Working memory binding and dual-tasking can mark onset and progression of Alzheimer's disease

Citation for published version:

Link:
Link to publication record in Edinburgh Research Explorer

Document Version:
Peer reviewed version

Publisher Rights Statement:

General rights
Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy
The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.
ICAD, Paris, France, 16-21 July 2011

Working memory binding and dual-tasking can mark onset and progression of Alzheimer’s disease

Sonia Moreno (1), Mario A. Parra (2), Sarah E. MacPherson (2), Francisco Lopera (1), Sergio Della Sala (2) and Robert H. Logie (2)

(1) Grupo de Neurociencias de Antioquia, University of Antioquia, Colombia
(2) Human Cognitive Neuroscience and Centre for Cognitive Ageing and Cognitive Epidemiology, University of Edinburgh, UK

Abstract

Background: The temporary retention of conjunctions of features (i.e., working memory binding) and the distribution of cognitive resources during concurrent tasks (i.e., dual tasking) are working memory (WM) functions which appear to be sensitive to Alzheimer’s disease (AD). However, recent data collected from asymptomatic carriers of the mutation E280A-PS1 which leads to early-onset familial AD and from patients with full blown AD suggest that these functions are differentially sensitive to the early stages of the disease and to its progression. Methods: A WM binding task (Parra et al., 2010) and a dual task (MacPherson et al., 2007) both known to be sensitive to sporadic AD were given to asymptomatic carriers of the mutation E280A-PS1 and to familial AD patients. Results: WM binding is impaired earlier, even in the asymptomatic stages of the disease, and deteriorates faster than the dual-tasking ability. When binding performances reach floor, dual-tasking continues to provide a measure of cognitive deterioration. Conclusions: These data suggest that WM binding can be a useful cognitive marker for the early detection of the disease whereas dual-task functions could better monitor its progression. These findings have both theoretical and practical implications for early intervention and follow up of AD.