4 NEURAL BASIS OF LEARNING AND MEMORY

KEY KNOWLEDGE

- neural plasticity and changes to connections between neurons (including long-term potentiation and long-term depression) as the fundamental mechanisms of memory formation that leads to learning
- the role of neurotransmitters and neurohormones in the neural basis of memory and learning (including the role of glutamate in synaptic plasticity and the role of adrenaline in the consolidation of emotionally arousing experiences).

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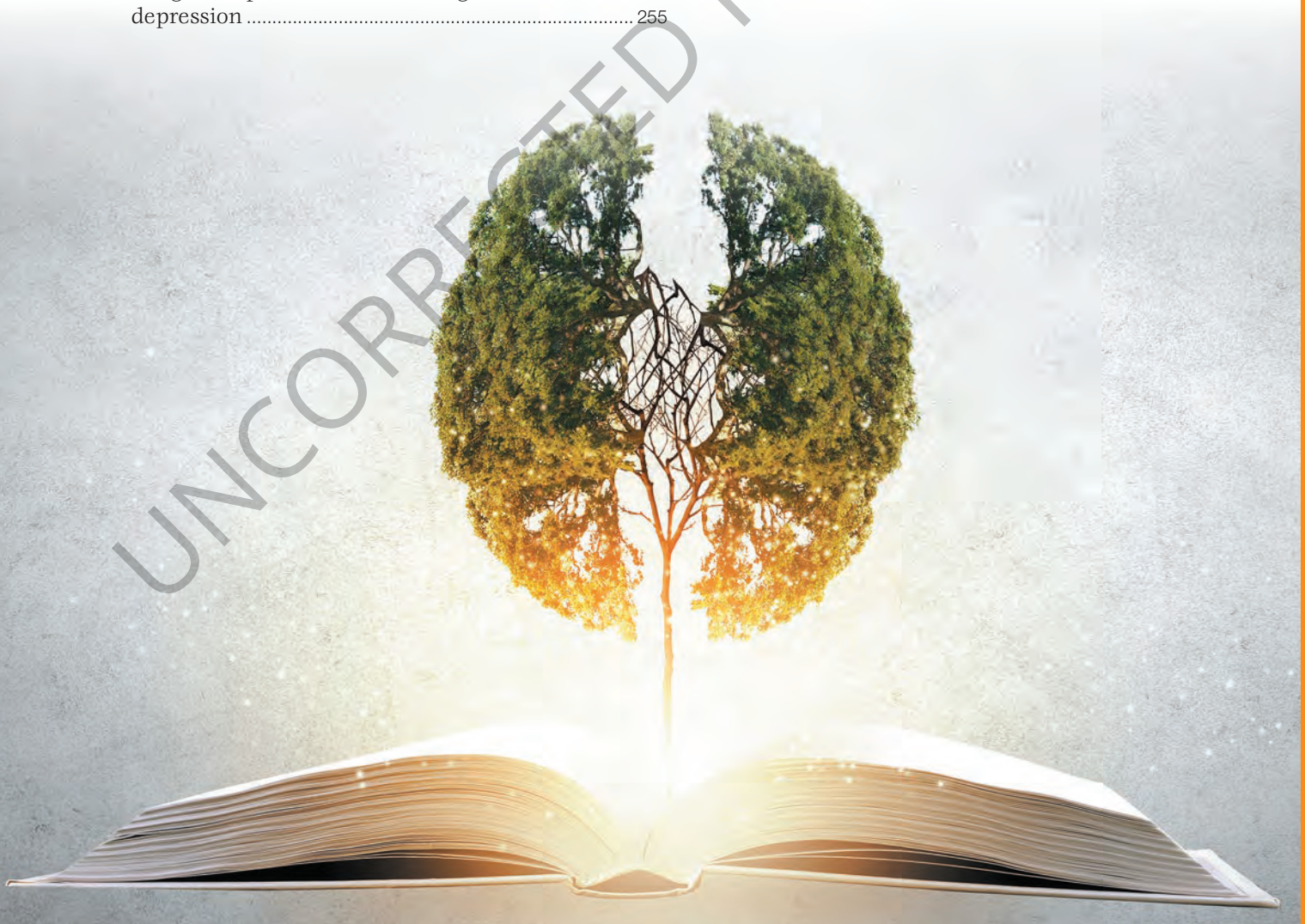
CHAPTER CONTENT



Can you think of something you do that you did not learn? It's a difficult task because learning is involved in nearly all our behaviours. Except for a range of physiological responses that are involuntary and normally occur automatically, such as breathing, digesting food, secreting hormones and blinking, most of what you do each day depends to a large degree on learning. For example, behaviours such as brushing your teeth, tuning in to your favourite television program, texting a friend and undertaking the VCE all depend on learning in a significant way. Your attitudes, values, beliefs, opinions, interests and decisions all involve learning. Many of our emotions are also learned or influenced significantly by learning. Learning is such an integral part of daily living that without the ability to learn from an early age, people would be unable to live independently and would need constant care in order to survive.

Next, imagine for a moment what life would be like without your memory. You would have no recollection of what happened to you 2 seconds ago, 10 minutes ago or even 10 years ago. Without memory, every moment would be a new experience. Each person you met would be a stranger and each task you tackled would be a new challenge. Even the most basic tasks that most of us take for granted, such as tying a shoelace or walking the dog, would be difficult because we would have no memory of how to do them.

Imagine the effect on your social life. You would not be able to hold a conversation and you would have no friends because you would have no memory of ever having met them or knowing anything about them from one encounter to the next. Without memory you would have no self-concept or true sense of yourself as an individual. Our self-concept develops from the many experiences we have during our lives. With no recollection of these experiences we would have no basis for developing an understanding of 'who I am'. Each time you looked in the mirror you would be confronted by a complete stranger.

In this sense, it is memory that provides meaning to our lives by integrating the past and the present, and enabling us to think about the future.

Learning is change that occurs through experience. Memory is very closely related to learning. The relationship is so close that learning and memory are often described as inseparable. Learning is the acquisition of skill or knowledge, while memory is the expression of what you have acquired. The existence of memory indicates that learning has occurred. If no learning occurs there is nothing to remember. Without memory, learning would not be possible because we need the capability to retain what we have learned. Nor would learning have any value if we could not remember — we usually learn with the understanding that at some future time we will be able to recall what we learned.

Memory is essentially the outcome of learning and enables knowledge and skills acquired through experience to be stored in the brain and retrieved when needed. The close relationship between learning and memory is evident not only from a psychological perspective, but also biologically as they both involve and are influenced by many of the same neural mechanisms and processes. All memory involves neurological changes that occur as a result of learning. Memory is not a recorded 'snapshot' of an event but a neurological representation of the event. From a biological perspective, learning may be viewed as the capability of modifying information already stored in memory based on new sensory input or learning experiences. Since memory is dependent on some kind of prior experience, the first step in memory formation is learning, which occurs when our sensory systems send information to the brain.

In this chapter we examine the neural basis of learning and memory, focusing on the brain's plasticity and changes to connections between neurons that enable learning and memory to take place and demonstrate that learning and memory are actually inseparable.

Figure 4.1 Learning and memory are inseparable. If no learning occurs there is nothing to remember, and to learn requires a capability to remember what will be learned.

NEURAL PLASTICITY AND CHANGES TO CONNECTIONS BETWEEN NEURONS

The human brain typically follows a predictable pattern of growth and development, with different structures and abilities progressing at different rates and maturating at different points in the lifespan. Although our genes ensure that the basic structure and organisation of our brain are established well before birth, our brain continues to mature and change long after birth. It is not a rigidly fixed organ. Nor are the neural circuits and pathways extending within and between different areas of our brain 'hardwired' like a computer or other human-made electronic device.

Neurons are soft, flexible living cells. They can change in size, shape and function. They can also change their connections with other neurons and their patterns of connections. These types of changes are influenced by the interaction of biological processes that are genetically determined and by experiences in everyday life.

From birth through to the end of life, neurons and the connections between them change in response to our experiences. They change to represent and store this information so that we can learn and remember. This fundamental and very important ability to change is referred to as neural plasticity, neuroplasticity, or simply plasticity.

Neural plasticity

Neural plasticity is the ability of the brain's neural structure or function to be changed by experience throughout the lifespan. This may involve a single neuron, a pair of neighbouring neurons or entire networks of neurons. The term plasticity is used because 'plastic' originally meant flexible, pliable or malleable. This property of the brain provides the physiological basis of learning and memory. It makes learning and memory possible, provides the brain with a way of being continually responsive to environmental input, and thereby assists us in adapting to life's ever-changing circumstances.

The brain's plasticity is a feature that persists from embryonic development through to and including old age. Lifelong plasticity accounts for many of the learning experiences we have throughout life, such as learning language as a child, learning to play a musical instrument as an adolescent, learning new job skills as an adult, learning how to use digital media at an older age, and so on. Our genes govern the overall architecture of our brain, but experience guides, sustains and maintains the details.

If a monkey is trained to push a lever with a finger several thousand times a day, the brain tissue which controls that finger changes to reflect the experience. Human brains function in a similar way. Whether learning to use a computer keyboard or to ride a skateboard, we will perform with increasing skill as our brain incorporates the learning within its neural structure. The neural activity underlying this process occurs in a systematic way and not haphazardly (Breedlove, Watson & Rosenzweig 2010; Myers, 2007).

Although some parts of the brain, such as the sensory and motor areas in the cerebral cortex, have a higher level of plasticity than others, it is unclear as to whether all brain parts have plasticity. However, the brain of a developing individual is even more plastic than that of an adult, particularly at specific times in development when it seems that the brain is more responsive to certain types of experiences. This is one reason why young children tend to learn a new language more quickly than do adults. Similarly, infants tend to recover more quickly from brain damage than adults due to the greater plasticity of their brain.

Figure 4.2 Lifelong plasticity accounts for many of the learning experiences we have throughout life, such as learning how to use digital media in young or old age.

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Weblinks

- TEDx Talk on learning, memory and neural plasticity 14m 24s
- TEDx Talk: Norman Doidge on neuroplasticity 44m 49s

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There also seems to be a relationship between the type of experience we have and the extent of the structural change that takes place in the brain. Generally, the more complex the experience in terms of the variety of sensory input, the more distinctive the structural change that will occur in neural tissue involved in that experience. This seems to be the case for both children and adults (Centre for Educational Research and Development [CERI], 2007; Kolb & Whishaw, 2014).

Changes to connections between neurons

Neural plasticity is evident in physical changes that take place at synapses where neurotransmission occurs and multiple neurons interconnect to form neural pathways. At the level of the synapse, neural plasticity is commonly called synaptic plasticity.

Synaptic plasticity refers to the ability of the synapse to change over time. For example, change may occur through growth or formation of new synaptic connections that strengthen the synapse or change may occur through disuse of synaptic connections that weaken or eliminate the synapse. Synaptic plasticity enables a flexible, efficient and effectively functioning nervous system. It is also the biological basis of learning and memory.

As we learn through the constant stream of new experiences in everyday life, our brain modifies its neural connections and pathways, thereby actually changing its structure and function by 'rewiring' itself. Existing connections between neurons can reorganise, and new networks or pathways can form and strengthen through use during the learning (and memory formation) process, thus making communication across a connection and along a pathway easier the next time. Furthermore, the brain can reorganise and reassign its neural connections, and pathways based on which parts of it are overused or underused. The result is a structure constantly remodelled by experience.

Canadian psychologist Donald Hebb is credited with the idea that learning involves the establishment and strengthening of neural connections at the synapse. For example, learning a list of new spelling words, to use a pogo stick, to play a harmonica or any other task will establish new neural connections, and regular practice of the task will strengthen these connections with the result that you get better at the task, become more efficient and make fewer mistakes.

Some 70 years ago, Hebb proposed that learning results in the creation of *cell assemblies* — interconnected groups of neurons that form networks or pathways. Neurons in a network send messages to other neurons within the network, but messages from one network may also be sent to other networks and small networks may also organise into bigger networks. Consequently, the same neurons may be involved in learning different things or in producing different patterns of behaviour, depending on which combination of neurons is active.

According to Hebb (1949), when neurotransmitter is repeatedly sent across the synaptic gap, presynaptic and postsynaptic neurons are repeatedly activated at the same time. When a presynaptic and a postsynaptic neuron are active at the same time, this changes the structure or chemistry of the synapse, strengthening the connections between these two neurons at the synapse. When the synaptic connection is strengthened, this makes them more likely to fire together again and to transmit their signals more forcibly and efficiently in the future. Conversely, not firing together — for example, through disuse — weakens the connections between neurons and also makes them less likely to fire together at the same time in the future.

Hebb's explanation of changes to synaptic connections between neurons during learning is known as *Hebb's rule* or *Hebbian learning* and is often summarised as 'neurons that fire together, wire together'. Subsequent research in the 1970s on neurological processes during learning found that the synaptic changes underlying the formation of cell assemblies described by Hebb were also involved in the formation and storage of new memories. In particular, the discovery of long-term potentiation provided evidence in support of Hebb's rule (Kandel, 2001).

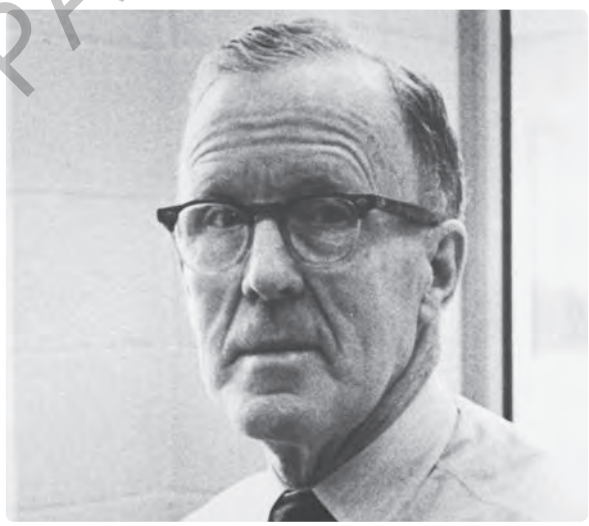

Figure 4.3 Canadian psychologist Donald Hebb (1904–1985) first proposed that the strength of a connection between neurons is determined by the neural activity of adjacent pre- and postsynaptic neurons. According to Hebb (1949 p. 62), 'when an axon of cell A is near enough to excite cell B or repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased'. This theory has become known as Hebb's rule and is often summarised more simply as *neurons that fire together, wire together*.

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Animation explaining Hebb's rule 1m 02s

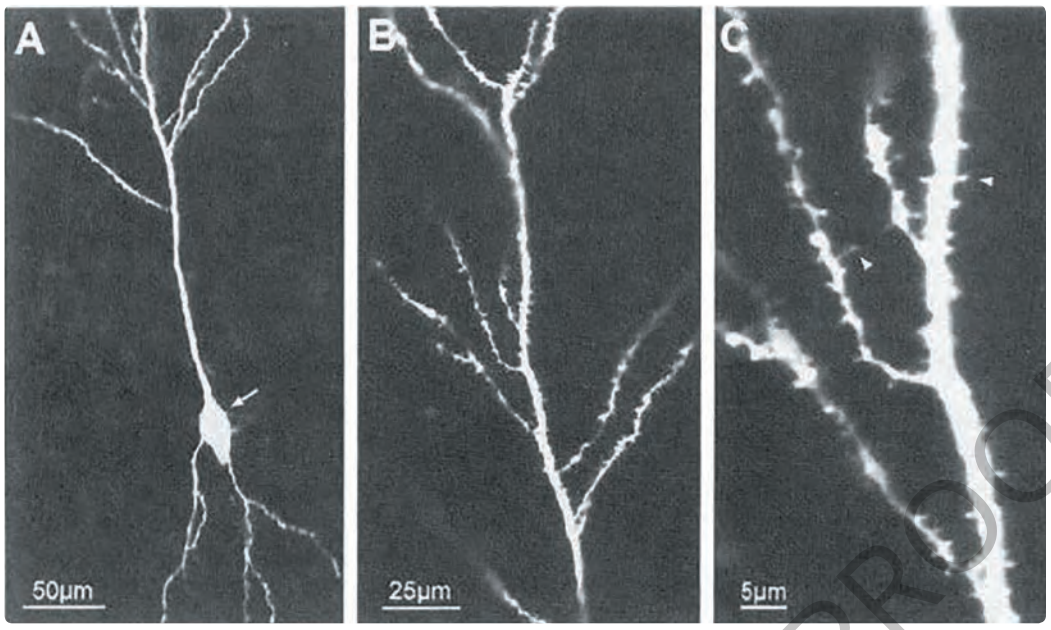

Figure 4.4 Change to connections on a postsynaptic neuron in the hippocampus of a laboratory rat when learning and forming a memory of that learning. A: the arrow is pointing to the soma. B: branches that have grown on a dendrite. C.: the dendritic branches are studded with numerous dendritic spines that have formed.

Long-term potentiation and long-term depression

Long-term potentiation and long-term depression are enduring (long-lasting) changes in synaptic strength that are brought about by specific patterns of activity at the synapse. These *activity-dependent* changes are thought to play a critical role in learning and subsequent memory formation. Both have been observed primarily in brain areas involved in learning and memory.

Long-term potentiation (LTP) refers to the long-lasting strengthening of synaptic connections, resulting in enhanced or more effective synaptic transmission. Basically, the effect of LTP is to improve the ability of two neurons — a presynaptic and a postsynaptic neuron — to communicate with one another at the synapse (Bliss & Lomo, 1973).

LTP strengthens synaptic connections in a way that enables postsynaptic neurons to be more easily activated. The postsynaptic neurons become more and more responsive to the presynaptic neurons as a consequence of repeated stimulation by neurotransmitters. The more that the connection is activated, the more the connection is strengthened. The more the connection is strengthened, the more the relevant neural pathway is strengthened, increasing the efficiency in transferring information along the pathway and decreasing the likelihood that what has been learned will be forgotten (and thereby enhancing memory storage of the information). In addition, the more we use the information being remembered, the more the LTP process strengthens the pathway, making it easier to retrieve that information. This suggests that simple repetitive 'rote learning' when studying for an exam is worthwhile (but not necessarily more effective than other study methods). With LTP, there also appear to be changes in the presynaptic neuron. For example, the terminal buttons on the neurons involved in LTP release more glutamate after the potentiation has been created (Thompson, 2000).

LTP was first reported in 1973 after it was observed in the brains (hippocampus) of anesthetised rabbits in a laboratory in Norway. It is the same kind of mechanism that Hebb had imagined 25 years earlier when he proposed that learning results from a strengthening of synaptic connections between neurons that fire together. The discovery of LTP confirmed Hebb's rule and helps explain in biological terms why 'neurons that fire together, wire together'.

Long-term depression (LTD) is the long-lasting decrease in the strength of synaptic transmission (which is the opposite of LTP). This results from lack of stimulation of pre- and postsynaptic neurons or prolonged low level stimulation. Basically, a postsynaptic neuron becomes less responsive to the neurotransmitter released by a presynaptic neuron and the effect is to weaken the synaptic connection and therefore weaken or even silence communication at the synapse (Bliss & Cooke, 2011).

LTD was discovered in the cortex of the cerebellum by Japanese researchers in 1981, then later found to

also occur in the hippocampus and elsewhere in the CNS (Ito & Kano, 1982; Ito, 1989).

It is believed that LTD may be just as important for learning and memory as LTP. The weakening or elimination of unused synapses through LTD may prune unimportant or unwanted connections, leaving only the important connections that have been strengthened through repeated use by LTP. The process occurs as if the rule 'use it or lose it' is being followed. LTD may, for example, enable old memories or unused connections and pathways for previously learned information or skills to be cleared out. LTD may be what allows us to correct our thinking when solving a problem, or to adjust our movements when learning how to serve in tennis or ride a surfboard. It may also provide the basis of blocking or erasing unwanted, inappropriate or incorrect thoughts, feelings and behaviours.

Although LTP and LTD have opposite outcomes in that they result in persistent increased vs decreased synaptic excitability and one increases neurotransmitter release in presynaptic neurons and the other does not, there are a number of similarities. For instance:

- both are activity dependent i.e. more or less activity
- both involve glutamate
- both occur at glutamate synapses
- both involve changes in excitability
- both are long-lasting effects
- both are forms of long-lasting neural plasticity.

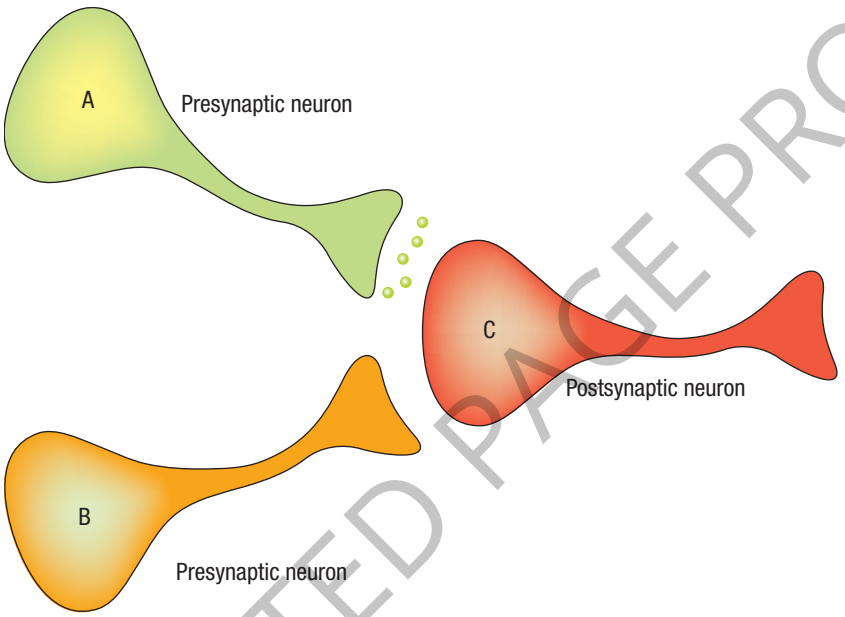

Figure 4.5 Long-term potentiation. The synapses between neuron A and neuron C and between neuron B and neuron C are initially weak. If neuron A fires and neuron C is activated immediately, and this occurs repeatedly for a sufficient number of times, neuron C will become more responsive to A than it was initially. This means that C will be more prepared to receive A's message (neurotransmitter) than B's message. In addition, the simultaneous activity between neurons A and C will grow and strengthen this synapse.

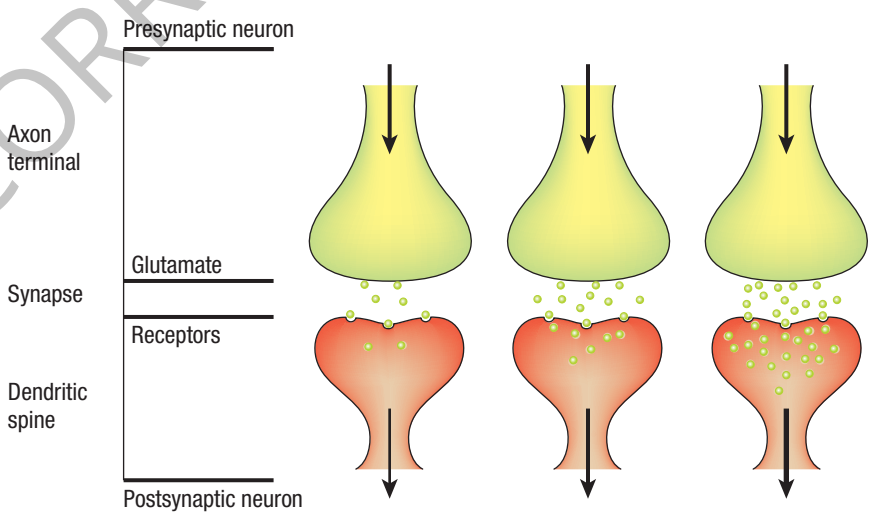

Figure 4.6 With LTP, there is an increase in the amount of neurotransmitter released by the presynaptic neurons, thereby enhancing communcation.

Although Hebb's rule, LTP and LTD are often described with reference to a pair of neurons, this is an oversimplification and it should be kept in mind that a single neuron in the human brain may have thousands of connections with other neurons, often in extremely complex ways. For example, a memory of a single bit of information may be stored within many connections, and each connection may be involved in several different memories. Thus, multiple memories may be stored within a single neural pathway, and have multiple synaptic connections. Similarly, a single memory may involve simultaneously activating several different groups of neurons in completely different areas of the brain so that the information can be brought into conscious awareness.

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Khan Academy presentation on LTP, LTD and neural plasticity 9m 39s

BOX 4.1 Animal studies on neural plasticity

A series of experiments conducted with rats in the 1960s by American psychologist Mark Rosenzweig and his colleagues provided some of the earliest evidence that the brain can be altered when learning. Simply living in a complex environment where new learning was possible produced distinctive anatomical and chemical changes in the brains of rats.

In a typical experiment, laboratory-born rat pups of the same sex and from the same litter were randomly allocated to different environments shortly after weaning (at 25 days after birth). Within each environment, the rats had different experiences and opportunities for informal learning. The three most common environments were:

- Condition 1 — a 'standard' environmental condition in which a small group of three rats were kept in a standard laboratory cage and provided with food and water. This is the typical environment for laboratory rats.
- Condition 2 — an 'impoverished' environmental condition in which a single rat was housed in a standard laboratory cage
- Condition 3 — an 'enriched' environmental condition in which a group of ten to 12 rats were kept in a large cage containing a wide variety of stimulus objects, which were changed daily and provided opportunities for complex stimulation and informal learning.

All rats were kept in these conditions for 80 days. When their brains were dissected, those rats reared in the enriched environment were found to have developed a thicker and heavier cerebral cortex than had their littermates raised in the other two conditions, particularly the impoverished environment rats. Significant changes were also found to occur at the neuronal level, particularly at the synapse. The brains of rats reared in the enriched environment had larger neurons with longer and bushier dendrites, existing synapses were bigger and new synapses had formed. In addition, there was evidence of heightened neurotransmitter activity.

In later experiments, the researchers found that spending shorter periods in the enriched condition could produce similar changes in the cerebral cortex, and that the brains of both young and adult rats changed, although changes in the young were more pronounced than those of the adults. Furthermore, these changes occurred even when rats were not placed in the differing environments until well into adulthood (Rosenzweig, Breedlove & Leiman, 2002).

Other researchers who replicated or substantially varied Rosenzweig's experiments have obtained similar results. American psychologists Bryan Kolb and Ian Whishaw (1998) analysed the results of these types of studies and reported that the weight of rats' brains following exposure to an enriched environment, and therefore opportunities for new experiences, can increase by up to 10% and the number of synapses can increase by as much as 20%. These neural changes may provide a greater number and variety of connections in the brain, thereby increasing the brain's ability to effectively deal with a more cognitively demanding and complicated environment. For instance, there is research evidence that rats raised in complex environments are much better than control group animals in solving various maze-learning tasks (Banich, 2004).

Enrichment and impoverishment studies have been carried out with many other species, including monkeys, cats, birds, honey bees and even fruit flies. In all cases, enriched environments are associated with measurable changes in the brain (Kolb & Whishaw, 2014).

Can the conclusions drawn from studies on rats, monkeys and other animals be applied to human brains? Obviously, for ethical reasons, researchers cannot conduct experiments on the effects of enriched or impoverished environments on human brain tissue as they can with animals. However, there is a growing body of evidence from studies that have used other research methods indicating that the human brain also seems to benefit from enriched, stimulating environments. For example, autopsies have been conducted to study differences in the brains of university graduates with those who had dropped out of high school. The brains of university graduates had up to 40% more synaptic connections than those of early school-leavers (Hockenbury & Hockenbury, 2006).

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Researchers have also conducted studies that have compared the life experiences of elderly people. The results suggest that a stimulating environment may delay the onset of some of the adverse effects associated with ageing. For example, in a long-term study of more than 5000 adults it was found that being involved in activities that are intellectually stimulating and challenging, both at work and at home, can reduce the risk of cognitive decline in old age, particularly earlier onset than might ordinarily occur. These activities include having a job involving a high level of complexity and a low level of routine, participating in continuing education such as a short course at a TAFE, having a habit of extensive reading, being active in social groups, and engaging in travel. Such an effect is also found when biological factors are controlled, or kept constant. For example, in a research study using sets of identical twins, one with a neurodegenerative disease (dementia) and one without, it was found that the twin with a low level of education (i.e. did not complete high school) and who tended not to be mentally active was more likely to get Alzheimer's disease (Banich, 2004).

Figure 4.7 The three conditions in Rosenzweig's 1960s experiments.

LEARNING ACTIVITY 4.1

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Word copy of table

Review questions

1. What is neural plasticity?
2. Explain the meaning of synaptic plasticity with reference to two examples of synaptic change.
3. (a) How does neural plasticity enable learning and memory?
   (b) Explain whether any learning or memory would be possible without neural plasticity.
4. Explain how learning and memory occur with reference to 'connections between neurons' and Hebb's rule.
5. To how many neural pathways might the memory of a single bit of information belong? Explain your answer.
6. Complete the following table to summarise similarities and differences between LTP and LTD.

Characteristic	LTP	LTD
Definition		
Neurotransmitter involved		
Where it occurs		
How it occurs		
Change in excitability (activation)		
How enduring		
Effect on neuronal communication		
Role in learning and memory		

7. Explain why LTP and LTD demonstrate neural or synaptic plasticity.
8. Why is LTP considered to be 'evidence' supporting Hebb's physiological explanation of learning?
9. Make a copy of Figure 4.5 (page 256) on LTP. Modify the drawing and caption to illustrate and explain LTD.
10. Briefly explain why learning and memory may be considered inseparable from:
    (a) a biological perspective
    (b) a psychological or behavioural perspective.

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LEARNING ACTIVITY 4.2

Reflection

Some psychologists who have adopted a biological perspective to the discipline describe learning and memory as processes of modifying existing neural pathways or building new neural pathways.

Considering the roles of learning and memory in shaping our identity, influencing our psychological development and supporting our adaptation to everyday life, is this a suitable description?

LEARNING ACTIVITY 4.3

Evaluation of research by Rosenzweig et al. on learning and neural plasticity

Consider the description of an experiment conducted by Rosenzweig and his colleagues in the 1960s summarised in Box 4.1 on the previous pages and answer the following questions.

1. Name the type of experimental research design.
2. Identify the operationalised independent and dependent variables.
3. Identify the experimental and control conditions (groups).
4. Why were the rats randomly allocated to different conditions?
5. Briefly state the results obtained.
6. Formulate a research hypothesis that could have been tested by the procedures used in the experiment and supported by the results obtained.
7. (a) List three synaptic changes attributed to the experimental procedure.
   (b) What three conclusions can be drawn about the relationship between experience and neural plasticity on the basis of the results obtained?
8. What are three other variables that the researchers tested in follow-up experiments?
9. To what extent can the results be applied to other animals? To people?
10. (a) What are two ethical issues of this type of research that prevent use of human participants?
    (b) Other than animal studies, how have researchers overcome ethical constraints for this type of research with people?

ROLE OF NEUROTRANSMITTERS AND NEUROHORMONES

Different neurotransmitters tend to have different roles in learning and memory. Researchers have yet to entirely isolate or explain every effect of each one. Generally, they all enable communication of the information being learned and initiate or contribute to important structural changes at the synapse that help ensure the memory is durable and long-lasting when formed. The neurotransmitter glutamate has been the target of considerable research. In this section we examine its role in synaptic plasticity that provides the neural basis of learning and memory.

Some neurohormones also have roles in learning and memory. Like neurotransmitters, **neurohormones** are chemical messengers that are manufactured by neurons and released from axon terminals. Unlike neurotransmitters, they are not released into the synaptic gap. Instead, they are released into capillaries (tiny blood vessels) where they are absorbed into the bloodstream and carried to target neurons or other cells. The effects of neurohormones can therefore be on distant cells or organs some time after their secretion, whereas neurotransmitters are released locally at a synapse and exert their effects on adjacent postsynaptic neurons within milliseconds if fast-acting (or up to minutes if slow-acting) (Thompson, 2000).

For example, the hypothalamus in the brain has neurons that produce different kinds of neurohormones. These are secreted into the blood and travel to the pituitary gland where they exert their effect. When we experience stress for a prolonged time, it is the neurohormone TRH that signals the pituitary gland to produce ACTH which then enters the bloodstream and travels down to the adrenal cortex where it stimulates secretion of cortisol and other corticosteroids.

Keep in mind that some neurotransmitters can also occur as neurohormones. In such cases, the neurotransmitter and neurohormone are essentially the same chemical substance. For example, epinephrine may be secreted by a neuron as neurotransmitter and adrenaline may be secreted by a neuron as neurohormone (as described on page 215). Consequently, neurotransmitters and neurohormones are best distinguished in terms of their *function* rather than their chemical structure. In the next section, we examine the role of adrenaline as a neurohormone in the consolidation of emotionally arousing experiences in memory.

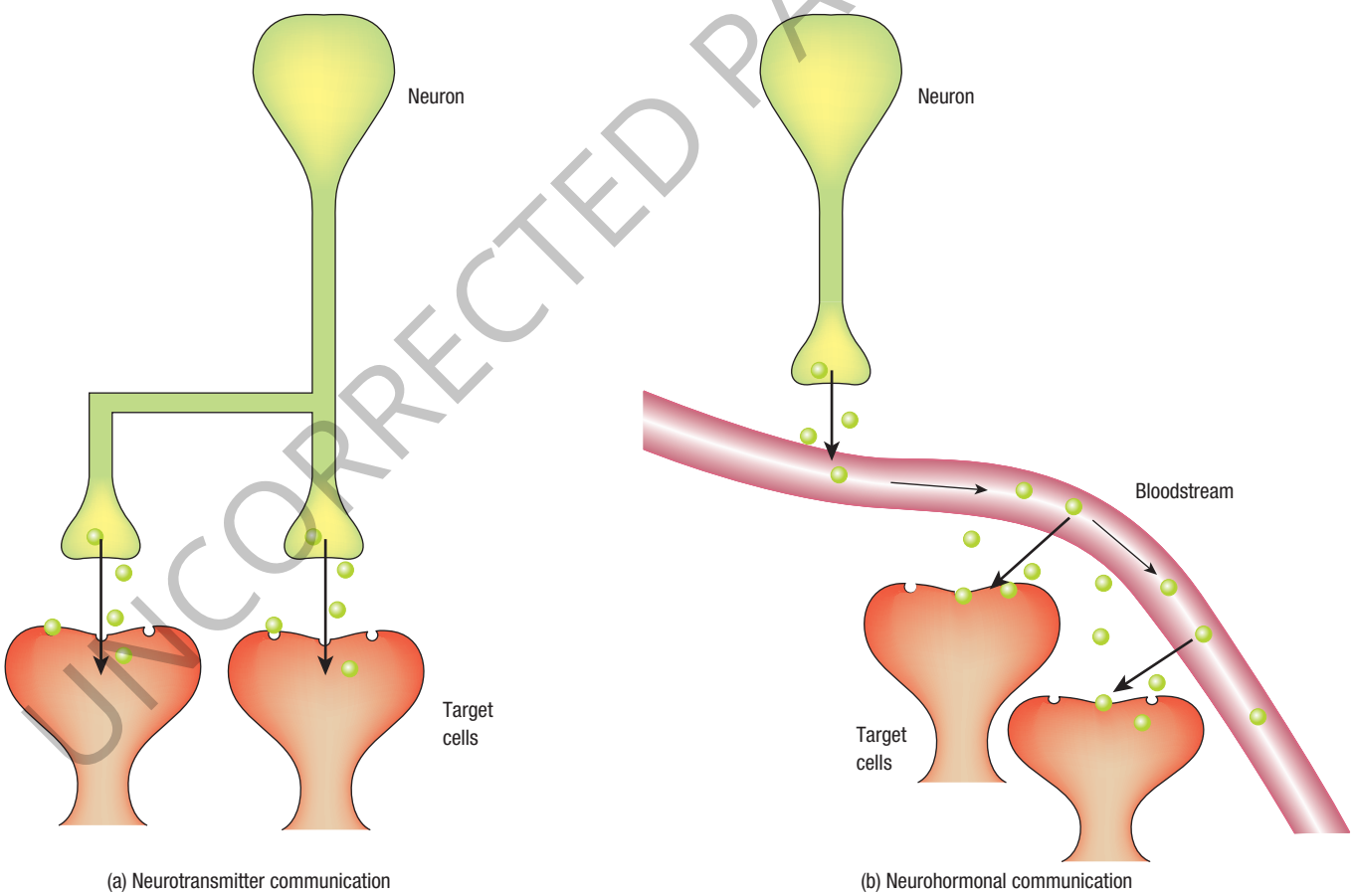

Figure 4.8 A comparison of neurotransmitter and neurohormonal communication. (a) Neurotransmitters are secreted into the synaptic gap and deliver their message to adjacent target cells, whereas (b) neurohormones are secreted into the blood for transport to target cells. Neurotransmission messages therefore travel rapidly, whereas neurohormone messages travel more slowly.

Role of glutamate in synaptic plasticity

When we learn, remember or engage in any other mental process or physical activity, neurons excite each other through the release of neurotransmitters. **Glutamate (Glu)** is the main excitatory neurotransmitter throughout the brain and enhances information transmission by making postsynaptic neurons more likely to fire.

In learning and memory, glutamate plays crucial roles in the synaptic changes that occur. In particular, it promotes the growth and strengthening of synaptic connections between neurons within a neural pathway that subsequently represents the memory of what has been learned.

LTP and LTD are important forms of synaptic plasticity that occur at synapses within several brain regions, particularly areas with significant roles in learning and memory (such as the cerebral cortex, hippocampus and cerebellum). Given glutamate's excitatory effect, it has a vital role in LTP and LTD. Generally, the more often that glutamate can excite an adjacent neuron, the more it contributes to LTP (and vice versa for LTD).

Specific types of glutamate receptors also have to be present on the dendrites of postsynaptic neurons for glutamate to have these effects. Two of these receptors are commonly called AMPA and NMDA and glutamate has to have an effect on both of them. Without these receptors at the specific sites where glutamate is received, any message carried in glutamate cannot be 'accepted' by a postsynaptic neuron (Gazzaniga, Ivry & Mungun, 2014; Zakharenko, Zablow & Siegelbaum, 2001).

Role of adrenaline in the consolidation of emotionally arousing experiences

Lasting memories are not created immediately at the time of a new experience. A period of time is required to ensure the experience becomes long-lasting when transferred to long-term memory for storage. Consolidation is the process by which this is achieved.

Consolidation

Consolidation is the biological process of making a newly formed memory stable and enduring after learning. Time is required after learning takes place to enable the new information to consolidate ('set') as a durable long-term memory. Consolidation is usually described as a process in its own right although some psychologists consider it to be part of the actual memory storage process.

New incoming information is temporarily stored in short-term memory before its transfer to long-term memory. Research evidence indicates that if consolidation is disrupted, new information may not transfer from short-term to long-term memory or will not be stored well in long-term memory if it arrives there. The outcome depends on the timing of the disruption. Consolidation appears to be a gradual process, and the information being remembered tends to be particularly vulnerable to disruption for at least 30 minutes following learning (Dudai, 2004).

The consolidation of information during transfer from short-term to long-term memory can be compared to writing your name in wet concrete. Once the concrete has set (the information has consolidated in long-term memory), your name (the information) is relatively permanently ingrained. But while it is setting (the process of consolidation), it can be interfered with (altered) or erased (completely lost).

Evidence in support of consolidation comes from studies of people who have experienced brain trauma resulting in memory failure or loss; for example, after suffering concussion or being knocked unconscious as a result of an accident, after acquiring certain diseases affecting the brain (such as encephalitis) or after receiving electroconvulsive therapy (ECT) as part of the treatment used in the more serious cases of depression. These people are frequently unable to report any memory of the events immediately before the accident or treatment, and in many instances they cannot remember anything that occurred during a period of about 30 minutes before the brain trauma (Breedlove, Rosenzweig & Watson, 2007; Squire & Kandel, 1999).

Other evidence for consolidation has come from research using animals. In one of the earliest and best known studies, researchers were interested in learning whether rats that were given ECT at various intervals after learning to run a maze would be able to remember the task they had learned.

In the 1960s, American psychologist William Hudspeth and his colleagues conducted an experiment using four groups of rats. ECT was administered to the rats in Group A immediately after they had learned the task, to Group B 20 seconds after learning, to Group C 30 minutes later and to Group D 60 minutes after learning. The results showed that consolidation of the experience occurred within about 60 minutes of the rats learning the task. None of the rats in Group A remembered the task they had learned. Those in Groups B and C showed partial retention (but Group C's retention was on average greater than that of Group B). All the rats in Group D remembered the task completely (Hudspeth, McGaugh & Thomson, 1964).

The hippocampus located deep within the brain has a crucial role in the consolidation of most of our memories. Once consolidated, memories are not

Figure 4.9 A severe blow to the head may disrupt consolidation and result in memory failure or loss. Eventually, any permanent memory loss is usually confined only to the contents of short-term memory during the time of the trauma since the information was never stored in long-term memory.

necessarily fixed. Whenever a memory is retrieved, it is open to further consolidation and has to be 're-stabilised' through the process called *reconsolidation*. If information in the original memory is changed, which is common when we rehash a memory, then the revised version is 'reconsolidated' (see Box 4.2 on page 265).

Consolidation is often described as comprising two phases — an initial rapid process for temporary storage, followed by a slower, more permanent process for long-term storage that may take days, weeks, months or years depending on such variables as the information, its storage requirements and how often the information is used (Gazzaniga, Ivry & Mungun, 2014).

Because consolidation is time-dependent, the process is exposed to various factors that can influence the strength or durability of the memory being formed during the consolidation period. One such factor involves stress hormones, particularly the adrenaline and cortisol secreted by the adrenal glands.

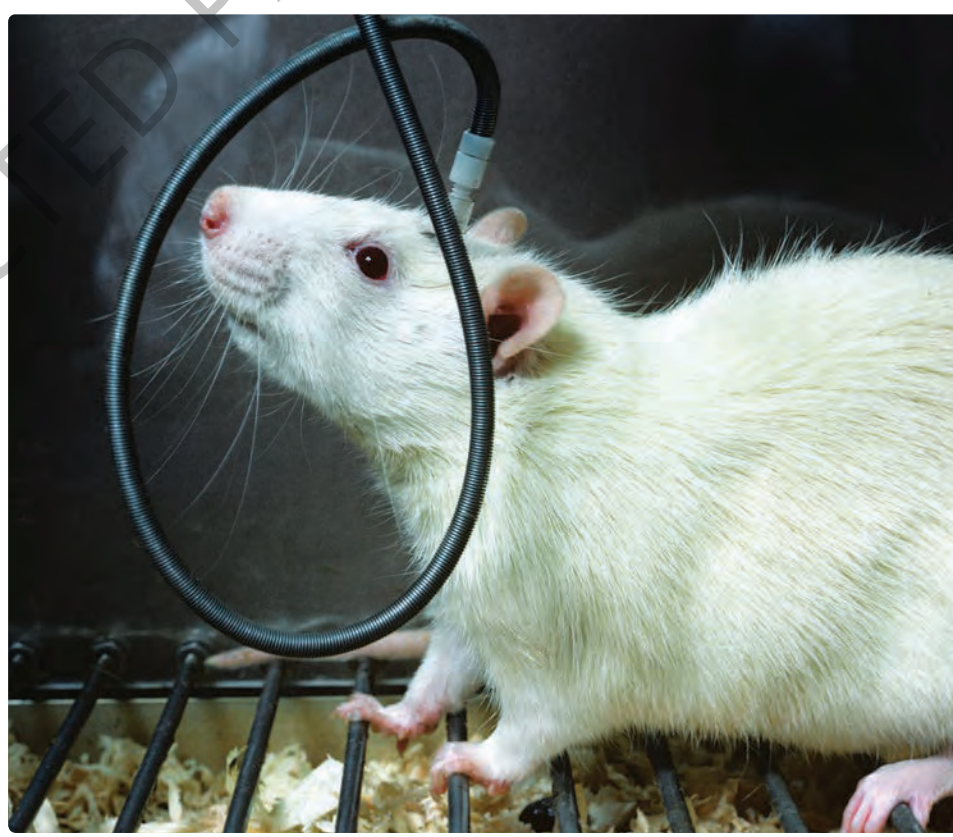

Figure 4.10 Administration of an electric shock after maze learning by rats enhanced understanding of consolidation and indicated it was a time-dependent process.

Role of adrenaline

There is considerable research evidence that adrenaline has an important role in the consolidation of specific types of memories. In particular, adrenaline can *enhance* the consolidation of long-term memories of *emotionally arousing experiences*. This means that these types of events are more likely to be well remembered (but not necessarily more accurately). In contrast, consolidation can be disrupted by brain trauma.

You probably know from personal experience that emotionally significant experiences tend to be well remembered. This is very common for both pleasant and unpleasant events.

Significant stress-inducing events are often unpleasant experiences that are emotionally arousing. They typically leave memories that are lasting, vivid and highly detailed from a personal perspective. For example, being a victim of a crime or a natural disaster will be remembered much better than the experiences of a routine day. Many years later, people can remember details about where they were, what they were doing, who they were with and what their emotional reaction was to the event. Memories of pleasant occasions that were emotionally arousing, such as a first kiss, first date, a wedding, or specific birthdays and holidays also tend to be well-retained.

When released during heightened emotional arousal, adrenaline induces the release of noradrenaline (also called norepinephrine) in the amygdala, which is located deep within the brain and has a crucial role in processing emotions. It is believed that the presence of noradrenaline during consolidation may then activate the amygdala to signal to the nearby hippocampus that details of the relevant experience are significant and its long-term storage should be strengthened. There is also interaction between the amygdala and other brain regions, so it is likely these are also involved in the consolidation process (Hamann, 2009; LeDoux, 2008; Phelps, 2004).

The exact way in which adrenaline as a neurohormone affects consolidation, either in isolation or together with other stress hormones, is not yet fully understood and is subject to further research. Nor is it fully understood how the amygdala actually interacts with the many other brain regions to which it is connected in order to strengthen the memory of an emotionally arousing experience. In addition, there is evidence that there may be an optimal level of adrenaline to enhance memory consolidation. Moderate doses seem to enhance consolidation, whereas lower or higher levels are less effective. Higher levels may even impair memory consolidation (Gazzaniga, Ivry & Mungun, 2014; Roozendaal, McEwen & Chattarji, 2009).

Research studies have also found that the strength of memories of events varies with the emotional significance of the events. The more emotionally significant the event to an individual, the longer-lasting its memory is likely to be and the more detail that will be recalled (and vice versa). For example, participants given a drug that promotes release of adrenaline when viewing emotionally arousing images will tend to have an enhanced memory of those images compared to control or placebo groups. Conversely, participants given a drug that inhibits the release of adrenaline later tend to have more trouble remembering details of the images (Cahill, et al., 1994).

Figure 4.11 Significant stress-inducing events may be pleasant or unpleasant experiences as either type can be emotionally arousing. They typically leave memories that are lasting, vivid and highly detailed.

Some psychologists have proposed that longer-lasting memories of emotionally arousing experiences, particularly for important or threatening events, has adaptive value as these memories can guide our behaviour in appropriate ways in the future. The ability to remember consequences can keep us from making the same mistakes again. For example, there is a greater chance of survival when we (and animals) remember situations that are dangerous and therefore need to be avoided. It may also be advantageous when we find ourselves in a dangerous situation and can remember exactly how we got into that situation and how we got out of it (McGaugh, 2013; McIntyre & Roozendaal, 2007).

The number of life-threatening, traumatic situations that are ordinarily encountered by most people has been greatly diminished in our modern world. Because of this, a mechanism that once may have kept us alive can become a hindrance more than a help. For example, people suffering from posttraumatic stress disorder are often haunted by memories and images of highly arousing, emotionally traumatic events to a point where it becomes unbearable. The ordeal they underwent keeps resurfacing over and over again. Each time the event resurfaces, adrenaline may be released, thereby maintaining the strength of the memory each time it is reconsolidated (Shenfield, 2013).

Adrenaline and amygdala-hippocampus interaction during emotionally arousing situations do not necessarily account for all the effects on consolidation and the enduring nature of arousal events. By their very nature, emotionally arousing events are more distinctive and unusual than everyday life events. Their novelty puts them in a special category of events quite unlike routine everyday events. They tend to be recalled more often than routine events so their neural pathways are also activated more often. These and other variables may influence their consolidation in ways that do not depend on adrenaline, noradrenaline or the amygdala (Gazzaniga, Ivry & Mungun, 2014; Roozendaal, Barsegyan & Lee, 2008).

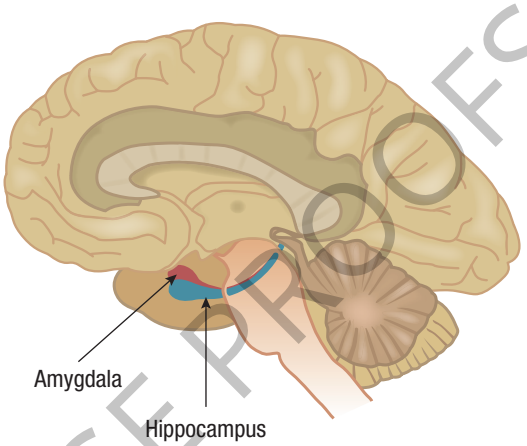

Figure 4.12 The amygdala and hippocampus are located near one another, deep within the brain, beneath the cerebral cortex.

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Joseph LeDoux presentation on the role of the amygdala in emotional memories 3m 24s

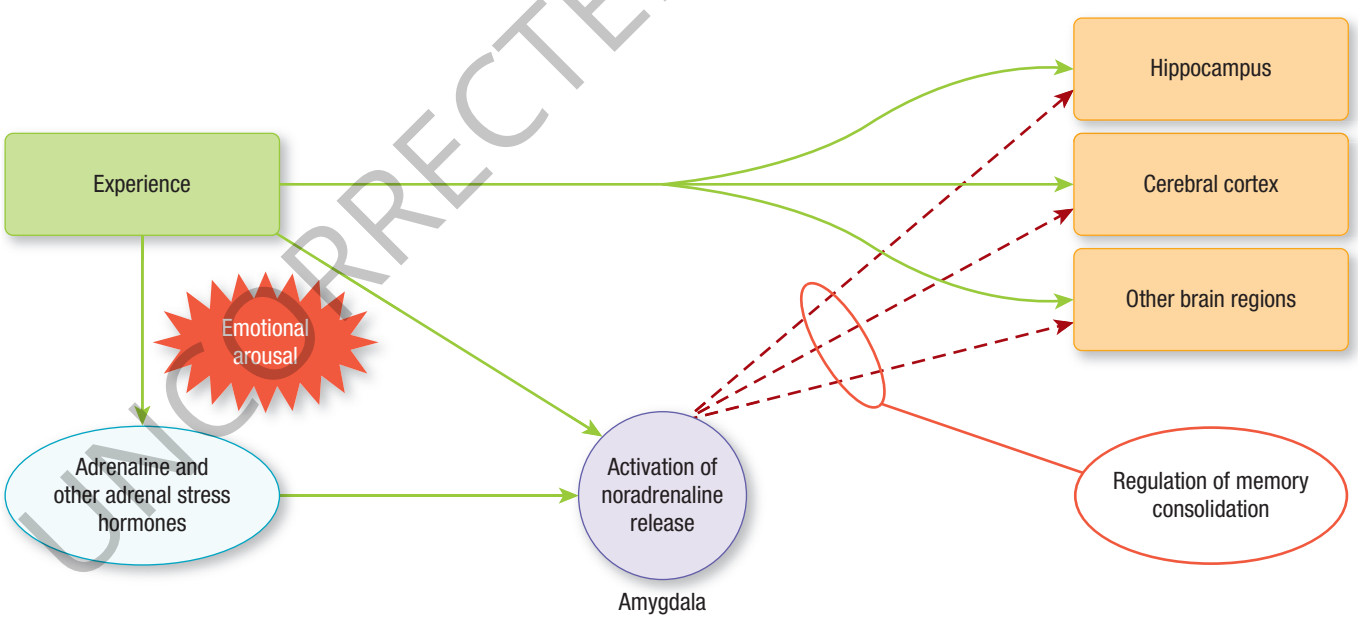


Figure 4.13 How memory consolidation of emotionally arousing experiences may occur. During periods of emotional arousal, stress hormone systems interact with the amygdala to regulate memory consolidation and storage processes occurring in other brain regions. Experiences can also be consolidated and stored in various brain regions with little or no involvement of stress hormone activation.

Source: Adapted from: McGaugh, J.L. (2006). Make mild moments memorable: add a little arousal. *Trends in Cognitive Science, 10*(8), 345–347.

BOX 4.2 Reconsolidation

It is believed that after a memory is retrieved from long-term memory it needs to be consolidated again in order to be stored back there. This process is known as *reconsolidation*.

According to American psychologists Michael Gazzaniga and Todd Heatherton (2006), evidence for reconsolidation has been obtained in studies with rats that were injected with drugs that interfered with memory storage following its retrieval. The rats were unable to reliably or accurately recall the information that was once stored in long-term memory. This suggests that memories for past events can be affected by new circumstances once they are retrieved, so that the newly reconsolidated memories may differ from their original versions.

This is similar to what would happen if you took a book out of the library and some pages were torn out before it was returned. The book that is placed back on the shelf is slightly different to the one that was taken out — the information contained in those torn-out pages is no longer available for retrieval.

The reconsolidation process is believed to repeat itself each time a memory is retrieved and placed back in storage, which may explain why our memories for events can change over time. For example, we frequently recall memories, rehash them, and integrate them with new information. In addition, we will integrate our revised, reconsolidated memories within our lifetime of stored memories.

The concept of reconsolidation has received considerable attention by researchers because it not only has implications for what it means to remember something, but it also opens up the possibility that bad memories could be altered or even erased by activating them and then interfering with reconsolidation. It seems that reconsolidation does occur, at least for some types of memories. However, much research on this memory process remains to be done.

Figure 4.14 Reconsolidation raises the possibility of intentionally altering or even erasing unwanted memories.

LEARNING ACTIVITY 4.4

Review questions

1. (a) What is a neurohormone?
(b) What are two characteristics that neurohormones have in common with neurotransmitters?
(c) What are two characteristics that distinguish neurohormones and neurotransmitters?
(d) Give an example of a substance that occurs as both a neurohormone and a neurotransmitter.

2. (a) Explain the role of glutamate in synaptic plasticity, ensuring you refer to LTP and synaptic connections.
(b) How does glutamate contribute to learning and memory?

3. (a) What is memory consolidation?
(b) Explain, with reference to consolidation, why a footballer who is knocked unconscious during a game may be unable to remember how that occurred.
(c) Jen and Sam were in a car accident. Jen was not wearing a seat belt, hit her head on the dashboard and was knocked unconscious for about a minute. Sam was wearing a seat belt and was not injured. Police arrived about half an hour after the accident and interviewed all involved.
i. Will Jen or Sam be more likely to recall how the accident occurred?
ii. Explain your answer with reference to consolidation theory.

4. (a) Formulate a research hypothesis for the experiment conducted by Hudspeth, McGaugh and Thomson (1964) which is described on page 261.
(b) Identify the operationalised independent and dependent variables.

5. (a) Why is memory consolidation vulnerable to the effects of adrenaline?
(b) What is the overall effect of adrenaline on consolidation of emotionally arousing experiences and how does this affect recall of those events?
(c) Briefly outline the role of adrenaline in the effects described in part b.
(d) What are three potential confounding variables requiring control in experiments on the role of adrenaline in the consolidation of emotionally arousing events?

6. (a) What is reconsolidation?
(b) Suggest how reconsolidation may be manipulated to change someone's memory of an event.

7. A friend who is not studying Psychology asks you to explain the neural basis of learning and memory. What five key points would you provide in your explanation?

LEARNING ACTIVITY 4.5

Reflection

The tendency of traumatic experiences to persist in memory can be debilitating rather than useful. Comment on whether this justifies the development of medications to erase unwanted or inappropriate memories, giving examples of how such medications could be used and abused.

LEARNING ACTIVITY 4.6

Role play on the neural basis of learning and memory

Working in a group of five or six, prepare a role play demonstrating synapse formation in learning and memory, the strengthening of neural connections through use, then weakening through disuse. During your presentation, name or refer to as many of each of the following concepts as possible: presynaptic neurons, postsynaptic neurons, axon terminals, dendrites, glutamate, receptors, LTP, LTD and consolidation. Ensure each anatomical feature and biological process can be clearly distinguished and understood by other members of the class.

LEARNING ACTIVITY 4.7

Flow chart on the neural basis of learning and memory

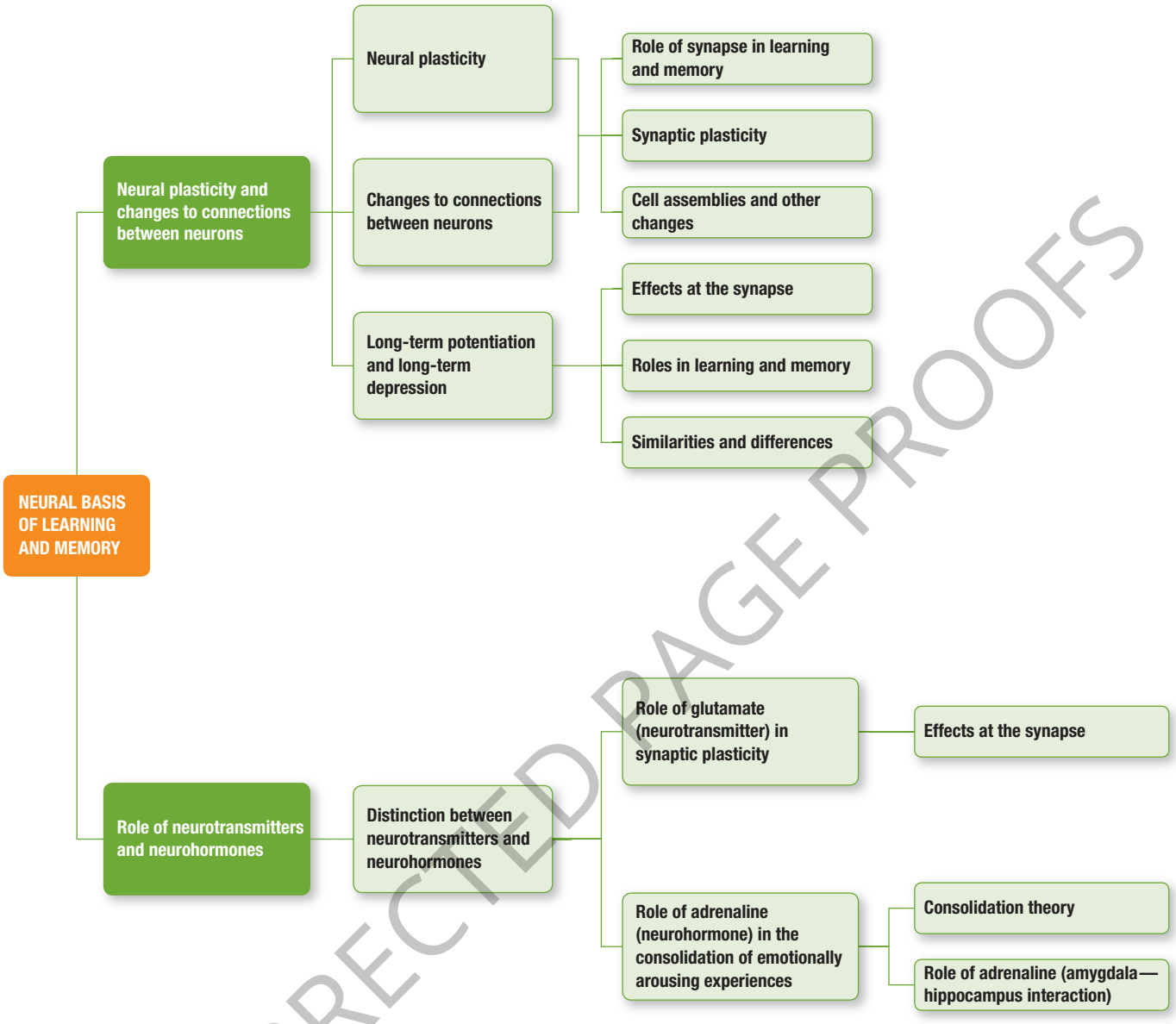
Create a flow chart that clearly outlines the neural basis of learning and memory.

In your chart, ensure that you:

- show changes at the synapse when learning and memory occurs (e.g. synapse formation)
- show the modification of a neural pathway for learning (and its associated memory)
- show the creation of a new neural pathway for learning (and its associated memory)
- demonstrate and briefly explain (e.g. in point form) the roles of neural plasticity, connections between neurons (including synapse formation and neural pathways), glutamate, long-term potentiation, long-term depression and consolidation
- demonstrate a possible role of adrenaline
- label all relevant anatomical structures and features and describe the roles of key structures and features.

In preparing your chart, base the presentation on learning of new knowledge or a specific new skill.

CHAPTER SUMMARY



KEY TERMS

adrenaline p. 263
amygdala p. 263
cell assembly (neural pathway) p. 254
consolidation p. 261
emotionally arousing p. 263
glutamate p. 261
hippocampus p. 261
learning p. 252
long-term depression (LTD) p. 255
long-term potentiation (LTP) p. 255
memory p. 252
neural plasticity p. 253
neurohormone p. 260
neuronal activation (excitability) p. 261
neurotransmitter p. 260
noradrenaline (norepinepherine) p. 263
postsynaptic neuron p. 255
presynaptic neuron p. 255
receptor p. 261
synapse p. 254
synaptic connection p. 254
synaptic plasticity p. 254

LEARNING CHECKLIST

Complete the self-assessment checklist below, using ticks and crosses to indicate your understanding of this chapter's key knowledge (a) before and (b) after you attempt the chapter test on pages 269–71. Use the 'Comments' column to add notes about your understanding.

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Word copy of checklist

Key knowledge I need to know about the neural basis of learning and memory	Self-assessment of key knowledge I understand *before* chapter test	Self-assessment of key knowledge I need to do more work on *after* chapter test	Comments
Neural plasticity and changes to connections between neurons			
Neural plasticity			
Synaptic plasticity			
Types of changes at the synapse i.e. to connections between neurons			
Long-term potentiation (including role in learning and memory)			
Long-term depression (including role in learning and memory)			
LTP and LTD similarities and differences			
Role of neurotransmitters and neurohormones			
Distinction between neurotransmitters and neurohormones			
Role of glutamate neurotransmitter in synaptic plasticity (including effects at the synapse)			
Role of adrenaline neurohormone in the consolidation of emotionally arousing experiences			
• Consolidation theory			
• Role of adrenaline			
• Amygdala-hippocampus interaction			

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Unit 4 › Area of study 1 › Topic 1

Concept screens and practice questions

CHAPTER 4 TEST

SECTION A — Multiple-choice questions

Choose the response that is **correct** or that **best answers** the question.
A correct answer scores 1, an incorrect answer scores 0.
Marks will **not** be deducted for incorrect answers.
No marks will be given if more than one answer is completed for any question.

Question 1

Where are memories most likely stored?

A. in a synapse
B. in glutamate
C. in a presynaptic neuron
D. in a postsynaptic neuron

Question 2

Neurohormones

A. are manufactured during synaptic plasticity.
B. are manufactured by the endocrine system.
C. communicate messages to dendrites.
D. communicate messages slower than neurotransmitters.

Question 3

When learning and memory occur

A. neurons change in structure and function.
B. there is an increase in the amount of synapses produced by neurons, thereby enabling them to flow more freely within a neural pathway.
C. new neurotransmitters grow and interconnect the neurons to form a pathway for the information.
D. neurons assemble in a formation that creates a neural pathway for the learning and its subsequent memory.

Question 4

Long-term potentiation is

A. the potential to learn and remember.
B. the potential to form a long-term memory.
C. the long-lasting release of glutamate at the synapse.
D. the long-lasting strengthening and efficient functioning of synaptic connections.

Question 5

If long-term potentiation is to occur between two neurons, then

A. the two neurons must be activated simultaneously.
B. the two neurons must be connected within a neural pathway.
C. the existing connection between the two neurons must be weak.
D. the existing connection between the two neurons must be strong.

Question 6

Long-term potentiation and long-term depression are _____ dependent processes.

A. time
B. activity
C. learning
D. learning and memory

Question 7

During chemical communication within the brain, neurohormones and neurotransmitters are both secreted from

A. the hypothalamus.
B. the pituitary gland.
C. synapses.
D. axon terminals.

Question 8

Which of the following statements about learning is not true?

A. Learning causes changes at the synapse.
B. Learning can create new neural pathways.
C. Learning causes weakening of synaptic connections.
D. Learning can reorganise neural pathways.

Question 9

Long-term potentiation and long-term depression cannot occur during learning unless

A. the organism also wants to remember the new information or skill.
B. the neurons involved in establishing a pathway already have synaptic connections.
C. prolonged simultaneous activity occurs in either adjacent presynaptic or postsynaptic neurons.
D. prolonged simultaneous activity occurs in both adjacent presynaptic and postsynaptic neurons.

Question 10

Simultaneous firing of two adjacent neurons makes those neurons

A. more inclined to fire together in the future.
B. less inclined to fire together in the future.
C. rearrange their connections.
D. prune connections that cannot adapt to the activity.

SECTION B

Answer **all** questions in the spaces provided. Write using blue or black pen.

Question 1 (2 marks)

Neurohormones secrete into the _____, whereas neurotransmitters secrete into the _____.

Question 2 (2 marks)

Describe the roles of the neuron and neurotransmitter as mechanisms of learning and memory.

Question 3 (4 marks)

(a) When considered from a neuronal perspective, no two human brains are identical. Explain why, with reference to neural changes associated without learning. 2 marks

(b) Explain how neural plasticity makes learning and memory possible. 2 marks

Question 4 (8 marks)

(a) What is synapse formation (or growth) and what role does it play in learning and memory? 2 marks

(b) Describe the role of glutamate and glutamate receptors in synapse formation. 2 marks

(c) Explain how long-term potentiation and long-term depression influence synapse formation. 4 marks

Question 5 (3 marks)

Explain the meaning of the phrase 'learning and memory involve the building of neural pathways in the brain', ensuring you refer to Hebb's rule.

Question 6 (6 marks)

An experiment found that rats remembered the place in an apparatus where they received electric shocks to a foot for much longer and better than rats that did not receive foot shocks.

Explain this finding with reference to the role of adrenaline in the consolidation of emotionally arousing experiences.

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The answers to the Section A multiple-choice questions are in the answer section at the end of this book and in eBookPLUS. The answers to the Section B questions are in eBookPLUS.