

13. NE.1 · Neurons, synapses and brain systems

Starter Quiz ·

GRE Subject Test · GRE Psychology

Name: _____ Score: _____

1. An SSRI's defining synaptic action is
 - ☐ blocking serotonin reuptake
 - ☐ mimicking dopamine
 - ☐ destroying serotonin
 - ☐ blocking serotonin receptors
2. A key feature of Parkinson's disease is degeneration of substantia nigra neurons that release
 - ☐ serotonin
 - ☐ GABA
 - ☐ dopamine
 - ☐ acetylcholine
3. In the standard axon model, intensity can be represented by the frequency of similar-sized spikes rather than proportionally larger spikes.
 - ☐ True ☐ False
4. During the absolute _____ period, another action potential cannot be triggered.

5. A local membrane depolarisation increases in size without producing an axonal spike. Which description fits?
 - ☐ A graded potential changed.
 - ☐ A chemical transmitter crossed the entire axon.
 - ☐ A half-sized all-or-none spike occurred.
6. Later in recovery, a stronger-than-usual depolarisation can trigger a second spike. Which phase best fits?
 - ☐ Relative refractory phase.
 - ☐ A phase with no excitability limit of any kind.
 - ☐ Absolute refractory phase.
7. Which description best explains saltatory conduction?
 - ☐ Transmitter molecules leap between myelin segments.
 - ☐ Each node produces a larger spike than the previous node.

- ☐ Local current spreads between nodes and action potentials are regenerated at nodes.
8. A manipulation blocks calcium entry into a presynaptic terminal while a spike still reaches it. Which immediate step in the common pathway is most directly impaired?
- ☐ The observer's ability to label the postsynaptic cell.
- ☐ Calcium-triggered vesicle fusion and transmitter release.
- ☐ Every electrical signal throughout the whole brain.
9. Compound B binds receptor R, does not activate it, and prevents agonist A from producing its response there. Which stated action fits?
- ☐ Reuptake inhibition.
- ☐ Direct receptor agonism.
- ☐ Receptor antagonism.
10. After a compound is added, extracellular transmitter increases. No target-binding or transporter measurement is available. What follows?
- ☐ The compound definitely directly activates the transmitter's receptor.
- ☐ The observation does not uniquely identify the mechanism.
- ☐ The compound must be a receptor antagonist.

1. blocking serotonin reuptake

Inhibition of serotonin reuptake targets transporters and can increase extracellular availability. This does not by itself establish direct serotonin-receptor agonism.

2. dopamine

Dopamine-producing neurons in the substantia nigra are affected. This is important for motor symptoms, but is not the whole disease mechanism.

3. True

Rate is one possible code under the stated model. This does not claim an unlimited rate increase for every stronger input or exclude other neural codes.

4. refractory

Refractory is the missing word. Sodium-channel inactivation is central to the absolute phase. The relative phase allows another spike with stronger depolarisation.

5. A graded potential changed.

Local graded signals vary in amplitude. They are distinct from the standard all-or-none model of an axonal spike.

6. Relative refractory phase.

A spike is possible during the relative phase but requires greater depolarisation. The absolute phase prevents another spike.

7. Local current spreads between nodes and action potentials are regenerated at nodes.

Nodes contain the membrane machinery for regenerative spikes; the intervening myelin changes current spread. Conduction along the axon is electrical.

8. Calcium-triggered vesicle fusion and transmitter release.

The intervention targets the release trigger at this terminal. It does not establish a global failure of all neural signalling.

9. Receptor antagonism.

B blocks the agonist's action at the receptor without activating it. No transporter mechanism was stated.

10. The observation does not uniquely identify the mechanism.

Release, uptake or breakdown changes could affect availability. A downstream observation alone does not specify the drug's target.