

**LOCATION: SWANSEA**

**LLEOLIAD: ABERTAWE**

**LEVEL 5**

**LEFEL 5**

**EXAMINATION PERIOD**  
**MAY 2025**

**CYFNOD ARHOLIADAU**  
**MAI 2025**

**MODULE CODE: ECPS5018**  
**THE BRAIN: BIOLOGY AND COGNITION**

**TIME: 2 HOURS**

**THIS PAPER CONSISTS OF THREE SECTIONS. YOU SHOULD:**

**ANSWER FOUR QUESTIONS OUT OF FIVE IN SECTION A**

**ANSWER THREE QUESTIONS OUT OF FIVE IN SECTION B**

**ANSWER ONE QUESTION OUT OF FIVE IN SECTION C**

**ALL answers must be written in your exam booklet.**

Please clearly indicate the section and question number of each question attempted.

*Equipment or materials required – None*

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## SECTION A: ANSWER FOUR QUESTIONS OUT OF FIVE

1. List any five major **external** features of a neuron (5 marks)

- Any five of the following:
  - Cell membrane,
  - Dendrites,
  - Axon Hillock,
  - Myelin,
  - Nodes of Ranvier,
  - Cell body,
  - Buttons,
  - Synapse.

2. List any five neurotransmitters (5 marks)

- Any five of the following:
  - Glutamate,
  - Aspartate,
  - Glycine,
  - Gamma-aminobutyric acid (GABA),
  - Dopamine,
  - Epinephrine
  - Norepinephrine,
  - Serotonin,
  - Acetylcholine,
  - Nitric oxide,
  - Carbon monoxide,
  - Anandamide,
  - Pituitary peptides,
  - Hypothalamic peptides,
  - Brain-gut peptides,
  - Opioid peptides,
  - Miscellaneous peptides

3. List any five sub-types of long-term memory (5 marks)

- Any five of the following
  - Episodic
  - Semantic
  - Implicit
  - Explicit
  - Procedural
  - Declarative

4. List the five layers of neurons that compose the retina (5 marks)

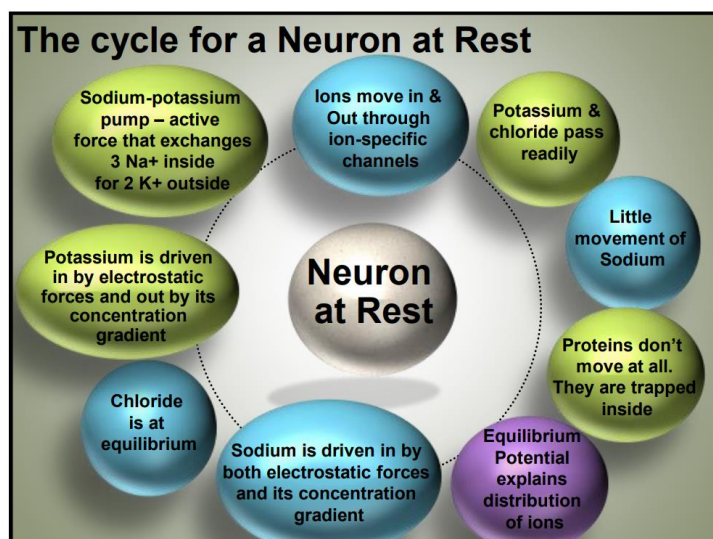
- Rods
- Cones
- Horizontal cells
- Amacrine cells
- Retinal Ganglion cells

5. List any five symptoms of epilepsy (5 marks)

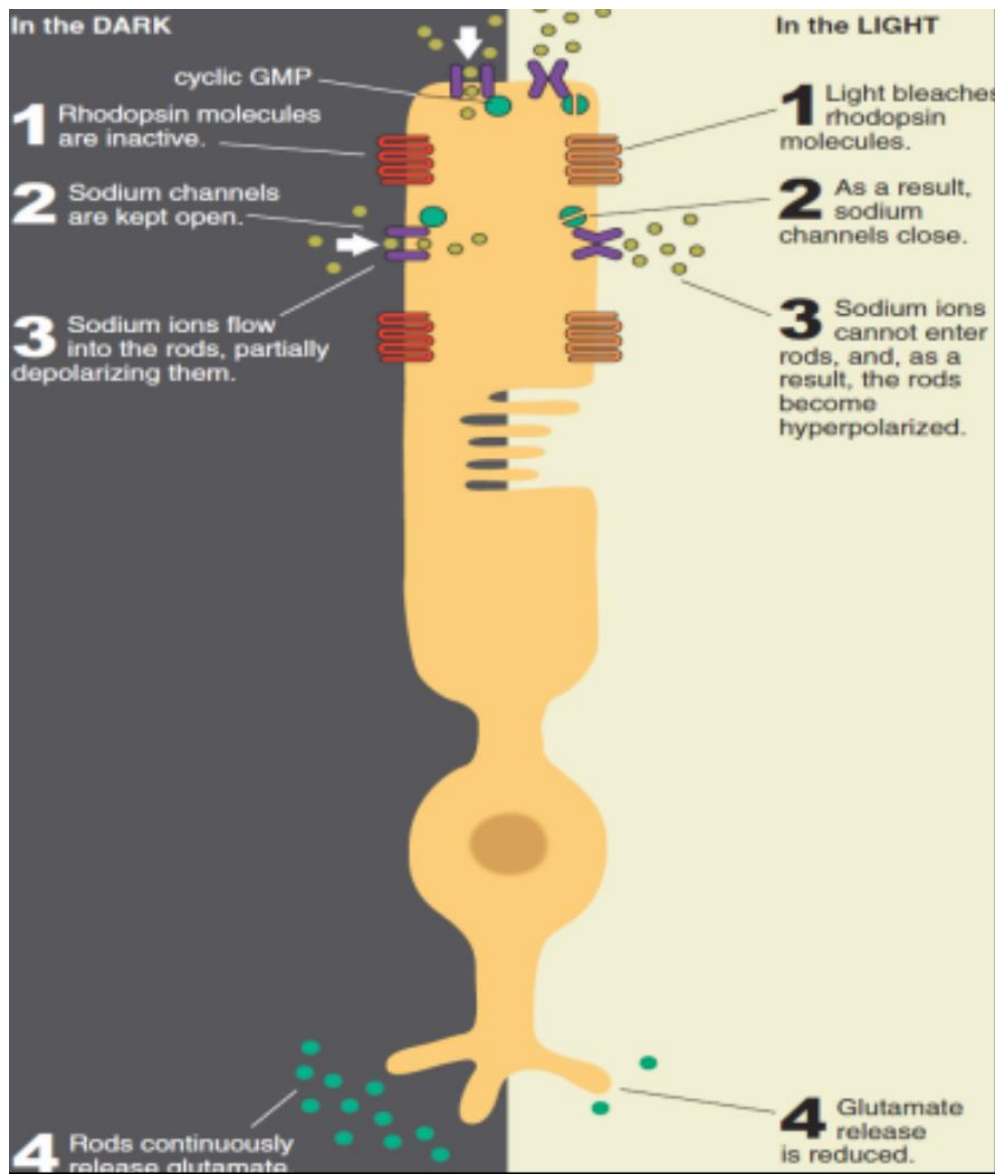
- Any five of the following
  - Seizure
  - Convulsions
  - Tremors
  - Rigidity
  - Loss of balance
  - Loss of consciousness
  - Subtle change to thought
  - Subtle change to mood
  - Subtle change to cognition
  - Subtle change to behaviour
  - Psychological change
  - Aura
  - Change in smell
  - Hallucination
  - Compulsions
  - Repetitive behaviour (automatisms)

## SECTION B: ANSWER THREE QUESTIONS OUT OF FIVE

1. Describe the cycle for a neuron at rest (10 marks)



2. Describe the response of the rods when in the light and the dark (10 marks)



3. Compare and contrast Functional magnetic resonance imaging (fMRI) with Electroencephalography (EEG) (10 marks)

fMRI provides images of brain structure and activity, uses strong magnetic field, two properties of oxygenated blood allow it to work. 1. Active areas of the brain take up more oxygenated blood than actually needed. 2. Oxygenated blood has magnetic properties through the iron. Structure is imaged using waves emitted by hydrogen ions, Function is imaged using signal created from interaction between oxygen and iron in the blood: Blood Oxygenated Level Dependent (BOLD) signal, Good spatial and temporal resolution, Produces 3D images of activity over the whole brain, But only provides indirect measure of neural activity through BOLD, Also, the 2 to 3 seconds to create image is too slow to capture neural responses e.g., action potentials that happen in milliseconds.

Electroencephalography (EEG) as techniques for studying the brain.

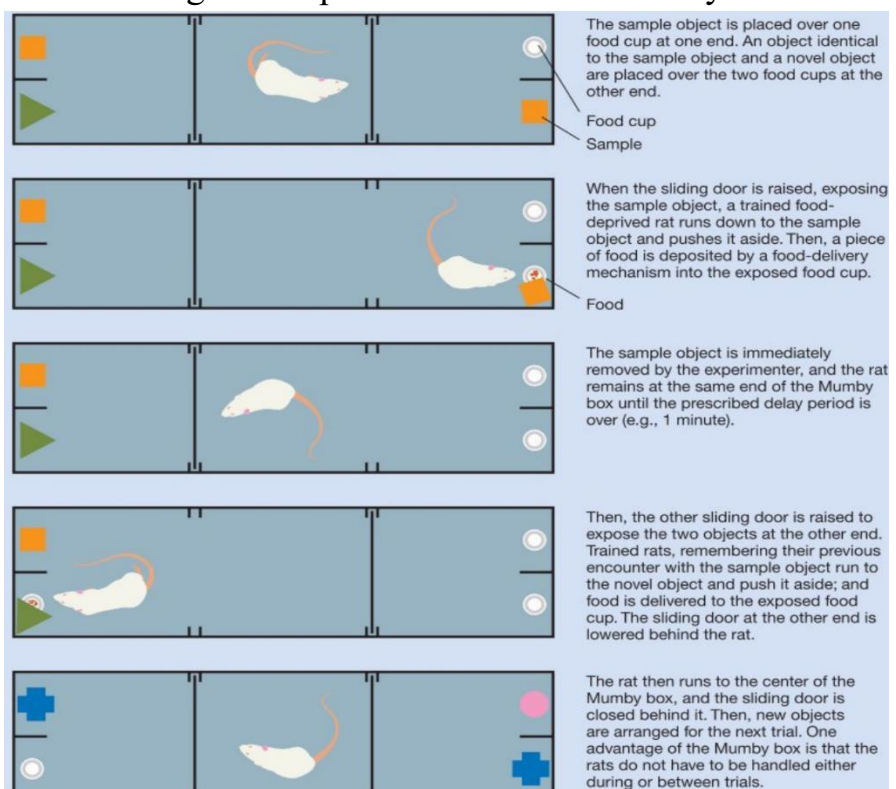
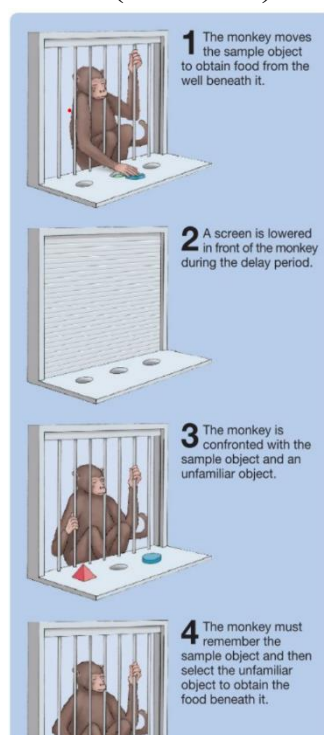
EEG measures gross electrical activity of brain with electrodes attached to scalp, including action potentials and postsynaptic potentials. Includes wave, ERP, & combined with MRI.

Main difference = one shows image (structure/activity), other informs of electrical activity.

#### 4. Describe the similarities and differences between somatosensory and neuropathic pain (10 marks)

- Similar:
  - Adaptive and needed,
  - No obvious cortical representation,
  - Anterior cingulate gyrus involved in emotional component
  - Descending pain control
- Different:
  - Somatosensory pain:
    - Clear pain stimulus causes experience of pain (e.g., broken leg)
    - Painful stimuli can elicit pain in primary & secondary somatosensory cortex, but removal not associated with changes in threshold for pain.
  - Neuropathic pain
    - Severe chronic pain in absence of recognizable pain stimulus
    - Exact mechanism unknown but likely from pathology of nervous system linked to injury
    - Some evidence for aberrant (diverging from normal function) glial cell signals triggering neural pain pathways

#### 5. Describe the delayed nonmatching-to-sample tests used with monkeys and rats (10 marks)



- Intact well-trained monkeys perform correctly 90% of time
- Monkeys with bilateral medial temporal lobe lesions had major deficit.
- With rats – conducted in mummy box and through electrical lesion rather than aspiration as hippocampus is too small.

## SECTION C: ANSWER ONE QUESTION OUT OF FIVE

### 1. Critically discuss the development of the dopamine theory of schizophrenia and the limitations of the current version (50 marks)

Discovery of the First Antipsychotic Drugs • Chlorpromazine: originally developed as antihistamine to counteract swelling from surgery • Surgeons noticed its calming effect, so given to calm agitated schizophrenics & to activate emotionally blunt schizophrenics • Does not cure schizophrenia, just reduces severity. • Reserpine: also found to be effective, but no longer used due to dangerous side effects. • Although chemical structure of both were different, their effects were similar (i.e., no effect for 2-3 weeks, also Parkinson-like motor effects were seen), suggesting they were both working through the same mechanisms. Dopamine Theory of Schizophrenia • 1960 – link between dopamine and Parkinson's disease established • Striatum depleted of dopamine, suggesting disruption of dopaminergic transmission might produce Parkinson's disease. • Due to this & already established relationship between symptoms of Parkinson's & anti-schizophrenic effects of chlorpromazine and reserpine, it was suggested anti-schizophrenic drug effects are produced in same way. • Led to development of dopamine theory – too much dopamine causes schizophrenia & conversely, anti-schizophrenic drugs exert effects by decreasing dopamine levels. • Supported by fact that: • Reserpine depletes brain of dopamine and other monoamines by making vesicles leaky • Chlorpromazine antagonizes dopamine activity by binding and blocking dopamine receptors – So, both antagonize transmission of dopamine but in different ways. Lack of activity at postsynaptic receptors sends feedback signal to presynaptic cells to increase release of dopamine. • This led to revision of the dopamine theory • Instead of high dopamine levels, the main factor is the high levels of activity at dopamine receptors. • 1970s: degree to which various anti-schizophrenia drugs bind to dopamine receptors was assessed. • Radioactively labelled dopamine added to samples of dopamine-receptor-rich neural membrane obtained from calf striatum • Unbound dopamine molecules rinsed away & amount of radioactivity left was measured to obtain number of dopamine receptors. Drugs ability to block binding of radioactive dopamine was assessed - assumed drugs with high affinity for dopamine receptors would leave fewer sites available for the dopamine. • Chlorpromazine & others had high affinity for dopamine receptors whereas ineffective drugs had low affinity..... • Except for haloperidol: very potent but had low affinity. • Solution: came from discovery that dopamine binds to more than one receptor subtype – 5 have been identified. • Chlorpromazine & others bind effectively to D1 & D2 receptors, whereas haloperidol bind effectively to D2 but not to D1. • This discovery led to further revision suggesting schizophrenia is caused by hyperactivity at D2 receptors rather than dopamine receptors in general. Limitations of the Dopamine Theory • Four key discoveries about the neural basis of schizophrenia cannot be explained by the D2 theory. 1. Other receptors are involved: glutamate, GABA, serotonin. • Evidence from studies on LSD, which produce schizophrenia-like symptoms by acting on serotonin & glutamate receptors. • Atypical neuroleptics, such as clozapine has affinity for D1, D4, & serotonin receptors but not for

D2. 2. Neuroleptics block D2 receptors within hours but it takes weeks to alleviate symptoms. • Time difference suggests blockage of D2 receptors is not only aspect involved. Limitations of the Dopamine Theory 3. Schizophrenia is associated with widespread brain damage, & meta-analysis of studies has shown reduced volume in 50 different brain regions. • Two aspects about pattern of brain damage observed that causes problems for the dopamine theory: A. Little evidence of damage to dopamine circuits B. Theory does not explain why there is diffuse damage 4. Neuroleptics only marginally effective. • If dopamine theory was correct neuroleptics should be effective to treat all patients. • They only help with some symptoms not others: • Conventional neuroleptics (D2 blockers) are effective for positive symptoms, while negative symptoms might be caused by brain damage.

## 2. Critically discuss the effectiveness of medication in treating affective disorders (50 marks)

Four major classes of drugs used in the treatment of affective disorders 1. Monoamine oxidase inhibitors (MAOIs): Iproniazid • Increases levels of monoamines by preventing their breakdown by oxidase enzymes in cytoplasm of neuron • Must avoid foods high in tyramine – “cheese effect” • Normally these foods have little effect on blood pressure because tyramine is metabolized in the liver by MAO, but people who take MAO inhibitors & eat food containing tyramine run risk of stroke caused by surges in blood pressure. Tricyclic antidepressants – Imipramine • Chemical structure includes 3 rings of atoms • They block reuptake of serotonin and norepinephrine – Thus, increasing levels of these in the brain • They are thought to be safer than MAOIs 3. Selective serotonin-reuptake inhibitors (SSRIs) • They are serotonin agonists that exert effects by blocking the reuptake of serotonin from synapses. • Include Prozac, Paxil, Zoloft, and others • No more effective than tricyclics, but side effects are few and they are effective at treating other disorders 4. Selective norepinephrine-reuptake inhibitors (SNRIs): effective & block reuptake of norepinephrine. Hollon et al. (2002) compared efficacy of various pharmacology treatments for depression and found they were about the same for MAOIs, tricyclics, & SNRIs: • ~50% of clinically depressed patients improved • But a control group showed a 25% improvement • So, really only 25% of the depressed group were helped. • Kirsch et al. (2008): meta-analysis indicated that placebo was about 82% as effective as anti-depressants in severely depressed individuals • Drugs even less effective for mild to moderately depressed individuals. MRI of bipolar patients shown reductions in overall brain size and particularly the amygdala, cingulate cortex, and prefrontal cortex. • But certain brain structures found to be of reduced size in one study is of normal size in other studies. • Not all patients with bipolar suffer from same disorder? • Two structures are found to be abnormal in many studies: amygdala and anterior cingulate cortex. • Connections between these also disrupted in patients with affective disorders. Monoamine theory of depression: Suggests depression is associated with underactivity at serotonergic and noradrenergic synapses. Support provided by autopsy: serotonin and norepinephrine receptors more numerous in brains of deceased clinically depressed individuals who had not had pharmacological treatment whilst alive, which implicates a deficit in monoamine release. • When not enough amount of a neurotransmitter is released at a synapse, there are usually compensatory increases in the number of receptors for that neurotransmitter – called Up-regulation. • However, overall support for the monoamine theory of depression is weak and not all patients respond to monoamine agonists.

### 3. Critically discuss the suitability of animal models in explaining human anxiety (50 marks)

- Assessment of Species-common behaviours: those displayed by all members of the species: e.g., grooming, eating, building nests, etc.). • Main tests

Open-field test: place animal in empty chamber & record activity (does it move around or stay close to wall, how much droppings it produces, etc.).

Tests of Sexual Behaviour.

Tests of Aggressive and Defensive Behaviour: Colony-intruder paradigm: examine no. of combat encounters between dominant alpha male of established colony & smaller male intruder. • Dominant alpha is aggressive: biting, putting hair upright. • Intruder is defensive: goes up on back legs to defend from biting & tries to push alpha away with its front paws. • Elevated plus maze: a maze mounted 50cm from the ground, two arms of maze have sides while other two do not • Amount of time rat spends in closed arms rather than the open edge arms is compared. • Giving rat anti-anxiety drugs increases amount of time spent in open arms of maze: used for understanding usefulness of anti-anxiety drugs in humans?

Traditional Conditioning Paradigms: • We can infer about the sensory, motor, motivational, and cognitive state of an animal from its ability to learn and perform various responses. • Pavlovian conditioning (pairing an unconditioned stimulus with a conditioned stimulus) • Operant conditioning & reinforcement.

Semi-natural Animal Learning Paradigms:

Conditioned Taste Aversion: pairing something that makes an animal ill with food so they learn to avoid it. • This really challenged traditional learning paradigms because taste aversion can be established after a single trial.

Conditioned Defensive Burying: give rat a single aversive stimulus (e.g., a shock/blast of air) delivered from an object. • They then spray their bedding/hay at the object to cover it.

- Anti-anxiety drugs decrease amount of burying behaviour.

### 4. Critically discuss the similarities and differences between human and animal communication (50 marks)

Should consider Aitchison's (1983) four features unique to humans:

Semanticity, Displacement, Structure Dependence, Creativity

- Similar
  - Both use symbols to represent events in the world
  - Rely on sender and receiver sharing representation
  - Primates can learn meaning of some arbitrary symbols for communication (as humans do)
- Different
  - Animals cannot communicate beyond 'here' & 'now' (abstract)
  - Syntax in language more human
  - Recursion is a human language sophistication
  - Semantics: difficult to fully test but possibly more human
  - Qualitative and Quantitative differences

5. Critically discuss what the formal assessment of H.M. tells us about the structure of memory (50 marks)

The formal methods of assessment should be explained in detail, along with details of his performance on these tasks:

Digit span + 1

Block-tapping memory-span task

Mirror-drawing

Rotary-Pursuit

Incomplete picture

Pavlovian condition

As well as the three major scientific contributions:

1. Medial temporal lobes are involved in memory
2. STM, remote memory, and LTM are distinctly separate – H.M. is unable to move memories from STM to LTM, a problem with memory consolidation
3. Memory may exist but not be recalled – as when H.M. exhibits a skill he does not know he has learned • Difference between explicit (conscious) vs. implicit (unconscious) memories.

**Diwedd/End**