PsychoAAD-2013

OMICS Group Conferences
Accelerating Scientific Discovery

International Conference on
Psychology, Autism and Alzheimer's Disease

September 30 - October 1, 2013 San Antonio, Texas, USA

Brain resilience and plasticity in the face of Alzheimer's disease

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Abstract

Alzheimer's disease (AD), an age-related neurodegenerative disorder and the most common cause of dementia, represents the culmination of neuropathological changes thought to evolve over several decades. Recent findings from prospective longitudinal studies with autopsy component suggest that neuritic plaques and neurofibrillary tangles, the neuropathological hallmarks of AD, are not limited to individuals with dementia. These 'hallmark' pathologic changes can also be present in the brains of clinically normal older adults - a condition we defined as Asymptomatic AD (ASYMAD). I will review the historical background and highlight the combined clinical, pathologic and morphometric evidence related to ASYMAD from the Baltimore Longitudinal Study of Aging (BLSA). The BLSA was established in 1958, supported since by the National Institute on Aging (NIA) intramural research program, with an autopsy program in place since 1986 in combination with comprehensive neurologic and cognitive evaluations. Although it remains unclear whether ASYMAD individuals would remain clinically normal with longer survival, our findings suggest that they seem to be able to compensate for or delay the appearance of clinical symptoms well into old age. Understanding the nature of changes during this apparently asymptomatic state may shed light on the mechanisms that forestall the progression of the disease and allow for maintenance of cognitive health, an important area of research that has been understudied relative to the identification of risks and pathways to negative health outcomes.

Biography

Dr. Driscoll has received her PhD in Neuroscience from the Canadian Centre for Behavioural Neuroscience at the University of Lethbridge in Canada and completed postdoctoral studies at the National Institute on Aging/NIH in Baltimore, MD. She is currently an Assistant Professor at the University of Wisconsin-Milwaukee.

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