Mechanisms underlying working memory in health and disease



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Abstract

Working memory (WM) — the means by which information is retained and manipulated over seconds — is a fundamental cognitive process that has been considered to have three phases: encoding, maintenance and retrieval. Although much research has focused on the first two of these, far less is understood about how information is retrieved from WM or how filtering of information in WM affects recall and forgetting, both in health and disease.

In the first part of the thesis, I gained mechanistic insight into the process of WM retrieval and the relationship between processing demands at different phases. Novel sensitive delayed-reproduction tests were developed, and mixture modelling of error applied to deconstruct the sources of error contributing to the pattern of recall performance. Additionally, to quantify the mechanisms of the decision process of recall, Hierarchical Bayesian Drift Diffusion modelling (HDDM) was implemented. In the second part, I investigated malfunctions of the underlying processes of WM in two different patient groups. First, I examined a rare group of people with medial temporal lobe (MTL) lesions following limbic encephalitis (LE). Second, as part of an experimental medicine study I assessed WM and the impact of emotional valence on WM in individuals with Parkinson's disease (PD) both on and off citalopram, a selective serotonin reuptake inhibitor (SSRI).

Overall, the results demonstrated that the nature of retrieval cues can significantly affect memory reports, with more information at retrieval improving access to items in WM. Additionally, the quality of associative retrieval depends not only on the

nature of the target, but also on features in a foil (distractor item) if this is also presented at probe, with the arrangement of non-spatial features modulating recall of relational information. Further studies provided data to suggest that filtering of information in WM is not a monolithic, single entity but rather underpinned by dissociable cognitive processes that engage with the different phases of memory.

Finally, results from the second part of this thesis provided evidence for a role of the MTL and hippocampal regions in relational WM binding. While serotonergic modulation with citalopram revealed a possible role of serotonin in processing negative emotional information in PD, no effects on WM were found. Together, this work provides novel insights into the mechanisms underlying WM retrieval, filtering and examination of the effects of processing demands induced at different stages of WM in the healthy brain. It also contributes to the understanding of memory disturbances in patients with MTL damage and PD.

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Acronyms

Abs-VGKC Antibodies to the Voltage-Gated Potassium Channels complex
ACE Addenbrooke's Cognitive Examination
AD Alzheimer's Disease
AIC Akaike Information Criterion
AMI Apathy Motivation Index
ANOVA Analysis of Variance
ATP Acute tryptophan depletion

BDI Beck Depression Inventory
BPIC Bayesian Predictive Information Criterion

CASPR2 Contactin-Associated Protein-like 2
CDA Contralateral Delayed Activity

DDM Diffusion Decision Model
DC Direct Current
DIC Deviance Information Criterion
DMC Dynamic Models of Choice

FAST FMRIB's Automated Segmentation Tool
FER Facial Emotional Recognition
FIRST FMRIB's Integrated Registration and Segmentation Tool
FIT Feature Integration Theory
FNIRT FMRIB's Nonlinear Image Registration Tool
FSL FMRIB Software Library

HDDM Hierarchical Bayesian estimation of the Drift Diffusion Model

LATER Linear Approach to Threshold with Ergodic Rate
LBA Linear Ballistic Accumulator Model
LE Limbic Encephalitis
LGI1 leucine-rich glioma-inactivated 1
LFP Local Field Potential
LTM Long Term Memory

MCMC Monte Carlo Markov Chain
MTL Medial Temporal Lobe

PET Positron Emission Tomography
PD Parkinson's disease

RBD REM sleep Behaviour Disorder
RT Response times

SWRs sharp-wave ripples
SD Standard Deviation
SERT Serotonin Transporter
SSRI Selective serotonin reuptake inhibitor
STM Short Term Memory

UPDRS Unified Parkinson's Disease Rating Scale

WM Working Memory

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List of Publications

Thesis material

Tabi, Y. A.*, Maio, M. R.*, Fallon, S. J., Udale, R., Dickson, S., Idris, M. I., … Husain, M. (2021). Impact of processing demands at encoding, maintenance and retrieval in visual working memory. Cognition, 214,104758.

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*The two authors contributed equally to this work, thus, share the first authorship.

All co-authors have given permission to include this material in this thesis.

Other material

Fallon SJ, Gowell M, Maio MR, Husain M. (2019). Dopamine affects short-term memory corruption over time in Parkinson's disease. *Njp Par Dis*. 5(1)

https://www.nature.com/articles/s41531-019-0088-2

Klar VS, Ang YS, Lockwood P, Attaallah B, Dickson S, Drew D, Kienast A; Maio MR, Plant O, Slavkova E, Toniolo S, Zambellas R, Irani SR, Husain M. (2021). Assessment of apathy in neurological patients using the Apathy Motivation Index caregiver version. Journal of Neuropsychology.

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Tabi YA, Maio MR, Attaallah B, Dickson S, Drew D, Idris MI, Kienast A, Klar V, Nobis L, Plant O, Saleh Y, Sandhu TR, Slavkova E, Toniolo S, Zokaei N, Manohar SG, Husain M. (2021). The vividness of visual imagery and its relationship to continuous measures of visual short-term memory. Cortex

Chapter 1. General Introduction

Memory is the description assigned to the processes and structures involved in the successful storage, maintenance and retrieval of information. Short-term memory (STM) is one of the components of WM, the means by which information is not only capable of being retained but also manipulated, over the order of seconds (A. Baddeley, 2003) and with its capacity intimately associated to cognitive ability and performance (S. J. Luck & Vogel, 2013). Research on WM has been primarily concerned with developing a proper understanding of its underlying architecture and limitations. In the first part of this introduction, I will focus on the mechanisms underlying WM limitations in health. I will start by describing the ongoing debate on the characterisation of WM capacity, followed by how studies designed to examine WM mechanisms have focused on precision methods to describe the pattern of errors contributing to memory performance. After presenting findings on how precision of WM decreases with memory load, I will highlight how response times (RT) from WM retrieval can also be used to provide a mechanistic perspective on cognitive processes. Finally, I will present a literature overview on the impact of attentional demands on recall performance at encoding and maintenance, using pre- or retro-cues, and how these have yet to be systematically examined across all three components of WM — encoding, maintenance and retrieval —using continuous measures of recall.

In the second part of this Introduction, I will address the diseased brain and how sensitive delayed-reproduction tasks have been able to dissociate WM impairment in different brain diseases: memory deficit resulting from MTL and hippocampal damage has been associated with higher misbinding rates, as opposed to patients with PD whose impairment has been shown to stem from increased random responses.

1.1. *Models of working memory*

1.1.1. The Modal Model or Multi-store Model

One of the most influential models of STM has been the Atkinson-Shiffrin's Modal model (Atkinson & Shiffrin, 1968). This representation of the memory process considered that stored information passes through three sequential memory structures that hold information for varying lengths of time (sensory registers, STM, and long-term memory) (Figure 1.1). The model posits that merely holding information in STM would guarantee its transfer to long-term memory (LTM). However, empirical evidence did not support this. For example, Craik and Lockart (1972) showed that deeper and more elaborate processing led to better learning. Another criticism raised against this model is that it assumes that information can only get in and out of LTM via the STM system, but Warrington and Shallice (1969) revealed that neurological patients with digit spans (verbal STM) reduced to one item could nevertheless be highly capable of learning and retaining lists of words and paired associations (verbal LTM).

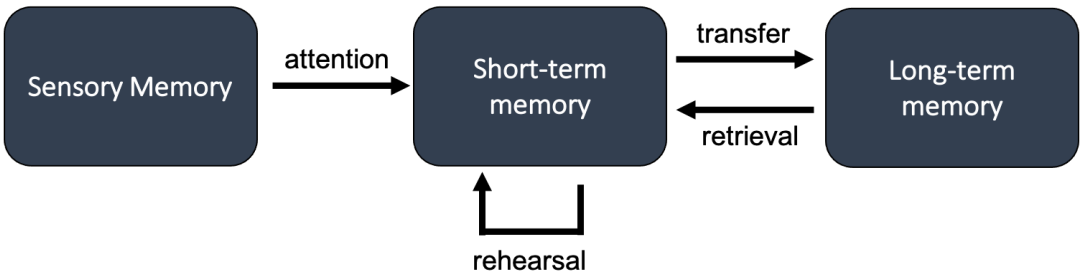


Figure 1.1| The multistore model of working memory.

Descriptive representation of the memory process multicomponent theory: stored and subsequently available information in memory passes through three sequential memory structures: sensory register, STM and LTM. The STM was regarded as essential for long-term learning, with greater rehearsal in STM associated with greater learning. This notion is no longer accepted. Adapted from Atkinson & Shiffrin, 1968.

1.1.2. Baddeley and Hitch Model: the visuospatial sketchpad

A few years later, in 1974, Baddeley and Hitch proposed a widely accepted multiple-component model of WM (A. Baddeley & Hitch, 1974) which has since evolved (A. Baddeley, 1992; A. Baddeley & Logie, 1992) with the postulation of three different components: the visuospatial sketchpad for remembering non-verbal information, the central executive for moving information in and out of storage, and the phonological loop for continuously repeating memorised verbal information (**Figure 1.2**).

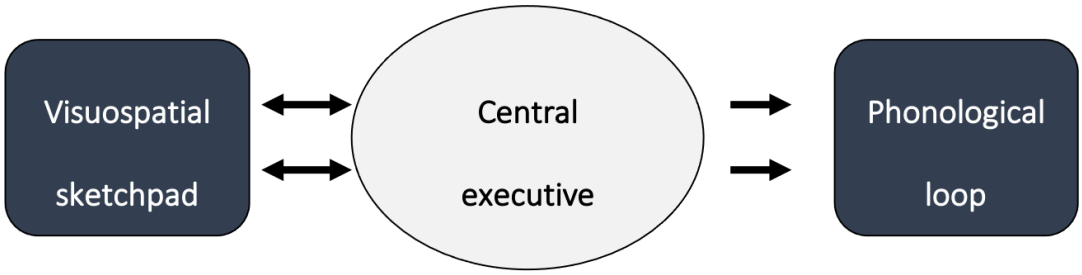


Figure 1.2| The visuospatial sketchpad.

Discontinuity of the one-dimensional system approach to WM and the shift towards a less peripheral understanding, with the inclusion of a central executive (central component) and three sub-systems: the phonological loop, the visuospatial sketchpad, and the episodic buffer. Adapted from Baddeley & Hitch, 1974.

According to this perspective, the *visuospatial sketchpad* is responsible for the temporary storage and manipulation of non-verbal information. It is thought to serve a similar function for visual or spatial material, playing a role in visual-imagery tasks, and to have obligatory access to the passive visual system (A. Baddeley, 1992; A. Baddeley & Logie, 1992; Salway & Logie, 1995). The *central executive* is a limited capacity attentional system responsible for coordinating information from the visuospatial sketchpad and the phonological loop, retrieval of strategic information from the LTM stores, and shifting attentional set, as well as inhibiting responses. It grants coordination while operating a

dual-task performance, both in the process of reasoning and problem solving (Gilhooly, Logie, Wetherick, & Wynn, 1993; Logie, Gilhooly, & Wynn, 1994). It is also posited to be involved in the allocation of attention (A. Baddeley, 1992). Finally, the *phonological loop* provides a temporary verbal storage. It incorporates a passive phonological store and an articulatory rehearsal process. Verbal information gains direct access to the passive store, which retains information in a phonological form and is subjected to both decay over time and interference from new verbal material. Nonetheless, the deterioration of stored information can be prevented by means of sub vocal rehearsal (A. Baddeley, 1992; A. Baddeley & Logie, 1992; Salway & Logie, 1995). However, although highly influential, this model might be best regarded as a theory as it is purely descriptive and lacks predictive power.

1.1.3. The Slot Model for visual working memory

Others have since developed more detailed models capable of testing the hypothesis of a limited capacity system that provides the temporary storage and manipulation of information, known as WM capacity. An ongoing debate has been on the characterisation of its limitations.

Traditionally, most studies have usually assessed WM using binary report measures (correct / incorrect response) and examine the quantity of information, i.e., number of items that can be maintained. Thus, the classic view, illustrated in **Figure 1.3**, describes visual WM as limited in nature with a storage capacity that varies between people, of about three to four items in average (Cowan, 2001; Vogel, Woodman, & Luck, 2001; for

a review on the classical view see Luck & Vogel, 2013). This perspective is based on the premise that WM is discrete and quantal, i.e., an object either enters into a memory slot– and is, then, remembered with high resolution – or it is not, which translates into the non-recollection of an object (J. S. Luck & Vogel, 1997).

Two main predictions of the slot model are that participants remember on a per-item basis regardless of the amount of features the items hold (Edward Awh, Barton, & Vogel, 2007; S. J. Luck & Vogel, 2013; Vogel et al., 2001), and that all memorised items are stored with the same resolution, even if at capacity. However, it has been acknowledged that the first prediction of this model — recall on a per-item basis — does not apply for more complex items holding a greater number of features (Alvarez & Cavanagh, 2002; Eng, Chen, & Jiang, 2005), despite some researchers positing that this is a consequence of higher shared similarities between complex stimuli (Fukuda, Awh, & Vogel, 2010).

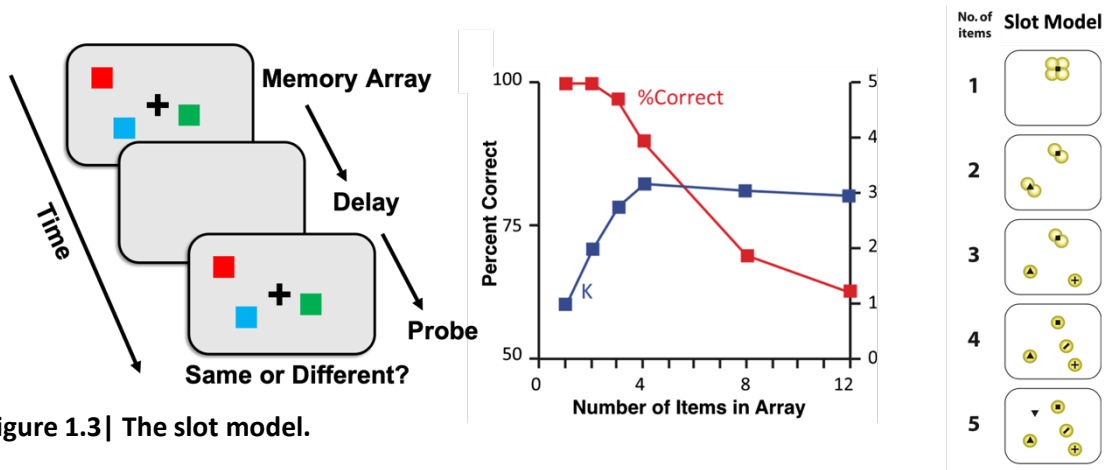


Figure 1.3| The slot model.

Luck and Vogel (1997) change-detection task, aimed at providing an estimation of the capacity of visual STM, consisted of varying the number of items to bear in mind. They observed an inverse relationship between the probability of correctly recalling an object (red) and the actual number of items to be remembered from the memory array (estimated capacity, k, in blue). Furthermore, in an instance where the items outnumber the set of slots available, not all items are stored. Thus, the slot model predicts that errors due to misreporting an item are comprised of a mixture of high *precision answers and random guesses.* Adapted from Eriksson, Vogel, Lansner, Bergström, & Nyberg, 2015 and Paul M. Bays & Husain, 2008b.

1.1.4. The Resource Model

On the other hand, many researchers have demonstrated the inadequacy of considering a hard limit on the number of items that can be stored, suggesting that WM might be better understood as a limited resource that can be distributed among retained items, with precision declining continuously as the number of items to be recalled increases (Wilken & Ma, 2004; for reviews see Fallon, Zokaei, & Husain, 2016; Ji Ma, Husain, & Bays, 2014) (**Figure 1.4**); contrary to the abrupt, steep decline (**Figure 1.3**) that would be expected when reaching the boundary of a set number of items (J. S. Luck & Vogel, 1997). Accordingly, in this model, precision is the fundamental metric of WM, not the number of objects to be remembered.

Therefore, the resource model predicts that memory resources are limited and finite: as memory load increases, the fidelity with which its visual features are stored decreases (Palmer, 1990; Wilken & Ma, 2004; Bays & Husain, 2008; Bays, Catalao, & Husain, 2009). Additionally, because the resource is limited and can be flexibly allocated, some items can be prioritised and therefore represented with greater fidelity, but this comes at the cost of other items in WM being recalled less precisely (Bays et al., 2009, 2011; Bays & Husain, 2008; Gorgoraptis, Catalao, Bays, & Husain, 2011). Also, by biasing memory resources to a specific item in memory, its representation can be recalled with the same fidelity as if there was a single item in WM (Pertzov, Manohar, & Husain, 2017).

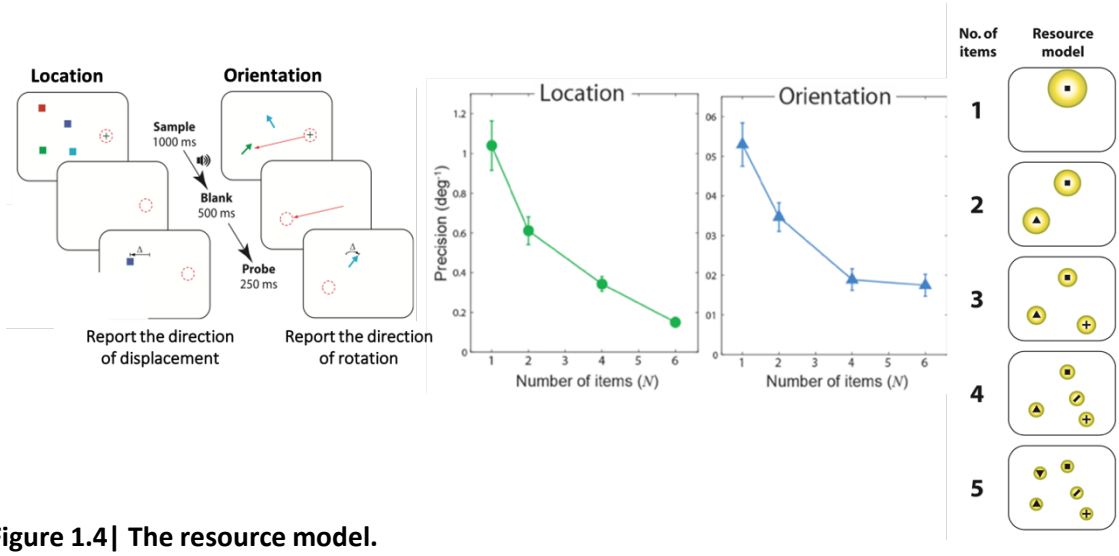


Figure 1.4| The resource model.

Bays and Husain (2008a) delayed-reproduction task tested subjects' ability to remember the location and orientation of multiple visual items using a continuous response space, as opposed to a discrete measure. They observed that, for both the location and orientation judgments, precision decreased with increasing numbers of items stored. Thus, the resource model predicts a trade-off between the number of items participants can remember and the representational precision of each memorised item, with the number of remembered items negatively correlating with the precision of recall. Adapted from Paul M. Bays & Husain, 2008a and Paul M. Bays & Husain, 2008b.

However, a topic of debate regarding the resource model is that when capacity is reached, the resource model (Paul M. Bays & Husain, 2008a) does not seem to be able to replicate the asymptote found in the slot model when participants are asked to remember memory arrays of 3 or 6 items (W Zhang & Luck, 2008), despite using a very similar method to measure WM precision.

1.1.5. Considerations on the introduced working memory models

The model proposed by Baddeley & Hitch (1974) has been highly influential, but difficult to refute because it is quite general and largely descriptive, lacks detailed predictive power, and fails to specify the underlying mechanisms at a computational level. Despite this multicomponent model being the leading theory of WM in the field,

there is still much disagreement regarding which neural areas are necessary and sufficient to carry out the tasks in each component, and how to best characterize WM capacity limits.

Later work on visual WM, however, can be considered more closely. As previously described, there are two main competing theories of WM capacity, the discrete-slot model (Luck & Vogel, 1997) and the flexible-resources model (Bays & Husain, 2008; Wilken & Ma, 2004). But no real consensus has yet been reached on capacity limits, or whether there is a single unified way to consider WM, particularly in terms of its functions and mechanisms (Baddeley, 2010; Cowan et al., 2005). Further, although these visual WM models emerged from different behavioural and neural findings, several 'hybrid' models have attempted to bridge the gaps between them (Alvarez & Cavanagh, 2004; Anderson, Vogel, & Awh, 2011; Weiwei Zhang & Luck, 2008).

In my opinion, the major differences between the slot and resource accounts can be captured in two key observations, both of which would seem to favour the latter. First, the finding that recall variability increases with increasing set size, without any discontinuity evident at any set size – even above the classical limit of 3 or 4 items – is not expected by any model with a deterministic limit such as the classical slot model (see Ji Ma, Husain, & Bays, 2014). Second, the original slot model lacks a stochastic mechanism, and therefore appears ill-suited to explain the significant deviations from the Normal distribution in WM errors (Bays, 2015).

Zhang & Luck's (2008) influential study proposed a revision of classical slot-based models by allowing multiple slots to be allotted to the same item. To my understanding,

this effectively mimics a resource model with the slots acting as a shared resource distributed between items, while at the same time maintaining a deterministic limit imposed by the number of slots. The adjustment reproduces the increase in variability with set size observed in experimental data, as well as some deviations from normality. However, it is important to note that every model that preserves a deterministic mechanism has thus far been unable to fully account for the magnitude of the breaches from normality observed in WM errors (Bays, 2015).

In summary, despite attempts to square slot and resource models, in my view they remain largely irreconcilable. In particular, because models which advance an understanding of WM as a limited resource are rooted in strongly established neural principles (Alexandre, Peter, & Richard, 2000; Carandini & Heeger, 2012), these are broadly opposed to a deterministic limit on the number of items stored. Ultimately, while recent slot-based models can produce randomly shifting limits to capture the innate stochasticity of neural activity, their theoretical underpinnings – concepts such as the spontaneous fade out of activity bumps – are not properly substantiated in neurophysiological observations (Bays, 2015).

1.2. *Precision methods as a way of measuring working memory capacity*

The manner in which WM is probed appears to be crucial to understand its underlying mechanisms, i.e., its capacity limitations and relationship with degradation and attention. Most previous studies have focused on quantifying the amount of

information that can be retained over the short-term, typically by resorting to binary reports (Luck & Vogel, 1997; Miller, 1956). More recently, a new approach has been to probe the fidelity with which an item is stored. By asking participants to reproduce the exact feature of a remembered item – for example, its location, colour or orientation – researchers have been able to investigate the resolution with which items are retained by using continuous, analogue report designs (Pertzov et al., 2012; Ma et al., 2014; Fallon et al., 2016).

Zhang and Luck (2008) presented participants with 1, 2, 3 or 6 items (squares defined by their location and colour) in each trial. They then asked them to indicate the colour of a random probed item on a continuous colour wheel. They fitted response data with a mixture of two components: the weighted sum of a Von Mises distribution (a circular normally distributed component of error centred on the true value of a probed item) and a uniform distribution (intended to capture guesses). The model predicts that participants' responses are either distributed around the target with a single fixed variability (the item has a slot, therefore is remembered) or random (the item does not have a slot, thus is not remembered). The model can be described by the sum of the gaussian variability in reporting the feature value belonging to the target and responding at random, as per the following equation (***1.1***):

$$P(\hat{\theta}) = (1-\gamma)\,\phi_\sigma(\hat{\theta}-\theta) + \gamma\frac{1}{2\pi} \qquad (1.1)$$

Where γ is the proportion of trials in which the subject responds at random, $(1-\gamma)$ is the probability of reporting the correct item, ϕ_σ denotes the Von Mises

distribution (the circular analogue of the Gaussian distribution with mean zero and standard deviation (SD) σ), $\hat{\theta}$ is the value reported by the participant and θ is the true value of the target.

The authors observed that the width of the fitted normally distributed component reached a plateau at around 3 items. In summary, the measure of precision used by Zhang and Luck (2008) was the SD of the Von Mises curve and they did not find a difference in this measure between the 3-item and 6-item conditions, in line with the slot model notion. Importantly, the authors only considered that an item could be remembered or forgotten.

Bays et al. (2009) were further able to explore the causes and consequences of how these representations are corrupted by deconstructing the pattern of recall error in three components, instead of two. These components are **precision**, which is the faithfulness with which an item location is reproduced; **misbinding**, referring to the probability of misreporting features of non-probed items (features belonging to other items in the memory array, for example reporting the colour of another item); and **random guessing**, which quantifies the proportion of times an item is completely forgotten. The model is depicted by the resulting equation (**1.2**):

$$P(\hat{\theta}) = (1 - \gamma - \beta)\,\phi_\sigma(\hat{\theta} - \theta) + \gamma\frac{1}{2\pi} + \beta\frac{1}{m}\sum_{i}^{m}\phi_\sigma(\hat{\theta} - \theta_{i^*}) \qquad (1.2)$$

Where misreporting non-target features (an index of errors due to misbinding) is in red and reflects the third component of recall error added to Zhang and Luck's model

(2008). Thus, β reflects the probability of reporting a non-target item, and $\{\theta_{1*}, \theta_{2*}, \theta_{3*}, \ldots, \theta_{m*}\}$ are the feature values of the m non-target items.

Notably, Bays and colleagues (2009) addressed the discrepancies of the two previously described findings (Paul M. Bays & Husain, 2008a; Weiwei Zhang & Luck, 2008) by replicating the Zhang and Luck (2008) study and finding that there is indeed an inverse relationship between the number of items held in STM and the precision with which they are retained, regardless of set size. In other words, they found no evidence for an item limit. By accounting for memory errors arising from misbinding different features belonging to the same item (the colour of an item with its location, for example), they were also able to find the predicted negative relationship between number of items and precision of its representation (**Figure 1.5**), irrespective of item limit (Paul M. Bays et al., 2009).

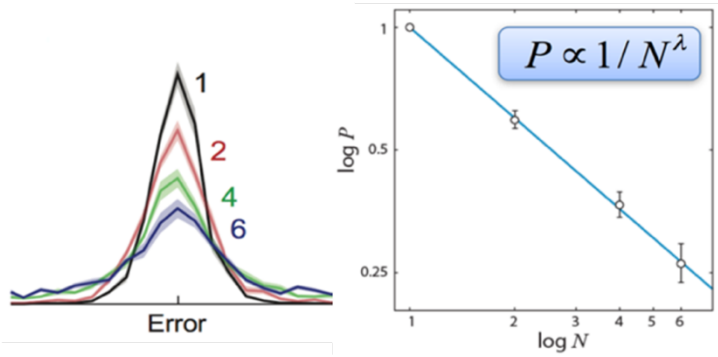


Figure 1.5| Precision is the fundamental metric of WM capacity, not number of items.

In their replication of Zhang and Luck's study (2008), Bays et. al. (2009) showed that distribution of responses relative to the colour delayed-estimation task depend on the number of items in the memory display, with increasing set sizes leading to greater recall error. But, when accounting for misbinding errors, as opposed to solely looking at whether an item is remembered or not, they reached the same no item limit finding from Bays and Husain (2008a), with precision (1/SD) beginning to deteriorate as soon as sample size exceeds one item. Note that SD is the measure of recall variability of error, which increases gradually with set size and saturates at items 3 and 4, with no capacity limit and a negative correlation between precision and N. In the slot model, this function would be flat up to set size 4. Figure adapted from Ji Ma et al., 2014.

1.3. *A dynamic model of WM invokes rapid neural plasticity and pattern completion to produce cued recall*

The susceptibility of the object-location association has traditionally been considered in the framework of hippocampal function theory in LTM (Rolls, 2013), with the hippocampal subfield CA3 acting as an association network able to form bindings between the object and its spatial representation (Rolls, 2013). As a result, the recovery of a complete stored representation is achieved through a partial, noisy or degraded retrieval cue triggering the mechanism of pattern completion (Bakker, Kirwan, Miller, & Stark, 2008; O'Reilly & McClelland, 1994).

Recently, the hippocampus has also been implicated in WM performance (Pertzov, Miller, et al., 2013). Additionally, a novel computational neural model of WM that

provides a mechanism for feature binding (Manohar, Zozaei, J., Vogels, & Husain, 2017), predicts that, similarly to LTM, features belonging to an object can be re-activated at retrieval through the process of pattern completion. It proposes that rapid plasticity in flexibly-coding neurons allows for features to be bound together into objects with recent items being preserved in synaptic traces.

The model establishes a dual functional architecture in which neurons hold information in both persistent activity and in changes in synaptic weights — in active (an item in the focus of attention) and silent (corresponding to the unattended item) representations, respectively. It is composed of two layers, corresponding to populations of neurons in the brain. *Freely-conjunctive units* (**Figure 1.6A in blue**), putatively in the prefrontal cortex, are able to rapidly form associations between the features of an object held in *feature-selective units* (**Figure 1.6A in green**), located in posterior cortical neurons responsible for encoding sensory-motor features, using a Hebbian associative rule (**Figure 1.6A**) (Manohar et al., 2017).

The model envisages that the coactivation of neurons (**Figure 1.6A in pink**) results in strengthening of synapses between them in both directions, producing a stable attractor state across brain areas. The proposed neural events during attention, encoding and retrieval are depicted in **Figure 1.6B**. First, a sensory input activates the corresponding feature neurons (**Figure 1.6B-1.**). These subsequently excite conjunctive neurons which, via mutual inhibition, compete to encode the combination of active features (**Figure 1.6B-2.**). The winning conjunction unit establishes a stable pattern that drives sustained activity *even without input* (**Figure 1.6B-3.**). If new input is provided,

firing activity is disrupted as a new conjunction forms. However the synaptic weighting is preserved and thereby continues to store the memory trace (**Figure 1.6B-4.**).

Therefore, even though a stable representation can fall silent when new information is encoded into the network (**Figure 1.6B-4.**), at recall, presenting a single probe feature can re-activate the original conjunction unit (**Figure 1.6B-5.**), which then restores the rest of the features units from its joined representation, resulting in recall (**Figure 1.6B-6.**). This is achieved because the concurrent activation between the conjunction unit and the features units drives rapid Hebbian plasticity, allowing for the feature pattern to be encoded into synaptic connections linking the two units.

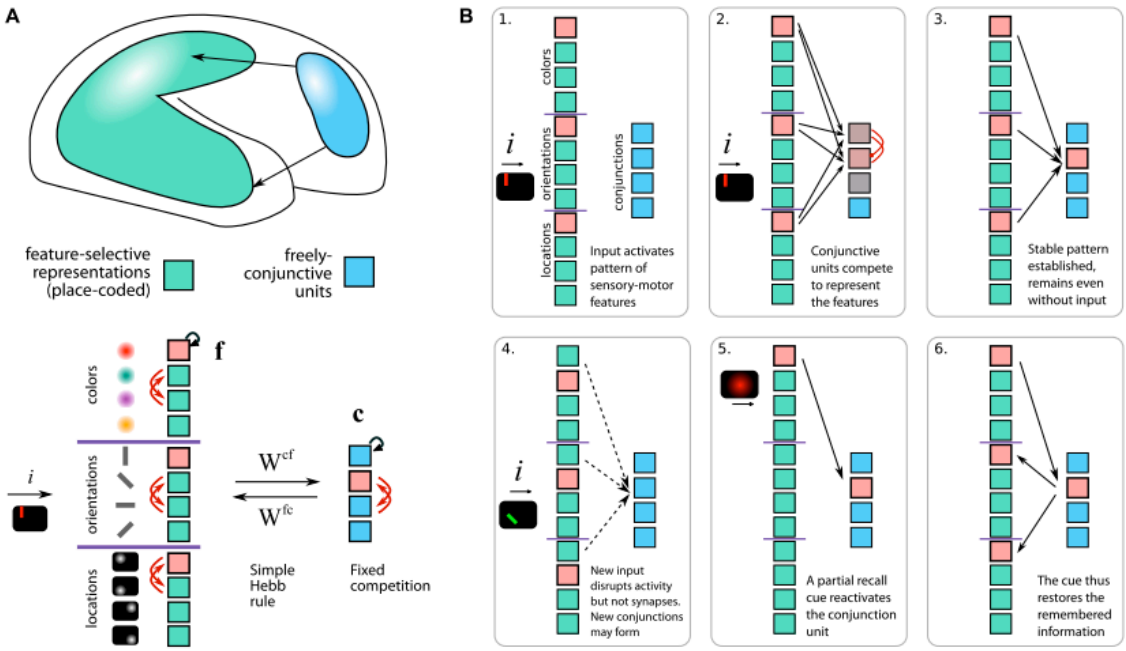


Figure 1.6| Dynamic WM model of freely conjunctive neurons.

A) The model's architecture encompasses two populations of neurons: posterior, feature-selective neurons (green) that encode sensory-motor features and prefrontal neurons (blue) that are freely-conjunctive units. The connections between the feature cells and conjunctive cells (c) change their synaptic weights (W) based on the patterns of activation (f) using a Hebbian plasticity. B) Depiction of the neuronal events during attention, encoding and retrieval after the presentation of a sensory input (i). Reprint from Manohar et al., 2017.

1.4. *Evidence accumulation methods as a way of measuring memory quality: the decision process of recall*

To date, the scientific effort to describe the limitations of WM has primarily focused on studying the pattern of errors made by participants, with greater memory load leading to increased variability around the target response because memory representations are noisy (Paul M. Bays & Husain, 2008a; Palmer, 1990; Wilken & Ma, 2004). But, RT, especially arising from repeated simple decisions, can also be used to infer latent cognitive processes underlying the decision process of memory retrieval, as well as a quantitative mechanistic explanation of the cognitive components contributing to observed effects (Christopher Donkin & Brown, 2018; Ratcliff, 1978).

Of particular interest to the WM field, Pearson et al. (2014) investigated whether the precision of memory report could vary as a function of RT distributions in participants' recall performance. They argued that retrieval from WM could be conceptualized as a decision made on the quality of internal evidence. The authors revealed that retrieval time increased with memory load. They also showed that the mean rate of rise of evidence accumulation for the decision process was strongly correlated with memory precision, i.e., the degree of response variability around the target determined the quality of evidence provided to the decision mechanism. Additionally, they found faster evidence accumulation for cued items — as this increased memory precision at the expense of others — as opposed to a reduced rate of accumulation for uncued items.

To explore the theory that memory guides choice by way of a decision mechanism acting on internal evidence, Pearson and colleagues (2014) used a simple "rise to

threshold" decision model, the Linear Approach to Threshold with Ergodic Rate (LATER) (Carpenter & Williams, 1995; Reddi, Asrress, & Carpenter, 2003). This is one of the many evidence-accumulation models of choice that have been described and extensively applied as a measurement tool in a range of cognitive tasks (Ratcliff, Smith, Brown, & McKoon, 2016 for a review). These models that have been introduced in the past fifty years allow for a joint analysis of RT and accuracy, and can differ in (1) how information is accumulated, either at discrete points in time (LaBerge, 1962; S W Link & Heath, 1975; Steve W Link, 1975; Smith & Van Zandt, 2000; Smith & Vickers, 1988) or continuously (S. D. Brown & Heathcote, 2008; Ratcliff, 1978; Stone, 1960; Usher & McClelland, 2001); and (2) how the decision-making process works based on different theoretical frameworks (S. D. Brown, Marley, Donkin, & Heathcote, 2008; Busemeyer & Townsend, 1993; Ratcliff, 1978; Ratcliff & Rouder, 1998; Usher & McClelland, 2001; Verdonck & Tuerlinckx, 2014) or just a simplification based on specific theoretical questions (S. D. Brown & Heathcote, 2008; S. Brown & Heathcote, 2005; Grasman, Wagenmakers, & van der Maas, 2009; E.-J. Wagenmakers, Van Der Maas, & Grasman, 2007; E. J. Wagenmakers, van der Maas, Dolan, & Grasman, 2008).

Despite their differences (Ratcliff & Smith, 2004 for an assessment of model architectures), all these models are based on the notion that a decision emerges from the process of gathering evidence over time at a certain drift rate in favour of each alternative response option until sufficient amount of evidence is accumulated for one option over another and a decision threshold is reached. They are able to decompose the choice RT distributions into latent parameters (which denote processes underlying how the decision is made), of which drift rate (rate at which information is processed),

threshold (response caution) and non-decision time (perceptual processing of stimulus) are the ones common across all variants of evidence accumulation models.

The two most prominent variants of cognitive models capturing the process of evidence accumulation are the Diffusion Decision Model (DDM) (Ratcliff, 1978; Ratcliff & McKoon, 2008) and the Linear Ballistic Accumulator Model (LBA) (S. D. Brown & Heathcote, 2008; S. Brown & Heathcote, 2005). The DDM, also known as the *Radcliffe diffusion model* in recognition to the large contribution of Radcliffe and colleagues, is the most broadly used decision-making model and posits that evidence accumulation is stochastic, i.e., to explain the variability in RT and accuracy, it assumes that evidence accumulated is noisy and varies from moment to moment (**Figure 1.7**). Thus, it accounts for both trial-to-trial variability in start point and drift rate, allowing the diffusion process to produce, within a single block of trials, both slower and faster errors for hard and easy conditions. To better report empirical data, it also accounts for variability in non-decision time such that, depending on the rate with which evidence is accumulated, the fastest responses do not take the same amount of time (**Figure 1.7**).

The LBA is an alternative evidence accumulation theory proposed by Brown and Heathcote which simplifies the characterisation of noise by assuming that within-trial evidence accumulation is ballistic, i.e., evidence from the environment is assumed to be noiseless. In the LBA, accumulation is theorised to be free of leakage (decay), excitation (tendency to grow in activation at a rate proportional to existing activation) and lateral inhibition (evidence in one accumulator inhibits the rate of evidence increase in the other accumulator). Thus, the model simply assumes deterministic linear evidence accumulation with two sources of trial-to-trial variability (in drift rate and start points),

which greatly facilitates computation (**Figure 1.7**) (Christopher Donkin & Brown, 2018, Donkin, Brown, Heathcote, & Wagenmakers, 2011 for a direct comparision between the two models).

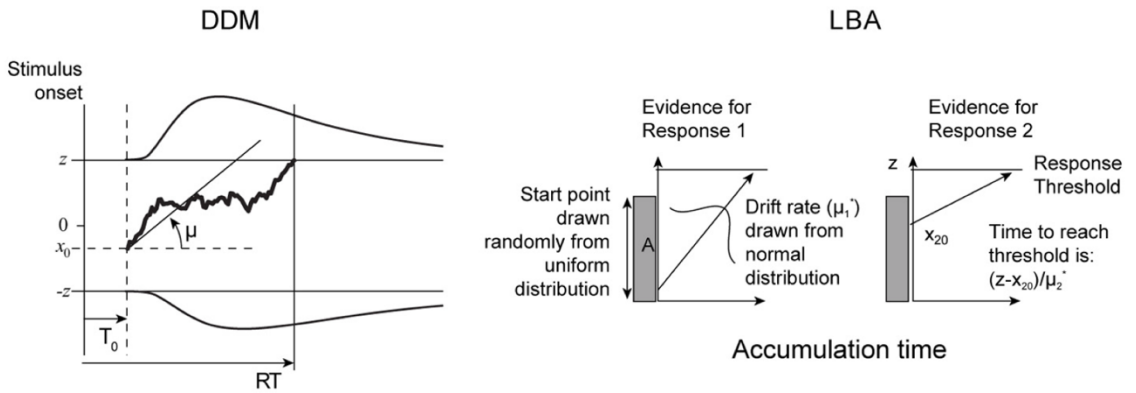


Figure 1.7| Illustration of the Decision process for the DDM versus the LBA.

The DDM and the LBA make different assumptions. The DDM considers that evidence is sampled from information stream arising from a continuous moment to moment match between a test stimulus and memory (jagged line). The accumulator starts gathering evidence with a specific drift rate (μ) at the start point (x_0) after a non-decision time (T_0) and a choice is made when a threshold is reached (± z). The LBA assigns a separate evidence accumulator in favour of each response. Each accumulator begins its activity at a start point randomly drawn from an uniform distribution within an interval [0,A] and gathers evidence with a specific drift rate (μ_1, μ_2) in a linear ballistic way. A decision is made when the response threshold is reached (z). Reprint from Goldfarb, Leonard, Simen, Caicedo-Núñez, & Holmes, 2014.

For both DDMs and LBA models, thresholds fundamentally represent caution parameters (Chris Donkin et al., 2011) which anchor the speed-accuracy trade-off: high caution denotes low speed and high accuracy, while lower caution is associated with higher response speeds but lower accuracy (Bogacz, Brown, Moehlis, Holmes, & Cohen, 2006; S. D. Brown & Heathcote, 2008).

Even though DDMs and LBA models tend to generate much of the same key results (S. D. Brown & Heathcote, 2008; Chris Donkin et al., 2011) an essential distinction between them concerns their treatment of variability, which in turn spurs differing

predictions regarding cautious behaviour in complex tasks. Variability is introduced in DDMs by adding Gaussian noise in the evidence accumulation process during each trial. Later iterations of DDMs can include further noise parameters capturing inter-trial variability in the drift rate, starting point, and non-decision time, which is especially useful to account for when errors are either faster or slower than correct responses. Hence, DDMs predict that participants are on average less cautious on the most difficult tasks, particularly when the optimal strategy to deal with complexity involves random guessing. On the other hand, because there is no additive noise in LBA models, and this method only deals with trial-to-trial variability in the starting point and drift rate, an LBA fit infers that participants are on average more cautious in tasks that display a greater relative difficulty (Goldfarb, Leonard, Simen, Caicedo-Núñez, & Holmes, 2014).

1.4.1. Hierarchical Bayesian Drift Diffusion Model

Contrary to frequentist methods which require plenty of RT measurements to impart meaningful estimates for each parameter, Hierarchical Bayesian parameter estimation methods enhance statistical power, granting both estimation of individual subject parameters and the group distribution they are drawn from, as well as providing measures of uncertainty for these parameters in the posterior distribution (Farrell & Ludwig, 2008; Heathcote et al., 2019; Shiffrin, Lee, Kim, & Wagenmakers, 2008; Wiecki, Sofer, & Frank, 2013). Thus, requiring less data per subject than non-hierarchical methods, hierarchical estimates are less variable, more precise and accurate.

Hence, in **Chapter 2** and **3** I adopt HDDM to provide a quantitative framework to the understanding of latent cognitive processes of retrieval manipulations on the

decision-making process of recall. This was my model of choice due to (1) the purported objective in these Chapters to examine the mechanisms underlying memory retrieval and how these are manifested in the speed-accuracy trade-off — which necessarily entail the study of diffusive noise dynamics within trials, which are sacrificed for simplicity in the LBA model; and (2) parameter recovery studies demonstrate that HDDM tend to outperform non-hierarchical and non-Bayesian models (Wiecki et al., 2013).

In summary, I will start by estimating the drift rate and threshold parameters for each participant separately and point estimates will be obtained through a process where the data generated by the model is continuously informed by the behavioural evidence using Bayes' Theorem. Information captured by the population-level parameters will be used to improve parameter estimation at the participant-level (during the course of sampling, the hierarchical model tunes the population-level parameter with each participant's data, which is able to then provide an incrementally more informative prior for the participant-level parameter). A hypothesis-driven approach will be preferred to determine which model (each with its unique parameterization of an RT model for every condition) gives a more parsimonious account of the behavioural data. To select the best fitting model, two standard model selection approaches will be implemented: the Deviance Information Criterion (DIC) and the Bayesian Predictive Information Criterion (BPIC) (Spiegelhalter, Best, Carlin, & Van Der Linde, 2002).

1.5. *Effects of processing demands in working memory performance*

Inferences on the limit of memory performance have also focused on manipulations of maintenance delays and item cueing. In fact, because WM is not a homogeneous store, the mechanisms underlying information loss over the short-term have also shown to influence memory precision.

Some researchers argue that forgetfulness results from information decaying over time, with longer periods of retention leading to greater decline of the fidelity of mental representation (A. Baddeley, Logie, Bressi, Sala, & Spinnler, 1986; Barrouillet, Bernardin, & Camos, 2004; Barrouillet, De Paepe, & Langerock, 2012; J. Brown, 1958; Towse, Hitch, & Hutton, 2000). Other investigators believe that forgetting is due to the interference between items in WM and the processing of distractors (Berman, Jonides, & Lewis, 2009; Lewandowsky, Geiger, Morrell, & Oberauer, 2010; Lewandowsky, Geiger, & Oberauer, 2008; Lewandowsky & Oberauer, 2009; Oberauer & Lewandowsky, 2008; Oberauer, Lewandowsky, Farrell, Jarrold, & Greaves, 2012; Saito & Miyake, 2004). Thus, interference-based models assume that processing involves the encoding of distractor representations in WM and time is used to process the removal of distractors — irrelevant information is introduced into memory which interposes with memoranda (Oberauer & Kliegl, 2006; Oberauer et al., 2012; Saito & Miyake, 2004). However, although interference predicts negative effects on memory performance, retro-cues, which are a form of positive interference, may provide a different way of looking into WM representations due to the positive effects they exert on later recall (Griffin & Nobre, 2003; Kuo, Stokes, & Nobre, 2012).

Some investigators have even provided evidence for the influence of both factors in healthy people (Pertzov et al., 2017). By examining how the interplay between items in memory adds to rapid forgetting over different durations, and how forgetfulness is affected by diverting attention towards a single representation in memory, the authors noted an interaction between time and the number of items kept in memory — with accelerated degradation of representations due to competition between stored memoranda. They have shown that misbinding can be affected by attentional manipulations and that the nature of these errors can be explored by manipulations of attention during the maintenance period. But it is still unclear whether all types of attentional manipulations during WM maintenance have the same effects on recall and misbinding.

Other researchers are sceptical of the proposition that sustained focal attention increases the ability to maintain information in a privilege state (Rerko, Souza, & Oberauer, 2014), associating the latter with other processes responsible for selectively improving accessibility of one object within a set, in response to a retro-cue. Therefore, the *retro-cue paradigm* has been used to investigate the mechanisms of attentional selection in WM (Souza & Oberauer, 2016) by presenting a spatial cue (named retro-cue because of its retroactive effect) following the retention interval, indicating which item will be tested. Interestingly, presenting participants with a cue, after encoding information, guides attention to a specific representation in WM, enhancing its relevance and improving recall, often over and above that seen for remembering an item for a similar period of time. Thus, preceding research has shown that retro-cues are proficient in producing strong benefits for the cued item on later recollection (Griffin & Nobre,

2003; Kuo et al., 2012), triggering a withdrawal of memory resources from the uncued item to attain a sustained maintenance of the cued memoranda (Pertzov, Bays, Joseph, & Husain, 2013). These results align with the notion of voluntary control over maintenance (Marshall & Bays, 2013).

Different – but not mutually exclusive – hypotheses have been presented to explain the retro-cue effect. One of these propositions is based in the idea that the focus of attention may be responsible for strengthening the binding between the content (e.g., colour) and the context (e.g., location) of a cued representation by improving its accessibility at retrieval (Loaiza & McCabe, 2012; Rerko & Oberauer, 2013; Rerko et al., 2014). Rather than reflecting changes in the cued representation, the benefits from retro cueing might instead emerge from the removal of irrelevant (non-cued) information: an increase in WM capacity an improved representation as a result of reduced competition and interference (Rerko et al., 2014; van Moorselaar, Olivers, Theeuwes, Lamme, & Sligte, 2015; Williams, Hong, Kang, Carlisle, & Woodman, 2013). Another suggested hypothesis, the retrieval head start hypothesis, implies that the retro-cue offers the opportunity to retrieve the representation of the cued object before using this information to decide. By assuming that retrieval is the gradual accumulation of information about the target's features in the focus of attention, it follows that delaying the memory test impels the accumulation of more information over time, predicting an increase in performance (Purcell et al., 2010; Souza & Oberauer, 2016).

The WM literature has consistently asserted the relationship between memory performance and constraints of encoding and storage. However, the retro-cue effect demonstrates that much of the same limitations are similarly present at the **retrieval**

stage. It additionally shows that information presented following encoding is capable of influencing the probability of retrieval, i.e., the actual event of a probe can, in and of itself, represent a retro-cue. Thus, in **Chapter 2** and **Chapter 3**, I explore how manipulating retrieval modulates performance; more specifically, I investigate whether, rather than acting as a form of interference and impairing memory, more information at retrieval can improve recall by serving as a more faithful retro-cue.

1.6. *Retrieval from working memory*

Results from episodic memory studies suggest that during encoding, cortical processing provides inputs to the MTL, specifically hippocampal circuitry, which binds the inputs into an integrated memory representation (Eldridge, Engel, Zeineh, Bookheimer, & Knowlton, 2005; Tulving, 1983). At recall, this conjunction of features is then retrieved through a process of pattern completion, i.e., a partial retrieval cue corresponding to part of the encoded information, which is bound to the rest of the memory representation, activates the remaining inputs (O'Reilly & McClelland, 1994; Rolls, 2013). The hippocampus has also been implicated in this retrieval mechanism (Eldridge et al., 2005; Eldridge, Knowlton, Furmanski, Bookheimer, & Engel, 2000; Nakazawa et al., 2002; O'Reilly & McClelland, 1994). For example, Eldridge's neuroimaging study provided evidence for a role of the MTL in episodic retrieval in healthy people, with successful recollection associated with activation of the hippocampus (Eldridge et al., 2000).

Thus, according to this view of integrated representations of memoranda, a feature presented at recall is a possible route to the memory conjunction because it triggers

pattern completion to access the other features that it was associated with. Even partial features holding a limited amount of information might in this way gain access to the encoded memory. Thus, hippocampal episodic memory studies suggest that the more information presented at retrieval, the better recall will be via pattern completion mechanisms (Rolls, 2013). However, findings from visual attention and WM studies suggest that the more information presented at retrieval the worse performance will be because more features have to be bound together and capacity is limited (Paul M. Bays & Husain, 2008a; Wheeler & Treisman, 2002).

In contrast with LTM retrieval, which has been studied in considerable detail, most research in WM has focused either on encoding (Ceraso, 1985; Wheeler & Treisman, 2002; W Zhang & Luck, 2008) or the effects of maintenance delay (Fallon, Mattiesing, Dolfen, Manohar, & Husain, 2018; Souza & Oberauer, 2014). But retrieval from WM has recently been shown to also affect memory report significantly. For example, Souza et al. (2016) found that delaying responses after the memory probe for at least 0.5s improves recall performance. This suggests that allocating time for retrieval before decision-making allows for greater accumulation of evidence about the memoranda, resulting in better recall. Yet it is left unclear how this decision-making process is modulated by the nature of the information carried by the memory probe.

Additionally, consistent with the idea that retrieval might not simply be a read-out of storage processes, the method by which memory is probed has also been shown to affect recollection, as redundant information in the probe (Tabi, Husain, & Manohar, 2019) or the presence of distractors at recall impairs memory performance (Makovski, Watson, Koutstaal, & Jiang, 2010; Udale, Farrell, & Kent, 2017). However, little is known

about how the quality of recall depends upon the amount and type of information carried by retrieval cues; or how the features carried by a foil (distractor) presented at probe affects the quality of associative retrieval. These questions will be investigated in Chapter 2 and Chapter 3.

1.7. *Working memory filtering*

One way to probe WM is to manipulate attentional demands at different times during the processing of information. This has usually been accomplished by investigating how well people can ignore or filter out distracting information at encoding (Jost, Bryck, Vogel, & Mayr, 2011; Vogel, McCollough, & Machizawa, 2005).

However, it is also possible to examine how effectively irrelevant stimuli presented during maintenance can be ignored or filtered out to determine how existing contents of STM can be shielded from being overwritten by the irrelevant information (Sean J. Fallon et al., 2018; Souza S., Rerko, & Oberauer, 2014). In addition to investigating the impact of distracting information, some studies have concurrently examined peoples' ability to jettison existing contents of STM and replace them with new contents during maintenance. Fallon and colleagues (2018) have shown that young adults' recall precision is considerably better for such updating with new information compared to ignoring distractors. Yet, there has been no systematic examination of how recall performance is affected when information held by a distractor foil presented at retrieval needs to be filtered out.

Because feature binding has been considered to be one important aspect of attention, in addition to the use of these strategies to study the interaction of attention and WM, some authors have also focused on *binding errors* in WM recall (Alan D. Baddeley, Allen, & Hitch, 2011; Johnson, Hollingworth, & Luck, 2008; Wheeler & Treisman, 2002). Some studies have revealed that attentional deployment during maintenance helps keep features accurately bound in WM. For example, Pertzov et al. (2017) have shown that misbinding rates increase with retention durations, especially with high memory loads. Fallon and colleagues' work (2018) revealed that presenting distractors during maintenance intervals can also lead to an increase in misreporting features of stored items, specifically to features that had to be ignored. Finally, consistent with misbinding errors resulting from high attentional requirements, experimental designs that employ taxing demands during the retention interval have displayed increased errors due to misbinding (Fougnie & Marois, 2009; Zokaei, Heider, & Husain, 2014). However, none of these studies implemented designs that systematically examine at which stage of memory feature binding of stimuli is affected by attentional demands.

Most previous studies on the effects of filtering demands have assumed that an item is either remembered or forgotten, measuring WM capacity using change-detection paradigms or the amplitude of contralateral delay activity (CDA), an electrophysiological marker associated to WM capacity (Cowan & Morey, 2006; Rhodes & Cowan, 2018; Robison, Miller, & Unsworth, 2018; Vogel & Machizawa, 2004; Vogel et al., 2005). Conversely, there has been little examination on how recall performance is modulated by attentional demands prompted by the retrieval cue. Theeuwes et al. (2009) showed that when retrieving information from WM, visual attention is allocated to the spatial location containing the feature to be recalled, even if unnecessary for the task at hand.

This suggests that manipulations of visual attention at retrieval can facilitate access to information in WM (Theeuwes et al., 2009; Theeuwes, Kramer, & Irwin, 2011).

Yet, it is left unclear how the nature of retrieval cues can modulate this effect. Besides, none of these studies have examined the relationships between attentional demands at encoding, maintenance and retrieval simultaneously. Therefore, in **Chapter 4**, I delve into the relationship between processing demands at all stages of WM to establish whether filtering is a monolithic entity or rather a dissociable cognitive process.

1.8. *Working memory dysfunction in brain disease*

Deficits in WM are a significant problem in patients with neurodegeneration, such as Alzheimer's disease (AD) and PD (Verbaan et al., 2007; Zokaei et al., 2020). Sensitive delayed-reproduction tasks able to measure memory precision have been instrumental in providing insight to the understanding of the mechanisms underlying memory deficit in these disorders — by enabling the dissociation of the type of errors contributing to the memory impairment. These measures have imparted evidence that, while patients with MTL dysfunction are impaired in their ability to associate object-location conjunctions over short delays, memory impairment in patients with PD has been associated with increased errors due to guessing (Zokaei & Husain, 2019b). In this thesis, I aim to extend this work by examining patients with MTL damage and with PD.

1.8.1. Working memory binding deficits associated with Medial Temporal Lobe damage

It has recently been proposed that the MTL is not just relevant in episodic LTM (Aggleton & Brown, 2006; H. Eichenbaum, Yonelinas, & Ranganath, 2007) but is also necessary for STM (Hannula & Ranganath, 2008; Olson, Page, Moore, Chatterjee, & Verfaellie, 2006; Ranganath & Blumenfeld, 2005; Squire, Stark, & Clark, 2004). Critically, it has been hypothesised that the MTL, specifically the hippocampus, is essential in binding information together, regardless of memory duration (Liang et al., 2016; Libby, Hannula, & Ranganath, 2014; Olson, Moore, Stark, & Chatterjee, 2006; Olson, Page, et al., 2006; Pertzov, Miller, et al., 2013; Yonelinas, 2013). For example, Koen and colleagues investigated the role of the hippocampus in forming and supporting high resolution complex bindings by testing patients with MTL damage resulting from viral encephalitis on a STM task where participants had to remember object-location and object-colour associations (Koen, Borders, Petzold, & Yonelinas, 2017). They found that they were impaired compared to controls, particularly in conditions with high resolution bindings. However, the paradigm implemented by this group used the frequency with which memoranda was correctly remembered as an index of memory function, thus failing to provide details on the nature of the memory deficit.

Rather than just testing the frequency of correct responses, a lesion study by Pertzov et al. (2013) implemented a sensitive delayed reproduction task in order to detect subtle variations in STM performance. They found that patients who had focal MTL (hippocampal) atrophy due to autoimmune LE had increased errors due to misbinding compared to controls, i.e., their ability to correctly associate different

features belonging to the same object (for example, identity and location) was hindered. However, although this is a rare condition, the sample size was relatively small (N= 7).

1.8.1.1. Autoimmune Limbic Encephalitis

Therefore, to further investigate the role of the MTL in memory, specifically the nature of memory deficit resulting from hippocampal damage, in **Chapter 5** I replicate Pertzov et al. (2013) study by testing twenty-eight focal hippocampal atrophy due to LE patients as they serve as a good model of human hippocampal lesions (Fox, 1996; Khan, Jeffree, Good, Macleod, & Al-Sarraj, 2009; Pertzov, Miller, et al., 2013; Vincent, Bien, Irani, & Waters, 2011).

LE in patients with antibodies (Abs) to the voltage-gated potassium channels complex (VGKC), are mainly directed to the VGKC-complex associated proteins LGI1 (leucenic-rich, glioma-inactivated 1) or CASPR2 (contactin-associated protein-like 2) and can display inflammatory changes of the MTL on acute neuroimaging with subsequent focal atrophy, **Figure 1.8** (Ances et al., 2005; Bettcher et al., 2014; Kotsenas et al., 2014; Reid, Foley, & Willison, 2009; Vincent et al., 2011), and focal, acute hippocampal pathology (Argyropoulos et al., 2019; Chan, Henley, Rossor, & Warrington, 2007; Finke et al., 2017; Malter et al., 2014).

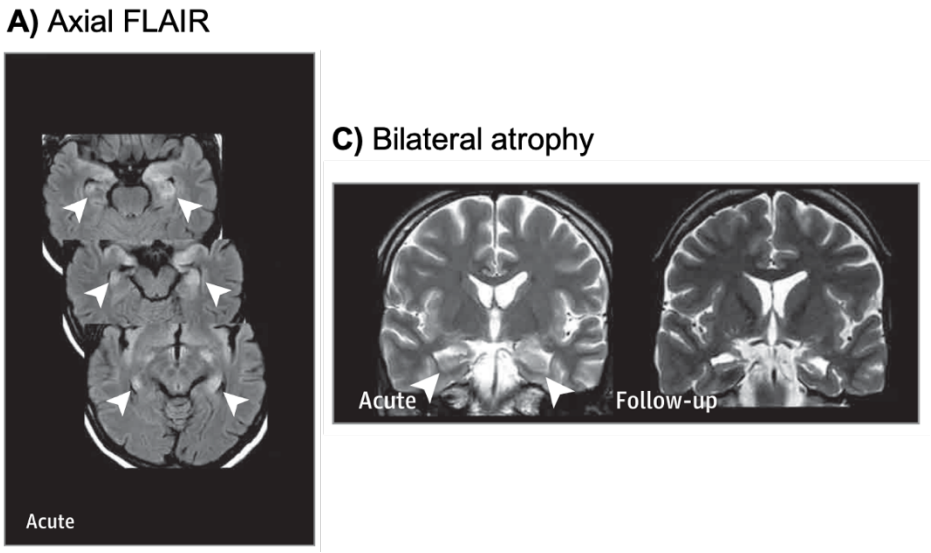


Figure 1.8| Acute MTL signal change and subsequent atrophy in representative MRIs of anti-LGI1 encephalitis patients.

A) Axial fluid-attenuated inversion recovery (FLAIR) imaging during the acute stage (on average 17 days after onset) displays signal hypertensities in both hippocampi and amygdalae. B) T2-weighted imaging reveals hippocampal edema as well as enlargement during the acute stage progressing into bilateral hippocampal atrophy (follow-up approximately 23.3 month after onset). Adapted from (Finke et al., 2017).

The pathogenic mechanism of this inflammatory brain disease associated with antibodies against ion channels, as illustrated in Figure 1.9, has been hypothesised to surge from a potential trigger that can be either a virus (Figure 1.9 A) or a tumour (Figure 1.9 B) that initiates an antibody response by releasing antigens. These antigens are loaded into dendritic cells and transported to regional lymph nodes where they are exposed to CD4+T cells and naïve B cells. Following this exposure, CD4+T cells and naïve B cells become antigen-conditioned and either differentiate into plasma cells or maturate and become memory B cells (Figure 1.9 C). When entering the brain, these memory B cells are restimulated and differentiate into antibody-producing plasma cells. These antibodies are thought to lead to neural dysfunction through different mechanisms; in these patients, it is hypothesised to occur by disrupting the LGI1 protein in the neuronal

synapse when binding to LGI1, altering neuronal excitability, i.e., because of the anti-LGI1 antibody, the LGI1 is no longer able to bind to the presynaptic ADAM23 and ADAM22 (which is a structure responsible for regulating the AMPAR receptor and the VGKC channel currents), thus interfering with synaptic transmission (**Figure 1.9 D**) (Dalmau & Graus, 2018; van Sonderen, Petit-Pedrol, Dalmau, & Titulaer, 2017).

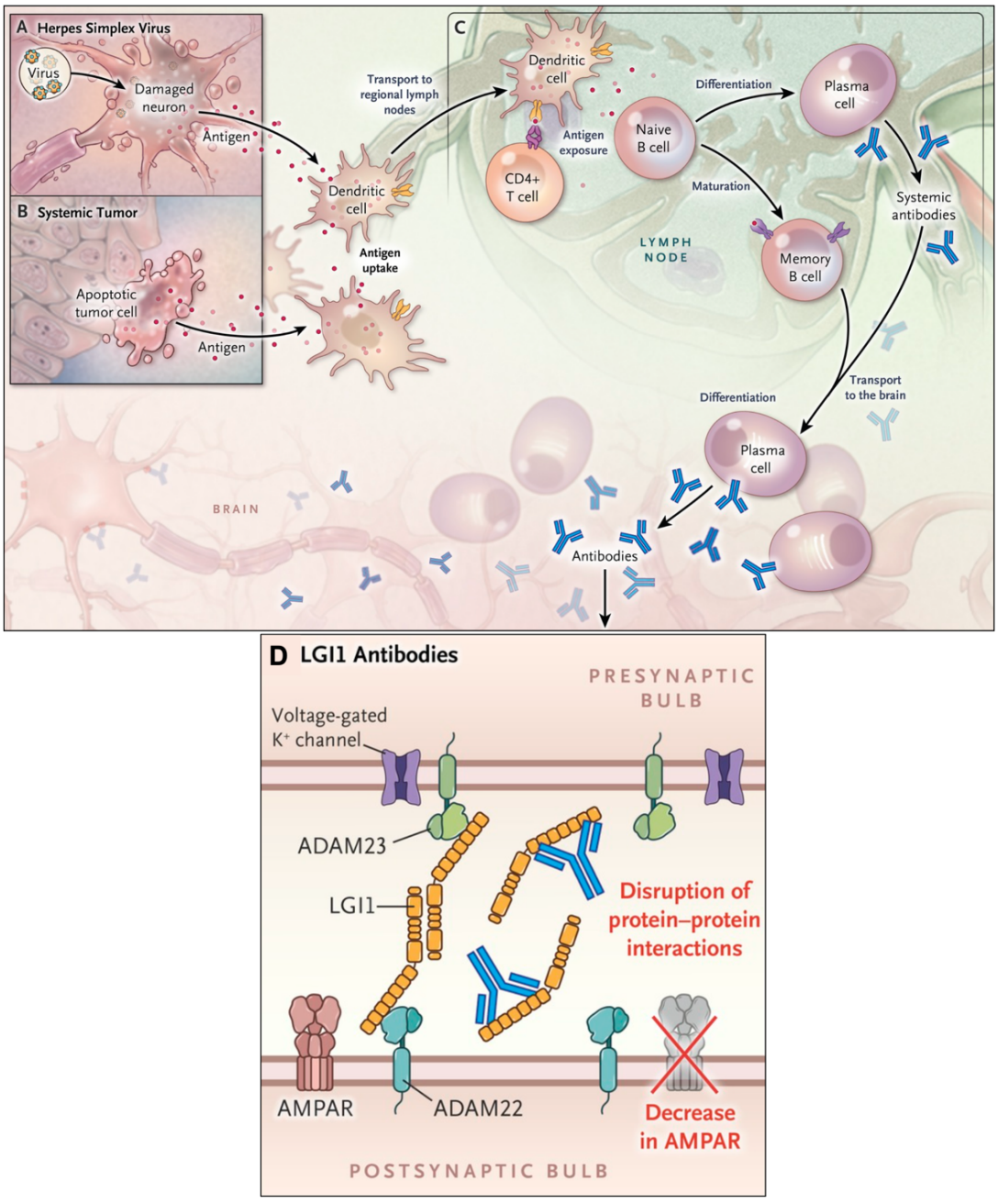


Figure 1.9| Schematic illustration of the postulated mechanism of Limbic Encephalitis with antibodies to the voltage-gated potassium channels complex directed to LG1. Reprint from (Dalmau & Graus, 2018).

1.8.2. Working memory in Parkinson's disease

PD is a chronic and progressive brain disorder of which a key neuropathological characteristic is the significant reduction of dopamine-producing neurons in the substantia nigra nuclei of the midbrain (Marsden, 1982) (**Figure 1.10**). The disease causes motor symptoms such as tremor, muscle rigidity, bradykinesia (slowness and difficulty to perform repeated movements), or postural unbalance (Postuma et al., 2015), but also cognitive impairment (Gratwicke, Jahanshahi, & Foltynie, 2015; Kehagia, Barker, & Robbins, 2010).

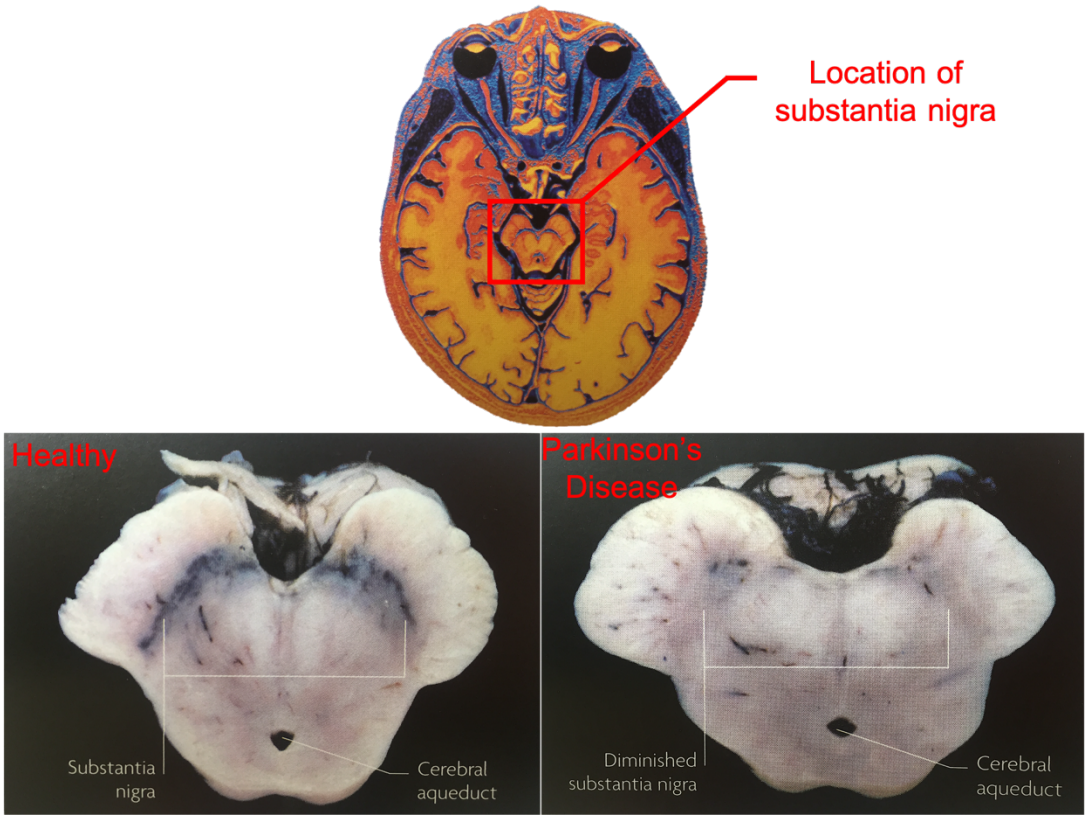


Figure 1.10| Changes in the substantia nigra nuclei of the midbrain in PD compared to controls.

The substantia nigra is the part of the nuclei known as the basal ganglia, in the midbrain (see colour-enhanced MRI scan of a horizontal section of the brain on top). In a healthy brain (see section of brain tissue in bottom left), the dark pigmented areas of the substantia nigra are clearly visible, as opposed to a PD patient where the pigmented neurons are significantly reduced (see section of brain tissue in bottom right). Reprint from Rita Carter, 2009.

WM has been found to be particularly impaired in PD patients (Lewis et al., 2003; Lewis, Slabosz, Robbins, Barker, & Owen, 2005), which is in alignment with a possible role of dopamine in affecting WM capacity found by Cools and colleagues (2008). In a positron emission tomography (PET) imaging study, they reported that dopamine synthesis in the striatum of young adults is associated with WM function, with greater capacity (**Figure 1.11**) — indexed by higher levels of listening span — correlated with higher dopamine tracer uptake, and hence greater rates of striatal dopamine synthesis (Roshan Cools, Gibbs, et al., 2008).

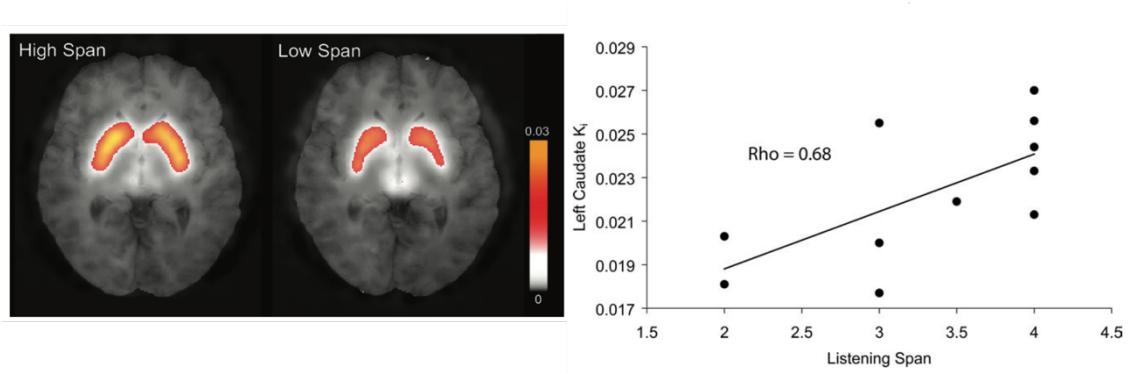


Figure 1.11| Dopamine synthesis in the dorsal striatum is predictive of working memory capacity in young adults.

Striatal dopamine synthesis using whole-brain 6-[18F] *fluoro-L-m-tyrosine* PET reveals that young adults with high span (index of WM capacity) scores have greater uptake of tracer in the striatum compared to those with low listening span. Moreover, the authors found a significant positive correlation between listening span and left dopamine synthesis in the left caudate nucleus. Reprint from Cools, Gibbs, Miyakawa, Jagust, & D'Esposito, 2008.

To dissociate the sources of errors arising from these patients' memory deficit, Zokaei and colleagues (2014) tested these patients on a sensitive delayed reproduction paradigm before they were started in their dopaminergic drug therapy. The task consisted of orientated, colour bars randomly presented on a screen and participants were instructed to remember their colour and orientation. At probe, they were asked to

replicate the orientation of the previously displayed item in order to match the orientation of the same colour item held in memory. Their results show a disease related decline in their ability to precisely remember orientation, i.e., the angular deviation of the response angle, compared to the target orientation, was greater for unmedicated PD compared to controls. Importantly, they showed that hindered precision was a result of an increased number of errors due to **guessing** (Zokaei, Mcneill, et al., 2014). Because the contribution of dopamine to patients' cognitive impairment is still poorly understood, researchers have focused on studying PD patients' performance both ON and OFF their dopaminergic medication, allowing for the mechanism associated with the WM deficit to be isolated from the neurotransmitter (Lange et al., 1992; Lewis et al., 2005; Poewe, Berger, Benke, & Schelosky, 1991).

1.8.2.1. The dopaminergic system and its role in visuospatial working memory

Conflicting results have surfaced from manipulating dopaminergic states: some have found that dopamine improves WM (Lange et al., 1992; Zokaei, Burnett Heyes, Gorgoraptis, Budhdeo, & Husain, 2015), others have shown evidence that it hinders performance (Fournet, Moreaud, Roulin, Naegele, & Pellat, 2000; Owen, Iddon, Hodges, Summers, & Robbins, 1997; Poewe et al., 1991). Cools et al. (2011) review these paradoxical results and suggest that dopaminergic state is not related to cognitive performance in a linear way, but can rather be represented by an inverted U-shape function, i.e., there is an optimal level of dopamine associated with top performance, but this can be shifted depending on the paradigm being used. Additionally, they found that dopamine depletion, or over-dosing, can also hinder performance (Roshan Cools & D'Esposito, 2011). They further point out that cognitive control may be multi-factorial,

involving an active equilibrium between cognitive flexibility and cognitive stability, i.e., depending on the type of task being studied, the levels of dopamine may have differential effects on the brain regions that are concerned (Roshan Cools & D'Esposito, 2011).

Fallon et al. (Sean James Fallon, Mattiesing, Muhammed, Manohar, & Husain, 2017) tackled this by delving into the separate consequences of maintaining and manipulating information in WM while controlling for patients' dopaminergic state. They found that, while dopamine improved performance when irrelevant memoranda had to be ignored or updated, it offered no benefit on the ability to maintain information over the delay period — even though the latter was impaired in patients compared to controls. Their findings are in agreement with the hypothesis that distinct mechanisms contribute to WM deficits in PD: a dopamine dependent frontal dysexecutive effect, responsible for gating relevant or irrelevant information into WM (R. G. Brown & Marsden, 1991; Dalrymple-Alford, Kalders, Jones, & Watson, 1994; Gruszka, Bor, Barker, Necka, & Owen, 2016), and a posterior cortical disorder unaffected by dopamine, responsible for simple maintenance of information (Gratwicke et al., 2015; Kehagia et al., 2010). Importantly, they denote that, even though patients were significantly impaired in their ability to maintain information over a delay period compared to controls, they still did not benefit from being ON their dopaminergic medication, potentially reflecting the involvement of non-dopaminergic neurotransmitters such as disruption in serotonin.

1.8.2.2. The role of serotonin in PD

Although PD is associated with a gradual depletion of nigrostriatal and mesocortical dopamine, other neurotransmitter systems such as the cholinergic, noradrenergic and

serotonergic systems (Dubois, Ruberg, Javoy-Agid, Ploska, & Agid, 1983; Kish et al., 2008; Zarow, Lyness, Mortimer, & Chui, 2003) are also affected.

The serotonergic system comprises of serotonergic neurons that are found in the raphe nuclei of the brainstem with extensive neuroanatomical ramifications to different regions of the brain: the forebrain (basal ganglia, amygdala, hypothalamus, cingulum, the medial cerebral cortex and portion of the hippocampus), as well as the caudal brainstem and spinal cord (Marios Politis & Niccolini, 2014). It also encompasses serotonin transporters (SERT or 5-HTT), monoamine proteins responsible for transporting serotonin (a neurotransmitter also known as 5-hydroxytryptamine or 5-HT) from the synaptic cleft to the presynaptic neuron. Importantly, because it provisions extensive areas of the brain, its dysfunction may be implicated in diverse domains (Roshan Cools, Roberts, & Robbins, 2008).

As the pathological process in serotonergic raphe nuclei transpires at stage 2 of Braak's staging of PD (a pre-symptomatic stage prior to lesions of midbrain dopaminergic neurons), loss of caudal brainstem serotonergic neurons has been hypothesised to precede the loss of dopaminergic midbrain neurons (Heiko Braak, Ghebremedhin, Rüb, Bratzke, & Del Tredici, 2004). According to a study by Politis and Nicollini (2014), this progression of serotonergic depletion is non-linear and affects different brain regions (**Figure 1.12**) (Marios Politis & Niccolini, 2014).

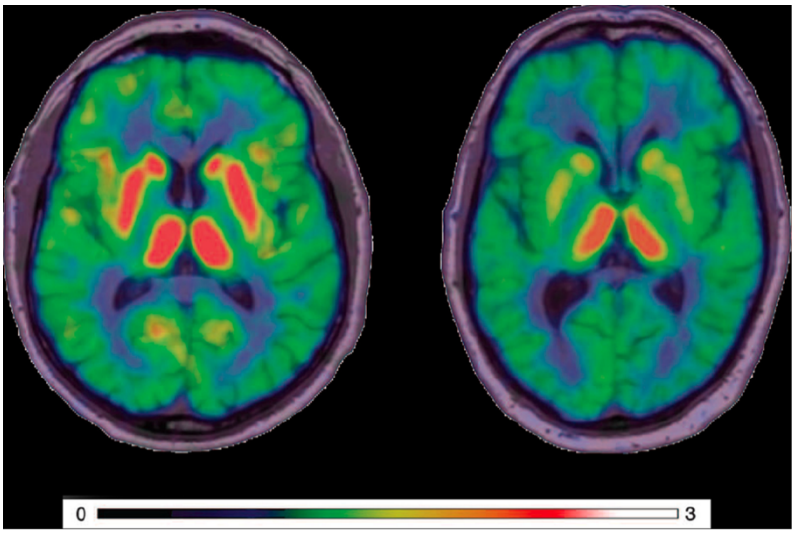


Figure 1.12| Reduction of serotonergic innervation in the basal ganglia in Parkinson's disease patients (left) as opposed to controls (right).

PET molecular imaging co-registered and fused with 1.5-T MRI images was implemented by Politis et. al (2014) to detect in vivo disease-related changes in the brain. A specific serotonin radioligand (^{11}C-DASB) to image SERT in three groups of PD patients depending on their disease duration was used. Results revealed a significant reduction of about 30% in ^{11}C-DASB binding to SERT across the course of the disease, irrespective of disease duration, disability or dopamine replacement therapy. Reprint from Politis & Niccolini, 2014.

Compromised emotional processing is an important link between the serotonergic system, regulation of mood and the experience of depression. Because SSRIs are capable of binding to SERT and blocking reuptake of serotonin in presynaptic cells, thereby increasing the level of serotonin within the synaptic cleft, they have been extensively used in the treatment of depression. Importantly, they have recently been shown to have a short-term probing effect of acute serotonergic modulation in the brain without changes in levels of mood or anxiety (Harmer et al., 2009; Harmer, Shelley, Cowen, & Goodwin, 2004; Ye et al., 2014). For example, Fu et al. (2004) showed that after administering SSRI to PD patients with depression for eight weeks, they displayed reduced activation in the amygdala, ventral striatum and frontoparietal cortex when presented with sad facial expressions. In agreement with these findings, Harmer and

colleagues (2009) have further revealed that SSRIs enhance positive biases in emotional perception and memory in PD patients. Taken together, these findings suggest that SSRI may normalize negative biases in processing information, and such changes in emotional processing may be effective in the management of depression.

Reduced serotonergic neurotransmission has also been related to LTM (Park et al., 1994; Riedel, Klaassen, Deutz, Van Someren, & Van Praag, 1999; Rubinsztein et al., 2001; Schmitt et al., 2000). However, little is known of its effects in WM. Harmer and colleagues reported a benefit on memory consolidation after administrating 10 mg of an SSRI to healthy volunteers, but no change in the immediate recall on the verbal memory task (Harmer, Bhagwagar, Cowen, & Goodwin, 2002). Yet, they failed to assess the resolution of memory representation. Therefore, in the last Chapter of this work, **Chapter 6**, I investigate the effects of SSRI on WM performance and emotional processing in both PD and age-matched controls. Importantly, the two behavioural experimental paradigms implemented assess performance in a sensitive continuous manner.

1.9. *Outline of Experimental Chapters*

This thesis aims to contribute to the understanding of the mechanisms underlying WM and how these might be impaired in clinical populations. This work is divided into two parts. In **Part I**, I will gain mechanistic insight into the process of memory retrieval (**Chapter 1** and **Chapter 2**) and the relationship between processing demands at all stages of WM (**Chapter 3**). In **Part II**, I will discuss malfunctions of the underlying processes and aspects of behavioural changes in two special cases of neurological disorders: autoimmune LE (**Chapter 5**) and PD (**Chapter 6**).

Chapter 2. The nature of retrieval cues affects recall performance

Abstract

Although most research has focused on understanding encoding and maintenance in WM, our ability to recall items may be dependent upon the type of information presented at retrieval. To test this hypothesis, in the first part of this study we employed a "what-was-where" design, where participants needed to remember the identity and corresponding location of memoranda. Participants always encoded the same information (circles divided vertically in two components – semi-circles – that differed in colour), but two factors were manipulated at the identification phase. Firstly, we varied whether both or only one of the components of the memoranda was presented. Secondly, we varied the level of competition at recall by asking participants to identify the target item from either a novel item or a horizontally flipped version of the remembered item. Results suggest that presenting more components of the memoranda at retrieval can improve identification and localisation performance. Importantly, more information in the retrieval cue improved identity-location binding, thus decreasing the likelihood of misreporting features belonging to another item presented in the memory array.

Because recall from WM can be envisaged as a decision process in which evidence is accumulated about different retrieval options, HDDM was implemented to test how changes in retrieval cues influence the decision-making process involved in identification of memoranda. Results show that increasing the amount of information in the target cue helps recall by increasing the rate of accumulation for the correct option. By contrast, greater similarity (high competition) between cues presented at retrieval leads to poorer recall due to greater interference between accumulators in the decision process.

Taken together, these findings suggest the nature of retrieval can significantly affect memory reports, and rather than acting as a form of interference and impairing memory, more information at retrieval can improve access to items in WM.

2.1. *Introduction*

WM is a fundamental cognitive function that allows storage, manipulation and retrieval of a limited amount of information over a brief period of time. However, WM can be corrupted: people can forget information even over short retention delays. Despite its significance, current research is still unclear about how forgetting occurs. To date, inferences on the limit of WM performance have focused largely on encoding by manipulating the perceptual contents of memory arrays, including memory load and complexity of feature conjunctions in each item (Ceraso, 1985; Irwin, 1991; Wheeler & Treisman, 2002; Zhang & Luck, 2008; Luck & Vogel, 2013); although, more recently, interest has shifted to the effects of maintenance delay (Fallon, Mattiesing, Dolfen, Manohar, & Husain, 2018; Souza & Oberauer, 2014) and of delaying retrieval cues (Souza S. et al., 2016). A consistent finding has been that recall decreases with higher load, which has been accounted for either by insufficient slots (J. S. Luck & Vogel, 1997) or a flexible WM resource being spread more thinly over the number of items to be stored (Paul M. Bays & Husain, 2008a). Evidence suggests that this resource is continuous and can be flexibly shifted among items, with increased imprecision — a decrease in fidelity of mental representation of any individual item — as memory load increases (P. M. Bays et

al., 2011; Paul M. Bays et al., 2009; Paul M. Bays & Husain, 2008a; Gorgoraptis et al., 2011).

However, recent studies have demonstrated that manipulations at retrieval also significantly affect memory report. For example, Souza and colleagues showed that simply delaying responses after the memory probe for at least 0.5s leads to benefits in recall performance (Souza S. et al., 2016). They propose that allocating time for retrieval before decision allows for higher accumulation of evidence about the memoranda and results in better recall. In line with this, Theeuwes et al. (Theeuwes et al., 2011) explored the role of visuospatial attention when retrieving information from WM. They asked participants to encode four colour circles displayed in different locations on a screen while concurrently asking participants to repeat "56" aloud, making sure the memoranda was not recoded into a verbal code. This was followed by the question on whether a specific colour was present in the memory array. When probed with the location that previously was occupied by the colour participants were asked to retrieve, reaction times were faster compared to trials where the probe was presented at any other location, suggesting that manipulations of visual attention at retrieval can facilitate access to information in WM.

Consistent with this finding, it has subsequently been demonstrated that the method by which memory is probed (the nature of the information presented in the cue) also affects recall precision, as unnecessary information in the probe (Tabi et al., 2019) or the presence of distractors at the time of recall hinders performance (Makovski et al., 2010; Udale et al., 2017).

These findings align well with a recent computational neural model of WM (Manohar et al., 2017) that establishes a dual functional architecture in which neurons hold information in both persistent activity and changes in synaptic weights – active and silent representations, respectively. The model proposes that rapid plasticity in flexibly-coding neurons allows features to be bound together into objects with recent items being preserved in synaptic traces. Even though they may be in a non-privileged state, the model predicts that features belonging to an object can be reactivated at retrieval through a process of pattern completion, thus suggesting that more information at the moment of recall affects how this process will unfold.

In the literature, there are two contrasting ideas regarding the effect of the amount of information at the time of recall. Findings from visual STM studies suggest that the more information presented at retrieval, the worse performance will be because more features have to be bound together (Wheeler & Treisman, 2002). On the other hand, results from hippocampal episodic memory studies have suggested that the more information presented at retrieval, the better recall will be via pattern completion mechanisms (Rolls, 2013). Thus, in the first part of this study we gain insight into the process of memory retrieval from WM by investigating how the quality of recall depends upon the amount and type of information presented at recall. I hypothesise that the amount of information carried by the retrieval cue can improve recall from WM. It might do so by facilitating associative retrieval, i.e., by presenting a partial cue, a process that retrieves other features belonging to an item in memory is triggered just as in pattern completion, perhaps by enabling integration of information held across disparate cortical regions. To test this, healthy young participants were asked to complete a sensitive

tablet-based behavioural task with a continuous measure of report. They had to remember both the identity and location of circles divided vertically into two semi-circles of different colours. After a brief maintenance interval, they were presented a test display containing a dual choice between a target (an item they had previously seen) and a foil. They were asked to select the target and drag it to its original location. While keeping the encoding display constant, at retrieval, two factors were varied: (1) *amount of information* by displaying either 'complete' items composed of both semi-circles, or 'incomplete' items composed of just a single semi-circle; (2) *amount of competition* by manipulating the level of similarity between cues, the foil either contained novel colours or had the same colour as the target but flipped in position.

Importantly, instead of solely probing participants in a binary manner by asking them to correctly identify memoranda, they were also requested to reproduce the exact quality of the remembered feature by dragging the item to its original location. This continuous measure of report allowed to deconstruct and quantify the sources of error into imprecision of recall, errors arising from misbinding or guessing using a mixture modelling analysis approach (Grogan, Fallon, Zokaei, Husain, & Manohar, 2019).

Even though inferences on the limit of performance have recently focused on precision methods to describe the pattern of errors contributing to memory performance (Paul M. Bays & Husain, 2008a; Palmer, 1990; Wilken & Ma, 2004), RT from WM retrieval have also been reported to provide a quantitative mechanistic perspective on cognitive processes. Pearson et al. (2014) postulated that recall from WM can be envisaged as a decision process in which information is accumulated about different retrieval options. The authors tested this by implementing a simple "rise to threshold" decision model, the

LATER. They found that retrieval times increase with memory load and that the quality of evidence provided to the decision mechanism — the mean rate of rise of evidence accumulation towards a decision threshold — is associated with the amount of response variability around the target (memory precision). Notably, they show that evidence is accumulated faster towards a decision threshold when an item is cued — when the resolution of its representation is boosted to the detriment of others — as opposed to a reduction in accumulation rate for uncued items.

By presenting evidence in support of the idea that the precision of memory report is modulated by reaction time distributions in participants' recall performance (Pearson et al., 2014), retrieval from WM can be conceptualized as a decision made on the quality of internal evidence. Thus, to quantify the impact in changes in the amount and type of information held by retrieval cues in the decision-making process involved in identification of memoranda from WM, the second part of the data collected here was modelled using HDDM. Greater similarity (high competition) between cues presented at retrieval is postulated to lead to poorer recall due to greater competition between accumulators in the decision process. By contrast, increasing the amount of information in the cue might help recall by increasing the rate of accumulation for the correct option.

2.2. *Experiment 1*

The first study investigated the effect of the nature of retrieval cues on the quality of WM recall. The key questions we sought to answer were:

1. Does the nature of the foil type, i.e., more or less information included in the foil, at retrieval have an impact on recall?
2. Does the nature of the foil type at retrieval influence recall depending on the amount of competition, i.e., the level of similarity between the foil and the target?
3. Are these manipulations modulated by delay? Does delay lead to worse performance overall or is there a selective effect of delay?

2.2.1. Methods

2.2.1.1. Participants and Consent

Twenty-one young adults aged 18-30 years (mean age = 23.3, SD = 3.7, 15 males, 6 females). Participants had no history of neurological or psychiatric disease and good vision and were screened for depression with the Beck Depression Inventory-II (BDI-II) (A.T. Beck, Steer, & Brown, 1996). All were reimbursed for their time (£10) and transport costs.

Participants were recruited from the Oxford Psychology Research Participant Recruitment Scheme on SONA. Permission for this study was obtained from the local ethics committee and all subjects gave their written informed consent.

2.2.1.2. Stimuli

Stimuli consisted of circles (visual angle of 3.2°), each split into half circles of different colours, presented on a grey background (RGB [171,171,171]) and drawn from

a library of 42 randomly selected foils without repetitions for every trial. Six colours were used (yellow, pink, cyan, red, green, blue) and randomly selected for each display with the constrain that no repetition of colour be within an object.

2.2.1.3. Design

Participants were asked to remember the identity and location of a set of stimuli (a target and a non-target) presented within a memory array for 2 seconds (Figure 2.1). After a brief blank maintenance interval (1 or 4 seconds), a test display with a dual choice containing one item that was previously shown during encoding (a target) and a distracter (foil) was shown. Participants were asked to select the object they saw in the initial array (identity identification) and to drag it to its remembered location (location identification). Two factors were manipulated at retrieval. First, we varied the amount of information in the retrieval cue by displaying either 'complete' targets composed of two semi-circles, or 'incomplete' targets composed of just a single semi-circle. Second, we manipulated the amount of competition, i.e., the level of similarity between cues: the foil either contained novel colours or had the same colours as the target but flipped in position.

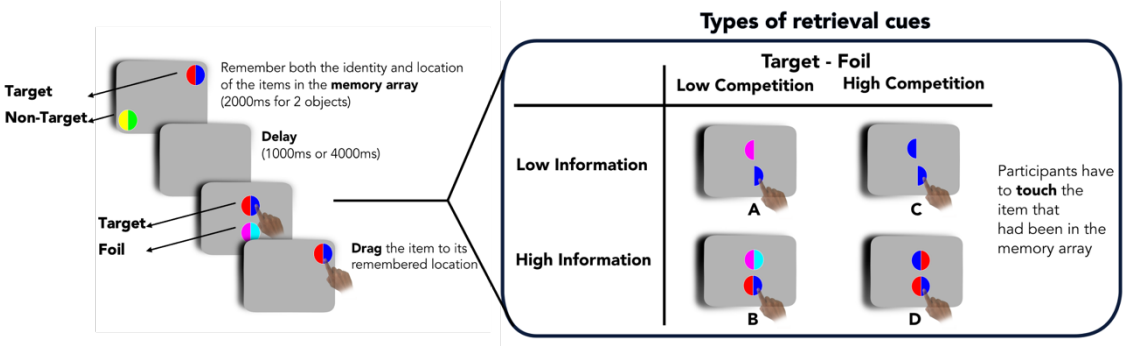


Figure 2.1| Schematic of experimental task used to examine the effects of the nature of retrieval cues on the quality of recall.

Participants were required to remember both the identity and location of memoranda. After a variable delay (1 or 4 seconds) they were given a two-alternative choice between a target and a foil. They had to touch the target, then drag it to where it originally appeared. Four conditions were used in the WM paradigm. (A) Low information & Low competition, (B) High information & Low competition, (C) Low information & High competition, (D) High information & High competition.

The task featured four conditions (Figure 2.1) with all possible combinations of the retrieval cues (low vs high information, novel vs flipped) and delay of either 1 or 4 seconds:

(A) Low Information & Low Competition: the *target* consisted of a single semi-circle from an item that previously appeared in the memory array and the *foil* consisted of a single semi-circle filled with a novel colour that had not appeared in the memory array (Figure 2.1, Condition A).

(B) High Information & Low Competition: the *target* was composed of two semi-circles that were previously presented as a complete item in the memory array; the *foil* consisted of two semi-circles filled with novel colours that had not appeared in the memory array (Figure 2.1, Condition B).

(C) Low Information & High Competition: the *target* consisted of a single semi-circle from an item that previously appeared in the memory array while the *foil* consisted of the flipped version of the target (Figure 2.1, Condition C).

(D) High Information & High Competition: the *target* consisted of two semi-circles that were previously presented as a complete item in the memory array; the foil consisted of the flipped version of the target (Figure 2.1, Condition D).

2.2.1.4. Procedure

The session took 1 hour to complete. Participants sat in front of an interactive touch-sensitive screen (iPad version 9.3.5 (13G36), model MGTX2B/A) with 1536x2048 pixel matrix. Experimental trials were randomized and presented within one block of 48 trials per condition (24 trials for 1 second delay maintenance and 24 trials for 4 seconds delay maintenance).

Data handling and statistical analysis were performed in *MATLAB* (The Math-Works inc., version 2018a) and *JASP* (JASP, version 0.12.1).

2.2.2. Behavioural Results

Behavioural analyses assessed the quality of recall using three measures: *identification accuracy*, i.e., the proportion of times participants touched the correct shape in the dual choice; *reaction time,* the time (in seconds) necessary to initiate a correct response; and *localisation error*, the distance (in pixels) between the centre of the reported location and the centre of the original location of the probed item.

In Experiment 1, we tested whether the amount of information presented at retrieval affected memory performance, by displaying one or two semi-circles as retrieval cues (**low vs high information**). We also tested whether the level of similarity between the features of the items retrieved affected recall, by presenting either a novel semi-circle or a horizontally flipped version of a semi-circle shown in the memory array (**low vs high competition**).

Accordingly, a 2 (Information: low vs high) x 2 (Competition: low vs high) x 2 (Delay: 1 second, 4 seconds) repeated measures analysis of variance (ANOVA) was conducted on identification accuracy, localisation error and reaction time (**Figure 2.2**). Note that only trials where participants correctly identified the target were included in the analysis of localisation error and reaction time. Significant interactions were unpacked with *Holm* corrected pairwise comparisons with a 95.0% confidence interval and results were considered significant if their p-value was less than .05. Furthermore, the data was also analysed using a Bayesian ANOVA. Unlike the classical approach, Bayesian statistics allows one to assess the relative evidence favouring the null hypothesis versus a specified alternative hypothesis, permitting a measure of the strength of the evidence favouring the null (Jeffreys, 1935).

The ***amount of information*** in the retrieval cue did not significantly affect identification accuracy [F(1, 20) = 3.027, p = .097, η_p^2 = .131, BF_{10} = .0321, BF_{incl} = 1.280] (**Figure 2.2, Identification Accuracy**). However, when given more information at retrieval, young adults were faster at identifying the correct memoranda compared to when they were given low information [F(1, 20) = 39.766, p < .001, η_p^2 = .668, BF_{10}= $7.669e^6$, BF_{incl} = $1.815e^7$] (**Figure 2.2, Reaction Time**), as well as remembering its location [F(1, 20) =

50.317, p = < .001, η_p^2 = .716, BF_{10} = 1.718e^6, BF_{incl} = 1.353e^1] (**Figure 2.2, Localisation error**).

Overall, the ***amount of competition***, i.e., the degree of shared similarities between retrieval cues, did have a significant effect on identification accuracy. Participants performed better when there were fewer competitive cues at recall [F(1, 20) = 23.891, p < .001, η_p^2 = .544, BF_{10} = 7.778e^7, BF_{incl} = 1.035e^8]. Additionally, there was a significant effect of Information by Competition interaction on the ability to correctly identify memoranda [F(1, 20) = 9.868, p = .005, η_p^2 = .330, BF_{incl} = 4.573]. Post-hoc t-tests showed that, in conditions of low competition, participants benefited from more information (t(21) = 3.498, p_{holm} = .005). However, this was no longer the case under high competition (t(21) = 1.166, p_{holm} = .251) (**Figure 2.2, Identification Accuracy**).

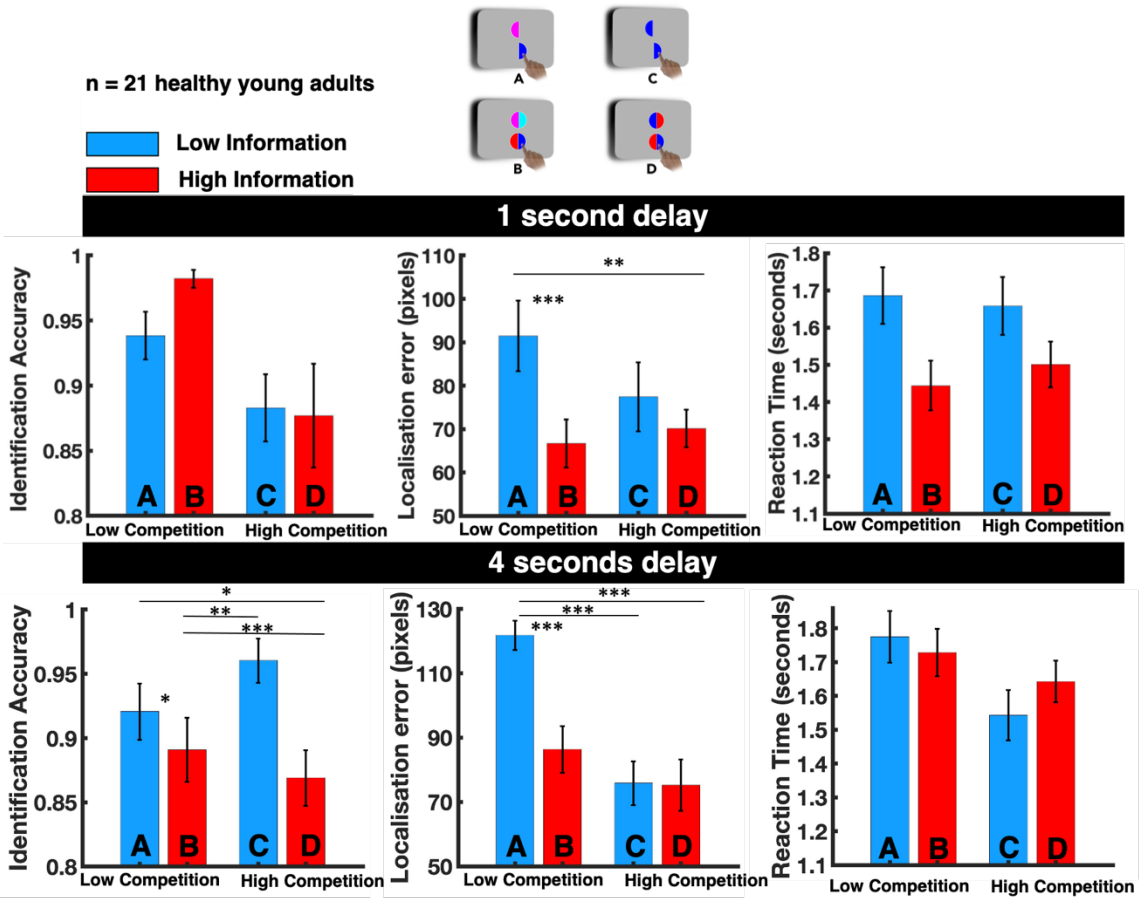


Figure 2.2| Results for Experiment 1 for both 1 and 4 second maintenance delays.

An increase in information at retrieval led to better localisation ability and faster reaction times while more competitive cues hindered identification accuracy and localisation performance. Similarly, longer delays (4 seconds) led to slower reaction times and greater localisation errors. Post-hoc t-test were applied, *** p-values < .001, ** p-values < .01, * p-values < .05. Details are summarized in Appendix Tables 2.10-2.16 (1 second delay) and Tables 2.17-2.24 (4 seconds delay). Error bars reflect standard error of the mean (SEM) and were calculated using the Loftus & Masson method (Loftus & Masson, 1994).

Localisation performance was also affected by the amount of competition at recall producing smaller location error when competition was low [$F(1, 20) = 24.323$, $p < .001$, $\eta_p^2 = .549$, $BF_{10} = 8.955$, $BF_{\text{incl}} = 1.283e^4$]. Information and competition were again found to interact, suggesting a different effect of competition on recall depending on the amount of information [$F(1, 20) = 12.910$, $p = .002$, $\eta_p^2 = .392$, $BF_{\text{incl}} = 1.232e^3$. Post-hoc t-tests revealed that presenting more information on the retrieval cue improves

localisation ability in low competition trials (t(21) = 7.353, p_{holm} < .001) but not under conditions of high competition (t(21) = 4.815, p_{holm} = .127) (**Figure 2.2, Localisation error).**

Reaction time was not influenced by the degree of similarities between retrieval cues, as suggested by no main effect of competition on reaction time [F(1, 20) = 0.333, p = .570, η_p^2 = .016, BF_{10} = .201, BF_{incl} = .283]. However, there was a significant interaction effect between Information and Competition [F(1, 20) = 6.475, p = .019, η_p^2 = .0245, BF_{incl} = .875]. Young adults were quicker to respond to the target in high information compared to low information trials for both conditions of high and low competition (t(20 = 3.337, p_{holm} = .006; t(20) = 6.514, p_{holm} < .001, respectively). Differences in reaction times were not statistically significant when manipulating competition in both high and low information trials (t(20) = 1.846, p_{holm} = 0.147, t(20) = .869, p_{holm} = .391, respectively) (**Figure 2.2, Reaction time**).

The length of the maintenance interval (delay) following encoding did not affect identification performance [F(1, 20) = 1.756, p = .200, η_p^2 = .081, BF_{10} = .237, BF_{incl} = .154] (**Figure 2.2, Identification Accuracy**). Conversely, localisation performance was significantly affected, with longer delays leading to greater localisation error [F(1, 20) = 47.940, *p* < .001, η_p^2 = .706, BF_{10} = 49.956, BF_{incl} = 1294.900]. Additionally, a Competition by Delay interaction [F(1, 20) = 4.972, *p* = .037, η_p^2 = .199, BF_{incl} = 3.968] revealed that 4 second delays significantly thwarted participants' localisation performance under high competition trials compared to low (t(20) = 4.861, p_{holm} < .001). Yet, this impairment was no longer observed for the 1 second maintenance delay (t(20) = 1.424, p_{holm} = .325) (**Figure 2.2, Localisation error**). Delay also affected the time participants took to correctly identify memoranda as per a main effect of delay on

reaction time [F(1, 20) = 12.811, p = .002, η_p^2 = .390, BF_{10} = 21.902 BF_{incl} = 46.015] (**Figure 2.2, Reaction Time**). Please see **Appendix Table 2.1 – 2.24** for further details.

2.2.3. Conclusion

This novel WM task tested on twenty-one healthy participants provides evidence that retrieval cues influence quality of recall from STM. Varying the amount of information in the retrieval cue – by displaying either 'complete' items composed of two semi-circles, or 'incomplete' items composed of just a single semi-circle – did *not* affect the frequency with which young adults select the correct memoranda. The presence of more information in the retrieval cue did allow participants to identify memoranda faster and improved their ability to locate them. Most importantly, varying the amount of competition, i.e., the level of similarity between foil and target, by either presenting a foil with novel colours or with the same colours as the target but flipped in position, had a detrimental impact on overall performance. Foils that were more similar – and therefore considered to be associated with greater competition for correct target retrieval – hindered identification accuracy and localisation performance. Likewise, longer maintenance delays slowed participants down and worsened their ability to locate memoranda.

Intriguingly, presenting more information for a short maintenance delay (1 second) improved participants' ability to identify memoranda, but only under conditions of low competition. To further explore why this was the case, twenty young adults were tested on a variant of this task in Experiment 2.

2.3. *Experiment 2*

Experimental manipulation involved changes in the amount of Information (low and high) and the amount of Competition (low and high) while keeping the delay constant (1 second). The experiment consisted of more trials than in Experiment 1: 100 per condition resulting in a total of 400 trials. Crucially, this higher number of trials allowed us to model the sources of error contributing to performance.

The aim of the second study was to answer the following questions:

1. What is the impact of the nature of the foil on the pattern of recall errors?
2. How is the decision process of recall modulated by the amount of information and competition in the retrieval cue?

2.3.1. Methods

2.3.1.1. Participants

Experiment 2 further explored the pilot results by recruiting twenty young adults aged 18-33 years (mean age = 24.1, SD = 3.39, 3 males, 17 females) that were not previously enrolled in Experiment 1. They were screened for any history of neurological or psychiatric conditions and had normal or corrected to normal vision. All participants were also screened for depression with the BDI-II (A.T. Beck et al., 1996) and all were reimbursed for their time (£10) and transport costs. Similarly to Experiment 1,

participants were recruited from a volunteer database on SONA and all subjects gave written informed consent to participate.

2.3.1.2. Stimuli, Design and Procedure

Stimuli, design and procedure of Experiment 2 was the same as in Experiment 1, except that the retention period was held constant at 1 second and participants performed one block of 100 randomized experimental trials per condition.

2.3.1.3. Mixture Modelling of error

The increased number of trials (100 trials per condition) allowed us to assess the fidelity of WM representations by measuring the pattern of recall errors, providing information about the quality of memory representations and enabling the analysis of the sources of errors contributing to the pattern of performance (Paul M. Bays et al., 2009; Paul M. Bays, Wu, & Husain, 2011; Paul M. Bays & Husain, 2008a; Palmer, 1990; Wilken & Ma, 2004). Location report errors, i.e., errors in the reported location of the item, could occur as a result of three different factors: imprecision, misbinding or guessing (Figure 2.3).

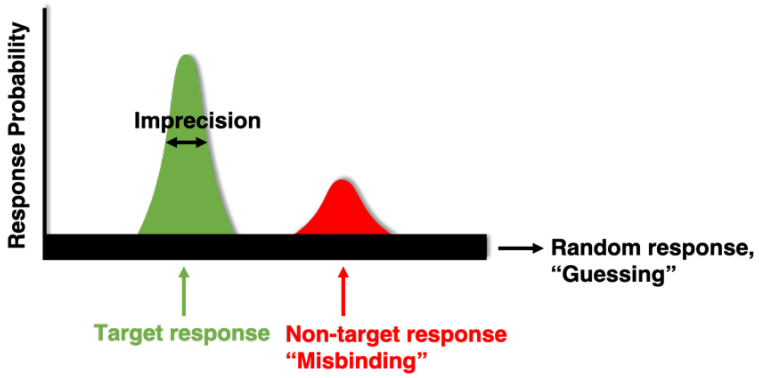


Figure 2.3| Schematic of the probability density function of responses of the different sources of memory error.

Localisation error could arise due to increased variability in responses towards the target (imprecision), by reporting the location of a non-target (misbinding) or by randomly misplacing memoranda (guessing). The height of each distribution illustrates the number of responses towards the target (green) and towards the non-target (red), the withs illustrate the imprecision with which the memory is recalled and uniform guessing across the screen (black).

- *Imprecision*, denoting the error in locating the target, with higher imprecision reflecting worse recall as per a larger width of the distribution around the target (width of the green distribution, Figure 2.3);
- *Misbinding,* referring to the proportion of times subjects misreport the location of the correctly identified item by dragging it to the location of the non-target item presented in the memory array (red distribution, Figure 2.3);
- *Guessing,* indicating the proportion of responses in which participants randomly located the target, i.e., there is no systematic relationship between the target location and the response location (uniform guessing across the screen in black, Figure 2.3).

In summary, this adaptation from 1D mixture models (Paul M. Bays et al., 2009) into 2D (Grogan et al., 2019), provides a much more powerful way to distinguish the error components thanks to two dimension of separation between objects as illustrated by equation (**2.1**):

$$P(\hat{\theta}) = \alpha\psi_{\sigma}(\hat{\theta} - \theta) + \beta\frac{1}{m}\sum_{i}^{m}\psi_{\sigma}(\hat{\theta} - \varphi_i) + \gamma\frac{1}{A} \quad (2.1)$$

Where $P(\hat{\theta})$ is the response density function, i.e., the probability of finding a response location. θ and φ_i are vectors indicating the target and non-target locations on the tablet screen, respectively. ψ_{σ} is the bivariate Gaussian distribution with SD σ and zero covariance. i to m are the number of non-target stimuli. Guessing is distributed across the screen, for simplicity the model assumes that this is drawn from a uniform distribution over the entire area of the screen (A). The parameters α, β and γ sum to 1, which together with σ (which is not included as a free parameter in the fitting) yield three free parameters that are estimated using maximum likelihood methods. These parameters control the proportion of target responding, non-target responding, guessing and the concentration of the bivariate Gaussian distribution, respectively.

Therefore, in order to deconstruct the three different sources of error, data analysis was performed using the *Matlab* package *MemToolBox2D* (Grogan et al., 2019).

2.3.2. Mixture Modelling Results

As in Experiment 1, we first investigated whether the amount of information (low and high information) and level of competition (similarity) between the features of the items at retrieval (low and high competition) affected recall. Accordingly, a 2 (Information: low vs high) x 2 (Competition: low vs high) x 2 (delay: 1 second vs 4 seconds) repeated measures ANOVA was conducted on identification accuracy, localisation error and reaction time (**Figure 2.4**). Significant interactions were unpacked with *Holm* corrected pairwise comparisons with a 95.0% confidence interval and results were considered significant if their p-value was less than .05.

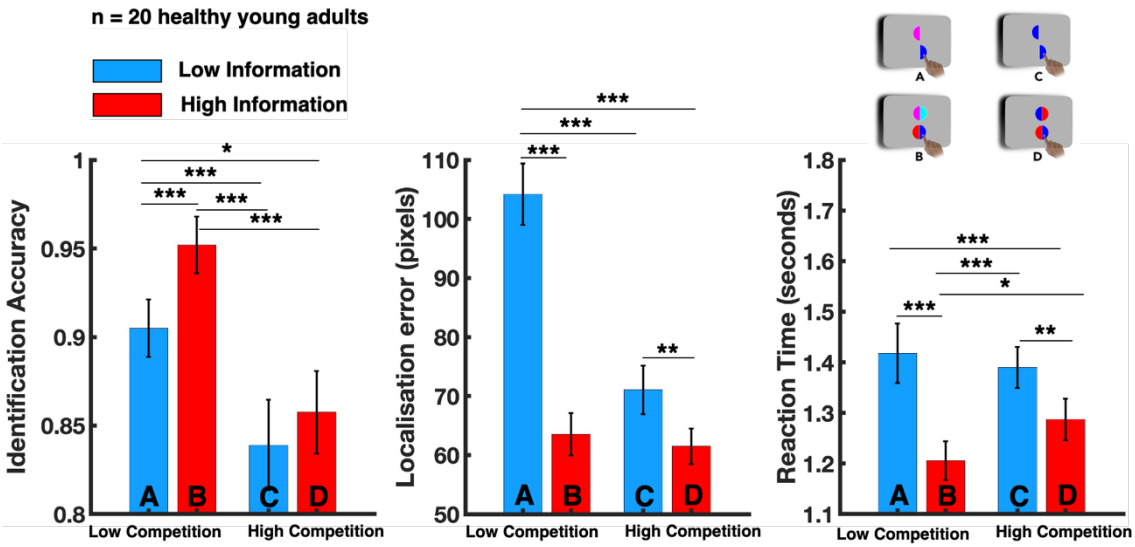


Figure 2.4| Overview of different cognitive patterns for Experiment 2.

Post-hoc t-test were applied, * p-values < .05, ** p-values < .01, *** p-values < .001. Error bars reflect SEM and were calculated using the Loftus & Masson method (Loftus & Masson, 1994).

Results suggest that participants benefited with respect to identification accuracy from more ***information*** at recall [F(1, 19) = 17.588, p < .001, η_p^2 = .0481, BF_{10} = 2.171,

BF_{incl} = 27.494] (**Figure 2.4, Identification Accuracy**). More information also improved localisation ability [F(1, 19) = 166.189, p < .001, η_p^2 = .897, BF_{10} = 4.842e^6, BF_{incl} = 6.005e^{15}] (**Figure 2.4, Localisation error**), as well as decreased the time required to identify the target [F(1, 19) = 50.630, p < .001, η_p^2 = .727, BF_{10} = 8.5350e^6, BF_{incl} = 1.495e^7] (**Figure 2.4, Reaction Time**).

There was also a main effect of ***competition***, i.e., the more competition at recall the worse participant's performance on identification accuracy [F(1, 19) = 38.404, p < .001, η_p^2= .669, BF_{10}= 3.378e^7, BF_{incl} = 2.999e^8] (**Figure 2.4, Identification Accuracy**) and localisation error [F(1, 19) = 79.291, p < .001, η_p^2= .807, BF_{10}= 245.346, BF_{incl} = 2.373e^{13}] (**Figure 2.4, Localisation error**) but not on reaction time [F(1, 19) = 1.652, p = .214, η_p^2= .080, BF_{10}=.325, BF_{incl} = 1.653] (**Figure 2.4, Reaction Time**).

Additionally, competition had a differential effect on identification accuracy depending on whether it was manipulated under high or low information as shown by a significant Information by Competition interaction [F(1, 19) = 4.895, p = .039, η_p^2= .205, BF_{incl} = 2.560] (**Figure 2.4, Identification Accuracy**). Post-hoc t-tests revealed that high competition trials exerted a cost on identification accuracy with participants selecting the target significantly less often when presented with a novel foil, compared to when presented with a foil similar to the target (t(20) = 4.563, p_{holm} < .001; t(19) = 6.534, p_{holm} < .001, for both low and high information trials respectively).

This interaction also affected the localisation error [F(1, 19) = 92.312, p < .001, η_p^2 = .829, BF_{incl} = 5.207e^8]. Under low information trials, participants' ability to remember the location of the target was significantly affected when more competitive cues were

presented at retrieval compared to less competitive cues (t(20) = 12.977, p_{holm} < .001), but this was no longer the case under high information trials (t(20) = .800, p_{holm} = 1 (**Figure 2.4, Localisation error**).

Competition also modulated the effect of information on reaction times [F(1, 19) = 9.217, *p* = .007, η_p^2 = .327, BF_{incl} = 5.603]. Unpacking this interaction revealed that, when provided with high information cue at recall, participants were faster to identify the target under low competition compared to high (t(20) = 2.965, p_{holm} = .032). However, this benefit was lost when they were presented with low information at recall (t(20) = 1.027, p_{holm} = 1) (**Figure 2.4, Reaction time**). Further details can be found in **Appendix Tables 2.25 – 2.33**.

Next, using mixture modelling, we analysed whether the amount of information (low and high) and level of similarity between features of items retrieved (low and high competition) contributed to the pattern of recall errors. Accordingly, a 2 (Information: low vs high) x 2 (Competition: low vs high) x 2 (delay: 1 second vs 4 seconds) repeated measures ANOVA was conducted on model parameters: imprecision, misbinding and guessing (**Figure 2.5**).

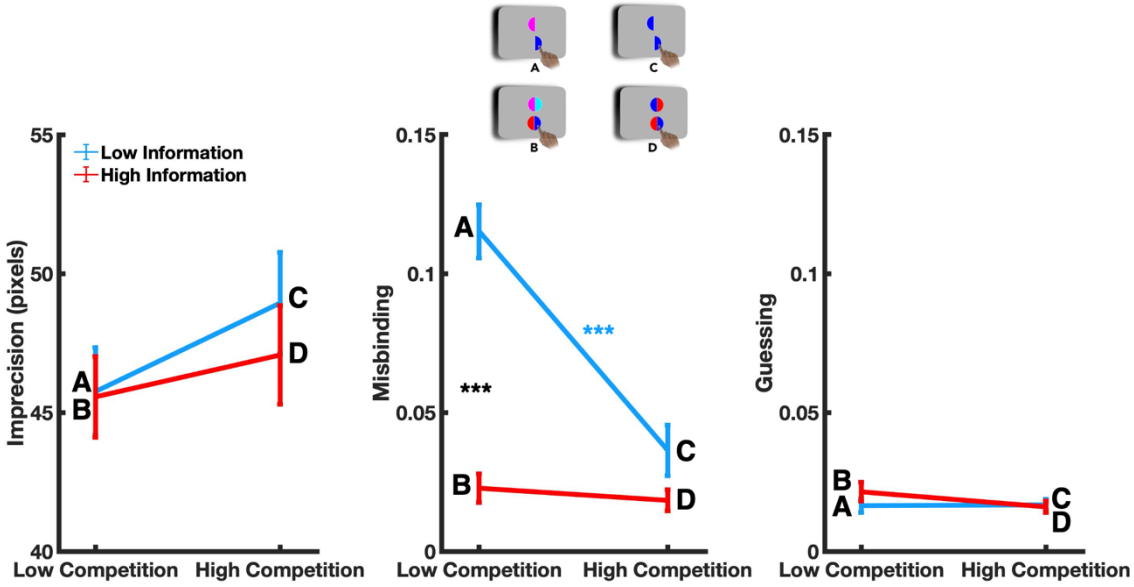


Figure 2.5| Mixture Model Results for Experiment 2.

Post-hoc t-test were applied, * p-values < .05, ** p-values < .01, *** p-values < .001. Error bars reflect SEM and were calculated using the Loftus & Masson method (Loftus & Masson, 1994).

The ***amount of information*** in the retrieval cue had a significant main effect on misbinding rates. Increased information was associated with less misbinding, i.e. it appeared to protect participants' ability correctly to bind the identity of memoranda with location [F(1, 19) = 116.756, p < .001, η_p^2 = .860, BF_{10} = 2.681e^6, BF_{incl} = 1.501e^{15}] (**Figure 2.5, Misbinding**). However, there was no significant main effect of amount of information on imprecision [F(1, 19) = 1.456, p = .242, η_p^2 = .071, BF_{10} = .503, BF_{incl} =.590] (**Figure 2.5, Imprecision**), nor on guessing rates [F(1, 19) = .837, p = .372, η_p^2 = .042, BF_{10} = .344, BF_{incl} = .268] (**Figure 2.5, Guessing**).

The ***amount of competition*** in the retrieval cue had a main effect on the precision with which participants localised the target. Increased competition led to greater imprecision of recall [F(1, 19) = 14.924, p = .001, η_p^2 = .516, BF_{10} = 18.888, BF_{incl} =

15.961] (**Figure 2.5, Imprecision**) but this effect was not modulated by Information [F(1, 19) = 1.897, p = .184, η_p^2 = .091, BF_{incl} = .782].

Misbinding rates were also affected by the amount of competition in the retrieval cues [F(1, 19) = 124.637, p < .001, η_p^2 = .858, BF_{10} = 740.303, BF_{incl} = 1.438e^{10}]. This effect was modulated by the amount of information in the retrieval cue as shown by a significant Information by Competition interaction on misbinding [F(1, 19) = 177.750, p < .001, η_p^2 = .903, BF_{incl} = 1.438e^{10}]. When participants were presented with high information trials, manipulations of competition made little difference in their ability to bind the identity with the location of memoranda (t(20) = .949, p_{holm} = .349). However, when presented with low information trials, misbinding rates were significantly increased under low competition compared to high competition conditions (t(20) = 16.927, p_{holm} < .001)] (**Figure 2.5, Misbinding**).

Additionally, increased competition at recall led to lesser guessing [F(1, 19) = .925, p = .004, η_p^2 = .348, BF_{10} = .411, BF_{incl} = .314] (**Figure 2.5, Guessing**) but this was not moderated by Information as per no interaction between competition and information on guessing [F(1, 19) = 2.421, p = .136, η_p^2 = .113, BF_{incl} = .168]. (**Appendix Tables 2.34 – 2.40**).

Thus, the most important findings revealed by modelling the sources of error related to the effects on misbinding identity with location as a function of information and competition.

2.3.3. Conclusion

The goal of Experiment 2 was to test whether the nature of the foil affects the type of recall errors. To summarize, results show that young adult participants benefit from more information provided at the time of recall when identifying memoranda. More information was shown to improve localisation performance. Importantly, this effect was driven specifically by significantly fewer errors due to misbinding: more information in the retrieval cue improved identity-location binding. These findings suggest that, rather than acting as a form of interference and impairing recall, more information at retrieval can improve access to items in STM.

Finally, reaction time results showed an interaction between information and competition. Although more information at retrieval led to faster responses, it improved participants' ability to identify memoranda only under conditions of low competition. However, conditions where participants performed less accurately were also paradoxically slower (Condition C, **Figure 2.1**), suggesting a violation of the speed-accuracy trade-off. To further explore this effect, we applied HDDM to both accuracy and reaction time data (Heathcote et al., 2019; Ratcliff & McKoon, 2008; Ratcliff et al., 2016).

2.4. *Hierarchical Bayesian Drift Diffusion Modelling*

Recall from WM has previously been conceptualized as a decision process (Pearson et al., 2014). Identification of information provided by a recall cue can be considered as a composite process, including both recognition of familiar information and rejection of unfamiliar information. Here, we test how changes in retrieval cues influenced the

decision process of recall by applying HDDM to both identification accuracy and reaction times. The evidence-accumulation approach provides a representation of the cognitive processes of speeded decision-making from gathering evidence from a stimulus (Ratcliff & Rouder, 1998; Ratcliff et al., 2016).

Cognitive models of the process of evidence accumulation were fit to the behavioural data using the open source, R-based Dynamic Models of Choice (DMC) software, together with the *rdists* Package (Heathcote et al., 2019; Singmann et al., 2019), v. 0.8-3, providing a Hierarchical Bayesian implementation of the DDM. The model assumes that evidence is continuous and stochastic, i.e., it varies from moment to moment during accumulation. Additionally, the DDM, as shown in **Figure 2.6**, assumes that, in a two-alternative choice task, evidence is accumulated for one alternative or the other (target or foil) at each time step and integrates that evidence until a decision threshold is reached (**Figure 2.6**). It is characterized by four main parameters:

- Decision bias, *z*, is the starting point parameter reflecting participants' bias
- Non-decision time, t_0, represents the delay between the memory probe and the start of the evidence accumulation process
- Drift rate, *v*, indicates the mean rate at which the evidence favouring the target (correct response) or the foil (wrong response) is accumulated over time until a decision is made by reaching a threshold.
- Decision threshold separation, *a*, denotes the amount of information accumulated over time until a response is made, reflecting response caution.

Additionally, the model assumes uniform variability in the start point from choice-to-choice with SD s_z and Gaussian choice-to-choice variability in the rate of evidence accumulation over a range of s_v. The moment-to-moment variability of the accumulation rate is fixed across conditions to 1.

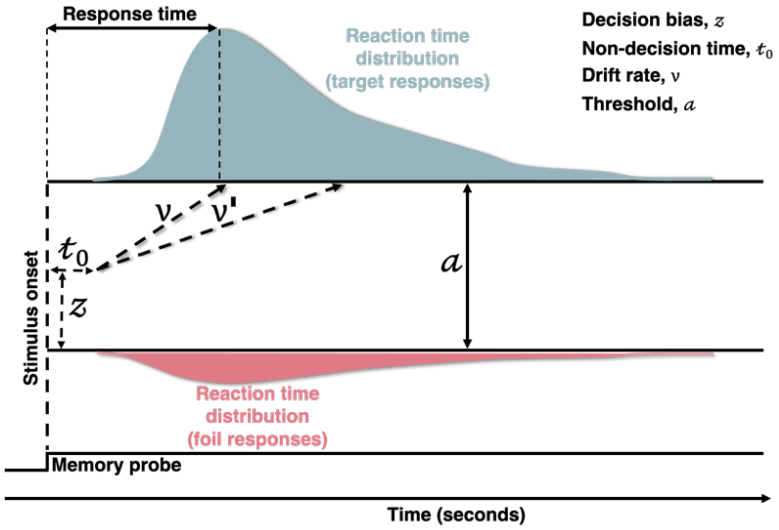


Figure 2.6| Modelling the dynamics of the decision process of recall using patterns of choice and response times.

Reaction time distributions and correct or incorrect answers are determined by underlying random accumulating evidence. The drift rate $\boldsymbol{v}$, is the rate at which evidence accumulates, faster drift rates leading to faster reaction times. The decision threshold $\boldsymbol{a}$, denotes the amount of evidence accumulated before a response is made. The non-decision time $\boldsymbol{t_0}$, represents the perceptual processing of stimuli and response production. Finally, a bias $\boldsymbol{z}$, in the offset towards one of the alternatives may be taken into account.

In order to unveil how changes in the amount and type of information at retrieval affect the neural mechanisms of the decision-making of recall, we used the HDDM (Appendix Chapter 2, Experiment 2 (HDDM)) to test the following three hypotheses:

Hypothesis 1: Increasing the amount of information helps recall by increasing the rate of evidence accumulation, while increasing the amount of competition acts as a form

of interference that hinders that accumulation process. Simultaneously, increasing the amount of competition leads to lower thresholds, reflected by faster incorrect responses due to noise generated by the retrieval cues (**Model 1:** *drift rate* modulated by amount of information & competition and *threshold* modulated by amount of competition).

Hypothesis 2: Increasing the amount of information helps recall by increasing the rate of evidence accumulation, while increasing the amount of competition between retrieval cues creates noise which adversely impacts the drift rate. Concurrently, more information at retrieval leads to faster mistakes reflecting less cautious responses (**Model 2:** *drift rate* modulated by amount of information & amount of competition and *threshold* modulated by amount of information).

Hypothesis 3: Increasing the amount of information helps recall by increasing the drift rate but more competition between retrieval cues decreases the mean rate of evidence accumulation. Likewise, more information and more competition led to noisier responses (**Model 3:** *drift rate* modulated by both amount of information & competition and *threshold* modulated by both amount of information & competition).

Model comparison was carried out using BPIC and DIC (Spiegelhalter et al., 2002) (**Figure 2.7**). The best fitting model (**Figure 2.8**) determined by the lowest deviance criterion (DIC = 12361.78) was Model 2, suggesting that the drift rate was modulated by both information and competition while threshold was solely influence by the amount of information held by the retrieval cue. The parameter that determined the winning model was the threshold that, in Model 2 was modulated by the amount of information, whereas was modulated by the amount of competition in Model 1 (DIC = 12390.02) and by both information and competition in Model 3 (DIC= 12367.81).

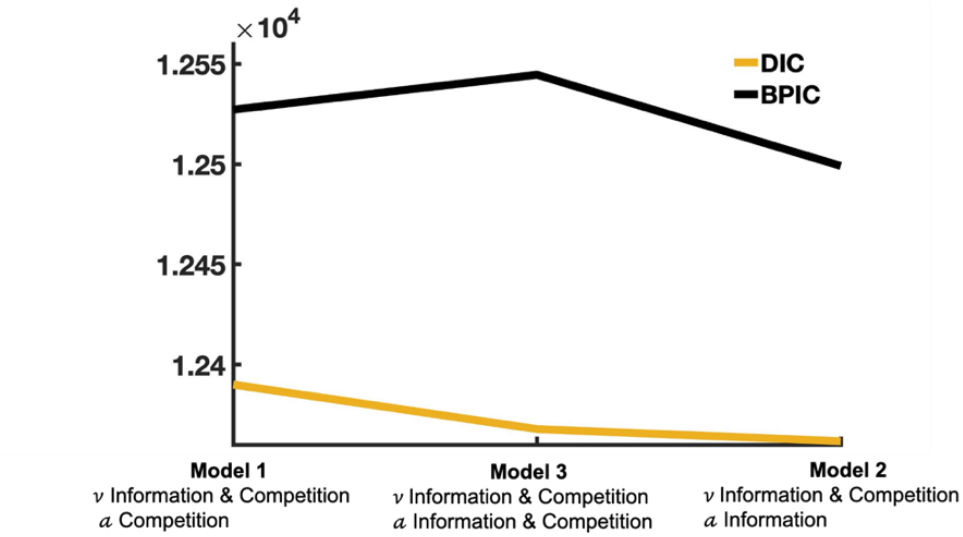


Figure 2.7| The best model fit is Model 2 where drift rate is modulated by amount of information and competition together with threshold being modulated by amount of information.

DIC is a hierarchical modelling generalization of the Akaike Information Criterion (AIC). BPIC avoids overfitting problems with DIC. Smaller DIC are preferred to models with larger DIC.

Subsequently, after all models were fitted using this Bayesian approach to parameter estimation, Model 2 (Figure 2.8) was further analysed. The winning hypothesis (Figure 2.7) posited that the rate of evidence accumulation was modulated by amount of *information and competition* while the threshold was solely modulated by amount of *information*.

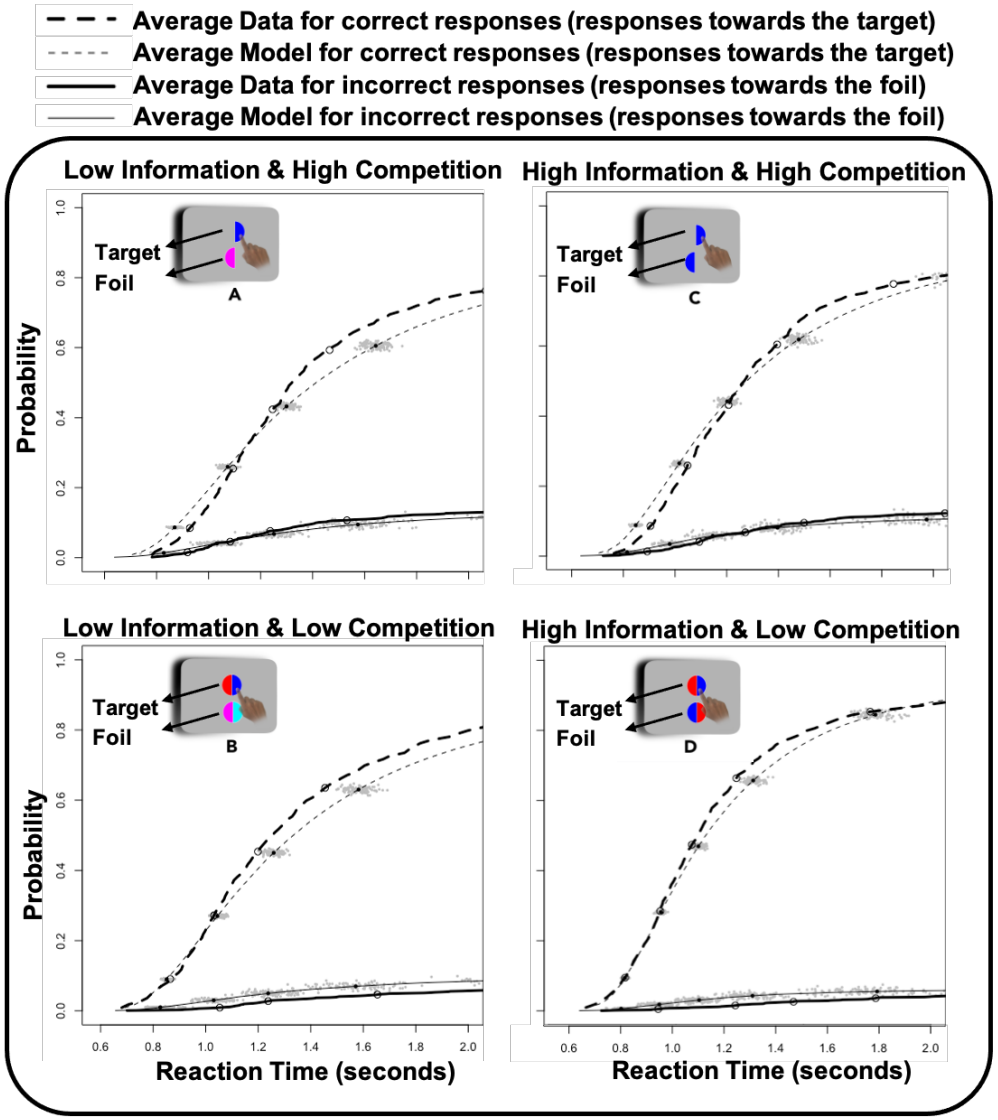


Figure 2.8| Data versus Model 2.

The average behavioural data is plotted against the average data simulated by the model for all four experimental conditions. Both the average data simulated for correct responses (responses towards the target) and the average data simulated for incorrect responses (responses towards the foil) are good fits for the behavioural data.

To further explore how the drift rate was modulated by both information and competition, a 2 (Information: low vs high) x 2 (Competition: low vs high) repeated measures ANOVA was conducted on drift rates. More information at recall led to higher drift rates as shown by the main effect of information [F(1,19) = 38.262 , p < .001, η_p^2= .668, BF_{10} = 5840.067, BF_{incl} = 1.249e^7]. Presenting less competitive recall cues allowed

for the evidence to be accumulated quicker compared to trials when more competitive cues were given at retrieval as revealed by a main effect of competition [F(1,19) = 43.420 , p < .001, η_p^2= .696, BF_{10} = 1013.013, BF_{incl} = 2.168e^6]. Additionally, information and competition were found to interact [F(1,19) = 21.454 , p < .001, η_p^2= .530, BF_{incl} = 99.784]. A post-hoc t-test showed that, under high information trials, participants benefited from less competitive cues (t(20) = 8.053, p < .001), i.e., under conditions of high information at recall, evidence in favour of the correct response is accumulated more quickly compared to conditions of low information in the retrieval cue. Similarly, even under low information conditions, participants also benefited from low competition trials (t(20) = 2.568, p = .031) (**Figure 2.9**).

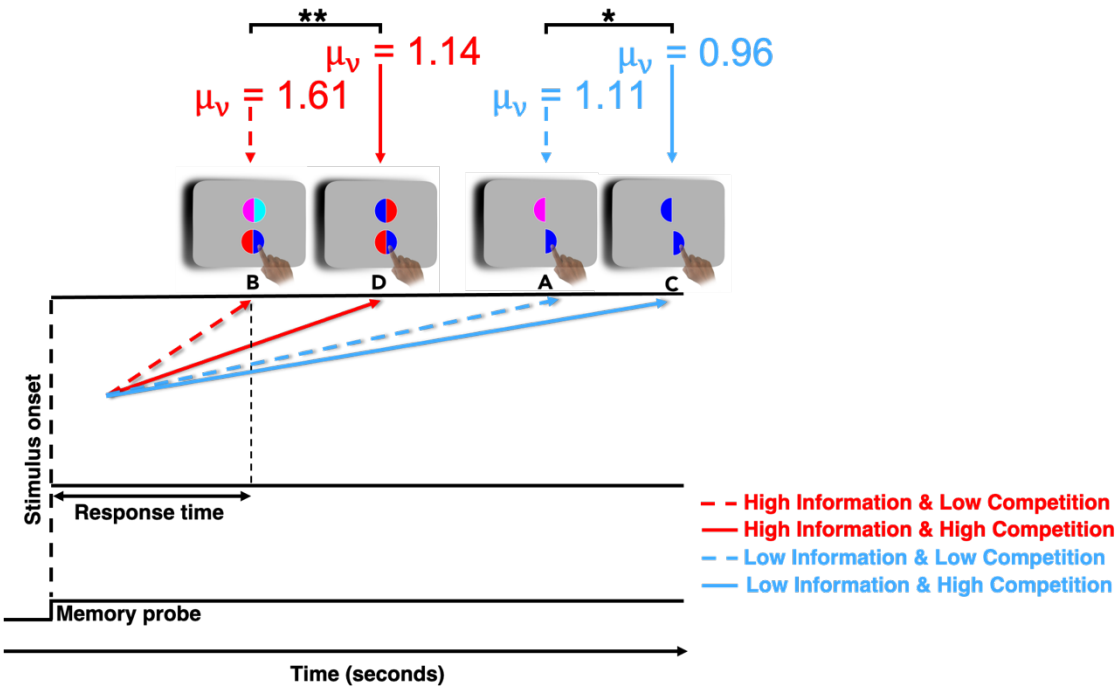


Figure 2.9| Effects of Information and Competition on drift rates for Model 2.

More information and lower competition at recall leads to higher drift rates allowing for the evidence to be accumulated quicker. Evidence is accumulated quicker in conditions where more retrieval cues are presented and when these cues don't share a lot of similarities. Post-hoc t-test were applied, * p-values < .05, ** p-values < .01.

Concurrently, lowering the threshold led to faster and less accurate responses (t(20) = 4.003, p < .001) because participants had a lower criterium to reach a decision i.e., they were willing to tolerate more errors when given more cues at retrieval under high competition trials (**Figure 2.9**). This was corroborated by the results from a 2 (information: low vs high) x 2 (competition: low vs high) on reaction time for inaccurate trials (**Figure 2.10**). There was no main effect of information [F(1, 15) = .071, p = .794, η_p^2= .005, BF_{10} = .256 , BF_{incl} = .256] but reaction time for inaccurate trials was significantly lower for high competition trials compared to low [F(1, 15) = .071, p = 14.403, η_p^2= .490, BF_{10} = 19.876, BF_{incl} = 14.668]. These two were not found to interact [F(1, 15) = 1.033, p = .325, η_p^2= .064, BF_{incl} = .381]. Further details can be found in **Appendix Tables 2.41 – 2.47**.

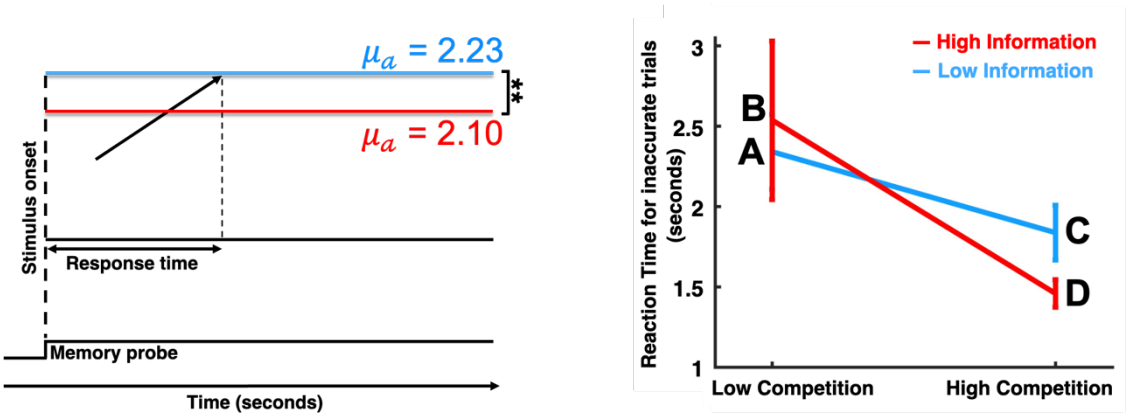


Figure 2.10| Effects of Information on threshold for Model 2.

More information leads to lower thresholds resulting in faster but less accurate responses. More information at recall leads to a smaller threshold boundary, thus participants make faster decisions at the detriment of accuracy, making faster mistakes. High competition trials (condition D) were shown to be more susceptible to noise compared to low competition trials (condition C). A paired sample t-test was applied, ** p-value < .01.

Thus, the most important findings revealed by applying HDDM to both identification accuracy and reaction times related to the effects on evidence accumulation as a function of both amount of information and competition whilst the effects on the decision threshold was only modulated by the amount of information.

2.4.1. Conclusion

Regarding our third question, on whether the decision process of recall was moderated by the amount of information and competition in the retrieval cue, the modelling approach provided evidence that the decision process of recall was differentially modulated by the amount of information held by the retrieval cue — by displaying either 'complete' targets composed of two semi-circles, or 'incomplete' targets composed of just a single semi-circle — and the amount of competition at retrieval, i.e., the level of similarity between cues — the foil either contained novel colours or had the same colours as the target but flipped in position.

In summary, the model demonstrates that evidence is accumulated towards the target more rapidly under conditions of high information compared to conditions of low information (Figure 2.9), i.e., the target was composed of two semi-circles instead of one (Figure 2.1). However, when the information carried by the target shared a lot of similarities with the foil (High competition, Figure 2.1) the rate of evidence accumulation was reduced possibly due to higher interference between memoranda (Figure 2.10) as shown by a significant interaction between information and competition on misbinding rates (Figure 2.5, Misbinding). When presented with high information trials,

manipulations of competition made little difference in participants' ability to bind the identity with the location of memoranda but, when presented with low information trials, misbinding rates were significantly increased under low competition compared to high competition (Figure 2.5, Misbinding).

Moreover, the model demonstrates that the amount of information held by the target affects the decision threshold, i.e., young adults have quicker inaccurate responses when presented with a target with two semi-circles instead of one as a consequence of less cautious responses. This is due to a lesser amount of evidence being required to trigger a decision threshold (Figure 2.10). Thus, in conditions with more competition at recall, participants do not benefit from more information because they are more susceptible to noise, i.e., they make mistakes due to speedy responses (Figure 2.10).

2.5. *Discussion*

Most research on the limitations of WM has largely focused on manipulations of encoding and maintenance periods, but the mechanisms underlying memory retrieval are yet to be fully understood. Using two behavioural experiments included in this Chapter, it was possible to gain better mechanistic insight into the process of memory retrieval from WM by investigating how the quality of recall depends on the amount and type of information presented at retrieval. Mixture modelling of error was also implemented to assess the fidelity of WM representation; and HDDM was used to measure how changes in retrieval cues influence the decision process of recall.

In Experiment 1, twenty-one healthy participants were tested on a novel paradigm aimed at answering three questions. First, whether more information in the foil at retrieval impacts recall. Behavioural results show that although varying the amount of information in the retrieval cue — by either displaying 'complete' memoranda composed of two semi-circles, or 'incomplete' items composed of a single semi-circle — had no effect on identification accuracy (**Figure 2.2, Identification Accuracy**). It did, however, augment the speed with which participants selected the correct item (**Figure 2.2, Reaction Time**) and improved their localisation performance (**Figure 2.2**, **Localisation error**).

This finding is consistent with the idea that performance is not only a reflection of the limitations imposed by encoding and storage (Tabi et al., 2019; Theeuwes et al., 2011). In fact, similar to the well known retro-cue effect (Griffin & Nobre, 2003; Kuo et al., 2012; Landman, Spekreijse, & Lamme, 2003; Souza S. et al., 2016)— where information presented after encoding can affect the probability of retrieval — these results suggest that the actual event of probing can be a retro-cue in itself. Thus, rather than acting as a form of interference and impairing memory, more information at retrieval can improve recall, specifically localisation performance, by serving as a more reliable retro-cue.

A different but not mutually exclusive hypothesis have been presented to explain the retro-cue effect (Souza & Oberauer, 2016). One of the propositions that fits well with the present results is based on the 'refreshing' hypothesis which states that a retro-cue strengthens the binding between the content (colours) and the context (location) by improving its accessibility (Loaiza & McCabe, 2012; Rerko & Oberauer, 2013; Rerko et al.,

2014). Even though it is unclear how pattern completion relates to the retro-cue hypothesis, similar to episodic memory, WM recollection might be dependent upon a pattern completion mechanism, which would explain the benefit of more information at recall since it enables the recovery of a complete stored representation based on a partial retrieval cue (Ally, Hussey, Ko, & Molitor, 2013; O'Reilly & McClelland, 1994). Notably, probing 'complete' memoranda was found to greatly facilitate access to its context (location), but not necessarily to its content, as no improvement of identification accuracy was observed. It may be the case that, as suggested by Wheeler and Treisman (2002), there is a memory mechanism characterised by the parallel representation of different levels of features, each with its own degree of accessibility.

The second question we sought to address with this experiment was whether the nature of the foil type at retrieval could influence recall depending on the level of similarities shared between the foil and the target. Evidence from the data presented here suggests that varying the amount of competition at recall — by either presenting a foil with novel colour or a foil with the same colours but flipped vertically in position — hindered identification accuracy (**Figure 2.2, Identification Accuracy**) as well as localisation performance (**Figure 2.2, Localisation error**). In accordance with the idea that forgetting from WM is due to interference between items and processing of distractors (Oberauer & Kliegl, 2006; Oberauer et al., 2012; Saito & Miyake, 2004), changing the way information had to be retrieved — by probing recall using different types of retrieval cues which can be perceived as a form of interference — while keeping encoding constant, induced a change in memory performance.

The third question was related to how manipulations of delay would modulate these effects. Longer delays of 4 seconds were found to worsen individuals' ability to locate memoranda (**Figure 2.2, Localisation error**) and led to slower reaction times (**Figure 2.2, Reaction Time**). In line with the hypothesis that a longer period of retention leads to poorer recall because time degrades the fidelity of mental representation (Barrouillet et al., 2012; J. Brown, 1958), the results show that memory resolution decreases with longer retention durations.

Intriguingly, participants' ability to correctly identify memoranda was found to benefit from 1 second delay maintenance intervals, but only under conditions of low competition. In Experiment 2, this was further explored by testing twenty young adults on a variant of this task, with increased number of trials per condition in order to model both the sources of errors contributing to performance and the decision process of recall. I aimed to answer two questions with the second experiment.

First, I wanted to explore how the nature of retrieval cues affects the pattern of recall errors. Results revealed a benefit from more information at the time of recall when identifying **(Figure 2.4, Identification Accuracy**) and localising memoranda (**Figure 2.4, Localisation error**). Importantly, more information in the retrieval cue improved identity-location binding as shown by a decrease in errors due to misbinding (**Figure 2.5, Misbinding**). The susceptibility of the object-location association to manipulations of amount of information is consistent with the idea that a 'complete' cue — a target containing both semi-circles — is a more reliable retro-cue, i.e., it corresponds to a higher number of pathways able to selectively access an object's characteristics. This mechanism of pattern completion is considered in the context of theory of hippocampal

function in episodic memory (Rolls, 2013), in which the hippocampal subfield CA3 system is thought to behave as an association network able to form links between spatial and object representations — and subsequently retrieve the completion of a whole memory from a partial cue. The hippocampus is now also implicated in WM performance (Pertzov, Miller, et al., 2013).

Second, conditions where participants performed less accurately were also paradoxically slower (Condition C: Low Information & High Competition, **Figure 2.1**), suggesting a violation of the speed-accuracy trade-off. This was further explored by implementing HDDM to both accuracy and reaction time data to measure the way in which the decision process of recall was modulated by the amount of information and competition in the retrieval cue. Results show that the recall process is differentially impacted by the amount of information (**Figure 2.9**) and competition (**Figure 2.10**) held by the retrieval cues, i.e., more information allows for evidence to be accumulated faster towards the decision threshold, but when the information carried by the target shares a lot of similarities with the foil, this rate of evidence accumulation is reduced. Concurrently, more information carried by the target lowers the decision threshold, leading to quicker inaccurate responses, making participants more susceptible to noise.

These results are consistent with predictions from a computational neural model proposed by Manohar and colleagues (Manohar et al., 2017). The model predicts that a single feature presented at retrieval can trigger pattern completion by activating the rest of the features from its joined representation. It also predicts that when competing information is presented at recall, retrieval is hindered as information activates multiple conjunction units (combinations of features represented as bound objects). In fact, the

reduction of the rate of accumulation when the information carried by the target shares a lot of similarities with the foil is consistent with the notion that memory performance is limited by interference, which can arise from multiple sources (Lewandowsky et al., 2010; Oberauer et al., 2012; Saito & Miyake, 2004): one being the superposition of overlapping memory representations, blurring each other; the other, the increase of confusion between target and foil because they share similar characteristics (Oberauer et al., 2012). This aligns well with Souza et. al (2016) findings on the benefit of delaying response selection for at least .5s to overcome visual interference and incomplete retrieval.

It is important to note that some characteristics of the study design used in this Chapter could impact the interpretation of the findings. Notably, a complementary angle may be that conditions of low competition vs. high competition test two different forms of memory, with the former requiring mere recognition of a memorized colour feature, while the latter requires recall of the spatial arrangement of the probed colour. It would be pertinent to add a control condition by presenting a dual choice between a full circle with a single colour and semi-circle with no colour (either the right-side or the left-side of a circle) to investigate if participants are just as quick at identifying memoranda based on its colour vs shape, making sure there is no object bias. Additionally, the fundamental mechanisms underlying the results from Experiment 1 arising from a 4s maintenance delay might be profitably explored in a future study.

In conclusion, the findings presented here suggest that the amount and type of information displayed at recall can significantly affect memory performance, and instead of taxing memory, more information at retrieval improves access to items in WM.

One important question that is left unanswered is how the foil itself drives WM selection when participants are presented with a dual choice between a target and a foil at the moment of recall. In Chapter 3, I consider WM driven selection in the context of visual search and binding.

Chapter 3. Successful recall relies on the information carried by the foil, not just the target

Abstract

Results from Chapter 2 suggest that recall from WM may be reliant on information associated with the foil presented at retrieval, not just the target. To test this hypothesis, here a similar experimental design was implemented. But instead of manipulating the nature of both retrieval cues, the focus was on investigating the impact of the quality of the information carried by the foil on memory performance.

At retrieval, when a choice had to be made between the target and a foil, the quality of information associated with the foil may vary depending on how helpful this information is — if at all — to the process of recalling the target. Across two experiments, the quality of the information in the foil was varied in two ways. First, we manipulated the *number of features* from the original memory array in the foil. Second, the *congruency of these features* with respect to the original memory array, i.e., spatial arrangement of colours within an object, was varied.

Results showed that presenting a foil with two features from the original memory display significantly hindered participants' ability to identify the target, leading to longer reactions times, but it had no detrimental effect on their localisation performance. However, congruency of the features in the foil with respect to those in the original memory actually had a beneficial impact on identification accuracy in young adults, although their ability to localise the correct memoranda was impaired when an incongruent foil was presented at retrieval. This detrimental effect was found to be specifically attributable to a greater number of errors due to misbinding, i.e., an increase in the inability to bind a target's features with their location.

In the second part of this study, the flexibility of decision making at retrieval was quantified using HDDM. This provided insights into how the decision process of retrieval is affected by the quality of evidence carried by the foil. Increasing the amount of information matching the memory array impaired recall by decreasing the rate of evidence accumulation towards the target. Concurrently, presenting a target together with a congruent foil — a foil with the same colour-position arrangement as in the memory array — led to poorer identification recall due to slower accumulation of evidence for the correct option.

In conclusion, these findings suggest that rather than selecting the correct memoranda solely based on the information held by the target, young adults also rely on information presented in a foil at retrieval. Furthermore, the arrangement of features in the foil are associated with successful binding of a target's identity with its location.

3.1. *Introduction*

The results in the previous chapter showed that the nature of retrieval cues affects memory performance, with more information presented at retrieval improving recall, possibly by facilitating associative retrieval mechanisms. Additionally, presenting a target together with a competing foil — a flipped version of the target — at retrieval worsened memory performance. This suggests that the nature of the foil, not just the target, may play a role in the recall decision process. A critical question left unanswered is whether the impact of the quality of the information carried by the foil can modulate memory performance.

To address this question, in the first part of this study, a delayed reproduction task with a continuous measure of report was implemented following the same design and analysis rationale employed in Chapter 1. Here, as previously, participants were instructed to remember a target and a non-target presented in a memory array. After a short delay, a choice between the target and a foil was displayed at recall. Participants first had to select the target and then drag it to its original location on the touchscreen. The effects of the nature of the information carried by the foil were investigated across two manipulations of complexity at retrieval: the *number of features* from the memory array *held by the foil* and the *congruency of these features* with the original memory array, i.e., the arrangement of the colours within the object. In the second part of this study, the flexibility of decision making of WM representation was quantified by employing HDDM. This approach provides insights into how the decision process at retrieval is affected by the nature of the evidence presented in the foil.

Participants' past experience remembering or interacting with a specific stimulus affects current visual processing (Desimore & Duncan, 1995; Olivers, Peters, Houtkamp, & Roelfsema, 2011; Pashler & Shiu, 1999). For example, the process of finding a target among distractors, also referred to as visual search, is slower when a distractor matches the information that is currently maintained in WM regarding the target (Moores, Laiti, & Chelazzi, 2003; Olivers, Meijer, & Theeuwes, 2006; Pashler & Shiu, 1999). In fact, the interactions between WM and visual search are so extensive that they have been posited to rely on shared mechanisms (Bocincova, Olivers, Stokes, & Manohar, 2020; Emrich, Al-Aidroos, Pratt, & Ferber, 2009; Mayer et al., 2007). It is possible that when participants are asked to select a memory item from an array of distractors, as is the case in a dual

choice WM paradigm, the process of target selection could be guided by similar principles as during visual search.

The efficiency of visual search is affected by the number of shared similarities between target and distractors (Bauer, Jolicoeur, & Cowan, 1996; Duncan & Humphreys, 1989; Pashler, 1987). Efficient search, also known as disjunctive or feature search, is fast and unaffected by the number of distractors (Mcelree & Carrasco, 1999), whereas non-efficient search, also called conjunction or serial search, is characterized by slower reaction times and is dependent on the number of distractors (Mcelree & Carrasco, 1999; Shen, Reingold, & Pomplun, 2003) (for other, non-attentional factors affecting visual search see (Eckstein, 2011)). These effects have been well documented when people search for a target item (held in WM) among many distractors.

Here, we investigated what happens to WM performance when a target held in WM is presented with a single foil at retrieval that varies with respect to specific parameters. To examine the impact of the number of features shared between a distractor and a target during retrieval in the current design we first manipulated the *number of features* from the original memory array that appeared *in the foil*. Specifically, a foil could hold either a single feature from the non-target item in the memory array, or one feature from the non-target and one feature from the target. We hypothesized that, because more unique targets are detected faster during visual search, excluding the target colour from the foil — by presenting a foil with one feature from the non-target in the memory array — would lead to more efficient recall decisions compared to when two features from the memory array were included in the foil (one colour from the target and one colour from the non-target).

Additionally, previous literature suggests that visual search is affected by contextual information (Chun & Jiang, 1998; Geyer, Shi, & Müller, 2010; Hout & Goldinger, 2010). For instance, finding a specific object in an array of other objects is facilitated when participants store the location of a distractor, as it restricts their attention to the relevant locations in space and therefore allows for rejection of non-targets efficiently (Hout & Goldinger, 2010). Location has also been shown to have a special role in WM. In fact, research on feature binding has shown that non-spatial features, e.g. colour, are associated through their shared location (Schneegans & Bays, 2018) and that the latter is remembered even if unnecessary for recall (A. Treisman & Zhang, 2006), almost in an obligatory manner.

Recently, more studies have implemented delayed reproduction tasks with a continuous measure of report, making it possible to distinguish misbinding errors — memory errors that arise from misreporting a feature belonging to another item presented in the memory array — from other types of errors. Some studies are consistent with the notion that non-spatial features of an object are bound to each other only indirectly via their common location. They show that when the target item is cued by other features, such as colour, participants tend to not just report the location of another item in memory but also *other features of the non-target* item at the reported location (Kovacs & Harris, 2019; Schneegans & Bays, 2017). In contrast, many have consistently shown that when the target is cued by its location, errors in reporting colour or orientation occur independently, i.e., the failure to remember one feature of an object is not necessarily accompanied by the failure to remember other features belonging to the

same object (Paul M. Bays et al., 2011; Fougnie & Alvarez, 2011; Fougnie, Cormiea, & Alvarez, 2013).

In line with the pivotal role of location described above, we hypothesized that the congruency of a retrieval cue — the spatial arrangement of colours within an object — would influence memory performance. To test whether the success of associative retrieval is reliant on the colour-position arrangement of an object's features, the *congruency of the features* carried by the foil with the original memory array is also explored. This second manipulation of the foil complexity entails presenting either congruent trials — trials where the foil contains features in their original intra-dimensional binding, i.e., the binding between position and colour is the same as in the memory array — or incongruent trials —where binding is broken by flipping the colours' position.

3.2. *Experiment 1*

The first study investigated the effect of foil complexity on the quality of WM recall. The key questions I sought to answer were:

1. What is the impact of presenting a *foil with different numbers of features from the memory array* (either a single feature from non-target or one feature from non-target combined with one feature from target) on the quality of recall?
2. Does the *colour-position arrangement of the foil's features* affect recall? We examined whether performance was affected by whether the foil contains

features that are spatially congruent (features in their original position: e.g., yellow on the left and pink on the right) or incongruent (features in their flipped position: e.g., green on the left and pink on the right) with an item presented in the memory array (for example: an item containing yellow on the left and green on the right)?

3.2.1. Methods

3.2.1.1. Participants and Consent

Thirty-five young adults (24 female) aged between 18 and 30 years old (mean age = 23.51, SD = 3.37) were included in this study. Participants were recruited from an online database, the Oxford Psychology Research Participant Recruitment Scheme on SONA. All had no history of neurological or psychiatric disease, good vision, and were screened for depression with the BDI-II (A.T. Beck et al., 1996). Two participants were removed from the analysis after their data was collected due to having a total score of 28 and 19 in BDI-II, classified as moderate and borderline clinical depression, respectively. A third participant was removed from the analysis for taking antidepressants (*Mirtazapin*). All were reimbursed for their time (£5) and transport costs. Ethical approval was obtained by the local ethics committee and written informed consent was given by all subjects.

3.2.1.2. Stimuli

Stimuli were the same as in Experiment 1 of Chapter 2.

3.2.1.3. Design

A delayed reproduction task was used to assess the quality of recall by asking participants to identify (identity identification) and reproduce the exact location (location identification) of memoranda (Figure 3.1). In this paradigm, two circles (a target and a non-target), each divided in two semi-circles of different colours, were randomly presented in a memory array, and displayed for 2 seconds. Following a 1-second-long blank screen, a dual choice between a target (the item presented in the memory array) and a foil (a distractor item that had not appeared in the memory array but contained some features of the memoranda presented at encoding) was shown. Participants first had to select the target and then drag it to its remembered location on the touchscreen. They signalled when they finalised the reproduction of the location by pressing the 'Done' button at the bottom of the screen. No feedback was given.

While keeping encoding constant, two factors were manipulated at retrieval by varying the nature of the foil type. First, the amount of information from the original array contained in the foil was altered by varying how many colours from the memory array were contained in the foil at retrieval (1 or 2 features). Second, we varied whether those colours were presented in their original position (congruent), or the incorrect position (incongruent) (Figure 3.1).

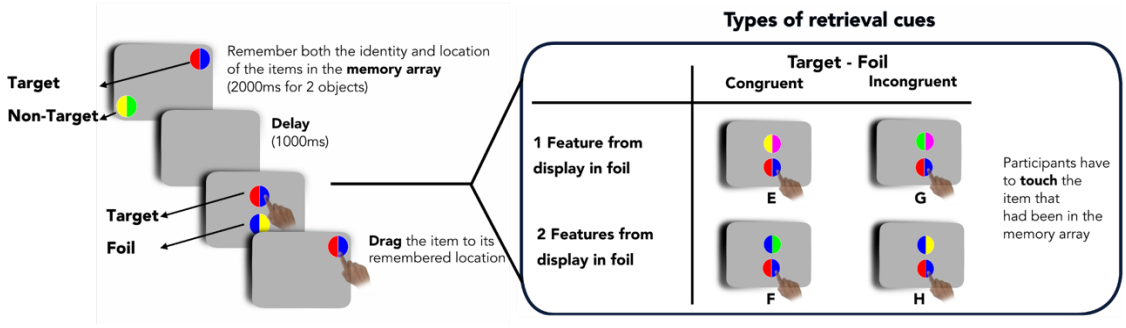


Figure 3.1| Paradigm used in Experiment 1.

Encoding was kept constant while retrieval was manipulated in two main ways. First, by varying the number of features from the memory array held by the foil (one or two features, Condition E and Condition F, respectively). Second, by varying the congruency of the features carried by the foil (congruent or incongruent, Condition G and Condition H, respectively), i.e., the retrieval foil could either have a feature with the same colour-position arrangement as in the memory array (congruent) or flipped (incongruent).

The task consisted of four experimental conditions (Figure 3.1) randomly presented within 1 Block of 24 trials per condition. In all four conditions, the foil was a circle consisting of two semi-circles, one of the left and the other on the right, but the exact constituents of the foil varied:

(E) One congruent feature from memory array in foil: In this condition, the foil consisted of a semi-circle with one colour that had appeared in the *non-target item* presented in the memory array, while the other semi-circle was filled with a colour that had *not appeared* in the memory array. Further, the semi-circle with the same colour as the non-target was *congruent*, i.e., occupied the same half of the circle as in the original memory array, thus sharing the same colour-position arrangement as the non-target (Figure 3.1, Condition E).

(F) Two congruent features from memory array in foil: In this condition, the foil consisted of *two semi-circles,* each with a colour that had appeared in the items in the

memory array: one colour from the target item and one from the non-target. Additionally, the semi-circle with the same colour as the non-target was *congruent*, i.e., occupied the same half of the circle to its position in the original memory array, thus sharing the same colour-position arrangement as the non-target (Figure 3.1, Condition F).

(G) One incongruent feature from memory array in foil: Here, the foil consisted of *one semi-circle* with a colour that had appeared in the non-target item in the memory array, and the other half of the circle filled with a colour that had *not appeared* in the memory array. Additionally, the semi-circle with the same colour as the non-target was incongruent, i.e., occupied the opposite half of the circle to its position in the original memory array (Figure 3.1, Condition G).

(H) Two incongruent features from memory array in foil: The foil consisted of *two semi-circles* with colours that had appeared in the items presented in the memory array: one colour from the target and one colour from the non-target. Additionally, the semi-circle with the same colour as the non-target was in*congruent*, i.e., occupied the opposite half of the circle to its position in the original memory array (Figure 3.1, Condition H).

3.2.1.4. Procedure

The session took half an hour to complete. The task consisted of four experimental conditions (Figure 3.1). Participants completed 4 practice trials and 24 randomized trials per condition on an interactive touch-sensitive screen (iPad version 9.3.5 (13G36), model MGTX2B/A) with 1536x2048 pixel matrix.

Data handling and statistical analysis were performed in *MATLAB* (The Math-Works inc., version 2018a) and *JASP* (JASP, version 0.12.1).

3.2.2. Behavioural Results

I started by testing whether the number of features from the memory array held by the foil — a single feature from non-target or one feature from non-target combined with one feature from target — and foil congruency — whether presenting these colours in their original arrangement or flipped in position (congruent or incongruent) — affected recall.

Therefore, a 2 x 2 repeated measures ANOVA with number of features (1 feature vs 2 features) and foil congruency (congruent vs incongruent) as within-subjects factors, was conducted on *identification accuracy*, i.e., the proportion of time the target was selected; *localisation error*, i.e., the distance (in pixels) between the target reported location and the original target location; and *reaction time, the time* (in seconds) taken to correctly identify the target (Figure 3.2). Significant interactions were unpacked with *Holm* corrected pairwise comparisons with a 95.0% confidence interval and results were considered significant if their p-value was less than .05.

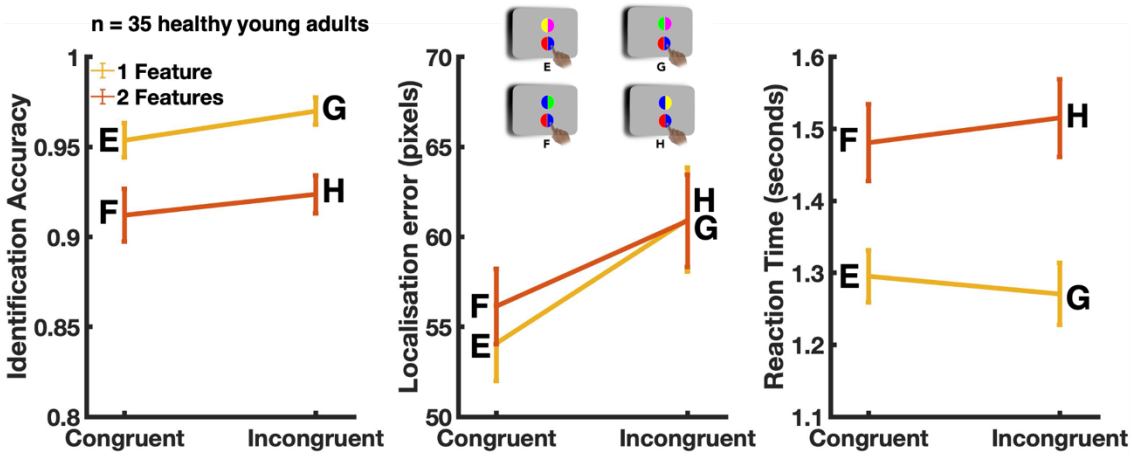


Figure 3.2| Behavioural results for Experiment 1.

Increasing the number of features held by the foil from the memory array led to worse identification performance and slower reaction times while presenting an incongruent foil hindered localisation performance. Error bars reflect the standard error of the mean (SEM) and were calculated using the Loftus & Masson method (Loftus & Masson, 1994).

The ***number of features*** from the original memory array presented as part of the foil (1 feature or 2 features) significantly affected participants' ability to select the correct memoranda. They had lower identification accuracy when presented with a foil with more information from the memory array – two features compared to one [F(1, 34) = 26.101, $p < .001$, $\eta_p^2 = .434$, $BF_{10} = 3.595e^4$, $BF_{\text{incl}} = 2.822e^4$] (Figure 3.2, Identification Accuracy). Also, on trials where the foil shared more similarity (more features) with the memory array, participants were slower to select the target, as per a main effect of number of features on reaction time [F (1, 34) = 45.947, $p < .001$, $\eta_p^2 = .575$, $BF_{10} = 2.444e^8$, $BF_{\text{incl}} = 1.720e^8$] (Figure 3.2, Reaction Time). However, this manipulation of the foil did not exert a cost on localisation error [F(1, 35) = .224, $p = .6391$, $\eta_p^2 = .007$, $BF_{10} = .196$, $BF_{\text{incl}} = .168$] (Figure 3.2, Localisation error).

Presenting features from the original array as part of the foil in a ***congruent or incongruent*** manner, i.e., in their original colour-position arrangement or the flipped position, did not affect participants' ability to correctly identity the target [F(1, 34) = 2.763, p = .106, η_p^2 = .075, BF_{10} = .432, BF_{incl} = .475] nor was there an interaction between congruency and amount of information [F(1, 34) = .085, p = .773, η_p^2 = .002, BF_{incl} = .382] (**Figure 3.2, Identification Accuracy**). Likewise, reaction time was also not affected by the congruency of the foil [F (1, 34) = .063, p = .803, η_p^2 = .002, BF_{10} = .182, BF_{incl} = .165] nor was there an interaction between foil congruency and number of features from the array on a distracter [F (1, 34) = 1.502, p = .229, η_p^2 = .042, BF_{incl} = .244] (**Figure 3.2, Reaction Time**). Yet, young adults were significantly worse in their ability to remember the correct location of the target when presented with an incongruent foil compared to when the foil was congruent, as denoted by a main effect of congruency on localisation error [F(1,34) = 13.781, p < .001, η_p^2 = .288, BF_{10} = 85.878, BF_{incl} = 59.977]. The effect of congruency on localisation error did not significantly change as a function of number of features [F(1, 34) = .244, p = .625, η_p^2 = .007, BF_{incl} = .180] (**Figure 3.2, Localisation error**). See **Appendix Table 3.1 – 3.6** for details.

3.2.2.1. Conclusion

This novel WM task provides evidence that the nature of the retrieval foil, not just the target, modulates the ability to accurately recall memoranda. Presenting a foil with two features from the memory array significantly hindered participants' ability to identify the target, leading to longer reactions times, but it had no detrimental effect on their

ability to localize the original position of the target. This suggest that, rather than selecting the correct memoranda solely based on the information held by the target, young adults seem to also rely on the information carried by the foil.

Additionally, when presented with a dual choice at retrieval, young adults' localisation performance, but not identification accuracy, was dependent on whether the target was presented together with a congruent foil or an incongruent foil. Participants made more localisation errors when the foil was incongruent, i.e., when the foil was composed of colours that occupied the opposite half of the circle compared to their arrangement in the original memory array. This suggests that the arrangement of non-spatial features in the foil also affects successful retrieval of a target's location. If this is the case, it is likely that congruency of the foil may affect the binding of the target's identity with its location by modulating misbinding. Therefore, a second experiment with an increased number of trials further explored these results.

3.3. *Experiment 2*

The second study also investigated the effect of foil complexity on the quality of recall but this time with an increased number of trials (100 trials per condition) to allow for modelling of sources of error and perform HDDM. A single delay period and the same two experimental manipulations of number of features and congruency were used.

This second study sought to answer the following questions:

1. How does the nature of the foil impact the pattern of recall errors?

2. Is the decision process of recall hindered by the amount of information coming from the memory array and carried by the foil?
3. How is the decision process of recall affected when breaking the colour-position arrangement of the non-spatial features held by the foil?

3.3.1. Methods

3.3.1.1. Participants

Twenty young adults, that did not take part in Experiment 1, aged between 18 and 33 years old (mean age = 24.55, SD = 4.06, 15 females), took part in Experiment 2. All participants had normal or corrected to normal vision at the time of testing. They were recruited from SONA, a volunteer database, and screened for depression with the BDI-II (A.T. Beck et al., 1996) and any history of neurological or psychiatric conditions. All were reimbursed for their time (£10) and transport costs. The study was approved and consistent with all relevant local regulations, with participants giving their written informed consent.

3.3.1.2. Stimuli, Design and Procedure

Stimuli, design and procedure were the same as in Experiment 1, except that participants performed 1 block of 100 trials per condition. This enabled to deconstruct the three different sources of error contributing to the pattern of memory performance: imprecision, misbinding and guessing. Data analysis was performed using the *Matlab* package *MemToolBox2D* (Grogan et al., 2019). Additionally, reaction times were

modelled to better understand the underlying mechanisms of the decision process of recall using HDDM (Heathcote et al., 2019; Singmann et al., 2019).

3.3.2. Mixture Modelling Results

The behavioural results from Experiment 1 were largely replicated using a 2 x 2 repeated measures ANOVA, with number of features from the memory held by the foil (1 feature vs 2 features) and congruency of these features with the memory array (congruent vs incongruent) as within-subjects factors, on identification accuracy, localisation error and reaction time (Figure 3.3).

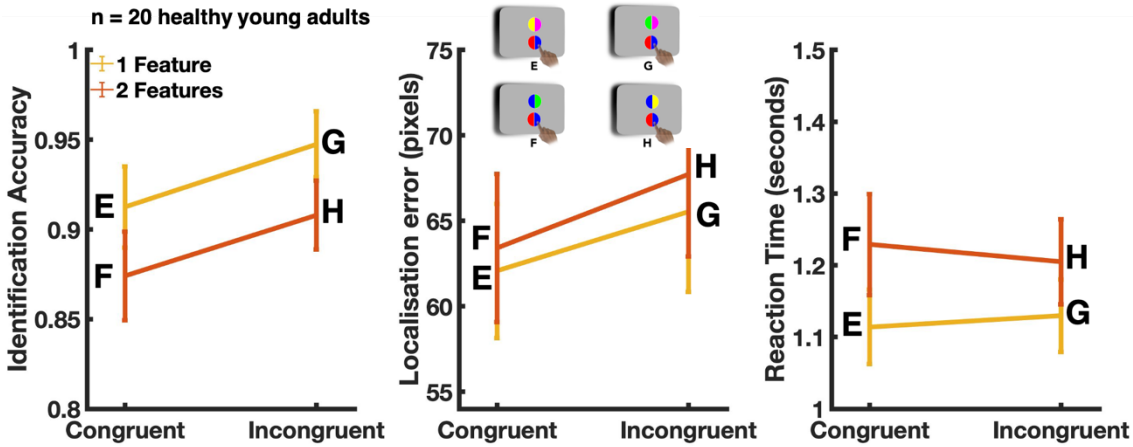


Figure 3.3| Behavioural results for Experiment 2.

Results from Experiment 1 were largely replicated. Presenting a foil with two features from the memory array led to worse identification performance and slower reaction times while, unlike in the previous experiment, presenting an incongruent foil not only hindered localisation performance but also identification accuracy. Error bars reflect SEM and were calculated using the Loftus & Masson method (Loftus & Masson, 1994).

As in Experiment 1, the ***number of features*** from the memory array held by the foil (1 feature or 2 features) significantly affected identification accuracy. Participants' ability to correctly identify the target was hindered when the foil carried two features from the memory array compared to one [F(1, 19) = 11.472, p = .003, η_p^2 = .376, BF_{10} = 15.621,

BF_{incl} = 22.286] (**Figure 3.3, Identification Accuracy**). It also increased their reaction time [F(1, 19) = 16.736, $p < .001$, η_p^2 = .468, BF_{10} = 3268.630, BF_{incl} = 2420.380] (**Figure 3.3, Reaction Time**) but did not significantly affect localisation performance [F(1, 19) = 1.169, p = .293, η_p^2 = .058, BF_{10} = .418, BF_{incl} = 3.216] (**Figure 3.3, Localisation error**).

Unlike Experiment 1, the ***congruency*** of the features held by the foil significantly impacted identification accuracy [F(1, 19) = 5.697, p = .028, η_p^2 = .231, BF_{10} = 5.765, BF_{incl} = 8.676] (**Figure 3.3, Identification Accuracy**). Thus, when the colour-position arrangement of the non-spatial features belonging to the foil was not the same as in the memory array (incongruent trials), participants' ability to correctly identify the target was hindered. Conversely, there was no main effect of foil congruency on reaction times [F(1, 19) = .076, p = .785, η_p^2 = .004, BF_{10} = .232, BF_{incl} = .246] nor an interaction between foil congruency and number of features from memory array on the foil [F(1, 19) = 1.320, p = .265, η_p^2 = .065, BF_{incl} = .358] (**Figure 3.3, Reaction Time**), replicating the results from Experiment 1. Likewise, localisation error was also significantly affected by foil congruency, i.e., presenting an incongruent foil resulted in poorer localisation performance [F(1, 19) = 5.993, p = .024, η_p^2 = .240, BF_{10} = 4.389, BF_{incl} = 3.216], but foil congruency and number of features were not found to interact [F(1, 19) = .140, p = .712, η_p^2 = .007, BF_{incl} = .308] (**Figure 3.3, Localisation error**). See **Appendix Table 3.7 – 3.12**).

Following initial investigations, the mixture model analysis was implemented as in Chapter 2, Experiment 2. The goal of this analysis was to deconstruct the contribution of different sources of error to responses: imprecision, misbinding and guessing (Paul M. Bays et al., 2009; Grogan et al., 2019). A 2 x 2 repeated measures ANOVA with number of features (1 feature vs 2 features) and foil congruency (congruent vs incongruent) as

within-subjects factors was conducted on imprecision, proportion of errors due to misbinding and guessing (**Figure 3.4**).

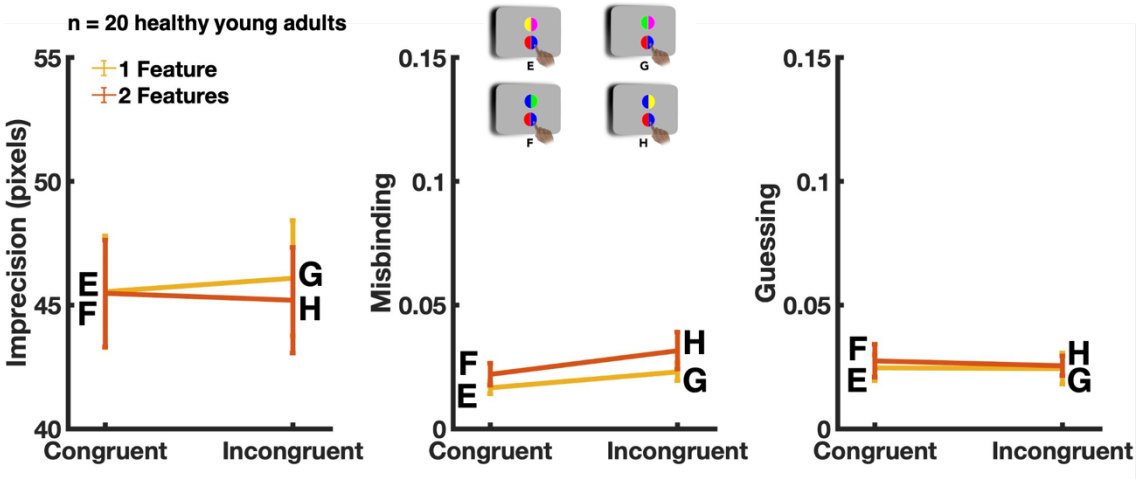


Figure 3.4| Mixture Model Results for Experiment 2.

Manipulations of the number of features in the foil and congruency with the memory display did not affect imprecision, nor proportion of errors due to guessing. Congruency of the foil did, however, affect the proportion of errors due to misbinding, with incongruent trials leading to greater misbinding. Error bars reflect the standard error of the mean (SEM) and were calculated using the Loftus & Masson method (Loftus & Masson, 1994).

The ***number of features*** from the memory array carried by the foil had no significant impact on imprecision [F(1, 19) = .398, p = .536, η_p^2 = .021, BF_{10} = .296, BF_{incl} = .214] (**Figure 3.4, Imprecision**). It also did not significantly affect errors due to misbinding, i.e., participants' ability to bind the identity of the target memoranda with its location was not affected by the colour-position arrangement of the non-spatial features belonging to the foil [F(1, 19) = 2.149, p = .159, η_p^2 = .102, BF_{10} = 1.016, BF_{incl} = .916] (**Figure 3.4, Misbinding**). Likewise, presenting a foil with more features from the memory array did not significantly affect the proportion of responses in which participants randomly located the target, as per no main effect of number of features on guessing [F(1, 19) = .259, p = .617, η_p^2 = .013, BF_{10} = .270, BF_{incl} = .193] (**Figure 3.4, Guessing**).

The ***congruency*** of the foil did not affect imprecision [F(1, 19) = .100, p = .756, η_p^2 = .005, BF_{10} = .228, BF_{incl} = .168] and this was not modulated by the number of features from the memory array held by the foil [F(1, 19) = .402, p = .534, η_p^2 = .021, BF_{incl} = .066] (**Figure 3.4, Imprecision**). But the colour-position arrangement of the features carried by the foil did affect the proportion of errors due to misbinding, with incongruent trials leading to significantly greater misbinding [F(1, 19) = 5.418, p = .031, η_p^2 = .222, BF_{10} = 1.674, BF_{incl} = 1.433] (**Figure 3.4, Misbinding**). The effect of congruency on the proportion of errors due to misbinding was not modulated by the amount of information held by the foil, as per no interaction between the two [F(1, 19) = .433, p = .518, η_p^2 = .022, BF_{incl} = .471] (**Figure 3.4, Misbinding**). Additionally, there was no significant main effect of guessing [F(1, 19) = .201, p = .659, η_p^2 = .010, BF_{10} = .244, BF_{incl} = .175] nor an interaction effect between the number of features and congruency of the foil on errors due to guessing [F(1, 19) = .067, p = .798, η_p^2 = .004, BF_{incl} = .052] (**Figure 3.4, Guessing**). Further information in **Appendix Table 3.13 – 3.18**.

In summary, the most important finding revealed by modelling the sources of error pertained to the effects on misbinding identity with location as a function of congruency of the foil. Presenting incongruent trials — where the colour-position arrangement of the non-spatial features carried by the foil was broken— was found to lead to an increased inability to correctly associate the identity of the target memoranda with its location.

3.3.2.1. Conclusion

The second experiment provides further evidence that the nature of the foil affects memory retrieval. Manipulations of the ***number of features*** from the memory array in the foil not only hindered accuracy of recall, but also increased reaction times. However, they did not affect localisation performance or memory resolution.

Manipulations of ***foil complexity*** had a detrimental impact on identification accuracy. Presenting a foil with features with the same colour-position arrangement as in the memory array led to poorer identification accuracy yet did not affect participants' reaction time. In contrast, presenting an incongruent foil at retrieval — where the colour-position arrangement of the foil's features was not the same as in the memory array — resulted in worse localisation performance. When deconstructing the different sources of error contributing to the pattern of localisation performance, errors due to misbinding were found to be disproportionally higher when this arrangement between colour and position was broken. Neither imprecision nor guessing were affected.

In summary, the colour-position arrangement in the foil had an impact on participants' ability to correctly identify the target. Importantly, it hindered localisation performance, which is specifically attributable to an increased number of errors due to misbinding — the inability to correctly bind the target with its location. Of course, these findings about the impact of the foil also suggest that the encoding of the non-target must also be important to these effects, depending on the nature of the foil and its similarity to the non-target.

Taken together, these results suggest that identification accuracy at the time of retrieval is affected when the foil matches the information that is currently maintained in WM. Moreover, localisation performance of a target is affected by the arrangement of non-spatial features (in this case, colour) in the foil. Participants reported the location of one of these non-spatial features from the foil significantly more often when their arrangement was incongruent with what was previously encoded, despite being irrelevant for the task at hand.

3.4. *Hierarchical Bayesian Drift Diffusion Modelling*

Analogously to Chapter 2, an HDDM was applied (**Appendix Chapter 3, Experiment 2 (HDDM)**) using the open-source, R-based DMC software together with the *rdists* Package (Heathcote et al., 2019; Singmann et al., 2019), v. 0.8-3.

Three hypotheses were explored to understand how the mechanisms underlying the decision process of recall are affected by the number of features from the memory array included in the foil (1 vs 2 features) and the congruency of these features with the original memory display (congruent vs incongruent).

Hypothesis 1: A foil with two features from the memory array hinders performance by decreasing the rate of evidence accumulation towards the target. Additionally, when the features held by the foil are presented with the same colour-position arrangement as in the memory array, the rate of evidence accumulation is slower due to higher interference with the target compared to when the features are incongruent. Simultaneously, increasing the number of features conveyed by the foil leads to noisier

answers due to faster, incorrect responses, reflecting a lower threshold (Model 1: *drift rate* modulated by number of features & congruency and *threshold* modulated by number of features).

Hypothesis 2: A foil with two features from the memory array hampers participants' performance by decreasing the rate of evidence accumulation towards the target. Additionally, when the features held by the foil are presented with the same colour-position arrangement as in the memory array, the rate of evidence accumulation is slower due to higher interference with the target. Simultaneously, increasing the degree of similarity between the foil and the memoranda presented in the memory array (presenting a congruent foil, i.e., a foil with the same colour-position arrangement as the features presented at encoding) leads to noisier, faster incorrect responses, reflecting a lower threshold (Model 2: *drift rate* modulated by number of features & congruency and *threshold* modulated by congruency).

Hypothesis 3: The rate of evidence accumulation decreases with both the number of features carried by the foil and its congruency, i.e., drift rate is slower when the foil holds two features from the encoding phase, and these features are in the same colour-position arrangement as they were in the memory array. Simultaneously, the decision threshold is negatively affected by both number of features and congruency as a consequence of less cautious responses: faster incorrect responses when participants are presented with a foil with features in the same position as the ones they were asked to encode (Model 3: both *drift rate* and *threshold* modulated by number of features and congruency).

Model comparison was carried out using BPIC and DIC (Spiegelhalter et al., 2002) (**Figure 3.5**). The best fitting model (**Figure 3.6**) determined by the lowest deviance criterion (DIC = 10075.509) was Model 2, suggesting that the *drift rate* was modulated by *both* number of *features and congruency*, while *threshold* was solely influenced by the *congruency* of the retrieval cue. The parameter that determined the winning model was threshold, which was modulated by congruency of the foil in Model 2, whereas it was modulated by number of features in Model 1 (DIC = 10090.354) and by both number of features and congruency in Model 3 (DIC= 10085.082).

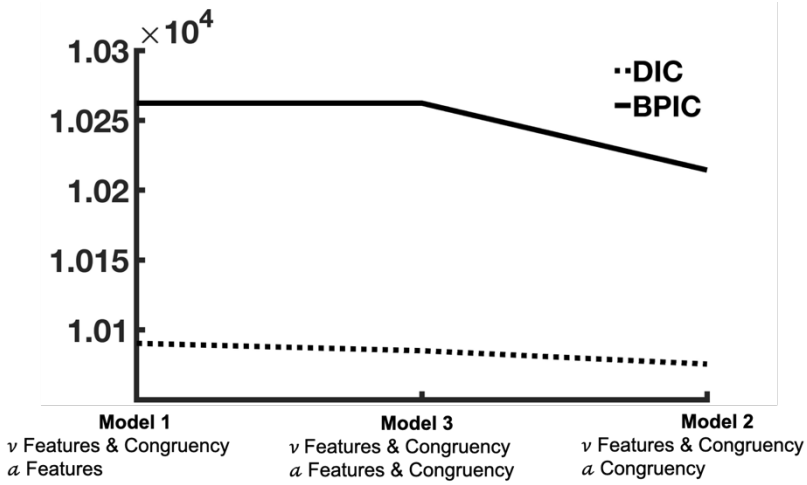


Figure 3.5| Best Model Fit.

The model that best fits the data is Model 2, where the drift rate is modulated by both number of features and congruency together with the threshold being modulated solely by congruency.

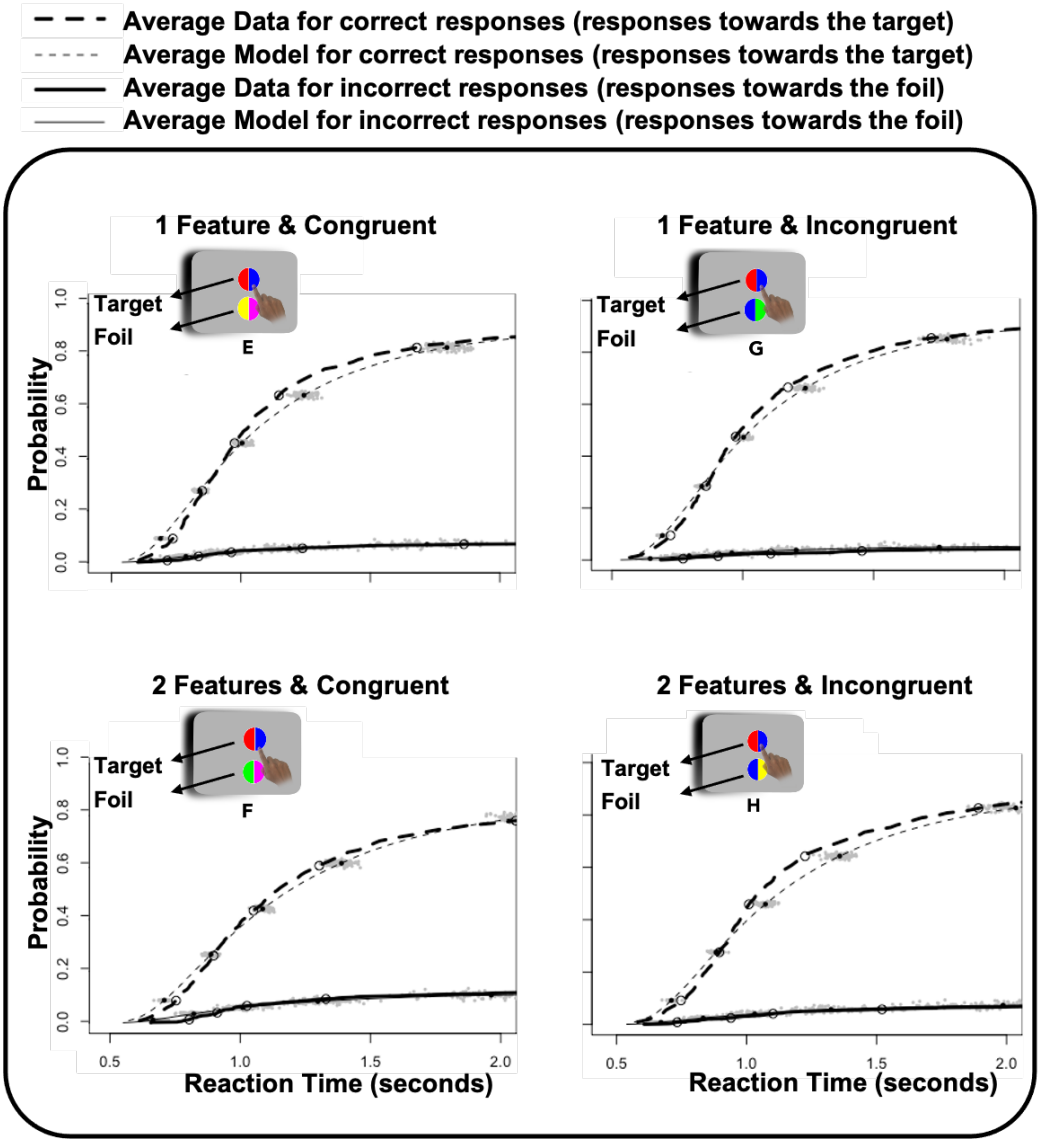


Figure 3.6| Behavioural data vs Model 2.

The average behavioural data is plotted against the average data simulated by the model for both the reaction time (seconds) for correct responses (trials where participants selected the target) and incorrect responses (trials where participants selected the distractor).

The winning model, Model 2 (Figure 3.6), was further explored by conducting a 2 (Number of features from the memory array held by the foil: 1 feature vs 2 features) x 2 (the congruency of these features with the memory array: congruent vs incongruent) ANOVA on drift rate and a paired sample t-test on threshold.

Presenting a foil with one feature from the memory array led to higher drift rates, as shown by a main effect of information [F(1,19) = 47.678 , p < .001, η_p^2 = .715, BF_{10} = 3.9223e^4, BF_{incl} = 5.2168e^4]. Congruency of the foil did not affect the rate of accumulation of evidence towards the target, as suggested by the lack of main effect of drift rate [F(1,19) = 3.808 , p = .066, η_p^2 = .167, BF_{10} = 1.747, BF_{incl} = 2.986]. Nonetheless, the inclusion of a Bayesian factor suggests that this effect is strong evidence for the null hypothesis (Jeffreys, 1961). The number of shared features and congruency were not found to interact [F(1,19) = .247 , p = .625, η_p^2 = .013, BF_{incl} = 1.073] (**Figure 3.7**). See **Appendix Table 3.19 – 3.22**.

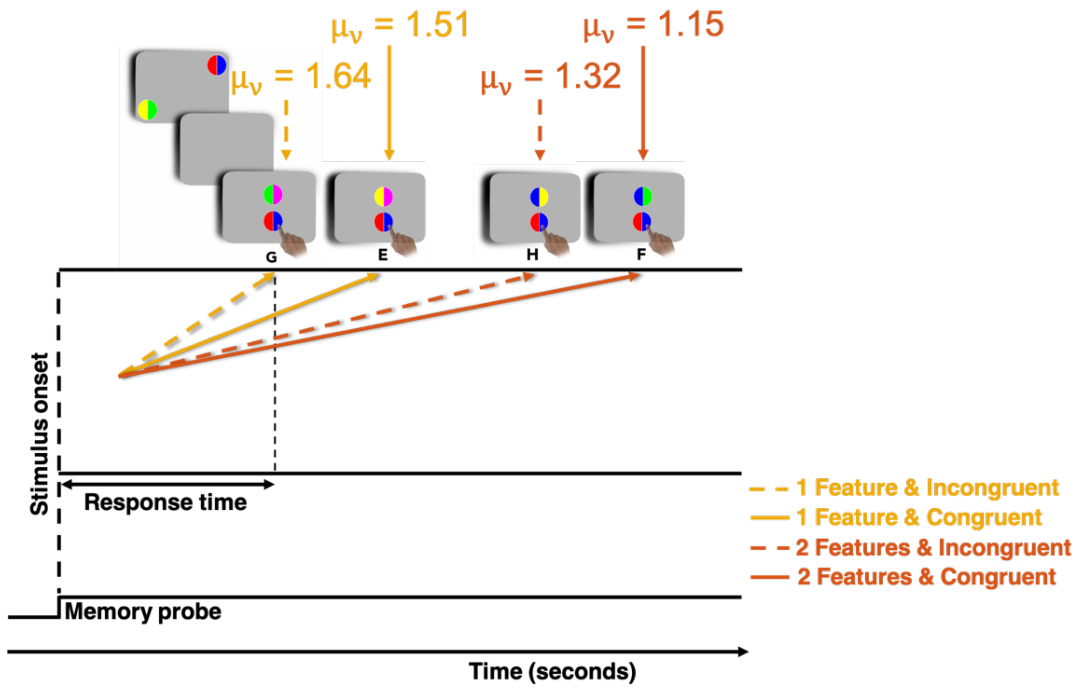


Figure 3.7| Effects of number of features and congruency on drift rate for Model 2.

When the foil holds one feature from the memory display (yellow) instead of two (orange), information is more rapidly accumulated towards the response threshold. Additionally, when the foil holds features in the same colour-position arrangement as in the memory display (congruent with the memory display, dashed lines), evidence towards the target is slowed down.

Moreover, presenting a foil that holds information that is congruent with the memoranda presented at encoding (i.e. a foil with the feature from the non-target item occupying the same half circle as in the memory array) did not result in significantly smaller decision thresholds (t(20) = 1.234, p = .232) (**Figure 3.8**). A 2 (number of features: 1 feature vs 2 features) x 2 (congruency: congruent vs incongruent) ANOVA on reaction time for inaccurate trials (**Figure 3.8**) further revealed that, though close to significant, reaction time for incorrectly identified trials were neither modulated by number of features [F(1,19) = 4.330 , p = .058, η_p^2 = .250, BF_{10} = 1.161, BF_{incl} = 1.007] nor congruency of the foil [F(1,19) = 1.773 , p = .206, η_p^2 = .120, BF_{10} = .597, BF_{incl} = .570]. Moreover, congruency and number of features were not found to interact [F(1,19) = 1.651 , p = .221, η_p^2 = .113, BF_{incl} = .585] (**Appendix Table 3.23 – 3.25**).

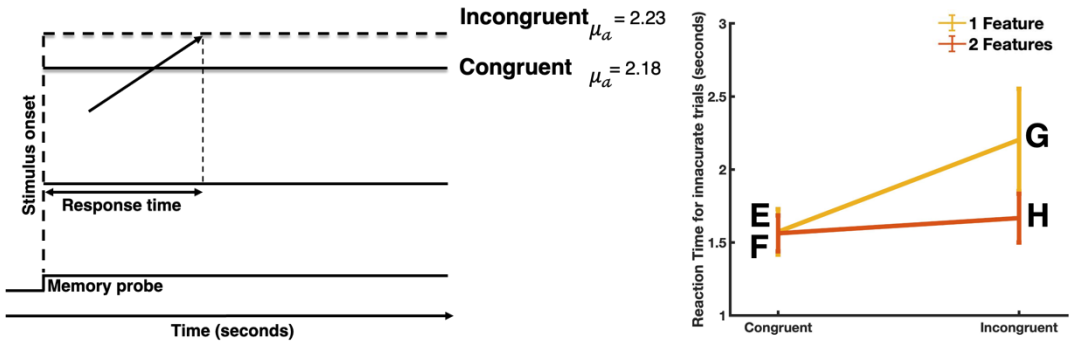


Figure 3.8| Effects of congruency on threshold for Model 2.

At recall, presenting a congruent foil (i.e., a foil with features with the same colour-position arrangement as the non-target) led to a smaller decision threshold. The amount of evidence required to trigger a decision response is lower as a consequence of less cautious responses.

Thus, the most important findings are that increasing the number of features from the memory array on the foil leads to slower accumulation of evidence towards the target. Concurrently, presenting a foil that is incongruent — a foil with the same colour-

position arrangement as in the memory array — increases the rate of accumulation for the correct option.

3.4.1.1. Conclusion

The findings from implementing HDDM to identification accuracy and reaction time data revealed that the decision process of recall is modulated by the nature of the foil, not just the target.

Regarding the question of whether the decision process of recall is hindered by the amount of information carried by the foil matching the memory array, the results show that the decision process of recall is affected by the amount of non-spatial features shared between the memory and test displays. Increasing the number of features from the memory array on the foil decreases the rate of evidence accumulation towards the target.

Concerning the last question, on how the decision process of recall is affected by the arrangement of the colours in the foil, results indicate that presenting a foil that is incongruent with the memory array assists the decision process of recall. As opposed to congruent conditions, trials where a foil holds a different colour-position arrangement from the memory exhibit increased rates of accumulation for the correct option.

3.5. *Discussion*

In Chapter 2, results revealed that recall performance is modulated by the nature of retrieval cues. In particular, presenting a target together with a competing foil was found to impair recall, suggesting that the nature of the foil, not just the target, may play a role in the decision process of WM retrieval. Here, the impact of information contained by the foil on performance was further investigated across two delayed reproduction tasks. Both mixture modelling analysis — to deconstruct the sources of memory errors — and HDDM — to quantify the impact of the information carried by the foil in the process of evidence accumulation towards retrieval from WM — were implemented.

Two questions were addressed. First, what is the consequence of presenting a *foil with different amounts of features from the memory array* on the quality of WM? Second, does the *colour-position arrangement* of these features differentially affect recall depending on whether the foil contained features in their original colour-position arrangement (congruent) or a flipped arrangement (incongruent)?

In Experiment 1, thirty-five heathy young adults were tested on a "what-was-where?" paradigm (**Figure 3.1**) to examine the effect of foil complexity on the quality of recall. Results showed that presenting a foil with two features from the memory array significantly hindered participants' ability to identify the target (**Figure 3.2, Identification Accuracy**), leading to longer reactions times (**Figure 3.2, Reaction Time**). Surprisingly, presenting different numbers of features from the memory array had no detrimental effect on their ability to localize the target (**Figure 3.2, Localisation error**). This suggests that young adults rely on the information in the foil, rather than recalling memoranda

solely based on the information held by the target. They use this information to make their decisions.

This detrimental effect on accuracy of recall — due to the number of features from the memory array in the foil — is consistent with findings from visual search. When presented with a dual choice at retrieval, the process of target selection is akin to visual search, where efficiency is limited by the number of shared similarities between the target and the foil (Bauer et al., 1996; Duncan & Humphreys, 1989; Pashler, 1987; Wolfe, Friedman-Hill, Stewart, & O'Connell, 1992). Increasing the similarity between them by displaying a foil with two features from the memory array — one feature from the target and one feature from the non-target — led to slower reaction times. Possibly because the process of target selection under these circumstances is more analogous to a conjunction search (Mcelree & Carrasco, 1999; Shen et al., 2003), which suffers from similarity between targets and distractors; as opposed to an efficient, feature search (Mcelree & Carrasco, 1999).

With regards to the second question on congruency, results showed that memory performance was modulated by whether the target was presented together with a congruent or an incongruent foil. A foil that held features in the same colour-position arrangement as in the memory array had a detrimental impact on identification accuracy (**Figure 3.2, Identification Accuracy**). However, when this arrangement was broken, as is the case when presenting incongruent trials in which the foil contained features where the colour-position arrangement was flipped, participants' localisation performance (**Figure 3.2, Localisation error**), not just identification accuracy, was impaired.

These findings indicate that the nature of the retrieval foil, not just the target, exerts an influence on the ability to accurately identify and localise memoranda. Moreover, the arrangement of the non-spatial features carried by the foil impacts successful retrieval of the target's location. This is in line with Treisman and Zhang (2006) findings on a special vulnerability of location when the arrangement of features belonging to an object is disrupted, even if irrelevant to the task at hand — suggesting that features are bound both to each other and to their location. Of course, these findings about the impact of the foil also suggest that information from the non-target in the original memory array has an impact on decision-making at the time of retrieval, depending on the nature of the foil and its similarity to the non-target.

A second experiment aimed at further investigating the impact of the information in the foil on the quality of recall by answering two questions. First, how does the nature of the foil modulate the pattern of recall errors? And, second, how can the decision-making of WM retrieval be quantified using HDDM?

Therefore, in Experiment 2, twenty young adults were tested on the same paradigm (**Figure 3.1**), but with an increased number of trials. This allowed to model both the different sources of error contributing to the pattern of localisation performance and the decision process of retrieval.

With respect to the first question, results provided evidence that manipulations of the n*umber of features* from the memory array held by the foil not only impaired accuracy of recall (**Figure 3.3, Identification Accuracy**), but also increased reaction times (**Figure 3.3, Reaction Time**). Importantly, this manipulation did not affect localisation performance (**Figure 3.3, Localisation error**) or memory resolution (**Figure 3.4**). However,

the detrimental effect of foil incongruency on participants' ability to correctly localise the target (**Figure 3.3, Localisation error**) was found to be specifically attributable to an increase in the number of errors due to misbinding (**Figure 3.4, Misbinding**). In other words, conditions where the target was presented together with a foil carrying features where the colour-position arrangement was not the same as in the memory array, led participants to drag the target to the location of the non-target. Conversely, neither imprecision (**Figure 3.4, Imprecision**) nor guessing (**Figure 3.4, Guessing**) were affected.

This finding on the foil congruency affecting localisation performance — by modulating the binding of the target's identity with its location — is again in accord with the proposition that location plays a special role in WM (Jiang, Olson, & Chun, 2000; Olson & Marshuetz, 2005; A. Treisman & Zhang, 2006; Udale et al., 2017), suggesting that non-spatial information is bound across features using location as a common index. This location-mediated-indexing finding fits well with the visual architecture hypothesised by The Feature Integration Theory (FIT) for visual search (Quinlan, 2003; A. Treisman, 1998; A. M. Treisman, 1980). This conceptualization of memory representation posits that spatial information is held in memory in the form of a master-map of locations, which is responsible for coordinating independent maps, wherein information on non-spatial features is stored (Quinlan, 2003; A. Treisman, 1998; A. M. Treisman, 1980). Our results may be accounted for by such a map-architecture because, in conditions where the target is presented together with an incongruent foil, the features belonging to the latter hold an increased number of spatial traces compared to when the foil is congruent. In other words, presenting a congruent foil activates the same colours and spatial traces previously encoded from the memory array. However, presenting an incongruent foil —

a foil with new spatial traces because it displays a flipped colour-position arrangement compared to the memory array — not only activates the spatial traces previously encoded, but also novel spatial traces indexed to colours belonging to another object presented in the memory array. Therefore, a larger re-activation of recent memory traces from one map with the memory traces for location available in other maps might lead to increased misbinding.

With respect to the second question, HDDM was implemented to quantify the effects of the nature of the foil on the decision process of recall. Results showed that the recall process is impacted by the quality of the information held by the foil. Increasing the number of features from the memory array led to slower accumulation of evidence towards the target (**Figure 3.7**). Simultaneously, presenting a foil with the same colour-position arrangement as in the memory array (congruent trials) increased the rate of accumulation for the correct option (**Figure 3.7**). While the drift rate was modulated by both number of features and complexity, the decision threshold was solely influenced by the congruency of the foil. When the colour-position arrangement of the non-spatial features was the same as in the memory array, the amount of evidence required to trigger the correct decision response was lower (but not significantly lower) compared to when this arrangement was broken, because of less cautious responses, i.e., quicker incorrect responses (**Figure 3.8**).

The model results on the effects of amount of information from the memory array on the foil are consistent with work by Bocincova and colleagues (2020), showing that a neural model recently proposed by Manohar et al. (2017) relying on pattern completion mechanisms to produce cued recall was also able to perform visual search. With

particular relevance to this study, these authors showed that the reactivation of recent patterns of input through pattern completion mechanisms could be employed to perform target selection and distraction rejection during visual search. Their analysis demonstrated that the model predicts that distracting information decreases the selectivity of the signal produced by the target unit activation, which could explain why in our data we found that conditions where a foil carried two features from the memory display — as opposed to one feature — were associated with longer drift rates.

Presenting a foil containing features with the same arrangement from that presented in the memory array (congruent trials) benefited the rate of accumulations of evidence towards the target. This finding could be explained based on Manohar et al. dynamic model of WM (2017), which utilizes pattern completion and mutual inhibition to implement cued recall and visual search. After the associations between colour and all locations presented at the memory array are encoded into a conjunction unit, the latter can be re-activated at recall when presented as a retrieval cue. Displaying a target together with a congruent foil, as opposed to an incongruent foil, allows for information to be gathered faster towards a decision threshold because the process of pattern completion is triggered more efficiently. In other words, presenting an incongruent foil is more costly because multiple different templates are activated and concurrently guide the selection of the target, as reflected by larger drift rates of evidence accumulation exhibited by our modelling approach.

It is important to note that an inherent limitation of quantitative models of choice, such as the one used in this Chapter, is that they provide an incomplete account of the behavioural data. In fact, the model here accounts for the distribution of reaction times

for correct and incorrect responses, but is incapable of quantifying the implications of the nature of the foil on localisation performance or misbinding rates. Future development of theoretical models on the flexibility of decision making of WM representations could significantly enrich the understanding of the underlying mechanisms of memory retrieval.

Taken together, the findings presented here provide initial evidence that the process of associative retrieval may also be reliant on the quality of the information held by the foil and its similarities to the non-target not just the target. Specifically, successful retrieval of the target's location is closely associated with the arrangement of non-spatial features belonging to the foil.

Chapter 4. Impact of attentional demands at encoding, maintenance and retrieval in visual working memory[1]

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*The two authors contributed equally to this work, thus, share the first authorship.

All co-authors have given permission to include this material in this thesis.

Abstract

There has been surprisingly little examination of how recall performance is affected by processing demands induced by retrieval cues, how manipulations at encoding interact with processing demands during maintenance or due to the retrieval cue, and how these are affected with aging. Here, we investigate these relationships by examining the fidelity of WM recall across two delayed reproduction tasks with a continuous measure of report across the adult lifespan. Participants were asked to remember and subsequently reproduce from memory the identity and location of a probed item from the encoding display. In Experiment 1, we examined the effect of filtering irrelevant information at encoding and the impact of filtering distracting information at retrieval simultaneously. In Experiment 2, we tested how ignoring distracting information during maintenance or updating current contents with new information during this period affects recall.

The results reveal that manipulating processing requirements induced by retrieval cues (by altering the nature of the retrieval foil) had a significant impact on memory recall: the presence of two previously viewed features from the encoding display in the retrieval foil led to a decrease in identification accuracy. Although irrelevant information can be filtered out well at encoding, both ignoring irrelevant information and updating the contents of memory during the maintenance delay had a detrimental effect on recall. These effects were similar across the lifespan, but older individuals were particularly affected by manipulations of processing demands at encoding as well as increasing set size of information to be retained in memory. Finally, analyses revealed that there were no systematic relationships between filtering performance at encoding, maintenance

and retrieval suggesting that these processing demands are independent of each other. Rather than filtering being a single, monolithic entity, the data suggest that it is better accounted for as distinctly dissociable cognitive processes that engage and articulate with different phases of WM.

4.1. *Introduction*

It is now generally agreed that there are extremely strong links between processes traditionally subsumed by the terms 'attention' and 'working memory' (Chun & Johnson, 2011; Sean James Fallon et al., 2016; Manohar, Zokaei, Fallon, Vogels, & Husain, 2019; Rhodes & Cowan, 2018). In the visual WM literature, these relationships have typically been probed by investigating the impact on recall performance of processing demands at encoding and during maintenance including studies of feature misbinding, but not generally at retrieval.

Demands on memory resources *at encoding* have often been studied by using pre-cues which provide information about the relevant properties of the upcoming task, such as the features of stimuli that are more likely to be probed (Jost et al., 2011; Vogel et al., 2005). For example, spatial cues can direct participants to locations where relevant information will be presented allowing them to encode fewer items and thereby improve performance (Ikkai, McCollough, & Vogel, 2010; Schmidt, Vogel, Woodman, & Luck, 2002; Vogel et al., 2005; Woodman, Vecera, & Luck, 2003).

Manipulations of processing demands on visual WM representations *during maintenance* have also been possible using either retro-cues which increase the

likelihood of some items being probed (Griffin & Nobre, 2003a; Landman, Spekreijse, & Lamme, 2003a; for a review see Souza & Oberauer, 2016a) or presenting distractors that need to be ignored during the retention interval (Sean J. Fallon et al., 2018; Souza S. et al., 2014; van Moorselaar et al., 2015; Williams et al., 2013). In this case, remembered information has to be protected from being overwritten by the irrelevant stimuli.

In addition to the use of these strategies to investigate the interaction of processing demands and WM, some authors have also focused on *binding errors* in WM recall, since feature binding has been considered to be one important aspect of attention (Alan D. Baddeley et al., 2011; Johnson et al., 2008; J. S. Luck & Vogel, 1997; Wheeler & Treisman, 2002). However, paradigms used to investigate this issue have usually not been designed to establish at which stage of memory – encoding, maintenance or retrieval – feature binding of stimuli has been affected. Some work has shown that attentional deployment during maintenance makes a contribution to keeping stimulus features correctly bound in WM. First, misbinding rates increase with retention durations particularly with high memory loads (Pertzov et al., 2017). Second, presentation of distractors during retention intervals can also increase misbinding, specifically to features in the to-be-ignored distractors (Sean J. Fallon et al., 2018). Finally, consistent with the proposal that high processing demands lead to more misbinding errors, paradigms that employ taxing demands during the maintenance interval have shown an increase in misreporting features of stored items (L. A. Brown & Brockmole, 2010; Fougnie & Marois, 2009; Zokaei, Heider, et al., 2014).

To the best of our knowledge, most investigations on the role of processing demands in WM, such as the ones described above, have focused on encoding and

maintenance periods. However, there has been little research on how recall performance is affected by processing demands induced by the retrieval cue. Theeuwes, Belopolsky, & Olivers (2009) showed that in retrieval from visual WM, observers allocate visual attention to the location in space that contains the information to be retrieved, even if this is not required to solve the task whatsoever. Hence, they concluded that directing attention in WM is similar to selecting actual visual input in form of visual stimuli (Theeuwes et al., 2009, 2011). We are not aware, though, of any study that has attempted to examine the relationships between processing demands at encoding, maintenance and retrieval. This would be important to establish whether there is a generic system that operates at all phases of WM when processing demands are increased or whether there might be dissociable or fractionated systems that operate at different phases in WM.

To investigate these issues, we designed two experiments to systematically examine how processing demands induced at different stages of WM relate to each other. The first experiment required participants on some trials to filter out information at encoding because it would never be probed at retrieval, while manipulating processing demands at retrieval by varying the nature of a distractor foil. Specifically, we altered whether the foil had one or two features that belonged to either another to-be-encoded item in the encoding display or one or two features that belonged to a task-irrelevant item that should have been filtered out at encoding. The second experiment compared the ability to filter out distractors presented during maintenance (*Ignoring*) with performance when new items presented during retention had to be uploaded into WM and older ones jettisoned (*Updating*).

Until now, the effects of filtering demands in most previous studies have relied on quantifying WM capacity by employing change-detection paradigms or using the amplitude of CDA, an electrophysiological marker putatively linked to WM capacity, assuming an item is either stored or not (Cowan & Morey, 2006; Gazzaley, Clapp, Kelley, McEvoy, & Knight, 2008; Jost et al., 2011; Rhodes & Cowan, 2018; Robison et al., 2018; Vogel & Machizawa, 2004; Vogel et al., 2005). For example, Vogel et al. showed that filtering ability correlates very closely with WM capacity and that people who can remember more objects from a spatial array are more efficient at excluding irrelevant information, arguing that individual differences in memory capacity can be predictive of filtering abilities (Vogel et al., 2005). In the current study, memory performance was measured using a delayed reproduction task, which uses a continuous response over an analogue scale. Therefore, instead of solely asking participants to report whether they remember a feature, here they were also requested to reproduce the exact quality of the remembered feature in an analogue response space (Paul M. Bays et al., 2009; Gorgoraptis et al., 2011; Pertzov et al., 2012). This method also allowed us to model the source of errors in terms of noisiness or imprecision of recall of the probed item or target, misbinding (reporting the features of a non-probed item that had been presented in the encoding display) and random guessing (Paul M. Bays et al., 2009; Grogan et al., 2019).

An ancillary aim of our study was to examine how processing demands at encoding, during maintenance and due to the retrieval cues, vary across the adult lifespan, with aging. Investigations of the effects of aging on storage have shown that the number of items that can be held in WM decreases with age (Chen, Hale, & Myerson, 2003; McNab & Dolan, 2014; Myerson, Emery, White, & Hale, 2003; Peich, Husain, & Bays, 2013;

Pertzov, Liang, Heider, & Husain, 2015). Additionally, it has been found that filtering abilities, at least as indexed by the amplitude of the CDA, is worse in older people but only just after encoding, improving over the retention delay (Jost et al., 2011). Another study has concluded that the ability to ignore distracting information at encoding might be a better predictor of age-related decline in WM capacity compared to filtering information at maintenance (McNab et al., 2015). Clearly, it remains to be resolved how processing demands impact on WM performance with aging in adults. Moreover, it is unknown whether processing effects induced by retrieval change with age.

4.2. *Experiment 1*

The first study investigated the effect of processing demands at encoding and the impact of distracting information at retrieval. The key questions we sought to answer were:

1. Is there an impact of the nature of the foil type at retrieval depending upon whether it had one or two features from an item presented in the encoding display which is to be expected in Identification Accuracy?
2. Does the nature of the foil type at retrieval impact upon recall, depending upon whether it had features that came from a task-irrelevant item, i.e., that was supposed to have been filtered out at encoding which is also to be expected in Identification Accuracy in particular?
3. How successful is filtering at encoding (by comparing performance in the set size 2 condition with the filtering condition in which 3 items were

presented but one item was task irrelevant, i.e., it had to be filtered out) reflected by Identification Accuracy and Localisation Performance?

4. What is the impact of these performance measures with aging? Does aging simply lead to worse performance across the board or is there any selective effect of age in the Identification Accuracy or Localisation Performance in 1-3?

4.2.1. Methods

4.2.1.1. Participants

We ran two experiments (**Experiment 1** and **Experiment 2**) with the order in which participants performed them randomized. 40 young adults aged between 18 and 34 years (26 females, 14 males) and 61 elderly adults aged between 52 and 81 years (34 females, 27 males) that reported to be neurologically normal were recruited to take part in this Experiment. See further demographics displayed in **Table 4.1**. Permission for this study was obtained from the local ethics committee and all subjects gave written informed consent. Cognitive function screening was tested using Addenbrookes Cognitive Examination-III (ACE-III) (Mathuranath, Nestor, Berrios, Rakowicz, & Hodges, 2000).

Table 4.1 | Participant Demographics.

Measure	Healthy young controls	Healthy elderly controls
n	40	61
Age (years old)	23.8 (± 4.1)	68.2 (± 7.5)
Gender (F/M)	26/14	34/27
Handedness (R/L)	38/2	48/12
Global Cognition (ACE-III score)	96.0 (± 4.1)	97.1 (± 2.7)

ACE = Addenbrooke's Cognitive Examination III; all values are mean (± SD).

4.2.1.2. Stimuli, Design and Procedure

This experiment investigated the effect of filtering at encoding and the impact of distracting information induced by retrieval cues on recall (Figure 4.1). Participants were asked to remember both the identity and location of circles, each of which consisted of semicircles of different colours. The orientation of a white arrow presented in the middle of the screen pointed to the items that had to be remembered. For example, if the arrow pointed left, participants had to recall memoranda presented on the left side of the screen while ignoring the *task-irrelevant item* presented on the opposite side of the screen, i.e., this was the item that had to be filtered out from the encoding display. In half of the trials, the memory array (the items that should be remembered) was presented on the left side of the screen, in the other half it was presented to the right. This arrow manipulation allowed us to examine processing demands *at encoding.*

After a 2s delay, participants were given a two-alternative choice between a *target* – the correct item to identify which had appeared in the encoding display – and a *foil* – a

distractor item that had not appeared in the memory display. They had to touch the target, then drag it to where it originally appeared.

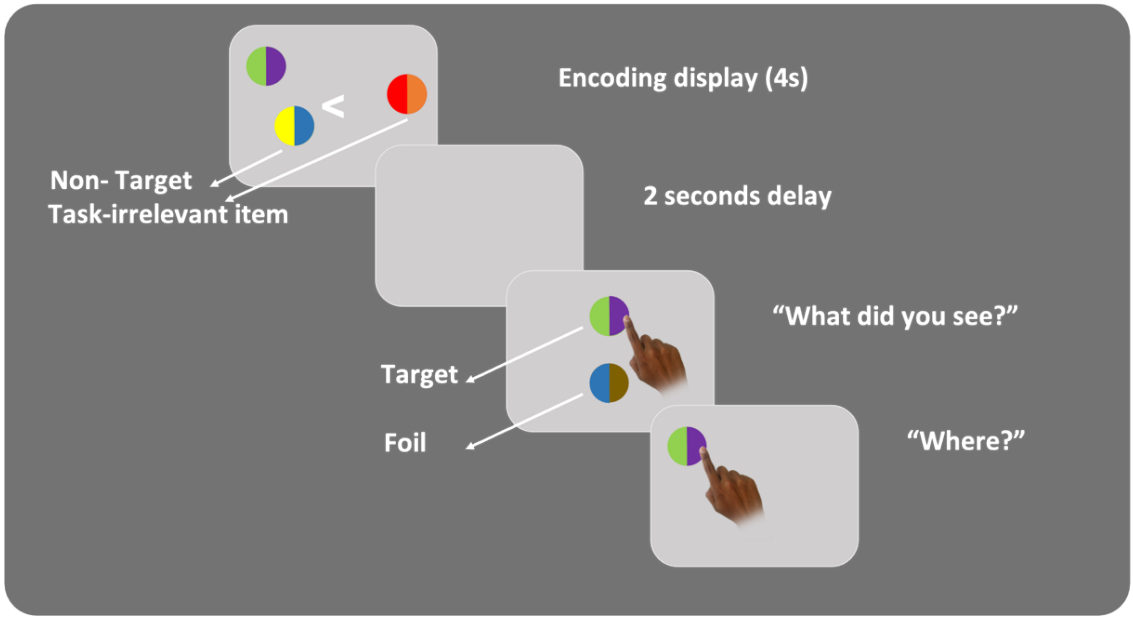


Figure 4.1| Schematic of Experiment 1: Manipulations of retrieval and filtering.

Participants were required to remember only the circles on that half of the screen that the arrow (< or >) pointed towards. After a 2 s delay, they were given a choice between a target and a foil. They needed to select the target and then drag it to where they remembered it to have originally appeared.

In the "set size 2" and "set size 3" conditions, two or three circles were presented respectively on one half of the screen, indicated by the arrow pointing to that side (**Figure 4.2, first and last row**). In the filtering at encoding condition, two circles were presented on the half of the screen that the arrow pointed towards while a *task- irrelevant item* was presented on the opposite side (**Figure 4.2, second row**).

The amount of information competing *at retrieval* was manipulated by including, in addition to the target item, a foil at retrieval. The foil was a circle which consisted of two semicircles, just like the target. Importantly, it contained either one or two features

(coloured semicircles) from an item in the original encoding display. Note that the semi-circle with the same colour as the target always occupied the opposite half of the circle to its location in the original encoding display.

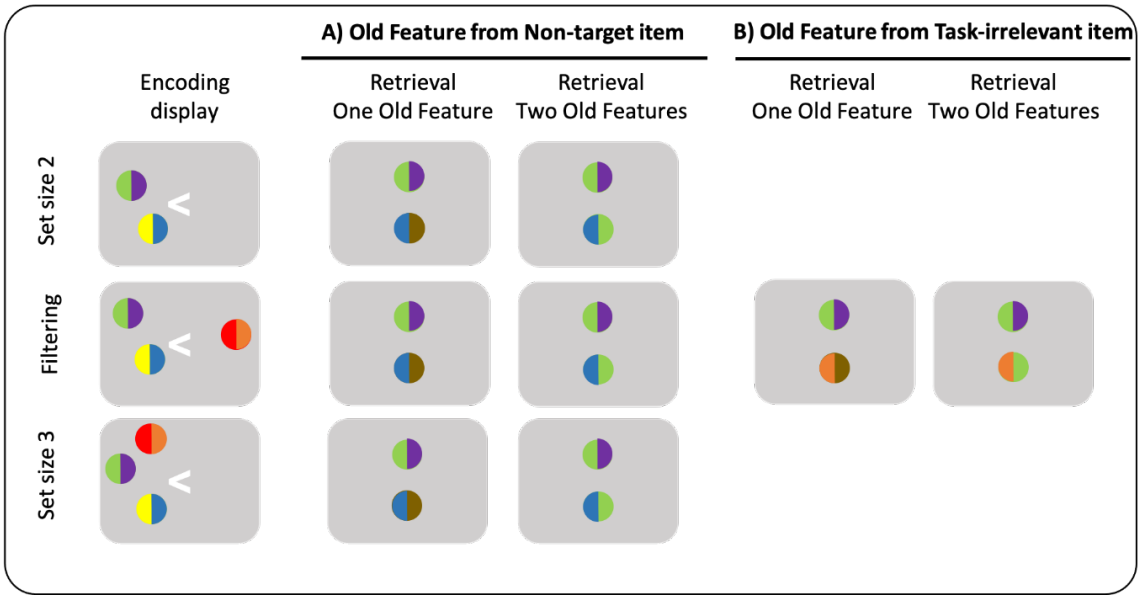


Figure 4.2| Schematic of all conditions for Experiment 1.

Encoding was manipulated by varying the number of items to be stored (Set size 2 and Set size 3) and processing demands at encoding were manipulated by asking participants to filter irrelevant information (Filtering). Retrieval was manipulated across all conditions in two main ways: A) retrieval cues had features from the non-filtered item or B) retrieval cues had features belonging to the task-irrelevant item. Additionally, in both A) and B), the retrieval foil could have either one or two old features from the encoding display.

The retrieval foil could consist of:

Features from non-target items:

- A semicircle filled with a colour that had appeared in one of the non-targets in the encoding display (one old feature from non-filtered items), with the other half of the circle filled with a colour that had not appeared in the

encoding display (one old feature from non-filtered items: **Figure 4.2, second column**).

- A semicircle filled with a colour that had appeared in one of the non-targets in the encoding display, but with the other half of the circle filled with a colour that had appeared in the *target item* (two old features from non-filtered items: **Figure 4.2, third column**).

Features from task-irrelevant items:

- A semicircle filled with a colour that had appeared in the task-irrelevant item that had to be filtered out in the encoding display (one old feature from the task-irrelevant item), with the other half of the circle filled with a colour that had not appeared in the encoding display (one old feature from the task-irrelevant item: **Figure 4.2, fourth column**).
- A semicircle filled with a colour that had appeared in the task-irrelevant item that had to be filtered out in the encoding display, but with the other half of the circle filled with a colour that had appeared in the *target item* (two old features from the task-irrelevant item: **Figure 4.2, fifth column**).

In total, there were eight different conditions: two manipulations of the number of features competing at retrieval (one old feature vs two old features from the encoding display) x four manipulations of set size (memory array composed of 2 items vs memory array composed of 3 items) and filtering (distracter composed of features from the task-irrelevant item vs distracter composed of features from the non-filtered items). Participants performed ten trials of each of these eight conditions. Each trial began with the presentation of either two or three circles (4000ms) that appeared in separate spatial

locations, with the symbol "<" or ">" appearing in the centre of the screen. These memoranda either had to be encoded or filtered from WM. The symbol "<" or ">" pointed to which memoranda had to be encoded (Figure 4.1). Then, after a delay period (2000ms) came the probe phase. In the probe phase a dual choice was presented. After touching the target shape, participants had to drag the shape to its original location and confirm by pressing a 'Done' button.

Participants sat in front of an interactive touch-sensitive screen (tablet Samsung, model Galaxy Tab S3, 2.15Ghz+1.6Ghz Quad Core Processor, 9.7 inches,) with a 2048x1536 pixel matrix. Stimuli were presented on a grey background (RGB [171,171,171]). In Experiment 1, colours were selected from a total of seven equally spaced-out colours on the same colour wheel, avoiding identical trial and colour-combinations. The stimuli were presented at a viewing distance of approximately 30 cm and subtended a visual angle of approximately 2.3^{o}.

4.2.1.3. Behavioural Recall Analysis

Data and analysis script are available on OSF: https://osf.io/75mju/?view_only=933a99445ddc4f97b75b58dc5ed25694. Identification performance was computed by taking the proportion of times participants touched the correct shape in the dual choice per condition. Localisation error was calculated by taking the distance between the centre of the reported location and the centre of the original location of the probed item. This was measured for only correctly identified targets. Errors due to misreporting the location of an item occurred as a result of different factors,

either misbinding errors or guessing. Misbinding errors refer to the proportion of times subjects misreport the location of the correctly identified item by dragging it to the location of another item presented in the encoding display (**Figure 4.3**). Imprecision was calculated based on the distance to the nearest neighbour, i.e., the closest distance between the response (where the participant placed the chosen item) and the location of any item in the encoding display (either a target, non-target or a task-irrelevant item).

Here, we calculated misbinding, using a permutation analysis approach based on the mixture model (Paul M. Bays et al., 2009) as follows: Per trial, distances between the response location and **i**) the target location, **ii**) the location of the closest non-target (the closest item that is not being probed) and **iii**) the location of another, randomly chosen trial's non-target were calculated. The locations of each of the target and non-target locations were randomly determined per trial, thus leading to a range of possible locations that is equal to the number of all non-targets in the experiment. Depending on which of these distances was the shortest, the response was either counted as target (**i**), non-target (**ii**, "misbinding") or random/guessing response (**iii**, "uniform"). We repeated this procedure 5000 times per trial, thus introducing a non-target from a randomly chosen trial (see **iii**) each time. This procedure allowed us to calculate proportions for these three sources of response per trial (absolute amount of response type/5000). The introduction of a non-target that was randomly chosen from another trial (**iii**) allowed us to differentiate whether an error was systematically linked to the very specific trial's non-target or whether it could be accounted for even by a randomly chosen non-target that was not present at trial. Guessing, quantifies the proportion of times an item is completely forgotten. This corresponds to the proportion of responding randomly to the

target location. To assess the performance of this permutation analysis, we simulated synthetic data and compared the simulation input to the results calculated using this analysis (**Appendix Chapter 4, Figure 4.1**). They show that for misbinding, the overall average of calculations will land on the true value without any particular bias towards over- or underestimating the parameter. But every single calculation will on average deviate about 0.03 from the true value and, thus, results of small magnitudes should be considered with due caution.

Data were analysed using *MATLAB* (The MathWorks inc., version 2018a) and *JASP* (JASP Team, 2020).

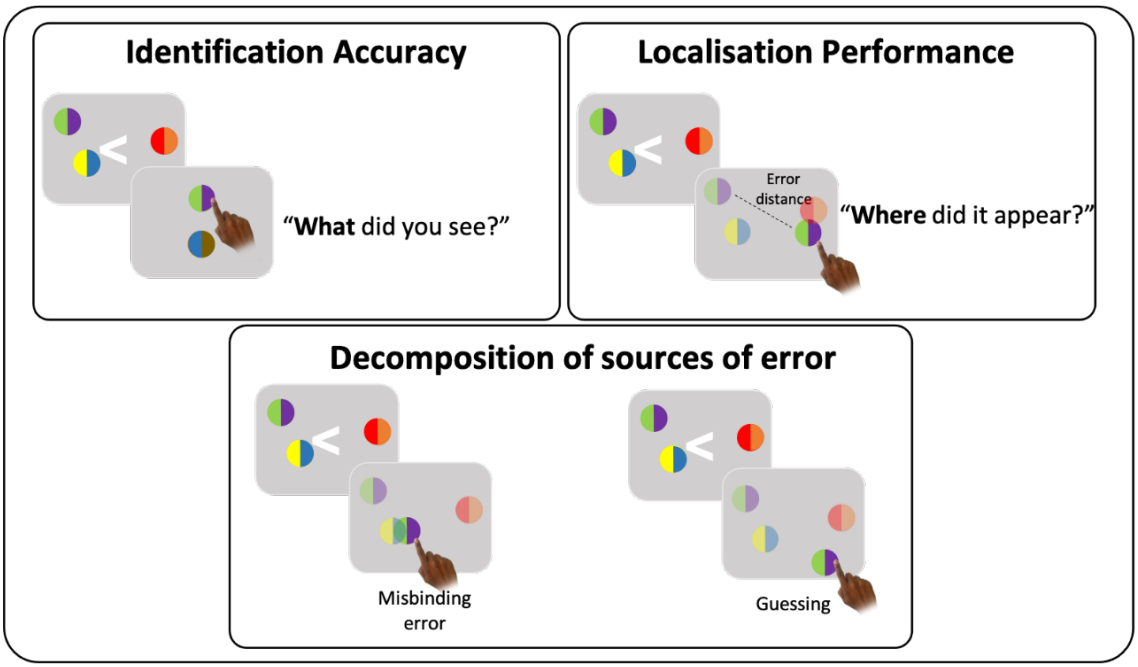


Figure 4.3| Recall measures.

Identification performance is the frequency with which participants touched the target in the two-alternative forced choice (top left). Localisation performance is the distance between the centre of the reported location and the original location of the target (top right). Decomposing the sources of error allowed extraction of two types of error due to misreporting the location of an item": 1) Misbinding errors, that occur when the participant dragged the correct item to the location of another item presented in the encoding display (bottom left) and 2) Errors due to guessing when a participant dragged the correct item to a random location (bottom right).

4.2.2. Results

4.2.2.1. Effect of set size, retrieval foil type and age group

2x2x2 repeated measures ANOVAs of set size (two vs three) x number of old features in the foil (one vs two) x group (young vs old participants) were first conducted to investigate the effects of set size and number of old features (from items in the encoding display) that appeared in the foil at retrieval on:

- Identification accuracy (proportion of correctly identified items)
- Localisation error
- Model parameters: misbinding, guessing and imprecision
- Reaction time (time taken to initiate the response)

Identification accuracy and Localisation performance were the variables of interest which were followed up with the model parameters and reaction time. The findings are depicted in Figure 4.4 and details are summarized in Appendix Tables 4.1 - 4.9.

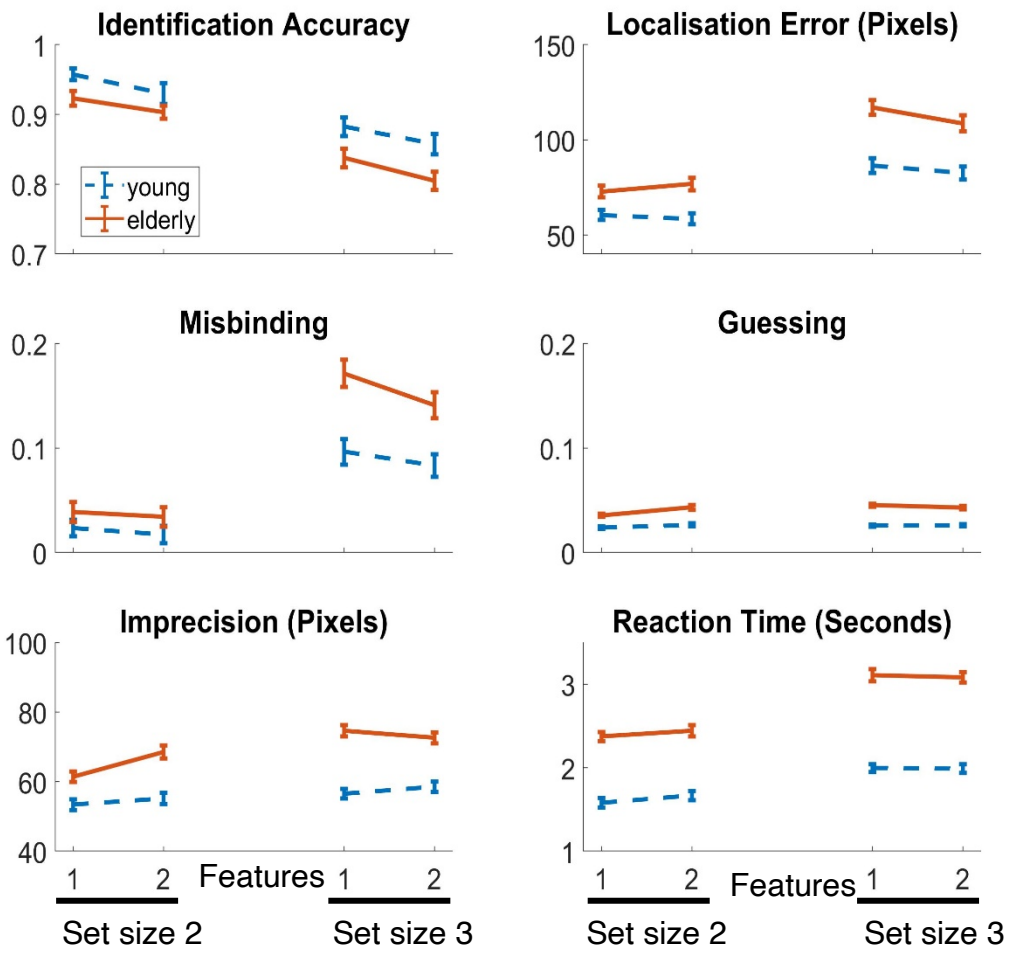


Figure 4.4| Impact on memory recall of number of old features in the foil at retrieval.

An increase of old features from 1 to 2 decreased Identification Accuracy. This was independent of Set Size. Higher set size led to an increase of Localisation Error driven by higher imprecision and higher misbinding, additionally participants responded significantly slower. Elderly participants generally performed worse than young controls. Error bars were calculated by subtracting each subject's grand mean away from their individual per-condition values and showing ± standard error (Loftus & Masson, 1994).

Set size

As expected, increase in set size from 2 to 3 items both decreased identification accuracy [$F(1,99) = 50.39$, $p < 0.001$, $\eta_p^2 = 0.34$] and increased localisation error [$F(1,99) = 82.62$, $p < 0.001$, $\eta_p^2 = 0.46$]. With respect to modelling parameters, increase in set size led to a significantly higher misbinding [$F(1,99) = 73.27$, $p < .001$, $\eta_p^2 = 0.43$] and

imprecision [$F(1,99) = 16.11$, $p < 0.001$, $\eta_p^2 = 0.14$] as well as slower reaction time [$F(1,99) = 74.71$, $p < 0.001$, $\eta_p^2 = 0.43$]. However, there was no significant main effect on guessing.

Number of old features in foil at retrieval

Increase in the number of features from the memory display in the foil at retrieval from 1 to 2 significantly decreased identification accuracy [$F(1,99) = 7.06$, $p = 0.009$, $\eta_p^2 = 0.07$]. However, it had no impact on localisation error, misbinding, guessing, imprecision or reaction time. Thus, the critical impact of number of features in the foil at retrieval was mainly on item identification. In addition, there was also a significant interaction between number of features and set size in guessing [$F(1,99) = 5.49$, $p = 0.021$, $\eta_p^2 = 0.05$]: Holm-corrected post-hoc t-tests show that participants performed worse for two features in the set size 2 condition compared to one feature [$t(100) = 2.924$, $p = 0.022$], but this was not the case in set size 3 [$t(100) = .540$, $p = 1.00$]. There was also a significant difference between set size 2 and set size 3 for one feature [$t(100) = 2.940$, $p = .022$] but not for two features [$t(100) = .166$, $p = 1.00$]. However, **Figure 4.4** suggests that the numeric value of this effect is marginal.

Young vs older participants

Younger participants performed significantly better on all metrics. Their identification accuracy was higher [$F(1,99) = 6.11$, $p = 0.015$, $\eta_p^2 = 0.06$], localisation error was smaller [$F(1,99) = 14.22$, $p < 0.001$, $\eta_p^2 = 0.13$], misbinding [$F(1,99) = 8.83$, $p = 0.004$,

η_p^2 = 0.08] and guessing [*F*(1,99) = 9.13, *p* = 0.003, η_p^2 = 0.08] were less, and they were more precise [*F*(1,99) = 18.21, *p* < 0.001, η_p^2 = 0.16] and reacted faster [*F*(1,99) = 49.02, *p* < 0.001, η_p^2 = 0.33]. There was a significant interaction of age group with the set size in misbinding (**Appendix Table 4.3**). This was further explored by two independent ANOVAs for each group on misbinding. In both young and older age groups there were significant main effects of set size [respectively *F*(1,39) = 30.82, *p* < 0.001, η_p^2 = 0.44; (*F*(1,60) = 56.77, *p* < 0.001, η_p^2 = 0.49], indicating greater misbinding when 3 items had to be remembered compared to 2. Holm-corrected post-hoc t-tests on the original ANOVA revealed that elderly participants were significantly worse than young controls in the 3 item condition but not in the 2 item condition [t(99) = 8.608, *p* < .001, t(99) = .911, *p* = .363, respectively; **Appendix Table 4.4**]. In addition, there was an interaction of group and set size in the reaction time (**Appendix Table 4.8**), which was followed up in an analogous manner. Both young and older people were slower on recall when there were 3 items [respectively *F*(31.02, *p* < 0.001, η_p^2 = 0.44); (*F*(1,60) = 59.72, *p* < 0.001, η_p^2 = 0.50]. Holm-corrected post-hoc t-tests following up on the original interaction showed that elderly controls are slower than young controls in both, the set size 2 [t(99) = 5.30, p < .001] and the set size 3 conditions [t(99) = 7.44, p < .001; **Appendix Table 4.9**].

In summary, regarding our first research question, the results show that the nature of the foil type – manipulation of processing demands induced by retrieval cues – had a significant impact on memory recall. Presenting two old features in the retrieval foil significantly decreased identification accuracy, independent of set size or age. The latter

did not reveal any specific impairment but generally reduced performance over all metrics.

4.2.2.2. Impact of filtered vs non-filtered features at retrieval

Next, we examined the data from the filtering trials (Figure 4.5, Filtering condition) by looking at the effects on recall performance of *filtering at encoding*, as a function of the number of old features from the encoding display present in the foil *at retrieval*. A 2x2x2 ANOVA of the number features x non-filtered vs filtered features in the foil x age group was performed (Figure 4.5, Appendix Tables 4.10 – 4.15).

Filtered vs non-filtered features in retrieval foil

Identification accuracy was significantly affected by whether features in the foil at retrieval were from the non-filtered vs filtered object at encoding [$F(1,99) = 8.23$, $p = 0.005$, $\eta_p^2 = 0.08$]. Thus, participants identified the correct item more often when the foil's features were taken from the task-irrelevant item, compared to when they were taken from the non-filtered item. This suggests that if at encoding participants successfully filtered out an item, they were less affected by features from it appearing in the foil. No other significant main effects of the task-irrelevant versus non-filtered item were observed: for localisation error, misbinding, guessing, imprecision or reaction time.

Number of old features in foil at retrieval

Increasing the number of old features in the foil at retrieval significantly decreased identification accuracy [$F(1,99) = 6.08$, $p = 0.015$, $\eta_p^2 = 0.06$] but also slowed participants down [$F(1,99) = 10.11$, $p = 0.002$, $\eta_p^2 = 0.09$]. There was, however, no significant effect on localisation error, misbinding, guessing or imprecision. There was a significant interaction for misbinding between filtered/non-filtered feature and number of features in the retrieval foil [$F(1,99) = 4.71$, $p = 0.032$, $\eta_p^2 = 0.05$]. Holm-corrected post-hoc t-tests showed that participants were significantly more likely to misbind in the two-previously-seen-features condition when one foil's feature was taken from the non-filtered item

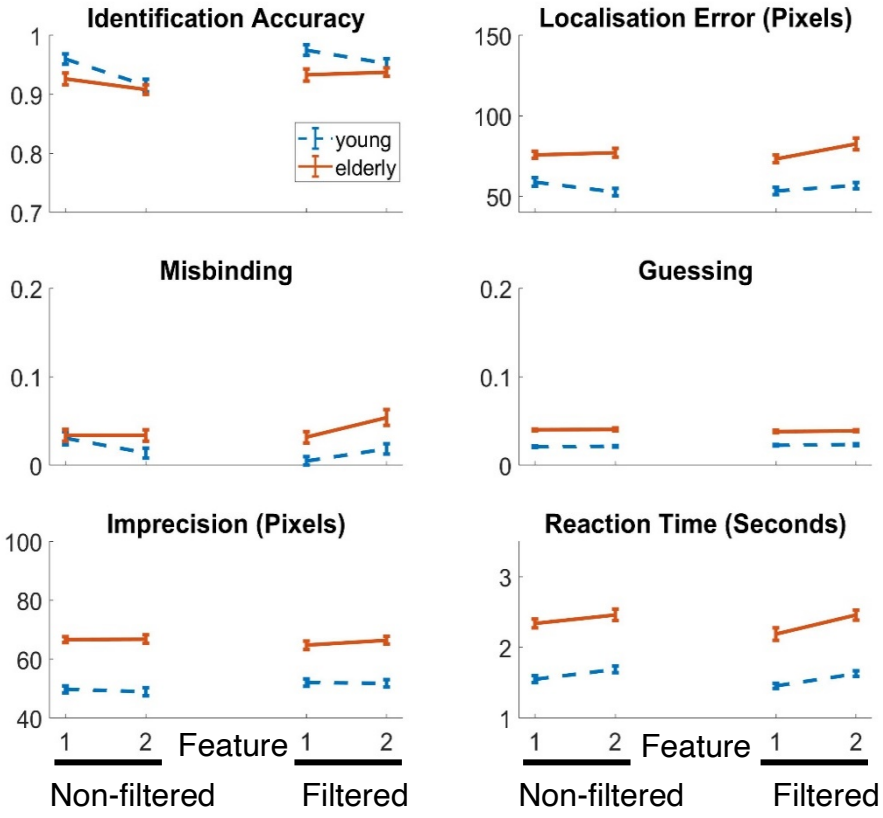


Figure 4.5| Impact on memory recall of filtered versus non-filtered features at retrieval.

If the retrieval foil's features were taken from the item that was to be filtered out at encoding, participants were faster and identified the correct item more often. Error bars were calculated by subtracting each subject's grand mean away from their individual per-condition values and showing ± standard error (Loftus & Masson, 1994).

[t(100) = -2.18, p = 0.032], but not if it was from the task-irrelevant item [t(100) = 0.85, p = 0.396].

Young vs older participants

Older participants performed at the levels of young participants in correctly identifying the memoranda [F(1,99) = 3.25, p = 0.074, η_p^2 = 0.03], but showed significantly higher localisation error [F(1,99) = 26.16, p < 0.001, η_p^2 = 0.21], more misbinding [F(1,99) = 7.09, p = 0.009, η_p^2 = 0.07], more guessing [F(1,99) = 12.55, p < 0.001, η_p^2 = 0.11] and higher imprecision [F(1,99) = 32.55, p < 0.001, η_p^2 = 0.25] as well as being slower to react [F(1,99) = 51.32, p < 0.001, η_p^2 = 0.34]. There were no significant interactions between age group with any of the factors or interactions outlined above.

In summary, with respect to our second research question, when previously presented features that were present in the foil originated from the task-irrelevant item, Identification Accuracy was better than when they were from the non-target, indicating they were likely filtered out. This suggests that irrelevant information is filtered out well at encoding. There was no evidence that age modified these effects.

4.2.2.3. Effects of filtering at encoding

A 2x2x2 ANOVA of set size (two vs three) x number of old features in the foil (one vs two) x group (young vs old). was conducted to compare directly the set size 2 condition with the condition in which participants were asked to filter out one of three items. If

filtering was perfect, the results of this analysis should show no difference in performance between these conditions. In both cases, the retrieval foil's features were taken from the items that were to be remembered (**Figure 4.6, Appendix Tables 4.16 – 4.22**).

Two items vs filter condition

There was no significant main effect of filtering between the conditions with respect to identification accuracy, localisation error, misbinding, guessing, imprecision or reaction time. In line with the previous analyses, the number of old features in the foil did, however, have a significant impact on identification accuracy [$F(1,99) = 12.19$, $p < 0.001$, $\eta_p^2 = 0.11$], as well as the reaction time [$F(1,99) = 5.37$, $p = 0.023$, $\eta_p^2 = 0.05$]. In this ANOVA, the number of features also increased guessing [$F(1,99) = 4.41$, $p = 0.038$, $\eta_p^2 = 0.04$]. All other measures– localisation error, misbinding and imprecision – were unaffected, as previously. There were also no significant interactions with identification accuracy, localisation error, misbinding, guessing or reaction time. However, there was a significant interaction of the two main factors in the imprecision measure [$F(1,99) = 3.98$, $p = 0.049$, $\eta_p^2 = 0.04$]. Participants performed significantly better with one repeated feature in the retrieval cue with set size 2 [t(100) = -2.61, p = 0.011] but not with the filtering condition [t(100) = 0.13, p = 0.898]. The three-way interaction (group x set size x number of features) was not significant but visual inspection of Figure 6 shows that elderly adults were generally less precise in the filtering condition and it is possible that their precision was already at floor in the set size two, two features condition.

Young vs older participants

Young participants did not differ significantly from older people on identification accuracy but were significantly better on localisation error [$F(1,99) = 16.39$, $p < 0.001$, $\eta_p^2 = 0.14$]. They also guessed significantly less [$F(1,99) = 10.06$, $p = 0.002$, $\eta_p^2 = 0.09$], were more precise [$F(1,99) = 23.22$, $p < 0.001$, $\eta_p^2 = 0.19$] and faster [$F(1,99) = 52.14$, $p < 0.001$, $\eta_p^2 = 0.35$] than their elderly counterparts.

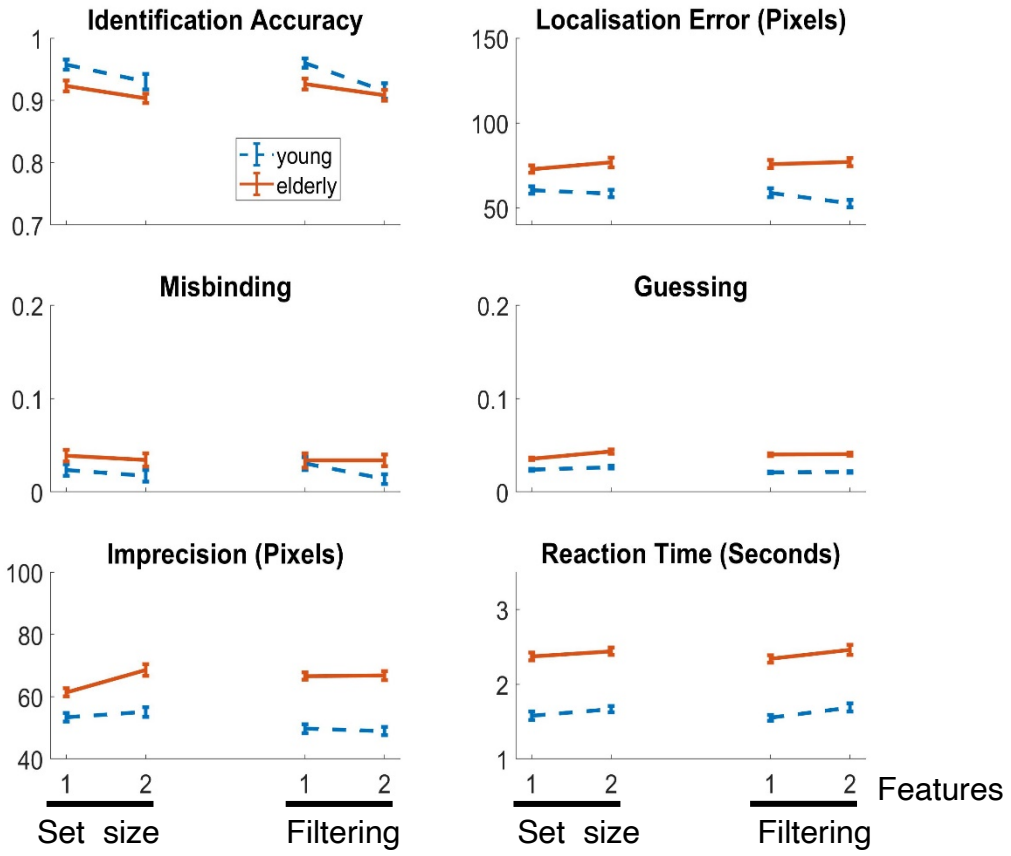


Figure 4.6| Impact on memory recall of filtering out one out of three items.

Participants performed at the same level when seeing only two items compared to when three items were presented of which one item was to be filtered out. Hence, they were able to successfully filter out an item at encoding. Error bars were calculated by subtracting each subject's grand mean away from their individual per-condition values and showing ± standard error (Loftus & Masson, 1994).

However, there was no significant difference between the groups on misbinding. Age group interacted significantly with the number of features in localisation error [$F(1,99) = 4.20$, $p = 0.043$, $\eta_p^2 = 0.04$, **Appendix Table 4.17**] and filtering with respect to imprecision of recall [$F(1,99) = 7.15$, $p = 0.009$, $\eta_p^2 = 0.07$, **Appendix Table 4.20**], but with none of the other factors or interactions.

Following up on the first of these interactions, neither within the group of young nor in older participants was there any significant difference between the filtering and set size two conditions [F(1,39) = 2.13, p = 0.153; F(1,60) = 0.30, p = 0.588, respectively] or between one or two repeated features in the retrieval cue [F(1,39) = 2.61, p = 0.114; F(1,60) = 1.57, p = 0.215, respectively]. With regard to the second interaction, within the young group, imprecision was significantly lower with the filtering condition [$F(1,39) = 10.87$, $p = 0.002$, $\eta_p^2 = 0.22$], but the number of repeated features did not affect imprecision. In the older group, however, although filtering did not affect imprecision, the number of old features in the foil did [$F(1,60) = 4.55$, $p = 0.037$, $\eta_p^2 = 0.07$]. In addition, there was an interaction of these two factors [$F(1,60) = 4.56$, $p = 0.037$, $\eta_p^2 = 0.07$] which shows that older participants did worse with two repeated features in the set size two condition (t(60) = -2.66, $p = 0.010$) but not in the filtering condition [t(60) = - 0.10, $p = 0.924$, **Appendix Table 4.22**]. However, **Figure 4.6** (bottom left graph) suggests that this might originate from performance being generally low in the filtering condition.

In summary, concerning our third research question, participants successfully filtered out irrelevant information at encoding, performing at equal levels for the set size 2 and the filtering condition (3 items with one item to be filtered out) in all metrics. Yet, filtering diminished the advantage of one repeated feature in the retrieval foil compared to two repeated features in precision. With respect to our fourth question, as for the previous analyses, there was mostly no selective main effect of age. Aging did lead to overall worse performance, except for the identification accuracy and misbinding which were not significantly different between age groups.

4.3. *Experiment 2*

In the second experiment we focused on processing demands during maintenance (effects of ignoring and updating). The key questions we sought to answer were:

1. Does ignoring during maintenance (filtering irrelevant items presented during retention) have a detrimental effect on recall measured in Identification Accuracy and Localisation Performance?
2. How does updating the contents of memory during retention affect recall measured in Identification Accuracy and Localisation Performance?
3. What is the impact of ignoring (filtering during maintenance) or updating with aging in Identification Accuracy and Localisation Performance?

4.3.1. Methods

4.3.1.1. Participants

Participants in this experiment were the same as in Experiment 1 (demographics in Table 4.1).

4.3.1.2. Stimuli, Design and Procedure

Stimuli and procedure were similar as to those in Experiment 1, except for the following changes. In Experiment 2, information during the maintenance interval was manipulated (Figure 4.7). As per Fallon et. al. (2018) each trial began with the presentation of two-coloured circles (4 seconds) that appeared in separate spatial locations, with the letter "T" appearing in the centre of the screen. After a blank screen delay (2 seconds), two different circles appeared on the screen. These could be accompanied either by a central "T" or simply a fixation cross. The presence of a central letter "T" indicated that the original stimuli held in memory should now be jettisoned, and these new circles remembered (Update condition). Conversely, the presence of a fixation cross meant that these new circles had to be ignored, i.e., ignore the new set of items and continue to remember the previous set (Ignore condition). Then, after another delay period (2 seconds) two further circles were presented, one of which was a circle that had to be remembered (target) and the other one a foil, the colour of which was not presented before within the trial (Figure 4.8). As in Experiment 1, after touching the target shape, participants had to drag the circle to its remembered location. We also included two control conditions with only one set of items and a short or long delay that matched the delays following the stimuli ultimately to be remembered in both ignore

and update conditions (**maintain long** and **short** conditions). In total, there were 4 randomised conditions of which participants performed 10 trials each, adding up to one block of 40 trials.

In this experiment, unlike to Experiment 1, six equally spaced-out colours were taken from a different colour wheel, avoiding same combinations across all trials.

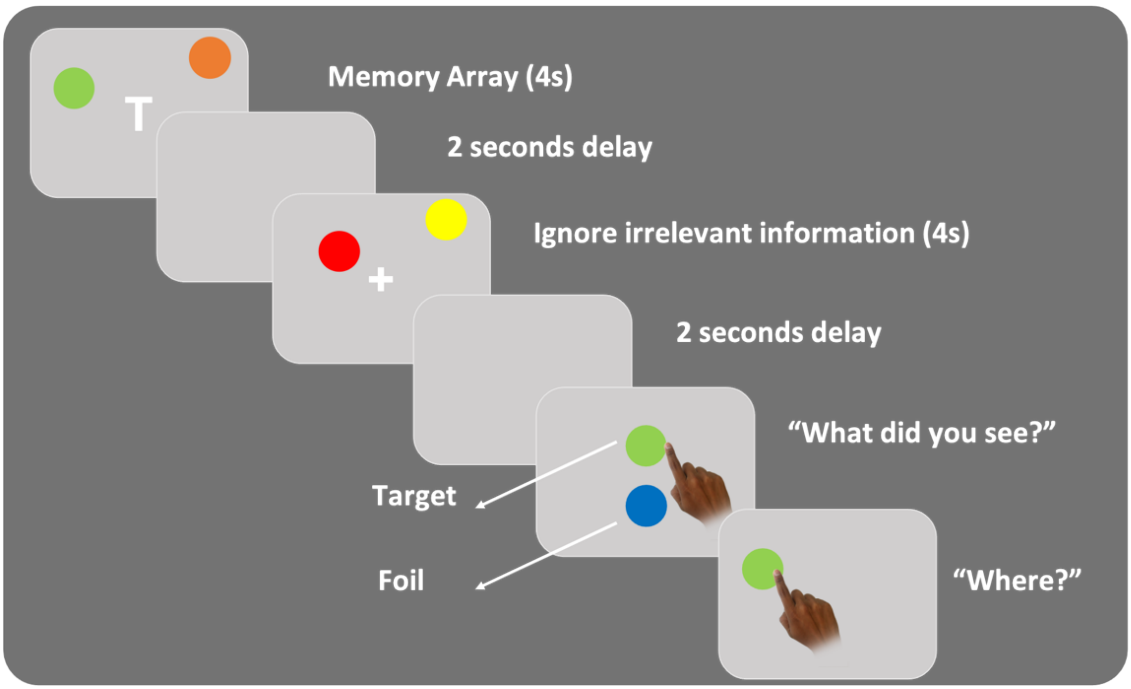


Figure 4.7| Schematic of Experiment 2: Manipulations of retrieval and filtering.

Participants were required to remember only the coloured circles when the letter 'T' (for 'targets') was present at screen centre. Moreover, they were told that only the most recent circles presented with a 'T' needed to be kept in memory. Later, they had to select the circle which was last presented with a 'T' and drag it to its remembered location.

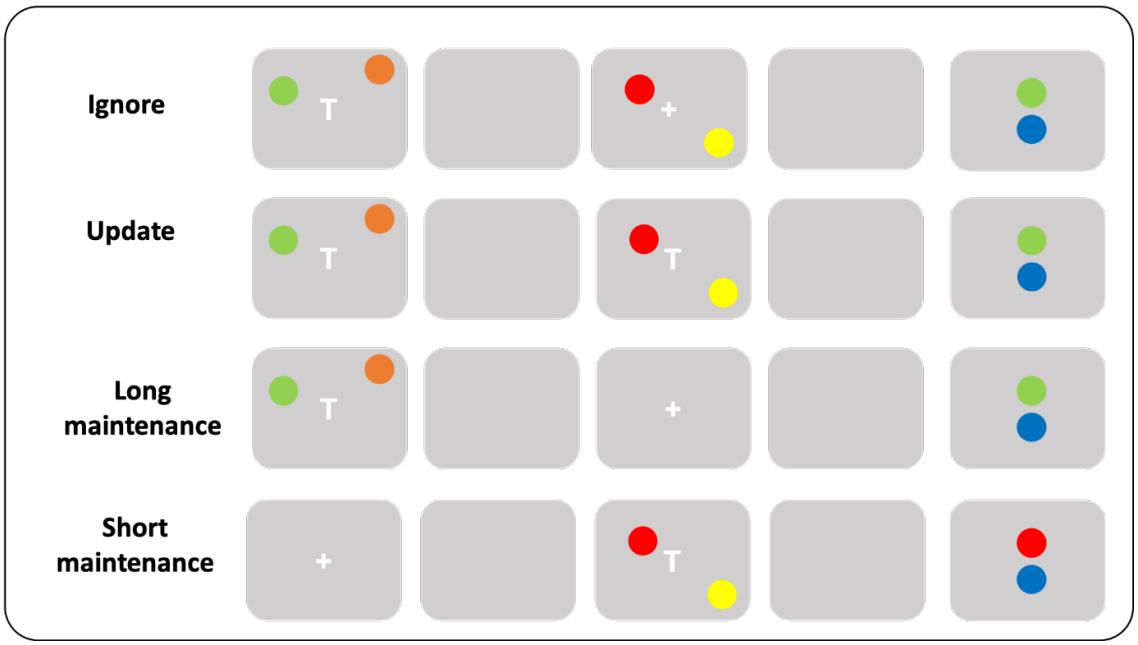


Figure 4.8| Schematic of all conditions for Experiment 2.

In the Ignore condition, participants had to retain information whilst ignoring an irrelevant pair of circles presented during maintenance. In contrast, in the Update condition, participants were presented with two pairs of circles consecutively, both of which are presented with the letter "T". They had to remember the last pair of circles and discard the previous pair. Two temporal control conditions were also used. They did not feature any irrelevant memoranda but differed only in the length of time for which items needed to be retained. The Long maintenance condition served as the temporal control for the Ignore condition, whereas the Short maintenance condition served as the temporal control for the Update condition.

4.3.2. Results

4.3.2.1. Impact of ignoring items during retention delay

A 2x2-ANOVA was conducted comparing performance in the ignoring versus the long maintenance delay conditions between the two age groups (Figure 4.9). When participants were asked to ignore two items during the retention period, their performance suffered significantly on several parameters: identification accuracy was lower [$F(1,99) = 7.63$, $p = 0.007$, $\eta_p^2 = 0.7$], they guessed more [$F(1,99) = 6.50$, $p = 0.012$, $\eta_p^2 = 0.06$] and misbinding increased [$F(1,99) = 69.89$, $p < 0.001$, $\eta_p^2 = 0.41$]. On average,

misbinding increased from 4.67% in the simple maintenance condition (when participants dragged the correctly identified target to the location of the other item that had appeared in the memory display) to 16.51% in the ignore condition. Most of the misbinding in the ignore condition (mean 66.4%; SD = 18.2%) was to the location of one of the to-be-ignored items, demonstrating the significant effect of these distractors, even on trials in which the target was correctly identified. Paradoxically there was a slight decrease in imprecision in the ignore condition compared to simple maintenance [$F(1,99) = 4.84$, $p = 0.030$, $\eta_p^2 = 0.05$]. Neither localisation error nor reaction time were significantly different between the two conditions.

Unlike in Experiment 1, there was no significant difference between young and old participants for any of our metrics (identification accuracy, localisation error, misbinding, guessing, imprecision or reaction time). The between subject factor group did not interact significantly with the factor ignore in any of these measures. Thus, ignoring was not significantly affected by age, under these experimental conditions (**Appendix Tables 4.23 – 4.28**).

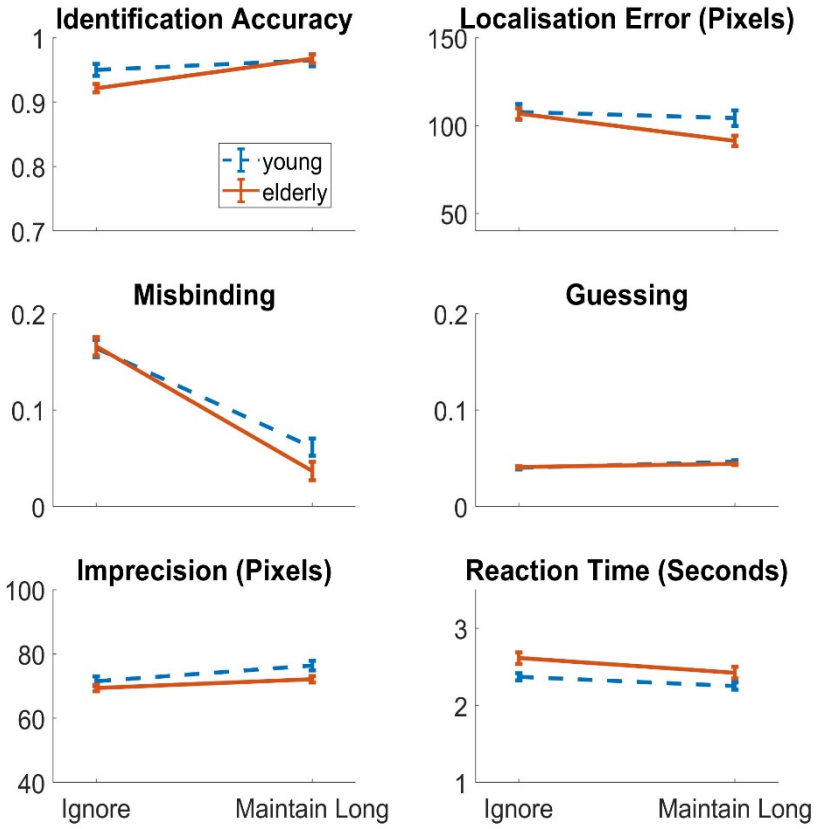


Figure 4.9| Impact on memory recall of filtering during maintenance (Ignoring).

Ignoring decreased identification accuracy, but increased misbinding and guessing. Effects were independent of age with elderly and young participants performing at similar levels. Error bars were calculated by subtracting each subject's grand mean away from their individual per-condition values and showing ± standard error (Loftus & Masson, 1994).

In summary, with respect to our first question, the findings show that ignoring information during the maintenance delay has a significant detrimental effect on recall. Filtering irrelevant items during retention affects identification accuracy, misbinding and guessing. With respect to our third research question, age did not interact with these effects; in this experiment, both age groups performed similarly.

4.3.2.2. Effect of updating contents of memory during retention delay

As for the Ignore condition, we conducted another 2x2-ANOVA of updating versus maintaining (this time for the short retention delay) and the between subject factor group (Figure 4.10). When participants were asked to update the contents of memory, there was no effect on identification accuracy but there was a significant increase in localisation error [$F(1,99) = 4.46$, $p = 0.037$, $\eta_p^2 = 0.04$], due to greater misbinding ($F(1,99) = 10.55$, $p = 0.002$, $\eta_p^2 = 0.10$). But participants were faster [$F(1,99) = 4.57$, $p = 0.035$, $\eta_p^2 = 0.04$]. In 82.42% (SD = 9.34%) of the cases, the item that was misbound to was *from the initial display*, demonstrating lingering effects of a previously retained (but now jettisoned) item on recall. Updating did not significantly impact on guessing or imprecision. As in the ignore condition, both groups performed at similar levels for identification accuracy, localisation error, misbinding, guessing, imprecision and reaction time. The between subject factor group did not interact with the factor update in any of the measures. Thus, like ignoring, updating was not significantly affected by age (Appendix Tables 4.29 – 4.34).

In summary, pertaining to our second research question, the results show that updating the contents of memory during retention did significantly impair localisation performance, with this being driven by increased misbinding to one of the original memoranda that should have been over-written by a new item.

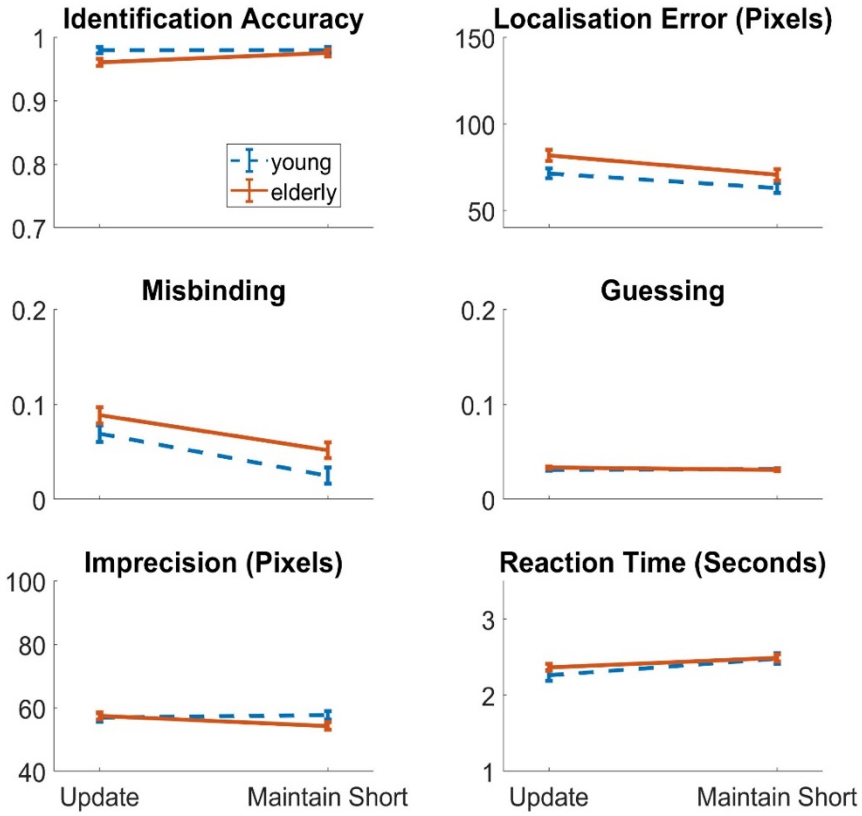


Figure 4.10| Impact on memory recall of Updating.

Updating increased localisation error driven by an increase in misbinding. However, participants were faster when updating. These effects were again independent of age. Error bars were calculated by subtracting each subject's grand mean away from their individual per-condition values and showing ± standard error (Loftus & Masson, 1994).

4.3.2.3. Effect of time — long vs short delay

A 2x2 ANOVA of delay (long vs short) and the between subject factor group was conducted (**Figure 4.11**). A longer delay did not significantly affect identification accuracy but it did increase localisation error [$F(1,99) = 53.94$, $p < 0.001$, $\eta_p^2 = 0.35$], driven by guessing [$F(1,99) = 52.54$, $p < 0.001$, $\eta_p^2 = 0.35$] and imprecision [$F(1,99) = 116.37$, $p < 0.001$, $\eta_p^2 = 0.54$]. Misbinding and reaction time were unaffected by a main effect of longer delay. Again, the two groups performed equally well across all the metrics we

studied (identification accuracy, localisation error, misbinding, guessing, imprecision or reaction time).

Delay duration interacted significantly with the between subject factor group on localisation error [$F(1,99) = 5.91$, $p = 0.017$, $\eta_p^2 = 0.06$]. Therefore, we followed up on this using pairwise t-tests within both groups individually. Both older and young participants had a greater localisation error with longer delay [$t(60) = 3.99$, $p < 0.001$); ($t(39) = 6.10$, $p < 0.001$, respectively]. In addition, group also interacted with the delay effect on misbinding [$F(1,99) = 9.90$, $p = 0.002$, $\eta_p^2 = 0.09$]. For this reason, we performed pairwise t-tests in each of the groups again. There was no difference between a short and long delay in misbinding for older participants [$t(60) = -1.57$, $p = 0.122$]. However, in the young group, misbinding increased more for longer delays [$t(39) = 2.58$, $p = 0.014$] than it did in their older counterparts. In younger controls the increase of misbinding in longer delays could have also contributed to the increase of localisation error for longer delays (Appendix Tables 4.35 – 4.40).

The results show a significant decay of location memory over longer delays, driven by an increase in guessing and imprecision.

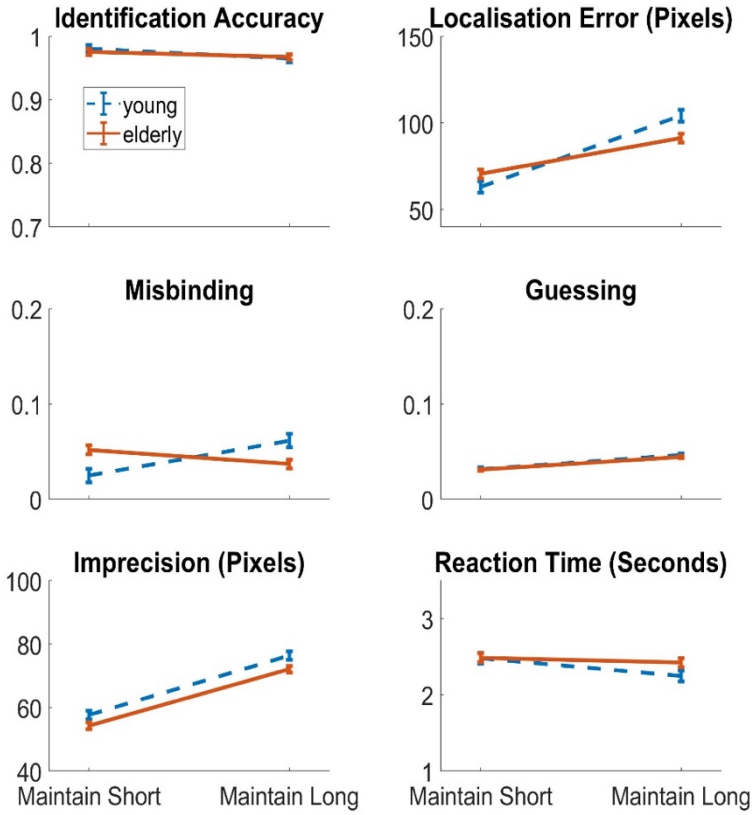


Figure 4.11| Impact on memory recall of Delay.

Longer delay increased localisation error in both young and elderly groups. Error bars were calculated by subtracting each subject's grand mean away from their individual per-condition values and showing ± standard error (Loftus & Masson, 1994).

4.3.2.4. Relationship between Updating and Ignoring

In a final step, the relationship between Updating and Ignoring performance was examined to assess whether these were correlated. For Identification accuracy, Imprecision and Misbinding: (i) participant-wise difference between the long delay trials and the ignore condition and (ii) participant-wise difference between the short delay trials and the update condition were computed. There were no significant correlations between these indices of Updating and Ignoring for either Identification accuracy or Imprecision [identification accuracy: r = 0.08, p = 0.412; imprecision: r = 0.17, p = 0.086],

suggesting that effects of ignoring and updating were independent of one another in these measures. However, there was a significant correlation between Updating and Ignoring performance for Misbinding [r = 0.24, p = 0.015]. This result suggests that the ability to bind features of an object together might depend on a single system, regardless of whether information has to be protected (Ignore condition) or jettisoned to store new information (Update).

4.4. *Filtering at encoding, maintenance and processing requirements induced by retrieval across experiments*

In the final analysis step, we examined the relationship between several parameters that pertain to processing demands at encoding, maintenance and processing demands due to retrieval across Experiments 1 and 2. In this analysis, we included the most important investigated measures. These were i) the quantitative WM result of identification accuracy, ii) misbinding and iii) imprecision which were the main sources of error of the qualitative WM task.

First, scores were calculated for the effect of the following on different performance measures:

- Filtering at encoding (Encoding): Difference between Set Size 2 versus the Filtering condition in which the foil's features were taken from the non-filtered item, collapsed over the number of features, in Experiment 1. *This provides a general measure of filtering at encoding.*

- **Filtering during maintenance** (**Maintenance):** Difference between the maintain long and the ignore condition in Experiment 2. *This is an index of filtering during maintenance.*
- **Processing requirements induced by the retrieval cue** (**Retrieval):** Difference between one- and two-repeated-features conditions, collapsed over all other conditions in Experiment 1. *This provides a measure of interference introduced by the retrieval cue.*
- **Set size:** *Difference between Set Size 2 and Set Size 3 conditions, collapsed over the number of features, in Experiment 1. This is an index of the impact of memory load.*

Next, correlation matrices for these four scores were examined separately for the Identification Accuracy, Misbinding and the Imprecision (**Figure 4.12**; details in **Appendix Tables 4.42 – 4.44**). Analyses revealed a significant positive correlation between *Set size* and *Encoding* across all three metrics [Identification Accuracy: r (99) = 0.52, $p < 0.001$, Misbinding: r(99) = 0.25, $p = 0.011$ and Imprecision: r(99) = 0.44, $p < 0.001$]. Thus, the more participants were affected by a manipulation of set size (difference between *Set Size 2 and 3*) the stronger they were also affected by a manipulation of processing demands at encoding (difference between *Set Size 2* and the filtering condition in which the foil's features were taken from the non-filtered item), and vice-versa. Most importantly, there were no significant correlations between filtering at encoding, maintenance and processing requirement induced by the retrieval cues across any of the three metrics of Identification accuracy, Misbinding and Imprecision. This suggests that,

within the limitations of this study, there was independence of filtering performance at encoding, maintenance and retrieval.

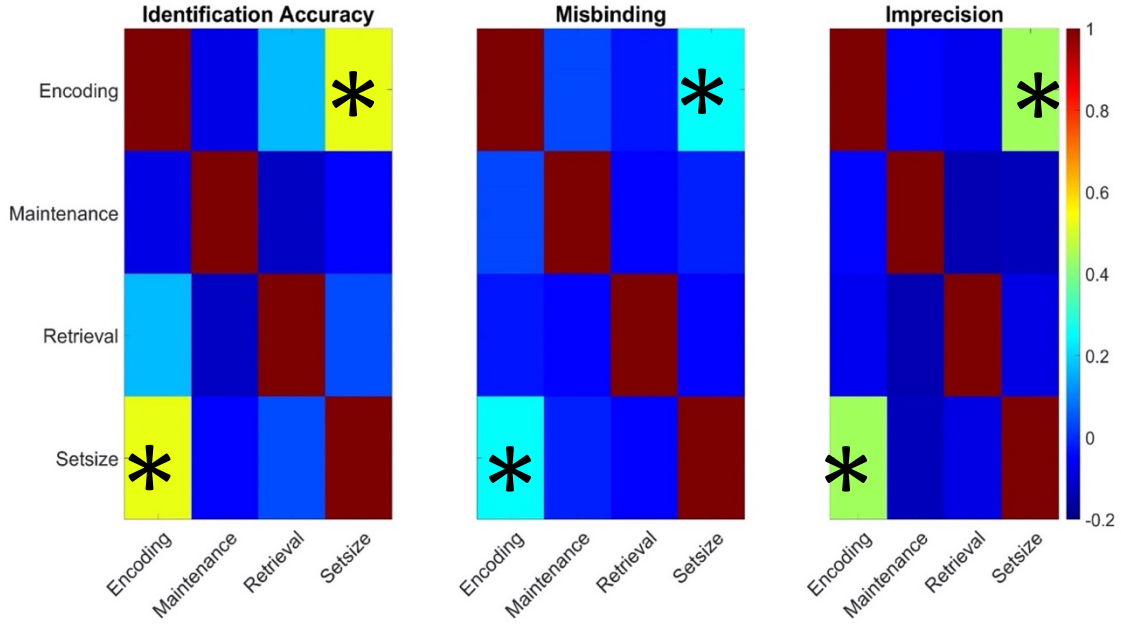


Figure 4.12| Correlation matrices of filtering at encoding, maintenance and retrieval, and set size.

Set size and processing demands at encoding were strongly correlated, but there were no significant correlations between filtering at different phases of WM – encoding, maintenance and retrieval. Significant correlations are indicated with an asterisk and df = 99.

4.5. *Discussion*

Although several previous investigations have examined the effect on WM of processing demands at encoding and maintenance, there has been little work on such demands induced by the retrieval cue or how this is related to the impact of processing demands at encoding and maintenance. In the present study, we conducted two experiments to examine how recall performance is affected by processing requirements induced by retrieval and to assess the relationships between such demands at encoding, maintenance and retrieval. In Experiment 1, we investigated the effects of filtering

irrelevant information at encoding and the impact of distracting information at retrieval by presenting a foil that could either contain one or two old features from the encoding display (either from the task-irrelevant or a non-filtered item). In Experiment 2, we explored the effect of ignoring - filtering during the retention period - by asking participants to ignore irrelevant information during the maintenance interval. Or, in an updating condition, they were asked to jettison and replace previously stored information. Across the two experiments, a secondary aim of the study was to examine how the effect of filtering at different stages of WM is affected by age.

The results from Experiment 1 showed that the nature of processing demands induced by the retrieval cue does indeed impact significantly on memory performance. Increasing the number of old features from an item presented in the encoding display in the retrieval foil decreased participants' ability to correctly identify an item (**Figure 4.4**). For example, it was harder for participants to correctly identify an item when the target was presented together with a foil containing the same two colours from another item they saw in the encoding display. These results expand on previous work which shows that adding unnecessary information in the probe (Tabi et al., 2019), and the presence of distractors at the time of probe (Makovski et al., 2010; Udale et al., 2017), disrupts performance. Including partial information from other representations currently maintained in memory negatively impacts recall performance.

These findings also align well with a recent computational neural model of WM (Manohar et al., 2019) which uses rapid synaptic plasticity to encode combinations of features represented in feature neuron layers as bound objects in flexibly-coding conjunction neurons. During recall, activation of a single feature neuron can trigger

pattern completion by guiding the activation down the strengthened synapses to the appropriate conjunction neuron that, in turn, activates the rest of the features from its joined representation. The model predicts that if competing information is presented in the retrieval cue, i.e., information that triggers the activation of multiple conjunction units, retrieval will suffer. However, this should be more evident if the irrelevant information is represented in WM. In line with this prediction, we found that when the retrieval foil had features that came from a task-irrelevant item that was supposed to be filtered out at encoding, participants identified the correct item more often and were faster at making this choice. This shows that filtering at retrieval interacts with processing demands at encoding.

A limitation to the retrieval cue manipulation, however, is that the results observed cannot exclusively be linked to effects that are at or due to retrieval. The retrieval cue manipulations affect the degree of similarity between foil and target and, thus, require participants to solve a computational problem that might increase with an increasing similarity of the choices available. In the signal detection framework, for example, similarity between the available choices at retrieval is claimed to explain participants' accuracy in the task (Schurgin, Wixted, & Brady, 2020). Thus, in trials in which target and foil were more similar, an increased number of false-alarms can be expected without any particular effects associated to retrieval. This would also be the case if participants would compute wrong colour-colour bindings at encoding to begin with and then consecutively fail at retrieval.

In Experiment 1, participants were able to successfully filter out an irrelevant item at encoding because they performed just as well as when presented with only two items

to encode, compared to when they were presented with three items but one had to be filtered out (task-irrelevant item) (**Figure 4.5**). However, ignoring irrelevant information during maintenance had a detrimental effect on recall (Experiment 2), leading to an increase in identification accuracy, misbinding rates and guessing (**Figure 4.9**). Moreover, updating the contents of memory during retention affected recall: Localisation performance was impaired driven by an increase of misbinding to one of the original memoranda's locations that should have been over-written by a new item (**Figure 4.10**). Unlike the results of a previous study (Sean J. Fallon et al., 2018) which reported an increase in misbinding solely to the to-be-ignored distractors in the Ignore condition, we also found an increase in misbindings to the to-be-updated item in the Update condition. Furthermore, performance in the Ignore and Update conditions were significantly correlated with respect to misbinding.

In line with previous work in which filtering at encoding has been closely related to WM capacity (McNab & Dolan, 2014; Vogel & Machizawa, 2004; Vogel et al., 2005), the results presented here show that filtering at encoding is related to the set size effect, i.e., participants with greater WM capacity were less affected by irrelevant information at encoding (**Figure 4.12**). This result is consistent with the proposal that individuals' WM capacity is related to their ability to filter irrelevant information at encoding (McNab & Dolan, 2014; McNab & Klingberg, 2008; Vogel & Machizawa, 2004), suggesting that attention serves as a gatekeeper for WM, biasing the encoding of information towards relevant memoranda (E. Awh, Vogel, & Oh, 2006). Importantly, these findings assume a direct relation between WM capacity and set size such as a decrease of available resource in the resource model of WM (Paul M. Bays et al., 2009) or in the slot model where $K = S$

(H-F) where *K* is the capacity, *S* the set size, *H* is hit and f the false alarm rate (McNab & Dolan, 2014; Vogel et al., 2005).

A key finding of the studies reported here is that there were no significant correlations between the ability to filter at encoding, maintenance, or at retrieval (**Figure 4.12)** suggesting that there is no single, monolithic mechanism responsible for all these processes. Thus people who are good at filtering information at one of the memory stages are not necessarily good at filtering at the other two. Of course, although we did not find a significant relationship between filtering at different memory stages, a negative result does not mean that it might not exist if tested for differently. One possible explanation for the lack of correlation between filtering efficiency at encoding, maintenance and retrieval might be the fact that the processing of information has to operate in different ways at each stage and interacts with different types of representation. During encoding, attention is considered to operate on perceptual information to filter out distractors and to selectively encode targets. During maintenance, it has been invoked as a mechanism to protect current WM contents from distraction if the information is irrelevant or to update existing contents if information is relevant. Finally, processing demands induced by the retrieval cues, as tested here, serve to select the appropriate response while filtering out irrelevant information that interferes with the process of recall.

An alternative explanation is that, if these are indeed truly unrelated metrics, different brain mechanisms and regions might be involved in each process. In support of this view, there is some evidence from imaging studies that have investigated neural correlates of protecting the contents of WM that have shown evidence for separate

mechanisms for distractor-filtering at encoding and during the maintenance interval (R. Cools, Miyakawa, Sheridan, & D'Esposito, 2010; Sean James Fallon & Cools, 2014; Gruber, Dayan, Gutkin, & Solla, 2006; McNab & Dolan, 2014; McNab & Klingberg, 2008; Mehta, Manes, Magnolfi, Sahakian, & Robbins, 2004). For example, some researchers have observed an improvement of distractor filtering during the maintenance delay as a result of a deficit in striatal dopamine (R. Cools et al., 2010; Mehta et al., 2004), whereas other studies have reported that presenting distractors during encoding is linked to prefrontal and basal ganglia activity, particularly in the globus pallidus (McNab & Klingberg, 2008). In line with this, Gruber and colleagues (2006) have posited that, since the basal ganglia have a high density of dopamine receptors critical to WM, dopamine can carry out a gating function by transiently strengthening the efficiency of inputs to the frontal cortex, and have modelled the effects of dopamine in the basal ganglia as gating stabilization against distractors by improving specific memories.

Previous studies have also consistently shown that aging is linked to a general deterioration of performance on WM tasks (Chen et al., 2003; Jost et al., 2011; McNab & Dolan, 2014; McNab et al., 2015; Myerson et al., 2003; Peich et al., 2013; Pertzov et al., 2015). We therefore tested whether age similarly correlated with general WM performance decrement. Evidence from manipulations of processing demands during encoding and processing demands due to the retrieval cues (Experiment 1) shows that elderly participants performed generally worse compared to young controls (**Figure 4.4, Figure 4.5, Figure 4.6**). When the effects of processing demands at the different memory stages were directly compared between young and elderly, filtering at encoding and set size had a greater negative effect on elderly participants compared to younger ones.

A few limitations of this study design need to be taken into consideration. Although the experiments were conducted on a single group of young and elderly participants – enabling examination of effects of aging on filtering at various stages, as well as a look at correlations amongst abilities to filter during encoding, maintenance, and retrieval – the use of slightly different memoranda across Experiment 1 and 2 might have had an impact on the results and their interpretation. Also, in Experiment 2, to make sure participants were solving the right computational problem (ignoring, updating, or maintaining information in WM), at the end of each trial a display with the question: "What have you just performed?" together with three possible answers: "Ignored irrelevant information", "Updated new information", "Maintained the same information" should be displayed. At the beginning of the experiment, participants would be instructed to choose from one of the three options displayed at the end of each trial. When analysing the data, this would help ensure that participants were indeed performing the task at hand, and that failure to correctly identify an item was not due to the inability to distinguish between the signs "T" and "+" that designated which trials were to be updated or ignored, respectively.

In conclusion, the findings of these studies provide evidence that additional irrelevant information presented at retrieval has a detrimental impact on performance and that the efficiency of filtering during encoding has an impact on recall. Moreover, filtering efficiency is associated with memory capacity. Finally, the findings point to the absence of a unified filtering system that operates across all phases of WM. Instead, evidence from the two experiments suggest that filtering at encoding, maintenance and retrieval are not related and might be underpinned by different cognitive processes.

Chapter 5. Hippocampal contributions to feature binding in working memory

Abstract

Traditionally, the hippocampus and related MTL structures have been considered to be essential for LTM, but not for WM. However, patients with MTL damage have been shown to be impaired in remembering relational information across delays. To investigate the role of the MTL in memory, specifically the nature of memory deficit resulting from hippocampal damage, a cohort of 28 LE patients was examined and compared to 28 age and gender-matched controls on a delayed reproduction STM task with a continuous measure of report. In addition, behavioural metrics were correlated with hippocampal volume (in 13 patients and 28 controls).

Behavioural results provided evidence for disease-related impairment in correctly identifying memoranda. Patients' localisation performance was also significantly worse than controls, and specifically attributable to an increase in the number of errors due to misbinding – the inability to bind an object to its location – but not by guessing. Structural imaging results show that patients have smaller hippocampi compared to controls and that misbinding negatively correlates with left hippocampus volume. Hippocampal subfield analysis revealed that misbinding was particularly associated to regions left CA4 and left molecular layer.

Together, these results contribute to the understanding of how the MTL participates in relational WM binding and raise the question of whether binding deficits might also be accompanied by remote effects on the function of the extended hippocampal system.

5.1. *Introduction*

One function attributed to the hippocampus is that of binding information together by maintaining and flexibly relating separate aspects of a memory that might be represented in different parts of the neocortex (Cashdollar, Duncan, & Duzel, 2011; Davachi, 2006; Howard Eichenbaum, 2006; Libby et al., 2014; Yonelinas, 2013). Even though, conventionally, associative binding has been considered relevant in LTM (Davachi, 2006; Davachi & Wagner, 2002; Howard Eichenbaum, 2006; Fernández & Tendolkar, 2001; Rawlins, Deacon, Yee, & Cassaday, 1994; Scoviille & Milner, 1957) the MTL, particularly the hippocampus, have been shown to be active during retention of information over short delays (Axmacher et al., 2007; Hannula & Ranganath, 2008; Piekema, Kessels, Mars, Petersson, & Fernández, 2006; Ranganath & Esposito, 2001). For example, an early study with MTL patients using serial recall could not reconcile their findings with a pure LTM impairment as they found a significant increase of intrusion errors from prior lists (A D Baddeley & Warrington, 1970). Graham and colleagues' review also highlighted the MTL contribution to binding and associative processes in general and not exclusively to declarative LTM (Graham, Barense, & Lee, 2010).

To investigate the role of the MTL in memory, specifically the nature of memory deficit resulting from hippocampal damage, focal hippocampal atrophy due to LE patients serves as a good model of human hippocampal lesions (Fox, 1996; Khan et al., 2009; Vincent et al., 2011). LE in patients with Abs-VGKC, mainly directed to the VGKC-complex proteins LGI1 or CASPR2 can display inflammatory changes of the MTL on acute neuroimaging (Ances et al., 2005; Bettcher et al., 2014; Butler et al., 2014; Harrower, Foltynie, Kartsounis, De Silva, & Hodges, 2006; Kotsenas et al., 2014; Reid et al., 2009;

Vincent et al., 2011), and focal, acute hippocampal pathology (Argyropoulos et al., 2019; Chan et al., 2007; Finke et al., 2017; Malter et al., 2014).

It has further been suggested that hippocampal atrophy in LE patients following early and later intervention is selective, affecting bilateral CA3, occurring in the absence of significant grey matter loss in brain regions outside the hippocampus (T. D. Miller et al., 2017). Other lines of evidence support this; for example, post-mortem studies demonstrate focal hippocampal damage in autoimmune LE patients (Khan et al., 2009). Animal studies have also shown focal limbic involvement (hippocampus, subiculum, amygdala and piriform lobe) in feline animal models (Tröscher et al., 2017). Additionally, in rodents, autoantibody binding patterns have been described as part of the pathophysiological cascade that involves VGKC- complex expression in the hippocampus (Vincent et al., 2011). Moreover, in their study, Irani et al. show that the expression of autoantibodies LGI1 and CASPR2 are favoured in CA3 rodent hippocampal subfields (Irani et al., 2010). These findings are in line with reports suggesting LGI1 gene expression to be very restricted to intrahippocampal circuitry (Herranz-Pérez, Olucha-Bordonau, Morante-Redolat, & Pérez-Tur, 2010), with greater neuronal loss in CA3 than in the CA1 region following seizures in a germline knockout mice for LGI1 (Chabrol et al., 2010).

Pertzov and colleagues (2013) were the first to use this unique cohort of patients with LE to examine visual STM performance although previous studies have investigated other cognitive aspects in this population (Binks et al., 2021; Chan et al., 2007; Hartley et al., 2007; Maguire, Nannery, & Spiers, 2006). They used a continuous measure of report (Gorgoraptis et al., 2011; Pertzov et al., 2012; Weiwei Zhang & Luck, 2008) to investigate the nature of STM deficits that result from MTL damage by examining not just the

frequency but also the quality of recall and types of errors made in seven LE patients (Paul M. Bays et al., 2009; Paul M. Bays & Husain, 2008a). These authors found that, following short delays, memory impairment in these patients was due to an increased number of misbinding errors, i.e., the inability to relationally bind the identity of memoranda with its location (Pertzov, Miller, et al., 2013).

The results on the nature of memory deficit in LE (Pertzov, Miller, et al., 2013) are in concordance with other lesion studies regarding MTL dysfunction in which patients have been shown to be impaired on tasks that required maintenance of associations and feature conjunctions, over short delays (Banta Lavenex, Lavenex, Colombo, Lambert, & Lavenex, 2014; Finke et al., 2007; Finke, Bruehl, Düzel, Heekeren, & Ploner, 2013; Hannula, Tranel, & Cohen, 2006; Olson, Page, et al., 2006; Zokaei & Husain, 2019a; Zokaei et al., 2020). This conceptualization of the nature of the memory deficit in LE patients converges with the results of other studies on people at risk of developing AD and MTL dysfunction, who might be asymptomatic carriers of a familial AD gene. They exhibit unhindered ability to maintain isolated features over few seconds but are significantly impaired when having to remember bound features that belong to a visual object (Liang et al., 2016; Parra et al., 2010, 2015).

In this study, we aim to test whether it is possible to replicate the findings from Pertzov et al. (2013) to show in a far larger group whether LE patients with focal damage to the MTL are specifically impaired in their ability to bind different features of the same memoranda. In the current investigation, twenty-eight patients clinically diagnosed with autoimmune LE and twenty-eight age matched controls were tested on the same "What was where?" WM task used in Pertzov's aforementioned study (2013). In this behavioural

task, undertaken on a touchscreen, participants were presented with a set of stimuli in different screen locations of which they were instructed to recall. At test, participants were presented with a two-alternative forced choice between the target and a distractor (never before seen memoranda). They were asked to select the target and drag it to its exact remembered location, providing a continuous measure of their memory recall performance but also an index of relational binding by looking at the frequency with which object-locations are swapped in memory (Liang et al., 2016). In the second part of this study, thirteen LE patients and twenty-eight age-matched controls previously tested on the behavioural task were scanned to acquire structural MRI images.

5.2. *Methods*

The aim of this study was to investigate the nature and type of errors arising from memory disorders associated with MTL damage by addressing the following questions:

1. What is the nature of the deficit in patients with hippocampal lesion? Is it a deterioration in memory capacity or simple degradation of memory resolution?
2. Is the memory error associated with MTL lesion reflected in increased misbinding, i.e., tendency to report features belonging to the wrong item?
3. Is the memory error associated with MTL lesion correlated with hippocampal volume and is this association selective to specific subfields of the hippocampus?

5.2.1. Participants, Demographics and Consent

Twenty-eight LE patients (7 female) and twenty-eight age and gender matched healthy controls were recruited through the specialist LE service at Oxford. Twenty-five of the patients were originally found to express LGI1 antibodies and three patients were found to be negative for both LGI1 and CASPR2. Patients included in this study were recruited post-acutely, after an initial assessment by a neurologist. Recruitment was on average done 3 years (SD = 2.7) after the initial diagnosis.

Demographics are presented in **Table 5.1**. Cognitive function screening was tested using ACE-III (Mathuranath et al., 2000). Patients did not differ from controls in terms of age or gender ratio. They had, however, significantly less years of full-time education and were more impaired in their cognitive function in terms of their memory and fluency.

Table 5.1 | Participant Demographics and baseline cognitive measurements. The amount of variation of the data set is quantify by ± SD.

Measure	Healthy elderly controls	Limbic Encephalitis patients	Controls versus Patients p-value
N	28	28	n/a
Age (years old)	63.8 (±8.5)	64.5 (±9.6)	0.781
Gender (F/M)	7/21	6/22	0.752[a]
Years of full-time education	17.3 (±2.9)	13.1(±3.1)	0.001[b]
Global Cognition (ACE-III score)	97.1 (±2.9)	91.9 (±7.0)	0.001[b]
Attention	17.7 (±0.8)	17.4 (±1.3)	.276
Memory	24.9 (±1.6)	23.1 (±3.1)	.009[b]
Fluency	12.9 (±1.4)	10.5 (±2.6)	<.001[b]
Language	25.8 (±0.5)	25.4 (±1.5)	.281[b]
Visuospatial	15.9 (±0.4)	15.5 (±0.9)	.241[b]

[a] Chi-squared test; [b] Mann-Whitney-U test was applied has the data was not normally distributed.

Permission for this study was obtained from the local ethics committee and all subjects gave written informed consent and only normal or corrected-to normal vision participants, not colour-blind, were included.

5.2.2. Stimuli, Design and Procedure

Stimuli were presented on a black background and were drawn from a library of 196 foils, randomly selected without repetitions for every trial. They were presented at a viewing distance of approximately 30 cm and subtended an angle of approximately 2.3°. The task featured four experimental conditions randomly presented, within 3 Blocks of 10 trials per condition. The number of trials per condition was determined in pilot studies to maximize the power of the analysis while keeping the task as short as possible.

Participants performed a variant of the "What was where" WM task (Pertzov et al., 2012). A schematic of the task is shown in **Figure 5.1**. In this task, two factors are manipulated. First, information to-be-encoded is varied by presenting either one or three fractals. Second, the delay period varied by presenting a blank screen for 1 or 4s. Thus, the task featured four experimental conditions. Within each of three blocks, 10 trials per condition were presented, leading to a total of 30 trials per condition.

Participants sat in front of an interactive touch-sensitive screen (iPad version 9.3.5 (13G36), model MGTX2B/A) with 1536x2048 pixel matrix. First, they were randomly presented with either 1 or 3 items (**Figure 5.1**). They had to remember what they looked like and where they were on the screen. The items then disappeared and, after a variable delay of 1 or 4 seconds, two shapes were presented. These consisted of a target (the item

they previously saw) and a distracter (a foil taken from the library of fractals used across the experiment but not presented in the memory array). When given this dual choice, participants had to touch the one they remembered from a few seconds before, then drag it to where it originally appeared. They were instructed to be as precise as possible, thus allowed to move the item and adjust its location as many times as they needed.

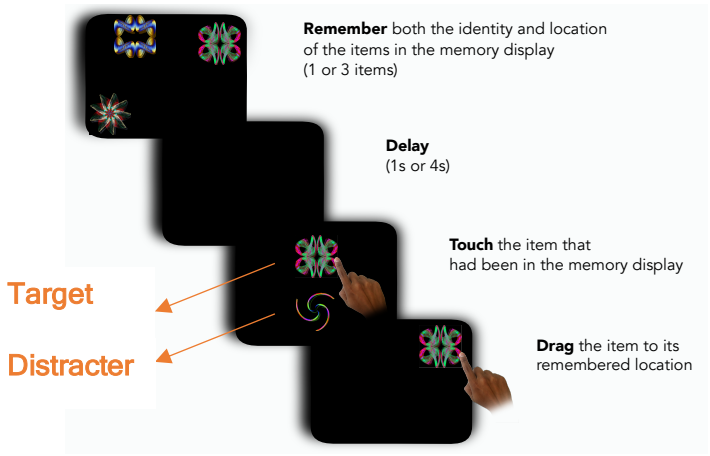


Figure 5.1| "What was where?" short-term memory task.

Participants are presented with either 1 or 3 items. They have to remember what they look like and where they are on the screen. After a variable delay (1s or 4s), a dual choice is presented. Participants have to touch the one they remember from a few seconds before, then drag it to where it originally appeared.

5.2.3. Behavioural Analysis and Mixture Model of error

This study used a delayed reproduction task allowing for the assessment of the fidelity of WM representations by measuring precision of recall (pattern of recall errors), which provided information on the quality of memory representations and enabled us to analyse the sources of errors contributing to the pattern of performance (P. M. Bays et al., 2011; Paul M. Bays et al., 2009; Paul M. Bays & Husain, 2008a; Gorgoraptis et al.,

2011; Ji Ma et al., 2014; Palmer, 1990; Wilken & Ma, 2004). Data was analysed in *MATLAB R2018b* using custom scripts and *JASP* version 0.12.1.

The proportion of trials in which participants correctly identified the target was defined as the **identification accuracy**, which was computed by taking the proportion of times participants touched the correct shape in the dual choice. The time taken to make a choice between the target and foil was defined as **reaction time**. The distance between the target's original location and the response was defined as the **localisation performance** and calculated by taking the Euclidian distance between the centre of the reported location and the centre of the original location of the probed item. Please note that the quality with which an item location is reproduced was calculated by solely considering trials where an object was correctly identified.

Errors due to misreporting the location of an item occurred as a result of different factors: imprecision, misbinding errors or errors due to guessing. These parameters were calculated using the extension to the *MemToolbox* of the mixture model analysis by Grogan and colleagues (Grogan et al., 2019), with **imprecision** referring to the width of the distribution around the target, representing the variability of responses around the target's location; **misbinding errors**, referring to the proportion of times subjects misreport the location of the correctly identified item by dragging it to the location of another item presented in the memory array; and **guessing**, corresponding to when participants randomly report the location on the screen.

Additionally, because years of full-time education and cognitive performance were significantly different between the two groups at test (**Table 5.1**), these metrics were added as covariates after being mean-centered using Delaney-Maxwell method of

rescaling (Delaney & Maxwell, 1981) to ensure that the covariates do not alter the main effect of "within-subjects" factors since the two are orthogonal. Years of full time education were controlled, firstly because this is a replication study and Pertzov (2013). reported data on only seven patients and their performance was compared to twelve aged and education-matched controls. Secondly, it is important to account for differences between the groups being due to their baseline mnemonic performance, since people with better mnemonic baseline abilities could be posited to have pursued more years of education. With regards to cognitive performance and subscales of the ACE, although both fluency and memory were impaired in patients compared to controls (Table 5.1), only fluency was added to the analysis as a covariate because the memory component of ACE already partially measures memory. Therefore, controlling for memory related impaired differences between the two groups would take away the effect of group in the behavioural task, i.e., the variance that explains differences in memory between the two groups in the "What was where?" task.

5.2.4. Neuroimaging Analysis

Thirteen LE patients (mean age: 62 ± 9.3, 7 male) previously tested on the behavioural task were scanned within 2 months (SD = 3.44) to acquire MRI images. Imaging data was also acquired for all twenty-eight age-matched controls (mean age: 62 ±8.68 years, 6 male) and was completed within 1 month after behavioural testing (SD = 1.52).

MRI images were acquired on a 3 Tesla *Siemens Verio* scanner at the Acute Vascular Imaging Centre at the John Radcliffe Hospital, Oxford, UK. The imaging sequence was aligned to the UK Biobank protocol, with more detail available elsewhere (Alfaro-Almagro et al., 2018; K. L. Miller et al., 2016). T1-weighted images were acquired using a magnetisation prepared rapid gradient echo protocol (MPRAGE) sequence in sagittal orientation (TR = 2000 msec, TE = 1.94 msec, TI = 880 msec, Flip angle = 8 degrees, FOV read = 256 mm, Voxel size = 1.0 x 1.0 x 1.0 mm). The same scanner was used to acquire T2-FLAIR images.

Other members of the Cognitive Neurology Lab research group pre-processed the data with FMRIB Software Library (FSL) according to the UK Biobank analysis pipeline (https://git.fmrib.ox.ac.uk/falmagro/UK_biobank_pipeline_ v_1) (Alfaro-Almagro et al., 2018). Additionally, they used FSL-FIRST (Patenaude, Smith, Kennedy, & Jenkinson, 2011) to calculate hippocampal volume for both hemispheres using T1 anatomical images and corrected for age and intracranial volume using FSL SIENAX. Finally, hippocampus subfields were decomposed using T1 and T2 image inputs using FreeSurfer (http://surfer.nmr. mgh.harvard.edu/).

Note that correlation plots were created using software *Grammar of graphics plotting* in *Matlab R2018b* (Morel, 2018).

5.3. *Results*

5.3.1. Behavioural Results

To investigate how LE affects WM identification accuracy, reaction time and identification error, a 2 x 2 x 2 repeated measures ANOVA, with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors, and years of full-time education together with the fluency domain of the ACE as covariates was applied.

Identification accuracy, i.e., the proportion with which participants touched the correct item in the two-alternative choice (Figure 5.2, Identification Accuracy), was worse when 3 items were presented compared to one [F(1,52) = 300.45, $p < .001$, $\eta_p^2 = .852$, $BF_{10} = .056$] and longer delays also lead to poorer identification recall [F(1,52) = 6.16, $p = .016$, $\eta_p^2 = .106$, $BF_{10} = 4.508e^{-42}$]. Additionally, patients identified the correct memoranda less often than controls, as per a significant effect of group [F(1,52) = 8.17, $p = 0.006$, $\eta_p^2 = .136$, $BF_{\text{incl}} = 4.35$]. Details in Appendix Table 5.1-5.2.

Both patients and controls took the same amount of time to correctly identify memoranda (Figure 5.2, Reaction Time) as per no effect of group on reaction time [F(1,52) = 2.56, $p = .115$, $\eta_p^2 = .047$, $BF_{\text{incl}} = .369$] nor any interactions with other factors (Appendix Tables 5.3-5.4). However, results show that higher memory load [F(1,52) = 203.638, $p < .001$, $\eta_p^2 = .797$, $BF_{10} = 5.739^{-4}$] and longer delays [F(1,52) = 32.45, $p < .001$, $\eta_p^2 = .384$, $BF_{10} = 2.766e^{-45}$] slowed participants down. Both main effects did not interact in the reaction time [F(1,52) = 1.599, $p = .212$, $\eta_p^2 = .030$, $BF_{\text{incl}} = .605$].

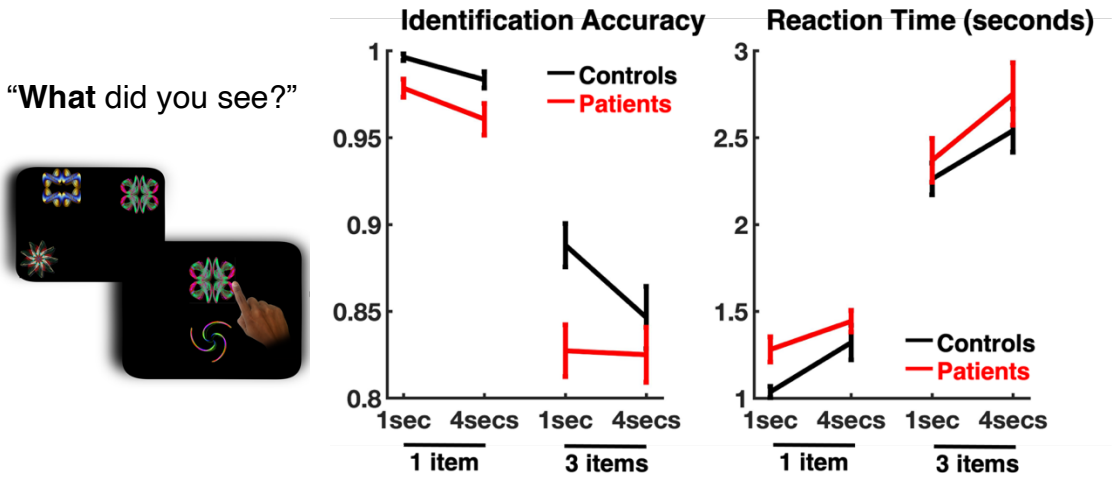


Figure 5.2| Results of "What was where?" task on the ability to correctly identify memoranda.

Patients with LE select the correct memoranda less often than elderly healthy controls (graph on Identification Accuracy). Regardless of disease occurrence, presenting three items as opposed to one slowed participants down when correctly identifying the target (graph on reaction Time).

Localisation error, i.e., the distance between the reported location and the original location of the memoranda was subsequently investigated, and trials with incorrect identifications excluded from further analysis (Figure 5.3, Localisation error). The main effect of number of items was significant [F(1,52) = 434.897, p < .001, η_p^2 = .893, BF_{10} = 4.387e^{-6}] reflecting larger errors when three items were presented. Additionally, a main effect of delay also increased participants' localization error [F(1,52) = 25.911, p < .001, η_p^2 = .333, BF_{10} = 1.662e^{-67}]. Patients showed significantly larger localization errors relative to controls, as reflected in the main effect of group [F(1,52) = 14.771, p < .001, η_p^2 = .221, BF_{incl} = 408.003] due to impaired localization performance for longer delays as suggested by the interaction effect between set size and delay with localisation error [F(1,52) = 8.243, p = .006, η_p^2 = .137, BF_{incl} = 4.336]. Post-hoc t-tests show that participants performed significantly worse with a four-second delay rather than with a

one-second delay for set size three [F(1,52) = 5.659, p_{holm} < .001], but this was no longer the case for set size one [F(1,52) = 1.679, p_{holm} = .096] (Appendix Tables 5.5-5.6).

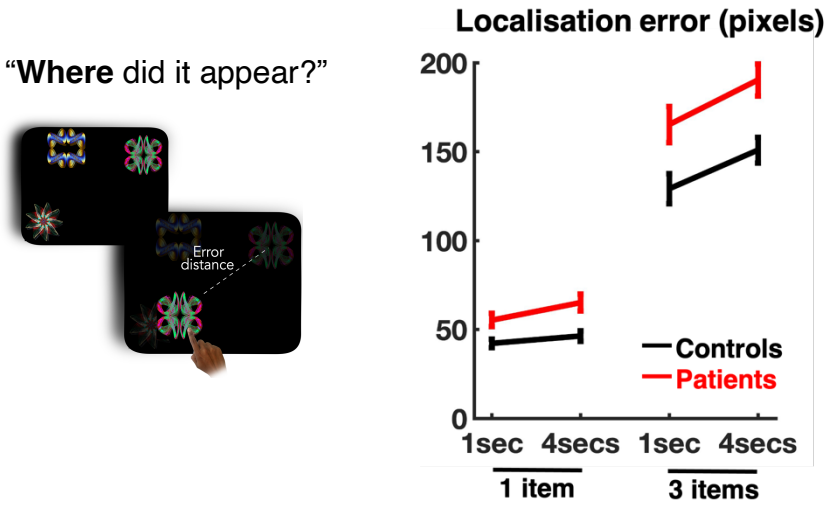


Figure 5.3 | Results of "What was where?" task on the ability to localise correctly identified memoranda.

Patients placed the shape further away from the original target's location compared to controls.

5.3.2. Mixture Model Results

To further explore the nature of poorer localization performance in patients compared to controls, a 2 x 2 ANOVA of factors delay and group was conducted for misbinding, guessing and imprecision, with years of full time education and fluency as covariates. Results (Figure 5.4) support lesion-related deficit in binding the identity of memoranda with its location [F(1,52) = 6.528, p = .014, η_p^2 = .112, BF_{incl} = 3.639] (Figure 5.4, Proportion of Misbinding). However, localization errors due to guessing were similar for both groups [F(1,52) = 2.637, p = .110, η_p^2 = .048, BF_{incl} = .527] (Figure 5.4, Proportion of Guessing), as well as imprecision (F(1,52) = 2.405, p = .127, η_p^2 = .044, BF_{incl} = .660] (Figure 5.4, Imprecision).

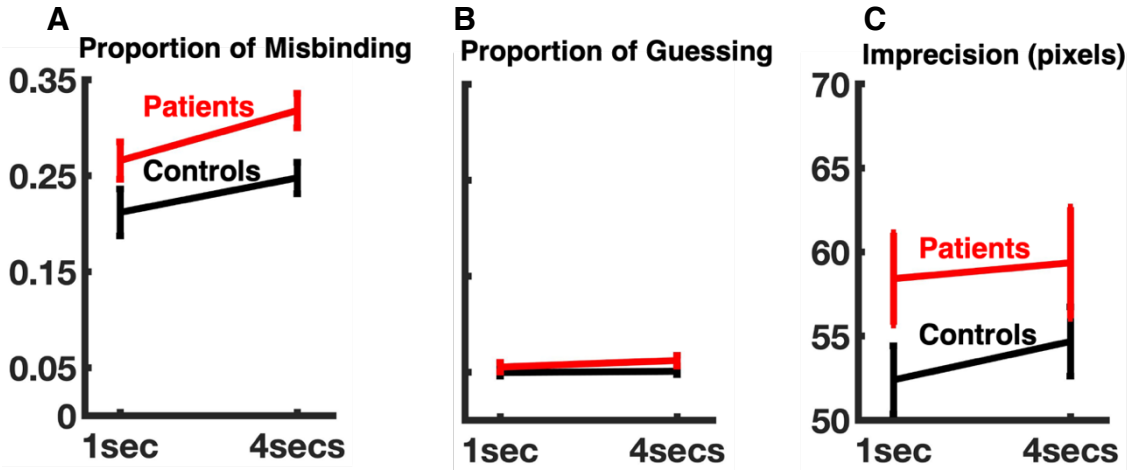


Figure 5.4 | Results on the types of memory errors on the "What was where?" task.

Patients' poorer localization performance can be explained by an increase in the number of errors due to misbinding (A) — the inability to bind together location and identify of memoranda — but not by guessing (B). Moreover, patients located the correct item more imprecisely than controls (C).

Additionally, results show that longer delays increased misbinding [F(1,52) = 10.591, p = .002, η_p^2 = .169, BF_{10} = .222], but neither guessing [F(1,52) = 1.125, p = .294, η_p^2 = .021, BF_{10} = .319], nor imprecision did [F(1,52) = 1.364, p = .248, η_p^2 = .026, BF_{10} = .440]. Moreover, group and delay did not interact with misbinding [F(1,52) = .364, p = .549, η_p^2 = .007, BF_{incl} = .928], guessing [F(1,52) = .475, p = .494, η_p^2 = .009, BF_{incl} = .137] or imprecision [F(1,52) = .233, p = .631, η_p^2 = .004, BF_{incl} = .166]. Details in Appendix Table 5.7-5.12.

5.3.3. Neuroimaging Results

All participants with a structural MRI were included in this correlation analysis, a total of forty-one data sets (28 controls and 13 patients). Differences in structural volumes of the hippocampus subfields between the two groups are displayed in

Table 5.2. Patients showed a significant reduction in bilateral hippocampal volume compared to controls [p = .006, BF_{10} = 7.422]. They exhibited reduced volume of both right and left hippocampus [p = .007, BF_{10} = 6.835; p = .029, BF_{10} = 2.250, respectively], left and right hippocampal tail [p < .001, BF_{10} = 176.849; p = .016 , BF_{10} = 3.546, respectively], right and left presubiculum [p = .007, BF_{10} = 6.463; p = .011, BF_{10} = 4.683, respectively], right subiculum [p = .012, BF_{10} = 4.234], right CA1 [p = .049, BF_{10} = 1.551], right CA4 [p = .025, BF_{10} =2.506], right granule cell layer of dentate gyrus (GC-DG) [p = .010, BF_{10} = 4.954], right molecular layer [p = .013, BF_{10} = 4.120] and both right and left fimbria [p = .019, BF_{10} = 3.075; p = .014, BF_{10} = 3.909, respectively]. Details in Appendix Table 5.13-5.15.

Table 5.2 | Structural differences between 28 healthy elderly controls and 13 LE patients as defined by independent samples t-test. The amount of variation of the data set is quantified by ± SD.

Volume (mm^3)	Healthy elderly controls	Limbic Encephalitis patients	Controls versus Patients p-value
Bilateral Hippocampus	7594.614(±621.933)	6687.217(±1390.022)	0.006
Right Hippocampus	514.496(±42.051)	445.594(±59.540)	0.007
Left Hippocampus	509.280(±50.045)	464.155(±59.656)	0.029

Right Hippocampal tail	514.496 (±)42.051	445.594 (±)59.540	< .001
Left Hippocampal tail	509.280 (±)50.045	464.155 (±)59.656	0.016
Right parasubiculum	58.546 (±9.650)	61.349 (±9.819)	0.395
Left parasubiculum	64.472 (±12.935)	60.650 (±14.447)	0.401
Right presubiculum	263.083 (±34.148)	229.165 (±38.518)	0.007
Left presubiculum	288.348 (±34.709)	253.953 (±45.285)	0.011
Right subiculum	434.670(±40.805)	390.694(±66.006)	0.012
Left subiculum	439.550(±44.419)	409.589(±64.160)	0.090
Right CA1	695.963 (±59.879)	637.387 (±126.286)	0.049
Left CA1	658.522 (±59.736)	621.245 (±133.037)	0.219
Right CA3	228.364 (±29.353)	211.221 (±42.466)	0.140
Left CA3	202.034 (±27.699)	203.013 (±40.087)	0.928
Right CA4	264.205 (±18.586)	241.380 (±44.624)	0.025
Left CA4	252.622 (±17.118)	240.852 (±36.476)	0.164
Right Hippocampus-amygdala transition area (HATA)	63.385 (±9.396)	63.723 (±12.084)	0.923
Left Hippocampus-amygdala transition area (HATA)	61.548 (±8.493)	60.389 (±10.641)	0.710
Right granule cell layer of dentate gyrus (GC-DG)	305.305 (±22.641)	274.162 (±51.633)	0.010
Left granule cell layer of dentate gyrus (GC-DG)	293.310 (±21.370)	278.704 (±45.481)	0.166
Right molecular layer	475.110 (±34.898)	433.869 (±66.835)	0.013
Left molecular layer	469.789 (±47.261)	443.789 (±53.38)	0.124
Right fimbria	64.163 (±20.240)	48.007 (±18.293)	0.019
Left fimbria	80.937 (±22.182)	61.141 (±24.291)	0.014

Right hippocampal fissure	148.897 (±20.508)	162.828 (±34.215)	0.112
Left hippocampal fissure	143.788 (±21.218)	160.188 (±31.439)	0.056

A correlation analysis (Figure 5.5) showed bilateral hippocampal volume to be associated with misbinding [r(39)= -.406, *p* = .008, BF_{10} = 5.514] but not guessing [r(39) = -.284, *p* = .072, BF_{10} = .925]. Furthermore, the volume of the left [r(39)= -.457, *p* = .003, BF_{10} = 15.3] but not the right hippocampus [r(39) = -.262, *p* = .098, BF_{10} = .731] was found to be related to misbinding. This was no longer true in relation to guessing for both the left [r(39) = - .290, *p* = .066, BF_{10} = .992] and right [r(39) = -.215, *p* = .176, BF_{10} = .471] hippocampus (Appendix Table 5.15 – 5.16).

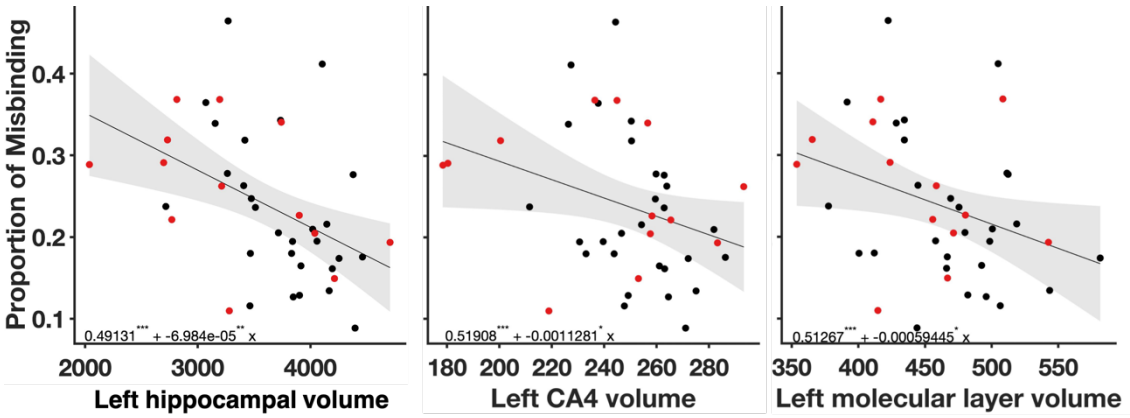


Figure 5.5 | Misbinding is negatively correlated with the left hippocampus and in particular the left CA4 and left molecular layer.

Pearson correlation analysis between the proportion of errors due to misbinding and left hippocampal volume, left CA4and left molecular layer**.**

Following up on the significant correlation with the left hippocampus, hippocampal subfield analysis revealed (Figure 5.5) that misbinding was specifically correlated to

regions left molecular layer [r(39) = -.334, *p* =.0333 , BF_{10} = 1.756] and left CA4 [r(39) = -.322, *p* = .040, BF_{10} = 1.484] but not to left CA3 [r(39) = -.290, *p* = .066, BF_{10} = .997] nor left CA1 [r(39) = -.291, *p* = .065 , BF_{10} = 1.007]. Details in **Appendix Table 5.16 – 5.17**.

Thus, the most important findings revealed by investigating the relationship between memory resolution and the hippocampi volumes and its subfields related to misbinding being a direct function of left hippocampal atrophy, specifically the left molecular layer and left CA4.

5.4. *Discussion*

The present study aimed to replicate Pertzov et al.'s (2013) findings which were based on only seven patients with LE. In order to determine if the authors research claims could be extended to a bigger sample size of twenty-eight LE patients, we used the same sensitive delayed reproduction task, the "What was where?" paradigm, but now using more modern analysis techniques (mixture modelling). Results were consistent with the original study. First, behavioural results reflect a disease-related impairment in correctly identifying memoranda with evident memory impairment when participants were asked to remember three items as opposed to one, particularly after longer retention intervals. Additionally, LE patients exhibited larger localisation errors compared to controls, especially after lengthier maintenance delays when given three items to remember as opposed to one. Secondly, the mixture model results suggest that this localisation impairment emerged from a lesion-related deficit in binding together different features belonging to the same memoranda, i.e., patients misreported the location of the

correctly identified item by dragging it to the location of another item presented in the memory array more often than controls. Importantly, their impairment was confined to these binding errors, as they did not display increased guessing.

An important aspect of this study was to enhance the generalizability of the original investigation by having participants performing the behavioural task on a portable tablet device (iPad version 9.3.5 (13G36) at a viewing distance of 30 cm and subtended an angle of 2.3°, model MGTX2B/A) with 1536x2048 pixel matrix, as opposed to the non-portable interactive touch-sensitive screen (Inspiron all-in-one 2320, Dell) with a 1920x1080 pixel matrix corresponding to approximately 62x35° of the visual angle employed by the authors. Furthermore, although measurements employed here were comparable to the original research to avoid misinterpretations caused by measurement errors, here a new toolbox was applied to distinguish the sources of memory error (Grogan et al., 2019) by means of implementing the technique of mixture modelling, commonly used in one dimensional report domains such as a colour wheel or rotation of a dial (Paul M. Bays et al., 2009; W Zhang & Luck, 2008), and adapting it to two dimensional data, i.e., spatial data.

Even though the binding problem has conventionally been examined in visual perception studies (Wheeler & Treisman, 2002), it has been shown to be very much relevant to memory as well. Indeed, analogously to Pertzov et al. (2013), our results show that patients with hippocampal lesions are capable of performing as well as controls when asked to hold single items in memory. But, when given three items to recall, they are significantly hindered at associating the correct identity of an object with its location. This is in rapport with Axmacher et al. (2007) findings on WM load engendering differing

sustained neural activity during maintenance in the MTL. In their EEG study, patients with MTL pathology and healthy controls were tested on a serial Sternberg paradigm to investigate WM-specific sustained activity patterns. They found an incrementally negative shift of the direct-current (DC) potential and an intensification in MTL gamma-band activity for the maintenance of multiple items, likely to be attributable to a sustained increase in neural activity. As well as a positive shift of the DC potential deactivation and decrease in MTL gamma-band activity during maintenance of a single item, probably resulting from a sustained decrease in neural activity (Axmacher et al., 2007).

The nature of the memory deficit in patients with LE presented here is in agreement with several other investigations on MTL dysfunction where these patients are hindered in their ability to associate different features belonging to the same item (Banta Lavenex et al., 2014; Finke et al., 2007, 2013; Hannula et al., 2006; Olson, Page, et al., 2006; Zokaei & Husain, 2019a; Zokaei et al., 2020). Importantly, and conforming with results suggesting that MTL is one of the earliest neural structures to be affected (Fox, 1996), a study from Parra and colleagues (2015) showed that asymptomatic individuals at risk of developing familial AD — carriers of the mutation E280A of the *PSEN1* gene — are also impaired in their ability to remember several features belonging to the same object over short delays, and that this is associated with reduced white matter integrity in frontal lobes, medial temporal lobes and corpus callosum (Parra et al., 2015).

A subsequent aim of this study was to investigate whether misbinding — the memory error found to be associated with MTL lesion — correlated with hippocampal volume and how selective was this association to specific subfields of the hippocampus.

The hippocampus is a complex structure that consists of different subfields —the cornu ammonis subfields (CA1, CA2, CA3, CA4), the molecular layer (ML), the granule cell layer of the dentate gyrus (GC-DG), subiculum, presubiculum, parasubiculum, fimbria, the hippocampal-amygdaloid transition area (HATA), hippocampal tail and hippocampal fissure — with diverse, specialized memory functions (Hunsaker, Lee, & Kesner, 2008; Reagh, Watabe, Ly, Murray, & Yassa, 2014; Yassa & Stark, 2011). For example, the CA2/CA3 and CA4/DG have been suggested to be further implicated in encoding, learning and recall over short delays; whilst CA1 and subiculum have been linked to delayed episodic memory and are regarded as output structures associated with retrieval roles (Eldridge et al., 2005; S. G. Mueller, Chao, Berman, & Weiner, 2011; Zammit et al., 2017).

Comprehensive volumetric studies of the hippocampal subfields reported in different disorders have started to reveal which regions are implicated in specific memory functions (Andersen, Morris, Amaral, Bliss, & Keefe, 2007; Geuze, Vermetten, & Bremner, 2005). For instance, reduced CA1, CA3 and DG volumes have been described in major depressive disorder (Huang et al., 2013; Travis et al., 2015), smaller CA4 and DG have been reported in post-traumatic stress disorder (Hayes et al., 2017) whereas diminished CA4/DG and CA1 have been related to schizophrenia (Nakahara, Matsumoto, & van Erp, 2018).

Studies in AD have shown that AD variability is also associated with distinct hippocampal subfields changes. Some researchers found that CA1 and subiculum are particularly affected in the early stages of AD through prominent tangle accumulation and neuron loss (Blanken et al., 2017; Rössler, Zarski, Bohl, & Ohm, 2002) while others have shown more extensive atrophy in DG and CA3 (see de Flores, La Joie, & Chételat,

2015 for a review). Carlesimo et. al (2015) have found that, unlike healthy individuals who don't show any correlation between hippocampal subfield volumes and memory performance, these patients' CA2-CA3, CA4-DG and subiculum subfield volumes are associated with immediate and delayed recall, whereas the presubiculum is only correlated with delayed recall. Moreover, findings on prodromal AD have suggested that atrophy of CA1 and CA2 is greater in patients with mild cognitive impairment (MCI) compared to normal aging (Susanne G. Mueller et al., 2010). This arises initially in the presubiculum and subiculum which have been reported to correlate with immediate recall (Carlesimo et al., 2015).

Connectivity to the hippocampus and prefrontal cortex (PFC) regions have been shown to correlate with binding in both episodic (Addis & McAndrews, 2006; McCormick, Moscovitch, Protzner, Huber, & McAndrews, 2010) and WM (Mitchell, Johnson, Raye, & Esposito, 2000; Prabhakaran, Narayanan, Zhao, & Gabrieli, 2000). Furthermore, increasing evidence from functional neuroimaging indicates that hippocampal subfield CA3 is not only engaged in the retrieval of both recent and remote vivid episodic memories (Bonnici, Chadwick, & Maguire, 2013; Chadwick, Bonnici, & Maguire, 2014; T. D. Miller et al., 2020) but is also responsible for helping signals resulting from different brain regions to be associated together (Kesner and Rolls, 2015). However, the nature and organization of the roles of individual structures within the MTL are yet unclear and little is known on the involvement of differing anatomical connectivity, and functional contribution of hippocampal subfields to relational processing of information in STM.

Here, analysis on the relationship between hippocampal subfield volumetry from structural imaging (13 patients and 28 controls) and memory resolution results (metrics

on memory imprecision, misbinding and guessing) revealed that LE patients have smaller bilateral hippocampus compared to controls, with a volumetric decrease in hippocampal tail, presubiculum, right subiculum, right CA1, right CA4, right CG-DG, right ML and fimbria. This is not in total accordance with other studies on hippocampal subfield changes in MTL patients, which have also failed to report consistent results (Carlesimo et al., 2015; Finke et al., 2017; Mak et al., 2017; Susanne G. Mueller et al., 2010; Wisse et al., 2015). For example, Finke and colleagues' study (2017) on 30 anti-LGI1 encephalitis patients reported significant bilateral hippocampal atrophy, comprising subfields CA2/ CA3, CA4/DG . While Wisse et. al observed a decrease in volume in the subiculum, CA1, CA4 and DG in patients with AD (Wisse et al., 2015), others have shown widespread subfield losses in both MCI and AD with the exception of CA2 and CA3 in the MCI cohort (Mak et al., 2017). Moreover, even though parasubiculum volume changes have been speculated to be considered to be a biomarker for dementia disorders (Zheng et al., 2018), no significant structural changes were found between controls and hippocampal atrophy patients, suggesting that a distinction between normal and pathological aging may not be as straightforward as envisaged.

Errors due misbinding — but not guessing — were found to be a direct function of left hippocampal atrophy; specifically, the left ML and left CA4 (**Figure 5.5**). The ML is located above the subiculum and underneath the hippocampal fissure, its volume — together with CA2/CA3 and CA4 — has been found to impact memory function and has been suggested to be positively associated with general cognitive ability across the lifespan (Tamnes, Bos, van de Kamp, Peters, & Crone, 2018). Zheng and colleagues (2018) investigated the association between hippocampal subfields and age-related memory

decline and found that immediate and delayed recall scores were strongly associated with volume of the bilateral ML as well as other subfields. Scheff et. al (2006), however, found that the number of synapses in ML was associated with both delayed recognition abilities and delayed recall but not with immediate recall aptitude. Other researchers have found a relationship between a decrease in both the DG and ML volumes to be associated with decreased delayed memory (Cantero, Iglesias, Van Leemput, & Atienza, 2016).

The CA4, which underlies the dentate gyrus, is in fact the "deep, polymorphic layer of the dentate gyrus" (Andersen et al., 2007). Together with DG, CA2 and CA3, CA4 volume has been found to be decreased in encephalitis patients with LE and associated with decreased recall of memory after interference and during recognition. Notably, Finke et. al (2017) found that patients with decreased CA4/DG volume tended to commit more intrusion errors — which misbiding errors are the common reminiscent of and that arise from misreporting an items' property during serial recall tasks (Burgess & Hitch, 1992; Henson, Norris, Page, & Baddeley, 1996)— during the recognition part of the auditory verbal learning test (AVLT).

But no relationship was found between misbinding and CA3, even though it has been observed that there is a predominant expression of LGI1 in the CA3 (Herranz-Pérez et al., 2010) a subfield that has been implicated to play a role in both pattern completion — assembling a total representation based on partial information — and pattern separation — adequately separating sensory inputs with large similarities to prevent memory interference and maintaining unique orthogonal representations (Yassa & Stark, 2011). For example, in their study, Mueller and colleagues (2011) tested participants'

immediate recall after reading them a list of 16 items over 5 learning trials (the AVLT test). Even though they used a binary assessment of recall (the item is either remembered or forgotten), they found a significant relationship between learning or encoding ability (performance in trials 2-5) and early retrieval of information (by introducing a second list of items to be recalled, 5 min after trial 5, and asking participants to recall the first list without allowing them to hear it again) with CA3 and DG volume. They also found a positive relationship between consolidation (by asking participants to recall the first item list freely or with the help of a cue, after a 20 min delay during which non-verbal tasks are performed) and delayed retrieval of verbal memoranda and CA1 subfield volumetry.

Unlike the entirety of the neuroimaging studies discussed here, which have looked at the relationship between hippocampal subfield volumetry and WM measures using binary neuropsychological tests that index span, this study uses a continuous measure of report in order to isolate and quantify the precise nature of WM deficit related to hippocampal dysfunction. This is a pertinent improvement on the current literature because span measures of WM are not always reliably replicable, are not sensitive to detect subtle variations in performance and do not provide any further details on the exact nature of the memory deficit.

This study has, however, some limitations. (1) The sample size is small and quite a few patients were not eligible to undergo MRI testing which further decreased statistical power. Nonetheless, it is important to consider that the patients included in this study are very unique and incredibly difficult to recruit. (2) The hippocampal subfield segmentation package in *FreeSurfer* used to pre-process the structural volumetric raises

some concerns as discussed by Wisse and colleagues (Wisse et al., 2015). Data requires low resolution T1 images, which do not contain the same amount of detail of high resolution T2 images other automated segmentation methods are developed for. Besides, the parcellation scheme used for the *FreeSurfer* segmentation results in large parts of the subfields being assigned to neighbourhood subfields leading to reporting results that differ from anatomical studies. Also, another shortcoming is that this protocol, which was applied to low resolution images, was initially developed on high resolution 3T images and has not been validated against manual segmentation on the 1.5-3T MR images. (3) The data was analysed using both a classical approach and Bayesian statistics. Even though Bayesian analysis, unlike the classical approach, allows to assess the relative evidence favouring the null hypothesis versus a specified alternative hypothesis (Jeffreys, 1935), permitting a measure of the strength of the evidence favouring the null, the significant statistical relationships found between structure and function do not necessarily mean that there is a causal link between the two. Finally, Fell and Axmacher (2011) have discussed that phase synchronization among distinct brain regions might be involved in the binding process impairment. Therefore, an important addition to the current study would be to ascertain whether hippocampal damage is accompanied by remote effects on the function of the extended hippocampal system as proposed by Aggleton and colleagues (2010).

Taken together, the results presented in this study suggest that LE patients display increased vulnerability in binding together the identity of an item with its location and that the binding process is hindered as a direct function of left hippocampal atrophy, particularly the left molecular layer and left CA4.This further supports the MTL role in

associative mechanisms responsible for retrieving different properties belonging to the same item, regardless of memory delay.

Chapter 6. The role of serotonin in cognitive and emotion processing in Parkinson's disease

Abstract

The mechanisms underlying memory deficits in PD are still unclear. Sensitive delayed reproduction tasks have been used to decompose the sources of errors arising from WM impairment and have shown that PD patients' deficit stems from increased guessing (random responses), as opposed to MTL patients who have been shown to have greater misbinding (inability to associate different features together). Conflicting results on the contributions of dopamine to WM impairment in PD have lead to the hypothesis that the increase in guess responses could potentially reflect involvement of non-dopaminergic neurotransmitters such as disruption in serotonin. Additionally, recent meta-analysis of emotion recognition studies in PD have shown an association between cognitive impairment and the variability in emotional valence processing in these patients.

Even though serotonergic dysfunction occurs early in the course of PD, there is still little understanding on how it affects WM and emotion processing. Therefore, this study aimed at investigating how serotonergic modulation through the administration of citalopram (20 mg) for seven days affects cognitive and emotional memory processing in patients with idiopathic PD. A double-blind, placebo-controlled within-groups cross-over design was implemented, where each patient received seven days of citalopram and a matched placebo in a counterbalanced, randomized order.

Results show that PD patients' WM identification accuracy and localisation performance was not impaired compared to controls. Administration of citalopram did not modulate their overall WM performance nor their memory resolution. Emotional

valence was found to affect visual spatial WM in PD patients as opposed to controls – patients found it harder to identify neutral and angry emotions compared to happy. The impact of emotional valence in PD seems to be moderated by the effects of citalopram in mood. Higher sensitivity to the antidepressant — reflected by greater decrease in depression scores — was allied to better identification accuracy for neutral faces, decreased localisation error for happy faces, decreased imprecision for both angry and happy faces and decreased guessing for angry faces.

Altogether, these findings highlight a possible role of the serotonergic degeneration in processing negative emotional information and show that WM is unaffected.

6.1. *Introduction*

WM can be disrupted in many different brain disorders. Delayed reproductions tasks have been used to examine the sources of errors contributing to impaired memory performance in order to understand the mechanisms underlying these deficits in diseases associated with MTL pathology and in PD (Pertzov, Miller, et al., 2013; Zokaei & Husain, 2019b; Zokaei et al., 2020). In patients with MTL lesions, WM deficits have been shown to specifically arise from misbinding errors — the inability to correctly associate different features belonging to the same object — (Banta Lavenex et al., 2014; Finke et al., 2017; Koen et al., 2017; Olson, Page, et al., 2006; Pertzov, Miller, et al., 2013; van Geldorp, Bouman, Hendriks, & Kessels, 2014; Zokaei & Husain, 2019b; Zokaei, Mcneill, et al., 2014; Zokaei et al., 2018, 2020) whereas in patients with PD, impairments in WM

have been associated with an increase in errors due to guessing (Zokaei, Mcneill, et al., 2014; Zokaei et al., 2020).

PD is characterised by progressive nigrostriatal dopaminergic cell loss (Heiko Braak et al., 2004; Vingerhoets et al., 1994) and several lines of research have suggested that the increase in errors due to guessing observed in this cohort could be due to a decrease in signal-to-noise ratio arising from deficits in their dopaminergic function (Sawaguchi & Goldman-Rakic, 1991; Winterer & Weinberger, 2004). However, others have advocated that memory deficits are not necessarily modulated by dopaminergic medication (Roshan Cools, 2006; Gratwicke et al., 2015; Kehagia et al., 2010) and may reflect involvement of non-dopaminergic neurotransmitters, such as disruption in noradrenaline (Zarow et al., 2003), acetylcholine (Dubois et al., 1983), GABA (Emir, Tuite, & Öz, 2012) or serotonin (Kish et al., 2008; Meyer et al., 2001; Marios Politis et al., 2010).

Fallon and colleagues (2017) have attempted to address the conflicting results on the contributions of dopamine to WM impairment by testing PD patients ON and OFF their dopaminergic medication on the separate consequences of maintaining and manipulating (ignoring and updating) information in WM. They found that dopamine improved performance on conditions where irrelevant information had to be jettisoned (ignored or updated), but had no effect on their ability to maintain information over variable delays (Sean James Fallon et al., 2017). These results suggest that, on the one hand, in line with a frontal dysexecutive effect of the dopamine neurotransmitter, these patients are impaired in gating of information into relevant versus irrelevant items in memory (R. G. Brown & Marsden, 1991; Dalrymple-Alford et al., 1994; Gruszka et al., 2016). On the other hand, in line with a posterior cortical syndrome that is not affected

by dopamine and is responsible for successful retention of information (Gratwicke et al., 2015; Kehagia et al., 2010), a null effect of dopaminergic medication on WM retention in the absence of competing information, together with a disease related impairment in simple retention of information, could potentially be associated with non-dopaminergic mechanisms.

Serotonin is involved in the regulation of mood, affection, aggression, cognition and other autonomic functions and most serotonergic input to the forebrain is provided by dorsal raphe nucleus (DRN) neurons (Jacobs & Azmitia, 1992). In PD, loss of caudal brainstem serotonergic neurons actually precedes degeneration of dopaminergic midbrain neurons (Heiko Braak et al., 2004), such that their depletion may be implicated in the development of significant non-motor and cognitive disturbances. For example, Maillet and colleagues have provided evidence that serotonergic degeneration is related to the severity of apathy and depression in PD (Maillet et al., 2016). In line with this, a number of researchers have linked serotonergic function to neuropsychiatric symptoms, reporting lower levels of serotonin metabolites or 5-HT1a receptor availability in patients with PD that were also depressed compared to those who were not (Aarsland, Påhlhagen, Ballard, Ehrt, & Svenningsson, 2012; Marios Politis & Niccolini, 2014; Tan, Hartung, Sharp, & Temel, 2011). Depressed PD patients have also been described as having increased limbic SERT binding, suggesting that loss of serotonergic neurons in the raphe nuclei and limbic structures, together with increased clearance of serotonin through SERT, may lead to these patients' depressive symptoms (Boileau et al., 2008; Pagano, Niccolini, Fusar-Poli, & Politis, 2017; M Politis et al., 2010).

Findings indicating reversal of brain functional abnormalities by antidepressant treatment are well documented in the literature and support the hypothesis that higher levels of serotonin may improve mood through its effects on emotion processing (Davidson, Ph, Irwin, Anderle, & Kalin, 2003; Fu, Walsh, & Drevets, 2003; Mayberg et al., 2013; Sheline et al., 2001). For instance, a study by Fu et al. (2004) shows evidence for serotonergic modulation of selective bias for negative information in patients with depression: after administrating fluoxetine hydrochloride — an SSRI — for eight weeks, they found that, when presenting these patients with a sad facial expression, they exhibited reduced activation in the amygdala, ventral striatum and frontoparietal cortex. Harmer and colleagues have also shown that impaired emotional processing is an important link between the 5-HT system, regulation of mood and the experience of depression, by reporting that citalopram and reboxetine enhanced positive biases in emotional perception and memory in PD patients (Harmer et al., 2009, 2004).

Intriguingly, however, many studies have reported impaired emotion recognition in PD patients without depression, particularly for negative emotions (Argaud, Vérin, Sauleau, & Grandjean, 2018; Gray & Tickle-Degnen, 2010; Péron, Dondaine, Le Jeune, Grandjean, & Vérin, 2012). Yet, reports on processing of emotional information in PD have presented contradictory findings, some failing to establish any such impairment (Adolphs, Schul, & Tranel, 1998; Dewick, Hanley, Davies, Playfer, & Turnbull, 1991; Madeley, Ellis, & Mindham, 1995; Pell & Leonard, 2005), others describing decreased facial expression recognition in PD compared to controls (Clark, Neargarder, & Cronin-Golomb, 2008; Dujardin et al., 2004; Kan, Kawamura, Hasegawa, Mochizuki, & Nakamura, 2002; Yip, Lee, Ho, Tsang, & Li, 2003). Importantly, this deficit appears to correlate with

various factors such as disease severity, dopamine replacement therapy, visual dysfunction and cognitive impairment (Argaud et al., 2018).

Memory and learning have also been reported to decrease with reduced serotonergic neurotransmission, with long term memory shown to be particularly affected as opposed to STM (Park et al., 1994; Riedel et al., 1999; Rubinsztein et al., 2001; Schmitt et al., 2000). Following recovery from depression, memory deficits have been shown to improve with antidepressants (Bondareff et al., 2000; Keegan et al., 1991; Peselow, Corwin, Fieve, Rotrosen, & Cooper, 1991), but it is unclear whether this is related to a non-specific effect of symptom improvement as opposed to a direct action of the antidepressant. The effects of increased serotonin levels on verbal short and long-term memory have recently been explored by administrating 10mg intravenous citalopram to healthy young females. Results showed that an increase of serotonergic transmission is accompanied by improved memory consolidation, but that immediate recall on the verbal memory test was unchanged (Harmer et al., 2002).

Even though there is a progressive, non-linear loss of serotonergic neurons in PD, there is still little understanding on how serotonergic modulation in PD affects WM and emotion processing measures. The aim of this study was therefore to investigate how a seven-day administration of 20mg of citalopram affects emotional memory processing in patients with idiopathic PD through a double-blind, placebo-controlled within-groups cross over design. Citalopram is an antidepressant with high affinity for the serotonin (5-hydroxytryptamine; 5-HT) transporter (5-HTT) (Owens, Morgan, Plott, & Nemeroff, 1997; Tatsumi, Groshan, Blakely, & Richelson, 1997). Thus, by binding to the 5-HTT, this SSRI blocks reuptake of serotonin in presynaptic cells, thereby increasing the level of

serotonin within the synaptic cleft. Importantly, commonly used SSRIs have been shown to have a short-term effect of acute serotonergic modulation in the brain without changes in levels of mood or anxiety (Harmer et al., 2009, 2004; Ye et al., 2014).

Because the aim of the current study was to investigate the serotonergic mechanisms of WM and emotion processing in PD, not the effectiveness of citalopram against depressive symptoms, seven days of citalopram were considered to be appropriate. To assess WM and emotion processing, patients and age-matched controls were examined on a set of questionnaires that index global cognition, and neuropsychiatric symptoms such as depression and apathy, as well as behavioural tasks that assess various aspects of WM and emotion processing: identification accuracy, reaction time, localisation error. Importantly, the delayed-reproduction paradigms used in this study allowed to assess the resolution of memory representation by modelling the sources of error contributing to the pattern of recall in imprecision, misbinding or guessing (Grogan et al., 2019; Ji Ma et al., 2014). We hypothesised that if PD patients, while on their regular dopamine medication, presented different patterns of WM resolution and emotion processing compared to controls, performance on serotonin might become similar to the healthy level. To account for individual differences in serotonergic responses, since several lines of evidence have suggested that apathy in people with and without PD is aggravated by SSRIs (Barnhart, Makela, & Latocha, 2004; Muhammed & Husain, 2016; Price, Cole, & Goodwin, 2009; Wongpakaran, van Reekum, Wongpakaran, & Clarke, 2007), baseline apathy motivation levels were also considered in the analysis.

6.2. *Methods*

6.2.1. Outline of the study Design

This experimental medicine study aimed at investigating how serotonergic modulation through administration of citalopram (20 mg) for seven days affects cognitive and emotion processing in patients with idiopathic PD. A double-blind, placebo-controlled within-groups cross-over design was implemented, where each patient received seven days of citalopram and a matched placebo in a counterbalanced, randomized order. This was then followed by at least a two-week wash-out period in-between the sessions. Behavioural tasks and questionnaires were completed after a week of taking the allocated compound. All capsules had the same appearance, so that participants would not be able to tell which compound they were receiving (the placebo looked like the citalopram capsule but was just a sugar pill that had no active substance in it). The order in which they took the drug or placebo capsules was assigned by chance. Information about the allocation was stored securely during the study and unblinding was only possible at the end of the study, or if it was deemed necessary for medical reasons. Note that to guarantee there was a gender balance between the two drug administration orders, the drug allocation was 'pseudo-random' as opposed to truly random since the sample was stratified by gender at a 1:1 ratio, and the randomisation scheme was made within each stratum.

The study involved five visits in total for each patient (Figure 6.1): a screening visit, two first dose visits (one for each allocated compound, either citalopram or placebo),

two research visits (one after a week of each allocated compound). Controls were tested once without medication.

The screening visit (**Figure 6.1**) was held at the John Radcliffe Hospital and lasted up to 3 hours. During this time patients underwent a cognitive assessment, questionnaires (taken as baseline measures, see Demographics **Table 6.1**) and a routine medical examination (medical history, pulse and blood pressure check, height and weight measurement), to make sure they fulfilled our criteria to take part in the study and make sure they were safe to take the citalopram tablets. Following the screening visit, participants' GPs were informed of their participation in the study.

The first dose visit (**Figure 6.1**) was also held at the John Radcliffe Hospital and lasted up to 2 hours. Patients were given the first dose of their assigned compound and were monitored for 1 hour. After a brief examination by my colleague, Dr. Saleh, they were discharged and received the remaining assigned compound to take every morning with full instructions. Additionally, they were given a wallet-sized card with key details about the study and contact information available 24h in case patients would experience any side effects causing concern.

On the seventh day, they were invited to complete a research visit at the Department of Experimental Psychology at the Anna Watts Building lasting approximately 5 hours, where a number of computer-based tasks and a set of questionnaires were completed (**Figure 6.1**). The First Dose Visit, and the Research Visit were repeated after a minimum break of 14 days. Note that healthy controls were invited for a combined screening and research visit where no drug was administered. See below for a schematic outline of the study (**Figure 6.1**).

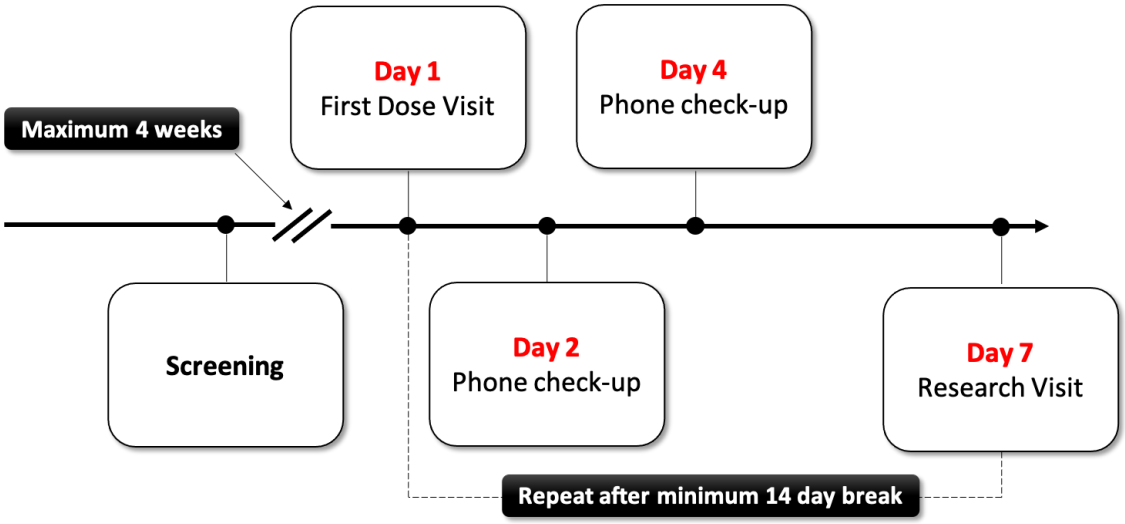


Figure 6.1 | Schematic outline of the study design.

The study consisted of five visits in total for each patient: a screening visit, within a 4-week period, followed by a first dose visit and a research visit on day seven of taking the allocated compound. After a wash-out period of a minimum of fourteen days, patients returned for a first dose visit to receive the second compound and respective research visit. Each patient received seven days of citalopram and a matched placebo in a counterbalanced, randomized order. Age and gender matched controls completed a combined screening and research visit.

6.2.2. Participants, Consent and Demographics

Twenty patients (11 females, mean age = 66.95 years, SD = 8.13) with a clinical diagnosis of idiopathic PD (Hoehn & Yahr stage = 1.95 ± .69) according to Parkinson's UK Biobank criteria (Hughes, Daniel, Kilford, & Lees, 1992) were recruited through an outpatient clinic to take part in this study. All patients included in this study had not participated in a study involving the use of medication within three months prior to the start of this research and had no history of bipolar disorder, schizophrenia or eating disorders, no diagnosis of familial or atypical PD, no history of stimulant abuse, alcohol dependence or alcohol abuse within one year. Additionally, they were not under treatment with MAO-B inhibitors (for example Rasagiline (*AZILECT*), Selegiline (*Eldepryl,*

Zelapar)) nor were they lactose intolerant. Another exclusion criterion was the use of antidepressant medication (for example, citalopram (*Cipramil*), dapoxetine (*Priligy*), escitalopram (*Cipralex*), fluoxetine (*Prozac/Oxactin*), fluvoxamine (*Faverin*), paroxetine (*Seroxat*), sertraline (*Lustral*), vortioxetine (*Viibryd*)).

Additionally, twenty age and gender matched controls were recruited from the local communities via public outreach events. All participants had normal or corrected-to-normal vision, were not colour blind, and gave written informed consent prior to participation. The study was reviewed and approved by the University of Oxford Central University Research Ethics Committee (Reference number: 18/SC/0524).

Demographics are displayed in **Table 6.1**. Levodopa equivalent daily dosage (LEDD) was calculated to summarise the total daily antiparkinsonian medication a patient was receiving (Tomlinson et al., 2010). Cognitive function screening was tested using ACE-III (Mathuranath et al., 2000), the MDS-Unified Parkinson's Disease Rating Scale (UPDRS) and the Hoehn and Yahr stage were used to gauge PD related disability and impairment (Goetz et al., 2008), the Anxiety motivation Index (AMI) was implemented to evaluate reduction in motivation (Ang, Lockwood, Apps, Muhammed, & Husain, 2017) and Beck's Depression Inventory (BDI) was used to measure characteristic symptoms of depression (A T Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Further details on these measures are reported below.

Baseline scores (**Table 6.1**) were collected at the screening visit for PD patients and at the research visit for healthy controls. At baseline, patients did not differ from controls in terms of age, gender ratio, or cognitive function, but they were, however, significantly more depressed and anxious (**Table 6.1**). Note that higher scores on the BDI and AMI are

typical of mild to moderate PD and do not in themselves establish a diagnosis of depression or apathy. Crucially, there were no significant differences in questionnaire scores between medication intake (Table 6.1, PD placebo vs PD citalopram).

Table 6.1 | Participants' demographics and differences between baseline measures for healthy controls and PD, as well as between medication intake (PD placebo vs PD citalopram).

Tests for differences between healthy controls and PD at baseline were done with unpaired t-tests, and test for differences between PD on placebo and PD on citalopram with paired t-tests. All values are mean (± SD) and p-values are uncorrected for multiple testing. Note that * significant $p < .05$ and ** significant $p < .01$.

Measure	Healthy controls	PD baseline	Controls vs PD baseline p-value	PD placebo	PD citalopram	Placebo vs citalopram p-value
N	20	20	n/a	20	20	n/a
Age (years old)	66.95(±8.13)	68.05(±8.40)	0.68	see baseline	see baseline	n/a
Gender (F/M)	11/09	10/10	0.75[a]	see baseline	see baseline	n/a
Disease duration (years)	n/a	5.59(±3.91)	n/a	see baseline	see baseline	n/a
LEDD[1]	n/a	299(±311)	n/a	see baseline	see baseline	n/a
ACE-III[2]	97.65(±2.50)	95.45(±4.42)	0.08	n/a	n/a	n/a
UPDRS[3]	n/a	29.95(±2.88)	n/a	29.95(±15.7)	33.16(±9.58)	.37
Hoehn & Yahr stage	n/a	1.95(±0.69)	n/a	see baseline	see baseline	n/a
BDI[4]	3.80(±4.44)	8.55(±4.22)	.001**	8.95(±6.00)	7.85(±4.63)	.15
AMI[5]	.98(±.42)	1.32(±.53)	.03*	1.37(±1.36)	1.36(±1.36)	.15
AMI behavioural	.89(±.56)	1.21(±.62)	.1	1.33(±1.63)	1.22 (±.64)	.20
AMI social	1.18(±.68)	1.61(±.76)	.07	1.53(±.79)	1.48(±.74)	.46
AMI emotional	.87(±.46)	1.15(±.50)	.08	1.24 (±.49)	1.19(±.51)	.45

[1]**LEDD** = Levodopa Equivalent Daily Dose; [2]**ACE-III** = Addenbrookes Cognitive Examination-III; [3]**UPDRS** = Unified Parkinson's Disease Rating Scale; [4]**BDI** = Beck Depression Inventory; [5]**AMI** = Anxiety motivation Index, n/a = not applicable. [a]Chi-squared test.

6.2.3. Procedure

Participants completed several questionnaires (ACE and UPDRS were completed by the researcher, whereas the BDI and AMI were self-reported) as well as two behavioural tasks (Experiment 1 and Experiment 2).

6.2.3.1. Questionnaires

Addenbrookes Cognitive Examination-III (ACE-III). The ACE is a neuropsychological test that measures cognitive status on a 24-item scale. It is divided in five domains: attention, memory, fluency, language and visuospatial up to a total sum of 100 (Mathuranath et al., 2000).

Unified Parkinson's Disease Rating Scale (UPDRS). The UPDRS is used to monitor PD-related disability and impairment on four components: Part I, mentation Behaviour and Mood; Part II, Activities of Daily Living; Part III, Motor, Part IV, Complications (Berven & Carl, 2003).

Anxiety Motivation Index (AMI). The AMI is a self-report anxiety motivation index on an 18-item scale that distinguishes apathy in terms of behavioural activation (tendency to self-initiate goal directed behaviour), emotional sensitivity (feelings or affective responses) and social motivation (level of engagement in social interactions). Every AMI sub score consists of 6 items, each scored from 0-4. AMI cut-off scores used were 1.91 for the AMI total, 2.34 for behavioural activation, 2.43 for social motivation, 1.68 emotional sensitivity (Ang et al., 2017).

Beck's Depression Inventory (BDI). The BDI measures the severity of depression on a 21-item scale (A T Beck et al., 1961). BDI cut-off scores have been set by the Centre for Cognitive Therapy (Aaron T. Beck, Epstein, Brown, & Steer, 1988) as no or minimal depression (0-9), mild to moderate depression (10-18) and moderate to severe depression (19 to 29).

6.2.3.2. Experiment 1: "What was where?" WM Task

The main purpose of this first study was to investigate the effects of serotonergic modulation in visual WM by addressing the following questions:

1. Is PD affecting identification accuracy and localisation performance?
2. Are PD patients hindered in their localisation performance due to higher guessing rates?
3. Does serotonin improve WM in PD?
4. Is memory performance modulated by changes in mood from the intake of antidepressant medication?

6.2.3.2.1. Stimuli, Design and Procedure

Stimuli to be remembered consisted of fractals randomly drawn from a library of 196 foils, without repetition for each trial. Stimuli were presented on a black background at a viewing distance of 30 cm and subtended an angle of 2.3°.

The WM task – an adaptation of the "What was Where?" experiment from Pertzov and colleagues (Pertzov et al., 2012) – had a 2 x 2 design where two factors were manipulated: the amount of information to be encoded and the maintenance delay period. The amount of information to be encoded was manipulated by either presenting one or three fractals in the memory display and the delay period was varied by presenting a blank screen for either 1 seconds or 4 seconds (Figure 6.2). The experimental design consisted of three trials with on-screen instructions, four practice trials for each of the conditions (one versus three shapes, one versus four seconds delay). After this introduction, participants performed a total of 120 trials over three blocks, leading to a total of 30 trials per condition presented in a randomized order.

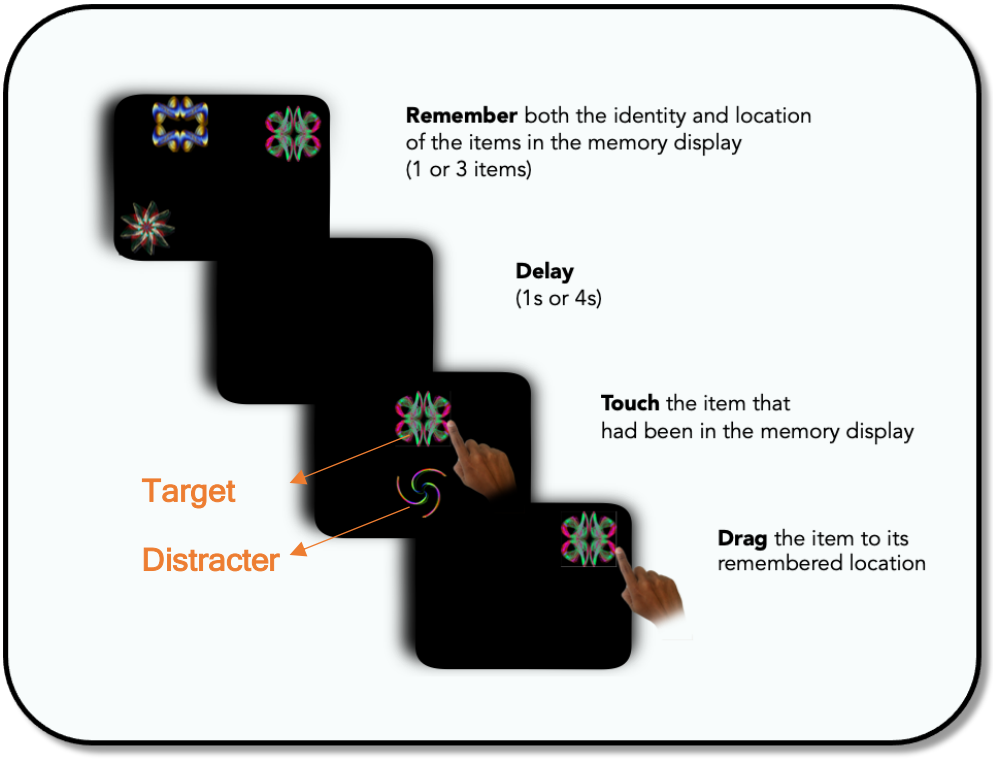


Figure 6.2 | "What was where?" short-term memory task.

Participants were presented with either 1 or 3 items. They had to remember what they looked like, and where they were located on the screen. After a variable delay (1s or 4s), a dual choice was presented. Participants had to touch the item they remembered from a few seconds before, then drag it to where it originally appeared. The task featured four experimental conditions randomly presented, within 3 Blocks of 10 trials per condition.

The behavioural task was performed on an interactive touch-sensitive screen (iPad version 9.3.5 (13G36), model MGTX2B/A) with 1536x2048 pixel matrix. Participants were asked to remember 1 or 3 fractals randomly presented on a black background. Importantly, they were asked to remember what the fractals looked like and where they were presented on the screen. After the items disappeared, a black screen was presented for a variable duration of either 1 second or 4 seconds. This was followed by the presentation of a two-alternative forced choice between the target (the item previously presented in the memory display) and a distracter (a foil taken from the library of fractals used across the experiment but not presented in the memory array). Participants were asked to select the target (identification accuracy) and drag it to its exact remembered location (localisation error). They were instructed to be as precise as possible, thus they were allowed to move the item and adjust its location as many times as they needed. As soon as participants stopped dragging the chosen shape, a "Done"-button appeared in the bottom centre of the screen. This button disappeared if participants started dragging the shape again and reappeared upon release of the shape. The final position was confirmed using this same button. To continue to the next trial, participants had to press a "Next" button. A schematic of the task is shown in Figure 6.2.

6.2.3.3. Experiment 2: "Which emotion was where?" WM and emotion recognition task.

The aim of this second experiment was to investigate the effects of serotonergic modulation on emotion processing. Therefore, the following questions were addressed:

1. Is visual-spatial memory dependent on emotional valence? If so, is this modulated by PD?
2. Is citalopram associated with changes in emotional memory processing? Does it improve overall performance or just processing of positive emotional information?
3. How is emotional valence in visual-spatial memory moderated by the effects in mood from the intake of citalopram?

6.2.3.3.1. Stimuli, Design and Procedure

Stimuli to be remembered consisted of black and white pictures of Caucasian amateur actors posing with three facial expressions: neutral, angry or happy. This sample corresponded to 26 models (13 female and 13 male) with all three emotional expressions, corresponding to a total of 78 face stimuli of the Karonlinska Directed Emotional Faces database (Lundqvist, Flykt, & Öhman, 1998). All the selected face stimuli involved faces at a straight viewing angle. Stimuli were presented on a black background at a viewing distance of approximately 60 cm and subtended an angle of approximately 4.7°.

The emotion recognition task manipulated the emotional expressions to be encoded by presenting three different facial expressions: neutral, angry and happy (Figure 6.3). The experimental design consisted of two practice trials followed by 26 trials per condition, leading to a total of 78 trials presented in a randomized order.

The behavioural task was performed on an interactive touch-sensitive screen (24-inch LCD Dell monitor) with 1920x1080 pixel matrix.

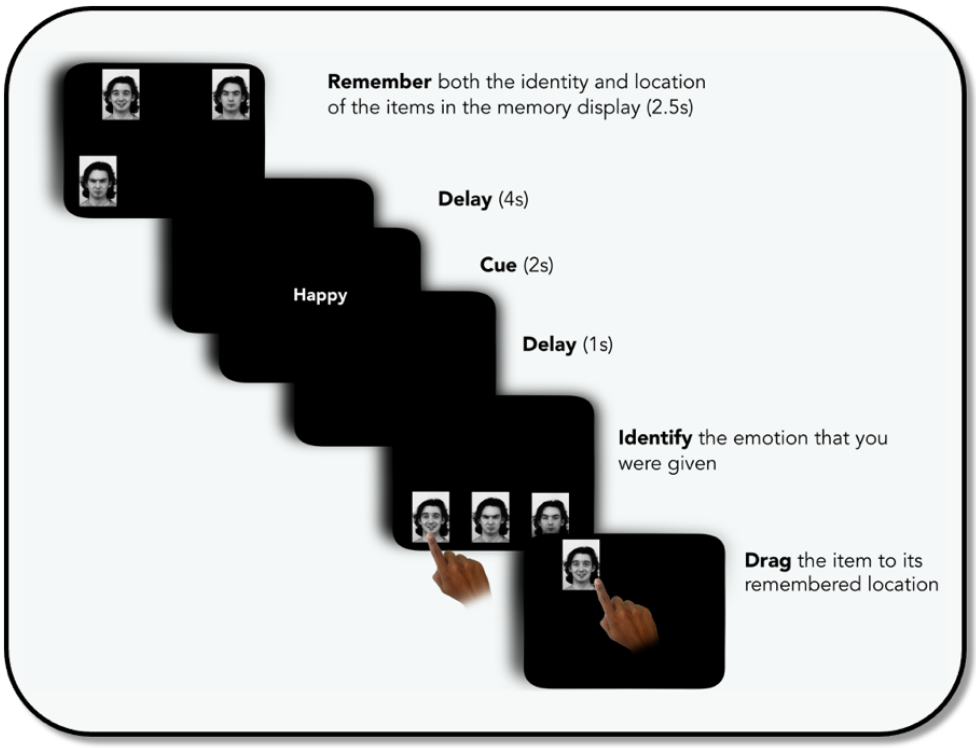


Figure 6.3 | "Which emotion was where?" working memory and emotion recognition task.

Participants were presented with 3 images of the same person with a different emotion (happy, neutral or angry). They had to remember where each emotion was presented on the screen. After a delay of 4 seconds, a cue was presented in the center of the screen. Participants had to identify the emotion they were cued on and then drag it to the location where it originally appeared. The task featured three experimental conditions randomly presented of 26 trials per condition.

Participants were asked to remember 3 emotional expressions. Importantly, they were asked to remember what the expressions looked like and where they were presented on the screen. After the items disappeared, a black screen was presented for 4 seconds, followed by a cue that could be either "Happy", "Neutral" or "Angry", presented in the centre of the screen for 2 seconds. Following a 1second delay, participants had to identify the emotion they were cued on (identification accuracy) and

drag it to its exact remembered location (localisation error). Similarly to the "What was Where?" WM task, they were instructed to be as precise as possible and given the chance to adjust its location as many times as necessary. Once they were happy with their response, they confirmed their answers using the space bar on the keyboard provided to continue to the next trial. A schematic of the task is shown in Figure 6.3.

6.2.3.4. Behavioural Data Analysis and Mixture Model Analysis

The data from the two delayed reproduction tasks used in this study (Experiment one and Experiment two) were analysed in *MATLAB R2018b* using custom scripts and *JASP* version 0.12.1. Behavioural analyses assessed the quality of recall using three measures: identification accuracy, reaction time and localisation error:

- Identification Accuracy: proportion of times the correct memoranda was selected.
- Reaction Time: time (in seconds) to select the target memoranda.
- Localization error: distance (in pixels) between the centre of the reported location and the centre of the target location, which was computed for only correctly identified targets.

Errors due to misreporting the target's location were calculated using the extension to the *MemToolbox* of the mixture model analysis by Grogan and colleagues (Grogan et al., 2019). This allowed to model the source of errors contributing to the pattern of recall performance in precision of recall (imprecision), misbinding and guessing:

- **Imprecision (pixels)**: the variability of responses around the target's location, i.e., the width of the distribution around the target.
- **Misbinding errors**: proportion of times subjects misreported the location of the target by dragging it to the location of another item presented in the memory array.
- **Errors due to chance**: proportion of responding randomly to the target location.

Additionally, because baseline AMI and depression scores (BDI) were significantly increased in patients compared to controls (**Table 6.1**) these were incorporated into the statistical analysis as covariates. Importantly, the covariates were mean-centered prior to running any ANOVA, known as the Delaney-Maxwell method of rescaling (Delaney & Maxwell, 1981). This rescaling of the covariate — a between-subject measure to a repeated measures analysis — ensures that it does not alter the main effect of "within-subjects" factors as the two are orthogonal. Thus, if a PD patient has a memory performance of 70% on citalopram and 80% on placebo, but for each data point his baseline measure of depression is the same, the mean performance of the whole group on placebo versus citalopram should not depend on each participant's baseline measure for depression.

6.2.4. Results

6.2.5. Experiment 1: "What was where?" WM Task

6.2.5.1. Behavioural Results

A total of nineteen patients (one patient was excluded from the analysis due to his dataset being incomplete) and twenty age and gender matched healthy controls completed the behavioural task (see Table 6.1 for detailed demographics).

PD does not affect WM performance

In order to investigate whether PD affected WM performance we started by running a 2 x 2 x 2 repeated measures ANOVA, with set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and both AMI and BDI as covariates.

Identification accuracy, i.e., the proportion with which participants touched the correct item in the two-alternative choice (Figure 6.4, Identification Accuracy), was not modulated by disease as per no significant effect of group [F(1,35) = 2.042, p = 0.162, η_p^2= .055, BF_{incl} = .486]. Identification recall was worse for higher set size [F(1,35) = 162.676, p < .001, η_p^2= .823, BF_{incl} = .376] and poorer for longer delays [F(1,35) = 6.740, p = .014, η_p^2= .161, BF_{incl} = 2.779e^{-24}] (Appendix Table 6.1 - 6.2).

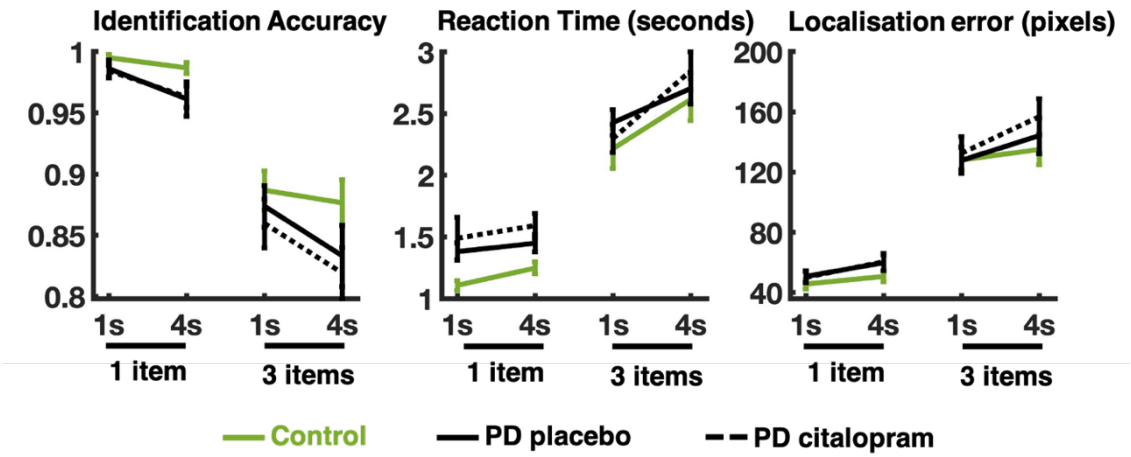


Figure 6.4 | Behavioural results for the "What was where?" short-term memory task.

Neither PD or citalopram were associated with changes in identification accuracy, reaction time or localisation error.

Both patients on placebo and controls took the same amount of time to correctly identify memoranda (Figure 6.4, Reaction Time) as per no effect of group on reaction time [F(1,35) = 2.498, p = .123, η_p^2= .067, BF_{incl} = .552] nor any interactions with other factors (Appendix Tables 6.3 - 6.4). However, results show that higher memory load slowed participants down [F(1,35) = 16.822, p < .001, η_p^2= .325, BF_{incl} = .007] as well as longer delays [F(1,35) = 39.274, p < .001, η_p^2= .529, BF_{incl} = 1.079e^{-39}]. Set size and delay were found to interact, suggesting a differential effect of delay depending on set size [F(1,35) = 16.822, p < .001, η_p^2= .325, BF_{incl} = 3.007]. Post-hoc t-tests revealed that after a delay of 4 seconds participants were significantly slower at correctly identifying memoranda when asked to remember 3 items [t(37) = 7.418, p_{holm} < .001] but when prompted to remember 1 item the delay effect was not as strong [t(37) = 2.305, p_{holm} = .024] (Appendix Tables 6.3 – 6.4).

The distance between the reported location and the original location of the memoranda was subsequently investigated, and trials with incorrect identifications

excluded from further analysis (**Figure 6.4, Localisation error**). Patients did not perform differently relative to controls, as per no main effect of group on localisation error [F(1,35) = .595, p = .446, η_p^2= .017, BF_{incl} = .195]. The main effect of set size was significant [F(1,35) = 207.698, p < .001, η_p^2= .856, BF_{incl} = .706] reflecting larger errors when three items were presented (**Figure 6.4, Localisation error**). Additionally, a main effect of delay [F(1,35) = 7.016, p = .012, η_p^2= .167, BF_{incl} = 7.597e^{-38}] exhibited an increased proportion of localization errors after a 4 second delay (**Appendix Tables 6.5 - 6.6**).

Citalopram has no effect on identification accuracy nor localisation ability

To investigate the mechanism of potential medication effect, we tested for differences between PD on placebo and PD on citalopram on their WM performance by running a 2 x 2 x 2 repeated measures ANOVA, with drug (patients on placebo versus citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and both AMI and BDI as covariates.

Citalopram did not modulate patients' ability to correctly identify memoranda [F(1,16) = .512, p = .485, η_p^2= .031, BF_{incl} = 1.013e^{-27}] (**Figure 6.4, Identification Accuracy**). As per the previous analysis, higher set sizes [F(1,16) = 91.400, p < .001, η_p^2= .851, BF_{incl} = .018] and longer delays [F(1,16) = 16.717, p < .001, η_p^2= .2511, BF_{incl} = 9.492 e^{-27}] hindered their performance (**Appendix Tables 6.13 - 6.14**).

Similarly, patients on citalopram did not differ from those on placebo in how fast they selected the correct memoranda [F(1,16) = 1.165, p = 0.296, η_p^2= .068, BF_{incl} =

1.101^{-34}] (**Figure 6.4, Reaction Time**). However, we did find a significant effect of set size [F(1,16) = 111.786, $p < .001$, $\eta_p^2 = .875$, $BF_{incl} = .002$] and delay [F(1,16) = 55.149, $p < .001$, $\eta_p^2 = .775$, $BF_{incl} = 8.611\ e^{-34}$]. These two factors were also found to interact [F(1,16) = 7.423, $p = .015$, $\eta_p^2 = .317$, $BF_{incl} = 9.732$], suggesting that delay had a differential effect on how fast patients selected the target depending on how many items they were presented at encoding. Post-hoc t-tests revealed that when asked to remember 3 items, participants took longer to identify the target after a 4 second delay compared to 1 second [t(17) = 5.996, $p_{holm} < .001$], but this was no longer the case when given 1 item to remember [t(17) = 1.232, $p_{holm} = .229$]. (**Appendix Tables 6.15 - 6.16**).

Localisation error was not modulated by citalopram as per no significant effect of drug [F(1,16) = .827, $p = .377$, $\eta_p^2 = .049$, $BF_{incl} = 2.821e^{-37}$] (**Figure 6.4, Localisation error**). Again, higher set sizes [F(1,16) = 139.898, $p < .001$, $\eta_p^2 = .897$, $BF_{incl} = .045$] and longer delays [F(1,16) = 8481.424, $p = .003$, $\eta_p^2 = .427$, $BF_{incl} = 1.017\ e^{-36}$] had a detrimental effect on localisation performance (**Appendix Tables 6.17 - 6.18**).

After taking citalopram, improvement in BDI scores correlates with enhanced identification accuracy

So far, any potential differences in performance that could be related to apathy or depression have been discounted by using these measures as covariates. Here, to investigate the relationship between the differences in memory performance (difference in scores on citalopram versus placebo) and the differences in apathy and BDI, a Pearson correlation analysis was implemented. Importantly, the BDI and AMI scores used in this

analysis were no longer the ones collected at baseline, but rather the scores measured on the respective research visit (either on placebo or citalopram).

Results show a significant correlation between differences in identification performance after citalopram intake and changes in BDI [r(17) = -.504, *p* =.028, BF_{10} = 2.693] but not AMI [r(17) = .071, *p* =.773, BF_{10} = .295]. This suggest that patients who underwent the greatest positive change in their mood, i.e., the ones who experienced the greatest improvement of their BDI scores after taking citalopram, exhibited better identification accuracy (**Figure 6.5**). Conversely, participants who displayed worse BDI scores after taking citalopram, or even participants who showed a lower positive sensitivity to the medication, i.e., the ones who improved only marginally, were associated with lower identification accuracy. None of the BDI and AMI scores correlated significantly with reaction time nor localisation error (*p* > .094). Details in **Appendix Table 6.19 - 6.20**.

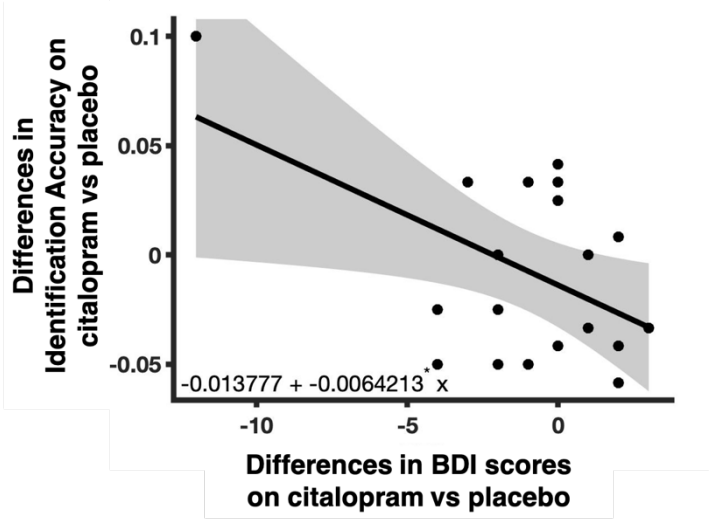


Figure 6.5 | Patients who displayed better BDI scores after taking citalopram also exhibited better identification accuracy.

Relationship between the effects of citalopram on BDI scores and the effects of citalopram on identification accuracy. Patients less depressed on citalopram compared to placebo, display better identification accuracy on citalopram compared to placebo.

Inspection of Figure 6.5 shows that there was one participant whose BDI score and identification accuracy improved substantially. The reported correlation analysis was based on the premise that this participant is not an outlier, but rather truly representative of a behaviour related to the pathology being investigated. Although they are ~3 SDs above the mean for BDI, this is not true for most of the behavioural metrics; importantly, their reaction times are within 1 SD from the mean. In fact, in a normal distribution, around 1 in 330 observations will be at least 3 SD away from the mean, and this extreme value found in our small sample size could be due to random chance but still a natural part of the data distribution, as opposed to a measurement error. Nevertheless, if in effect this is an outlier, then a parametric test is less than ideal and either removing the outlier or applying a nonparametric hypothesis test, which is robust to outliers, would be most appropriate. Therefore, Spearman's correlation — a nonparametric alternative to Pearson's correlation — was also implemented.

Results from the nonparametric analysis no longer showed a significant relationship between differences in identification performance after citalopram intake and changes in BDI [r(17) = -.209, *p* =.391, BF_{10} = .442]. However, there was a significant relationship between differences in reaction time and changes in AMI after intake of citalopram [r(17) = .480, *p* =.038, BF_{10} = .509], which was absent using its parametric alternative. This relationship suggests that patients who display lower apathy scores after taking citalopram are also quicker at correctly identifying memoranda (Figure 6.6, Appendix Table 6.19 - 6.20). The limited sample size limits definitive conclusions.

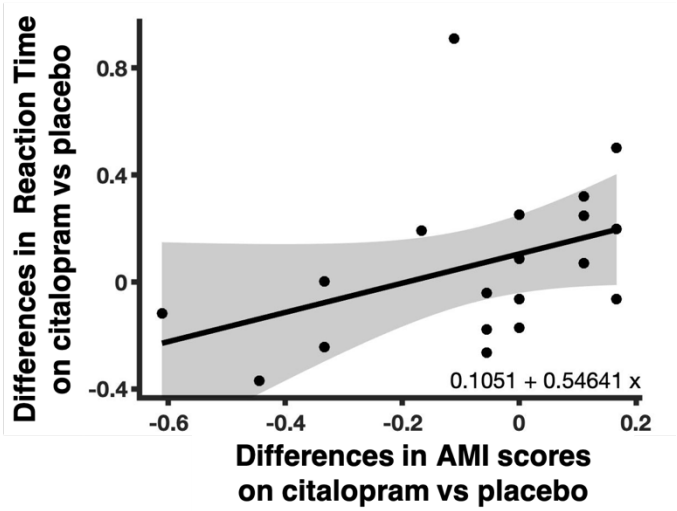


Figure 6.6 | Patients who displayed better AMI scores after taking citalopram also exhibited shorter reaction times.

Relationship between the effects of citalopram on AMI scores and the effects of citalopram on reaction time when applying a nonparametric test. Patients less apathetic on citalopram compared to placebo are also faster at identifying the correct memoranda after citalopram intake.

6.2.5.2. Mixture Model Results

PD is not associated with poorer memory resolution

A 2 x 2 ANOVA of delay as a "within-subjects" factor, group as a "between-subjects" factor, and both AMI and BDI as covariates was conducted for imprecision, misbinding and guessing (**Figure 6.7**).

Patients on placebo and controls did not differ in how precisely they located memoranda [F(1,35) = 724.016, p = .177, η_p^2= .051, BF_{incl} = .669] (**Figure 6.7, Imprecision**). However, we found that longer delays lead to greater imprecision [F(1,35) = 7.346, p = .010, η_p^2= .173, BF_{incl} = 1.00] (**Appendix Tables 6.7 - 6.8**).

Results do not support a disease-related deficit in binding the identity of memoranda with its location [F(1,35) = .196, p = .661, η_p^2= .006, BF_{incl} = .283] (**Figure 6.7, Misbinding**), nor do they support a greater number of localization errors due to guessing, as they were similar for both groups [F(1,35) = .721, p = .402, η_p^2= .020, BF_{incl} = .375] **(Figure 6.7, Guessing**). Even though group and delay were found to interact [F(1, 35) = 4.317, p = .044, η_p^2= .111, BF_{incl} = .564], post-hoc t-tests show there is no support for this interaction (**Appendix Table 6.11**). Details in **Appendix Tables 6.9 - 6.12**.

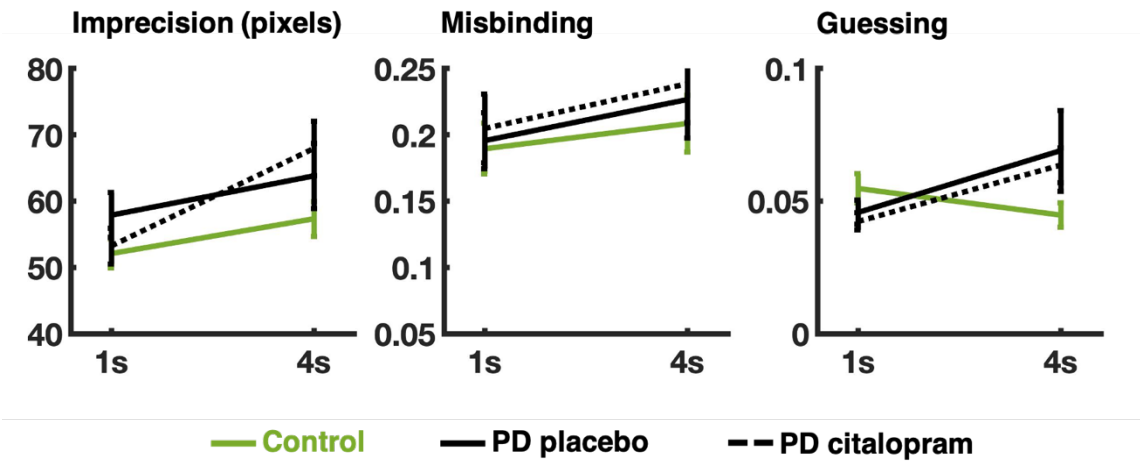


Figure 6.7 | Mixture model results for the "What was where?" short-term memory task.

Patients and controls performed similarly for imprecision, misbinding and guessing. However, the intake of citalopram was found to increase patients' imprecision after 4 second maintenance delay.

Citalopram increases patients' imprecision after a 4-second delay

A 2 x 2 ANOVA of drug and delay as "within-subjects" factors, and AMI and BDI as covariates, was conducted for imprecision, misbinding and guessing (Figure 6.7). Neither delay [F(1,16) = 17.030, $p < .001$, $\eta_p^2 = .516$, $BF_{incl} = 1.00$] nor imprecision [F(1,16) = .002, $p = .888$, $\eta_p^2 = .001$, $BF_{incl} = 8.581\ e^{-5}$] were significantly affected by the intake of citalopram (Figure 6.7, Imprecision). However, drug and delay were found to interact, suggesting that delay had a differential effect on imprecision depending on whether or not participants were on the serotonergic medication [F(1,16) = 379.997, $p = .004$, $\eta_p^2 = .419$, $BF_{incl} = 1.518$]. Post-hoc t-tests show that citalopram significantly increased imprecision after a delay of 4 seconds [t(17) = 5.226, $p_{holm} < .001$]. However, this was not the case for placebo [t(17) = 2.080, $p_{holm} = .145$] (Appendix Tables 6.21 - 6.22).

The proportion of errors due to misbinding (**Figure 6.7, Misbinding**) was not modulated by citalopram as per no main effect of drug [F(1,16) = .185, *p* = .673, η_p^2= .011, BF_{incl} = .276]. Likewise, guessing (**Figure 6.7, Guessing**) was not moderated by drug [F(1,16) = .294, *p* = .595, η_p^2= .018, BF_{incl} = .034], but we found a significant increase in the proportion of errors due to guessing after a 4-second delay [[F(1,16) = 6.424, *p* = .022, η_p^2= .286, BF_{incl} = 1.00] (**Appendix Tables 6.23 - 6.26**).

Greater improvement in BDI scores after citalopram intake correlates with decreased imprecision

Pearson correlation analysis was applied to investigate the relationship between the differences in memory performance (difference in scores on citalopram versus placebo) and the differences in apathy and depression. Note that the BDI and AMI scores used in this analysis were collected on both research visits: either after citalopram intake or placebo.

A significant correlation was found between differences in imprecision after citalopram intake and changes in BDI scores [r(17) = -.509, *p* =.026, BF_{10} = 2.847] but not AMI r(17) = -.162, *p* =.506, BF_{10} = .349]. In effect, this suggests that depression plays a significant role on memory performance, i.e., people that were very responsive to citalopram who showed greater improvement in their BDI scores also displayed decreased imprecision. BDI and AMI scores did not correlate with any of the other mixture model measures: neither guessing nor misbinding (*p* > .061, **Figure 6.8**). Details in **Appendix Table 6.27 - 6.28**.

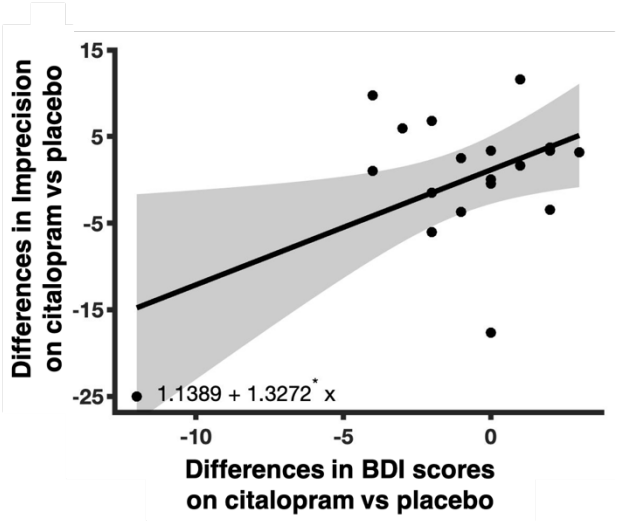


Figure 6.8 | Improvement of BDI scores after citalopram intake is associated with decreased imprecision.

The relationship between the effects of citalopram on imprecision and BDI scores. Patients less depressed on citalopram compared to placebo displayed lower imprecision on citalopram compared to placebo.

Again, note that although the participant who displays an extreme difference in their BDI (Figure 6.8) has not been classified as an outlier, but rather as a true representation of a behaviour related to PD pathology, nonparametric tests were applied in case this was indeed a measurement error. Spearman's correlations no longer found significant differences between imprecision after citalopram intake and changes in BDI scores [r(17) = .155, p =.526, BF_{10} = .377].

6.2.6. Experiment 2: "Which emotion was where?" WM and Emotion recognition task

6.2.6.1. Behavioural Results

A total of 20 patients and 20 age and gender matched healthy controls completed the emotion behavioural task (Table 6.1). Note that because of non-normality in the reaction time data, a log transform was computed on this metric. Also note that cases where the assumption of sphericity was violated (Mauchly's test of sphericity had a $p <$ .05) were corrected for by reporting Greenhouse-Geisser.

PD patients take longer to identify emotions compared to controls, particularly when presented with neutral and angry faces

In order to investigate whether PD was associated with changes in the ability to correctly identify and locate emotions, we started by running a 3 x 2 repeated measures ANOVA, with emotion (neutral, angry, happy) as a "within-subjects" factor, group (controls versus patients on placebo) as a "between-subjects" factor, and both AMI and BDI as covariates.

The proportion with which participants touched the correct item in the two-alternative choice (Figure 6.9, Identification Accuracy) was not modulated by disease [F(1,36) = .614, p = .438, η_p^2= .01, BF_{incl} = .320] nor emotion [F(1.207,43.460) = 3.437, p = .038, η_p^2= .087, BF_{incl} = 1.00], nor was there an interaction between the two

[F(1.207,43.460) = 1.641, p = 0.209, η_p^2= .044, BF_{incl} = .296] (**Appendix Tables 6.29 - 6.30**).

PD patients took longer than controls to correctly identify memoranda [F(1,36) = 8.656, p = .006, η_p^2= .194, BF_{incl} = 5.216] (**Figure 6.9, Reaction Time**). Additionally, a main effect of emotion [F(2,72) = 95.416, p <.001, η_p^2= .726, BF_{incl} = .160] revealed that selecting the neutral emotion took longer compared to both the angry [t(38) = 4.409 < .001] and happy emotions [t(38) = 13.578 < .001]. These two factors were not found to interact [F(2,72) = .666, p = .517, η_p^2= .018, BF_{incl} = .127]. See **Appendix Tables 6.31 - 6.32** for further details.

Localisation performance **(Figure 6.9, Localisation error)** was not modulated by disease as per no significant effect of group [F(1,36) = 2.247, p = .143, η_p^2= .059, BF_{incl} = .700], nor was it affected by emotion [F(2,72) = .640, p = .530, η_p^2= .017, BF_{incl} = .087]. No emotion by group interaction was found [F(2,72) = .108, p = .898, η_p^2= .003, BF_{incl} = .028] (**Appendix Tables 6.33 - 6.34**).

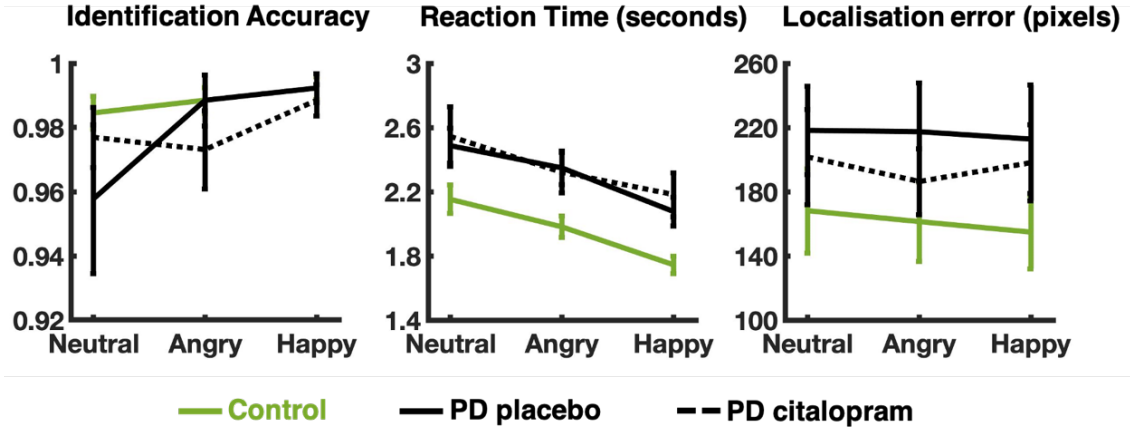


Figure 6.9 | Behavioural results for the Emotion task.

Patients took longer to identify emotions compared to controls, particularly when selecting neutral and angry faces, compared to happy. Moreover, citalopram does not change PD patients' ability to identify and localise emotional memoranda.

Citalopram does not modulate patients' ability to identify or localise memoranda with emotional valence

Effects of medication on WM were tested looking at the differences between PD on placebo and PD on citalopram. Therefore a 2 x 3 repeated measures ANOVA, with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors and both AMI and BDI as covariates was applied.

Citalopram did not modulate patients' identification accuracy [F(1,17) = 8.036 e^{-8}, p = 1, η_p^2= 4.727e^{-9}, BF_{incl} = .195] (**Figure 6.9, Identification Accuracy**). There was also no significant main effect of emotion [F(1.300,22.107) = 2.005, p = .169, η_p^2= .106, BF_{incl} = .611] nor was there an interaction between drug and emotion [F(2,34) = 2.889, p = .069, η_p^2= .145, BF_{incl} = .097] (**Appendix Tables 6.41 - 6.42**).

Even though reaction time (**Figure 6.9, Reaction Time**) was not affected by the intake of citalopram, as suggested by no main effect of drug [F(1,17) = .040, p = .843, η_p^2=.002, BF_{incl} = 1.197e^{-10}], it was, however, modulated by emotion [F(2,34) = 66.532, p < .001, η_p^2= .796, BF_{incl} = 1], with PD selecting happy emotions faster compared to both neutral [t(17) = 11.335 < .001] and angry emotions [t(17) = 7.524 < .001]. Drug and emotion were not found to interact [F(2,34) = 1.103, p = .343, η_p^2= .061, BF_{incl} = .134] (**Appendix Tables 6.43 - 6.44**).

Localisation error (**Figure 6.9, Localisation error**) was similarly unaffected by any of the "within-subjects" factors (all p >.147). See **Appendix Tables 6.45 - 6.46** for further details on null results.

Improvement of BDI after citalopram intake, but not AMI, correlate with better identification accuracy for neutral faces and decreased localisation error for happy faces

Pearson correlation analysis was applied to investigate the relationship between the differences on the impact of emotional valence in memory performance (difference in scores on citalopram versus placebo) and the differences in apathy and depression. Note that the BDI and AMI scores used in this analysis were collected on both research visits: either after citalopram intake or placebo.

Results indicate that greater positive change in mood (BDI scores) after citalopram intake is correlated with improved identification accuracy for neutral faces [r(18) = -.573, *p* =.008, BF_{10} = 7.138] and decreased localisation error for happy faces [r(18) = .472, *p* =.036, BF_{10} = 2.180] (Figure 6.10). This suggest that people who were more responsive to citalopram — experiencing greater positive BDI changes — had increased ability to correctly identify memoranda with neutral valence, as well as greater localisation ability for memoranda with happy valence (Appendix Table 6.47 - 6.48).

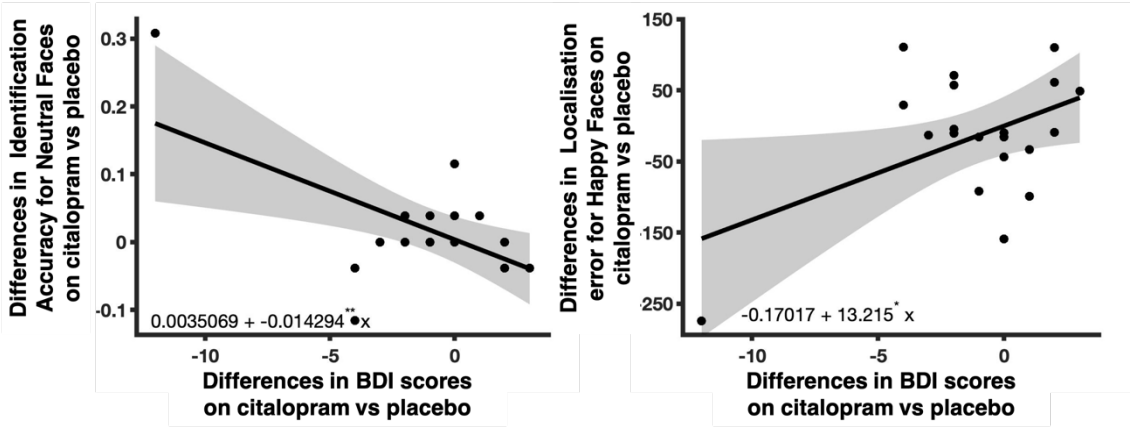


Figure 6.10 | Improvement of BDI scores after citalopram intake was associated with increased identification accuracy for neutral faces (left) and decreased localisation error for happy faces (right).

The relationship between the effects of citalopram on identification accuracy for neutral faces and BDI scores (graph on the left) showed that patients who were less depressed on citalopram compared to placebo also displayed increased identification accuracy. Additionally, the relationship between the effects of citalopram on localisation error for happy faces and BDI scores (graph on the right) illustrates that participants who undergone the greatest positive change in their mood displayed decreased localisation error.

Again, Spearman's correlations no longer found a significant relationship between BDI scores after citalopram intake and identification accuracy for neutral faces [r(18) = -.050, p =.833, BF_{10} = .291] nor with localisation error for happy faces [r(18) = .031, p =.896, BF_{10} = .286]. Details in Appendix Table 6.47 - 6.48.

6.2.6.2. Mixture Model Results

Memory resolution for emotions is not modulated by PD

In order to investigate whether PD was associated with changes in memory resolution (imprecision, errors due to misbinding or guessing) a 3 x 2 repeated measures ANOVA with emotion (neutral, angry, happy) as a "within-subjects" factor, group

(controls versus patients on placebo) as a "between-subjects" factor, and both AMI and BDI as covariates, was conducted.

Disease did not affect how precisely participants located memoranda [F(1,36) = .799, p = .377, η_p^2= .022, BF_{incl} = .357], nor did emotion [F(1.54, 41.528) = .727, p = 0.417, η_p^2= .020, BF_{incl} = .012]. These two factors were not found to interact [F(1.54, 41.528) = .165, p = 0.723, η_p^2= .005, BF_{incl} = .024] (**Figure 6.11, Imprecision**). (**Appendix Tables 6.35 - 6.36**).

Patients do not appear to be impaired in their ability to correctly bind the correct emotion to its location (**Figure 6.11, Misbinding**) as per no main effect of group [F(1,36) = 3.152, p = .084, η_p^2= .081, BF_{incl} = .744] or emotion [F(1.477,53.154) = .193, p = .758, η_p^2= .005, BF_{incl} = .083]. We also did not find an interaction effect between the two [F(1.477,53.154) = .858, p = .400, η_p^2= .023, BF_{incl} = .047] (**Appendix Tables 6.37 - 6.38**). Similarly, they did not exhibit increased errors due to guessing (**Figure 6.11, Guessing**) as per no main effect of group [F(1,36) = .240, p = .627, η_p^2= .007, BF_{incl} = .371]. Guessing was also not modulated my emotion [F(1.276,45.940) = .897, p = .373, η_p^2= .024, BF_{incl} = .041] nor was there an interaction between emotion and group [F(1.276,45.940) = .566, p = .496, η_p^2= .015, BF_{incl} = .043] (**Appendix Tables 6.39 - 6.40**).

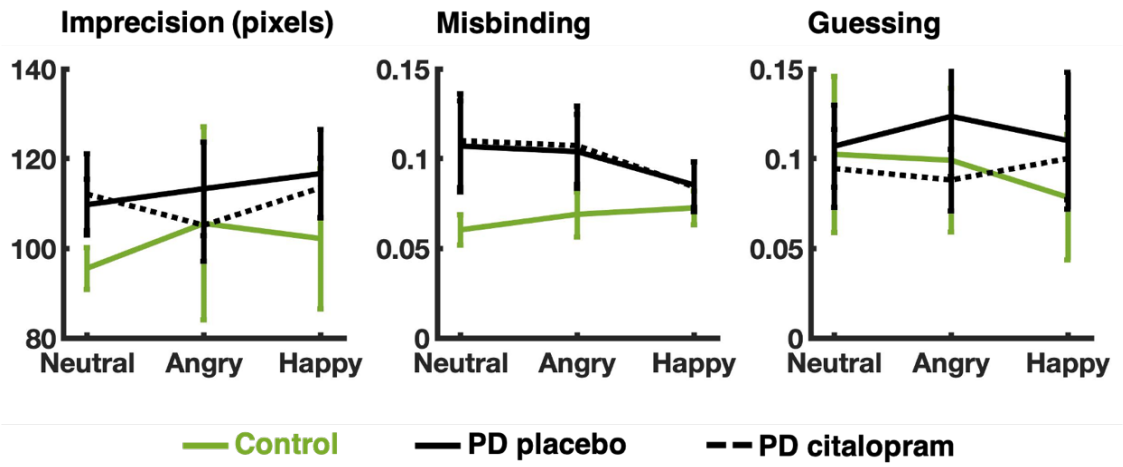


Figure 6.11 | Mixture model results for the Emotion task.

PD is not predictive of changes in memory resolution for memoranda with emotional valence, nor is citalopram.

Citalopram does not affect memory resolution for emotional memoranda

No "within-subjects" factors were found to significantly impact imprecision, misbinding or guessing (p > .082 and p > .342, respectively) (Figure 6.11). Details in Appendix Tables 6.49 - 6.54.

Differences in BDI and AMI after citalopram correlate with changes in WM resolution

To investigate the relationship between the differences on the impact of emotional valence in memory resolution (differences in scores on citalopram versus placebo) and differences in apathy and depression, Pearson correlation analysis was applied. Note that the BDI and AMI scores used in this analysis were collected on both research visits: after the citalopram intake or placebo.

Participants who experienced the greatest improvement of their BDI scores, exhibited lower imprecision for memoranda with angry and happy valence (**Figure 6.12, first row**) [r(18) = .479, *p* =.033, BF_{10} = 2.342, r(18) = .556, *p* =.011, BF_{10} = 5.668], respectively] as well as lower proportion of guessing for angry faces [r(18) = .609, *p* =.004, BF_{10} = 12.275] (**Figure 6.12, last row**). Conversely, participants who displayed worse BDI and AMI scores after taking citalopram, and participants who showed a lower positive sensitivity to the medication, were associated with higher rates of misbinding for angry faces and lower rates of misbinding for happy faces, respectively [r(18) = -.548, *p* =.012, BF_{10} = 5.120, r(18) = .476, *p* =.034, BF_{10} = 2.271] (**Figure 6.12, second row**). See **Appendix Table 6.55 - 6.56**.

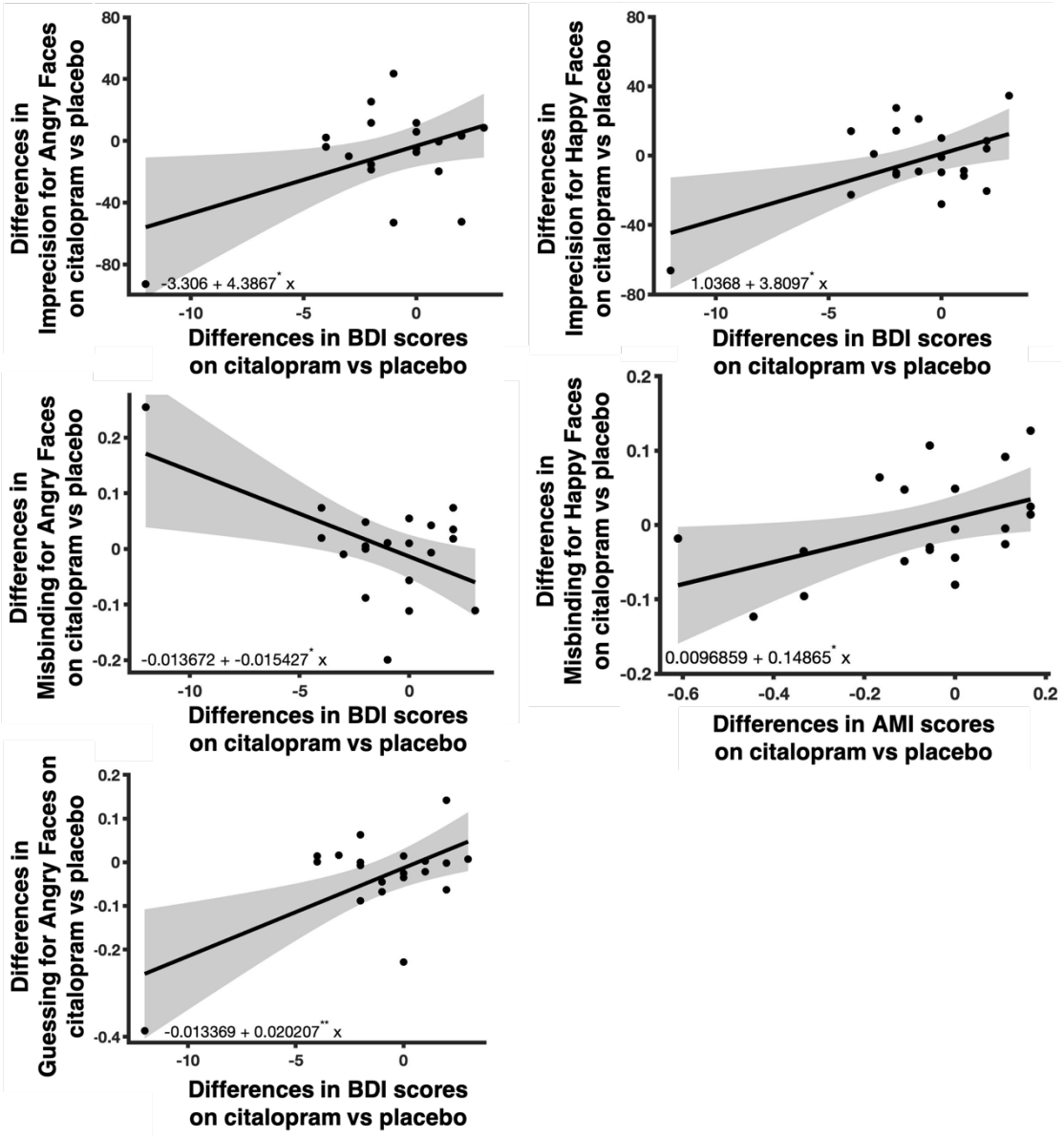


Figure 6.12 | Relationship between mood changes after citalopram intake and the mixture model results.

Patients who had the greatest improvement in their BDI scores following citalopram intake, exhibited decreased imprecision for both angry (first row, graph on the left) and happy faces (first row, graph on the right). They also showed a decreased number of random responses (las row). Conversely, greater proportion of errors due to misbinding for angry faces was associated with greater improvement of BDI scores (second row, graph on the right). Decreased misbinding for happy faces was related to improvement in AMI scores following citalopram intake (second row, graph on the left).

Note that, similarly to previous correlation analysis, all participants were treated as a normal part of the patient data distribution, as opposed to outliers due to

measurement inaccuracies. However, nonparametric tests were applied in case these patients scores were indeed measurement errors. Using Spearman's correlations there was no longer a significant relationship between changes in BDI scores after citalopram intake and imprecision for angry [r(18) = .160, *p* =.502, BF_{10} = .341] or happy faces [r(18) = .163, *p* =.493, BF_{10} = .373]. Nor did results show a significant correlation between variations in BDI scores and differences in misbinding memoranda with angry valence [r(18) = -.165, *p* =.487, BF_{10} = .356] or guessing for angry faces [r(18) = .062, *p* =.794, BF_{10} = .309]. However, the significant association between higher rates of misbinding for faces with a happy valence and AMI scores after citalopram intake [r(18) = .478, *p* =.033, BF_{10} = 2.867] remained (**Figure 6.12, second row**). See **Appendix Table 6.55 - 6.56**.

6.3. *Discussion*

This study aimed to investigate how serotonergic modulation through the administration of 20 mg citalopram affects cognitive and emotional memory processing in patients with idiopathic PD. Age and gender-matched healthy controls were included to test for general effects of PD. A double-blind, placebo-controlled within-groups cross-over design was implemented, where each patient received seven days of citalopram and a matched placebo in a counterbalanced, randomized order. Patients' WM identification accuracy and localisation performance was not impaired compared to controls (**Figure 6.4**) nor was their memory resolution (**Figure 6.7**). However, emotional valence was found to affect visuospatial WM in PD as opposed to controls: PD patients took longer to identify neutral and angry emotions compared to happy (**Figure 6.9**). Finally, when investigating the relationship between differences in memory performance and

differences in apathy and depression following citalopram intake, citalopram modulates mood — reflected by a greater decrease in depression scores over just seven days — which correlated with better identification accuracy for neutral faces, decreased localisation error for happy faces (**Figure 6.10**), decreased imprecision for both angry and happy faces, and decreased guessing for angry faces (**Figure 6.12**).

WM performance in patients with PD on placebo and healthy controls was explored using a sensitive, continuous analogue reproduction task (Pertzov et al., 2012) that provides, not just a measure of responses in a binary manner, systematically used in change detection studies to estimate how many items people can recall (J. S. Luck & Vogel, 1997), but further estimates the quality of the memory representation. Thus, making it possible to assess memory resolution, dissociating the sources of error in imprecision, misbinding, and guessing, by applying a computational model of response error (Grogan et al., 2019). Results on the "What was where?" WM task (**Figure 6.2**) did not find a disease related impairment on identification accuracy, reaction time or localisation performance in this group of PD patients (**Figure 6.4**).

Like other studies using this paradigm (Pertzov et al., 2012, 2015), participants' recall was hindered by higher set sizes (3 items compared to 1 item) and longer maintenance delays (4-second delay as opposed to 1 second). However, we failed to replicate previous findings and did not observe increased rates of guessing in PD compared to controls (**Figure 6.7**) (Rolinski et al., 2016; Zokaei & Husain, 2019b; Zokaei, Mcneill, et al., 2014; Zokaei et al., 2020). Although our sample size was similar to previous studies on STM using a similar task (Rolinski et al., 2016; Zokaei et al., 2020), this could be due to small sample size and overall high general cognitive screening test scores; in

this study, there was no significant difference between patients and controls, just as in Zokaei and colleagues' work (2020), but their PD cohort presented lower ACE scores (93.8 ± 4.6) compared to the one in this study (95.5 ± 4.4). Another possible explanation is that the memory task was performed on a smaller screen size in this study: we used an interactive touch-sensitive screen (iPad version 9.3.5 (13G36) at a viewing distance of 30 cm and subtended an angle of 2.3°, model MGTX2B/A) with 1536x2048 pixel matrix, whereas they used a bigger touchscreen (Inspiron All-in-One 2320; DELL at a viewing distance of approximately 62 cm, corresponding to 62 x 35 of visual angle) with a 1920 x 1080 pixel resolution. Additionally, the fact that PD patients in this study were chosen on the basis of several exclusion criteria, with a mere 20% being eligible to take part, could have biased our sample, which may not be representative of the cohort as a whole.

Even though serotonergic dysfunction occurs early in PD, a 7-day administration of 20mg of citalopram did not affect patients' WM identification accuracy nor localisation ability (**Figure 6.4**). This is in accordance with a study by Harmer et al. (Harmer et al., 2002) which shows that administering 10mg of citalopram intravenously in twenty-four healthy young females did not improve immediate recall on the verbal memory task where participants had to repeat a 15-item word list. Nevertheless, the authors did find that increased citalopram facilitated recall if participants were asked to repeat the same list fifteen minutes later. In contrast, by modulating serotonin in the opposing direction, with acute tryptophan depletion (ATD) in healthy people, others have consistently found decreased memory consolidation while leaving STM intact (Park et al., 1994; Riedel et al., 1999; Rubinsztein et al., 2001; Schmitt et al., 2000). However, these studies employed a paradigm in which the index used to measure WM is the number of items which people

can recall correctly but does not take into account the quality with which the memory representation regarding that item is stored. The delayed-reproduction task used in this study allowed to assess the resolution of memory representation by modelling the sources of error contributing to the pattern of memory recall and found that delay had a differential effect on imprecision depending on whether or not participants were on the serotonergic medication. Patients on citalopram displayed increased imprecision after a 4-second delay, but not for 1 second (**Figure 6.7**). No effect on misbinding nor guessing was found (**Figure 6.7**).

Previously, depression associated with impaired WM performance has been shown to improve following antidepressant treatment (Bondareff et al., 2000; Keegan et al., 1991; Peselow et al., 1991). In line with this, results arising from the present study show a significant correlation between differences in identification performance after citalopram intake and changes in depression, i.e., patients who underwent the greatest improvement in their depression scores after taking citalopram exhibited higher identification accuracy (**Figure 6.5**) and decreased imprecision (**Figure 6.8**). This suggests that antidepressant drug treatment effects on WM may be reliant on mood improvement. However, the small sample size contributes to uncertainty of the effect of a person who showed a big change in their BDI. For a definitive conclusion, future larger investigations or a replication study may be able to confirm this link.

To further characterise the effects of PD and citalopram, patients were tested on the "Who was where?" WM and emotional recognition task (**Figure 6.3**). Firstly, a PD related impairment in face recognition was investigated. Identification accuracy and localisation performance was not affected by disease. However, PD was found to affect

the speed with which memoranda was selected, with patients finding it harder than controls to identify emotions: neutral and angry emotions were harder to identify compared to happy (**Figure 6.9**). These results are somewhat consistent with a facial emotional recognition (FER) deficit in PD, particularly for negative emotions (Argaud et al., 2018; Gray & Tickle-Degnen, 2010; Péron et al., 2012). In fact, studies of emotional facial expression have yielded ambivalent results, some reporting diminished facial expression recognition in PD compared to controls (Clark et al., 2008; Dujardin et al., 2004; Kan et al., 2002; Yip et al., 2003) and others failing to demonstrate any such differences (Adolphs et al., 1998; Dewick et al., 1991; Madeley et al., 1995; Pell & Leonard, 2005).

Gray and Tickle-Degnen's meta-analysis showed a strong link between PD and impaired recognition of emotion; importantly, they revealed that this cross-model deficit appeared to be greater from the recognition of emotion from prosody, as opposed to facial expression (Gray & Tickle-Degnen, 2010). Furthermore, despite using a sensitive delayed-reproduction task to assess subtle behavioural impairment, patients were not found to be impaired in their memory resolution (imprecision, misbinding, guessing) compared to controls in our study (**Figure 6.11**). However, again it is unclear how these results might have been confounded by an interaction with pathological dopaminergic baseline. A review by Lawrence et. al. (2007) evidenced the ventral striatal dopamine systems as the source of a selective impairment in the recognition of negative emotions, particularly anger, disgust and fear. Moreover, emotion discrimination was not appraised with this paradigm and it is conceivable that an intensity rating at the end of each trial could have improved the current paradigm.

Inspection of the medication effect related to memory performance for memoranda with different emotional valence (neutral, angry and happy) revealed that citalopram did not modulate patients' ability to identify or localise memoranda with emotional valence (**Figure 6.9**) nor did it bear any effect on memory resolution (**Figure 6.11**). A failure to observe any serotonergic-related changes in emotional processing could be due to 1) insufficient amount of serotonin administered, 2) an exceedingly short duration, or 3) evidence for a lack of sensitivity to the serotonergic pathway in the dopamine-repleted group at test. Future research that manipulates both dopaminergic and serotonergic levels would be in a position to rule out these alternative possibilities.

Finally, we investigated how emotional valence in visuospatial memory is manipulated by the effects in mood from the intake of citalopram. Taking the SSRI was found to reflect a greater decrease in depression scores, which correlated with better identification accuracy for neutral faces, decreased localisation error for happy faces (**Figure 6.10**), decreased imprecision for both angry and happy faces, and decreased guessing for angry faces (**Figure 6.12**). This intimates that it is not the antidepressant itself that is regulating the effects of emotional valence directly on visual WM, but indirectly through its effect on mood. Although Gray and Tickle-Degnen's meta-analysis posits that emotion recognition deficits in PD are independent of depression (Gray & Tickle-Degnen, 2010), the results presented in this study corroborate that face emotion recognition deficits are not specific to PD but may be linked to mood disturbances arising from disruption of emotional processing in depression (Bourke, Douglas, & Porter, 2010; Delle-Vigne, Wang, Kornreich, Verbanck, & Campanella, 2014), accompanied by a higher incidence of depression in PD (Reijnders, Ehrt, Weber, Aarsland, & Leentjens, 2008;

Remy, Doder, Lees, Turjanski, & Brooks, 2005). Nonetheless, the small sample size contributes to uncertainty of the effect of an individual who displayed a large variation in their BDI. For a definitive conclusion free from the limitations of the present investigation, a replication or larger study are necessary to corroborate this.

Together, these findings provide support for serotonergic effects on WM to be reliant on mood improvement and a possible role of the serotonergic degeneration in processing negative emotional information.

Chapter 7. General Discussion

This thesis set out to contribute to the understanding of the mechanisms underlying WM and how these might be disrupted in clinical populations. This was achieved in two parts: **Part I** aimed at gaining mechanistic insight into the process of WM retrieval and the relationship between processing demands, including filtering of information, at all cognitive stages. **Part II** was motivated by its clinical relevance to patients with neurological disorders and intended at investigating malfunctions of the underlying processes and aspects of behavioural changes in LE and PD.

Here, I will start by outlining the main findings reported in all five empirical chapters and assess the overall conclusions that can be drawn from the data therein. This will be followed by a short discussion on how these findings contribute to existing WM theoretical frameworks. The relevance of translational research, such as the ones reported in Part II of the thesis, and its impact on supporting clinical interventions will also be discussed. Finally, future directions will be contemplated.

7.1. *Overview of findings*

Part I of this work aimed to contribute to the understanding of the cognitive processes underpinning WM in healthy people. Across all experiments, a delayed reproduction "What-was-where?" design was implemented where identification and corresponding location of memoranda needed to be remembered. At recall, a dual choice was presented between the item participants had been tested on (the target) and a distracting item that was not previously displayed in the memory array (a foil).

Chapter 2 focused on gauging the role of the nature of retrieval cues on the quality of recall by keeping the encoding phase constant and manipulating two factors at the retrieval stage: the amount of information carried by retrieval cues and the levels of competition/ similarities between them. Results showed that more information in the retrieval cue improved identification accuracy, localisation performance and identity-location binding. Additionally, the quantitative changes produced by these manipulations were tested using HDDM. Findings showed that increasing the amount of information displayed at retrieval helps recall specifically by increasing the rate of evidence accumulation towards the correct option. Simultaneously, greater similarity between cues presented at retrieval hinders recall due to interference between accumulators in the decision process. Therefore, the main conclusion from Chapter 2 was that the nature of retrieval cues related to the target can significantly affect memory reports, with more information at retrieval improving access to items in WM.

Yet, results arising from manipulations of similarities between retrieval cues seemed to suggest that recall from WM may be reliant also on information held by the foil, not just the target. In Chapter 3, this hypothesis was investigated by examining the impact of the quality of the information carried by the foil on memory performance while keeping the target unchanged. To do so, the foil's presentation was varied in two ways: the number of features from the memory array carried by the foil and the congruency of these features with the memory array (a congruent foil, with the same colour-position arrangement as in the memory array, or an incongruent foil, with its non-spatial features flipped in position).

Presenting a foil with two features from the memory array was found to impair identification of the target but had no disadvantageous outcome on localisation performance. Congruency of the features held by the foil, not only hindered identification accuracy, but also localisation abilities. However, while presenting an incongruent foil was beneficial to the identification accuracy of the target, it was simultaneously detrimental to localisation performance, which was found to be specifically attributable to an increase in the number of errors due to misbinding. These effects — quantified by means of HDDM — revealed that increasing the amount of information from the memory array in the foil impairs recall by decreasing the rate of evidence accumulation towards the target. At the same time, presenting a target together with a congruent foil leads to poorer identification recall due to slower accumulation of evidence for the correct option. Taken together, these findings suggest that recall is reliant on the information held by the foil and its similarities to the non-target, not just the target. Moreover, the arrangement of the features belonging to the foil (congruent or incongruent) was found to be related to the success of the target-location binding.

Chapter 4 was dedicated to a systematic examination of the effects of processing demands induced at different stages of WM, how these relate to each other, and how they vary across the adult lifespan. Manipulations of processing requirements at retrieval were found to impact memory recall, with a decline in identification accuracy when the foil carried two features previously presented in the memory array, as opposed to one. While irrelevant information was found to be filtered out well at encoding, both ignoring irrelevant information and updating the contents of memory during the maintenance

delay had a negative impact on recall. These results were similar across the lifespan, except older adults were especially affected by manipulations of processing demands at encoding, as well as greater set sizes. Finally, no significant relationships between filtering performance at encoding, maintenance and retrieval were found, suggesting these attentional processes are independent from one another. Thus filtering at different phases of WM does not appear to be the role of one single, monolithic system, but rather different cognitive processes that are dissociable at different stages of WM.

Part II of the thesis aimed to investigate how brain diseases disrupt these behavioural processes and whether it is possible in PD to modulate them with drugs that target serotonergic systems. As previously described in the literature, the dissociation of the sources of errors arising from WM impairment have imparted evidence that, while patients with MTL dysfunction are impaired in their ability to associate object-location conjunctions over short delays, memory impairment in patients with PD stems from an increase in errors due to guessing (Zokaei & Husain, 2019b).

In **Chapter 5**, the role of the MTL in remembering relational information was investigated, in particular the nature of memory deficit resulting from focal hippocampal damage in a rare group of patients with limbic encephalitis. Additionally, these behavioural metrics were correlated with hippocampal volume in the largest series of such patients studied on WM tasks. Evidence was found for disease-related impairment in identification accuracy and localisation performance, which resulted from an increase in misbinding. Furthermore, structural imaging showed that patients have smaller hippocampi than controls, with misbinding rates negatively correlated with left hippocampus, specifically the left CA4 and left molecular layer.

Conflicting results on the contributions of dopamine to WM impairment in PD have led to the postulation that the increase in errors due to guessing in these patients could reflect the involvement of non-dopaminergic neurotransmitters, such as disruption in serotonin. Therefore, in Chapter 6, an experimental medicine study was implemented to investigate the contribution of serotonin modulation on WM via administration of 20mg of citalopram for seven days. Additionally, because recent meta-analysis of emotion recognition studies in PD have shown a relationship between cognitive impairment and the variability in emotional valence processing in these patients, this study also aimed at understanding how serotonergic dysfunction in PD affects emotion processing. The effects on cognitive and emotional memory processing in patients with idiopathic PD was measured using two computerized tasks and clinical questionnaires. Results indicated that citalopram did not modulate patients' overall WM performance nor their memory resolution. However, WM was found to be affected by emotional valence in PD, reflected by worse identification accuracy for neutral and angry emotions compared to happy. This appeared to be influenced by the effects of citalopram in mood since higher sensitivity to the antidepressant was associated to better identification accuracy for neutral faces, decreased localisation error for happy faces, decreased imprecision for both angry and happy faces and decreased guessing for angry faces. However, interpretation needs to be cautious, given the small sample size.

7.2. *Contributions to WM Theoretical frameworks*

The present findings make contributions to four aspects of WM: to the discussion on WM capacity, retrieval being conceptualized as a decision-making process, feature integration theory and the role of the hippocampus in relational binding.

Limitations of WM performance have focused largely on encoding by manipulating the contents of memory displays, either through variations of memory load or complexity of feature conjunctions in each item (Ceraso, 1985; S. J. Luck & Vogel, 2013; Wheeler & Treisman, 2002). More recently, this interest has shifted to the effects of maintenance delay (Fallon, Mattiesing, Dolfen, Manohar, & Husain, 2018; Souza & Oberauer, 2014) and even of delaying retrieval cues (Souza S. et al., 2016). A systematic finding has been that recall declines with greater load, which was traditionally explained by reference to either an insufficient fixed number of slots (J. S. Luck & Vogel, 1997) or a flexible resource extended more and more thinly over the amount of items to be held in WM (Paul M. Bays & Husain, 2008a). But retrieval might not simply be a read-out of storage processes. The novel delayed-reproduction test of WM, developed to test young adults' ability to recall information as a function of the nature of retrieval cues, has the potential to broaden current understanding. The results presented in this thesis extend the view on WM capacity as being determined via how information is stored (slots versus resources) by providing evidence for the importance of retrieval mechanisms as well. The findings here show that retrieval cues significantly modulate the quality of memory performance and distracting information presented at the same time can influence – and bias – the decision process of recall.

It has been proposed that recall from working memory can be envisaged as a decision process in which evidence is accumulated about different retrieval options (Pearson et al., 2014). Findings from HDDM expand on this by revealing how changes in retrieval cues influence this **decision-making process of recall**. Displaying retrieval cues with greater amount of information assisted recall by increasing the rate of evidence accumulation towards the correct option. Simultaneously, greater similarity between cues presented at retrieval led to slower accumulation of evidence for the correct option due to interference between accumulators in the decision process. Additionally, the decision process of recall depended not only on the nature of the target, but also the features in a foil (distractor item) if this was also presented at probe, with the arrangement of non-spatial features modulating the rate of accumulation towards the target.

Findings on associative retrieval being contingent on both the information carried by the foil and its similarities to the non-target in the original memory array cannot be well captured by **FIT**, the highly influential psychological model of visual attention (Quinlan, 2003; A. Treisman, 1998; A. M. Treisman, 1980). The data are indeed in line with the conceptualization that spatial features are stored in memory in the form of a master-map of locations, which is responsible for synchronising independent maps, wherein information on non-spatial features (such as colour) is stored (Quinlan, 2003; A. Treisman, 1998; A. M. Treisman, 1980). But the way in which these are re-activated at retrieval is perhaps better conceptualized by a more recent, dynamic computational neural model of WM, **Figure 1.6** (Manohar et al., 2017).

This relies on pattern completion mechanisms, inspired by models of hippocampal function, to produce cued recall. It proposes that rapid plasticity in flexibly-coding neurons allows features, possibility stored in different maps, to be bound together into a conjunction unit. These can be re-activated at recall by a retrieval cue which might contain one of the features belonging to the conjunction unit. Pattern completion would allow other features that are conjoined or bound to that in the retrieval cue to be accessed. However, when multiple templates are activated simultaneously — because the foil shares similarities with the non-target — the retrieval process is more costly because several different templates are activated and concurrently guide the selection of the target.

Finally, findings from the study of limbic encephalitis patients suggest that pattern completion might indeed be reliant on the MTL, with a particular involvement of left hippocampal regions CA4 and molecular layer when the task requires the retrieval of associative information. This further supports the **role of the hippocampus in relational binding**, maintaining and flexibly relating separate features of an object that might be represented in different parts of the neocortex (Cashdollar et al., 2011; Libby et al., 2014).

7.3. *The value of translational research*

Cognitive disorders in neurological diseases are a major challenge. There is no one-to-one mapping: one disease can manifest itself in a multitude of ways, while distinct pathologies can be associated with the same cognitive phenotype. For instance, two

diseases that are seemingly very different, AD and PD, can both ultimately lead to deficits in WM in some patients (Zokaei et al., 2020). Whereas the criteria for AD dementia encompasses the cognitive plus functional impairment in everyday life (McKhann et al., 2011), PD has classically been thought of as a disease primarily affecting motor ability (Kalia & Lang, 2015). But PD is not just a motor disorder; non-motor symptoms such as cognitive deficits also constitute a very important part of the disease (Kalia & Lang, 2015).

Although they are two separate clinical disorders with different underlying pathologies, AD and PD have some important similarities, one of which is that they start many years, perhaps more than a decade, before becoming clinically obvious. In AD, amyloid starts its deposition in certain regions of the neocortex before spreading to cover large amounts of the brain (H. Braak & Braak, 1991). Similarly, tau deposition starts in the MTL and the locus coeruleus before spreading throughout the whole brain (H. Braak & Braak, 1991). On the other hand, the α-synuclein Lewy body pathology of PD displays a very different pattern: it starts in the olphactory tubercle and lower parts of the brainstem before spreading up to involve areas characteristically involved in motor control, including the basal ganglia (Heiko Braak et al., 2004). Later, there is spread into the cortex, which is possibly one reason why cognitive impairments are observed in these patients.

Thus these diseases start long before a clinical diagnosis is made (Bateman et al., 2012; Kalia & Lang, 2015) or even shrinkage of the hippocampus in AD (Bateman et al., 2012). Therefore, a window in which interventions could occur is made potentially available. However, in order to detect its earliest cognitive signs, sensitive measurements able to detect early cognitive decline are of utmost relevance. Indeed, a significant effort

in detecting these early signs have focused on biomarkers — manifestations of these proteins being laid down in different parts of the body. These can be clinical markers (in PD, for example, the loss of smell can be a very early marker of incipient disease; or REM sleep behaviour disorder), imaging markers (PET or MRI scans), pathological markers (in PD, the α-synuclein can be laid down in the skin, even in the gut), biochemical markers (cerebrospinal or plasma amyloid and tau) or genetic markers (the apolipoprotein-E ε4 allele gene —APOE4 — increases the risk of developing AD) (Bateman et al., 2012; Kalia & Lang, 2015). However, most researchers have focused on the prodromal phase of the illness, very close to the diagnosis of dementia, when cognition begins to decline more rapidly. What is really relevant, however, is investigating these changes at the preclinical phase, long before the degeneration process starts and when intervention will presumably be most effective. The difficulty is that the changes that occur at the preclinical phase are quite small compared to those that occur in the prodromal phase.

To meet this challenge, the development of novel cognitive tests such as the ones in **Part I** of this work, which have been shown to be more sensitive than the ones traditionally used in clinical settings (Zokaei & Husain, 2019b), might be important to understand not only the mechanisms underlying WM in healthy populations but also detect normal declines with aging. They also have the potential of being administered online to a large number of people and used as a behavioural marker in different clinical populations to diagnose and differentiate different types of WM deficit. The "What-was-where?" paradigm is a case in point and was purposely used in **Chapter 5** and **6** due to having been previously validated with normative data from several different cohorts (Liang et al., 2016; Pertzov et al., 2012; Pertzov, Miller, et al., 2013; Zokaei et al., 2019,

2020). Its continuous estimation method allows for the deconstruction of memory errors arising from WM failure and has been particularly successful at differentiating healthy populations at greater risk of developing neurodegenerative diseases. For example, asymptomatic carriers of the pathological mutation in presenilin-1 or APP genes, i.e., people with the familial AD gene (FAD) — who are good candidates for examining cognitive impairment that may point to early signs of AD since they do not yet exhibit cognitive deficits on standard neuropsychological tests — have been shown to make greater misbinding errors than controls (Liang et al., 2016).

Misbinding errors turn out to be a very important signature of hippocampal dysfunction and occur when participants remember the item they had been tested on but move it to the location of another object that was present in the memory array — as if it's being corrupted by the other memoranda being held in memory (Pertzov, Miller, et al., 2013; Yonelinas, 2013). The findings of the WM performance of limbic encephalitis patients in this thesis provide further support for this view. In contrast, people who are at high risk of developing PD display significantly more random errors than controls (Rolinski et al., 2016). Despite being asymptomatic, these populations are nevertheless displaying early signs of memory problems that are being detected by a sensitive test — and which could potentially benefit from disease-modifying treatments that, albeit not particularly effective in late stages, might produce a significant difference if introduced earlier.

Accordingly, understanding manifestations of brain disease at the systemic level by linking cognitive/behavioural components to brain structure, such as the study in **Chapter 5,** and testing whether it is possible to modulate component processes with drugs to

improve function in patients, as in Chapter 6, provide insights into the mechanisms underlying diseases in which WM deficit occur. Simultaneously, the development of novel experimental paradigms that better deconstruct the cognitive processes of WM, such as the ones developed in part I of this work, can provide a systems-level conceptual framework to understand cognitive and behavioural syndromes in neurological diseases such that, in the future, treatments focusing on modulating cognitive processes — and the brain networks supporting them— can be developed.

7.4. *Future directions*

Even though the role of retrieval has been well characterized in LTM, it has yet to be fully explored in STM. Therefore, future investigation on whether the results from manipulations of the nature of retrieval cues are contingent upon maintenance duration can help further elucidate similarities between retrieval from STM and LTM.

This can be addressed by comparing how information is retrieved after short vs. long retention periods, using the paradigm developed in Chapter 2 with different maintenance durations and with the implementation of Hierarchical Bayesian Drift Diffusion modelling to evaluate the mechanistic differences in the decision-making process from STM and LTM. Because one of the fundamental conclusions reached in LTM is that the success of episodic retrieval is cue dependent, and the data from Chapter 2 and 3 reveals that this is also the case for STM, recall quality for both short and long delays can be postulated to be a function of the type of information held by both the target and the probe, not simply a function of delay.

Findings presented here may also be taken forward by using the novel delayed reproduction task from **Chapter 2** in patients with hippocampal recordings. Recording of rapid bursts of synchronized neuronal activity produced by the hippocampus — also known as sharp-wave ripples (SWRs), which are large amplitude negative polarity deflections that usually appear in the local field potential (LFP) of the hippocampal CA1 pyramidal layer — have been identified to anticipate the onset of episodic recall (Buzsáki, 2015; Norman et al., 2019). Recent evidence by Norman and colleagues (2019), from simultaneous intracranial recording of electrophysiological events in hippocampus and cortex revealed that the hippocampus SWRs are associated with forming and activating spontaneous recall. Results disclose that these hippocampal ripples might be fundamental in managing the mnemonic reactivation and interchange between the hippocampus and cerebral cortex, i.e., between memory centres and high-level representations that underlie the retrieval of memoranda.

Given the findings from **Chapter 5** — suggesting that pattern completion is reliant on the MTL, with a particular involvement of the hippocampus when the task requires the retrieval of associative information from STM — these results suggest that the function of SWRs that contributes to recall from LTM may also be engaged in STM. Therefore, the paradigm developed in **Chapter 2** could be used in patients with hippocampal recording to investigate whether a transient increase in SWR rate anticipates the onset of STM recall, and if this is modulated by the nature of the information carried by the retrieval cue.

A third avenue of research would incorporate retrieval in the current debate on whether slot and resource models are incompatible or can be reconciled into a unified

framework. The problem of how to accurately describe the complex processes underlying WM is one that has long divided experts: in particular, the question of whether visual WM is best understood as a distributed resource or discrete slots. There are obvious differences in how both types of model inform our expectations concerning human performance. On the one hand, the stochastic and continuous mechanisms of resource models predict a smooth deterioration of memory with an increasing set size (Bays & Husain, 2008; Wilken & Ma, 2004); on the other hand, one would expect an abrupt change in WM fidelity due to the deterministic limits imposed by classic slot models.

As such, because these paradigms yield distinct and easily identifiable predictions, the discussions pertaining to the limited capacity of WM, and how best to define it, have traditionally been centred around memory load at encoding — and how their relative departure from experimental data gives support for one model over the other. Recently, several hybrid models (Alvarez & Cavanagh, 2004; Anderson et al., 2011; Weiwei Zhang & Luck, 2008) have attempted to blur these distinctions and impart possible unified frameworks that better explain behavioural data, and which provide interesting avenues for future research.

In spite of the difficulty of framing our results in light of the classical arguments which largely focus on memory load at encoding, the work in the present thesis contributes to the debate by highlighting that retrieval, not solely encoding, plays an important role in the quality of recall. Consequently, future directions should focus on exploring the noisiness of responses and how their pattern might provide further insight

into how different conceptions of WM might be successfully integrated into a unified framework.

To examine this, a similar design to the one used in Chapters 2 & 3 could be implemented where, instead of having a binary design, i.e., presenting either low or high conditions, it could instead follow a gradation when manipulating information and competition. It would be interesting to observe if, as complexity in the retrieval cues increases, metrics of WM performance such as imprecision or misbinding would follow a smooth and continuous deterioration consistent with the concept of WM as a limited resource. Or, it just might be the case that some of these metrics would follow a gradual degradation while others would display abrupt changes characteristic of deterministic limits.

7.5. *Conclusions*

In summary, the work presented in this thesis indicates that: **(i)** the nature of retrieval can significantly affect memory reports, with more information at retrieval improving access to items in WM; **(ii)** associative retrieval is reliant on the foil's information — not just the target — and the arrangement of its non-spatial features modulates retrieval of relational information; **(iii)** filtering is a dissociable cognitive process that engages and articulates with different phases of WM, rather than being a single entity; **(iv)** the MTL plays a key role in relational WM binding, particularly CA4 and molecular layer; and **(v)** serotonin might potentially be responsible for processing negative emotional information in PD.

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Appendix

Appendix Chapter 2. The nature of retrieval cues affects recall performance

Experiment 1

Appendix Table 2.1 | Experiment 1 (n =21) Repeated Measures ANOVA: Identification accuracy
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	0.008	1	0.008	3.027	0.097	0.131
Residuals	0.054	20	0.003			
Competition	0.208	1	0.208	23.891	< .001	0.544
Residuals	0.174	20	0.009			
Delay	0.004	1	0.004	1.756	0.200	0.081
Residuals	0.047	20	0.002			
Information ✻ Competition	0.032	1	0.032	9.868	0.005	0.330
Residuals	0.066	20	0.003			
Information ✻ Delay	0.001	1	0.001	0.362	0.554	0.018
Residuals	0.057	20	0.003			
Competition ✻ Delay	0.004	1	0.004	0.819	0.376	0.039
Residuals	0.101	20	0.005			
Information ✻ Competition ✻ Delay	3.720e-4	1	3.720e-4	0.099	0.756	0.005
Residuals	0.075	20	0.004			

Note. Type III Sum of Squares

Appendix Table 2.2 | Experiment 1 (n =21) Post Hoc Tests: Identification accuracy
Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
Low, Low	High Low	-0.042	0.012	-3.498	0.005
	Low, High	0.043	0.017	2.523	0.033
	High, High	0.057	0.016	3.433	0.005
High, Low	Low, High	0.084	0.016	5.119	< .001
	High, High	0.098	0.017	5.809	< .001
Low, High	High, High	0.014	0.012	1.166	0.251

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Delay

Appendix Table 2.3 | Experiment 1 (n =21) Bayesian Repeated Measures ANOVA: Identification accuracy
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.053	2.088e-9	3.759e-8	1.000	
Information + Competition + Information ✻ Competition	0.053	0.472	16.092	2.260e+8	3.278
Competition	0.053	0.162	3.491	7.778e+7	0.983

Appendix Table 2.3 | Experiment 1 (n =21) Bayesian Repeated Measures ANOVA: Identification accuracy

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Information + Competition + Delay + Information ✻ Competition	0.053	0.120	2.461	5.759e+7	3.325
Information + Competition	0.053	0.064	1.231	3.066e+7	2.347
Information + Competition + Delay + Information ✻ Competition + Competition ✻ Delay	0.053	0.042	0.784	1.999e+7	3.270
Competition + Delay	0.053	0.041	0.766	1.954e+7	1.302
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay	0.053	0.029	0.540	1.394e+7	2.830
Information + Competition + Delay	0.053	0.024	0.447	1.159e+7	32.243
Competition + Delay + Competition ✻ Delay	0.053	0.015	0.273	7.163e+6	2.895
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay + Competition ✻ Delay	0.053	0.012	0.216	5.688e+6	8.411
Information + Competition + Delay + Information ✻ Delay	0.053	0.008	0.145	3.829e+6	46.612
Information + Competition + Delay + Competition ✻ Delay	0.053	0.006	0.102	2.686e+6	3.368
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay + Competition ✻ Delay + Information ✻ Competition ✻ Delay	0.053	0.003	0.063	1.658e+6	8.819
Information + Competition + Delay + Information ✻ Delay + Competition ✻ Delay	0.053	0.001	0.026	696050.859	4.749
Information	0.053	6.710e-10	1.208e-8	0.321	2.403
Delay	0.053	4.960e-10	8.927e-9	0.237	2.803
Information + Delay	0.053	1.570e-10	2.826e-9	0.075	2.445
Informaion + Delay + Information ✻ Delay	0.053	3.682e-11	6.628e-10	0.018	2.322

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.737	0.782	1.280
Competition	0.737	1.000	1.035e+8
Delay	0.737	0.302	0.154
Information ✻ Competition	0.316	0.679	4.573
Information ✻ Delay	0.316	0.054	0.123
Competition ✻ Delay	0.316	0.079	0.186
Information ✻ Competition ✻ Delay	0.053	0.003	0.063

Appendix Table 2.4 | Experiment 1 (n =21) Repeated Measures ANOVA: Localisation error (pixels)
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	20880.806	1	20880.806	50.317	< .001	0.716
Residuals	8299.752	20	414.988			
Competition	5707.531	1	5707.531	24.323	< .001	0.549
Residuals	4693.071	20	234.654			
Delay	7553.870	1	7553.870	47.940	< .001	0.706
Residuals	3151.406	20	157.570			
Information ✻ Competition	7212.832	1	7212.832	12.910	0.002	0.392
Residuals	11174.384	20	558.719			
Information ✻ Delay	1656.171	1	1656.171	2.959	0.101	0.129
Residuals	11194.415	20	559.721			
Competition ✻ Delay	1707.289	1	1707.289	4.972	0.037	0.199
Residuals	6867.600	20	343.380			
Information ✻ Competition ✻ Delay	800.412	1	800.412	1.713	0.205	0.079
Residuals	9344.012	20	467.201			

Note. Type III Sum of Squares

Appendix Table 2.5 | Experiment 1 (n =21) Post Hoc Tests: Localisation error (pixels)

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
Low, Low	High, Low	35.402	4.815	7.353	< .001
	Low, High	24.762	4.346	5.697	< .001
	High, High	33.954	3.933	8.633	< .001
High, Low	Low, High	-10.640	3.933	-2.705	0.031
	High, High	-1.447	4.346	-0.333	0.741
Low, High	High, High	9.192	4.815	1.909	0.127

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Delay

Post Hoc Comparisons - Competition ✻ Delay

		Mean Difference	SE	t	p_{holm}
Low, X1second	High, X1second	5.282	3.710	1.424	0.325
	Low, X4seconds	-19.787	3.454	-5.729	< .001
	High, X4seconds	-1.754	3.056	-0.574	0.569
High, X1second	Low, X4seconds	-25.068	3.056	-8.203	< .001
	High, X4seconds	-7.035	3.454	-2.037	0.148
Low, X4seconds	High, X4seconds	18.033	3.710	4.861	< .001

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Information

Appendix Table 2.6 | Experiment 1 (n =21) Bayesian Repeated Measures ANOVA: Localisation error (pixels)
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.053	3.215e-14	5.788e-13	1.000	

Appendix Table 2.6 | Experiment 1 (n =21) Bayesian Repeated Measures ANOVA: Localisation error (pixels)
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay + Competition ✻ Delay	0.053	0.262	6.376	8.135e+12	3.614
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay	0.053	0.207	4.691	6.430e+12	8.667
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay + Competition ✻ Delay + Information ✻ Competition ✻ Delay	0.053	0.193	4.292	5.988e+12	6.406
Information + Competition + Delay + Information ✻ Competition + Competition ✻ Delay	0.053	0.192	4.271	5.965e+12	3.139
Information + Competition + Delay + Information ✻ Competition	0.053	0.145	3.061	4.520e+12	4.610
Information + Competition + Delay + Information ✻ Delay + Competition ✻ Delay	0.053	4.772e-4	0.009	1.484e+10	4.005
Information + Competition + Delay + Competition ✻ Delay	0.053	4.419e-4	0.008	1.374e+10	2.583
Information + Competition + Delay + Information ✻ Delay	0.053	4.362e-4	0.008	1.357e+10	2.565
Information + Competition + Delay	0.053	3.697e-4	0.007	1.150e+10	2.270
Information + Competition + Information ✻ Competition	0.053	2.743e-4	0.005	8.530e+9	4.258
Information + Delay + Information ✻ Delay	0.053	1.955e-5	3.519e-4	6.080e+8	56.761
Information + Delay	0.053	8.213e-6	1.478e-4	2.554e+8	1.855
Information + Competition	0.053	1.414e-6	2.544e-5	4.397e+7	1.266
Information	0.053	5.523e-8	9.942e-7	1.718e+6	0.694
Competition + Delay	0.053	1.373e-11	2.471e-10	426.979	2.007
Competition + Delay + Competition ✻ Delay	0.053	1.074e-11	1.934e-10	334.150	1.658
Delay	0.053	1.606e-12	2.891e-11	49.956	29.809
Competition	0.053	2.879e-13	5.183e-12	8.955	1.240

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.737	1.000	1.353e+10
Competition	0.737	1.000	12839.116
Delay	0.737	1.000	1294.900
Information ✻ Competition	0.316	0.998	1232.970

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information ✻ Delay	0.316	0.662	4.240
Competition ✻ Delay	0.316	0.647	3.968
Information ✻ Competition ✻ Delay	0.053	0.193	4.292

Appendix Table 2.7 | Experiment 1 (n =21) Repeated Measures ANOVA: Reaction time (seconds)
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	1.349	1	1.349	39.766	< .001	0.665
Residuals	0.679	20	0.034			
Competition	0.018	1	0.018	0.333	0.570	0.016
Residuals	1.090	20	0.055			
Delay	0.419	1	0.419	12.811	0.002	0.390
Residuals	0.654	20	0.033			
Information ✻ Competition	0.140	1	0.140	6.475	0.019	0.245
Residuals	0.434	20	0.022			
Information ✻ Delay	0.018	1	0.018	0.687	0.417	0.033
Residuals	0.521	20	0.026			
Competition ✻ Delay	0.002	1	0.002	0.103	0.752	0.005
Residuals	0.333	20	0.017			
Information ✻ Competition ✻ Delay	0.010	1	0.010	0.541	0.470	0.026
Residuals	0.362	20	0.018			

Note. Type III Sum of Squares

Appendix Table 2.8 | Experiment 1 (n =21) Post Hoc Tests: Reaction time (seconds)
Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
Low, Low	High, Low	0.237	0.036	6.514	< .001
	Low, High	0.037	0.043	0.869	0.391
	High, High	0.158	0.046	3.453	0.006
High, Low	Low, High	-0.200	0.046	-4.359	< .001
	High, High	-0.079	0.043	-1.846	0.147
Low, High	High, High	0.121	0.036	3.337	0.006

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Delay

Appendix Table 2.9 | Experiment 1 (n =21) Bayesian Repeated Measures ANOVA: Reaction time (seconds)
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.053	6.845e-10	1.232e-8	1.000	
Information + Delay	0.053	0.429	13.521	6.267e+8	2.744
Information + Competition + Delay + Information ✻ Competition	0.053	0.177	3.867	2.583e+8	4.431
Information + Delay + Information ✻ Delay	0.053	0.123	2.532	1.802e+8	2.463
Information + Competition + Delay	0.053	0.091	1.793	1.323e+8	1.902

Appendix Table 2.9 | Experiment 1 (n =21) Bayesian Repeated Measures ANOVA: Reaction time (seconds)
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay	0.053	0.053	1.000	7.692e+7	4.641
Information + Competition + Delay + Information ✻ Competition + Competition ✻ Delay	0.053	0.041	0.774	6.019e+7	4.540
Information + Competition + Delay + Competition ✻ Delay	0.053	0.030	0.548	4.319e+7	25.086
Information + Competition + Delay + Information ✻ Delay	0.053	0.027	0.499	3.944e+7	2.671
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay + Competition ✻ Delay	0.053	0.012	0.211	1.690e+7	4.237
Information + Competition + Delay + Information ✻ Delay + Competition ✻ Delay	0.053	0.007	0.121	9.786e+6	6.029
Information	0.053	0.005	0.095	7.669e+6	11.681
Information + Competition + Delay + Information ✻ Competition + Information ✻ Delay + Competition ✻ Delay + Information ✻ Competition ✻ Delay	0.053	0.004	0.071	5.729e+6	4.471
Information + Competition + Information ✻ Competition	0.053	0.001	0.027	2.165e+6	1.787
Information + Competition	0.053	9.704e-4	0.017	1.418e+6	3.051
Delay	0.053	1.499e-8	2.699e-7	21.902	0.928
Competition + Delay	0.053	3.143e-9	5.657e-8	4.591	2.341
Competition + Delay + Competition ✻ Delay	0.053	7.205e-10	1.297e-8	1.053	3.031
Competition	0.053	1.378e-10	2.481e-9	0.201	1.492

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.737	1.000	1.815e+7
Competition	0.737	0.442	0.283
Delay	0.737	0.992	46.015
Information ✻ Competition	0.316	0.288	0.875
Information ✻ Delay	0.316	0.225	0.630
Competition ✻ Delay	0.316	0.093	0.222
Information ✻ Competition ✻ Delay	0.053	0.004	0.071

Experiment 1 (1second delay trials)

Appendix Table 2.10 | Experiment 1, delay=1second (n =21) Repeated Measures ANOVA: Identification accuracy
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Information	0.007	1	0.007	3.445	0.078	0.015	0.147

Appendix Table 2.10 | Experiment 1, delay=1second (n =21) Repeated Measures ANOVA: Identification accuracy

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Residuals	0.043	20	0.002				
Competition	0.136	1	0.136	13.567	0.001	0.266	0.404
Residuals	0.200	20	0.010				
Information ✻ Competition	0.013	1	0.013	2.332	0.142	0.025	0.104
Residuals	0.111	20	0.006				

Note. Type III Sum of Squares

Appendix Table 2.11 | Experiment 1, delay=1second (n =21) Bayesian Repeated Measures ANOVA: Identification accuracy

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	4.195e-4	0.002	1.000	
Competition	0.200	0.575	5.403	1369.904	5.237
Information + Competition + Information ✻ Competition	0.200	0.214	1.091	510.744	25.249
Information + Competition	0.200	0.211	1.067	502.041	2.404
Information	0.200	1.508e-4	6.032e-4	0.359	7.923

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.425	0.493
Competition	0.600	0.999	1168.442
Information ✻ Competition	0.200	0.214	1.091

Appendix Table 2.12 | Experiment 1, delay = 1second (n=21) Repeated Measures ANOVA: Localisation error

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Information	5387.826	1	5387.826	14.371	0.001	0.194	0.418
Residuals	7498.009	20	374.900				
Competition	585.807	1	585.807	1.731	0.203	0.021	0.080
Residuals	6769.175	20	338.459				
Information ✻ Competition	1603.866	1	1603.866	5.355	0.031	0.058	0.211
Residuals	5990.699	20	299.535				

Note. Type III Sum of Squares

Appendix Table 2.13 | Experiment 1, delay = 1second (n=21) Post Hoc Tests: Localisation error

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
Low., Low	High, Low	24.757	5.667	4.369	< .001
	Low., High	14.021	5.512	2.544	0.060
	High, High	21.299	5.828	3.654	0.004
High, Low	Low., High	-10.736	5.828	-1.842	0.219
	High, High	-3.458	5.512	-0.627	0.534
Low., High	High, High	7.278	5.667	1.284	0.413

Post Hoc Comparisons - Information ✻ Competition

	Mean Difference	SE	t	p_{holm}

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.14 | Experiment 1, delay = 1 second (n=21) Bayesian Repeated Measures ANOVA: Localisation error

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.005	0.020	1.000	
Information	0.200	0.431	3.026	88.501	1.432
Information + Competition + Information ✻ Competition	0.200	0.370	2.349	76.025	1.920
Information + Competition	0.200	0.192	0.953	39.548	2.201
Competition	0.200	0.002	0.008	0.408	2.232

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.993	96.625
Competition	0.600	0.564	0.864
Information ✻ Competition	0.200	0.370	2.349

Appendix Table 2.15 | Experiment 1, delay=1second (n =21) Repeated Measures ANOVA: Reaction Time (seconds)

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	0.839	1	0.839	46.173	< .001	0.698
Residuals	0.363	20	0.018			
Competition	0.004	1	0.004	0.220	0.644	0.011
Residuals	0.397	20	0.020			
Information ✻ Competition	0.038	1	0.038	3.857	0.064	0.162
Residuals	0.197	20	0.010			

Note. Type III Sum of Squares

Appendix Table 2.16 | Experiment 1, delay=1second (n =21) Bayesian Repeated Measures ANOVA: Reaction Time (seconds)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	9.605e-8	3.842e-7	1.000	
Information	0.200	0.696	9.163	7.248e+6	1.956
Information + Competition	0.200	0.169	0.814	1.760e+6	1.756
Information + Competition + Information ✻ Competition	0.200	0.135	0.624	1.404e+6	2.287
Competition	0.200	2.452e-8	9.806e-8	0.255	5.823

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	1.000	5.530e+6
Competition	0.600	0.304	0.291
Information ✻ Competition	0.200	0.135	0.624

Experiment 1 (4 seconds delay trials)

Appendix Table 2.17 | Experiment 1, delay=4seconds (n =21) Repeated Measures ANOVA: Identification Accuracy

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	0.002	1	0.002	0.497	0.489	0.024
Residuals	0.067	20	0.003			
Competition	0.077	1	0.077	20.389	< .001	0.505
Residuals	0.075	20	0.004			
Information ✻ Competition	0.020	1	0.020	13.219	0.002	0.398
Residuals	0.030	20	0.002			

Note. Type III Sum of Squares

Appendix Table 2.18 | Experiment 1, delay=4seconds (n =21) Post Hoc Tests: Identification Accuracy

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
Low, Low	High, Low	-0.040	0.015	-2.606	0.040
	Low, High	0.030	0.016	1.878	0.138
	High, High	0.052	0.018	2.798	0.032
High, Low	Low, High	0.069	0.018	3.766	0.003
	High, High	0.091	0.016	5.759	< .001
Low, High	High, High	0.022	0.015	1.433	0.161

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.19 | Experiment 1, delay=4seconds (n =21) Bayesian Repeated Measures ANOVA: Identification Accuracy

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	1.431e-4	5.724e-4	1.000	
Information + Competition + Information ✻ Competition	0.200	0.459	3.393	3207.760	3.928
Competition	0.200	0.440	3.147	3077.371	19.385
Information + Competition	0.200	0.101	0.447	702.933	3.356
Information	0.200	3.948e-5	1.579e-4	0.276	3.552

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.560	0.847
Competition	0.600	1.000	3651.292
Information ✻ Competition	0.200	0.459	3.393

Appendix Table 2.20 | Experiment 1, delay=4seconds (n =21) Repeated Measures ANOVA: Localisation error (pixels)

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	17149.151	1	17149.151	28.591	< .001	0.588
Residuals	11996.159	20	599.808			
Competition	6829.013	1	6829.013	28.505	< .001	0.588
Residuals	4791.496	20	239.575			
Information ✻ Competition	6409.378	1	6409.378	8.824	0.008	0.306
Residuals	14527.697	20	726.385			

Note. Type III Sum of Squares

Appendix Table 2.21 | Experiment 1, delay=4seconds (n =21) Post Hoc Tests: Localisation error (pixels)

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
Low, Low	High, Low	46.047	7.947	5.794	< .001
	Low, High	35.503	6.782	5.235	< .001
	High, High	46.610	6.322	7.372	< .001
High, Low	Low, High	-10.544	6.322	-1.668	0.314
	High, High	0.563	6.782	0.083	0.934
Low, High	High, High	11.106	7.947	1.398	0.340

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.22 | Experiment 1, delay=4seconds (n =21) Bayesian Repeated Measures ANOVA: Localisation error (pixels)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	4.985e-7	1.994e-6	1.000	
Information + Competition + Information ✻ Competition	0.200	0.969	126.592	1.945e+6	1.949
Information + Competition	0.200	0.029	0.121	58683.435	2.367
Information	0.200	0.001	0.006	2756.524	1.083
Competition	0.200	3.236e-6	1.295e-5	6.493	3.811

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	1.000	178499.036
Competition	0.600	0.999	484.344
Information ✻ Competition	0.200	0.969	126.592

Appendix Table 2.23 | Experiment 1, delay=4seconds (n =21) Repeated Measures ANOVA: Reaction time (seconds)

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	0.528	1	0.528	12.626	0.002	0.387

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	0.837	20	0.042			
Competition	0.016	1	0.016	0.302	0.588	0.015
Residuals	1.026	20	0.051			
Information ✻ Competition	0.112	1	0.112	3.748	0.067	0.158
Residuals	0.599	20	0.030			

Note. Type III Sum of Squares

Appendix Table 2.24 | Experiment 1, delay=4seconds (n =21) Bayesian Repeated Measures ANOVA: Reaction time (seconds)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.017	0.069	1.000	
Information	0.200	0.647	7.332	37.993	1.035
Information + Competition	0.200	0.170	0.817	9.955	1.846
Information + Competition + Information ✻ Competition	0.200	0.162	0.774	9.517	7.945
Competition	0.200	0.004	0.017	0.255	0.889

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.979	30.519
Competition	0.600	0.336	0.337
Information ✻ Competition	0.200	0.162	0.774

Experiment 2

Appendix Table 2.25 | Experiment 2, (n =20) Repeated Measures ANOVA: Identification Accuracy

Within Subjects Effects

Cases	df	Mean Square	F	p	η^2	η^2_p
Information	1.000	0.021	17.588	< .001	0.083	0.481
Residuals	19.000	0.001				
Competition	1.000	0.129	38.404	< .001	0.501	0.669
Residuals	19.000	0.003				
Information ✻ Competition	1.000	0.004	4.895	0.039	0.016	0.205
Residuals	19.000	8.297e-4				

Note. Type III Sum of Squares

Appendix Table 2.26 | Experiment 2, (n =20) Post Hoc Tests: Identification Accuracy

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{bonf}
Low, Low	High, Low	-0.047	0.010	-4.643	< .001
	Low, High	0.066	0.014	4.563	< .001
	High, High	0.047	0.015	3.141	0.022
High, Low	Low, High	0.113	0.015	7.473	< .001
	High, High	0.095	0.014	6.534	< .001

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{bonf}
Low, High	High, High	-0.018	0.010	-1.828	0.455

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.27| Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Identification Accuracy
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	7.009e-10	2.804e-9	1.000	
Information	0.200	1.522e-9	6.087e-9	2.171	0.909
Competition	0.200	0.024	0.097	3.378e+7	0.941
Information + Competition	0.200	0.586	5.664	8.362e+8	4.690
Information + Competition + Information ✻ Competition	0.200	0.390	2.560	5.567e+8	2.323

Note. All models include subject.

Analysis of Effects

Effects	P(incl)	P(incl|data)	$BF_{Inclusion}$
Information	0.600	0.976	27.494
Competition	0.600	1.000	2.999e+8
Information ✻ Competition	0.200	0.390	2.560

Appendix Table 2.28 | Experiment 2, (n =20) Repeated Measures ANOVA: Localisation error (pixels)
Within Subjects Effects

	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	12611.523	1	12611.523	166.189	< .001	0.897
Residual	1441.843	19	75.886			
Competition	6197.083	1	6197.083	79.291	< .001	0.807
Residual	1484.964	19	78.156			
Information ✻ Competition	4840.607	1	4840.607	92.312	< .001	0.829
Residual	996.308	19	52.437			

Note. Type III Sum of Squares

Appendix Table 2.29 | Experiment 2, (n =20) Post Hoc Tests: Localisation error (pixels)
Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{bonf}
Low, Low	High, Low	40.669	2.533	16.055	< .001
	Low, High	33.160	2.555	12.977	< .001
	High, High	42.714	2.775	15.391	< .001
High, Low	Low, High	-7.509	2.775	-2.706	0.061
	High, High	2.045	2.555	0.800	1.000
Low, High	High, High	9.554	2.533	3.772	0.003

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.30 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Localisation error (pixels)
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	5.796e-21	2.319e-20	1.000	

Appendix Table 2.30 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Localisation error (pixels)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Information	0.200	2.807e-14	1.123e-13	4.842e+6	0.707
Competition	0.200	1.422e-18	5.689e-18	245.346	3.820
Information + Competition	0.200	7.682e-9	3.073e-8	1.325e+12	1.206
Information + Competition + Information ✻ Competition	0.200	1.000	5.207e+8	1.725e+20	2.298

Note. All models include subject.

Analysis of Effects

Effects	P(incl)	P(incl|data)	$BF_{Inclusion}$
Information	0.600	1.000	6.005e+15
Competition	0.600	1.000	2.373e+13
Information ✻ Competition	0.200	1.000	5.207e+8

Appendix Table 2.31 | Experiment 2, (n =20) Repeated Measures ANOVA: Reaction Time (seconds)

Within Subjects Effects

Cases	df	Mean Square	F	p	η^2_p
Information	1.000	0.496	50.630	< .001	0.727
Residuals	19.000	0.010			
Competition	1.000	0.014	1.652	0.214	0.080
Residuals	19.000	0.009			
Information ✻ Competition	1.000	0.060	9.217	0.007	0.327
Residuals	19.000	0.006			

Note. Sphericity corrections not available for factors with 2 levels.

Note. Type III Sum of Squares

Appendix Table 2.32 | Experiment 2, (n =20) Post Hoc Tests: Reaction Time (seconds)

Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{bonf}
Low, Low	High, Low	0.212	0.029	7.435	< .001
	Low, High	0.028	0.027	1.027	1.000
	High, High	0.131	0.030	4.326	< .001
High, Low	Low, High	-0.184	0.030	-6.080	< .001
	High, High	-0.081	0.027	-2.965	0.032
Low, High	High, High	0.103	0.029	3.602	0.006

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.33 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Reaction Time (seconds)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	3.367e-8	1.347e-7	1.000	
Information	0.200	0.287	1.613	8.535e+6	1.215
Competition	0.200	1.093e-8	4.374e-8	0.325	1.553

Appendix Table 2.33 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Reaction Time (seconds)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Information + Competition	0.200	0.129	0.593	3.836e+6	2.050
Information + Competition + Information ✻ Competition	0.200	0.583	5.603	1.733e+7	1.643

Note. All models include subject.

Analysis of Effects

Effects	P(incl)	P(incl|data)	$BF_{Inclusion}$
Information	0.600	1.000	1.495e+7
Competition	0.600	0.713	1.653
Information ✻ Competition	0.200	0.583	5.603

Appendix Table 2.34 | Experiment 2, (n =20) Repeated Measures ANOVA: Imprecision (pixels)

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	21.417	1	21.417	1.456	0.242	0.071
Residuals	279.535	19	14.712			
Competition	110.425	1	110.425	14.924	0.001	0.440
Residuals	140.581	19	7.399			
Information ✻ Competition	13.986	1	13.986	1.897	0.184	0.091
Residuals	140.106	19	7.374			

Note. Type III Sum of Squares

Appendix Table 2.35 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Imprecision (pixels)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.027	0.110	1.000	
Competition	0.200	0.504	4.063	18.888	0.812
Information + Competition	0.200	0.292	1.653	10.959	1.956
Information + Competition + Information ✻ Competition	0.200	0.164	0.782	6.131	2.836
Information	0.200	0.013	0.054	0.503	2.129

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.469	0.590
Competition	0.600	0.960	15.961
Information ✻ Competition	0.200	0.164	0.782

Appendix Table 2.36 | Experiment 2, (n =20) Repeated Measures ANOVA: Misbinding

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	0.061	1	0.061	116.756	< .001	0.860
Residuals	0.010	19	5.222e-4			
Competition	0.035	1	0.035	124.637	< .001	0.868

Appendix Table 2.36 | Experiment 2, (n =20) Repeated Measures ANOVA: Misbinding
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	0.005	19	2.782e-4			
Information ✻ Competition	0.028	1	0.028	177.750	< .001	0.903
Residuals	0.003	19	1.558e-4			

Note. Type III Sum of Squares

Appendix Table 2.37 | Experiment 2, (n =20) Post Hoc Tests: Misbinding
Post Hoc Comparisons - Information ✻ Competition

		Mean Difference	SE	t	p_{holm}
low, LOW	high, LOW	0.092	0.006	15.874	< .001
	low, HIGH	0.079	0.005	16.927	< .001
	high, HIGH	0.097	0.006	15.310	< .001
high, LOW	low, HIGH	-0.014	0.006	-2.146	0.078
	high, HIGH	0.004	0.005	0.949	0.349
low, HIGH	high, HIGH	0.018	0.006	3.091	0.013

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.38 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Misbinding
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	1.233e-22	4.930e-22	1.000	
Information + Competition + Information ✻ Competition	0.200	1.000	1.438e+10	8.113e+21	3.760
Information + Competition	0.200	2.781e-10	1.112e -9	2.256e+12	2.293
Information	0.200	3.305e-16	1.322e-15	2.681e +6	0.933
Competition	0.200	9.124e-20	3.650e-19	740.303	1.472

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	1.000	1.501e+15
Competition	0.600	1.000	8.578e+14
Information ✻ Competition	0.200	1.000	1.438e+10

Appendix Table 2.39 | Experiment 2, (n =20) Repeated Measures ANOVA: Guessing
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	8.858e-5	1	8.858e-5	0.837	0.372	0.042
Residuals	0.002	19	1.059e-4			
Competition	1.331e-4	1	1.331e-4	0.925	0.348	0.046
Residuals	0.003	19	1.438e-4			
Information ✻ Competition	1.596e-4	1	1.596e-4	2.421	0.136	0.113
Residuals	0.001	19	6.591e-5			

Note. Type III Sum of Squares

Appendix Table 2.40 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA: Guessing
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.506	4.094	1.000	
Competition	0.200	0.208	1.048	0.411	1.177
Information	0.200	0.174	0.843	0.344	3.230
Information + Competition	0.200	0.072	0.311	0.143	4.845
Information + Competition + Information ✻ Competition	0.200	0.040	0.168	0.080	2.117

Note. All models include subject

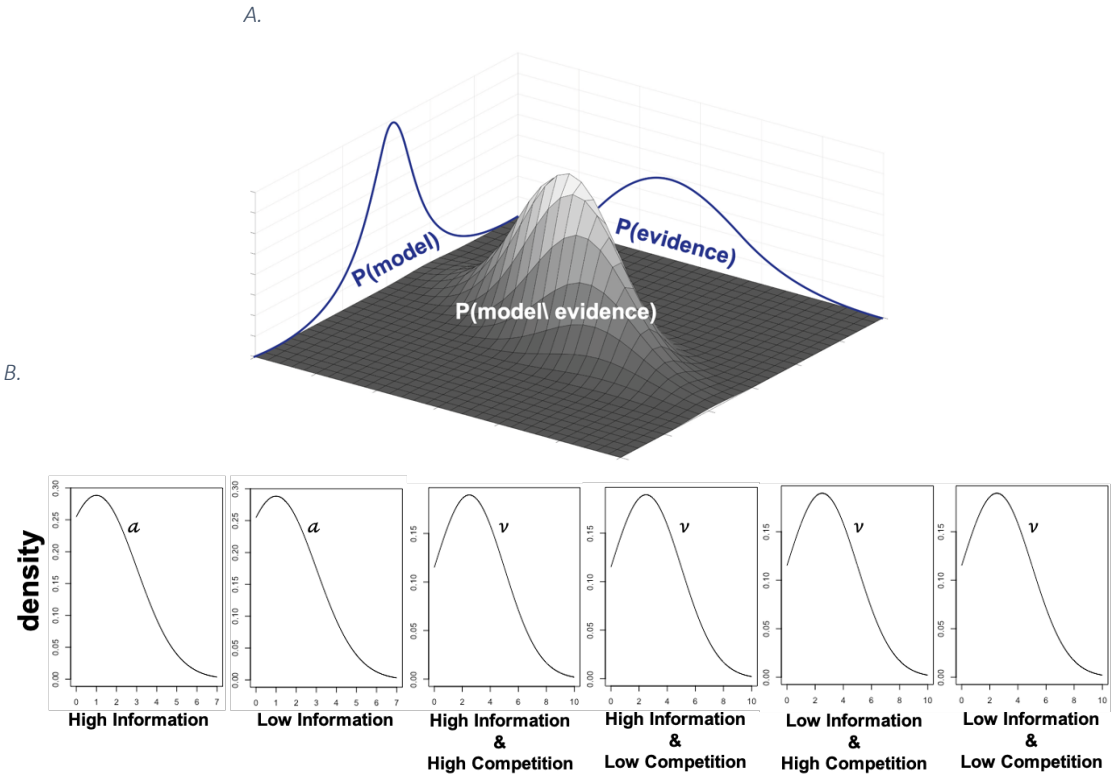
Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.287	0.268
Competition	0.600	0.320	0.314
Information ✻ Competition	0.200	0.040	0.168

Experiment 2 (HDDM)

All models were fitted using a Bayesian approach to parameter estimation, where the prior distribution (**Appendix Figure 2.1**) — describing our initial parameter assumptions, uninformed by any data — was updated by the evidence using Bayes' theorem of conditional probability, as per the following equation:

$$P(A\backslash B) = \frac{P(B\backslash A).P(A)}{P(B)} \sim P(model\backslash evidence) = \frac{P(evidence\backslash model).P(model)}{P(evidence)}$$



Appendix Figure 2.1| The Bayesian approach to parameter estimation.

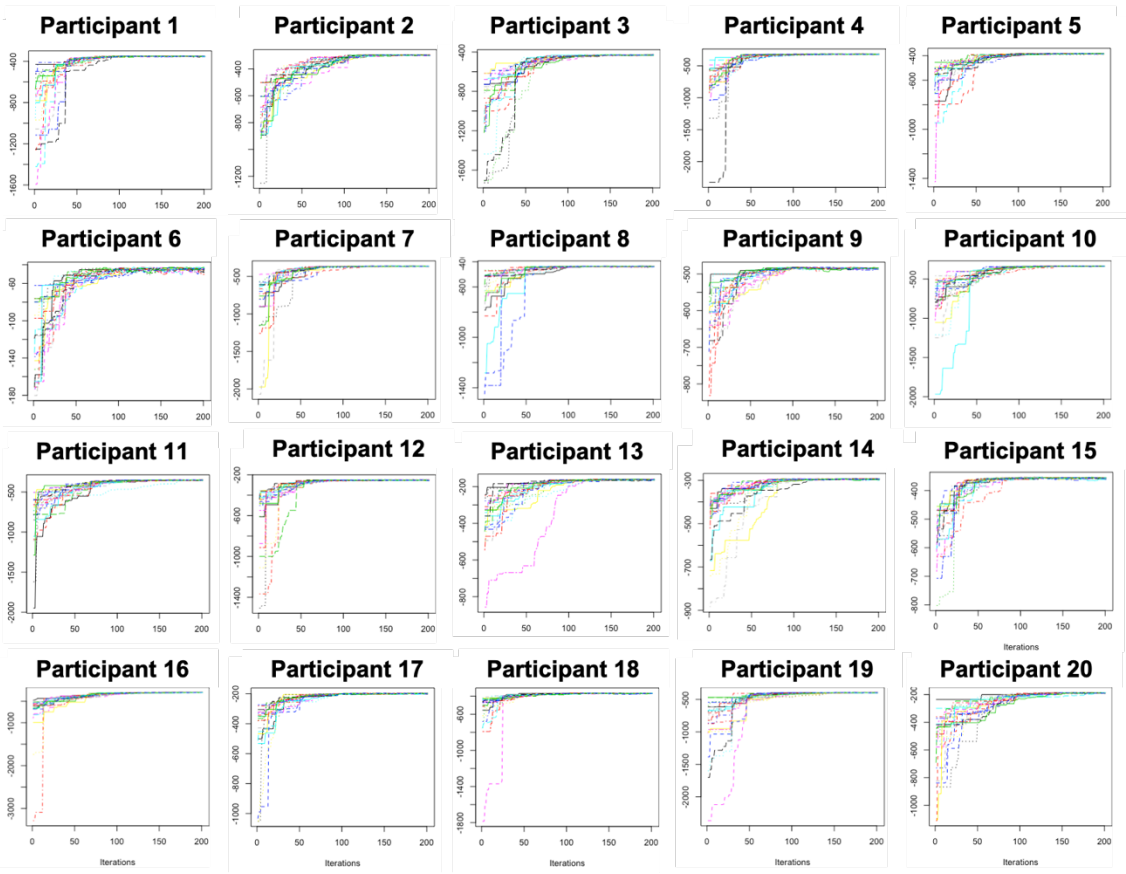
(A) Point estimates are obtained through a process where the data generated by the model is continuously informed by the behavioural evidence using Bayes' Theorem. In other words, how likely are the results produced by the model in light of our behavioural evidence (P(model\evidence). (B) Illustrates the density of the prior distributions of the winning hypothesis (Model 2), randomly generated using the plot.prior function.

Point estimates were subsequently inferred from the central tendency measures of the posterior distribution, which also quantifies the uncertainty of the parameter estimates. However, deriving the posterior distribution analytically can prove computationally intractable even for slightly non-trivial models. Hence, an indirect method of simulating complex distributions using Markov chain theory was adopted (Gamerman & Lopes, 2006; Robert, 2015).

The posterior distribution was therefore approximated using Monte Carlo Markov Chain (MCMC), a class of algorithms that create samples from a desired distribution by producing Markov chains — systems that transition from one state to another — where the target distribution is stationary for the chain.

A Markov chain is said to be stationary if the moments of a probability distribution in the chain remain constant as time progresses. In other words, given a probability density π, associated with our target distribution and defined on the state space χ, MCMC methods propose generic ways to construct an ergodic chain, i.e., a chain which forgets its initial (often noninformative) value, that is stationary with respect to π: if $\mathcal{X}(t)\sim\pi(x)$, then $\mathcal{X}(t+1)\sim\pi(x)$ — and converges in distribution to π. Intuitively, stationarity is achieved by a gradual and slow process dependent on the chain's ability to move and reach any region of the parameter space, irrespective of its starting value. Markov chains are thus able to construct a progressively more precise representation of the posterior distribution, proceeding by local exploration of the state space χ until uncovering all the regions of interest. Transitions of state in the Markov Chain are performed until the chain reaches stationarity, at which point we can take samples from the chain to approximate the target distribution by Monte Carlo methods.

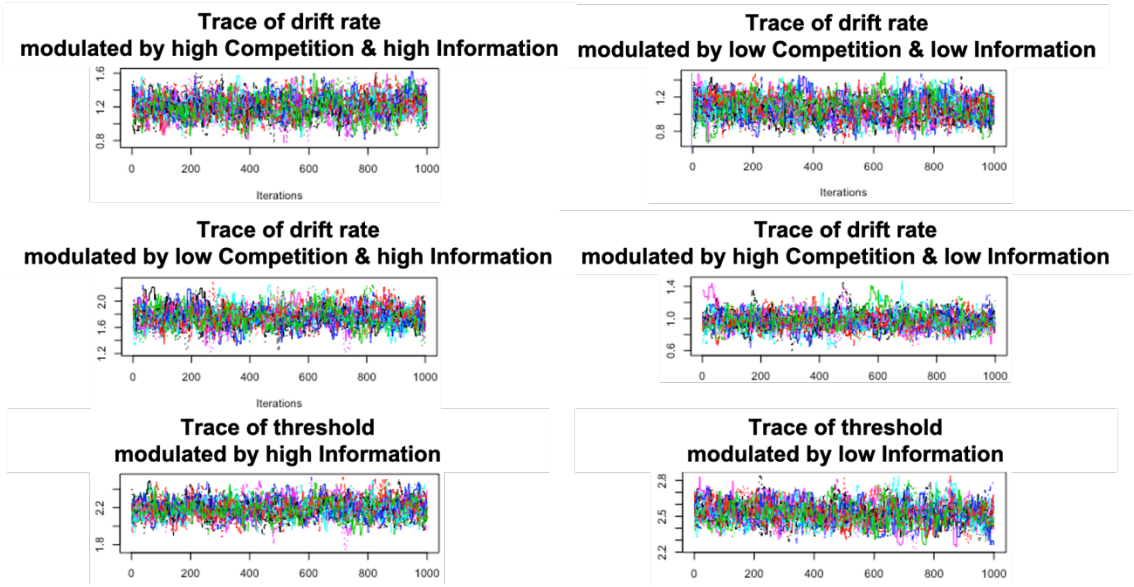
In practice, MCMC can fail to produce useful estimates in evidence-accumulation models, particularly in cases where the parameters and chains are highly correlated. Fitting the DDM using the R-based Dynamic Models of Choice (DMC) software solved these technical difficulties by relying on a Differential-Evolution (DE-MCMC) sampler that generates a large set of Markov Chains. I started with a burn-in of 250 samples (using the *h.run.dmc* function) for each of the 21 MCMC chains (3 x 7 parameters of Model 2) with the initial values being randomly generated from the prior distribution (using *h.samples.dmc* function). The chains for each participant rapidly converged to the higher likelihood regions in the parameter space (see **Appendix Figure 2.2**, drawn using *plot.dmc*). Correlations between chains were determined in "crossover" steps — amplified resorting to "migration" steps in 5% of the iterations, facilitating chain convergence in higher-likelihood areas — which guided the DE-MCMC in the exploration of the state space. Sampling proceeded until an equilibrium was reached, i.e., the data exerted its full influence on the sampling process.



Appendix Figure 2.2| Posterior log likelihoods for Model 2 (plotted using the plot.dmc function) for the last 200 out of 400 iterations originating from randomly generated start values.

The DMC software was particularly useful in guaranteeing convergence because the run.dmc function automatically corrects for convergence issues. It augments crossover steps with a migration step until "stuck" chains were removed — replacing lower-likelihood chains with slightly perturbed copies of higher-likelihood chains.

However, these results (Appendix Figure 2.2) cannot reliably be used in parameter estimation because migration steps can bias the posterior samples. The initial "burn-in" samples were thus discarded; migration was turned off and 1000 additional converged samples were generated (using the *h.run.dmc* function) to adequately approximate the posterior distributions and the associated uncertainty of the parameter estimates. Chains for all participants were visually inspected (using the h.*plot.dmc* function), as per in the example provided by Appendix Figure 2.3, which revealed the expected "flat, fat, hairy caterpillars".



Appendix Figure 2.3| Visual inspection of the quality of sampling.

After migration was turned off, new and old samples were added and removed until chains were mixed (assessed with the $\widetilde{\boldsymbol{R}}$ statistic) and flat (checked by comparing the location and spread of the first and last third of the samples), resembling fat, flat, hairy caterpillars (drawn using plot.dmc). Mixing here is meant to refer to "similarity of within-and between chains variability," while flat refers to no discernible temporal trend. The run.dmc function in DMC repeatedly performs all steps, starting with stuck chains, then moving on to mixing and flatness, but going back to stuck chains if necessary, until convergence is reached.

This visual inspection was further corroborated (using the *h.gelman.diag.dmc* function) by examining the $\tilde{R}$ statistic, which was lower than 1.05 for all participants, well below the ideal cut-off value of 1.1. Additionally, the mean effective sample size, which accounts for redundancy due to autocorrelation between chains (using *effectiveSize.dmc* function), was 964.257 providing a good approximation of the posterior distribution.

Appendix Table 2.41 | Experiment 2, (n =20) Bayesian Drift Diffusion Modelling parameters

Drift rate, v	Threshold, a	Summed Minimum Deviance & DIC	Summed Minimum Deviance & BPIC	Summary					
Amount of Information (high, low)	Amount of Competition (HIGH, LOW)	2328.48 12527.04	12328.48 12626.99	Participant	a.HIGH	a.LOW	v.high	v.low	t0

				1	2.36	2.44	.61	10.93	0.61
				2	1.41	1.52	0.64	0.62	0.67
				3	2.44	2.29	1.21	0.87	0.59
				4	1.69	1.58	0.88	0.64	0.59
				5	2.83	2.63	1.58	0.98	0.61
				6	2.33	1.97	2.57	1.71	0.59
				7	1.66	1.67	0.85	0.64	0.87
				8	2.47	2.25	1.14	0.87	0.53
				9	2.87	2.83	1.26	0.85	0.63
				10	2.54	2.04	1.40	1.01	0.56
				11	1.75	1.78	1.02	0.58	0.44
				12	2.38	1.93	1.57	1.20	0.65
				13	2.00	1.92	2.00	1.15	0.69
				14	2.31	2.29	1.27	1.40	0.53
				15	1.96	1.98	1.30	0.69	0.64
				16	1.50	1.63	1.12	0.71	0.65
				17	2.28	2.19	1.72	1.47	0.62
				18	2.81	2.66	1.42	1.18	0.53
				19	2.20	2.19	1.28	0.85	0.65
				20	2.44	2.22	1.93	1.57	0.64
				Mean	2.21	2.10	1.39	1.00	0.61

Amount of Competition (HIGH, LOW)	Amount of Information (high, low)	12312.52 12509.94	12312.52 12608.77	Participant	a.high	a.low	v.HIGH	v.LOW	t0
				1	2.04	2.59	1.05	1.35	0.62
				2	1.40	1.54	0.48	0.74	0.67
				3	2.19	2.53	0.91	1.13	0.59
				4	1.62	1.66	0.54	1.00	0.59
				5	2.34	2.92	1.17	1.21	0.62
				6	1.86	2.28	1.82	2.23	0.60
				7	1.52	1.81	0.68	0.78	0.88
				8	2.28	2.45	0.85	1.15	0.52
				9	2.64	2.95	1.15	0.89	0.63
				10	2.22	2.39	0.96	1.46	0.54
				11	1.66	1.88	0.45	1.07	0.45
				12	2.03	2.28	1.11	1.68	0.64
				13	1.65	2.19	1.27	1.66	0.70
				14	2.23	2.40	1.28	1.41	0.53
				15	1.78	2.09	0.83	1.03	0.64
				16	1.38	1.70	0.70	1.01	0.66
				17	2.16	2.32	1.43	1.77	0.62
				18	2.69	2.74	1.26	1.31	0.53
				19	2.02	2.32	0.91	1.13	0.66
				20	2.18	2.46	1.62	1.84	0.63
				Mean	2.00	2.28	1.02	1.29	0.62

Amount of Information (high, low) & Amount of Competition (HIGH, LOW)	Amount of Information (high, low) & Amount of Competition (HIGH, LOW)	12014.82 12367.81	12014.82 12544.59	Participant	a.HIGH.high	a.LOW.high	a.HIGH.low	a.LOW.low	v.HIGH.high	v.LOW.high	v.HIGH.low	v.LOW.low	t0
				1	2.07	2.43	2.44	2.69	1.14	1.95	0.93	1.13	0.61
				2	1.34	1.46	1.47	1.61	0.39	0.80	0.52	0.77	0.67
				3	2.33	2.18	2.47	2.49	1.09	1.23	0.81	1.03	0.59
				4	1.63	1.64	1.71	1.60	0.62	1.19	0.50	0.82	0.59
				5	2.59	2.50	2.98	2.71	1.37	1.64	1.14	0.95	0.62
				6	2.48	1.84	2.2	2.10	2.83	2.34	1.57	1.94	0.59
				7	1.55	1.49	1.77	1.84	0.75	0.86	0.63	0.74	0.88
				8	2.52	2.18	2.33	2.45	1.04	1.45	0.75	1.03	0.53
				9	2.97	2.78	3.12	2.84	1.18	1.35	1.30	0.63	0.63
				10	2.51	2.30	2.44	2.16	1.09	2.08	0.99	1.04	0.55
				11	1.62	1.87	1.84	1.88	0.53	1.51	0.38	0.85	0.44

				12	2.42	1.86	2.17	2.1	1.33	1.91	1.09	1.39	0.65
				13	1.74	1.99	2.10	2.16	1.39	2.56	1.16	1.34	0.69
				14	2.11	2.27	2.57	2.52	1.06	1.34	1.50	1.58	0.53
				15	1.88	1.88	1.98	2.13	1.17	1.33	0.57	0.87	0.64
				16	1.40	1.43	1.53	1.83	0.78	1.31	0.58	0.92	0.66
				17	2.24	2.30	2.18	2.41	1.42	2.14	1.38	1.65	0.61
				18	2.79	2.90	2.87	2.53	1.40	1.55	1.23	1.10	0.53
				19	2.05	2.19	2.25	2.29	1.02	1.48	0.81	0.94	0.65
				20	2.40	2.13	2.40	2.38	1.83	1.93	1.55	1.68	0.64
				Mean	2.13	2.08	2.24	2.24	1.17	1.60	0.97	1.12	0.61

				Participant	a.high	a.low	v.HIGH.high	v.LOW.high	v.HIGH.low	v.LOW.low	t0
Amount of Information (high, low) & Amount of Competition (HIGH, LOW)	Amount of Information (high, low)	12087.48 12361.78	12087.48 12499.14	1	2.19	2.53	1.21	1.78	0.97	1.06	0.62
				2	1.40	1.53	0.42	0.76	0.54	0.73	0.67
				3	2.25	2.48	1.04	1.26	0.82	1.03	0.59
				4	1.64	1.65	0.61	1.19	0.47	0.85	0.59
				5	2.54	2.82	1.34	1.67	1.08	0.98	0.62
				6	2.12	2.19	2.39	2.65	1.53	2.00	0.59
				7	1.53	1.80	0.71	0.88	0.65	0.73	0.88
				8	2.36	2.40	0.97	1.57	0.76	1.00	0.52
				9	2.89	2.93	1.14	1.40	1.22	0.65	0.62
				10	2.46	2.30	1.06	2.19	0.92	1.10	0.54
				11	1.71	1.85	0.56	1.39	0.37	0.83	0.44
				12	2.18	2.22	1.17	2.17	1.09	1.39	0.63
				13	1.81	2.10	1.46	2.37	1.16	1.31	0.70
				14	2.18	2.53	1.11	1.30	1.48	1.59	0.53
				15	1.87	2.05	1.17	1.32	0.59	0.83	0.64
				16	1.40	1.68	0.77	1.29	0.64	0.85	0.66
				17	2.25	2.28	1.45	2.09	1.44	1.56	0.62
				18	2.84	2.66	1.42	1.52	1.14	1.16	0.53
				19	2.10	2.27	1.05	1.42	0.82	0.93	0.65
				20	2.26	2.40	1.72	2.05	1.54	1.68	0.63
				Mean	2.10	2.23	1.14	1.61	0.96	1.11	0.61

				Participant	a.HIGH	a.LOW	v.HIGH.high	v.LOW.high	v.HIGH.low	v.LOW.low	t0
Amount of Information (high, low) & Amount of Competition (HIGH, LOW)	Amount of Competition (HIGH, LOW)	12117.45 12390.02	12117.45 12527.22	1	2.29	2.62	1.24	2.08	0.88	1.09	0.61
				2	1.40	1.53	0.41	0.83	0.51	0.73	0.67
				3	2.41	2.34	1.11	1.31	0.79	0.97	0.58
				4	1.67	1.62	0.62	1.18	0.47	0.83	0.59
				5	2.81	2.66	1.46	1.71	1.06	0.92	0.61
				6	2.35	1.97	2.67	2.49	1.63	1.81	0.59
				7	1.65	1.68	0.76	0.95	0.59	0.68	0.87
				8	2.41	2.33	0.99	1.55	0.77	0.99	0.53
				9	3.05	2.82	1.21	1.38	1.26	0.62	0.62
				10	2.46	2.19	1.07	2.00	1.01	1.07	0.55
				11	1.72	1.88	0.56	1.51	0.35	0.84	0.44
				12	2.30	2.07	1.25	2.09	1.13	1.29	0.64
				13	1.92	2.11	1.51	2.70	1.06	1.31	0.69
				14	2.28	2.35	1.15	1.40	1.34	1.48	0.53
				15	1.94	2.02	1.20	1.42	0.56	0.82	0.63
				16	1.49	1.68	0.79	1.49	0.55	0.84	0.65
				17	2.20	2.37	1.40	2.17	1.38	1.61	0.61
				18	2.82	2.65	1.41	1.43	1.22	1.14	0.53
				19	2.16	2.26	1.07	1.51	0.78	0.93	0.65
				20	2.40	2.25	1.82	2.04	1.54	1.58	0.63
				Mean	2.19	2.17	1.19	1.66	0.94	1.08	0.61

Amount of Information (high, low)	Amount of Information (high, low) & Amount of Competition (HIGH, LOW)	12196.59 12469.99	12196.59 12606.75	Participant	a.HIGH.high	a.LOW.high	a.HIGH.low	a.LOW.low	v.high	v.low	t0
				1	2.18	2.13	2.43	2.63	1.46	0.99	0.62
				2	1.34	1.44	1.48	1.60	0.60	0.66	0.67
				3	2.34	2.14	2.51	2.42	1.15	0.92	0.59
				4	1.66	1.59	1.72	1.57	0.88	0.64	0.59
				5	2.66	2.40	2.87	2.75	1.49	1.03	0.62
				6	2.32	1.91	2.32	2.01	2.54	1.73	0.60
				7	1.57	1.50	1.77	1.85	0.79	0.70	0.88
				8	2.59	2.10	2.37	2.38	1.18	0.88	0.53
				9	3.02	2.67	2.70	2.92	1.25	0.84	0.63
				10	2.67	1.94	2.42	2.11	1.41	1.01	0.56
				11	1.65	1.71	1.85	1.83	0.98	0.61	0.44
				12	2.53	1.71	2.21	2.10	1.55	1.23	0.66
				13	1.83	1.67	2.10	2.09	1.81	1.25	0.70
				14	2.16	2.19	2.56	2.47	1.20	1.53	0.53
				15	1.90	1.85	2.01	2.08	1.25	0.72	0.64
				16	1.41	1.39	1.55	1.79	1.02	0.78	0.66
				17	2.35	2.07	2.21	2.31	1.70	1.50	0.62
				18	2.84	2.81	2.80	2.55	1.47	1.15	0.53
				19	2.11	2.08	2.27	2.26	1.23	0.88	0.65
				20	2.42	2.10	2.44	2.34	1.87	1.61	0.64
				Mean	2.18	1.97	2.23	2.20	1.34	1.03	0.62
Amount of Competition (HIGH, LOW)	Amount of Information (high, low) & Amount of Competition (HIGH, LOW)	12222.31 12496.25	12222.31 12633.25	Participant	a.HIGH.high	a.LOW.high	a.HIGH.low	a.LOW.low	v.HIGH	v.LOW	t0
				1	2.01	2.12	2.43	2.83	1.01	1.44	0.62
				2	1.34	1.46	1.46	1.61	0.45	0.79	0.67
				3	2.26	2.14	2.52	2.52	0.93	1.12	0.59
				4	1.62	1.61	1.72	1.61	0.55	0.99	0.59
				5	2.48	2.19	3.04	2.80	1.24	1.16	0.63
				6	1.99	1.71	2.42	2.13	1.95	2.08	0.60
				7	1.55	1.50	1.75	1.87	0.67	0.80	0.88
				8	2.43	2.10	2.37	2.51	0.88	1.17	0.53
				9	2.97	2.39	3.00	2.89	1.23	0.84	0.64
				10	2.46	1.92	2.43	2.22	1.03	1.34	0.56
				11	1.60	1.76	1.84	1.94	0.44	1.12	0.44
				12	2.34	1.74	2.21	2.25	1.20	1.58	0.65
				13	1.68	1.63	2.11	2.29	1.26	1.70	0.70
				14	2.18	2.29	2.40	2.43	1.26	1.44	0.53
				15	1.78	1.80	2.02	2.16	0.81	1.04	0.64
				16	1.39	1.40	1.54	1.85	0.67	1.06	0.66
				17	2.22	2.16	2.18	2.51	1.40	1.84	0.61
				18	2.71	2.67	2.91	2.61	1.31	1.28	0.53
				19	2.02	2.05	2.28	2.36	0.90	1.15	0.65
				20	2.31	2.07	2.48	2.45	1.68	1.80	0.64
				Mean	2.07	1.93	2.26	2.29	1.04	1.29	0.62
Amount of Information (high, low)	Amount of Information (high, low)	12322.09 12517.41	12322.09 12615.17	Participant	a.high	a.low	v.high	v.low	t0		
				1	2.15	2.53	1.46	0.99	0.62		

				2	1.39	1.53	0.60	0.64	0.67
				3	2.24	2.47	1.15	0.92	0.59
				4	1.62	1.64	0.89	0.65	0.59
				5	2.54	2.82	1.48	1.03	0.62
				6	2.11	2.17	2.51	1.71	0.59
				7	1.53	1.80	0.79	0.69	0.88
				8	2.34	2.39	1.12	0.88	0.52
				9	2.86	2.86	1.26	0.85	0.62
				10	2.29	2.29	1.39	1.01	0.54
				11	1.67	1.84	0.97	0.60	0.45
				12	2.08	2.21	1.51	1.22	0.64
				13	1.75	2.10	1.82	1.24	0.70
				14	2.16	2.52	1.20	1.53	0.53
				15	1.87	2.04	1.24	0.72	0.64
				16	1.40	1.68	1.02	0.77	0.66
				17	2.19	2.26	1.68	1.50	0.62
				18	2.83	2.66	1.47	1.15	0.53
				19	2.09	2.26	1.22	0.88	0.66
				20	2.24	2.40	1.86	1.61	0.63
				Mean	2.07	2.22	1.33	1.03	0.61
Amount of Competition (HIGH, LOW)	Amount of Competition (HIGH, LOW)	12490.56 12681.80	12490.56 12780.11	Participant	a.HIGH	a.LOW	v.HIGH	v.LOW	t0
				1	2.26	2.49	1.02	1.44	0.61
				2	1.40	1.53	0.45	0.78	0.67
				3	2.40	2.33	0.93	1.11	0.58
				4	1.67	1.61	0.54	0.98	0.59
				5	2.77	2.56	1.22	1.17	0.61
				6	2.22	1.93	1.95	2.06	0.59
				7	1.65	1.68	0.67	0.79	0.87
				8	2.42	2.32	0.88	1.12	0.52
				9	3.03	2.72	1.23	0.87	0.63
				10	2.45	2.10	1.04	1.36	0.56
				11	1.72	1.85	0.44	1.13	0.44
				12	2.30	2.01	1.19	1.58	0.64
				13	1.90	1.96	1.23	1.70	0.69
				14	2.27	2.34	1.24	1.43	0.53
				15	1.91	1.99	0.82	1.05	0.63
				16	1.50	1.68	0.65	1.06	0.64
				17	2.21	2.33	1.40	1.83	0.61
				18	2.81	2.64	1.30	1.27	0.53
				19	2.15	2.23	0.90	1.16	0.65
				20	2.39	2.23	1.67	1.78	0.63
				Mean	2.17	2.13	1.04	1.28	0.61

Appendix Table 2.42 | Experiment 2, (n =20) Repeated Measures ANOVA on best Model fit drift rates
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	2.295	1	2.295	38.262	< .001	0.668
Residuals	1.140	19	0.060			
Competition	1.963	1	1.963	43.420	< .001	0.696
Residuals	0.859	19	0.045			
Competition ✻ Information	0.523	1	0.523	21.454	< .001	0.530
Residuals	0.859	19	0.045			

Note. Type III Sum of Squares

Appendix Table 2.43 | Experiment 2, (n =20) Post-Hoc Tests on best Model fit drift rates

Post Hoc Comparisons - Competition ✻ Information

		Mean Difference	SE	t	p_{holm}
HIGH, high	LOW, high	-0.475	0.059	-8.053	< .001
	HIGH, low	0.177	0.065	2.725	0.031
	LOW, low	0.025	0.073	0.352	0.727
LOW, high	HIGH, low	0.652	0.073	8.991	< .001
	LOW, low	0.501	0.065	7.706	< .001
HIGH, low	LOW, low	-0.151	0.059	-2.568	0.031

Note. P-value adjusted for comparing a family of 6

Appendix Table 2.44 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA on best Model fit drift rates

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	5.264e-11	2.106e-10	1.000	
Information + Competition + Information ✻ Competition	0.200	0.961	99.784	1.826e+10	3.899
Information + Competition	0.200	0.039	0.160	7.321e +8	3.061
Information	0.200	3.074e -7	1.230e -6	5840.067	0.650
Competition	0.200	5.333e -8	2.133e -7	1013.013	0.948

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	1.000	1.249e+7
Competition	0.600	1.000	2.168e+6
Information ✻ Competition	0.200	0.961	99.784

Appendix Table 2.45 | Experiment 2, (n =20) Paired Samples T-test on best Model fit threshold

Paired Samples T-Test

			t	df	p
threshold_highInfo_a.high	-	threshold_lowInfo_a.low	-4.003	19	< .001

Note. Student's t-test.

Appendix Table 2.46 | Experiment 2, (n =20) Repeated Measures ANOVA on Reaction time (seconds) for inaccurate trials

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Information	0.128	1	0.128	0.071	0.794	0.005
Residuals	27.247	15	1.816			
Competition	12.052	1	12.052	14.403	0.002	0.490
Residuals	12.551	15	0.837			
Information ✻ Competition	0.944	1	0.944	1.033	0.325	0.064
Residuals	13.707	15	0.914			

Note. Type III Sum of Squares

Appendix Table 2.47 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA on Reaction time (seconds) for inaccurate trials

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.035	0.143	1.000	
Competition	0.200	0.688	8.810	19.876	0.916
Information + Competition	0.200	0.182	0.889	5.256	1.498
Information + Competition + Information ✻ Competition	0.200	0.087	0.381	2.512	2.912
Information	0.200	0.009	0.036	0.256	0.741

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.278	0.256
Competition	0.600	0.957	14.668
Information ✻ Competition	0.200	0.087	0.381

Appendix Chapter 3. Successful recall relies on the information carried by the foil, not just the target

Experiment 1

Appendix Table 3.1 | Experiment 1, (n =35) Repeated Measures ANOVA: Identification Accuracy
Within Subjects Effects

	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of features	0.072	1	0.072	26.101	< .001	0.434
Residual	0.093	34	0.003			
Congruency	0.006	1	0.006	2.763	0.106	0.075
Residual	0.074	34	0.002			
Number of features ✻ Congruency	1.984e-4	1	1.984e-4	0.085	0.773	0.002
Residual	0.080	34	0.002			

Note. Type III Sum of Squares

Appendix Table 3.2 | Experiment 1, (n =35) Bayesian Repeated Measures ANOVA: Identification Accuracy
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	1.650e-5	6.601e-5	1.000	
Number of features	0.200	0.593	5.832	35945.671	1.908
Congruency	0.200	7.121e-6	2.848e-5	0.432	1.027
Number of features + Congruency	0.200	0.320	1.878	19364.494	1.312
Number of features + Congruency + Number of features ✻ Congruency	0.200	0.087	0.382	5288.560	2.669

Note. All models include subject.

Analysis of Effects

Effects	P(incl)	P(incl|data)	$BF_{Inclusion}$
Number of features	0.600	1.000	28221.350
Congruency	0.600	0.407	0.457
Number of features ✻ Congruency	0.200	0.087	0.382

Appendix Table 3.3 | Experiment 1, (n =35) Repeated Measures ANOVA: Localisation error (pixels)
Within Subjects Effects

	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of features	22.053	1	22.053	0.224	0.639	0.007
Residual	3346.573	34	98.429			
Congruency	1342.237	1	1342.237	13.781	< .001	0.288
Residual	3311.410	34	97.394			
Number of features ✻ Congruency	22.672	1	22.672	0.244	0.625	0.007
Residual	3161.317	34	92.980			

Note. Type III Sum of Squares

Appendix Table 3.4 | Experiment 1, (n =35) Bayesian Repeated Measures ANOVA: Localisation error (pixels)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.009	0.037	1.000	
Number of features	0.200	0.002	0.007	0.196	0.929
Congruency	0.200	0.789	14.982	85.878	1.816
Number of features + Congruency	0.200	0.157	0.744	17.057	2.562
Number of features ✻ Congruency + Number of features	0.200	0.043	0.180	4.675	5.723

Note. All models include subject.

Analysis of Effects

Effects	P(incl)	P(incl|data)	$BF_{Inclusion}$
Number of features	0.600	0.202	0.168
Congruency	0.600	0.989	59.977
Number of features ✻ Congruency	0.200	0.043	0.180

Appendix Table 3.5 | Experiment 2a, (n =35) Repeated Measures ANOVA: Reaction Time (seconds)

Within Subjects Effects

	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of features	1.533	1	1.533	45.947	< .001	0.575
Residual	1.134	34	0.033			
Congruency	0.002	1	0.002	0.063	0.803	0.002
Residual	0.933	34	0.027			
Number of features ✻ Congruency	0.034	1	0.034	1.502	0.229	0.042
Residual	0.761	34	0.022			

Note. Type III Sum of Squares

Appendix Table 3.6 | Experiment 1, (n =35) Bayesian Repeated Measures ANOVA: Reaction Time (seconds)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	3.278e-9	1.311e-8	1.000	
Number of features	0.200	0.801	16.125	2.444e+8	1.571
Congruency	0.200	5.980e-10	2.392e-9	0.182	1.006
Number of features + Congruency	0.200	0.141	0.658	4.310e+7	1.680
Number of features ✻ Congruency + Number of features	0.200	0.057	0.244	1.754e+7	1.709

Note. All models include subject.

Analysis of Effects

Effects	P(incl)	P(incl|data)	$BF_{Inclusion}$
Number of features	0.600	1.000	1.720e+8
Congruency	0.600	0.199	0.165
Number of features ✻ Congruency	0.200	0.057	0.244

Experiment 2

Appendix Table 3.7 | Experiment 2, (n = 20) Repeated Measures ANOVA: Identification Accuracy
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Number of features	0.030	1	0.030	11.472	0.003	0.150	0.376
Residuals	0.050	19	0.003				
Congruency	0.024	1	0.024	5.697	0.028	0.117	0.231
Residuals	0.079	19	0.004				
Number of features ✻ Congruency	5.000e-6	1	5.000e-6	0.005	0.944	2.465e-5	2.660e-4
Residuals	0.019	19	9.892e-4				

Note. Type III Sum of Squares

Appendix Table 3.8 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Identification Accuracy
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.004	0.017	1.000	
Number of features + Congruency	0.200	0.687	8.761	159.912	1.511
Number of features + Congruency + Number of features ✻ Congruency	0.200	0.217	1.111	50.624	3.763
Number of features	0.200	0.067	0.288	15.621	1.183
Congruency	0.200	0.025	0.102	5.765	0.749

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of features	0.600	0.971	22.286
Congruency	0.600	0.929	8.676
Number of features ✻ Congruency	0.200	0.217	1.111

Appendix Table 3.9 | Experiment 2, (n = 20) Repeated Measures ANOVA: Localisation error (pixels)
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of features	62.419	1	62.419	1.169	0.293	0.058
Residuals	1014.073	19	53.372			
Congruency	303.329	1	303.329	5.993	0.024	0.240
Residuals	961.675	19	50.614			
Number of features ✻ Congruency	3.526	1	3.526	0.140	0.712	0.007
Residuals	477.944	19	25.155			

Note. Type III Sum of Squares

Appendix Table 3.10 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Localisation error (pixels)
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.121	0.551	1.000	
Congruency	0.200	0.532	4.538	4.389	2.324

Appendix Table 3.10 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Localisation error (pixels)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of features + Congruency	0.200	0.225	1.163	1.861	1.809
Number of features + Congruency + Number of features ✻ Congruency	0.200	0.071	0.308	0.590	2.290
Number of features	0.200	0.051	0.213	0.418	2.039

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of features	0.600	0.347	0.355
Congruency	0.600	0.828	3.216
Number of features ✻ Congruency	0.200	0.071	0.308

Appendix Table 3.11 | Experiment 2, (n = 20) Repeated Measures ANOVA: Reaction time (seconds)

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of features	0.180	1	0.180	16.736	< .001	0.468
Residuals	0.204	19	0.011			
Congruency	3.356e-4	1	3.356e-4	0.076	0.785	0.004
Residuals	0.084	19	0.004			
Number of features ✻ Congruency	0.008	1	0.008	1.320	0.265	0.065
Residuals	0.116	19	0.006			

Note. Type III Sum of Squares

Appendix Table 3.12 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Reaction time (seconds)

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	2.235e-4	8.943e-4	1.000	
Number of features	0.200	0.731	10.847	3268.630	0.698
Number of features + Congruency	0.200	0.187	0.921	837.010	3.022
Number of features + Congruency + Number of features ✻ Congruency	0.200	0.082	0.358	367.076	2.208
Congruency	0.200	5.185e-5	2.074e-4	0.232	1.294

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of features	0.600	1.000	2420.380
Congruency	0.600	0.269	0.246
Number of features ✻ Congruency	0.200	0.082	0.358

Appendix Table 3.13 | Experiment 2, (n = 20) Repeated Measures ANOVA: Imprecision
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of Features	4.621	1	4.621	0.398	0.536	0.021
Residuals	220.625	19	11.612			
Congruency	0.336	1	0.336	0.100	0.756	0.005
Residuals	64.009	19	3.369			
Number of Features ✻ Congruency	3.300	1	3.300	0.402	0.534	0.021
Residuals	156.128	19	8.217			

Note. Type III Sum of Squares

Appendix Table 3.14 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Imprecision
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.616	6.427	1.000	
number of Features	0.200	0.183	0.893	0.296	1.350
Congruency	0.200	0.141	0.655	0.228	0.877
number of Features + Congruency	0.200	0.044	0.186	0.072	2.598
number of Features + Congruency + number of Features ✻ Congruency	0.200	0.016	0.066	0.026	3.001

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
number of Features	0.600	0.243	0.214
Congruency	0.600	0.201	0.168
number of Features ✻ Congruency	0.200	0.016	0.066

Appendix Table 3.15 | Experiment 2, (n = 20) Repeated Measures ANOVA: Misbinding
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number Features	9.886e-4	1	9.886e-4	2.149	0.159	0.102
Residuals	0.009	19	4.599e-4			
Congruency	0.001	1	0.001	5.418	0.031	0.222
Residuals	0.005	19	2.405e-4			
Congruency ✻ Number Features	4.725e-5	1	4.725e-5	0.433	0.518	0.022
Residuals	0.005	19	2.405e-4			

Note. Type III Sum of Squares

Appendix Table 3.16 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Misbinding
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.157	0.748	1.000	
Number of Features + Congruency	0.200	0.314	1.827	1.991	2.615
Congruency	0.200	0.264	1.432	1.674	0.921
Number of Features	0.200	0.160	0.762	1.016	1.221
Number of Features + Congruency + Number of Features ✻ Congruency	0.200	0.105	0.471	0.669	7.817

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of Features	0.600	0.579	0.916
Congruency	0.600	0.683	1.433
Number of Features ✻ Congruency	0.200	0.105	0.471

Appendix Table 3.17 | Experiment 2, (n = 20) Repeated Measures ANOVA: Guessing
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of Features	7.669e-5	1	7.669e-5	0.259	0.617	0.013
Residuals	0.006	19	2.964e-4			
Congruency	3.028e-5	1	3.028e-5	0.201	0.659	0.010
Residuals	0.003	19	1.508e-4			
Number of Features ✻ Congruency	1.406e-5	1	1.406e-5	0.067	0.798	0.004
Residuals	0.004	19	2.094e-4			

Note. Type III Sum of Squares

Appendix Table 3.18 | Experiment 2, (n = 20) Bayesian Repeated Measures ANOVA: Guessing
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.624	6.632	1.000	
Number of features	0.200	0.169	0.811	0.270	1.571
Congruency	0.200	0.152	0.717	0.244	1.595
Number of features + Congruency	0.200	0.043	0.179	0.069	2.347
Number of features + Congruency + Number of features ✻ Congruency	0.200	0.013	0.052	0.021	2.141

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of features	0.600	0.224	0.193
Congruency	0.600	0.208	0.175
Number of features ✻ Congruency	0.200	0.013	0.052

Experiment 2 (HDDM)

Similarly to Chapter 2, Experiment 2, 250 iterations were run (using the *run.dmc* function) for each of the 21 MCMC chains (3 x 7 parameters for Model 2). Initial values were randomly drawn from the prior distribution (using the h.*samples.dmc* function) and thus had very similar (low) likelihoods. After the initial burn-in phase, I ran an additional 1000 iterations with migration turned off. Although migration can be very useful during burn-in because it deals with chains that become stuck in low probability areas of the parameter space, driving the sampler to the right region of the state space, it can lead to false convergence (i.e., all chains converge on the same suboptimal solution) when used extensively. The fresh 1000 samples

were visually inspected, and successful convergence was further corroborated by both the $\tilde{R}$ statistic being lower than 1.04 (below the ideal cut-off of 1.1) and the effective sample size, which adjusted the actual sample size (21 chains x 1000 iterations) for redundancy due to autocorrelations (assessed with *effectiveSize.dmc* function), being 952.83.

Appendix Table 3.19 | Modelling parameters Experiment 2

Features= high, low Congruency = HIGH, LOW	Drift rate, v	Threshold, a	Summed Minimum Deviance & DIC	Summed Minimum Deviance & BPIC	Summary					
Model 1	Features	Congruency	9932.557 10128.878	9932.557 10227.271		a.HIGH	a.LOW	v.high	v.low	t0
					1	2.79	2.97	0.98	1.18	0.59
					2	2.53	2.31	1.38	1.76	0.45
					3	1.86	1.95	0.73	1.54	0.55
					4	2.02	1.88	0.70	1.05	0.43
					5	2.59	2.67	1.60	1.98	0.40
					6	2.57	2.64	1.69	2.00	0.49
					7	1.47	1.60	0.60	0.53	0.49
					8	1.53	1.72	1.16	1.45	0.58
					9	1.67	1.86	0.99	1.21	0.50
					10	2.24	2.16	1.93	2.29	0.54
					11	1.85	1.82	0.78	0.91	0.38
					12	2.43	2.49	1.73	1.85	0.49
					13	2.39	2.43	1.26	1.72	0.51
					14	3.09	2.91	1.00	1.25	0.53
					15	1.47	1.54	0.93	1.50	0.52
					16	1.92	1.87	1.82	2.42	0.49
					17	1.72	1.73	0.85	1.11	0.50
					18	2.43	2.44	2.00	2.56	0.53
					19	2.82	2.77	0.92	1.54	0.58
					20	2.21	2.11	1.18	1.27	0.36
					Mean	2.18	2.19	1.21	1.56	0.49
Model 2	Congruency	Features	10015.40 10212.25	10015.4 10310.8		a.high	a.low	v.HIGH	v.LOW	t0
					1	3.04	2.69	1.11	1.04	0.60
					2	2.59	2.21	1.43	1.66	0.46
					3	1.92	1.79	0.97	1.13	0.55
					4	1.94	1.95	0.89	0.84	0.42
					5	2.79	2.40	1.84	1.69	0.41
					6	2.72	2.38	1.93	1.73	0.50
					7	1.58	1.49	0.52	0.60	0.49
					8	1.74	1.51	0.87	1.65	0.58
					9	1.72	1.81	0.91	1.23	0.50
					10	2.24	2.15	2.08	2.09	0.54
					11	1.89	1.79	0.67	1.05	0.38
					12	2.44	2.51	1.82	1.80	0.49
					13	2.53	2.22	1.38	1.53	0.51

					14	3.08	2.85	1.07	1.14	0.54
					15	1.51	1.46	1.23	1.14	0.52
					16	1.90	1.84	2.00	2.15	0.49
					17	1.78	1.67	0.96	0.98	0.50
					18	2.43	2.34	2.13	2.31	0.53
					19	2.92	2.38	1.10	1.15	0.61
					20	2.21	2.11	1.19	1.26	0.36
					Mean	2.25	2.08	1.30	1.41	0.50

Model 3	Features&Congruency	Features&Congruency	9730.614 10085.082	9730.614 10262.316		a.high.HIGH	a.low.HIGH	a.high.LOW	a.low.LOW	v.high.HIGH	v.low.HIGH	v.high.LOW	v.low.LOW	t0
					1	2.99	2.59	3.04	2.91	1.14	1.02	0.95	1.25	0.60
					2	2.61	2.36	2.45	2.18	1.37	1.66	1.54	1.66	0.46
					3	1.84	1.89	1.92	2.12	0.58	1.51	0.86	1.70	0.54
					4	1.97	2.18	1.85	1.90	0.65	1.29	0.71	0.92	0.42
					5	2.76	2.46	2.78	2.56	1.94	1.71	1.50	2.09	0.40
					6	2.92	2.41	2.52	2.50	1.94	2.08	1.62	1.77	0.50
					7	1.55	1.39	1.62	1.58	0.55	0.47	0.69	0.55	0.49
					8	1.61	1.39	2.10	1.94	0.80	0.77	1.86	2.21	0.57
					9	1.60	1.69	1.84	2.06	0.70	1.02	1.22	1.54	0.50
					10	2.20	2.39	2.13	2.22	1.95	2.42	1.84	2.35	0.54
					11	1.89	1.76	1.88	1.85	0.59	0.76	1.08	1.06	0.38
					12	2.39	2.57	2.44	2.63	1.57	2.14	1.99	1.80	0.49
					13	2.37	2.27	2.56	2.51	1.21	1.51	1.37	1.97	0.51
					14	3.27	2.95	2.82	3.04	1.07	1.17	0.94	1.32	0.53
					15	1.48	1.46	1.49	1.63	1.08	1.36	0.77	1.69	0.52
					16	1.90	1.99	1.76	2.24	1.71	2.51	1.82	2.84	0.49
					17	1.74	1.70	1.78	1.67	0.78	1.15	0.95	1.02	0.50
					18	2.22	2.68	2.35	2.94	1.65	2.86	2.11	3.01	0.52
					19	3.02	2.53	2.64	2.77	0.95	1.50	0.95	1.46	0.60
					20	2.31	2.13	2.12	2.14	1.28	1.17	1.14	1.35	0.36
					Mean	2.23	2.14	2.21	2.27	1.18	1.50	1.30	1.68	0.50

Model 4	Features&Congruency	Features	9814.058 10090.354	9814.058 10228.502		a.high	a.low	v.high.HIGH	v.low.HIGH	v.high.LOW	v.low.LOW	t0
					1	3.02	2.73	1.15	1.07	0.94	1.17	0.60
					2	2.54	2.28	1.32	1.59	1.59	1.74	0.45
					3	1.87	2.00	0.58	1.60	0.84	1.61	0.54
					4	1.91	2.02	0.63	1.20	0.73	0.97	0.42
					5	2.77	2.50	1.95	1.76	1.50	2.05	0.40
					6	2.69	2.46	1.79	2.13	1.74	1.75	0.50
					7	1.59	1.48	0.55	0.50	0.69	0.52	0.49
					8	1.74	1.52	0.88	0.87	1.57	1.78	0.58
					9	1.70	1.84	0.75	1.10	1.13	1.37	0.50
					10	2.16	2.30	1.91	2.32	1.87	2.43	0.54
					11	1.88	1.79	0.59	0.76	1.08	1.04	0.38
					12	2.42	2.61	1.60	2.16	1.96	1.78	0.49
					13	2.44	2.37	1.25	1.57	1.31	1.85	0.51
					14	3.01	2.98	0.99	1.19	1.01	1.30	0.53
					15	1.48	1.53	1.08	1.44	0.77	1.60	0.52
					16	1.81	2.09	1.63	2.64	1.86	2.65	0.49
					17	1.77	1.69	0.79	1.14	0.93	1.03	0.50
					18	2.27	2.79	1.69	2.97	2.05	2.86	0.53

					19	2.84	2.66	0.88	1.57	1.01	1.40	0.60
					20	2.20	2.13	1.22	1.17	1.19	1.35	0.36
					Mean	2.21	2.19	1.16	1.54	1.29	1.61	0.50
Model 5	Features&congruency	Congruency	9797.759 10075.509	9797.759 10214.383		a.HIGH	a.LOW	v.high.HIGH	v.low.HIGH	v.high.LOW	v.low.LOW	t0
					1	2.78	2.98	1.06	1.09	0.93	1.27	0.59
					2	2.51	2.34	1.30	1.75	1.46	1.77	0.45
					3	1.84	1.99	0.58	1.48	0.89	1.61	0.55
					4	2.04	1.87	0.68	1.21	0.72	0.91	0.42
					5	2.61	2.69	1.83	1.82	1.46	2.19	0.40
					6	2.71	2.56	1.78	2.30	1.62	1.79	0.49
					7	1.47	1.60	0.52	0.49	0.69	0.56	0.49
					8	1.50	2.04	0.75	0.82	1.80	2.30	0.57
					9	1.63	1.94	0.72	0.98	1.29	1.44	0.50
					10	2.27	2.14	2.02	2.30	1.86	2.28	0.54
					11	1.82	1.87	0.57	0.78	1.08	1.07	0.38
					12	2.45	2.53	1.62	2.05	2.09	1.74	0.49
					13	2.33	2.55	1.18	1.54	1.36	2.00	0.50
					14	3.08	2.91	1.01	1.24	0.98	1.27	0.53
					15	1.47	1.54	1.06	1.38	0.80	1.61	0.52
					16	1.90	1.89	1.72	2.43	1.95	2.42	0.49
					17	1.72	1.73	0.77	1.17	0.92	1.06	0.50
					18	2.37	2.58	1.78	2.54	2.32	2.64	0.53
					19	2.86	2.74	0.89	1.67	0.97	1.42	0.58
					20	2.21	2.12	1.22	1.21	1.15	1.36	0.36
					Mean	2.18	2.23	1.15	1.51	1.32	1.64	0.49
Model 6	Features	Features&Congruency	9852.474 10123.719	9852.474 10259.501		a.high.HIGH	a.low.HIGH	a.high.LOW	a.low.LOW	v.high	v.low	t0
					1 vv	2.90	2.67	3.11	2.81	1.03	1.13	0.59
					2	2.66	2.35	2.40	2.17	1.45	1.66	0.46
					3	1.86	1.93	1.89	2.07	0.71	1.60	0.54
					4	1.97	2.10	1.84	1.94	0.68	1.09	0.42
					5	2.55	2.53	2.88	2.41	1.66	1.88	0.41
					6	2.77	2.28	2.59	2.57	1.76	1.90	0.50
					7	1.56	1.40	1.61	1.58	0.62	0.51	0.49
					8	1.64	1.41	1.83	1.61	1.24	1.36	0.58
					9	1.62	1.74	1.78	1.97	0.95	1.26	0.50
					10	2.17	2.37	2.14	2.24	1.88	2.38	0.54
					11	1.92	1.78	1.82	1.81	0.80	0.90	0.38
					12	2.44	2.43	2.29	2.71	1.68	1.93	0.49
					13	2.40	2.37	2.50	2.35	1.28	1.70	0.51
					14	3.20	2.98	2.86	2.96	1.00	1.24	0.53
					15	1.47	1.48	1.50	1.58	0.92	1.53	0.52
					16	1.90	2.03	1.74	2.15	1.75	2.67	0.49
					17	1.75	1.68	1.77	1.68	0.87	1.08	0.50
					18	2.30	2.68	2.18	2.86	1.84	2.91	0.53
					19	3.02	2.49	2.63	2.76	0.95	1.48	0.60
					20	2.27	2.16	2.13	2.10	1.21	1.25	0.36
					Mean	2.22	2.14	2.17	2.22	1.21	1.57	0.50

Model 7	Congruency	Features&Congruency	9922.102 10198.637	9922.102 10336.912		a.high.HIGH	a.low.HIGH	a.high.LOW	a.low.LOW	v.HIGH	v.LOW	t0
					1	2.92	2.62	3.14	2.77	1.07	1.07	0.60
					2	2.69	2.27	2.49	2.14	1.49	1.60	0.46
					3	1.88	1.70	1.98	1.89	0.93	1.18	0.55
					4	2.01	2.03	1.86	1.88	0.92	0.80	0.42
					5	2.67	2.50	2.93	2.30	1.82	1.71	0.41
					6	2.97	2.36	2.55	2.44	2.01	1.69	0.50
					7	1.54	1.40	1.60	1.59	0.50	0.62	0.49
					8	1.60	1.39	2.16	1.85	0.79	2.00	0.57
					9	1.61	1.66	1.88	2.00	0.84	1.37	0.50
					10	2.29	2.24	2.19	2.05	2.15	2.03	0.54
					11	1.90	1.75	1.89	1.85	0.66	1.08	0.38
					12	2.49	2.32	2.34	2.65	1.79	1.87	0.49
					13	2.42	2.20	2.68	2.29	1.34	1.59	0.51
					14	3.30	2.89	2.91	2.85	1.12	1.11	0.53
					15	1.50	1.43	1.52	1.51	1.21	1.16	0.52
					16	1.97	1.77	1.83	1.91	2.00	2.15	0.49
					17	1.77	1.66	1.80	1.67	0.95	0.98	0.50
					18	2.36	2.16	2.50	2.53	2.02	2.45	0.53
					19	3.11	2.26	2.71	2.51	1.12	1.15	0.61
					20	2.28	2.15	2.15	2.09	1.22	1.24	0.36
					Mean	2.26	2.04	2.26	2.14	1.30	1.44	0.50
Model 8	Features	Features	9912.082 10109.378	9912.082 10208.307		a.high	a.low	v.high	v.low	t0		
					1	3.01	2.73	1.03	1.12	0.59		
					2	2.53	2.27	1.44	1.67	0.46		
					3	1.87	2.00	0.71	1.62	0.54		
					4	1.90	2.02	0.67	1.08	0.42		
					5	2.73	2.49	1.66	1.89	0.40		
					6	2.70	2.44	1.77	1.89	0.49		
					7	1.59	1.49	0.61	0.51	0.49		
					8	1.70	1.48	1.22	1.31	0.58		
					9	1.69	1.84	0.93	1.25	0.50		
					10	2.16	2.31	1.89	2.39	0.54		
					11	1.87	1.79	0.81	0.90	0.38		
					12	2.38	2.60	1.66	1.94	0.48		
					13	2.45	2.36	1.28	1.69	0.51		
					14	3.00	2.98	0.99	1.24	0.53		
					15	1.48	1.53	0.91	1.52	0.52		
					16	1.82	2.10	1.74	2.65	0.49		
					17	1.77	1.69	0.86	1.08	0.50		
					18	2.24	2.78	1.84	2.92	0.53		
					19	2.83	2.65	0.94	1.47	0.60		
					20	2.20	2.12	1.20	1.25	0.36		
					Mean	2.20	2.18	1.21	1.57	0.50		
Model 9	Congruency	Congruency	10049.16 10245.63	10049.16 10344.16		a.HIGH	a.LOW	v.HIGH	v.LOW	t0		
					1	2.78	2.94	1.07	1.07	0.59		
					2	2.49	2.34	1.48	1.57	0.45		
					3	1.79	1.94	0.95	1.18	0.55		
					4	2.03	1.86	0.92	0.81	0.43		
					5	2.61	2.59	1.82	1.69	0.40		
					6	2.66	2.55	1.97	1.70	0.49		

7	1.47	1.60	0.50	0.63	0.49
8	1.50	2.01	0.78	2.00	0.57
9	1.63	1.94	0.84	1.36	0.50
10	2.26	2.13	2.15	2.03	0.54
11	1.83	1.87	0.66	1.07	0.38
12	2.43	2.50	1.79	1.82	0.49
13	2.31	2.49	1.34	1.59	0.50
14	3.07	2.88	1.10	1.11	0.53
15	1.46	1.52	1.22	1.17	0.52
16	1.87	1.88	2.00	2.15	0.49
17	1.72	1.73	0.96	0.98	0.50
18	2.30	2.56	2.04	2.46	0.53
19	2.74	2.70	1.12	1.15	0.58
20	2.20	2.12	1.21	1.25	0.36
Mean	2.16	2.21	1.30	1.44	0.49

Appendix Table 3.20 | Experiment 2, (n =20) Repeated Measures ANOVA on best Model fit drift rates
Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2	η^2_p
Congruency	0.412	1	0.412	3.808	0.066	0.065	0.167
Residuals	2.055	19	0.108				
Information	2.298	1	2.298	47.678	< .001	0.363	0.715
Residuals	0.916	19	0.048				
Information ✻ Congruency	0.008	1	0.008	0.247	0.625	0.001	0.013
Residuals	0.916	19	0.048				

Note. Type III Sum of Squares

Appendix Table 3.21 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA on best Model fit drift rates
Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	4.653e-6	1.861e-5	1.000	
Congruency + Information	0.200	0.606	6.152	130244.049	1.764
Congruency + Information + Congruency ✻ Information	0.200	0.212	1.073	45462.094	9.730
Information	0.200	0.182	0.893	39223.148	5.354
Congruency	0.200	8.126e-6	3.251e-5	1.747	26.270

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Congruency	0.600	0.818	2.986
Information	0.600	1.000	52168.254
Information ✻ Congruency	0.200	0.212	1.073

Appendix Table 3.22 | Experiment 2, (n =20) Paired Samples T-test on best Model fit threshold
Paired Samples T-Test

			t	df	p
a.HIGHCongruency_a.HIGH	-	a.LOWCongruency_a.LOW	-1.234	19	0.232

Note. Student's t-test.

Appendix Table 3.23 | Experiment 2, (n =20) Repeated Measures ANOVA on Reaction time (seconds) for inaccurate trials

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of features	1.870	1	1.870	4.330	0.058	0.250
Residuals	5.614	13	0.432			
Congruency	1.047	1	1.047	1.773	0.206	0.120
Residuals	7.679	13	0.591			
Number of features ✻ Congruency	0.868	1	0.868	1.651	0.221	0.113
Residuals	6.835	13	0.526			

Note. Type III Sum of Squares

Appendix Table 3.24| Experiment 2, (n =20) Bayesian Repeated Measures ANOVA on Reaction time (seconds) for inaccurate trials

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.249	1.329	1.000	
Number of features	0.200	0.289	1.630	1.161	0.787
Number of features + Congruency	0.200	0.185	0.906	0.740	1.493
Congruency	0.200	0.149	0.700	0.597	1.038
Number of features + Congruency + Number of features ✻ Congruency	0.200	0.128	0.585	0.511	2.301

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of features	0.600	0.602	1.007
Congruency	0.600	0.461	0.570
Number of features ✻ Congruency	0.200	0.128	0.585

Appendix Table 3.25 | Experiment 2, (n =20) Bayesian Repeated Measures ANOVA on Reaction time (seconds) for inaccurate trials

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.200	0.035	0.143	1.000	
Competition	0.200	0.688	8.810	19.876	0.916
Information + Competition	0.200	0.182	0.889	5.256	1.498
Information + Competition + Information ✻ Competition	0.200	0.087	0.381	2.512	2.912
Information	0.200	0.009	0.036	0.256	0.741

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Information	0.600	0.278	0.256
Competition	0.600	0.957	14.668
Information ✻ Competition	0.200	0.087	0.381

Appendix Chapter 4. Impact of attentional demands at encoding, maintenance and retrieval in visual working memory

Experiment 1

Appendix Table 4.1 | 2x2x2 ANOVA on the Proportion of Correctly Identified Shapes including set size (two versus three), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Set size	0.662	1	0.662	50.391	< .001
Set size x group	0.008	1	0.008	0.599	0.441
Residual	1.301	99	0.013		
Number of features	0.067	1	0.067	7.060	0.009
Number of features x group	$1.01e^{-8}$	1	$1.014e^{-8}$	$1.076e^{-6}$	0.999
Residual	0.933	99	0.009		
Set size x number of features	$6.805e^{-4}$	1	$6.805e^{-4}$	0.080	0.778
Set size x number of features x group	0.001	1	0.001	0.174	0.678
Residual	0.840	99	0.008		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.152	1	0.152	6.111	0.015
Residual	2.463	99	0.025		

Note: Type III Sum of Squares

Appendix Table 4.2 | 2x2x2 ANOVA on Localisation Performance including set size (two versus three), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Set size	95874.024	1	95874.024	83.623	< .001
Set size x group	4064.365	1	4064.365	3.545	0.063
Residual	113503.217	99	1146.497		
Number of features	646.903	1	646.903	0.950	0.332
Number of features x group	13.183	1	13.183	0.019	0.890
Residual	67405.641	99	67405.641		
Set size x number of features	1205.295	1	1205.295	1.506	0.223
Set size x number of features x group	668.505	1	668.505	0.835	0.363
Residual	79236.269	99	800.366		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	45979.824	1	45979.824	14.219	< .001
Residual	320135.251	99	3233.689		

Note: Type III Sum of Squares

Appendix Table 4.3 | 2x2x2 ANOVA on Misbinding including set size (two versus three), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Set size	0.864	1	0.864	73.272	<.001
Set size x group	0.061	1	0.061	5.172	0.025
Residual	1.167	99	0.012		
Number of features	0.018	1	0.018	2.811	0.097
Number of features x group	0.001	1	0.001	0.230	0.633
Residual	0.631	99	0.006		
Set size x number of features	0.006	1	0.006	0.945	0.333
Set size x number of features x group	0.002	1	0.002	0.327	0.569
Residual	0.662	99	0.007		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.165	1	0.165	8.833	0.004
Residual	1.847	99	0.019		

Note: Type III Sum of Squares

Appendix Table 4.4 | Holm-corrected post-hoc t-tests on the Interaction of set size and group in Misbinding.

		Mean Difference	SE	t	p_{holm}
Set size 3, young controls	Set size 3, elderly controls	-0.066	0.018	-3.742	< .001
	Set size 2, young controls	0.069	0.017	4.044	< .001
	Set size 2, elderly controls	0.053	0.018	3.000	0.006
Set size 3, elderly controls	Set size 2, young controls	0.136	0.018	7.653	< .001
	Set size 2, elderly controls	0.120	0.014	8.608	< .001
Set size 2, young controls	Set size 2, elderly controls	-0.016	0.018	-0.911	0.363

Note. Bonferroni adjusted confidence intervals.

Appendix Table 4.5 | 2x2x2 ANOVA on Guessing including set size (two versus three), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Set size	$7.236e^{-4}$	1	$7.236e^{-4}$	3.431	0.067
Set size x group	$3.670e^{-4}$	1	$3.670e^{-4}$	1.740	0.190
Residual	0.021	99	$2.109e^{-4}$		
Number of features	$4.295e^{-4}$	1	$4.295e^{-4}$	3.134	0.080
Number of features x group	$2.587e^{-5}$	1	$2.587e^{-5}$	0.189	0.665
Residual	0.014	99	$1.370e^{-4}$		
Set size x number of features	$9.070e^{-4}$	1	$9.070e^{-4}$	5.488	0.021
Set size x number of features x group	$3.359e^{-4}$	1	$3.359e^{-4}$	2.033	0.157
Residual	0.016	99	$1.653e^{-4}$		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.025	1	0.025	9.129	0.003
Residual	0.276	99	0.003		

Note: Type III Sum of Squares

Appendix Table 4.6 | Holm-corrected post-hoc t-tests on the Interaction of set size and number of features

		Mean Difference	SE	t	p_{holm}
Set size 3, one feature	Set size 3, two features	9.555e-4	0.002	0.540	1.000
	Set size 2, one feature	0.006	0.002	2.940	0.022
	Set size 2, two features	6.283e-4	0.002	0.331	1.000
Set size 3, two features	Set size 2, one feature	0.005	0.002	2.553	0.046
	Set size 2, two features	-3.272e-4	0.002	-0.166	1.000
Set size 2, one feature	Set size 2, two features	-0.005	0.002	-2.924	0.022

Note. Bonferroni adjusted confidence intervals.

Appendix Table 4.7 | 2x2x2 ANOVA on Imprecision including set size (two versus three), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Set size	3428.791	1	3428.791	16.113	<.001
Set size x group	696.825	1	696.825	3.275	0.073
Residual	21066.270	99	212.791		
Number of features	465.679	1	465.679	3.215	0.076

Number of features x group	12.227	1	12.227	0.084	0.772
Residual	14340.496	99	144.853		
Set size x number of features	491.759	1	491.759	2.693	0.104
Set size x number of features x group	536.195	1	536.195	2.936	0.090
Residual	18077.218	99	182.598		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	17375.314	1	17375.314	18.211	<.001
Residual	94456.998	99	954.111		

Note: Type III Sum of Squares

Appendix Table 4.8 | 2x2x2 ANOVA on Reaction Time including set size (two versus three), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Set size	27.208	1	27.208	74.709	<.001
Set size x group	2.418	1	2.418	6.641	0.011
Residual	36.055	99	0.364		
Number of features	0.096	1	0.096	0.397	0.530
Number of features x group	0.012	1	0.012	0.048	0.828
Residual	23.941	99	0.242		
Set size x number of features	0.215	1	0.215	1.218	0.272
Set size x number of features x group	$6.516e^{-5}$	1	$6.516e^{-5}$	$3.697e^{-4}$	0.985
Residual	17.449	99	0.176		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	85.974	1	85.974	49.016	<.001
Residual	173.648	99	1.754		

Note: Type III Sum of Squares

Appendix Table 4.9 | Holm-corrected post-hoc t-tests on the Interaction of set size and group in Reaction Time

		Mean Difference	SE	t	p_{holm}
Set size 3, young controls	Set size 3, elderly controls	-1.101	0.148	-7.439	< .001
	Set size 2, young controls	0.372	0.095	3.903	< .001
	Set size 2, elderly controls	-0.413	0.148	-2.787	0.006
Set size 3, elderly controls	Set size 2, young controls	1.474	0.148	9.955	< .001
	Set size 2, elderly controls	0.689	0.077	8.915	< .001
Set size 2, young controls	Set size 2, elderly controls	-0.785	0.148	-5.302	< .001

Appendix Table 4.9 | Holm-corrected post-hoc t-tests on the Interaction of set size and group in Reaction Time

		Mean Difference	SE	t	p_{holm}

Note. Bonferroni adjusted confidence intervals.

Appendix Table 4.10| 2x2x2 ANOVA on Identification Accuracy including whether the retrieval foil's feature was taken from the task-irrelevant versus non-filtered item.

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Non-filtered/task-irrelevant	0.047	1	0.047	8.227	0.005
Non-filtered/task-irrelevant x group	0.002	1	0.002	0.283	0.596
Residual	0.570	99	0.006		
Number of features	0.039	1	0.039	6.078	0.015
Number of features x group	0.018	1	0.018	2.766	0.099
Residual	0.639	99	0.006		
Non-filtered/task-irrelevant x number of features	0.012	1	0.012	2.779	0.099
Non-filtered/task-irrelevant x number of features x group	$1.227e^{-6}$	1	$1.227e^{-6}$	$2.734e^{-4}$	0.987
Residual	0.444	99	0.004		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.058	1	0.058	3.254	0.074
Residual	1.750	99	0.018		

Note: Type III Sum of Squares

Appendix Table 4.11 | 2x2x2 ANOVA on Localisation Performance including whether the retrieval foil's feature was taken from the task-irrelevant versus non-filtered item, the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Non-filtered/task-irrelevant	14.969	1	14.969	0.033	0.856
Non-filtered/task-irrelevant x group	121.521	1	121.521	0.268	0.606
Residual	44848.772	99	453.018		
Number of features	340.441	1	340.441	0.858	0.357
Number of features x group	1094.081	1	1094.081	2.757	0.100
Residual	39289.835	99	396.867		
Non-filtered/task-irrelevant x number of features	1870.087	1	1870.087	2.977	0.088

Non-filtered/task-irrelevant x number of features x group	15.336	1	15.336	0.024	0.876
Residual	62180.021	99	628.081		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	45779.975	1	45779.975	26.160	<.001
Residual	173251.805	99	1750.018		

Note: Type III Sum of Squares

Appendix Table 4.12. | 2x2x2 ANOVA on Misbinding including whether the retrieval foil's feature was taken from the task-irrelevant versus non-filtered item, the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Non-filtered/task-irrelevant	$4.293e^{-5}$	1	$4.293e^{-5}$	0.014	0.904
Non-filtered/task-irrelevant x group	0.009	1	0.009	3.059	0.083
Residual	0.294	99	0.003		
Number of features	0.002	1	0.002	0.685	0.410
Number of features x group	0.004	1	0.004	1.169	0.282
Residual	0.324	99	0.003		
Non-filtered/task-irrelevant x number of features	0.017	1	0.017	4.712	0.032
Non-filtered/task-irrelevant x number of features x group	$3.943e^{-4}$	1	$3.943e^{-4}$	0.112	0.739
Residual	0.349	99	0.004		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.044	1	0.044	7.085	0.009
Residual	0.614	99	0.006		

Note: Type III Sum of Squares

Appendix Table 4.13 | 2x2x2 ANOVA on Guessing including whether the retrieval foil's feature was taken from the task-irrelevant versus non-filtered item, the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Non-filtered/task-irrelevant	$1.228e^{-6}$	1	$1.228e^{-6}$	0.010	0.920
Non-filtered/task-irrelevant x group	$2.811e^{-4}$	1	$2.811e^{-4}$	2.339	0.129
Residual	0.012	99	$1.202e^{-4}$		
Number of features	$3.836e^{-5}$	1	$3.836e^{-5}$	0.396	0.530

Number of features x group	$7.692e^{-7}$	1	$7.692e^{-7}$	0.008	0.929
Residual	0.010	99	$9.677e^{-5}$		
Non-filtered/task-irrelevant x number of features	$1.638e^{-6}$	1	$1.638e^{-6}$	0.017	0.898
Non-filtered/task-irrelevant x number of features x group	$3.352e^{-9}$	1	$3.352e^{-9}$	$3.399e^{-5}$	0.995
Residual	0.010	99	$9.864e^{-5}$		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.029	1	0.029	12.46	<.001
Residual	0.226	99	0.002		

Note: Type III Sum of Squares

Appendix Table 4.14 | 2x2x2 ANOVA on Imprecision including whether the retrieval foil's feature was taken from the task-irrelevant versus non-filtered item, the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Non-filtered/task-irrelevant	51.298	1	51.298	0.480	0.490
Non-filtered/task-irrelevant x group	327.383	1	327.383	3.065	0.083
Residual	10573.817	99	106.806		
Number of features	4.624	1	4.624	0.039	0.843
Number of features x group	51.341	1	51.341	0.436	0.511
Residual	11666.878	99	117.847		
Non-filtered/task-irrelevant x number of features	25.162	1	25.162	0.209	0.649
Non-filtered/task-irrlevant x number of features x group	5.883	1	5.883	0.049	0.826
Residual	11920.682	99	120.411		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	23229.109	1	323229.109	32.548	<.001
Residual	70655.977	99	713.697		

Note: Type III Sum of Squares

Appendix Table 4.15 | 2x2x2 ANOVA on the Reaction Time including whether the retrieval foil's feature was taken from the task-irrelevant versus non-filtered item, the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P

Non-filtered/task-irrelevant	0.581	1	0.581	1.430	0.235
Non-filtered/task-irrelevant x group	$6.266e^{-6}$	1	$6.266e^{-6}$	$1.542e^{-5}$	0.997
Residual	40.225	99	0.406		
Number of features	2.989	1	2.989	10.112	0.002
Number of features x group	0.037	1	0.037	0.125	0.724
Residual	29.259	99	0.296		
Non-filtered/task-irrelevant x number of features	0.203	1	0.203	0.780	0.379
Non-filtered/task-irrelevant x number of features x group	0.079	1	0.079	0.302	0.584
Residual	25.792	99	0.261		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	58.806	1	58.806	51.806	<.001
Residual	113.441	99	1.146		

Note: Type III Sum of Squares

Appendix Table 4.16 | 2x2x2 ANOVA on the Identification Accuracy including the filtering effect (set size two versus the filtering condition in which the non-filtered feature was presented in the foil), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Filtering	$1.118e^{-4}$	1	$1.118e^{-4}$	0.021	0.886
Filtering x group	0.003	1	0.003	0.477	0.492
Residual	0.537	99	0.005		
Number of features	0.0736	1	0.0736	12.189	<.001
Number of features x group	0.007	1	0.007	1.215	0.273
Residual	0.596	99	0.006		
Filtering x number of features	0.002	1	0.002	0.285	0.594
Filtering x number of features x group	0.002	1	0.002	0.416	0.521
Residual	0.527	99	0.005		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.063	1	0.063	2.501	0.117
Residual	2.480	99	0.025		

Note: Type III Sum of Squares

Appendix Table 4.17 | 2x2x2 ANOVA on the Localisation Performance including the filtering effect (set size two versus the filtering condition in which the non-filtered feature was presented in the foil), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Filtering	109.414	1	109.414	0.263	0.609
Filtering x group	682.834	1	682.834	1.640	0.203
Residual	41227.883	99	416.443		
Number of features	60.143	1	60.143	0.228	0.634
Number of features x group	1104.877	1	1104.877	4.196	0.043
Residual	26068.187	99	263.315		
Filtering x number of features	290.302	1	290.302	0.526	0.470
Filtering x number of features x group	14.088	1	14.088	0.026	0.873
Residual	54625.691	99	551.775		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	31260.995	1	31260.995	16.392	<.001
Residual	188797.909	99	1907.050		

Note: Type III Sum of Squares

Appendix Table 4.18 | 2x2x2 ANOVA on Misbinding including the filtering effect (set size two versus the filtering condition in which the non-filtered feature was presented in the foil), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Filtering	$2.373e^{-5}$	1	$2.373e^{-5}$	0.007	0.934
Filtering x group	$4.977e^{-4}$	1	$4.977e^{-4}$	0.143	0.706
Residual	0.345	99	0.003		
Number of features	0.005	1	0.005	2.209	0.140
Number of features x group	0.002	1	0.002	0.974	0.326
Residual	0.207	99	0.002		
Filtering x number of features	$1.831e^{-4}$	1	$1.831e^{-4}$	0.051	0.821
Filtering x number of features x group	0.001	1	0.001	0.375	0.542
Residual	0.354	99	0.004		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P

Group	0.019	1	0.019	2.838	0.095
Residual	0.652	99	0.007		

Note: Type III Sum of Squares

Appendix Table 4.19 | 2x2x2 ANOVA on Guessing including the filtering effect (set size two versus the filtering condition in which the non-filtered feature was presented in the foil), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Filtering	$2.561e^{-4}$	1	$2.561e^{-4}$	1.223	0.271
Filtering x group	$5.187e^{-4}$	1	$5.187e^{-4}$	2.477	0.119
Residual	0.021	99	$2.094e^{-4}$		
Number of features	$7.771e^{-4}$	1	$7.771e^{-4}$	4.412	0.038
Number of features x group	$1.468e^{-4}$	1	$1.468e^{-4}$	0.833	0.363
Residual	0.017	99	$1.761e^{-4}$		
Filtering x number of features	$5.273e^{-4}$	1	$5.273e^{-4}$	3.660	0.059
Filtering x number of features x group	$1.276e^{-4}$	1	$1.276e^{-4}$	0.886	0.349
Residual	0.014	99	$1.441e^{-4}$		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	0.027	1	0.027	10.064	0.002
Residual	0.262	99	0.003		

Note: Type III Sum of Squares

Appendix Table 4.20 | 2x2x2 ANOVA on the Imprecision including the filtering effect (set size two versus the filtering condition in which the non-filtered feature was presented in the foil), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Filtering	242.549	1	242.549	1.637	0.204
Filtering x group	1059.070	1	1059.070	7.150	0.009
Residual	14664.575	99	148.127		
Number of features	417.984	1	417.984	2.582	0.111
Number of features x group	246.370	1	246.370	1.522	0.220
Residual	16025.687	99	161.876		
Filtering x number of features	543.383	1	543.383	3.979	0.049
Filtering x number of features x group	120.042	1	120.042	0.879	0.351

Residual	13520.787	99	136.574		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	19033.339	1	19033.339	23.215	<.001
Residual	81166.894	99	819.868		

Note: Type III Sum of Squares

Appendix Table 4.21 | 2x2x2 ANOVA on the Reaction Time including the filtering effect (set size two versus the filtering condition in which the non-filtered feature was presented in the foil), the number of features in the retrieval foil that were taken from the original memory display (one versus three) and the between subject factor group (young versus elderly).

Within Subjects Effects	Sum of Squares	df	Mean Square	F	P
Filtering	0.002	1	0.002	0.014	0.907
Filtering x group	$6.532e^{-4}$	1	$6.532e^{-4}$	0.004	0.950
Residual	16.451	99	0.166		
Number of features	1.052	1	1.052	5.369	0.023
Number of features x group	0.009	1	0.009	0.045	0.833
Residual	19.398	99	0.196		
Filtering x number of features	0.064	1	0.064	0.264	0.608
Filtering x number of features x group	$3.025e^{-5}$	1	$3.025e^{-5}$	$1.255e^{-4}$	0.991
Residual	23.857	99	0.241		

Note: Type III Sum of Squares

Between Subjects Effects	Sum of Squares	df	Mean Square	F	P
Group	59.160	1	59.160	52.144	<.001
Residual	112.320	99	1.135		

Note: Type III Sum of Squares

Appendix Table 4.22 | Post Hoc Comparisons of the interaction of filtering at encoding and the number of features in Imprecision.

		Mean Difference	SE	t	p_{holm}
Filtering, One Feature	Filtering, Two Features	-0.191	2.374	-0.080	1.000
	Set Size 2, One Feature	5.212	2.394	2.177	0.149
	Set Size 2, Two Features	-1.950	2.457	-0.794	1.000
Filtering, Two Features	Set Size 2, One Feature	5.403	2.457	2.199	0.149
	Set Size 2, Tow Features	-1.760	2.394	-0.735	1.000

Appendix Table 4.22 | Post Hoc Comparisons of the interaction of filtering at encoding and the number of features in Imprecision.

		Mean Difference	SE	t	p_{holm}
Set Size 2, One Feature	Set Size 2, Two Features	-7.162	2.374	-3.017	0.019

Note. Bonferroni adjusted confidence intervals.

Experiment 2

Appendix Table 4.23 | 2x2 ANOVA on the Identification Accuracy including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
ignore	0.045	1	0.045	7.631	0.007
ignore ✻ group	0.012	1	0.012	1.965	0.164
Residual	0.581	99	0.006		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	0.008	1	0.008	1.297	0.258
Residual	0.646	99	0.007		

Note. Type III Sum of Squares

Appendix Table 4.24 | 2x2 ANOVA on the Localisation Performance including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
ignore	4361.103	1	4361.103	3.339	0.071
ignore ✻ group	1703.749	1	1703.749	1.305	0.256
Residual	129288.555	99	1305.945		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	2356.270	1	2356.270	0.457	0.501
Residual	510870.748	99	5160.311		

Note. Type III Sum of Squares

Appendix Table 4.25 | 2x2 ANOVA on Misbinding including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
ignore	0.646	1	0.646	69.888	< .001
ignore ✻ group	0.009	1	0.009	0.928	0.338
Residual	0.916	99	0.009		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	0.006	1	0.006	0.287	0.594
Residual	2.102	99	0.021		

Appendix Table 4.24 | 2x2 ANOVA on the Localisation Performance including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p

Note. Type III Sum of Squares

Appendix Table 4.26 | 2x2 ANOVA on Guessing including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
ignore	0.001	1	0.001	6.499	0.012
ignore ✻ group	9.492e-5	1	9.492e-5	0.550	0.460
Residual	0.017	99	1.726e-4		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	2.858e-5	1	2.858e-5	0.008	0.928
Residual	0.345	99	0.003		

Note. Type III Sum of Squares

Appendix Table 4.27 | 2x2 ANOVA on Imprecision including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
ignore	706.491	1	706.491	4.838	0.030
ignore ✻ group	55.902	1	55.902	0.383	0.538
Residual	14456.755	99	146.028		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	494.026	1	494.026	0.337	0.563
Residual	145298.790	99	1467.665		

Note. Type III Sum of Squares

Appendix Table 4.28 | 2x2 ANOVA on Reaction Time including the ignoring effect (ignore vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
ignore	1.179	1	1.179	2.435	0.122
ignore ✻ group	0.055	1	0.055	0.114	0.736
Residual	47.927	99	0.484		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	2.099	1	2.099	1.609	0.208
Residual	129.186	99	1.305		

Note. Type III Sum of Squares

Appendix Table 4.29 | 2x2 ANOVA on the Identification Accuracy including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update	0.003	1	0.003	0.872	0.353

Appendix Table 4.29 | 2x2 ANOVA on the Identification Accuracy including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update ✻ group	0.003	1	0.003	0.872	0.353
Residual	0.298	99	0.003		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	0.007	1	0.007	1.463	0.229
Residual	0.468	99	0.005		

Note. Type III Sum of Squares

Appendix Table 4.30 | 2x2 ANOVA on the Localisation Performance including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update	4664.274	1	4664.274	4.456	0.037
update ✻ group	98.207	1	98.207	0.094	0.760
Residual	103628.991	99	1046.757		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	3899.529	1	3899.529	1.001	0.319
Residual	385527.955	99	3894.222		

Note. Type III Sum of Squares

Appendix Table 4.31 | 2x2 ANOVA on Misbinding including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update	0.079	1	0.079	10.552	0.002
update ✻ group	6.857e-4	1	6.857e-4	0.091	0.763
Residual	0.742	99	0.007		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	0.026	1	0.026	2.195	0.142
Residual	1.163	99	0.012		

Note. Type III Sum of Squares

Appendix Table 4.32| 2x2 ANOVA on Guessing including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update	4.249e-5	1	4.249e-5	0.349	0.556
update ✻ group	1.266e-4	1	1.266e-4	1.041	0.310
Residual	0.012	99	1.217e-4		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	3.783e-5	1	3.783e-5	0.014	0.905

Appendix Table 4.32| 2x2 ANOVA on Guessing including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Residual	0.263	99	0.003		

Note. Type III Sum of Squares

Appendix Table 4.33| 2x2 ANOVA on Imprecision including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update	74.756	1	74.756	0.491	0.485
update ✻ group	183.912	1	183.912	1.208	0.274
Residual	15075.764	99	152.280		

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	95.498	1	95.498	0.077	0.782
Residual	123167.662	99	1244.118		

Note. Type III Sum of Squares

Appendix Table 4.34 | 2x2 ANOVA on the Reaction Time including the updating effect (updating vs maintain short) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
update	1.458	1	1.458	4.570	0.035
update ✻ group	0.113	1	0.113	0.355	0.552
Residual	31.580	99	0.319		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	0.150	1	0.150	0.169	0.682
Residual	87.521	99	0.884		

Note. Type III Sum of Squares

Appendix Table 4.35| 2x2 ANOVA on the Identification Accuracy including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Delay	0.006	1	0.006	2.311	0.132
Delay ✻ group	5.591e-4	1	5.591e-4	0.199	0.657
Residual	0.278	99	0.003		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	6.825e-5	1	6.825e-5	0.018	0.895
Residual	0.384	99	0.004		

Note. Type III Sum of Squares

Appendix Table 4.36| 2x2 ANOVA on the Localisation Performance including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Delay	46221.012	1	46221.012	53.941	< .001
Delay ✻ group	5066.179	1	5066.179	5.912	0.017
Residual	84831.089	99	856.880		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	347.480	1	347.480	0.105	0.747
Residual	327820.714	99	3311.320		

Note. Type III Sum of Squares

Appendix Table 4.37 | 2x2 ANOVA on Misbinding including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Delay	0.006	1	0.006	1.792	0.184
Delay ✻ group	0.032	1	0.032	9.902	0.002
Residual	0.319	99	0.003		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	6.483e-5	1	6.483e-5	0.008	0.931
Residual	0.855	99	0.009		

Note. Type III Sum of Squares

Appendix Table 4.38 | 2x2 ANOVA on Guessing including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Delay	0.010	1	0.010	52.538	< .001
Delay ✻ group	2.493e-5	1	2.493e-5	0.136	0.714
Residual	0.018	99	1.839e-4		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	1.019e-4	1	1.019e-4	0.033	0.857
Residual	0.308	99	0.003		

Note. Type III Sum of Squares

Appendix Table 4.39 | 2x2 ANOVA on Imprecision including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Delay	16114.717	1	16114.717	116.365	< .001
Delay ✻ group	10.143	1	10.143	0.073	0.787
Residual	13709.882	99	138.484		

Note. Type III Sum of Squares

Between Subjects Effects

Appendix Table 4.39 | 2x2 ANOVA on Imprecision including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
	Sum of Squares	df	Mean Square	F	p
group	703.235	1	703.235	0.582	0.447
Residual	119535.657	99	1207.431		

Note. Type III Sum of Squares

Appendix Table 4.40 | 2x2 ANOVA on the Reaction Time including the delay effect (maintain short vs maintain long) and the between subject factor group (young versus elderly).

	Sum of Squares	df	Mean Square	F	p
Delay	1.059	1	1.059	2.496	0.117
Delay ✻ group	0.339	1	0.339	0.798	0.374
Residual	42.002	99	0.424		

Note. Type III Sum of Squares

Between Subjects Effects

	Sum of Squares	df	Mean Square	F	p
group	0.399	1	0.399	0.360	0.550
Residual	109.731	99	1.108		

Note. Type III Sum of Squares

Simulations of the parameters Misbinding and Guessing and Parameter Recovery

For the purpose of demonstrating the reliability of our approach of calculating misbinding and guessing, we used synthetic data which we present below.

In a first step, we ask whether we can recover model parameters (**Appendix Figure 4.1A/B**). 10 trials for 100 simulated participants were generated as follows: Response types were drawn per subject for 10 trials from a categorical distribution comprising of target response, misbinding and guessing. The Misbinding prior was initially set to a proportion of 0.1 and then consecutively increased by 0.1 until it would reach a value of 1. The Guessing prior was kept fixed at 0 and the Target response was defined as Target = 1- (Misbinding + Guessing) for **Appendix Figure 4.1A**. This procedure was also repeated for guessing with an increase of 0.1 to 1 and Misbinding being fixed at 0, for **Appendix Figure 4.1B**. For each simulated trial, a random set of locations that appeared in the actual dataset in one condition was chosen. According to which response was chosen from the categorical distribution, one of three next steps would follow:

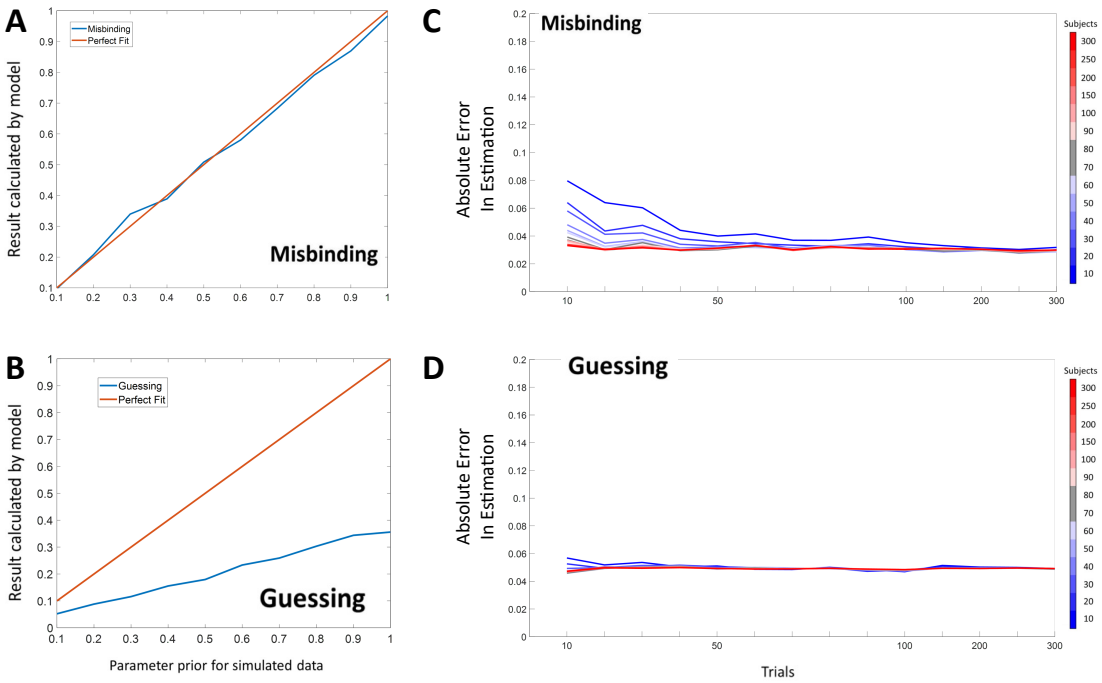
1) Target: The categorical distribution defined this trial to be a target response. An x- and y-response were drawn from distributions that were identical to the calculated distributions around the target's x- and y-coordinates in said condition of the actual data. Thus, a response around the target was simulated.
2) Misbinding: If the trial came out to be defined as a Misbinding trial, the same procedure as in 1) applied except for the response now being drawn from distributions around the non-target.
3) Guessing: If the trial came out to be a guess, the x- and y-coordinates were randomly drawn from random distributions around x and y.

The response and trial's stimulus locations were then run through our permutation model and the results compared to the values set for the simulation. A visual comparison of these can be found in the graph below (**Appendix Figure 4.1 A, B**).

The graph for guessing shows an obvious deviation between simulated input and calculated output with an increase of guessing. This is, however, expected as the permutations do not use random coordinates but coordinates that are actually part of the experiment and thus avoid unusual locations such as the screen centre or the screen edges which are locations that are, strategically, also avoided by a participant that fully forgot the correct location whatsoever.

In a second step, we ask how estimation error varies according to the number of participants and trials. The process described in 1 was repeated, however, Guessing and Misbinding were both fixed to a proportion of 0.1. The amount of trials and of participants were each varied from 10 in steps of 10 to 100 and from there in steps of 50 up to 300. Priors in the simulation were subtracted from the results extracted from the permutations method presented in the paper. The unsigned absolute value of this result was then calculated. This process was repeated 5000 times and averages for the differences calculated. The result was plotted for the different numbers of trials and subjects below (**Appendix Figure 4.1 C,D**).

The results of these simulations in the first and second step (**Appendix Figure 4.1**) suggest the following: In misbinding, there is no bias towards over- or underestimating true values but the second step shows that independently, each calculation will on average deviate about 0.03 from the true value. Thus, misbinding values of very small magnitude should be considered with the necessary levels of caution. Guessing does not follow the same pattern given that guessing in this study was based on the hypothesis that participants are unlikely to drag the response to the very centre or edge of the screen and are more likely to report locations that are likely to be part of the experiment.



Appendix Figure 4.1| Misbinding (A) and Guessing (B) Simulation Priors versus Results calculated by the model and the averaged absolute distance between measures and real data for Misbinding (C) and Guessing (D). Calculations for Misbinding on average land on the true value and do not show a particular bias towards an over- or underestimation of the true parameter. For guessing, the model presented here does not assume participants to make completely random guesses but rather assumes that they will most likely only move their chosen response stimulus to locations that could have possibly been presented in the task (i.e. not to the screen centre or the very edge of the screen). It is, thus, not surprising that the values deviate from one another. For Misbinding, each calculation will on average deviate from the true value by about 0.03 though all calculations together will land close to the true value. For Guessing, this value is at about 0.05. The absolute error on the y-axis of graphs C and D depicts the unsigned absolute difference between simulation priors and calculated results.

Appendix Table 4.41| Unpaired-samples T-tests and Mann-Whitney U-tests assessing the different effects manipulated in Experiments 1 and 2.

Independent Samples T-test	Test	Statistic	df	p
Identification _ Retrieval	Student	1.028	99.000	0.306
	Mann-Whitney	1362.500		0.320
Identification_ Set Size	Student	-0.774	99.000	0.441
	Mann-Whitney	1076.000		0.313
Identification_ Encoding	Student	-0.690	99.000	0.492
	Mann-Whitney	1133.000		0.537
Identification_ Maintenance	Student	-1.402	99.000	0.164
	Mann-Whitney	1131.500		0.503
Misbinding _ Retrieval	Student	0.238	99.000	0.812[a]
	Mann-Whitney	1213.000		0.964[a]
Misbinding _ Set Size	Student	2.274	99.000	0.025[a]
	Mann-Whitney	1511.000		0.044
Misbinding _ Encoding	Student	0.378	99.000	0.706
	Mann-Whitney	1226.000		0.968
Misbinding _ Maintenance	Student	0.963	99.000	0.338
	Mann-Whitney	1280.500		0.676
Imprecision _ Retrieval	Student	0.721	99.000	0.473
	Mann-Whitney	1314.000		0.516
Imprecision _ Set Size	Student	1.810	99.000	0.073[a]
	Mann-Whitney	1462.000		0.094[a]
Imprecision _ Encoding	Student	-2.674	99.000	0.009
	Mann-Whitney	759.000		0.001
Imprecision _ Maintenance	Student	0.619	99.000	0.538
	Mann-Whitney	1362.000		0.326

[a] Levene's test is significant (p<.05), suggesting violation of the equal variance assumption.

Appendix Table 4.42 | Correlation matrix for the effects of the number of features (numfeat), the set size, whether the foil's feature was taken from the filtered or non-filtered item (filtunfilt), filtering, ignoring, updating and delay on the proportion of trials in which participants identified the correct shape.

Pearson Correlations

		Encoding	Maintenance	Retrieval	Set size
Identification _Encoding	Pearson's r	—			
	p-value	—			
Identification _Maintenance	Pearson's r	-0.081	—		
	p-value	0.419	—		
Identification _Retrieval	Pearson's r	0.170	-0.122	—	
	p-value	0.089	0.224	—	
Identification _Set size	Pearson's r	0.526	-0.050	0.039	—
	p-value	< .001	0.616	0.701	—

Appendix Table 4.43 | Correlation matrix for the effects of the number of features (numfeat), the set size, whether the foil's feature was taken from the filtered or non-filtered item (filtunfilt), filtering, ignoring, updating and delay on misbinding.

Pearson Correlations

		Encoding	Maintenance	Retrieval	Set size
Misbinding _Encoding	Pearson's r	—			
	p-value	—			
Misbinding_Maintenance	Pearson's r	0.032	—		
	p-value	0.749	—		
Misbinding_Retrieval	Pearson's r	-0.029	-0.051	—	
	p-value	0.774	0.611	—	
Misbinding_Set size	Pearson's r	0.251	-0.015	-0.054	—
	p-value	0.011	0.883	0.589	—

Appendix Table 4.44 | Correlation matrix for the effects of the number of features (numfeat), the set size, whether the foil's feature was taken from the filtered or non-filtered item (filtunfilt), filtering, ignoring, updating and delay on imprecision.

Pearson Correlations

		Encoding	Maintenance	Retrieval	Set size
Imprecision_Encoding	Pearson's r	—			
	p-value	—			
Imprecision_Maintenance	Pearson's r	-0.050	—		
	p-value	0.620	—		
Imprecision_Retrieval	Pearson's r	-0.069	-0.143	—	
	p-value	0.490	0.154	—	
Imprecision_Set size	Pearson's r	0.438	-0.131	-0.087	—
	p-value	<0.001	0.191	0.388	—

Appendix Chapter 5. Hippocampal contributions to feature binding in working memory

Behavioural Analysis: Controls versus LE

Appendix Table 5.1 | Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of items	0.991	1	0.991	300.446	< .001	0.852
Number of items ✻ Group	0.006	1	0.006	1.842	0.181	0.034
Number of items ✻ Centered Years of full time education	4.923e-5	1	4.923e-5	0.015	0.903	2.869e-4
Number of items ✻ Centered Fluency	3.450e-4	1	3.450e-4	0.105	0.748	0.002
Residuals	0.172	52	0.003			
Delay	0.020	1	0.020	6.161	0.016	0.106
Delay ✻ Group	0.004	1	0.004	1.305	0.258	0.024
Delay ✻ Centered Years of full time education	4.984e-5	1	4.984e-5	0.016	0.901	2.998e-4
Delay ✻ Centered Fluency	0.001	1	0.001	0.424	0.518	0.008
Residuals	0.166	52	0.003			
Number of items ✻ Delay	6.002e-4	1	6.002e-4	0.231	0.633	0.004
Number of items ✻ Delay ✻ Group	0.007	1	0.007	2.614	0.112	0.048
Number of items ✻ Delay ✻ Centered Years of full time education	8.814e-6	1	8.814e-6	0.003	0.954	6.524e-5
Number of items ✻ Delay ✻ Centered Fluency	1.709e-5	1	1.709e-5	0.007	0.936	1.265e-4
Residuals	0.135	52	0.003			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.053	1	0.053	8.174	0.006	0.136
Centered Years of full time education	0.001	1	0.001	0.226	0.636	0.004
Centered Fluency	0.011	1	0.011	1.687	0.200	0.031
Residuals	0.335	52	0.006			

Note. Type III Sum of Squares

Appendix Table 5.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Group	0.013	0.148	12.991	1.000	
Number of items + Delay + Group + Centered Fluency	0.013	0.097	8.024	0.655	21.291
Number of items + Delay + Group + Number of items ✻ Group	0.013	0.079	6.402	0.533	8.328
Number of items + Delay + Group + Delay ✻ Group	0.013	0.057	4.555	0.388	3.954
Number of items + Group	0.013	0.044	3.455	0.298	2.953
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Group	0.013	0.041	3.210	0.278	37.403
Number of items + Delay + Group + Centered Years of full time education	0.013	0.039	3.036	0.264	4.941
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Group	0.013	0.036	2.766	0.241	4.223
Number of items + Delay + Group + Centered Fluency + Delay ✻ Group	0.013	0.033	2.593	0.226	22.171
Number of items + Delay + Group + Number of items ✻ Delay	0.013	0.032	2.513	0.220	3.799
Number of items + Delay + Group + Number of items ✻ Group + Delay ✻ Group	0.013	0.029	2.249	0.197	5.222
Number of items + Delay	0.013	0.026	2.036	0.179	3.505
Number of items + Group + Number of items ✻ Group	0.013	0.025	1.934	0.170	7.544
Number of items + Group + Centered Fluency	0.013	0.025	1.902	0.168	16.376
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency	0.013	0.024	1.869	0.165	4.425
Number of items + Group + Centered Years of full time education	0.013	0.018	1.377	0.122	35.665
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.016	1.233	0.110	4.061
Number of items + Delay + Group + Centered Years of full time education + Delay ✻ Group	0.013	0.016	1.204	0.107	12.861
Number of items + Delay + Group + Number of items ✻ Delay + Delay ✻ Group	0.013	0.015	1.161	0.103	21.805
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay	0.013	0.015	1.126	0.100	4.305
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Group + Delay ✻ Group	0.013	0.014	1.032	0.092	6.116
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group	0.013	0.013	0.984	0.088	5.238
Number of items + Delay + Centered Fluency	0.013	0.013	0.961	0.086	8.819
Number of items + Group + Centered Fluency + Number of items ✻ Group	0.013	0.010	0.768	0.069	3.584
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.013	0.009	0.661	0.059	3.680

Appendix Table 5.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay	0.013	0.009	0.644	0.058	4.479
Number of items	0.013	0.008	0.630	0.056	3.349
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.008	0.619	0.055	7.057
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Group + Delay ✻ Group	0.013	0.008	0.591	0.053	6.349
Number of items + Group + Centered Years of full time education + Centered Fluency	0.013	0.007	0.562	0.050	9.542
Number of items + Delay + Centered Years of full time education	0.013	0.007	0.494	0.044	3.192
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.006	0.484	0.043	4.328
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Delay ✻ Group	0.013	0.006	0.438	0.039	5.322
Number of items + Group + Centered Years of full time education + Number of items ✻ Group	0.013	0.006	0.432	0.039	5.447
Number of items + Delay + Number of items ✻ Delay	0.013	0.005	0.413	0.037	3.176
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay	0.013	0.005	0.402	0.036	7.044
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.005	0.340	0.031	5.992
Number of items + Delay + Centered Years of full time education + Centered Fluency	0.013	0.004	0.338	0.030	10.380
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group + Delay ✻ Group	0.013	0.004	0.331	0.030	3.638
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.004	0.331	0.030	5.682
Number of items + Centered Fluency	0.013	0.004	0.272	0.024	3.459
Number of items + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group	0.013	0.003	0.255	0.023	4.314
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Delay ✻ Group	0.013	0.003	0.229	0.021	4.376
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.003	0.224	0.020	5.719
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.003	0.212	0.019	6.161

Appendix Table 5.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Centered Fluency + Number of items ✻ Delay	0.013	0.003	0.192	0.017	3.839
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.002	0.182	0.016	17.355
Number of items + Centered Years of full time education	0.013	0.002	0.154	0.014	3.251
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Delay ✻ Group	0.013	0.002	0.140	0.013	3.769
Number of items + Delay + Centered Years of full time education + Number of items ✻ Delay	0.013	0.002	0.120	0.011	6.412
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.002	0.119	0.011	4.922
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.001	0.096	0.009	6.653
Number of items + Centered Years of full time education + Centered Fluency	0.013	0.001	0.095	0.009	3.201
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.001	0.077	0.007	7.327
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	9.280e -4	0.070	0.006	14.090
Number of items + Delay + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay	0.013	8.641e -4	0.065	0.006	3.197
Group	0.013	1.888e-42	1.416e-40	1.278e-41	2.842
Null model (incl. subject)	0.013	1.416e-42	1.062e-40	9.588e-42	2.697
Delay + Group	0.013	9.146e-43	6.859e-41	6.194e-42	3.339
Delay	0.013	6.656e-43	4.992e-41	4.508e-42	3.137
Group + Centered Fluency	0.013	5.295e-43	3.972e-41	3.587e-42	3.005
Group + Centered Years of full time education	0.013	4.091e-43	3.069e-41	2.771e-42	8.932
Centered Fluency	0.013	3.872e-43	2.904e-41	2.622e-42	2.812
Delay + Group + Centered Fluency	0.013	3.364e-43	2.523e-41	2.279e-42	20.503

Appendix Table 5.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Centered Years of full time education	0.013	2.762e-43	2.071e-41	1.870e-42	3.128
Delay + Centered Fluency	0.013	2.408e-43	1.806e-41	1.631e-42	23.760
Delay + Group + Delay ✻ Group	0.013	2.354e-43	1.765e-41	1.594e-42	3.760
Delay + Group + Centered Years of full time education	0.013	1.760e-43	1.320e-41	1.192e-42	3.911
Group + Centered Years of full time education + Centered Fluency	0.013	1.458e-43	1.094e-41	9.875e-43	3.122
Delay + Centered Years of full time education	0.013	1.267e-43	9.506e-42	8.585e-43	3.681
Centered Years of full time education + Centered Fluency	0.013	1.108e-43	8.307e-42	7.502e-43	3.027
Delay + Group + Centered Years of full time education + Centered Fluency	0.013	7.118e-44	5.338e-42	4.821e-43	3.442
Delay + Group + Centered Fluency + Delay ✻ Group	0.013	6.565e-44	4.924e-42	4.447e-43	5.405
Delay + Centered Years of full time education + Centered Fluency	0.013	5.057e-44	3.793e-42	3.425e-43	3.044
Delay + Group + Centered Years of full time education + Delay ✻ Group	0.013	4.312e-44	3.234e-42	2.920e-43	3.471
Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.013	1.788e-44	1.341e-42	1.211e-43	3.829

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of items	0.737	1.000	4.021e+14
Delay	0.737	0.846	1.964
Group	0.737	0.924	4.351
Centered Years of full time education	0.500	0.236	0.309
Centered Fluency	0.500	0.347	0.531
Number of items ✻ Delay	0.316	0.150	0.383
Number of items ✻ Group	0.316	0.320	1.020
Delay ✻ Group	0.316	0.217	0.601
Number of items ✻ Delay ✻ Group	0.053	0.009	0.164

Appendix Table 5.3 | Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of items	82.264	1	82.264	203.638	< .001	0.797
Number of items ✻ Group	0.008	1	0.008	0.021	0.886	4.006e-4
Number of items ✻ Centered Years of full time education	0.001	1	0.001	0.003	0.954	6.355e-5
Number of items ✻ Centered Fluency	0.011	1	0.011	0.027	0.870	5.179e-4
Residuals	21.007	52	0.404			
Delay	4.280	1	4.280	32.446	< .001	0.384
Delay ✻ Group	0.001	1	0.001	0.008	0.929	1.528e-4
Delay ✻ Centered Years of full time education	0.259	1	0.259	1.966	0.167	0.036
Delay ✻ Centered Fluency	7.124e-5	1	7.124e-5	5.401e-4	0.982	1.039e-5
Residuals	6.860	52	0.132			
Number of items ✻ Delay	0.159	1	0.159	1.599	0.212	0.030
Number of items ✻ Delay ✻ Group	0.179	1	0.179	1.802	0.185	0.033
Number of items ✻ Delay ✻ Centered Years of full time education	0.006	1	0.006	0.057	0.812	0.001
Number of items ✻ Delay ✻ Centered Fluency	0.084	1	0.084	0.846	0.362	0.016
Residuals	5.175	52	0.100			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	1.652	1	1.652	2.563	0.115	0.047
Centered Years of full time education	1.127	1	1.127	1.748	0.192	0.033
Centered Fluency	1.119	1	1.119	1.736	0.193	0.032
Residuals	33.511	52	0.644			

Note. Type III Sum of Squares

Appendix Table 5.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay	0.013	0.146	12.782	1.000	
Number of items + Delay + Group	0.013	0.097	8.084	0.668	5.455
Number of items + Delay + Centered Fluency	0.013	0.094	7.789	0.646	7.047
Number of items + Delay + Centered Years of full time education	0.013	0.084	6.905	0.579	5.603
Number of items + Delay + Group + Centered Years of full time education	0.013	0.079	6.435	0.543	20.693
Number of items + Delay + Group + Centered Fluency	0.013	0.062	4.940	0.424	5.309

Appendix Table 5.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Centered Years of full time education + Centered Fluency	0.013	0.058	4.615	0.398	4.507
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency	0.013	0.041	3.234	0.284	4.753
Number of items + Delay + Number of items ✻ Delay	0.013	0.041	3.168	0.278	8.289
Number of items + Delay + Centered Fluency + Number of items ✻ Delay	0.013	0.028	2.134	0.190	14.492
Number of items + Delay + Group + Number of items ✻ Delay	0.013	0.027	2.051	0.183	6.211
Number of items + Delay + Centered Years of full time education + Number of items ✻ Delay	0.013	0.025	1.961	0.175	6.302
Number of items + Delay + Group + Number of items ✻ Group	0.013	0.023	1.728	0.155	14.351
Number of items + Delay + Group + Delay ✻ Group	0.013	0.019	1.466	0.132	6.255
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay	0.013	0.019	1.462	0.131	6.842
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay	0.013	0.017	1.323	0.119	5.338
Number of items + Delay + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay	0.013	0.016	1.234	0.111	4.683
Number of items + Delay + Group + Centered Years of full time education + Delay ✻ Group	0.013	0.013	0.999	0.090	6.588
Number of items + Delay + Group + Centered Fluency + Delay ✻ Group	0.013	0.013	0.977	0.088	10.662
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Group	0.013	0.013	0.958	0.087	6.559
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Group	0.013	0.012	0.941	0.085	5.686
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay	0.013	0.011	0.851	0.077	4.979
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.013	0.009	0.660	0.060	5.390
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group	0.013	0.008	0.609	0.055	4.741
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.006	0.460	0.042	7.504
Number of items + Delay + Group + Number of items ✻ Delay + Delay ✻ Group	0.013	0.005	0.388	0.035	5.658
Number of items + Delay + Group + Number of items ✻ Group + Delay ✻ Group	0.013	0.004	0.294	0.027	8.945
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.003	0.261	0.024	7.419

Appendix Table 5.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.003	0.258	0.024	5.724
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Delay ✻ Group	0.013	0.003	0.256	0.023	6.168
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Delay ✻ Group	0.013	0.003	0.246	0.022	5.600
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Delay ✻ Group	0.013	0.003	0.209	0.019	12.168
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Group + Delay ✻ Group	0.013	0.003	0.196	0.018	8.213
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.002	0.181	0.017	6.481
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Group + Delay ✻ Group	0.013	0.002	0.171	0.016	5.136
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group + Delay ✻ Group	0.013	0.002	0.130	0.012	6.268
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.001	0.083	0.008	8.950
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	6.998e -4	0.053	0.005	6.101
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	6.681e -4	0.050	0.005	5.376
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	4.439e -4	0.033	0.003	5.525
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	4.256e -4	0.032	0.003	8.783
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	2.980e -4	0.022	0.002	9.917
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	2.393e -4	0.018	0.002	5.601
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻	0.013	1.885e -4	0.014	0.001	7.400

Appendix Table 5.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group					
Number of items	0.013	8.356e -5	0.006	5.739e -4	9.360
Number of items + Group	0.013	5.133e -5	0.004	3.525e -4	4.601
Number of items + Centered Fluency	0.013	4.538e -5	0.003	3.117e -4	4.643
Number of items + Centered Years of full time education	0.013	4.520e -5	0.003	3.104e -4	4.673
Number of items + Group + Centered Fluency	0.013	3.217e -5	0.002	2.209e -4	5.077
Number of items + Group + Centered Years of full time education	0.013	3.011e -5	0.002	2.068e -4	4.657
Number of items + Centered Years of full time education + Centered Fluency	0.013	3.011e -5	0.002	2.067e -4	4.495
Number of items + Group + Centered Years of full time education + Centered Fluency	0.013	2.162e -5	0.002	1.485e -4	4.692
Number of items + Group + Number of items ✻ Group	0.013	1.070e -5	8.029e -4	7.351e -5	7.203
Number of items + Group + Centered Years of full time education + Number of items ✻ Group	0.013	8.825e -6	6.619e -4	6.060e -5	31.048
Number of items + Group + Centered Fluency + Number of items ✻ Group	0.013	5.922e -6	4.441e -4	4.067e -5	4.622
Number of items + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group	0.013	4.667e -6	3.501e -4	3.205e -5	7.158
Delay	0.013	4.027e-46	3.020e-44	2.766e-45	4.529
Delay + Group	0.013	1.585e-46	1.189e-44	1.089e-45	5.021
Delay + Centered Fluency	0.013	1.467e-46	1.100e-44	1.008e-45	5.080
Delay + Centered Years of full time education	0.013	1.460e-46	1.095e-44	1.003e-45	4.701
Null model (incl. subject)	0.013	1.207e-46	9.054e-45	8.291e-46	4.324
Delay + Centered Years of full time education + Centered Fluency	0.013	6.353e-47	4.765e-45	4.363e-46	4.588
Delay + Group + Centered Years of full time education	0.013	5.598e-47	4.198e-45	3.844e-46	6.644
Delay + Group + Centered Fluency	0.013	5.483e-47	4.112e-45	3.766e-46	4.865
Group	0.013	4.421e-47	3.316e-45	3.036e-46	4.408
Centered Years of full time education	0.013	4.280e-47	3.210e-45	2.939e-46	5.074

Appendix Table 5.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Centered Fluency	0.013	4.094e-47	3.071e-45	2.812e-46	4.459
Delay + Group + Delay ✻ Group	0.013	3.079e-47	2.309e-45	2.115e-46	4.891
Delay + Group + Centered Years of full time education + Centered Fluency	0.013	2.496e-47	1.872e-45	1.714e-46	5.346
Centered Years of full time education + Centered Fluency	0.013	1.791e-47	1.344e-45	1.230e-46	4.505
Group + Centered Fluency	0.013	1.556e-47	1.167e-45	1.068e-46	4.888
Group + Centered Years of full time education	0.013	1.499e-47	1.124e-45	1.029e-46	4.483
Delay + Group + Centered Years of full time education + Delay ✻ Group	0.013	1.191e-47	8.935e-46	8.182e-47	7.468
Delay + Group + Centered Fluency + Delay ✻ Group	0.013	1.097e-47	8.224e-46	7.531e-47	5.206
Group + Centered Years of full time education + Centered Fluency	0.013	6.695e-48	5.021e-46	4.598e-47	4.515
Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.013	4.721e-48	3.541e-46	3.242e-47	5.132

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of items	0.737	1.000	∞
Delay	0.737	1.000	965.928
Group	0.737	0.508	0.369
Centered Years of full time education	0.500	0.395	0.653
Centered Fluency	0.500	0.388	0.633
Number of items ✻ Delay	0.316	0.218	0.605
Number of items ✻ Group	0.316	0.086	0.203
Delay ✻ Group	0.316	0.083	0.196
Number of items ✻ Delay ✻ Group	0.053	0.001	0.021

Appendix Table 4.5 | Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Number of items	637730.869	1	637730.869	434.897	< .001	0.893
Number of items ✻ Group	6688.335	1	6688.335	4.561	0.037	0.081
Number of items ✻ Centered Years of full time education	234.956	1	234.956	0.160	0.691	0.003
Number of items ✻ Centered Fluency	27.660	1	27.660	0.019	0.891	3.626e-4

Appendix Table 4.5 | Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	76252.591	52	1466.396			
Delay	12855.304	1	12855.304	25.911	< .001	0.333
Delay ✻ Group	285.533	1	285.533	0.576	0.452	0.011
Delay ✻ Centered Years of full time education	496.494	1	496.494	1.001	0.322	0.019
Delay ✻ Centered Fluency	191.674	1	191.674	0.386	0.537	0.007
Residuals	25798.969	52	496.134			
Number of items ✻ Delay	3782.992	1	3782.992	8.243	0.006	0.137
Number of items ✻ Delay ✻ Group	20.207	1	20.207	0.044	0.835	8.460e-4
Number of items ✻ Delay ✻ Centered Years of full time education	520.692	1	520.692	1.135	0.292	0.021
Number of items ✻ Delay ✻ Centered Fluency	0.950	1	0.950	0.002	0.964	3.982e-5
Residuals	23864.811	52	458.939			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	40394.726	1	40394.726	14.771	< .001	0.221
Centered Years of full time education	5.869	1	5.869	0.002	0.963	4.127e-5
Centered Fluency	1084.679	1	1084.679	0.397	0.532	0.008
Residuals	142210.432	52	2734.816			

Note. Type III Sum of Squares

Post Hoc Tests

Post Hoc Comparisons - Number of items ✻ Delay

		Mean Difference	SE	t	p_{holm}
X1.item., X1.second	X3.items, X1.second	-98.496	5.864	-16.798	< .001
	X1.item., X4.seconds	-6.932	4.130	-1.679	0.096
	X3.items, X4.seconds	-121.866	5.920	-20.586	< .001
X3.items, X1.second	X1.item., X4.seconds	91.564	5.920	15.467	< .001
	X3.items, X4.seconds	-23.370	4.130	-5.659	< .001
X1.item., X4.seconds	X3.items, X4.seconds	-114.934	5.864	-19.601	< .001

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Group

Appendix Table 5.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.270	27.680	1.000	

Appendix Table 5.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Group + Number of items ✻ Group	0.013	0.151	13.351	0.561	11.959
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.113	9.579	0.420	33.523
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.071	5.751	0.264	13.331
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.061	4.842	0.225	9.575
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Group	0.013	0.042	3.256	0.154	5.962
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Group	0.013	0.035	2.740	0.131	6.292
Number of items + Delay + Group + Number of items ✻ Group + Delay ✻ Group	0.013	0.031	2.365	0.113	5.883
Number of items + Delay + Group + Number of items ✻ Delay	0.013	0.028	2.168	0.104	8.077
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group	0.013	0.028	2.128	0.102	6.396
Number of items + Delay + Group + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.019	1.421	0.069	12.246
Number of items + Delay + Group	0.013	0.018	1.354	0.066	6.335
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.017	1.335	0.065	7.900
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group	0.013	0.015	1.123	0.055	5.887
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.013	0.995	0.049	6.163
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Group + Delay ✻ Group	0.013	0.010	0.762	0.037	6.830
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group	0.013	0.009	0.662	0.032	31.492
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Group + Delay ✻ Group	0.013	0.008	0.636	0.031	7.800
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay	0.013	0.008	0.627	0.031	6.586
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay	0.013	0.007	0.493	0.024	5.917

Appendix Table 5.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Delay + Group + Number of items ✻ Delay + Delay ✻ Group	0.013	0.006	0.461	0.023	5.931
Number of items + Delay + Group + Centered Fluency	0.013	0.005	0.369	0.018	5.800
Number of items + Delay + Group + Centered Years of full time education	0.013	0.005	0.347	0.017	8.788
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.004	0.338	0.017	6.662
Number of items + Delay + Group + Delay ✻ Group	0.013	0.004	0.309	0.015	6.301
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group + Delay ✻ Group	0.013	0.004	0.288	0.014	8.241
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.004	0.285	0.014	7.002
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay	0.013	0.003	0.208	0.010	5.805
Number of items + Delay + Group + Centered Fluency + Number of items ✻ Delay + Delay ✻ Group	0.013	0.002	0.153	0.008	8.710
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency	0.013	0.002	0.145	0.007	6.072
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Number of items ✻ Group + Delay ✻ Group + Number of items ✻ Delay ✻ Group	0.013	0.002	0.123	0.006	6.959
Number of items + Delay + Group + Centered Years of full time education + Number of items ✻ Delay + Delay ✻ Group	0.013	0.002	0.120	0.006	7.196
Number of items + Delay + Group + Centered Fluency + Delay ✻ Group	0.013	0.001	0.086	0.004	6.421
Number of items + Delay + Group + Centered Years of full time education + Delay ✻ Group	0.013	9.945e -4	0.075	0.004	7.507
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay + Delay ✻ Group	0.013	7.474e -4	0.056	0.003	13.341
Number of items + Group + Number of items ✻ Group	0.013	7.402e -4	0.056	0.003	9.364
Number of items + Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.013	5.016e -4	0.038	0.002	12.850
Number of items + Delay + Number of items ✻ Delay	0.013	3.096e -4	0.023	0.001	6.328
Number of items + Delay	0.013	2.006e -4	0.015	7.442e -4	8.826
Number of items + Group + Centered Fluency + Number of items ✻ Group	0.013	1.894e -4	0.014	7.027e -4	7.029

Appendix Table 5.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Number of items + Group + Centered Years of full time education + Number of items ✻ Group	0.013	1.578e -4	0.012	5.855e -4	5.945
Number of items + Group	0.013	1.076e -4	0.008	3.993e -4	5.625
Number of items + Delay + Centered Fluency + Number of items ✻ Delay	0.013	9.340e -5	0.007	3.465e -4	8.976
Number of items + Delay + Centered Years of full time education + Number of items ✻ Delay	0.013	7.916e -5	0.006	2.937e -4	7.554
Number of items + Group + Centered Years of full time education + Centered Fluency + Number of items ✻ Group	0.013	7.110e -5	0.005	2.637e -4	9.578
Number of items + Delay + Centered Years of full time education + Centered Fluency + Number of items ✻ Delay	0.013	6.238e -5	0.005	2.314e -4	42.172
Number of items + Delay + Centered Fluency	0.013	5.681e -5	0.004	2.107e -4	5.764
Number of items + Delay + Centered Years of full time education	0.013	4.882e -5	0.004	1.811e -4	5.870
Number of items + Group + Centered Fluency	0.013	3.007e -5	0.002	1.115e -4	5.708
Number of items + Group + Centered Years of full time education	0.013	2.850e -5	0.002	1.057e -4	11.922
Number of items + Delay + Centered Years of full time education + Centered Fluency	0.013	2.180e -5	0.002	8.087e -5	5.766
Number of items + Group + Centered Years of full time education + Centered Fluency	0.013	1.043e -5	7.826e -4	3.871e -5	5.512
Number of items	0.013	1.183e -6	8.870e -5	4.387e -6	5.482
Number of items + Centered Fluency	0.013	3.543e -7	2.657e -5	1.314e -6	5.783
Number of items + Centered Years of full time education	0.013	3.067e -7	2.300e -5	1.138e -6	5.566
Number of items + Centered Years of full time education + Centered Fluency	0.013	1.369e -7	1.026e -5	5.077e -7	5.640
Group	0.013	2.969e-67	2.227e-65	1.101e-66	6.783
Delay + Group	0.013	1.906e-67	1.429e-65	7.069e-67	5.621
Null model (incl. subject)	0.013	7.001e-68	5.251e-66	2.597e-67	5.372
Group + Centered Fluency	0.013	5.209e-68	3.907e-66	1.932e-67	5.554
Group + Centered Years of full time education	0.013	4.790e-68	3.593e-66	1.777e-67	5.583
Delay	0.013	4.480e-68	3.360e-66	1.662e-67	5.671
Delay + Group + Delay ✻ Group	0.013	3.994e-68	2.996e-66	1.482e-67	6.165

Appendix Table 5.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + Group + Centered Fluency	0.013	3.523e-68	2.642e-66	1.307e-67	5.868
Delay + Group + Centered Years of full time education	0.013	3.408e-68	2.556e-66	1.264e-67	8.323
Group + Centered Years of full time education + Centered Fluency	0.013	1.390e-68	1.043e-66	5.156e-68	5.532
Centered Fluency	0.013	1.302e-68	9.768e-67	4.831e-68	5.523
Centered Years of full time education	0.013	1.224e-68	9.180e-67	4.540e-68	5.683
Delay + Group + Centered Years of full time education + Centered Fluency	0.013	1.113e-68	8.349e-67	4.129e-68	19.546
Delay + Centered Fluency	0.013	8.508e-69	6.381e-67	3.156e-68	5.810
Delay + Centered Years of full time education	0.013	7.548e-69	5.661e-67	2.800e-68	5.577
Delay + Group + Centered Fluency + Delay ✻ Group	0.013	6.859e-69	5.145e-67	2.544e-68	5.833
Delay + Group + Centered Years of full time education + Delay ✻ Group	0.013	6.571e-69	4.928e-67	2.438e-68	5.930
Centered Years of full time education + Centered Fluency	0.013	3.639e-69	2.729e-67	1.350e-68	7.244
Delay + Centered Years of full time education + Centered Fluency	0.013	2.265e-69	1.699e-67	8.403e-69	6.114
Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.013	1.943e-69	1.457e-67	7.207e-69	7.816

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Number of items	0.737	1.000	4.233e+13
Delay	0.737	0.999	266.721
Group	0.737	0.999	408.003
Centered Years of full time education	0.500	0.208	0.263
Centered Fluency	0.500	0.266	0.363
Number of items ✻ Delay	0.316	0.667	4.336
Number of items ✻ Group	0.316	0.907	21.096
Delay ✻ Group	0.316	0.199	0.537
Number of items ✻ Delay ✻ Group	0.053	0.028	0.528

Mixture model Analysis: Controls versus LE

Appendix Table 5.7 | Repeated Measures ANOVA on Proportion of Misbinding with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay	0.054	1	0.054	10.591	0.002	0.169
Delay ✻ Group	0.002	1	0.002	0.364	0.549	0.007
Delay ✻ Centered Years of full time education	0.042	1	0.042	8.268	0.006	0.137
Delay ✻ Centered Fluency	0.006	1	0.006	1.172	0.284	0.022
Residuals	0.266	52	0.005			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.108	1	0.108	6.528	0.014	0.112
Centered Years of full time education	0.005	1	0.005	0.289	0.593	0.006
Centered Fluency	5.057e-4	1	5.057e-4	0.031	0.862	5.874e-4
Residuals	0.860	52	0.017			

Note. Type III Sum of Squares

Appendix Table 5.8 | Bayesian Repeated Measures ANOVA on Proportion of Misbinding with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + Group	0.050	0.347	10.105	1.000	
Delay + Group + Centered Years of full time education	0.050	0.110	2.358	0.318	2.686
Delay + Group + Delay ✻ Group	0.050	0.100	2.111	0.288	2.881
Delay + Group + Centered Fluency	0.050	0.098	2.056	0.281	2.476
Delay	0.050	0.077	1.588	0.222	2.270
Delay + Group + Centered Years of full time education + Centered Fluency	0.050	0.049	0.974	0.140	2.278
Delay + Group + Centered Years of full time education + Delay ✻ Group	0.050	0.042	0.837	0.122	14.978
Group	0.050	0.030	0.594	0.087	2.306
Delay + Group + Centered Fluency + Delay ✻ Group	0.050	0.030	0.593	0.087	3.999
Delay + Centered Years of full time education	0.050	0.028	0.552	0.081	4.879
Delay + Centered Fluency	0.050	0.024	0.473	0.070	2.616
Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.050	0.016	0.305	0.045	3.719
Delay + Centered Years of full time education + Centered Fluency	0.050	0.013	0.241	0.036	2.843
Group + Centered Years of full time education	0.050	0.010	0.187	0.028	2.359
Group + Centered Fluency	0.050	0.009	0.167	0.025	2.378
Null model (incl. subject)	0.050	0.007	0.137	0.021	1.750
Group + Centered Years of full time education + Centered Fluency	0.050	0.004	0.079	0.012	2.571

Appendix Table 5.8 | Bayesian Repeated Measures ANOVA on Proportion of Misbinding with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Centered Years of full time education	0.050	0.002	0.045	0.007	2.124
Centered Fluency	0.050	0.002	0.041	0.006	1.976
Centered Years of full time education + Centered Fluency	0.050	0.001	0.020	0.003	1.827

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Delay	0.600	0.934	9.496
Group	0.600	0.845	3.639
Centered Years of full time education	0.500	0.275	0.380
Centered Fluency	0.500	0.245	0.325
Delay ✻ Group	0.200	0.188	0.928

Appendix Table *5.9* | Repeated Measures ANOVA on Proportion of Guessing with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay	4.792e-4	1	4.792e-4	1.125	0.294	0.021
Delay ✻ Group	2.022e-4	1	2.022e-4	0.475	0.494	0.009
Delay ✻ Centered Years of full time education	0.001	1	0.001	2.452	0.123	0.045
Delay ✻ Centered Fluency	0.003	1	0.003	6.502	0.014	0.111
Residuals	0.022	52	4.259e-4			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.002	1	0.002	2.637	0.110	0.048
Centered Years of full time education	1.091e-4	1	1.091e-4	0.150	0.700	0.003
Centered Fluency	6.389e-5	1	6.389e-5	0.088	0.768	0.002
Residuals	0.038	52	7.281e-4			

Note. Type III Sum of Squares

Appendix Table 5.10 | Bayesian Repeated Measures ANOVA on Proportion of Guessing with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.260	6.678	1.000	
Group	0.050	0.191	4.488	0.735	1.857
Delay	0.050	0.083	1.722	0.319	0.945

Appendix Table 5.10 | Bayesian Repeated Measures ANOVA on Proportion of Guessing with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Centered Years of full time education	0.050	0.068	1.386	0.261	1.039
Centered Fluency	0.050	0.067	1.361	0.257	0.892
Delay + Group	0.050	0.060	1.220	0.232	1.358
Group + Centered Years of full time education	0.050	0.050	1.009	0.194	1.893
Group + Centered Fluency	0.050	0.049	0.986	0.190	2.386
Centered Years of full time education + Centered Fluency	0.050	0.027	0.529	0.104	1.128
Delay + Centered Fluency	0.050	0.023	0.439	0.087	2.042
Delay + Centered Years of full time education	0.050	0.022	0.424	0.084	1.369
Delay + Group + Delay ✻ Group	0.050	0.020	0.384	0.076	2.095
Group + Centered Years of full time education + Centered Fluency	0.050	0.019	0.363	0.072	1.075
Delay + Group + Centered Years of full time education	0.050	0.017	0.320	0.064	3.138
Delay + Group + Centered Fluency	0.050	0.015	0.299	0.060	2.456
Delay + Centered Years of full time education + Centered Fluency	0.050	0.009	0.175	0.035	2.479
Delay + Group + Centered Years of full time education + Centered Fluency	0.050	0.006	0.121	0.024	3.205
Delay + Group + Centered Fluency + Delay ✻ Group	0.050	0.005	0.102	0.021	3.348
Delay + Group + Centered Years of full time education + Delay ✻ Group	0.050	0.005	0.100	0.020	3.237
Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.050	0.003	0.052	0.010	22.915

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Delay	0.600	0.268	0.245
Group	0.600	0.441	0.527
Centered Years of full time education	0.500	0.226	0.292
Centered Fluency	0.500	0.224	0.288
Delay ✻ Group	0.200	0.033	0.137

Appendix Table 5.11 | Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay	73.984	1	73.984	1.364	0.248	0.026

Appendix Table 5.11 | Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay ✻ Group	12.645	1	12.645	0.233	0.631	0.004
Delay ✻ Centered Years of full time education	35.953	1	35.953	0.663	0.419	0.013
Delay ✻ Centered Fluency	2.831	1	2.831	0.052	0.820	0.001
Residuals	2820.971	52	54.249			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	801.708	1	801.708	2.405	0.127	0.044
Centered Years of full time education	19.139	1	19.139	0.057	0.812	0.001
Centered Fluency	56.636	1	56.636	0.170	0.682	0.003
Residuals	17332.970	52	333.326			

Note. Type III Sum of Squares

Appendix Table 5.12 | Bayesian Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.171	3.917	1.000	
Group	0.050	0.156	3.506	0.911	1.318
Delay	0.050	0.075	1.544	0.440	15.188
Centered Fluency	0.050	0.074	1.516	0.432	0.754
Centered Years of full time education	0.050	0.072	1.467	0.419	0.789
Group + Centered Fluency	0.050	0.070	1.432	0.410	2.429
Group + Centered Years of full time education	0.050	0.066	1.340	0.385	2.264
Delay + Group	0.050	0.060	1.204	0.349	3.014
Group + Centered Years of full time education + Centered Fluency	0.050	0.042	0.830	0.245	2.787
Centered Years of full time education + Centered Fluency	0.050	0.040	0.799	0.236	2.623
Delay + Centered Fluency	0.050	0.028	0.543	0.163	1.635
Delay + Centered Years of full time education	0.050	0.027	0.528	0.158	1.602
Delay + Group + Centered Fluency	0.050	0.025	0.489	0.147	2.034
Delay + Group + Centered Years of full time education	0.050	0.024	0.471	0.142	2.097
Delay + Group + Delay ✻ Group	0.050	0.018	0.348	0.105	2.997
Delay + Centered Years of full time education + Centered Fluency	0.050	0.016	0.305	0.093	3.152
Delay + Group + Centered Years of full time education + Centered Fluency	0.050	0.015	0.288	0.087	3.514
Delay + Group + Centered Years of full time education + Delay ✻ Group	0.050	0.009	0.172	0.053	17.280

Appendix Table 5.12 | Bayesian Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factor, group (controls versus LE patients) as "between-subjects" and Years of full time education and Fluency as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + Group + Centered Fluency + Delay ✻ Group	0.050	0.008	0.161	0.049	3.513
Delay + Group + Centered Years of full time education + Centered Fluency + Delay ✻ Group	0.050	0.005	0.086	0.026	4.280

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Delay	0.600	0.310	0.299
Group	0.600	0.497	0.660
Centered Years of full time education	0.500	0.315	0.460
Centered Fluency	0.500	0.323	0.477
Delay ✻ Group	0.200	0.040	0.166

Imaging Analysis

Appendix Table 5.13| Descriptive statistics on structural imaging results for both hippocampus and hippocampus subfields.

	Bilateral_Hipp_HSA		Right_Hipp_HSA		Left_Hipp_HSA	
	Controls	LE	Controls	LE	Controls	LE
Valid	28	13	28	13	28	13
Missing	0	15	0	15	0	15
Mean	7594.614	6687.217	3837.907	3353.507	3756.706	3333.710
SD	621.933	1390.022	317.984	770.705	445.110	748.228
Minimum	6129.878	4231.952	2983.338	2194.101	2716.548	2037.852
Maximum	8669.709	9375.801	4467.126	4669.050	4461.950	4706.751

	Right_Hippocampal_tail_HSA		Left_Hippocampal_tail_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	514.496	445.594	509.280	464.155
SD	42.051	59.540	50.045	59.656
Minimum	448.121	365.598	441.997	399.497
Maximum	647.827	568.490	661.841	595.909

	right_parasubiculum_HSA		left_parasubiculum_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	58.546	61.349	64.472	60.650
SD	9.650	9.819	12.935	14.447
Minimum	35.354	49.562	40.287	48.450
Maximum	76.314	82.435	95.493	86.750

	right_presubiculum_HSA		left_presubiculum_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	263.083	229.165	288.348	253.953
SD	34.148	38.518	34.709	45.285
Minimum	191.629	160.785	214.564	166.649
Maximum	326.968	296.588	363.704	306.369

	right_subiculum_HSA		left_subiculum_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	434.670	390.694	439.550	409.589
SD	40.805	66.006	44.419	64.160
Minimum	321.031	266.276	351.414	288.252
Maximum	498.111	494.945	541.120	500.648

	right_CA1_HSA		left_CA1_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	695.963	637.387	658.522	621.245
SD	59.879	126.286	59.736	133.037
Minimum	590.854	444.787	528.641	317.436
Maximum	828.377	813.600	774.210	822.811

	right_CA3_HSA		left_CA3_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	228.364	211.221	202.034	203.013
SD	29.353	42.466	27.699	40.087
Minimum	176.604	154.596	146.577	135.017
Maximum	274.947	290.054	243.863	253.475

	right_CA4_HSA		left_CA4_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	264.205	241.380	252.622	240.852
SD	18.586	44.624	17.118	36.476
Minimum	234.008	176.983	208.843	176.775
Maximum	294.258	312.022	283.245	290.106

	right_HATA_HSA		left_HATA_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13

Missing	0	15	0	15
Mean	63.385	63.723	61.548	60.389
SD	9.396	12.084	8.493	10.641
Minimum	48.766	50.486	40.242	38.066
Maximum	88.655	90.613	76.379	73.233

	right_GC-ML-DG_HSA		left_GC-ML-DG_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	305.305	274.162	293.310	278.704
SD	22.641	51.633	21.370	45.481
Minimum	269.476	196.587	237.981	190.901
Maximum	349.445	352.239	340.918	335.361

	right_molecular_layer_HP_HSA		left_molecular_layer_HP_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	475.110	433.869	469.789	443.789
SD	34.898	66.835	47.261	53.389
Minimum	431.439	301.460	377.691	353.839
Maximum	569.287	544.331	581.388	542.530

	right_fimbria_HSA		left_fimbria_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	64.163	48.007	80.937	61.141
SD	20.240	18.293	22.182	24.291
Minimum	30.435	19.379	32.474	21.928
Maximum	110.457	68.717	120.886	97.253

	right_hippocampal-fissure_HSA		left_hippocampal-fissure_HSA	
	Controls	LE	Controls	LE
Valid	28	13	28	13
Missing	0	15	0	15
Mean	148.897	162.828	143.788	160.188
SD	20.508	34.215	21.218	31.439
Minimum	124.540	124.741	92.988	113.467
Maximum	215.454	225.711	190.614	212.261

Appendix Table 5.14 | Independent Samples T-test on hippocampus and hippocampus subfields volumes, between healthy elderly controls and limbic encephalitis' patients.

	t	df	p
Bilateral_Hipp_HSA	2.912	39	0.006 [a]
Right_Hipp_HSA	2.871	39	0.007 [a]
Left_Hipp_HSA	2.266	39	0.029 [a]
right_Hippocampal_tail_HSA	4.267	39	< .001
left_Hippocampal_tail_HSA	2.528	39	0.016

Appendix Table 5.14 | Independent Samples T-test on hippocampus and hippocampus subfields volumes, between healthy elderly controls and limbic encephalitis' patients.

	t	df	p
right_parasubiculum_HSA	-0.861	39	0.395
left_parasubiculum_HSA	0.849	39	0.401
right_presubiculum_HSA	2.843	39	0.007
left_presubiculum_HSA	2.677	39	0.011
right_subiculum_HSA	2.624	39	0.012 [a]
left_subiculum_HSA	1.740	39	0.090
right_CA1_HSA	2.030	39	0.049 [a]
left_CA1_HSA	1.248	39	0.219 [a]
right_CA3_HSA	1.505	39	0.140 [a]
left_CA3_HSA	-0.091	39	0.928
right_CA4_HSA	2.330	39	0.025 [a]
left_CA4_HSA	1.417	39	0.164 [a]
right_HATA_HSA	-0.098	39	0.923
left_HATA_HSA	0.375	39	0.710
right_GC-ML-DG_HSA	2.707	39	0.010 [a]
left_GC-ML-DG_HSA	1.410	39	0.166 [a]
right_molecular_layer_HP_HSA	2.609	39	0.013 [a]
left_molecular_layer_HP_HSA	1.574	39	0.124
right_fimbria_HSA	2.448	39	0.019
left_fimbria_HSA	2.581	39	0.014
right_hippocampal-fissure_HSA	-1.626	39	0.112 [a]
left_hippocampal-fissure_HSA	-1.969	39	0.056

Note. Student's t-test.

[a] Levene's test is significant ($p < .05$), suggesting a violation of the equal variance assumption

Appendix Table 5.15 | Bayesian Independent Samples T-test on hippocampus and hippocampus subfields volumes, between healthy elderly controls and limbic encephalitis' patients.

	BF_{10}	error %
Bilateral_Hipp_HSA	7.422	7.363e-5
Right_Hipp_HSA	6.835	8.139e-5
Left_Hipp_HSA	2.250	8.612e-4
right_Hippocampal_tail_HSA	176.849	1.312e-7
left_Hippocampal_tail_HSA	3.546	4.866e-6
right_parasubiculum_HSA	0.431	0.010
left_parasubiculum_HSA	0.428	0.010
right_presubiculum_HSA	6.463	8.569e-5
left_presubiculum_HSA	4.683	7.362e-5
right_subiculum_HSA	4.234	5.044e-5
left_subiculum_HSA	1.031	0.004
right_CA1_HSA	1.551	0.003
left_CA1_HSA	0.591	0.009
right_CA3_HSA	0.773	0.011
left_CA3_HSA	0.324	0.008
right_CA4_HSA	2.506	4.819e-4
left_CA4_HSA	0.701	0.010
right_HATA_HSA	0.325	0.008
left_HATA_HSA	0.342	0.008
right_GC-ML-DG_HSA	4.954	8.209e-5

Appendix Table 5.15 | Bayesian Independent Samples T-test on hippocampus and hippocampus subfields volumes, between healthy elderly controls and limbic encephalitis' patients.

	BF_{10}	error %
left_GC-ML-DG_HSA	0.696	0.010
right_molecular_layer_HP_HSA	4.120	4.259e-5
left_molecular_layer_HP_HSA	0.838	0.013
right_fimbria_HSA	3.075	9.007e-5
left_fimbria_HSA	3.909	2.655e-5
right_hippocampal-fissure_HSA	0.893	0.014
left_hippocampal-fissure_HSA	1.416	0.004

Appendix Table 5.16 | Pearson Correlations between misbinding, guessing and hippocampus and hippocampus subfields volumes.

Variable		Mean_MisbMCMC	Mean_GuessMCMC
Bilateral_Hipp_HSA	Pearson's r	-0.406 **	-0.284
	p-value	0.008	0.072
Right_Hipp_HSA	Pearson's r	-0.262	-0.215
	p-value	0.098	0.176
Left_Hipp_HSA	Pearson's r	-0.457 **	-0.290
	p-value	0.003	0.066
right_Hippocampal_tail_HSA	Pearson's r	-0.110	-0.214
	p-value	0.495	0.180
left_Hippocampal_tail_HSA	Pearson's r	-0.215	-0.152
	p-value	0.177	0.343
right_parasubiculum_HSA	Pearson's r	-0.055	-0.097
	p-value	0.733	0.545
left_parasubiculum_HSA	Pearson's r	-0.184	-0.068
	p-value	0.249	0.674
right_presubiculum_HSA	Pearson's r	-0.092	-0.231
	p-value	0.569	0.147
left_presubiculum_HSA	Pearson's r	-0.113	-0.121
	p-value	0.482	0.451
right_subiculum_HSA	Pearson's r	-0.007	-0.118
	p-value	0.966	0.464
left_subiculum_HSA	Pearson's r	-0.161	-0.157
	p-value	0.314	0.325
right_CA1_HSA	Pearson's r	-0.067	-0.107
	p-value	0.676	0.507
left_CA1_HSA	Pearson's r	-0.291	-0.226
	p-value	0.065	0.154
right_CA3_HSA	Pearson's r	-0.152	-0.158
	p-value	0.342	0.324
left_CA3_HSA	Pearson's r	-0.290	-0.155
	p-value	0.066	0.333
right_CA4_HSA	Pearson's r	-0.233	-0.124
	p-value	0.143	0.440
left_CA4_HSA	Pearson's r	-0.322 *	-0.120
	p-value	0.040	0.456
right_HATA_HSA	Pearson's r	0.133	-0.147
	p-value	0.408	0.358
left_HATA_HSA	Pearson's r	-0.038	-0.013

Appendix Table 5.16 | Pearson Correlations between misbinding, guessing and hippocampus and hippocampus subfields volumes.

Variable		Mean_MisbMCMC	Mean_GuessMCMC
	p-value	0.814	0.935
right_GC-ML-DG_HSA	Pearson's r	-0.228	-0.175
	p-value	0.151	0.274
left_GC-ML-DG_HSA	Pearson's r	-0.228	-0.095
	p-value	0.152	0.555
right_molecular_layer_HP_HSA	Pearson's r	-0.084	-0.074
	p-value	0.603	0.644
left_molecular_layer_HP_HSA	Pearson's r	-0.334 *	-0.152
	p-value	0.033	0.342
right_fimbria_HSA	Pearson's r	0.037	-0.267
	p-value	0.820	0.091
left_fimbria_HSA	Pearson's r	-0.163	-0.288
	p-value	0.308	0.068
right_hippocampal-fissure_HSA	Pearson's r	0.126	0.248
	p-value	0.432	0.118
left_hippocampal-fissure_HSA	Pearson's r	0.018	0.290
	p-value	0.913	0.066

* p < .05, ** p < .01, *** p < .001

Appendix Table 5.17 | Bayesian Pearson Correlations between misbinding, guessing and hippocampus and hippocampus subfields volumes.

Variable		Mean_MisbMCMC	Mean_GuessMCMC
Bilateral_Hipp_HSA	Pearson's r	-0.406	-0.284
	BF_{10}	5.514	0.925
Right_Hipp_HSA	Pearson's r	-0.262	-0.215
	BF_{10}	0.731	0.471
Left_Hipp_HSA	Pearson's r	-0.457 *	-0.290
	BF_{10}	15.300	0.992
right_Hippocampal_tail_HSA	Pearson's r	-0.110	-0.214
	BF_{10}	0.244	0.464
left_Hippocampal_tail_HSA	Pearson's r	-0.215	-0.152
	BF_{10}	0.469	0.300
right_parasubiculum_HSA	Pearson's r	-0.055	-0.097
	BF_{10}	0.206	0.232
left_parasubiculum_HSA	Pearson's r	-0.184	-0.068
	BF_{10}	0.370	0.212
right_presubiculum_HSA	Pearson's r	-0.092	-0.231
	BF_{10}	0.228	0.538
left_presubiculum_HSA	Pearson's r	-0.113	-0.121
	BF_{10}	0.247	0.256
right_subiculum_HSA	Pearson's r	-0.007	-0.118
	BF_{10}	0.195	0.252
left_subiculum_HSA	Pearson's r	-0.161	-0.157
	BF_{10}	0.318	0.310
right_CA1_HSA	Pearson's r	-0.067	-0.107
	BF_{10}	0.212	0.241
left_CA1_HSA	Pearson's r	-0.291	-0.226

Appendix Table 5.17 | Bayesian Pearson Correlations between misbinding, guessing and hippocampus and hippocampus subfields volumes.

Variable		Mean_MisbMCMC	Mean_GuessMCMC
	BF_{10}	1.007	0.518
right_CA3_HSA	Pearson's r	-0.152	-0.158
	BF_{10}	0.301	0.311
left_CA3_HSA	Pearson's r	-0.290	-0.155
	BF_{10}	0.997	0.306
right_CA4_HSA	Pearson's r	-0.233	-0.124
	BF_{10}	0.548	0.260
left_CA4_HSA	Pearson's r	-0.322	-0.120
	BF_{10}	1.484	0.255
right_HATA_HSA	Pearson's r	0.133	-0.147
	BF_{10}	0.271	0.293
left_HATA_HSA	Pearson's r	-0.038	-0.013
	BF_{10}	0.200	0.195
right_GC-ML-DG_HSA	Pearson's r	-0.228	-0.175
	BF_{10}	0.527	0.347
left_GC-ML-DG_HSA	Pearson's r	-0.228	-0.095
	BF_{10}	0.525	0.230
right_molecular_layer_HP_HSA	Pearson's r	-0.084	-0.074
	BF_{10}	0.222	0.216
left_molecular_layer_HP_HSA	Pearson's r	-0.334	-0.152
	BF_{10}	1.756	0.301
right_fimbria_HSA	Pearson's r	0.037	-0.267
	BF_{10}	0.199	0.774
left_fimbria_HSA	Pearson's r	-0.163	-0.288
	BF_{10}	0.322	0.969
right_hippocampal-fissure_HSA	Pearson's r	0.126	0.248
	BF_{10}	0.262	0.635
left_hippocampal-fissure_HSA	Pearson's r	0.018	0.290
	BF_{10}	0.196	1.001

* $BF_{10} > 10$, ** $BF_{10} > 30$, *** $BF_{10} > 100$

Appendix Chapter 6. The role of serotonin in cognitive and emotion processing in Parkinson's disease

Experiment 1

Behavioural Analysis: Controls versus PD placebo

Appendix Table 6.1 | Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Set size	0.513	1	0.513	162.676	< .001	0.823
Set size ✻ Group	0.001	1	0.001	0.347	0.560	0.010
Set size ✻ AMI centered	3.470e-4	1	3.470e-4	0.110	0.742	0.003
Set size ✻ BDI centered	8.493e-4	1	8.493e-4	0.270	0.607	0.008
Residuals	0.110	35	0.003			
Delay	0.017	1	0.017	6.740	0.014	0.161
Delay ✻ Group	0.005	1	0.005	1.908	0.176	0.052
Delay ✻ AMI centered	3.154e-4	1	3.154e-4	0.123	0.728	0.004
Delay ✻ BDI centered	0.007	1	0.007	2.560	0.119	0.068
Residuals	0.090	35	0.003			
Set size ✻ Delay	7.845e-4	1	7.845e-4	0.308	0.582	0.009
Set size ✻ Delay ✻ Group	4.035e-4	1	4.035e-4	0.159	0.693	0.005
Set size ✻ Delay ✻ AMI centered	2.530e-5	1	2.530e-5	0.010	0.921	2.840e-4
Set size ✻ Delay ✻ BDI centered	0.003	1	0.003	1.046	0.314	0.029
Residuals	0.089	35	0.003			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.019	1	0.019	2.042	0.162	0.055
AMI centered	0.005	1	0.005	0.480	0.493	0.014
BDI centered	0.006	1	0.006	0.612	0.439	0.017
Residuals	0.333	35	0.010			

Note. Type III Sum of Squares

Appendix Table 6.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay	0.013	0.140	12.235	1.000	
Set size + Delay + Group	0.013	0.098	8.193	0.702	2.614
Set size + Delay + Group + Delay ✻ Group	0.013	0.090	7.397	0.640	43.914
Set size	0.013	0.053	4.178	0.376	1.846
Set size + Delay + AMI centered	0.013	0.046	3.636	0.330	3.753
Set size + Delay + BDI centered	0.013	0.046	3.596	0.326	2.273
Set size + Delay + Set size ✻ Delay	0.013	0.040	3.103	0.283	6.277
Set size + Group	0.013	0.037	2.863	0.262	2.106
Set size + Delay + Group + BDI centered	0.013	0.034	2.630	0.242	4.448
Set size + Delay + Group + AMI centered	0.013	0.032	2.510	0.231	4.289
Set size + Delay + Group + Set size ✻ Delay	0.013	0.027	2.112	0.195	5.948
Set size + Delay + Group + Set size ✻ Group	0.013	0.027	2.083	0.193	3.678
Set size + Delay + AMI centered + BDI centered	0.013	0.026	1.966	0.182	4.438
Set size + BDI centered	0.013	0.018	1.360	0.127	3.063
Set size + AMI centered	0.013	0.017	1.284	0.120	3.377
Set size + Delay + Group + AMI centered + BDI centered	0.013	0.017	1.282	0.120	5.200
Set size + Delay + Group + BDI centered + Delay ✻ Group	0.013	0.017	1.277	0.119	3.283
Set size + Delay + Group + Set size ✻ Group + Delay ✻ Group	0.013	0.016	1.234	0.115	9.664
Set size + Delay + Group + AMI centered + Delay ✻ Group	0.013	0.016	1.213	0.114	5.046
Set size + Delay + Group + Set size ✻ Delay + Delay ✻ Group	0.013	0.014	1.075	0.101	4.719
Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.012	0.907	0.085	4.288
Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.012	0.888	0.083	2.471
Set size + Group + AMI centered	0.013	0.012	0.886	0.083	3.727
Set size + Group + BDI centered	0.013	0.012	0.876	0.082	2.677
Set size + Group + Set size ✻ Group	0.013	0.010	0.792	0.075	3.078
Set size + Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.013	0.010	0.791	0.074	5.629
Set size + Delay + Group + AMI centered + Set size ✻ Group	0.013	0.009	0.708	0.067	5.435
Set size + AMI centered + BDI centered	0.013	0.009	0.678	0.064	3.888
Set size + Delay + Group + BDI centered + Set size ✻ Group	0.013	0.009	0.663	0.062	2.950
Set size + Delay + Group + BDI centered + Set size ✻ Delay	0.013	0.009	0.650	0.061	5.577
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group	0.013	0.008	0.634	0.060	4.939
Set size + Delay + Group + AMI centered + Set size ✻ Delay	0.013	0.008	0.629	0.059	5.279

Appendix Table 6.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + AMI centered + BDI centered + Set size ✻ Delay	0.013	0.006	0.476	0.045	4.950
Set size + Group + AMI centered + BDI centered	0.013	0.006	0.446	0.042	4.583
Set size + Delay + Group + BDI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.005	0.409	0.039	6.525
Set size + Delay + Group + AMI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.005	0.404	0.038	5.526
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.005	0.359	0.034	8.069
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay	0.013	0.004	0.327	0.031	5.834
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Group	0.013	0.004	0.325	0.031	5.875
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.004	0.319	0.030	5.532
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.004	0.289	0.027	4.093
Set size + Group + AMI centered + Set size ✻ Group	0.013	0.004	0.276	0.026	4.316
Set size + Group + BDI centered + Set size ✻ Group	0.013	0.003	0.247	0.023	3.366
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.003	0.201	0.019	6.550
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.002	0.177	0.017	7.166
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.002	0.169	0.016	4.144
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.002	0.161	0.015	5.929
Set size + Group + AMI centered + BDI centered + Set size ✻ Group	0.013	0.002	0.129	0.012	5.402
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.001	0.100	0.009	5.640
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.001	0.097	0.009	6.946
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.001	0.092	0.009	6.481
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	0.001	0.091	0.009	3.803
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	8.278e -4	0.062	0.006	7.710

Appendix Table 6.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	4.282e -4	0.032	0.003	5.161
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	4.055e -4	0.030	0.003	6.861
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	2.139e -4	0.016	0.002	7.694
Null model (incl. subject)	0.013	7.317e-25	5.488e-23	5.217e-24	1.667
Delay	0.013	3.897e-25	2.923e-23	2.779e-24	2.248
Group	0.013	3.434e-25	2.576e-23	2.449e-24	3.338
BDI centered	0.013	1.813e-25	1.360e-23	1.293e-24	2.116
Delay + Group	0.013	1.777e-25	1.333e-23	1.267e-24	2.220
AMI centered	0.013	1.715e-25	1.286e-23	1.223e-24	1.750
Delay + BDI centered	0.013	1.066e-25	7.994e-24	7.600e-25	11.920
Delay + AMI centered	0.013	9.081e-26	6.811e-24	6.475e-25	2.357
Group + BDI centered	0.013	8.803e-26	6.602e-24	6.277e-25	7.595
Group + AMI centered	0.013	7.733e-26	5.800e-24	5.514e-25	2.105
AMI centered + BDI centered	0.013	7.667e-26	5.750e-24	5.467e-25	5.447
Delay + Group + Delay ✻ Group	0.013	5.761e-26	4.321e-24	4.108e-25	2.692
Delay + Group + BDI centered	0.013	4.630e-26	3.473e-24	3.302e-25	4.033
Delay + Group + AMI centered	0.013	4.241e-26	3.181e-24	3.024e-25	3.500
Delay + AMI centered + BDI centered	0.013	3.761e-26	2.820e-24	2.681e-25	3.634
Group + AMI centered + BDI centered	0.013	3.222e-26	2.417e-24	2.297e-25	3.206
Delay + Group + AMI centered + BDI centered	0.013	1.845e-26	1.384e-24	1.316e-25	7.767
Delay + Group + AMI centered + Delay ✻ Group	0.013	1.419e-26	1.064e-24	1.012e-25	5.309

Appendix Table 6.2 | Bayesian Repeated Measures ANOVA on Identification Accuracy with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + Group + BDI centered + Delay ✻ Group	0.013	1.418e-26	1.063e-24	1.011e-25	2.747
Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.013	5.377e-27	4.033e-25	3.834e-26	3.466

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Set size	0.737	1.000	∞
Delay	0.737	0.819	1.612
Group	0.737	0.576	0.486
AMI centered	0.500	0.261	0.354
BDI centered	0.500	0.264	0.359
Set size ✻ Delay	0.316	0.167	0.435
Set size ✻ Group	0.316	0.122	0.300
Delay ✻ Group	0.316	0.197	0.533
Set size ✻ Delay ✻ Group	0.053	0.002	0.041

Appendix Table 6.3 | Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Set size	55.740	1	55.740	176.687	< .001	0.835
Set size ✻ Group	0.075	1	0.075	0.238	0.629	0.007
Set size ✻ AMI centered	0.103	1	0.103	0.325	0.572	0.009
Set size ✻ BDI centered	0.119	1	0.119	0.376	0.544	0.011
Residuals	11.042	35	0.315			
Delay	1.914	1	1.914	39.274	< .001	0.529
Delay ✻ Group	0.099	1	0.099	2.032	0.163	0.055
Delay ✻ AMI centered	0.116	1	0.116	2.382	0.132	0.064
Delay ✻ BDI centered	0.063	1	0.063	1.296	0.263	0.036
Residuals	1.705	35	0.049			
Set size ✻ Delay	0.527	1	0.527	16.822	< .001	0.325
Set size ✻ Delay ✻ Group	0.006	1	0.006	0.197	0.660	0.006
Set size ✻ Delay ✻ AMI centered	0.014	1	0.014	0.449	0.507	0.013
Set size ✻ Delay ✻ BDI centered	0.009	1	0.009	0.280	0.600	0.008
Residuals	1.097	35	0.031			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	1.384	1	1.384	2.498	0.123	0.067
AMI centered	0.837	1	0.837	1.510	0.227	0.041
BDI centered	1.334	1	1.334	2.407	0.130	0.064
Residuals	19.389	35	0.554			

Note. Type III Sum of Squares

Post Hoc Comparisons - Set size ✻ Delay

		Mean Difference	SE	t	p_{holm}
X1.item, X1.second	X3.items, X1.second	-1.079	0.094	-11.437	< .001
	X1.item, X4.seconds	-0.104	0.045	-2.305	0.024
	X3.items, X4.seconds	-1.415	0.097	-14.639	< .001
X3.items, X1.second	X1.item, X4.seconds	0.974	0.097	10.080	< .001
	X3.items, X4.seconds	-0.336	0.045	-7.418	< .001
X1.item, X4.seconds	X3.items, X4.seconds	-1.311	0.094	-13.893	< .001

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Group

Appendix Table 6.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + Set size ✻ Delay	0.013	0.099	8.203	1.000	
Set size + Delay + Group + Set size ✻ Delay	0.013	0.086	7.076	0.874	3.712
Set size + Delay	0.013	0.071	5.739	0.721	2.832
Set size + Delay + Group	0.013	0.061	4.845	0.615	2.756
Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.055	4.388	0.561	13.309
Set size + Delay + Group + BDI centered + Set size ✻ Delay	0.013	0.047	3.698	0.477	7.583
Set size + Delay + AMI centered + BDI centered + Set size ✻ Delay	0.013	0.041	3.194	0.414	5.221
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay	0.013	0.036	2.825	0.368	5.727
Set size + Delay + BDI centered	0.013	0.036	2.801	0.365	4.713
Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.036	2.801	0.365	5.056
Set size + Delay + Group + Delay ✻ Group	0.013	0.036	2.794	0.364	39.619
Set size + Delay + Group + AMI centered + Set size ✻ Delay	0.013	0.030	2.354	0.309	5.319
Set size + Delay + Group + BDI centered	0.013	0.030	2.296	0.301	3.838
Set size + Delay + Group + Set size ✻ Delay + Delay ✻ Group	0.013	0.028	2.128	0.280	4.082
Set size + Delay + AMI centered + BDI centered	0.013	0.027	2.109	0.277	4.753
Set size + Delay + AMI centered	0.013	0.026	2.012	0.265	4.458
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group	0.013	0.026	2.002	0.264	4.806
Set size + Delay + Group + AMI centered	0.013	0.023	1.773	0.234	4.838

Appendix Table 6.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + Group + AMI centered + BDI centered	0.013	0.022	1.694	0.224	5.424
Set size + Delay + Group + Set size ✻ Group	0.013	0.018	1.360	0.181	3.395
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.015	1.177	0.157	23.596
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.014	1.087	0.145	8.643
Set size + Delay + Group + BDI centered + Set size ✻ Group	0.013	0.012	0.931	0.124	17.508
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.012	0.901	0.120	6.590
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.010	0.775	0.104	6.476
Set size + Delay + Group + BDI centered + Delay ✻ Group	0.013	0.010	0.771	0.103	5.593
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.010	0.770	0.103	6.723
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.009	0.699	0.094	6.715
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Group	0.013	0.008	0.607	0.081	6.546
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.008	0.600	0.081	4.152
Set size + Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.013	0.008	0.600	0.080	6.504
Set size + Delay + Group + AMI centered + Delay ✻ Group	0.013	0.007	0.546	0.073	5.550
Set size + Delay + Group + AMI centered + Set size ✻ Group	0.013	0.007	0.521	0.070	5.575
Set size + Delay + Group + Set size ✻ Group + Delay ✻ Group	0.013	0.006	0.460	0.062	4.309
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.005	0.369	0.050	7.411
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.004	0.267	0.036	8.556
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.003	0.244	0.033	7.159
Set size + Delay + Group + BDI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.003	0.201	0.027	8.389
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	0.003	0.198	0.027	6.096
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.002	0.186	0.025	6.811

Appendix Table 6.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + Group + AMI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.002	0.166	0.022	6.801
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	0.001	0.100	0.013	8.094
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	0.001	0.088	0.012	9.050
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	0.001	0.077	0.010	7.667
Set size	0.013	6.768e -4	0.051	0.007	5.182
Set size + Group	0.013	5.264e -4	0.040	0.005	2.721
Set size + BDI centered	0.013	3.082e -4	0.023	0.003	2.917
Set size + Group + BDI centered	0.013	2.656e -4	0.020	0.003	4.501
Set size + AMI centered + BDI centered	0.013	2.324e -4	0.017	0.002	4.385
Set size + AMI centered	0.013	2.229e -4	0.017	0.002	4.077
Set size + Group + AMI centered + BDI centered	0.013	1.976e -4	0.015	0.002	4.853
Set size + Group + AMI centered	0.013	1.770e -4	0.013	0.002	4.531
Set size + Group + Set size ✻ Group	0.013	1.503e -4	0.011	0.002	3.138
Set size + Group + BDI centered + Set size ✻ Group	0.013	6.971e -5	0.005	7.071e -4	3.219
Set size + Group + AMI centered + Set size ✻ Group	0.013	5.508e -5	0.004	5.586e -4	5.180
Set size + Group + AMI centered + BDI centered + Set size ✻ Group	0.013	5.371e -5	0.004	5.448e -4	5.637
Null model (incl. subject)	0.013	1.257e-40	9.429e-39	1.275e-39	2.229
Delay	0.013	1.064e-40	7.981e-39	1.079e-39	2.420
Group	0.013	5.783e-41	4.337e-39	5.865e-40	7.460
Delay + Group	0.013	4.649e-41	3.486e-39	4.715e-40	2.668
BDI centered	0.013	3.744e-41	2.808e-39	3.797e-40	3.025
Delay + BDI centered	0.013	3.232e-41	2.424e-39	3.278e-40	3.466
AMI centered	0.013	2.883e-41	2.162e-39	2.924e-40	2.384
Delay + AMI centered	0.013	2.402e-41	1.801e-39	2.436e-40	2.486
AMI centered + BDI centered	0.013	1.759e-41	1.319e-39	1.784e-40	2.284
Group + BDI centered	0.013	1.553e-41	1.165e-39	1.575e-40	3.016

Appendix Table 6.4 | Bayesian Repeated Measures ANOVA on Reaction Time with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + AMI centered + BDI centered	0.013	1.518e-41	1.139e-39	1.540e-40	2.847
Delay + Group + BDI centered	0.013	1.361e-41	1.021e-39	1.381e-40	3.480
Delay + Group + Delay ✻ Group	0.013	1.255e-41	9.414e-40	1.273e-40	7.354
Group + AMI centered	0.013	1.250e-41	9.372e-40	1.267e-40	3.092
Delay + Group + AMI centered	0.013	1.062e-41	7.964e-40	1.077e-40	3.144
Group + AMI centered + BDI centered	0.013	7.988e-42	5.991e-40	8.102e-41	6.188
Delay + Group + AMI centered + BDI centered	0.013	6.382e-42	4.786e-40	6.473e-41	4.056
Delay + Group + BDI centered + Delay ✻ Group	0.013	3.551e-42	2.663e-40	3.602e-41	5.248
Delay + Group + AMI centered + Delay ✻ Group	0.013	2.669e-42	2.002e-40	2.708e-41	3.471
Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.013	1.511e-42	1.133e-40	1.532e-41	4.030

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Set size	0.737	1.000	8.694e+13
Delay	0.737	0.997	121.294
Group	0.737	0.607	0.552
AMI centered	0.500	0.328	0.489
BDI centered	0.500	0.402	0.672
Set size ✻ Delay	0.316	0.581	3.007
Set size ✻ Group	0.316	0.145	0.369
Delay ✻ Group	0.316	0.164	0.426
Set size ✻ Delay ✻ Group	0.053	0.006	0.112

Appendix Table 6.5 | Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Set size	261668.524	1	261668.524	207.698	< .001	0.856
Set size ✻ Group	40.940	1	40.940	0.032	0.858	9.276e-4
Set size ✻ AMI centered	127.748	1	127.748	0.101	0.752	0.003
Set size ✻ BDI centered	2304.298	1	2304.298	1.829	0.185	0.050

Appendix Table 6.5 | Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	44094.861	35	1259.853			
Delay	3390.048	1	3390.048	7.016	0.012	0.167
Delay ✻ Group	439.242	1	439.242	0.909	0.347	0.025
Delay ✻ AMI centered	34.587	1	34.587	0.072	0.791	0.002
Delay ✻ BDI centered	9.864	1	9.864	0.020	0.887	5.829e-4
Residuals	16912.695	35	483.220			
Set size ✻ Delay	188.910	1	188.910	0.449	0.507	0.013
Set size ✻ Delay ✻ Group	77.260	1	77.260	0.184	0.671	0.005
Set size ✻ Delay ✻ AMI centered	42.954	1	42.954	0.102	0.751	0.003
Set size ✻ Delay ✻ BDI centered	39.215	1	39.215	0.093	0.762	0.003
Residuals	14721.483	35	420.614			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	1308.882	1	1308.882	0.595	0.446	0.017
AMI centered	1295.708	1	1295.708	0.589	0.448	0.017
BDI centered	153.445	1	153.445	0.070	0.793	0.002
Residuals	77006.252	35	2200.179			

Note. Type III Sum of Squares

Appendix Table 6.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay	0.013	0.185	17.062	1.000	
Set size	0.013	0.131	11.282	0.706	3.433
Set size + Delay + AMI centered	0.013	0.066	5.309	0.357	4.312
Set size + Delay + Group	0.013	0.066	5.294	0.356	2.541
Set size + Delay + BDI centered	0.013	0.051	3.997	0.273	2.421
Set size + Delay + Set size ✻ Delay	0.013	0.050	3.922	0.268	3.863
Set size + Group	0.013	0.046	3.657	0.251	2.801
Set size + AMI centered	0.013	0.046	3.646	0.250	3.743
Set size + BDI centered	0.013	0.036	2.783	0.193	2.482
Set size + Delay + AMI centered + BDI centered	0.013	0.026	2.033	0.142	5.264
Set size + Delay + Group + AMI centered	0.013	0.026	1.998	0.140	5.137
Set size + Delay + Group + Delay ✻ Group	0.013	0.021	1.593	0.112	5.429
Set size + Delay + Group + BDI centered	0.013	0.019	1.472	0.104	3.447
Set size + Delay + Group + Set size ✻ Delay	0.013	0.018	1.361	0.096	4.387

Appendix Table 6.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.018	1.361	0.096	5.365
Set size + AMI centered + BDI centered	0.013	0.017	1.319	0.093	4.625
Set size + Delay + Group + Set size ✻ Group	0.013	0.017	1.276	0.090	3.755
Set size + Group + AMI centered	0.013	0.015	1.173	0.083	4.385
Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.013	1.011	0.072	3.527
Set size + Group + BDI centered	0.013	0.012	0.937	0.067	2.492
Set size + Group + Set size ✻ Group	0.013	0.011	0.854	0.061	3.526
Set size + Delay + Group + AMI centered + BDI centered	0.013	0.011	0.837	0.060	6.192
Set size + Delay + Group + AMI centered + Delay ✻ Group	0.013	0.008	0.595	0.042	6.124
Set size + Delay + AMI centered + BDI centered + Set size ✻ Delay	0.013	0.007	0.542	0.039	6.425
Set size + Group + AMI centered + BDI centered	0.013	0.006	0.487	0.035	5.219
Set size + Delay + Group + AMI centered + Set size ✻ Delay	0.013	0.006	0.476	0.034	6.456
Set size + Delay + Group + AMI centered + Set size ✻ Group	0.013	0.006	0.474	0.034	6.146
Set size + Delay + Group + Set size ✻ Delay + Delay ✻ Group	0.013	0.006	0.437	0.031	12.390
Set size + Delay + Group + BDI centered + Delay ✻ Group	0.013	0.006	0.426	0.030	3.779
Set size + Delay + Group + BDI centered + Set size ✻ Delay	0.013	0.005	0.408	0.029	6.817
Set size + Delay + Group + Set size ✻ Group + Delay ✻ Group	0.013	0.005	0.399	0.029	5.714
Set size + Delay + Group + BDI centered + Set size ✻ Group	0.013	0.005	0.361	0.026	6.292
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group	0.013	0.004	0.319	0.023	5.158
Set size + Group + AMI centered + Set size ✻ Group	0.013	0.004	0.302	0.022	5.163
Set size + Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.013	0.003	0.247	0.018	6.440
Set size + Group + BDI centered + Set size ✻ Group	0.013	0.003	0.239	0.017	8.398
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay	0.013	0.003	0.194	0.014	6.780
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Group	0.013	0.002	0.178	0.013	7.393
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.002	0.156	0.011	7.194
Set size + Delay + Group + AMI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.002	0.149	0.011	8.220
Set size + Group + AMI centered + BDI centered + Set size ✻ Group	0.013	0.002	0.128	0.009	6.335

Appendix Table 6.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.002	0.126	0.009	7.863
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Delay ✻ Group	0.013	0.002	0.117	0.008	3.819
Set size + Delay + Group + BDI centered + Set size ✻ Group + Delay ✻ Group	0.013	0.001	0.109	0.008	7.282
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	0.001	0.093	0.007	3.424
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group	0.013	0.001	0.087	0.006	3.275
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Delay ✻ Group	0.013	8.207e -4	0.062	0.004	9.728
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group	0.013	6.855e -4	0.051	0.004	9.417
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Group + Delay ✻ Group	0.013	6.698e -4	0.050	0.004	9.160
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	4.503e -4	0.034	0.002	7.738
Set size + Delay + Group + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	4.438e -4	0.033	0.002	11.943
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	3.742e -4	0.028	0.002	6.511
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group	0.013	1.984e -4	0.015	0.001	15.054
Set size + Delay + Group + AMI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	1.651e -4	0.012	8.908e -4	8.879
Set size + Delay + Group + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	1.172e -4	0.009	6.324e -4	6.130
Set size + Delay + Group + AMI centered + BDI centered + Set size ✻ Delay + Set size ✻ Group + Delay ✻ Group + Set size ✻ Delay ✻ Group	0.013	7.449e -5	0.006	4.019e -4	13.044
Null model (incl. subject)	0.013	4.573e-38	3.430e-36	2.468e-37	1.482
Delay	0.013	1.408e-38	1.056e-36	7.597e-38	2.064
AMI centered	0.013	1.141e-38	8.555e-37	6.155e-38	3.733
Group	0.013	1.091e-38	8.184e-37	5.888e-38	1.783

Appendix Table 6.6 | Bayesian Repeated Measures ANOVA on Localisation error with number of items (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
BDI centered	0.013	9.542e-39	7.156e-37	5.148e-38	1.780
Delay + AMI centered	0.013	4.180e-39	3.135e-37	2.255e-38	19.901
AMI centered + BDI centered	0.013	3.502e-39	2.627e-37	1.890e-38	1.535
Delay + Group	0.013	3.428e-39	2.571e-37	1.850e-38	2.235
Delay + BDI centered	0.013	2.869e-39	2.151e-37	1.548e-38	2.003
Group + AMI centered	0.013	2.700e-39	2.025e-37	1.457e-38	2.216
Group + BDI centered	0.013	2.504e-39	1.878e-37	1.351e-38	7.914
Delay + AMI centered + BDI centered	0.013	1.115e-39	8.364e-38	6.017e-39	3.839
Group + AMI centered + BDI centered	0.013	8.444e-40	6.333e-38	4.556e-39	3.415
Delay + Group + Delay ✻ Group	0.013	8.399e-40	6.299e-38	4.532e-39	2.652
Delay + Group + AMI centered	0.013	8.263e-40	6.197e-38	4.458e-39	2.371
Delay + Group + BDI centered	0.013	7.489e-40	5.617e-38	4.041e-39	3.771
Delay + Group + AMI centered + BDI centered	0.013	2.576e-40	1.932e-38	1.390e-39	2.576
Delay + Group + AMI centered + Delay ✻ Group	0.013	2.017e-40	1.512e-38	1.088e-39	3.384
Delay + Group + BDI centered + Delay ✻ Group	0.013	1.836e-40	1.377e-38	9.904e-40	3.589
Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.013	9.028e-41	6.771e-39	4.871e-40	25.941

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Set size	0.737	1.000	∞
Delay	0.737	0.669	0.722
Group	0.737	0.353	0.195
AMI centered	0.500	0.283	0.395
BDI centered	0.500	0.236	0.308
Set size ✻ Delay	0.316	0.141	0.356
Set size ✻ Group	0.316	0.071	0.164
Delay ✻ Group	0.316	0.060	0.139
Set size ✻ Delay ✻ Group	0.053	8.006e-4	0.014

Mixture model Analysis: Controls versus PD placebo

Appendix Table 6.7 | Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay	605.694	1	605.694	7.346	0.010	0.173
Delay ✻ Group	1.038	1	1.038	0.013	0.911	3.595e-4
Delay ✻ AMI centered	0.147	1	0.147	0.002	0.967	5.101e-5
Delay ✻ BDI centered	163.812	1	163.812	1.987	0.168	0.054
Residuals	2885.868	35	82.453			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	724.016	1	724.016	1.896	0.177	0.051
AMI centered	296.503	1	296.503	0.776	0.384	0.022
BDI centered	84.373	1	84.373	0.221	0.641	0.006
Residuals	13368.721	35	381.963			

Note. Type III Sum of Squares

Appendix Table 6.8 | Bayesian Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay	0.050	0.179	4.141	1.000	
Delay + Group	0.050	0.144	3.184	0.802	1.770
Delay + AMI centered	0.050	0.089	1.855	0.497	2.124
Delay + BDI centered	0.050	0.088	1.836	0.492	15.094
Delay + Group + AMI centered	0.050	0.075	1.537	0.418	2.270
Delay + Group + BDI centered	0.050	0.060	1.223	0.338	2.862
Delay + AMI centered + BDI centered	0.050	0.051	1.012	0.283	2.468
Delay + Group + Delay ✻ Group	0.050	0.045	0.894	0.251	2.252
Delay + Group + AMI centered + BDI centered	0.050	0.045	0.888	0.250	2.634
Null model (incl. subject)	0.050	0.044	0.869	0.244	0.843
Group	0.050	0.036	0.700	0.199	2.323
Delay + Group + AMI centered + Delay ✻ Group	0.050	0.023	0.450	0.129	2.716
AMI centered	0.050	0.021	0.399	0.115	1.825
Delay + Group + BDI centered + Delay ✻ Group	0.050	0.019	0.367	0.106	2.762
Group + AMI centered	0.050	0.017	0.331	0.096	1.999
BDI centered	0.050	0.017	0.324	0.094	1.220
Group + BDI centered	0.050	0.014	0.273	0.079	2.783
Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.050	0.014	0.268	0.078	3.156
AMI centered + BDI centered	0.050	0.012	0.224	0.065	2.169

Appendix Table 6.8 | Bayesian Repeated Measures ANOVA on Imprecision with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Group + AMI centered + BDI centered	0.050	0.009	0.182	0.053	2.364

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Delay	0.600	0.831	3.278
Group	0.600	0.501	0.669
AMI centered	0.500	0.355	0.550
BDI centered	0.500	0.329	0.489
Delay ✻ Group	0.200	0.101	0.449

Appendix Table 6.9 | Repeated Measures ANOVA on Misbinding with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay	0.012	1	0.012	2.142	0.152	0.058
Delay ✻ Group	7.434e-4	1	7.434e-4	0.134	0.716	0.004
Delay ✻ AMI centered	0.006	1	0.006	1.098	0.302	0.030
Delay ✻ BDI centered	7.680e-4	1	7.680e-4	0.139	0.712	0.004
Residuals	0.194	35	0.006			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.003	1	0.003	0.196	0.661	0.006
AMI centered	7.662e-4	1	7.662e-4	0.048	0.827	0.001
BDI centered	0.002	1	0.002	0.139	0.711	0.004
Residuals	0.554	35	0.016			

Note. Type III Sum of Squares

Appendix Table 6.10 | Bayesian Repeated Measures ANOVA on Misbinding with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.220	5.372	1.000	
Delay	0.050	0.135	2.973	0.614	1.556
BDI centered	0.050	0.086	1.794	0.392	1.904
AMI centered	0.050	0.082	1.708	0.374	1.581
Group	0.050	0.081	1.682	0.369	0.587
Delay + BDI centered	0.050	0.054	1.080	0.244	1.767
Delay + AMI centered	0.050	0.053	1.073	0.243	1.867

Appendix Table 6.10 | Bayesian Repeated Measures ANOVA on Misbinding with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + Group	0.050	0.051	1.018	0.231	1.295
AMI centered + BDI centered	0.050	0.043	0.849	0.194	1.986
Group + BDI centered	0.050	0.033	0.654	0.151	2.311
Group + AMI centered	0.050	0.031	0.615	0.142	1.915
Delay + AMI centered + BDI centered	0.050	0.028	0.543	0.126	2.378
Delay + Group + AMI centered	0.050	0.020	0.395	0.092	2.251
Delay + Group + BDI centered	0.050	0.020	0.395	0.092	2.204
Group + AMI centered + BDI centered	0.050	0.017	0.330	0.077	2.353
Delay + Group + Delay ✻ Group	0.050	0.017	0.320	0.075	1.760
Delay + Group + AMI centered + BDI centered	0.050	0.010	0.197	0.047	2.785
Delay + Group + AMI centered + Delay ✻ Group	0.050	0.007	0.127	0.030	2.702
Delay + Group + BDI centered + Delay ✻ Group	0.050	0.006	0.122	0.029	1.570
Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.050	0.003	0.062	0.015	3.317

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Delay	0.600	0.405	0.454
Group	0.600	0.298	0.283
AMI centered	0.500	0.295	0.419
BDI centered	0.500	0.301	0.431
Delay ✻ Group	0.200	0.033	0.136

Appendix Table 6.11 | Repeated Measures ANOVA on Guessing with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay	8.918e-4	1	8.918e-4	0.718	0.403	0.020
Delay ✻ Group	0.005	1	0.005	4.371	0.044	0.111
Delay ✻ AMI centered	0.004	1	0.004	3.219	0.081	0.084
Delay ✻ BDI centered	4.279e-4	1	4.279e-4	0.344	0.561	0.010
Residuals	0.043	35	0.001			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.001	1	0.001	0.721	0.402	0.020
AMI centered	0.001	1	0.001	0.790	0.380	0.022
BDI centered	1.569e-5	1	1.569e-5	0.010	0.922	2.773e-4
Residuals	0.057	35	0.002			

Note. Type III Sum of Squares

Post Hoc Comparisons - Group ✻ Delay

		Mean Difference	SE	t	p_{holm}
EC, X1.second	PD_placebo, X1.second	0.009	0.012	0.740	1.000
	EC, X4.seconds	0.010	0.011	0.913	1.000
	PD_placebo, X4.seconds	-0.014	0.012	-1.177	0.973
PD_placebo, X1.second	EC, X4.seconds	0.001	0.012	0.100	1.000
	PD_placebo, X4.seconds	-0.023	0.011	-2.030	0.286
EC, X4.seconds	PD_placebo, X4.seconds	-0.024	0.012	-2.017	0.286

Note. P-value adjusted for comparing a family of 6

Appendix Table 6.12 | Bayesian Repeated Measures ANOVA on Guessing with delay (1 second versus 4 seconds) as "within-subjects" factors, group (controls versus patients on placebo) as "between-subjects" and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.264	6.803	1.000	
AMI centered	0.050	0.106	2.254	0.402	0.524
Group	0.050	0.098	2.066	0.372	2.901
BDI centered	0.050	0.080	1.656	0.304	2.233
Delay	0.050	0.079	1.627	0.299	0.748
Delay + Group + Delay ✻ Group	0.050	0.062	1.257	0.235	2.491
AMI centered + BDI centered	0.050	0.043	0.861	0.164	3.187
Group + AMI centered	0.050	0.038	0.752	0.144	1.736
Delay + AMI centered	0.050	0.031	0.615	0.119	1.669
Delay + Group + AMI centered + Delay ✻ Group	0.050	0.031	0.605	0.117	15.019
Group + BDI centered	0.050	0.030	0.594	0.115	2.208
Delay + Group	0.050	0.030	0.582	0.113	2.651
Delay + BDI centered	0.050	0.023	0.452	0.088	1.327
Delay + Group + BDI centered + Delay ✻ Group	0.050	0.020	0.378	0.074	3.565
Group + AMI centered + BDI centered	0.050	0.016	0.307	0.060	3.707
Delay + AMI centered + BDI centered	0.050	0.013	0.256	0.050	3.764
Delay + Group + AMI centered	0.050	0.011	0.217	0.043	2.323
Delay + Group + AMI centered + BDI centered + Delay ✻ Group	0.050	0.011	0.213	0.042	4.494
Delay + Group + BDI centered	0.050	0.008	0.163	0.032	1.943
Delay + Group + AMI centered + BDI centered	0.050	0.005	0.087	0.017	4.186

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Delay	0.600	0.324	0.320
Group	0.600	0.360	0.375
AMI centered	0.500	0.306	0.441
BDI centered	0.500	0.250	0.333
Delay ✻ Group	0.200	0.124	0.564

Behavioural Analysis: PD placebo versus PD citalopram

Appendix Table 6.13 | Repeated Measures ANOVA on Identification Accuracy with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	0.002	1	0.002	0.512	0.485	0.031
Drug ✻ BDI centered	1.786e-4	1	1.786e-4	0.053	0.821	0.003
Drug ✻ AMI centered	0.010	1	0.010	2.847	0.111	0.151
Residuals	0.054	16	0.003			
Set size	0.612	1	0.612	91.400	< .001	0.851
Set size ✻ BDI centered	4.707e-4	1	4.707e-4	0.070	0.794	0.004
Set size ✻ AMI centered	0.004	1	0.004	0.600	0.450	0.036
Residuals	0.107	16	0.007			
Delay	0.037	1	0.037	16.717	< .001	0.511
Delay ✻ BDI centered	0.003	1	0.003	1.206	0.288	0.070
Delay ✻ AMI centered	1.674e-5	1	1.674e-5	0.007	0.932	4.662e-4
Residuals	0.036	16	0.002			
Drug ✻ Set size	0.002	1	0.002	0.510	0.485	0.031
Drug ✻ Set size ✻ BDI centered	1.490e-4	1	1.490e-4	0.042	0.841	0.003
Drug ✻ Set size ✻ AMI centered	0.001	1	0.001	0.379	0.547	0.023
Residuals	0.057	16	0.004			
Drug ✻ Delay	2.292e-5	1	2.292e-5	0.009	0.924	5.905e-4
Drug ✻ Delay ✻ BDI centered	0.008	1	0.008	3.164	0.094	0.165
Drug ✻ Delay ✻ AMI centered	0.003	1	0.003	1.341	0.264	0.077
Residuals	0.039	16	0.002			
Delay ✻ Set size	0.003	1	0.003	1.774	0.202	0.100
Delay ✻ Set size ✻ BDI centered	0.001	1	0.001	0.735	0.404	0.044
Delay ✻ Set size ✻ AMI centered	7.008e-5	1	7.008e-5	0.044	0.837	0.003
Residuals	0.039	16	0.002			
Drug ✻ Delay ✻ Set size	4.816e-5	1	4.816e-5	0.049	0.828	0.003
Drug ✻ Delay ✻ Set size ✻ BDI centered	0.007	1	0.007	7.012	0.018	0.305
Drug ✻ Delay ✻ Set size ✻ AMI centered	7.198e-5	1	7.198e-5	0.073	0.791	0.005
Residuals	0.039	16	0.002			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	0.009	1	0.009	0.411	0.530
AMI centered	0.004	1	0.004	0.168	0.687
Residuals	0.335	16	0.021		

Note. Type III Sum of Squares

Appendix Table 6.14 | Bayesian Repeated Measures ANOVA on Identification Accuracy with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay	0.013	0.265	26.998	1.000	
Set size + Delay + BDI centered	0.013	0.103	8.610	0.389	4.442
Set size + Delay + AMI centered	0.013	0.100	8.297	0.376	5.117
Set size + Delay + Set size ✻ Delay	0.013	0.091	7.537	0.345	5.254
Set size + Delay + BDI centered + AMI centered	0.013	0.060	4.786	0.227	5.410
Drug + Set size + Delay	0.013	0.059	4.672	0.222	6.190
Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.040	3.144	0.152	6.680
Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.035	2.752	0.134	5.438
Drug + Set size + Delay + BDI centered	0.013	0.025	1.887	0.093	5.492
Drug + Set size + Delay + AMI centered	0.013	0.024	1.832	0.090	5.547
Set size + Delay + BDI centered + AMI centered + Set size ✻ Delay	0.013	0.024	1.813	0.089	5.964
Drug + Set size + Delay + Set size ✻ Delay	0.013	0.023	1.733	0.085	7.088
Drug + Set size + Delay + Drug ✻ Set size	0.013	0.016	1.244	0.062	4.454
Drug + Set size + Delay + BDI centered + AMI centered	0.013	0.014	1.073	0.053	6.047
Drug + Set size + Delay + Drug ✻ Delay	0.013	0.014	1.044	0.052	5.878
Drug + Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.009	0.658	0.033	6.037
Drug + Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.008	0.631	0.032	5.000
Drug + Set size + Delay + BDI centered + Drug ✻ Set size	0.013	0.008	0.620	0.031	9.740
Drug + Set size + Delay + AMI centered + Drug ✻ Set size	0.013	0.007	0.564	0.028	5.962
Drug + Set size + Delay + Drug ✻ Set size + Set size ✻ Delay	0.013	0.007	0.512	0.026	5.957
Drug + Set size + Delay + BDI centered + Drug ✻ Delay	0.013	0.006	0.468	0.023	6.460
Drug + Set size + Delay + AMI centered + Drug ✻ Delay	0.013	0.006	0.426	0.021	6.121
Drug + Set size + Delay + Drug ✻ Delay + Set size ✻ Delay	0.013	0.005	0.396	0.020	6.213
Drug + Set size + Delay + BDI centered + AMI centered + Set size ✻ Delay	0.013	0.005	0.378	0.019	6.716
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay	0.013	0.005	0.361	0.018	8.164
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size	0.013	0.005	0.355	0.018	6.536
Set size	0.013	0.005	0.352	0.018	4.222
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.013	0.003	0.257	0.013	6.703
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.003	0.225	0.011	7.537
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.003	0.208	0.010	6.623

Appendix Table 6.14 | Bayesian Repeated Measures ANOVA on Identification Accuracy with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Set size + Delay + BDI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.002	0.160	0.008	7.564
Drug + Set size + Delay + AMI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.002	0.154	0.008	6.640
Set size + BDI centered	0.013	0.002	0.147	0.007	4.414
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.002	0.140	0.007	6.992
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.002	0.135	0.007	8.293
Set size + AMI centered	0.013	0.002	0.134	0.007	4.927
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.002	0.127	0.006	6.738
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.002	0.114	0.006	8.474
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.001	0.086	0.004	7.596
Drug + Set size	0.013	0.001	0.083	0.004	4.801
Set size + BDI centered + AMI centered	0.013	0.001	0.080	0.004	5.201
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.001	0.076	0.004	7.123
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	6.443e -4	0.048	0.002	8.386
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	5.984e -4	0.045	0.002	7.884
Drug + Set size + BDI centered	0.013	4.373e -4	0.033	0.002	4.897
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	4.316e -4	0.032	0.002	4.861
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	4.091e -4	0.031	0.002	9.256
Drug + Set size + AMI centered	0.013	4.076e -4	0.031	0.002	5.293
Drug + Set size + Drug ✻ Set size	0.013	3.268e -4	0.025	0.001	4.913
Drug + Set size + BDI centered + AMI centered	0.013	2.412e -4	0.018	9.112e -4	5.741
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	2.107e -4	0.016	7.959e -4	8.801
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	1.888e -4	0.014	7.134e -4	7.813
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	1.332e -4	0.010	5.032e -4	9.075
Drug + Set size + BDI centered + Drug ✻ Set size	0.013	1.281e -4	0.010	4.839e -4	4.769

Appendix Table 6.14 | Bayesian Repeated Measures ANOVA on Identification Accuracy with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Set size + AMI centered + Drug ✻ Set size	0.013	1.273e -4	0.010	4.809e -4	5.769
Drug + Set size + BDI centered + AMI centered + Drug ✻ Set size	0.013	7.523e -5	0.006	2.842e -4	6.427
Delay	0.013	2.512e-27	1.884e-25	9.492e-27	4.914
Null model (incl. subject)	0.013	1.410e-27	1.057e-25	5.325e-27	4.140
Delay + BDI centered	0.013	7.834e-28	5.876e-26	2.960e-27	4.347
Delay + AMI centered	0.013	7.535e-28	5.651e-26	2.847e-27	4.588
Drug + Delay	0.013	4.784e-28	3.588e-26	1.807e-27	4.494
BDI centered	0.013	4.425e-28	3.319e-26	1.672e-27	4.234
AMI centered	0.013	4.178e-28	3.133e-26	1.578e-27	4.221
Delay + BDI centered + AMI centered	0.013	3.629e-28	2.722e-26	1.371e-27	5.679
Drug	0.013	2.682e-28	2.012e-26	1.013e-27	4.266
BDI centered + AMI centered	0.013	1.840e-28	1.380e-26	6.950e-28	5.277
Drug + Delay + AMI centered	0.013	1.528e-28	1.146e-26	5.774e-28	5.735
Drug + Delay + BDI centered	0.013	1.502e-28	1.126e-26	5.673e-28	4.523
Drug + Delay + Drug ✻ Delay	0.013	1.202e-28	9.014e-27	4.541e-28	7.179
Drug + BDI centered	0.013	8.334e-29	6.250e-27	3.148e-28	4.364
Drug + AMI centered	0.013	7.932e-29	5.949e-27	2.997e-28	4.463
Drug + Delay + BDI centered + AMI centered	0.013	6.911e-29	5.183e-27	2.611e-28	6.392
Drug + BDI centered + AMI centered	0.013	4.063e-29	3.047e-27	1.535e-28	5.813
Drug + Delay + BDI centered + Drug ✻ Delay	0.013	3.977e-29	2.983e-27	1.503e-28	8.830
Drug + Delay + AMI centered + Drug ✻ Delay	0.013	3.457e-29	2.592e-27	1.306e-28	5.308
Drug + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.013	1.756e-29	1.317e-27	6.636e-29	7.049

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.737	0.273	0.134
Set size	0.737	1.000	∞
Delay	0.737	0.988	28.625
BDI centered	0.500	0.318	0.465
AMI centered	0.500	0.307	0.443
Drug ✻ Set size	0.316	0.065	0.151
Drug ✻ Delay	0.316	0.053	0.121
Set size ✻ Delay	0.316	0.264	0.778
Drug ✻ Set size ✻ Delay	0.053	9.643e-4	0.017

Appendix Table 6.15 | Repeated Measures ANOVA on Reaction Time with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	0.184	1	0.184	1.165	0.296	0.068
Drug ✻ BDI centered	0.184	1	0.184	1.163	0.297	0.068
Drug ✻ AMI centered	0.192	1	0.192	1.214	0.287	0.071
Residuals	2.532	16	0.158			
Set size	45.446	1	45.446	111.786	< .001	0.875
Set size ✻ BDI centered	0.486	1	0.486	1.196	0.290	0.070
Set size ✻ AMI centered	0.332	1	0.332	0.817	0.379	0.049
Residuals	6.505	16	0.407			
Delay	2.341	1	2.341	55.149	< .001	0.775
Delay ✻ BDI centered	6.781e-6	1	6.781e-6	1.598e-4	0.990	9.986e-6
Delay ✻ AMI centered	1.150e-5	1	1.150e-5	2.709e-4	0.987	1.693e-5
Residuals	0.679	16	0.042			
Drug ✻ Set size	0.127	1	0.127	0.677	0.423	0.041
Drug ✻ Set size ✻ BDI centered	0.007	1	0.007	0.036	0.853	0.002
Drug ✻ Set size ✻ AMI centered	0.166	1	0.166	0.889	0.360	0.053
Residuals	2.993	16	0.187			
Drug ✻ Delay	0.226	1	0.226	3.745	0.071	0.190
Drug ✻ Delay ✻ BDI centered	3.486e-5	1	3.486e-5	5.784e-4	0.981	3.615e-5
Drug ✻ Delay ✻ AMI centered	0.013	1	0.013	0.212	0.651	0.013
Residuals	0.964	16	0.060			
Set size ✻ Delay	1.015	1	1.015	7.423	0.015	0.317
Set size ✻ Delay ✻ BDI centered	0.006	1	0.006	0.045	0.835	0.003
Set size ✻ Delay ✻ AMI centered	0.010	1	0.010	0.074	0.790	0.005
Residuals	2.189	16	0.137			
Drug ✻ Set size ✻ Delay	0.136	1	0.136	2.137	0.163	0.118
Drug ✻ Set size ✻ Delay ✻ BDI centered	0.004	1	0.004	0.066	0.800	0.004
Drug ✻ Set size ✻ Delay ✻ AMI centered	0.012	1	0.012	0.184	0.674	0.011
Residuals	1.017	16	0.064			

Appendix Table 6.15 | Repeated Measures ANOVA on Reaction Time with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	0.395	1	0.395	0.315	0.583
AMI centered	0.070	1	0.070	0.056	0.816
Residuals	20.076	16	1.255		

Note. Type III Sum of Squares

Post Hoc Comparisons - Set size ✻ Delay

		Mean Difference	SE	t	p_{holm}
X1.item, X1.second	X3.items, X1.second	-0.930	0.120	-7.776	< .001
	X1.item, X4.seconds	-0.085	0.069	-1.232	0.229
	X3.items, X4.seconds	-1.342	0.109	-12.343	< .001
X3.items, X1.second	X1.item, X4.seconds	0.845	0.109	7.776	< .001
	X3.items, X4.seconds	-0.412	0.069	-5.996	< .001
X1.item, X4.seconds	X3.items, X4.seconds	-1.257	0.120	-10.512	< .001

Note. P-value adjusted for comparing a family of 6

Note. Results are averaged over the levels of: Drug

Appendix Table 6.16 | Bayesian Repeated Measures ANOVA on Reaction Time with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay + Set size ✻ Delay	0.013	0.241	23.808	1.000	
Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.104	8.702	0.431	4.338
Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.102	8.495	0.422	3.106
Drug + Set size + Delay + Set size ✻ Delay	0.013	0.069	5.547	0.286	3.119
Set size + Delay + BDI centered + AMI centered + Set size ✻ Delay	0.013	0.060	4.790	0.249	4.669
Set size + Delay	0.013	0.057	4.531	0.236	6.644
Drug + Set size + Delay + Drug ✻ Delay + Set size ✻ Delay	0.013	0.035	2.752	0.147	6.325
Drug + Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.032	2.511	0.134	6.901
Drug + Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.029	2.245	0.121	5.115
Drug + Set size + Delay + Drug ✻ Set size + Set size ✻ Delay	0.013	0.025	1.889	0.102	7.553
Set size + Delay + BDI centered	0.013	0.025	1.884	0.102	4.061
Set size + Delay + AMI centered	0.013	0.023	1.729	0.094	3.973

Appendix Table 6.16 | Bayesian Repeated Measures ANOVA on Reaction Time with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Set size + Delay + BDI centered + AMI centered + Set size ✻ Delay	0.013	0.018	1.411	0.077	5.263
Drug + Set size + Delay	0.013	0.015	1.163	0.063	3.946
Drug + Set size + Delay + AMI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.014	1.049	0.057	5.596
Drug + Set size + Delay + BDI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.013	1.025	0.056	4.208
Set size + Delay + BDI centered + AMI centered	0.013	0.013	1.007	0.055	4.365
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.012	0.918	0.050	5.830
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.011	0.846	0.046	4.393
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.011	0.824	0.045	4.304
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.009	0.693	0.038	5.946
Drug + Set size + Delay + Drug ✻ Delay	0.013	0.007	0.543	0.030	4.329
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.007	0.535	0.029	6.505
Drug + Set size + Delay + AMI centered	0.013	0.007	0.493	0.027	4.633
Drug + Set size + Delay + BDI centered	0.013	0.006	0.486	0.027	4.310
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	0.006	0.417	0.023	9.394
Drug + Set size + Delay + Drug ✻ Set size	0.013	0.005	0.406	0.022	4.838
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.005	0.404	0.022	6.697
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.005	0.377	0.021	6.316
Drug + Set size + Delay + BDI centered + AMI centered	0.013	0.004	0.285	0.016	5.252
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.003	0.251	0.014	9.338
Drug + Set size + Delay + AMI centered + Drug ✻ Delay	0.013	0.003	0.243	0.013	5.091
Drug + Set size + Delay + BDI centered + Drug ✻ Delay	0.013	0.003	0.220	0.012	3.600
Drug + Set size + Delay + BDI centered + Drug ✻ Set size	0.013	0.002	0.174	0.010	4.510
Drug + Set size + Delay + AMI centered + Drug ✻ Set size	0.013	0.002	0.173	0.010	5.431
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay	0.013	0.002	0.169	0.009	3.474
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	0.002	0.163	0.009	6.234

Appendix Table 6.16 | Bayesian Repeated Measures ANOVA on Reaction Time with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	0.002	0.161	0.009	4.712
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.013	0.002	0.133	0.007	6.142
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.001	0.103	0.006	19.919
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	0.001	0.097	0.005	7.046
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size	0.013	0.001	0.097	0.005	5.721
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.001	0.080	0.004	5.753
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	6.104e -4	0.046	0.003	6.701
Set size	0.013	3.977e -4	0.030	0.002	2.956
Set size + BDI centered	0.013	1.788e -4	0.013	7.419e -4	6.733
Set size + AMI centered	0.013	1.531e -4	0.011	6.355e -4	3.650
Drug + Set size	0.013	1.065e -4	0.008	4.418e -4	3.923
Set size + BDI centered + AMI centered	0.013	9.028e -5	0.007	3.747e -4	4.111
Drug + Set size + BDI centered	0.013	4.681e -5	0.004	1.943e -4	4.003
Drug + Set size + AMI centered	0.013	4.024e -5	0.003	1.670e -4	4.237
Drug + Set size + Drug ✻ Set size	0.013	3.254e -5	0.002	1.351e -4	2.812
Drug + Set size + BDI centered + AMI centered	0.013	2.546e -5	0.002	1.056e -4	4.677
Drug + Set size + AMI centered + Drug ✻ Set size	0.013	1.535e -5	0.001	6.372e -5	4.602
Drug + Set size + BDI centered + Drug ✻ Set size	0.013	1.396e -5	0.001	5.792e -5	3.217
Drug + Set size + BDI centered + AMI centered + Drug ✻ Set size	0.013	8.211e -6	6.158e -4	3.408e -5	5.245
Delay	0.013	2.075e-34	1.556e-32	8.611e-34	2.507
Null model (incl. subject)	0.013	1.324e-34	9.933e-33	5.497e-34	2.335
Delay + AMI centered	0.013	7.012e-35	5.259e-33	2.910e-34	14.798
Delay + BDI centered	0.013	6.959e-35	5.219e-33	2.888e-34	4.139
BDI centered	0.013	4.191e-35	3.143e-33	1.739e-34	3.329
Drug + Delay	0.013	4.183e-35	3.137e-33	1.736e-34	2.934
AMI centered	0.013	3.855e-35	2.891e-33	1.600e-34	2.416

Appendix Table 6.16 | Bayesian Repeated Measures ANOVA on Reaction Time with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay + BDI centered + AMI centered	0.013	2.812e-35	2.109e-33	1.167e-34	4.758
Drug	0.013	2.652e-35	1.989e-33	1.101e-34	2.608
BDI centered + AMI centered	0.013	1.846e-35	1.385e-33	7.662e-35	4.193
Drug + Delay + BDI centered	0.013	1.317e-35	9.874e-34	5.464e-35	3.135
Drug + Delay + Drug ✻ Delay	0.013	1.255e-35	9.411e-34	5.208e-35	3.926
Drug + Delay + AMI centered	0.013	1.246e-35	9.345e-34	5.171e-35	3.503
Drug + BDI centered	0.013	8.648e-36	6.486e-34	3.589e-35	4.768
Drug + AMI centered	0.013	7.759e-36	5.820e-34	3.220e-35	3.464
Drug + Delay + BDI centered + AMI centered	0.013	5.780e-36	4.335e-34	2.399e-35	5.489
Drug + Delay + AMI centered + Drug ✻ Delay	0.013	3.833e-36	2.875e-34	1.591e-35	11.216
Drug + Delay + BDI centered + Drug ✻ Delay	0.013	3.831e-36	2.873e-34	1.590e-35	3.533
Drug + BDI centered + AMI centered	0.013	3.620e-36	2.715e-34	1.502e-35	4.900
Drug + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.013	1.661e-36	1.246e-34	6.893e-36	5.998

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.737	0.375	0.214
Set size	0.737	1.000	∞
Delay	0.737	0.999	321.698
BDI centered	0.500	0.324	0.479
AMI centered	0.500	0.322	0.475
Drug ✻ Set size	0.316	0.107	0.260
Drug ✻ Delay	0.316	0.128	0.319
Set size ✻ Delay	0.316	0.818	9.732
Drug ✻ Set size ✻ Delay	0.053	0.011	0.203

Appendix Table 6.17 | Repeated Measures ANOVA on Localisation error with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	693.257	1	693.257	0.827	0.377	0.049
Drug ✻ BDI centered	2242.777	1	2242.777	2.677	0.121	0.143
Drug ✻ AMI centered	5761.524	1	5761.524	6.877	0.018	0.301
Residuals	13405.007	16	837.813			
Set size	274569.777	1	274569.777	139.898	< .001	0.897
Set size ✻ BDI centered	52.711	1	52.711	0.027	0.872	0.002
Set size ✻ AMI centered	1668.035	1	1668.035	0.850	0.370	0.050
Residuals	31402.310	16	1962.644			
Delay	8481.424	1	8481.424	11.935	0.003	0.427
Delay ✻ BDI centered	772.310	1	772.310	1.087	0.313	0.064
Delay ✻ AMI centered	417.453	1	417.453	0.587	0.455	0.035
Residuals	11369.865	16	710.617			
Drug ✻ Set size	699.050	1	699.050	1.151	0.299	0.067
Drug ✻ Set size ✻ BDI centered	1012.912	1	1012.912	1.668	0.215	0.094
Drug ✻ Set size ✻ AMI centered	3974.194	1	3974.194	6.544	0.021	0.290
Residuals	9716.123	16	607.258			
Drug ✻ Delay	190.572	1	190.572	0.412	0.530	0.025
Drug ✻ Delay ✻ BDI centered	8.504	1	8.504	0.018	0.894	0.001
Drug ✻ Delay ✻ AMI centered	22.964	1	22.964	0.050	0.826	0.003
Residuals	7393.144	16	462.071			
Set size ✻ Delay	1105.134	1	1105.134	2.889	0.109	0.153
Set size ✻ Delay ✻ BDI centered	196.711	1	196.711	0.514	0.484	0.031
Set size ✻ Delay ✻ AMI centered	702.515	1	702.515	1.836	0.194	0.103
Residuals	6120.976	16	382.561			
Drug ✻ Set size ✻ Delay	115.398	1	115.398	0.232	0.636	0.014
Drug ✻ Set size ✻ Delay ✻ BDI centered	0.015	1	0.015	3.019e-5	0.996	1.887e-6
Drug ✻ Set size ✻ Delay ✻ AMI centered	3.329	1	3.329	0.007	0.936	4.183e-4
Residuals	7954.760	16	497.173			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	529.196	1	529.196	0.090	0.768
AMI centered	302.316	1	302.316	0.051	0.824
Residuals	94430.349	16	5901.897		

Note. Type III Sum of Squares

Appendix Table 6.18 | Bayesian Repeated Measures ANOVA on Localisation error with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Set size + Delay	0.013	0.230	22.438	1.000	
Set size + Delay + AMI centered	0.013	0.099	8.233	0.430	8.214
Set size + Delay + BDI centered	0.013	0.095	7.860	0.412	8.172
Set size + Delay + Set size ✻ Delay	0.013	0.088	7.275	0.384	7.701
Drug + Set size + Delay	0.013	0.073	5.866	0.315	23.680
Set size + Delay + BDI centered + AMI centered	0.013	0.047	3.703	0.204	8.535
Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.044	3.458	0.191	8.501
Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.039	3.074	0.171	8.428
Drug + Set size + Delay + Set size ✻ Delay	0.013	0.026	2.020	0.114	11.132
Drug + Set size + Delay + AMI centered	0.013	0.025	1.911	0.108	8.510
Drug + Set size + Delay + BDI centered	0.013	0.024	1.827	0.103	8.238
Drug + Set size + Delay + Drug ✻ Set size	0.013	0.021	1.575	0.089	8.490
Set size + Delay + BDI centered + AMI centered + Set size ✻ Delay	0.013	0.020	1.566	0.089	8.926
Drug + Set size + Delay + Drug ✻ Delay	0.013	0.015	1.118	0.064	8.116
Drug + Set size + Delay + BDI centered + AMI centered	0.013	0.013	0.957	0.055	8.849
Drug + Set size + Delay + AMI centered + Set size ✻ Delay	0.013	0.011	0.810	0.046	8.976
Set size	0.013	0.010	0.788	0.045	7.612
Drug + Set size + Delay + BDI centered + Set size ✻ Delay	0.013	0.009	0.690	0.040	7.889
Drug + Set size + Delay + Drug ✻ Set size + Set size ✻ Delay	0.013	0.009	0.689	0.040	12.524
Drug + Set size + Delay + BDI centered + Drug ✻ Set size	0.013	0.008	0.598	0.034	8.027
Drug + Set size + Delay + AMI centered + Drug ✻ Set size	0.013	0.007	0.545	0.031	8.770
Drug + Set size + Delay + Drug ✻ Delay + Set size ✻ Delay	0.013	0.007	0.524	0.030	9.103
Drug + Set size + Delay + AMI centered + Drug ✻ Delay	0.013	0.006	0.480	0.028	9.096
Drug + Set size + Delay + BDI centered + Drug ✻ Delay	0.013	0.006	0.476	0.027	8.423
Drug + Set size + Delay + BDI centered + AMI centered + Set size ✻ Delay	0.013	0.006	0.463	0.027	9.486
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size	0.013	0.005	0.383	0.022	9.439
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay	0.013	0.005	0.378	0.022	8.349
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.004	0.329	0.019	18.511
Set size + AMI centered	0.013	0.004	0.317	0.018	8.103
Set size + BDI centered	0.013	0.004	0.309	0.018	7.635
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.004	0.270	0.016	9.716

Appendix Table 6.18 | Bayesian Repeated Measures ANOVA on Localisation error with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.013	0.004	0.265	0.015	9.394
Drug + Set size + Delay + BDI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.003	0.227	0.013	9.570
Drug + Set size + Delay + AMI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.003	0.209	0.012	9.440
Drug + Set size	0.013	0.003	0.190	0.011	7.612
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.002	0.179	0.010	9.399
Set size + BDI centered + AMI centered	0.013	0.002	0.178	0.010	8.252
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.002	0.168	0.010	9.259
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.002	0.158	0.009	8.039
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Set size ✻ Delay	0.013	0.002	0.149	0.009	9.913
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Delay + Set size ✻ Delay	0.013	0.002	0.114	0.007	9.916
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay	0.013	0.001	0.103	0.006	10.791
Drug + Set size + BDI centered	0.013	0.001	0.099	0.006	18.059
Drug + Set size + AMI centered	0.013	0.001	0.084	0.005	8.403
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	0.001	0.079	0.005	10.068
Drug + Set size + Drug ✻ Set size	0.013	9.238e -4	0.069	0.004	8.244
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	8.427e -4	0.063	0.004	8.347
Drug + Set size + Delay + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	7.615e -4	0.057	0.003	9.880
Drug + Set size + BDI centered + AMI centered	0.013	6.122e -4	0.046	0.003	8.668
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay	0.013	5.290e -4	0.040	0.002	10.330
Drug + Set size + BDI centered + Drug ✻ Set size	0.013	4.009e -4	0.030	0.002	8.806
Drug + Set size + Delay + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	3.858e -4	0.029	0.002	11.062
Drug + Set size + AMI centered + Drug ✻ Set size	0.013	3.798e -4	0.028	0.002	8.723
Drug + Set size + Delay + BDI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	2.929e -4	0.022	0.001	9.972
Drug + Set size + BDI centered + AMI centered + Drug ✻ Set size	0.013	2.018e -4	0.015	8.762e -4	9.153

Appendix Table 6.18 | Bayesian Repeated Measures ANOVA on Localisation error with drug (patients on placebo versus patients on citalopram), set size (1 item versus 3 items) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Set size + Delay + BDI centered + AMI centered + Drug ✻ Set size + Drug ✻ Delay + Set size ✻ Delay + Drug ✻ Set size ✻ Delay	0.013	1.738e -4	0.013	7.548e -4	11.505
Null model (incl. subject)	0.013	3.385e-37	2.539e-35	1.470e-36	7.524
Delay	0.013	2.341e-37	1.756e-35	1.017e-36	7.562
BDI centered	0.013	1.005e-37	7.537e-36	4.364e-37	7.654
AMI centered	0.013	9.992e-38	7.494e-36	4.339e-37	7.537
Delay + BDI centered	0.013	7.047e-38	5.285e-36	3.060e-37	7.744
Delay + AMI centered	0.013	6.977e-38	5.233e-36	3.030e-37	7.628
Drug	0.013	6.496e-38	4.872e-36	2.821e-37	7.566
Drug + Delay	0.013	4.653e-38	3.490e-36	2.021e-37	7.956
BDI centered + AMI centered	0.013	4.074e-38	3.055e-36	1.769e-37	8.554
Delay + BDI centered + AMI centered	0.013	2.898e-38	2.173e-36	1.258e-37	8.988
Drug + BDI centered	0.013	2.059e-38	1.544e-36	8.941e-38	8.651
Drug + AMI centered	0.013	1.904e-38	1.428e-36	8.267e-38	7.600
Drug + Delay + AMI centered	0.013	1.397e-38	1.048e-36	6.069e-38	8.059
Drug + Delay + BDI centered	0.013	1.380e-38	1.035e-36	5.994e-38	7.867
Drug + Delay + Drug ✻ Delay	0.013	1.117e-38	8.380e-37	4.852e-38	7.923
Drug + BDI centered + AMI centered	0.013	8.983e-39	6.737e-37	3.901e-38	9.019
Drug + Delay + BDI centered + AMI centered	0.013	5.999e-39	4.499e-37	2.605e-38	9.696
Drug + Delay + BDI centered + Drug ✻ Delay	0.013	3.544e-39	2.658e-37	1.539e-38	8.521
Drug + Delay + AMI centered + Drug ✻ Delay	0.013	3.289e-39	2.467e-37	1.428e-38	7.801
Drug + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.013	1.457e-39	1.093e-37	6.329e-39	9.953

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.737	0.315	0.165
Set size	0.737	1.000	∞
Delay	0.737	0.971	12.148
BDI centered	0.500	0.301	0.431
AMI centered	0.500	0.312	0.453
Drug ✻ Set size	0.316	0.079	0.186
Drug ✻ Delay	0.316	0.062	0.144
Set size ✻ Delay	0.316	0.284	0.860
Drug ✻ Set size ✻ Delay	0.053	0.002	0.029

Correlation Analysis on Behavioural Results: PD citalopram versus PD placebo

Appendix Table 6.19 | Pearson and Spearman's correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and identification accuracy, reaction time (seconds) and localisation error (pixels).

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
OMT_effect of drug on accuracy (citalopram-placebo)	Pearson's r	-0.504 *	0.071
	p-value	0.028	0.773
	Spearman's rho	-0.209	-0.019
	p-value	0.391	0.940
OMT_effect of drug on LOGRT(citalopram-placebo)	Pearson's r	0.131	0.396
	p-value	0.592	0.094
	Spearman's rho	0.212	0.480 *
	p-value	0.384	0.038
OMT_effect of drug on locerror(citalopram-placebo)	Pearson's r	0.200	-0.217
	p-value	0.412	0.372
	Spearman's rho	0.220	-0.224
	p-value	0.364	0.358

* p < .05, ** p < .01, *** p < .001

Appendix Table 6.20 | Bayesian Pearson and Kendall's Tau Correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and identification accuracy, reaction time (seconds) and localisation error (pixels).

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
OMT_effect of drug on accuracy (citalopram-placebo)	Pearson's r	-0.504	0.071
	BF_{10}	2.693	0.295
	Kendall's tau	-0.156	-0.019
	BF_{10}	0.442	0.294
OMT_effect of drug on LOGRT(citalopram-placebo)	Pearson's r	0.131	0.396
	BF_{10}	0.324	1.052
	Kendall's tau	0.159	0.363
	BF_{10}	0.448	2.672
OMT_effect of drug on locerror(citalopram-placebo)	Pearson's r	0.200	-0.217

Appendix Table 6.20 | Bayesian Pearson and Kendall's Tau Correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and identification accuracy, reaction time (seconds) and localisation error (pixels).

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
	BF_{10}	0.389	0.412
	Kendall's tau	0.147	-0.181
	BF_{10}	0.421	0.509

* $BF_{10} > 10$, ** $BF_{10} > 30$, *** $BF_{10} > 100$

Mixture Model Analysis: PD placebo versus PD citalopram

Appendix Table 6.21 | Repeated Measures ANOVA on Imprecision with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	1.659	1	1.659	0.020	0.888	0.001
Drug ✻ BDI centered	20.297	1	20.297	0.250	0.624	0.015
Drug ✻ AMI centered	11.524	1	11.524	0.142	0.712	0.009
Residuals	1301.258	16	81.329			
Delay	2011.981	1	2011.981	17.030	< .001	0.516
Delay ✻ BDI centered	177.175	1	177.175	1.500	0.238	0.086
Delay ✻ AMI centered	139.853	1	139.853	1.184	0.293	0.069
Residuals	1890.300	16	118.144			
Drug ✻ Delay	379.997	1	379.997	11.559	0.004	0.419
Drug ✻ Delay ✻ BDI centered	48.200	1	48.200	1.466	0.244	0.084
Drug ✻ Delay ✻ AMI centered	1.617	1	1.617	0.049	0.827	0.003
Residuals	526.005	16	32.875			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	364.326	1	364.326	0.375	0.549
AMI centered	880.012	1	880.012	0.907	0.355
Residuals	15531.646	16	970.728		

Note. Type III Sum of Squares

Post Hoc Comparisons - Drug ✻ Delay

		Mean Difference	SE	t	p_{holm}
PD.placebo, X1.second	PD.citalopram., X1.second	4.693	2.452	1.914	0.145
	PD.placebo, X4.seconds	-5.865	2.819	-2.080	0.145
	PD.citalopram., X4.seconds	-10.041	3.240	-3.099	0.016
PD.citalopram., X1.second	PD.placebo, X4.seconds	-10.557	3.240	-3.258	0.014
	PD.citalopram., X4.seconds	-14.733	2.819	-5.226	< .001
PD.placebo, X4.seconds	PD.citalopram., X4.seconds	-4.176	2.452	-1.703	0.145

Note. P-value adjusted for comparing a family of 6

Appendix Table 6.22 | Bayesian Repeated Measures ANOVA on Imprecision with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay	0.200	0.589	5.738	1.000	
Drug + Delay + Drug ✻ Delay	0.200	0.275	1.518	0.467	2.380
Drug + Delay	0.200	0.135	0.626	0.230	2.126
Null model (incl. subject)	0.200	2.151e-4	8.607e-4	3.651e-4	1.662
Drug	0.200	5.056e-5	2.023e-4	8.581e-5	2.255

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.411	0.464
Delay	0.600	1.000	2508.478
Drug ✻ Delay	0.200	0.275	1.518

Appendix Table 6.23 | Repeated Measures ANOVA on Misbinding with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	0.002	1	0.002	0.185	0.673	0.011
Drug ✻ BDI centered	0.030	1	0.030	2.707	0.119	0.145
Drug ✻ AMI centered	0.038	1	0.038	3.490	0.080	0.179
Residuals	0.175	16	0.011			
Delay	0.020	1	0.020	3.148	0.095	0.164
Delay ✻ BDI centered	0.002	1	0.002	0.269	0.611	0.017
Delay ✻ AMI centered	7.105e-4	1	7.105e-4	0.113	0.741	0.007
Residuals	0.101	16	0.006			
Drug ✻ Delay	4.021e-5	1	4.021e-5	0.008	0.931	4.781e-4
Drug ✻ Delay ✻ BDI centered	1.033e-4	1	1.033e-4	0.020	0.890	0.001
Drug ✻ Delay ✻ AMI centered	0.001	1	0.001	0.220	0.645	0.014
Residuals	0.084	16	0.005			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	6.171e-4	1	6.171e-4	0.019	0.893
AMI centered	0.023	1	0.023	0.696	0.416
Residuals	0.532	16	0.033		

Note. Type III Sum of Squares

Appendix Table 6.24 | Bayesian Repeated Measures ANOVA on Misbinding with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.192	4.505	1.000	
Delay	0.050	0.143	3.167	0.746	1.179
AMI centered	0.050	0.107	2.275	0.558	1.477
BDI centered	0.050	0.081	1.670	0.422	0.776
Delay + AMI centered	0.050	0.079	1.628	0.412	1.796
Delay + BDI centered	0.050	0.065	1.318	0.338	5.090
BDI centered + AMI centered	0.050	0.057	1.146	0.297	1.814
Drug	0.050	0.053	1.060	0.276	4.126
Delay + BDI centered + AMI centered	0.050	0.045	0.892	0.234	2.165
Drug + Delay	0.050	0.038	0.761	0.201	2.064
Drug + AMI centered	0.050	0.028	0.541	0.144	1.912
Drug + Delay + AMI centered	0.050	0.022	0.425	0.114	2.276
Drug + BDI centered	0.050	0.022	0.425	0.114	1.942
Drug + Delay + BDI centered	0.050	0.016	0.304	0.082	1.974
Drug + BDI centered + AMI centered	0.050	0.015	0.292	0.079	2.251
Drug + Delay + BDI centered + AMI centered	0.050	0.011	0.220	0.060	2.695
Drug + Delay + Drug ✻ Delay	0.050	0.011	0.218	0.059	1.568
Drug + Delay + AMI centered + Drug ✻ Delay	0.050	0.007	0.127	0.035	2.757
Drug + Delay + BDI centered + Drug ✻ Delay	0.050	0.005	0.104	0.028	4.176
Drug + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.050	0.004	0.070	0.019	3.208

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.232	0.202
Delay	0.600	0.446	0.537
BDI centered	0.500	0.321	0.472
AMI centered	0.500	0.374	0.598
Drug ✻ Delay	0.200	0.027	0.111

Appendix Table 6.25 | Repeated Measures ANOVA on Guessing with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	3.905e-4	1	3.905e-4	0.294	0.595	0.018
Drug ✻ BDI centered	3.489e-5	1	3.489e-5	0.026	0.873	0.002
Drug ✻ AMI centered	3.562e-4	1	3.562e-4	0.268	0.612	0.016
Residuals	0.021	16	0.001			
Delay	0.010	1	0.010	6.424	0.022	0.286
Delay ✻ BDI centered	1.348e-4	1	1.348e-4	0.091	0.767	0.006

Appendix Table 6.25 | Repeated Measures ANOVA on Guessing with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Delay ✻ AMI centerd	0.002	1	0.002	1.484	0.241	0.085
Residuals	0.024	16	0.001			
Drug ✻ Delay	2.516e-5	1	2.516e-5	0.024	0.880	0.001
Drug ✻ Delay ✻ BDI centered	9.481e-6	1	9.481e-6	0.009	0.926	5.580e-4
Drug ✻ Delay ✻ AMI centered	9.120e-4	1	9.120e-4	0.859	0.368	0.051
Residuals	0.017	16	0.001			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	3.868e-4	1	3.868e-4	0.173	0.683
AMI centered	0.003	1	0.003	1.202	0.289
Residuals	0.036	16	0.002		

Note. Type III Sum of Squares

Appendix Table 6.26 | Bayesian Repeated Measures ANOVA on Guessing with drug (patients on placebo versus patients on citalopram) and delay (1 second versus 4 seconds) as "within-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Delay	0.050	0.325	9.143	1.000	
Delay + AMI centered	0.050	0.150	3.350	0.461	2.133
Delay + BDI centered	0.050	0.106	2.260	0.327	1.914
Drug + Delay	0.050	0.092	1.933	0.284	3.997
Delay + BDI centered + AMI centered	0.050	0.072	1.479	0.222	3.260
Null model (incl. subject)	0.050	0.041	0.817	0.127	1.611
Drug + Delay + AMI centered	0.050	0.041	0.804	0.125	2.290
Drug + Delay + BDI centered	0.050	0.030	0.578	0.091	3.023
Drug + Delay + Drug ✻ Delay	0.050	0.027	0.534	0.084	4.191
Drug + Delay + BDI centered + AMI centered	0.050	0.021	0.409	0.065	3.805
AMI centered	0.050	0.019	0.368	0.058	1.718
BDI centered	0.050	0.014	0.268	0.043	1.986
Drug + Delay + AMI centered + Drug ✻ Delay	0.050	0.014	0.267	0.043	5.417
Drug	0.050	0.011	0.214	0.034	2.327
BDI centered + AMI centered	0.050	0.009	0.182	0.029	3.185
Drug + Delay + BDI centered + Drug ✻ Delay	0.050	0.009	0.173	0.028	2.734
Drug + Delay + BDI centered + AMI centered + Drug ✻ Delay	0.050	0.007	0.129	0.021	4.078
Drug + AMI centered	0.050	0.005	0.098	0.016	3.111
Drug + BDI centered	0.050	0.004	0.071	0.012	3.487
Drug + BDI centered + AMI centered	0.050	0.003	0.048	0.008	3.766

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.263	0.238
Delay	0.600	0.894	5.612
BDI centered	0.500	0.275	0.379
AMI centered	0.500	0.341	0.516
Drug ✻ Delay	0.200	0.057	0.242

Correlation Analysis on Mixture Model Results: PD citalopram versus PD placebo

Appendix Table 6.27 | Pearson and Spearman's correlations: Relationship between the effect of B (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placeb and imprecision (pixels), misbinding and guessing.

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
OMT_ effect of drug on imprecision(citalopram-placebo)	Pearson's r	0.509 *	-0.162
	p-value	0.026	0.506
	Spearman's rho	0.155	-0.268
	p-value	0.526	0.268
OMT_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.179	-0.263
	p-value	0.463	0.277
	Spearman's rho	-0.112	-0.093
	p-value	0.649	0.706
OMT_effect of drug on guess(citalopram-placebo)	Pearson's r	0.438	0.073
	p-value	0.061	0.766
	Spearman's rho	0.092	-0.037
	p-value	0.708	0.880

* p < .05, ** p < .01, *** p < .001

Appendix Table 6.27 | Bayesian Pearson and Spearman's correlations: Relationship between the effect of B (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placeb and imprecision (pixels), misbinding and guessing.

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
OMT_ effect of drug on imprecision(citalopram-placebo)	Pearson's r	0.509	-0.162
	BF_{10}	2.847	0.349
	Kendall's tau	0.122	-0.206
	BF_{10}	0.377	0.596
OMT_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.179	-0.263
	BF_{10}	0.365	0.493
	Kendall's tau	-0.086	-0.085
	BF_{10}	0.331	0.330
OMT_effect of drug on guess(citalopram-placebo)	Pearson's r	0.438	0.073
	BF_{10}	1.460	0.296
	Kendall's tau	0.073	-0.024
	BF_{10}	0.320	0.295

* BF_{10} > 10, ** BF_{10} > 30, *** BF_{10} > 100

Experiment 2

Behavioural Analysis: Controls versus PD placebo

Appendix Table 6.28 | Repeated Measures ANOVA on Identification Accuracy with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion	None	0.010	[a] 2.000	[a] 0.005	[a] 3.437	[a] 0.038	[a] 0.087
	Greenhouse-Geisser	0.010	1.207	0.008	3.437	0.063	0.087
Emotion ✻ Group	None	0.005	[a] 2.000	[a] 0.002	[a] 1.641	[a] 0.201	[a] 0.044
	Greenhouse-Geisser	0.005	1.207	0.004	1.641	0.209	0.044
Emotion ✻ AMI centered	None	0.003	[a] 2.000	[a] 0.001	[a] 1.007	[a] 0.371	[a] 0.027
	Greenhouse-Geisser	0.003	1.207	0.002	1.007	0.337	0.027
Emotion ✻ BDI centered	None	5.832e-4	[a] 2.000	[a] 2.916e-4	[a] 0.198	[a] 0.821	[a] 0.005
	Greenhouse-Geisser	5.832e-4	1.207	4.831e-4	0.198	0.705	0.005
Residuals	None	0.106	72.000	0.001			
	Greenhouse-Geisser	0.106	43.460	0.002			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.002	1	0.002	0.614	0.438	0.017
AMI centered	0.005	1	0.005	1.396	0.245	0.037
BDI centered	2.783e-4	1	2.783e-4	0.072	0.790	0.002
Residuals	0.139	36	0.004			

Note. Type III Sum of Squares

Appendix Table 6.29 | Bayesian Repeated Measures ANOVA on Identification Accuracy with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Emotion	0.050	0.183	4.262	1.000	
Null model (incl. subject)	0.050	0.141	3.123	0.770	0.605
Emotion + AMI centered	0.050	0.102	2.165	0.558	1.546
AMI centered	0.050	0.075	1.546	0.411	1.982
Emotion + Group	0.050	0.071	1.455	0.388	1.887

Appendix Table 6.29 | Bayesian Repeated Measures ANOVA on Identification Accuracy with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Emotion + BDI centered	0.050	0.058	1.162	0.314	1.355
Group	0.050	0.053	1.053	0.287	1.662
BDI centered	0.050	0.044	0.871	0.239	1.788
Emotion + AMI centered + BDI centered	0.050	0.042	0.827	0.228	3.058
Emotion + Group + AMI centered	0.050	0.036	0.717	0.198	1.579
Emotion + Group + Emotion ✻ Group	0.050	0.032	0.636	0.177	1.684
AMI centered + BDI centered	0.050	0.030	0.596	0.166	2.452
Group + AMI centered	0.050	0.028	0.553	0.154	2.163
Emotion + Group + BDI centered	0.050	0.023	0.448	0.126	3.693
Emotion + Group + AMI centered + Emotion ✻ Group	0.050	0.019	0.369	0.104	5.247
Emotion + Group + AMI centered + BDI centered	0.050	0.016	0.318	0.090	3.711
Group + BDI centered	0.050	0.016	0.313	0.089	2.311
Group + AMI centered + BDI centered	0.050	0.012	0.226	0.064	2.864
Emotion + Group + BDI centered + Emotion ✻ Group	0.050	0.010	0.198	0.056	1.805
Emotion + Group + AMI centered + BDI centered + Emotion ✻ Group	0.050	0.007	0.135	0.039	4.294

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Emotion	0.600	0.601	1.003
Group	0.600	0.325	0.320
AMI centered	0.500	0.369	0.584
BDI centered	0.500	0.258	0.348
Emotion ✻ Group	0.200	0.069	0.296

Appendix Table 6.30 | Repeated Measures ANOVA on Reaction Time with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion	0.672	2	0.336	95.416	< .001	0.726
Emotion ✻ Group	0.005	2	0.002	0.666	0.517	0.018
Emotion ✻ AMI centered	0.019	2	0.009	2.685	0.075	0.069
Emotion ✻ BDI centered	0.031	2	0.015	4.370	0.016	0.108
Residuals	0.254	72	0.004			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.652	1	0.652	8.656	0.006	0.194
AMI centered	0.090	1	0.090	1.200	0.281	0.032
BDI centered	0.145	1	0.145	1.932	0.173	0.051

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	2.712	36	0.075			

Note. Type III Sum of Squares

Post Hoc Comparisons - Emotion

		Mean Difference	SE	t	p_{holm}
neutral	angry	0.059	0.013	4.409	< .001
	happy	0.180	0.013	13.578	< .001
angry	happy	0.122	0.013	9.169	< .001

Note. P-value adjusted for comparing a family of 3

Note. Results are averaged over the levels of: Group

Appendix Table 6.31 | Bayesian Repeated Measures ANOVA on Reaction Time with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Emotion + Group	0.050	0.250	6.323	1.000	
Emotion + Group + AMI centered + BDI centered	0.050	0.172	3.955	0.690	5.309
Emotion + Group + BDI centered	0.050	0.163	3.694	0.652	3.336
Emotion + Group + AMI centered	0.050	0.156	3.509	0.624	5.358
Emotion + Group + Emotion ✻ Group	0.050	0.053	1.054	0.211	5.276
Emotion	0.050	0.040	0.793	0.160	2.978
Emotion + Group + BDI centered + Emotion ✻ Group	0.050	0.032	0.630	0.129	3.693
Emotion + Group + AMI centered + BDI centered + Emotion ✻ Group	0.050	0.032	0.620	0.127	7.748
Emotion + Group + AMI centered + Emotion ✻ Group	0.050	0.030	0.582	0.119	6.966
Emotion + BDI centered	0.050	0.027	0.527	0.108	2.813
Emotion + AMI centered + BDI centered	0.050	0.024	0.459	0.094	4.973
Emotion + AMI centered	0.050	0.023	0.442	0.091	4.928
Group	0.050	2.405e-18	4.569e-17	9.631e-18	2.592
Group + BDI centered	0.050	1.357e-18	2.578e-17	5.433e-18	3.451
Group + AMI centered + BDI centered	0.050	1.070e-18	2.034e-17	4.287e-18	3.279
Group + AMI centered	0.050	1.060e-18	2.014e-17	4.245e-18	3.124
Null model (incl. subject)	0.050	2.903e-19	5.516e-18	1.163e-18	2.238
BDI centered	0.050	1.647e-19	3.129e-18	6.595e-19	2.711
AMI centered	0.050	1.295e-19	2.461e-18	5.187e-19	3.048
AMI centered + BDI centered	0.050	1.243e-19	2.362e-18	4.979e-19	3.220

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Emotion	0.600	1.000	∞
Group	0.600	0.887	5.216
AMI centered	0.500	0.436	0.772

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
BDI centered	0.500	0.449	0.816
Emotion ✻ Group	0.200	0.146	0.684

Appendix Table 6.32 | Repeated Measures ANOVA on Localisation error with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion	1683.067	2	841.534	0.640	0.530	0.017
Emotion ✻ Group	282.815	2	141.407	0.108	0.898	0.003
Emotion ✻ AMI centered	11357.493	2	5678.747	4.321	0.017	0.107
Emotion ✻ BDI centered	5077.990	2	2538.995	1.932	0.152	0.051
Residuals	94619.577	72	1314.161			

Note. Type III Sum of Squares

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	92342.154	1	92342.154	2.247	0.143	0.059
AMI centered	61789.465	1	61789.465	1.504	0.228	0.040
BDI centered	22401.015	1	22401.015	0.545	0.465	0.015
Residuals	1.479e+6	36	41095.242			

Note. Type III Sum of Squares

Appendix Table 6.33 | Bayesian Repeated Measures ANOVA on Localisation error with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Group + AMI centered	0.050	0.144	3.195	1.000	
AMI centered	0.050	0.133	2.916	0.924	4.068
Group + BDI centered	0.050	0.110	2.342	0.762	4.107
BDI centered	0.050	0.105	2.227	0.729	5.880
Group + AMI centered + BDI centered	0.050	0.100	2.108	0.694	4.174
Group	0.050	0.097	2.052	0.677	4.018
AMI centered + BDI centered	0.050	0.096	2.011	0.665	4.129
Null model (incl. subject)	0.050	0.096	2.010	0.665	2.891
Emotion + AMI centered	0.050	0.020	0.381	0.137	4.585
Emotion + Group + AMI centered	0.050	0.018	0.348	0.125	17.056
Emotion + Group + AMI centered + BDI centered	0.050	0.014	0.272	0.098	5.482
Emotion + AMI centered + BDI centered	0.050	0.014	0.262	0.095	5.246
Emotion + Group	0.050	0.013	0.256	0.092	5.109
Emotion + BDI centered	0.050	0.013	0.242	0.087	13.307
Emotion	0.050	0.013	0.241	0.087	2.935
Emotion + Group + BDI centered	0.050	0.009	0.169	0.061	9.629
Emotion + Group + BDI centered + Emotion ✻ Group	0.050	0.002	0.040	0.015	30.222
Emotion + Group + AMI centered + BDI centered + Emotion ✻ Group	0.050	0.002	0.036	0.013	7.784

Appendix Table 6.33 | Bayesian Repeated Measures ANOVA on Localisation error with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Emotion + Group + AMI centered + Emotion ✻ Group	0.050	0.002	0.032	0.012	5.489
Emotion + Group + Emotion ✻ Group	0.050	0.001	0.026	0.009	11.243

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Emotion	0.600	0.120	0.091
Group	0.600	0.512	0.700
AMI centered	0.500	0.542	1.181
BDI centered	0.500	0.463	0.863
Emotion ✻ Group	0.200	0.007	0.028

Mixture Model Analysis: Controls versus PD placebo

Appendix Table 6.34 | Repeated Measures ANOVA on Imprecision with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion	None	1045.266	[a] 2.000	[a] 522.633	[a] 0.727	[a] 0.487	[a] 0.020
	Greenhouse-Geisser	1045.266	1.154	906.132	0.727	0.417	0.020
Emotion ✻ Group	None	236.871	[a] 2.000	[a] 118.436	[a] 0.165	[a] 0.848	[a] 0.005
	Greenhouse-Geisser	236.871	1.154	205.342	0.165	0.723	0.005
Emotion ✻ AMI centered	None	1846.022	[a] 2.000	[a] 923.011	[a] 1.285	[a] 0.283	[a] 0.034
	Greenhouse-Geisser	1846.022	1.154	1600.301	1.285	0.270	0.034
Emotion ✻ BDI centered	None	10871.332	[a] 2.000	[a] 5435.666	[a] 7.565	[a] 0.001	[a] 0.174
	Greenhouse-Geisser	10871.332	1.154	9424.267	7.565	0.007	0.174
Residuals	None	51736.790	72.000	718.567			
	Greenhouse-Geisser	51736.790	41.528	1245.839			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	4956.930	1	4956.930	0.799	0.377	0.022
AMI centered	7897.654	1	7897.654	1.272	0.267	0.034

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
BDI centered	24958.381	1	24958.381	4.021	0.053	0.100
Residuals	223466.293	36	6207.397			

Note. Type III Sum of Squares

Appendix Table 6.35 | Bayesian Repeated Measures ANOVA on Imprecision with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
BDI centered	0.050	0.279	7.358	1.000	
AMI centered + BDI centered	0.050	0.173	3.966	0.619	1.988
Group + BDI centered	0.050	0.148	3.299	0.530	0.921
AMI centered	0.050	0.098	2.063	0.351	1.059
Group + AMI centered + BDI centered	0.050	0.092	1.928	0.330	2.296
Group + AMI centered	0.050	0.050	1.003	0.180	1.267
Emotion + BDI centered	0.050	0.037	0.724	0.132	0.780
Null model (incl. subject)	0.050	0.025	0.489	0.090	0.404
Emotion + AMI centered + BDI centered	0.050	0.023	0.453	0.083	3.089
Emotion + Group + BDI centered	0.050	0.020	0.379	0.070	2.721
Emotion + AMI centered	0.050	0.013	0.250	0.047	1.415
Group	0.050	0.013	0.250	0.047	0.846
Emotion + Group + AMI centered + BDI centered	0.050	0.012	0.227	0.042	3.703
Emotion + Group + AMI centered	0.050	0.007	0.126	0.024	2.055
Emotion	0.050	0.003	0.063	0.012	0.946
Emotion + Group + BDI centered + Emotion ✻ Group	0.050	0.003	0.054	0.010	1.722
Emotion + Group + AMI centered + BDI centered + Emotion ✻ Group	0.050	0.002	0.034	0.006	5.105
Emotion + Group	0.050	0.002	0.033	0.006	1.183
Emotion + Group + AMI centered + Emotion ✻ Group	0.050	0.001	0.019	0.004	2.635
Emotion + Group + Emotion ✻ Group	0.050	2.529e-4	0.005	9.058e-4	1.554

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Emotion	0.600	0.122	0.092
Group	0.600	0.349	0.357
AMI centered	0.500	0.470	0.888
BDI centered	0.500	0.788	3.715
Emotion ✻ Group	0.200	0.006	0.024

Appendix Table 6.36 | Repeated Measures ANOVA Misbinding with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion	None	0.001	[a] 2.000	[a] 6.424e-4	[a] 0.193	[a] 0.825	[a] 0.005
	Greenhouse-Geisser	0.001	1.477	8.701e-4	0.193	0.758	0.005
Emotion ✻ Group	None	0.006	[a] 2.000	[a] 0.003	[a] 0.858	[a] 0.428	[a] 0.023
	Greenhouse-Geisser	0.006	1.477	0.004	0.858	0.400	0.023
Emotion ✻ AMI centered	None	0.001	[a] 2.000	[a] 7.407e-4	[a] 0.222	[a] 0.801	[a] 0.006
	Greenhouse-Geisser	0.001	1.477	0.001	0.222	0.733	0.006
Emotion ✻ BDI centered	None	0.006	[a] 2.000	[a] 0.003	[a] 0.894	[a] 0.414	[a] 0.024
	Greenhouse-Geisser	0.006	1.477	0.004	0.894	0.387	0.024
Residuals	None	0.240	72.000	0.003			
	Greenhouse-Geisser	0.240	53.154	0.005			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.029	1	0.029	3.152	0.084	0.081
AMI centered	0.011	1	0.011	1.260	0.269	0.034
BDI centered	0.013	1	0.013	1.376	0.248	0.037
Residuals	0.328	36	0.009			

Note. Type III Sum of Squares

Appendix Table 6.37 | Bayesian Repeated Measures ANOVA on Misbinding with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Group	0.050	0.239	5.971	1.000	
Null model (incl. subject)	0.050	0.219	5.325	0.915	1.128
Group + BDI centered	0.050	0.089	1.861	0.373	2.886
Group + AMI centered	0.050	0.086	1.781	0.358	2.364
BDI centered	0.050	0.081	1.670	0.338	1.537
AMI centered	0.050	0.076	1.570	0.319	2.164
Group + AMI centered + BDI centered	0.050	0.058	1.180	0.245	2.769
AMI centered + BDI centered	0.050	0.057	1.146	0.238	2.490
Emotion + Group	0.050	0.022	0.419	0.090	1.674
Emotion	0.050	0.020	0.385	0.083	1.435
Emotion + Group + AMI centered	0.050	0.008	0.154	0.034	3.544
Emotion + Group + BDI centered	0.050	0.008	0.153	0.033	2.637
Emotion + BDI centered	0.050	0.008	0.144	0.032	2.125
Emotion + AMI centered	0.050	0.007	0.140	0.031	3.140
Emotion + Group + Emotion ✻ Group	0.050	0.006	0.109	0.024	2.397

Appendix Table 6.37 | Bayesian Repeated Measures ANOVA on Misbinding with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Emotion + Group + AMI centered + BDI centered	0.050	0.006	0.106	0.023	4.125
Emotion + AMI centered + BDI centered	0.050	0.005	0.096	0.021	3.739
Emotion + Group + AMI centered + Emotion ✻ Group	0.050	0.002	0.042	0.009	4.999
Emotion + Group + BDI centered + Emotion ✻ Group	0.050	0.002	0.040	0.009	3.493
Emotion + Group + AMI centered + BDI centered + Emotion ✻ Group	0.050	0.002	0.031	0.007	5.535

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Emotion	0.600	0.095	0.070
Group	0.600	0.527	0.744
AMI centered	0.500	0.307	0.443
BDI centered	0.500	0.315	0.460
Emotion ✻ Group	0.200	0.012	0.047

Appendix Table 6.38 | Repeated Measures ANOVA on Guessing with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion	None	0.006	[a] 2.000	[a] 0.003	[a] 0.897	[a] 0.412	[a] 0.024
	Greenhouse-Geisser	0.006	1.276	0.004	0.897	0.373	0.024
Emotion ✻ Group	None	0.004	[a] 2.000	[a] 0.002	[a] 0.566	[a] 0.571	[a] 0.015
	Greenhouse-Geisser	0.004	1.276	0.003	0.566	0.496	0.015
Emotion ✻ AMI centered	None	0.008	[a] 2.000	[a] 0.004	[a] 1.335	[a] 0.270	[a] 0.036
	Greenhouse-Geisser	0.008	1.276	0.007	1.335	0.263	0.036
Emotion ✻ BDI centered	None	0.012	[a] 2.000	[a] 0.006	[a] 1.932	[a] 0.152	[a] 0.051
	Greenhouse-Geisser	0.012	1.276	0.010	1.932	0.169	0.051
Residuals	None	0.228	72.000	0.003			
	Greenhouse-Geisser	0.228	45.940	0.005			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Group	0.015	1	0.015	0.240	0.627	0.007
AMI centered	0.059	1	0.059	0.950	0.336	0.026
BDI centered	0.181	1	0.181	2.922	0.096	0.075

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	2.228	36	0.062			

Note. Type III Sum of Squares

Appendix Table 6.39 | Bayesian Repeated Measures ANOVA on Guessing with emotion (neutral, angry, happy) as "within-subjects" factors, group (controls versus patients) as "between-subjects" factors and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
BDI centered	0.050	0.203	4.826	1.000	
AMI centered + BDI centered	0.050	0.139	3.057	0.684	2.661
Group + BDI centered	0.050	0.121	2.628	0.600	3.030
AMI centered	0.050	0.106	2.252	0.523	1.048
Emotion + BDI centered	0.050	0.097	2.046	0.480	63.850
Group + AMI centered + BDI centered	0.050	0.080	1.656	0.396	2.957
Group + AMI centered	0.050	0.063	1.288	0.313	2.875
Null model (incl. subject)	0.050	0.048	0.965	0.239	0.599
Group	0.050	0.029	0.561	0.142	2.782
Emotion + AMI centered + BDI centered	0.050	0.023	0.450	0.114	3.969
Emotion + Group + BDI centered	0.050	0.021	0.413	0.105	4.528
Emotion + AMI centered	0.050	0.018	0.356	0.091	1.856
Emotion + Group + AMI centered + BDI centered	0.050	0.015	0.293	0.075	4.349
Emotion + Group + AMI centered	0.050	0.012	0.233	0.060	4.281
Emotion	0.050	0.008	0.158	0.041	0.921
Emotion + Group	0.050	0.005	0.090	0.023	4.096
Emotion + Group + BDI centered + Emotion ✻ Group	0.050	0.004	0.078	0.020	6.369
Emotion + Group + AMI centered + BDI centered + Emotion ✻ Group	0.050	0.003	0.060	0.015	5.996
Emotion + Group + AMI centered + Emotion ✻ Group	0.050	0.002	0.043	0.011	5.718
Emotion + Group + Emotion ✻ Group	0.050	0.001	0.020	0.005	5.695

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Emotion	0.600	0.211	0.178
Group	0.600	0.358	0.371
AMI centered	0.500	0.462	0.860
BDI centered	0.500	0.707	2.410
Emotion ✻ Group	0.200	0.011	0.043

Behavioural Analysis: PD placebo versus PD citalopram

Appendix Table 6.40 | Repeated Measures ANOVA on Identification Accuracy with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	None	2.971e-10	1.000	2.971e-10	8.036e-8	1.000	4.727e-9
Drug ✻ AMI centered	None	0.002	1.000	0.002	0.628	0.439	0.036
Drug ✻ BDI centered	None	5.825e -4	1.000	5.825e -4	0.158	0.696	0.009
Residuals	None	0.063	17.000	0.004			
Emotion	None	0.011	[a] 2.000	[a] 0.005	[a] 2.005	[a] 0.150	[a] 0.106
	Greenhouse-Geisser	0.011	1.300	0.008	2.005	0.169	0.106
Emotion ✻ AMI centered	None	0.004	[a] 2.000	[a] 0.002	[a] 0.664	[a] 0.521	[a] 0.038
	Greenhouse-Geisser	0.004	1.300	0.003	0.664	0.462	0.038
Emotion ✻ BDI centered	None	0.002	[a] 2.000	[a] 8.654e -4	[a] 0.323	[a] 0.726	[a] 0.019
	Greenhouse-Geisser	0.002	1.300	0.001	0.323	0.634	0.019
Residuals	None	0.091	34.000	0.003			
	Greenhouse-Geisser	0.091	22.107	0.004			
Drug ✻ Emotion	None	0.006	2.000	0.003	2.889	0.069	0.145
	Greenhouse-Geisser	0.006	1.799	0.003	2.889	0.076	0.145
Drug ✻ Emotion ✻ AMI centered	None	0.002	2.000	0.001	1.071	0.354	0.059
	Greenhouse-Geisser	0.002	1.799	0.001	1.071	0.349	0.059
Drug ✻ Emotion ✻ BDI centered	None	9.559e -4	2.000	4.780e -4	0.444	0.645	0.025
	Greenhouse-Geisser	9.559e -4	1.799	5.314e -4	0.444	0.625	0.025
Residuals	None	0.037	34.000	0.001			
	Greenhouse-Geisser	0.037	30.579	0.001			

Note. Sphericity corrections not available for factors with 2 levels.

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
AMI centered	0.007	1	0.007	0.972	0.338
BDI centered	0.004	1	0.004	0.541	0.472
Residuals	0.119	17	0.007		

Note. Type III Sum of Squares

Appendix Table 6.41 | Bayesian Repeated Measures ANOVA on Identification Accuracy with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.263	6.764	1.000	
Emotion	0.050	0.160	3.632	0.611	0.540
AMI centered	0.050	0.101	2.129	0.384	2.074
BDI centered	0.050	0.086	1.797	0.329	1.108
Emotion + AMI centered	0.050	0.064	1.298	0.244	2.791
BDI centered + AMI centered	0.050	0.055	1.102	0.209	2.439
Emotion + BDI centered	0.050	0.052	1.049	0.199	1.190
Drug	0.050	0.051	1.022	0.195	3.248
Emotion + BDI centered + AMI centered	0.050	0.036	0.707	0.137	3.254
Drug + Emotion	0.050	0.031	0.617	0.120	3.247
Drug + AMI centered	0.050	0.020	0.386	0.076	2.574
Drug + BDI centered	0.050	0.016	0.313	0.062	1.699
Drug + Emotion + AMI centered	0.050	0.012	0.238	0.047	3.476
Drug + Emotion + Drug ✻ Emotion	0.050	0.012	0.233	0.046	1.879
Drug + BDI centered + AMI centered	0.050	0.011	0.208	0.041	3.070
Drug + Emotion + BDI centered	0.050	0.010	0.200	0.040	2.179
Drug + Emotion + BDI centered + AMI centered	0.050	0.007	0.132	0.026	3.944
Drug + Emotion + AMI centered + Drug ✻ Emotion	0.050	0.005	0.093	0.019	4.453
Drug + Emotion + BDI centered + Drug ✻ Emotion	0.050	0.004	0.078	0.016	1.953
Drug + Emotion + BDI centered + AMI centered + Drug ✻ Emotion	0.050	0.003	0.049	0.010	5.209

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.183	0.149
Emotion	0.600	0.397	0.440
BDI centered	0.500	0.280	0.390
AMI centered	0.500	0.313	0.455
Drug ✻ Emotion	0.200	0.024	0.097

Appendix Table 6.42 | Repeated Measures ANOVA on Reaction Time with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	0.001	1	0.001	0.040	0.843	0.002
Drug ✻ BDI centered	9.510e-4	1	9.510e-4	0.037	0.850	0.002
Drug ✻ AMI centered	0.002	1	0.002	0.095	0.761	0.006
Residuals	0.439	17	0.026			
Emotion	0.530	2	0.265	66.532	< .001	0.796
Emotion ✻ BDI centered	0.011	2	0.006	1.407	0.259	0.076

Appendix Table 6.42 | Repeated Measures ANOVA on Reaction Time with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Emotion ✻ AMI centered	0.011	2	0.006	1.409	0.258	0.077
Residuals	0.136	34	0.004			
Drug ✻ Emotion	0.008	2	0.004	1.103	0.343	0.061
Drug ✻ Emotion ✻ BDI centered	0.008	2	0.004	1.039	0.365	0.058
Drug ✻ Emotion ✻ AMI centered	0.002	2	9.874e-4	0.267	0.767	0.015
Residuals	0.126	34	0.004			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	0.051	1	0.051	0.184	0.673
AMI centered	0.015	1	0.015	0.055	0.817
Residuals	4.673	17	0.275		

Note. Type III Sum of Squares

Post Hoc Comparisons - Emotion

		Mean Difference	SE	t	p_{holm}
neutral	angry	0.054	0.014	3.811	< .001
	happy	0.160	0.014	11.335	< .001
angry	happy	0.106	0.014	7.524	< .001

Note. P-value adjusted for comparing a family of 3

Note. Results are averaged over the levels of: Drug

Appendix Table 6.43 | Bayesian Repeated Measures ANOVA on Reaction Time with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Emotion	0.050	0.295	7.951	1.000	
Emotion + BDI centered	0.050	0.188	4.394	0.637	3.903
Emotion + AMI centered	0.050	0.180	4.157	0.608	3.296
Emotion + BDI centered + AMI centered	0.050	0.147	3.267	0.497	3.490
Drug + Emotion	0.050	0.058	1.174	0.197	1.857
Drug + Emotion + BDI centered	0.050	0.037	0.723	0.124	2.040
Drug + Emotion + AMI centered	0.050	0.036	0.713	0.123	3.993
Drug + Emotion + BDI centered + AMI centered	0.050	0.027	0.536	0.093	4.331
Drug + Emotion + Drug ✻ Emotion	0.050	0.012	0.224	0.040	2.493
Drug + Emotion + BDI centered + Drug ✻ Emotion	0.050	0.007	0.142	0.025	3.620
Drug + Emotion + AMI centered + Drug ✻ Emotion	0.050	0.007	0.141	0.025	5.482

Appendix Table 6.43 | Bayesian Repeated Measures ANOVA on Reaction Time with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug + Emotion + BDI centered + AMI centered + Drug ✻ Emotion	0.050	0.006	0.115	0.020	5.436
Null model (incl. subject)	0.050	1.763e-10	3.350e -9	5.977e-10	1.431
BDI centered	0.050	1.030e-10	1.957e -9	3.491e-10	2.740
AMI centered	0.050	9.690e-11	1.841e -9	3.285e-10	2.450
BDI centered + AMI centered	0.050	6.883e-11	1.308e -9	2.333e-10	2.651
Drug	0.050	3.532e-11	6.712e-10	1.197e-10	2.124
Drug + AMI centered	0.050	2.006e-11	3.811e-10	6.800e-11	2.899
Drug + BDI centered	0.050	1.993e-11	3.787e-10	6.756e-11	1.796
Drug + BDI centered + AMI centered	0.050	1.389e-11	2.640e-10	4.709e-11	3.125

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.191	0.157
Emotion	0.600	1.000	1.248e+9
BDI centered	0.500	0.412	0.701
AMI centered	0.500	0.403	0.676
Drug ✻ Emotion	0.200	0.032	0.134

Appendix Table 6.44 | Repeated Measures ANOVA on Localisation error with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	13037.532	1	13037.532	2.314	0.147	0.120
Drug ✻ BDI centered	1.717	1	1.717	3.047e-4	0.986	1.792e-5
Drug ✻ AMI centered	11844.400	1	11844.400	2.102	0.165	0.110
Residuals	95771.220	17	5633.601			
Emotion	1303.746	2	651.873	0.544	0.585	0.031
Emotion ✻ BDI centered	2700.311	2	1350.155	1.128	0.336	0.062
Emotion ✻ AMI centered	2923.477	2	1461.738	1.221	0.308	0.067
Residuals	40707.302	34	1197.274			
Drug ✻ Emotion	1626.863	2	813.432	0.527	0.595	0.030
Drug ✻ Emotion ✻ BDI centered	2030.711	2	1015.356	0.658	0.525	0.037
Drug ✻ Emotion ✻ AMI centered	9752.774	2	4876.387	3.158	0.055	0.157
Residuals	52502.653	34	1544.196			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	56205.958	1	56205.958	0.643	0.434
AMI centered	46687.706	1	46687.706	0.534	0.475
Residuals	1.487e+6	17	87475.733		

Note. Type III Sum of Squares

Appendix Table 6.45 | Bayesian Repeated Measures ANOVA on Localisation error with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Drug	0.050	0.227	5.569	1.000	
Drug + BDI centered	0.050	0.147	3.280	0.650	4.042
Drug + AMI centered	0.050	0.143	3.161	0.629	4.519
Drug + BDI centered + AMI centered	0.050	0.126	2.729	0.554	4.613
Null model (incl. subject)	0.050	0.090	1.887	0.398	3.683
BDI centered	0.050	0.059	1.193	0.261	3.782
AMI centered	0.050	0.059	1.182	0.258	4.298
BDI centered + AMI centered	0.050	0.048	0.968	0.214	4.400
Drug + Emotion	0.050	0.022	0.431	0.098	4.266
Drug + Emotion + AMI centered	0.050	0.015	0.299	0.068	5.463
Drug + Emotion + BDI centered	0.050	0.015	0.280	0.064	4.210
Drug + Emotion + BDI centered + AMI centered	0.050	0.012	0.232	0.053	5.738
Emotion	0.050	0.009	0.170	0.039	3.843
Emotion + BDI centered	0.050	0.007	0.127	0.029	12.312
Emotion + AMI centered	0.050	0.006	0.109	0.025	5.161
Emotion + BDI centered + AMI centered	0.050	0.005	0.094	0.022	5.248
Drug + Emotion + Drug ✻ Emotion	0.050	0.004	0.071	0.016	4.056
Drug + Emotion + BDI centered + Drug ✻ Emotion	0.050	0.003	0.053	0.012	7.392
Drug + Emotion + AMI centered + Drug ✻ Emotion	0.050	0.003	0.049	0.011	7.219
Drug + Emotion + BDI centered + AMI centered + Drug ✻ Emotion	0.050	0.002	0.037	0.009	6.813

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.717	1.693
Emotion	0.600	0.101	0.075
BDI centered	0.500	0.423	0.734
AMI centered	0.500	0.418	0.718
Drug ✻ Emotion	0.200	0.011	0.045

Correlation Analysis on Behavioural Results: PD citalopram versus PD placebo

Appendix Table 6.46 | Pearson and Spearman's correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and identification accuracy on neutral, angry and happy memoranda.

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
Neutral_effect of drug on accuracy(citalopram-placebo)	Pearson's r	-0.573 *	0.090
	p-value	0.008	0.705
	Spearman's rho	-0.050	-0.036
	p-value	0.833	0.879
Angry_effect of drug on accuracy(citalopram-placebo)	Pearson's r	-0.226	0.085
	p-value	0.337	0.721
	Spearman's rho	-0.216	-0.319
	p-value	0.361	0.170
Happy_effect of drug on accuracy(citalopram-placebo)	Pearson's r	-0.298	0.232
	p-value	0.202	0.324
	Spearman's rho	-0.076	0.069
	p-value	0.749	0.774
Neutral_effect of drug on LOGRT(citalopram-placebo)	Pearson's r	-0.408	0.067
	p-value	0.074	0.778
	Spearman's rho	0.022	0.250
	p-value	0.927	0.288
Angry_effect of drug on LOGRT(citalopram-placebo)	Pearson's r	-0.228	-0.168
	p-value	0.334	0.478
	Spearman's rho	-0.156	-7.567e-4
	p-value	0.510	0.997
Happy_effect of drug on LOHRT(citalopram-placebo)	Pearson's r	-0.268	-0.043
	p-value	0.254	0.856
	Spearman's rho	-0.237	0.090
	p-value	0.314	0.706
Neutral_effect of drug on locerror(citalopram-placebo)	Pearson's r	-0.152	-0.390
	p-value	0.523	0.089
	Spearman's rho	-0.151	-0.452 *
	p-value	0.525	0.046
Angry_effect of drug on locerror(citalopram-placebo)	Pearson's r	0.238	-0.217
	p-value	0.313	0.358
	Spearman's rho	-0.064	-0.251
	p-value	0.789	0.285
Happy_effect of drug on locerror(citalopram-placebo)	Pearson's r	0.472 *	-0.033
	p-value	0.036	0.891
	Spearman's rho	0.031	0.054
	p-value	0.896	0.820

* p < .05, ** p < .01, *** p < .001

Appendix Table 6.47 | Bayesian Pearson and Kendall's correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and identification accuracy on neutral, angry and happy memoranda.

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
Neutral_effect of drug on accuracy(citalopram-placebo)	Pearson's r	-0.573	0.090
	BF_{10}	7.138	0.296
	Kendall's tau	-0.032	-0.031
	BF_{10}	0.291	0.291
Angry_effect of drug on accuracy(citalopram-placebo)	Pearson's r	-0.226	0.085
	BF_{10}	0.426	0.294
	Kendall's tau	-0.194	-0.270
	BF_{10}	0.561	1.050
Happy_effect of drug on accuracy(citalopram-placebo)	Pearson's r	-0.298	0.232
	BF_{10}	0.594	0.436
	Kendall's tau	-0.064	0.047
	BF_{10}	0.307	0.297
Neutral_effect of drug on LOGRT(citalopram-placebo)	Pearson's r	-0.408	0.067
	BF_{10}	1.232	0.287
	Kendall's tau	0.055	0.196
	BF_{10}	0.302	0.568
Angry_effect of drug on LOGRT(citalopram-placebo)	Pearson's r	-0.228	-0.168
	BF_{10}	0.428	0.350
	Kendall's tau	-0.100	0.000
	BF_{10}	0.341	0.286
Happy_effect of drug on LOHRT(citalopram-placebo)	Pearson's r	-0.268	-0.043
	BF_{10}	0.510	0.281
	Kendall's tau	-0.155	0.076
	BF_{10}	0.439	0.317
Neutral_effect of drug on locerror(citalopram-placebo)	Pearson's r	-0.152	-0.390
	BF_{10}	0.335	1.069
	Kendall's tau	-0.055	-0.348
	BF_{10}	0.302	2.499
Angry_effect of drug on locerror(citalopram-placebo)	Pearson's r	0.238	-0.217
	BF_{10}	0.446	0.411
	Kendall's tau	-0.044	-0.163
	BF_{10}	0.296	0.460
Happy_effect of drug on locerror(citalopram-placebo)	Pearson's r	0.472	-0.033
	BF_{10}	2.180	0.279
	Kendall's tau	0.000	0.044
	BF_{10}	0.286	0.295

* $BF_{10} > 10$, ** $BF_{10} > 30$, *** $BF_{10} > 100$

Mixture Model Analysis: PD placebo versus PD citalopram

Appendix Table 6.48 | Repeated Measures ANOVA on Imprecision with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	None	265.822	1.000	265.822	0.907	0.354	0.051
Drug ✻ BDI centered	None	1001.849	1.000	1001.849	3.419	0.082	0.167
Drug ✻ AMI centered	None	35.650	1.000	35.650	0.122	0.732	0.007
Residuals	None	4981.272	17.000	293.016			
Emotion	None	727.211	2.000	363.606	2.033	0.147	0.107
	Greenhouse-Geisser	727.211	1.638	443.909	2.033	0.156	0.107
Emotion ✻ BDI centered	None	185.501	2.000	92.750	0.519	0.600	0.030
	Greenhouse-Geisser	185.501	1.638	113.235	0.519	0.565	0.030
Emotion ✻ AMI centered	None	393.490	2.000	196.745	1.100	0.344	0.061
	Greenhouse-Geisser	393.490	1.638	240.196	1.100	0.336	0.061
Residuals	None	6080.416	34.000	178.836			
	Greenhouse-Geisser	6080.416	27.849	218.332			
Drug ✻ Emotion	None	548.622	[a] 2.000	[a] 274.311	[a] 0.821	[a] 0.449	[a] 0.046
	Greenhouse-Geisser	548.622	1.501	365.524	0.821	0.420	0.046
Drug ✻ Emotion ✻ BDI centered	None	770.235	[a] 2.000	[a] 385.117	[a] 1.152	[a] 0.328	[a] 0.063
	Greenhouse-Geisser	770.235	1.501	513.175	1.152	0.318	0.063
Drug ✻ Emotion ✻ AMI centered	None	704.589	[a] 2.000	[a] 352.294	[a] 1.054	[a] 0.360	[a] 0.058
	Greenhouse-Geisser	704.589	1.501	469.438	1.054	0.344	0.058
Residuals	None	11363.215	34.000	334.212			
	Greenhouse-Geisser	11363.215	25.516	445.343			

Note. Sphericity corrections not available for factors with 2 levels.

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	5456.752	1	5456.752	0.717	0.409
AMI centered	7286.007	1	7286.007	0.957	0.342
Residuals	129449.148	17	7614.656		

Note. Type III Sum of Squares

Appendix Table 6.49 | Bayesian Repeated Measures ANOVA on Imprecision with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.216	5.226	1.000	
AMI centered	0.050	0.142	3.133	0.656	2.097
BDI centered	0.050	0.132	2.901	0.614	1.032
BDI centered + AMI centered	0.050	0.119	2.556	0.550	2.210
Drug	0.050	0.065	1.317	0.301	2.000
Emotion	0.050	0.052	1.041	0.241	0.932
Drug + AMI centered	0.050	0.043	0.851	0.199	2.567
Drug + BDI centered	0.050	0.039	0.779	0.183	0.962
Drug + BDI centered + AMI centered	0.050	0.038	0.745	0.175	2.752
Emotion + AMI centered	0.050	0.036	0.702	0.165	3.041
Emotion + BDI centered	0.050	0.032	0.634	0.150	1.357
Emotion + BDI centered + AMI centered	0.050	0.030	0.584	0.138	3.379
Drug + Emotion	0.050	0.016	0.308	0.074	2.406
Drug + Emotion + AMI centered	0.050	0.010	0.191	0.046	3.967
Drug + Emotion + BDI centered	0.050	0.010	0.186	0.045	1.729
Drug + Emotion + BDI centered + AMI centered	0.050	0.009	0.169	0.041	4.069
Drug + Emotion + Drug ✻ Emotion	0.050	0.004	0.085	0.021	1.928
Drug + Emotion + AMI centered + Drug ✻ Emotion	0.050	0.003	0.057	0.014	4.755
Drug + Emotion + BDI centered + Drug ✻ Emotion	0.050	0.003	0.057	0.014	4.643
Drug + Emotion + BDI centered + AMI centered + Drug ✻ Emotion	0.050	0.002	0.047	0.011	5.618

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.242	0.213
Emotion	0.600	0.207	0.174
BDI centered	0.500	0.414	0.707
AMI centered	0.500	0.430	0.755
Drug ✻ Emotion	0.200	0.013	0.052

Appendix Table 6.50 | Repeated Measures ANOVA on Misbinding with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	9.623e-5	1	9.623e-5	0.026	0.874	0.002
Drug ✻ BDI centered	3.461e-7	1	3.461e-7	9.275e-5	0.992	5.456e-6
Drug ✻ AMI centered	0.003	1	0.003	0.676	0.422	0.038
Residuals	0.063	17	0.004			
Emotion	0.013	2	0.007	1.373	0.267	0.075
Emotion ✻ BDI centered	0.006	2	0.003	0.598	0.556	0.034
Emotion ✻ AMI centered	0.004	2	0.002	0.375	0.690	0.022

Appendix Table 6.50 | Repeated Measures ANOVA on Misbinding with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p	η^2_p
Residuals	0.165	34	0.005			
Drug ✻ Emotion	1.206e-4	2	6.028e-5	0.017	0.984	9.745e-4
Drug ✻ Emotion ✻ BDI centered	0.001	2	7.058e-4	0.194	0.824	0.011
Drug ✻ Emotion ✻ AMI centered	2.420e-4	2	1.210e-4	0.033	0.967	0.002
Residuals	0.124	34	0.004			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	0.015	1	0.015	0.429	0.521
AMI centered	0.017	1	0.017	0.498	0.490
Residuals	0.593	17	0.035		

Note. Type III Sum of Squares

Appendix Table 6.51 | Bayesian Repeated Measures ANOVA on Misbinding with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.277	7.275	1.000	
AMI centered	0.050	0.128	2.788	0.462	1.707
BDI centered	0.050	0.127	2.767	0.459	1.517
Emotion	0.050	0.092	1.927	0.333	0.510
BDI centered + AMI centered	0.050	0.090	1.868	0.323	1.991
Drug	0.050	0.054	1.082	0.195	1.265
Emotion + AMI centered	0.050	0.045	0.898	0.163	2.546
Emotion + BDI centered	0.050	0.044	0.874	0.159	3.145
Emotion + BDI centered + AMI centered	0.050	0.030	0.580	0.107	2.907
Drug + AMI centered	0.050	0.025	0.496	0.092	2.211
Drug + BDI centered	0.050	0.025	0.484	0.090	1.764
Drug + Emotion	0.050	0.018	0.352	0.066	1.643
Drug + BDI centered + AMI centered	0.050	0.016	0.318	0.060	2.552
Drug + Emotion + AMI centered	0.050	0.009	0.177	0.033	3.190
Drug + Emotion + BDI centered	0.050	0.008	0.155	0.029	1.997
Drug + Emotion + BDI centered + AMI centered	0.050	0.006	0.115	0.022	3.690
Drug + Emotion + Drug ✻ Emotion	0.050	0.002	0.046	0.009	2.049
Drug + Emotion + AMI centered + Drug ✻ Emotion	0.050	0.001	0.023	0.004	4.582
Drug + Emotion + BDI centered + Drug ✻ Emotion	0.050	0.001	0.021	0.004	2.502
Drug + Emotion + BDI centered + AMI centered + Drug ✻ Emotion	0.050	7.929e-4	0.015	0.003	5.037

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.168	0.134
Emotion	0.600	0.258	0.232
BDI centered	0.500	0.348	0.533
AMI centered	0.500	0.351	0.542
Drug ✻ Emotion	0.200	0.006	0.022

Appendix Table 6.52 | Repeated Measures ANOVA on Guessing with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Within Subjects Effects

Cases	Sphericity Correction	Sum of Squares	df	Mean Square	F	p	η^2_p
Drug	None	0.011	1.000	0.011	1.182	0.292	0.065
Drug ✻ BDI centered	None	0.005	1.000	0.005	0.508	0.486	0.029
Drug ✻ AMI centered	None	0.021	1.000	0.021	2.175	0.159	0.113
Residuals	None	0.160	17.000	0.009			
Emotion	None	6.147e-4	2.000	3.073e-4	0.092	0.913	0.005
	Greenhouse-Geisser	6.147e-4	1.940	3.168e-4	0.092	0.908	0.005
Emotion ✻ BDI centered	None	0.004	2.000	0.002	0.646	0.530	0.037
	Greenhouse-Geisser	0.004	1.940	0.002	0.646	0.526	0.037
Emotion ✻ AMI centered	None	0.007	2.000	0.003	1.000	0.378	0.056
	Greenhouse-Geisser	0.007	1.940	0.003	1.000	0.377	0.056
Residuals	None	0.114	34.000	0.003			
	Greenhouse-Geisser	0.114	32.988	0.003			
Drug ✻ Emotion	None	0.004	[a] 2.000	[a] 0.002	[a] 0.377	[a] 0.688	[a] 0.022
	Greenhouse-Geisser	0.004	1.320	0.003	0.377	0.603	0.022
Drug ✻ Emotion ✻ BDI centered	None	0.009	[a] 2.000	[a] 0.004	[a] 0.815	[a] 0.451	[a] 0.046
	Greenhouse-Geisser	0.009	1.320	0.007	0.815	0.409	0.046
Drug ✻ Emotion ✻ AMI centered	None	0.011	[a] 2.000	[a] 0.005	[a] 0.984	[a] 0.384	[a] 0.055
	Greenhouse-Geisser	0.011	1.320	0.008	0.984	0.356	0.055
Residuals	None	0.182	34.000	0.005			
	Greenhouse-Geisser	0.182	22.437	0.008			

Note. Sphericity corrections not available for factors with 2 levels.

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

Between Subjects Effects

Cases	Sum of Squares	df	Mean Square	F	p
BDI centered	0.036	1	0.036	0.538	0.473
AMI centered	0.026	1	0.026	0.388	0.542
Residuals	1.127	17	0.066		

Note. Type III Sum of Squares

Appendix Table 6.53 | Bayesian Repeated Measures ANOVA on Guessing with drug (PD on placebo versus PD on citalopram) and emotion (neutral, angry, happy) as "within-subjects" factors, and AMI (anxiety motivation index) and BDI (depression scores) as covariates.

Model Comparison

Models	P(M)	P(M|data)	BF_M	BF_{10}	error %
Null model (incl. subject)	0.050	0.252	6.386	1.000	
BDI centered	0.050	0.133	2.922	0.530	0.944
Drug	0.050	0.129	2.813	0.513	0.931
AMI centered	0.050	0.128	2.794	0.510	1.778
BDI centered + AMI centered	0.050	0.093	1.942	0.369	2.025
Drug + BDI centered	0.050	0.068	1.380	0.269	1.334
Drug + AMI centered	0.050	0.066	1.346	0.263	2.215
Drug + BDI centered + AMI centered	0.050	0.051	1.014	0.201	2.435
Emotion	0.050	0.020	0.395	0.081	0.713
Drug + Emotion	0.050	0.011	0.214	0.044	3.217
Emotion + BDI centered	0.050	0.011	0.211	0.044	1.827
Emotion + AMI centered	0.050	0.010	0.197	0.041	3.042
Emotion + BDI centered + AMI centered	0.050	0.008	0.153	0.032	3.405
Drug + Emotion + BDI centered	0.050	0.006	0.110	0.023	3.467
Drug + Emotion + AMI centered	0.050	0.006	0.107	0.022	3.520
Drug + Emotion + BDI centered + AMI centered	0.050	0.004	0.076	0.016	4.083
Drug + Emotion + Drug ✻ Emotion	0.050	0.002	0.035	0.007	1.398
Drug + Emotion + AMI centered + Drug ✻ Emotion	0.050	0.001	0.020	0.004	5.447
Drug + Emotion + BDI centered + Drug ✻ Emotion	0.050	0.001	0.019	0.004	2.077
Drug + Emotion + BDI centered + AMI centered + Drug ✻ Emotion	0.050	7.729e-4	0.015	0.003	6.896

Note. All models include subject

Analysis of Effects

Effects	P(incl)	P(incl|data)	BF_{incl}
Drug	0.600	0.345	0.351
Emotion	0.600	0.081	0.059
BDI centered	0.500	0.375	0.600
AMI centered	0.500	0.367	0.581
Drug ✻ Emotion	0.200	0.005	0.019

Correlation Analysis on Mixture Model Results: PD citalopram versus PD placebo

Appendix Table 6.54 | Pearson and Spearman's correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and imprecision (pixels) on neutral, angry and happy memoranda.

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
11. Neutral_effect of drug on imprecision(citalopram-placebo)	Pearson's r	-0.149	-0.106
	p-value	0.530	0.657
	Spearman's rho	0.073	-0.096
	p-value	0.760	0.687
12. Angry_effect of drug on imprecision(citalopram-placebo)	Pearson's r	0.479 *	-0.177
	p-value	0.033	0.457
	Spearman's rho	0.160	0.047
	p-value	0.502	0.844
13. Happy_effect of drug on imprecision(citalopram-placebo)	Pearson's r	0.556 *	0.037
	p-value	0.011	0.876
	Spearman's rho	0.163	0.114
	p-value	0.493	0.634
14. Neutral_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.116	-0.481 *
	p-value	0.627	0.032
	Spearman's rho	-0.115	-0.328
	p-value	0.628	0.158
15. Angry_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.548 *	-0.168
	p-value	0.012	0.480
	Spearman's rho	-0.165	-0.254
	p-value	0.487	0.279
16. Happy_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.021	0.476 *
	p-value	0.929	0.034
	Spearman's rho	0.073	0.478 *
	p-value	0.760	0.033
17. Neutral_effect of drug on guess(citalopram-placebo)	Pearson's r	-0.040	-0.003
	p-value	0.868	0.992
	Spearman's rho	0.021	-0.207
	p-value	0.929	0.382
18. Angry_effect of drug on guess(citalopram-placebo)	Pearson's r	0.609 **	0.024
	p-value	0.004	0.918
	Spearman's rho	0.062	0.060
	p-value	0.794	0.802
19. Happy_effect of drug on guess(citalopram-placebo)	Pearson's r	0.411	-0.221
	p-value	0.072	0.349
	Spearman's rho	0.134	-0.122
	p-value	0.574	0.609

* p < .05, ** p < .01, *** p < .001

Appendix Table 6.55 | Bayesian Pearson and Kendall's correlations: Relationship between the effect of BDI (difference in scores on citalopram and on placebo), AMI (difference in scores on citalopram and on placebo) and imprecision (pixels) on neutral, angry and happy memoranda.

Variable		BDI effect (citalopram-placebo)	AMI effect (citalopram-placebo)
Neutral_effect of drug on imprecision(citalopram-placebo)	Pearson's r	-0.149	-0.106
	BF_{10}	0.333	0.304
	Kendall's tau	0.089	-0.098
	BF_{10}	0.329	0.339
1Angry_effect of drug on imprecision(citalopram-placebo)	Pearson's r	0.479	-0.177
	BF_{10}	2.342	0.359
	Kendall's tau	0.100	0.022
	BF_{10}	0.341	0.288
Happy_effect of drug on imprecision(citalopram-placebo)	Pearson's r	0.556	0.037
	BF_{10}	5.668	0.280
	Kendall's tau	0.122	0.076
	BF_{10}	0.373	0.317
Neutral_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.116	-0.481
	BF_{10}	0.309	2.379
	Kendall's tau	-0.089	-0.207
	BF_{10}	0.329	0.614
Angry_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.548	-0.168
	BF_{10}	5.120	0.350
	Kendall's tau	-0.111	-0.163
	BF_{10}	0.356	0.460
Happy_effect of drug on misb(citalopram-placebo)	Pearson's r	-0.021	0.476
	BF_{10}	0.278	2.271
	Kendall's tau	0.022	0.359
	BF_{10}	0.288	2.867
Neutral_effect of drug on guess(citalopram-placebo)	Pearson's r	-0.040	-0.003
	BF_{10}	0.280	0.277
	Kendall's tau	0.011	-0.152
	BF_{10}	0.286	0.433
Angry_effect of drug on guess(citalopram-placebo)	Pearson's r	0.609 *	0.024
	BF_{10}	12.275	0.278
	Kendall's tau	0.066	0.054
	BF_{10}	0.309	0.301
Happy_effect of drug on guess(citalopram-placebo)	Pearson's r	0.411	-0.221
	BF_{10}	1.263	0.418
	Kendall's tau	0.100	-0.109
	BF_{10}	0.341	0.353

* $BF_{10} > 10$, ** $BF_{10} > 30$, *** $BF_{10} > 100$