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DEPARTMENT: Pharmacology

Matric no: 17/MHS07/012

Course code: Pha 308

PHA 308 MOCK TEST ANSWERS

NEUROPHARMACOLOGY

1. Blood-brain barrier and Cerebrospinal barrier system
	* 1. Synthesis of the neurotransmitter. This can take place in the [cell body](https://en.wikipedia.org/wiki/Cell_body), in the axon, or in the [axon terminal](https://en.wikipedia.org/wiki/Axon_terminal%22%20%5Co%20%22Axon%20terminal).
		2. Storage of the neurotransmitter in storage granules or vesicles in the axon terminal.
		3. Calcium enters the axon terminal during an action potential, causing [release](https://en.wikipedia.org/wiki/Exocytosis%22%20%5Co%20%22Exocytosis) of the neurotransmitter into the synaptic cleft.
		4. After its release, the transmitter binds to and activates a receptor in the postsynaptic membrane.
		5. Deactivation of the neurotransmitter. The neurotransmitter is either destroyed enzymatically, or taken back into the terminal from which it came, where it can be reused, or degraded and removed
2. α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and N-methyl-d-aspartate (NMDA) for glutamate
3. Glycine and GABA (γ-Amino butyric acid) opens Cl- channels, resulting in post-synaptic target hyperpolarization.
4. Acetylcholine
5. Dopamine
6. Serotonin and Dopamine
7. Parkinson’s diseases
8. Sedatives and Hypnotics
9. Anticonvulsant, Skeletal muscle relaxant, Amnesic, Sedatives, Hypnotics, and Anxiolytic.
10. The monoamine hypothesis of depression predicts that the underlying pathophysiologic basis of depression is a depletion in the levels of serotonin, norepinephrine, and/or dopamine in the central nervous system. that depression is caused by a functional deficit of the monoamine transmitters, noradrenaline and 5-hydroxytryptamine (5-HT) at certain sites in the brain, while mania results from a functional excess.
11. **T T T T**
12. **T T F F**
13. **T F T T**
14. **T F F T**