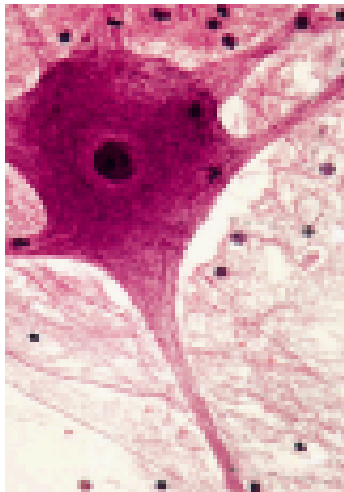


BIO 211:
ANATOMY & PHYSIOLOGY I



Ch 10 A

Ch 10 B

← This set

CHAPTER 10

NERVOUS SYSTEM 1

BASIC STRUCTURE and FUNCTION

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Naugatuck
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Synapses

A. Discovery of Neurotransmitters:

A space, the **synapse**, occurs between two adjacent neurons.

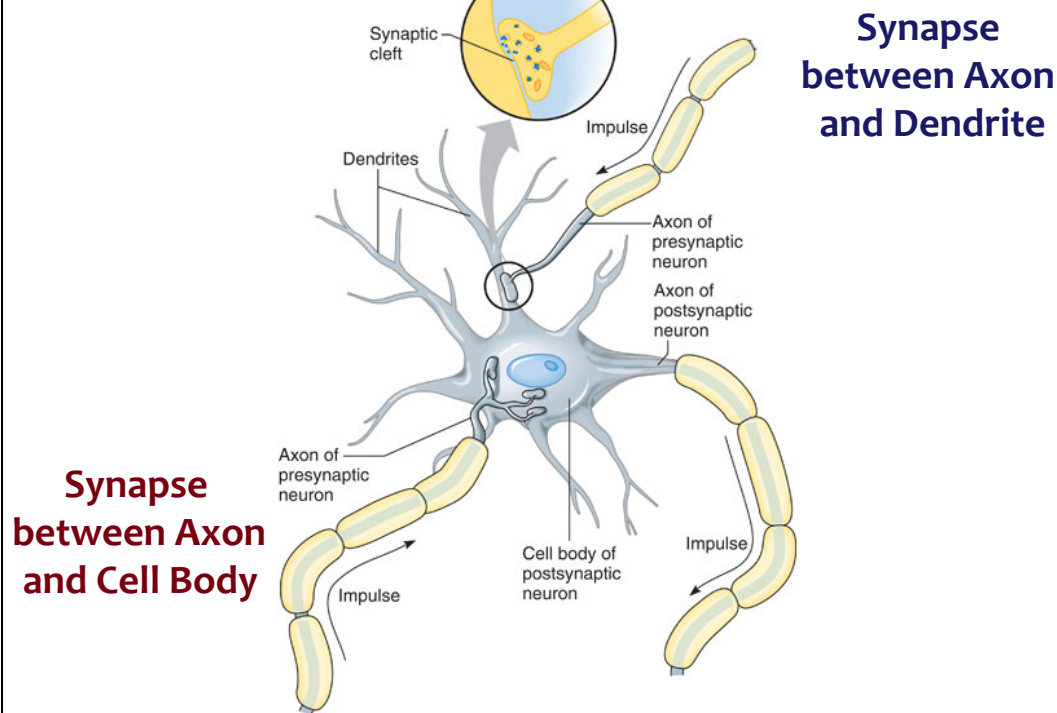
Acetylcholine: first known neurotransmitter.

Neurons, muscle cells, and neuroglia do communicate through **gap junctions**.

Much of the communication within the nervous system is accomplished using **neurotransmitters**.

B. Chemical Synapse Structure

1. The **presynaptic neuron** houses vesicles filled with neurotransmitter in its **synaptic knob**.
2. The **postsynaptic neuron** contains *no specializations* other than **proteins** that function as **receptors** and **ion gates**.



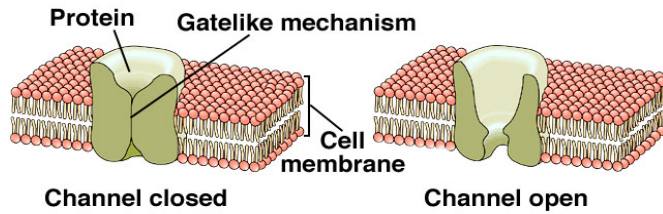
C. Neurotransmitters and Related Messengers

1. More than 100 different chemicals have been identified as **neurotransmitters**.
 2. **Major neurotransmitters:**
 - acetylcholine
 - epinephrine (adrenalin)
 - serotonin
 - norepinephrine (noradrenalin)
- Neuropeptides:** may **modify** neurotransmitter activity

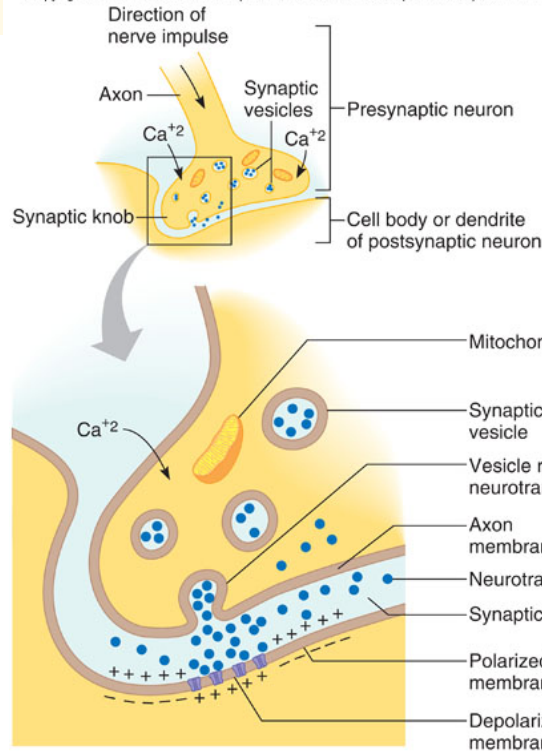
D. Synaptic Transmission

1. **Cholinergic synapse:** mediated by **acetylcholine**
2. **Presynaptic neuron** transmits an impulse to its **synaptic knob** --> **synaptic vesicles** --> **Ach** --> **cleft**.
3. **Postsynaptic neuron:** **ACh** binds to gated channels, causing them to open; sodium and potassium cross the membrane ---> a **local postsynaptic potential (PSP)**.
4. If strong enough, the **PSP** opens voltage-gated ion channels, causing the neuron to fire. (**ionotropic** effect)

TABLE 10.7	Events Leading to Neurotransmitter Release
<ol style="list-style-type: none"> 1. Action potential passes along a nerve fiber and over the surface of its synaptic knob. 2. Synaptic knob membrane becomes more permeable to calcium ions, and they diffuse inward. 3. In the presence of calcium ions, synaptic vesicles fuse to synaptic knob membrane. 4. Synaptic vesicles release their neurotransmitter by exocytosis into the synaptic cleft. 5. Synaptic vesicles become part of the membrane. 6. The added membrane provides material for endocytotic vesicles. 	



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Synaptic Transmissions

TABLE 10.4 Neurotransmitters and Actions		
Neurotransmitter	Location	Major actions
Acetylcholine	CNS	Involved in control of skeletal muscle actions
	PNS	Stimulates skeletal muscle contraction at neuromuscular junctions. May excite or inhibit in autonomic nervous system actions, depending on receptors
Biogenic amines Norepinephrine	CPNS	Creates a sense of feeling good; low levels may lead to depression
	PNS	May excite or inhibit autonomic nervous system actions, depending on receptors
Dopamine	CNS	Creates a sense of feeling good; deficiency in some brain areas associated with Parkinson disease
	PNS	Limited actions in autonomic nervous system; may excite or inhibit, depending on receptors
Serotonin	CNS	Primarily inhibitory; leads to sleepiness; action is blocked by LSD, enhanced by Prozac
Histamine	CNS	Release in hypothalamus involved in "waking up"
Amino acids		
● GABA	CNS	Generally inhibitory
Glutamate	CNS	Generally excitatory
Neuropeptides		
Substance P	PNS	Excitatory; results in perception of pain
Endorphins	CNS	Generally inhibitory; reduce pain by inhibiting substance P release
enkephalins		

● Gamma-aminobutyric acid

TABLE 10.5 Neurotransmitter Imbalances – Disorders		
Condition	Symptoms	Imbalance of Neurotransmitter in Brain
Alzheimer disease	Memory loss, depression, disorientation, dementia, hallucinations, death	Deficient acetylcholine
Clinical depression	Debilitating, inexplicable sadness	Deficient norepinephrine and/or serotonin
Epilepsy	Seizures, loss of consciousness	Excess GABA leads to excess norepinephrine and dopamine Deficient GABA
Huntington disease	Personality changes, loss of coordination, uncontrollable dancelike movements, death	Deficient GABA
Hypersomnia	Excessive sleeping	Excess serotonin
Insomnia	Inability to sleep	Deficient serotonin
Mania	Elation, irritability, overtalkativeness, increased movements	Excess norepinephrine
Myasthenia gravis	Progressive muscular weakness	Deficient acetylcholine receptors at neuromuscular junctions
Parkinson disease	Tremors of hands, slowed movements, muscle rigidity	Deficient dopamine
Schizophrenia	Inappropriate emotional responses, hallucinations	Deficient GABA leads to excess dopamine
Sudden infant death syndrome ("crib death")	Baby stops breathing, dies if unassisted	Excess dopamine
Tardive dyskinesia	Uncontrollable movements of facial muscles	Deficient dopamine

TABLE 10.6		Drugs That Alter Neurotransmitter Levels	
Drug	Neurotransmitter Affected*	Mechanism of Action	Effect
Tryptophan	Serotonin	Stimulates neurotransmitter synthesis	Sleepiness
Reserpine	Norepinephrine	Decreases packaging of neurotransmitter into vesicles	Decrease blood pressure
Curare	Acetylcholine	Decreases neurotransmitter in synaptic cleft	Muscle paralysis
Valium	GABA	Enhances receptor binding	Decreases anxiety
Nicotine	Acetylcholine	Stimulates synthesis of enzyme that degrades neurotransmitter	Increases alertness
Cocaine	Norepinephrine	Blocks reuptake	Euphoria
Tricyclic antidepressants	Norepinephrine	Blocks reuptake	Mood elevation
Monoamine oxidase inhibitors	Norepinephrine	Blocks enzymatic degradation of neurotransmitter in presynaptic cell	Mood elevation
Selective serotonin reuptake inhibitors	Serotonin	Blocks reuptake	Mood elevation, anti-anxiety agent

E. Cessation of the Signal

1. **ACh** binds to its receptors for only *a very short time*, then **dissociates from the receptor**.
2. **Removal** of additional neurotransmitter in synapse:
 - diffusion**
 - reuptake** by the synaptic knob
 - chemical degradation** by **enzymatic** activity:
 - **acetylcholinesterase** *or*
 - **monoamine oxidase (MAO)**

F. Other Modes of Chemical Communication

To qualify as a "neurotransmitter", a substance must be **synthesized by a presynaptic neuron**, *released in response to stimulation*, bind to specific **receptors** on **postsynaptic cells**, and alter the physiology of the same.

Other:

- neuropeptides** gut-brain peptides
- neuromodulators** hormones/neuropeptides
- inorganic gases** i.e., Nitric oxide (**NO**)

Re: **NO**, see <http://faculty.washington.edu/chudler/no.html>

A. Postsynaptic Potentials

1. **Neural integration** refers to the information-processing, decision-making, and memory mechanisms of neurons. This ability is **based on the postsynaptic potentials (PSPs)** produced by a neurotransmitter.
2. An **excitatory postsynaptic potential (EPSP)** is the **likelihood** of the postsynaptic cell reaching an **action potential**.

*If a neurotransmitter instead makes the postsynaptic membrane **hyperpolarize**, it is called an **inhibitory postsynaptic potential (IPSP)**.*

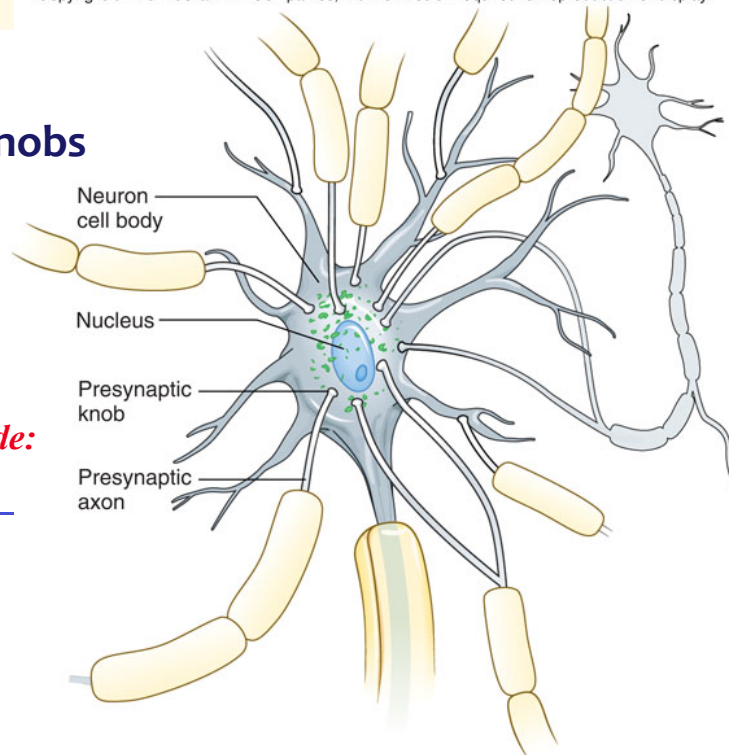
B. Summation and Facilitation

1. **Summation** is the process of **adding up incoming information** and responding to the net effect of it. This occurs in the **trigger zone** of the neuron.
2. **Summation** can be:
 - temporal** threshold reached via quick incoming **EPSPs**, *cumulative effect of-*
 - spatial** threshold reached via the cumulative addition of local potentials from several synapses

Since each synapse makes it easier for the others to cause firing, presynaptic cells converging on a single neuron are said to **facilitate** each other.

Synaptic Knobs

See previous slide:
Facilitation



C. Neural Coding

1. The way in which the nervous system *converts information to a meaningful pattern of action potentials* is called **neural coding**.
2. The nervous system employs a phenomenon known as **recruitment (or multiple motor unit summation)**, in which it is able to judge stimulus strength by which neurons, and how many of them, are firing.

D. Synaptic Potentiation and Inhibition

1. **Synaptic potentiation (presynaptic facilitation)** occurs at synapses when thought or actions occur **repeatedly**, producing a well-worn pathway. Prolonged use of the pathway causes **more calcium to accumulate in the synaptic knob**, making impulse transmission **easier in the future**. This is the process involved in **learning**.
2. **Presynaptic inhibition**: *one neuron suppresses the release of neurotransmitter by another.*

E. Neuronal Pools

Neurons actually function in much larger groups (thousands to millions of **interneurons**) called **neuronal pools**. (Recall: *interneurons only in CNS*)

As an **input fiber** enters a neuronal pool, it branches and synapses with **numerous neurons**.

Nature of stimulus:

- excitatory
- inhibitory
- or both!!!

F. Neuronal Circuits

Neuronal circuit: the connection pathway among a series of neurons:

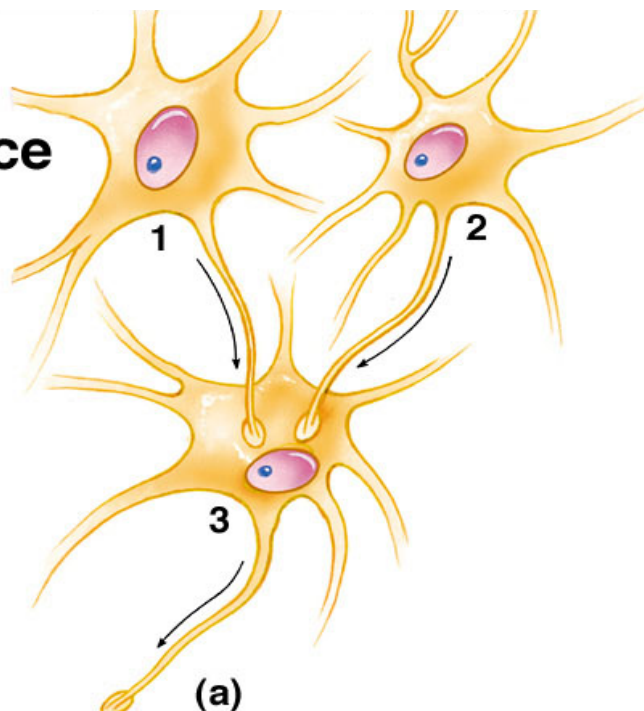
diverging circuits

converging circuits

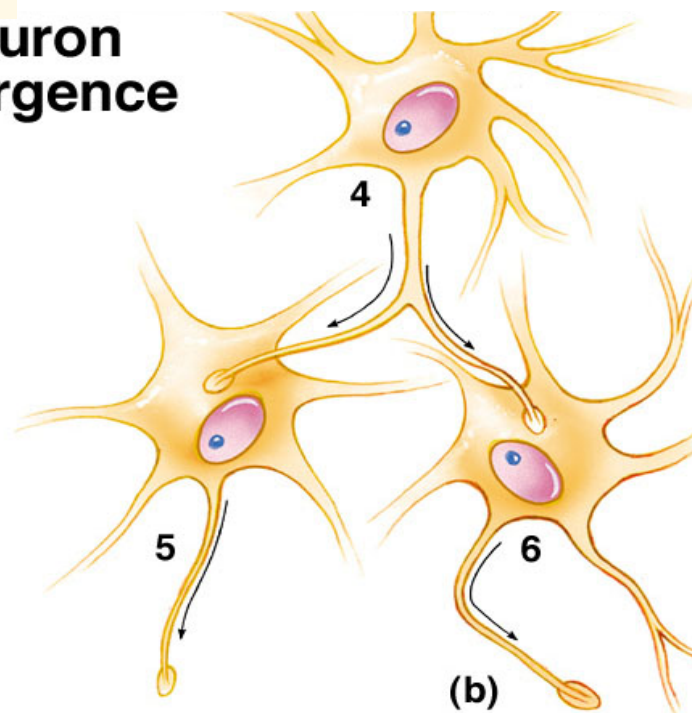
reverberating (oscillating)

parallel processing

Neuron Convergence



Neuron Divergence



Alzheimer disease is characterized by progressive memory loss, dementia, and eventually, death.

The cause is unknown, but neurofibrillary tangles in the gyri of the cerebral cortex and the hippocampus (memory areas) appear in those afflicted with this disease.

Parkinson disease is a progressive **loss of motor function** due to *degeneration of dopamine-releasing neurons* (neurotransmitter: DOPA) in the *substantia nigra* (portion of the brain).

Nicotine Addiction

