

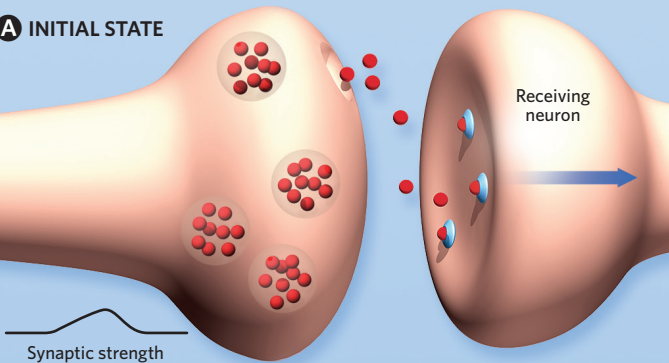
MOLECULAR LEARNING

Long-term potentiation (LTP), discovered in the 1970s, was later shown to be the molecular basis of memory. Since many diseases of aging affect memory, could memory formation and storage be altered by the same mechanisms in normal aging and diseased brains? LTP was the perfect tool to find out.

LONG-TERM POTENTIATION

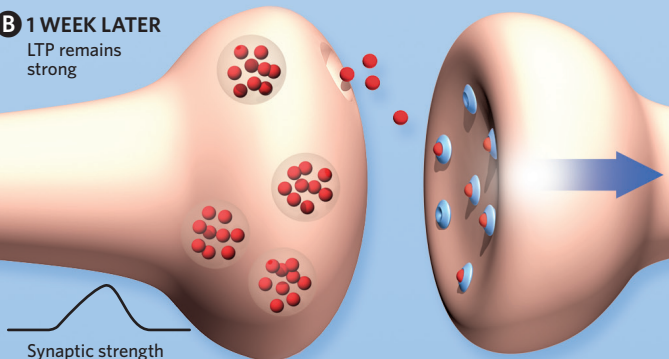
The neurotransmitters and receptors at the synapses between neurons propagate the electrical signal that transmits information **A**. During memory formation, synapses between neurons are strengthened as the number of receptors are increased on the surface of the receiving neuron **B**. This molecular strengthening, or LTP, decays faster in old rats than in young ones **C**. Indeed, researchers showed that old synapses are more susceptible to a process that weakens synaptic strength, called long-term depression (LTD), which may explain why LTP is not as durable in the aged rat brain.

A INITIAL STATE



B 1 WEEK LATER

LTP remains strong



C 2 WEEKS LATER

In an aged animal

