# Study Suggests Sleep Apnea Link to Alzheimer's

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DENVER — The well-documented association between the apolipoprotein E ε4 allele and development of cognitive decline and Alzheimer's disease may be mediated at least in part by obstructive sleep apnea, Ruth O'Hara, Ph.D., said at the annual meeting of the Associated Professional Sleep Societies.

This is an exciting possibility, because although no therapies are available to delay the onset of dementia, continuous positive airway pressure (CPAP) offers a highly effective treatment for obstructive sleep apnea. It's possible that identifying and treating this disorder in apo E ε4–positive patients could delay or perhaps even prevent the onset of cognitive decline and Alzheimer's dementia, according to Dr. O'Hara of Stanford (Calif.) University.

She presented a cross-sectional study of 36 community-dwelling nondemented older adults—mean age 70 years—half of whom possessed the apo E ε4 allele. All were assessed for cognitive performance status by the Mini-Mental State Examination and Rey Auditory Verbal Learning Test. The presence and severity of obstructive sleep apnea were assessed using home ventilatory polygraphy.

The most striking study finding was that although there was no difference in cognitive function between the apo E ε4–positive and –negative groups overall, apo E ε4–positive individuals with sleep apnea as defined by a higher apnea/hypopnea index had lower memory scores as reflected by worse performance on the delayed recall and short-term recall components of the Rey test. The higher an apo E ε4–positive subject's apnea/hypopnea index, the lower their memory scores. In contrast, the apnea/hypopnea index was unrelated to memory function in individuals who didn't carry the apo E ε4 allele.

Daytime sleepiness was unrelated to cognitive performance in either group.

Dr. O'Hara said that it's impossible to tell from a cross-sectional study such as this whether sleep apnea is mediating the effect of the apo E ε4 allele as a risk factor for development of cognitive decline and Alzheimer's disease. That's a question that can be addressed only in a longitudinal study. On the strength of the provocative cross-sectional study findings, the National Institute of Mental Health has granted funding for Dr. O'Hara and coworkers to conduct a 150-subject prospective study. ■

## Lifestyle Factors May Affect Gene Carriers' AD Risk

STOCKHOLM — Individuals who carry the apolipoprotein E ε4 allele that increases the risk of developing Alzheimer's disease may lower their risk to that of a noncarrier through regular exercise, moderate fat intake, and low alcohol consumption, reported Tiia Ngandu at the 12th Congress of the International Psychogeriatric Association.

Ms. Ngandu and her associates studied 1,449 Finnish people who participated in the longitudinal, population-based Cardiovascular Risk Factors, Aging, and Dementia study (CAIDE) in 1972, 1977, 1982, or 1987. After an average follow-up of 21 years, the participants were aged 65-79 years when they were re-examined in 1998.

Based on questions answered at midlife, active individuals (exercised at least twice per week) who carried the ε4 allele had a significantly lower likelihood of developing Alzheimer's disease (AD) than sedentary carriers, said Ms. Ngandu, a doctoral student at the Aging Research Center at the Karolinska Institute, Stockholm.

Intake of polyunsaturated fatty acids did not alter the odds of developing AD in carriers, but high intake of saturated fatty acids was associated with significantly greater odds of AD in carriers, compared with a low intake.

Carriers who frequently consumed alcohol had a significantly higher likelihood of developing AD than carriers who drank infrequently or never.

None of the lifestyle factors reduced the risk of AD in noncarriers, she noted.

People with a parent with late-onset Alzheimer's disease may undergo genotyping for apolipoprotein E. A positive finding is not diagnostic of Alzheimer's disease, even in a symptomatic patient.

**—Jeff Evans**