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The effects of aging on visual memory: evidence for functional reorganization of cortical networks

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Abstract

Recent evidence suggests that the mature human brain is capable of substantial functional reorganization following injury. The fact that the brain retains a great deal of plasticity raises the possibility that cortical reorganization may occur during normal aging. We examined this issue by using positron emission tomography (PET) to measure the brain activity associated with short-term memory for simple visual attributes in young and old observers. A two-interval forced choice procedure was used to measure spatial frequency discrimination thresholds for sine wave gratings presented at different inter-stimulus intervals (ISI). Memory load was manipulated by varying the duration of the ISI and by presenting an irrelevant masking stimulus in the middle of the ISI. Old and young observers performed the experiment equally well. However, the neural systems correlated with good performance differed for the two age groups. The results support the hypothesis that the functional networks that underlie visual memory undergo reorganization during aging. © 2001 Published by Elsevier Science B.V.

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1. Introduction

How are psychological processes instantiated in the brain? One classical technique for addressing this issue is to measure how damage to different parts of the brain affects behaviour. Using this approach, several studies have shown that focal lesions often produce deficits in specific aspects of visual perception (Huxlin & Merigan, 1998; Merigan, Freeman, & Meyers, 1997; Merigan, 1996; Merigan & Pham, 1998; Newsome & Paré, 1988; Newsome & Wurtz, 1988; Pasternak & Merigan, 1994) and memory (Greenlee, Lang, Mergner, & Seeger, 1995; Greenlee, Rischewski, Mergner, & Seeger, 1993; Markowitsch, 2000; Zola & Squire, 2000). For example, Newsome and Paré (1988) found that lesions to visual cortical area MT reduced the performance of monkeys in a motion discrimination task, but not in an orientation discrimination task. Based on the specificity of the induced deficit, Newsome and Paré argued that activity in MT contributes selectively to motion perception. However, Newsome and Paré also found that the effects of MT lesions were not permanent: over a period of several days, monkeys fully recovered their ability to discriminate motion. The transient nature of the deficit suggested that the pathways subserving motion perception were reorganized after the lesion. Indeed, there is now substantial evidence that sensorimotor pathways in the mature brain are capable of extensive reorganization following injury (Buonomano & Merzenich, 1998; Kaas, Florence, & Jain, 1999; Liepert et al., 2000).

The fact that the mature brain retains a surprising degree of plasticity has implications for theories of cognitive aging. It is generally agreed that performance on a wide range of perceptual and cognitive tasks declines with age, and that age-related changes are much greater in some tasks than in others (Grady & Craik, 2000). Within the area of memory, for example, age differences are very small in priming tasks, but are very large in episodic retrieval tasks (Balota, Dolan, & Duchek, 2000). What are the biological underpinnings of these deficits? One possibility is that priming and episodic memory tasks tap into different sets of neural structures that support different memory systems (Tulving & Schacter, 1990), and that these systems age at different rates. For example, visual priming is associated with neural activity in extrastriate areas (Schacter & Buckner, 1998), whereas episodic memory is thought to be linked to activity in frontal areas (Lepage, Ghaffar, Nyberg, & Tulving, 2000). The effects of aging are not uniform throughout the brain (Anderson & Craik, 2000), so it is reasonable to suggest that the differential effects of aging in priming and episodic retrieval tasks reflects the structural integrity of extrastriate and frontal areas in old adults. Note that, according to this view, the failure to find a difference between old and young people in priming tasks means that the same neural circuitry underlies priming in both age groups. This interpretation of a null result is entirely appropriate, but the results reported by Newsome and Paré (1988) and others suggest that it may not always be correct. Specifically, demonstrations of plasticity in the adult brain raise the possibility that functional networks may be reorganized during normal aging, and therefore that the same behaviour in young and old adults may be controlled by different neural networks.

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