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Two processes are not necessary to understand memory deficits

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Abstract: Bastin et al. propose a dual process model to understand memory deficits. However, results from state-trace analysis have suggested a single underlying variable in behavioral and neural data. We advocate the usage of unidimensional models which are supported by data and have been successful in understanding memory deficits and in linking to neural data.

Bastin et al. advocate a dual process model to understand memory deficits. While this model is a popular framework, the evidence for it is weak. Much of the argument for the dual process model hinges on double dissociations in behavioral data, including remember-know responses and parameters of the dual process signal detection model (Yonelinas 2002), and in neural data, such as between the frontal-negativity component and the parietal late-positive component in event-related potentials (Rugg & Curran 2007). Neural data also include dissociations between the hippocampus and surrounding cortical regions such as the perirhinal cortex in functional magnetic resonance imaging (fMRI) (Eichenbaum et al. 2007).

Unfortunately, a double dissociation is not sufficient evidence to infer the existence of more than one latent variable or processes (Dunn & Kirsner 1988). A more principled method is provided by state-trace analysis (Bamber 1979; Dunn & Kalish 2018; Newell & Dunn 2008). State-trace analysis evaluates the number of latent variables that are required to explain performance across multiple dependent variables. State-trace analysis in recognition memory consistently refutes dual process theory, as it has not revealed evidence for more than one latent variable in remember-know responses (Dunn 2008), item recognition and source memory across development (Hayes et al. 2017), and event-related potentials (Brezis et al. 2017; Freeman et al. 2010).

Double dissociations in these paradigms are consistent with a monotonic but non-linear relationship between dependent measures, as illustrated in the hypothetical demonstration in our Figure 1. Although state-trace analysis has not yet been applied to fMRI data, Squire et al. (2007) proposed that dissociations between the hippocampus and perirhinal cortex can be explained by non-linear relationships between the two regions, and evidence for such a non-linearity has been found using fMRI (Song et al. 2011).

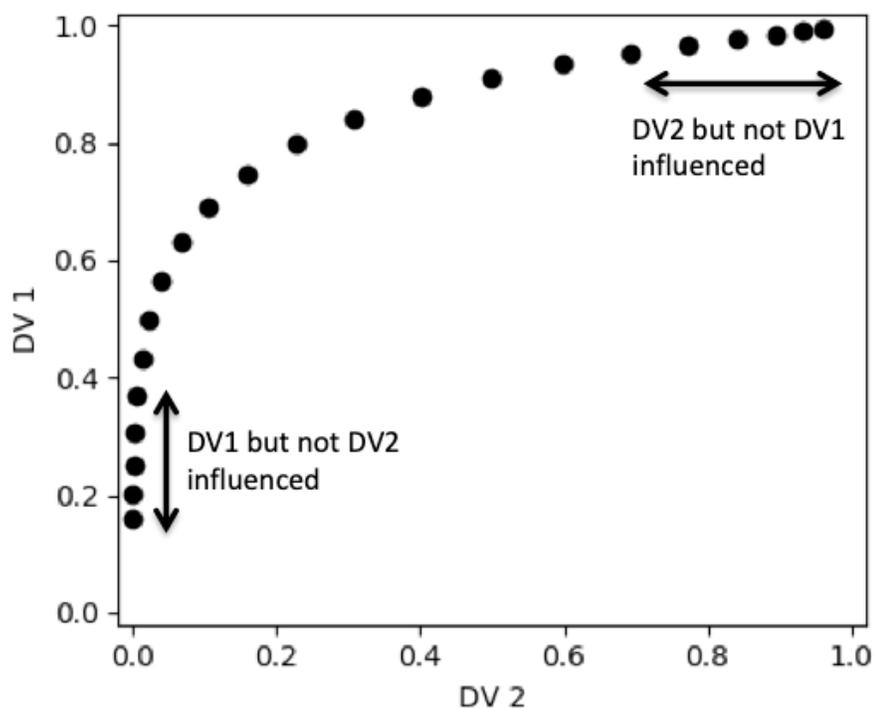


Figure 1 (Osth et al.): A hypothetical example of how manipulations can produce double dissociations between two dependent variables (DVs) within a unidimensional model where the relation between the two DVs is non-linear. The black dots depict performance across a range of manipulations.

We argue that a more fruitful approach to understanding cognitive and neurological deficits in memory is to use models that contain a single underlying latent variable. One-dimensional models, such as signal-detection theory, have often been found to provide a better fit to receiver-operating characteristics than the dual process model (Hayes et al. 2017; Heathcote 2003; Heathcote et al. 2006). Signal-detection theory has also been highly successful as a

measurement model; even graded levels of memory deficits in Alzheimer's disease have been able to be identified using this model (Pooley et al. 2011).

The diffusion model of Ratcliff (1978) is an even better candidate for understanding memory deficits because it can also account for response times. In the diffusion model, evidence accumulates toward one of the two response boundaries corresponding to response alternatives, such as "old" and "new" in the case of recognition memory. Once a boundary is reached, the associated response is given and the time taken to reach the boundary plus time for non-decision processes is the response time. The rate at which evidence is accumulated is called the *drift rate* and is analogous to memory strength in signal-detection theory. As drift rate increases, the rate of correct responses increases and latency decreases.

The diffusion model is advantageous because, unlike signal-detection theory and the dual-process model, it leverages both accuracy and latency into relevant psychological variables. These include memory strength (measured by drift rate), speed of perception and motor processes (measured by non-decision time), and response caution (measured by response boundaries). The diffusion model has been highly successful in explaining data from recognition memory paradigms (Osth et al. 2017; 2018; Ratcliff 1978). Although recollection in the dual-process model has been described as being slower than the familiarity process, to date there is no formal instantiation of the dual-process model that has made contact with latency data.

The diffusion model has also been extremely fruitful as a measurement model. A noteworthy example is the study by Ratcliff et al. (2004), which compared younger and older adults' recognition performance. While both groups exhibited similar accuracy, latencies were much longer in older adults. Diffusion modeling revealed that older adults were more cautious in their responding and had higher non-decision times, but otherwise exhibited very similar drift

rates. Without the aid of the model, researchers could easily be misled into believing that the older adults had slower rates of processing in the task. In other applications, diffusion model parameters such as the drift rate have been shown to be more sensitive to group-level differences than measures based on accuracy or latency alone (White et al. 2010).

Diffusion modeling applied to Alzheimer's disease is in its infancy, but shows promise. Memory deficits associated with a family history of Alzheimer's disease have been best described by differences in the drift-rate parameter (Aschenbrenner et al. 2016). Even more critically, the drift-rate parameter predicted group-level differences better than neuropsychological tests.

In addition, diffusion models have been extremely successful in linking to neural data. Ratcliff et al. (2016a) were able to explain variability in single-trial indices of memory strength using only the drift-rate parameter. In addition, a great deal of work in neuroscience has uncovered neural mechanisms that resemble evidence accumulation in the diffusion model (Gold & Shadlen 2007), suggesting that the neurological underpinnings of dementia may be able to be understood through the lens of diffusion models.

We suggest that adoption of a dual-process framework for recognition memory is unlikely to lead to progress in understanding memory deficits. It is not strongly supported by existing evidence and, if it is the wrong model of memory, will lead to misleading conclusions (Pazzaglia et al. 2013). Models that contain a single latent variable are consistent with the neural evidence and provide a framework for unifying accuracy and latency; they are suitable measurement models for memory impairment. In our view, the application of this framework will lead to a deeper understanding of the nature of memory deficits.

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