

## Effects of the use of alcohol and cigarettes on cognition in elderly adults

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(RECEIVED February 2, 2001; REVISED September 19, 2001; ACCEPTED November 2, 2001)

### Abstract

The objective of this study was to examine the independent and interactive effects of lifetime patterns of drinking and smoking on cognitive performance in the elderly. A sample of 395 individuals with varying histories of alcohol and cigarette use was drawn from the Charlotte County Healthy Aging Study, a community-based, cross-sectional study of randomly selected older adults of age 60 to 84. Dependent variables were the results of a neuropsychological battery that provided measures of general cognitive ability, executive function, and memory. Specifically, we examined (1) differences in performance among groups of abstainers, drinkers, and smokers, (2) the effects of lifetime drinking and smoking dose on cognition within the group of users, and (3) the effects of intensity of drinking and smoking on cognition. Potential methodological confounds, such as age, education, and medical history, were controlled by means of sampling and covariance procedures. Analyses failed to provide evidence for a beneficial *J*-curve or threshold effect for drinking, but did not reveal any detrimental effect. No detrimental effect of smoking was found in any analysis; nor was there any evidence of an interaction between alcohol and cigarette use on any cognitive measure. (*JINS*, 2002, 8, 811–818.)

**Keywords:** Aging, Cognition, Neuropsychological tests, Risk factors, Alcohol drinking, Smoking

### INTRODUCTION

The influence of alcohol and cigarette use on cognitive function in the elderly (age 60+) has become a focal target of research, following reports suggesting an increase in risk among alcohol users (Fratiglioni et al., 1993) and a decrease in risk among smokers (Lee, 1994) for Alzheimer's disease. Several recent studies of community samples (Elias et al., 1999; Hendrie et al., 1996) have found that curvilinear or threshold effects best described the relation between alcohol consumption and cognitive performance. In a study by Elias et al. (1999), for example, occasional or light drinkers performed at the same, or even slightly lower, level than abstainers, but moderate drinkers (2–8 drinks per day) per-

formed at a higher level than abstainers and light drinkers on several cognitive measures. This effect was found for both men and women, but at a lower level of consumption for women. These findings are not uniformly reported, however. Positive effects for women, but not for men, have been reported (Dufouil et al., 1997), and a failure to find any differences in cognitive performance between current and abstinent individuals has also been reported (Dent et al., 1997). Studies have also shown differences in the drinking–cognition relationship based on the specific cognitive ability measured (Cerhan et al., 1998).

Fewer studies have examined the impact of smoking on cognitive function in the elderly. Results from these studies are inconsistent, reporting no loss of cognitive function in smokers (Carmelli et al., 1997; Dufouil et al., 1997), a loss in smokers (Kilander et al., 1997), a loss in smokers and ex-smokers (Galanis et al., 1997), or a loss in smokers but not ex-smokers (Launer et al., 1996). Surprisingly, there is

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little research that has examined the joint impact of smoking and drinking on cognition in the elderly, despite the fact that these behaviors are frequently concurrent. A single study has included analyses of both risk factors (Dufouil et al., 1997), but did not report findings for their interactive effects.

Inconsistency in the results of studies examining the impact of smoking and drinking on cognitive function in the elderly to date would be expected, given the variability across studies in several methodological parameters. These parameters include participant sampling (age ranges, gender, national origin of sample), method of approach (cross-sectional, longitudinal), measures of cognition (global cognitive measures, batteries of domain-specific measures), range and type of consumption (drinker, ex-drinker, duration since quitting, amount and frequency of consumption, alcohol content for preferred drink), control over confounding variables (age, education, history of head trauma, vascular disorder), and statistical power. Several of these variables appear to be especially critical. Because alcohol and cigarette consumption are related to education (Slater et al., 1999), and education is an especially strong predictor of cognitive ability (Vanderploeg & Schinka, 1995), statistical control of years of education is critical. Other variables that potentially confound comparisons between abstainers and users because of their association with use of alcohol and/or cigarettes and their impact on cognitive function include history of vascular disorder, diabetes, and hypertension (Haan et al., 1999). A comprehensive analysis of the impact of drinking and smoking on cognition should also include analyses not only by category of use (abstainer, user, ex-user), but of lifetime consumption as well. In the Galanis et al. (1997) study, for example, significant findings were reported for comparisons of groups based on category of smoking use, but not for analyses based on pack-year history. Finally, while brief measures of global cognitive function, such as the Mini-Mental Status Exam (Folstein et al., 1975), can serve an important role in screening for significant cognitive loss in the elderly, they are not strong measures of differences in cognitive function in normal populations. Adequate measurement of cognitive function should include more sensitive measures of global function, as well as measures of important domains of cognition.

In this study we examined the individual and interactive effects of lifetime drinking and smoking on cognition in a community-based sample of elderly adults. The research design addressed several of the methodologic issues described above and included multiple cognitive measures, both sampling and statistical control of potential confounding variables, and analyses based on both category of use and lifetime dose of alcohol and cigarettes. Specifically, we addressed whether (1) individuals with a lifetime history of sustained smoking and/or drinking show cognitive loss in comparison to lifetime abstainers; (2) cumulative lifetime doses of alcohol or cigarettes, or of the two substances in interaction, have an effect on cognition within the group of individuals with a history of smoking or drinking; or (3)

individuals who have histories including periods of intense use of either alcohol or cigarettes show cognitive loss in comparison to lifetime abstainers.

## METHODS

### Research Participants

Data for the present study were obtained from the Charlotte County Healthy Aging Study (CCHAS) (Small et al., 2000). The CCHAS is a representative, population-based, cross-sectional study of individuals, aged 60 to 85, living in two census tracts of Charlotte County, Florida. Sequential sampling procedures in the CCHAS identified 808 individuals who were eligible for participation in the study. Of these, 466 (58%) individuals consented to participate and completed the study protocol. Protocol information was obtained by structured interviews with participants on demographic variables, personal and family medical history (but not psychiatric history), smoking and alcohol consumption, depression, anxiety, social support, work history, physical and mental exercise, and risk factors for dementia. Participants also completed a battery of cognitive ability measures. More complete information on CCHAS participant selection and data collection procedures is provided in Small et al. (2000).

Exploratory data analyses were conducted to identify outliers in the distributions of scores for cognitive measures and drinking and smoking estimates, cases with missing or inconsistent demographic data, and participants who reported any period of drinking in which consumption exceeded 8 drinks per day. The criterion of 8 drinks per day was based on the conclusion of Parsons and Nixon (1996) that the risk for mild cognitive impairment occurs with extended drinking at the level of 7 to 9 drinks per day and of the data reported by Elias et al. (1999) that beneficial effects of drinking on cognition can occur with up to 8 drinks per day. Data for 34 participants were dropped from the data set as a result of these analyses (5 identified as outliers, 11 for missing or inconsistent data, and 18 for exceeding the criterion of greater than 8 drinks per day). Cases were also excluded if they reported a history of any of the following: endarterectomy, transient ischemic attacks, cerebrovascular accidents, Parkinson's disease, or traumatic head injury with loss of consciousness and retrograde amnesia. A total of 47 cases were dropped for meeting one or more of these criteria. Hypertension and diabetes were common in the sample (145 participants) and these cases were retained. The final data set available for analysis consisted of 383 individuals; 176 men and 209 women. Of these, 61 denied any history of regular drinking and smoking and reported consuming fewer than 1 drink and 1 cigarette per month. These individuals were considered to be abstinent. The remaining 324 individuals all had a history of a substantive period (at least 1 year) of drinking and/or smoking, although only 239 continued to drink and 32 continued to smoke at the time the study was conducted. The sample was 98% White in ethnic composition.

## Cognitive Measures

Cognitive measures included measures of general cognitive ability, executive function, and memory. The general ability measure was the Modified Mini-Mental Status Exam (3MS; Teng & Chui, 1987), a modification and extension of the Mini Mental Status Exam (MMSE: Folstein et al., 1975) designed to provide a more reliable and sensitive measure of overall ability than the MMSE. Executive ability was measured by the Stroop Test (Golden, 1978) color-word trial and the Trail Making Test (Reitan, 1958) Trial B time. An executive ability score was derived by taking the average of sample-based  $z$  scores for correct items on the Stroop color-word trial and total time for Trailmaking Trial B. Memory was assessed by the Hopkins Verbal Learning Test (Benedict et al., 1998), a list learning task in which subjects are presented with three learning trials, followed by delayed recall, cued recall, and recognition trials. Because of a ceiling effect the distribution of scores for the recognition trial were highly skewed in this sample, and so only scores for the sum of recalled items for Trials 1 to 3, delayed recall, and cued recall were used. A memory ability score was calculated by taking the average of sample-based  $z$  scores for total correct for learning, delayed recall, and cued recall trials.

## Drinking, Smoking, and Control Variables

Two indices of drinking and smoking were used in the analyses, lifetime dose and intensity. Lifetime dose for smoking was calculated as the number of pack-years of cigarette use (packs per day multiplied by the number of years of smoking). A similar index was used to measure drinking. A drink-year was defined as follows: number of drinks per day multiplied by the number of years drinking. An individual who consumed 3 drinks a day for 30 years would therefore have a 90 drink-year history. No distinction was made between various forms of alcoholic beverage (1 glass/can/bottle beer = 1 glass wine = 1 hard liquor drink). Participants' drinking histories were examined, however, for periods of heavy drinking. Drink-year estimates were based on histories of both regular drinking and heavy drinking.

The intensity of smoking and drinking was calculated as the maximum amounts consumed over a sustained (minimum of 1 year) period of time. For smoking, this was calculated as the number of cigarettes per day; for drinking, the number of drinks per month.

Because information on the impact of diabetes and hypertension on overall health was not captured in the interview, the number of years of treatment for each disorder was used as an indicator of the potential cumulative effect of these chronic diseases. Age at the time of the study and number of years of formal education also served as control variables. For one set of analyses, intensity of smoking and drinking were also used as covariate control variables.

## Analyses

Three procedures were used in the analysis of data. Analyses of covariance (ANCOVA) and multivariate analyses of covariance (MANCOVA) were conducted, with groups defined by drinking and smoking indices. Control variables served as covariates for these analyses. Power to find a medium-sized effect with alpha set at .05 was greater than .80 for all analyses. When several analyses were conducted for a subgroup of the sample, family-wise error corrections were made for tests of significance. Hierarchical multiple linear regression analyses were conducted with simultaneous entry of variables in sequential blocks of sets of variables in this order: control variables, study variables, and interactions. ANCOVAs and MANCOVAs did not include sex as an independent variable, because of the substantial loss of statistical power. Regression analyses, however, did examine sex and its interactions. For analyses of variance, Eta squared was used to estimate the amount of variance explained by variables and their interactions. For regression analyses, the change in  $R^2$  was used to estimate variance contributions at sequential steps in the analysis.

## RESULTS

### Preliminary Analyses

Because the sample contained many individuals who had substantive life histories of drinking and/or smoking, but were ex-users at the time of the study, we first examined differences in cognitive performance between current and ex-users. A single factor (drinkers vs. ex-drinkers) analysis of covariance (ANCOVA), with age, years of education, years of diabetes treatment, and years of hypertension treatment as covariates, was used to test the hypothesis of differences in performance on the 3MS. Separate multivariate analyses of covariance (MANCOVA), employing the same design, were used to examine differences in performance for the three memory scores and for the two executive scores. Parallel analyses were performed to compare the cognitive performance of smokers and ex-smokers. In no case did the results achieve corrected significance levels. Further, in no analysis did the group variable account for more than 3% of the variance in cognitive performance scores. In all subsequent analyses, therefore, data for current and ex-users was combined.

### Performance of Abstainers and Users on Tests of Cognition

Univariate and multivariate analyses of covariance were used to examine the simple and interactive effects of drinking and smoking versus abstinence on cognitive performance. Four groups of individuals were used in this analysis: 61 lifetime abstainers, 36 individuals with a history of smoking but not drinking, 104 individuals with a history of drinking but not smoking, and 182 individuals with a history of

drinking and smoking. Both current and ex-users were included in the drinking and smoking groups. Characteristics of these groups are provided in Table 1. A 2 (drinking vs. abstinence)  $\times$  2 (smoking vs. abstinence) ANCOVA, with age, years of education, years of diabetes treatment, and years of hypertension treatment as covariates, was used to test the hypothesis of differences in performance on the 3MS. Separate MANCOVAs, employing the same design, were used to examine differences in performance for the three memory scores and for the two executive scores.

Results of the ANCOVA for general cognitive ability and the MANCOVAs for memory and executive ability are presented in Table 2. Age was found to be a significant ( $p < .001$ ) covariate for all three abilities, while years of education was found to be a significant ( $p < .01$ ) covariate for general cognitive and executive abilities. Years of diabetes and hypertension were not found to significantly covary with any ability measure. Smoking, drinking, and their interaction were not found to have an effect ( $p > .10$  for all analyses) on the cognitive measures, indicating that there were no differences in performance on any of the cognitive measures among the groups of abstainers, drinkers, smokers, and those who had a history of both drinking and smoking.

## Effects of Drinking and Smoking on Cognition in Users

Hierarchical multiple linear regression analysis was used to assess the influence of lifetime cumulative doses of alcohol and cigarettes on cognitive performance for the group ( $n = 322$ ) of current and ex-users. Descriptive data for demographic information, smoking and drinking variables, control variables, and cognitive measures for these individuals are provided in Table 1.

The regression analyses were performed with simultaneous entry of variables in sequential blocks. Control variables were entered on Blocks 1 (gender, age, and years of education) and 2 (hypertension and diabetes). On Block 3, pack-years and drink-years were entered. On Block 4 the possible two-way interactions of age, pack-years, and drink-years were entered. The three-way interaction of Age  $\times$  Pack-Years  $\times$  Drink-Years was entered on Block 5. Separate analyses were performed for the 3MS total, the memory ability score, and the executive ability score.

Results of the multiple linear regression analyses for all three cognitive measures are provided in Table 3. The set of demographic variables consisting of gender, age, and years of education accounted for significant variance in general

**Table 1.** Demographic characteristics and cognitive performance statistics for drinking and smoking groups

Group characteristic/ score		Abstinent	Smoking nondrinker	Drinking nonsmoker	Drinking smoker	Smoker or drinker	Smoker- med intensity	Smoker- high intensity	Drinker- med intensity	Drinker- high intensity
<i>N</i>		61	36	104	182	322	114	46	90	44
% Male		18.0	22.2	42.3	62.1	51.2	51.8	84.8	59.8	79.5
% Female		82.0	77.8	57.7	37.9	48.8	48.2	15.2	40.2	20.5
Age		73.7	70.1	71.5	72.0	71.7	72.1	71.9	71.4	70.8
Years of education		13.1	16.1	14.3	14.2	14.5	14.8	14.2	14.7	14.2
Cigarettes per day		—	22.4	—	22.6	15.3	16.9	48.3	14.4	22.8
Years of smoking		—	34.5	—	30.1	21.3	32.1	32.6	19.4	25.9
Pack-years		—	39.4	—	36.1	24.8	27.2	78.3	22.4	39.0
Drinks per month		—	—	44.3	62.0	49.4	44.9	75.9	42.9	186.2
Years of drinking		—	—	35.0	34.1	30.6	28.1	32.5	31.5	34.3
Drink-years		—	—	39.1	58.3	45.6	43.6	75.7	37.8	150.1
3MS score	<i>M</i>	91.4	92.9	91.9	92.6	92.5	92.5	93.3	91.8	92.8
	<i>SD</i>	6.7	5.0	6.0	6.4	6.0	5.7	4.3	7.1	5.3
HVLT Trials 1–3	<i>M</i>	21.3	21.7	20.7	19.6	20.2	19.9	19.7	19.2	19.0
	<i>SD</i>	5.1	5.0	5.8	5.1	5.4	5.3	4.4	5.3	5.1
HVLT recall	<i>M</i>	7.7	8.5	7.9	7.4	7.6	7.5	7.6	7.4	7.4
	<i>SD</i>	2.8	2.6	2.6	2.9	2.8	2.9	2.4	2.9	2.9
HVLT cued recall	<i>M</i>	8.8	9.2	8.6	8.4	8.5	8.5	8.4	8.4	7.9
	<i>SD</i>	2.4	2.2	2.5	2.3	2.4	2.4	2.0	2.3	2.4
Trail-Making Test	<i>M</i>	123.0	107.4	112.1	113.0	113.3	110.3	104.6	107.9	99.1
	<i>SD</i>	70.6	50.9	57.9	61.7	59.3	57.4	45.9	54.4	49.9
Stroop Test	<i>M</i>	27.3	28.9	29.2	28.9	29.1	29.4	30.4	28.2	30.1
	<i>SD</i>	8.8	10.3	9.1	9.6	9.6	9.5	9.2	9.3	9.5

Note. The smoker or drinker group is comprised of the smoking–nondrinker, drinking–nonsmoker, and drinking–smoker groups.

**Table 2.** Results of ANCOVA on general cognitive ability and of MANCOVA on memory and executive measures for presence of lifetime drinking and smoking

Source	<i>F</i>	<i>p</i>	$\eta^2$
<b>General cognitive</b>			
Years of education	17.85	.00	.04
Age	26.23	.00	.06
Years of diabetes	1.64	.20	.00
Years of hypertension	0.45	.50	.00
Smoking	0.12	.73	.00
Drinking	0.53	.82	.00
Smoking $\times$ Drinking	0.27	.82	.00
<b>Memory</b>			
Years of education	2.17	.09	.02
Age	6.73	.00	.05
Years of diabetes	0.67	.57	.00
Years of hypertension	1.16	.33	.01
Smoking	1.06	.36	.01
Drinking	1.66	.18	.01
Smoking $\times$ Drinking	1.15	.33	.01
<b>Executive</b>			
Years of education	5.12	.01	.03
Age	27.62	.00	.13
Years of diabetes	2.49	.08	.01
Years of hypertension	0.23	.79	.00
Smoking	0.05	.96	.00
Drinking	0.28	.75	.00
Smoking $\times$ Drinking	0.10	.90	.00

cognitive and memory ability scores, but not for executive ability scores. The only other significant finding was for the set of two-way interactions for drink-years, pack-years, and age in predicting memory ability. Examination of individual interaction terms revealed this result to be due specifically to the interaction of drink-years and pack-years. However, this interaction term accounted for only 1.6% of the variance in memory ability scores. In fact, the drinking and smoking variables, and their interactions together and with age, produced at best (for the prediction of the memory function measure) an incremental 2.3% in contributed variance beyond that of the control variables.

To examine possible gender interactions with smoking and drinking variables, an additional set of multiple linear regression analyses was conducted. In these analyses, all variables were entered on Block 1. Two- and three-way interactions for gender, drink-years, and pack-years were entered on subsequent blocks. No significant results ( $p > .05$  for all analyses) were found for any interaction term for analysis of any cognitive measure.

### Effects of Drinking and Smoking Intensity on Cognition

Univariate and multivariate analyses of covariance were used to examine the impact of intensity of alcohol and cig-

**Table 3.** Results of regression analyses for effects of lifetime dose of alcohol and cigarettes on general cognitive, memory, and executive abilities

Model	<i>R</i>	<i>R</i> <sup>2</sup>	<i>R</i> <sup>2</sup> <sub>Change</sub>	<i>F</i> <sub>Change</sub>	<i>p</i>
<b>General cognitive</b>					
1	.334(a)	.112	.112	13.35	.00
2	.338(b)	.114	.003	0.44	.64
3	.343(c)	.118	.003	0.61	.55
4	.355(d)	.126	.008	0.98	.40
5	.355(e)	.126	.000	0.00	.96
<b>Memory</b>					
1	.393(a)	.154	.147	19.356	.00
2	.396(b)	.157	.003	0.501	.61
3	.399(c)	.159	.002	0.348	.71
4	.424(d)	.180	.021	2.613	.05
5	.427(e)	.182	.002	0.869	.35
<b>Executive</b>					
1	.099(a)	.010	.010	1.042	.37
2	.118(b)	.014	.004	0.643	.53
3	.141(c)	.020	.006	0.948	.39
4	.151(d)	.023	.013	0.331	.80
5	.153(e)	.023	.000	0.110	.74

*Note.* Variables entered in each sequential block: (a) sex, years of education, age; (b) years of diabetes, years of hypertension; (c) pack-years, drink-years; (d) two-way interactions of pack-years, drink-years, and age; (e) three-way interaction of drink-years, pack-years, age.

arette use on cognitive function. Separate sets of analyses were conducted for alcohol and cigarette use. For drinking, current or ex-drinkers with a sustained period of 30 to 60 drinks per month, regardless of length of drinking history, were identified as medium intensity drinkers. Current or ex-drinkers with a sustained period of over 119 drinks per month, regardless of length of drinking history, were identified as high intensity drinkers. Three groups of individual were used in this analysis: 61 lifetime abstainers, 90 medium-intensity drinkers, and 34 high-intensity drinkers. A one-way ANCOVA, with age, years of education, years of diabetes treatment, years of hypertension treatment, and smoking amount (number of cigarettes per day) as covariates, was used to test the hypothesis of differences in performance on the 3MS. Separate multivariate analyses of covariance (MANCOVA), employing the same design, were used to examine differences in performance for the three memory scores and for the two executive scores. Characteristics of the groups used in these analyses are provided in Table 1.

Similar analyses were performed for smoking intensity, using 61 lifetime abstainers, 114 medium-intensity (10–19 cigarettes per day) smokers, and 46 high-intensity (>39 cigarettes per day) smokers. For these analyses, drinking amount (number of drinks per month) was used as a covariate in addition to age, years of education, years of diabetes treatment, and years of hypertension treatment. Characteristics of the groups used in these analyses are provided in Table 1.

Results of the ANCOVA on general cognitive ability and of the MANCOVAs on memory and executive ability for the effects of drinking and smoking intensity are presented in Tables 4 and 5, respectively.

For the analyses on drinking intensity, education was found to be a significant ( $p < .05$ ) covariate for all three abilities, while age was found to be a significant ( $p < .01$ ) covariate for general cognitive and executive abilities. Years of diabetes, years of hypertension, and number of cigarettes daily were not found to significantly covary with any ability measure. In no analysis were there significant differences among abstainer, low-intensity drinkers, and high-intensity drinkers.

For the analyses on smoking intensity, age was found to be a significant ( $p < .05$ ) covariate for all three abilities, while years of education was found to be a significant ( $p < .01$ ) covariate for general cognitive ability. Years of diabetes, years of hypertension, and number of drinks per month were not found to significantly covary with any ability measure. In no analysis were there significant differences among abstainer, low-intensity smokers, and high-intensity smokers.

## DISCUSSION

In this study we took several approaches to study the effect of drinking and smoking on cognition in the elderly. We examined (1) the effects of lifetime dose of alcohol and cigarette consumption on cognition among groups of abstainers, drinkers, and smokers, (2) the effects of lifetime drinking and smoking dose on cognition in users, and

**Table 4.** Results of ANCOVA on general cognitive ability and of MANCOVA on memory and executive measures for drinking intensity

Source	<i>F</i>	<i>p</i>	$\eta^2$
General cognitive			
Years of education	37.99	.00	.18
Age	12.34	.00	.06
Years of diabetes	1.44	.23	.01
Years of hypertension	0.22	.64	.00
Number of cigarettes daily	0.44	.51	.00
Drinking intensity	1.37	.26	.02
Memory			
Years of education	3.19	.02	.05
Age	2.91	.04	.05
Years of diabetes	0.43	.73	.01
Years of hypertension	0.33	.80	.01
Number of cigarettes daily	0.74	.53	.01
Drinking intensity	1.23	.29	.02
Executive			
Years of education	8.72	.00	.09
Age	13.35	.00	.13
Years of diabetes	1.53	.22	.02
Years of hypertension	0.22	.80	.00
Number of cigarettes daily	0.28	.76	.00
Drinking intensity	0.85	.50	.01

**Table 5.** Results of ANCOVA on general cognitive ability and of MANCOVA on memory and executive measures for smoking intensity

Source	<i>F</i>	<i>p</i>	$\eta^2$
General cognitive			
Years of education	10.44	.00	.05
Age	13.48	.00	.06
Years of diabetes	0.85	.36	.00
Years of hypertension	0.13	.72	.00
Number of drinks daily	0.35	.58	.00
Smoking intensity	0.81	.44	.01
Memory			
Years of education	1.25	.29	.08
Age	3.06	.03	.04
Years of diabetes	0.32	.81	.01
Years of hypertension	0.57	.64	.01
Number of drinks daily	0.87	.46	.01
Smoking intensity	1.01	.42	.01
Executive			
Years of education	2.43	.09	.02
Age	12.64	.00	.11
Years of diabetes	0.64	.53	.01
Years of hypertension	0.24	.78	.00
Number of drinks daily	1.34	.26	.01
Smoking intensity	0.18	.95	.00

(3) the effect of intensity of drinking and smoking on cognition. These analyses were well controlled by excluding individuals with medical histories of disorders associated with cognitive loss or decline and adjusting cognitive measures for the effects of hypertension, diabetes, age, and education. The use of multiple cognitive measures allowed a broad examination of function in several domains. However, the analyses produced only a single significant result and no findings of substance, even when considering the interaction of Smoking  $\times$  Drinking History. In no analysis did drinking and smoking variables explain as much as 3% of the variance in performance on any of the cognitive measures.

Our results add to a growing literature that reveals no substantive harmful effects of drinking, within the broad limits of consumption of social drinking, on cognitive performance. The results of our intensity analyses also failed to provide evidence for a beneficial *J*-curve or threshold effect for drinking, despite the fact that intensity of drinking in the sample covered the range of drinking intensity within which such results are usually found (i.e., 1–8 drinks per day). Previous studies (Dufouil et al., 1997; Elias et al., 1999; Hendrie et al., 1996) have reported that the intensity level at which the beneficial effect of drinking occurs is likely to be gender-dependent. Our regression analyses, however, failed to find any effect for the interaction of Gender  $\times$  Drinking on cognition.

The failure to find an effect of smoking on cognition in the elderly is consistent with reports finding no effect in

smokers or ex-smokers (Carmelli et al., 1997; Dufouil et al., 1997), but contrary to other research reporting detrimental effects in both smokers and ex-smokers (Galanis et al., 1997). Because individuals in our sample with a lifetime history of smoking were primarily ex-smokers at the time of the study, it would be tempting to conclude that our results are also consistent with the study by Launer et al. (1996), who reported detrimental findings in smokers, but not ex-smokers. However, we found no difference in cognitive performance in our sample between current and ex-smokers.

While other studies have controlled for the effects of smoking in the study of drinking, or for drinking in the study of smoking, our study is the first to examine the interactive effects of both behaviors on cognition. In our regression analysis of memory function in users we did find a single significant interaction of Smoking  $\times$  Drinking  $\times$  Age, but it accounted for only 2.1% of the variance. Thus, though statistically notable, the finding has little predictive value and provides little of meaning to potential models of cognitive aging.

Despite our attempts to attend to methodological issues that could influence outcome, our study can be criticized from the standpoint of its external validity. Our sample is characterized by 14+ years of mean education, a higher level of educational achievement than would be expected for individuals in the age range of 60 to 84. Education is positively related to lifetime income, availability of health care, and positive health status. Additionally, more highly educated individuals are thought to have more cognitive reserve (Mortimer, 1997), a protection against the effects of cortical insult. Thus, the impact of alcohol and cigarette consumption may be attenuated in our sample. While our use of years of education as a control variable argues against these concerns for individuals with the level of education of our sample, it is possible that samples in the lower ranges of education might produce different results.

A second threat to external validity follows from restrictions applied to sampling. Our sample was almost exclusively White in ethnic origin and thus our results may not generalize to other ethnic populations. We also excluded individuals with disorders known to impact cognitive function, several of which (e.g., vascular disorders) are associated with drinking and smoking. While these restrictions allowed examination of the direct effects of drink and smoking on cognition, they precluded examination of the potential indirect and interactive effects that might be produced in the presence of such disorders. These effects can only be examined in large samples with representative numbers of individuals who are both afflicted with the disorder and for whom there is a sufficient range of use of alcohol and cigarettes.

A third validity threat can be attributed to the sample rate of nonparticipation. While the majority of individuals (58%) solicited for participation did in fact complete the study, it is possible that heavier drinkers and smokers were disproportionately represented among nonparticipants. Thus, our results could only be considered to generalize within the range of drinking and smoking of the participants.

Finally, our study is susceptible to the shortcomings of retrospective designs that rely heavily on the integrity of participant recall of critical data such as amount and duration of use of alcohol and cigarettes. A single prospective, longitudinal study (Carmelli et al., 1997) has failed to show an impact of drinking or smoking on cognition over a period of 4 to 6 years in men of age 70 and older at the time of the baseline cognitive evaluation. Notably, this time frame was sufficient, however, to demonstrate significant changes in cognition attributable to factors such as depression, activity level, and health status. The body of research on the cumulative effects of drinking and smoking on cognition in old age would most benefit from prospective, longitudinal studies that would cover substantial (e.g., 5–10 years) segments of the age range of 50 to 80.

## ACKNOWLEDGMENTS

We thank Glenn Curtiss and Amy Lazev for helpful comments in the preparation of this article.

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