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Research report

Spatial structure normalises working memory performance in Parkinson's disease

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ABSTRACT

Cognitive deficits are a frequent symptom of Parkinson's disease (PD), particularly in the domain of spatial working memory (WM). Despite numerous demonstrations of aberrant WM in patients, there is a lack of understanding about how, if at all, their WM is fundamentally altered. Most notably, it is unclear whether span – the yardstick upon which most WM models are built – is compromised by the disease. Moreover, it is also unknown whether WM deficits occur in all patients or only exist in a sub-group who are executively impaired. We assessed the factors that influenced spatial span in medicated patients by varying the complexity of to-be-remembered items. Principally, we manipulated the ease with which items could enter – or be blocked from – WM by varying the level of structure in memoranda. Despite having similar levels of executive performance to controls, PD patients were only impaired when remembering information that lacked spatial, easy-to-chunk, structure. Patients' executive function, however, did not influence this effect. The ease with which patients could control WM was further examined by presenting irrelevant information during encoding, varying the level of structure in irrelevant information and manipulating the amount of switching between relevant and irrelevant information. Disease did not significantly alter the effect of these manipulations. Rather, patients' executive performance constrained the detrimental effect of irrelevant information on WM. Thus, PD patients' spatial span is predominantly determined by level of structure in to-be-remembered information, whereas their level of executive function may mitigate against the detrimental effect of irrelevant information.

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1. Introduction

Cognitive deficits, in addition to the characteristic motoric problems, are a frequent symptom of Parkinson's disease (PD; Brown & Marsden, 1988; Cools, 2006; Kehagia, Barker, & Robbins, 2012; Monchi, Hanganu, & Bellec, 2016; Owen, 2004). Aberrant performance on working memory (WM) tasks form a core component of these deficits (Bublak, Müller, Grön, Reuter, & von Cramon, 2002; Cools, Miyakawa, Sheridan, & D'Esposito, 2010; Lewis, Slabosz, Robbins, Barker, & Owen, 2005; Poewe, Berger, Benke, & Schelosky, 1991), with impairments most robustly seen in the spatial domain (Owen, Iddon, Hodges, Summers, & Robbins, 1997; Postle, Jonides, Smith, Corkin, & Growdon, 1997). Deficits on these tasks are largely thought to occur due to the dopaminergic abnormalities that characterise the disease (Sawamoto et al., 2008).

However, despite these demonstrations, we still lack an understanding of whether the basic architecture of WM – how information is encoded, stored and recalled – is altered in PD. The amount of information that can be recalled – WM span – is the basic currency in which different mnemonic models are constructed, compared and evaluated (Fallon, Zokaei, & Husain, 2016). Measures of WM span are also related to measures of real-world success (Gathercole, Brown, & Pickering, 2003). Thus, any exploration of the architecture of WM deficits in PD should seek to identify the factors that determine WM span.

The Corsi block-tapping task is a common test of spatial span (Milner, 1971). The basic structure of this task is to require individuals to observe and encode a sequence of spatial locations and then, after a short delay, reproduce this sequence by touching the remembered locations. Although there have been several studies of spatial span in PD patients using Corsi-like tasks, the results have been mixed. Despite numerous demonstrations of intact spatial spans in early medicated PD, reduced spans have also been reported (Fournet, Moreaud, Roulin, Naegele, & Pellat, 2000; Kemps, Szmalec, Vandierendonck, & Crevits, 2005; Stoffers, Berendse, Deijen, & Wolters, 2003). There are likely to be two principal reasons for this: a failure to control for the complexity of memoranda and cognitive heterogeneity (executive performance) in patients.

Here, we consider four factors that may influence the complexity of memoranda: level of structure in the to-be-remembered information, presence of irrelevant information, structure in the irrelevant information and degree of switching that is required between relevant and irrelevant information. Firstly, with regards to structure, most studies that have examined spatial span in PD patients have failed to control for the extent to which the to-be-remembered sequences can be re-organised into familiar or regular structures, i.e., the extent to which information can be chunked (Miller, 1956). This factor has been found to be a key determinant of performance on span tasks, with higher spans and concomitant increases in dorsolateral prefrontal cortex (DLPFC) activity observed during the encoding of structured verses unstructured material (Bor & Owen, 2007; Bor, Cumming, Scott, & Owen, 2004; Bor, Duncan, Lee, Parr, & Owen, 2006; Bor, Duncan, Wiseman, & Owen, 2003). The failure to control for this factor may also lead to a misrepresentation of patients' mnemonic abilities, either because encoding easily-chunked information 'normalises' their span (due to it being easier), or, because they are unable to derive the normative enhancement in spatial span when encoding easily-chunked information. For example, patients with moderate Alzheimer's disease fail to show improvements in span when remembering structured material (Huntley, Bor, Hampshire, Owen, & Howard, 2011).

Secondly, impaired span in PD patients may only appear when irrelevant information has to be ignored. This line of reasoning stems from observations that the basal ganglia – particularly its modulation by dopamine – are thought to be essential for filtering out irrelevant information (Baier et al., 2010; Gruber, Dayan, Gutkin, & Solla, 2006; McNab & Klingberg, 2008). In line with this, PD patients' WM deficits have been found to be exacerbated by irrelevant information (Lee et al., 2010).

Thirdly, however, there may be a modulatory role of salience in influencing the detrimental effect of irrelevant information. As the mirror-image of what occurs when relevant information is structured, were irrelevant information to contain structure its salience may increase and thus be harder to ignore. Such an effect could be anticipated on the basis that PD patients have already been shown to have impaired capacity to ignore salient information in the attentional domain (Cools, Rogers, Barker, & Robbins, 2010). Therefore, we sought to determine whether a similar effect can be detected in the mnemonic domain by varying the level of spatial structure in the irrelevant as well as the relevant information.

Finally, the detrimental effect of irrelevant information on patients' mnemonic performance may be contingent upon its prior relevance, especially given the established literature showing that this group has impairments in switching attention and task sets (Cools, Barker, Sahakian, & Robbins, 2001; Fales, Vanek, & Knowlton, 2006; Hayes, Davidson, Keele, & Rafal, 1998; Owen et al., 1993; Pollux, 2004). Indeed, in support of this claim, Moustafa, Sherman, and Frank (2008) found that mnemonic impairments in PD patients became more pronounced when they had to remember previously irrelevant information. Therefore, as a final manipulation of the complexity of memoranda, this study varied the extent to which participants had to update their demarcation between relevant and irrelevant information by including a condition in which they had to switch to attending to previously irrelevant information (where the relevant and irrelevant information were defined by colour; Fig. 1).

A perennial problem in characterising cognitive performance in PD is patient heterogeneity (Owen, 2004). For example, only a subgroup of PD patients, in the absence of dementia, exhibit deficits on so-called executive tasks such as planning, WM and attention (Kehagia et al., 2012; Tremblay, Achim, Macoir, & Monetta, 2013; Williams-Gray et al., 2013), though estimates of prevalence vary (Aarsland et al., 2010). Thus, the appearance of WM deficits in patients, and the resulting conclusions, may greatly depend upon the baseline executive performance level of that sample. One way to circumvent this problem is to test a larger group of patients with varying levels of executive performance. Performance on the Tower of London (TOL) task has been used to stratify