Decoding the Inactive Engram: Can Memory Content be Read from Brain Structure?

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One question that neuroscientists have debated for a long time is whether it is possible to read out memories simply by looking at brain structure. This review focuses on the question: Is there enough information in the brain's physical neural circuits to reconstruct experiences learned by an animal earlier in life? By using *Drosophila melanogaster* as a model, the study investigates the possibility of deciphering data from the brain post-mortem. Although available evidence shows that long-term memories are encoded in the brain as physical changes that can remain even after death, this varies by specific type; meanwhile, short-term memories are generally thought to leave no physical traces after death. Additionally, our ability to read out memories is also limited by many challenges. Mapping even small brain regions can require petabytes of data and human proofreading despite advances in automated reconstruction. Without knowing the original contexts of animal life, it is almost impossible to interpret overlapping neural networks after death and link them to specific memories. The research, finally, concludes that even though several types of long-term memories are actually encoded as physical structures in the brain and remain there post-mortem, a readout of memories remains unattainable with current methods - even in smaller organisms; decoding a brain's content based on structure remains a distant goal that requires improvement in automated neural reconstruction and availability of context-aware mapping.

Keywords: Connectomics, *Drosophila melanogaster*, Structural Engram, Synaptic Plasticity, Mushroom Body, Associative Learning, Electron Microscopy, Neural Decoding.

Introduction

The nervous system is the basis for an organism's behavior, cognition, and sensory perception. The basic unit of the nervous system is a neuron; neurons can form connections with each other at synapses and form vast networks[1]. Intra-neuronal communication happens due to electrical gradients and impulses, while inter-neuronal signaling happens at synapses via the transfer of chemicals called neurotransmitters. Neurotransmitters are released from the presynaptic terminal and diffuse across the synaptic cleft to bind with postsynaptic receptors[2]. When neurotransmitters bind to postsynaptic receptors (mostly ionic channels), they activate and allow ions to flow into the postsynaptic terminal. This process initiates the electrical impulse so the signal can travel further.

The synaptic strength is neither uniform across neurons nor static over time. It depends on the number and size of synaptic connections, which can be changed after some experience. This ability to change the strength of signaling is called synaptic plasticity[3]. When the circuit fires repeatedly, the properties of synapses between neurons change. The change can be either chemical or physical: Chemical changes, for example, phosphorylation of receptors, can increase the duration of pore opening. Physical changes, in contrast, include transformation of the structure of the synapse, such as insertion of new receptors from the receptor pool to allow bigger ion influx or formation of an entirely new synaptic connection[4].

Based on duration, memory can be divided into short-term, intermediate-term, and long-term memory. Short-term memory is generally thought to last less than 30 seconds and relies on temporary electrical activity that quickly fades[1,5]. Intermediate-term memory persists for a longer time (from minutes to hours) and involves chemical changes, such as transient changes in synaptic proteins[6]. Finally, long-term memory is widely believed to involve stable structural changes, for example, changes in the size of a synapse or changes in the total number of connections[7]. These transformations can persist for years and are thought to contribute to the persistence of stable knowledge, learned skills, and long-term behavioral patterns.

Understanding the difference is important when deciding which type of memory research should focus on. Neither short-term memory nor intermediate-term memory leaves stable and lasting structural modifications[5,8]. Thus, after death, these memory traces will quickly fade away, and it will be im-

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possible to reconstruct them. In contrast, long-term memory, which includes changes in the physical configuration of neural circuits, may be detected even after death[7]. That is why long-term memory is the most plausible candidate for "reading out" memories.

Beyond temporal division, long-term memory can also be classified as declarative (explicit) and non-declarative (implicit). Explicit memory is associated with episodic memory for personal events or facts. An individual is typically consciously aware of explicit memories. However, implicit memory is procedural memory for motor skills and habits. An individual uses implicit memory automatically in daily life without conscious effort[1].

In mammalian brains, these memories are formed by extremely intricate, highly distributed neural networks. Such memory can depend on many external cues and be deeply context-dependent, making it difficult to isolate[4]. Because post-mortem brain tissue does not generate electrical signals and the context of memory is not always available, decoding specific memories from complex neural webs can be highly challenging. Consequently, this review centers exclusively on long-term associative memory (a type of implicit memory). Associative memory can be defined as the ability to link two or more different experiences or external stimuli. This type of memory tends to be simpler because it often involves directly linking sensory cues to behavioral outcomes through synaptic plasticity.

The main challenge addressed in this review is whether it is possible to "read out" memory using modern technologies such as imaging that allow scientists to observe the brain at the level of individual synapses. Investigating this problem can test the limits of current understanding of engrams and define the possibility of translating neural maps (connectomics) into a record of an organism's learned experience. If memories are stored in the brain's physical structure, translation should theoretically be possible; however, this research faces many difficulties, e.g., lack of pre-mortem context, automated reconstruction errors, etc.

This review has several conceptual, biological, and technological limitations. First, a significant conceptual boundary is neural degeneracy, a phenomenon in which distinct memories can result in identical neural networks, making it impossible to distinguish specific memories from connectomic data alone[9]. Second, the research elaborates on some aspects of memory readout techniques using *Drosophila melanogaster* as a model organism. However, these findings can be extrapolated to larger, more complex brains. Additionally, technological barriers such as errors in automated reconstruction can result in loss of subtle but important memory traces.

The objectives of the review are to identify which type of memory is the best candidate for reconstruction based on its post-mortem preservation. After that, using the model organism *Drosophila melanogaster*, especially its mushroom body olfactory circuits, the review examines which steps are necessary to "read out" specific memory. Finally, the research aims to evaluate current technologies (electron microscopy and automated tracing) and their role in this process, while also identifying bottlenecks such as data scale and segmentation error.

Memory is an highly complex phenomenon that is still not entirely understood. As was mentioned before, the study focuses exclusively on associative long-term memory. It also excludes non-structural memory storage mechanisms such as epigenetics and localized metabolic states. Furthermore, it omits post-mortem tissue degradation, such as the rapid decay of protein phosphorylation states and the breakdown of cellular membrane structure. Conceptually, the review does not address the fact that identifying which Kenyon Cells respond to a specific odorant is impossible unless genetic tagging happened pre-mortem. Finally, all assumptions made based on laboratory studies may not apply to all species and lack ecological validity.

The theoretical framework of the review relies on the Structural Engram Theory, a model of Feedforward Circuit Tracing, and a Synaptic Weight Matrix model. Structural Engram Theory posits that while short-term and intermediate-term memories depend on transient chemical changes, long-term memories are thought to be stable and expressed through physical alterations in structure[7,10]. Feedforward Circuit Tracing simplifies information flow into a direct path from Kenyon Cells (KCs) to Mushroom Body Output Neurons (MBONs)[11]. Ultimately, the Synaptic Weight Matrix model considers that synapses have measurable strength, allowing for numerical comparison of connection weights[12,13].

This study utilizes a qualitative narrative review approach to connect findings across connectomics, molecular biology, and behavioral neuroscience. By examining existing literature on electron microscopy, automated neural tracing, and circuit mapping, it compares how memory is physically stored in *Drosophila melanogaster* with the massive data and technical challenges of scaling up to mammalian brains. This comparison forms the basis for evaluating whether we can truly read out stored memories from static brain maps.

Methodology

The methodology uses a qualitative review approach. Literature was gathered from databases like PubMed and Google Scholar. Search terms covered synaptic plasticity, the *Drosophila* mushroom body, volume electron microscopy, and segmentation error rates. Additionally, citation tracking provided several extra sources.

Data were gathered from peer-reviewed journal articles, review papers, and reliable neuroscience texts. Studies were included if they focused on structural mechanisms of mem-

ory storage or specifically on long-term memory, rather than non-structural mechanisms such as epigenetics or short-term/intermediate-term memory. Another exclusion criterion was a lack of methodological detail.

Information was extracted based on its relevance to the study and to the research question. The study used a scale-based approach, so findings were organized starting from mapping memory from large-scale brain structures down to synapses. Afterward, the study employed a comparative and assessment analysis to compare the more complex mammalian brain to model organisms and evaluate current technology, respectively.

The source quality was judged from the publication venue, the citation value of the sources, the methodology, the relevance of the literature to the research question, and the consistency of results in several independent studies. The preference was for highly cited foundational papers, recent peer-reviewed publications, and neuroscience journal publications.

Memory Mapping: how different brain areas store our thoughts?

The first step in reconstructing memory from brain structure is to identify which brain lobe memory could potentially be located in. To do so, it is important to understand how different types of memory are distributed across brain regions (lobes). Recognizing even approximate memory location can significantly ease further processing.

Memory is not stored in one place - it is distributed across different brain lobes. Brain lobes generally have certain types of memory they are responsible for[4]. For example, in humans, the cerebellum is important for motor learning, the hippocampus handles spatial memory, the amygdala processes emotional associations, the basal ganglia is in charge of reward-based associative learning, and the cortex is responsible for higher-level cognition, including problem-solving and planning[1]. To learn this information about memory distribution across the brain in humans, researchers utilized lesion studies and modern genetic tools.

Lesion studies are a method that involves deliberately damaging certain parts of the brain in model systems to observe how an animal's behavior changes after impairment and draw conclusions from it. For example, studies involving hippocampal damage in rats showed that the hippocampus plays a significant role in spatial memory, as demonstrated by rats' decreased ability to navigate and remember locations in Morris Water Maze task[14]. In humans, however, where lesion studies are unethical, scientists can learn about the different brain lobes' roles from accidental trauma, stroke, surgical resections, or neurodegeneration of some brain areas. A famous example is patient H.M., whose hippocampus was removed to treat severe epilepsy. After the procedure, patient H.M suffered severe anterograde amnesia and lost the ability to form new episodic and declarative memories[1]. In model systems, observations can also be made in mutants that lack a particular brain area. For example, certain Drosophila mutants lack mushroom bodies, which are important for olfactory learning; studies focusing on these mutants illustrated that absence of this structure leads to severe impairments in behavioral responses[15]. Thus, they established the role of Drosophila's mushroom bodies.

Modern techniques like optogenetics and chemogenetics allow researchers to control brain activity more precisely. Optogenetics uses light to activate or inhibit neurons with modified genes[16], while chemogenetics uses designed receptors (proteins that detect and respond to specific chemicals)[17]. These methods can reversibly silence or stimulate particular brain areas, showing their roles in learning and memory. Gene editing can also disrupt specific genes involved in memory processes, helping understand their role in memory[18].

Once the brain regions responsible for different types of memory are identified, the next step is decoding the memory's content. This requires focusing on the specific neural circuits that encode and express the memory.

How memory might be interpreted?

Mapping brain circuits from sensory to motor systems including the portions of the circuits that write and store memories

To map how memories are stored in neural pathways, it is first important to understand the neurons involved at each stage. This includes sensing the stimulus, storing the memory, and producing the behavioral response.

This process begins by locating sensory neurons —such as sensory neurons in the vertebrate nose or olfactory receptor neurons (ORNs) in the insect antennae for odors[19]. Researchers use techniques like calcium imaging or electrophysiological recordings (microelectrodes placed in sensory organs to measure neural responses to specific stimuli)[20,21]. This is essential to establish which neurons represent the initial sensory input and to link specific stimuli with neural activity.

Next, scientists trace the neural pathway downstream to identify the circuit where memory formation and storage could take place[11]. Following pathway tracing, functional investigations using electrophysiological recording or imaging are conducted during learning to observe changes in neural activity patterns[20,21]. These experiments can show which neurons are the first in the feedforward chain that change their responses to stimuli after a memory is formed. The synapses of neurons whose responses are altered after learning are the candidates for structural plasticity. Tools like optogenetics,

combined with behavioral tests, allow researchers to test the necessity or sufficiency of these neurons for memory formation and expression[16].

Finally, researchers identify output neurons. To do so, they use an analogous approach: first, trace downstream to find candidate neurons; second, conduct behavioral experiments combined with electrophysiological recording to confirm that the neuron is participating in learning; and ultimately, use tools like optogenetics during behavioral tests to selectively silence candidate neurons and observe the effect silencing has on behavior.

Even if precise mapping of each neuron in a neural circuit is difficult, by using the method described above, researchers can discover the structure of the neural circuit and its function[11].

Mapping synaptic changes that occur when specific memories are written

Next, researchers examine how synaptic strengths have changed. A primary component of memory consolidation is hypothesized to involve long-term adjustments in the patterns of strengthened or weakened synapses[1]. Identifying these changes allows scientists to infer where and how a memory pathway is organized.

Electrophysiological recordings or functional imaging of neural activity before, during, and after learning reveal which neurons change their responses after learning. To determine which exact connections onto these neurons have changed, it is necessary to activate the presynaptic neurons and record the postsynaptic neurons (that have changed after learning) – and show that a particular connection is different after learning[11,22].

This still does not show that the change that occurred is structural. To show that a synapse has grown in size (or shrunk) or that the number of synaptic connections between two neurons has changed, researchers must examine the structure of synapses. This can be done by labelling synapses with particular markers (e.g. fluorescently tagged synaptic proteins) and imaging with light-microscopy. Alternatively, researchers can use electron microscopy to obtain detailed information about the structure and size of synapses.

However, structural changes alone do not reveal what the memory means. Behavioral analysis is crucial for interpretation[10]. By linking specific synaptic changes to observed shifts in behavior—such as avoidance or preference—scientists can confirm that the neural changes reflect meaningful memory content. For instance, if synapses related to approach behavior weaken after a negative experience, the organism should avoid the associated stimulus. Finally, by looking at how synaptic shapes change for different sensory inputs, we can trace how these physical patterns link to specific memories[23]. In short, analyzing memory content from structure involves matching changes in neural connectivity to past experience, behavior, and reinforcement history.

To illustrate how this is possible in practice, we now turn to the fruit fly (*Drosophila melanogaster*) as a model organism. Its small brain, completely mapped connectome, and powerful genetic tools make it uniquely suited for studying how specific experiences are encoded in structural changes to neural circuits—making it an ideal model to study how well physical memory traces are preserved after death[24,25].

Example circuit where memory traces are well understood: memory encoding and behavioral translation in fruit flies

In fruit flies, associative learning and memory mainly depend on a structure in the brain called the mushroom body (MB), which consists of Kenyon cells (KCs) that encode stimuli and Mushroom Body Output Neurons (MBONs) that encode the learnt behavior[12,25].

Dopaminergic neurons (DANs) give reinforcement signals that change the strength of connections between KCs and MBONs[26]. When a fruit fly smells something, the sensory input activates KCs[25,27]. This begins with odorant molecules being detected by olfactory receptor neurons located in the antennae. Each type of odor activates a different set of these sensory neurons, which project to distinct regions (called glomeruli) in the antennal lobe. From there, the signals are passed to higher-order neurons that reach the mushroom body and activate specific combinations of Kenyon cells. Each odor activates a small subset of KCs (sparse neural code)[28]. These KCs then connect with MBONs. Dopamine from DANs changes these KC-to-MBON synapses. Each KC helps to activate certain MBONs, based on the smell. How strong the synapses are between KCs and MBONs depends on what the fly has experienced before. In naïve animals, Kenyon cells connect broadly to MBONs that promote both approach and avoidance, so any given odor activates both pathways equally — resulting in no net behavioral preference. While animals often possess innate, baseline olfactory preferences, associative conditioning paradigms dynamically shift this balance by selectively altering specific pathways.

Specific groups of DANs, signaling either reward or punishment, can change these synapses, which then affects how the fly acts[12,26]. Depending on whether the fly is rewarded or punished, certain DANs are activated to modulate the synapses between KCs and MBONs, so the fly remembers the experience when it smells the same odor again[25].

To illustrate how this process might work in practice, consider a fruit fly that experienced a series of strong events throughout its life. Each time it approached apples, it was attacked by a parasitoid wasp and had to escape. In contrast, when it encountered pears, there was no danger, and it

safely consumed food. To confirm how these experiences influenced behavior, behavioral assays—such as olfactory preference tests—would be necessary [29]. These experiments can reveal whether the fly learned to avoid or approach specific odors based on prior outcomes.

To determine how fruit flies learn to react to apples, we should examine how dopaminergic neurons (DANs) affect synaptic plasticity. When a fly gets attacked by a parasitoid wasp while it's near apples, this aversive experience activates DANs from the PPL1 group that drive punishment learning. Whenever the apple odor activates specific KCs at the same time as the negative experience activates the PPL1-DAN, the synaptic links between Kenyon cells (KCs)—which get turned on by the apple smell—and mushroom body output neurons (MBONs) that usually make the fly want to go toward the apple (approach behavior) are weakened [30]. Because of this, the output changes: the synaptic signal from KCs to MBONs that cause avoidance gets stronger than the signal to MBONs that cause approach. This shift in synaptic weight serves as a primary neural mechanism for expressing the learned avoidance of the stimulus. Because of that, the next time the fly smells apples, it will likely try to avoid them. As a result, the fly learns to suppress its approach to the odor, because the corresponding neural pathway has been weakened by aversive reinforcement [13]. To verify this behaviorally, experiments would need to compare the fly's response to apple odor before and after conditioning to see whether avoidance is reliably expressed.

By contrast, in the case of pear odor, when the fruit fly smells a pear and nothing happens except that it receives tasty and nutritious food, this fruit becomes associated with a positive (appetitive) experience. Similar to the reaction to apples, different Kenyon cells (KCs) are activated by the pear odor and then innervate mushroom body output neurons (MBONs). Because different odors activate different sets of olfactory receptor neurons and glomeruli, the pear smell results in a distinct pattern of KC activation compared to apple odor. This means that the memory for each odor is stored in a stimulus-

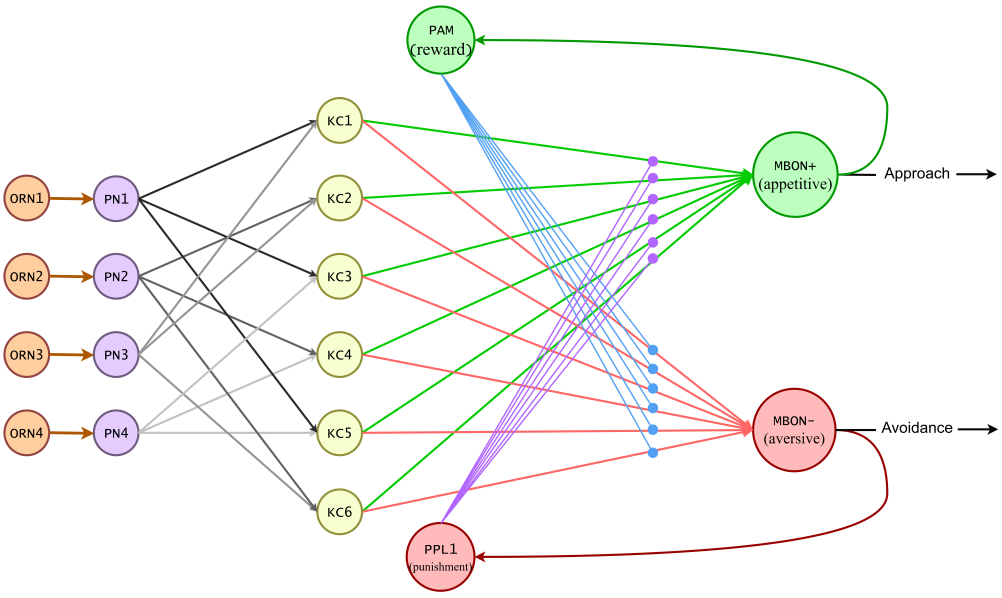

Figure. 1 Conceptual schematic of the Drosophila olfactory and associative learning network. The circuit demonstrates the feedforward pathway from olfactory receptor neurons (ORN1–4) to projection neurons (PN1–4), which randomly diverge into the Kenyon cell layer (KC1–6) of the mushroom body. Sensory signals undergo dopaminergic gating at the KC–MBON synapses, mediated by reward-associated PAM neurons (blue) and punishment-associated PPL1 neurons (purple). Modulated outputs diverge into appetitive ($MBON^{+}$) and aversive ($MBON^{-}$) pathways to drive approach or avoidance behaviors, respectively. Curved lines indicate feedback loops providing behavioral reinforcement back to the dopaminergic systems. Adapted from mushroom body learning circuit model [11,12].

specific way[12,28]. However, based on previous experience linking the smell of pears with a positive outcome, the fly is more likely to approach the pear. The process responsible for this is analogous to the one described for apples, but in this case, appetitive DANs (such as those from the PAM cluster) are activated and weaken synapses that promote avoidance behavior[26]. Therefore, the KCs signal to the MBONs leading the fly to move towards a stimulus, and the signal's strength intensifies, which makes the fly like pears more. As with aversive conditioning, appetitive behavior should be tested using preference or feeding assays to confirm that the learned attraction has taken place.

These synaptic strength changes are hypothesized to leave structural traces that can potentially be seen in the fly's brain under specific preservation conditions. If the brain is examined using electron microscopy , they can analyze how the Kenyon cells (KCs) and MBONs are connected. Assuming the tissue is mapped prior to post-mortem degradation, researchers may observe that the KCs that react to the apple smell link up more strongly with the MBONs that make the fly avoid things, and not as strongly with the MBONs that make the fly go toward things. On the other hand, the KCs that react to the pear smell will keep stronger links with the MBONs that make the fly want to approach. Since each odor activates a distinct set of Kenyon cells—determined by the odor-specific input from receptor neurons—observing which KC-MBON synapses were strengthened or weakened can reveal not just the presence of a memory, but the specific stimulus it relates to. By checking the relative physical sizes or numbers of these connections between KCs and MBONs for each smell, researchers could potentially infer which odors the fly learned to associate with reward or punishment[27,31]. In principle, such a pattern could be interpreted as evidence that the fly learned to avoid apples and approach pears.

These synaptic patterns reflect the fly's unique experiences by leaving physical correlates associated with memory at the cellular level. Studying these neural changes helps reveal fundamental principles of how memories are stored and modified over time. Thus, under ideal experimental models, a structural trace of learned preferences and aversions can be inferred from the pattern of synaptic strengths between odor-specific Kenyon cell combinations and MBONs, though this process remains heavily constrained by the challenges of unlabelled post-mortem tissue and circuit degeneracy.

Challenges in mapping and reading memories from brain structure: technological and conceptual barriers

Even though there has been remarkable progress in mapping neural circuits, there are still major difficulties in isolating, mapping, and interpreting these complex networks. The most significant obstacle is technological. For example, it took years of work to map the entire brain of *Drosophila melanogaster*, which required specialized imaging techniques such as Electron Microscopy (EM). Although EM offers the high resolution necessary to examine individual brain cells and synapses in detail, tissue preparation is complicated and delicate. The brain has to be fixed, stained with heavy metals, embedded in resin, and then sliced into very thin sections—usually between 50 and 100 nanometers thick[32]. Since the tissue is so brittle, even a small error when slicing can damage the sample and result in the loss of important data before transmission electron microscopy can send electrons through the sections to image them.

In order to eliminate the need for sectioning, scientists have begun to use scanning electron microscopy—where the electrons reflect off an extremely flat surface of tissue and therefore do not need to pass through a thin section—such as Serial Block-Face Electron Microscopy (SBEM) and Focused Ion Beam Scanning Electron Microscopy (FIB-SEM). SBEM aids in the production of 3D models by taking images of successive layers of tissue. With FIB-SEM, an ion beam is used to remove material step by step before imaging takes place. Although these techniques are faster and more reliable than conventional EM, great care must be taken when handling the tissue to avoid structural distortions which could affect the integrity of the data[33].

Even with better modern technologies, it is difficult to manage the complexity of the brain (millions of axons, dendrites and synapses). Thus, modern connectomics relies on advanced artificial intelligence and machine learning algorithms to automatically trace neural networks and locate synapses from imagery[34]. However, because tissue is so densely packed, automated tracing models still make many errors. Hence, a substantial amount of human proofreading is necessary to correct misalignments and false connections[35]. This hybrid workflow is a major bottleneck; for context, it has been estimated that reconstructing the complete connectome of a single mouse brain would require the equivalent of 10 experts working full-time for over half a century.

Additionally, researchers struggle to scan the whole brain. Each scan generates an enormous amount of data, often terabytes for each section. This data then should be processed by the hybrid workflow described above to make high-quality 3D models from it. Right now, computers can only work with some of this data, which causes errors in alignment and potentially wrong reconstructions[36]. To illustrate the purely computational scale of this challenge, an automated EM reconstruction of just one cubic millimeter of the human temporal cortex generated roughly 1.4 petabytes of data, containing approximately 57,000 cells and 150 million synapses[37]. These numbers represent how it is extremely hard to create a static

anatomical map of the brain at the synaptic level. Trying to decode each of these neural circuits is far more challenging - it requires tracing of subtle shifts in synapse sizes which require immense effort.

Beyond technological limitations, there are a number of conceptual barriers. First, reliance of memory to molecular states such as protein phosphorylation cascades, metabolic shifts, and active neurotransmitter pooling at synaptic active zones. Upon death, cellular membranes start to degrade. Because a post-mortem state cannot preserve these molecular components, the structure of the brain alone offers incomplete data of a once-living memory trace.

Second, while electron microscopy provides high-quality anatomical resolution, a raw post-mortem EM volume is completely anonymous[11]. In model organisms like *Drosophila*, specific Kenyon cell functions are mapped using live calcium imaging or targeted genetic lines pre-mortem. Without this functional tagging prior to death, a static map cannot identify which anonymous neuron corresponded to a specific sensory experience, making the whole neural pathway uninterpretable.

Third, even if cell identities are successfully mapped, distributed neural networks exhibit significant circuit degeneracy. In complex circuits, multiple distinct experiences can produce completely overlapping structural outcomes. Because different cognitive inputs can be mapped as identical physical network layouts, it may be impossible without additional information to perform a clean "readout" of a specific past memory from structure alone.

Fourth, much of our structural understanding of memory comes from rigid, simplified laboratory paradigms, like classical conditioning tests. Such highly controlled experiments often lack ecological validity and do not replicate the way an organism interacts with a rich unconstrained natural environment[38]. The effort to interpret human episodic or semantic memory in terms of the strict structural rules of model organism conditioning is a fundamental category error.

Finally, memories do not exist as separate, unchanging files stored in specific places. They are very much influenced by context and depend on the changing, overall state of the brain. Moreover, memories are strongly driven by the dynamics of recurrent neural networks, which involve ongoing loops of electrical activity moving through the circuit in real time. Because a post-mortem connectome is completely unchanging, it completely ignores these live, looping electrical signals needed to express or recognize a cognitive state.

Conclusion

This review looked at whether memory content can be reconstructed from brain structure after death. It focused on the structural ways long-term associative memory works, especially in the olfactory learning circuits of *Drosophila melanogaster*. The evidence points to long-term memories leaving lasting structural changes through altered synaptic connections, making them good candidates for study after death. However, these physical changes alone cannot reliably reconstruct an organism's past experiences.

The findings show that while connectomics has improved our understanding of how memories exist in neural circuits, a fixed anatomical map cannot capture the molecular, physiological, and contextual details needed for a complete memory readout. Therefore, this review achieved its goals by identifying the memory type best suited for structural reconstruction, examining how associative memories are encoded in the mushroom body, and looking at the technological and conceptual challenges that prevent decoding stored experiences from brain structure alone.

This review is limited because it focuses on long-term associative memory, uses *Drosophila melanogaster* as a model organism, and excludes non-structural memory storage methods. Future research should work on better automated connectome reconstruction while combining structural data with functional and molecular methods to gain a fuller understanding of memory.

Ultimately, this review suggests that the biggest challenge in decoding memory is not just getting a complete brain map. It is also about understanding how structure, function, and experience come together to form memory. While structural traces of learning may last after death, decoding the inactive memory trace remains a long-term goal that will need progress in several areas of neuroscience.

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