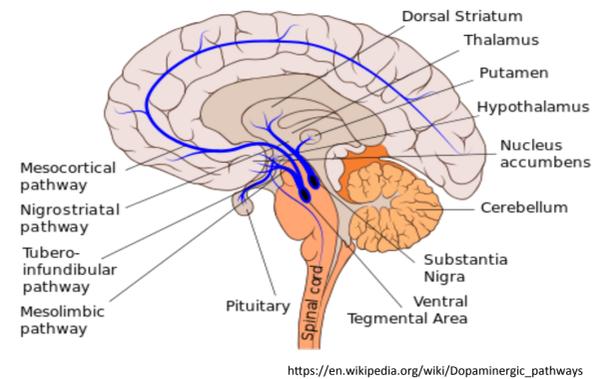


Major Neurotransmitters in the Brain

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Dopamine



https://en.wikipedia.org/wiki/Dopaminergic_pathways

This diagram shows the four major pathways of dopamine transmission: the mesocortical pathway, nigrostriatal pathway, tubero-infundibular pathway, and mesolimbic pathway.

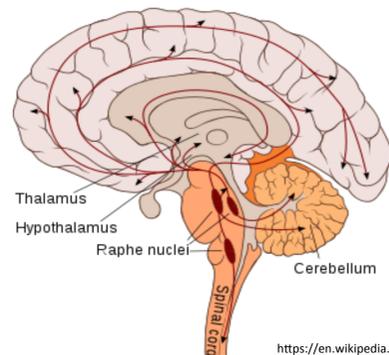
Mesolimbic (reward) pathway: increases motivation for reward stimuli, mediates reward-related learning, involved in motivation cognition

Mesocortical pathway: governs executive functions (attention, planning, working memory, etc.)

Nigrostriatal pathway: involved in motor functions

Tuberoinfundibular pathway: regulates prolactin secretion from the anterior pituitary gland

Serotonin

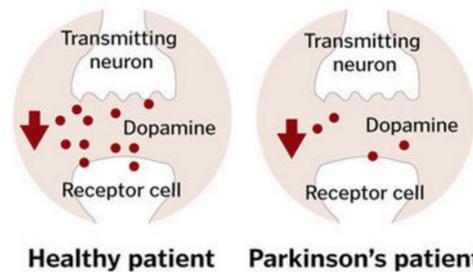


https://en.wikipedia.org/wiki/Serotonin_pathway

The neurotransmitter serotonin is found primarily in blood platelets, the central nervous system, and the gastrointestinal tract. Raphe nuclei, labeled above in the diagram, produce serotonin and project it to every part of the brain. The sleep-wakefulness cycle is majorly regulated by serotonin. While awake, the activity of serotonin neurons varies little; while asleep, activity slows and comes to a complete stop when dreaming. Serotonin is also a dominant factor in mediating calmness and happiness.

Parkinson's Disease:

an imbalance of acetylcholine and dopamine in the brain

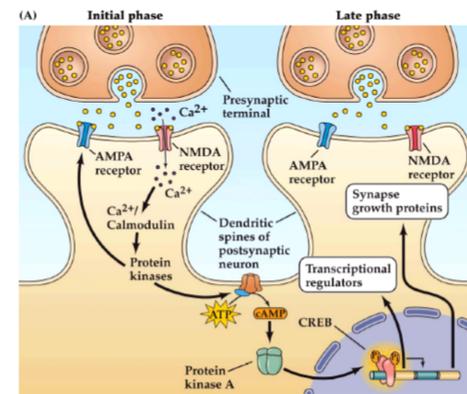


http://www.cleveland.com/healthfit/index.ssf/2011/05/parkinsons_and_the_brain_graph.html

While dopamine is an inhibiting neurotransmitter, meaning it signals motor neurons to stop producing movement, acetylcholine does the opposite. A healthy brain contains a delicate balance of acetylcholine and dopamine; however, when the substantia nigra, a dopamine-producing part of the brain, stops generating sufficient amounts of dopamine, the balance is disrupted. There is then too much acetylcholine signaling to the motor neurons to produce movement and not enough dopamine to control this motion, thereby producing tremors in the body.

Long-term Potentiation:

a mechanism underlying learning and memory formation

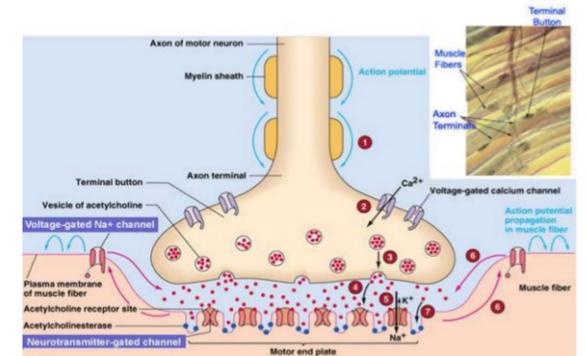


<http://bigtone.zone/2013/12/05/long-term-potentialion-depression/>

The process of long-term potentiation strengthens neuronal synaptic connections, as a mechanism for how the brain responds to experience. When glutamate is released from the presynaptic cell to the post-synaptic cell, glutamate receptors, known as AMPA receptors, depolarize and cause magnesium to flow to other glutamate receptors, called NMDA receptors. This discharge of magnesium allows for the passage of calcium ions through the NMDA receptors, which then activate more AMPA receptors on the membrane. These additional AMPA receptors lead to the strengthening of the post-synaptic cell, the hallmark of long-term potentiation, as the cell has been conditioned to respond more strongly to future glutamate release.

Acetylcholine

The Neuromuscular Junction

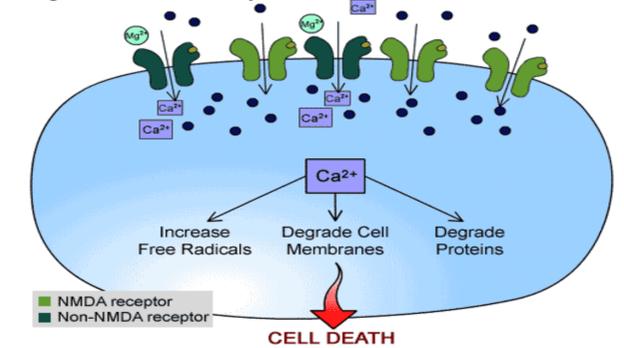


<https://www.slideshare.net/danielveladuaremd/neuromuscular-junction>

The neurotransmitter acetylcholine activates the muscles used for all types of voluntary movement (skeletal muscles). Spinal cord motor neurons that directly control these muscles send electrical impulses through motor nerves, which connect to the neuromuscular junction (above). Once acetylcholine is released into the synaptic cleft and binds to postsynaptic receptors, ion channels allow sodium to discharge into the muscle cell, producing a muscle contraction.

Glutamate

Figure L-2: Ca²⁺ Entry into the Cell



http://web.stanford.edu/group/hopes/cgi-bin/hopes_test/about-glutamate-toxicity/

Used by every significant function in the vertebrate brain, glutamate is the most bountiful neurotransmitter in the vertebrate nervous system. It plays a large role in memory and learning under normal circumstances. However, excess glutamate causes cell death and leads to impaired spatial learning; a depletion of glutamate similarly decreases information processing abilities. Glutamate mediates the formation of memory, but when too much calcium enters the neuron, cell death occurs and memory is lost.

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