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Smoking is associated with neurocognitive deficits in alcoholism^{*}

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Abstract

Background— Impaired problem solving, visual–spatial processing, memory, and cognitive proficiency are consequences of severe alcoholism. Smoking is much more prevalent among alcoholics than the general population, yet the possible neurocognitive effects of cigarette smoking in alcoholism have not been studied, despite evidence that long-term smoking is associated with neurocognitive deficits.

Objective— Determine whether smoking contributes to neurocognitive deficits associated with alcoholism.

Design— Neurocognitive function was examined in a community-recruited ($n = 172$) sample of men. Alcohol problems/alcoholism were measured by the lifetime alcohol problems score (LAPS), DSM-IV diagnosis, and monthly drinking rate. Smoking was measured in pack-years. Neurocognitive function was measured with IQ (short version of WAIS-R), and cognitive proficiency (fast, accurate performance).

Results— Both alcoholism and smoking were negatively correlated with neurocognitive function. When alcoholism and smoking were included in regression models, smoking remained a significant predictor for both measures, but alcoholism remained significant only for IQ.

Conclusions— Both smoking and alcoholism were related to neurocognitive function. Smoking may explain some of the relationship between alcoholism and neurocognitive function, perhaps especially for measures that focus on proficiency. Future studies are necessary to more fully understand the effects of smoking on neurocognitive function in alcoholism.

Keywords

Cognitive function; Alcoholism; Cigarette smoking

1. Introduction

Alcohol use disorders entail considerable personal and economic costs (Anthony et al., 1997) and a substantial literature documents neurocognitive deficits in severely and chronically alcoholic men (Brewer and Perrett, 1971; Butters et al., 1977; Knight and Longmore, 1994;

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Parsons and Leber, 1981). These include difficulties with visual-spatial processing, problem solving, memory, and cognitive proficiency (Ahveninen et al., 2000; Nixon et al., 1995, 1998). Thus it is well established that chronic alcoholism is associated with neurocognitive deficits. However, the specific type and magnitude of the cognitive deficit caused by alcoholism has been difficult for the field to agree upon, resulting in multiple descriptive neuropsychological theories (Gilman et al., 1998; Knight and Longmore, 1994; Oscar-Berman, 2000; Parsons and Farr, 1981; Parsons and Leber, 1981; Sullivan et al., 1993).

This variability likely reflects multiple pathways and moderators of brain and cognitive dysfunction among persons with alcoholism (Adams and Grant, 1986; Tarter and Edwards, 1986), including comorbid psychological and medical conditions. In particular, abuse of drugs other than alcohol is common with alcoholism and may also affect neurocognitive function (Nixon et al., 1998; Rogers and Robbins, 2001). As a result, studies have increasingly taken into account the impact of other illicit substances in cognitive studies of alcoholism (Nixon et al., 1998). In striking contrast, nicotine use/cigarette smoking is virtually unstudied in this regard even though people with alcoholism also have high rates of smoking. Estimates of cigarette smoking among persons with alcoholism range from 47% to 87%, compared to 18% to 28% in the general population in the United States (John et al., 2003; Marks et al., 1997) and approximately 33% globally (World Health Organization, 2002). Nonetheless, nicotine is rarely mentioned in papers where other drugs (e.g. marijuana, cocaine) were excluded or controlled. This is a potentially important gap in understanding because chronic cigarette smoking is linked to decreased cognitive performance, and thus may explain some of the cognitive decline in alcoholism.

Cigarette smoking has two potentially different effects on neurocognitive performance: an acute effect of nicotine and a chronic effect due to long-term smoking (related to health effects such as vascular disease and chronic obstructive pulmonary disease). Acute administration of nicotine leads to some improvement in cognitive performance, especially on tasks of sustained attention or vigilance (Ernst et al., 2001; Lawrence et al., 2002; Rezvani and Levin, 2001). This effect is present even in patients with alcoholism (Craddock et al., 2003). On the other hand, chronic cigarette smoking is associated with cognitive impairment in several epidemiological studies (Cervilla et al., 2000). Smokers perform more poorly than both never-smokers and ex-smokers on tests of general cognitive function (Galanis et al., 2000), working memory (Ernst et al., 2001; Jacobsen et al., 2005), psychomotor speed (Hill, 1989; Kalmijn et al., 2002), cognitive flexibility (Kalmijn et al., 2002), and verbal memory and visual search (Richards et al., 2003). Berkman (Berkman et al., 1993) reported that among adults age 70–79 years, those classified as “high functioning” smoked fewer cigarettes. Meyer (Meyer et al., 1986) found that among multi-infarct dementia patients, improved cognitive function was related to cessation of smoking. Smokers also self-report more everyday and long-term prospective memory errors than non smokers (Heffernan et al., 2005). In sum, although there are beneficial *acute* effects of nicotine on cognitive performance, the *long-term* effects of smoking are clearly negative and seem most pronounced on measures of neurocognitive function that emphasize the ability to rapidly and flexibly process information.

We hypothesized that smoking may contribute to neurocognitive impairment in alcoholics. Because smoking is prevalent in alcoholism, previous studies of cognitive deficits in alcoholism may have been subject to the unknown and unmeasured influence of the different rates and duration of smoking in the alcoholic and control groups. This hypothesis is consistent with recent findings that cigarette smoking is associated with increased neuronal injury in chronic alcoholism (Durazzo et al., 2004). Regression analyses were used to examine the relationships of smoking and alcoholism with neurocognitive function. Because most of the literature has addressed men and we wished to evaluate the likely effects in this literature, we studied a sample of men with chronic alcoholism and controls.

2. Methods

2.1. Participants

Participants were 172 men who had completed the MicroCog Assessment of Cognitive Functioning (Powell et al., 1994) battery as part of an ongoing, prospective, multi-wave study that is following a community sample of families with high levels of alcohol use disorder (AUD), along with a community contrast sample of families drawn from the same neighborhoods but without the high substance abuse profile (Zucker et al., 2000). We restricted our analyses to men only because the original recruitment strategy (described below) resulted in a significantly higher density of male alcoholic participants than female alcoholic participants. Families are assessed every 3 years. The cognitive data are from Wave 4 of testing, except for 33 cases where the MicroCog battery was not obtained during Wave 4. For those cases, Wave 5 data were substituted. Written informed consent, approved by the University of Michigan Medical School Institutional Review Board and the Michigan State University Institutional Review Board, was obtained prior to study.

The families involved in the longitudinal study were initially ascertained through men identified by a network covering all courts in a four county wide area. All men with a drunk driving conviction involving a blood alcohol concentration of at least 0.15% if first conviction (or at least 0.12% if a previous drinking-related legal problem had occurred) were potential study candidates. In addition, they were required to meet diagnosis for probable or definite alcoholism (Feighner et al., 1972) and, due to offspring studies also conducted with this sample, have at least one biological son between 3 and 5 years of age and be living with the child and his biological mother. Since family recruitment was based on the men's alcoholism, substantially fewer women were alcoholic and their *N* was insufficient to permit parallel analysis.

A contrast/control group of nonalcoholic families (neither parent alcoholic) residing in the same neighborhoods as the alcoholic families was recruited using door-to-door canvassing. This strategy also resulted in the recruitment of an intermediate risk group, since some families with an alcohol abuse/dependence diagnosis were found during the community canvass. Although the original recruitment used the Feighner et al. criteria, in later years all parents were re-diagnosed using the alcohol abuse/dependence criteria of the DSM-IV. A more detailed description of study procedures, recruitment strategies and eligibility criteria is found in Zucker et al. (2000). Full assessments involving both parents and participating children occurred at 3 year intervals, starting at baseline (Wave 1).

2.2. Clinical behavioral assessment

2.2.1. Alcoholism—Alcohol use disorders (AUD) were assessed at Wave 1 by the diagnostic interview schedule (DIS) Version III (Robins et al., 1980), the short michigan alcohol screening test (SMAST) (Selzer, 1975), and the drinking and drug history questionnaire. This questionnaire incorporates items from the 1978 NIDA survey (Johnston et al., 1979) and from the American drinking practices survey (Cahalan et al., 1969). All of the items have been extensively used in a variety of survey and clinical settings. They provide data on quantity, frequency and variability of alcohol consumption, frequency of drug use, and multiple questions on consequences and troubles related to the use of these substances. On the basis of information collected by all three instruments, a diagnosis of AUD lifetime, as well as current (met criteria during past 3 years) was made at Waves 1 and 4 (Wave 5 for those cases with Wave 5 cognitive data) by a trained clinician using DSM-IV criteria (American Psychiatric Association, 2000). A dichotomous variable was created from the DSM-IV diagnoses (diagnosis of dependence ever in lifetime versus no diagnosis of dependence). Out of the sample, 103 met a lifetime diagnosis (DSM-IV) of alcohol dependence, (mean age = 43 years

(S.D. = 5.4), mean education = 14 years (S.D. = 3.2)) and 69 had never met diagnostic criteria for dependence (mean age = 42 years (S.D. = 4.8), mean education = 15 years (S.D. = 2.4)). The group difference in education was not significant. Among those who had ever met diagnosis, 40 had both past and current diagnoses (i.e. met diagnostic criteria at Wave 4 or 5 and at Wave 1) and 63 had a past but not current diagnosis.

Other measures of alcohol problem severity were also used: the average number of alcoholic drinks per month over the study time period from Waves 1–4 (9 years at Wave 4, for most participants or 12 years for those for whom we used Wave 5 cognitive data), the average number of alcoholic drinks per month during the 6 months prior to testing, and the lifetime alcohol problems scale (LAPS) (Zucker et al., 1997). LAPS has been validated with regard to diagnosis, alcohol-specific problems, and having been in treatment (Zucker et al., 1997). It provides a dimensional measure of extensiveness of alcohol problems over the life course and includes three components: onset (measured as age of first drunkenness), breadth (i.e. number of different alcohol-related problems encountered over the lifetime), and a life percent component that scales the degree to which alcohol-related problems have been present in a Pearson's life.

¹ The LAPS score was highly correlated with diagnosis (presence or absence of alcoholism; Pearson $r = .705$, $p < .01$). The average (across the time of study) number of drinks per month was calculated with data from the drinking and other drug history questionnaire, administered at each wave of testing. Two items were used to calculate average monthly drinking rate. Participants indicated: (1) how many days per month they drank over the past 6 months, and (2) how many drinks were consumed on average on drinking days. The final score was the monthly drinking rate averaged over all four waves. Data from the past 6 months from the current wave of testing (Wave 4) was used to measure recent alcohol consumption.

2.2.2. Smoking—Rate of smoking was also obtained from the drinking and drug history questionnaire. At the time of cognitive testing, 106 participants (mean age = 43 years; S.D. = 4.8, mean education = 15 years; S.D. = 3.0) reported no smoking or less than one cigarette per day over the 30 days prior to testing. Sixty-six participants reported smoking “one to five cigarettes per day” or more (mean age = 43 years; S.D. = 5.5, mean education = 13 years; S.D. = 2.8). Education was significantly lower among those who reported current smoking, $t(173) = 3.03$, $p < .01$. The median number of cigarettes smoked per day for those who reported smoking was “about one pack per day”. As expected, the prevalence of smoking was higher for the alcohol-dependent group (63% current smokers) than the control group (24% current smokers). A continuous variable, called *pack-years*, was created by multiplying average daily use (in packs) by the number of years smoking. Daily use was estimated from self-reported rate of smoking from Waves 1–4 or 5. Years of smoking was estimated from self-reported age at smoking onset. Pack-years was used as the index of lifetime amount of smoking in the regression analyses.

2.2.3. Drug use and other comorbidities—Data on the use of drugs other than alcohol or cigarettes were also gathered from the drinking and drug history questionnaire, Wave 4 (or Wave 5). Participants were asked on how many occasions during the last 3 years they had used: marijuana, LSD, psychedelics other than LSD, cocaine, amphetamines, quaaludes, barbiturates, tranquilizers, heroin, narcotics other than heroin, or sniffed glue. Marijuana had the highest frequency of use, with 24% of the sample reporting any use, followed by cocaine (6.4%) and amphetamines (4.5%). A composite variable of any drug use was computed by summing the frequency of use for each of the individual drug categories, for each participant. For the total drug-use score, 25% of the sample reported some use. Depression at the time of

¹LAPS = 10 + Z (component A) + Z (component B) + Z (component C). Component A = 100/(age first drunk)², component B = number of problem areas ever experienced, component C = [(age most recent problem experienced – age first problem experienced) + 1]/respondent's age².

testing was measured by clinician ratings using the Hamilton depression scale (Hamilton, 1960). Data concerning health status (heart and lung disease, loss of consciousness, head injury, HIV infection) that may affect cognitive function was obtained from the study's Wave 4 or 5 health history questionnaire. Thirty-eight percent of participants reported the presence of any heart or lung disease. No participants reported a loss of consciousness during the past 3 years, no participants reported being HIV positive, and only two participants reported a head injury during the past 3 years.

2.3. Neurocognitive assessment

2.3.1. Procedure—Assessment occurred in the participant's home. A home-based protocol was used to increase participation and cooperation. Several precautions were instituted to ensure valid administration of the measures. Testing occurred at a table in a room of the home that permitted adequate privacy and limited the possibility of distractions. Breaks were allowed between measures, but participants were told that interruptions during testing would not be permitted. Since testing did not take place in a controlled environment, such as a treatment center, care was taken to ascertain whether a participant had recently consumed alcohol or other drugs. A short questionnaire was completed to assess barriers to collecting valid data (e.g. how much sleep the respondent had the previous night, any use of alcohol, recreational drugs, or prescription drugs in the past 24 h). If a person reported more than two alcoholic drinks within the hour prior to testing, or appeared intoxicated or "high" to the administrator, the assessment was not administered at that time and was rescheduled for another date. Only ten participants reported drinking alcoholic beverages on the day of testing. Participants could smoke before the assessment and during breaks. Therefore, participants who were regular smokers were not tested in a nicotine withdrawal state and to the extent that recent cigarette smoking improves cognitive function this should have acted to counteract any findings with regard to cigarette smoking.

2.3.2. Cognitive proficiency—Proficiency, which refers to an index combining speed and accuracy, has been a more sensitive measure of neurocognitive function in alcoholism than accuracy alone (Glenn and Parsons, 1992; Nixon et al., 1995, 1998), and information processing proficiency impairments are reported for smokers as we noted earlier. To assess cognitive proficiency, we administered the short form of the MicroCog battery (Powell et al., 1994). This computerized battery tests six distinct aspects of neurocognitive function in a standardized automated format. The cognitive components include short-term memory (similar to digit span forward), immediate and delayed story recall, verbal reasoning (analogies), mathematical reasoning, and visual-spatial processing. As noted earlier, visual-spatial processing has been one hypothesized area of difficulty in relation to alcoholism. Error rates and response latency were automatically recorded for all tests. The program combines errors and response times into proficiency scores for each cognitive component. High proficiency scores indicate fast, accurate performance. The program also calculates a global proficiency score by combining the component proficiency scores. The global score, as might be expected, has better reliability than the individual component scores. A further benefit of the global proficiency score is that it is normalized with respect to age and education. This is of crucial importance because education in our sample was negatively correlated with both alcoholism ($r = -.188, p < .05$) and smoking ($r = -.253, p < .01$).

2.3.3. Intelligence—IQ, one of the most widely accepted and psychometrically well-established index of intellectual functioning (Sattler, 2001), was estimated with a four-subtest short form of the WAIS-R (Reynolds et al., 1983) consisting of information, picture completion, arithmetic, and block design. This short form predicts full scale IQ in normal adults (Reynolds et al., 1983) and in neurologically impaired individuals (Ryan, 1985). Evidence

supports the use of the short form for characterizing group performance (Silverstein, 1990). The estimated IQ score is normalized for age of the respondent.

2.4. Analyses

To examine the hypothesis that smoking may be associated with neurocognitive function, we conducted correlation (Pearson) analyses and linear regression analyses for each of the two global neurocognitive measures. Our primary analyses focused on the global measures because of their desirable psychometric properties and age and education normative correction. Predictor variables for the multivariate regression models included LAPS, pack-years, the interaction term for LAPS and pack-years, Hamilton current depression score, and other drug use. The interaction term was not significant in any of the regressions performed, and thus is omitted in the subsequent regression models. Predictor variables were entered simultaneously.

For completeness of description, we also report the results for the individual component proficiency scores from the MicroCog battery. We included education in these regressions since the component scores were not normalized for education. Other drug use was not correlated with any of the component measures and so is omitted.

To present the means and S.D. for the neurocognitive measures, participants were characterized as control (never met alcohol diagnosis at any time), past, but not current alcohol-dependence diagnosis, or current alcohol-dependence diagnosis. Diagnoses were based on DSM-IV criteria. Participants were also characterized as current smokers (smoked one or more cigarettes per day in the past 30 days), or non-smokers.

3. Results

Participant characteristic for the alcoholism groups subdivided by smoking are shown in Table 1. Zero order correlations (Pearson) between the neurocognitive measures and the smoking, alcoholism, and comorbidity variables are shown in Table 2. As expected, the smoking score (pack-years) was correlated with worse performance on both of our main neurocognitive indices. Among the alcoholism problems/alcoholism measures, LAPS was most highly and consistently correlated with worse neurocognitive function and therefore was the measure used for subsequent regression analyses. Means and S.D. for the neurocognitive measures for alcoholism groups subdivided by smoking are shown in Table 3.

Multiple linear regression analyses were conducted to examine the combined effects of smoking and alcoholism rather than the more common group analyses (e.g. ANOVA), since pack-years and LAPS are both continuous measures. To be sure that the observed associations between neurocognitive function, smoking and alcoholism in the whole sample are not driven simply by clustering of the smoking or drinking scores at the lower and higher ends for the control and alcoholic groups, respectively, correlations were computed with the sample restricted to alcoholic participants only. Pack-years and LAPS were both significantly negatively related to IQ and global proficiency in this restricted sample (Pearson $r = -.200$ to $-.235$, $p < .05$). For our main measures of neurocognitive function (IQ and global proficiency, Table 4), smoking was a significant unique predictor even with alcoholism controlled. Alcoholism dropped to nonsignificant for global cognitive proficiency, indicating that smoking statistically mediated the association between drinking and neurocognitive function on this measure. Alcoholism, however remained a significant unique statistical predictor for IQ, even with smoking controlled. Other drug use was not a significant predictor for either neurocognitive measure. The interaction between alcoholism and smoking was not significant for either measure (data are not shown).

Among the component cognitive proficiency measures, pack-years was significantly associated with all six measures (Pearson $r = -.194$ to $-.275$, $p < .01$). LAPS was also significantly associated with all six measures (Pearson $r = -.145$ to $-.276$, $p < .05$). Multiple linear regressions for each component measure (Table 5) were conducted, including LAPS, pack-years, as well as depression and education (since the component scores are not normalized for education). Alcoholism was significantly associated with less efficient mathematical reasoning, even with smoking and education controlled. Smoking was significantly associated with weaker verbal and visual-spatial reasoning, even with alcoholism and education controlled.

4. Discussion

Lifetime alcohol problem severity (as measured by LAPS), was significantly correlated with poorer neurocognitive function, consistent with previous research (Brewer and Perrett, 1971; Butters et al., 1977; Glenn and Parsons, 1992; Knight and Longmore, 1994; Nixon et al., 1995; Parsons and Leber, 1981). However, an important portion of this association, as measured by proficiency, was mediated by concurrent lifetime smoking history. This effect was not explained by concurrent illicit drug use history, health or current depression and held with scores normalized for age and education.

First, it is important to note that we confirmed an association of chronic alcohol use with neurocognitive weakness; smoking did not account for all of the associations between neurocognitive function and alcoholism. Second, however, as noted in the introduction, there are likely multiple paths that lead to the association of chronic excessive drinking and cognitive impairments, including other drug use. We found that comorbid chronic smoking statistically explained some of the observed cognitive impairments associated with drinking, particularly global cognitive proficiency. In view of the widespread smoking in alcoholic samples and the virtually universal neglect of this potential confound in cognitive studies of alcoholism this result may require re-assessment of prior findings. Crucially, this effect was over and above the effects of presence or absence of illicit drug use (marijuana was the most common in this sample), indicating that nicotine is an additional substance that should be controlled and considered in alcoholism research.

Although we do not know the direction of causal effects from this analysis, one possibility is that smoking could lead over time to poor cognitive proficiency. For example, we speculate that chronic smoking may have damaging effects on the brain either via direct neurotoxic action (Jacobsen et al., 2005) or by reducing blood flow. Risks for cardiovascular disease (Burns, 2003a,b; Foody et al., 2001; Kalmijn et al., 2002) and chronic obstructive pulmonary disease (COPD) (Burns, 2003b; Sutherland and Martin, 2003) are increased by smoking. In turn, both cardiovascular disease (DeCarli, 2003; Geroldi et al., 2003) and chronic obstructive pulmonary disease (Grant et al., 1987; Heaton et al., 1983; Prigatano et al., 1983) are associated with reduced neurocognitive function. The fact that our data show a dose-related effect between smoking and poorer neurocognitive function is in accord with this view.

Our data also indicated that global cognitive proficiency is more strongly related to smoking than to alcoholism. In prior studies, effects of smoking seem to be most pronounced on measures that emphasize rapid, flexible information processing, consistent with the reduced cognitive proficiency observed in the present study. Therefore, the pattern of findings is consistent with the causal relationship just suggested.

However, an alternative causal effect is also possible. Less proficient neurocognitive function may be a predisposing factor for initiating cigarette smoking (Dinn et al., 2004). The fact that education and smoking are negatively correlated in our sample is consistent with this

possibility. However, the fact that the global cognitive proficiency score is normalized for age and education renders it less likely that what we observed here was simply due to those with lower education being more likely to smoke and to have lower proficiency scores. Likewise, the fact that some of the component cognitive proficiency findings held with education controlled suggests we are detecting effects other than a spurious effect of low education driving low proficiency and smoking. Nonetheless, further evaluation of causal direction of effects is clearly needed.

Other limitations should be borne in mind in this initial study. Our analyses included only men and it is not known if the results would generalize to women. On average, the severity of alcoholism in this community-based sample is lower than might be expected in a treatment-based sample; this may have reduced our ability to find relationships between alcohol consumption measures and neurocognitive function. Examination of the relationship between smoking, alcoholism and neurocognitive function was limited by the fact that smoking and alcoholism were confounded in this naturalistic sample. As a result, it was difficult to assess possible interactions between smoking and alcoholism. In addition, a wide range of potentially important cognitive functions were not assessed in this initial study, most notably executive function. Nonetheless, the study features a population-based sample of well characterized alcoholic and control men followed over a 9-year period or longer. The results should thus have reasonable generalizability, although it will be important in the future to study similar effects in women.

In summary, our results show that smoking is related to neurocognitive performance. It statistically mediated the relationship between alcoholism and cognitive proficiency. To our knowledge, this is the first report to document these effects. Further study is warranted to replicate this finding.

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Participant characteristics by alcoholism and smoking groups

Table 1

	Controls		Formerly alcoholic		Currently alcoholic	
	Smoke—	Smoke+	Smoke—	Smoke+	Smoke—	Smoke+
<i>N</i>	56	13	34	29	16	24
Age (years; mean (S.D.))	42.1 (4.6)	41.9 (5.8)	42.9 (5.0)	43.1 (6.5)	45.1 (4.5)	42.3 (4.6)
Education (years; mean (S.D.))	15.1 (2.3)	13.8 (2.6)	14.2 (3.9)	13.2 (3.8)	14.1 (3.3)	13.2 (1.8)
LAPS (mean (S.D.))	7.7 (1.7)	9.2 (0.9)	11.1 (1.6)	11.1 (2.0)	11.7 (0.9)	12.5 (1.9)
Drinking rate (long-term; median (range))	2.5 (96)	8.0 (80)	18.5 (93)	21.0 (341)	49.0 (200)	82.5 (368)
Drinking rate (6 month; median (range))	2.0 (72)	5.0 (60)	2.0 (60)	8.0 (240)	39.0 (200)	60.0 (240)
Recent drinking (24 h)	<i>N</i> = 0	<i>N</i> = 0	<i>N</i> = 0	<i>N</i> = 3	<i>N</i> = 2	<i>N</i> = 5
Pack-years (mean (S.D.))	0.4 (2.2)	15.0 (21.7)	1.4 (5.1)	26.8 (19.6)	4.3 (9.3)	27.0 (16.5)
Health	<i>N</i> = 15	<i>N</i> = 3	<i>N</i> = 10	<i>N</i> = 6	<i>N</i> = 8	<i>N</i> = 9
Depression (mean (S.D.))	4.3 (5.0)	5.2 (6.6)	4.3 (5.1)	4.0 (4.6)	4.1 (4.1)	6.8 (7.7)

Note: Formerly alcoholic indicates a DSM-IV diagnosis of alcohol dependence from any of the previous test Waves (1–3), but no dependence diagnosis at Wave 4. Smoke–, no smoking in the past 30 days at Wave 4 testing; Smoke+, current smoker; LAPS, lifetime alcohol problems score; drinking rate, mean number of alcoholic drinks consumed per month, averaged over entire 9-year study period (Waves 1–4; long-term) or 6 months prior to Wave 4 testing; recent drinking, number of participants who reported consuming any alcoholic drink in 24 h prior to testing; health, number of participants who reported any heart or lung disease; depression, mean Hamilton current depression score.

Table 2

Correlations between global neurocognitive function, measures of alcoholism, smoking, depression and health

	IQ	Cognitive proficiency
Pack-years	-.286**	-.203**
LAPS	-.314**	-.170*
Alcohol diagnosis (none/current/past)	-.171*	.012
Alcohol diagnosis (lifetime, yes/no)	-.210**	-.039
Drinking rate (long-term)	-.210**	-.016
Drinking rate (past 6 months)	-.101	-.016
Recent drinking (past 24 h)	.073	.055
Other drug use	-.169*	-.050
Current depression	-.025	-.239**
Health	-.021	.022

Note: LAPS, lifetime alcohol problems score; drinking rate, mean number of alcoholic drinks consumed per month averaged over entire 9-year study period (Waves 1–4; long-term) or 6 months prior to Wave 4 testing; health, presence of heart or lung disease; depression, Hamilton current depression index.

*
 $p < .05$.

**
 $p < .01$.

Table 3

Neurocognitive means and S.D. by alcohol and smoking group

	Controls		Formerly alcoholic		Currently alcoholic	
	Smoke–	Smoke+	Smoke–	Smoke+	Smoke–	Smoke+
IQ	113.3 (11.5)	104.1 (15.2)	108.1 (14.3)	103.1 (10.2)	108.2 (12.5)	104.8 (13.6)
Cognitive proficiency	95.11 (15.7)	90.1 (14.4)	94.1 (18.3)	89.6 (12.1)	102.5 (12.3)	91.8 (18.8)
Short-term memory	12.0 (4.8)	10.9 (4.9)	10.5 (4.0)	10.6 (4.2)	12.4 (4.8)	10.4 (5.5)
Immediate recall	4.8 (1.3)	4.3 (1.7)	4.5 (1.3)	4.5 (1.1)	5.1 (1.2)	4.3 (1.4)
Delayed recall	8.8 (2.4)	7.6 (2.7)	8.1 (2.4)	8.0 (2.3)	9.9 (2.6)	8.0 (2.7)
Verbal reasoning	4.2 (1.2)	3.4 (1.3)	3.9 (1.3)	3.4 (1.2)	4.1 (1.3)	3.5 (1.2)
Mathematical reasoning	3.6 (1.4)	2.8 (1.8)	3.0 (1.5)	2.8 (1.4)	3.3 (1.3)	2.7 (1.3)
Visual–spatial reasoning	4.3 (0.8)	4.1 (0.7)	4.1 (0.8)	3.9 (0.6)	4.3 (0.5)	3.8 (0.7)

Note: Formerly alcoholic, DSM-IV diagnosis of alcohol dependence from any of the previous test waves (1–3) but no dependence diagnosis at Wave 4; Smoke–, no smoking in the past 30 days at Wave 4 testing; Smoke+, current smoker, all table entries except IQ represent MicroCog proficiency scores (accuracy scaled by response speed).

Table 4
Multiple regression results for alcoholism and smoking predicting global neurocognitive function

	Outcome variables	
	IQ	Cognitive proficiency
Standardized regression coefficients		
LAPS	-.229**	-.127
Pack-years	-.230**	-.168*
Other drug use	-.058	.073
Depression	.038	-.195**
R^2 (model)	.152	.097

Note: LAPS, lifetime alcohol problems score; depression, Hamilton current depression score.

*
 $p < .05$.

**
 $p < .01$.

Table 5
Multiple regression results for alcoholism and smoking predicting individual component MicroCog proficiency scores

Outcome variables					
STM					
	I-recall	D-recall	V-reasoning	M-reasoning	V-P-reasoning
Standardized regression coefficients					
LAPS	-.045	-.095	-.096	-.201**	-.133*
Pack-years	-.100	-.083	-.142*	-.077	-.153*
Depression	-.170*	-.277**	-.196**	-.087	-.171*
Education	.255**	.244**	.385**	.352**	.269**
R ² (model)	.127	.179	.267	.228	.193

Note: LAPS, lifetime alcohol problems score; depression, Hamilton current depression score; STM, short-term memory; I-recall, immediate recall; D-recall, delayed recall; V-reasoning, verbal reasoning; M-reasoning, mathematical reasoning; V-P-reasoning, visual-spatial reasoning.

* $p < .05$.
** $p < .01$.