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

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Abstract

In this study we examined the independent and interactive effects of lifetime patterns of drinking and smoking on cognitive performance in elderly African Americans. A sample of 230 individuals with varying histories of alcohol and cigarette use was drawn from the Hillsborough Elder African American Life Study, a community-based, cross-sectional study of older adults aged 60 to 84. Dependent variables were the results of a neuropsychological battery that provided measures of general cognitive ability, executive function, and memory. Specifically, our study addressed (1) whether individuals with a lifetime history of sustained smoking and/or drinking show lower levels of cognitive performance in comparison to lifetime abstainers, (2) whether cumulative lifetime doses of alcohol or cigarettes, or of the two substances in interaction, have an effect on cognition, and (3) whether individuals who have histories of periodic, intense use of either alcohol or cigarettes show lower levels of cognitive performance in comparison to lifetime abstainers. When significant results were obtained, effect sizes were small, not exceeding 5% of the variance. A single exception occurred for the intensity analyses, in which drinking explained approximately 16% of the variance in global cognitive ability after adjusting for the contributions of control variables. In these analyses, drinking was found to have a *U*-shaped effect on global cognitive ability and total acquisition in the memory trials. Specifically, moderate users performed at a lower level than abstainers or heavy users, who did not differ from each other. (*JINS*, 2003, *9*, 690–697.)

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

INTRODUCTION

Research to date has demonstrated that the risk for Alzheimer's disease may be 2 to 3 times higher in African Americans as compared to Caucasians (Tang et al., 1998, 2001). This increased risk remains even after adjustments are made for education, illiteracy, or a history of stroke, heart disease, hypertension, or diabetes (Tang et al., 2001). As census data have revealed that the proportion of African American and Hispanic Americans living beyond age 65 in the United States is increasing more rapidly than the proportion of white individuals (Day, 1996), it is important to determine the behavioral factors associated with cognitive status among these minority groups.

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Two behavioral factors that have received attention are smoking and alcohol consumption. In the elderly population at large, it has been demonstrated that smoking causes structural changes in the brain (Liao et al., 1996; Longstreth et al., 2001; Swan et al., 2000). Likewise, heavy alcohol consumption in older persons is associated with brain atrophy (Kubota et al., 2001; Mukamal et al., 2001) and may cause dementia. The association of white matter lesions in the brain with smoking and alcohol intake is greater in African Americans than in Whites (Liao et al., 1997). This again underscores the potential need to consider subpopulations when studying the effects of alcohol and smoking on cognitive status.

To our knowledge, only one published study has examined the relationship between drinking and cognitive status in an elderly, African American sample (Hendrie et al., 1996). This study, which involved 2040 elderly (M age = 74.1 years) African Americans randomly selected from the Indi-

anapolis region, revealed a variable dose relationship between alcohol consumption and cognitive performance. Heavy consumption was associated with poorer cognitive status. Light drinkers outperformed lifelong abstainers, however, suggesting a possible protective effect of light alcohol consumption. The potentially facilitative effect of alcohol on cognition has also been demonstrated in other samples that were not exclusively African American (Carmelli et al., 1999; Cervilla et al., 2000; Galanis et al., 2000; Launer et al., 1996). On the other hand, many studies have failed to find a protective effect (Carmelli et al., 1997; Dent et al., 1997; Elwood et al., 1999; Herbert et al., 1993; Schinka et al., 2002) or an effect moderated by gender (Dufouil et al., 1997; Edelstein et al., 1998).

There are no specific studies on the effects of smoking on cognition in African American elderly. Studies with Caucasian or mixed samples have reported either poorer cognitive functioning associated with smoking (Carmelli et al., 1999; Galanis et al., 1997; Kilander et al., 1997; Launer et al., 1996) or no effect on cognition (Carmelli et al., 1997; Cervilla et al., 2000; Elwood et al., 1999; Herbert et al., 1993; Schinka et al., 2002). One study which specifically examined the potential interactive effects of smoking and drinking on cognition (Schinka et al., 2002) failed to find such an interaction.

The inconsistencies inherent in the literature pertaining to Caucasian or mixed samples is not surprising given the variability in methodology, participant sampling, and statistical power in these studies. Variables such as comprehensiveness of cognitive assessment and method of calculating substance use (both current and lifetime) are particularly important to consider. In addition, given that African Americans as a group have more problems with hypertension than Caucasians (e.g., Resnick et al., 2001; Sacco, 2001; Weir, 1997), vascular risk factors will obviously be particularly important to consider.

In this study, we examined the individual and interactive effects of lifetime drinking and smoking on cognition in a community-based sample of elderly African American adults. Specifically, this study addressed (1) whether individuals with a lifetime history of sustained smoking and/or drinking show lower levels of cognitive performance in comparison to lifetime abstainers, (2) whether cumulative lifetime doses of alcohol or cigarettes, or of the two substances in interaction, have an effect on cognition, and (3) whether individuals who have histories of periodic, intense use of either alcohol or cigarettes show lower levels of cognitive performance in comparison to lifetime abstainers.

METHODS

Research Participants

Data for this study were obtained from a larger epidemiologic study of 255 community-dwelling elderly African Americans (ages 60–84) in three Tampa, Florida neighborhoods. The study used epidemiologic sampling procedures to identify and solicit participation from elderly individuals living in private residences. All individuals provided University of South Florida IRB-approved consent prior to participation in the study. For participants, data collection was scheduled across a 1-week interval. The first visit was scheduled either at the participant's home or in a neutral location (e.g., church). At this visit, a trained interviewer administered a risk factor questionnaire. Participants were then left with a packet of self-administered questionnaires. A second interview, which involved sensory, cognitive, and other testing, occurred approximately 1 week later. Interview variables included demographic information, personal and family medical history, smoking and alcohol consumption, social support, work history, physical and mental exercise, and risk factors for dementia. Information was not collected on mental health disorders. Cognitive testing consisted of a 2-hr battery of cognitive ability measures that were administered by trained technicians.

Exploratory data analyses were conducted to identify outliers (defined *a priori* as a score that was 3 *SD*s from the mean and obviously discontinuous from the tail of the distribution) in the distributions of scores for cognitive measures and smoking and drinking estimates, cases with missing or inconsistent demographic data, and participants who reported any period of drinking in which they consumed more than eight drinks per day. Participants drinking at this level were excluded because (1) Parsons and Nixon (1998) reported that the risk for mild cognitive impairment rises with extended drinking at the level of seven to nine drinks per day; and (2) Elias et al. (1999) found that beneficial effects of alcohol consumption can occur with up to eight drinks per day. Data for 11 participants were dropped from the data set as a result of these exploratory analyses (3 identified as outliers, 5 for missing or inconsistent data, and 3 for exceeding the criterion of greater than eight 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 any loss of consciousness and retrograde amnesia. A total of 14 cases were dropped for meeting one or more of these criteria. The final data set available for analysis thus consisted of 230 individuals, 114 men and 126 women. Of these, 86 denied any history of drinking and smoking and were considered to be abstinent. The remaining 144 individuals all had a history of a substantive period (at least 1 year) of drinking and/or smoking.

Cognitive Measures

Cognitive measures included measures of general cognitive ability, memory, and executive function. The general ability measure was the Modified Mini-Mental Status Exam (3MS; Teng & Chui, 1997), 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 cognitive ability than the MMSE. Memory was

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assessed using the Hopkins Verbal Learning Test (Benedict et al., 1998), a list learning task in which participants 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 1–3, 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. Executive ability was assessed using the Stroop Test (Golden, 1978) color-word trial. This test entails first reading color words, then naming ink colors, and finally naming ink colors of color words printed in incongruent colors (e.g., the word "red" printed in green ink). The color-word trial requires inhibiting the customary response of reading and is thought to be associated with prefrontal functioning (Perret, 1974; Peterson et al., 2002).

Drinking, Smoking, and Control Variables

Two indices of drinking and smoking were utilized 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 the number of drinks per day multiplied by the number of years drinking. For example, an individual who consumed four drinks per day for 30 years would have a 120 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 and heavy drinking. We have found that the drink-year measure correlates modestly but significantly with pack-years in separate samples of 182 white elderly individuals (r = .27, p <.01) and 1147 middle-aged adults (r = .23, p <.001). These findings are consistent with the well-known common association of smoking and drinking and of their shared genetic risk factors (Swan et al., 1996; True et al., 1999).

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

A majority of the sample carried a diagnosis of hypertension and/or diabetes (125 hypertensives, 2 diabetics, 25 hypertensives with diabetes). Because measures of the impact of diabetes and hypertension on overall health was not captured in the interview, treatment years for each disorder were used as an indicator of the potential cumulative effect of these chronic diseases. Age and education also served as control variables.

Analyses

Several statistical procedures were used in the data analyses. 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. The statistical power to find a medium-sized effect with alpha set at .05 was at least .72 for each analysis. When several analyses were conducted for a subgroup of the sample, family-wise corrections were made for significance tests. Multiple linear regression analyses were conducted with simultaneous entry of the entire set of variables in sequential blocks of sets of variables in this order: control variables, study variables, and interactions. ANCOVAs and MANCOVAs did not include gender as an independent variable due to the substantial loss of statistical power. For analyses of variance, eta^2 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 of unequal cell sizes and heterogeneity of variance estimates across groups, we examined the F_{max} statistics and the ratios of cell *n*s for each ANCOVA analysis. Following the guidelines of Tabachnick and Fidell (1996), all ANCOVA analyses appeared to be safe from the threat of inflated Type 1 error rates. A similar potential problem with MANCOVA analyses was addressed by calculating Box's *M* statistic. For the one MANCOVA for which the *M* statistic was significant, there were no significant findings at the .05 level. No adjustments were therefore made to the use of .05 as a criterion significance level.

As the sample contained many individuals who had life histories of drinking and/or smoking, but were ex-users at the time of the study, we examined differences in cognitive performance between current and ex-users. Ex-drinkers had a mean drinking history of 17.5 years and all had been abstinent for at least 5 years; ex-smokers had a mean smoking history of 15.6 years and all had been abstinent for at least 2 years. 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 and on the color-word trial of the Stroop. A separate multivariate analysis of covariance (MANCOVA), employing the same design, was used to examine differences in performance for the three memory 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 only one analysis did the group variable account for as much as 4% of the variance in cognitive performance scores; the group variable accounted for less

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than 2% of the variance in all other analyses. In all subsequent analyses, therefore, data for current and ex-users were combined.

We also examined possible gender interactions with smoking and drinking variables. In these multiple linear regression analyses, all variables were entered on block one. 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. Data for both genders were therefore combined for all following analyses.

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: 86 lifetime abstainers from both alcohol and cigarettes, 30 individuals with a history of smoking but not drinking, 28 individuals with a history of drinking but not smoking, and 86 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) × 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 for the global cognitive ability and executive measures. A MANCOVA, employing the same design, was used to examine differences in performance for the three memory scores.

Results of the ANCOVAs for general cognitive and executive abilities and the MANCOVA for memory ability are presented in Table 2. Both education and years of treatment for hypertension were found to be significant covariates for all three abilities, and age was found to be a significant covariate for general cognitive and memory abilities. Years of treatment for diabetes were not found to covary significantly with any ability measure. Smoking and drinking interacted to have a small, but significant, deleterious effect on general cognitive ability as measured by the 3MS ($p < .02$, $\text{eta}^2 = .03$). Examination of covariate-adjusted scores revealed that individuals who both smoked and drank had scores that were about 6% lower on average than other groups. Smoking, drinking, and their interaction were not found to have an effect on the executive or memory measures.

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 ($n = 144$) 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 in sequential blocks with simultaneous entry of variables. Control variables were entered on Blocks 1 (gender, age, and years of education) and 2 (hypertension and diabetes). Preliminary stepwise analyses had determined that drink-years always entered the equation prior to pack-years. Drink-years and pack-years were therefore entered on Blocks 3 and 4. On

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

Variable	Abstinent	Smoking nondrinker	Drinking nonsmoker	Drinking smoker	Smoker or drinker	Nondrinker	Drinker–med intensity	Drinker–high intensity
N	86	30	28	86	144	116	25	18
% male	17.4	30.0	35.7	81.4	61.8	79.3	92.0	94.4
% female	82.6	70.0	64.3	18.6	38.2	20.7	8.0	5.6
Age	73.1	69.2	71.0	70.7	70.4	72.0	72.5	67.8
Years of education	9.87	9.50	10.8	9.37	9.68	9.8	8.8	10.0
Cigarettes per day	—	16.1	—	12.8	11.0	4.2	11.0	12.3
Years of smoking	—	16.9	—	20.6	15.8	4.4	17.8	16.8
Pack-years	—	14.6	—	15.3	12.2	3.8	11.4	12.5
Drinks per month	—	—	35.3	74.2	51.2	—	45.2	169.3
Years of drinking	—	—	15.5	22.5	16.4	—	19.3	25.1
Drink-years	—	—	25.9	46.9	33.0	—	25.9	106.0
3MS score, *M* (*SD*)	85.7 (8.0)	85.8 (8.7)	89.2 (8.6)	79.5 (12.5)	82.7 (11.8)	85.7 (8.2)	74.0 (14.2)	82.8 (12.1)
HVLT Trials 1–3, *M* (*SD*)	16.9 (3.8)	16.4 (3.4)	18.5 (5.0)	15.6 (4.2)	16.4 (4.3)	16.7 (3.7)	14.0 (3.4)	17.7 (5.0)
HVLT recall, *M* (*SD*)	6.3 (1.7)	6.3 (1.9)	6.5 (2.4)	5.7 (1.9)	6.0 (2.0)	6.3 (1.7)	5.3 (1.6)	6.7 (2.0)
HVLT cued recall, *M* (*SD*)	7.0 (1.7)	6.9 (1.7)	7.5 (2.2)	6.4 (1.9)	6.7 (2.0)	7.0 (1.7)	6.1 (1.6)	7.1 (2.1)
Stroop Test Color-Word, score *M* (*SD*)	21.3 (5.2)	22.3 (4.7)	24.2 (6.3)	20.7 (5.8)	21.7 (5.8)	21.9 (5.1)	19.8 (5.1)	23.6 (8.4)

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

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

Source	*F*	*p*	Eta^2
General cognitive			
Years of education	58.88	.00	.21
Age	6.60	.01	.03
Years of diabetes	0.27	.60	.00
Years of hypertension	9.11	.00	.04
Smoking	6.48	.01	.03
Drinking	1.76	.19	.00
Smoking × Drinking	5.58	.02	.03
Executive			
Years of education	35.63	.00	.14
Age	.27	.60	.00
Years of diabetes	6.39	.24	.01
Years of hypertension	6.73	.01	.03
Smoking	0.55	.46	.00
Drinking	.28	.60	.00
Smoking × Drinking	3.11	.08	.01
Memory			
Years of education	24.19	.00	.25
Age	3.88	.01	.05
Years of diabetes	1.06	.37	.01
Years of hypertension	7.76	.00	.10
Smoking	1.94	.12	.03
Drinking	1.34	.26	.02
Smoking × Drinking	0.53	.66	.01

Block 5 the possible two-way interactions of age, pack-years, and drink-years were entered. The three-way interaction of Age × Pack-Years × Drink-Years was entered on Block 6. 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 the general cognitive score, the memory ability scores, and the executive ability scores. Drink-years explained a significant amount of variance on all cognitive measures, while pack-years explained a significant amount of variance only in memory scores. Specifically, drink-years explained 2.3% of the variance in the 3MS scores, 4.8% of the variance in the memory scores, and 4.9% of the variance in the color-word scores above and beyond the control variables. These effects of drinking were facilitative. Smoking explained only 1.8% of the variance in memory scores above and beyond the control variables and had a negative impact on memory.

Effects of Drinking Intensity on Cognition

We explored the impact of drinking intensity on cognitive performance by examining the performance scores, adjusted for covariates, of three groups of drinkers: 116 lifetime nondrinkers, 25 medium-intensity drinkers, and 18 high-intensity drinkers. Because of insufficient sample sizes in subgroups, analyses of intensity of cigarette use could not be conducted.

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 more than 119 drinks per month, regardless of length of drinking history, were identified as 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 global cognitive and executive measures. A MANCOVA, employing the same design, was used to examine differences in performance for the three memory scores. Characteristics of the groups whose data was used in these analyses are provided in Table 1. Performance scores for cognitive measures are also presented in Table 1.

Education was found to be a significant ($p < .01$) covariate for all three abilities and age was found to be a significant ($p < .05$) covariate for general cognitive ability. While years of hypertension treatment significantly ($p < .01$) covaried with all three abilities, years of diabetes treatment was found to significantly ($p < .05$) covary only with memory. Number of cigarettes per day was not found to be a significant covariate.

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

Model	*R*	R^2	R^2_{Change}	F_{Change}	*p*
General cognitive					
1	.641(a)	.411	.411	32.57	.000
2	.646(b)	.417	.006	.73	.485
3	.664(c)	.440	.023	5.69	.018
4	.665(d)	.442	.002	.38	.537
5	.669(e)	.448	.006	.45	.716
6	.669(f)	.448	.001	.15	.701
Executive					
1	.421(a)	.177	.177	10.04	.000
2	.445(b)	.198	.021	1.78	.173
3	.497(c)	.247	.049	8.98	.003
4	.498(d)	.248	.001	.24	.628
5	.506(e)	.256	.008	.45	.719
6	.512(f)	.262	.006	1.13	.289
Memory					
1	.550(a)	.303	.303	20.27	.000
2	.559(b)	.313	.010	.98	.377
3	.601(c)	.361	.048	10.30	.002
4	.616(d)	.379	.018	3.98	.048
5	.628(e)	.394	.015	1.12	.342
6	.629(f)	.396	.002	.34	.560

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

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Drinking intensity was found to have a significant ($p < .001$) and substantial (explaining approximately 16% of the variance) effect on general cognitive ability. The pattern of covariate-adjusted 3MS scores was *U*-shaped, with nondrinkers and high-intensity drinkers both performing significantly better than the low-intensity group ($p < .01$) but not differently from each other. A smaller (explaining approximately 4% of the variance) but still significant ($p < .05$) pattern of results was found in the multivariate analysis for the memory measures. Univariate follow-up tests revealed that only the total score over trials was significantly different ($p < .05$) among the groups. Again, the pattern of covariate-adjusted scores was *U*-shaped, with nondrinkers and high-intensity drinkers both performing significantly better than the low-intensity group ($p < .05$) but not differently from each other. Drinking intensity did not have a significant ($p > .05$) effect on executive ability.

DISCUSSION

This study was designed to examine the potential effects of lifetime alcohol and tobacco consumption in elderly African Americans. We approached this task in a variety of ways, including studying (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 (3) the effects of intensity of lifetime dose of alcohol or nicotine on cognition. These analyses were controlled by excluding those individuals with medical histories of disorders associated with cognitive decline and by adjusting cognitive measures for the effects of hypertension, diabetes, age, and education. The use of multiple cognitive measures allowed for examination of function in several domains.

In summarizing the results, it is important to emphasize that each of our analyses addressed a slightly different question. The group analysis revealed that African American elders may not suffer much consequence, relative to abstainers, from drinking or smoking in isolation. The combination, however, may produce a small, deleterious effect on general cognitive ability. The regression analyses, focused only on those who used either or both alcohol and tobacco, revealed that greater consumption of alcohol was associated with better cognitive performance across domains. Higher levels of cigarette smoking, however, were associated with slightly lower memory performance. The somewhat inconsistent pattern of results from these two sets of analyses were clarified by analyses of intensity data, which produced evidence of nonlinear relationships. In these analyses, drinking was found to have a *U*-shaped effect on global cognitive ability and total acquisition in the memory trials. Specifically, moderate users (approximately 1.5 drinks per day) performed at a lower level than abstainers or heavy users (approximately 5 drinks per day), who did not differ from each other.

It should be noted that, when significant results were obtained, effect sizes were small, not exceeding 5% of the variance. A single exception was the effect of intensity of drinking. In the intensity analyses, drinking explained approximately 16% of the variance in global cognitive ability after adjusting for the contributions of control variables. Practically, this was reflected in scores for nonusers and heavy users obtaining covariate-adjusted scores that were 10–15% higher than those of medium drinkers.

Our results are inconsistent with the reported conclusions of one other study of elderly, African Americans. In that study, Hendrie et al. (1996) found an inverted *J*-shaped function between alcohol consumption and cognitive performance in which light drinkers (<4 drinks per week) outperformed lifelong abstainers, suggesting a possible facilitative effect of light alcohol consumption. Heavy consumption (defined as >10 drinks per week) was associated with poorer cognitive status. Effect sizes in the Hendrie et al. study were uniformly small, however. For example, the effect size (d) for the difference between abstainers and heavy drinkers was less than .1 (see Hendrie et al., Table 2).

Our results are also inconsistent with those of our previous research (Schinka et al., 2002) with a White sample. In that study, conducted with healthy, high SES, elderly on the west coast of Florida and employing similar sets of analyses, revealed no impact of drinking on essentially the same set of cognitive measures. Notably, a curvilinear relationship such as that revealed in our intensity analysis have been found in Caucasian samples. For example, Elias et al. (1999) found that higher cognitive performance was associated with heavy drinking (2–4 drinks per day for women, 4–8 drinks per day for men) relative to abstainers, while lighter drinkers performed at a lower level than abstainers. In this study again, however, effect sizes were very small. Although the effect sizes cannot be calculated from information provided in tables in the Elias et al. study, significance levels beyond .01 were not achieved in any analysis despite samples sizes of 1,053 for women and 733 for men.

While we found that smoking, in itself, had no impact on cognition, the combination of smoking and drinking was found to have a small, deleterious effect on general cognitive ability. Previous studies of the impact of smoking on cognition have focused on predominantly Caucasian samples. Smoking has sometimes been found to be associated with a decrement in cognitive function (Carmelli et al., 1999; Galanis et al., 1997; Kilander et al., 1997; Launer et al., 1996), but studies finding this association have not typically controlled for the impact of drinking. Because drinking and smoking co-occur with such frequency, these findings may reflect an underlying interaction of both behaviors. Notably, there are a number of reports failing to find a detrimental effect on cognition in smokers or ex-smokers (Carmelli et al., 1997; Cervilla et al., 2000, Elwood et al., 1999; Herbert et al., 1993; Schinka et al., 2002). Again, with one exception (Schinka et al., 2002), these studies did not examine the interactive effect of both drinking and smoking. In the Schinka et al. study, neither smoking nor the smoking by drinking interaction was found to have any effect on cognition.

While not a primary focus of the study, it was interesting that hypertension chronicity accounted for a significant por-

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tion of the variance in cognitive measures (usually second only to education) in every analysis. Given that African Americans as a group have more problems with hypertension than Whites (e.g., Resnick et al., 2001; Sacco, 2001; Weir, 1997), this is a particularly disturbing finding. Preventive intervention, to avoid the cognitive sequelae associated with chronic hypertension, is paramount.

Despite our attempts to attend to methodological issues that could influence outcome, our study can be criticized from the standpoint of its external validity. We 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 drinking 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.

Other potential criticisms could be leveled against our sample. In general, they had few years of education, thereby limiting the generalizability of our findings. In addition, we could not adequately test hypotheses with regard to smoking intensity due to the dearth of heavy smokers in our sample. Another weakness of our study was the inclusion of only one executive measure. While the Stroop is associated with frontal lobe functioning, the addition of other measures would likely have allowed for greater generalizability. The addition of measures of other cognitive domains, such as visuospatial ability, and of daily instrumental functioning would also allow a more comprehensive examination of the impact of drinking and smoking. The study would have also benefited from the use of better indices of disease severity for hypertension and diabetes.

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 in a mixed sample of elderly men (Carmelli et al., 1997) failed to show an impact of drinking or smoking on cognition over a period of 4–6 years 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.

Despite these limitations, this study is an important step in understanding the cognitive sequelae of smoking and drinking in the elderly African American population. In Whites, a summary of studies to date would probably conclude that there is little evidence for meaningful detrimental effect of lifelong social drinking on cognition in the elderly, but some evidence for a small beneficial effect at heavier levels of consumption. For smoking, a summary would probably conclude that there is insufficient evidence to conclude whether there is any effect on cognition. A summary of drinking studies in African Americans would likely note the contradictory results of this study and those of Hendrie et al. (1996). The most significant result in the two studies is our finding of a detrimental effect of moderate drinking on cognition, with no apparent harmful effect of heavy drinking. Explanations for such a phenomenon implicate relationships with mechanisms such as lipid production and plasma concentrations (see Hendrie et al., 1996). If confirmed in future studies, this finding may play a role in helping to explain the complex interactions among factors that produce risk for Alzheimer's disease that is 2 to 3 times higher for African Americans than for Whites (Tang et al., 1998, 2001).

Additional studies would be most productive by examining the impact of drinking and smoking in combined samples of White and African American individuals. As we have demonstrated, control of a large list of variables, including hypertension, education, and age will be critical in providing a focused examination of direct effects of these behaviors across cognitive domains.

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