NEUROSCIENCE

# Hippocampal encoding of memories in human infants

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Humans lack memories for specific events from the first few years of life. We investigated the mechanistic basis of this infantile amnesia by scanning the brains of awake infants with functional magnetic resonance imaging while they performed a subsequent memory task. Greater activity in the hippocampus during the viewing of previously unseen photographs was related to later memory-based looking behavior beginning around 1 year of age, suggesting that the capacity to encode individual memories comes online during infancy. The availability of encoding mechanisms for episodic memory during a period of human life that is later lost from our autobiographical record implies that postencoding mechanisms, whereby memories from infancy become inaccessible for retrieval, may be more responsible for infantile amnesia.

Infancy is a period of dramatic learning, yet memories for individual experiences from this period are absent later in childhood and adulthood. A prevailing theory of this paradoxical phenomenon, known as infantile amnesia (*1*–*3*), is that the hippocampus may not be able to support the encoding of episodic memories during infancy because of its protracted maturation into adolescence (*4*–*7*). However, this encoding-based account conflicts with evidence from rodents that the hippocampus forms memory engrams during infancy (*8*–*12*). For example, although mice who learn the location of an escape hole in a maze during infancy forget this location by maturity, optogenetic reactivation of the hippocampal neurons that were activated during encoding can elicit the learned behavior (*12*). These findings suggest that infantile amnesia in rodents occurs because of postencoding mechanisms related to retrieval.

It is unknown whether infantile amnesia in humans results from developmental changes in encoding and/or postencoding mechanisms. There is behavioral evidence for memory in human infants, including conditioned responses when placed back into a familiar context (*13*), deferred imitation after observing another person (*14*), and delayed preferential looking after repeated habituation to a stimulus (*15*). Whether these behaviors depend on the hippocampus or other brain structures has long been debated without direct neural evidence (*5*, *16*, *17*).

A recent functional magnetic resonance imaging (fMRI) study of statistical learning suggested that the human hippocampus is functional as early as 3 months of age (*18*). The hippocampal pathway associated with episodic memory formation develops slowly (*19*), suggesting that such encoding may emerge later during infancy and may be supported by the posterior hippocampus, where the subfields in this pathway are overrepresented (*20*). Indeed, there is reason to believe that hippocampal encoding of episodic memories may onset as early as 9 to 12 months of age, in line with behavioral changes in relational memory (*4*, *21*, *22*), or later around 18 to 24 months of age, in line with improvements in memory for arbitrary order, spatial locations, and individual events (*23*–*25*).

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## Subsequent memory paradigm based on looking time

We used fMRI in awake infants to investigate whether the hippocampus can encode individual memories. We relied on the subsequent memory paradigm, a well-established task for assessing long-term episodic memory in adult cognitive neuroscience (*26*, *27*). In this task, participants view a series of images or words (each presented once) in the scanner and then complete a recognition memory test. The encoding trials are then sorted post hoc based on whether the items were remembered or forgotten at test. A subsequent memory effect is defined as greater neural activity during the initial encoding of items that are later remembered. Meta-analyses of fMRI studies in adults have shown robust subsequent memory effects in the bilateral hippocampus (*28*).

We adapted the subsequent memory paradigm for infants by interleaving encoding and test trials and using preferential looking during a visual paired comparison as the behavioral assay of memory (Fig. 1). Preferential looking predicts explicit recall in adults (*29*), relates to memory over development (*30*), and requires the hippocampus in nonhuman primates (*31*, *32*). The lag between encoding and test (mean = 59.9 s, SD = 15.8 s) and the interruption by intervening trials (3 to 12 items) place the memory demands of this task outside of the range of working memory. We predicted that hippocampal activity at encoding would track looking to the old item during the visual paired comparison test. Such familiarity preferences are more likely when a stimulus is not fully habituated, as was likely in our study because items were presented only once during encoding and very briefly (2 s) by infant standards (*33*–*35*). Thus, our design may reflect early stages of encoding that might be partial or incomplete.

We collected usable awake fMRI data and anatomical scans from 26 infants aged 4.2 to 24.9 months of age (table S1), with half of the infants younger ($N$ = 13) and half older ($N$ = 13) than 1 year of age. We sorted each encoding trial based on whether the infant subsequently looked longer at that old item during test (that is, whether a familiarity preference was elicited). We excluded items for which the infant was moving their head excessively or not looking at the display during either encoding or test. We did not find an overall familiarity preference (proportion of test trials with more looking to old item) in the full sample [mean = 0.510, 95% confidence interval (CI) (0.473 to 0.545), versus 0.5 bootstrap $P$ = 0.540]. This was true within younger [mean = 0.501, 95% CI (0.451 to 0.554), $P$ = 0.962] and older [mean = 0.515, 95% CI (0.457 to 0.566), $P$ = 0.558] infants; there was also neither a difference between age groups [mean = −0.014, 95% CI (−0.087 to 0.063), $P$ = 0.724] nor a continuous effect of age on familiarity preference (Spearman's rank correlation coefficient ρ = 0.220, $P$ = 0.306). Results were similar when the familiarity preference was quantified continuously as the average proportion of time looking at the old item per test trial rather than the proportion of test trials with a binary preference (all $P$ values > 0.598). Bootstrap resampling of the older infants (1000 iterations) suggested that we would not have obtained an overall familiarity preference even with a full sample of older infants [95% CI (0.476 to 0.548), $P$ = 0.398].

## Hippocampal activity during memory encoding

What matters most in the subsequent memory paradigm is how trial-wise variance in memory behavior relates to brain activity during encoding within-participant (*36*); namely, what is different in the brain during the encoding of items that are later remembered (in our case, looked at longer)? We fit blood oxygenation level–dependent (BOLD) activity measured with fMRI using a general linear model (GLM) with regressors for encoding trials that yielded a later familiarity preference at test and for encoding trials that did not as well as with a third task regressor for all of the test trials and additional nuisance regressors. To measure the neural subsequent memory effect, we extracted the normalized difference ($z$-score) of parameter estimates between the two types of encoding trials from an automatically segmented region of interest (ROI) for the bilateral hippocampus and across the whole brain. We manually subdivided the hippocampal ROI into anterior and posterior regions (Fig. 2A).

There was significantly greater BOLD activity in the whole hippocampus during the encoding of items for which infants later showed a familiarity preference versus items for which

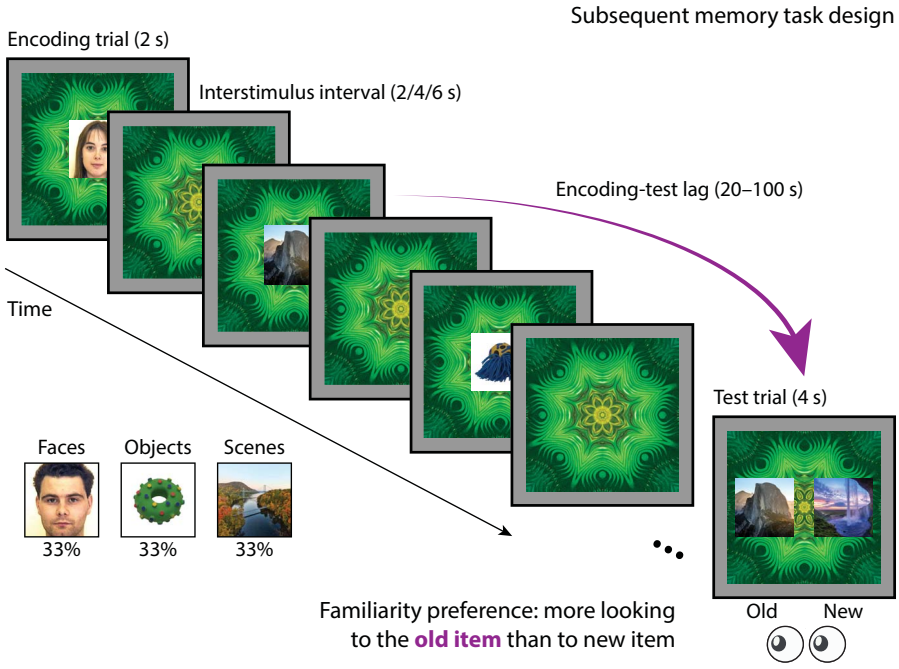


**Fig. 1. Design of subsequent memory task for infant behavior.** Schematic of a trial sequence shown to infants during fMRI depicting encoding trials with a previously unseen face, object, or scene photograph; a test trial with an old item from an earlier encoding trial opposite a new item from the same category; and a green background pattern that moved dynamically during the jittered interstimulus interval to maintain infant attention. The cartoon eyeballs below the test trial indicate that gaze behavior to the old versus new item was used to assess memory.

they did not [mean *z*-score = 0.295, 95% CI (0.087 to 0.515), bootstrap $P = 0.008$; Fig. 2B]. These results suggest that rapid, one-shot encoding of individual visual experiences can be supported by the hippocampus in human infants. This subsequent memory effect was evident in the posterior hippocampus [mean = 0.369, 95% CI (0.139 to 0.629), $P = 0.002$; Fig. 2C] but not the anterior hippocampus [mean = 0.191, 95% CI (−0.064 to 0.431), $P = 0.140$]; however, the comparison between posterior and anterior did not reach significance [mean = 0.178, 95% CI (−0.039 to 0.411), $P = 0.118$]. The role of the posterior hippocampus in episodic encoding accords with adult findings (*37*), theoretical models (*38*), and rodent studies [their dorsal hippocampus (*9, 10, 12*)].

We replicated this pattern of results with a continuous rather than binary measure of memory (fig. S1). In control analyses, we showed that these results cannot be explained parsimoniously by common confounds in subsequent memory studies (*39*), such as item effects or memorability (fig. S2) or serial position (fig. S3). Moreover, these effects were specific to familiarity preferences for the old item at test, as hippocampal activity at encoding was unrelated to the general strength of the test preference when preferences for the old and new items were combined (fig. S4).

Although we did not observe a familiarity preference at the group level, several individual infants showed a familiarity preference on average across trials. Insofar as these infants had better overall behavioral memory, we would expect the overall hippocampal subsequent memory effect to be strongest in these infants. To test this prediction, we split participants on the basis of whether their average familiarity preference across trials was higher or lower than the median ($N = 13$ in each group). The hippocampal subsequent memory effect was robust in infants with a higher average familiarity preference [mean = 0.417, 95% CI (0.117 to 0.705)], $P < 0.001$; Fig. 2D and fig. S5] but unreliable in infants with a lower average familiarity preference [mean = 0.173, 95% CI (−0.124 to 0.491), $P = 0.276$]. The effect in infants with a higher average familiarity preference was driven by the posterior hippocampus [mean = 0.542, 95% CI (0.243 to 0.832), $P < 0.001$; Fig. 2E] and not the anterior hippocampus [mean = 0.244, 95% CI (−0.162 to 0.630), $P = 0.242$]. No effects were found in infants with a lower average familiarity preference in the posterior [mean = 0.195, 95% CI (−0.160 to 0.566), $P = 0.294$] or anterior [mean = 0.138, 95% CI (−0.149 to 0.421), $P = 0.318$] hippocampus.

### Emergence of hippocampal encoding across infancy

Given the protracted maturation of hippocampal anatomy (*19, 40*) and behavioral changes in memory around 9 to 12 months (*4, 21, 22*), we used median values to split our sample into younger (4 to 9 months; $N = 13$) and older (12 to 24 months; $N = 13$) infant age groups (Fig. 3A). The subsequent memory effect in the whole hippocampus was driven by the older infants [mean = 0.577, 95% CI (0.268 to 0.898), $P < 0.001$; Fig. 3B], with no effect in younger infants [mean = 0.014, 95% CI (−0.198 to 0.242), $P = 0.898$]; the group difference was also significant [mean = 0.563, 95% CI (0.196 to 0.944), $P = 0.002$]. This age effect did not depend on using a median split, as it was also evident when age in months was considered as a continuous variable (Spearman's $\rho = 0.385$, $P = 0.034$; Fig. 3C). These age effects were present in both the anterior and posterior hippocampus, though numerically stronger in the posterior hippocampus (fig. S6).

We ran control analyses to determine whether the age effects reflect differences in data quality between younger and older infants. Firstly, we replicated the relationship between age and subsequent memory in the hippocampus after partialing out six potential age-related confounds, including measures of the number of usable trials, attention to the screen, looking preferences, and hippocampal volume (table S2). Secondly, we confirmed that the variance in hippocampal activity (as a measure of noise) was comparable between older and younger infants (fig. S7). Thirdly, we demonstrated robust and comparable visual evoked BOLD responses in category-selective areas for faces, objects, and scenes (fig. S8); this further indicates that similar attention was paid to the stimuli, in addition to the fact that trials were only included in the main analysis if fixated.

We included items from three categories to increase visual interest for the infants and to allow for the possibility that the infant hippocampus may prioritize certain content types for encoding. Although all three categories independently showed the same pattern of results, objects were the most reliable, followed by scenes (fig. S9A). The results from this analysis should be interpreted with caution because of the loss of statistical power from subdividing in three an already modest number of usable pairs of encoding and test trials.

The nascent memory abilities of older infants may still be a rudimentary form of adult episodic memory. For example, the subsequent memory effect in the whole hippocampus for older infants was clearer for shorter than longer study-test lags (fig. S9B). The fleeting nature of these memories may be expected given the brief, one-shot exposure during encoding. Future work could introduce repetitions to examine memory savings and increase durability (*41*).

### Subsequent memory effects beyond the hippocampus

We investigated the anatomical specificity of hippocampal subsequent memory effects by running an exploratory voxel-wise GLM analysis. Across all infants, the contrast of encoding

trials in which infants did versus did not show a later familiarity preference at test yielded bilateral BOLD responses in the posterior hippocampus ($P < 0.01$, uncorrected; Fig. 4). When separated by age, younger infants showed no whole-brain subsequent memory effect, whereas older infants showed effects in the bilateral hippocampus ($P < 0.01$, uncorrected) and in the right orbitofrontal cortex ($P < 0.05$, corrected). The hippocampal involvement in this whole-brain analysis mirrors the results of the main analysis with individualized ROIs. The orbitofrontal result in older infants is in line with prior findings of memory-related activity in the orbitofrontal cortex and its connectivity with the hippocampus in children 4 to 8 years old (*42*).

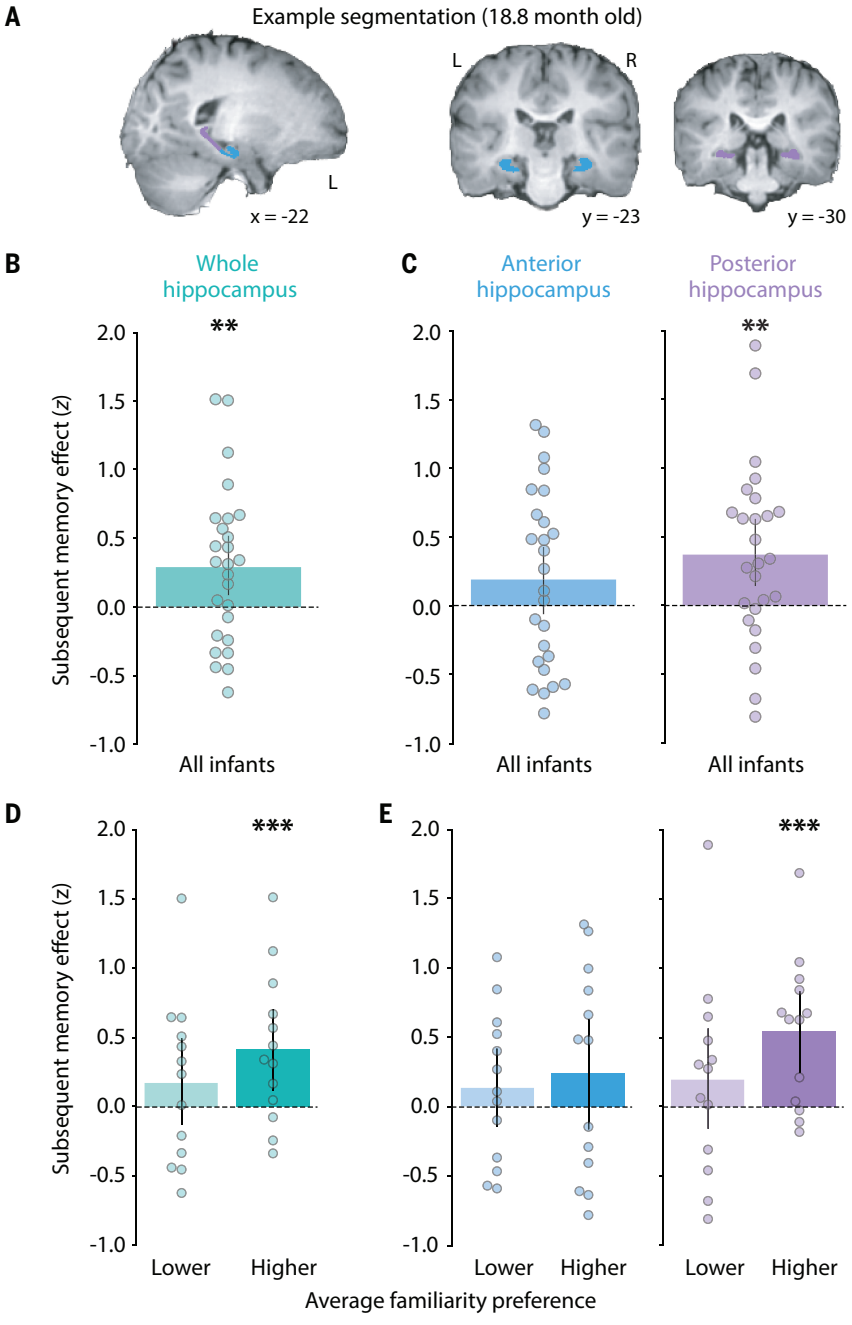


**Fig. 2. Subsequent memory effect in the infant hippocampus.** (**A**) Sagittal and coronal (L, left hemisphere; R, right hemisphere) sections of the hippocampal ROI segmented from the structural MRI scan of an 18.8-month-old infant participant [visualized with *x* and *y* coordinates in Montreal Neurological Institute (MNI) standard space]. The anterior hippocampus is shown in blue, and the posterior hippocampus is shown in purple. (**B** and **C**) Normalized difference of BOLD activity during the encoding of items for which infants later did versus did not show a familiarity preference at test in the whole hippocampus (B) and in the anterior and posterior hippocampus (C). (**D** and **E**) This subsequent memory effect split into subgroups of infants whose average familiarity preferences across test trials were lower and higher than the group median in the whole hippocampus (D) and in the anterior and posterior hippocampus (E). Dots indicate individual participants, and error bars represent 95% CIs from bootstrap resampling. ***$P < 0.001$; **$P < 0.01$.

Familiarity preferences in our visual paired comparison test required recognizing the old item and discriminating it from the new item. In adults, such item recognition is related to encoding in both the hippocampus (*28*) and the surrounding medial temporal lobe (MTL) cortex (*43*). Yet, we did not observe clear involvement of the MTL cortex in the whole-brain analysis or in ROIs that encompassed entorhinal, perirhinal, and parahippocampal cortices (fig. S10). The difficulty of the encoding task for infants may help explain why only the hippocampus was recruited: The memoranda were new to infants at encoding, they were shown only once and briefly, they were tested only after multiple intervening items, and this test required fine discrimination against new items from the same category. For these reasons, the expression of memory in looking behavior at test may have required the specialized capability of the hippocampus (especially posterior) for rapid encoding of distinctive memory traces through pattern separation (*37*). Indeed, the hippocampus is more involved in item recognition in children than teenagers or adults (*44*).

### The ontogeny of episodic memory in humans

We found that the human hippocampus is more active during the encoding of items for which infants later show a familiarity looking preference at test. Although observed in the full sample, this subsequent memory effect was clearer in infants who showed an overall familiarity preference and in those who were older than 12 months. The effect was also strongest in the posterior hippocampus, with additional involvement of orbitofrontal cortex in older infants. These findings provide neural evidence that at least some form of rapid, one-shot hippocampal encoding of individual experiences emerges by around the first year of life.

Task difficulty may help explain the lack of a subsequent memory effect in younger infants (*33*). Younger and older infants did not differ in their overall behavioral looking preferences, which is inconsistent with an explanation of the observed age effects in the hippocampus based on differential performance (*45*). However, stimulus duration was fixed in the current study, which may complicate the interpretation of age effects given developmental changes in the time needed for encoding (*46*). Future work could consider alternative paradigms, such as infant-controlled habituation (*47*), to ensure sufficient encoding time. This could help refine the developmental trajectory of episodic memory by providing greater confidence

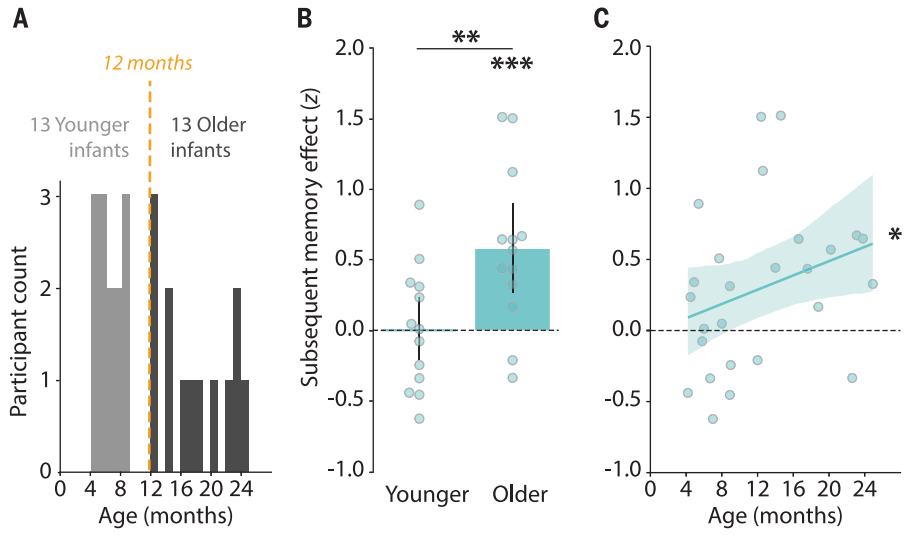


**Fig. 3. Development of hippocampal subsequent memory effect.** (**A**) Histogram depicting age distribution of participants split into subgroups of infants who were younger (gray) and older (black) than 12 months of age. (**B**) Subsequent memory effect in the whole hippocampus for younger and older infants. (**C**) Linear relationship between age in months and the subsequent memory effect in the whole hippocampus. Dots indicate individual participants, and error bars and bands represent 95% CIs from bootstrap resampling. ***$P < 0.001$; **$P < 0.01$; *$P < 0.05$.

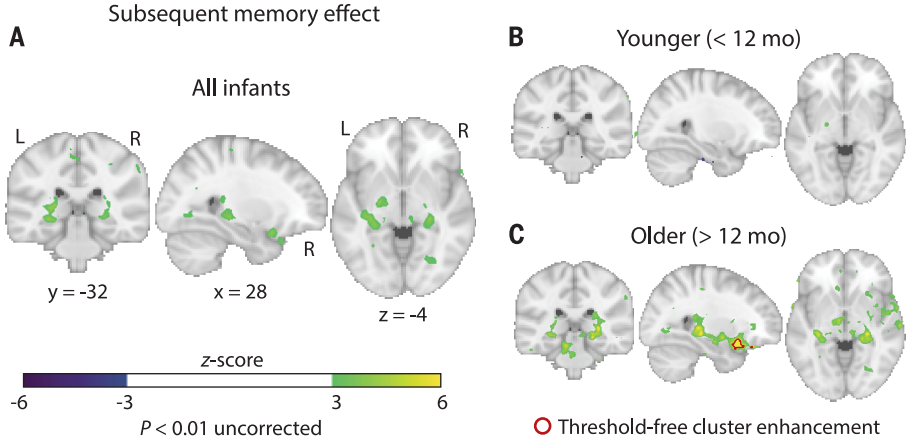


**Fig. 4. Subsequent memory effect in the whole brain.** (**A** to **C**) Exploratory GLM contrast of encoding trials that did versus did not yield a subsequent familiarity preference in (A) all infants and then separately in (B) younger and (C) older infants. Voxels significant at $P < 0.01$ (uncorrected) are colored by the average $z$ statistic across participants. Clusters of significant voxels after threshold-free cluster enhancement at $P < 0.05$ (corrected) are outlined in red.

in the presence or absence of hippocampal encoding in younger infants.

We predicted that hippocampal activity at encoding would be linked to greater looking at the old versus the new item at test (a familiarity preference). However, infants sometimes look longer at the new item in visual paired comparison tasks (a novelty preference). Many factors contribute to familiarity versus novelty preferences (*34*), but we expected a link between hippocampal encoding and familiarity preferences principally because old items had been seen only once before, unlike the multiple exposures needed for habituation linked to novelty preferences (*48*). The pacing of the task, number of items, complexity of items, intervening trials, and delay between study and test further favored familiarity preferences. Consistent with this prediction, the hippocampal subsequent memory effect was found in infants who showed a stronger familiarity preference. However, we did not obtain a familiarity preference on average across all participants. This reflects variance in memory-related looking behavior across items, which we capitalized on in our within-participant subsequent memory analyses. We are not claiming that all familiarity preferences depend on hippocampal encoding, only that we have found evidence for this link given the parameters of our task. Because familiarity preferences have traditionally been associated with partial or incomplete encoding (*34*), our results may reflect still-maturing hippocampal memory function. Future studies could test paradigms that elicit novelty preferences and assess their basis in the hippocampus (*49*, *50*).

In exploratory analyses of the test trials, we found tentative evidence that the posterior hippocampus may be involved in memory retrieval in older infants (fig. S11). However, the visual paired comparison paradigm used for the test trials was not designed to assess neural mechanisms of memory retrieval because of the presence of both old and new items that could yield a mixture of retrieval and encoding processes, respectively. Future work could use alternative test designs, such as an item recognition task in which a single old or new item is presented on each test trial, as has been used in adult fMRI studies (*51*) and infant pupillometry research (*52*). Moreover, future studies will be needed to address richer forms of memory that are hallmarks of adult hippocampal processing, such as spontaneous or free recall (*53*, *54*), associative inference (*55*), and relational binding (*21*). Until then, it remains possible that infantile amnesia may be partly attributable to real but impoverished encoding in infancy.

Episodic memory depends on the trisynaptic pathway of the hippocampus (entorhinal cortex to dentate gyrus, CA3, and CA1 subfields) (*38*), which develops later in nonhuman primates than the monosynaptic pathway (entorhinal cortex to CA1) (*19*) thought to support statistical learning. Consistent with this, episodic encoding in the current study was observed only in infants older than 12 months, whereas statistical learning has been observed in the hippocampus throughout infancy, starting at around 3 months (*18*). The emergence of hippocampal encoding near the end of the first year of life aligns with prior behavioral studies (*4*, *21*, *22*), though it is earlier than predictions based on spatial memory and structural development in nonhuman animals (*5*, *56*). Understanding the onset of hippocampal encoding more precisely will require denser sampling of the months before and after 1 year and may be better assessed in longitudinal studies that account for variation within individuals. This longitudinal approach may be valuable for determining the relative onsets of different types of memory, including more complex forms of relational memory.

In addition to age, there are other cognitive, demographic, and environmental factors that contribute to individual differences in infant memory, including attentional abilities (*57*), socioeconomic status (*58*), and infant-caregiver

relationships (*59*). Understanding the impact of these factors on the hippocampus and its emerging ability to encode episodic memories in infancy may be helpful for predicting later cognitive outcomes and for potential interventions to support or enhance memory in young children. Such questions would need to be addressed in larger samples with adequate statistical power for individual differences analyses (*60*).

## Conclusions

Why grown humans have a years-long blind spot in their episodic memory for the period of infancy remains a puzzle. It had been unclear which stage(s) in the life of a memory are responsible for this infantile amnesia: encoding, consolidation, storage, and/or retrieval. By showing that the hippocampus has at least some capacity to encode individual experiences beginning around 1 year of age, this study establishes a boundary condition for accounts of infantile amnesia that assume broad failures of encoding from hippocampal immaturity. Our findings are consistent with recent studies in rodents showing that memory engrams formed during infancy in the dorsal hippocampus, homologous to the posterior hippocampus in humans, can persist into adulthood but remain inaccessible at retrieval without direct stimulation (*10*, *12*) or reminders (*9*). Whether the encoding capacity of the infant hippocampus extends beyond items to include the contextual and relational information central to rich autobiographical memories is an open question. Determining the precise developmental trajectory of hippocampal memory processes and the role of postencoding mechanisms in infantile amnesia will require long-term studies that capture the ground truth of what infants experience and track the persistence and accessibility of these memories across childhood and beyond.

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## SUPPLEMENTARY MATERIALS

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Materials and Methods
Figs. S1 to S12
Tables S1 and S2
References (*64*–*70*)
MDAR Reproducibility Checklist

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