

ECPS5003 Brain, Biology and Cognition
Exam Answers 2020-2021

SECTION A: ANSWER FOUR QUESTIONS OUT OF FIVE

1. List any five major internal features of a neuron (5 marks).
 - Any of the following: Endoplasmic reticulum, Nucleus, Mitochondria, Cytoplasm, Ribosomes, Golgi Complex, Microtubules, Synaptic Vesicle, Neurotransmitters
2. List any five types of agnosia (5 marks).
 - Any of the following: Prosopagnosia, Astereognosia, Asomatognosia, Akinetopsia, any agnosia related to the senses (hearing, tactile, visual, smell, and taste), Semantic agnosia, Phonagnosia, Simultagnosia, Anosognosia, Visuo-spatial, etc.
3. List any five symptoms of schizophrenia (5 marks).
 - Any of the following:
 - Positive symptoms: Delusions, Hallucinations, Inappropriate Affect, Incoherent speech or thought, Odd behaviour, catatonia, etc.
 - Negative symptoms: Affective flattening, Alogia (no speech), Avolition (no motivation), Apathy, Anhedonia (no experience of pleasure), etc.
4. List the five main identified functions of consciousness (5 marks).
 1. Associated with perceiving the environment
 2. Crucial role in social communication
 3. Role in controlling our actions
 4. Allows us to think about events and issues far removed from the present
 5. Integrating and combining numerous types of information
5. List any five structures of the eye (5 marks).
 - Any of the following: Ligament, Eye muscle, Iris, Pupil, Lens, Cornea, Fovea, Blind spot, Ciliary muscles, Sclera, Retina, Optic nerve.

SECTION B: ANSWER THREE QUESTIONS OUT OF FIVE

6. Describe five main forms of forgetting from long-term memory (10 marks).

- Any of the following: Trace-dependent Forgetting, Interference, Repression, Motivated Forgetting, Directed Forgetting, Cue-dependent Forgetting, Encoding Specificity, State-dependent Forgetting, Context-dependent Forgetting, Memory biases, Misleading information – would need to give a brief explanation of it.

7. Explain how the human brain is protected (10 marks).

- Physical Protection: Skull – CNS encased in bone & covered by: 3 Meninges – Dura mater – tough outer membrane – Arachnoid membrane – quite fine & web-like – Pia mater – inner & delicate membrane that adheres to CNS surface.
- Cerebrospinal fluid (CSF) - serves as cushion by filling in space underneath arachnoid membrane, central canal of the spinal cord, and the cerebral ventricles of the brain. 4 large internal chambers of the brain: two lateral, third, and fourth.
- Chemical protection: The blood-brain barrier: – Because blood can contain many toxins, the body needs to stop these toxins passing in to the brain. The cells of the blood vessel walls in the brain are so tightly packed together it forms a barrier which stops any molecules from passing through.

8. Describe the components and function of the limbic system (10 marks).

- Circuit of midline structures that circle the thalamus. Involved in regulating motivational behaviours: the four Fs: fleeing, fighting, feeding, and Sexual behaviour. Includes the following: Amygdala - emotion (almond shaped nucleus in anterior temporal lobe), Hippocampus – memory for locations (three major layers in the medial edge of the cerebral cortex as it folds back on itself in the medial temporal lobe), Cingulate cortex (large strip of cortex in the cingulate gyrus on the medial surface of the cerebral hemispheres and encircles the dorsal thalamus), Fornix (major tract that encircles the dorsal thalamus), Septum (midline nucleus at anterior tip of the cingulate cortex), and Mammillary body (pair of spherical nuclei located on inferior surface of the hypothalamus).

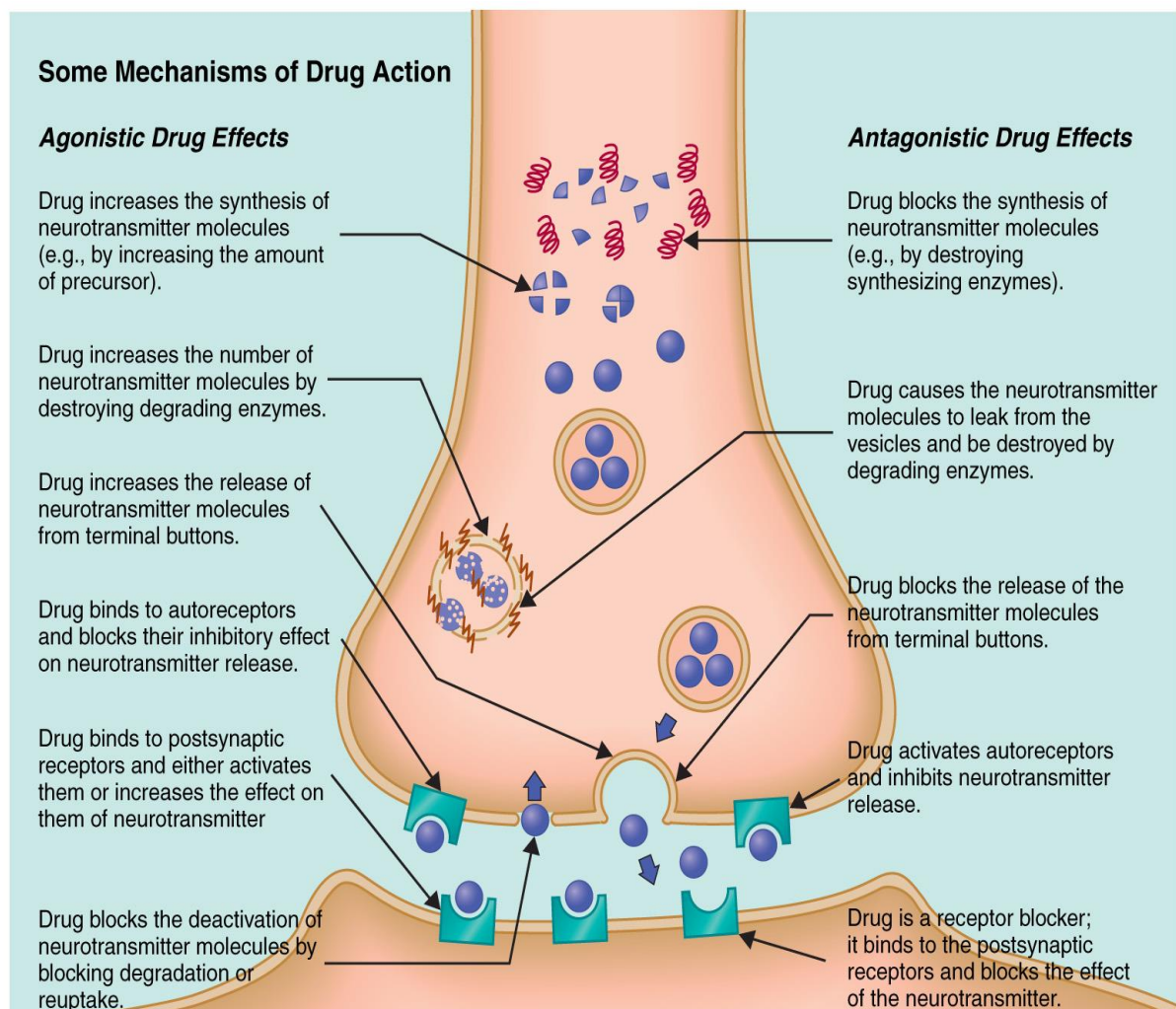
9. Describe the four features Aitchison (1983) argued were unique to human language (10 marks).

- Semanticity: Human language – each word has at least one specific meaning
 - Vervet monkeys – each call elicits a different response – is this semanticity?
- Displacement: Across Time / space ; true / hypothetical situations
 - Step away from current true state of affairs
 - We can talk about what ‘might’ be true or a time that has not yet been reached (e.g., “where is the exam?”)

- Abstract concepts represented (e.g., Love, justice, truth)
- Structure Dependence: Human language is characterised by a series of symbols that are abstract
 - Words put into units of meaning in which their position in the sentence is important to the meaning generated
 - E.g. “water bird” vs. “bird water”
 - Set of rules that govern how words are combined is syntax (Chomsky, 1960)
- Creativity: Human language allows us to produce utterances that have never been expressed before
 - Novel utterances
 - Flexible and infinitely productive

10. Describe the agonistic and antagonistic effects of drugs on synaptic transmission (10 marks).

- Would need to specify what each does overall and describe their effects on each process.



SECTION C: ANSWER TWO QUESTIONS OUT OF FIVE

11. With reference to the work of Zeki, the binding problem, research from cognitive neuroscience, and to patients with visual deficits, critically discuss the theory of visual functional specialisation in the brain and explain what this means for visual cognition (25 marks).

Zeki's work with animals, distinction of V1,2,3,4,5, brain imaging research (both showing activity in particular brain area thought to deal with specific type of visual stimuli (e.g., present Ps with colour vs. black and white version of film to assess activity in V4), research with patients who have brain damage to specific areas and subsequently show visual deficit linked to that area (e.g., V4 Achromatopsia, V5 Akinestopia) – but also key to discuss when there is damage to area but no corresponding visual deficit. Binding problem – the different types of binding problems (e.g., work of Treisman, Feldman) and evidence from cognitive psychology, including role of attention. Critique that there is no binding problem (DiLollo). Most importantly must always link back to the last part of the question – what does all this mean for visual cognition – how do we see in a coherent whole if different visual properties are processed in different brain areas?

12. Describe the characteristics and causes of epileptic seizures and evaluate the methods used to diagnose and treat the condition (25 marks).

Primary symptom is seizures, but not all who have seizures have epilepsy • Epileptics have seizures generated by brain dysfunction • Affects about 1% of the population • Difficult to diagnose due to the diversity and complexity of epileptic seizures. Types of seizures • Convulsions – motor seizures: involve tremors, rigidity, loss of balance and consciousness. • However, others involve merely subtle changes of thought, mood, or behaviour • Causes • Brain damage • Faulty Genes – over 70 have been linked so far • Faults at inhibitory synapses, particularly GABA where neurons end up just firing in synchronized bursts. • Diagnosis • EEG – electroencephalogram • Why?.....Seizures are associated with high amplitude EEG spikes during an attack. Some epileptics experience psychological changes before a convulsion – known as auras • Such as a smell, hallucination, or feeling • Aura's nature suggests the epileptic focus • Warns epileptic of an impending seizure • Once diagnosis is given, it's usual to assign the epilepsy to one of 2 categories: • Partial epilepsy – does not involve whole brain • Generalized epilepsy – involves the entire brain. Simple • Symptoms are primarily sensory or motor or both • Known as Jacksonian seizures after neurologist Hughlings Jackson • Symptoms spread as epileptic discharge spreads. Complex • Often restricted to the temporal lobes (temporal lobe epilepsy) • Patient engages in compulsive and repetitive simple behaviours (automatisms) • More complex behaviours seem normal & although conscious they do not have recollection of seizure. Grand mal • Loss of consciousness • Loss of Equilibrium • Violent Tonic-clonic convulsions • Rigidity (tonus) • Tremors (clonus) • Petit mal • Not associated with convulsions • Instead there is a disruption of consciousness associated with cessation of ongoing behaviour • The EEG of a petit mal seizure is different from other types: it is a bilateral symmetrical 3 per second spike and wave discharge. No cure,

but frequency and severity can be reduced by antiepileptic medication (Rogawski & Löscher, 2004). However, these types of medication have severe side effects (Toledano & Gil-Nagel, 2008). Brain surgery is performed in grave situations (Guerrini et al., 2008).

13. Critically discuss the causal factors, brain pathology, theories, and treatments of affective disorders (25 marks).

Affective Disorders: Depression & Mania • Depression: normal reaction to loss, abnormal when it persists or has no cause. • Experience anhedonia • Unable to meet essential requirements of daily life • Mania: overconfidence, impulsivity, distractibility, and high energy for no apparent reason. • When extreme it is a clinical problem: wake up in total enthusiasm with nonstop talking that switches rapidly between topics & overconfidence can lead to disaster. Affective disorders: • Psychiatric disorders characterized by mood or emotion disturbances. • Known as mood disorders • Depression and mania • Can be seen as opposite to each other but many depressive patients can experience periods of mania. Types of depression • Unipolar & Bipolar • Reactive (triggered by negative experience) • Endogenous (from within with no apparent cause). Causal Factors in Affective Disorders • Genetic factors contribute to differences. • Twin studies suggest concordance rate of ~60% for identical twins & ~15% for fraternal twins regardless if brought up together or apart. • Concordance higher for bipolar than unipolar However, most research on causal role of experience on affective disorders focuses on stress. Although evidence suggests depression is a stress-related disorder, evidence linking them is sparse. • Stress can trigger attacks in those already depressed but little evidence showing stress increases susceptibility. • Extreme stress is more likely to cause post-traumatic stress disorder (PTSD) than depression. • Two affective disorders where the cause is more apparent due to timings of the attacks: 1. Seasonal Affective Disorder (SAD) • Wintertime depression and lethargy, due to reduction of sunlight • Evidence: incidence rates different between N. America & Florida • Light therapy thought to reduce symptoms 2. Postpartum depression. 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. Diathesis-stress model of depression: Suggests some people inherit a diathesis (genetic susceptibility) incapable of initiating the disorder by itself. • If susceptible people are exposed to stress early in life their systems become permanently sensitized and end up over reacting to mild stressors for the rest of their lives. • Diathesis + stress = depression • Support is indirect: depressed people... • Tend to release more stress hormones. Also, fail the dexamethasone suppression test: • Assesses adrenal gland function by measuring how cortisol levels change in response to injection of dexamethasone.... • which is an exogenous steroid that provides negative feedback to the pituitary gland to suppress the secretion of adrenocorticotrophic hormone. • Weakness of theory: • Evidence of early stress based on recollections of distant past - likely faded or perhaps even distorted • May not be any way of confirming the events recalled. • Sometimes there is no evidence of excessive early stress in the lives of a number of depressed patients. Treatment of depression with brain stimulation. Sleep deprivation • More than 50% of depressed patients improve after one night of sleep deprivation • Depression returns with normal sleep pattern • Not explained by any current theory Exercise • Helps reduce depression • Increases adult hippocampal neurogenesis • Findings suggests that depression may be caused by reduced adult hippocampal neurogenesis.

14. Critically discuss the aetiology, neural basis, animal models, and treatments of anxiety disorders (25 marks).

- Anxiety – chronic fear in the absence of threat
- Anxiety disorder – when anxiety interferes with normal functioning
- Associated with feeling fear & worry
- Accompanied by physiological symptoms – rapid heartbeat (tachycardia), hypertension, sleep disturbances, breathing difficulties, nausea, etc.
 - Generalized anxiety disorders – stress and anxiety in the absence of a causal stimulus

- Phobic anxiety disorders – similar to generalized, but triggered by a particular stimulus (e.g., birds)
- Panic disorders – attacks of extreme fear and stress; may occur with other disorders or alone
- Obsessive-compulsive disorders (OCDs) – obsessive, recurring, uncontrollable thoughts alleviated by compulsive actions
- Posttraumatic stress disorder – pattern of psychological distress following extreme stress
- Aetiology
- Given that anxiety disorders are often triggered by an identifiable stressful event or is focused on a particular object or situation, the role of experience in shaping the disorder seems clear.
- But there is also a significant genetic component with heritability estimates ranging from 30-40% and concordance rates are substantially higher for identical twins than for fraternal twins.
- However, timing & focus of anxiety disorders often reflects the particular experiences of the patient and no specific gene or genes have yet to be linked to anxiety disorders.
- Neural Basis
- Current theories rely on analysis of the effects of drugs.
- The fact that many anxiolytic drugs are agonists at either GABA or serotonin receptors has focused attention on the role of deficits in both GABA and serotonin transmission in anxiety disorders.
- There is also substantial overlap between the brain structures involved in affective and anxiety disorders
- e.g., amygdala and anterior cingulate cortex have been implicated in affective disorders & in anxiety disorders.
- Although both these structures have been implicated the patterns of evidence has differed.....
- Affective disorders = shrinkage to these structures.
- Anxiety disorders = no overall damage.
- Most evidence linking amygdala & anterior cingulate cortex to anxiety disorders has come from functional brain imaging studies
- Abnormal activity in these areas has been recorded during the performance of various emotional tasks
- For example, there is increased activity in the amygdalae of patients with spider phobias when they viewed photographs of spiders.
- Pharmacological Treatments
- Benzodiazepines (Librium, Valium): Also used as hypnotics, anticonvulsants, muscle relaxants
- GABA_A agonists – bind to receptor and facilitate effects of GABA; highly addictive
- Serotonin agonists (buspirone, SSRI) Reduce anxiety without sedation, side effects
- Antidepressants – effective due to comorbidity of anxiety and depression.
- Animal Models
- have helped with understanding of anxiety disorders & development of anti-anxiety drugs

- Weakness: they involve animal defensive behaviors – implicit assumption being that defense is motivated by fear and that fear and anxiety are similar states.
- Elevated-plus-maze test: rats placed in a 4 arm plus sign 50cm off the floor, where 2 of the arms have sides and 2 do not – the measure of anxiety is the proportion of time the rats spend in the enclosed arms rather than going in to the exposed arms.
- Defensive-burying test: rats receive unpleasant stimulus expelled from dowl in test chamber – measure of anxiety is amount of bedding they throw over source of unpleasant stimuli.
- Risk assessment test: after single exposure to a cat, rats run away to their burrows and freeze & then engage in risk assessment behaviors before behavior returns to normal – measure of anxiety is amount of time rat spends frozen and risk assessing.
- All have been validated by demonstrations that benzodiazapines reduce the indices of anxiety used in the tests whereas non-anti-anxiety drugs do not.
- But many cases of anxiety do not respond to benzodiazapines

15. Critically discuss the evidence for a distinction between semantic, episodic, and procedural memory (25 marks).

Brief account needed to begin describing Tulving's work/ideas on the distinctions between different LTM systems. Then a definition of episodic, semantic, and procedural memory with examples should have been included. Answers should then have provided a detailed account of the various empirical literature and studies from brain-damaged patients, including those with amnesia (Spiers et al., 2001; Vargha-Khadem et al., 1997) and the evaluation of the Vargha-Khadem et al. study by Gardiner et al. (2008) and an explanation for the discrepancy. The evidence that episodic memory depends on the hippocampus whereas semantic memory depends on the entorhinal, perirhinal, and parahippocampal cortices (Vargha-Khadem et al., 1997; Bindschaedler et al., 2011). Evidence from patients with retrograde amnesia in particular (Bayley et al., 2006; Manns et al., 2003), the theory of consolidation to explain the difference, the evidence from the alcohol (Moulton et al., 2005) and sleep (Jenkins & Dallenback, 1924) studies to support the consolidation theory, and the critique of the sleep study regarding time of day (Hockey et al., 1972), and the alternative theory of semantisation. Evidence from patients with semantic dementia (Adlam et al., 2009) and that these patients show a double-dissociation with amnesic patients. An evaluation of using patients with brain damage in general (i.e., loss of other cognitive abilities, or strategies may be used to overcome issues). The essay could have also focused on patients with Korsakoff syndrome and the issues associated with basing evidence from this sample. Discussions on the distinction could have also focused on how different types of information can be forgotten and the influence of interference. Furthermore, an acknowledgment that perhaps the systems are not so distinct and are instead interdependent was also required (e.g., Burianova et al., 2010 on similar brain regions of activation; Kan et al., 2009 on learning). Stronger answers would have included work from additional readings (Renoult et al., 2016; Kim, 2016; Takashima et al., 2017).

Diwedd/End