dimensions, showed that YoD was the most significant predictor of ToM impairments and negatively impacted almost every dimension of

ToM. These findings may be relevant to understanding problems with everyday living, specifically in the formation, management, and maintenance of interpersonal relationships (Hunter-Reel et al., 2009; Wadd et al., 2013). While no data was collected relating directly to perceived motivations for alcohol use in the participants with AUD, many psychoactive substances, including alcohol, are very effective both in blunting one's own emotions and the ability to detect/perceive emotions in others (Khantzian, 2003). Furthermore, awareness of these impairments may be stressful to the individual. Thus, years of problematic alcohol use coupled with other emotional difficulties may well perpetuate future drinking through the desire to resolve or manage interpersonal problems.

For specific ToM sub-components and YoD, intention was also predicted by anxiety, which is consistent with a large body of evidence showing anxiety disorders are associated with differences in the processing of emotion and an association of threat and fear for future events (e.g., Mathews and MacLeod, 2005). Empathy was also predicted by another variable: the response inhibition score from the colour-naming Stroop task. Such a finding is consistent with the view that to infer what others feel, it is necessary to inhibit one's own perspective (Le Bouc et al., 2012; see Augustinova and Ferrand, 2014, for a critical review of this task). This is a common view in the field of social neurosciences and is compatible with recent cognitive models of ToM postulating that the representation of other's mental states is the result of an interaction between low-level (e.g., gaze direction processing) and high-level processing (e.g., executive functions) (Stone and Gerrans, 2006). However, it is interesting to note that attribution of intention and attribution of knowledge scores were not predicted by the response inhibition score, though both appear to require inhibiting one's own perspective. An alternative explanation is that, although responses to the other questions are largely binary (requiring yes/no answers), the empathy question (asking about how a protagonist/character felt) is open to a wider set of both scenario-appropriate and inappropriate choices. Problems

with impulse control, inhibition, and other cognitive domains might therefore make this final question more difficult and open to error in the AUD cohort. Overall, more data relating to how ToM and other cognitive function are related in populations where there is neurological dysfunction is needed (e.g., Bertoux et al., 2015), and these factors should be given consideration in treatment planning.

Participants in the AUD group also, as expected, showed errors in recognising emotional facial expressions such as fear, anger, disgust, as well as neutral expressions. This is in line with previous findings by Philippot et al. (1999) and Clark et al. (2007), thus adding more evidence that AUD is associated with poorer recognition of negative facial stimuli. Whether such impairments in facial recognition predate the onset of alcohol addiction remain unclear.

The mini-SEA and its use of social scenarios, and in particular the narrative responses of AUD participants in this study, highlight additional potential clinical utility for this measure. The scenarios invite qualitative exploration of a client's beliefs, knowledge, and understanding of intentions and empathy, and they may well give insight into current distress or change. Detailed qualitative analysis was beyond the initial scope and aims of this study, but a cursory examination of responses highlighted various aspects of the lived experiences, biases, and mental state of the participants with AUD taking part in this project. This examination forms the basis of additional qualitative analysis currently in progress (Cox et al., in preparation). However, use of this measure in a clinical setting or to support clinical evaluation outside of a traditional healthcare setting could afford practitioners both a quick and engaging method to assess social functioning in AUD (and indeed other clinical populations such as stroke, traumatic brain injury, developmental disorders, and dementia) and provide a more nuanced narrative dataset which may highlight additional aspects of wellbeing and general psychosocial functioning.

This study naturally has a number of limitations and raises broader questions. The data presented here— as with many other studies in this field— is only cross-sectional, and a longitudinal design would be needed to confirm with more accuracy the significance of key factors (such as YoD), particularly in reference to causation and how social cognition impacts future drinking. Furthermore, our sample size is small given the number of competing factors within the analysis. The Stroop task is considered to measure more than response inhibition alone (see Augustinova and Ferrand, 2014), and future studies should consider finer tasks (e.g., the attentional networking task). The current work also looks at a particular subcategory of people with an AUD diagnosis and excludes those with affective problems, other mental health issues, and other substance use. Whilst this allows exploration of a possibly less confounded AUD effect, it could be argued that this data lacks wider applicability to AUD populations more generally. Future studies could explore these more complex samples to see the extent to which the alcohol variables identified remain a part of the core pathology.

The current findings may also point to the need to contextualise social cognition more widely, not just in the understanding of the nature of problems associated with AUD but within the context of recovery. Abstinence from alcohol is a difficult process for those who have experienced problems with AUD, and high rates of relapse are a testament to this (Moos and Moos, 2006). Social support and the ability to maintain interpersonal relationships are crucial to recovery, and thus they are especially relevant in group-based treatment settings which are often based and developed on the premise of experiential learning (i.e., 12-step models). Thus, as also recommended by Thoma et al. (2013), treatments which work directly with service users to develop their capacity for understanding their own and others' mindsets, emotions, and actions are needed, especially in relation to understanding how social experiences may underpin and perpetuate future drinking.

In sum, the current results suggest that deficits in social and more generalized

cognitive functioning contribute to YoD and enhance the volume of alcohol consumption, and so YoD appears to be a clear indicator of the need for treatment to be received as early as possible and sustained long-term. In this context, although more data are needed to confirm this conclusion and to redress the relative poverty of work looking at social cognition in AUD, the mini-SEA may represent a quick and effective tool for identifying problems with social cognition in groups with AUD.

Author Disclosures

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Contributors

The authors all contributed significantly to different aspects of the research preparation, data collection and the writing of paper (as specified in the contributions section at the end of the manuscript). All authors approved of the final version of the manuscript.

Conflict of Interest

The authors have no conflicts of interest to declare.

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Figure Legends

Figure 1. Mean scores on the emotional recognition task for both participants with AUD, and the control group participants. *p<.05, ** p<.001.
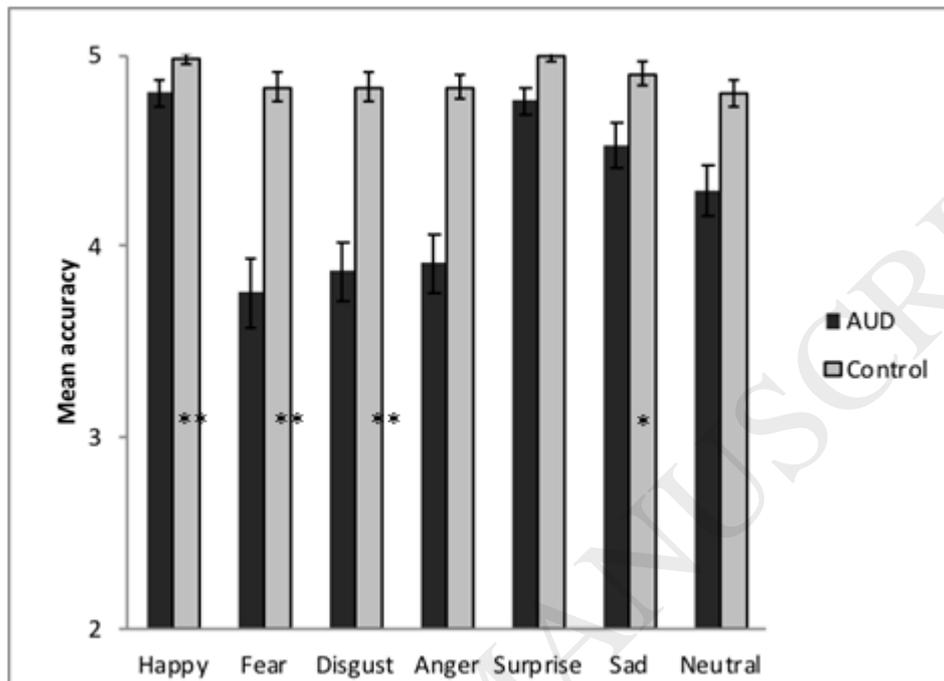
Table 1: Participants' demographics and AUD characteristics. Results given as means and

	Controls (N=40)	AUD (N= 45)
Age	40.23 (11.99)	40.43 (12.56)
Gender	25 males 15 female	29 male 16 female
Years of education	14.8 (0.56)	14.2 (1.20)
Professional background	Manual = 16 Professional = 10 Home maker = 8 Unemployed = 2 Student = 4	Manual = 11 Professional = 11 Home maker = 8 Unemployed = 15 Student = 0
Smoking status **Tobacco** **E-cigarettes** **Cigarettes per day**	$N = 4$ $N = 1$ 20.4 (2.30)	$N= 18$ 17.02 (1.97)
Estimated age started drinking alcohol (ASD)	15.23 (2.18)	14.90 (1.79)
Age started drinking problematically (AoPD)	-	32.31 (4.27)
Years of problematic drinking (YoD)	-	8.23 (8.25)
Units consumed per week (UC) **(prior to treatment for the AUD participants)**	12.10 (3.66)	51.69 (46.84)**
Detox history **Unspecified Benzodiazepine (3+ weeks)** **Detox (previous 12 months)** **Detox (>1 in lifetime)**	- - -	$N = 4$ $N= 9$ $N= 5$
Depression (BDI)	15.10 (9.88)	24.38 (17.06)**
Anxiety (STAI)	27.45 (3.41)	33.24 (8.08)**

standard deviations indicated in parentheses. *P*-values have been adjusted for multiple comparisons. BDI (Beck Depression Inventory. STAI (State-Trait Anxiety Inventory).

** Significant at $p < 0.001$.

Table 2: Between subject multi- and univariate analysis effects for the ToM sub-scores derived from

Task	Multivariate Effects					Univariate Effects				
	Variable	F	df	p	η_p^2	Dependent Variable	F	df	p	η_p^2
ToM sub-scores	Group (AUD, Control)	3.67	1,81	<.001	.193	Detection	3.70	1,81	.003	.148
						Identification	4.10	1,81	.006	.048
						Knowledge	5.88	1,81	.018	.068
						Belief	13.91	1,81	<.001	.147
						Intention	17.83	1,81	<.001	.180
						Empathy	18.14	1,81	<.001	.264

the faux pas task. Exact *p*-values are shown. Bonferonni adjustments were made for multiple

comparisons.

Predictor variables	Detection		Identification		Knowledge		Intention		Belief		Empathy	
	β	p	β	p	β	p	β	p	β	p	β	p
Cognitive assessment												
Similarities (SIM)	-.096	.585	-.155	.371	-.076	.661	.332	.121	.080	.588	.054	.746
Vocabulary (VB)	.145	.348	.128	.395	.161	.291	.101	.405	.190	.146	-.087	.554
Sequencing (SQ)	-.186	.443	-.178	.452	-.168	.480	.119	.528	-.059	.768	-.031	.893
Block design (BD)	.006	.970	-.001	.993	.122	.407	.244	.142	.052	.673	.005	.971
Digit span forwards (DSF)	-.031	.890	-.050	.821	.034	.897	.134	.448	.117	.534	-.245	.258
Digit span backwards (DSB)	.048	.772	.051	.754	-.079	.630	.094	.473	.098	.480	.076	.634
Stroop task	.417	.064	.407	.064	.224	.301	-.034	.841	.129	.480	**.518**	**.018**
AUD related-behaviours												
Estimated age started drinking alcohol (ASD)	-.169	.219	-.162	.227	-.002	.988	-.110	.306	-.100	.380	-.070	.588

Age started drinking problematically (AoPD)	.042	.321	-234	.301	.177	.401	-.203	.263	-.542	.008	.216	.187
Years of problematic drinking (YoD)	**-.791**	**.002**	**-.859**	**.001**	**-.741**	**.002**	**.-789**	**.019**	-.444	.068	**-.557**	**.015**
Units consumed per week (UC)	.039	.865	.106	.636	-.235	.301	-.120	.501	.079	.528	-.135	.534
Alcohol craving (ACQ)	.061	.686	-.021	.884	-.039	.790	-.034	.826	.135	.413	-.122	.396
Depression (BDI)	.085	.666	.013	.944	.167	.389	.254	.310	-.091	.617	.121	.517
Anxiety (STAI)	-.065	.639	.015	.912	-.079	.560	**-.270**	**.017**	-.261	.262	-.086	.513
Cigarettes per day	-.198	.201	-.136	.367	-.015	.918	-.004	.970	-197	.222	.206	.164
R^2	.425		.449		.440		.647		.598		.478	

Table 3: Regression coefficients for the ToM sub-scores in participants with AUD. Significant correlations are highlighted in bold.