Alcohol & Alcoholism Vol. 40, No. 6, pp. 498–503, 2005
Advance Access publication 26 September 2005
doi:10.1093/alcalc/agh203

IS THERE COGNITIVE IMPAIRMENT IN CLINICALLY 'HEALTHY' ABSTINENT ALCOHOL DEPENDENCE?

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(*First received* 30 *November* 2004; *first review notified* 9 *January* 2005; *in final revised form* 26 *July* 2005; *accepted* 27 *July* 2005; *advance access publication* 26 *September* 2005)

Abstract — Aims: The aim of this study was to determine neuropsychological performance in apparently cognitively, mentally, and physically healthy abstinent alcohol-dependent subjects compared with control subjects who were recruited for a number of different neuroimaging studies. **Methods:** All subjects completed a battery of neuropsychological tests as part of the neuroimaging protocol. **Results:** The group dependent on alcohol performed as well as controls on a non-verbal memory test and verbal fluency but performed worse in the verbal memory task, Trail A + B, and total IQ derived from Silverstein's short-form of the WAIS-R. However, the IQ performance of both groups was above average. In both groups, age was associated with slower performance on the Trail A + B task. In the alcohol-dependent group, severity of dependence and length of abstinence was not associated with performance of any task. **Conclusions:** In this apparently clinically healthy population of abstinent alcohol-dependent subjects, frontal lobe dysfunction was detectable using the Trail A + B and digit symbol tasks. This was despite above-average WAIS-R IQ scores. Consideration needs to be given to routine incorporation of cognitive testing in alcohol dependence since subtle deficits may not be easily apparent and may impact on treatment outcome.

INTRODUCTION

Alcohol misuse is common in the UK, with more than one in four men and one in seven women drinking more than the recommended limits (Office for National Statistics, 2000). Excessive alcohol intake has numerous physical consequences (Arria and Van Thiel, 1992) including liver disease, gastrointestinal problems, and neurological complications. Alcohol misuse has adverse social (Caetano, 1993) and psychological consequences (Ross *et al*., 1988; Marshall and Alam, 1997).

Cognitive deficits occur commonly in alcohol dependence (Parsons, 1977) and may arise through direct toxic effects of alcohol or withdrawal, associated deficiency of vitamins such as thiamine, or via cirrhosis of the liver. Deficits in problem solving, verbal and non-verbal abstraction, visuo-motor coordination, learning, and memory have been reported (Tarter and Edwards, 1985; Parsons, 1998). In severe cases gross deficits may be evident, including Wernicke's encephalopathy (Harper *et al*., 1986), Korsakoff's syndrome (Kopelman, 1995) and alcohol-related dementia. Many cognitive studies in alcohol dependence utilise chronically dependent clinical and/or inpatient populations who have been referred to clinical psychologists. Tuck and Jackson (1991) reported that male patients with no apparent neurological disorder showed significant impairment on a variety of neuropsychological tests compared with controls, and postulated that such subtle cognitive deficits may precede alcohol-related gross neurological disorders by up to ten years. Indeed, heavy so-called social alcohol drinking has been shown to result in poor neuropsychological performance in tasks that can also be impaired in alcoholism (Waugh *et al*., 1989; Page and Cleveland, 1987). There is limited published evidence relating to cognitive deficits in 'healthy' abstinent alcohol-dependent individuals that are not clinically obvious and would not generally warrant referral to a clinical psychologist.

In the present study we report on the neuropsychological performance of subjects with and without alcohol dependence who were otherwise healthy and had no clinically obvious cognitive problems. Subjects had all been recruited for one of a number of neuroimaging studies (Lingford-Hughes *et al*., 1998, 2000, 2002, 2005). A battery of tests was performed to include cognitive functions likely to be affected by alcohol dependence e.g. problem solving, executive functions, visuo-spatial processing, and less likely to be affected, e.g. vocabulary.

METHODS

Subjects

One hundred and four subjects were recruited for one of a number of different neuroimaging single photon emission tomography and positron emission tomography studies measuring GABA-benzodiazepine receptor levels in the brain (Lingford-Hughes *et al*., 1998, 2000, 2002, 2005). Forty-three abstinent alcohol-dependent subjects who fulfilled DSM IV criteria for alcohol dependence and fifty-eight control subjects, who had never fulfilled such criteria were recruited. Three more subjects were recruited for the neuroimaging studies but were familiar with the neuropsychological package and so did not complete the battery. As part of the protocol, subjects also received a structural MRI scan that was assessed by a clinical neuroradiologist. No scans were identified as having significant atrophy. In the control group, three currently

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drank no alcohol and in their lifetime had consumed no more than one drink per year. The alcohol-dependent group was recruited from local outpatient Alcohol services and the control group, through colleagues and advertisements. Alcohol-dependent subjects had been abstinent from alcohol for a minimum of 6 weeks. The majority of patients were in the early few months of abstinence, resulting in a skewed distribution (9 median = 5.25 months, mean = 23.5 months; Kolmogorov–Smirnov < 0.01). A range of assessments of abstinence was used, depending on the study, including reports from their key-worker, liver function tests, routine breathaliser testing. For those patients undergoing residential rehabilitation or attending day patient abstinence focused treatment programme, relapse would have been noted and unexplained non-attendance led to exclusion from the study. In general if there was any suspicion patients had been drinking, they were excluded. All but one study aimed to recruit men only (for reasons concerning radiation exposure).

The subjects recruited for all these studies all had no clinically apparent sequelae of their alcohol dependence such as abnormal liver function test or cognitive impairment or warranted referral for medical or psychological opinions. The population was, therefore, referred to as being 'clinically healthy' and representative of many individuals receiving treatment for alcohol dependence. Therefore, subjects with clinical evidence of hepatic, cognitive, or neurological impairment or medical disorder, including epilepsy were excluded. Recreational or occasional use of illicit drugs (e.g. cannabis, Ecstasy), but not dependency (DSM IV defined) was admissible. Subjects with a history of psychosis were excluded (The Schedule of Affective Disorders and Schizophrenia—Lifetime; Endicott, 1978).

Anxiety and depression were assessed using the Beck Depression Inventory (BDI; Beck, 1961) and Spielberger State—Trait Anxiety Inventory (STAIS, STAIT; Spielberger, 1983). The neuropsychological testing was performed generally within a few weeks and during a stable period in their drinking behaviour. All subjects were assessed for their severity of dependency (The Severity of Dependency Questionnaire; SADQ; Stockwell *et al*., 1983).

Eight abstinent alcohol-dependent and eleven control subjects had previously suffered from major depression. Six abstinent alcohol-dependent subjects were receiving antidepressant medication (specific serotonin reuptake inhibitors, venlafaxine, amitriptyline) and one control subject (clomipramine). Removing these subjects from the analyses performed did not substantially alter the results found with the significant differences between the groups remaining. Analysis without these subjects removes the effects of medication rather than those with higher depressive ratings since there were some individuals in both groups who were not taking antidepressant medication and reported higher scores on the BDI. The BDI was not completed at the time of the neuropsychological testing, and therefore may not have reflected mental state at this time. Therefore, the relationship between BDI score and other neuropsychological variables were not explored.

Neuropsychological battery

The following neuropsychological tests were generally completed in one sitting lasting between 60 and 90 min. Subtests of the Wechsler Adult Intelligence Scale [WAIS-R (Wechsler, 1981)] including verbal tests (i) vocabulary, and (ii) arithmetic, and performance tests (1) block design, (2) picture arrangement, and (3) digit symbol were chosen. Age graded scaled scores were converted to verbal, performance, and total IQ with Silverstein's four subtest short-form (verbal: vocabulary, and arithmetic; performance: picture arrangement, and block design) (Silverstein, 1982). Verbal memory was tested with the Weschler Memory Scale [WMS(R); Wechsler, 1987]: logical memory (immediate and 30-min delayed recall) and visuo-spatial memory with the Rey–Osterrieth Complex Figure Test (R–OCF) (Taylor, 1959). In addition, the Trail Making Tests (Trail A and B) from the Halstead–Reitan Battery (Reitan, 1955) and verbal fluency using the 'controlled oral word association test' (CWAT) (Benton and Hamsher, 1976) with letters FAS were administered. All testing was conducted by psychiatrists (A.L-H., A.F., S.J.C.D., S.A.P., and B.J.S.). A.L.H. had received training in neuropsychological test administration from a clinical neuropsychologist (S.B.) and trained the remaining psychiatrists. Although the administering clinicians were not blind to the diagnosis of the subjects, results were analysed by a rater who was blind to subject identity.

All studies received approval from the appropriate local ethics and research committees.

Statistical analysis

Data relating to each neuropsychological test were examined using Student's *t*-test, regression analysis, and Spearman's correlation with the SPSS statistical package. The Kolmogorov–Smirnov test was used to test for normal distribution of scores.

RESULTS

Data from 101 subjects were available, 43 were alcohol-dependent subjects and 58 were controls. The majority of subjects were men, but there was no significant difference in the representation of women between the groups, with female subjects accounting for 21% of the alcohol-dependent group and 29% of the controls (see Table 1; results are described as mean ± SD throughout). Due to the small number of women, formal comparisons between the genders were not conducted; however, no obvious different patterns of impairment were evident. There was no significant difference in age (control: 43.0 ± 8.6; alcohol-dependent: 43.7 ± 8). As

Table 1. Characteristics of control and abstinent alcohol-dependent subjects

	Control group	Alcohol-dependent group	*P*-value
Number	58	43	
Males	41 (71%)	34 (79%)	NS
Age (years)	43.0 ± 8.6	43.7 ± 8	NS
Length of abstinence (months)	N/A	23.5 ± 42.4	
SADQ	2.2 ± 3.4	37.0 ± 13.3	<0.01
Beck Depression Inventory (BDI)	4.7 ± 4.2	9.8 ± 8.6	<0.01
Spielberger State Anxiety	36.9 ± 9.8	38.9 ± 10.1	NS
Spielberger Trait Anxiety	38.7 ± 8.0	43.1 ± 11.4	0.03

Results are described as mean ± SD. N/A, not available; NS, non-significant.

anticipated, the SADQ score was significantly higher in the alcohol-dependent group (control: 2.2 ± 3.4; alcohol-dependent: 37.0 ± 13.3), where scores over 31 denote severe dependence (Stockwell *et al*., 1983). The length of abstinence ranged from 3 weeks to 180 months, though accurate data were not available for three subjects (23.5 ± 42.4 months). Although the BDI score was higher in the alcohol-dependent group (9.8 ± 8.6) compared with controls (4.7 ± 4.2), this higher level is still below the threshold for mild depression (Beck, 1961). The STAIT (control: 38.7 ± 8.0; alcohol-dependent: 43.1 ± 11.4), but not STAIS (control: 36.9 ± 9.8; alcohol-dependent: 38.9 ± 10.1), was significantly different between the two groups ($P \leqslant 0.05$; Table 1). Data were missing in the control group for one SADQ, seven STAIS, three STAIT, and five BDI, and in the alcohol-dependent group for three BDI. These questionnaires were not filled in at the time of the neuropsychological evaluation, so may not necessarily reflect their mental state at the time of neuropsychological testing; however, all tests were completed in a stable period of abstinence from alcohol.

Neuropsychological outcome measures

The total IQ derived from the WAIS-R Silverstein short-form (Silverstein, 1982) was significantly lower in the alcohol-dependent group compared with the control group (control: 117.0 ± 12.0; alcohol-dependent: 110.9 ± 14.7; Table 2; $P \leqslant 0.05$ uncorrected). However, in both groups the IQ was above average. The performance scores for the individual tasks contributing to the total IQ are also described in Table 2. The alcohol-dependent group performed significantly worse ($P \leqslant 0.05$, uncorrected) on the vocabulary (control: 14.1 ± 2.7; alcohol-dependent: 12.5 ± 3.3; Table 2) and digit symbol (control: 10.9 ± 2.5; alcohol-dependent: 9.3 ± 2.7; Table 2) tasks of the WAIS-R. There were no significant differences in performance in the other tasks: arithmetic, picture arrangement, or block design (Table 2). The pro-rated verbal and performance IQ derived from Silverstein (1982) (verbal: vocabulary + arithmetic; performance: picture arrangement + block design) were also not significantly different between the two groups. After correcting for multiple comparisons with Bonferroni correction, only performance on the digit symbol task remained significantly lower in the alcohol-dependent compared to control group ($P \leqslant 0.003$). The total IQ and pro-rated verbal and performance IQ scores showed a normal distribution ($P > 0.05$).

The alcohol-dependent group performed significantly worse on a verbal memory task, the Wechsler logical memory task, than the control group. On immediate recall, the alcohol-dependent group recalled significantly fewer items, 12.0 ± 3.5 compared with 13.3 ± 3.0 in the control group and on delayed recall, the scores were 10.1 ± 3.9 and 11.7 ± 3.2, respectively ($P \leqslant 0.05$, uncorrected). This gave combined scores i.e. immediate ± delayed of 25.0 ± 6.0 in the control group and 22.2 ± 7.1 in the alcohol-dependent group which were significantly different ($P \leqslant 0.05$, uncorrected). None of these differences between the groups remained significant after Bonferroni correction ($P < 0.003$).

By contrast, assessment of non-verbal memory with the R–OCF task revealed no significant differences in the performance between the alcohol-dependent group and the control group (Table 2). When copying the R–OCF, there was little difference between the two groups [total score (maximum = 36); controls: 34.7 ± 2.7 vs alcohol-dependent: 34.3 ± 2.7], and although the alcohol-dependent group performed slightly worse than the control group on immediate recall and delayed recall at 20 min, the differences were not significant (Table 2).

Table 2. Scores from the individual neuropsychological tasks from which outcome scores were derived

	Controls ($n = 58$)	Alcohol-dependent ($n = 43$)	Uncorr. P-value
WAIS-R tests			
Vocabulary: age scaled scores	14.1 ± 2.7	12.5 ± 3.3	0.006
Arithmetic: age scaled scores	12.0 ± 2.7	11.9 ± 3.1	NS
Picture arrangement: age scaled scores	13.7 ± 2.8	12.6 ± 3.4	NS
Block design: age scaled scores	12.7 ± 3.3	12.1 ± 2.9	NS
Digit symbol: age scaled scores	10.9 ± 2.5	9.3 ± 2.7	0.002*
Pro-rated verbal IQ	114.6 ± 12.2	109.7 ± 15	NS
Pro-rated performance IQ	114.5 ± 11.4	109.8 ± 13.3	NS
Pro-rated full scale IQ	117.0 ± 12.0	110.9 ± 14.7	0.024
Trail-Making Tests			
Trail A (s)	33.0 ± 10	36.9 ± 12.8	NS
Trail B (s)	66.1 ± 23.9	84.4 ± 42.9	0.007
Total: Trail A ± B (s)	99 ± 28	121 ± 51	0.007
WMS: logical memory test			
Immediate recall	13.3 ± 3.0	12.0 ± 3.5	0.05
30-min recall	11.7 ± 3.2	10.1 ± 3.9	0.03
Total Wechsler memory score: (immediate + recall).	25.0 ± 6.0	22.2 ± 7.1	0.033
R–OCF Test			
Copy	34.7 ± 2.7	34.3 ± 2.7	NS
Immediate recall	24.4 ± 7.4	21.6 ± 7.9	NS
Delayed recall (20 min)	23.8 ± 7.2	21.8 ± 7.4	NS
Verbal fluency	53.9 ± 18.2	54.4 ± 15.5	NS
(CWAT-FAS)	56.8 ± 13.4	55.7 ± 13	

Results are described as mean ± SD. Uncorr., uncorrected; NS, non-significant.
*Significant after Bonferroni correction for multiple comparisons; $P < 0.003$.

The alcohol-dependent group took significantly longer than controls to complete the Trail B task (control: 66.1 ± 23.9 vs alcohol-dependent: 84.4 ± 42.9 s; P (0.05), whilst there was no significant difference in time taken to complete Trail A (control: 33.0 ± 10 vs alcohol-dependent: 36.9 ± 12.8 s). Combined Trail A + B scores were 99 ± 29 s in the control group vs 121 ± 51 s in the alcohol-dependent group ($P \leqslant 0.05$, uncorrected). None of these differences remained significant after Bonferroni correction ($P < 0.003$).

Lastly, there were no significant differences between the control and alcohol-dependent groups in their verbal fluency in performing the controlled word association test. The control group generated 53.9 ± 18.2 words and the alcohol-dependent group generated 54.4 ± 15.5 words (see Table 2).

Regression analysis was undertaken to explore the influence of age and trait anxiety (STAIT score) on a single outcome measure reflecting the tasks that were significantly different between the control and alcohol-dependent group i.e. pro-rated full scale IQ, Trail A + B, and total score at Wechsler logical memory task. Other demographic details were not investigated since they may not have reflected mental state at the time of neuropsychological testing. After controlling for age, the two groups remained significantly different for Trail A + B scores, pro-rated full scale IQ and Wechsler logical memory task scores ($P \leqslant 0.05$). After controlling for

trait anxiety (STAIT score), the difference between the two groups for the Trail A + B scores was still significant but not for pro-rated full scale IQ or Wechsler logical memory task.

The relationship between neuropsychological task performance and clinical measures was also explored for each group of subjects individually. A significant positive correlation was found between age and Trail A + B in the control group (r = 0.269; P = 0.04), and between age and Trail B, and Trail A + B in the alcohol-dependent group (r = 0.333, P = 0.028; r = 0.348, P = 0.022). No significant correlations were found between age and pro-rated full scale IQ, Wechsler logical memory total score (immediate, delay, and total), verbal fluency, or R–OCF score (delay recall/copy was selected to represent this task). Trait anxiety was also not found to be significantly associated with any of these outcome measures in either group. Lastly, no significant correlations were found between these outcome measures with SADQ score or length of abstinence in the alcohol-dependent group.

The relationship between total IQ with task performance Trail A + B, verbal fluency, R–OCF score (delay recall/copy was selected to represent this task), and total score at Wechsler logical memory task was explored. Analysing the group together, total IQ correlated with Trail A + B (r = –0.626, $P < 0.05$), verbal fluency (r = 0.355, $P < 0.05$), Wechsler logical memory total score (r = 0.557, $P < 0.05$), and R–OCF score (delay/copy; r = –0.250, $P < 0.05$). In the alcohol-dependent group, total IQ correlated with Trail A + B (r = –0.701, $P < 0.05$), verbal fluency ($r = 0.486$, $P < 0.05$), Wechsler logical memory total score (r = 0.630, $P < 0.05$), but not R–OCF score (delay/copy; $r = –0.274$, $P > 0.05$). In the control group, total IQ correlated with Trail A + B (r = –0.471, $P < 0.05$), Wechsler logical memory total score (r = 0.434, $P < 0.05$), but not verbal fluency (r = 0.244, $P > 0.05$) or R–OCF score (delay/copy; r = –0.228, $P > 0.05$). Concerning Trail A + B, the time taken to complete the Trail B part of the task, and not Trail A, was significantly correlated with total IQ in both groups. This analysis, therefore, shows that the contribution of total IQ to the variance in performance scores on these tasks was always greater in the alcohol-dependent group compared to the controls. As an example, an increase in IQ by 10 points resulted in faster performance in Trail A + B by 11 s in the control group and by 23 s in the alcohol-dependent group.

DISCUSSION

In a population of apparently clinically healthy abstinent alcohol-dependent subjects we found impaired frontal lobe function as evidenced by poorer task performance on the Trail Making Test and digit symbol test of the WAIS-R compared with control subjects. This was despite an above-average IQ score derived from the WAIS-R Silverstein short-form (Silverstein, 1982) in the alcohol-dependent group. In addition, verbal memory was impaired in the alcohol-dependent group with poorer recall on the WMS evident. This is consistent with previous studies showing frontal lobe dysfunction (Moselhy *et al*., 2001), and adds to the literature since this alcohol-dependent outpatient population studied showed no signs of cognitive impairment, and were in good medical and mental health.

The Trail-making tests of the Halstead–Reitan Battery have been widely used in assessing cognitive impairment and, in particular, frontal lobe function in alcohol misuse. Trail B is described as testing visuo-spatial scanning skills, divided attention, suppression of the impulse to revert to the more familiar task of merely connecting numbers, and of coping with the shifting paradigm. Although much of the early literature focussed on patients where cognitive deficits were clinically obvious, such as patients with confabulation characteristic of Korsakoff's syndrome or frank alcoholic dementia (Brown *et al*., 1958), subsequent studies (Loberg, 1980; Eckhardt and Matarazzo, 1981; Moselhy *et al*., 2001) showed that performance on the Trail B could be impaired in alcohol dependence not associated with any clinically obvious neurological deficits. More recently, Noel *et al*. (2001) also reported that 'non-amnesic' alcohol-dependent subjects were slower on Trails A and B, and similar to our study, greater impairment was seen in completing the Trail B. Comparing this study with ours, the performance in the control groups was similar but the alcohol-dependent group in the present study performed better than in Noel *et al*. (2001), which may reflect the fact that their subjects were inpatients and ours were outpatients with a longer period of abstinence.

Other tasks that reflect psychomotor speed and frontal lobe function were also incorporated into our battery. Impaired performance on the digit symbol task of the WAIS-R has been previously reported and was chosen because of this (Moselhy *et al*., 2001). Of all the subtests of the WAIS-R chosen, it was the only test for which significant differences remained after correction for multiple testing. By contrast, performance in the verbal fluency task by our population of alcohol-dependent subjects was not impaired compared with controls. Noel *et al*. (2001) also used a verbal fluency task and found no difference in their group of 'non-amnesic' alcohol-dependent subjects abstinent for ~3 weeks. Ratti *et al*. (1999) also found no difference but notably this was in the presence of widespread cortical and sub-cortical atrophy in their population of 'heavy alcohol drinkers'. However, Joyce and Robbins (1991) did find impaired verbal fluency but the average age of their population was 53, a decade older than in the present study.

Noel *et al*. (2001) specifically set out to re-examine the frontal brain vulnerability hypothesis and found that performance at easier or early stages of tasks showed little, if any, impairment of executive functions. However, as previously reported by Joyce and Robbins (1991) impairment was evident at more difficult tasks, or where realising and addressing a mistake was required. The digit symbol task requires all of these skills. The Trail B requires greater levels of flexibility and exploring planning ability compared with Trail A. It appears, therefore, that Trail B has sufficient complexity compared with Trail A and other easily deliverable frontal tasks such as verbal fluency, to be of clinical use in determining if there is any frontal lobe dysfunction prior to overt clinical signs.

We found impairment in performance in the verbal memory task (Wechsler logical memory scale) but not non-verbal memory task (R–OCF). Such impaired verbal memory has been reported elsewhere, in the presence of no significant difference in the WAIS full scale IQ (Joyce and Robbins, 1991; Ratti *et al*., 1999). The Silverstein (1982) short-form

of the WAIS-R battery (Wechsler, 1981) was used to generate verbal, performance, and full scale IQ scores. The difference seen in the full scale IQ between the groups was driven by lower scores on the digit symbol and vocabulary tasks in the alcohol-dependent group. We did not see a significant difference in the block design that has been widely reported to be impaired in alcohol dependence, and it was chosen for this purpose (Loberg and Miller, 1986). The reason for this might be that the population we studied is healthier than those previously described. The poorer performance on the vocabulary test in the alcohol-dependent group is not consistent with previous literature, and possibly reflects the fact that many controls were recruited through colleagues in academic institutions (Miller and Orr, 1980; Loberg, 1980; Smith and Smith, 1977). In addition, the mean vocabulary score (12.5) for the alcohol-dependent group puts this group in the highest 25 percentile, suggesting little, if any, impairment. It was notable that both abstinent alcohol-dependent and control groups had IQ scores greater than the population mean. This may have been due to the fact they were recruited for neuroimaging studies and required a good understanding of the concepts involved. The fact that the volunteers who had been alcohol-dependent were, as a group, functioning above the population norm in terms of global IQ scores reinforces the point that the cognitive deficits elicited are sufficiently subtle as to be only identifiable by targeted neuropsychological testing.

The fact that total IQ (derived from vocabulary, arithmetic, block design, and picture arrangement) correlated with performance at verbal and visual memory tasks, and the two reflecting frontal lobe functions (Trail A + B; verbal fluency) when the groups are analysed together and separately is not surprising given that it is likely the same factors, such as alcohol consumption, influenced their scores. A significant correlation between performance at the Trail A + B task with intellectual ability has been previously reported (Yeudall *et al*., 1987). However, a study in healthy individuals suggests that the difference seen here between the two groups is greater than would be expected just from different IQ levels. The difference in performance at Trails A + B tasks in people with above average IQ (115) compared with those with average (97) or superior (125) was only between 2 and 5 s, whereas in our study a much greater difference of 20 s was seen between the two groups (Waldmann *et al*., 1992).

We found no correlation between task performance on the total IQ of the WAIS-R, the Trail Making Test, WMS in the alcohol-dependent group with their severity of their alcohol dependence, nor length of abstinence. Although cognitive impairment has been shown to be related to abstinence, the amount, and length of alcohol abuse, this is not consistently so (Eckardt *et al*., 1995; Parsons, 1998; Mann *et al*., 1999; Noel *et al*., 2001). For example, Eckardt *et al*. (1995) reported better function in the Trail B task in alcohol-dependent patients abstinent for >70 days compared with those for <14 days. Similarly, Mann *et al*. (1999) reported that after 6-week abstinence, improvement in the range of neuropsychological tasks was seen, including Trail B. Noel *et al*. (2001) found no correlation with age, lifetime amount of alcohol drunk, and length of abuse. The duration of abstinence was less (19.8 ± 2.8 days) than in the current study (∼750 days). It may be that 'recovery' occurs early in the course of abstinence and the fact that the majority of patients in our study had been abstinent for at least 3 months precluded finding a relationship between abstinence and task performance in abstinent alcohol-dependent subjects who appear clinically healthy.

The implications of our findings for treatment are considerable. Deficits in executive functioning, learning, and concentration are likely to lead to poor treatment outcomes in alcohol-dependent patients, even those who appear to be 'healthy'. Higher levels of cognitive functioning in patients were shown in preliminary analysis to be associated with greater likelihood of a successful completion of inpatient treatment, to fewer relapses, longer abstinence periods, and lower rates of alcohol consumption at 1 year (O'Leary *et al*., 1979). In addition, poor abstracting and problem solving skills have been shown to be associated with clinicians' rating of likelihood of treatment success (Leber *et al*., 1985). If benefits are to be derived from identifying subtle cognitive deficits among alcohol-dependent subjects, the question arises as to how this can be achieved. Whilst a comprehensive history is essential to the assessment of any patient with an alcohol problem, it may offer few clues specific to the elucidation of subtle cognitive problems. Patients with alcohol dependence often voice worries about their memory that are more likely to be a reflection of impaired attention and concentration. Questionnaires often used for screening, diagnosis, and further characterization of alcohol misuse such as AUDIT (Conigrave *et al*., 1995), and Paddington Alcohol Test (Smith *et al*., 1996), Alcohol Problem Questionnaire (Drummond, 1990), and the SADQ (Stockwell *et al*., 1983), were not designed to assess cognitive difficulties. In addition, patients may not be aware of the existence of subtle impairments.

In conclusion, cognitive testing deserves a prominent role in the routine clinical assessment of patients with alcohol problems, alongside assessment of the severity of dependence, co-existing depression, and anxiety because it may have considerable impact on outcomes. This study has shown that despite appearing clinically 'healthy' and scoring above average IQ derived from the WAIS-R, this group of alcohol-dependent subjects had clear deficits on further testing. Whilst clinicians are aware that neuropsychological impairment occurs in alcohol dependence, cognitive testing does not often occur regularly during assessment, nor is it taken into account in treatment planning. We propose that the Trail A and B making test, in particular the Trail B section, and the digit symbol test should be considered for routine clinical use in the assessment and treatment of alcohol dependence, even in apparently cognitively, mentally, and physically healthy patients. Whilst digit symbol may not be easily available or deliverable, this does not apply to the Trail Making Test. This test requires minimal training, can be easily used by a range of workers, takes about 5 min to complete, and requires no special equipment.

Acknowledgements — The neuroimaging studies were funded through a Wellcome Foundation Training Fellowship to A.L.H., with E.J.M., and R.W.K., and an MRC Programme Grant to D.J.N.

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