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Working memory recall precision is a more sensitive index than span

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Delayed adjustment tasks have recently been developed to examine working memory (WM) precision, that is, the resolution with which items maintained in memory are recalled. However, despite their emerging use in experimental studies of healthy people, evaluation of patient populations is sparse. We first investigated the validity of adjustment tasks, comparing precision with classical span measures of memory across the lifespan in 114 people. Second, we asked whether precision measures can potentially provide a more sensitive measure of WM than traditional span measures. Specifically, we tested this hypothesis examining WM in a group with early, untreated Parkinson's disease (PD) and its modulation by subsequent treatment on dopaminergic medication. Span measures correlated with precision across the lifespan: in children, young, and elderly participants. However, they failed to detect changes in WM in PD patients, either pre- or post-treatment initiation. By contrast, recall precision was sensitive enough to pick up such changes. PD patients pre-medication were significantly impaired compared to controls, but improved significantly after 3 months of being established on dopaminergic medication. These findings suggest that precision methods might provide a sensitive means to investigate WM and its modulation by interventions in clinical populations.

Working memory (WM) provides a temporary storage system for maintenance, storage, and manipulation of information in order to support cognitive processes (Baddeley, 2003). It is a critical contributor to many essential cognitive functions such as reasoning, language comprehension, learning, planning, and general fluid intelligence (Baddeley, 1986; Conway *et al.*, 2005; Engle, Tuholski, Laughlin, & Conway, 1999).

Classically, the storage or short-term memory component of WM has been tested using 'span' measures where participants are asked to remember a string of stimuli (e.g., digits) and recall them in the same order (forwards). Such simple tasks measure short-term maintenance (Groeger, Field, & Hammond, 1999). However, WM is not just the simple retention of information but might also involve additional processing demands. In many studies, the effects of such extra demands have been indexed by performance on

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backwards span tasks, where participants are asked to recall stored information in reverse order (Miller, Price, Okun, Montijo, & Bowers, 2009). This combines simple retention of information with a processing demand (Atkinson & Shiffrin, 1968; Oberauer, Lange, & Engle, 2004).

Span tasks such as these have been shown to be both reliable and valid measures of WM capacity (Conway *et al.*, 2005) and are considered to directly tap into the ability to manipulate maintained information (Cornoldi & Mammarella, 2008; Groeger *et al.*, 1999). Both factor analysis (Kail & Hall, 2001; Oberauer, Süß, Wilhelm, & Wittman, 2003) and neuroimaging studies (Fletcher & Henson, 2001; Postle, Berger, & D'Esposito, 1999) have shown a dissociation between simple (i.e., forwards) versus complex (i.e., backwards) memory tasks (Rosen & Engle, 1997).

Although such 'quantal' measures of performance have been fundamental to developing our understanding of memory function, they might not be sensitive to detect differences in memory *resolution*. A key aspect of span tasks is that they rely on binary (correct versus incorrect recall) measures: for any given item in a sequence, the participant either gets the correct answer or not, and the assessor obtains the number of items that can be retained in WM by that individual. But just because a participant makes an error on an item, it does not mean they had no memory of it at all. Conversely, a correct response does not tell us how well they remembered an item, that is, the resolution with which that item was retained (see (Ma, Husain, & Bays, 2014).

Recently, an alternative theoretical and empirical approach to WM has been developed that investigates the *precision* with which items are maintained in WM using delayed adjustment tasks (Gorgoraptis, Catalao, Bays, & Husain, 2011; Ma *et al.*, 2014; Wilken & Ma, 2004; Zhang & Luck, 2008; Zokaei, Heider, & Husain, 2014). Adjustment tasks rely on remembering a feature and *reproducing* the exact qualities of the stored feature after a retention period. For example, participants are presented with a sequence of oriented coloured bars and are asked to reproduce the exact orientation of one of the bars, probed by its colour, following a delay. Despite their emerging use in studies of WM (Peich, Husain, & Bays, 2013; Pertzov *et al.*, 2013), adjustment tasks have received little attention in testing patient populations.

The aim of current paper is first to determine whether WM precision shows convergent validity with commonly used clinical measures of WM – specifically span. One possibility is that precision of recall might simply correlate with forward span in traditional tests. However, adjustment tasks do not simply rely on passive maintenance of single features. They require participants to retain the correct conjunction of features belonging to an item *and* to retrieve one of the remembered features (e.g., orientation of bar) when probed by another feature (e.g., colour). One feature of an object is used to probe another feature that belonged to the object, thereby increasing processing demand compared to simple storage and free recall of a sequence consisting of say letters. Therefore an alternative hypothesis might be that precision of recall might correlate with performance on backwards span tasks, which also increase processing demand compared to forward span tests.

In order to establish the relationship between precision and span measures we first tested a group of children, young and elderly healthy participants on both types of task. Second, we asked whether precision measures can potentially provide a more sensitive measure of WM that can aid researchers working with clinical populations, including intervention studies and using relatively small sample sizes. Specifically, we examined WM deficits in early Parkinson's disease (PD) and its modulation by dopaminergic medication.

Working memory impairments have long been associated with PD, though studies using classical 'quantal' measures of memory have reported inconsistent results in the early stages of the disease are inconsistent (Dujardin, Degreef, Rogelet, Defebvre, & Destee, 1999; Muslimovic, Post, Speelman, & Schmand, 2005; Owen *et al.*, 1992; Owen, Iddon, Hodges, Summers, & Robbins, 1997; Savica, Rocca, & Ahlskog, 2010; Verbaan *et al.*, 2007). Because of the potentially more sensitive nature of precision measures, we hypothesized that it might be possible to detect small WM impairments in early PD and examine improvement of this cognitive function later on medication using this methodology.

Methods and materials

Participants

In total, we assessed 114 healthy individuals across the lifespan. Seventy-three children (aged 9–13) were recruited from a single-sex (male) school, randomly selected from each school year, excluding individuals with a known developmental disorder (participants from study by Burnett Heyes, Zokaei, van der Staaij, Bays, & Husain, 2012). In addition, 41 adults – 21 young (aged 18–29) and 20 older volunteers (aged 53–80) – participated in this study. Information on all participants are presented in Table 1.

For the assessment of a clinical group, 12 newly diagnosed PD patients (aged 51–79) were tested pre-medication and later ~3 months after being established on dopaminergic medication. Half the patients were on dopamine agonist while the remaining half were on Levodopa with average daily levodopa equivalent dose of 647 mg (Tomlinson *et al.*, 2010).

All participants reported having normal or corrected to normal vision and colour vision. They provided written informed consent (parental consent for children) to the procedure of the experiment, approved by local ethics committee.

Tasks

Delayed adjustment WM task

Stimuli were presented on a laptop monitor (32° × 19°) at a viewing distance of ~52 cm. A schematic representation of the task is shown in Figure 1A. In each trial, a sequence of coloured bars (2° × 0.2°) was presented at screen centre on a grey background. Each bar was presented for 500 ms followed by a 500 ms blank interval prior to the presentation of

Table 1. Demographics and participant information

	Mean age (Age range – years)	Gender		Years of education Mean (*SD*)	Daily Levedopa Equivalent dose
		Male	Female		
Child participants (n = 73)	11.5 (9–13.5)	73	-	–	–
Young participants (n = 21)	22 (18–29)	15	7	16 (2)	–
Healthy Elderly (n = 20)	62 (53–80)	9	11	15 (3)	–
PD patients (n = 12)	65 (51–79)	6	6	14 (3)	647 (on medication)

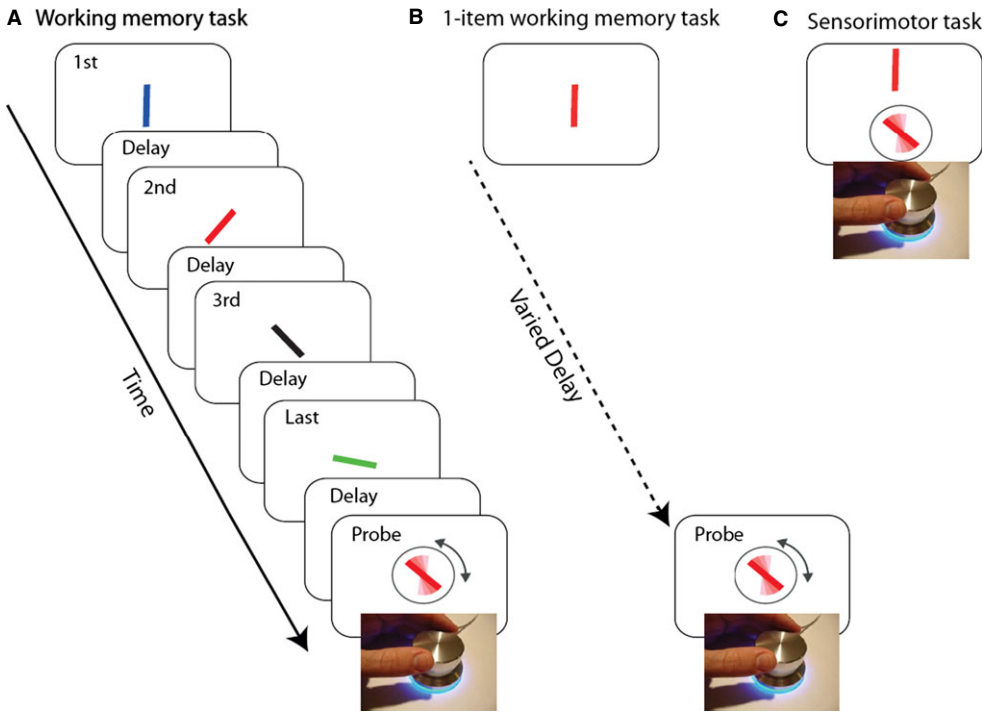

Figure 1. Tasks. (A) Working memory paradigm: A sequence of 4 coloured oriented bars (3 for children) were presented sequentially. Any of the bars could be probed by colour of the response stimuli. Participants were asked to adjust the orientation of the response stimuli to their memory of the orientation of the bar with same colour. (B) Control delay task: 1-item working memory task. A rotating dial is used to orient the probe bar (surrounded by circle) to match the orientation of the target bar presented following a delay which was varied to match the different delays for items at each serial position in the working memory task. (C) Control sensorimotor task: A rotating dial is used to orient the probe bar (surrounded by circle) to match the orientation of the target bar presented directly above the probe.

the next bar. For the children, we used sequences of three bars, while adults viewed 4 bars.

The colours in each trial were selected randomly, with no repetition from five easily distinguished colours (red, yellow, green, blue, and purple). Minimum angular separation between the orientation of bars within the same sequence was 10°; the orientation were chosen randomly otherwise. Participants were asked to remember the orientation of each bar.

At the end of each trial, a *probe bar* oriented randomly, in the same colour as one of the bars in the sequence was presented. A circle surrounding this probe made it easier to distinguish from bars in the sequence to-be-remembered. Participants were instructed to use a rotating dial to match the orientation of the probe to that of the same coloured bar in the sequence. The black circle surrounding the probe item disappeared upon rotating the dial. Participants clicked on the dial once they had rotated the dial to their selected orientation.

Stimuli presented in any of the serial positions within the sequence were probed with equal probability and participants did not know beforehand which item would be tested.

Children completed 90 trials while adult participants completed 200 trials. PD patients completed 100–200 trials depending on their availability.

Control tasks

Poor performance on the adjustment WM task might be due to factors other than the ability to maintain four/three items. To control for some of these, the following tasks were administered in random order, across the adult participants, including PD patients.

In the *pre-cueing task*, one of a four bars was cued, with a 100% valid cue, at the beginning of each trial where participants later had to recall. This test the ability to pay attention and filter out the non-cued items (bars) presented in a sequence. Both young and healthy participants completed 200 trials of this task while PD patients completed 100–200 trials depending on their availability.

In the *1-item task* a single bar was presented, following by a delay matching the durations from presentation of the probed bar at different serial positions within the WM adjustment task (Figure 1B). This tests for the simple effect of time (delay) on WM recall precision. Both young and healthy participants completed 200 trials of this task while PD patients completed 100–200 trials depending on their availability.

In PD patients, children and a subset of healthy elderly participants (n = 10), we also administered a *sensorimotor control task* (25 trials), where a probe appeared on screen below a target bar and participants had to adjust the probe bar's orientation to match the orientation of the target which remained on screen until response (Figure 1C). This is particularly important in the context of testing PD patients who might have difficulties with dexterity in using the dial and children who might have continuing maturation of fine motor precision and sensorimotor co-ordination across the age range in our sample (Pehoski, Henderson, & Tickle-Degnen, 1997).

Digit span

All participants performed both forwards and backwards digit span where letter sequences of 2 to ≤8 with two sequences per list length, up to and including the span where participants failed.

Corsi spatial span

Elderly participants and PD patients also completed forwards and backwards Corsi spatial blocks. Spatial sequences of 2 to ≤8 were used, with two sequences per list length, up to and including the span where participants failed.

Analysis

Delayed adjustment WM and control tasks

Recall performance was defined as the difference in response angle from target angle (i.e., the probed item). Precision was defined as the reciprocal of the circular standard deviation of error response (Bays & Husain, 2008) with less variability corresponding to more precise memory.

Performance in the sensorimotor control task in children correlated with age. If one assumes that sensorimotor error and recall error in the WM task contribute independently

to performance in the WM task, we can estimate recall precision, corrected for changes in sensorimotor performance by calculating precision as 1/sqrt (variance (WM error) – variance (sensorimotor error)) (Burnett Heyes *et al.*, 2012).

Digit and spatial span

We calculated span as the maximum sequence length which participants reported a minimum of one sequence per list length correctly. Half a score was deduced if participants performed only one of two correct per sequence length.

Results

WM precision correlates with span, across different age ranges

We first examined whether precision measures of WM correlate with digit or spatial span tasks of memory in healthy participants. Interestingly the positive effects were with backwards span measures, not forwards. WM precision correlated with backwards *digit span* across the lifespan: in children (r^2 = .26, p = .028; Figure 2A), young (r^2 = .612, p = .003; Figure 2B) and elderly healthy participants (r^2 = .5, p = .026; Figure 2C). Furthermore, WM precision correlated with backwards *spatial span* in elderly healthy

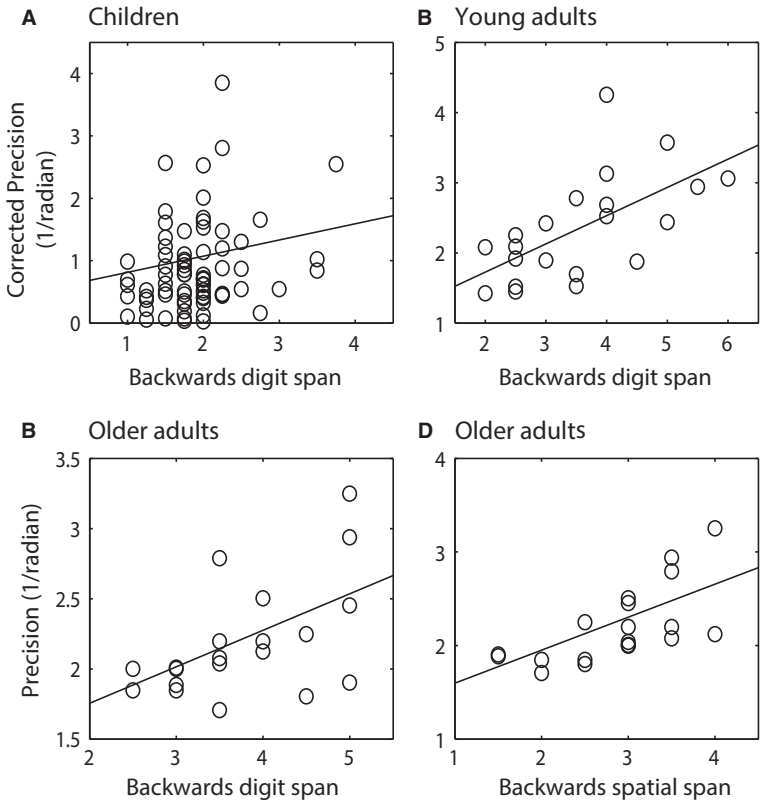

Figure 2. Relationship between span and precision of recall. Correlation between precision (corrected precision for children) and (A) backwards digit span in Children, (B) backwards digit span in Young adults, (C) backwards digit span in Older adults and (D) backwards spatial span in Older adults.

people (r^2 = .65, p = .002; Figure 2D). Thus WM precision correlated with traditional measures of WM, but only an index of 'processing demand plus storage' – backwards span – rather than forwards span, which is considered a measure of simple storage. Moreover, there was no significant correlation between backwards and forwards digit or spatial spans and our control measures (1-item or 1-item pre-cue conditions) in children, young or elderly participants.

WM precision, but not span, is modulated by dopamine in PD patients

Working memory precision in newly diagnosed unmedicated PD patients was significantly impaired compared to 16 healthy age-matched controls from the 21 elderly participants tested, $t(26) = 2.77$, $p = .010$; Figure 3A. After being established on daily dopaminergic treatment for ~3 months, there was a significant increase in precision, $t(11) = 3.01$, $p = .012$, reaching levels comparable – and not significantly different – to normal levels on medication, $t(26) = 1.05$, $p = .3$.

Working memory impairments in PD patients pre-medication compared to healthy controls was demonstrable at all serial positions within the sequence (Figure 3B; main effect of group, $F(1,104) = 15$, $p < .001$). Post-medication, there was a significant improvement in performance compared to pre-medication, across all positions (main effect of medication, $F(1,11) = 9.08$, $p = .012$, main effect of serial position, $F(3,33) = 12.6$, $p < .001$, with no significant interaction between medication and serial position: $F(3,33) = 2.5$, $p = .07$).

Parkinson's disease patients were also tested on forwards and backwards digit and spatial spans. Further, there was no significant impairment on these measures in patients pre-medication compared to healthy controls, on forwards digit ($p = .25$) and spatial span ($p = .78$) or backwards digit ($p = .44$) and spatial spans ($p = .84$). Furthermore, there was no significant improvement on dopaminergic medication in PD patients in either forwards digit ($p = .9$) or spatial span ($p = .6$), or backwards digit ($p = .8$) and spatial spans ($p = .21$; Figure 3C).

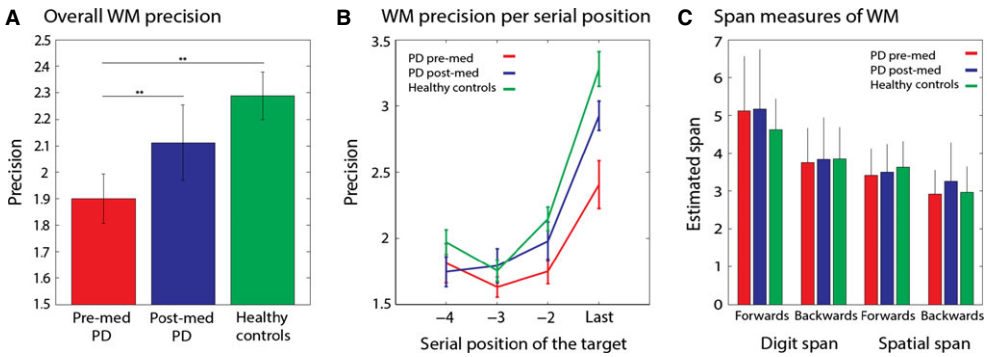

Figure 3. Precision of recall in Parkinson's disease before and after dopaminergic treatment. (A) Overall WM precision was significantly impaired in PD patients pre-medication and improved significantly after 3 months of dopaminergic medication. (B) Same pattern of findings was demonstrated at different serial positions of the target within the sequence. (C) There was no difference in forwards and backwards span measures of digit and spatial span in PD patients pre- and post-medication compared to healthy controls.

These findings demonstrate that although WM precision was impaired in PD patients pre-medication and later improved on medication, no such change in span measures was observed, suggesting that recall precision might be a more sensitive specifically when dealing with small sample sizes and subtle changes in WM performance.

WM deficits in PD patients cannot be explained by impairments in attentional filtering, temporal decay or sensorimotor performance

To examine whether the differences in performance on our WM task in PD patients were caused by impairments in factors other than in WM, we investigated performance on our three control tasks. There was no significant difference in the attention filtering (by pre-cueing), decay over time (1-item memory), and sensorimotor control tasks in PD patients pre- versus post-medication, and compared to healthy controls. Thus, changes in WM precision in PD patients cannot be explained by changes in attentional filtering, temporal decay or sensorimotor ability in PD.

Discussion

The primary aim of the present study was to establish whether WM precision tasks might provide useful measures in neuropsychological studies. The findings demonstrate a significant correlation between *backwards* – but not forwards – digit or spatial span and WM recall precision in healthy people. It has been suggested that forwards span measures short-term *storage*, while backwards span reflects storage *plus* processing demand (e.g., manipulation of information) within WM (Groeger *et al.*, 1999). Although the distinction between backwards and forward tasks is debated (Rosen & Engle, 1997), here we observed a correlation between only the backwards span measures and delayed adjustment tasks performance, potentially pointing to different underlying mechanisms, as previously suggested.

Delayed adjustment tasks, such as the one used here to measure recall precision, do not simply rely on passive storage of single features. They also require maintenance of the correct conjunction of features belonging to an item *and* retrieval of one of the remembered features (here, orientation) when probed by another feature (colour). Thus, we would contend that the processing demands of such a task is beyond simple storage. Consistent with this view, our measure of WM using a delayed adjustment task demonstrated a correlation with backwards span.

If these measures correlate with one another, then why bother to use WM precision? Adjustment tasks of WM provide a means to examine the *quality* of information maintained in WM rather than classic binary measures, investigating how well the information is retained, rather than whether an item was remembered or not (Ma *et al.*, 2014). Thus, these tasks potentially provide a more sensitive measure of WM with the potential to detect changes in performance when span tasks fail to do so. The second aim of our study was to compare WM precision and performance on backwards digit and spatial span, in a small group of PD, pre- versus post-medication, and compared to healthy controls.

Working memory precision was significantly impaired in PD patients pre-medication compared to healthy controls and importantly there was a significant improvement in recall precision after ~3 months of being established on a daily dopaminergic medication. Our findings on WM deficits associated with PD are supported by several studies (Morris

et al., 1988; Owen *et al.*, 1997; Postle *et al.*, 1999). However, the role of disease progression on WM processes is highly complex and some investigations have failed to identify WM impairments in non-medicated mild PD patients using variants of span tasks (Owen *et al.*, 1997; Owen *et al.*, 1992; Owen, 2004). Using sensitive precision measures of WM, we were able to demonstrate deficits in WM associated with early stages of the disease.

The role of dopaminergic treatment on WM in PD patients is also unclear. Dopamine has been shown to improve performance on spatial (Lange *et al.*, 1992) and N-back WM tasks (Mattay *et al.*, 2002) in PD patients, but other studies have failed to demonstrate any differences in WM between medicated versus non-medicated PD patients (Owen *et al.*, 1992). In the current report, WM precision significantly improved in early, newly diagnosed PD patients, following only 3 months of dopaminergic medication. Crucially, however, there was no difference in digit or spatial span, either forwards or backwards, in PD patients pre- versus post-medication.

Taken together the findings from the present research suggest that measurements of recall precision provide a potentially useful tool that can aid researchers working on clinical populations, intervention studies and small sample sizes to detect WM deficits and their modulation by interventions. Importantly, this methodology provides a means to dissect out sources of error contributing to the pattern of performance. Using computational modelling, it is possible to gain further mechanistic insight into the nature of WM deficits (see Bays *et al.*, 2009 for in depth description of the model and Zokaei, Heider *et al.*, 2014; Zokaei, McNeill *et al.*, 2014 for an example).

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References

Atkinson, R., & Shiffrin, R. (1968). Human memory: A proposed system and its control processes. *The Psychology of Learning and Motivation*, *8*, 13–113.

Baddeley, A. (1986). *Working Memory*. (New edition) Oxford, UK: Clarendon Press.

Baddeley, A. (2003). Working memory: Looking back and looking forward. *Nature Reviews Neuroscience*, *4*, 829–839. doi:10.1038/nrn1201

Bays, P. M., Catalao, R. F. G., & Husain, M. (2009). The precision of visual working memory is set by allocation of a shared resource. *Journal of Vision*, *9*, 1–11.

Bays, P. M., & Husain, M. (2008). Dynamic shifts of limited working memory resources in human vision. *Science (New York, N.Y.)*, *321*, 851–854. doi:10.1126/science.1158023

Burnett Heyes, S., Zokaei, N., van der Staaij, I., Bays, P. M., & Husain, M. (2012). Development of visual working memory precision in childhood. *Developmental Science*, *15*, 528–539. doi:10.1111/j.1467-7687.2012.01148.x

Conway, A. R. A., Kane, M. J., Bunting, M. F., Hambrick, D. Z., Wilhelm, O., & Engle, R. W. (2005). Working memory span tasks: A methodological review and user's guide. *Psychonomic Bulletin & Review*, *12*, 769–786. doi:10.3758/BF03196772

Cornoldi, C., & Mammarella, I. C. (2008). A comparison of backward and forward spatial spans. *The Quarterly Journal of Experimental Psychology*, *61*, 674–682. doi:10.1080/17470210701774200

Dujardin, K., Degreef, J. F., Rogelet, P., Defebvre, L., & Destee, A. (1999). Impairment of the supervisory attentional system in early untreated patients with Parkinson's disease. *Journal of Neurology*, *246*, 783–788. doi:10.1007/s004150050455

Engle, R. W., Tuholski, S. W., Laughlin, J. E., & Conway, A. R. (1999). Working memory, short-term memory, and general fluid intelligence: A latent-variable approach. *Journal of Experimental Psychology: General*, *128*, 309–331. doi:10.1037/0096-3445.128.3.309

Fletcher, P. C., & Henson, R. N. A. (2001). Frontal lobes and human memory Insights from functional neuroimaging. *Brain*, *124*, 849–881. doi:10.1093/brain/124.5.849

Gorgoraptis, N., Catalao, R. F. G., Bays, P. M., & Husain, M. (2011). Dynamic updating of working memory resources for visual objects. *The Journal of Neuroscience*, *31*, 8502–8511. doi:10.1523/JNEUROSCI.0208-11.2011

Groeger, J. A., Field, D., & Hammond, S. M. (1999). Measuring memory span. *International Journal of Psychology*, *34*, 359–363. doi:10.1080/002075999399693

Kail, R., & Hall, L. K. (2001). Distinguishing short-term memory from working memory. *Memory & Cognition*, *29*, 1–9. doi:10.3758/BF03195735

Lange, K. W., Robbins, T. W., Marsden, C. D., James, M., Owen, A. M., & Paul, G. M. (1992). L-dopa withdrawal in Parkinson's disease selectively impairs cognitive performance in tests sensitive to frontal lobe dysfunction. *Psychopharmacology (Berl)*, *107*, 394–404. doi:10.1007/BF02245167

Ma, W. J., Husain, M., & Bays, P. M. (2014). Changing concepts of working memory. *Nature Neuroscience*, *17*, 347–356. doi:10.1038/nn.3655

Mattay, V. S., Tessitore, A., Callicott, J. H., Bertolino, A., Goldberg, T. E., Chase, T. N., . . . Weinberger, D. R. (2002). Dopaminergic modulation of cortical function in patients with Parkinson's disease. *Annals of Neurology*, *51*, 156–164. doi:10.1002/ana.10078

Miller, K. M., Price, C. C., Okun, M. S., Montijo, H., & Bowers, D. (2009). Is the N-back task a valid neuropsychological measure for assessing working memory? *Archives of Clinical Neuropsychology*, *24*, 711–717. doi:10.1093/arclin/acp063

Morris, R. G., Downes, J. J., Sahakian, B. J., Evenden, J. L., Heald, A., & Robbins, T. W. (1988). Planning and spatial working memory in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, *51*, 757–766. doi:10.1136/jnnp.51.6.757

Muslimovic, D., Post, B., Speelman, J. D., & Schmand, B. (2005). Cognitive profile of patients with newly diagnosed Parkinson disease. *Neurology*, *65*, 1239–1245. doi:10.1212/01.wnl.0000180516.69442.95

Oberauer, K., Lange, E., & Engle, R. W. (2004). Working memory capacity and resistance to interference. *Journal of Memory and Language*, *51*, 80–96. doi:10.1016/j.jml.2004.03.003

Oberauer, K., Süß, H.-M., Wilhelm, O., & Wittman, W. W. (2003). The multiple faces of working memory: Storage, processing, supervision, and coordination. *Intelligence*, *31*, 167–193. doi:10.1016/S0160-2896(02)00115-0

Owen, A. M. (2004). Cognitive dysfunction in Parkinson's disease: The role of frontostriatal circuitry. *The Neuroscientist*, *10*, 525–537. doi:10.1177/1073858404266776

Owen, A. M., Iddon, J. L., Hodges, J. R., Summers, B. A., & Robbins, T. W. (1997). Spatial and non-spatial working memory at different stages of Parkinson's disease. *Neuropsychologia*, *35*, 519–532. doi:10.1016/S0028-3932(96)00101-7

Owen, A. M., James, M., Leigh, P. N., Summers, B. A., Marsden, C. D., Quinn, N. P., . . . Robbins, T. W. (1992). Fronto-striatal cognitive deficits at different stages of Parkinson's disease. *Brain*, *115*, 1727–1751. doi:10.1093/brain/115.6.1727

Pehoski, C., Henderson, A., & Tickle-Degnen, L. (1997). In-hand manipulation in young children: Rotation of an object in the fingers. *The American Journal of Occupational Therapy: Official Publication of the American Occupational Therapy Association*, *51*, 544–552. doi:10.5014/ajot.51.7.544

Peich, M.-C., Husain, M., & Bays, P. M. (2013). Age-related decline of precision and binding in visual working memory. *Psychology and Aging*, *28*, 729–743. doi:10.1037/a0033236

Pertzov, Y., Miller, T. D., Gorgoraptis, N., Caine, D., Schott, J. M., Butler, C., & Husain, M. (2013). Binding deficits in memory following medial temporal lobe damage in patients with voltage-gated potassium channel complex antibody-associated limbic encephalitis. *Brain*, *136*, 2474–2485. doi:10.1093/brain/awt129

Postle, B. R., Berger, J. S., & D'Esposito, M. (1999). Functional neuroanatomical double dissociation of mnemonic and executive control processes contributing to working memory performance. *Proceedings of the National Academy of Sciences of the United States of America*, *96*, 12959–12964. doi:10.1073/pnas.96.22.12959

Rosen, V. M., & Engle, R. W. (1997). Forward and backward serial recall. *Intelligence*, *25*, 37–47. doi:10.1016/S0160-2896(97)90006-4

Savica, R., Rocca, W. A., & Ahlskog, J. (2010). WHen does Parkinson disease start? *Archives of Neurology*, *67*, 798–801. doi:10.1001/archneurol.2010.135

Tomlinson, C. L., Stowe, R., Patel, S., Rick, C., Gray, R., & Clarke, C. E. (2010). Systematic review of levodopa dose equivalency reporting in Parkinson's disease. *Movement Disorders: Official Journal of the Movement Disorder Society*, *25*, 2649–2653. doi:10.1002/mds.23429

Verbaan, D., Marinus, J., Visser, M., van Rooden, S. M., Stiggelbout, A. M., Middelkoop, H. a. M., & van Hiltenvan, J. J. (2007). Cognitive impairment in Parkinson's disease. *Journal of Neurology, Neurosurgery & Psychiatry*, *78*, 1182–1187. doi:10.1136/jnnp.2006.112367

Wilken, P., & Ma, W. J. (2004). A detection theory account of change detection. *Journal of Vision*, *4*, 1120–1135. doi:10.1167/4.12.11

Zhang, W., & Luck, S. J. (2008). Discrete fixed-resolution representations in visual working memory. *Nature*, *453*, 233–235. doi:10.1038/nature06860

Zokaei, N., Heider, M., & Husain, M. (2014). Attention is required for maintenance of feature binding in visual working memory. *Quarterly Journal of Experimental Psychology (2006)*, *67*, 1191–1213. doi:10.1080/17470218.2013.852232

Zokaei, N., McNeill, A., Proukakis, C., Beavan, M., Jarman, P., Korlipara, P., . . . Husain, M. (2014). Visual short-term memory deficits associated with GBA mutation and Parkinson's disease. *Brain: A Journal of Neurology*, *137*, 2303–2311. doi: 10.1093/brain/awu143

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