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Opinion

A neural state space for episodic memories

Aidan J. Horner ^{1,2,*}

Episodic memories are highly dynamic and change in nonlinear ways over time. This dynamism is not captured by existing systems consolidation theories that predict a unidirectional process where memories are first supported by the hippocampus and then the neocortex. Here, I propose a 3D state space for episodic memories. The first two dimensions relate to whether episodic retrieval is driven by the hippocampus and the neocortex, critically allowing for independent and additive contributions from both regions. The third dimension relates to the episodic specificity of retrieval. Memories can be located at any point in this state space and move to any other location. The state space captures the dynamic nature of episodic memory and broadens the search space of possible memory states and transformations across time.

The dynamic nature of episodic memory

Episodic memory, our ability to recollect previous life events, is highly dynamic and reconstructive [1–4]. Once encoded, memories change, and their expression relies on different neural mechanisms and brain regions. To explain this dynamism, multiple theories of **systems consolidation** (see [Glossary](#)) have been proposed that capture certain aspects of this process ([Box 1](#)) [5–12], such as the potential time-limited role of the hippocampus in supporting episodic memory. However, these theories fail to fully capture the dynamic and reconstructive nature of episodic memory. For example, the role of the hippocampus may not always be time limited and it may remerge as a critical structure [13,14], reversing the predicted hippocampal–neocortical transfer of regions that supports recollection. Although this dynamism and flexibility have recently been noted [15,16], we lack a theoretical framework for dealing with such dynamism. Here, I propose a new ‘state space’ framework to understand how episodic memories are expressed and how we can track changes to this expression over time. Critically, the proposed neural state space for episodic memories allows for dynamic changes in memory expression over time, which most theories of systems consolidation are not able to explain.

I first outline current theories of episodic memory retrieval and systems consolidation before challenging some key assumptions of theories of systems consolidation, highlighting how most theories are not able to fully capture the dynamic nature of episodic memory over time. I use this critique to introduce a new framework, a neural state space for episodic memory, which allows for the retrieval of memories to change dynamically over time. I demonstrate how existing theories sit within this neural state space and outline experimental approaches that would allow for an exploration of it.

Neocortical reinstatement underpins episodic recollection

When we experience an event, the individual elements of the event, such as where we are and who we are with, are represented in patterns of neural activity in distinct neocortical regions ([Figure 1](#)). These independently represented elements are bound into a **memory trace** in the hippocampus [17–20], allowing for the retrieval of the event later in time. If I return to the same location I visited recently, the hippocampus retrieves the other elements of the event via **pattern completion** [21–23], driving the **reinstatement** of that information in the neocortex [24–27].

Highlights

Systems consolidation theories predict that the retrieval of a memory is initially hippocampus dependent but, over time, is supported by the neocortex, and that retrieval becomes less episodically specific during this process.

Research suggests that memories are more dynamic than these theories predict: the time course of the contributions made by the hippocampus and neocortex are highly flexible and mechanistically independent.

Episodic memories can be located in a 3D state space: reinstatement is driven by two independent mechanisms (Dimensions 1 and 2) and can be highly specific or more gist-like (Dimension 3).

Episodic memories can occupy any location in this state space and move to any other location.

The episodic memory state space captures the dynamic and flexible nature of episodic memory over time and highlights currently underexplored memory states.

¹Department of Psychology, University of York, York, UK

²York Biomedical Research Institute, University of York, York, UK

*Correspondence: aidan.horner@york.ac.uk (A.J. Horner).



Box 1. Theories of systems consolidation

Standard Systems Consolidation Theory

Standard Systems Consolidation Theory (SSCT) proposes that the hippocampus rapidly encodes new episodic experiences and is critical to their retrieval [5,7]. Over time, there is a ‘transfer’ (see Box 2 in the main text) from the hippocampus to neocortex, such that the retrieval of events becomes less hippocampal dependent over time. This hippocampal–neocortical ‘transfer’ is thought to occur via offline reactivation of hippocampal traces, leading to the gradual formation of nonhippocampal connections between neocortical representations.

Multiple Trace Theory

Multiple Trace Theory (MTT) proposes that the hippocampus is always critical to the retrieval of episodic events and that, each time an event is retrieved, a new hippocampal trace will be formed, creating multiple similar traces of the original experience [10]. The overlapping traces are thought to allow for the extraction of generalities over time, supporting the emergence of semantic knowledge from episodic events.

Trace Transformation Theory

Trace Transformation Theory (TTT) builds on SSCT and MTT, proposing that the hippocampus is always critical to the retrieval of episodic events; however, overtime, the neocortex can support the retrieval of more ‘gist-like’ or semanticised memories [8,9,12]. Thus, the ‘transfer’ from the hippocampus to neocortex is accompanied by a change from episodically specific to more gist-like retrieval. Once both a detail-rich and a gist-like representation has been formed, TTT allows for either trace to dominate at retrieval over time.

Scene Construction Theory

Scene Construction Theory (SCT) proposes that the hippocampus is always needed for the construction of the episodic context or ‘scene’ of a previous event [11]. While hippocampal traces are predicted to have a relatively short lifespan, a new hippocampal trace is re-established at longer time delays via the reconstruction of consolidated neocortical representations associated with the event. Thus, whereas the elements of an event can be ‘consolidated’ in the neocortex, the reconstruction of the event into a spatially coherent ‘scene’ requires the hippocampus regardless of delay.

However, the hippocampus does not directly project to all neocortical regions where reinstatement occurs [28–30]. Instead, reinstatement is driven by a process where neocortical representations are ‘reconstructed’ via back projections along specific neocortical processing pathways [31,32]. Indeed, reinstatement has been shown in both higher-order [24–27] and primary visual regions [33–36], demonstrating that reinstatement can flow back along the entire processing pathway. This process should occur along multiple processing pathways related to the elements of an event. For example, reinstatement of object and location representations might independently occur via reconstructing representations in the ventral and dorsal visual pathways [37]. Therefore, recollection is underpinned by neocortical reinstatement, which occurs via a reconstructive process along neocortical processing pathways.

Current theories of systems consolidation

Theories of systems consolidation propose a time-limited role for the hippocampus in driving this reinstatement [5,7]. When an event is first experienced, no direct connections exist between the neocortically represented event elements, and the hippocampus is needed to bind together and subsequently retrieve these elements. Over time, an active process of systems consolidation is thought to occur, where connections between the independent neocortical representations form, decreasing the need for the hippocampus to drive reinstatement (Figure 1). These new connections are thought to be formed either directly between neocortical regions [38], or via a second ‘hub’, such as the ventromedial prefrontal cortex [39]. These connections are predicted to form gradually via the ‘reactivation’ of hippocampal traces, potentially during offline periods [7], such as non-REM sleep [40,41].

Despite variations across different theories of systems consolidation (Box 1), there is general agreement to the predictions that: (i) episodic recollection is underpinned by neocortical

Glossary

Electrical temporal interference

stimulation (TIS): non-invasive neuromodulation technique that uses multiple electrical fields at different frequencies, allowing for stimulation at superficial and deep (e.g., the hippocampus) brain regions where the fields converge.

Memory trace: interconnected network of neurons in the brain that allows for the retrieval of a previously experienced event at a later time point.

Multivariate pattern analysis

(MVPA): analysis and comparison of patterns of activity in the brain that are associated with particular experimental conditions or mental states.

Neurogenesis: creation of new neurons throughout the lifespan, often seen in the dentate gyrus of the hippocampus.

Pattern completion: retrieval of a complete neural representation or pattern in the presence of an ambiguous or partial input, commonly associated with subfield CA3 of the hippocampus.

Reinstatement: re-establishment of a similar distributed pattern of neocortical activity during retrieval to that seen when the event was first experienced.

Systems consolidation:

reorganisation of a memory trace such that it is supported by different brain regions, typically between an initially acquired hippocampal memory trace to a trace that is less hippocampal dependent.

Systems reconsolidation: process by which a memory that has undergone systems consolidation to become hippocampus independent becomes hippocampal dependent following reactivation of the memory via a reminder cue.

Transcranial focussed ultrasound

stimulation (tFUS): non-invasive neuromodulation technique that focusses ultrasound waves to modulate both superficial and deep (e.g., the hippocampus) brain regions.

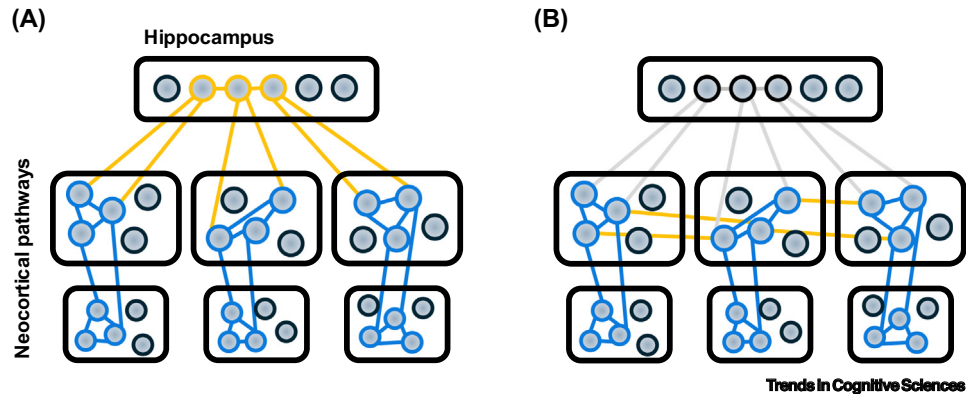


Figure 1. A modal neurocognitive model of episodic retrieval and systems consolidation. (A) When experiencing an event, a distributed pattern of activity occurs along neocortical processing pathways (pathways shown here are intentionally 'generic' but could map to ventral and dorsal visual pathways as well as other sensory processing pathways; e.g., auditory). These pathways converge on the hippocampus and are bound into a memory trace allowing for their retrieval later in time. At encoding, all neocortical representations are active, forming a new hippocampal memory trace. At retrieval, a subset of neocortical representations will be active, allowing for the retrieval of the hippocampal memory trace and subsequent reinstatement of the remaining neocortical representations. (B) Over time, connections between neocortical regions form, allowing for their retrieval without the involvement of the hippocampus. Blue lines indicate pre-existing connections/representations; yellow lines indicate newly formed connections/representations; grey lines indicate previously formed connections that have weakened.

reinstatement; (ii) neocortical reinstatement is initially driven by the hippocampus; and (iii) over time, the role of the hippocampus as a driver of reinstatement decreases and another nonhippocampal mechanism drives reinstatement. A final key prediction by specific theories is that, over the course of systems consolidation, reinstatement becomes less episodically specific and more 'gist-like' [8,9]. Critically, recent evidence suggests that none of these theories are able to fully capture the dynamic nature of episodic memory.

From hippocampal to neocortical retrieval

Given that recollection is underpinned by the reconstruction of representations along neocortical processing pathways [21,32,37], how do the above tenets of systems consolidation fit with this retrieval mechanism? According to systems consolidation, reinstatement can occur via two mechanisms: (i) the retrieval of a hippocampal memory trace that drives reinstatement (Figure 1A); and/or (ii) a nonhippocampal mechanism (Figure 1B). Critically, reinstatement should become less hippocampally driven and more driven by a nonhippocampal mechanism over time. However, despite this general prediction, theories are rarely explicit in relation to how reinstatement occurs when both mechanisms are available to drive reinstatement and how these two mechanisms are related over time.

How do the two drivers of reinstatement interact at the point of retrieval? Are they compensatory, such that, if one fails to drive reinstatement, the other takes over, or are they instead additive, such that the strength of reinstatement is driven by the combined contribution of both mechanisms? Evidence has recently been provided for an additive relationship, that is, that both mechanisms cumulatively contribute to reinstatement. Using fMRI, neocortical reinstatement was correlated with a neural marker of hippocampal pattern completion (hippocampal activity during a memory task where complex events were retrieved in a 'holistic' manner, consistent with the presence of pattern completion). Immediately after encoding, a positive relationship was seen, whereby the greater the evidence for hippocampal pattern completion the greater the evidence for neocortical reinstatement [21,42,43]. Importantly, when there was no evidence for hippocampal pattern completion,

there was no evidence for reinstatement (formally, the intercept of the correlation is zero). This suggests that, immediately after encoding, the hippocampus is the sole driver of reinstatement.

After 24 h, there was still a significant correlation between the hippocampus and reinstatement, suggesting that the hippocampus was still driving reinstatement, but now the correlation had a non-zero intercept [43]. This positive intercept provides evidence for reinstatement in the absence of evidence for hippocampal pattern completion. From this, it can be inferred that another, nonhippocampal, mechanism was contributing to reinstatement after 24 h. Critically, although there was a significant increase in the intercept over 24 h (from 0 to >0), implying an increase in a nonhippocampal mechanism, there was no evidence for a decrease in the slope of the relationship, implying there was no decrease in the hippocampal mechanism. These data suggest that the two mechanisms additively contribute to neocortical reinstatement: the hippocampus still drives reinstatement to the same degree after 24 h, but reinstatement is now being boosted by a nonhippocampal mechanism. Further empirical studies need to assess the additive nature of reinstatement, ideally using within-subject correlations (as opposed to the across-subject correlations used in [21,43]) that allow for the assessment of reinstatement at the level of individual episodic memories.

In relation to how the two mechanisms are related over time, Complementary Learning Systems [a computational instantiation of Standard Systems Consolidation Theory (SSCT)] makes the explicit prediction that the nonhippocampal mechanism increases in strength as a function of repeated interleaved reactivation during offline periods [7]. There is ample evidence supporting this prediction [44–48]. The hippocampal mechanism in this model decreases as a consequence of passive decay over time, and recent research supports such a decay mechanism [49], potentially driven via hippocampal **neurogenesis** [50,51]. Thus, the causes of an increase in one mechanism and a decrease in the other are mechanistically distinct: reactivation versus decay.

Current empirical research suggests that the two mechanisms that drive reinstatement are independent and additively contribute to reinstatement and that the increase/decrease of these mechanisms are independent. Further, if they are mechanistically distinct, then the time course with which they increase/decrease could differ. They may be loosely temporally correlated, but that does not mean that they are mechanistically related to one another or follow the same precise time course. The conclusion is that two independent mechanisms with different time courses additively contribute to neocortical reinstatement.

From detail-rich to gist-like retrieval

A second key concept is the process of semanticisation, where episodic memories become more generalised or ‘gist-like’ as a function of systems consolidation. This is specifically a prediction of Trace Transformation Theory (TTT) [8,9,12]. Under this theory, the hippocampus supports episodically specific, detail-rich representations and the neocortex supports more abstract, gist-like representations.

There is evidence showing decreased specificity of the remembered event over time, particularly for the specific perceptual and/or peripheral aspects of the event [52,53]. However, it is not clear whether this more rapid forgetting of event details is mechanistically linked to systems consolidation. If recollection is underpinned by the reconstruction of neocortical representations, then it is not clear why the hippocampus should drive more detail-rich reinstatement than would a nonhippocampal mechanism, particularly given that the hippocampus does not have direct projections to lower-level sensory regions [28–30].

'Episodic specificity' can be conceptualised in two ways: in relation to: (i) the perceptual specificity of reinstated neocortical representations; and (ii) the forgetting of certain aspects of the event and not others (i.e., the 'coherence' of memory retrieval). In the former, we might reinstate all event elements, but less specifically. For example, we might forget that the apple was green and not red. If perceptual specificity at retrieval is a consequence of how far back along a given processing pathway reactivation occurs, there is no clear reason why the hippocampus should drive reinstatement further back down processing pathways compared with a nonhippocampal mechanism. Critically, there are currently no empirical data showing that hippocampally driven reinstatement occurs further back along a given processing pathway relative to nonhippocampally driven reinstatement. Thus, there is no reason to expect a loss in perceptual specificity as a function of systems consolidation.

If episodic specificity is a function of forgetting certain aspects of the event and not others, such as forgetting where we found the apple but still remembering the apple was green, there is no reason to assume this should occur as a direct consequence of systems consolidation. While offline reactivation might cause certain direct neocortical connections to form at the expense of others, thereby prioritising some aspects of the event, this forgetting of specific elements could occur independently of the active process of systems consolidation (e.g., via forgetting mechanisms, such as interference or decay [54–58]).

In sum, there is no reason that an active process of offline reactivation should cause a change in what is being reactivated. Two independent mechanisms could drive the reinstatement of the same pattern, which would result in the same degree of episodic specificity at retrieval. Instead, the loss in specificity might be temporally correlated with systems consolidation, but caused by forgetting. It should not be assumed that a loss in episodic specificity at retrieval is mechanistically related to systems consolidation simply because it is temporally correlated.

The dynamic nature of episodic memory

I have argued that three processes that often temporally co-occur are not mechanistically related, as is assumed in systems consolidation theories. Neocortical reinstatement can be driven, in an additive manner, by two independent mechanisms and the increase and decrease over time of these mechanisms are independent. Furthermore the specificity with which reinstatement occurs is related to mechanisms that are theoretically independent of what is driving the reinstatement. For example, a strong input from either a hippocampal or nonhippocampal mechanism could both cause a high degree of episodic specificity.

A further critical assumption in most theories of systems consolidation is that the process is unidirectional, from hippocampally driven reinstatement to nonhippocampally driven reinstatement over time. Agreeing with TTT [8,12], I challenge this assumption. First, the hippocampal mechanism can be maintained over longer periods of time by the application of norepinephrine to the amygdala shortly after initial encoding [59]. Second, the nonhippocampal mechanism does not have to slowly emerge over time but can be engaged rapidly either through retrieval-based mechanisms [60–62] or when events are aligned with our schematic understanding of the world [63,64]. Thus, the time course of both mechanisms can be substantially altered. More critically, the hippocampal mechanism can be increased [65] or even re-engaged [13,14] as a driver of reinstatement at longer delays, suggesting that the typical direction of systems consolidation can be entirely reversed. This hippocampal re-engagement is highlighted in studies of **systems reconsolidation** (a process related to the broader concept of reconsolidation [66–69]), where a memory trace that is thought to have undergone systems consolidation and, therefore, is hippocampus independent, becomes dependent on the hippocampus again following memory reactivation via a reminder cue [13,14]. Thus, the extent to which the hippocampal and

nonhippocampal mechanisms contribute to reinstatement can differ both after initial encoding and over time in nonlinear and non-unidirectional ways.

Finally, the degree to which reinstatement is episodically specific is also likely highly dynamic. Although specific episodic detail will often be lost over time, some situations likely allow for maintained specificity at the point of retrieval. For example, you might experience an event with a specific object (e.g., your favourite coffee cup). Given that you are then likely to encounter the same coffee cup repeatedly, when retrieving the first event you will be able to reinstate a high degree of perceptual specificity in relation to the cup. Conversely, if you saw lots of different coffee cups in the intervening period, the specificity of reinstatement for the cup would likely be less detailed and more 'gist-like'. Given that neocortical reinstatement is a reconstructive process, 'episodic specificity' is more likely to be driven by a range of factors, including long-term (personal) semantic knowledge [70,71] and experiences following the critical event, rather than what mechanism is driving reinstatement. Indeed, recent research shows that the hippocampus can drive both specific and gist-like retrieval, a process mediated by hippocampal neurogenesis after initial encoding [72,73], underlining how the specificity of reinstatement can be independent of the mechanisms driving reinstatement. Although TTT allows for dynamic changes in episodic specificity over time, it does not allow for this specificity to be decoupled from the mechanism driving reinstatement [8].

A neural state space for episodic memories

I have argued for at least three independent processes that are often mechanistically linked in systems consolidation theories: (i) the creation and loss of a hippocampal trace that drives reinstatement; (ii) the creation and loss of a nonhippocampal mechanism that drives reinstatement; and (iii) the episodic specificity of reinstatement at retrieval. I have also argued that these time courses can differ markedly dependent on encoding conditions and subsequent experiences. To better conceptualise these processes, I propose a 3D neural 'state space', where any given episodic event can be located at any given point in this space at the point of retrieval and move to any other point within the space under specific conditions (Figure 2, Key figure).

The first dimension of the state space is the strength of the hippocampal trace that drives reinstatement (from weak to strong), the second dimension is the strength of the nonhippocampal mechanism that drives reinstatement (from weak to strong), and the third dimension is the degree to which reinstatement is episodically specific (from low to high specificity). It is possible that more independent dimensions exist. For example, there may be multiple nonhippocampal mechanisms that drive reinstatement (e.g., both direct long-range neocortical connections and new neocortical 'hubs', including the ventromedial prefrontal cortex, medial parietal cortex, and angular gyrus). Furthermore, episodic specificity may differ in several ways, including (but not limited to) the perceptual specificity of reinstatement and the degree to which certain aspects of an event are remembered (the 'coherence' of episodic retrieval [74–77]). Here, I focus on these three dimensions for theoretical clarity and because of their importance in relation to theories of systems consolidation. I refer to the second dimension as a 'nonhippocampal' mechanism, because it is still an open question as to what is driving nonhippocampal reinstatement (or whether multiple nonhippocampal mechanisms exist). A similar multidimensional memory space has been proposed [16]; however, the specific dimensions were not formally defined and the emphasis of the framework was related to how memories change to be behaviourally relevant as opposed to the focus here on the neurocognitive mechanisms that support episodic retrieval.

This state space is not a mechanistic account of systems consolidation. However, I have outlined a model of episodic retrieval that focusses on reconstructive reinstatement, which provides a mechanistic basis for interpreting movement through this state space. The state space of episodic memory

Key figure

A neural state space for episodic memories

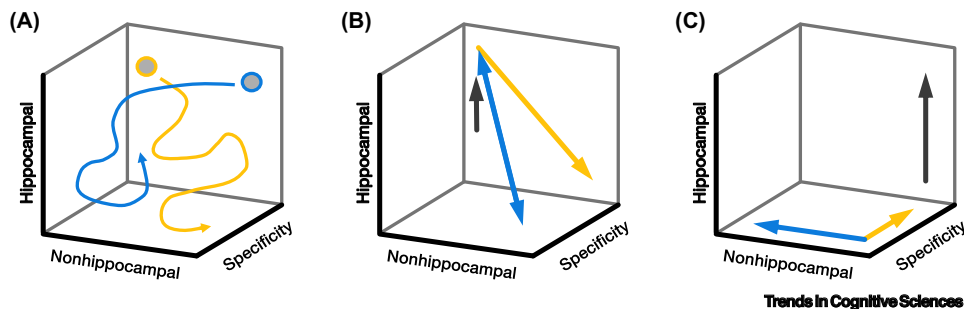


Figure 2. The state space comprises three dimensions: the degree to which reinstatement is driven by a hippocampal mechanism (Hippocampal; weak to strong); the degree to which reinstatement is driven by a nonhippocampal mechanism (Nonhippocampal; weak to strong); and the degree to which reinstatement is episodically specific (Specificity; low to high). (A) The trajectory of two hypothetical memories. When an event is experienced, it forms a memory trace in the hippocampus (yellow circle; strong hippocampal mechanism) and, depending on the congruence of the event with pre-existing schematic knowledge, can also be supported by the nonhippocampal mechanism (blue circle). Memories take dynamic routes through state space over time. (B) Theories of systems consolidation mapped as vectors in the state space. Standard Systems Consolidation Theory (SSCT) starts with strong hippocampal, weak nonhippocampal, and high specificity and moves to weak hippocampal, strong nonhippocampal, and high specificity (yellow arrow). Trace Transformation Theory (TTT) starts with strong hippocampal, weak nonhippocampal, and high specificity and moves to weak hippocampal, strong nonhippocampal, and low specificity (blue arrow). Multiple Trace Theory (MTT) starts with medium hippocampal, weak nonhippocampal, and high specificity and (as more hippocampal traces form) moves to strong hippocampal (the other two dimensions remain the same; grey arrow). Scene Construction Theory (SCT) predicts no movement in this space: the hippocampus is always required for recollection [not shown in (B)]. (C) Potential trajectories through state space that are underexplored and/or not currently predicted by existing theories. The forgetting of ‘consolidated’ memories, from strong nonhippocampal to weak nonhippocampal (blue arrow); an increase in episodic specificity (from low to high) despite reinstatement being driven by the nonhippocampal mechanism (yellow arrow); the reintroduction of the hippocampal mechanism (from weak to strong) following ‘consolidation’ (grey arrow).

is a broader theoretical framework that starts with the assumptions that: (i) three processes that are mechanistically linked in most systems consolidation theories are independent; and (ii) memories can move from any location in this state space to any other location (Figure 2A). The advantage of this state-space framework is that it allows for the mapping of current theories of systems consolidation (Figure 2B) and highlights the locations and directions of travel in the state space that these theories do not predict (Figure 2C). For example, TTT predicts that a recently encoded event will be reinstated by the hippocampus in a detail-rich manner and, following systems consolidation, will often be reinstated by a nonhippocampal mechanism in a more gist-like manner (Box 1). Critically, TTT does not allow memories to move to specific locations in the state space, such as reinstatement that is detail rich occurring via a nonhippocampal mechanism.

This mapping of current systems consolidation theories in the state space highlights regions of the space that have not been considered. For example, strengthening of the hippocampal and nonhippocampal mechanism would result in both mechanisms additively contributing to reinstatement. It is plausible that offline reactivation, or active retrieval, strengthens both mechanisms, leading to memories moving to such a location in the state space. Indeed, it appears unlikely that the offline reactivation of a hippocampal trace would result in the strengthening of the nonhippocampal mechanism without a similar strengthening of the hippocampal trace itself (Box 2). A further underexplored region of this space is the weakening of the nonhippocampal

Box 2. The fallacy of hippocampal–neocortical transfer

The concept of systems consolidation is often associated with a ‘transfer’ from a hippocampal to a neocortical memory trace. Although perhaps not explicitly stated, there is often an implicit assumption that mnemonic information is transferred from the hippocampus to neocortex. However, the expression of an episodic memory is via the reconstructive reinstatement of representations in the neocortex. As such, there is no ‘transfer’ of mnemonic information, but instead, at most, a transfer in relation to what mechanism is driving reinstatement. The representational content is always in the neocortex, it is simply reactivated via different mechanisms over time.

However, a transfer process implies that one mechanism takes over from the other: as the nonhippocampal mechanism increases, the hippocampal mechanism should decrease in an equal manner. Two issues undermine this concept. First, as discussed in the main text, the decrease in hippocampal mechanism is likely driven by an independent process (decay) relative to the process that increases the nonhippocampal mechanism (offline reactivation). Second, it is unclear how the hippocampal mechanism decrease occurs in the presence of continual offline reactivation of the hippocampal trace. Why is reactivation of a hippocampal trace not reinforcing of the trace itself? The default assumption in neuroscience is that synaptic strength increases as a function of neural firing via Hebbian learning. Under this assumption, the reactivation of a hippocampal trace should strengthen itself (as well as increasing any nonhippocampal mechanism). McClelland *et al.* [7] speculate that an additional mechanism might exist, for example the suppression of hippocampal synaptic plasticity during sleep [92] or synaptic downscaling during sleep [93], which suppresses this reinforcement. However, such mechanisms would likely only prevent further strengthening and, in the case of synaptic downscaling, would not be trace specific. As such, offline reactivation could lead to the strengthening of both the hippocampal and nonhippocampal mechanism.

Given that I have argued for two independent mechanisms that drive reinstatement at retrieval and that the decrease in one mechanism is not related to the increase in the other mechanism, then the notion of ‘transfer’ is a fallacy. There is no transfer, but simply two distinct drivers of reinstatement that increase/decrease over time via different mechanisms.

mechanism. While most theories assume that hippocampal traces decay over time, it is unknown how long-range neocortical connections weaken. One possibility is via interference mechanisms that are thought to occur locally in neocortical regions [49,54,57], but it is again unknown whether interference also occurs for more long-range connections.

By highlighting unexplored regions of this state space, the idea is to test the strong assumptions of independence and non-unidirectional movement within it. It is possible that these two assumptions do not hold. For example, there might be some areas of the state space that memories are not able to move to, or certain directions of travel that are not possible. Fully exploring the state space would allow for the identification of planes or manifolds in this space that memories are constrained to, or attractor states that memories gravitate toward. Therefore, this broader theoretical framework allows for a wider range of theoretical perspectives, with the intention that fully exploring the state space will enable the development of more accurate mechanistic models in the future.

Exploring the state space

One critical question is how best to explore this proposed state space. How do we test for the assumptions of independence and non-unidirectional movement? First, we need experimental and analysis approaches that can assess the degree to which reinstatement is driven by the hippocampal and nonhippocampal mechanism. Critically, we need to assess how reinstatement is driven at the point of retrieval, which standard univariate analyses that focus on hippocampal and neocortical changes in activity over time are not able to do [78–84]. The approach outlined earlier, assessing the slope and intercept of the relationship between hippocampal pattern completion and neocortical reinstatement, is able to do this [43], as are functional connectivity measures between the hippocampus and neocortex [78,85]. Second, we need to assess the degree of ‘episodic specificity’ at retrieval. This can be done behaviourally, for example by probing the degree to which participants remember perceptual versus conceptual features of elements in an event [61,86], or more gist-like versus specific features of an event [53]. It can also be achieved

using **multivariate pattern analysis (MVPA)** of neuroimaging data, assessing the specificity of reinstatement in neocortical regions [62]. Critically, these approaches would need to be combined, and used at multiple time points after encoding, to track their proposed independent time courses.

The above approach would assess the independence of the three state space dimensions, but not the extent to which movement is non-unidirectional. One direction of travel that SSCT does not currently predict is the re-emergence of the hippocampus in driving reinstatement (Figure 2C). Recent advances in causally manipulating deeper brain regions, for example with **transcranial focussed ultrasound stimulation (tFUS)** [87–89] or **electrical temporal interference stimulation (TIS)** [90], allow for the up- and downregulation of the hippocampus. One approach would be to assess the hippocampal contribution to reinstatement at longer delays (where the hippocampus is less likely to be contributing to reinstatement under ‘normal’ conditions) following upregulation of the hippocampus prior to retrieval. This potential re-emergence of a hippocampal contribution would fit with recent optogenetic research showing that hippocampal engrams can be reactivated following a period of forgetting [91]. A further possibility is to use reminder cues shortly before retrieval following a long delay between encoding and retrieval, promoting systems reconsolidation mechanisms [14,66].

A second direction of travel that systems consolidation theories do not currently predict is that an older memory that is currently being retrieved with little episodic detail can increase in episodic specificity (Figure 2C). Here, it might be possible to upregulate specific cortical regions where reinstatement occurs to drive increased specificity, despite the reinstatement being largely driven by a nonhippocampal mechanism. Finally, it might be possible to increase episodic specificity experimentally with increased exposure to specific event elements across novel events, similar to the example earlier of using the same coffee cup repeatedly across experiences. Although not exhaustive, I have outlined specific experimental, analysis, and methodological approaches that might allow us to both track and change the location of an episodic memory in this state space in humans.

Concluding remarks

Memories dynamically change over time. To account for these changes, theories of systems consolidation predict that the reinstatement of events is first driven by the hippocampus and, over time, is driven by a nonhippocampal mechanism, and that memories become less episodically specific during this process. Although all three processes are typically temporally correlated, I have argued that the present evidence suggests that they are mechanistically independent and should be assumed so until evidence to the contrary is provided. I have introduced a 3D state space to allow, and further test, for this mechanistic independence, where a given episodic memory can be located anywhere in this state space at any given moment in time. Furthermore, the location of a given memory can move from any location in this state space to any other. These assumptions of mechanistic independence and non-unidirectional movement challenge current theories of systems consolidation and broaden the search space of possible changes to memories over time. I have further outlined recent experimental, analysis, and methodological advances that enable us to search this proposed state space, allowing for more mechanistic models of memory change to be developed. More broadly, setting out a wider theoretical space in which current mechanistic models of systems consolidation can be placed allows for better comparison between models and highlights areas of theoretical space that have received less attention or have not been envisaged (see [Outstanding questions](#)). Critically, the state-space framework of episodic memory captures the dynamic nature of memory and provides a theoretical grounding for how this dynamism occurs.

Outstanding questions

Are there regions of the state space where memories are not able to be located, or are there directions of travel in the state space that memories are unable to take?

Are there preferred locations or directions of travel in the state space that memories gravitate toward or move between?

Under what conditions is a hippocampal trace maintained despite the building of neocortical connections that can support reinstatement?

How does a weakening of the nonhippocampal mechanism occur, that is, how are ‘consolidated’ memories forgotten?

How is reinstatement additively combined in the neocortex when hippocampal and nonhippocampal mechanisms are driving reinstatement?

Given that neocortical reactivation does not solely occur in sensory regions seen during encoding, but also in regions such as the default mode network, how do these multisensory regions contribute to retrieval when reinstatement is driven by either the hippocampal or nonhippocampal mechanism?

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Declaration of interests

None declared by author.

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