Cognitive Deficits of People with Alcohol Dependence

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INTRODUCTION: Excessive alcohol consumption is associated with a wide range of cognitive deficits observed in varying degrees among users. The aim of the research was to compare performance across different domains of cognition in a clinical group of people with alcohol dependence and a control group of people without a dependence. **METHODS:** The research population consisted of 53 male patients with alcohol dependence treated at the Specialized Psychiatric Institute Predná Hora. For each participant from the clinical group, a participant of the same age and level of educational attainment was selected from the non-clinical population. The NEUROPSY battery tests were used to assess cognitive performance. Significance of differences between clinical and control groups was analyzed using the Mann-Whitney U test and indicators of effect size. Sensitivity and specificity of the scales were determined using ROC analyses. **RESULTS:** Research findings indicate a lower level of cognitive ability in people with alcohol dependence. The greatest differences were found in the areas of executive function, attention, and memory. The Symbol Encoding Test, Trail Making, and the Verbal Learning Test showed the highest discriminative ability for performance between groups. Differences in phonemic fluency were not found.

CONCLUSIONS: These research findings provide a relatively comprehensive picture of the cognitive performance of people with alcohol dependence. They indicate a lower level of cognitive ability in people with alcohol dependence across all areas measured, except phonemic fluency.

Keywords | Cognition – Cognitive Deficits – Alcohol – Alcohol Dependence – NEUROPSY Battery

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1 INTRODUCTION

Brain matter loss is one of the typical consequences of long-term and excessive alcohol consumption. The prevalence of cerebral atrophy in people with alcohol dependence is approximately 60% (Pfefferbaum et al., 2004). Functional changes and brain damage due to chronic alcohol use occur most frequently in the frontal lobes, limbic system, cerebellum, and partially in the hippocampus, hypothalamus, and mamillary bodies (Beresford et al., 2006; Bleich et al., 2003; Moselhy et al., 2001). These changes can manifest in a wide range of disorders characterized by long-term impairment of cognitive function, thinking and intellect caused by excessive and chronic alcohol use combined with associated physical complications (alcohol-related brain damage). The deficits can vary from initial mild cognitive deficit to severe cognitive impairment (Korsakoff syndrome, Wernicke encephalopathy) or dementia (Jauhar et al., 2014; Xu et al., 2009). Mild cognitive impairment, in contrast to severe deficits, occurs more frequently in people with alcohol dependence, with reports in approximately 30-40% of patients during the first two months of abstinence. This is often only a transient condition that diminishes over time with long-term abstinence and proper nutrition, but in 50% of users it can progress to dementia within 5 years (Gauthier et al., 2006).

Neafsey and Collins (2011) analyzed 143 studies looking at the relationship between the amount of alcohol drinking and the risk of cognitive impairment it poses for adults. They found that heavy drinking (3–4 drinks per day) was associated with the development of cognitive impairment and dementia, while light to moderate drinking showed no such association. However, acute alcohol intoxication can also have an impact on cognitive processes, with the regions of cerebellum, hippocampus and amygdala being affected in particular (Jacob & Wang, 2020; Van Skike et al., 2019). The cerebellum is involved in cognitive and non-motor functions, including learning, spatial perception, executive functions or changes in affectivity and behavior in the form of disinhibited and inadequate actions, in addition to ensuring motor coordination and balance when standing or walking (Husárová & Bareš, 2008; Van Skike et al., 2019). In mild intoxication, loss of motor coordination and prolonged reaction time may occur; however, ingesting higher doses of alcohol may result in slower thinking, and, due to disruption of hippocampal activity, impaired learning, and memory functions, particularly in visuo-spatial working memory (Söderlund et al., 2007; Van Skike et al., 2019; Zorumski et al., 2014). Episodes of amnesia (so-called blackouts) that cause partial or complete memory loss of events experienced during acute intoxication are also common (White, 2003). On the other hand, alcohol ingestion involves the amygdala in facilitating the recall of certain types of content, in particular memories of events with a strong emotional valence experienced before intoxication (Van Skike et al., 2019).

Long-term memory deficits in chronic alcohol users without associated comorbidities are largely absent (Jauhar et al., 2014). On the contrary, working memory impairments related to damage to the prefrontal cortex, frontal cortex, frontal lobes, and parietal cortex, which are responsible for the temporary storage of information, were reported (Hort et al., 2007). Significant differences were also found in the ability to learn target material (Iome et al., 2018; Krabbendam et al., 2000). The frontal lobe and its cortex play a crucial role in decision-making, logical thinking, cognitive flexibility, information processing speed, abstraction, inhibitory control, and psychomotor speed (Moselhy et al., 2001; Ratti et al., 2002). People with alcohol dependence show lower scores in motor programming, sensitivity to interference, and inhibitory control (Zago-Gomes & Nakamura-Palacois, 2009), as well as in tests of conceptualization and mental flexibility (Adhikari et al., 2016). Furthermore, research findings indicate reduced attentional capacity (Liappas et al., 2007), impaired attentional distribution (Ioime, et al., 2018), or impaired information encoding ability (Krabbendam et al., 2000; Ratti et al., 2002) in people with alcohol dependence. Although attentional deficits in people with alcohol dependence are noticeable, focused research studies in this area of cognition have been missing (Maurage et al., 2014).

Empirical evidence in the field of language impairment in people with alcohol dependence is inconclusive. Most research comparing verbal fluency between people with alcohol dependence and controls found only minimal differences in either phonemic or semantic fluency (Chanraud, et al., 2007; Juardo-Barba, et al., 2017). However, findings to the contrary can also be found in the literature (Liappas et al., 2007). Impairment in verbal fluency may reflect an intellectual or memory disorder, but it also reflects a person's ability to organize their thoughts. It has been suggested that the deterioration of verbal fluency is caused by abnormal activity in or damage to the frontal and temporal lobes (Birn et al., 2010). The brain regions mediating verbal fluency overlap considerably with areas of frontal, i.e. executive functions.

A targeted neuropsychological diagnosis is necessary to assess the severity of cognitive deficits and to select an appropriate intervention, for instance in the form of cognitive training. This has been complicated until recently, due to the lack of standardized assessment methods. Responding to the needs of clinical practice, Hajdúk et al. (2021) developed a neuropsychological battery consisting of tests assessing cognitive functions, emotion experience and personality pathology under the NEUROPSY project, and standardized it for the adult Slovak population. The aim of the current research was to use the NEUROPSY battery to investigate differences in selected cognitive domains between people with alcohol dependence and non-clinical population. Based on previous research, we hypothesized lower cognitive performance in participants with alcohol dependence (Adhikari et al., 2016; Dubois et al., 2000; Ioime, et al., 2018; Krabbendam et al., 2000; Liappas et al., 2007; Ratti et al., 2002; Zago-Gomes & Nakamura-Palacois, 2009). We predicted impaired performance in learning (H1), memory (H2), attention (H3), executive and frontal functioning (H4). We did not predict a difference between the clinical and control groups in the language area (H5). We also focused on examining the relationships between different domains of cognition, as well as their associations with selected sociodemographic and clinical variables. As the NEUROPSY test battery has not been previously validated in the population of people with alcohol dependence, the present study also aimed to validate the diagnostic accuracy of the ad-

ministered tests in this particular clinical population, and to establish a preliminary cut-off score to differentiate between the clinical and control groups.

2 METHODS

2.1 Participant sample

The research sample consisted of patients treated at the Predná Hora Specialized Psychiatric Institute at the time of data collection and who met the following inclusion criteria: 1. age over 18 years; 2. in treatment for alcohol dependence without significant psychological or physical consequences of alcohol use; 3. current treatment duration at least 1 month (patients individually cleared by the resident psychologist as well-adapted to the regime at the institute were included too). Due to the gender representation of patients currently undergoing treatment for alcohol dependence, only men were included in the research sample. The size of the research sample was set to 106 participants (53 participants in each group) using a priori power analysis, considering the minimum required statistical power of .80, the expected medium effect size (d = .50), and the level of statistical significance (p = .05).

The participant sample consisted of 53 male patients from Wards II and III of the Predná Hora Specialized Psychiatric Institute, aged 18–55 years (Mdn = 34, IQR = 15.5). The present duration of abstinence at the time of data collection was 2.3 months on average (SD = 1.7); the duration of excessive drinking ranged from 18 to 360 months (Mdn = 96, IQR = 132). 37 participants (70%) were undergoing their first treatment, while 16 participants (30%) reported a relapse. At the time of data collection, 20 participants were undergoing pharmacological treatment. Acute increase in symptoms of anxiety was found in 15% of the clinical group participants. We also observed increased levels of depressive symptoms in 19% of patients with dependence. However, differences in the rates of anxiety (U = 1403.0, p = .992, d = .01) and depression (U = 1189.5, p =. 172, d = .27) between the clinical and control groups were insignificant, or small. Performance on cognitive tests was not related to indicators of depression and anxiety.

Participants from the control group were required to meet the following criteria: 1. male; 2. no history of alcohol dependence; 3. absence of psychiatric disorder or major neurological conditions. Each participant in the clinical group (N = 53), was matched with a participant from the non-clinical population of the same age (U = 1397.0, p = .96, d = .01) and educational attainment (X^2 (2, N = 106) = .04, p = .98, V = .02).

The research project was approved by the ethics committee of the Pan-European University and the ethics committee of the Specialized Psychiatric Institute Predná Hora. Participants were informed about the important ethical aspects of the research (voluntary participation, anonymity, possibility to withdraw participation at any time, no consequences for refusal to participate). The dataset has been anonymized and is available on here - https://osf.io/g6ayw/.

2.2 Measures

Socio-demographic characteristics and clinical markers

We administered a short socio-demographic questionnaire to the participants to collect data on their age and the highest level of educational attainment. Data on the duration of excessive alcohol use, length of abstinence, relapse, presence of co-morbid disorders and pharmacological treatment was obtained from the patient records.

Neuropsychological tests

The NEUROPSY battery (Hajdúk et al., 2021) includes comprehensive neuropsychological tests, as well as cognitive function screening and self-assessment methods focusing on current emotion experience and personality pathology. Given the scope of the battery, we selected 9 methods of measuring cognitive functioning, for the purposes of the research presented here.

The Montreal Cognitive Assessment (hereinafter MoCA; Nasreddine et al., 2005) provides a baseline assessment of cognitive function in the following domains: visuo-spatial abilities and executive functions, naming, memory, attention, language, abstraction, and orientation.

In the Word Fluency Test (Thurstone & Thurstone, 1962), the participant's task is to name as many words as possible that begin with a chosen letter (K, P, S) or belong to a certain category (animals, vegetables, tools) within one minute. The score serves as an indicator of phonemic and semantic fluency.

Learning ability and memory function were assessed using the Auditory Verbal Learning Test (hereinafter AVLT; Rey, 1958). The participant repeatedly learns a list of 15 words, with the number of words memorized in five trials (A1 - A5) and the occurrence of qualitative errors (confabulations, distortions, or perseverations) that may indicate unreliability of memory functions is recorded. The participant reproduces the learned material immediately following an interference by Set B, and then 30 minutes later.

The Symbol Encoding Test adapted from the authors of the NEUROPSY battery (Hajdúk et al., 2021) is an indicator of the level of psychomotor speed and attention, capturing the functionality of the visuo-motor connection. Within a time limit of 90 seconds, the participant writes the correct number under a series of 143 symbols according to the provided key. The Forward and Backward Digit Span Test was adopted from the Wechsler battery (Wechsler, 1997) to measure attention span, concentration, immediate and short-term verbal memory. The number of recalled rows of digits is recorded, along with the length of the longest row the participant was able to remember. Performance on the Trail Making Test (Reitan, 1955) acts as an indicator of brain damage and impairment in several cognitive functions (psychomotor speed, visuo-motor search, attention, working memory, mental flexibility, and executive functioning). In Part A, the participant is asked to connect digits in ascending order as quickly as possible; in Part B, the participant is asked to mark the path from a digit to a letter in ascending order.

Table 1 | Comparison of cognitive performance in clinical and control groups

	Median		U	p	d
	Clinical group	Control group			
MoCA	24.7	26.9	766.0	<.001	.85
KPS	46.7	47.6	1339.0	.679	.08
Animals	25.0	25.1	1346.5	.714	.07
Vegetables	13.8	14.4	955.5	.004	.57
Tools	13.8	16.6	855.0	<.001	.72
A1-A5	36.5	42.3	905.5	.002	.64
A30	6.6	9.0	773.5	<.001	.84
Symbols	53.4	63.7	688.5	<.001	.98
DS F1	6.9	7.5	1182.0	.156	.28
DS F2	5.9	6.4	1164.5	.121	.30
DS B1	5.6	6.2	1183.0	.156	.27
DS B2	4.3	4.6	1247.5	.300	.19
TMT-A	31.8	27.6	1059.5	.029	.43
TMT-B	87.0	64.0	775.0	<.001	.84
FAB	17.1	17.6	972.0	.003	.55

Note. KPS – phonemic fluency; A1-A5 – the total number of items learned on the 5 list learning trials; A30 – the total number of items recalled after 30 minutes; DS F1 – Digit Span Forwards – the maximum number of digits correctly produced; DS F2 – Digit Span Forwards – total score; DS B1 – Digit Span Backwards – the maximum number of digits correctly produced; DS B2 – Digit Span Backwards – total score; FAB – Frontal Assessment Battery

The Frontal Assessment Battery (hereinafter FAB; Dubois et al., 2000) consists of six subtests assessing measures of conceptualization ability, mental flexibility, motor programming, sensitivity to interference, inhibitory control, and environmental autonomy (or adherence to the environment).

Self-assessment questionnaires – anxiety, depression

The GAD-7 (General Anxiety Disorder - 7; Spitzer et al., 2006) self-assessment screening scale is used to measure the frequency of symptoms of generalized anxiety disorder. It consists of seven items. We used the nine-item PHQ-9 (Patient Health Questionnaire; Kroenke et al., 2001) to measure the frequency of depressive symptoms in the past two weeks. The Cronbach's alpha values in our study were acceptable for the clinical group (α = .79 for GAD-7; and α = .81 for PHQ-9). Scores from the GAD-7 and PHQ-9 questionnaires were used only to describe the research sample in greater detail and to control for the relationship between cognitive performance and emotional experiencing. Given the absence of difference in depression and anxiety scores between the clinical and control groups, as well as the negligible relationship with cognitive test performance, we did not use the data from the GAD-7 and PHQ-9 questionnaires in further analyses.

2.3 Procedures

Differences between the clinical and non-clinical groups in terms of socio-demographic characteristics were analyzed using the Mann-Whitney U test (age) and chi-square test with Cramer's V coefficient (education). Comparative analyses with regard to education in the clinical group were performed using the Kruskal-Wallis test and η^2 coefficient. Statistical significance of differences between clinical and control groups was analyzed using the Mann-Whitney U test. Cohen's d with correction for non-parametric tests (accounting for the U value and the size of the groups being compared) was used as indicator of effect size difference in cognitive performance between clinical and control group. We used receiver operating characteristic curves (ROC) to assess the diagnostic accuracy of cognitive performance tests as a function of their sensitivity and specificity, and to determine cut-off scores for differentiating the performance of participants with alcohol dependence from the control group. The internal consistency of self-assessment questionnaires (GAD-7, PHQ-9) was determined by calculating Cronbach's alpha. The strength of relationships between variables (age, duration of excessive drinking, cognitive performance, depression, anxiety) was expressed by Spearman's correlation coefficient.

3 RESULTS

3.1 Comparison of cognitive performance in clinical and control groups

Based on the screening assessment, we found mild levels of cognitive impairment in 60% of the clinical group. In one participant, the presence of moderate cognitive deficit could be observed. The mean MoCA test score in the clinical group was M = 24.0 (SD = 3.3). *Table 1* shows the results of the compar-

Table 2 | Results from the ROC analysis for administered cognitive tests

	AUC	95% CI		Sensitivity	Specificity	Cut off score/theoretical maximum
MoCA	.727	.632	.823	62.30	69.80	25.50/30
KPS	.523	.412	.634	54.70	50.90	47.50*
Animals	.521	.410	.631	54.70	49.10	25.50*
Vegetables	.660	.557	.763	67.90	49.10	14.50*
Tools	.696	.597	.795	79.20	50.90	16.50*
A1-A5	.678	.574	.781	62.30	73.60	38.50/75
A30	.725	.630	.819	67.90	60.40	7.50/15
Symbols	.755	.661	.849	73.60	69.80	60.50/143
TMT-A	.623	.515	.730	58.50	62.30	29.50*
TMT-B	.724	.623	.825	69.80	67.90	70.50*
DS F1	.579	.470	.689	56.60	49.10	7.50/14
DS F2	.585	.476	.695	64.20	47.20	6.50/9
DS B1	.579	.469	.689	67.90	37.70	6.50/14
DS B2	.556	.446	.666	60.40	47.20	4.50/8
FAB	.654	.550	.758	58.50	66.00	17.50/18

Note. KPS – phonemic fluency; A1-A5 – the total number of items learned on the 5 list learning trials; A30 – the total number of items recalled after 30 minutes; DS F1 - Digit Span Forwards – the maximum number of digits correctly produced; DS F2 – Digit Span Forwards – total score; DS B1 – Digit Span Backwards – the maximum number of digits correctly produced; DS B2 – Digit Span Backwards – total score; FAB – Frontal Assessment Battery; * unlimited number of points / seconds

ison of cognitive performance in the clinical group of people with dependence vs. the control group. The largest difference was found in the level of executive function (d = .84 - .98, p < .001), long-term memory (d = .84, p < .001), and the global screening assessment of cognitive function (d = .85, p < .001). In contrast, the differences found in phonemic fluency were negligible (d = .08, p = .679).

Of the cognitive tests, the instruments administered to measure executive function in terms of psychomotor speed and mental flexibility have the best discriminative ability, namely Symbols test (AUC = 75.50%, 95% CI [66.10-84.90], sensitivity 73.60% and specificity 69.80%) and the Trail Making Test Part B (AUC = 72.40%, 95% CI [63.20-82.50], sensitivity 69.80% and specificity 67.90%). Relatively satisfactory values can also be noted for the scores in delayed recall after thirty minutes in the Auditory Verbal Learning Test (AUC = 72.50%, 95% CI [63.00 -81.90], sensitivity 67.90% and specificity 60.40%). The optimal cut-off score to differentiate between patients with dependence from healthy controls in the MoCA screening was 25.5 points (AUC = 72.70%, 95% CI [63.20 - 82.30], sensitivity 62.30% and specificity 69.80%). We report the results of the ROC analyses in *Table 2*.

3.2 Relationships between performance in different cognitive domains

The values of the correlation coefficients between the scores of individual cognitive tests for the participants in the clinical group are shown in *Table 3*. Moderately strong relationships were found between the tests measuring frontal, i.e. executive functions (Symbols, Trail Making Test in both parts, and Frontal Assessment Battery). The number of correctly marked symbols is also positively correlated with backward digit recall. Both tests are an indicator of the attention level as well as working memory. Correlations between phonemic and semantic fluency were also moderately strong. The number of words reproduced during delayed recall after 30 min correlated with the memory subtest in the MoCA screening (r_s = .51, p < .001).

3.3 Relationship between cognitive performance, sociodemographic and clinical measures

Age correlated with the total number of words recalled after five repetitions on the AVLT (r_s = - .36, p = .008), the number of symbols correctly assigned (r_s = - .47, p < .001), and the time taken to make the trail between numbers in Part A (r_s = .30, p = .030). Lower performance on a memory test of learning, for recalling learned material with a 30-min delay (r_s = -.29, p =.036) was associated with increasing duration of excessive drinking (*Table 4*). Education differentiated between performance on the TMT-B (η^2 = .22, p = .003) and Symbols (η^2 = .20, p = .006), but also scores on the backward digit recall (η^2 = .19, p = .006, alt. p = .008) and on one subtest of phonemic fluency (η^2 = .18, p = .010), with participants with a secondary education without a diploma scoring lower.

Table 3 | Correlations between scores in administered cognitive tests (clinical group)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1 MoCA														
2 KPS	.51**													
3 Animals	.24	.31*												
4 Vegetables	.32*	.40**	.32*											
5 Tools	.21	.45**	.49**	.30*										
6 A1-A5	.45**	.41**	.22	.23	.06									
7 A30	.44**	.43**	.23	.26	.09	.83**								
8 Symbols	.38**	.10	.29*	.32*	-.05	.36**	.26							
9 TMT-A	-.24	-.17	-.13	-.21	-.22	-.19	-.13	-.45**						
10 TMT-B	-.32*	-.27	-.33*	-.24	-.25	-.13	-.11	-.49**	.70**					
11 DS F1	.47**	.63**	.31*	.26	.37**	.23	.30*	.07	-.17	-.37**				
12 DS F2	.43**	.62**	.32*	.21	.36**	.24	.32*	.03	-.13	-.28*	.96**			
13 DS B1	.35**	.23	.02	.23	-.05	.28*	.34*	.45**	-.18	-.32*	.36**	.28*		
14 DS B2	.37**	.28*	-.01	.31*	-.02	.25	.29*	.41**	-.15	-.28*	.40**	.36**	.93**	
15 FAB	.48**	.31*	.23	.21	.23	.19	.23	.39**	-.40**	-.63**	.43**	.39**	.45**	.44**

Note. KPS – phonemic fluency; A1-A5 – the total number of items learned on the 5 list learning trials; A30 – the total number of items recalled after 30 minutes; DS F1 – Digit Span Forwards – the maximum number of digits correctly produced; DS F2 – Digit Span Forwards – total score; DS B1 – Digit Span Backwards – the maximum number of digits correctly produced; DS B2 – Digit Span Backwards – total score; FAB – Frontal Assessment Battery; * $p < .05$; ** $p < .01$

Table 4 | Correlations between cognitive performance, age and length of excessive drinking (clinical group)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Age	-.16	-.02	.01	.10	.04	-.36**	-.17	-.47**	.30*	.24	.01	.04	-.17	-.04	-.17
Duration of excessive drinking	-.23	-.18	.12	.03	-.07	-.24	-.29*	-.24	.23	.15	-.08	-.09	-.14	-.17	-.18

Note. 1 MoCA; 2 KPS – phonemic fluency; 3 Animals; 4 Vegetables; 5 Tools; 6 the total number of items learned on the 5 list learning trials in AVLT ;7 A30 – the total number of items recalled after 30 minutes in AVLT; 8 Symbols; 9 TMT-A; 10 TMT-B; 11 Digit Span Forwards – the maximum number of digits correctly produced; 12 Digit Span Forwards – total score; 13 Digit Span Backwards – the maximum number of digits correctly produced; 14 Digit Span Backwards – total score; 15 Frontal Assessment Battery; * $p < .05$; ** $p < .01$

4 DISCUSSION

Excessive alcohol consumption is associated with a spectrum of cognitive deficits observed to a varying degree among alcohol users. The aim of the present research was to investigate these differences in cognitive performance between people with alcohol dependence and non-clinical populations across multiple domains. Based on MoCA cognitive screening scores, a mild cognitive impairment (18–25 points) can be assumed in 60% of participants. In one participant, the presence of moderate cognitive deficit could be observed. The overall mean score in the clinical group (M = 24.0, SD = 3.3) was higher compared to the findings of several international authors. The results of Sharma et al. (2017) reported a mean MoCA test score of $M = 21.5$ ($SD = 3.3$). A very similar score ($M = 21.0$, $SD = 2.7$) was also reported by Sawant et al. (2017). This result may be due to the different composition of participant samples in terms of sociodemographic and clinical characteristics across these studies.

Based on our results, we posit that the learning process is impaired in men with alcohol dependence. We only found a negligible between-groups difference after the first reading of the target material to be learned by the participants. However, with repeated repetitions, the difference between the clinical and non-clinical group became small (second trial) to moderate (third to fifth trial), but in both cases statistically significant in favor of the control group. According to Pitel et al. (2007), people with alcohol dependence require more repetitions to learn the target material. We also observed a large between-group difference in spontaneous delayed recall after 30 minutes, consistent with other studies (Ioime et al., 2018; Krabbendam et

al., 2000). We found empirical support for hypotheses H1 and H2. Performance on the test is an indicator of episodic memory and, according to our findings, is related to the duration of excessive drinking, but not differentiated by age or educational attainment. A higher tendency towards confabulations and perseverations was observed in the group with alcohol dependency. Confabulations are defined as errors, illusions or distortions of memory that can occur with brain damage and are most often discussed in the context of amnestic syndrome (e.g., Korsak syndrome) or dementia in people with alcohol dependence (Borsutzky et al., 2008). Distortion of information or excessive repetition often indicates the unreliability of memory functions and can be a compensatory means for people with alcohol dependence in everyday life.

Our findings of impaired attention in people with alcohol dependence are consistent with the findings of other authors (Krabbendam et al., 2000; Ratti et al., 2002). We observed impaired performance in the capacity (Digit Recall), distribution (Trail Making Test), and encoding (Symbols Test) areas (H3). In everyday life, impairments in these domains can manifest themselves in a reduced amount of information that one can process in a single moment, problems with dividing attention, difficulty focusing on multiple tasks at the same time, as well as a reduced ability to briefly hold information in memory and perform certain operations with it. The administered tests are also an indicator of the level of executive function. They enable us to perform goal-directed behavior, i.e. to formulate a goal, plan, prepare and execute actions that will lead to its fulfilment (Lezak et al., 2012). The frontal lobes and prefrontal cortex are primarily responsible for the processes above and tend to be among the brain regions most susceptible to damage due to chronic alcohol use (Moselhy et al., 2001). Cognitive deficits in this area can manifest as difficulties in decision-making and logical or abstract thinking, impaired inhibitory control (including control of the impulse to ingest alcohol), or cognitive rigidity (Moselhy et al., 2001; Oscar-Berman & Marinkovic, 2007; Ratti et al., 2002). Based on the scores in tests of executive function, the reduced speed of information processing and impaired psychomotor speed can be observed among the participants in our clinical group (H4). Deficits in frontal functions as measured by the Frontal Assessment Battery were not as pronounced in people with alcohol dependence as in other studies (Adhikari et al., 2016; Viswam et al., 2018), but this may also be attributed to the limitations of the assessment tool used, which we address in the limitations to the study.

Our results further corroborate the findings of Chanraud et al. (2007) or Noël et al. (2001), according to which impairment in verbal initiation ability is either absent or negligible in people with alcohol dependence (H5). Participants from the clinical group were more likely to commit rule violations in listing words, which may again indicate impaired executive functioning, especially inhibitory control. In the test of semantic fluency, only the scores on the most difficult subtest, Tools, differentiated the performance of the clinical and control groups.

The present research provides information on the psychometric properties of the methods administered, in addition to the substantive findings on the cognitive functioning of people with alcohol dependence. Based on the results, we recommend the suitability of administering several tests from the NEUROPSY battery in this specific clinical population, for both screening purposes, and for more comprehensive assessment of cognitive performance. The Symbols Test, the Trail Making Test Part B, and the Auditory Verbal Learning Test, which measure mental flexibility, psychomotor speed, attention, and memory function, show the highest utility in differentiating performance between groups. In line with Noël et al. (2001), we conclude that the Trail Making Test Part B is a more sensitive instrument for measuring executive functions than Part A, since its completion is more demanding, requiring a considerable involvement of inhibitory control and mental flexibility in terms of switching between numbers and letters while maintaining their correct sequence, as well as the functioning of working memory for remembering the given rule. Based on our findings, the Symbols Test, as adapted by the authors of the NEUROPSY battery (Hajdúk et al., 2021), and the Trail Making Test Part B can be considered suitable tools for differentiating the performance of people with addiction from the non-clinical population. To the contrary, the Frontal Assessment Battery does not show satisfactory values of sensitivity and specificity from a psychometric point of view, mainly due to the ceiling effect present in both groups. As many as 42% of the participants in the clinical group scored full points on the test, and only 25% of the participants scored below 16 points, with a theoretical maximum of 18. All participants achieved full scores in the environmental autonomy subtest. Low variability in performance was found in all areas except for the Similarities subtest, focusing on abstract thinking. The moderately strong relationships found between the tests measuring executive functions and attention (Symbols, Trail Making Test in both Parts, and Frontal Assessment Battery, Digit Recall), verbal fluency, or the level of memory functions (Auditory Verbal Learning Test, Memory subtest in the MoCA inventory) can be considered an argument in favor of the convergent validity of the above-mentioned methods.

We interpret these results in the light of the limitations of the present research, consisting of a relatively heterogeneous composition of the clinical group in terms of sociodemographic and clinical characteristics, and in the absence of symptom severity assessment by a clinical psychologist or psychiatrist. The performance of some participants in the clinical group may also have been influenced by ongoing treatment with psychopharmaceuticals (such treatment was indicated by 20 of 53 patients) or by an unrelated psychiatric condition.

5 CONCLUSIONS

These research findings provide a relatively comprehensive picture of the performance of male patients with alcohol dependence across different domains of cognition. Future knowledge in this area could benefit from longitudinal research focusing on the long-term assessment of cognitive performance, and the prevalence of mild cognitive impairment and dementia in abstinent males and females, with a focus on specific gender differences in their cognition. Mild cognitive impairment has

been identified as a prodrome of dementia in several longitudinal studies (Xu et al., 2009) looking at the relationship between alcohol drinking and the subsequent onset of dementia in a predominantly elderly population, with the critical factor being the amount of alcohol consumed daily. From a psychometric and diagnostic viewpoint, we would also recommend validating other parts of the NEUROPSY battery (Story, Stroop Effect Test, Rey's Figure) to establish their potential for assessing cognitive function or identifying mild cognitive impairment in people with alcohol dependence without significant psychological or physical consequences.

Authors' contributions: VB proposed the study design, performed the statistical analysis and data interpretation She participated in article preparation. FF conducted the literature review, collected data and participated in the preparation of the article. All authors contributed to the creation of the article and approved the final version of the manuscript.

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REFERENCES

Adhikari, S., Rana, M., Shakya, S., & Ojha, S. P. (2016). Cognitive dysfunction in patients with alcohol dependence syndrome in a tertiary hospital in Kathmandu. *Journal of the Nepal Medical Association*, *54*(201), 17–23.

Beresford, T. P., Arciniegas, D. B., Alfers, J., Clapp, L., Martin, B., Liu, Y. D. D., Shen, D., & Davatzikos, C. (2006). Hippocampus volume loss due to chronic heavy drinking. *Alcohol: Clinical and Experimental Research*, *30*(11), 1886–1870. https://doi.org/10.1111/j.1530-0277.2006.00223.x

Birn, R. M., Kenworthy, L., Case, L., Caravella, R., Jones, T. B., Bandettini, P. A., & Martin, A. (2010). Neural systems supporting lexical search guided by letter and semantic category cues: A self-paced overt response fMRI study of verbal fluency. *NeuroImage*, *49*(1), 1099–1107. https://doi.org/10.1016/j.neuroimage.2009.07.036

Bleich, S., Sperling, W., Degner, D., Graesel, E., Bleich, K., Wilhelm, J., Havemann-Reinecke, U., Javaheripour, K., & Kornhuber, J. (2003). Lack of association between hippocampal volume reduction and first-onset alcohol withdrawal seizure. A volumetric MRI study. *Alcohol and Alcoholism*, *1*(1), 40–44. https://doi.org/10.1093/alcalc/agg017

Borsutzky, S., Fujiwara, E., Brand, M., & Markowitsch, H. J. (2008). Confabulations in alcoholic Korsakoff patients. *Neuropsychologia*, *46*(13), 3133–3143. https://doi.org/10.1016/j.neuropsychologia.2008.07.005

Dubois, B., Slachevsky, A., Litvan, I., & Pillon, B. (2000). The FAB: A frontal assessment battery at bedside. *Neurology, 55*(11), 1621–1626. https://doi.org/10.1212/WNL.55.11.1621

Gauthier, S., Reisberg, B., Zaudig, M., Petersen, R. C., Ritchie, K., Broich, K., Belleville, S., Brodaty, H., Bennett, D., Chertkow, H., Cummings, J. L., de Leon, M., Feldman, H., Ganguli, M., Hampel, H., Scheltens, P., Tierney, M. C., Whitehouse, P., Winblad, B., & International Psychogeriatric Association Expert Conference on mild cognitive impairment (2006). Mild cognitive impairment. *Lancet*, *367*(9518), 1262–1270. https://doi.org/10.1016/S0140-6736(06)68542-5

Hajdúk, M., Brandoburová, P., Pribišová, K., Krakovská, S., Gergely, S., Svingerová, A., Abrahámová, M., Dančík, D., Smolejová, E., Cviková, V., Heretik, A. ml., & Heretik, A st. (2021). *NEUROPSY: Štandardizácia neuropsychologickej testovej batérie na dospelej slovenskej populácii*. Univerzita Komenského v Bratislave.

Hort, J., & Rusina, R. (2007). *Pamäť a její poruchy*. Maxdorf.

Husárová, I., & Bareš, M. (2008). Účasť cerebella na kognitívnych a nemotorických funkciách v obraze funkčnej magnetickej rezonancie. *Neurologie pro Praxi, 9*(4), 236–239.

Chanraud, S., Martelli, C., Delain, F., Kostogianni, N., Douaud, G., Aubin, H. J., Reynaud, M., & Martinot, J. L. (2007). Brain morphometry and cognitive performance in detoxified alcohol-dependents with preserved psychosocial functioning. *Neuropsychopharmacology, 32*(2), 429–438. https://doi.org/10.1038/sj.npp.1301219

Ioime, L., Guglielmo, R., Affini, G. F., Quatrale, M., Martinotti, G., Callea, A., Savi, E., & Janiri, L. (2018). Neuropsychological performance in alcohol dependent patients: A one-year longitudinal study. *Psychiatry Investigation*, *15*(5), 505–513. https://doi.org/10.30773/pi.2017.09.27.1

Jacob, A., & Wang, P. (2020). Alcohol intoxication and cognition: Implications on mechanisms and therapeutic strategies. *Frontiers in Neuroscience, 14*, 1–10. https://doi.org/10.3389/fnins.2020.00102

Jauhar, S., Marshall, E. J., & Smith, I. (2014). Alcohol and cognitive impairment. *Advances in Psychiatric Treatment*, *20*(5), 304–313. https://doi.org/10.30773/pi.2017.09.27.1

Juardo-Barba, R., Martínez, A., Sion, A., Álvarez-Alonso, M., Robles, A., Quinto-Guillen, R., & Rubio, G. (2017). Development of a screening test for cognitive impairment in alcoholic population: TEDCA. *Actas Españolas de Psiquiatría*, *45*(5), 201–217.

Krabbendam, L., Visser, P. J., Derix, M. M., Verhey, F., Hofman, P., Verhoeven, W., Tuinier, S., & Jolles, J. (2000). Normal cognitive performance in patients with chronic alcoholism in contrast to patients with Korsakoff's syndrome. *Journal of Neuropsychiatry and Clinical Neurosciences*, *12*(1), 44–50. https://doi.org/10.1176/jnp.12.1.44

Kroenke K., Spitzer R. L., & Williams J. B. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine*, *16*(9), 606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological Assessment*. University Press.

Liappas, I., Theotoka, I., Kapaki, E., Ilias, I., Paraskevas, G. P., & Soldatos, C. R. (2007). Neuropsychological assessment of cognitive function in chronic alcohol-dependent patients and patients with Alzheimer's disease. *In Vivo*, *21*(6), 1115–1118.

Maurage, P., de Timary, P., Billieux, J., Collignon, M., & Heeren, A. (2014). Attentional alterations in alcohol dependence are underpinned by specific executive control deficits. *Alcoholism: Clinical and Experimental Research*, *38*(7), 2105–2112. https://doi.org/10.1111/acer.12444

Moselhy, H.F., Georgiou, C., & Kahn, A. (2001). Frontal lobe changes in alcoholism: A review of literature. *Alcohol and Alcoholism*, *36*(1), 357–368. https://doi.org/10.1093/alcalc/36.5.357

Nasreddine, Z. S., Phillips, N. A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J. L., & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, *4*, 695–699. https://doi.org/10.1111/j.1532-5415.2005.53221.x

Neafsey, E. J., & Collins, M. A. (2011). Moderate alcohol consumption and cognitive risk. *Neuropsychiatric Disease and Treatment*, *7*(1), 465–484. https://doi.org/10.2147/NDT.S23159

Noël, X., Van der Linden, M., Schmidt, N., Sferrazza, R., Hanak, C., Le Bon, O., De Mol, J., Kornreich, C., Pelc, I., & Verbanck, P. (2001). Supervisory attentional system in nonamnesic alcoholic men. *Archives of General Psychiatry*, *58*(12), 1152–1158. https://doi.org/10.1001/archpsyc.58.12.1152

Oscar-Berman, M., & Marinkovic, K. (2007). Alcohol: Effects on neurobehavioral functions and the brain. *Neuropsychology review*, *17*(3), 239–257. https://doi.org/10.1007/s11065-007-9038-6

Pfefferbaum, A., Rosenbloom, M. J., Serventi, K. S., & Sullivan, E. V. (2004). Brain volumes, RBC status, and hepatic function in alcoholics after 1 and 4 weeks of sobriety: Predictors of outcome. *American Journal of Psychiatry, 161*(7), 1190–1196. https://doi.org/10.1176/appi.ajp.161.7.1190

Pitel, A. L., Beaunieux, H., Witkowski, T., Vabret, F., Guillery-Girard, B., Quinette, P., Desgranges, B., & Eustache, F. (2007). Genuine episodic memory deficits and executive dysfunctions in alcoholic subjects early in abstinence. *Alcoholism, Clinical and Experimental Research*, *31*(7), 1169–1178. https://doi.org/10.1111/j.1530-0277.2007.00418.x

Ratti, M. T., Bo, P., Giardini, A., & Soragna, D. (2002). Chronic alcoholism and the frontal lobe: which executive functions are impaired? *Acta Neurologica Scandinavica*, *105*(4), 276–281. https://doi.org/10.1034/j.1600-0404.2002.0o315.x

Reitan, R. M. (1955). The relation of the Trail Making Test to organic brain damage. *Journal of Consulting Psychology, 19*(5), 393–394. https://doi.org/10.1037/h0044509

Rey, A. (1958). *L'examen clinique en psychologie.* Presse Universitaire de France.

Sawant, P., Gokhale, P., & Ferzandi, Z. (2017). Screening of chronic alcoholics for cognitive impairment using Montreal cognitive assessment

- occupational therapy perspective. *Journal of Health Management, 19*(4), 634–648. https://doi.org/10.1177/0972063417727620

Sharma, D., Padam, A., Gharu, Y., Sharma, D., & Sharma, S. (2017). Assessment of cognitive function in patients with alcohol dependence: A cross-sectional study. *National Journal of Physiology, Pharmacy and Pharmacology*, *8*(3), 337–340. https://doi.org/10.5455/njppp.2018.8.0935121092017

Söderlund, H., Grady. C. L., Easdon. C., & Tulving. E. (2007). Acute effects of alcohol on neural correlates of episodic memory encoding. *Neuroimage, 35*(2), 928–939. https://doi.org/10.1016/j.neuroimage.2006.12.024

Spitzer, R. L., Kroenke, K., & Williams, J. B. W., & Löwe, B. (2006). A brief measure for assessing generalized anxiety disorder. *Archives of Internal Medicine*, *166*(10), 1092– 1097. https://doi.org/10.1001/archinte.166.10.1092

Thurstone, L. L & Thurstone, T. G. (1962). *Primary mental abilities* (rev.). Science Research Associates.

Van Skike, C. E., Goodlett, C., & Matthews, D. B. (2019). Acute alcohol and cognition: Remembering what it causes us to forget. *Alcohol, 79,* 105–125. https://doi.org/10.1016/j.alcohol.2019.03.006

Viswam, A., Nagarajan, P., Kuppili, P. P., & Bharadwaj, B. (2018). Cognitive functions among recently detoxified patients with alcohol dependence and their association with motivational state to quit. *Indian Journal of Psychological Medicine*, *40*(4), 310–314. https://doi.org/10.4103/IJPSYM.IJPSYM_72_18

Wechsler, D. (1997). *Wechsler Memory Scale-Third Edition administration and scoring manual*. The Psychological Corporation.

White, A. M. (2003). What happened? Alcohol, memory blackouts, and the brain. *Alcohol, Research and Health, 27*(2), 186–96.

Xu, G., Liu, X., Yin, Q., Zhu, W., Zhang, R., & Fan, X. (2009). Alcohol consumption and transition of mild cognitive impairment to dementia. *Psychiatry and Clinical Neurosciences*, *63*(1), 43–49. https://doi.org/10.1111/j.1440-1819.2008.01904.x

Zago-Gomes, M. D. P., & Nakamura-Palacios, E. M. (2009). Cognitive components of frontal lobe function in alcoholics classified according to Lesch's typology. *Alcohol and Alcoholism, 44*(5), 449–457. https://doi.org/10.1093/alcalc/agp043

Zorumski, C. F., Mennerick, S., & Izumi, Y. (2014). Acute and chronic effects of ethanol on learning-related synaptic plasticity. *Alcohol, 48*(1), 1–17. https://doi.org/10.1016/j.alcohol.2013.09.045