

260-2017-02-10

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*2017-02-08 08:44:58*

## Today's Topics

- How neurons talk to one another
- Synaptic communication

## In the beginning

- Soma receives input from dendrites
- Axon hillock sums/integrates
- If sum > threshold, AP “fires”

## Illustration of summation

## Steps in synaptic transmission

- Rapid change in voltage triggers neurotransmitter (NT) release
- *Voltage-gated calcium  $Ca^{++}$  channels* open
- $Ca^{++}$  causes *synaptic vesicles* to bind with presynaptic membrane, merge, *exocytosis*
- NTs diffuse across *synaptic cleft*

## Steps in synaptic transmission

- NTs bind with *receptors* on *postsynaptic membrane*
- Receptors respond
- NTs unbind, are inactivated

## Synaptic transmission

## Exocytosis

<http://dx.doi.org/doi:10.1038/nrn2948>

## Why do NTs move from presynaptic terminal toward postsynaptic cell?

- Electrostatic force pulls them
- Force of diffusion

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## Postsynaptic receptor types

- *Ionotropic* (receptor + ion channel)
  - Ligand-gated
  - Open/close ion channel
  - Ions flow in/out depending on membrane voltage and ion type
  - Faster, but short-acting effects

## Postsynaptic receptor types

- *Metabotropic* (receptor only)
  - Trigger 2nd messengers
  - G-proteins
  - Open/close adjacent channels, change metabolism
  - Slower, but longer-lasting effects

## Receptor types

### Receptors generate postsynaptic potentials (PSPs)

- Small voltage changes
- Amplitude scales with # of receptors activated
- *Excitatory PSPs (EPSPs)*
  - Depolarize neuron (make more +)
- *Inhibitory (IPSPs)*
  - Hyperpolarize neuron (make more -)

## NTs inactivated

- *Buffering*
  - e.g., glutamate into astrocytes (Anderson and Swanson 2000)
- *Reuptake* via *transporters*
  - molecules in membrane that move NTs inside
  - e.g., serotonin via serotonin transporter (SERT)
- *Enzymatic* degradation
  - e.g., AChE degrades ACh

## Questions to ponder

- Why must NTs be inactivated?

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- Why must NTs be inactivated?
  - Keeps messages discrete, localized in time and space

**What sort of PSP would opening a Na<sup>+</sup> channel produce?**

- Excitatory PSP, Na<sup>+</sup> flows in
- Excitatory PSP, Na<sup>+</sup> flows out
- Inhibitory PSP, Na<sup>+</sup> flows in
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**What sort of PSP would opening a Cl<sup>-</sup> channel produce?**

Remember  $[Cl_{out}] \gg [Cl_{in}]$ ; Assume resting potential  $\sim -60$  mV

- Excitatory PSP, Cl<sup>-</sup> flows in
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**Types of synapses**

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- Axodendritic (axon to dendrite)
- Axosomatic (axon to soma)
- Axoaxonic (axon to axon)
- Axosecretory (axon to bloodstream)

**Synapses on**

- dendrites
  - usually excitatory
- cell bodies
  - usually inhibitory
- axons
  - usually modulatory (change  $p(\text{fire})$ )

## Summary of chemical transmission

### Neurotransmitters

Family	Neurotransmitter
Amino acids	<b>Glutamate (Glu)</b> <b>Gamma aminobutyric acid (GABA)</b> Glycine Aspartate

### Glutamate

- Primary excitatory NT in CNS
- Role in learning (via NMDA receptor)
- Transporters on neurons and glia (astrocytes and oligodendrocytes)
- Linked to umami (savory) taste sensation, think monosodium glutamate (MSG)
- Dysregulation in schizophrenia? (Javitt 2010)

### Glutamate

Type	Receptor	Esp Permeable to
Ionotropic	<b>AMPA</b>	Na+, K+
	Kainate	
Metabotropic	<b>NMDA</b>	Ca++
	mGlu	

### GABA

- Primary inhibitory NT in CNS
- Excitatory in developing CNS,  $[Cl^-]_{in} \gg [Cl^-]_{out}$
- Binding sites for benzodiazepines (e.g., Valium), barbiturates, ethanol, etc.

Type	Receptor	Esp Permeable to
Ionotropic	<b>GABA-A</b>	Cl-
Metabotropic	<b>GABA-B</b>	K+

### GABA

“GABAA-receptor-protein-example” by Chemgirl131 at English Wikipedia - Transferred from en.wikipedia to Commons by Sreejithk2000 using CommonsHelper.. Licensed under Public Domain via Commons.

### Next time...

- More on NTs!

## References

Anderson, Christopher M., and Raymond A. Swanson. 2000. "Astrocyte Glutamate Transport: Review of Properties, Regulation, and Physiological Functions." *Glia* 32 (1). John Wiley & Sons, Inc.: 1–14. doi:10.1002/1098-1136(200010)32:1<1::AID-GLIA10>3.0.CO;2-W.

Javitt, Daniel C. 2010. "Glutamatergic Theories of Schizophrenia." *Israel Journal of Psychiatry and Related Sciences* 47 (1): 4.