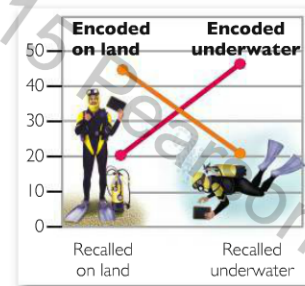


Factoid

TOT occurs in those who use sign language as well as spoken language; psychologists call this the *tip-of-the-fingers* phenomenon. Deaf signers who are unable to retrieve the names of fairly famous people but feel that they're on the verge of remembering can depict at least some part of the famous person's name with their fingers about 80 percent of the time (Thompson, Emmorey, & Gollan, 2005).

FIGURE 7.13 Research Shows That the Word Learning of Scuba Divers Depends on Context. If the divers learned words underwater, they recalled them best when underwater again (Godden & Baddeley, 1975).



REPLICABILITY ►

Can the results be duplicated in other studies?

encoding specificity

phenomenon of remembering something better when the conditions under which we retrieve information are similar to the conditions under which we encoded it

context-dependent learning

superior retrieval of memories when the external context of the original memories matches the retrieval context

state-dependent learning

superior retrieval of memories when the organism is in the same physiological or psychological state as it was during encoding

REPLICABILITY ►

Can the results be duplicated in other studies?

ENCODING SPECIFICITY: FINDING THINGS WHERE WE LEFT THEM. Why is it easier to retrieve some things from memory than others? One answer to this mystery lies in the principle of **encoding specificity** introduced by Endel Tulving (1982; Tulving & Thompson, 1973). We're more likely to remember something when the conditions present at the time we encoded it are also present at retrieval. We can see this principle at work in several psychological phenomena, two of which we'll examine here: context-dependent learning and state-dependent learning.

Context-Dependent Learning. Context-dependent learning refers to superior retrieval when the external context of the original memories matches the retrieval context (Vlach & Sandhofer, 2011). Duncan Godden and Alan Baddeley (1975) provided an example of this effect in an ingenious study of scuba divers. They presented divers with 40 unrelated words while the divers were either standing on the beach or submerged in about 15 feet of water. Godden and Baddeley then tested the divers in either the same or a different context from which they originally presented the words. The divers' memory was best when the original context matched the retrieval context, regardless of whether they were on land or underwater, as shown in **FIGURE 7.13**.

There's even evidence for context-dependent learning when undergraduates take exams. Students tend to do slightly better on their exams when tested in the same classroom in which they learned the material (Smith, 1979). You may want to gently remind your introductory psychology instructor of this fact when he or she schedules the room for your next test. Still, this effect isn't all that powerful, and not all researchers have replicated it (Saufley, Otaka, & Bavaresco, 1985). That's probably because you've acquired the information not only in the classroom but also in other settings, such as the room in which you're now reading this textbook.

State-Dependent Learning. Despite its name, state-dependent learning doesn't mean that if you learned something while on vacation in the state of Montana, you need to go back to Montana to recall it. Instead, state-dependent learning is similar to context-dependent learning, except that it refers to the internal "state" of the organism rather than the external context. That is, **state-dependent learning** refers to superior retrieval of memories when the organism is in the same physiological or psychological state as it was during encoding (McNamara, Trimmer, & Houston, 2012).

There's anecdotal evidence for this phenomenon among alcoholics, who often report that they need to get drunk to locate items—including their favorite bottles of liquor—that they'd hidden while drinking (Goodwin, 1995). Of course, we've learned that anecdotes are limited as sources of scientific evidence (see Chapter 2). However, in this case, controlled studies bear out the anecdotes: People who've learned a task while under the influence of alcohol tend to remember it better when under the influence than when sober (Goodwin et al., 1969). Still, researchers haven't always replicated these findings (Lisman, 1974), suggesting that state-dependent effects probably depend in complex ways on the participants tested and stimuli administered.

State-dependent learning sometimes extends to mood, in which case it's termed *mood-dependent learning* (Bower, 1981). Studies show that both younger and older adults find it easier to recall and recognize unpleasant memories than pleasant ones when they're sad and easier to recall and recognize pleasant memories than unpleasant ones when they're happy (Knight, Maines, & Robinson, 2002; Nelson & Craighead, 1977; Robinson & Rollings, 2011).

Mood-dependent learning can create nasty difficulties for researchers who want to draw conclusions about people's life histories. Specifically, it can result in a *retrospective bias*: Our current psychological state can distort memories of our

past (Dawes, 1988; Ross, 1989; Taylor, Russ-Eft, & Taylor, 2009). For example, most individuals with clinical depression report having been treated more harshly by their parents in childhood than do individuals without clinical depression. One explanation for this finding is that harsh parental treatment predisposes to later depression. But there's another explanation: Perhaps people's bad moods distort their memories of their childhoods.

To evaluate this possibility, researchers asked three groups of participants—(1) people who had clinical depression, (2) people who had a history of depression but weren't currently depressed, and (3) people who'd never been depressed—about how their parents treated them as children. Currently depressed participants recalled their parents as having been more rejecting and domineering toward them as children than did participants in the other two groups (Lewinsohn & Rosenbaum, 1987). So participants' moods seem to have influenced their evaluations of how their parents had treated them. In this case, we don't know whether the participants with clinical depression were less *accurate* than the participants in the other groups, only that their memories were different (although as we'll learn in Chapter 15, mildly—but not severely—depressed people may sometimes be *more* accurate in their memories than nondepressed people).

◀ RULING OUT RIVAL HYPOTHESES

Have important alternative explanations for the findings been excluded?

Assess Your Knowledge

FACT or FICTION?

1. We encode virtually all of our life experiences, even though we can't retrieve more than a tiny proportion of them. **True / False**
2. We need to practice mnemonics to use them successfully. **True / False**
3. Schemas only distort memories, but don't enhance them. **True / False**
4. In general, recall is more difficult than recognition. **True / False**
5. Cramming for exams, although stressful, is actually a good strategy for enhancing long-term recall of material. **True / False**

Answers: 1. F (p. 259); 2. T (pp. 259–260); 3. F (p. 263); 4. T (p. 264); 5. F (p. 265)



Study and Review in MyPsychLab

The Biology of Memory

- 7.8** Describe the role of long-term potentiation in memory.
- 7.9** Distinguish different types of amnesia and the relevance of amnesia to the brain's organization of memory.
- 7.10** Identify the key impairments of Alzheimer's disease.

Although few of us think about it, the biology of memory plays a pivotal role in our daily lives, whether it's remembering where we left our keys or the name of that friendly person we met at last night's party. What's more, understanding how our brains store memory may help us find ways of treating devastating diseases that impair our ability to recall everyday events.



Explore in MyPsychLab the **Concept:**
Virtual Brain: Learning and Memory

The Neural Basis of Memory Storage

Locating where a library book is stored is generally pretty easy. We look it up in our library's online system, write down its number, go to the shelf, and—unless someone's recently plucked it away—find it. If we're lucky, it's right there on the shelf where it's supposed to be. Yet as we'll soon see, memory storage in the brain isn't quite this cut and dry.

THE ELUSIVE ENGRAM. Beginning in the 1920s, psychologist Karl Lashley went in search of the *engram*: the physical trace of each memory in the brain (see Chapter 6). He taught rats how to run mazes and lesioned different parts of their brains to see if they forgot how

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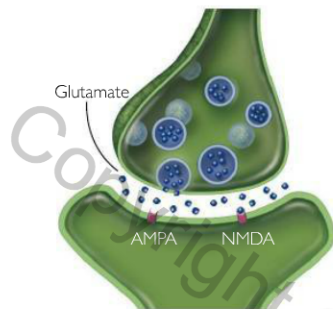


FIGURE 7.14 Neural Basis of Long-Term Potentiation. LTP enhances the release of glutamate and activates postsynaptic receptors for NMDA and AMPA

REPLICABILITY ►

Can the results be duplicated in other studies?

RULING OUT RIVAL HYPOTHESES ►

Have important alternative explanations for the findings been excluded?

Factoid

One source of evidence for the role of the hippocampus in memory comes from research on Clark's nutcracker, a remarkable North American bird. To help it prepare for long winters, Clark's nutcracker buries up to 33,000 seeds in about 5,000 places spread over about 150 square miles—and manages to locate most of them months later—even though they're often buried under several feet of snow! Clark's nutcracker has an unusually large hippocampus, which may help to explain its exceptional spatial memory (Basil et al., 1996).

long-term potentiation (LTP)

gradual strengthening of the connections among neurons from repetitive stimulation

to find their way. By doing so, Lashley hoped to discover where memory is stored in the brain. Yet after years of painstaking work, he came up empty-handed.

Still, Lashley learned two important things. First, the more brain he removed, the worse the rat performed on the maze: There's no great surprise there. Second, no matter where he removed brain tissue, the rats retained at least some memory of the maze (Lashley, 1929). Even removing up to half of the rat's cortex didn't erase the memory. These findings led Lashley to conclude that we can't simply point to a spot in the brain and say, "There's the memory of my first kiss," because that memory isn't located in a single place, like a library book sitting on a shelf. Instead, as scientists have since learned, memories of different features of experiences, like their sound, sight, and smell, are almost certainly stored in different brain regions (see Chapter 3).

Over a half century ago, Donald Hebb (1949) suggested that the engram is instead located in *assemblies* (organized groups) of neurons in the brain. According to Hebb, one neuron (A) becomes connected to another neuron (B) when it repeatedly activates that neuron. As we learned in Chapter 3, neurons, fed by a rich blend of neurotransmitters, form circuits, integrate sensory information in meaningful ways, and transform our experience of the world into lasting, perhaps even lifelong, memories.

LONG-TERM POTENTIATION—A PHYSIOLOGICAL BASIS FOR MEMORY. As we learned in Chapter 3, **long-term potentiation (LTP)** refers to a gradual strengthening of the connections among neurons by repetitive stimulation over time (Abrari et al., 2009; Bliss, Collingridge, & Morris, 2004). Terje Lomo first observed LTP in the hippocampus of rabbits in 1966, a finding replicated by many later researchers in other animals and humans. The gist of what neuroscientists have learned since the discovery of LTP is that neurons that "fire together wire together" (Malenka & Nicoll, 1999). To a large extent, Hebb was right.

Today, many researchers believe that our ability to store memories depends on strengthening the connections among neurons arranged in sprawling networks that extend to the far and deep recesses of our brains (Shors & Matzel, 1999). The question of whether LTP is directly responsible for the storage of memories or whether it affects learning indirectly by increasing arousal and attention, remains unresolved (Shors & Matzel, 1999). Still, most scientists agree that LTP plays a key role in learning and that the hippocampus plays a key role in forming lasting memories.

LTP and Glutamate LTP tends to occur at synapses where the sending neuron releases the neurotransmitter glutamate into the synaptic cleft—the space between the sending and receiving neuron (see Chapter 3). As shown in **FIGURE 7.14**, glutamate interacts with receptors for NMDA and another substance (AMPA). LTP enhances the release of glutamate into the synaptic cleft, resulting in enhanced learning (Lisman & Raghavachari, 2007; Navakkode & Korte, 2012). A research team was even able to create a "super smart mouse" by manipulating its genes to create extra receptors for NMDA. Compared with everyday mice, the super-smart mouse is an especially quick and effective learner (Lee & Silva, 2009; Tsien, 2000).

Where Is Memory Stored?

Clearly, the hippocampus is critical to memory. As we saw in Chapter 4, some researchers have even identified neurons in the hippocampus that fire in response only to certain celebrities, such as actress Halle Berry (Quiroga et al., 2005).

But is the hippocampus, or any single brain structure, the site of the elusive engram? We can say with some certainty that the answer is no. fMRI studies reveal that learned information isn't stored permanently in the hippocampus itself. Rather, the prefrontal cortex seems to be one of the major "banks" from which we withdraw our memories (Zeinab et al., 2003). But as Lashley discovered, damage to isolated areas of the prefrontal cortex—or other cortical regions, for that matter—doesn't wipe out long-established

memories. Much as the smell of a rose diffuses throughout a room, our memories distribute themselves throughout many areas of the cortex.

AMNESIA—BIOLOGICAL BASES OF EXPLICIT AND IMPLICIT MEMORY. Earlier we learned about explicit and implicit memory. Research demonstrates that two forms of memory are governed largely by different brain systems (Squire, 1987; Voss & Paller, 2008). The best evidence comes from individuals with severe amnesia. The two most common types of amnesia are **retrograde amnesia**, in which we lose some memories of our past, and **anterograde amnesia**, in which we lose the capacity to form new memories.

Amnesia Fictions and Facts. Contrary to popular belief, generalized amnesia—in which people have lost all details of their previous life (American Psychiatric Association, 2000), is quite rare (Baxendale, 2004). Even retrograde amnesia isn't especially common; anterograde amnesia is much more frequent among people with brain damage (Lilienfeld et al., 2010). Also, although many Hollywood films depict memory recovery from amnesia as abrupt, recovery from amnesia tends to occur gradually, if at all (APA, 2000).

Case Studies of Amnesia: H. M. and Clive Wearing. By far the best-known person with amnesia in the psychological literature was a man from Connecticut known only by the initials H. M. He suffered from severe epileptic seizures that his doctors couldn't control with medication. In March 1953, in a last-ditch attempt to eliminate these seizures, surgeons removed large chunks of H. M.'s temporal lobes, including both his left and right hippocampi, where they had reason to believe the seizures originated. (The surgeons of the time didn't anticipate the disastrous impact of this radical operation, which would probably never be performed today.) At the time, H. M. was 26 years old. Following the operation, H. M. developed virtually complete anterograde amnesia: He could recall almost no new information. Although he also experienced some retrograde amnesia for the 11 years prior to the surgery (Corkin, 1984), his memories from the first 15 years of his life remained pretty much intact.

In the decades following his surgery, H. M.'s life was, for all intents and purposes, frozen in time. H. M. himself put it eloquently: "Every day is alone by itself, whatever enjoyment I have had, whatever sorrow I have had." Among other things, he was oblivious to the fact that he had undergone surgery. Two years after the operation, in 1955, he reported the current date as March 1953. H. M. read the same magazines and completed the same jigsaw puzzles over and over again without any awareness of having seen them before. He didn't recall having met physicians whom he met just a few minutes earlier or remember what he ate for lunch 30 minutes ago (Milner, 1972; Scoville & Milner, 1957). Even when informed repeatedly of the death of his uncle, he showed the same dramatic grief to the news each time (Shimamura, 1992). H. M.'s identity was revealed as Henry Molaison only after his death in December 2008 at the age of 82. For 55 years, he acquired virtually no new explicit memories.

H. M.'s tragic case, like that of Damasio's patient David, illustrates a striking dissociation between explicit and implicit memory. Researchers asked H. M. to trace simple geometrical shapes from a mirror (see **Figure 7.15**), a task that just about all people find infuriatingly difficult when they first try it. Although H. M. had no recollection of ever having performed this task before, his performance improved steadily over time (Milner, 1964, 1965). So although H. M. had no explicit memory for this task, he displayed clear-cut implicit—specifically, procedural—memory for it.

When researchers examined H. M.'s brain using imaging techniques, they found that not only his hippocampus but also his surrounding cortex and neighboring amygdala (see Chapter 3) were damaged (Corkin et al., 1997). This finding and others led researchers to hypothesize that large circuits connecting different parts of the limbic system, including the hippocampus, and amygdala, are critical to memory (see **FIGURE 7.16** on page 270).

Similar evidence for a distinction between explicit and implicit memory comes from the case of Clive Wearing, a former music producer in Great Britain whose hippocampi (along with several other brain structures) were destroyed by a herpes virus in 1985



FIGURE 7.15 A Mirror Tracing Task Similar to That Administered to H. M. How well can you draw while looking in a mirror? On this task, used to assess implicit memory, participants must trace a star while looking only at a mirror.



Watch in MyPsychLab the Video: Special Topics: When Memory Fails

retrograde amnesia

loss of memories from our past

anterograde amnesia

inability to encode new memories from our experiences

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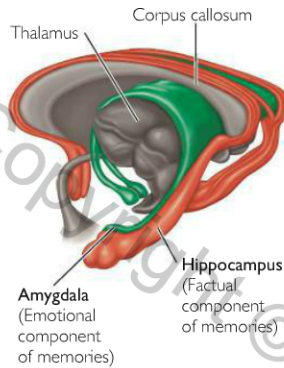


FIGURE 7.16 Emotional Memories and the Brain. Research suggests that the amygdala helps us recall the fear associated with scary experiences and the hippocampus helps us recall the experiences themselves.

(Wearing, 2005). Like H. M., Clive has virtually complete anterograde amnesia. When his wife leaves the room for a few minutes and returns, he showers her with immense affection, as though he hasn't seen her in years. Yet Clive shows implicit memory in the form of priming effects. When his wife says "St. Mary's," he quickly responds "Padding-ton," entirely oblivious of why he says that. The name of the hospital to which Clive Wearing was taken after his viral infection was—you guessed it—St. Mary's Paddington (Wearing, 2005). The bottom line: Damage to the hippocampus impairs explicit memory, but leaves implicit memory intact.

EMOTIONAL MEMORY. We usually think of memory as our good friend, as a lifelong companion that helps us to store useful information, allowing us to cope with our environment. Yet our memories can also bring us distress, as in the case of a 53-year-old woman who reported olfactory (smell) memories tracking back to a brutal gang rape decades earlier (Vermetten & Bremner, 2003). Olfactory hallucinations of leather, alcohol, and the aftershave Old Spice—all of which were present at the rape scene—triggered intense fear responses that led her to retreat to a closet and engage in self-destructive behavior.

The Role of the Amygdala The amygdala is where the emotional components of these and other memories, especially those governing fear, are stored. The amygdala interacts with the hippocampus during the formation of memory, but each structure contributes different information (refer again to Figure 7.16). Researchers uncovered the specific roles of the amygdala and hippocampus in a study of two patients identified by their initials, S. M. and W. S. The first suffered damage to the amygdala; the second, to the hippocampus (LeBar & Phelps, 2005). The patient with amygdala damage (S. M.) remembered facts about the fear-producing experience, but not the fear. In contrast, the patient with hippocampal damage (W. S.) remembered the fear, but not the facts about the fear-producing experience. So the amygdala and hippocampus play distinctive roles in memory, with the amygdala helping us recall the emotions associated with fear-provoking events and the hippocampus helping us recall the events themselves (Fitzgerald et al., 2011; Marschner et al., 2008).

Erasing Painful Memories What if it were possible to erase or take the sting out of traumatic or painful memories, like witnessing someone's death or experiencing the devastating breakup of a relationship?

As we've learned, emotional memories can persist, even if they often become distorted over time. The hormones adrenaline and norepinephrine (see Chapter 3) are released in the face of stress and stimulate protein (beta-adrenergic) receptors on nerve cells, which solidify emotional memories.

Lawrence Cahill and James McGaugh (1995) demonstrated the staying power of emotional memories in an elegant study. They created two stories regarding 12 slides they showed to participants. They told half of the participants an emotionally neutral story about a boy's visit to a hospital where his father works. They told the other half a far more disturbing story about the same slides; in the middle of the story, they informed participants that the boy was injured and operated on at a hospital to reattach his severed legs. Participants returned for a memory test 24 hours later, and Cahill and McGaugh asked them what they remembered. Participants who heard the emotionally arousing story displayed the best recall for the part of the story about the boy's trauma. In contrast, participants who heard the neutral story recalled the same amount of detail for all parts of the story.

Cahill and his colleagues (Cahill et al., 1994) conducted a similar experiment with an interesting twist. This time, they gave some participants a drug called *propranolol*, which blocks the effects of adrenaline on beta-adrenergic receptors (doctors also use it to treat high blood pressure). When participants' adrenaline was inhibited by propranolol, they didn't display especially good recall for the emotionally arousing part of the story. In fact, their recall was no different from that of individuals who listened to the emotionally neutral story.

 **Watch in MyPsychLab the Video:** In the Real World: The Memories We Don't Want

Psychiatrist Roger Pitman reasoned that propranolol might blunt the memories of real-life traumas, such as automobile accidents. Pitman and his colleagues (Pitman et al., 2002) administered propranolol to people for 10 days after they experienced a traumatic event, such as a car accident and, a month later, examined their physical reactions to individually prepared tapes that replayed key aspects of the event. Forty-three percent of participants who received a placebo showed a physical response to the tape that re-created their traumatic experience. Yet none of the people who received the drug did.

Pitman's pill only dampened the effects of traumatic memories; it didn't erase them. These findings have been replicated by other investigators using different designs (Brun et al., 2011; Kindt, Soeter, & Vervliet, 2009). Still, this research hasn't laid to rest difficult questions about whether such procedures are ethical, much less desirable. After all, if we could choose to forget every negative experience, would we learn and grow from our mistakes? The mere fact that we *can* do something doesn't mean we *should*, so the debate continues.

◀ REPLICABILITY

Can the results be duplicated in other studies?

The Biology of Memory Deterioration

As we humans pass the ripe old age of 65, we usually begin to experience memory problems and some degeneration in the brain. Yet despite what many people believe (Lilienfeld et al., 2010), senility isn't an unavoidable part of aging, and some manage to make it past 100 with only modest amounts of everyday forgetfulness. But scientists disagree as to how much memory loss is "normal" during the advanced years. Some argue that we needn't accept any memory impairment as normal. Nevertheless, a longitudinal study of participants aged 59 to 84 years at baseline showed small but consistent reductions in the overall area of the cortex at two-year and four-year intervals (Resnick et al., 2003). We might assume that subtle cognitive decline would accompany these tissue losses, but alternative hypotheses are possible. For example, cognition may be fully preserved until a critical amount of tissue loss occurs.

Many people equate senility with one cause: Alzheimer's disease. Yet Alzheimer's disease is only the most frequent cause of senility, accounting for about 50 to 60 percent of cases of *dementia*, that is, severe memory loss (another common cause of senility is the accumulation of multiple small strokes in the brain). Alzheimer's disease occurs at alarming rates as people age—one American develops Alzheimer's disease every 72 seconds (Alzheimer's Disease Facts and Figures, 2007). The risk for Alzheimer's disease is 13 percent for those over 65 years of age, but a whopping 42 percent for those over 85 years of age. With the "graying" (aging) of the U.S. population, Alzheimer's disease is expected to become even more of a concern in coming decades, with approximately 13 million Americans projected to develop the disease by 2050 if a cure can't be found (Alzheimer's Association, 2012).

The cognitive impairments of Alzheimer's disease are both memory- and language-related, which corresponds to the patterns of cortical loss in this illness (see **FIGURE 7.17**). The memory loss begins with recent events, with memories of the distant past being the last to go. Patients with Alzheimer's forget their grandchildren's names well before forgetting their children's names. They also experience disorientation and are frequently at a loss as to where they are, what year it is, or who the current president is.

As we learned in Chapter 3, the Alzheimer's brain contains many senile plaques and neurofibrillary tangles. These abnormalities contribute to the loss of synapses and death of cells in the hippocampus and cerebral cortex. They may also contribute to memory loss and intellectual decline. Loss of synapses is correlated with intellectual status, with greater loss as the disease progresses (Scheff et al., 2007). But this result doesn't necessarily mean that the reduction in synapses causes the memory decline. Along with loss of synapses comes degeneration and death of acetylcholine neurons in the forebrain. Accordingly, the

◀ RULING OUT RIVAL HYPOTHESES

Have important alternative explanations for the findings been excluded?

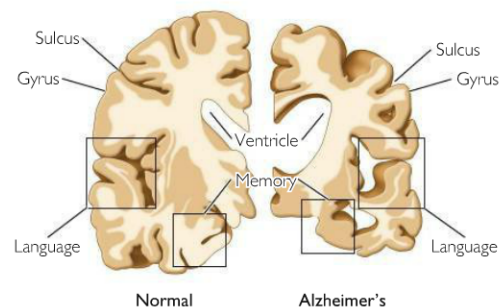


FIGURE 7.17 Changes in the Brain of Patients with Alzheimer's Disease. Changes include enlargement of the ventricles and severe loss of the cortex in areas involved in language and memory. (Source: Courtesy of Alzheimer's Disease Research, a program of the American Health Assistance Foundation)

◀ CORRELATION VS. CAUSATION

Can we be sure that A causes B?



In 2001, the year of the *Time* magazine photoessay on the “Nun Study,” Sister Ester was 106, the oldest nun in the order. The results of this study suggest that physical and mental activity may protect against memory loss.

CORRELATION VS. CAUSATION ►

Can we be sure that A causes B?

most common treatments for Alzheimer’s disease today are drugs, like Donepezil (whose genetic name is *Aricept*), that boost the amount of acetylcholine in the brain by inhibiting its breakdown. There are also experimental procedures, such as gene therapies that enhance the production of neurotrophic (growth) factors, which enable acetylcholine neurons to survive and thrive (see Chapter 3). Still other promising medications block the actions of the neurotransmitter glutamate (Francis, 2008), which as we learned in Chapter 3 tends to be toxic to neurons in high doses. Yet no treatment to date halts or reverses the course of Alzheimer’s disease. At best, these treatments only slow its progression slightly (Birks, 2012).

For this reason, researchers have evaluated people’s lifestyles to see if anything can be done to reduce the risk of Alzheimer’s disease. A massive study assessing over 4,000 people over 65 years of age showed that being physically active reduces the risk of cognitive impairment and Alzheimer’s disease (Laurin et al., 2001). This large study dovetails with earlier findings from a small but telling study of 678 nuns who were hard-working, were active, and had strong social networks. What’s most remarkable about these nuns is their advanced age—ranging from 87 to over 100 years—along with their low incidence of cognitive impairment, including Alzheimer’s disease (Snowdon, 2003). Numerous other studies suggest that people who are highly educated and intellectually active are at decreased risk of Alzheimer’s disease (Ngandu et al., 2007; Sattler et al., 2012). Admittedly, these correlational findings are ambiguous in their causal direction: Perhaps people who are more mentally and physically fit have more brain capacity to begin with. Yet these findings certainly raise the possibility that the old maxim “use it or lose it” may contain more than a grain of truth (Cracchiolo et al., 2007; Wilson et al., 2007).



Study and Review in MyPsychLab

Assess Your Knowledge

FACT or FICTION?

1. Long-term potentiation appears to play a key role in learning. True / False
2. The hippocampus is the site of the engram. True / False
3. Memory recovery from amnesia is usually quite sudden. True / False
4. Explicit and implicit memory are controlled by the same brain structures. True / False
5. Alzheimer’s disease is only one cause of dementia. True / False

Answers: 1. T (p. 268); 2. F (p. 268); 3. F (p. 268); 4. F (p. 269); 5. T (p. 271)

The Development of Memory: Acquiring a Personal History

7.11 Identify how children’s memory abilities change with age.

How early can children remember, and what do they remember? The answer depends on what kind of memory we’re discussing. In one sense, we can remember information even before we’re born. That’s because fetuses display *habituation*—a decrease in attention to familiar stimuli. As we learned in Chapter 6, fetuses as young as 32 weeks exhibit a decline in their reactions to vibratory stimulators over time. Habituation is a form of implicit memory—to interpret a stimulus as familiar, we need to recall we’ve experienced it before. It’s a far cry from explicitly recalling the words to a song or remembering what we wore to our last birthday party, but it’s still a form of remembering.

Memory over Time

Memory changes as we age, but there's considerable continuity over the course of development. On average, infants have worse memories than do children, who have worse memories than adults, and young adults have better memories than older adults. But the same basic processes operate across the life span. For example, infants display a serial position curve just as adults do (Cornell & Bergstrom, 1983; Gulya et al., 2001). Nevertheless, the span of memory and the ability to use strategies increase dramatically across the infant, toddler, preschool, and elementary school years.

Over time, children's memories become increasingly sophisticated. Several factors explain why. First, children's memory spans increase with age (Pascual-Leone, 1989). In fact, their Magic Number doesn't become seven plus or minus two until age 12 or so. If we ask a 3-year-old to remember a string of letters or numbers, she'll remember only about three on average. A 5-year-old will remember about four. By age 9, children are getting close to the adult's Magic Number, remembering six items on average.

Is this increase in span a result of better use of strategies, like rehearsal? That's certainly part of the story (Flavell, Beach, & Chinsky, 1966; McGilly & Siegler, 1989), but there's a large physical maturational component, too. So in an odd turn of events, shoe size is actually more highly correlated with memory span in children than with either age or intelligence. Nevertheless, we can assure you that this correlation isn't a causal one! Because different children grow at different rates, this correlation reflects a biological maturity component to memory span, for which variables like shoe size and height are the best predictors.

Second, our conceptual understanding increases with age. This fact is important because our ability to chunk related items and store memories in meaningful ways depends on our knowledge of the world. For example, without knowing that "CIA" stands for Central Intelligence Agency, children can't chunk the letters *C*, *I*, and *A* into one unit.

Third, over time, children develop enhanced **meta-memory** skills: knowledge about their memory abilities and limitations. These skills help children to identify when they need to use strategies to improve their memories, as well as which strategies work best (Schneider, 2008; Schneider & Bjorklund, 1998; Weinert, 1989; Zabrocky & Ratner, 1986). If we show a 4-year-old ten pictures and ask her how many she thinks she can remember, she'll probably tell you with supreme confidence that she can remember all ten. She can't. Children at this age don't appreciate their own memory limitations and overestimate their capacities as a result. Older children, who actually remember *more* than younger children do, estimate they'll remember *less*. As a result, they're more accurate in their ability to gauge their recall (Flavell, Friedrichs, & Hoyt, 1970).

Infants' Implicit Memory: Talking with Their Feet

Carolyn Rovee-Collier developed an innovative technique to study infants' implicit memory. Her research capitalizes on the fact that we can operantly condition (see Chapter 6) infants to perform specific behaviors. Rovee-Collier placed infants in a crib with a mobile positioned over their heads. She first watched their behaviors for a few minutes to assess their activity levels in a "baseline" condition. Then, she took a ribbon tied to the mobile and attached it to the infant's ankle. The next time the infant kicked her foot, she was in for a pleasant surprise: The mobile shook and jiggled in response. Infants find the motion of the mobile inherently reinforcing. Because the movement is dependent on infants' behavior, they quickly become conditioned to kick their legs to get the mobile moving.

Once she conditioned infants to kick their legs in response to the mobile, Rovee-Collier sent them home. Then, after a delay—of a day, a week, or even a month—she brought them back to the lab and placed them in the crib again. This time, the mobile wasn't attached to the infant's leg, so there was no reinforcement. The question was: Would infants show an increased kicking rate in response to seeing the mobile? If so, it would imply that they remembered the conditioning experience.

◀ CORRELATION VS. CAUSATION

Can we be sure that A causes B?



Carolyn Rovee-Collier and other researchers have used mobiles to study infants' implicit memory. Although infants can't tell you they remember the mobiles, their memories "vote with their feet."

meta-memory

knowledge about our own memory abilities and limitations



? Some people claim to remember their own birth. What well-established psychological phenomenon renders this claim extremely implausible? (See answer upside down on bottom of page.)

infantile amnesia

inability of adults to remember personal experiences that took place before an early age

EXTRAORDINARY CLAIMS ►

Is the evidence as strong as the claim?

Rovee-Collier (1993) found that children as young as two months retained a memory of this experience, although they forgot it after just a few days. Nevertheless, their span of recall increased quickly. Three-month-olds could remember the conditioning for over a week, and six-month-olds for over two weeks. Infants' memories of the experience were surprisingly specific. If researchers modified even a few elements of the mobile or changed the pattern of the crib liner ever so slightly, infants didn't seem to recognize the mobile: Their kicking rate returned to baseline.

from inquiry to understanding

WHY CAN'T WE REMEMBER THE FIRST FEW YEARS OF OUR LIVES?

Take a brief break from reading this chapter, and try to recall your earliest memory. What was it, and how old were you? Most students say their earliest memory falls somewhere between 3 and 5 years of age. **Infantile amnesia** is the inability of adults to retrieve accurate memories before an early age (Malinoski, Lynn, & Sivec, 1998; Wetzler & Sweeney, 1986).

Few, if any, of us correctly recall events before 2 or 3 years of age, the lowest cutoff for infantile amnesia (West & Bauer, 1999; Winograd & Killinger, 1983), although there are differences among people in the boundary of this amnesia (Peterson, Warren, & Short, 2011). Memories before that age just aren't trustworthy. So if you have a distinct memory of something that happened at age 1 or before, it's almost certainly either a false memory or a true memory of something that happened several years later.

Recent research suggests that culture may shape the age and content of our first memories. European Americans report earlier first memories than do people from Taiwan. Moreover, European Americans' earliest memories more often focus on themselves, whereas Taiwanese's earliest memories more often focus on others (Wang, 2006). These findings dovetail with research we'll describe later in the text (see Chapter 10) showing that European-American cultures tend to be individually oriented, whereas many Asian cultures tend to be other-oriented (Lehman, Chiu, & Schaller, 2004).

Unfortunately, proponents of some fringe psychological treatments have largely ignored the scientific evidence concerning infantile amnesia. Many advocates of hypnotic age regression, which Nadean Cool's therapist used, claim to be able to retrieve memories from well before age 2, sometimes even before birth (Nash, 1987). At least one therapist even tried to recover his female client's memory of being trapped as an egg in her mother's fallopian tube prior to fertilization (*Frontline*, 1995). Similarly, proponents of the school of Scientology, popular among many Hollywood celebrities, believe that long-buried memories of negative statements overheard by fetuses, embryos, and even zygotes can be reactivated in adulthood, especially under stress. These memories, Scientologists claim, can trigger low self-esteem and other psychological problems (Carroll, 2003; Gardner, 1958). For example, if a fetus overhears her mother say "I hate you" during a bitter argument with her husband, the grown adult may later misinterpret this statement as referring to her rather than to her father. Fortunately, for both fetuses and adults, there's no evidence for this extraordinary claim. Fetuses can't accurately make out most sentences they hear from outside the womb (Smith et al., 2003), let alone remember them decades later.

No one knows for sure why the first few years of our lives are lost to us forever, but psychological science offers several promising leads (Bauer, 2006). The hippocampus, which as we learned plays a key role in long-term memory, especially episodic memory, is only partially developed in infancy (Mishkin, Malamut, & Bachevalier, 1984; Schacter & Moscovitch, 1984;

to be genuine.
prior to age two and a half or three are extremely unlikely.
Answer: Infantile amnesia; research suggests that memories

Zola, 1997). So before age 2 or so, we may not possess the brain architecture needed to retain memories of events (Josselyn & Frankland, 2012; see Chapter 10).

Also, as infants, we possess little or no concept of self (Fivush, 1988; Howe & Courage, 1993). Before about 18 months of age, infants can't recognize themselves in mirrors (Lewis, Brooks-Gunn, & Jaskir, 1985). Without a well-developed schema of self, infants may not be able to encode or store memories of their experiences in a meaningful fashion. So these experiences will never be remembered.



Research suggests that other than humans, gorillas, orangutans, dolphins, and perhaps elephants are among the handful of species that exhibit mirror self-recognition—often regarded as one important indicator of the presence of a self-concept (Plotnik, de Waal, & Reiss, 2006). Here a baby reacts to his mirror image.

Assess Your Knowledge

FACT or FICTION?

1. Most young children underestimate their memory abilities. True / False
2. Children as young as two months have implicit memories of their experiences. True / False
3. Most adults can accurately recall events that took place before they were 3 years old. True / False
4. One explanation for infantile amnesia is that the hippocampus is only partially developed in infancy. True / False

Answers: 1. F (p. 273); 2. T (p. 274); 3. F (p. 274); 4. T (p. 274)



Study and Review in MyPsychLab

False Memories: When Good Memory Goes Bad

7.12 Identify factors that influence people's susceptibility to false memories and memory errors.

7.13 Describe some of the real-world implications of false memories and memory errors.

We generally trust our memories to provide us with an accurate recounting of our past. In many cases, our memories do the job well enough. Over the past few decades, however, researchers have shown that our memories can be more fallible than any of us could have imagined. Moreover, we're often far more confident of our recollections of events than we should be.

False Memories

At first blush, our everyday experience strongly suggests that we can safely rely on our memories, because many of our recollections seem to be as crisp as scenes from a movie. Do you remember where you were and what you were doing when you heard about the terrorist attacks on September 11, 2001? Most Americans say yes, and many say that, even today, they can "relive" those frightening moments with astonishing clarity. Many older Americans report equally vivid memories of the assassination of President John F. Kennedy on November 22, 1963. Powerful memories of the attempted assassination of President Ronald Reagan (Pillemer, 1984), the explosion of the space shuttle *Challenger* (McCloskey, Wible, & Cohen, 1988), and the deaths of Princess Diana (Krackow, Lynn, & Payne, 2005–2006) and singer Michael Jackson are other examples.

FLASHBULB MEMORIES. It's no wonder that Roger Brown and James Kulik (1977) referred to these recollections as **flashbulb memories**, emotional memories that seem so vivid that people appear to recount them in remarkable, even photographic, detail.

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flashbulb memory

emotional memory that is extraordinarily vivid and detailed