

Smoking Worsens Neuro Ills in Heavy Drinkers

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SANTA BARBARA, CALIF. — Smoking appears to heighten neuropsychological deficits found in heavy social drinkers, researchers reported at the annual meeting of the Research Society on Alcoholism.

Specifically, deficits in executive functioning and balance seen in people who both smoked and drank heavily were significantly worse than those seen in heavy-

drinking nonsmokers, said Timothy C. Durazzo, Ph.D., a neuropsychologist and neuroscience researcher at the San Francisco Veterans Administration Medical Center.

“We believe smoking may compound alcohol-induced neurobiologic and neurocognitive dysfunction among individuals with alcohol use disorders,” said Dr. Durazzo following the meeting.

The neuropsychological results build on Dr. Durazzo’s earlier identification—by MRI and magnetic resonance spec-

troscopy—of specific brain metabolite deficits in the frontal lobes and subcortical structures of smokers who had recently undergone alcohol detoxification (*Alcohol. Clin. Exp. Res.* 2004;1849-60).

The study concluded that smoking exacerbates alcohol-related frontal lobe neuronal injury and cell membrane damage, but also has an independent adverse effect on subcortical structures.

In the current study, Dr. Durazzo and associates administered neuropsychologi-

cal tests to 33 socially functioning heavy drinkers, 13 of whom were also daily smokers, as well as to 22 nonsmoking light drinkers.

Heavy drinking was defined as consuming more than 80 drinks per month, but study subjects actually consumed considerably more. Nonsmoking heavy drinkers averaged 141 lifetime drinks per month, while heavy drinkers who smoked drank an estimated 227 drinks per month.

Subjects were mostly in their early 40s and had a relatively high level of education (14-15 years, on average). Most were males. No subject suffered a medical condition that could impair neurocognition.

Significant differences between smoking and nonsmoking heavy drinkers were seen on the Wisconsin Card Sorting Test, reflecting executive function, and on the Fregly-Graybiel Ataxia Battery, reflecting balance.

Nonsmoking heavy drinkers performed better than smoking heavy drinkers on every Wisconsin Card Sorting Test measure except nonperseverative errors.

Perseverative response scores, for example, averaged 14.7 for nonsmoking heavy drinkers and 24.5 for heavy drinkers who smoked. As a point of comparison, the matched controls who neither smoked nor drank heavily had an average perseverative response score of 12.1.

Ataxia and balance-related scores showed significant differences as well. Total raw scores on the Fregly-Graybiel Ataxia Battery and the Sharpened Romberg Test were 151.8 for nonsmoking heavy drinkers and 107.7 for smoking heavy drinkers. The nonsmoking light drinkers who served as controls scored significantly higher at 208.5.

By contrast, no significant differences were found in visuospatial memory and working memory among heavy drinkers who smoked, nonsmoking heavy drinkers, and controls.

Previous research has concluded that heavy drinking is associated with depleted cortical gray matter volume and diminished levels of N-acetylaspartate (a marker of neuronal viability) in the lower frontal white matter and gray matter, said Dr. Durazzo.

Cigarette smoke contains many toxic compounds that may have an added negative effect on brain structure and function, particularly the cortical gray matter and the frontal lobes—“critical components of functional circuits mediating executive and motor functions,” he explained in a poster presented at the meeting.

“Thus, chronic cigarette smoking may account for a portion of the dysfunction in executive functioning and balance that had been previously attributed solely to long-term heavy alcohol consumption,” he said.

METABOLIC SYNDROME: THE CLUSTER OF CARDIOMETABOLIC RISK FACTORS¹

- Decreased HDL-C
- Elevated blood pressure
- Elevated triglycerides
- Elevated fasting glucose
- Increased waist circumference (excess adipose tissue)

ADIPOSE TISSUE IS A METABOLICALLY ACTIVE ENDOCRINE ORGAN²

- More than just a storage facility for fat—it has metabolic effects²
- Associated with abnormal endocrine function—impacts secretions of bioactive substances that help regulate lipid and glucose metabolism²
- May lead to development of cardiometabolic risk factors like dyslipidemia, elevated blood glucose, and insulin resistance^{2,3}

A NEWLY DISCOVERED PHYSIOLOGIC SYSTEM

- The endocannabinoid system (ECS) impacts metabolic functions⁴
- Consists of signaling molecules and their receptors, including the cannabinoid receptors [CB₁ and CB₂]^{5,6}

CB₁ RECEPTORS MAY IMPACT LIPID LEVELS AND INSULIN SENSITIVITY⁴

- Located centrally in the brain and peripherally in liver, muscle, and adipose tissue^{4,8}
—ECS overactivity in adipose tissue is associated with decreases in the hormone adiponectin, which may be linked to dyslipidemia, insulin resistance, and intra-abdominal adiposity⁴
- At the center of a cascade of events with potential impact on cardiometabolic risk⁴
- May assist in regulating physiologic processes, eg, lipid and glucose metabolism⁴

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