



**HAL**  
open science

## Short-term neuropsychological recovery in alcohol use disorder: A retrospective clinical study

Angéline Maillard, Hélène Poussier, Céline Boudehent, Coralie Lannuzel, Angel Vicente, François Vabret, Nicolas Cabe, Anne-Lise Pitel

### ► To cite this version:

Angéline Maillard, Hélène Poussier, Céline Boudehent, Coralie Lannuzel, Angel Vicente, et al.. Short-term neuropsychological recovery in alcohol use disorder: A retrospective clinical study. *Addictive Behaviors*, 2020, 105, pp.106350 -. 10.1016/j.addbeh.2020.106350 . hal-03489785

**HAL Id: hal-03489785**

**<https://hal.science/hal-03489785v1>**

Submitted on 22 Aug 2022

**HAL** is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial 4.0 International License

## SHORT-TERM NEUROPSYCHOLOGICAL RECOVERY IN ALCOHOL USE DISORDER: A RETROSPECTIVE CLINICAL STUDY

Angéline MAILLARD<sup>1</sup>, Hélène POUSSIER<sup>2</sup>, Céline BOUDEHENT<sup>1,3</sup>, Coralie  
LANNUZEL<sup>1</sup>, Angel VICENTE<sup>2</sup>, François VABRET<sup>1,3</sup>, Nicolas CABE<sup>1,3</sup>, Anne-Lise  
PITEL<sup>1,4</sup>

- (1) Normandie Univ, UNICAEN, PSL Université Paris, EPHE, INSERM, U1077, CHU de Caen, GIP Cyceron, Neuropsychologie et Imagerie de la Mémoire Humaine, 14000 Caen, France
- (2) Clinique de Soins de Suite et de Réadaptation Korian Côte Normande, 14123 Ifs, France
- (3) Service d'Addictologie, Centre Hospitalier Universitaire de Caen, 14000 Caen, France
- (4) Institut Universitaire de France (IUF)

### **Corresponding author:**

Dr. Anne Lise Pitel (pitel@cyceron.fr)  
Centre Cyceron, Campus Jules Horowitz  
Boulevard Henri Becquerel  
BP 5229  
14074 Caen Cedex 5

## ABSTRACT

**Background:** Neuropsychological impairments found in recently detoxified patients with alcohol use disorder (AUD) can limit the benefit of psychosocial treatments and increase the risk of relapse. These neuropsychological deficits are reversible with abstinence. The aim of this retrospective clinical study was to investigate whether a short-term stay as inpatients in a convalescent home enables neuropsychological deficits observed in recently detoxified AUD patients to recover and even performance to return to normal.

**Methods:** Neuropsychological data were collected in 84 AUD patients. Five neuropsychological components were assessed before and after a three-week stay in a convalescent home offering multidisciplinary support. Baseline and follow-up performance were compared in the entire group of patients and in subgroups defined by the nature and intensity of the therapy (OCCASIONAL: occasional occupational and physical therapy; INTENSIVE: intensive occupational and physical therapy and neuropsychological training).

**Results:** In the entire group of patients, neuropsychological performance significantly improved between baseline and follow-up for all 5 components and even returned to a normal level for 4 of them. The ratio of patients with impaired performance was significantly lower at follow-up than baseline examination for 3 components in the INTENSIVE group only.

**Conclusion:** Recently detoxified AUD patients with cognitive deficits benefit from a short-term stay in an environment ensuring sobriety and healthy nutrition. Cognitive recovery may be enhanced by intensive care including neuropsychological training. Alcohol programs could be postponed in patients with cognitive deficits in order to offer psychosocial treatment when patients are cognitively able to benefit from it.

**KEYWORDS:** alcohol use disorder, neuropsychology, cognitive recovery, training

## 1 1. INTRODUCTION

2 It is well-known that alcohol use disorder (AUD), characterized by chronic and excessive  
3 alcohol consumption, is associated with brain damage and cognitive deficits (Epstein, Pisani,  
4 & Fawcett, 1977; Fama et al., 2019; Oscar-Berman, Shagrin, Evert, & Epstein, 1997; Parsons,  
5 1977; Pitel et al., 2011). Indeed, executive functions and episodic memory abilities were  
6 repeatedly reported as impaired in recently detoxified AUD patients (Le Berre, Fama, &  
7 Sullivan, 2017; Oscar-Berman et al., 2014 for a review). Dysfunctions of these cognitive  
8 components can alter motivation to change behavior, decision-making abilities and new  
9 complex learning (Blume, Schmaling, & Marlatt, 2005; Le Berre et al., 2012), which are  
10 crucial processes involved in the motivation to abandon excessive drinking behavior and to  
11 engage in alcohol treatment (DiClemente, Bellino, & Neavins, 1999). Altered decision-  
12 making abilities would hamper patients to modify alcohol habits in order to maintain  
13 abstinence. For these reasons, it seems clinically evident that efficient cognitive abilities are  
14 crucial when sobriety or reduction of alcohol consumption is considered. All together,  
15 neuropsychological impairments observed early in abstinence may thus limit the benefit of  
16 psychosocial treatment and increase the risk of relapse. As found in other psychopathological  
17 states such as schizophrenia (Harvey, Green, Keefe, & Velligan, 2004) and depression  
18 (Culpepper, Lam, & McIntyre, 2017), cognitive deficits limit the benefit of psychosocial  
19 treatments and notably of cognitive behavioral therapies (CBT). In AUD patients,  
20 psychoeducation, CBT or motivational interview, which are routinely proposed during the  
21 first weeks of sobriety, may thus not be clinically appropriate for AUD patients with impaired  
22 neuropsychological abilities (Fein, Bachman, Fisher, & Davenport, 1990; McCrady & Smith,  
23 1986; Tapert, Ozyurt, Myers, & Brown, 2004).

24 It is now clear that cognitive deficits observed early after detoxification can be  
25 partially or totally reversible with drinking cessation even in the absence of any stimulation.

26 Several studies indicated improvements of cognitive abilities in long-term abstinent AUD  
27 patients (more than one year) and even a return to a normal level of performance (Fein,  
28 Torres, Price, & Di Sclafani, 2006; Reed, Grant, & Rourke, 1992; Stavro, Pelletier, & Potvin,  
29 2013). However, Munro et al. (2000) highlighted that memory deficits and certain executive  
30 dysfunctions were still present after a two-year period of abstinence. Similarly, Nowakowska-  
31 Domagała et al. (2017) showed that executive deficits persist in AUD patients who have been  
32 abstinent for one year. Regarding medium-term abstinence (several months), some studies  
33 showed that six months without alcohol consumption allow neuropsychological functions to  
34 recover (Ioime et al., 2018; Loeber et al., 2010). Pitel et al. (2009) even highlighted that  
35 patients abstinent for six months perform on par with controls regarding memory and  
36 executive functions. On the opposite, other studies only found partial or nonsignificant  
37 improvement during the first year of abstinence (Ros-Cucurull et al., 2018; Stavro et al.,  
38 2013). With regard to the effects of short-term abstinence (several weeks), results are also  
39 inconsistent. Some studies reported that one month of sobriety is sufficient to recover from  
40 alcohol-related cognitive deficits (Kish, Hagen, Woody, & Harvey, 1980; Mann, Günther,  
41 Stetter, & Ackermann, 1999) while other investigations showed that neuropsychological  
42 impairments can still be observed after the first weeks of sobriety (Mulhauser, Weinstock,  
43 Ruppert, & Benware, 2018; Stavro et al., 2013). Manning et al. (2008) and Petit et al. (2017)  
44 both found that working memory recovers after three weeks of abstinence while executive  
45 dysfunction persists.

46 The discrepancies in these results may be related to different factors modulating the  
47 neuropsychological recovery. Indeed, the number of previous detoxifications could influence  
48 the recovery of flexibility abilities (Loeber et al., 2010). It is also important to take account of  
49 the smoking status of AUD patients as non-smoker AUD patients seem to better recover than  
50 smoker patients (Durazzo, Mon, Gazdzinski, Yeh, & Meyerhoff, 2015). The age of drinking

51 cessation can also explain the heterogeneity in the findings (Munro et al., 2000; Ros-Cucurull  
52 et al., 2018). A slowdown in the dynamics of cognitive recovery, which may be related to  
53 decreased brain plasticity, has been described in older AUD patients. Cognitive recovery may  
54 also be affected by the concurrent use of benzodiazepines (Manning et al., 2008; Petit et al.,  
55 2017). Further studies including AUD patients without psychotropic drugs are thus required.  
56 In addition, because of the heterogeneity in the cognitive profile of AUD patients, the use of  
57 cross-sectional design comparing patients with different lengths of abstinence (e.g.  
58 Nowakowska-Domagala et al., 2017) yields less reliable results than longitudinal studies that  
59 investigate the same group of patients over the course of abstinence (e.g. Ioime et al., 2018).  
60 Finally, several studies suggest that neuropsychological recovery can be influenced by  
61 cognitive training. After three months of cognitive training, AUD patients had higher memory  
62 and learning abilities than after three months of working therapy (Bell, Vissicchio, &  
63 Weinstein, 2016). In the same vein, Rupp et al. (2012) showed that 12 sessions of cognitive  
64 stimulation during six weeks favor cognitive improvement in certain executive functions and  
65 memory components compared to conventional treatments. All together, these results are  
66 encouraging since they suggest that psychosocial treatments could be postponed in AUD  
67 patients with neuropsychological deficits in order to offer psychoeducation, CBT or  
68 motivational interview when patients are cognitively able to benefit from them (McCrary &  
69 Smith, 1986, for review).

70 The main purpose of this retrospective clinical study was thus to investigate, in recently  
71 detoxified benzodiazepine-free AUD patients, whether a short-term stay as inpatients in a  
72 convalescent home enables neuropsychological deficits to recover and even performance to  
73 return to normal. In a more exploratory perspective, an additional objective was to examine,  
74 in a clinical setting, the effect of intensive and multidisciplinary care including  
75 neuropsychological training compared to occasional physical and/or occupational therapy.

76 Finally, the present study also explored the relations between recovery on the one hand and  
77 demographic and clinical data on the other hand.

78

79 2. MATERIAL AND METHODS

80 2.1. *Patients*

81 For this retrospective study, data were collected for 84 AUD inpatients (71 men and 13  
82 women) through clinical practice in Caen University Hospital and the convalescent home  
83 Korian Côte Normande at Ifs from 2011 to 2018. The inclusion criteria were the presence of  
84 the DSM IV criteria for alcohol dependence (American Psychiatric Association, 1994) or  
85 DSM 5 criteria for AUD (American Psychiatric Association, 2013). Patients were included  
86 when they had performed a neuropsychological assessment (in the Addiction department of  
87 Caen University Hospital) both before and after a short-term stay in a convalescent home  
88 (Korian Côte Normande at Ifs). Alcohol history is presented in Table 1. Because they reflect  
89 the reality of the population in addiction departments in France, patients who presented  
90 comorbidities were included when clinicians hypothesized that these comorbidities would not  
91 hinder them to benefit from the therapies proposed in the convalescent home. Nine patients  
92 presented psychiatric comorbidities (4 bipolar, 4 anxiety syndrome, and 1 major depression)  
93 and 15 patients had previous neurological history including head injuries, stroke, and glioma.  
94 However, patients with intelligence quotient below 70 as well as patients with suspicion of  
95 neurodegenerative disorders, Korsakoff's syndrome or fetal alcohol spectrum disorder were  
96 excluded. Fifty-nine patients were smokers and 10 had consumed other substances before  
97 alcohol detoxification (benzodiazepines, cannabis, buprenorphine). All patients were free  
98 from psychotropic medication during the follow-up period, except for those who already had  
99 a stable mood regulator treatment (5 out of the 9 patients with psychiatric comorbidities).

100

101

Table 1 about here

102

103



104        2.2. Study Design

105        The overall design of the study is illustrated in Fig. 1. Patients followed a symptom-  
106 triggered detoxification protocol at Caen University Hospital consisting of monitoring  
107 patients and providing long-acting benzodiazepines only when symptoms of alcohol  
108 withdrawal developed. Symptoms were identified with a validated assessment tool, the  
109 Cushman score (Cushman, Forbes, Lemer, & Stewart, 1985). A baseline neuropsychological  
110 assessment was administered to AUD inpatients at least 48 hours after the detoxification  
111 program (ie no physical symptoms of alcohol withdrawal and no benzodiazepine when they  
112 were assessed). The neuropsychological status was examined through five neuropsychological  
113 components: episodic memory, working memory, flexibility, inhibition and processing speed.  
114 When at least one neuropsychological component was impaired, patients were offered to stay  
115 in the convalescent home Korian Côte Normande for three weeks during which all patients  
116 remained abstinent. The first objective of this stay is to favor time-dependent physical and  
117 cognitive recovery. This convalescent home also offers multidisciplinary care. Physical  
118 therapy, occupational therapy, and dietary advice can systematically be offered to patients  
119 whereas access to neuropsychological training is limited to the scarce availability of the only  
120 neuropsychologist of the convalescent home. The neuropsychological training sessions  
121 consist of exercises that stimulate cognitive functions such as inhibition, flexibility, attention,  
122 and working memory. Physical therapy, occupational therapy, dietary advice and  
123 neuropsychological training were offered to the patients who were free to participate or not.  
124 Since the beginning of the collaboration between the addiction department and the  
125 convalescent home, the nature and frequency of each type of treatment were documented for  
126 each patient.

127        To meet our secondary objectives, patients were *a posteriori* subdivided in two groups  
128 depending on the nature and the intensity of the therapy they could benefit from. The main

129 criterion we used to split the group was the possibility to attend neuropsychological training  
130 sessions or not. When we compared patients who had benefited from neuropsychological  
131 training sessions (N=54) with those who could not attend neuropsychological training (N=30),  
132 we found that the first group had on the overall benefited from more occupational/physical  
133 therapy sessions ( $15.07 \pm 6.01$ ) than the second one ( $6.00 \pm 9.62$ ). Thus, the two groups  
134 differed not only on the nature of the treatment (neuropsychological and  
135 occupational/physical therapy vs occupational/physical therapy only) but also on the  
136 frequency of the training sessions. The two groups were consequently named INTENSIVE  
137 (N=54) and OCCASIONAL (N=30) to emphasize that given our clinical retrospective study  
138 design, the present study cannot disentangle the effect of the nature versus frequency of the  
139 sessions. After a short-term stay, all the patients underwent a follow-up neuropsychological  
140 assessment at Caen University Hospital.

141 Figure 1 about here

142

### 143 2.3. Neuropsychological Assessment

144 Because data were collected retrospectively through clinical practice, neuropsychological  
145 examinations sometimes included different tests classically used to assess the same cognitive  
146 component. For the same reason, the sample size can vary depending on the cognitive task  
147 (Figure 3).

- 148 • *Baseline examination*

149 *Episodic Memory.* A French version of the Free and Cued Selective Reminding Test (FCSRT,  
150 Grober & Buschke, 1987; Van der Linden et al., 2004) or a French version of the California  
151 Verbal Learning Test (CVLT, Delis, Kramer, Kaplan, & Ober, 1987; Nolin, 1999) were used.

152 For the FCSRT, performance on the 3 free recalls of learning trials was used. For the CVLT,  
153 performance on the 5 free recalls of learning trials was used.

154 *Executive Functions.* Inhibition was assessed by means of the Stroop test (Stroop, 1935). The  
155 time (in seconds) needed to complete the interference condition (Word-Color condition)  
156 minus the time needed for the denomination condition (Color condition) was calculated.  
157 Flexibility was assessed by means of the Modified Card Sorting Test (MCST, Cianchetti,  
158 Corona, Foscol, Scalas, & Sannio-Fancello, 2005). The number of perseverative errors was  
159 recorded.

160 *Working Memory.* Verbal working memory was assessed by means of the digit span task  
161 (forward and backward, WAIS-III; Wechsler, 1997). The final score corresponded to the  
162 number of correctly reported sequences. The standardized score was used.

163 *Processing speed.* Time needed to complete the denomination task (Color condition) of the  
164 Stroop test (Stroop, 1935) was recorded.

165 • *Follow-Up examination*

166 Episodic memory was assessed using a parallel version of the FCSRT (Van der Linden et al.,  
167 2004) to avoid a potential test-retest effect, which is particularly problematic for memory  
168 assessment. The other functions were assessed using the same tasks as those administered  
169 during the baseline session since no parallel forms are available for these tests, and executive  
170 tasks are less affected by test-retest effect (Bachoud-Levi et al., 2001).

171

172 *2.4. Statistical analyses*

173 All neuropsychological measures were first transformed into z-scores which were  
174 obtained using the mean and standard deviation of the standardized norms for each

175 neuropsychological test and each patient adjusted for age, gender, and level of education  
176 when available. Regarding episodic memory, an average of the z-score for the free recall trials  
177 was computed for each patient. Thus all cognitive variables were on the same scale with 0 as  
178 the mean and 1 as the standard deviation of the reference population. When necessary, the  
179 direction of the z-score was reversed so that all the z-scores had the same direction: the higher  
180 the z-score, the better the performance.

181       Given that the sample size varied depending on the cognitive components studied and the  
182 fact that some data were not normally distributed (according to Shapiro Wilk tests), we chose  
183 to conduct non-parametric tests for all analyses.

184       First, in order to determine whether a short-term stay as inpatients in a convalescent home  
185 enables neuropsychological recovery, we compared cognitive performance between baseline  
186 and follow-up examinations by means of Wilcoxon's tests in the entire group of patients. We  
187 also investigated whether the cognitive changes between baseline and follow-up were  
188 clinically meaningful. To this end, both at baseline and follow-up examinations and for each  
189 patient, the neuropsychological performance was classified as impaired or preserved  
190 according to a binary z-score approach. The individual performance was considered as  
191 impaired when a z-score was strictly below -1.65 standard deviations from the mean. The  
192 proportion of patients with preserved and impaired performance was then compared between  
193 baseline and follow-up using Yates corrected Chi-square tests.

194       Second, we explored whether an intensive and multidisciplinary therapy including a  
195 neuropsychological training would result in better recovery than occasional physical and/or  
196 occupational therapy. To this end, we first compared the INTENSIVE and OCCASIONAL  
197 groups on demographic and clinical variables, as well as on the neuropsychological  
198 performance both at baseline and follow-up using Mann-Whitney's tests or Yates corrected  
199 Chi-square tests. We then used Wilcoxon's tests to examine neuropsychological changes

200 between baseline and follow-up in each group, and Yates corrected Chi-square tests to  
201 compare the proportion of patients with preserved and impaired performance between  
202 baseline and follow-up examinations in each group.

203 Last, we gauged the cognitive recovery by computing a ratio (follow-up performance -  
204 baseline performance divided by the baseline performance for each patient). Spearman's  
205 correlations were conducted to examine the relationships between this score of cognitive  
206 recovery on the one hand and the number of days in the convalescent home, the number of  
207 therapy sessions, and demographic as well as disease-related variables on the other hand.

208 We performed statistical analyses with the complete sample of patients as well as 1)  
209 without patients who presented neurological comorbidities, 2) without patients who presented  
210 psychiatric comorbidities, and 3) without these two samples.

211

## 212 3. RESULTS

### 213 *3.1. Comparison between neuropsychological performance at Baseline and Follow-up in* 214 *the entire group*

215 In the entire group of patients, flexibility, inhibition, and processing speed were  
216 impaired (z-scores < -1.65) at baseline, whereas episodic memory and working memory  
217 components were relatively preserved (z-scores > -1.65). Wilcoxon's comparisons (Fig. 2.)  
218 revealed significant differences between baseline and follow-up examinations: cognitive  
219 performance was better at follow-up than baseline for all components (episodic memory [ $T =$   
220 394,  $p < 0.001$ ]; working memory [ $T = 172$ ,  $p < 0.001$ ]; flexibility [ $T = 120$ ,  $p < 0.001$ ];  
221 inhibition [ $T = 84$ ,  $p < 0.001$ ]; and speed processing [ $T = 96$ ,  $p < 0.001$ ]). These results were  
222 unchanged when patients with comorbidities were removed from the sample. Episodic  
223 memory and working memory were still preserved at follow-up. Inhibition and processing  
224 speed abilities were also preserved on this second assessment (z-scores > -1.65). Yet, the

225 flexibility component remained impaired ( $z\text{-score} < -1.65$ ), whereas when analyses were  
226 performed without patients who presented comorbidities, flexibility performance always  
227 returned to a normal level.

228 Figure 2 about here

229 Chi-square tests showed that the proportion of patients with preserved or impaired  
230 performance in the entire group was significantly different between baseline and follow-up  
231 assessments for episodic memory ( $X^2 = 9.31, p < 0.01$ ), working memory ( $X^2 = 8.94, p <$   
232  $0.01$ ), flexibility ( $X^2 = 14.94, p < 0.001$ ), and inhibition ( $X^2 = 5.11, p = 0.02$ ), indicating that  
233 there were fewer patients with impaired performance at follow-up than baseline (Figure 3).

234

235 *3.2. Comparisons between demographic and clinical variables in the INTENSIVE and*  
236 *OCCASIONAL groups*

237 The statistical analyses did not reveal any significant difference in terms of  
238 demographic, clinical, or disease-related variables between the patients of the INTENSIVE  
239 and OCCASIONAL groups at baseline (Table 1). However, patients in the INTENSIVE  
240 group benefited from more therapy sessions than patients in the OCCASIONAL group ( $U =$   
241  $341, p < 0.001$ ). More precisely, the number of physical therapy sessions was significantly  
242 higher in the INTENSIVE group than in the OCCASIONAL group ( $U = 357, p < 0.001$ ) as  
243 well as for the number of occupational therapy sessions ( $U = 349, p < 0.001$ ). Only patients in  
244 the INTENSIVE group benefited from neuropsychological training sessions ( $5,50 \pm 3,23$ ).

245

246 *3.3. Comparisons between neuropsychological performance in the INTENSIVE and*  
247 *OCCASIONAL groups at Baseline*

248 Analyses did not reveal any significant difference between the INTENSIVE and  
249 OCCASIONAL groups at baseline, except for the flexibility component (Figure 4): patients in

250 the INTENSIVE group had lower flexibility performance than patients in the OCCASIONAL  
251 group ( $U = 129.5$ ,  $p = 0.04$ ). When comorbid patients were excluded, the difference was no  
252 more significant. However, regarding the proportion of patients with impaired performance,  
253 no between-group difference was found at baseline (Figure 3).

254 Figure 3 about here

255

#### 256 3.4. Comparisons between neuropsychological performance at Baseline and Follow-up in 257 each group

258 In the INTENSIVE group, analyses revealed a significant improvement between  
259 baseline and follow-up performance for all five components (episodic memory [ $T = 149.5$ ,  $p <$   
260  $0.001$ ]; working memory [ $T = 40.5$ ,  $p < 0.001$ ]; flexibility [ $T = 43$ ,  $p < 0.001$ ]; inhibition [ $T =$   
261  $46$ ,  $p < 0.001$ ]; and processing speed [ $T = 50.5$ ,  $p < 0.001$ ]).

262 In the OCCASIONAL group, analyses revealed a significant improvement between  
263 baseline and follow-up performance for episodic memory ( $T = 58$ ,  $p < 0.01$ ); inhibition ( $T = 6$ ,  
264  $p < 0.01$ ) and processing speed ( $T = 10$ ,  $p = 0.01$ ) only (Figure 4).

265 Regarding the comparisons of the proportion of patients with impaired and preserved  
266 performance between baseline and follow-up, Chi-square tests showed more patients with  
267 preserved performance at follow-up than at baseline only in the INTENSIVE group (Fig. 3)  
268 for episodic memory ( $X^2 = 5.52$ ,  $p = 0.02$ ), working memory ( $X^2 = 6.45$ ,  $p = 0.01$ ), and  
269 flexibility ( $X^2 = 17.08$ ,  $p < 0.001$ ).

270 There were no major changes when patients with comorbidities were removed from  
271 the samples, as for the two next parts.

272 Figure 4 about here

273        *3.5. Comparisons between neuropsychological performance in the INTENSIVE and*  
274        *OCCASIONAL groups at Follow-up*

275        No differences were found between the two groups regarding neuropsychological  
276 performance at follow-up (Figure 4).

277

278        *3.6. Relationships between neuropsychological changes and clinical variables*

279        There was no significant relationship between cognitive changes on the one hand and  
280 demographic, clinical, or disease-related variables (all p values > 0.05).

281

282        4. DISCUSSION

283        The present investigation aimed at determining whether a short-term stay (three  
284 weeks) as inpatients in a convalescent home enables neuropsychological deficits observed  
285 early in abstinence to recover and even performance to return to normal. In accordance with  
286 previous studies (Oscar-Berman et al., 2014; Pitel et al., 2007), baseline results collected early  
287 in abstinence indicated that AUD patients exhibited a slowdown in the processing speed, low  
288 memory performance and executive dysfunction including altered flexibility and inhibition  
289 abilities. Comparisons between baseline and follow-up examinations confirm that cognitive  
290 performance significantly improves during the first weeks of sobriety (Kish et al., 1980;  
291 Mann et al., 1999). Contrary to Petit et al. (2017) who described persistent executive  
292 impairments after 3 weeks of abstinence, we found recovery of inhibition abilities within the  
293 same time period.

294        For four cognitive components (episodic memory, working memory, inhibition, and  
295 processing speed), performance returned to a normal level (z-scores > -1.65) at follow-up.  
296 Improvement of flexibility was not sufficient to observe a normalization of the performance at



297 follow-up (zscore<-1.65) because baseline results were very severely impaired. Regarding the  
298 proportion of patients with preserved and impaired performance, there were fewer patients  
299 with impaired episodic and working memory as well as executive performance at follow-up  
300 than at baseline. The absence of clinically meaningful changes regarding the ratio of patients  
301 with impaired and preserved processing speed performance may be related to the low  
302 proportion of impaired patients at baseline. Both when considered the average performance of  
303 the group or the individual results, our data suggest that neuropsychological abilities can  
304 return to normal not only after several years (Rosenbloom, Pfefferbaum, & Sullivan, 2004;  
305 Rourke & Grant, 1999) or several months of abstinence (Pitel et al., 2009) but also after  
306 several weeks without alcohol consumption.

307         Psychosocial treatments are usually proposed early in abstinence, right at the time  
308 when alcohol-related neuropsychological deficits are the most severe. Thus, the timing of  
309 most psychosocial treatments may not be appropriate for patients with cognitive impairments  
310 (McCrary & Smith, 1986). Our results suggest that the neuropsychological recovery observed  
311 after just a few weeks of sobriety could enable AUD patients to be cognitively able to  
312 effectively benefit from motivational interview, psychoeducation and CBT. After short-term  
313 abstinence, efficient inhibition abilities would make patients more able to control alcohol  
314 craving but the use of novel adaptative strategies in risky situations (Giancola & Moss, 1998)  
315 may still be difficult because of residual flexibility deficits. Such persistent flexibility  
316 impairments could reflect premorbid cognitive alterations that would make patients  
317 particularly vulnerable to develop AUD (Mulhauser et al., 2018). Interestingly, when patients  
318 with psychiatric and/or neurological comorbidities were excluded from the analyses,  
319 flexibility performance returned to a normal level at follow-up. This result suggests that  
320 cognitive impairments related to psychiatric and/or neurological diseases may slow down the

321 course of recovery (Bourne et al., 2013; Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari,  
322 & Lönnqvist, 2008; Whiting, Deane, Simpson, McLeod, & Ciarrochi, 2017).

323 While drinking cessation is the main factor that influences cognitive recovery, other  
324 variables have been suggested to modulate its dynamics and extent. The present study does  
325 not confirm the role of age (Munro et al., 2000; Ros-Cucurull et al., 2018) and smoking status  
326 (Durazzo et al., 2015), and does not indicate a relationship between alcohol-related variables  
327 and cognitive recovery. Diet may also support neuropsychological recovery through regular  
328 and healthy meals provided in the convalescent home. Biological comorbidities including  
329 malnutrition (Ritz et al., 2016) and altered thiamine metabolism (Coulbault et al., 2019) are  
330 associated with neuropsychological impairments. As all patients included in the present study  
331 were well-fed and benefited from nutritional monitoring during convalescence, the effect of  
332 this variable could not be examined.

333 Another factor has been described to influence cognitive recovery. The concept of  
334 experience-dependent recovery suggests that neuropsychological skills may indeed be  
335 enhanced by the practice of cognitive tasks (Goldman, 1990). In an exploratory perspective,  
336 the present study enabled us to investigate the effect of intensive and multidisciplinary care on  
337 cognitive recovery. Between baseline and follow-up, the INTENSIVE group significantly  
338 improved all five cognitive components, whereas the OCCASIONAL group only improved  
339 episodic memory, inhibition, and processing speed performance. For flexibility, the difference  
340 in the recovery of the two groups may be related to a larger margin of improvement observed  
341 in the INTENSIVE group. Indeed, at baseline, the INTENSIVE group performed lower than  
342 the OCCASIONAL group only on the flexibility task. However, this difference may be driven  
343 by the presence of patients with psychiatric and neurological comorbidities as there was no  
344 more significant difference at baseline when these patients were excluded from the analyses.  
345 Our results also show that in the OCCASIONAL group, the proportion of patients with

346 preserved performance did not increase from baseline to follow-up. On the opposite, in the  
347 INTENSIVE group, there were significantly more patients with preserved episodic memory,  
348 working memory and flexibility performance at follow-up than at baseline. To sum up, even a  
349 short-term mild intensity program based on occasional physical and occupational therapy  
350 allows cognitive improvement during the first weeks of sobriety (Kish et al., 1980; Mann et  
351 al., 1999). But a more intensive program including cognitive training seems to favor an  
352 overall neuropsychological recovery and even a normalization of some cognitive components.  
353 The present results are in agreement with those of Bell et al. (2016) and Rupp et al. (2012),  
354 who showed that cognitive training programs (respectively three months and six weeks) allow  
355 a better cognitive recovery than usual treatment.

356         It is worthwhile keeping in mind that the present investigation is a retrospective  
357 clinical study, which does not permit to determine whether cognitive recovery is modulated  
358 by the nature and/or the intensity of the program. Another limitation of this study relates to  
359 the tasks used for the cognitive assessment. Performance was analyzed with  
360 neuropsychological tasks classically used in routine practice. Other experimental tasks, not  
361 frequently used in clinical settings, would be more sensitive to alcohol effects (Stephan et al.,  
362 2017). It would thus be interesting to complete the neuropsychological assessment using  
363 decision-making tasks, social cognition tasks, and more ecological tests, which would  
364 probably better reflect daily life difficulties. Ideally, the use of parallel versions of the tasks,  
365 when available, should also be used, not only for memory assessment, to avoid test-retest  
366 effects. Moreover, given the retrospective nature of this research, the experimental design  
367 (exact modalities of treatment, randomization of the different treatment options and use of  
368 blind experimenters) is not as well controlled as it would be with a randomized controlled  
369 trial (RCT). Further RCT are thus required to set up solid programs favoring cognitive  
370 recovery and specifying whether the nature or intensity of the care matters. For a better

371 differentiation between time-dependent or treatment-dependent recovery, future studies need  
372 to include non-treated comparison groups. In a more sophisticated prospective study with  
373 three experimental groups, a passive control group with no treatment could be proposed to  
374 measure spontaneous recovery only related to sobriety. The clinical setting in which this study  
375 was conducted offering systematically occasional physical and occupational therapy, the  
376 OCCASIONAL group was considered as an active control group. From a clinical perspective,  
377 psychosocial treatments involving efficient cognitive abilities should be postponed in order to  
378 propose programs including physical, cognitive and nutritional support that seem to favor  
379 cognitive recovery of AUD patients with neuropsychological deficits. While the present  
380 study was conducted in AUD inpatients, day center programs could be a relevant alternative  
381 so that patients can face their daily life without alcohol. Finally, it would also be interesting to  
382 examine whether the recovery observed after short-term sobriety allows AUD patients to  
383 engage more actively and efficiently in psychosocial treatment ultimately decreasing the rate  
384 of relapse.

385         To conclude, early detoxified AUD patients who are cognitively impaired could be  
386 transferred to an environment in which they would be well-fed, alcohol would be prohibited,  
387 and a multidisciplinary treatment would be offered. The cognitive improvement observed  
388 during a short-term stay in such environment would enable patients to effectively benefit from  
389 psychosocial programs to eventually reduce alcohol relapse.

390

391

392

393

394

395

396 ACKNOWLEDGMENTS

397 The authors are grateful to Alice Laniepce and Claire André for their helpful comments on the  
398 manuscript.

399

400 CONFLICTS OF INTEREST

401 The authors report no conflicts of interest.

402

403 FUNDING

404 This research did not receive any specific grant from funding agencies in the public,  
405 commercial, or not-for-profit sectors.

406

407

408

409

410

411 **Table 1.** Demographic, clinical and disease-related characteristics of the INTENSIVE and  
 412 OCCASIONAL groups

<b>Variables</b>	<b>ENTIRE GROUP (N=84) (M ± SD or N)</b>	<b>INTENSIVE GROUP (N=54) (M ± SD or N)</b>	<b>OCCASIONAL GROUP (N=30) (M ± SD or N)</b>
<u>Demographic variables</u>			
Men/Women ratio	71/13	46/8	25/5
Age (years)	53,38 ± 8,57	53,04 ± 8,72	54,00 ± 8,40
Education (years)	11,31 ± 2,10	11,50 ± 2,11	10,97 ± 2,08
<u>Clinical variables</u>			
Number of days in the convalescent home	23,29 ± 7,21	22,61 ± 6,68	24,50 ± 8,05
Number of days between assessments	33,17 ± 11,90	33,50 ± 12,38	32,57 ± 11,17
Overall number of therapy sessions	11,83 ± 8,63	15,07 ± 6,01	6,00 ± 9,62 ***
Number of neuropsychological training sessions	3,23 ± 2,97	5,02 ± 2,16	--
Number of physical therapy sessions	7,54 ± 5,93	9,57 ± 4,84	3,87 ± 6,02 ***
Number of occupational therapy sessions	4,30 ± 3,78	5,50 ± 3,23	2,13 ± 3,79 ***
<u>Disease-related factors</u>			
Daily alcohol consumption (units <sup>a</sup> )	17,76 ± 10,35 (5MD)	18,02 ± 11,05 (1MD)	17,23 ± 8,92 (4MD)
AUD duration (years)	21,73 ± 11,66 (9MD)	21,85 ± 11,18 (6MD)	21,52 ± 12,68 (3MD)
Smokers	59 (2MD)	37 (1MD)	22 (1MD)
Polysubstance users	10 (1MD)	6 (1MD)	4
Psychiatric comorbidity	9 (1MD)	7 (1MD)	2
Neurological history	15 (1MD)	12 (1MD)	3

413 *N: sample size; M: mean; SD: standard deviation; MD: missing data*

414 <sup>a</sup>: an alcohol unit = 10g of pure ethanol

415 \*\*\**p* < 0.001, Mann-Whitney's tests used to compare demographic and clinical characteristics between the  
 416 INTENSIVE and OCCASIONAL groups

417

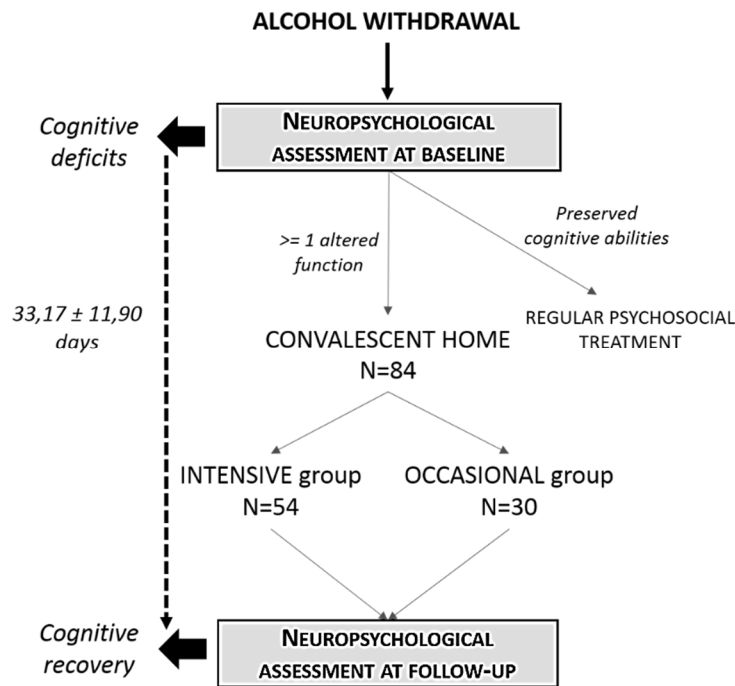
418

419

420

421

422



424

425

426 **Figure 1.** Study design.

427 After alcohol withdrawal, AUD patients performed a first neuropsychological assessment.

428 When they had preserved cognitive abilities, patients attended the regular psychosocial

429 treatment, whereas when at least one neuropsychological component was impaired, they were

430 offered to stay in a convalescent home. The entire group of patients was *a posteriori*

431 subdivided in two groups (INTENSIVE versus OCCASIONAL) depending on the therapy

432 they could benefit from. One month after the first neuropsychological assessment, patients

433 underwent a follow-up assessment. In order to study the cognitive recovery, we compared

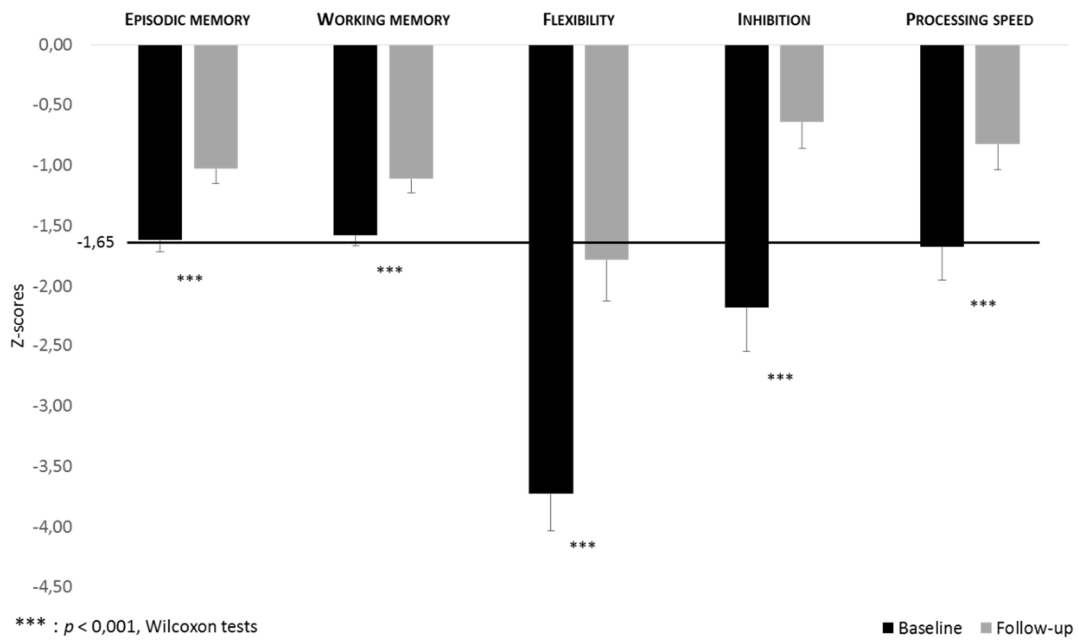
434 baseline and follow-up neuropsychological assessments in the entire group and in each group

435 separately.

436

437

438



440

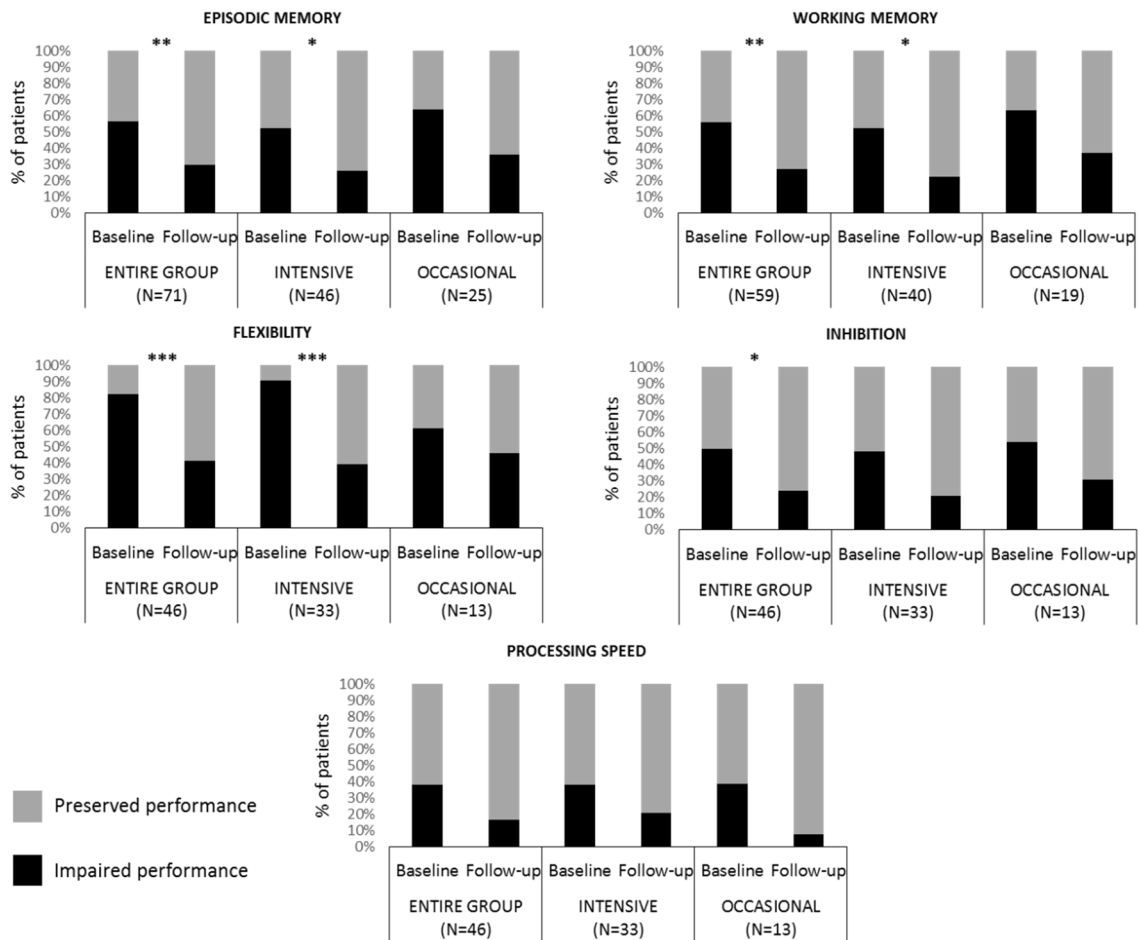
441

442 **Figure 2.** Neuropsychological performance at Baseline and Follow-up.

443 \*\*\*: significant difference between baseline and follow-up performance, with a p-value  
 444 strictly below 0.001 at Wilcoxon test. The black line represents the threshold for considering  
 445 performance as impaired ( $z\text{-score} < -1.65$ ) or preserved ( $z\text{-score} > -1.65$ ).

446





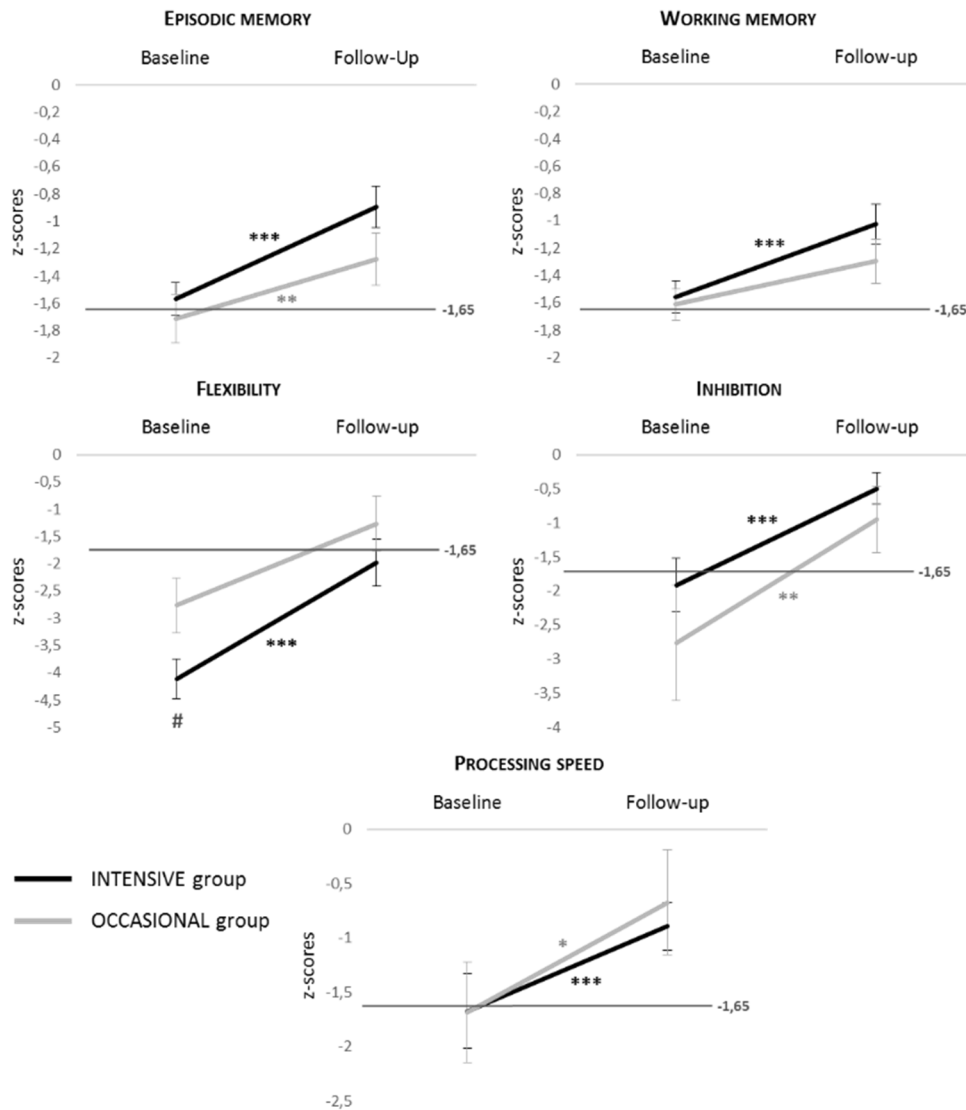
448

449

450 **Figure 3.** Ratio of patients with preserved and impaired neuropsychological performance at  
 451 Baseline and Follow-up in the ENTIRE group, as well as in the INTENSIVE and  
 452 OCCASIONAL groups.

453 The performance of each patient was classified as impaired or preserved according to a binary  
 454 z-score approach (Cut-off z-score: -1.65). \*, \*\*, \*\*\* represent significant differences in the  
 455 proportion of patients with impaired and preserved performance between baseline and follow-  
 456 up, with p-values respectively below 0.05, 0.01, and 0.001 at Chi-square tests. The proportion  
 457 of patients with impaired performance differed significantly between the two groups neither at  
 458 baseline nor at follow-up examinations.

459



460

461

462 **Figure 4.** Neuropsychological performance at Baseline and Follow-up in the INTENSIVE  
 463 and OCCASIONAL groups.

464 Means and standard errors. # represents a significant difference between the performance of  
 465 the two groups at baseline, with a  $p < 0.05$  at the Mann-Whitney test.

466 \*, \*\*, \*\*\* represent significant differences between baseline and follow-up performance  
 467 within each group, with p-values respectively below 0.05, 0.01, and 0.001 at Wilcoxon tests.

468 The dark gray lines represent the threshold for considering performance as impaired (z-  
 469 score  $< -1.65$ ) or preserved (z-score  $> -1.65$ ).

470

471 REFERENCES

472 American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders*

473 American Psychiatric Association. *Washington, DC*.

474 American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders, 5th*

475 *Edition. Diagnostic and Statistical Manual of Mental Disorders, 5th Edition*.

476 <https://doi.org/10.1176/appi.books.9780890425596.744053>

477 Bachoud-Levi, A.-C., Maison, P., Bartolomeo, P., Boisse, M.-F., Dalla Barba, G., Ergis, A.-M., ...

478 Peschanski, M. (2001). Retest effects and cognitive decline in longitudinal follow-up of patients

479 with early HD. *Neurology*, *56*(8), 1052–1058. <https://doi.org/10.1212/WNL.56.8.1052>

480 Bell, M. D., Vissicchio, N. A., & Weinstein, A. J. (2016). Cognitive Training and Work Therapy for

481 the Treatment of Verbal Learning and Memory Deficits in Veterans With Alcohol Use Disorders.

482 *Journal of Dual Diagnosis*, *12*(1), 83–89. <https://doi.org/10.1080/15504263.2016.1145779>

483 Blume, A. W., Schmaling, K. B., & Marlatt, G. A. (2005). Memory, executive cognitive function, and

484 readiness to change drinking behavior. *Addictive Behaviors*, *30*(2), 301–314.

485 <https://doi.org/10.1016/j.addbeh.2004.05.019>

486 Bourne, C., Aydemir, Ö., Balanzá-Martínez, V., Bora, E., Brissos, S., Cavanagh, J. T. O., ...

487 Goodwin, G. M. (2013). Neuropsychological testing of cognitive impairment in euthymic bipolar

488 disorder: an individual patient data meta-analysis. *Acta Psychiatrica Scandinavica*, *128*(3), 149–

489 162. <https://doi.org/10.1111/acps.12133>

490 Castaneda, A. E., Tuulio-Henriksson, A., Marttunen, M., Suvisaari, J., & Lönnqvist, J. (2008). A

491 review on cognitive impairments in depressive and anxiety disorders with a focus on young

492 adults. *Journal of Affective Disorders*, *106*(1–2), 1–27. <https://doi.org/10.1016/j.jad.2007.06.006>

493 Cianchetti, C., Corona, S., Foscol, M., Scalas, F., & Sannio-Fancello, G. (2005). Modified Wisconsin

494 Card Sorting Test: proposal of a supplementary scoring method. *Archives of Clinical*

495 *Neuropsychology*, 20(4), 555–558. <https://doi.org/10.1016/j.acn.2004.12.002>

496 Coulbault, L., Ritz, L., Vabret, F., Lannuzel, C., Boudehent, C., Nowoczyn, M., ... Pitel, A. L. (2019).  
497 Thiamine and phosphate esters concentrations in whole blood and serum of patients with alcohol  
498 use disorder: a relation with cognitive deficits. *Nutritional Neuroscience*, 1–12.  
499 <https://doi.org/10.1080/1028415X.2019.1652438>

500 Culpepper, L., Lam, R. W., & McIntyre, R. S. (2017). Cognitive Impairment in Patients With  
501 Depression: Awareness, Assessment, and Management. *The Journal of Clinical Psychiatry*,  
502 78(9), 1383–1394. <https://doi.org/10.4088/JCP.tk16043ah5c>

503 Cushman, P., Forbes, R., Lemer, W., & Stewart, M. (1985). Alcohol Withdrawal Syndromes: Clinical  
504 Management with Lofexidine. *Alcoholism: Clinical and Experimental Research*, 9(2), 103–108.  
505 <https://doi.org/10.1111/j.1530-0277.1985.tb05527.x>

506 Delis, D., Kramer, J., Kaplan, E., & Ober, B. (1987). California Verbal Learning Test (CVLT)  
507 Manual. *The Psychological Corporation*. [https://doi.org/10.1207/s15328023top2503\\_18](https://doi.org/10.1207/s15328023top2503_18)

508 DiClemente, C. C., Bellino, L. E., & Neavins, T. M. (1999). Motivation for change and alcoholism  
509 treatment. *Alcohol Research & Health : The Journal of the National Institute on Alcohol Abuse*  
510 *and Alcoholism*, 23(2), 86–92. Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/10890801>

511 Durazzo, T. C., Mon, A., Gazdzinski, S., Yeh, P.-H., & Meyerhoff, D. J. (2015). Serial longitudinal  
512 magnetic resonance imaging data indicate non-linear regional gray matter volume recovery in  
513 abstinent alcohol-dependent individuals. *Addiction Biology*, 20(5), 956–967.  
514 <https://doi.org/10.1111/adb.12180>

515 Epstein, P. S., Pisani, V. D., & Fawcett, J. A. (1977). Alcoholism and Cerebral Atrophy. *Alcoholism:*  
516 *Clinical and Experimental Research*, 1(1), 61–65. [https://doi.org/10.1111/j.1530-](https://doi.org/10.1111/j.1530-0277.1977.tb05769.x)  
517 [0277.1977.tb05769.x](https://doi.org/10.1111/j.1530-0277.1977.tb05769.x)

518 Fama, R., Le Berre, A.-P., Sassoon, S. A., Zahr, N. M., Pohl, K. M., Pfefferbaum, A., & Sullivan, E.  
519 V. (2019). Relations between cognitive and motor deficits and regional brain volumes in

520 individuals with alcoholism. *Brain Structure and Function*, 224(6), 2087–2101.  
521 <https://doi.org/10.1007/s00429-019-01894-w>

522 Fein, G., Bachman, L., Fisher, S., & Davenport, L. (1990). Cognitive impairments in abstinent  
523 alcoholics. *The Western Journal of Medicine*, 152(5), 531–537.  
524 <https://doi.org/10.1016/j.optlastec.2010.08.002>

525 Fein, G., Torres, J., Price, L. J., & Di Sclafani, V. (2006). Cognitive Performance in Long-Term  
526 Abstinent Alcoholics George. *Alcohol Clin Exp Res*, 30(9), 1538–1544. Retrieved from  
527 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1868685/pdf/nihms20940.pdf>

528 Giancola, P. R., & Moss, H. B. (1998). Executive Cognitive Functioning in Alcohol Use Disorders. In  
529 *Recent Developments in Alcoholism* (pp. 227–251). Boston, MA: Springer US.  
530 [https://doi.org/10.1007/0-306-47148-5\\_10](https://doi.org/10.1007/0-306-47148-5_10)

531 Goldman, M. S. (1990). Experience-Dependent Neuropsychological Recovery and the Treatment of  
532 Chronic Alcoholism. *Neuropsychology Review*, 1(1), 75–101.

533 Grober, E., & Buschke, H. (1987). Genuine memory deficits in dementia. *Developmental*  
534 *Neuropsychology*, 3(1), 13–36. <https://doi.org/10.1080/87565648709540361>

535 Harvey, P. D., Green, M. F., Keefe, R. S. E., & Velligan, D. I. (2004). Cognitive Functioning in  
536 Schizophrenia. *The Journal of Clinical Psychiatry*, 65(3), 361–372.  
537 <https://doi.org/10.4088/JCP.v65n0312>

538 Ioime, L., Guglielmo, R., Affini, G. F., Quatrone, M., Martinotti, G., Callea, A., ... Janiri, L. (2018).  
539 Neuropsychological Performance in Alcohol Dependent Patients: A One-Year Longitudinal  
540 Study. *Psychiatry Investigation*, 15(5), 505–513. <https://doi.org/10.30773/pi.2017.09.27.1>

541 Kish, G. B., Hagen, J. M., Woody, M. M., & Harvey, H. L. (1980). Alcoholics' recovery from cerebral  
542 impairment as a function of duration of abstinence. *Journal of Clinical Psychology*, 36(2), 584–  
543 589. <https://doi.org/10.1002/jclp.6120360234>

544 Le Berre, A.-P., Fama, R., & Sullivan, E. V. (2017). Executive Functions, Memory, and Social

545 Cognitive Deficits and Recovery in Chronic Alcoholism: A Critical Review to Inform Future  
546 Research. *Alcoholism, Clinical and Experimental Research*, 41(8), 1432–1443.  
547 <https://doi.org/10.1111/acer.13431>

548 Le Berre, A.-P., Vabret, F., Cauvin, C., Pinon, K., Allain, P., Pitel, A.-L., ... Beaunieux, H. (2012).  
549 Cognitive barriers to readiness to change in alcohol-dependent patients. *Alcoholism, Clinical and*  
550 *Experimental Research*, 36(9), 1542–1549. <https://doi.org/10.1111/j.1530-0277.2012.01760.x>

551 Loeber, S., Duka, T., Welzel Márquez, H., Nakovics, H., Heinz, A., Mann, K., & Flor, H. (2010).  
552 Effects of repeated withdrawal from alcohol on recovery of cognitive impairment under  
553 abstinence and rate of relapse. *Alcohol and Alcoholism (Oxford, Oxfordshire)*, 45(6), 541–547.  
554 <https://doi.org/10.1093/alcalc/agq065>

555 Mann, K., Günther, A., Stetter, F., & Ackermann, K. (1999). Rapid recovery from cognitive deficits in  
556 abstinent alcoholics: a controlled test-retest study. *Alcohol and Alcoholism (Oxford,*  
557 *Oxfordshire)*, 34(4), 567–574. <https://doi.org/10.1093/alcalc/34.4.567>

558 Manning, V., Wanigaratne, S., Best, D., Hill, R. G., Reed, L. J., Ball, D., ... Strang, J. (2008).  
559 Changes in neuropsychological functioning during alcohol detoxification. *European Addiction*  
560 *Research*, 14(4), 226–233. <https://doi.org/10.1159/000156479>

561 McCrady, B. S., & Smith, D. E. (1986). Implications of Cognitive Impairment for the Treatment of  
562 Alcoholism. *Alcoholism: Clinical and Experimental Research*, 10(2), 145–149.  
563 <https://doi.org/10.1111/j.1530-0277.1986.tb05061.x>

564 Mulhauser, K., Weinstock, J., Ruppert, P., & Benware, J. (2018). Changes in Neuropsychological  
565 Status during the Initial Phase of Abstinence in Alcohol Use Disorder: Neurocognitive  
566 Impairment and Implications for Clinical Care. *Substance Use & Misuse*, 53(6), 881–890.  
567 <https://doi.org/10.1080/10826084.2017.1408328>

568 Munro, C. A., Saxton, J., & Butters, M. A. (2000). The neuropsychological consequences of  
569 abstinence among older alcoholics: a cross-sectional study. *Alcoholism, Clinical and*

570 *Experimental Research*, 24(10), 1510–1516. <https://doi.org/10.1097/00000374-200010000->  
571 00008

572 Nolin, P. (1999). Analyses psychométriques de l'adaptation française du California Verbal Learning  
573 Test (CVLT). = Psychometric analyses of the French version of the California Verbal Learning  
574 Test (CVLT). *Revue Québécoise de Psychologie*, 20(1), 39–55. Retrieved from  
575 [https://www.researchgate.net/profile/Pierre\\_Nolin/publication/264925983\\_ANALYSES\\_PSYC](https://www.researchgate.net/profile/Pierre_Nolin/publication/264925983_ANALYSES_PSYC)  
576 [HOMETRIQUES\\_DE\\_L'ADAPTATION\\_FRANCAISE\\_DU\\_CALIFORNIA\\_VERBAL\\_LEA](https://www.researchgate.net/profile/Pierre_Nolin/publication/264925983_ANALYSES_PSYC_HOMETRIQUES_DE_L'ADAPTATION_FRANCAISE_DU_CALIFORNIA_VERBAL_LEARNING_TEST_CVLT/links/53f5f22e0cf2888a7491fbd3/ANALYSES-PSYCHOMETRIQUES-DE-LADAPTATION-FRANCAISE-DU-CALIFORN)  
577 [RNING\\_TEST\\_CVLT/links/53f5f22e0cf2888a7491fbd3/ANALYSES-PSYCHOMETRIQUES-](https://www.researchgate.net/profile/Pierre_Nolin/publication/264925983_ANALYSES_PSYC_HOMETRIQUES_DE_L'ADAPTATION_FRANCAISE_DU_CALIFORNIA_VERBAL_LEARNING_TEST_CVLT/links/53f5f22e0cf2888a7491fbd3/ANALYSES-PSYCHOMETRIQUES-DE-LADAPTATION-FRANCAISE-DU-CALIFORN)  
578 [DE-LADAPTATION-FRANCAISE-DU-CALIFORN](https://www.researchgate.net/profile/Pierre_Nolin/publication/264925983_ANALYSES_PSYC_HOMETRIQUES_DE_L'ADAPTATION_FRANCAISE_DU_CALIFORNIA_VERBAL_LEARNING_TEST_CVLT/links/53f5f22e0cf2888a7491fbd3/ANALYSES-PSYCHOMETRIQUES-DE-LADAPTATION-FRANCAISE-DU-CALIFORN)

579 Nowakowska-Domagala, K., Jabłkowska-Górecka, K., Mokros, Ł., Koprowicz, J., & Pietras, T.  
580 (2017). Differences in the verbal fluency, working memory and executive functions in  
581 alcoholics: Short-term vs. long-term abstainers. *Psychiatry Research*, 249, 1–8.  
582 <https://doi.org/10.1016/j.psychres.2016.12.034>

583 Oscar-Berman, M., Shagrin, B., Evert, D. L., & Epstein, C. (1997). Impairments of brain and  
584 behavior: the neurological effects of alcohol. *Alcohol Health and Research World*, 21(1), 65–75.  
585 Retrieved from <http://www.ncbi.nlm.nih.gov/pubmed/15706764>

586 Oscar-Berman, M., Valmas, M. M., Sawyer, K. S., Ruiz, S. M., Luhar, R. B., & Gravitz, Z. R. (2014).  
587 Profiles of impaired, spared, and recovered neuropsychologic processes in alcoholism. In  
588 *Handbook of Clinical Neurology* (pp. 183–210). [https://doi.org/10.1016/B978-0-444-62619-](https://doi.org/10.1016/B978-0-444-62619-6.00012-4)  
589 [6.00012-4](https://doi.org/10.1016/B978-0-444-62619-6.00012-4)

590 Parsons, O. A. (1977). Neuropsychological Deficits in Alcoholics: Facts and Fancies. *Alcoholism:*  
591 *Clinical and Experimental Research*, 1(1), 51–56. <https://doi.org/10.1111/j.1530->  
592 [0277.1977.tb05767.x](https://doi.org/10.1111/j.1530-0277.1977.tb05767.x)

593 Petit, G., Luminet, O., Cordovil De Sousa Uva, M., Zorbas, A., Maurage, P., & De Timary, P. (2017).  
594 Differential spontaneous recovery across cognitive abilities during detoxification period in  
595 alcohol-dependence. *PLoS ONE*, 12(8), 1–18. <https://doi.org/10.1371/journal.pone.0176638>

596 Pitel, A.-L., Zahr, N. M., Jackson, K., Sassoon, S. A., Rosenbloom, M. J., Pfefferbaum, A., &  
597 Sullivan, E. V. (2011). Signs of preclinical Wernicke's encephalopathy and thiamine levels as  
598 predictors of neuropsychological deficits in alcoholism without Korsakoff's syndrome.  
599 *Neuropsychopharmacology: Official Publication of the American College of*  
600 *Neuropsychopharmacology*, 36(3), 580–588. <https://doi.org/10.1038/npp.2010.189>

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

605 Pitel, A. L., Rivier, J., Beaunieux, H., Vabret, F., Desgranges, B., & Eustache, F. (2009). Changes in  
606 the episodic memory and executive functions of abstinent and relapsed alcoholics over a 6-month  
607 period. *Alcoholism, Clinical and Experimental Research*, 33(3), 490–498.  
608 <https://doi.org/10.1111/j.1530-0277.2008.00859.x>

609 Reed, R. J., Grant, I., & Rourke, S. B. (1992). Long-term abstinent alcoholics have normal memory.  
610 *Alcoholism, Clinical and Experimental Research*, 16(4), 677–683.  
611 <https://doi.org/10.1111/j.1530-0277.1992.tb00660.x>

612 Ritz, L., Coulbault, L., Lannuzel, C., Boudehent, C., Segobin, S., Eustache, F., ... Beaunieux, H.  
613 (2016). Clinical and Biological Risk Factors for Neuropsychological Impairment in Alcohol Use  
614 Disorder. *PLOS ONE*, 11(9), 1–14. <https://doi.org/10.1371/journal.pone.0159616>

615 Ros-Cucurull, E., Palma-Álvarez, R. F., Cardona-Rubira, C., García-Raboso, E., Jacas, C., Grau-  
616 López, L., ... Roncero, C. (2018). Alcohol use disorder and cognitive impairment in old age  
617 patients: A 6 months follow-up study in an outpatient unit in Barcelona. *Psychiatry Research*,  
618 261, 361–366. <https://doi.org/10.1016/j.psychres.2017.12.069>

619 Rosenbloom, M. J., Pfefferbaum, A., & Sullivan, E. V. (2004). Recovery of Short-Term Memory and  
620 Psychomotor Speed but Not Postural Stability With Long-Term Sobriety in Alcoholic Women.  
621 *Neuropsychology*, 18(3), 589–597. <https://doi.org/10.1037/0894-4105.18.3.589>



622 Rourke, S. B., & Grant, I. (1999). The interactive effects of age and length of abstinence on the  
623 recovery of neuropsychological functioning in chronic male alcoholics: a 2-year follow-up study.  
624 *Journal of the International Neuropsychological Society : JINS*, 5(3), 234–246.  
625 <https://doi.org/10.1017/S1355617799533067>

626 Rupp, C. I., Kemmler, G., Kurz, M., Hinterhuber, H., & Fleischhacker, W. W. (2012). Cognitive  
627 remediation therapy during treatment for alcohol dependence. *Journal of Studies on Alcohol and*  
628 *Drugs*, 73(4), 625–634. <https://doi.org/10.15288/jsad.2012.73.625>

629 Stavro, K., Pelletier, J., & Potvin, S. (2013). Widespread and sustained cognitive deficits in  
630 alcoholism: a meta-analysis. *Addiction Biology*, 18(2), 203–213. [https://doi.org/10.1111/j.1369-](https://doi.org/10.1111/j.1369-1600.2011.00418.x)  
631 [1600.2011.00418.x](https://doi.org/10.1111/j.1369-1600.2011.00418.x)

632 Stephan, R. A., Alhassoon, O. M., Allen, K. E., Wollman, S. C., Hall, M., Thomas, W. J., ... Grant, I.  
633 (2017). Meta-analyses of clinical neuropsychological tests of executive dysfunction and  
634 impulsivity in alcohol use disorder. *American Journal of Drug and Alcohol Abuse*, 43(1), 24–43.  
635 <https://doi.org/10.1080/00952990.2016.1206113>

636 Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental*  
637 *Psychology*, 18(6), 643–662. <https://doi.org/10.1037/h0054651>

638 Tapert, S. F., Ozyurt, S. S., Myers, M. G., & Brown, S. A. (2004). Neurocognitive ability in adults  
639 coping with alcohol and drug relapse temptations. *The American Journal of Drug and Alcohol*  
640 *Abuse*, 30(2), 445–460. <https://doi.org/10.1081/ADA-120037387>

641 Van der Linden, M., Coyette, F., Poitrenaud, J., Kalafat, M., Calicis, F., Wyns, C., ... GREMEM.  
642 (2004). L'épreuve de rappel libre/ rappel indicé à 16 items (RL/RI-16). In *L'évaluation des*  
643 *troubles de la mémoire: présentation de quatre tests de mémoire épisodique avec leur*  
644 *étalonnage* (pp. 25–47). Marseille: Solal.

645 Wechsler, D. (1997). *WAIS-III administration and scoring manual*. The Psychological Corporation,  
646 San Antonio, TX.

647 Whiting, D. L., Deane, F. P., Simpson, G. K., McLeod, H. J., & Ciarrochi, J. (2017). Cognitive and  
648 psychological flexibility after a traumatic brain injury and the implications for treatment in  
649 acceptance-based therapies: A conceptual review. *Neuropsychological Rehabilitation*, 27(2),  
650 263–299. <https://doi.org/10.1080/09602011.2015.1062115>

651