

Neurons targeted by dementing illness may have evolved for complex social cognition

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Von Economo neurons (VENs) are uniquely shaped brain cells that seem to have evolved in a select group of socially complex species: great apes, humans, and, as reported last month, whales.

Across species, VENs are localized to frontal brain regions associated with cognition, emotion and social behavior. Frontotemporal dementia (FTD), a common neurodegenerative condition, is characterized by early breakdown in social and emotional awareness and is accompanied by atrophy and dysfunction in the brain areas where VENs are located.

A new study published in the December 2006 issue of *Annals of Neurology*, the official journal of the American Neurological Association, examined brain tissue acquired at autopsy and found that VENs were devastated in FTD.

Led by William W. Seeley, M.D., of the Department of Neurology at the University of California, San Francisco, researchers quantified anterior cingulate cortex VENs in seven patients with FTD, five with Alzheimer's disease (AD), and seven control subjects who were not demented. All FTD patients had prominent changes in social behavior and emotion, sometimes accompanied by deficits in cognitive function.

In contrast, AD patients had an array of cognitive symptoms, including memory and language impairment, with little change in social behavior. The researchers found early, severe, and selective loss of VENs in FTD, which showed a 69% reduction compared to AD and controls after



controlling for overall neuronal loss. "Our findings suggest that selective VEN loss is a defining feature of FTD but does not apply to AD," the researchers state, adding that future research should explore how VEN loss relates to specific social/behavioral deficits in FTD and other disorders where such deficits are a defining feature.

VENs receive chemical signals from dopamine, serotonin, and vasopressin systems that play important roles in social and emotional bonding. These functions are disrupted early in FTD but spared in AD, the authors note. While drugs aimed at preserving VEN function may help curtail FTD symptoms, basic studies of VEN biophysical and molecular properties are needed to clarify the mechanisms that cause VENs to degenerate in FTD. "Distinctive functions of these unique cells may prove invaluable in health, yet may also expose us to specific forms of developmental or later-life illness," the authors conclude. "The link forged here between VENs and FTD should spawn further studies of how human brain evolution relates to human brain disease."

Article: "Early Frontotemporal Dementia Targets Neurons Unique to Apes and Humans," William W. Seeley, Danielle A. Carlin, John M. Allman, Marcelo N. Macedo, Clarissa Bush, Bruce L. Miller, Stephen J. DeArmond. Annals of Neurology, December 2006, (DOI: 10.1002/ana.21055).

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