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Effects of Alcoholism Severity and Smoking on Executive Neurocognitive Function

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Abstract

Aims—Neurocognitive deficits in chronic alcoholic men are well documented and include impairments in memory, visual-spatial processing, problem solving and executive function. The cause of these deficits is unclear, but could include direct effects of alcohol toxicity, pre-existing cognitive deficits that may predispose towards substance abuse, comorbid psychiatric disorders (e.g., depression) and comorbid abuse of substances other than alcohol. For example, cigarette smoking occurs at a much higher rate among persons with alcoholism and has been linked to poor cognitive performance. Until recently, the negative effects of smoking on cognitive function in alcoholism have been ignored.

Methods—The effects of alcoholism and smoking are examined in a community recruited sample of alcoholic and non-alcoholic men (N=240) using standard neuropsychological measures and reaction-time measures of executive function. Alcoholism severity was measured as an average of alcoholism diagnoses across the study duration (12 yrs). Smoking was measured in pack-years.

Results—Both alcoholism and smoking were negatively correlated with a composite executive function score. For component measures, alcoholism was negatively correlated with a broad range of measures, whereas smoking was negatively correlated with measures that emphasize response speed. In regression analyses, both smoking and alcoholism are significant predictors of executive function composite. However; when IQ is included in the regression analyses, alcoholism severity is no longer a significant predictor.

Conclusions—Both smoking and alcoholism were related to executive function. However, the effect of alcoholism on EF was not independent of IQ, suggesting that the alcoholism effect was generalized, perhaps affecting a wide range of cognitive abilities of which executive function is a component. On the other hand, the effect of smoking on measures relying on response speed were

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independent of IQ, suggesting a more specific processing speed deficit associated with chronic smoking.

Alcohol use disorders (AUD) represent a major public health problem with substantial costs (1;2). Neurocognitive deficits in severe, chronic alcoholic men (3-7) are well documented and include visual-spatial processing, problem solving, memory, and cognitive proficiency (3). Problem solving and higher-order impairments led to theories of frontal lobe dysfunction; recent examinations of neurocognitive function in alcoholics have focused on executive function (8;9) in part because neuroimaging studies have linked alcoholism to changes in brain structure and function, especially the frontal lobes and their connections to other brain areas, including the cerebellum (10-15).

Although alcoholism is known to have neuropsychological consequences linked with brain impairment, the direct versus indirect role of alcohol toxicity is unclear (16;17). Several cognitive abilities are theoretically related to substance use onset and outcome, especially executive function because it is linked to poor planning and impulsive behavior (2;18). Thus, some cognitive impairment observed in alcoholism may have been present before the onset of alcohol use. Alcoholism is typically accompanied by co-morbid conditions, such as depression, that negatively impact cognitive function. Abuse of drugs other than alcohol is more prevalent in AUD and also impact cognitive function (9;19-22).

For example, cigarette smoking occurs at a much higher rate among alcoholic samples than in the general population. Surprisingly, the negative effects of smoking on neurocognitive function have been largely ignored until recently (23-25). Smoking has two potentially different effects on neurocognitive performance: an acute effect of nicotine that may be beneficial (26-30) and a chronic effect due to long-term smoking. Impairments among smokers compared to non-smokers include general cognitive function (31), working memory (26;32), psychomotor speed (33;34), cognitive flexibility (34), and verbal memory and visual search (35;36).

Previously, we reported that smoking mediated the effect of alcoholism on cognitive proficiency (rapid *and* accurate cognitive function), and that both smoking and alcoholism were negatively related to IQ. The current study examines effects of alcoholism and smoking in a community sample of alcoholic and non-alcoholic men using neuropsychological and reaction-time measures of executive function. Executive function is defined here as the ability to maintain an appropriate mental set in order to fulfill a future goal (37;38). It involves planning, filtering competing information, maintaining a goal despite distraction, and inhibiting goal-inconsistent responses.

The constructs of intelligence and executive function overlap in that both involve higher-order cognitive functions and the ability to learn and adapt (39;40). Neuropsychological measures of executive function correlate with IQ (although the correlations are not large) (41-43). In some models, EF is a component of general intelligence (44). Therefore, the interpretation of distinct versus overlapping cognitive effects may not be straightforward.

Nonetheless, intelligence and executive function are at least partially separable (45), though the extent of this separation remains a point of contention in the literature (46;47). A classic neuropsychological observation is that injury to the frontal lobes can dramatically impair performance on tests of executive function, whereas IQ is relatively insensitive to such injuries (42). In this paper we examine whether chronic alcoholism and chronic smoking have effects on executive function and on response speed that are separable from IQ.

To the extent that executive function impairments exist in our sample, they may be part of a more general impairment, or they may be specific to executive function. Furthermore, IQ and

executive function may be more highly correlated among alcoholics than non-alcoholics. In comparison to controls, alcoholics use higher-order executive functions to perform a perceptual-learning task (48) and neuroimaging studies suggest increased reliance on frontal lobe systems during cognitive tasks (10;14;15). The recruitment of higher-order functions may be a compensatory strategy leading to a stronger relationship between executive function measures and IQ.

Most studies showing impaired executive/frontal lobe function among alcoholic men have focused on more “macro” (i.e., performance based on several component processes) tests. Other tests offer more specific focus on individual components; one such test is the Stopping Task. The Stopping Task is a reaction-time procedure designed to isolate the ability to inhibit an ongoing behavioral response, an important component of executive function (49;50; 50-52). This test involves a two-choice reaction time task. Occasionally, a “stop signal” occurs to indicate the response should be withheld. Interpretation is based on a cognitive model where there are competing activating and inhibiting processes. The two processes “race” each other, and the process that is completed first “wins”. People with good inhibition have relatively fast inhibition processes and can inhibit prepotent responses more often. The Stopping Task is sensitive to differences in inhibition in many disorders such as ADHD (41;53) and cocaine abuse (54), and normal variations in impulsivity (55).

To our knowledge, the Stopping Task has not been tested in AUD, but there is reason to suspect impairment. Our group reported poorer Stopping Task inhibition in children of alcoholics (18), and Stopping Task inhibition predicts the onset of adolescents' alcohol and drug use (2); those who develop AUD may have a pre-existing weakness in response inhibition. Furthermore, acute alcohol intake selectively disrupts Stopping Task inhibition, but not reaction time to regular (no stop signal) trials (56). AUD may lead to long-term impairment in this ability; thus, poor response inhibition may be both a risk for and a consequence of alcoholism. Since chronic cigarette smoking affects psychomotor and information processing speed (23;33;34); it is expected that smoking will also be associated with slower response inhibition.

In this paper, executive function is examined in a community-recruited sample of alcoholic and non-alcoholic men. Executive function impairments in alcoholic samples from treatment sites have been reported; the current results generalize those findings to a larger population of alcohol abusing men, using both traditional neuropsychological measures and reaction time measures. The effects of smoking on executive function in alcoholism are novel.

Methods

Participants

Data were from 240 men[1] who completed an executive functioning battery as part of Wave 5 data collection in the Michigan Longitudinal Study (MLS) (57), a family study of the development of substance use disorders. Families were recruited through men identified through courts in a four county area. Men with driving convictions involving a blood alcohol concentration of at least 0.15% (first conviction, 0.12% if prior conviction(s)) were potential enrollees if they met diagnosis for probable or definite alcoholism (58) and, had at least one biological son between 3-5 years of age (for family risk studies). The men had to be living with the child and his biological mother. Alcoholic status of the mothers was free to vary. A contrast/ control group of nonalcoholic families was recruited by door to door canvass in the same

[1]The original recruitment was based on male alcoholism among the parents; nonetheless, some alcoholic women were also recruited. On average, severity of alcoholism in the women participants was lower than the men. Effects of alcoholism on the women's neurocognitive function will be reported separately.

neighborhoods as the alcoholic families. This also resulted in recruitment of an intermediate risk group, since some families of parallel composition were identified with alcoholic fathers without a history of alcohol related legal problems. Original recruitment used Feighner criteria (58); thereafter parents were re-diagnosed using DSM-IV AUD (59) criteria. Inter-rater reliability for diagnosis was good (kappa=.81). Detailed description of the study is provided in Zucker et al. (57). Although some participants sought outpatient treatment, either voluntarily or upon court order, the recruitment pathway for this study was not clinic or inpatient based.

Procedure

Data were collected by trained project staff, blind to diagnostic status, as part of the regular data collection for the MLS. Data were collected in individuals' homes, although sometimes it was collected at an office on campus when convenient for the participant. Home administration assured privacy and freedom from distractions. A short questionnaire assessed if there were barriers to collecting valid data (e.g., lack of sleep, immediately prior use of alcohol, recreational or prescription drugs). If two or more alcoholic drinks within the hour prior to testing were reported, or the person appeared intoxicated or “high”, the assessment was rescheduled. Participants were allowed to smoke cigarettes prior to testing and during breaks.

The specific tests were selected to address our hypotheses with a combination of instruments having strong psychometric properties. The focus was executive functioning, broadly construed as a multifaceted construct (18). The assessment battery was constructed with the best evidence-based tools from the neuropsychology literature on alcoholism that addressed inhibition (60), along with the more traditional parameters.

Measures of Executive Control

Stroop Color and Word Test (Stroop Test)—The Stroop Test (61) is a timed task in which the participant has to read through three sets of stimuli (words and colors). For word reading, color names are printed in black ink, for color naming a string of X's (XXXX) are printed in colored ink and in the interference set, color names are printed in colored ink and the participant must name the color of the ink, and resist interference from the printed color word which does not match the ink color (e.g., the word blue printed in red ink). We report the number of items correctly completed in a 90 second interval for each set.

Controlled Oral Word Association Test (COWAT)—The COWAT (62) is a timed test used to measure verbal word fluency. Participants are asked to generate as many words as they can that begin with various letters of the alphabet, during a one-minute period. The score that the participant receives is the sum of all acceptable words produced during three trials.

Wisconsin Card Sorting Test (WCST; (63))—The WCST consists of four stimulus cards and 64 response cards that depict figures of varying forms, colors, and numbers of figures. Participants are told to match each consecutive card from the deck with one of the four stimulus cards. The participant is told whether his/her response is correct or wrong, but is not told the sorting principle involved. The participant must match the sorting principle (color, form, or number) for a specified number of responses before the sorting principle is changed. For this study we used the computer version of the instrument. The variables employed for this study were the number of categories achieved, and the number of perseverative errors.

Paced Auditory Serial Addition Test (PASAT; (64))—The PASAT measures verbal working memory, attention, concentration, and speed of information processing. The test requires the participant to add 60 pairs of randomized digits so that each is added to the digit immediately preceding it. The digits are presented at four rates of speed. A taped representation

is used to provide precise control over the rate at which stimuli are presented. The total number of correct responses was the dependent variable used from this task.

Trail Making Test (TMT; (65))—The TMT is a widely-used measure of visual conceptual and visual-motor tracking as well as set switching. Trails consists of two parts, A and B, with sequential circles containing ascending numbers (Part A), and alternating numbers and letters (Part B). Participants draw lines between the sequential circles with the examiner pointing out errors to the subject as they occur. The amount of time it takes to complete each part is the dependent variable.

Stopping Task (50;52)—During this two-alternative choice reaction time task, participants see an X or an O on a computer screen and respond rapidly with one of two keys. On some trials a tone sounded shortly after the X or O appeared, indicating that participants should withhold the response. After two practice blocks of 32 trials each, four blocks of 64 trials are administered. The final three blocks are averaged unless data quality checks suggest otherwise (53). The most reliable estimates of stop signal reaction time (SSRT) are obtained with a response-reaction time tracking methodology (66) where the delay between the visual stimulus and the warning tone is varied to maintain 50% success rate at withholding the response. A quantitative model of reaction time (RT) processes enables calculation of each participant's speed of stopping or inhibiting a response (SSRT) by subtracting average stop signal delay from average RT to the trials without a stop signal (GoRT) (50;55). This SSRT estimate is a measure of inhibitory control. The GoRT is a measure of response activation, as is the within-subject variability of GoRT.

Measures of Substance Abuse

Alcoholism Severity—Since our alcoholic sample was not recruited from treatment sites, the assignment of participants to either control or alcoholic group is not always clear. Some participants met diagnostic criteria for AUD early in life, but not at the time of testing, others more recently but not earlier, and still others met diagnosis steadily from the time of initial diagnosis. To address this issue of diagnostic variation over time, we developed a continuous variable of alcohol severity. The alcoholism severity index was computed by averaging severity code across T1 (baseline) to T5 (12 year). The severity at each wave was coded as: 0 for negative diagnosis; 1 for alcohol abuse; 2 for alcohol dependence without physical dependence; and 3 for alcohol dependence with physical dependence. The resulting index is a continuous scale ranging from 0 to 3.

Cigarette Smoking—Rate of smoking was obtained from the Drinking and Drug History Questionnaire. At the time of testing, 41.5% reported regular smoking. 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 Study Waves 1 through 5. Years of smoking was estimated from self-reported age at smoking onset to current age. Among those who had ever smoked, average pack-years was 20.17 years.

Other Drug Use—Data on the use of drugs other than alcohol or cigarettes were also gathered from the Drinking and Drug History Questionnaire. 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 22.2% of the sample reporting any use, followed by cocaine (6.1%) and tranquilizers (5.9%). 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, 24.5% of the sample reported some use in the past 3 years.

Depression

Depression at the time of testing was measured by clinician ratings using the Hamilton Depression Scale (67).

Education

The highest level of education achieved at the time of testing was calculated as years of education.

Intelligence (IQ)

IQ, one of the most widely accepted and psychometrically well-established index of intellectual functioning (40), was estimated with a 4-subtest short form of the WAIS-R (68-70) consisting of Information, Picture Completion, Arithmetic, and Block Design.

Data Analyses

Each variable was standardized and if necessary reverse coded. An executive function composite variable was created by averaging each of the variables for each participant.

Missing Data—There were about 10% missing data across variables. Multiple imputation proposed by Rubin was adopted to deal with missing data (71). We assumed *missing at random* (i.e. the probability of response does not depend on missing values) that was satisfied by the protocol of MLS data collection. SAS PROC MI was used to generate 5 plausible alternative versions of the complete data. Each of the 5 data sets was analyzed in the same fashion by a complete data method. The 5 sets of results were then aggregated by using SAS PROC MIANALYZE to obtain overall estimates and standard errors that reflect missing data uncertainty and finite sample variation.

Statistical Analysis—We examined zero-order correlations between smoking, alcoholism severity and executive function; first with the composite variable, and then if significant with the component measures. All probabilities are one-tailed.

Next we conducted a series of regression analyses, starting with the executive-function composite, if significant, we followed up with regression analyses using component measures. For each executive function variable, we tested up to three different regression models. Model 1 investigated whether alcoholism severity and smoking were independent predictors of executive function by including both severity and smoking in the regression model. Note that this model was only tested if both smoking and alcoholism severity were significantly correlated with the particular executive function measure.

Model 2 added education to Model 1, since education was lower in the alcoholic group. Some differences associated with alcoholism could be simply due to lower education attainment rather than dysfunction caused by alcoholism. However, given the lifestyle associated with alcoholism there may be fewer opportunities for education and therefore lower education is part of the syndrome. To examine whether alcoholism causes brain dysfunction, then education should be included to control for its effects. To examine the overall consequences of alcoholism, controlling for education may underestimate the overall effect. Because this is a descriptive paper documenting executive function in a community-based sample, we present our analyses both with and without education included as a predictor.

Model 3 added IQ to Model 1. As with education, whether or not to include IQ as a factor can be argued in either direction. It is theoretically interesting to know whether effects of alcoholism or smoking on executive function are separable from IQ. Thus, we present our regression analyses both with and without IQ included in the models.

Finally, we tested whether executive function and IQ become more correlated with increasing alcoholism severity by dividing creating two dummy variables based on severity. One variable coded diagnosis of abuse or higher as 1, the other variable coded diagnosis of dependence as 1. These variables were included in a regression model along with IQ and the interaction between IQ and the dummy variables to predict executive function (composite).

Results

Correlations

Shown in Table 1 are the zero-order correlations relating alcoholism severity and smoking to IQ, education, other drug use, and depression. Alcoholism was significantly correlated with each of these variables. Pack-years was significantly correlated with each of these variables with the exception of depression.

Table 2 shows the zero-order correlations between the executive function measures and alcoholism severity, smoking, education, depression, other drug use and IQ. Alcoholism severity, smoking, education, depression and IQ were all significantly correlated with the executive function composite score. For the individual executive function scores, alcoholism was moderately negatively correlated with at least one measure from each of the tests. Smoking, on the other hand showed strong correlations with tests that emphasize response speed. Education was positively correlated with all of the executive function measures except GORT and GORT variability. Depression showed moderate correlations with the composite variable, Stroop color-word task, TMT A and B, and GORT variability. Other drug use was not correlated with any executive function measure except SSRT. Table 3 shows the standardized executive function means for each quartile of the alcohol severity measure. For most of the measures, the quartiles show decreasing performance with increasing alcoholism severity.

Regression Models

Table 4 shows the regression coefficients for the three models described above, for each of the executive function measures. The alcoholism by smoking interaction was not significant and was dropped from all models. For Model 1, severity and smoking remained significant predictors of the executive function composite, Stroop word reading, TMT-A, GoRT, GoRT variability, and SSRT, indicating that for these measures alcoholism and smoking have independent effects. For Stroop color naming, smoking dropped out; for this measure alcoholism mediated the effect of smoking.

For Model 2 (education), severity and smoking remained significant predictors for the executive function composite, TMT-A, and GoRT variability. Thus, even with education included both smoking and alcoholism were significant predictors of general executive function and for those measures with an emphasis on response speed. However, the effects of smoking and alcoholism severity were reduced to non-significant levels for Stroop word reading and Stroop color naming. The effects of alcoholism severity were reduced to non-significant levels for COWAT, Stroop color-word, and WCST categories. Alcoholism severity remained a significant predictor of PASAT.

For Model 3 (IQ), the effects of severity drop to non-significant levels for all measures, including the executive function composite. On the other hand, pack-years remained a significant predictor for the composite, TMT-A, GoRT, GoRT variability, and SSRT. Thus, even with IQ included, smoking continued to be a significant predictor of performance on tasks with an emphasis on response speed.

There was no evidence that executive function and IQ became more correlated with increasing severity; the interaction terms between IQ and severity dummy variables were not significant ($p>.06$).

Discussion

This study follows up prior work to attempt to isolate associations of alcoholism and smoking with higher order cognitive abilities. Several interesting results emerged. To begin, among a community recruited sample of alcoholic men and controls, a broad range of executive function measures were significantly correlated with alcoholism severity. This extends the results of previous studies with samples recruited from treatment sites where it is expected that the average severity of alcoholism will be worse than in our sample. The present data show a clear trend towards lower performance in the precise way one would expect given neuropsychological risk from alcohol use, even though the sample is in middle life and not taken from a treatment setting. These subjects, while not performing in clinically significant ranges of impairment, are not performing at the level of a socio-economically and carefully neighborhood-matched non-alcoholic group. However, the effects of alcoholism severity on executive function in our sample were not independent of IQ. Our results could indicate that the executive function effects are subsumed under more general effects on overall intelligence.

The present results also show that smoking is an important variable for some measures of executive function and has a pattern of effects that are distinct from alcoholism. In particular, smoking was correlated with measures that emphasize response speed. This adds to our previous work on smoking and alcoholism effects on neurocognitive function. It also shows that the effects of smoking are quite robust because they remained significant even with education or IQ included in the regression models.

Previous research suggested that smoking may mediate the effects of alcoholism on cognitive proficiency (23). The current results reinforce the importance of smoking when considering cognitive function in alcoholism (24;25;72), and suggest the possibility that the effect of smoking is to primarily slow information processing speed, whereas the effects of alcoholism are more wide-spread across cognitive domains. The effects of smoking on response execution are consistent with other studies that have linked smoking to reduced information-processing speed or efficiency (26;34;36).

Finally the effects of smoking and alcoholism severity on behavioral inhibition measured by the Stopping Task are new. The existing literature indicates executive function deficits in alcoholics from traditional neuropsychological tests that measure more global aspects of executive function. The present study demonstrates that a particular component of executive function, the ability to inhibit a response is also impaired in alcoholics and fits well with the view that drug abuse and addiction involve failures of response inhibition (73;74). Although in this adult sample we cannot determine whether response-inhibition impairments preceded problem alcohol use or were caused by problem alcohol use in our sample, it is nonetheless a critical finding in regards to executive function in alcoholism. With its relation to impulsive, stimulus-driven behavior and the inability to resist short-term gratification, poor response inhibition may underlie or exacerbate problem alcohol use, and likewise has significant implications for treatment outcomes(75-77).

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Table 1

Correlations of Alcoholism Severity and Smoking with Demographic and Comorbidity Variables

	Alcoholism Severity	Smoking Level
Pack-Years	.255***	--------
Education	-.181**	-.219***
Other Drug	.363***	.137*
Depression	.109*	.020
IQ	-.211**	-.195**

* $p < .05$,

** $p < .01$,

*** $p < .001$.

Table 2

Correlations of Predictor Variables with Executive Function.

	Severity	Pack-Years	Education	Depression	Other Drug	IQ
Executive Function Composite						
	243***	-.236***	.353***	-.144**	-.019	.590***
COWAT	-.115*	.038	.318***	-.056	.185	.554***
PASAT	-.221***	-.080	.408***	-.076	-.051	.468***
STROOP-w	-.143**	-.162**	.282***	-.091	-.087	.342***
STROOP-c	-.154**	-.130*	.199***	-.066	.006	.321***
STROOP-cw	-.119*	-.056	.217***	-.113*	.013	.395***
WCST-pe	-.045	-.050	.219***	-.073	.082	.302***
WCST-cat	-.113*	-.078	.165**	-.037	.044	.203***
TMT-A	-.195***	-.229***	.120*	-.187**	-.024	.374***
TMT-B	-.095	-.213***	0.244***	-.120*	.048	.467***
Go-RT	-.181**	-.262***	.082	-.061	-.070	.156**
Go-RT var.	-.194***	-.254***	.184**	-.108*	-.059	.331***
SS-RT	-.167**	-.221***	.099	-.044	-.119*	.294***

* p < .05,

** p < .01,

*** p < .001.

Table 3

Standardized Means (Standard Deviations) for Executive Function by Alcoholism Severity Quartile.

	Quartile 1 N=89	**Quartile 2 N=65**	**Quartile 3 N=53**	**Quartile 4 N=33**
Composite	0.12 (0.57)	0.07 (0.56)	-0.17 (0.60)	-0.19 (0.67)
COWAT	0.15 (0.95)	-0.06 (0.96)	-0.14 (0.93)	-0.02 (1.19)
PASAT	0.17 (0.97)	0.17 (1.10)	-0.40 (0.96)	-0.35 (0.93)
STROOP-w	0.11 (1.00)	0.09 (1.05)	-0.15 (0.93)	-0.17 (0.96)
STROOP-c	0.12 (0.96)	0.06 (1.03)	-0.15 (0.98)	-0.21 (1.11)
STROOP-cw	0.06 (1.00)	0.08 (1.06)	-0.09 (0.94)	-0.20 (0.89)
WCST-pe	-0.06 (1.15)	0.15 (0.75)	-0.03 (0.84)	-0.09 (1.20)
WCST-cat	0.02 (0.97)	0.17 (0.81)	-0.12 (1.08)	-0.22 (1.25)
TMT-A	0.19 (1.02)	0.18 (0.81)	-0.36 (1.14)	-.18 (0.99)
TMT-B	0.11 (1.16)	0.06 (0.90)	-0.25 (0.81)	-0.03 (0.97)
STOP-TASK-goRT	0.19 (0.90)	-0.04 (1.00)	-0.13 (1.09)	-0.31 (1.00)
STOP-TASK-var	0.20 (0.94)	-0.06 (1.04)	-0.13 (1.09)	-0.30 (0.89)
STOP-TASK-stopRT	0.17 (0.94)	0.04 (0.92)	-0.15 (1.02)	-0.24 (1.16)

Table 4
Regression Models predicting Executive Function

	Model 1	Model 2	Model 3
	Coeff. (Std. Err.)	Coeff. (Std. Err.)	Coeff. (Std. Err.)
Composite			
Severity	-0.127*** (0.042)	-0.095** (0.040)	-0.047 (0.035)
Pack-Years	-0.007** (0.002)	-0.005* (0.002)	-0.004* (0.002)
Education	----	0.069*** (0.015)	----
IQ	----	----	0.027*** (0.002)
COWAT			
Severity	----	-0.081 (0.070)	-0.011 (0.062)
Education	----	0.114*** (0.027)	----
IQ	----	----	0.044*** (0.006)
PASAT			
Severity	----	-0.155* (0.072)	-0.102 (0.068)
Education	----	0.156*** (0.027)	----
IQ	----	----	0.039*** (0.006)
STROOP-word reading			
Severity	-0.123* (0.090)	-0.085 (0.071)	-0.049 (0.072)
Pack-Years	-0.008* (0.004)	-0.005 (0.004)	-0.005 (0.004)
Education	----	0.103*** (0.026)	----
IQ	----	----	0.026*** (0.005)
STROOP-color naming			
Severity	-0.137* (0.072)	-0.113 (0.072)	-0.059 (0.070)
Pack-Years	-0.006 (0.004)	-0.004 (0.004)	-0.003 (0.002)
Education	----	0.065** (0.027)	----
IQ	----	----	0.028*** (0.005)
STROOP-color word			
Severity	----	-0.081 (0.070)	-0.049 (0.012)
Education	----	0.083*** (0.027)	----
IQ	----	----	0.032*** (0.005)
WCST-categories			
Severity	----	-0.077 (0.072)	-0.057 (0.072)
Education	----	0.071** (0.027)	----
IQ	----	----	0.016*** (0.005)
TMT-A			
Severity	-0.153* (0.074)	-0.121* (0.074)	-0.056 (0.071)
Pack-Years	-0.011** (0.004)	-0.010** (0.004)	-0.008 *(0.004)
Education	----	0.030 (0.027)	----
IQ	----	----	0.029*** (0.005)

	Model 1	Model 2	Model 3
	Coeff. (Std. Err.)	**Coeff. (Std. Err.)**	**Coeff. (Std. Err.)**
STOP-TASK go RT			
Severity	-0.140* (0.075)	----	-0.115 (0.079)
Pack-Years	-0.014*** (0.004)	----	-0.013*** (0.004)
IQ	----	----	0.009* (0.005)
STOP-TASK go RT variability			
Severity	-0.167** (0.073)	-0.145* (0.075)	-0.096 (0.074)
Pack-Years	-0.014*** (0.004)	-0.013*** (0.004)	-0.011** (0.004)
Education	----	0.036 (0.027)	----
IQ	----	----	0.023*** (0.005)
STOP-TASK stop-signal RT			
Severity	-0.123* (0.075)	----	-0.047 (0.082)
Pack-Years	-0.011** (0.004)	----	-0.009** (0.004)
IQ	----	----	0.017*** (0.005)

Note : For each executive function variable, alcoholism severity and pack-years were only included if they had a significant correlation with that variable.

* $p < .05$,

** $p < .01$,

*** $p < .001$.