University of Central Florida (UCF) ZOO3744 Neurobiology Practice Exam 2 (Sample)

Study Guide



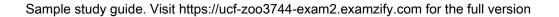
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Questions



- 1. What is long-term potentiation (LTP)?
 - A. A process that weakens synaptic connections
 - B. A mechanism that strengthens synaptic connections
 - C. A type of reflex action
 - D. A neurodevelopmental disorder
- 2. How does the paracrine signaling from the postsynaptic cell affect neurotransmitter release?
 - A. It enhances the effects of serotonin
 - B. It increases the release of glutamate
 - C. It inhibits neurotransmitter synthesis
 - D. It triggers the degradation of neurotransmitters
- 3. What occurs during the process of sensory adaptation?
 - A. Sensory receptors become more responsive to stimuli
 - B. Sensory receptors become less responsive to constant stimuli
 - C. The brain increases its sensitivity to changes in stimuli
 - D. Sensory pathways are altered to reduce input
- 4. What is the primary function of the basal ganglia?
 - A. Regulation of sleep patterns
 - B. Coordination and execution of voluntary movements
 - C. Processing of sensory information
 - D. Emotion regulation
- 5. What does the central nervous system (CNS) consist of?
 - A. The brain and spinal cord
 - B. The brain and peripheral nerves
 - C. The spinal cord and cranial nerves
 - D. The brain, spinal cord, and all peripheral nerves

- 6. Which of the following is an example of a neurotransmitter peptide?A. AcetylcholineB. DynorphinC. GABA
- 7. What role does acetylcholine play in neurotransmission?
 - A. Facilitates muscle contractions at neuromuscular junctions
 - B. Inhibits the release of other neurotransmitters
 - C. Modulates pain responses in the brain
 - D. Stimulates the production of cortisol
- 8. Which of the following phases follows depolarization in an action potential?
 - A. Resting phase

D. Dopamine

- B. Repolarization
- C. Hyperpolarization
- D. Restoration
- 9. Which peptide is specifically involved in appetite regulation?
 - A. Enkephalins
 - B. Neuropeptide Y
 - C. Substance P
 - D. CCK
- 10. How does the peripheral nervous system regenerate after injury?
 - A. By forming scar tissue around the injury
 - B. By activation of glial cells
 - C. By the activation of Schwann cells
 - D. By producing new neurons from the spinal cord

Answers



- 1. B
- 2. B
- 3. B
- 4. B
- 5. A
- 6. B
- 7. A
- 8. B
- 9. B
- 10. C

Explanations



- 1. What is long-term potentiation (LTP)?
 - A. A process that weakens synaptic connections
 - B. A mechanism that strengthens synaptic connections
 - C. A type of reflex action
 - D. A neurodevelopmental disorder

Long-term potentiation (LTP) is a fundamental process that underlies synaptic plasticity, which is the ability of synapses to strengthen or weaken over time in response to increases or decreases in their activity. Specifically, LTP refers to the long-lasting enhancement in signal transmission between two neurons that occurs when they are repeatedly stimulated simultaneously. This strengthening of synaptic connections is believed to be a critical mechanism for learning and memory formation. During LTP, repeated stimulation can lead to a variety of cellular changes, including an increase in the number of neurotransmitter receptors at the postsynaptic membrane or changes in the structure of the synapse itself. This enhanced synaptic efficacy means that subsequent stimulation will produce a stronger postsynaptic response than before. The concept of LTP is crucial in understanding how experiences can lead to lasting changes in the neural circuitry of the brain, allowing for the storage of memories and the learning of new information.

- 2. How does the paracrine signaling from the postsynaptic cell affect neurotransmitter release?
 - A. It enhances the effects of serotonin
 - B. It increases the release of glutamate
 - C. It inhibits neurotransmitter synthesis
 - D. It triggers the degradation of neurotransmitters

Paracrine signaling refers to the process by which a cell produces a signal to induce changes in nearby cells. In the context of neurotransmitter release, when a postsynaptic cell is activated, it can release signaling molecules that influence the presynaptic cell's activity. The correct answer indicates that paracrine signaling from the postsynaptic cell increases the release of glutamate. Glutamate is a principal excitatory neurotransmitter in the brain. When released from the postsynaptic cell, it acts on the presynaptic cell, enhancing its excitability and potentially prompting greater release of neurotransmitters like glutamate in subsequent signaling events. This mechanism allows for modulation and amplification of synaptic transmission, contributing to processes such as synaptic plasticity, which is critical for learning and memory. In this case, the interaction highlights how communication between neurons is not only a direct effect through synaptic transmission but also involves complex feedback loops that influence overall neurotransmitter dynamics in the neuronal circuit. Understanding these interactions is fundamental in neurobiology, particularly in addressing how synaptic efficacy can be modified under different physiological and pathological conditions.

3. What occurs during the process of sensory adaptation?

- A. Sensory receptors become more responsive to stimuli
- B. Sensory receptors become less responsive to constant stimuli
- C. The brain increases its sensitivity to changes in stimuli
- D. Sensory pathways are altered to reduce input

During the process of sensory adaptation, sensory receptors become less responsive to constant stimuli. This phenomenon allows the nervous system to prioritize novel stimuli that may indicate changes in the environment or signal potential threats, rather than continuously reacting to unchanging conditions. When a stimulus is constant and unchanging, sensory receptors gradually decrease their rate of firing over time. This results in a diminished perception of that stimulus, which helps avoid sensory overload and allows individuals to focus on more relevant changes in their surroundings. For example, when you first enter a room with a strong odor, you may notice it intensely, but over time, you become less aware of it as your receptors adapt. In contrast, the other options describe processes or effects that do not align with the definition of sensory adaptation. Sensory receptors do not become more responsive to stimuli when adaptation occurs, nor does the brain increase its sensitivity to unchanging stimuli. Additionally, while sensory pathways can adjust for various reasons, the specific mechanism of sensory adaptation is characterized by a decrease in receptor responsiveness to constant stimuli rather than an alteration of the pathways.

4. What is the primary function of the basal ganglia?

- A. Regulation of sleep patterns
- B. Coordination and execution of voluntary movements
- C. Processing of sensory information
- D. Emotion regulation

The primary function of the basal ganglia is indeed the coordination and execution of voluntary movements. This group of nuclei located deep within the cerebral hemispheres plays a crucial role in motor control. The basal ganglia are involved in the planning and smoothing of movements, helping to initiate and facilitate voluntary motor actions while also inhibiting unwanted movements. Through connections with the motor cortex and other brain regions, the basal ganglia integrate information regarding movement and contribute to the initiation of action, which is vital for activities requiring coordinated muscle movement, precise timing, and posture. Their role in motor learning also underscores their importance in adapting movements based on experience. While the other functions mentioned in the choices are significant in their own contexts, they do not accurately define the core responsibility of the basal ganglia. For example, regulation of sleep patterns is primarily associated with brain structures such as the hypothalamus. Processing of sensory information generally involves other brain areas such as the thalamus and sensory cortices. Emotion regulation is more closely tied to the limbic system, including structures like the amygdala and hippocampus. Thus, the correct choice aligns with the established understanding of the basal ganglia's pivotal role in motor function.

5. What does the central nervous system (CNS) consist of?

- A. The brain and spinal cord
- B. The brain and peripheral nerves
- C. The spinal cord and cranial nerves
- D. The brain, spinal cord, and all peripheral nerves

The central nervous system (CNS) specifically consists of the brain and spinal cord. This definition is fundamental in neuroanatomy, as the CNS is responsible for processing and integrating information, coordinating body functions, and facilitating communication throughout the body. The brain serves as the control center, interpreting sensory information, making decisions, and initiating motor commands. The spinal cord acts as a conduit for signals between the brain and the rest of the body. Together, they form the core structure that enables complex behaviors, reflexes, and higher cognitive functions. In contrast, peripheral nerves, which connect the CNS to the limbs and organs, are part of the peripheral nervous system (PNS). The cranial nerves, although they originate in the brain, are also considered part of the peripheral nervous system, as they extend beyond the CNS. Understanding this distinction is crucial for grasping the overall organization and function of the nervous system.

6. Which of the following is an example of a neurotransmitter peptide?

- A. Acetylcholine
- B. Dynorphin
- C. GABA
- D. Dopamine

Dynorphin is indeed classified as a neuropeptide, which is a specific type of neurotransmitter peptide. Neuropeptides are larger than traditional neurotransmitters and consist of chains of amino acids. They play critical roles in modulating a wide range of biological functions, including pain regulation, stress response, and emotional processing. Dynorphin, in particular, is known for its role in pain perception and its involvement in the body's response to stress. In contrast, acetylcholine, GABA (gamma-aminobutyric acid), and dopamine are classified as classical neurotransmitters. Acetylcholine is essential for muscle activation and cognitive functions, GABA serves primarily as an inhibitory neurotransmitter in the central nervous system, and dopamine is involved in reward, motivation, and several neurological functions. These neurotransmitters typically function by acting on specific receptors to transmit signals across synapses, but they do not fall within the peptide category. This distinction is crucial in understanding the different types of signaling molecules in the nervous system and their varying mechanisms of action.

7. What role does acetylcholine play in neurotransmission?

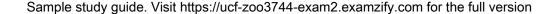
- A. Facilitates muscle contractions at neuromuscular junctions
- B. Inhibits the release of other neurotransmitters
- C. Modulates pain responses in the brain
- D. Stimulates the production of cortisol

Acetylcholine is a critical neurotransmitter in both the central and peripheral nervous systems, serving a vital function in neurotransmission, especially at the neuromuscular junction. When released from motor neurons, acetylcholine binds to receptors on muscle cells, leading to depolarization of the muscle membrane. This process initiates a cascade of events that ultimately results in muscle contraction. In the context of neuromuscular transmission, acetylcholine's role is fundamentally about stimulating contraction by facilitating communication between neurons and muscles. When acetylcholine binds to its receptors on the muscle fibers, it triggers an influx of sodium ions, leading to an action potential that causes the muscle to contract. This mechanism is essential for all voluntary movements in the body, making acetylcholine indispensable in the neuromuscular junction. Other options, while related to different functions of other neurotransmitters or hormones, do not accurately capture the primary role of acetylcholine in neurotransmission as it pertains specifically to muscle contractions.

8. Which of the following phases follows depolarization in an action potential?

- A. Resting phase
- B. Repolarization
- C. Hyperpolarization
- D. Restoration

Following depolarization in an action potential, the next phase is repolarization. During depolarization, the membrane potential of the neuron becomes more positive due to the influx of sodium ions (Na+) through voltage-gated sodium channels. Once the membrane reaches its peak depolarization, these sodium channels begin to close, and voltage-gated potassium channels open. In the repolarization phase, potassium ions (K+) exit the neuron, causing the membrane potential to decrease back toward the resting membrane potential. The efflux of potassium counteracts the previous influx of sodium, leading to a return to a more negative membrane potential. This transition is essential for resetting the neuronal membrane and preparing it for the next potential action potential, following the all-or-nothing principle. The repolarization is critical in ensuring that the neuron can fire in a single direction and maintain a proper signal transmission in response to stimuli.



- 9. Which peptide is specifically involved in appetite regulation?
 - A. Enkephalins
 - B. Neuropeptide Y
 - C. Substance P
 - D. CCK

Neuropeptide Y (NPY) plays a crucial role in appetite regulation by influencing energy balance and food intake. It is produced primarily in the brain, specifically within the hypothalamus, which is a key region involved in regulating hunger and satiety. NPY acts to stimulate appetite and promote food intake. When energy levels are low, such as during fasting, the release of NPY increases, signaling the body that it needs to consume more food. This feedback mechanism is essential for maintaining energy homeostasis. Furthermore, NPY interacts with other hormones and neuropeptides involved in appetite control, making it a central player in the complex network of signals that govern feeding behavior. Understanding its function is critical for exploring potential treatments for eating disorders and obesity, where appetite regulation becomes dysfunctional. Other peptides, such as enkephalins, substance P, and Cholecystokinin (CCK), have their own unique roles but are not as directly involved in the regulation of appetite as NPY. Enkephalins are primarily associated with pain modulation and reward pathways, substance P is linked to pain and stress responses, and CCK is involved in digestion and satiety but does not exert the strong appetite-stimulating effects that characterize NPY

- 10. How does the peripheral nervous system regenerate after injury?
 - A. By forming scar tissue around the injury
 - B. By activation of glial cells
 - C. By the activation of Schwann cells
 - D. By producing new neurons from the spinal cord

The regeneration of the peripheral nervous system (PNS) after injury largely hinges on the role of Schwann cells, which are a type of glial cell that supports and insulates nerve fibers in the PNS. When a peripheral nerve is injured, Schwann cells play a crucial role in the repair process. They not only help in the disintegration of damaged axons but also create a supportive environment for regeneration. Following an injury, Schwann cells proliferate and align themselves along the pathway of the damaged axon. They form a structure known as a "regeneration tube," which guides the regrowth of the axon toward its target. This is critical because the environment provided by the Schwann cells encourages axonal growth through the secretion of growth factors and extracellular matrix proteins, which are essential for the regeneration process. Furthermore, Schwann cells also remove debris from the site of injury, which is vital for a conducive healing environment. Their ability to facilitate remyelination of newly formed axons is another critical component of effective regeneration in the PNS. Therefore, the activation of Schwann cells is the primary mechanism through which the peripheral nervous system can regenerate after an injury, effectively restoring function to damaged nerves.