PYB3O4 Exam Revision Guide

Exam info

- Worth 60%
- 2hrs 10 mins
- 80 multiple choice questions
    - 7 Qs from first 3 weeks
    - 8-9 from following weeks
- Questions are factual, conceptual, and application Qs
    - Parts of brain effected

Week 1: Intro and brain structures

Overview

What does the brain look like?

- The brain is divided into the cerebrum, cerebellum, and brainstem – each with distinct functions
- There are 4 lobes
    1. Frontal lobe
        a. decision-making
        b. planning
        c. personality
        d. voluntary movement (motor cortex)
        e. Broca's area (speech production)
    2. Parietal lobe
        a. processes sensory info – touch, pain, temp
        b. spatial awareness and navigation
    3. Temporal lobe
        a. Hearing
        b. language comprehension
        c. Wernicke's area,
        d. memory
        e. contains the hippocampus = damage to the hippocampus can cause anterograde amnesia, where a person cannot form new memories – case of H.M.
    4. Occipital lobe =
        a. visual processing, damage causes blindness or visual distortions
- Main structures
    1. Cerebellum
        a. Balance
        b. Coordination
        c. fine motor control
        d. procedural learning
    2. Brainstem
        a. Controls automatic survival functions: breathing, heartbeat, sleep-wake cycles
        b. Includes
            i. Midbrain = upper part of brainstem, coordinates movement and sensory info
            ii. Pons = middle part, communication bridge between brain regions
            iii. medulla = lowest part, controls automatic functions
    3. Limbic system
        a. Emotion and memory hub
        b. Key structures:
            i. Amygdala = fear / emotion

ii. Hippocampus = memory
iii. Hypothalamus = homeostasis

4. Corpus callosum = thick bands of fibres connecting the left and right hemispheres, allowing them to communicate

How does the brain work?

Neurons, neurotransmissions, and brain communication

- Neurons communicate via electrical signals (action potentials) and chemical signals (neurotransmitters) across synapses
- Neuron structure

1. Dendrites receive signals
2. Cell body processes
3. Axon transmits
4. Axon terminals release neurotransmitters into the synapse

- Action potential – signal
  - Depolarisation = Na+ (sodium) in
  - Repolarization = K+ out (potassium)
- Synaptic transmission – signal
  1. Neurotransmitter released from vesicles
  2. Crosses synaptic cleft
  3. Binds to receptor
  4. Reuptake or degradation follows
- Neuron types
  - Sensory (afferent) – carries info to central nervous system
  - Motor (efferent) – from CNS to muscles
  - Interneurons = connect neurons within CNS
  - Key neurotransmitters
    - Dopamine = reward, motivation, movement
    - Serotonin = mood, sleep, appetite
    - GABA – main inhibitory neurotransmitter, reduces neural excitability
    - Glutamate – main excitatory neurotransmitter, critical for learning and memory
    - Norepinephrine – arousal, attention, fight-or-flight
    - Acetylcholine – muscle activation, attention, memory

Genetic mechanisms underlying brain function and behaviour

- Genes and epigenetics
- Genes don't determine behaviour directly – they influence brain development, neurotransmitter systems, and sensitivity to environments
- Behaviour is always the product of gene x environment interactions
- Twin studies of monozygotic (identical twins, share 100% DNA) and dizygotic share 50%
- Adoption studies – if adopted children resemble biological parents more than adoptive parent = genetic influence on a trait
- Heritability of traits changes with environment
- Epigenetic = gene expressions can be switched on/off by experience e.g., stress, diet, without changing DNA sequence
- Intelligence = polygenetic – influenced by thousands of genes, each with tiny effect

Hunger and substance use

- Reward circuits, hypothalamus, and addiction neuroscience
- Both hunger and substance use tap into the brains mesolimbic dopamine reward system
- Hunger – hypothalamic regulation
    - Lateral hypothalamus = hunger centre – lesion=anorexia
    - Ventromedial hypothalamus = satiety centre lesion-hyperphagia=overweight
- Hormones
    - Ghrelin (stomach) = increases appetite
    - Leptin (fat cells) = signals fullness to hypothalamus
    - Insulin = regulates glucose and feeding
- Reward = mesolimbic pathway
    - Dopamine release from engaging in eating and drugs – drugs hijack this circuit
- Neuroadaptation
    - Repeated drug use = downregulation of dopamine receptors = tolerance (need more for same effect)
    - Withdrawal = dopamine crash
- Drug mechanisms
    - Cocaine/amphetamines = block dopamine reuptake, flood of dopamine in synapse
    - Opioids = trigger dopamine release, inhibits pain signals
    - Alcohol = enhances GABA (inhibitory), suppresses glutamate (excitatory) – sedation

- Nicotine = binds acetylcholine receptors, dopamine release

How do we form memories?

- Memory types, encoding, consolidation, and retrieval
- Memory is multiple interacting systems
  - Hippocampus = critical for forming new explicit memories
  - Amygdala = enhances emotional memories
  - Basal ganglia = handles habits
- 2 Types of memory
  1. Explicit = declarative
     a. Conscious memory
     b. Personal events and semantic memory - facts and knowledge
  2. Implicit = non-declarative
     a. Unconscious memory
     b. Procedural = skills
     c. Conditioning = learned associations
     d. Priming = exposure alters response
- Stages of memory
  1. Encoding = attending to info
  2. Storage
  3. Retrieval = reactivating stored trace – influenced by context and cues
- Long-term potentiation (LTP)
  - Repeated synaptic firing = stronger connections between neurons, neurons that fire together wire together
  - NMDA glutamate receptors
- Patient H.M. had his hippocampus removed = could not form new explicit memories (anterograde amnesia) but kept implicit memory intact
- Misinformation effect – post-event info can alter memory
- Reconsolidation – memories become labile when retrieved and must be re-stored

Language – how does the brain produce language?

- Language areas, aphasia, and the neuroscience of speech
- 2 key areas connected by the arcuate fasciculus
  - Brocas = production
  - Wernickes = comprehension
- Areas affecting language
  - Brocas area
    - left inferior frontal gyros

        - responsible for speech production and grammar
        - damage = brocas aphasia – slow effortful speech, comprehension relatively preserved
    - wernickes area
        - left posterior superior temporal gyrus
        - responsible for language comprehension
        - damage = wernickes aphasia, fluent but meaningless speech, poor comprehension
    - arcuate fasciculus = white matter tract connecting Brocas and Wernickes areas
        - damage = conduction aphasia – can speak and understand but cannot repeat words
- wenicke-geschwind model = model of spoken language processing
    - auditory cortex = wernickes
    - arcuate cortex = brocas
    - motor cortex = speech output

Emotion and Stress – how does the brain process and manage emotion and stress

- limbic system, HPA axis, theories of emotion
- emotion involves rapid subcortical processing (amygdala) and slower cortical appraisals
- chronic stress activates the HPA axis and floods the body with cortisol
- amygdala
    - rapid threat detection
    - Fear conditioning
    - Receives sensory input before conscious processing
    - Overactive in anxiety disorders
- Prefrontal cortex PFC
    - Emotions regulation
    - Reduced PFC activity = poor impulse control and emotional dysregulation
- HPA axis
    - Hypothalamus = pituitary = adrenal glands = cortisol
    - Cortisol mobilises energy but chronic elevation damages hippocampus and immune function
- Theories of emotion
    - James-lange = body sensation = emotion
    - Cannon-bard = simultaneous body and feeling
- Acute stress = adaptive, sharpens attention, boosts memory encording
- Chronic stress = hippocampal atrophy, impaired memory and immune function

- PTSD = hyperactive amygdala, reduced hippocampal volume
- Emotion regulation strategies
    - Reappraisal
    - Suppression

Brain recovery – how does the brain recover from damage

- Neuroplasticity, recovery mechanisms, and rehabilitation
- The brain is not fixed – neuroplasticity allows it to reorganise, form new connections, and compensate for damage
- Recovery is greatest in young brains
- Neuroplasticity = lifelong ability to change synaptic connections and cortical maps in responds to experience, learning, or damage
- Synaptic sprouting = intact neurons grow new axon branches to reinnervate areas abandoned by damaged neurons
    - Can restore functions or cause maladaptive changes
- Cortical remapping = brain areas can take over functions of damaged regions
- Neurogenesis = new neurons are generated in the hippocampus
    - Stress reduces neurogenesis
- Diaschisis = temporary dysfunction in areas connected to but not directly damaged by the injury

Behavioural neuroscience = study between brain, behaviour, and environment and how they relate and interplay

2 systems

1. Central nervous system – CNS (located within the skull and spine)
    a. Brain
    b. Spinal cord
2. Peripheral nervous system – PNS (located outside the skull and spine)
    a. Somatic nervous system = sensations, hearing, and sight
    b. Autonomic nervous system = automatic processes

- Somatic and autonomic consist of
    - Afferent nervous system = send messages to CNS
    - Efferent nervous system = signals exiting CNS
        - Sympathetic nervous system = physiological arousal
        - Parasympathetic = relaxation

Protection mechanisms

- Meninges = membrane around the brain
    - Dura mater = outer, toughest layer
    - Arachnoid meninx = spider-like membrane, subarachnoid space (filled with cerebrospinal fluid (CSF) – a shock absorber)
    - Pia mater = delicate
- Blood-brain barrier
    - Barrier of tightly packed walls
    - Only small molecules, lipid-soluble molecules, and some gasses can pass freely and passively
    - Prevents influx of big molecules, some toxic substances, but allows the passage of important ones through transporter proteins

Structures

Anatomical directions

- Anterior-posterior = front-back
- Dorsal-ventral = towards top/at back – under/belly/front
- Medial-lateral = towards the midline/away from the middle
    - Dorsal = top
    - Ventral = bottom
    - Posterior = behind
    - Anterior = front
    - Medial = middle, centre
    - Lateral = away, further away

Planes in the brain

- Saggital = split from left to right = across
- Frontal plane = split from front to back = anterior and posterior
- Horizontal = top and bottom, dorsal and ventral

Spinal cord

- Grey matter = cell bodies and unmyelinated interneurons
- White matter = myelinated axons
- Dorsal route (back route) = sensory afferent route, pairing sensory info to approach brain
- Ventral route (front route) = motor info, efferent route, pairing motor info to exit brain and create movement

Components of an adult brain

Forebrain = telencephalon and diencephalon

Midbrain = mesencephalon

Hindbrain = metencephalon, myelencephalon

- Tel = biggest and rest in alphabetical order

Sulcus/fissure and gyrus

- Provides groves to create more surface space in brain
- Deep sulcus = fissures – help orientate where we are in brain

Telencephalon – major divisions of cerebral cortex

- Frontal lobe = thinking, planning, organising, and problem-solving
- Motor cortex = movement
- Temporal lobe = memory, understanding language
- Sensory cortex = sensations
- Parietal lobe = perceptions, making sense of world
- Occipital lobe = vision
- Limbic system = motivated behaviour, emotion, memory
    - Amygdala – fear conditioning, emotions
    - Hippocampus – memory, learning, spatial navigation
- Basal ganglia = regulates movement

Diencephalon

- Thalmus = sensory relay station of brain – senses go through brain and then passed onto other parts of brain
- Hypothalamus = motivated behaviour e.g., eating, sleeping, and sexual behaviour

Mesencephalon (midbrain)

- Tectum
    - Superior colliculi = visual-motor
    - Inferior colliculi = auditory
- Tegmentum = movement

Metencephalon

- Pons = bridge connecting systems
- Cerebellum = movement, coordination, balance

Myelencephalon

- Myelencephalon (medulla) = comprised of tracts carrying signals between brain and body
- Reticular formation = survival functions

Week 2: Neuroanatomy and Neural Transmission

Overview

- What do neurons look like?
- How do neurons communicate with each other?

Brain terminology

- CNS
    - Nuclei = cluster of cell bodies
    - Tracts = bundles of axons
- PNS
    - Ganglia = cluster of cell bodies
    - Nerves = bundle of axons
- Glial cells = transduce info, respond to injury, regulate blood flow, and control what chemicals pass from blood into brain
- Myelinated neurons = myelin wraps around axons to massively speed up communication
    - Gaps in myelin (nodes of Ranvier) = allow fast jumping transmission

Neuron types

- Multipolar = standard, many dendrites connect to many other neurons
- Unipolar & bipolar = unipolar = 1 connection, bipolar = 2 connections
- Interneurons = connect neurons to other neurons within the CNS
    - Brains internal relay network

Neuron structure

- Cell body (soma) = decision-making, regulates and integrates all incoming info
    - Contains nucleus = control centre of cell
- Dendrites = branch-like extensions that receive signals from other neurons
    - Multipolar neurons = multiple dendrites
- Cell membrane = semi-permeable – only some things can pass through
    - 2 proteins – channel, signal
- Axon hillock = decided whether to fire an action potential
- Axon = long, narrow cable projecting from cell body, wrapped in myelin to speed communication, carries signal away from body

- Synapse and terminal buttons
    - Synapse = tiny gaps between one neuron terminal button and the next dendrite
    - Terminal buttons = at the end of the axon

Resting membrane potential = neuron doing nothing

- When a neuron is at rest (not firing), its inside is negatively charged at about neg 70mV relative to the outside. This charge difference is the stored energy that allows neurons to fire
- Potassium K+ = most concentrated inside the cell
- Sodium Na- = more concentrated outside the cell

2 ways the potential can change

- **Depolarization =** cell becomes more positive, caused by excitatory postsynaptic potentials (EPSPs)
- **Hyperpolarization =** cell becomes more negative, caused by inhibitory postsynaptic potentials (IPSPs), makes it harder to fire
- **Repolorisation =** neuron returns to its resting state after an action potential has fired

Summation – how signals add up

- Spatial summation = 2 signals arrive same time from diff locations, if both EPSPs = push cell towards firing
- Temporal summation = 2 signals arrive same location at diff times

Action potential – the electrical signal that travels down the axon

- An action potential is an all-or-nothing electrical spike. Once the threshold of -65mV is crossed, there's no going back – the signal fires fully or not at all

Step by step sequence

1. Resting potential (axon doing nothing) -70mV
2. Threshold reached -65mV
3. Na channels open – rushes in
4. Depolorisation peak (+40mV)
5. Na channels close
6. K channels open – flows out
7. Repolorisation back to -70mV

8. Brief hyperpolorisation

Key concepts

- All-or-nothing = if threshold reached, the AP will fire at full strength every time
- Absolute refractory period = immediately after an AP, the neuron cannot fire again = ensures signals travel in one direction
- Relative refractory period = after the absolute refractory period, the neuron can fire again – but only with a stronger-than-normal stimulus – the cell is still repolarising
- Saltatory conduction = in myelinated axons, the AP jumps between nodes of ranvier

Neurotransmission – how neurons pass messages to each other

- When an AP reaches the terminal button, it triggers the release of chemical messengers (neurotransmitters) that cross the synaptic gap and tell the next neuron to excite or inhibit

1. **AP arrives at terminal button**
    - The electrical signal reaches the end of the axon, which triggers synaptic vesicles to fuse with the membrane
2. **Exocytosis**
    a. Vesicles fuse the terminal membrane and release neurotransmitters into the synaptic cleft – the tiny gap between 2 neurons
3. **Receptor binding**
    a. Neurons only bind to specific receptors on the postsynaptic membrane
4. **Effect on next neuron – can excite or inhibit**
    a. Binding causes:
        i. Ionotropic effect – direct ion channel opening – fast
        ii. Metabotropic effect – slower signal cascade via protein

Cleanup mechanisms

- Reuptake = the presynaptic neuron sucks unused neurotransmitters back in for recycling
- Enzymatic degradation = enzymes in the synaptic cleft break the neurotransmitter apart, deactivating it so it cannot keep stimulating the receptor

Types of neurotransmitters

- **Small neurotransmitters =** made in cytoplasm, packaged in terminal buttons. Fast acting e.g., dopamine, serotonin, GABA, glutamate
- **Neuropeptides =** large molecules made on ribosomes in cell body, transported to terminal buttons, slower-acting e.g., endorphins

Week 3: Behavioural Neuroscience and Behavioural Genetics

Overview:

1. How genetics alone shape traits and behaviour
2. How genes and environment interact

- No gene acts alone – traits are polygenic (many genes) and always shaped by environment

Theories

- **Lamarck –** use trait or lose it, traits acquired during a lifetime e.g., a giraffe stretching its neck (wrong)
- **Darwin –** natural selection: individuals with traits that help them survive and reproduce pass those traits on, evolution
- **Mendel –** studied pea plants to discover inheritance rules – dominant/recessive alleles, and how traits are passed via discrete factors (genes), filled gaps of Darwins theory

Evolution and inheritance – how traits are passed down through generations

- **Mendel's key insight –** we inherit one version of each gene (allele) from each parent. Whether a trait shows up depends on if its dominant or recessive
- **Allele –** a diff version of the same gene e.g., blue vs brown eyes, you have 2 alleles for every gene – one from each parent
- **Homozygous =** both alleles are the same (e..g, both brown or both blue)
    - can be homozygous dominant or recessive
- **heterozygous =** 2 different alleles e.g., one blue and one brown
    - the dominant allele will usually determine which trait is displayed
- **dominant =** a trait that shows up whether you have 1 or 2 copies of the allele
- **recessive =** only shows up if you have 2 copies of that allele (only homozygous recessive), one dominant allele will mask it
- **genotype =** the actual gene combination you carry e.g.,
    - AA – homozygous dominant
    - Aa – heterozygous
    - aa – homozygous recessive
- **phenotype =** the physically observable trait e.g., blue eyes, curly hair

Genetic similarity between relatives

- parents = child 50% shared DNA
- grandparents = grandchild 25%
- identical twins (monozygotic) = 100% shared DNA
- non-identical twins (dizygotic) = 50% shared, same as siblings

Chromosomes

- humans have 23 matched chromosome pairs = 46 total
- 22 pairs are autosomes (non-sex chromosomes)
- 1 pair sex chromosomes = XX – female, XY = male
- X chromosome larger, so females have twice chance of dominant traits
- Mitosis = cell duplication with full chromosome set
- Meiosis = produces sperm/egg cells with only half the chromosomes

DNA and Genes – carries genetic info

- DNA = instruction manual, a Gene = one chapter – a specific segment that encodes a protein. Base pairing rules:
    - Adenine-thymine (A-T)
    - Guanine-Cytosine (G-C)
- DNA has a double helix structure
- **Chromatin and Chromosomes** – DNA wraps around proteins called histones = chromatin, a chromatin coils tightly, forms a chromosome
    - Chromosomes are only visible during cell division
- **Gene** – a specific segment of DNA that encodes instructions for making a particular protein
    - One gene does not equal one trait, most traits are polygenic (influenced by many genes)
- Polygenic = many genes affect one trait e.g., height, intelligence
- Pleiotropic = one gene affects many traits

Gene expression – how DNA becomes a physical trait or behaviour

- **The central dogma =** path from gene to behaviour: DNA is transcribed into RNA, which is translated into proteins

- **Proteins =** do almost everything in the body, build structures, act as neurotransmitters, and shape behaviour

The pathway

1. **Transcription =** DNA double helix unwinds. One strand is used as a template to create a matching strand of messenger RNA (Mrna). Here DNA -> RNA
2. **Translation =** ribosomes read the mRNA in codon each codon codes for an amino acid, a chain of amino acids = a protein
3. **Protein folding =** how the protein folds determine its function e.g., how tightly a hair-protein folds determines how curly your hair is

Regulation

- **Enhancers and promoters =** stretches of DNA that acts like volume controls – they determine whether a gene gets turned on and starts making proteins or stays silent
- **Transcription factors =** proteins that bind to DNA and influence how much a gene is expressed, key mechanism to which environment can change gene activity

Gene meets environment

- Behavioural traits can be passed across generations, but the environment shapes how genes are expressed
- **Selective breeding =** animals can be bred for behavioural traits e.g., aggression, tameness, shows genetics influences behaviour, selective breeding cannot fully control outcomes due to environmental factors
- **Twin studies =** Identical twins (MZ) = 100% same DNA. Fraternal twins (DZ) = 50% same DNA. If MZ twins are more similar on a trait, that trait has a genetic component
    - **Limitation =** many classical twin studies used middle-class families – limited environmental variation. Also, MZ twins become more diff with age = environment increasingly matters over time
- **PKU (phenylketonuria) =** single recessive gene disorder - body can't break down phenylalanine (an amino acid), causing toxic buildup → intellectual disability, seizures. But: managed entirely with diet.
- **MAOA gene =** encodes enzymes that break down dopamine, norepinephrine, and serotonin
    - The risk allele only increased aggression and reduced empathy in people who had experienced childhood abuse

- **Correlation does not equal causation –** cannot prove genes cause behaviour directly

Epigenetics = changes in gene expression caused by experience, not changes to the DNA sequence itself. Your environment, diet, stress, and relationships can switch genes on or off

- **Epigenetics** = Changes in how genes are turned on or off without altering the underlying DNA sequence. The DNA letters stay the same — but the volume on certain genes goes up or down.
- **DNA methylation =** a methyl group (small chemical tag) is added to DNA. This usually silences or reduces the activity of that gene
- **Dynamic and reversible =** Unlike your DNA sequence, epigenetic marks can change across your lifetime. Diet, lived experience, social interactions, and stress can all alter them.
- **Chronic stress =** Sustained stress can trigger epigenetic changes that affect genes involved in regulating the stress response — making someone more reactive to future stressors.
- **Epigenetic inheritance =** fear responses can be instilled in one generation and passed on epigenetically e.g., your grandparents traumas may influence your gene expression
- **Triggers**
    - Diet and nutrition
    - Chronic stress and trauma
    - Social interactions and relationships
    - Exercise and physical environment
    - Childhood maltreatment or adverse experiences