Unit 4 Psychology

Study Notes

Learning Mechanisms (part 1)
**What is Learning?**

*Read p155 of text*

Define **learning**

Why does it say **relatively permanent**?

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**Behaviours not dependent on learning**

*Read p156-157 of text*

List the 3 behaviours **not** dependent on learning:

Define **reflex action** and provide an example.

Define **fixed action pattern** and provide an example.

Define **maturation** and provide an example.
DP2 - mechanisms of learning:
– areas of the brain and neural pathways involved in learning, synapse formation, role of neurotransmitters
– developmental plasticity and adaptive plasticity of the brain: changes to the brain in response to learning and experience; timing of experiences
– use of imaging technologies in identification of localised changes in the brain due to learning specific tasks

Physiology of Learning

Read p157-159 of text

What does it mean to say ‘physiological basis of learning’?

Describe the complexity of the neural circuits involved in learning, thinking, feeling, behaving.

Why do researchers use a simple systems approach to research? (give eg.)

Brain areas involved in learning

read p158-159

Cerebellum

What is the cerebellum and how is it involved in learning?
(Brain areas involved in learning cont...)

Limbic System

Define the limbic system, and its key components

Summarise the role of the hippocampus in learning (take note of what it is not involved in):

Summarise the role of the amygdala in learning:

Frontal Lobe

Define the varied roles of the frontal lobe and thus its role in learning

Complete Ex 5.2 p159-160, all Q's
Neural basis of learning

The human brain follows a predictable pattern of growth and development, with different structures and abilities progressing at different rates and maturing at different points in the lifespan. Although the basic structure and organisation of our brain in terms of a cerebral cortex with hemispheres, lobes, lower brain structures and so on are irreversibly established well before birth, our brain continues to develop after birth. It is not a solid fixed organ. Nor are the neural 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 their sizes, shapes, functions, connections with other neurons and patterns of connections. These types of changes can occur at any time in the human lifespan, including old age. They are influenced by the interaction of biological processes that are genetically determined and by experiences in everyday life.

When neurons communicate with one another, they do so by sending a neurotransmitter comprising electrochemical messages across the tiny space between the axon ending of one neuron (which sends the neurotransmitter) and the dendrite of another (which receives the neurotransmitter). This tiny space is called the synaptic gap. The synaptic gap is one component of the synapse. The other two components of the synapse are the axon ending of the ‘sending’, or presynaptic neuron, and the dendrite of the ‘receiving’, or postsynaptic neuron.

The synapse is the site of communication between adjacent neurons. The act of sending a neurotransmitter across the synaptic gap actually changes the synapse. Some dendrites that receive the neurotransmitter messages from other neurons can grow longer and ‘sprout’ new branches or tips when used, whereas others are ‘pruned’ away if not used. Every day we form, or ‘grow’, millions of new synapses and millions of others disappear through disuse. At least some of these changes seem to depend on our unique experiences of that day.
As we learn through the constant stream of new experiences in everyday life, our brain modifies its neural pathways (or circuits) and neural connections within and between pathways, thereby literally changing its structure and function by ‘rewiring’ itself. Existing connections between neurons can reorganise, and new pathways can form and strengthen during the learning process, thus making communication across a connection and along a pathway easier the next time.

This ability of the brain to change is commonly called *plasticity*, a property that 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. In the next section, we examine areas of the brain involved in learning, the plasticity of the brain, and the effects of experience on the brain.

**Areas of the brain and neural pathways involved in learning**

As you are aware, different types of incoming information are processed by different areas of the brain; for example, visual information is processed by the visual cortex in the occipital lobe and auditory information is processed by the auditory cortex in the temporal lobe. Similarly, areas of the parietal lobe are involved in processing somatosensory and spatial information, and areas of the frontal lobe are responsible for directing and maintaining attention, developing and acting on plans, and so on. Consequently, many areas of the brain are involved in responding to input during learning and different brain areas interact by exchanging information in the learning process. Like memory, most human learning is not located in any single area of the brain, although some brain areas process different types of information and some areas seem to be more actively involved in specific types of learning.

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**Box 9.2**

**Between-neuron transmission: neurotransmitters**

The activation of a single neural impulse is not sufficient to produce any noticeable response, even something as small as the twitch of a muscle, let alone complex mental activity such as thinking of a creative solution to a problem. It is only when one neuron stimulates others around them that any noticeable activity begins to occur. It usually takes billions of neurons to be activated together for complex behaviour or mental processes to occur.

Neurons do not link together like a chain. Each neuron is separated from the next neuron by a tiny gap called a synaptic gap (or synaptic cleft, as it also called). The synaptic gap is about 300 times thinner than the thinnest strand of your hair. Neural information passes across the gap from the axon terminal and connects with any number of dendrites from adjacent neurons (see figure 9.8). The point, or site, of chemical communication between neurons is called a synapse. The synapse includes the synaptic gap and a small area of the membrane of each of the connecting neurons.

The transmission of neural impulses within the neuron basically involves electrical activity. However, when the neural impulse reaches the end of the axon, the transmission of the information from one neuron to the next primarily involves a chemical process.

When the neural impulse reaches the end of each axon, neurotransmitter is released from tiny vesicles within the synaptic buttons. A neurotransmitter is a chemical substance that is made by the neuron and enables communication between neurons. Each neurotransmitter contains ions that travel across the synapse from the neuron releasing it (the presynaptic neuron) to the receptors on the dendrites of the receiving neuron (the postsynaptic neuron). The neurotransmitter works by attaching (‘binding’) itself to the receptor site on the receiving neuron. Generally, the neurotransmitter will have either of two
effects. Sometimes, the neurotransmitter will have an excitatory effect, and consequently stimulates or activates a neural impulse in another neuron. At other times, the neurotransmitter will have an inhibitory effect, and blocks or prevents the receiving neuron from firing (see figure 9.9).

Each type of neurotransmitter has a chemically distinct shape. When released by the presynaptic neuron, the neurotransmitter searches for the correctly shaped receptor sites on the dendrites of the postsynaptic neurons. Like a key in a lock or a piece of a jigsaw puzzle, a neurotransmitter’s shape must precisely match the shape of the receptor site on the postsynaptic neuron’s dendrites for the neurotransmitter to have an effect on that neuron. However, a postsynaptic neuron can have many different-shaped receptor sites on its dendrites and may therefore be able to receive several different neurotransmitters (Hockenbury & Hockenbury, 2006) (see figure 9.10).

The number of neurotransmitters that a neuron can manufacture varies. Some neurons manufacture only one type of neurotransmitter, whereas other neurons manufacture two or more. Although estimates vary, researchers have identified more than 100 different neurotransmitters. For example, two of the major neurotransmitters believed to be involved...
in learning and memory are glutamate and dopamine.

While communication between one neuron and another is usually a chemical process involving neurotransmitters, communication between neurons also occurs in other ways. In some instances, communication between neurons is electrical; for example, when axons transmit messages directly to other axons or directly to the cell body of other neurons and when dendrites of one neuron communicate directly with the dendrites of other neurons.

What happens within the brain when something new is learned? As in the process of memory formation, when learning occurs, physical changes take place in the brain at the neuronal, or ‘cellular’, level. The most prominent of these changes is in the area of the synapses where transmission between neurons occurs and neurons interconnect with others in forming new synapses and neural pathways. In some cases, learning changes the strength of connections between neurons at the synapses within neural pathways that also become our memory of an experience. In other cases, learning can cause new synapses to form (Breedlove, Rosenzweig & Watson, 2007). This has led some psychologists who adopt the biological perspective to describe learning as a process that involves synapse formation and the building of neural pathways in the brain. Although this description is simplistic as it overlooks many other influences on learning, it is not an inaccurate description of the relationship between learning and the resulting physical changes in the brain. Most psychologists believe that the ability of the brain to ‘rewire’ itself by modifying existing neural connections and pathways, or by forming new neural connections and pathways, provides the biological basis, or ‘foundation’, of learning.

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 the piano will establish new neural connections and regular piano practice will strengthen the connections. Hebb (1949) proposed that learning results in the creation of cell assemblies, or inter-connected 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 different learning or in producing different patterns of behaviour, depending on which combination of neurons is active.

According to Hebb (1949), when a neurotransmitter is repeatedly sent across the synaptic gap, the presynaptic (‘sending’) neuron and the postsynaptic (‘receiving’) neuron are repeatedly activated at the same time. This has the effect of actually changing the chemistry of the synapse, leading to a strengthening of the connections between the neurons at the synapse. When the synaptic connection between neurons is strengthened, this makes them more likely to fire together again and to transmit their signals more forcibly 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. Known as ‘Hebbian learning’, this biological theory of learning is summarised by Hebb’s rhyme ‘neurons that fire together, wire together’. Hebb’s theory has since been supported by a considerable amount of research evidence and many biological models of learning have been, and continue to be, based on his theory. For example, Kandel’s research on memory formation was influenced by and provided evidence for Hebb’s theory.

When Kandel studied the neuronal basis of memory in the sea snail Aplysia, he had to induce learning. In doing so, he was able to observe what was happening at the synapse as Aplysia learn and remember. A neuron was observed to release more neurotransmitter across the synaptic gap and dendrites on postsynaptic neuron receiving the neurotransmitter became bushier. According to Kandel, these changes at the synapse strengthened the connections between adjacent neurons. As a result, a neural pathway for the

Unit 4 Brain, behaviour and experience 431

(from Grivas, Letch, Down & Carter, 2011)

Unit 3 Psychology - Course Notes - Mind Brain Body. Pg 7 of 14

learning and memory of the relevant information was created along which there was greater efficiency in the transmission of information.

The synaptic changes that take place within a neural pathway during learning are believed to have long-term potentiation. **Long-term potentiation (LTP)** refers to the long-lasting strengthening of the synaptic connections of neurons, resulting in the enhanced or more effective functioning of the neurons whenever they are activated. Basically, the effect of LTP is to improve the ability of two neurons—a presynaptic neuron and a postsynaptic neuron—to communicate with one another at the synapse.

It is now widely believed that LTP is a crucial neural mechanism that makes learning possible in humans, as well as in all animals with nervous systems (Breedlove, Rosenzweig & Watson, 2007; Gazzaniga & Heatherton, 2006).

The earliest evidence that LTP is likely to be involved in learning comes from studies with animals. In one study, British psychologist Richard Morris and his colleagues (1982) investigated the role of both LTP and the hippocampus in spatial learning using rats and a water maze. The researchers set up a circular tank filled with milky water that obscured a platform submerged just below the surface. The researchers used this apparatus to compare the performance of three groups of rats in swimming through the water maze to the platform. Group 1 comprised rats with a cerebral cortex that had been surgically damaged in the upper area of the frontal lobe, group 2 comprised rats with a surgically damaged hippocampus and group 3 comprised rats with no surgically damaged brain area or structure. When a group 3 ‘normal’ rat was placed in the tank, it would swim around until it found the platform and then pull itself up. Each time it was placed in the tank, it located the platform more quickly, eventually working out the most direct route and thereby demonstrating learning. When a group 1 rat (with cortical damage) was placed in the tank, it performed about as well as a group 3 rat. After several trials, it would learn a direct route through the maze to the platform. However, whenever a group 2 rat (with hippocampal damage) was placed in the tank, it showed little evidence of learning.

As shown in figure 9.11, these rats failed to learn a direct path to the platform, performing in each trial as if it was the first trial. The results of this study indicate that the hippocampus is important in spatial learning. It also suggests that LTP is important in learning because the hippocampus has been found in many previous and subsequent studies on learning and memory to influence neuronal changes that result in LTP.

Further evidence of the possible role of LTP in learning comes from studies indicating that drugs which enhance transmission of information across the synapse also tend to enhance learning. NMDA (N-methyl-D-aspartate) is a neurotransmitter receptor found on the dendrites of neurons, particularly neurons in the hippocampal region. NMDA is specialised to receive the neurotransmitter called glutamate and, together with glutamate, is believed to have an important role in LTP. Without NMDA at the site of a dendrite where glutamate is received, any message carried in glutamate from a neuron cannot be ‘accepted’ by a postsynaptic neuron. Research findings that NMDA glutamate receptors are involved in LTP led researchers to examine whether they could influence learning by manipulating the capability of NMDA receptors in postsynaptic neurons during learning tasks. In one study, American psychologist

![Figure 9.11 Typical swimming paths shown by rats within a water maze. Normal rats (c) rapidly acquire a direct path, as do rats with cortical damage (a), whereas hippocampal damage results in a failure to learn (b).](image-url)
Joseph Tsien (2000) used genetic engineering to produce a strain of mice that had more-efficient NMDA receptors. When tested on various learning and memory tasks, these mice performed better on all tasks than did normal unaltered mice in the control group. For example, they outperformed the normal mice in maze-learning and object recognition tasks. They also showed significantly better memory when tested a day or more later. These findings raise the possibility of developing drugs that might enhance the learning process (and memory) by activating or mimicking NMDA. However, much research on the role of LTP and NMDA in learning remains to be done. Although LTP has been recorded in the brains of higher order animals and human research participants during learning (and memory), the role of LTP in complex forms of learning in humans has yet to be extensively investigated. Generally, it is now widely accepted that LTP is necessary for learning and that NMDA receptors are necessary for the changes at the synapse assumed to underlie learning. However, this does not mean that other biological processes, as well as psychological processes, are of lesser importance in learning (Breedlove, Rosenweig & Watson, 2007; Gazzaniga & Heatherton, 2006).

Learning Activity 9.4

Review questions

1. Name the three components of the synapse and their roles in communication between neurons.
2. What role does neurotransmitter play at the synapse?
3. Explain the meaning of synapse formation and the role of this process in learning.
4. Explain the meaning of Hebb’s rhyme ‘cells that fire together, wire together’ in relation to learning.
5. a. What is long-term potentiation (LTP)?
   b. Why is LTP believed to be a crucial biological process for learning to occur?
   c. Why is the study by Morris and others (1982) considered to provide evidence for LTP having a role in learning?
6. Explain why some psychologists who adopt the biological perspective have described learning as a process of modifying existing neural pathways or building new neural pathways.

Figure 9.12 In Tsien’s (2002) experiment, the ‘smart’ (genetically altered) mice were exposed to two objects: one that they had explored previously and one that was new. Like other mammals, mice prefer to explore new objects more than familiar ones. The ‘smart’ mice explored the new object (red top) more than the original object (orange top), even when several days had passed since the first session. The unaltered mice explored the new object more than the familiar one only when a shorter period of time had elapsed. To Tsien and his colleagues, this was evidence that the ‘smart’ mice remembered the original object for longer than did the unaltered mice.
Neural Development in Learning

read p160-165

Describe the formation of a neuron through the process of cell division (p160)

Define synaptogenesis

Define filipodia and the role they play in synaptogenesis

When, during our lifetime, does synaptogenesis occur?

What role does experience play in the process of synaptogenesis consolidation?

Summarise the work/theories of Donald Webb (p160) regarding neural development in learning

Complete Ex 5.3 p163-164, all Q’s
Role of neurotransmitters in learning

Define the **pre-synaptic** neuron

Define the **post-synaptic** neuron

Define **action potential**

Define **long-term potentiation (LTP)** (see p8 of notes previously)

Define **receptor**. Explain its role in learning.

The nerve impulse (action potential) within a neuron is primarily ___________. In contrast, communication between neurons is ____________, so messages are sent between neurons chemically (neurotransmitters).

Compare the **excitory** or **inhibitory** actions of neurotransmitters.

Two of the major neurotransmitters involved in memory and learning are:

**Complete Ex 5.4 p165, all Q’s**
Developmental & Adaptive Plasticity
read p166-168

Define plasticity

Define developmental plasticity

Define adaptive plasticity

What changes occur in the brain in response to learning and experience?

Give an example of adaptive plasticity

What are the implications of plasticity, and what conditions encourage plasticity?

Complete Ex 5.5 p168, all Q’s
Critical Periods - The effect of timing of experience on learning

read p.168-170

**Define critical periods**

**Give an example of a critical period**

**List some evidence in support of 'critical periods'**

**What does plasticity tell us about critical periods? Does critical period mean there is no development outside of this time? (see p169)**

Evidence in support of plasticity

read p.170-172

**List some evidence in support of neuroplasticity**
Imaging the learning brain

read p172-173

List the brain imaging techniques used to monitor the learning brain:

What have been some of the discoveries about the learning brain from these imaging techniques?

Complete Ex 5.6 p173, all Q’s

Complete Chapter Review p174-176