Is Episodic Memory a Natural Kind? - A Defense of the Sequence Analysis

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Abstract

Colloquially, episodic memory is described as "the memory of personally experienced events". Here we ask how episodic memory should be characterized in order to be validated as a natural kind. We propose to conceive of episodic memory as a knowledge-like state that is identified with an experientially based mnemonic representation of an episode. We discuss selected experimental results that provide exemplary evidence for uniform causal mechanisms underlying the properties of episodic memory and argue that episodic memory is a natural kind. The argumentation proceeds along two cornerstones: First, empirical results support the claim that the principal anatomical substrate of episodic memory is the hippocampus. Second, we can pin down causal mechanisms onto neural activities in the hippocampus to explain the psychological states and processes constituting episodic memory.

Keywords: natural kind, neural sequences, replay, mental time travel, memory trace, events, reliable process

Introduction

In many textbooks the notion of episodic memory is introduced in a taxonomical manner: In a first step, a distinction between explicit and implicit memory is made (Graf & Schacter, 1985). In a second step, two subordinate categories are introduced within the superordinate category of declarative memory, namely, semantic memory and episodic memory (Tulving, 1972). Episodic memories are proposed to be those of personally experienced episodes, such as "I met my wife on my first day at work at Acme Co." By contrast, semantic memories are thought to consist of knowledge about the world such as "Abraham Lincoln was assassinated at Ford's Theatre in 1865". To further distinguish episodic from semantic memory, Tulving later added to his definition of episodic memory the requirement of conscious reliving (autonoetic consciousness) (Tulving, 1985). Suddendorf and Corballis (2007) went even further and suggested that episodic memory is mental time travel into the past and facilitates mental time travel into the future.

In this paper, we digress from the taxonomical pathway and focus on the question of whether episodic memory is a natural kind and what implications this has for what episodic memory is best taken to be.

We will start with the proposal that the content of each instance of episodic memory is an episode defined as follows:

Definition:

An episode *E* is a sequence of events e_i , i.e., $E = \langle e_1, \dots, e_n \rangle$., where an event is a concrete particular located in space and time and, for every pair of subsequent members of the sequence e_i and e_{i+1} the event e_i occurs before the event e_{i+1} occurs.

By saying that an event is a particular (rather than a universal) we mean that it occurs only once: It neither repeats in time, nor does it occur as a whole at a different place at the same time. By assuming that events are concrete (rather than abstract) we mean that each event occupies a distinctive region in space-time, which no other therefrom independent event occupies (for the composition of event representations see Werning, 2003, 2012). For our proposed analysis, what counts is the relationship between episodes and events. Our definition of episodes implies that an episode is an ordered list of events. As such, an episode is distinct from a set of events, which is an unordered list of events. The definition of episode can be applied recursively to link episodes together into an episode on a longer timescale such that the shorter episodes are the events in the longer episode.

We suggest that episodic memory is the memory of an episode, but characterizing the content of episodic memory is insufficient to fully constrain it. So other conditions have to be satisfied. In Cheng & Werning (submitted) we have in detail argued for the following analysis:

Sequence Analysis of Episodic Memory:

A subject *S* has episodic memory with content *E* at a time t_1 if and only if the following conditions are fulfilled:

(i) E is an episode with $E = \langle e_1, \dots, e_n \rangle$. E is called the *mnemonic content*.

(ii) At some time t_1, S compositionally represents E as an episode of temporally succeeding events $e_1, \dots, e_n$. S's representation of E at t_1 is called the *mnemonic representation*.

(iii) At a time $t_0 < t_1$, S has a reliable experience of the temporally succeeding events $e_1^*, \dots, e_m^*$, which make up an episode $E^* = \langle e_1^*, \dots, e_m^* \rangle$. E^* is called the *experiential base*.

(iv) The episode E^* occurs at or before t_0 (*factivity*).

(v) The mnemonic content E is *ontologically grounded* in the experiential base E^* in the following sense of counterfactual dependence: Were E^* to occur at or before t_0, E would also occur at that time.

(vi) S's representation with content E at t_1 is *causally grounded* in S's experience of E^* through a reliable memory trace.

(vii) On the basis of its mnemonic representation with content E, S is capable of generating a temporally explicit simulation with content E at some time $t_2 \geq t_1$. The generated simulation is called a *mnemonic simulation*.

When we ask if episodic memory is a natural kind, we presuppose a notion of natural kind that can be traced back to Boyd (1991). It is commonly labelled "the homeostatic property cluster view" (HPC view) of natural kinds. The core idea is that, in science, entities should be clustered together in a way that (i) optimizes the inductive and explanatory potential of theories that make reference to those clusters and (ii) that this inductive and explanatory potential should rest on uniform causal mechanisms underlying each cluster. In the spirit of the HPC view, we will use the notion of natural kind as defined in the following way:

Definition:
A class C of entities is a natural kind if and only if there is a large set of properties that subserve relevant inductive and explanatory purposes such that C is the maximal class whose members are likely to share these properties because of some uniform causal mechanism.

In the following, we will argue that episodic memory as analyzed above indeed is a natural kind. Our argumentation will proceed along two cornerstones:

S1: The principal anatomical substrate of episodic memory is the hippocampus.
- S1.1: The principal function of the hippocampus is episodic memory. That is, all processes hosted by the hippocampus directly or indirectly contribute to episodic memory.
- S1.2: Episodic memory is principally hosted by the hippocampus. That is: Even though episodic memory involves interactions with other cognitive processes, which are supported by a variety of brain regions, processes specific to episodic memory are exclusively hosted by the hippocampus.

S2: Neural processes in the hippocampus provide uniform causal mechanisms for all non-external conditions of the Sequence Analysis.
- S2.1: The hippocampus provides a uniform causal mechanism that aligns the sequential representation of mnemonic content with the sequential representation of the experiential base (conjunction of conditions ii & iii & v).
- S2.2: The hippocampus provides a uniform causal mechanism for the mnemonic representation of episodes and its mnemonic simulation (condition ii and vii).
- S2.3: Interventions in the memory trace warrant that mnemonic representations are causally grounded in experiences (condition vi).

The anatomical basis of episodic memory

Our case for episodic memory being a natural kind would be strengthened, if there were a dedicated brain region to support episodic memory. Here we argue that the hippocampus is this region (S1).

The role of the hippocampus in episodic memory

The first and most important hint that the hippocampus is involved in episodic memory was the observation in patient HM. After both his hippocampi were removed in a surgery to control his epileptic seizures, he could no longer form new episodic memories (Scoville & Milner, 1957). This condition is called anterograde amnesia. Intriguingly, HM did not suffer apparent impairments on most other cognitive functions such as language, perception and working memory. Over the years, these basic and many other observation have been confirmed in a number of hippocampal patients (Squire & Zola-Morgan, 1988). Amnesics also lose memories of past episodes, i.e., from the period before the hippocampal damage (retrograde amnesia). Intriguingly, memories from the remote past appear to be less affected than recently formed memories. This gradient of retrograde amnesia had been observed earlier after head trauma that did not involve permanent brain damage (Ribot, 1881). The process by which episodic memories become less prone to disruption is known as systems consolidation.

It remains controversial whether the hippocampus is important for semantic memory as well. Acquisition of new semantic memory was reported to be very slow and laborious in amnesics (Levy, Bayley, & Squire, 2004). However, studies on subjects who became amnesic during childhood revealed that they had acquired sufficient semantic memory to do well in secondary education (Vargha-Khadem et al., 1997), suggesting that semantic memories were learned without a functioning hippocampus. A resolution between these opposing conclusion was suggested by a more recent study that showed that a special learning protocol, called fast mapping, allows amnesics to rapidly learn new semantic information (Sharon, Moscovitch, & Gilboa, 2011). These results suggest that there are multiple ways in which semantic memories can be formed, only one of which depends on the hippocampus and thus on episodic memory.

The hippocampus is required for learning tasks that require associations across temporal gaps and processing of temporal sequences. For instance, in learning sequences of odors in the same location (Fortin, Agster, & Eichenbaum, 2002), disambiguation of overlapping sequences (Agster, Fortin, & Eichenbaum, 2002) and for trace conditioning (Weiss, Bouwmeester, Power, & Disterhoft, 1999). Sequence learning has a clear relationship to episodic memory in the Sequence Analysis. In trace conditioning, animals learn to associate an initially neutral stimulus, such as a tone, with a stimulus that elicits an automatic response, such as an electric shock. The crucial point is that the two stimuli do not overlap in time. If they do (delay conditioning), learning is independent of the hippocampus. We suggest that trace conditioning requires the hippocampus because episodic memory is required to learn the task. Since episodes are extended in time in the Sequence Analysis, episodic memory is apt to bridge the temporal gap between the two stimuli. Since these experiments were performed extensively in rodents and monkeys with consistent results, we conclude that non-human animals probably have episodic memory.

Another prominent example of hippocampally-dependent process is spatial memory. Eichenbaum et al. (1999) suggest that episodic memory is the primary function of the hippocampus and spatial information is only one aspect thereof. By contrast, O'Keefe and Nadel (1978) argue that a

cognitive map evolved in the hippocampus of non-human mammals to support spatial navigation and that this cognitive map is used in humans to support episodic memory. Either way, spatial information can be considered to be a part of the content of episodic memory (Cheng, 2013).

The above experimental results, taken together, suggest that the hippocampus is largely dedicated to the storage and retrieval of episodic memories (S1.1).

The role of other brain regions for episodic memory

Experimental evidence suggests that other brain regions, in particular the prefrontal cortex, are involved in the formation and retrieval of episodic memories. For instance, after lesions of the prefrontal cortex (PFC), patients have a deficit in effortful memory tasks such as recognition, cued-recall and free recall (Wheeler, Stuss, & Tulving, 1995). In addition, imaging studies revealed that the PFC is activated during encoding and retrieval of episodic memories. It was suggested that the two hemispheres are activated asymmetrically during different memory phases with the left more active during encoding and the right more during retrieval. However, later studies suggest that the left-right asymmetry depends on the content of the memory rather than on the memory phase (Golby et al., 2001). The left PFC was more active for verbal tasks, whereas the right PFC was more active in non-verbal tasks. Interestingly, these asymmetries are similar to the verbal/non-verbal asymmetries observed after hippocampal lesions.

However, general episodic memory is only slightly impaired after lesions of the PFC. So patients with restricted frontal lesions are not usually considered amnesic (Wheeler et al., 1995). In addition, cognitive deficits are much more widespread after frontal than after hippocampal lesions. The affected functions are collectively referred to as executive control and include, among others, task switching (Milner, 1963), decision making (Bechara, Damasio, Damasio, & Anderson, 1994), and working memory (Jacobsen, 1935). In summary, to the best of our knowledge, there is no convincing evidence that any other brain region is as central for the formation of episodic memory as the hippocampus. While it is always possible that future studies will reveal such a brain region, until that time, it is most parsimonious to assume that the hippocampus plays a unique role in episodic memory.

The preceding statement does not imply that episodic memory is stored and retrieved in the hippocampus alone. On the contrary, we believe that the hippocampus is part of a network that performs these functions and that neocortex is critical for processing the sensory information to be stored, for initiating memory retrieval and for processing the retrieved information (Nadel & Moscovitch, 1998). Specifically, what we mean by "the hippocampus plays a unique role in episodic memory" is that the hippocampus endows the cortico-hippocampal network with a capability that the network does not have without the hippocampus. For instance, a recent modeling study suggested that the hippocampus enables the cortico-hippocampal network to associate two inputs across significant time gaps (Pyka and Cheng, 2013 in preparation). This function emerges from two simple anatomical properties of the biological network. Heterogeneous synaptic conductance delays between neocortex and hippocampus, and a high degree of convergence from cortical to hippocampal cells. Without the hippocampus, the network can still learn associations, but not across large time gaps. Without the neocortex, the model cannot learn any associations. So both neocortex and hippocampus are required, but the hippocampus adds a more specialized functionality to the network. We are therefore justified in saying that, in the model, the hippocampus plays a special role in learning an association across larger time gaps. We think that a similar characterization can be applied to the biological cortico-hippocampal network.

Here one could object that even if the hippocampus played a special role in the formation of episodic memory, remote episodic memories that are consolidated can be retrieved without a hippocampus. And that, therefore, other brain regions might sustain at least some episodic memories. This is exactly what the transfer hypothesis proposes (McClelland, McNaughton, & O'Reilly, 1995) and consistent with imaging studies that find activations above baseline in the hippocampus for retrieval of recent memories, but activity in neocortical regions for remote memories (Bontempi, Laurent-Demir, Destrade, & Jaffard, 1999). If the transfer hypothesis were correct, it would significantly weaken our case for episodic memory being a natural kind, since it would reduce the strong link between the hippocampus and episodic memory.

However, there are alternative hypotheses that account for the gradient in retrograde amnesia. For instance, the multiple memory trace theory proposes that multiple memory traces are established through repeated retrievals (Nadel & Moscovitch, 1997) and that the nature of these traces can differ (Cheng, 2013). Only episodic memories are stored in the hippocampus, semantic information is extracted from episodic memories and stored in the neocortex. The gradient of retrograde amnesia is thought to arise because more remote memories are more likely to have been retrieved repeatedly, and therefore to have spawned more memory traces. As a consequence, when amnesics remember their remote past, they retrieve semantic memories grounded in episodic memories, not the episodic memory. Episodic memories, even remote ones, would always require the hippocampus. This is the view we adopt here.

In summary, no other brain region is known to support episodic memory to the degree that the hippocampus does (S1.2).

Neural mechanisms of episodic memory

Neural activity can give us a window into what S represents at a given time and how the representation at one time is related to that at another time (S2). Since the hippocampus is the crucial brain structure for episodic memory, we review below experimental observations of the neural activity in the hippocampus with a special focus on neuronal sequences and their mnemonic function. Many authors have suggested that the hippocampal circuitry is optimized for storing neural sequences. More specifically, it has been suggested that the dense recurrent network in subarea CA3 is well suited to generate neural sequences (Azizi, Wiskott,

& Cheng, 2013). Perhaps, the ability to store extended neural sequences is what distinguishes the hippocampus from neocortex.

Corballis (2013) has argued previously that neural sequences are an indication that non-human animals have episodic memory. However, his view of episodic memory depends on mental time travel and he needs to argue that neural sequences are a correlate of the subjective experience of the animal, something we avoid in our approach.

Phase precession and theta sequences in the hippocampus

Principal cells in the hippocampus are active in specific, circumscribed spatial regions (place fields) and are therefore called place cells (Fig. 1A). The precise timing of a place cell's action potentials, or spikes, is further regulated by three prominent network oscillations in the hippocampus: theta (5-12 Hz), gamma (30-120 Hz) and sharp-wave ripples (150-250 Hz). Spatial location and network oscillations have a combined influence on the firing activity of place cells.

A theta oscillation occurs when animals are actively involved in a task, which we refer to as online state (Buzsaki, 1989). During spatial exploration, place cells initially fire spikes at the peak of this theta oscillation and then at earlier and earlier phases of the theta oscillation as the animal enters and traverses the place field (O'Keefe & Recce, 1993). This phenomenon is observed when the spiking activity of a single place cell is followed across multiple cycles of the theta oscillation and is called theta phase precession (that is the correlation between the phase of theta and the position of the animal within the place field of the place cell as shown in Fig. 1B). However, an alternative view emerges, when the spiking of multiple place cells are observed within a single theta cycle: While the animal runs along a trajectory in the online state, place cells fire in a temporal sequence that corresponds to the spatial succession of their respective overlapping place fields (W. Skaggs, McNaughton, Wilson, & Barnes, 1996) (Fig. 1C). In the online state, thus an episode $E^* = \langle e_1^*, e_2^*, e_3^* \rangle$ is represented.

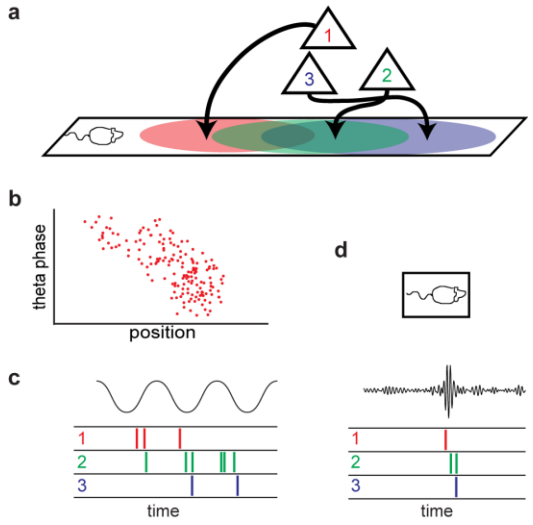

Figure 1. *Schematic illustration of neural activity of hippocampal neurons.* **a**: As the animal explores the linear track, place cells (1,2,3) fire spikes when the animal is located in a circumscribed region in space, the place field (indicated by three colored ellipses). **b**: In addition, the spiking of place cells is modulated by the phase of the theta oscillation. Each red dot marks the theta phase and position of the animal when neuron 1 fired a spike as the animal run from left to right. The correlation between phase and position associated with spikes is known as theta phase precession. **c**: When spiking of a group of place cells is analyzed within one cycle of the theta oscillation (black trace at the top), temporal sequences emerge across neurons (theta sequences). **d**: During the offline state, sharp wave/ ripples occur in the local field potential (black trace in middle, filtered between 150 and 250 Hz) and place cells are reactivated in a sequence that is related to the theta sequences.

Theta phase precession demonstrates a remarkably precise temporal coordination between cells in the hippocampus. Initial suggestions favored the view that phase precession in individual neurons gives rise to the sequential activity of groups of place cells (W. Skaggs et al., 1996). However, other studies have argued that it is rather the other way around and that phase precession might instead be the product of sequential activity (Tsodyks, Skaggs, Sejnowski, & McNaughton, 1996). More recent experimental studies claim that the timing of action potentials within a theta cycle is more precise than would be expected from phase precession and coined the term "theta sequences" (Foster & Wilson, 2007). Whichever phenomenon is primary, either one can serve to align the sequential representation of mnemonic content in the offline state (discussed in the next section) with the sequential representation of the experiential base in the online state (S2.1).

This last conclusion needs to be explained in more detail. Take, for instance, the episode of the animal running from left to right on the linear track in Fig. 1A, where the experiential base E^* consists of three events $E^* = \langle e_1^*, e_2^*, e_3^* \rangle$. Each of the events occurs at one of the locations marked by arrows on the track. We can assign each location to the place cell that has a place field center closest to that location. Thus, the place cells 1, 2, and 3 would be assigned to the representation of the events e_1^*,e_2^*, and e_3^* respectively (Fig. 1A). In principle, the three place cells could fire spikes at any time while the animal is located in their respective place fields. If some place fields overlapped, as they do in the case of place cells 1, 2 and 3, then the spikes of the corresponding place cells would occur in random order. However, due to phase precession or theta sequences, the spiking of place cells is temporally ordered along the succession of the positions of their place fields, thus the representation of the experiential base is sequential. Since the same place cells also participate in the mnemonic representation of $E = \langle e_1, e_2, e_3 \rangle$ (more on this below), the two representations become aligned.

Offline sequential activity and replay in the hippocampus

In the offline state, when the animal sits quietly or falls asleep, sharp-wave ripples (SWRs, Fig. 1D) dominate network oscillations. SWRs have been observed, e.g., in rodents (Buzsáki, Leung, & Vanderwolf, 1983), and human hippocampus and entorhinal cortex (Bragin, Engel, Wilson, Fried, & Buzsáki, 1999), suggesting that SWRs are part of a general, conserved mechanism. Concurrent with SWRs in

the hippocampus, populations of place cells fire spikes in a temporal sequence within a 50-400 ms time-window.

The critical point is that the offline sequence is correlated with and influenced by preceding online activity (Fig 1D; for a more detailed review see (Buhry, Azizi, & Cheng, 2011). Individual place cells that are active during behavior are more likely to be active again during subsequent sleep and quiescence than those place cells that were not active during explorations. Subsequent studies reported the reactivation of pairs of cells, which also preserve their ordering (W. E. Skaggs & McNaughton, 1996). Most importantly, populations of neurons become active in the offline state in a sequence that correlates with the sequence, in which they were active at an earlier time in the online state. This means that the temporal sequence of the mnemonic content episode is aligned with the temporal sequence of the episode of the experiential content. Thus, neural activity reactivated in the offline state is a replay of prior experience. Replay has been observed across species and brain regions, such as rodent hippocampus, rodent PFC, primate motor, somatosensory, and parietal cortex (but not prefrontal cortex) and during free recall of movie sequences in humans (Gelbard-Sagiv, Mukamel, Harel, Malach, & Fried, 2008). These results strongly suggest that offline neural sequences are the representation of the mnemonic content $E = \langle e_1, e_2, e_3 \rangle$ of episodic memory (S2.2).

Replay in the hippocampus is linked to the formation and consolidation of episodic memory

The final crucial aspect missing from our discussion is evidence for a memory trace that causally links the experienced episode to the mnemonic representation. The most relevant studies in this regard are those that examine the link between offline sequences and the systems consolidation process. Much time and effort has been devoted to understanding the exact properties and neural mechanisms of consolidation. Buzsaki (1989) proposed that, first, a labile memory trace is formed in the hippocampus during the online state. Then, during subsequent offline states, hippocampal replay gradually transfers the memory trace to the sensory cortical areas (McClelland et al., 1995).

To examine the functional role of neural sequences, a number of studies exploited its co-occurrence with SWR (Fig. 1D). Mounting experimental evidence suggests that SWRs are important for learning and memory. For instance, the rate of SWRs was found to be higher in a novel than in a familiar part of an environment and so is the spiking probability of place cells (Cheng & Frank, 2008). SWRs were observed to increase during slow-wave sleep after learning. The number of rhinal SWRs in humans during a daytime nap appears to be correlated with the number of successfully recalled items learned prior to sleep. Disrupting SWR in rats during sleep after a learning session interferes with the formation of long-term memories. Disrupting SWRs in rat hippocampus during the awake state disrupted learning a spatial working memory task. Taken together, these results suggest that offline sequences in the hippocampus are involved in maintaining the memory trace of episodic memories (Cheng & Werning, 2013), and that the memory trace causally links experiences to their mnemonic representation (S2.3).

For completeness, we note that not all sequential activity in the hippocampus is causally grounded in previously experienced sequences. During exploration, theta sequences appear to begin in the past and sweep to anticipated locations (Gupta, van der Meer, Touretzky, & Redish, 2012). These results suggest that memories are retrieved during exploration, as would be required if memory has to influence future behaviors. Offline sequences also do not strictly correlate with sequences experienced in the past. In the awake state, Gupta et al. (2010) reported that trajectories that the animal had never traveled were replayed in the hippocampus, and Dragoi and Tonegawa (2011) reported evidence for pre-play. Neural sequences recorded during rest were predictive of the sequence of the neurons' place fields a linear track that the animal had never experienced before. These results suggest that sequences are an intrinsic feature of the hippocampal network. However, this conclusion does not undermine our argument for a causal memory trace since reactivated sequences occur more often and/ or exhibit stronger correlations than spontaneous, intrinsic sequences (Kudrimoti, Barnes, & McNaughton, 1999; Wilson & McNaughton, 1994).

Conclusion

The starting point for us in this paper has been the insight that the two questions: "What is episodic memory?" and "Is episodic memory a natural kind?" are inherently connected to each other. The first question cannot be answered intelligently without aiming at a positive answer of the second: It would be scientifically and philosophically rather futile to have a notion of episodic memory that, for better or worse, matches our conceptual intuitions, our linguistic practice and perhaps some introspective phenomenology, but does not refer to a natural kind. Such a notion would be fictive or, at best, allow for a *fiat* entity. Falling under a natural kind is a prerequisite for a phenomenon to be justly regarded as real.

In turn, an answer to the second question has to be assessed in light of the consequences it has for the first. It would not suffice to enlist a number of neural mechanisms that amount to particular psychological properties and label them "episodic memory". What has to be done in addition is to show that uniform causal mechanisms explain why the psychological properties are shared such that the cluster of those properties subserve inductive and explanatory purposes of what we are to understand is episodic memory. Any other approach could easily be criticized as a change of subject.

In search for an answer to the conditional question "What is episodic memory if it is a natural kind?" we have tied analytical and empirical approaches most closely together. In the Sequence Analysis episodic memory is conceived of as a factive, knowledge-like state that consists of an experientially based mnemonic representation. We have stressed the sequential character of the mnemonic content as being an episode. That is, a temporally ordered list of particular concrete events.

We have tried to validate the Sequence Analysis of episodic memory as corresponding to a natural kind by proceeding along two empirical cornerstones: First, Do the

empirical data support a claim of what the principal anatomical substrate of episodic memory is, given that the Sequence Analysis holds? We have pointed to a great deal of evidence that there is one: the hippocampus. Secondly, do we know the neural activities in the hippocampus onto which we can pin down causal mechanisms in order to explain the psychological states and processes appealed to by the Sequence Analysis? Also here we could call on a body of evidence from neuroscience.

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