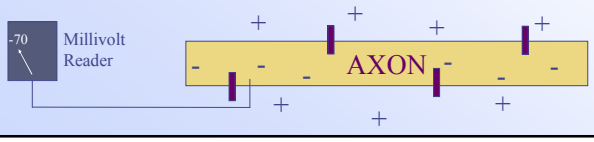
**Neural Communication**
**Lecture 11**

## III. NEURAL COMMUNICATION
### A. Resting Potential

- In this section, we will consider the basic unit of the nervous system – the neuron – and how neurons communicate with each other.
  - The story of neural communication is electrical within a cell and chemical between cells.
- Neurons have cell membranes that separate them from the environment outside the neuron.
  - On the membrane, selective tunnels or channels connect the inside of the cell to the outside, extra cellular environment.

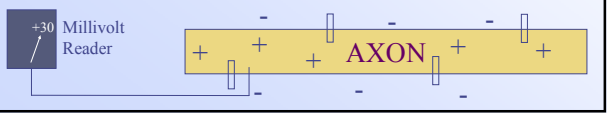
## III. NEURAL COMMUNICATION
### A. Resting Potential

- There is an imbalance of ions (electrically charged particles) inside and outside the cell
  - **Positive (+) ions** are concentrated outside the cell, whereas **negative (-) ions** are concentrated inside the cell.
  - In this state, the charge in the cell is at -70 millivolts, and this is the **resting potential**




## III. NEURAL COMMUNICATION
### B. Action Potential

- When the cell is stimulated, the channels open and there is a reversal of ions.
  - Predominately positive (+) ions rush into the cell and negative (-) ions rush out.
  - This results in a voltage spike in the cell to +30 millivolts, called the **action potential.**
  - The cell then pumps out + ions, causing - to return and the cell returns to its resting potential



## III. NEURAL COMMUNICATION
### C. Rate and Nature of Neural Firing

- The action potential works its way down the axon much like a fire works down a fuse.
  - A cell can fire once then returns to resting potential. In an **all-or-nothing** manner.
  - A myelinized axon causes the action potential to travel faster because action potentials occurs at the nodes.
  - A highly stimulated cell will fire repeatedly in a short period of time.
    - High rate -/-/-/-/-/-/-
    - Slow rate /-----/-----/

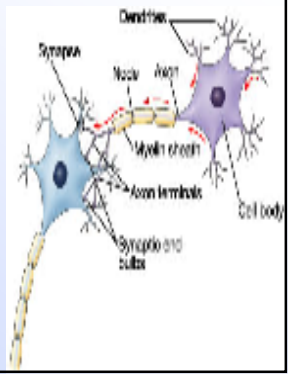
## III. NEURAL COMMUNICATION
### C. Rate and Nature of Neural Firing

The terminal endings come into close contact with another neuron at a synapse.

**Presynaptic membrane**: The terminal endings of the axon of a target neuron.

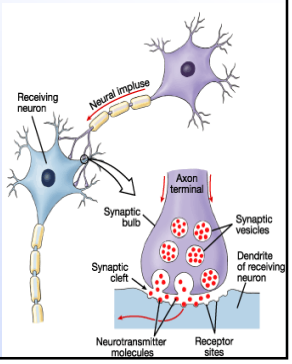
**Synaptic Cleft**: The space between neurons

**Postsynaptic membrane**: Another neuron dendrite or cell body

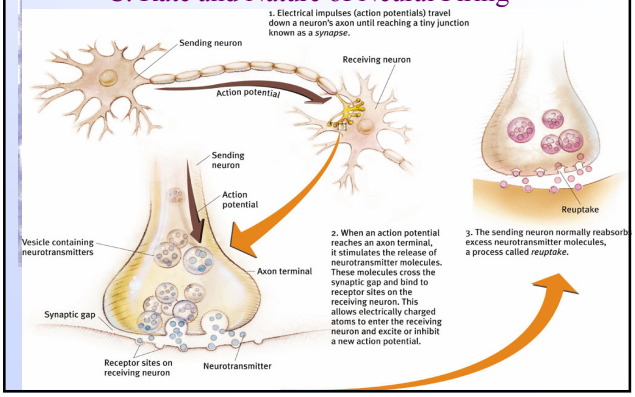



## III. NEURAL COMMUNICATION
### C. Rate and Nature of Neural Firing

- When firing, neurons have a chemical effect on other cells.
- **Neurotransmitters** are chemicals contained in **synaptic vesicles.**
- Neurotransmitters travel across the synaptic cleft and stimulate receptor sites on the postsynaptic membrane.




## III. NEURAL COMMUNICATION
### C. Rate and Nature of Neural Firing



## III. NEURAL COMMUNICATION
### C. Rate and Nature of Neural Firing

- Neurotransmitters function at receptor sites like a key fitting into a lock.
  - Other chemicals interact with receptor sites too.
    - **Agonist**: Chemical which functions like the neurotransmitter targeted for the receptor site.
    - **Antagonist**: Chemical which blocks receptor sites inhibiting the neurotransmitter.
  - After it is released, neurotransmitters are either returned to the presynaptic cell (called **reuptake**) or chemically deactivated by enzymes (call **deactivation**).

## III. NEURAL COMMUNICATION
### C. Rate and Nature of Neural Firing

- A neuron firing and releasing neurotransmitter can have one of two effects on other neurons:
  1. It could **facilitate** the firing of another neuron, making it more likely to fire.
  2. It could **inhibit** the firing of another neuron, making it less likely to fire.
     - Neurons fire as a result of the sum total of facilitating and inhibiting stimulation
- Neurons are sensitive to different types of neurotransmitters which have important consequences for behavior.

## III. NEURAL COMMUNICATION
### D. Neurotransmitters

- **1. Acetylcholine (Ach)**
  - Found at the neuromuscular junctions and other peripheral synapses of the ANS (e.g., heart).
  - Ach is also found in the brain and may be critical for normal intellectual functioning
    - If Ach receptors are blocked by mimicking drugs (so there is a deficit of Act), animals', performance on memory and learning tasks are also impaired.
    - Also, if Ach is increased, there appears to be increased learning and retention.
    - Ach **deficit** in extreme is found in the brain of Alzheimer's patents

## III. NEURAL COMMUNICATION
### D. Neurotransmitters

- **2. Gamma-aminobutyric acid (GABA)**
  - It is a simple amino acid which is effectively picked up by the post synaptic receptor sites.
  - GABA **inhibits** the action potential of other cells.
  - GABA is difficult to deactivate after release and tends to be picked up by the neuron that releases it.
    - A lack of GABA is associated with epileptic seizures which are "runaway firing of neurons."
    - GABA synapses have been implicated in anxiety neurosis. Valium and Librium appear to facilitate GABA receptors thereby reduce anxiety by increasing inhibition of neuronal activity at site.

III. NEURAL COMMUNICATION
D. Neurotransmitters

- **3. Dopamine**
  - Three dopamine circuits have been discovered.
    1. Between the hypothalamus and pituitary gland
    2. Lower midbrain
       - Important for regulating movement. Parkinson disease (tremors, repetitive movements, difficulty standing, and initiating bodily movements) is due to a loss of dopamine neurons in this region.
    3. Parkinson's: Lower midbrain to higher cortical regions.
       - Abundance in schizophrenia. Thorazine and Haldol, antipsychotic drugs which control schizophrenic symptoms, interfere with synaptic transmission at dopamine sites.
  - Excess: Involuntary movements & schizophrenia.
  - Deficit: Impaired movement (Parkinson) Memory impairment, depression.

III. NEURAL COMMUNICATION
D. Neurotransmitters

- **4. Norepinephrine**
  - Norepinephrine identified in peripheral nervous system, particularly in the Sympathetic NS, and functions for arousal.
    - Norepinephrine circuits are all over brain. The circuits are sparse, dispersed and widespread.
    - Also related to learning and memory. More Norepinephrine better performance. Don't study tired.
  - Deficit: Memory impairment and perhaps depression.
  - Excess: Anxiety and symptoms resembling schizophrenia.

III. NEURAL COMMUNICATION
D. Neurotransmitters

- **5. Serotonin**
  - Serotonin is involved in many behaviors
  - Sleep induction
    - Serotonin may play a role in sleep induction. Grandmother's suggestion to drink a glass of warm milk before sleep may be sound, since milk is a good source of tryptophan, which is the amino acid needed by the brain for the synthesis of serotonin.
  - Regulation of appetite
    - Hunger is reduced by drugs which elevate serotonin levels in the brain (e.g. fenfluramine/Pondimin or dexfenfluramine/Redux) making these drugs popular in the treatment of obesity.

III. NEURAL COMMUNICATION
D. Neurotransmitters

- **5. Serotonin (continued)**
  - Human violence
    - Aggressive behaviors and suicide have all been associated with reduced levels of serotonin in the brain.
  - Human psychiatric disorders
    - Depression and OCD (obsessive compulsive disorder) are effectively treated with drugs which specifically block the reuptake of serotonin into the presynaptic axon terminal (Prozac).

III. NEURAL COMMUNICATION
D. Neurotransmitters

- **6. Neuropeptides**
  - Neuropeptides are short chains of amino acids, which include endorphins
    - Endorphin gets its name from the contraction of "endogenous morphine".
    - Endorphins are similar in structure and action to opiates
  - Endorphins are involved in pain reduction, pleasure, and memory
    - Excess of Endorphins: Inhibition of pain
    - Deficit: Increased pain
  - Endorphins also functions as a neuromodulator, intensifying the effects of other neurotransmitters.

III. NEURAL COMMUNICATION
D. Neurotransmitters

- **7. Glutamate**
  - Glutamate is the most common neurotransmitter in the brain.
    - It is always excitatory, usually due to simple receptors that increase the flow of positive ions by opening ion-channels.
      - Glutamate is involved in cognitive functions like learning and memory in the brain.
      - The form of plasticity known as long-term potentiation (LTP) takes place at glutamatergic synapses in the hippocampus, neocortex, and other parts of the brain.
    - Oversupply can overstimulate brain, producing migraines or seizures (which is why some people avoid MSG, monosodium glutamate, in food).