

From Neuroplasticity to Scaffolding: A Giant Step for Cognitive Aging Research?

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ABSTRACT

This paper is a review of cognitive aging research centred on the Scaffolding Theory of Aging and Cognition (STAC), a theory which brings together much of the previous research into cognitive aging over the past century and suggests directions for future work. From Santiago Ramon y Cajal, with his microscope and talented drawings, to today's researchers with psychological and neurobiological methods and technology, particularly neuroimaging techniques, such as fMRI, sMRI, PET, etc., enormous progress has been made, through cognitive reserve, dedifferentiation, compensation, hemispherical asymmetry, inhibition and neurotransmission, to the Scaffolding theory of aging and cognition and beyond. Prior to 1990, research was almost entirely behavioural, but the advent of neuroimaging has boosted research and given rise to a new domain known as cognitive neuroscience, combining behavioural and neurobiological approaches to investigate structural and functional changes in the aging brain. Having reviewed the existing literature on cognitive aging research, the author concludes that although the scaffolding theory brings together a significant body of work and ideas, it is not yet the single, unifying theory for researchers. However, it does represent a giant step toward that theory.

Keywords: Cognitive Aging Research, Neurobiology, Neuroimaging, Neuroimaging Techniques, Scaffolding Theory of Aging and Cognition (STAC)

1. INTRODUCTION

The Scaffolding Theory of Aging and Cognition (Park & Reuter-Lorenz, 2009) represents a giant step in cognitive aging research, a culmination of research done over 10-15 years, notably cognitive reserve, compensation and dedifferentiation theories.

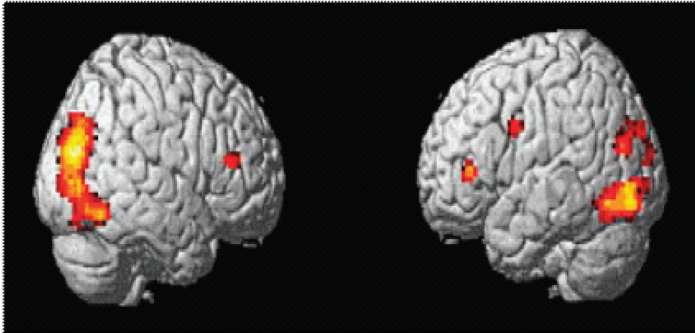
In elderly people, processing is activated more in the frontal cortices, with less in the mediotemporal and occipital cortices. Falling processing speed (Salthouse, 1996), auditory

and visual decline (Baltes & Lindenberger, 1997), the decline in grey matter (due to volume shrinkage, particularly in the hippocampus) and white matter integrity, with increasing evidence of white matter hyperintensities affecting neurotransmission are also put forward as reasons for cognitive decline (Figure 1).

Since the 1990s, the increasing development and use of neuroimaging techniques, such as positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) has revealed the loss of processing specificity in the elderly brain, with concomitant compensation by recruitment in other regions, mainly

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Figure 1. fMRI showing regions of activation common to young and old adults (Gutchess et al., 2005, used with permission)



prefrontal and contralateral - leading to the compensation (CRUNCH) (Reuter-Lorenz & Mikels, as cited in Reuter-Lorenz & Lustig, 2005), dedifferentiation (Baltes & Lindenberger, 1997), hemispherical asymmetry (HAROLD) (Cabeza, 2002) and PASA (Grady et al., 1994) models to explain hemispherical processing asymmetry in the elderly. In 2001, Park et al. were already working on compensation involving lifelong plastic, dynamic brain properties. In 2003, they focused on frontal and hippocampal activation at different ages (Park et al., 2003). The young showed increased left anterior hippocampal activity whereas the elderly showed both left and right activity and the authors suggested a compensatory role. With their eventual development of the STAC theory, scaffolding could be used to explain almost all the previous theories in an important intermediate step in this research. The future will focus on searching for scaffolding elsewhere in the brain and molecular and genetic research may define further details to contribute to our knowledge of cognition and aging. This review follows the STAC theory to the present day (results of the associated Synapse programme are due out in December 2011) and notes that thus far the theory seems to hold up remarkably well!

2. WHAT IS NEUROPLASTICITY?

2.1. Historical Developments Since 1890

The first recorded use of the word “plasticity” in the context of neuroscience was by William James, author of the *The Principles of Psychology*, in 1890, who defined plasticity as: “...the possession of a structure weak enough to yield to an influence, but strong enough not to yield all at once...” (*Principles of Psychology*, p. 68).

In 1904, Santiago Ramon y Cajal, the famous Spanish anatomist suggested that behavioural changes must have an anatomical basis in the brain. He said that

“... mental exercise leads to a greater development of the dendritic apparatus and of the system of axonal collaterals in the most utilized cerebral regions. In this way, associations already established among certain groups of cells would be notably reinforced by means of the multiplication of the small terminal branches of the dendritic appendages and axonal collaterals; but, in addition, completely new intercellular connections could be established thanks to the new formation of [axonal] collaterals and dendrites.” (The Cronian Lecture: La fine

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