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The unitary zROC slope in amnesics does not reflect the absence of recollection: critical simulations in healthy participants of the zROC slope

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ABSTRACT

The functional deficit underlying amnesia has been argued to be in recollective processing. This idea is based on the DPSD model, wherein recognition comprises a mixture of recollection and familiarity signals, with familiarity conforming to an equal-variance signal-detection mechanism while recollection is binary. This model interprets the greater variance for targets than for lures revealed in sub-unit zROC slopes, to be a consequence of the mixture of two signals. Importantly, equal variance between targets and lures is found in amnesic, and is consequently interpreted by DPSD to reflect impairment to recollection alongside the sparing of familiarity. Here, we pointed to a logical fallacy in this interpretation. We then asked participants, in two experiments, to make remember-know (RK) and confidence judgments. Simulating equal variance in healthy participants, we either excluded from the analysis 'remember' responses, reflecting recollection, or the most accurate memories, reflecting strength. We found that only the exclusion of the strongest responses led to equal-variance distributions. In addition, we found that accuracy was associated with an interlaced ordering of RK response groups nested under confidence, a pattern hard to reconcile with classic recognition models (DPSD, UVSD). This pattern can, however, be accommodated by the Continuous Dual Process (CDP) model (Wixted and Mickes, 2010), wherein both familiarity and recollection are continuous signals. Amnesia may thus be characterized as the inability to form strong memories, recollection as well as familiarity.

1. Introduction

An important goal in the cognitive neuropsychology of memory is to delineate the functional loss in amnesia. Over the past 50 years, a multitude of processes have been suggested as being impaired in anterograde amnesia, beginning with the idea that it comprises a deficit in long term memory (e.g., Milner, 1966; Wickelgren, 1968; cf., Davelaar et al., 2005; Talmi and Goshen-Gottstein, 2006), and moving on to a long list of suggestions of possible deficits within long-term memory, including a deficit to declarative memory (as compared to procedural memory; Cohen and Squire, 1980; Manns et al., 2003), a deficit to episodic memory (as compared to semantic memory; Viskontas et al., 2000), a deficit in retrieval (Nadel and Moscovitch, 1997), a storage deficit (e.g., Hardt et al., 2009; Mayes, 1995), a deficit in conscious recollection as indexed by explicit tests of memory (for reviews, see Cohen and Eichenbaum, 1993; Moscovitch, 1982), a deficit in relational memory (Cohen et al., 1997; Ryan et al., 2000; but see Goshen-Gottstein et al., 2000), as an impairment of detail generation and binding (Rosenbaum et al., 2009), as an impairment in autobiographical memory (e.g., Rosenbaum et al., 2004), and—most relevant to our present concerns—as an impairment in recollective processing, sparing familiarity (Gilboa et al., 2006; Hirst et al., 1988; Schacter et al., 1984; Turriziani et al., 2008; Yonelinas et al., 1998). In this article, we argue against the idea that a recollective deficit characterizes amnesia. Instead, we suggest that amnesia may represent a deficit in the formation of strong traces, comprising both recollection and familiarity.

The investigations reported in this article are guided by the notion that a better understanding of the nature of the deficit underlying (anterograde) amnesia can be obtained if pursuant to a carefully designed manipulation, performance in neurologically intact participants can be shown to simulate the deficit observed in amnesia. Such manipulations include pharmaceutical interventions (e.g., Hardt et al., 2009), transcranial magnetic stimulations (e.g., Bolognini and Ro, 2010) and cognitive tasks (e.g., Dunbar and Sussman, 1995; Moscovitch, 1994). Experimentally-induced impairments have the benefit that they can be subject to systematic

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investigations, with the purpose of expanding our understanding of the mechanism underlying the observed behavior.

Here, we focus on a pivotal finding regarding the performance of amnesic patients in recognition tests, which was uncovered in analyses of ROC curves. The pattern revealed in ROC analysis (described in detail below), suggests that amnesia may be characterized as impairment in the recollective process. In validation of this suggestion was the finding that the amnesic ROC pattern (Aggleton et al., 2005; Gilboa et al., 2006; Yonelinas et al., 1998) could be revealed in healthy participants, when applying a simulation procedure aimed at disabling recollective processing (Yonelinas, 2001). In this article, we demonstrate how critical investigations of this simulation procedure should change our conceptualization of the functional deficit underlying amnesia.

The suggestion that recollective processes are impaired in amnesia is primarily based on studies of recognition memory. In recognition, participants are presented with items at study, and at test are asked to distinguish between targets (studied items) and lures (unstudied items). Dual-process models of recognition (the Dual-Process Signal Detection (DPSD), Yonelinas (1994); the Variable-Recollection Dual-Process (VRDP), Onyper et al. (2010); the Continuous Dual-Process (CDP), Wixted and Mickes (2010); the Sum-difference Theory of Remembering and Knowing (STREAK), Rotello et al. (2004)) propose that performance on this task is an outcome of two qualitatively different signals, recollection and familiarity. As we shall see below, impaired processing of one of these signals—recollection—has been argued to be the source of deficit in amnesia.

The recollection signal entails memory for details of an episodic event, which may, or may not, be available to consciousness. For example, remembering not only that you met a certain person before, but also that person's shirt color, a background song, and topics of conversation in the specific encounter. The recollection process may fail to provide episodic details, yet often people still report experiencing a feeling of knowing that the event occurred (e.g., that the person has been previously encountered). This feeling of knowing corresponds to output from the second signal—familiarity. The notion that recognition comprises both recollection and familiarity has been supported in numerous studies using behavioral methodologies, fMRI, scalp electroencephalography, human neuropsychology and the study of recognition in non-humans (Aggleton et al., 2005; Curran, 2000; Diana et al., 2006; Eichenbaum et al., 2007; Fortin et al., 2004; Rugg and Curran, 2007; Rugg and Yonelinas, 2003; Woodruff et al., 2006; Yonelinas et al., 2005; Yonelinas, 2002; Yovel and Paller, 2004).

A triad of findings (see details below) turns out to be critical in constraining theories of recognition memory as well as for delineating the functional deficit underlying amnesia. The three findings were uncovered in the analysis of ROC curves. An ROC curve describes the function relating the proportion of correctly recognized target items (i.e., hit rate) to the proportion of incorrectly recognized lure items (i.e., false alarm rate) across variations in response criteria. The notion of response criteria refers to the bias in making a positive recognition response, irrespective of the amount of recognition-relevant information (e.g., familiarity and/or recollection) that is available for a particular stimulus. When the underlying lure and target distributions are normal—a standard assumption of signal-detection theory (SDT; Swets and Green, 1963)—the ROC curve plotted in z-space (zROC) is linear. Importantly, the slope of the linear zROC curve has been shown to represent the ratio of lure-to-target distribution variance (or, strictly speaking, standard deviation). Specifically, a zROC slope equal to 1 indicates equal variance between the target and lure distributions and a slope smaller than 1 indicates a larger variance of the target distribution.

The first of the triad of findings was that the zROC slope in standard tests of recognition in healthy participant is smaller than 1 ($\sim$0.8), indicating a higher variance of the target distribution than the lure distribution (e.g., Ratcliff et al., 1992). This finding is important, in that standard SDT models assume equal variance for target and lure distributions. To account for the inequality of variance, profoundly different models have been postulated, ranging from an assortment of dual-process models to models that posit only a single process. The second finding was that the slope of the zROC curve in amnesics is equal to 1, indicating equal variance for target and lure distributions, as assumed by SDT. As we shall see, this finding has been interpreted as evidence that the function evidence in amnesia is that of impairment to recollective processing (an interpretation we challenge in this article). The third finding was that of equal variance of targets and lures, when a simulation procedure was used to eliminate recollective processing in neurologically intact participants (Yonelinas, 2001). We now describe two classic models, DPSD—a dual-process model, and UVSD—a single process model. The two models represent radically different interpretations of the inequality of variance observed in healthy individuals and of the equality observed in amnesia and under the simulation procedure.

1.1. DPSD

The Dual Process Signal Detection (DPSD) model (Yonelinas, 1994), suggests that a familiarity signal is available for all items. In contrast, recollection is available for some, but not all, of the target items. Specifically, the familiarity signal, assumed to be governed by the principles of SDT, is argued to mediate both the lure and the target distributions. Here, the effect of studying the items is reflected by a uniform—constant—boost (increase) to the lure distribution. Because the boost is uniform for all studied items, the resultant target distribution is equal in variance to that of the lure distribution. Thus, the familiarity signal conforms to an equal-variance signal detection mechanism. In addition, the target distribution alone is influenced by retrieval of a binary, high threshold (Macmillan and Creelman, 1991) recollection signal that is generated only for some of the items that have been studied. Importantly, the empirically-observed inequality of variance is interpreted by DPSD to be the outcome of the equal-variance familiarity signal available for both the lure and target items, and the binary recollection signal available for only target items.

According to DPSD, if recognition were to be tested in patients with a deficit in recollective processing, then their performance should reflect the spared familiarity process, yielding a zROC slope of 1 (reflecting an equal-variance distribution). Such a unitary zROC slope was in fact observed in densely amnesic patients and patients with hippocampal injury (Aggleton et al., 2005; Yonelinas et al., 2002, 1998), suggesting an impairment of recollection as the source of the functional deficit in amnesia. Bolstering the notion of a function deficit in recollection in amnesia was the finding of the simulation procedure, which presumably mimicked the functional deficit of the recollective process in healthy participants. To this end, the Remember-know task (Gardiner, 1988; Tulving, 1985) was used in conjunction with zROC analysis. In the Remember-know (R-K) task, participants are required to provide a subjective classification of positive recognition judgments into recognition that entails 'remembering' (R) something specific about the study episode as opposed to recognition that only comprises 'knowing' (K) that an item was studied, with failure to retrieve any specific detail from the learning episode. R judgments have been argued to reflect the operation of the recollection signal, with K judgments associated with familiarity signal (with a possible correction for an underestimation of this process; Yonelinas and Jacoby, 1995). Systematic investigation of performance on the R-K task have supplied converging evidence in support of DPSD (for review, see Yonelinas, 2002; but see Dunn, 2004).