

The Relation between Alcohol and Tobacco Use and Cognitive Function in the Cochran County Aging Study

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Abstract

Purpose: To examine the relation between alcohol and tobacco use and cognitive function and to determine if any relations were specific to ethnic origin. **Methods:** Data were collected via interviews conducted with adults aged 40 years or older in Cochran County, Texas. Demographics, self report of current or past alcohol and tobacco use, and Mini-Mental Status Examination (MMSE) performance was examined. Participants who were without dementia and were either of Mexican American (n = 127) or non-Hispanic white (n = 100) ancestry were included. **Results:** Mexican American participants were significantly younger and had fewer years of self-reported education compared to non-Hispanic white participants. Alcohol use status (never used, previously used, or currently use) did not differ between groups. Tobacco use pattern did significantly differ with Mexican American participants reporting higher rates of current (29% versus 14%) and lower rates of no history of tobacco use (50% versus 60%). No significant relation between alcohol use pattern and MMSE cognitive performance was found; however, a trend was noted between current smoking among Mexican American participants and lower MMSE scores. **Conclusion:** In Phase 2 more detailed and quantitative assessments of alcohol, tobacco, and drug use as well as cognitive functioning will be conducted, which will better quantify the impact of substance use on cognitive function during aging as well as better determine if substance use patterns interact with other variables (e.g., cardiovascular disease and diabetes) to alter cognitive function during aging.

Deficits in multiple cognitive domains have been reported among the alcohol use disorders (AUD) including memory, working memory, executive functioning, abstract reasoning, *TPHA Journal*

processing speed, and visuospatial abilities.^{1,2,3} However, impairments in abstract reasoning and processing speed are of particular concern given that deficits of similar magnitude are observed among short- and long-term alcoholics indicating these deficits can be produced by relatively brief drinking histories.⁴ Given these cognitive deficits, it should come as no surprise that the neuropathology of AUD's include prominent atrophy in the parahippocampal and prefrontal cortex, hippocampus, and cerebellum.^{5,6} Interestingly, numerous large scale studies have found a "J-shaped" relation between alcohol use and cognitive function in later life. Specifically, it has been noted that light to moderate drinkers have better cognitive functioning compared to abstainers or heavy drinkers (with heavy drinkers having the worst overall outcomes across groups).^{7,8} In fact, it appears that among individuals with mild cognitive impairment, light to moderate alcohol intake appears to slow the progress of the development of dementia.⁹ Although not totally understood, the relation between moderate alcohol intake and preserved cognitive function may be causal in nature via the ameliorating effect of alcohol on cardiovascular risk factors such as vessel ischemia and hypertension or its effect of reducing insulin resistance. Inflammatory markers related to cardiovascular disease and cognitive decline have been shown to increase or decrease based on pattern or absence of alcohol intake.^{10,11,12,13} Likewise, the association may be unrelated to direct alcohol intake. Specifically, light to moderate drinkers have been shown to have better overall health habits as well as a higher level of education, and come from higher socioeconomic groups, each of which are predictive of better cognitive functioning later in life.

Cognitive deficits are also associated with tobacco smoking including problems with memory, working memory, executive functioning, and cognitive flexibility.^{14,15,16,17,18} MRI has demonstrated reduced volume of prefrontal and anterior cingulate cortices of adult smokers.¹⁹ These cognitive deficits and patterns of neuropathology are strikingly similar to the deficits observed among individuals with alcohol use disorders, and these problems could worsen when smoking is comorbid with problem drinking. This is an important consideration given that 98% of current smokers drink and 45-50% of binge drinkers smoke.²⁰ In fact, alcoholics who smoke have greater frontal and parietal gray matter hypoperfusion compared to nonsmoking alcoholics or nonsmoking light drinkers.²¹

The purpose of the current study was to examine whether reported past or current alcohol or tobacco use history was related to level of cognitive functioning as measured by the Mini Mental Status Examination (MMSE)²² among participants in the first wave of the Cochran County Aging Study. Additionally, this study sought to examine whether there was a differential impact on cognitive functioning of use of the substances between Mexican American and non-Hispanic white Caucasians in this rural sample.

Methods

Participants

The sample recruitment methodology and data collection pro-

cedures for the Cochran County Aging Study are described in detail by O'Bryant et al earlier in this issue. In the current analysis, African American and non Mexican Hispanic participants were excluded from the study due to limited representation in the sample. Also, those who met criteria for diagnosis of dementia were excluded. The final sample used included 227 Mexican American and 100 non-Hispanic white participants.

Results

Demographics for each group are presented in Table 1. Group differences in reported alcohol and tobacco use history (e.g., never used, previously used, or currently use) are presented in Table 2. As seen from this table, there was a significant difference between groups in tobacco use history with a greater percentage of the Mexican American participants being current smokers and a lesser percentage reporting never smoking tobacco.

Table 1.

Demographics of Participant Sample

	Mexican American	European American	Total Sample
Number	127 (55.9%)	100 (44.1%)	227 (100%)
Female	82 (64.6 %)	70 (70.0%)	152 (67.0%)
Age (Years) ¹	57.2 ± 10.9	69.1 ± 12.4	62.4 ± 13.0
Education (Years) ¹	6.4 ± 3.9	12.6 ± 2.1	9.2 ± 4.4

¹ Significant difference between groups ($p < .001$, independent t-tests, $df = 225$)

Table 2.

Self-Report of Alcohol and Tobacco Use History in the Participant Sample

	Mexican American	European American	Total Sample
Never Used Alcohol	68 (53.5 %)	61 (61.0%)	129 (56.8%)
Prior Alcohol Use	40 (31.5 %)	18 (18.0%)	58 (25.6%)
Current Alcohol Use	19 (15.0 %)	21(21.0%)	40 (17.6%)
Never Used Tobacco ¹	63 (49.6 %)	60 (60.0%)	123 (54.2%)
Prior Tobacco Use ¹	27 (21.3 %)	26 (26.0%)	53 (23.3%)
Current Tobacco Use ¹	37 (29.1 %)	14 (14.0%)	21 (22.5%)

¹ Significant difference between groups ($p < .05$, contingency coefficient = .177)

Given the significant group differences between age and education, analysis of covariance (ANCOVA) analyses were performed to examine if ethnic group and either alcohol or tobacco use classifications were related to MMSE performance while controlling for age and education. With regard to alcohol, education ($F_{1,219} = 35.3$, $p < .001$) and age ($F_{1,219} = 11.8$, $p = .001$) were found to be significant predictors. Specifically, among those participants who reported drinking alcohol, those that were younger who had more years of education tended to have higher MMSE scores. There was also a significant main effect for ethnicity ($F_{1,219} = 5.7$, $p < .05$) observed with non-Hispanic whites scoring higher than their Mexican Americans peers. However, alcohol use groupings (e.g., never, previous, or current alcohol use) predicted essen-

tially no variance in MMSE scores ($F_{2,219} = 0.1$) and the mean difference in MMSE scores between ethnic groups was similar across alcohol use classifications (see Table 3). The interaction between ethnicity and alcohol history was not significant.

Data on tobacco use also revealed that education ($F_{1,219} = 43.4$, $p < .001$) and age ($F_{1,219} = 14.0$, $p < .001$) were again significant predictors with younger and more educated participants who reported smoking tending to have higher MMSE scores. There was again a significant main effect for ethnicity ($F_{1,219} = 4.9$, $p < .05$), however, tobacco use groupings (i.e., never, previous, or current) predicted minimal variance in MMSE scores ($F_{2,219} = 0.8$). The interaction between ethnicity and tobacco history predicted notably more variance than tobacco classification alone ($F_{1,219} = 2.4$, $p < .10$) indicating a potential differential impact of smoking on cognitive function based on ethnicity. This interaction was driven by the larger MMSE scores for the non-Hispanic whites compared to the Mexican Americans in the current user smoking classification as compared to the other tobacco use patterns classifications (see Table 3).

Table 3.

Estimated MMSE Scores Controlling for Age and Education¹

	Mexican American	European American	Difference
Never Used Alcohol	25.7	28.1	2.4
Prior Alcohol Use	26.3	28.4	2.1
Current Alcohol Use	26.6	29.1	2.5
Never Used Tobacco	26.2	28.3	2.1
Prior Tobacco Us	26.3	28.2	1.9
Current Tobacco Use	25.5	29.0	3.5

¹ Values are Marginal Means from the ANCOVA Analyses

Discussion and Future Directions

These preliminary analyses of the first wave of data collected as part of the Cochran County Aging Study (CCAS) did not reveal a significant relation between self-reported alcohol and tobacco use histories with current performance on the MMSE. There was, however, some indication of a potential interaction between ethnicity and current smoking such that Mexican American current smokers demonstrated lower MMSE scores than current non-Hispanic white smokers. The significant difference noted in MMSE scores between Mexican American and non-Hispanic whites participants found in the current analysis has been noted in multiple studies and is likely secondary to language, educational opportunity, and cultural differences between the ethnic groups (see Hobson et al, this issue).^{23,24}

A clear limitation to these preliminary analyses was the coarseness of the measures used to define patterns of alcohol and tobacco use. In particular, these measures asked individuals to describe themselves in terms of never used, past but not current use, or current use. Phase 2 of the CCAS project will employ more comprehensive, well established measures

such as the Alcohol Use Disorder Identification Test,²⁵ and the Fagerström Test of Nicotine Dependence,²⁶ as well as selected brief questioning on specific current use pattern. This assessment will provide more detailed information on duration, frequency, intensity, and latency of use over the lifespan yet not be burdensome in terms of length or time required. These measures will also serve to identify individuals who subsequently could be followed in far greater detail. Such participants will be contacted at a later date (with prior participant permission) and invited to take part in more intensive structured interviews assessing their alcohol, drug, and/or tobacco use. In these cases, the Semi-structured Assessment for the Genetics of Alcoholism (SSAGA-II) ²⁷ developed by the Collaborative Study on the Genetics of Alcoholism (COGA) for its large scale study of the genetics of alcohol dependence will be used.²⁸ The SSAGA II is part of a battery of detailed and comprehensive interviews regarding substance use that can be used for future genetic linkage or biomarker studies.

Phase 2 of the CCAS project will also include a biobank of stored serum and DNA, a medical examination, clinical lab work, as well as more detailed neuropsychological testing. With more detailed assessments of alcohol and tobacco use, more comprehensive cognitive assessment, the collection of biological samples allowing serum biomarker and genetic analyses, and more detailed data collection on health history and status, Phase 2 of the study will allow a far more comprehensive examination of the impact of alcohol and tobacco use on cognitive function during aging. It will then be possible to look at the impact of the use of alcohol and tobacco on age-related cognitive decline as well as genetic factors that may mediate or moderate the effects of use on this decline. Further, it will be possible to explore possible mechanisms of how alcohol and tobacco use impacts cognitive decline by assessing the role of inflammatory biomarkers or other indicators of pathological processes. Finally, Phase 2 also will allow assessment of how co-morbid substance use interacts with other disease states such as cardiovascular disease and diabetes in terms of cognitive decline. Studies of this nature made possible by the expansion of the Cochran County Aging Study will provide valuable and unique information both on the natural course of cognitive decline in a rural setting as well as potential differential impact based on ethnic and other key demographic variables.

References

1. Kokavec A, Crowe SF. A comparison of cognitive performance in binge versus regular chronic alcohol misusers. *Alcohol Alcohol*. 1999; 34:601-608.
2. Rourke, SB, Loberg, T. The neurobehavioral correlates of alcoholism. In Grant I, Adams KM, eds. *Neuropsychological assessment of neuropsychiatric disorders*. New York, NY: Oxford University Press; 1996: 423-485.
3. Schrimsher GW, Parker JD, Burke RS. Relation between cognitive testing performance and pattern of substance use in males at treatment entry. *Clin Neuropsychol*. 2007; 1:498-510.
4. Beatty WW, Tivis R, Stott HD, Nixon SJ, Parsons OA. Neuropsychological deficits in sober alcoholics: Influences of

chronicity and recent alcohol consumption. *Alcohol Clin Exp Res*. 2000; 24:149-154.

5. Beresford TP, Arciniegas DB, Alfers J, Clapp L, Martin B, Du Y, Liu D, Shen D, Davatzikos C. Hippocampus volume loss due to chronic heavy drinking. *Alcohol Clin Exp Res*. 2006; 30:1866-1870.
6. Sullivan EV. Human brain vulnerability to alcoholism: Evidence from neuroimaging studies. In Noronha A, Eckardt M, Warren K, eds. *Review of NIAAA's Neuroscience and Behavioral Research Portfolio*, NIAAA Research Monograph No. 34. Washington DC: NIAAA; 2000: 473-508.
7. Mukamal KJ, Kuller LH, Fitzpatrick AL, Longstreth WT Jr, Mittleman MA, Siscovick DS. Prospective study of alcohol consumption and risk of dementia in older adults. *JAMA*. 2003; 289:1405-1413.
8. Anttila T, Helkala EL, Viitanen M, Kåreholt I, Fratiglioni L, Winblad B, Soininen H, Tuomilehto J, Nissinen A, Kivipelto M. Alcohol drinking in middle age and subsequent risk of mild cognitive impairment and dementia in old age: a prospective population based study. *BMJ*. 2004; 329:539-542.
9. Solfrizzi V, D'Introno A, Colacicco AM, et al. Alcohol consumption, mild cognitive impairment, and progression to dementia. *Neurology*. 2007; 68:1790-1799.
10. Bermudez EA, Rifai N, Buring J, Manson JE, Ridker PM. Interrelationships among circulating interleukin-6, C-reactive protein, and traditional cardiovascular risk factors in women. *Arterioscler Thromb Vasc Biol*. 2002; 22:1668-1673.
11. Imhof A, Froehlich M, Brenner H, et al. Effect of alcohol consumption on systemic markers of inflammation. *Lancet*. 2001; 357:763-767.
12. Pai JK, Hankinson SE, Thadhani R, Rifai N, Pischon T, Rimm EB. Moderate alcohol consumption and lower levels of inflammatory markers in US men and women. *Atherosclerosis*. 2006; 186:113-120.
13. Volpato S, Pahor M, Ferrucci L, et al. Relationship of alcohol intake with inflammatory markers and plasminogen activator inhibitor-1 in well-functioning older adults: the Health, Aging and Body Composition study. *Circulation*. 2004; 109:607-612.
14. Ernst M, Heishman SJ, Spurgeon L, London ED. Smoking history and nicotine effects on cognitive performance. *Neuropsychopharmacology*. 2001; 25:313-319.
15. Gazdzinski S, Durazzo TC, Studholme C, Song E, Banys P, Meyerhoff DJ. Quantitative brain MRI in alcohol dependence: preliminary evidence for effects of concurrent chronic cigarette smoking on regional brain volumes. *Alcohol Clin Exp Res*. 2005; 29:1484-1495.
16. Kalmijn S, van Boxtel MP, Verschuren MW, Jolles J, Launer LJ. Cigarette smoking and alcohol consumption in relation to cognitive performance in middle age. *Am J Epidemiol*. 2002; 156:936-944.
17. Razani J, Boone K, Lesser I, Weiss D. Effects of cigarette smoking history on cognitive functioning in healthy older adults. *Am J Geriatr Psychiatry*. 2004; 12:404-411.
18. Schinka JA, Belanger H, Mortimer JA, Borenstein Graves A. Effects of the use of alcohol and cigarettes on cognition in elderly African American adults. *J Int Neuropsychol Soc*. 2003; 9:690-697.

19. Brody AL, Mandelkern MA, Jarvik ME, Lee GS, Smith EC, Huang JC, Bota RG, Bartzokis G, London ED. Differences between smokers and nonsmokers in regional gray matter volumes and densities. *Biol Psychiatry*. 2004; 55:77-84.
20. Weitzman ER, Chen YY. The co-occurrence of smoking and drinking among young adults in college: national survey results from the United States. *Drug Alcohol Depend*. 2005; 80:377-386.
21. Gazdzinski S, Durazzo T, Jahng GH, Ezekiel F, Banys P, Meyerhoff D. Effects of chronic alcohol dependence and chronic cigarette smoking on cerebral perfusion: a preliminary magnetic resonance study. *Alcohol Clin Exp Res*. 2006; 30:947-958.
22. Folstein MF, Folstein SE, McHugh PR. "Mini-Mental State": a practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*. 1975; 12:189-198.
23. Black SA, Espino DV, Mahurin R, Lichtenstein MJ, Hazuda HP, Fabrizio D, Ray LA, Markides KS. The influence of noncognitive factors on the Mini-Mental State Examination in older Mexican-Americans: findings from the Hispanic EPESE. Established Population for the Epidemiologic Study of the Elderly. *J Clin Epidemiol*. 1999; 52:1095-1102.
24. Jones RN. Identification of measurement differences between English and Spanish language versions of the Mini-Mental State Examination. Detecting differential item functioning using MIMIC modeling. *Med Care*. 2006; 44:S124-S133.
25. Babor TF, Higgins-Biddle JC, Saunders JB, Monteiro MG. AUDIT: The Alcohol Use Disorders Identification Test. Guidelines for Use in Primary Care. 2nd ed. Geneva, Switzerland: World Health Organization; 2001.
26. Heatherton TF, Kozlowski LT, Frecker RC, Fagerström KO. The Fagerström Test for Nicotine Dependence: a revision of the Fagerström Tolerance Questionnaire. *Br J Addict*. 1991; 86:1119-1127.
27. Bucholz KK, Cadoret R, Cloninger CR, Dinwiddie SH, Hesselbrock VM, Nurnberger JI Jr, Reich T, Schmidt I, Schuckit MA. A new, semi-structured psychiatric interview for use in genetic linkage studies: a report on the reliability of the SSAGA. *J Stud Alcohol*. 1994; 55:149-158.
28. Reich T. A genomic survey of alcohol dependence and related phenotypes: results from the Collaborative Study on the Genetics of Alcoholism (COGA). *Alcohol Clin Exp Res* 1996; 20: A133-A137.