The better way to learning

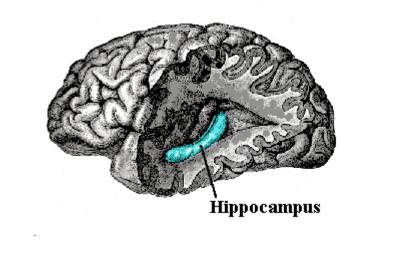
Work steadily rather than last-minute cram is sound advice, says S.Ananthanaryanan.

Teachers, trainers, students –need to know the best way – study in small and many doses or in a short and packed programme? The slow method is recommended because it is easier to administer and corresponds to the principles of hard work, thrift and prudence and seems right by experience. But managers of quick start undertakings would like hard science to know how best to train executives for error free work – should it be over extended time or can short, concentrated input be as useful? And it is equally true of learning to drive or to play the violin!

Learning and the brain

It is understood that learning involves formation of long term memory, which takes place via formation of stable connections between neurons, or brain cells. Repeated connection between areas of the brain, for instance for salivation, with the area that perceives food, results in consolidating the neural highway and creates a well learnt stimulus-response sequence.

This general mechanism of learning - the idea of 'plasticity' of the brain, or its ability to add or remove connections based on experience, is well documented and the role of the *hippocampus*, a structure in the brain that mediates learning, and specific enzymes that get active in long term memory formation, are understood. But still, while it is found that 'spaced' learning, over many sessions, is more effective than 'massed' learning – over short intervals, for this there has been no clear explanation. This raises the important question of whether the it is indeed true that slow learning is the better way?



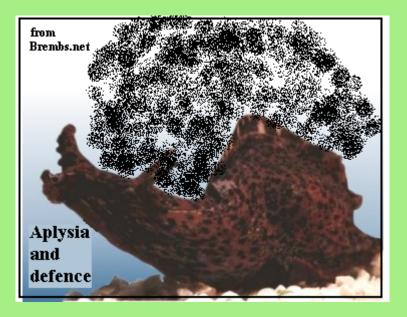
There are two kinds of mechanisms suggested for memory formation. The first is that stimulation of inter-neural connectors, the 'synapses', result in changes in the existing proteins, known as *protein kinases*, resulting in changes in the way the synapses work.

The other mechanism depends on agents that affect the genetic protein generation machinery at synapses. The effectiveness of synapses depend on the density of channels that can convey electric signals and there is evidence that synapses adapt these features in response to stimulus.

Aplysia and learning

The journal, Neuroscience, has carried a report by Dr Wayne Sossin and others at Mc Gill University in Canada who have discovered a link at the molecular level to explain the different types of learning. The discovery was made with the help of neural cells of the Aplysia, a marine slug, which is able to 'learn' to invoke its natural defense mechanism, the release of a cloud of black ink to blind its attacker, in response to particular signals.

But the interest in the Aplysia is because ink release behaviour is caused by electrical activity in several connections between the limited number of large size nerve cells found in the animal. This feature makes it easy to study the mechanism of memory formation and the Aplysia is a favourite for research in the field.



The way the Aplysia is 'trained' is with the help of a light touch or prod, which would normally not be alarming, so as to set off any defense reaction. But if a touch is repeatedly associated with a distinctly unpleasant experience, like an electric shock, the animal begins to associate the touch with the shock and reacts to the touch like it would to a shock.

It has been established that the strength of connections between nerve cells, which is called *synaptic facilitation*, is the basis of learning that takes place in the Aplysia. The actual transmission of a message from one cell to another is via chemicals, called *neurotransmitters*, released by the originator cell, when electrical pressures build up

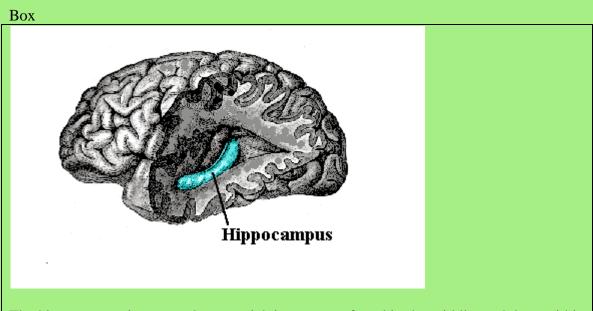
within the cell. The chemical then binds to regions at the other end of the synapse, to set off changes within the receiver cell.

In the case of the Aplysia, the Mc Gill Univ team found that it is a neurotransmitter called *serotonin* which controls the changes in nerve cells when they communicate. But it was found that the generation of another agent, called Potein Kinase C App II (PKC App II) is quite different during different learning sequences. This difference may contain the answer to why there are different learning results.

PKC holds the key

The study showed that four to five spaced application of serotonin resulted in long term changes in the strength of the synapse, or the junction between neurons. Spaced applications led to less activation and build up of PKC App II and in four to five applications, separated from one another, the reduced activation of PKC was associated with increased learning and retention. In contrast, if the application of serotonin was continuous, as would happen in massed learning, there was more activation of PKC App II – which suggests that it is PKC that blocks the formation of long term memory.

This is the first study of a clear difference in the biochemistry of spaced and massed learning and promises to explain why one kind is more effective.



The hippocampus is a curved organ, rich in neurons, found in the middle and deep within the brain and it is known to mediate the working of the long term memory. The theory that memory works by strengthening connections between neurons that are active at the same time was formalized in 1948. It was in 1970 that evidence was found of this effect, in the hippocampus of the rabbit. While similar changes have now been observed in other areas of the brain as well, the role of the hippocampus has been demonstrated in the decay of this organ in progress of Alzheimer's disease, an ailment marked by inability to form long term memory, and also in the memory related effects of surgical removal of the hippocampus.

The organ gets its name from its similarity to the horns of a ram, maybe a seahorse. A 16th century anatomist coined the name from the Greek works, 'hippos' for horse and 'kampos' for sea monster.