

Constructing a past in infancy: a neuro-developmental account

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The ability to recall the past is fundamental yet until relatively recently, infants were assumed to lack the capacity. Contrary to this perspective, non-verbal tests indicate that developments in recall are well underway by late in the first year of life; by the end of the second year, long-term recall is reliable and robust. New research combining electrophysiological and behavioral measures is identifying the loci of age-related changes: they are attributed to more effective and efficient encoding, consolidation and storage processes associated with developments in the temporal-cortical network that subserves recall. The emerging framework, which applies to episodic and autobiographical memory, highlights the essential developmental continuities in memory from infancy onwards and sheds new light on the phenomenon of childhood amnesia.

Introduction

How long is the tale of your past? For most adults, the first chapter of the autobiography seems to have gone missing. We have few (if any) memories from the first years of life. Until the mid 1980s this ‘amnesia of childhood’ [1] was widely attributed to late development of the ability to recall. Children younger than 3 years were assumed to be unable to create accessible memories, which provided a ready explanation for the sparse representation of such memories in adults’ corpora. Changing perspectives on the nature of the infant mind [2] and brain [3], coupled with methodological advances [4], challenged the prevailing view. Twenty years of research using a non-verbal analogue to verbal report has made clear that before their first birthdays, infants recall the past. Nevertheless there are pronounced changes in recall throughout infancy and beyond [5,6].

To explain the changes, researchers have begun focusing on the processes involved in memory trace construction and retrieval, and on the developmental course of the structures that subserve them. Parsing memory into its constituent processes has proven very powerful in the adult cognitive science and neuroscience literatures. It has substantially increased the specificity of our knowledge of relations between the various mnemonic functions and the structures (and networks) that subserve them [7]. In the developmental literature, the approach

is new but is already bringing refinements of our perspective on the source(s) of age-related change. As a result, we have stepped closer to the brink of explanation of the changes. Moreover, the new perspective not only alters our view of the mnemonic life of infants, but also informs us about the ‘fates’ of early memories and thus why they are such rare commodities.

Age-related changes in long-term recall in infancy

How do you test recall in an organism that cannot speak? One of the most prominent developmental scientists, Jean Piaget [8], nominated imitation after a delay as a means. The experimental version of Piaget’s candidate – ‘elicited imitation’ – has proven to be a valid and productive test of recall (see Box 1). Use of imitation with infants as young as 6 months has revealed early mnemonic competence as well as clear development [9,10]. Two changes are especially salient [5].

First, there are changes in the *reliability* with which recall is observed and in particular, in recall of the temporal order of events (as opposed to the ‘items’ or actions that constitute them). Even after seeing a multi-step sequence demonstrated six times, only 25% of 6-month-olds recall the sequence in order 24 h later [11]. About 50% of 9-month-olds demonstrate ordered recall 1 month after seeing a sequence of events [12,13]. By 20 months, long-term recall is the rule: 100% of 20-month-olds recall the order of multi-step sequences over a 1-month period [14]. The second major change is in the *temporal extent* of memory. Six-month-olds remember for 24 h [11]. Nine-month-olds remember for 1 month but not for 3 months, whereas 10-month-olds remember over the longer delay [13]. A substantial proportion of 20-month-olds recall the order of events even after 1 year [14].

Sources of age-related change in recall

A full explanation of changes in long-term recall will involve multiple levels of analysis, from proteins and synapses, to neural systems, to cultural influences on memory and its expression [15,16]. At present, much progress is being made by adopting an intermediate level of analysis – one connecting behavior with the processes and neural systems that support it [17,18]. Perhaps because of their salience, changes in prefrontal cortex and associated changes in retrieval are frequent nominees for ‘what develops’ in early memory. For example, Liston and Kagan [19] invoked more effective retrieval to explain

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Box 1. Elicited imitation: a non-verbal test of recall

Elicited imitation involves using objects or props to produce an action or a sequence of actions that the subject is invited to imitate either immediately (elicited imitation), after a delay (deferred imitation), or both. The example sequence shown in Figure 1a involves moving a lever from the front of a door and then opening the door to reveal an infant-friendly character. In an initial baseline or premodeling period, the props are given to the infant for a period of observation of spontaneous behavior. Then the action or sequence of actions is demonstrated by a model (typically an adult experimenter). Either immediately, after a delay, or both, the props are returned to the infant whose behavior is observed. Two primary dependent measures are derived: the number of actions produced and the number of ordered pairs of actions. If postmodeling values on these variables are higher than baseline values (and/or if performance on familiar sequences is higher than on novel sequences), memory is inferred (Figure 1b). Ordered reproduction of sequences provides the strongest indication that the task taps recall as opposed to recognition. Whereas the props

can be said to cue recall of the actions to be produced, once the model is gone, there is no information about the temporal order of the actions available in the perceptual array. To reproduce an ordered sequence, temporal order information must be encoded at the time of presentation and later retrieved from a representation of the event. In this regard the task is analogous to verbal report. Moreover, the technique has been validated as a measure of declarative or explicit memory, on the bases that: (i) individuals who sustained medial temporal lobe damage as either adults [56] or children [57] show impaired performance on the task; (ii) once children acquire the verbal skills necessary to do so, they provide verbal descriptions of sequences experienced as preverbal infants in the context of elicited imitation [58–61]. Verbal accessibility would not be possible if the behavior acquired in imitation was encoded as a procedural memory. (3) Performance on imitation tasks in infancy is correlated with later performance on declarative memory tasks including sort-recall and the 'Children's Memory Scale' [62].

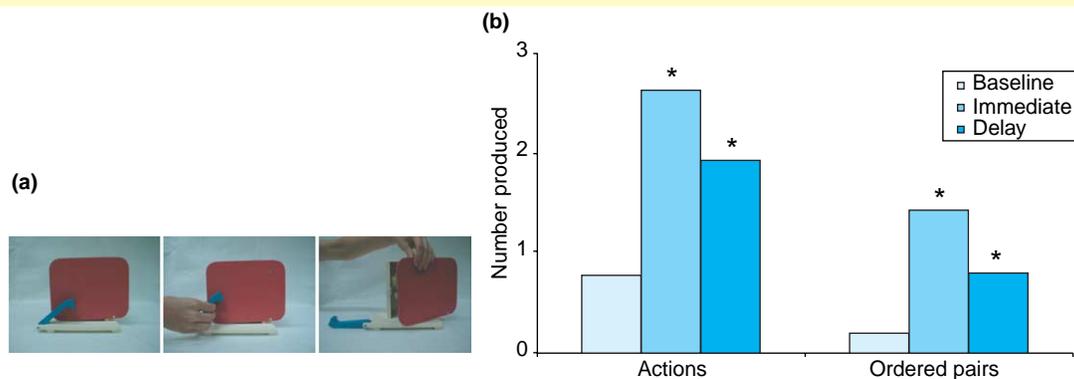


Figure 1. (a) Example of a sequence to be imitated. (b) The number of actions and ordered pairs of actions produced by 20-month-old infants in a baseline period, at immediate recall after the sequence is demonstrated, and after a two-week delay. Data redrawn from [55].

the observation that infants of 17 and 24 months of age at the time of experience of events remembered them 4 months later, whereas 9-month-old infants did not. However, attribution of change to retrieval processes implies that memory storage did not differ between the older and younger infants. Equivalent storage in turn implies no age-related differences in encoding. Consideration of the processes of memory trace construction and maintenance [20], and of the development of the structures and network that support them [21], suggests that these are not 'safe' assumptions. New research is highlighting the peril associated with them. After review of the relevant literatures, an emerging alternative conceptualization that places the locus of early age-related differences in long-term recall not at the 'rear end' of retrieval, but at the 'front end' of encoding and consolidation [22] is discussed. In this framework, age differences associated with retrieval are later to develop.

Memory traces 'under construction'

Formation, maintenance and subsequent retrieval of memories depends on a multi-component network involving medial temporal and cortical structures [23–25]. Primary, secondary and association cortices are involved in initial registration of experiences. Cortical and medial temporal structures consolidate the elements of experience into enduring memory traces. The storage sites for

long-term memories are in the cortices that gave rise to the original experiences. Prefrontal cortex in particular is implicated in retrieval of traces from long-term stores.

In the mature organism, the integrity of these neural structures affects mnemonic processes. Hippocampal lesions are associated with impairments in the formation of new declarative memories [20]. Lesions to prefrontal cortex are associated with deficits in retrieval, especially of episodic and autobiographical memories [26]. Logic dictates that in the developing human, mnemonic processes are affected by maturation of the structures and network in which they participate [3,27]. Portions of the medial temporal structures, including the cell fields of the hippocampus, mature relatively early [28]. By contrast, prefrontal cortex and the dentate gyrus of the hippocampus are later to mature. It is not until 20 to 24 months that the numbers of synapses in these structures reach a peak [29], heralding their functional maturity [30]. And it is not until late in the preschool years and adolescence or early adulthood that adult numbers of synapses are apparent in the dentate gyrus and prefrontal cortex, respectively [29,31], indicating full maturity of these structures [30]. The connections between the structures also are slow to develop [32,33]. Late development of cortical structures is important because they are implicated in all phases of the life of a memory. Late development of the dentate gyrus is crucial because in the mature organism, it is

the major 'route in' to the hippocampus. Less effective and efficient communication between cortical structures and the hippocampus would present challenges to consolidation, and therefore storage, of new information [34]. As these structures and connections between them develop we should see age-related changes in long-term recall (as well as in spatial memory tasks: [35]).

Changes in encoding

Although encoding cannot be directly observed, we can test for age differences in memory shortly after learning. Both behavioral and electrophysiological measures indicate age-related changes in the first 2 years of life. When they are tested immediately after a single experience of an event, 16-month-olds remember fewer actions and less information about temporal order than 20-month-olds [36]. Studies in which children are brought to a criterion level of learning – implying complete encoding – reveal that 12-month-olds require more learning trials than 15-month-olds who in turn require more trials than 18-month-olds [37].

Event-related potentials (ERPs) also suggest age-related changes in encoding (see Box 2). In a longitudinal study [38], infants were exposed to sequences and then

ERPs were recorded as they viewed photographs of the props used to produce the sequences interspersed with props from novel sequences. Differential responses to the different classes of stimuli indicated recognition and thus, encoding. Infants had more robust ERP responses to sequences encoded at 10 months of age than of sequences encoded at 9 months; responses to novel sequences did not differ at the two ages (Figure 1a). Differential encoding related to differential recall: infants had more robust recall of sequences encoded at 10 months than of sequences encoded at 9 months (Figure 1b) These studies establish encoding processes as a source of developmental differences in long-term recall in infancy.

Changes in consolidation and storage

Age-related differences in encoding account for a portion – but not all – of the age-related variance in long-term recall. With encoding processes controlled statistically [14], by matching [39], or by criterion learning [37], developmental differences are still apparent: Older infants and children remember more, and for longer periods of time, than their younger counterparts. This indicates that we must look beyond encoding to the next phases of memory trace consolidation and storage.

A clear indication that consolidation and storage processes are a source of variance in long-term recall comes from another study in which behavior and ERPs were combined [40]. To minimize encoding as a source of variability, 9-month-olds were exposed to sequences (A, B and C) at each of three sessions. At the third session encoding was tested via ERP (old Sequence A versus new Sequence D). One week later consolidation and storage was tested in another ERP test (Sequences B and E). Recall of the sequences was tested 1 month later (along with three new sequences as a within-subjects control). As a group the infants showed evidence of encoding (see Figure 2a) but there was differential long-term recall that in turn related to differential consolidation and storage. Infants who did not recall the sequences after 1 month also did not recognize them after 1 week (Figure 2b, left bars). By contrast, infants who recalled the sequences showed successful consolidation and storage (Figure 2b, right bars) ([40]; see also [41]). The subgroups did not differ at encoding nor was individual variability in encoding a significant predictor of long-term recall. By contrast, successful consolidation and storage over 1 week accounted for 28% of the variance in recall after 1 month. As discussed below, differential savings in relearning suggests that the less successful group experienced storage failure, as opposed to retrieval failure.

Consolidation and storage processes continue to account for variance in the second year. For infants 20 months of age, the amount of information retained 48 h after exposure to events explains 25% of variance in recall after 1 month [42]. Moreover, variability in initial consolidation is observed in populations of infants with suspected hippocampal damage associated with maternal gestational diabetes (which in animal models impairs hippocampal function [43]). At immediate testing, infants born to mothers with gestational diabetes do not differ from infants from non-diabetic pregnancies. By contrast,

Box 2. Event-related potentials as measures of memory in infancy

Event-related potentials (ERPs) are recorded from electrodes on the surface of the scalp. The ERP components of interest in infancy research are different from those in the adult literature [63]. In infant research, two major components are analyzed. As illustrated in Figure 1, the first component occurs in a middle-latency window roughly 400–800 ms after stimulus onset and is indicative of attention modulated by memory. Over anterior scalp locations the effect is observed at midline leads and is negative in deflection. Over lateral-posterior scalp locations the component is positive. In tests administered immediately after experience of sequences, recognition is indicated by differential amplitude of response to familiar relative to novel stimuli [38]. After a delay, recognition may be inferred by longer latency to peak amplitude for familiar than for novel stimuli [40]. The second component is observed 800 to 1200–1500 ms after stimulus onset (long-latency window). This slow wave does not feature clear components but rather, amplitude that either returns to baseline (after the middle-latency deflection; associated with familiar or well encoded stimuli) or that continues at greater amplitude, indicative of continued processing.

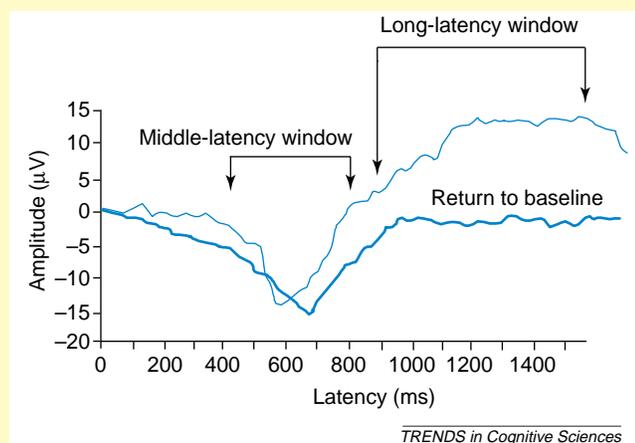


Figure 1. ERP measurement in infancy. Two main components are analyzed, in middle- and long-latency windows (see text for details).

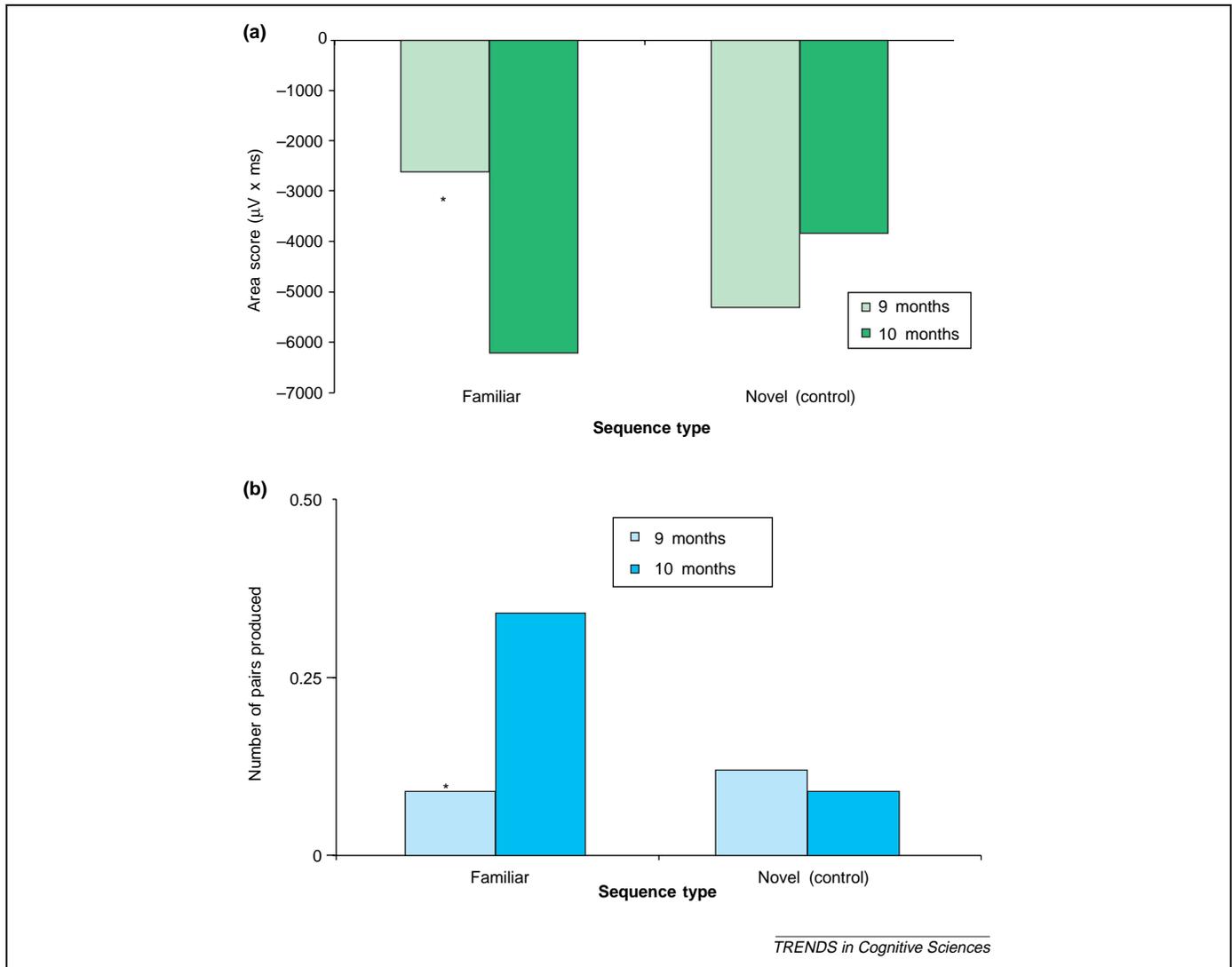


Figure 1. (a) Amplitude of event-related potential (ERP) responses to familiar and novel (control) sequences in the long-latency window (900–1500 ms after stimulus onset). The amplitude of response to familiar sequences was significantly (denoted by asterisk) larger to stimuli experienced at 10 months of age than at 9 months. Responses to novel, control stimuli experienced at the two ages did not differ significantly. (b) The number of pairs of actions produced in target order (a measure of ordered recall) for familiar and novel (control) sequences. For familiar sequences, ordered recall was greater for stimuli experienced at 10 months of age relative to stimuli experienced at 9 months of age. Levels of ordered reproduction of novel, control sequences did not differ at the two ages. Data redrawn from [38].

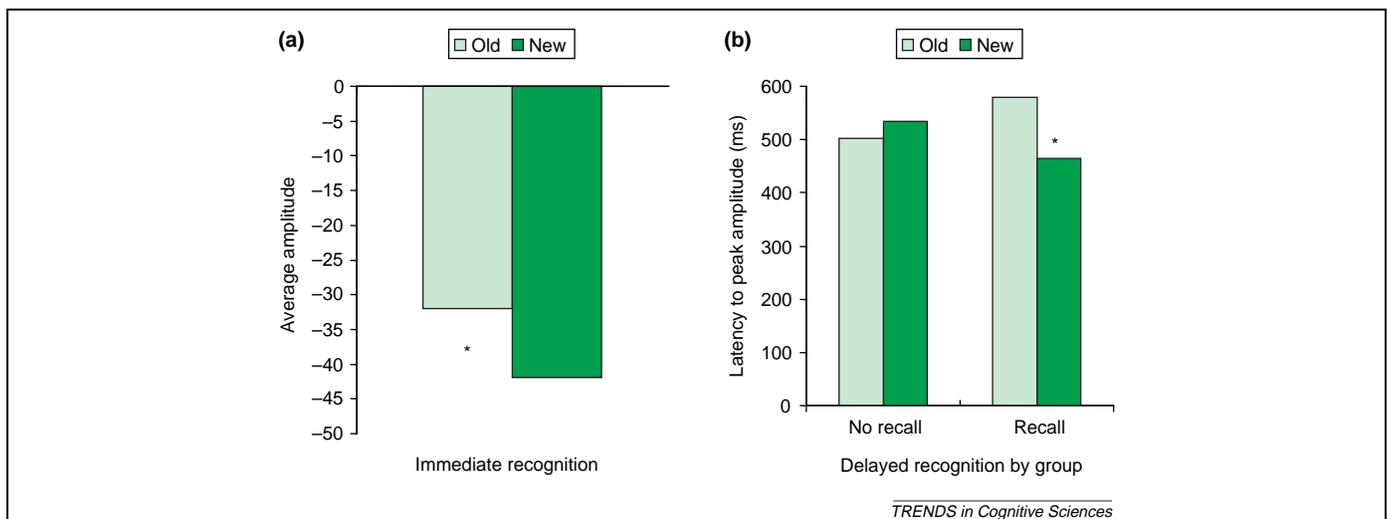


Figure 2. (a) Average ERP amplitude at electrode site Fz, providing evidence of differential response to familiar ('Old' sequence) and novel ('New' sequence) stimuli. This indicates successful encoding of the sequences. (b) The 1-week delayed ERP responses (latency to peak amplitude) for infants who failed to show ordered recall after 1 month (left bars). The lack of differentiation of responses to familiar and novel stimuli indicates failed consolidation and storage. The right bars reflect the 1-week delayed ERP responses for infants who later showed evidence of ordered recall. Differential responses to old and new stimuli indicate successful consolidation and storage. Data redrawn from [40].

after a 10-min delay, performance by affected infants is significantly lower whether tested at 12 months [44] or 24 months [45]. Because differences were not observed at immediate testing, consolidation and storage of information, as opposed to initial encoding of it, is implicated.

Changes in retrieval

Evidence that encoding and consolidation and storage processes account for age-related and individual variability in long-term recall makes clear that we cannot explain developmental changes only by examining end-stage processes such as retrieval. In fact, the observation that together, encoding and consolidation and storage processes account for as much as 70% of the variance in long-term recall [42] begs the question of whether retrieval processes explain *any* age-related variance in long-term recall in infancy. This question is surprisingly difficult to address because in most studies there is no experimental means of determining whether age effects occur because younger infants experience retrieval failure (memory traces remain intact but become inaccessible) or storage failure (memory traces lose their integrity and become unavailable).

One means of distinguishing storage from retrieval failure is to provide multiple test trials without intervening study trials [46]. The logic is that because each retrieval attempt entails re-encoding [47], even though the material is not presented again overtly, the associated strengthening of the memory and route to retrieval of it can render an intact trace more accessible on a subsequent trial. Conversely, lack of improvement across trials implies that the trace is not available. Relearning also can distinguish between an inaccessible trace and one that has disintegrated. Classically, when the number of trials required to relearn a stimulus was smaller than the number required for initial learning, savings in relearning was said to have occurred [48]. Savings presumably accrues because the products of relearning are integrated with an existing (although not necessarily accessible) trace. Conversely, the absence of savings is attributed to storage failure: there is no residual trace upon which to build. Age-related differences in relearning would suggest that the residual traces available to children of different ages are differentially intact.

Because the strategy of examining the constituent processes of memory is new to the infancy literature, means of differentiating storage and retrieval failure have rarely been invoked. When they are, they suggest storage, rather than retrieval, as the major source of age-related change. In Bauer [39], 13- to 20-month-olds were matched for levels of encoding before imposition of 1- to 6-month delays (see Figure 3 for data from 16- and 20-month-olds). In spite of the matching, age-related differences in memory loss were apparent. At the longer delays in particular, younger infants showed more forgetting than older infants; the differences were apparent on two test trials, the second of which also featured additional retrieval cues (thus further reducing retrieval demands). Storage processes also were implicated by age-related differences in relearning. In each case, older children showed greater preservation of memory

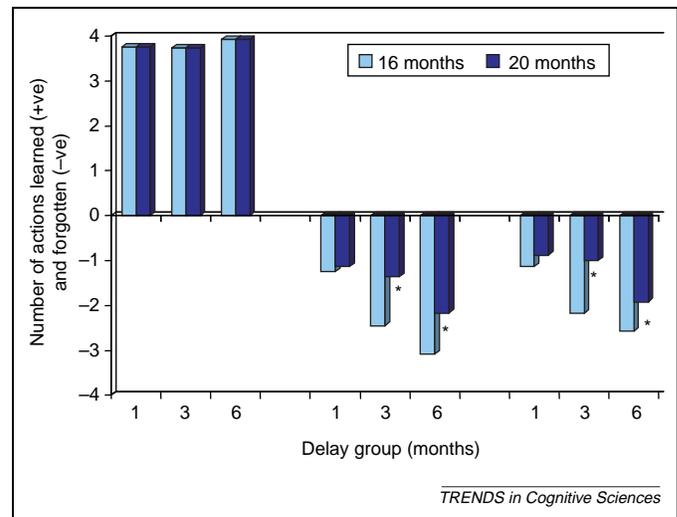


Figure 3. Learning and forgetting for 16- and 20-month-old infants tested after 1, 3 and 6 month delay intervals. Positive values indicate the number of actions produced immediately after learning by the two age groups in each of the three delay conditions (between-subjects). Younger and older infants were matched for levels of learning before imposition of the delays. Negative values indicate the number of actions lost or forgotten by each age group in each delay condition. The left set of bars indicates performance on the first retrieval trial, when the event-related props served as the only retrieval cues. The right set of bars indicates performance on the second retrieval trial, when retrieval was supported both by the event-related props and by verbal reminders of the to-be-remembered sequences. Although the age groups were matched for level of learning, the younger infants lost more information over time, relative to the older infants. The age difference was apparent on both retrieval trials and was pronounced at the longer delays (3 and 6 months). Data redrawn from [39].

traces in storage, relative to younger children. Similarly, group differences in relearning were apparent in Bauer *et al.* [40]: infants with apparently more successful consolidation and storage as indexed by an ERP test 1 week after exposure to events also had higher levels of relearning.

Does the relative paucity of evidence of variance explained by retrieval processes early in life imply that the dramatic postnatal changes in prefrontal cortex have no implications for developments in long-term recall? Certainly not. For one thing, the variance associated with retrieval processes has yet to be systematically identified. Much additional research is needed to map age-related changes associated with each phase of memory trace construction, storage and subsequent retrieval (see Box 3). Second, prefrontal cortex plays many roles in memory,

Box 3. Questions for future research

Further progress in construction of a neuro-developmental account of age-related changes in infant memory will require:

- More information about the time course of basic brain development in the young human
- Systematic identification of the variance associated with encoding, consolidation and storage, and retrieval processes across the first two years of life and beyond
- Further development of conceptual links between observed age-related changes in the basic processes of encoding, consolidation and storage, and retrieval, and developments in the neural substrates that presumably underlie them
- Establishment of converging tests of declarative or explicit memory development and relations between neural structures and behavior
- Determination of how different memory networks (those subserving declarative and different types of non-declarative memory) 'talk' to one another

only one of which is retrieval. Cortical and medial temporal structures interact during encoding and consolidation. As a consequence, developments in both regions are likely to contribute to age-related changes. Third, the ultimate storage sites for long-term memories are the association cortices [49]. Prefrontal cortex plays an especially significant role in storage of the *where* and *when* of events and experiences [7], the very features that locate memories in specific place and time. Thus, even if not through retrieval processes, changes in prefrontal cortex will no doubt be found to make substantial contributions to developments in long-term recall [5].

Shift in the locus of forgetting and its implications for childhood amnesia

Although the approach of parsing memory into its constituent processes is new in the literature on early development of recall memory, it has already permitted refinements of our perspective on the source(s) of age-related change and thus the mechanisms of change. Together, the data suggest that over development, as medial temporal structures reach maturity, the locus of forgetting shifts from the initial phases of memory trace construction to the later phases of trace retrieval [50]. Consistent with this suggestion, storage failure rates decline over childhood [46]. Additional research is necessary to determine the time course and rate of changes in the variance accounted for by encoding, consolidation and storage and retrieval processes over development.

As described in Bauer [50], the shift in locus of forgetting has implications for autobiographical memory and the phenomenon of childhood amnesia. As more and more memories survive the initial periods of vulnerability associated with encoding and consolidation, more memories are successfully stored. Moreover, more effective and efficient consolidation implies that the traces that are stored include more features that make memories distinctive from one another and relevant to one's self (elements such as the *who*, *what*, *where*, *when*, *why* and *how* of events). Thus, not only are more traces stored, they are of higher quality, and higher quality traces are more available for retrieval. In these ways, changes in the basic processes of encoding, consolidation and storage, and retrieval, contribute to an increase in the number of memories that feature personal or autobiographical elements (see [16] for additional contributing factors). It is probably no coincidence that the adult-like distribution of autobiographical memories that signals the offset of childhood amnesia becomes apparent in the early school years [51], as medial temporal structures responsible for consolidation mature. As more newly-formed memories survive into maturity, they are then subjected to the 'normal forgetting' that characterizes adults' autobiographies [52,53]. This perspective on the changing distribution of autobiographical memories lacks some of the glamour and mystery of mechanisms such as repression [54]. It compensates by making more apparent the developmental continuity of personal memory that might otherwise be obscured.

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References

- 1 Freud, S. (1905/1953) Childhood and concealing memories. *The Basic Writings of Sigmund Freud*. (Brill, A.A., transl. and ed.), The Modern Library
- 2 Mandler, J.M. (2004) *The Foundations of Mind: Origins of Conceptual Thought*, Oxford University Press
- 3 Nelson, C.A. et al. (2006) Neural bases of cognitive development. In *Handbook of Child Psychology: Cognition, Perception, and Language* (6th Edn) (Kuhn, D. and Siegler, R., eds), pp. 3–57, John Wiley & Sons
- 4 Bauer, P.J. (2004) New developments in the study of infant memory. In *Blackwell Handbook of Research Methods in Developmental Science* (Teti, D.M., ed.), pp. 467–488, Blackwell
- 5 Bauer, P.J. (2004) Getting explicit memory off the ground: Steps toward construction of a neuro-developmental account of changes in the first two years of life. *Dev. Rev.* 24, 347–373
- 6 Hayne, H. (2004) Infant memory development: Implications for childhood amnesia. *Dev. Rev.* 24, 33–73
- 7 Cabeza, R. et al. (2004) Brain activity during episodic retrieval of autobiographical and laboratory events: An fMRI study using a novel photo paradigm. *J. Cogn. Neurosci.* 16, 1583–1594
- 8 Piaget, J. (1952) *The Origins of Intelligence in Children*, International Universities Press
- 9 Bauer, P.J. et al. In the language of multiple memory systems, defining and describing developments in long-term declarative memory. In *Short- and Long-Term Memory in Infancy and Early Childhood: Taking the First Steps Toward Remembering* (Oakes, L.M. and Bauer, P.J., eds), Oxford University Press (in press)
- 10 Hayne, H. Infant memory development: New questions, new answers. In *Short- and Long-Term Memory in Infancy and Early Childhood: Taking the First Steps Toward Remembering* (Oakes, L.M. and Bauer, P.J., eds), Oxford University Press (in press)
- 11 Barr, R. et al. (1996) Developmental change in deferred imitation by 6- to 24-month-old infants. *Infant Beh. Dev.* 19, 159–170
- 12 Carver, L.J. and Bauer, P.J. (1999) When the event is more than the sum of its parts: Nine-month-olds' long-term ordered recall. *Memory* 7, 147–174
- 13 Carver, L.J. and Bauer, P.J. (2001) The dawning of a past: The emergence of long-term explicit memory in infancy. *J. Exp. Psychol. Gen.* 130, 726–745
- 14 Bauer, P.J. et al. (2000) Parameters of remembering and forgetting in the transition from infancy to early childhood. *Monogr. Soc. Res. Child Dev.* 65, No. 4 (Serial No. 263)
- 15 Bauer, P.J. (2006) Event memory. In *Handbook of Child Psychology: Cognition, Perception, and Language* (6th Edn) (Kuhn, D. and Siegler, R., eds), pp. 373–425, John Wiley & Sons
- 16 Nelson, K. and Fivush, R. (2004) The emergence of autobiographical memory: A social cultural developmental theory. *Psychol. Rev.* 111, 486–511
- 17 Bachevalier, J. and Vargha-Khadem, F. (2005) The primate hippocampus: Ontogeny, early insult and memory. *Curr. Opin. Neurobiol.* 15, 168–174
- 18 Nelson, C.A. and Webb, S. (2003) A cognitive neuroscience perspective on early memory development. In *The Cognitive Neuroscience of Development* (de Haan, M. and Johnson, M.H., eds), pp. 99–125, Psychology Press
- 19 Liston, C. and Kagan, J. (2002) Memory enhancement in early childhood. *Nature* 419, 896
- 20 Squire, L.R. (2004) Memory systems of the brain: A brief history and current perspective. *Neurobiol. Learn. Mem.* 82, 171–177
- 21 Bachevalier, J. (2001) Neural bases of memory development: Insights from neuropsychological studies in primates. In *Handbook of Developmental Cognitive Neuroscience* (Nelson, C.A. and Luciana, M., eds), pp. 365–379, MIT Press
- 22 Bauer, P.J. Toward a neuro-developmental account of the development of declarative memory. *Dev. Psychobiol.* (in press)

- 23 Eichenbaum, H. and Cohen, N.J. (2001) *From Conditioning to Conscious Recollection: Memory Systems of the Brain*, Oxford University Press
- 24 Kandel, E.R. and Squire, L.R. (2000) Neuroscience: Breaking down scientific barriers to the study of brain and mind. *Science* 290, 1113–1120
- 25 Zola, S.M. and Squire, L.R. (2000) The medial temporal lobe and the hippocampus. In *The Oxford Handbook of Memory* (Tulving, E. and Craik, F.I.M., eds), pp. 485–500, Oxford University Press
- 26 Barnett, M.P. et al. (2000) The constituent structure of autobiographical memory: Autobiographical fluency in people with chronic epilepsy. *Memory* 8, 413–424
- 27 Johnson, M.H. and Munakata, Y. (2005) Processes of change in brain and cognitive development. *Trends Cogn. Sci.* 9, 152–188
- 28 Seress, L. (2001) Morphological changes of the human hippocampal formation from midgestation to early childhood. In *Handbook of Developmental Cognitive Neuroscience* (Nelson, C.A. and Luciana, M., eds), pp. 45–58, MIT Press
- 29 Huttenlocher, P.R. and Dabholkar, A.S. (1997) Regional differences in synaptogenesis in human cerebral cortex. *J. Comp. Neurol.* 387, 167–178
- 30 Goldman-Rakic, P.S. (1987) Circuitry of primate prefrontal cortex and regulation of behavior by representational memory. In *Handbook of Physiology, The Nervous System, Higher Functions of the Brain, Vol. 5* (Plum, F., ed.), pp. 373–417, American Physiological Society
- 31 Bourgeois, J.-P. (2001) Synaptogenesis in the neocortex of the newborn: The ultimate frontier for individuation?. In *Handbook of Developmental Cognitive Neuroscience* (Nelson, C.A. and Luciana, M., eds), pp. 23–34, MIT press
- 32 Durston, S. et al. (2002) Anatomical MRI of the developing human brain: What have we learned? *J. Am. Acad. Child Adolesc. Psychiatry* 40, 1012–1020
- 33 Schneider, J.F. et al. (2004) Fast quantitative diffusion-tensor imaging of cerebral white matter from the neonatal period to adolescence. *Neuroradiology* 46, 258–266
- 34 Gluck, M.A. and Myers, C.E. (2001) *Gateway to Memory: An Introduction to Neural Network Modeling of the Hippocampus and Learning*, MIT Press
- 35 Sluzenski, J. et al. (2004) Knowing where things are in the second year of life: Implications for hippocampal development. *J. Cogn. Neurosci.* 16, 1443–1451
- 36 Bauer, P.J. and Dow, G.A.A. (1994) Episodic memory in 16- and 20-month-old children: Specifics are generalized, but not forgotten. *Dev. Psychol.* 30, 403–417
- 37 Howe, M.L. and Courage, M.L. (1997) Independent paths in the development of infant learning and forgetting. *J. Exp. Child Psychol.* 67, 131–163
- 38 Bauer, P.J. et al. (2006) Electrophysiological indices of encoding and behavioral indices of recall: Examining relations and developmental change late in the first year of life. *Dev. Neuropsychol.* 29, 293–320
- 39 Bauer, P.J. (2005) Developments in declarative memory: Decreasing susceptibility to storage failure over the second year of life. *Psychol. Sci.* 16, 41–47
- 40 Bauer, P.J. et al. (2003) Developments in long-term explicit memory late in the first year of life: Behavioral and electrophysiological indices. *Psychol. Sci.* 14, 629–635
- 41 Carver, L.J. et al. (2000) Associations between infant brain activity and recall memory. *Dev. Sci.* 3, 234–246
- 42 Bauer, P.J. et al. (2002) Short-term forgetting: Charting its course and its implications for long-term remembering. In *Advances in Psychology Research. Vol. 9* (Shohov, S.P., ed.), pp. 53–74, Nova Science Publishers
- 43 Georgieff, M.K. and Rao, R. (2001) The role of nutrition in cognitive development. In *Handbook of Developmental Cognitive Neuroscience* (Nelson, C.A. and Luciana, M., eds), pp. 491–504, MIT Press
- 44 DeBoer, T. et al. Explicit memory performance in infants of diabetic mothers at 1 year of age. *Dev. Med. Child Neurol.* (in press)
- 45 DeBoer, T.L. et al. Declarative memory performance in infants of diabetic mothers. In *Varieties of Early Experience: Influences on Declarative Memory Development* (Bauer, P.J., ed.), Erlbaum (in press)
- 46 Howe, M.L. and O'Sullivan, J.T. (1997) What children's memories tell us about recalling our childhoods: A review of storage and retrieval processes in the development of long-term retention. *Dev. Rev.* 17, 148–204
- 47 Markowitsch, H.J. (2000) Neuroanatomy of memory. In *The Oxford Handbook of Memory* (Tulving, E. and Craik, F.I.M., eds), pp. 465–484, Oxford University Press
- 48 Ebbinghaus, H. (1885) *On Memory* (Ruger, H.A. and Bussenius, C.E., transl.), Teachers' College (1913); pbk edn, Dover (1964)
- 49 Takehara, K. et al. (2003) Time-dependent reorganization of the brain components underlying memory retention in trace eyeblink conditioning. *J. Neurosci.* 23, 9897–9905
- 50 Bauer, P.J. (2006) *Remembering the Times of our Lives: Memory in Infancy and Beyond*, Erlbaum
- 51 Van Abbema, D.L. and Bauer, P.J. (2005) Autobiographical memory in middle childhood: Recollections of the recent and distant past. *Memory* 13, 829–845
- 52 Rubin, D.C. (1982) On the retention function for autobiographical memory. *J. Verbal Learn. Verbal Behav.* 21, 21–38
- 53 Wetzler, S.E. and Sweeney, J.A. (1986) Childhood amnesia: An empirical demonstration. In *Autobiographical Memory* (Rubin, D.C., ed.), pp. 191–201, Cambridge University Press
- 54 Freud, S. (1916/1966) The archaic features and infantilism of dreams. In *Introductory Lectures on Psychoanalysis*. (Strachey, J., transl. and ed.), pp. 199–212, Norton
- 55 Bauer, P.J. and Mandler, J.M. (1989) One thing follows another: Effects of temporal structure on one- to two-year-olds' recall of events. *Dev. Psychol.* 25, 197–206
- 56 McDonough, L. et al. (1995) The deferred imitation task as a nonverbal measure of declarative memory. *Proc. Natl. Acad. Sci. U. S. A.* 92, 7580–7584
- 57 Adlam, A.-L.R. et al. (2005) Deferred imitation of action sequences in developmental amnesia. *J. Cogn. Neurosci.* 17, 240–248
- 58 Bauer, P.J. et al. (1998) If memory serves, will language? Later verbal accessibility of early memories. *Dev. Psychopathol.* 10, 655–679
- 59 Bauer, P.J. et al. (2002) Making the past present: Later verbal accessibility of early memories. *J. Cogn. Dev.* 3, 21–47
- 60 Bauer, P.J. et al. (2004) Props, not pictures, are worth a thousand words: Verbal accessibility of early memories under different conditions of contextual support. *Appl. Cogn. Psychol.* 18, 373–392
- 61 Cheatham, C.L. and Bauer, P.J. (2005) Construction of a more coherent story: Prior verbal recall predicts later verbal accessibility of early memories. *Memory* 13, 516–532
- 62 Bauer, P.J. Learning and memory: Like a horse and carriage. In *Learning and the Infant Mind*. (Woodward, A. and Needham, A., eds), Oxford University Press (in press)
- 63 Nelson, C.A. and Monk, C. (2001) The use of event-related potentials in the study of cognitive development. In *Handbook of Developmental Cognitive Neuroscience* (Nelson, C.A. and Luciana, M., eds), pp. 125–136, MIT Press

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