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BDNF gene expression signals cognitive reserve against AD progression

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By Lucy Piper, Senior medwireNews Reporter

Brain-derived neurotrophic factor (*BDNF*) gene expression contributes to slowing of cognitive decline in older adults and may protect against the effects of Alzheimer's disease (AD) pathology, researchers report in *Neurology*.

"This may have substantive clinical implications, since slower rate of cognitive decline may postpone major AD-related negative outcomes, from patient and family suffering to institutionalization and early death", note Michal Schnaider Beeri (The Icahn School of Medicine at Mount Sinai, New York, USA) and Joshua Sonnen (University of Utah Medical Center, Salt Lake City, USA) in an accompanying editorial.

The findings arise from the study of 535 individuals who were assessed annually for cognitive decline and dementia for an average of 6 years prior to death, when the average age was 88.5 years.

The rate of cognitive decline measured from a composite of 17 tests was 0.10 U/year on average, but for individuals with high post-mortem *BDNF* expression levels in the 90th percentile cognitive decline was reduced by 48.3%, compared with those with levels in the 10th percentile.

The association between cognitive decline and high *BDNF* expression was strongest for patients with dementia, becoming weaker for those with mild cognitive impairment and was absent in those with normal cognition.

BDNF expression was independently associated with cognitive decline after taking into account common age-related brain pathologies. In a linear regression model adjusted for age, gender and education, *BDNF* expression explained 2.1% of the variance in cognitive decline, common neuropathologies 26.8% and demographics 3.3%.

In addition to this independent association, *BDNF* expression negatively correlated with AD pathology with high *BDNF* expression associated with a low extent of AD pathology. But in patients with high levels of AD pathology, high *BDNF* expression was strongly associated with slower cognitive decline. For example in two individuals with the highest AD pathology, in the 90th percentile, cognitive decline was 40% slower in one with high *BDNF* expression (90th percentile) than in the other with low expression (10th percentile).

This suggests that a higher level of brain *BDNF* expression may promote cognitive reserve against the effects of AD pathology in older adults, say the researchers led by Aron Buchman (Rush University Medical Center, Chicago, Illinois, USA).

This could be through compensatory mechanisms such as neurogenesis, synaptic plasticity and dendritic density, suggest Beeri and Sonnen.

"*BDNF* gene expression or its gene products might thus serve as a biomarker for cognitive reserve against AD progression", they say, adding: "This specific finding promotes the idea that increasing *BDNF* gene expression might be a reasonable therapeutic strategy for AD in humans."