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**Knowledge Rich Curriculum Plan**

GCE Biology- Unit 3.6a Responding to the Environment



| **Lesson/Learning Sequence**  | **Intended Knowledge:***Students will know that…* | **Prior Knowledge:***In order to know this, students need to already know that…* | **Tiered Vocabulary and Reading Activity** |
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| **Lesson 1:** **Neurones** | *The structure of a neurone is more complex than previously learned: the axon is insulated by a protective sheath called the myelin sheath. This is not a continuous layer, but rather it is made of a series of cells known as Schwann Cells. The membrane of a nerve cell has many intrinsic proteins that support transmission of an impulse. Motor neurones transmit impulses to muscles/glands. Sensory neurones detect energy changes in the environment and produce an impulse. Relay neurones transmit impulses between the peripheral neurones.* | *GCSE Biology covered structure of a general neurone: cell body, axon, dendrites, nucleus, insulated axon prevents loss of energy along axon length. Correct word for the signal is impulse. Three distinct forms of neurone: sensory, relay and motor.**Neurotransmitters are chemicals that are produced by neurons and released into the synapse to diffuse across to the next neurone.**Structure of a membrane involves a range of specialised proteins that support movement of ions across the selectively permeable structure. (Topic 2, Y12)* | *Schwann cells**Myelin**READ Structure of Neurones* |
| **Lesson 2:** **Neurones are polarised when ions are exchanged** | When neurones are resting, the membranes are polarised. This means that the outside of the membrane is positively charged compared to the inside. (There are more positive ions outside than inside the membrane) We call this the resting state. The resting potential is -70mV. The balance of Na+ ions and K+ ions on opposite sides of the membrane creates this potential difference. Key idea: the membrane is not permeable to Na+ ions so they cannot pass freely through. If the ions are actively pumped out of the neurone, an electrochemical gradient will be established. The movement of the ions is done by proteins called the Na/K pump. K+ ions are moved in to the axon but since they can pass out freely and so the difference in charge is why there is a potential difference/electrochemical gradient. (3 x Na+ for every 2 K+ are exchanged) | Membrane structure (Topic 2, Y12) includes proteins that are concerned with transport of ions/molecules eg., Na/K pump. Energy is supplied by ATP (Topic 1,Y12)GCSE Physics: potential difference is measured in Volts. Small values would be expressed in milliVolts. | *Polarised**Resting state**Potential difference**Electrochemical gradient.**Channel proteins* |

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| **Lesson 3:** ***Action Potential*** | *The membrane of a neurone is depolarised when a stimulus triggers the change. The Na+ channels open. If the stimulus is large, the difference in potential is large and an action potential is established across the membrane. If sodium channels are open, the membrane is more permeable to sodium ions which diffuse into the neurone. This makes the inside less negative than when at rest. If the difference reaches a threshold value (-55mV) more sodium channels open and so diffusion occurs rapidly. If the potential difference reaches =30mV, the Na+ channels close. At this value, K+ channels open. More K+ ions diffuse outwards. This restores the potential difference to the resting potential. After an action potential the neurone cannot be excited again immediately as the ion channels need to recover the resting position: this is the refractory period.**These changes can be represented graphically and the labels for target language applied. NB: action potentials ensure that impulse are discrete and unidirectional.* | *Previous lesson including the Y12 work on membranes and transport processes.* | *Polarisation**Diffusion**Repolarisation**Permeable**Hyperpolarisation**Depolarisation**Refractory period**Unidirectional* |
| **Lesson 4:** **Speed of conduction of action potentials** | Myelin is an electrical insulator and is provided by the series of Schwann cells arranged along an axon length. There are small gaps between these cells known as the Node of Ranvier. It is at these locations that the sodium ion channels are concentrated. The consequence is that for myelinated axons, the depolarisation sequence only occurs here. The impulse appears to ‘jump’ from node to node. This is called saltatory conduction and is faster than in non-myelinaed neurones.Other factors that affect the speed of conduction are temperature and diameter of axon. Temperature allows ions to diffuse faster. The limitation is that at temperatures above 40oC, proteins denature and so the membrane function of transport ceases.When axon diameter is increased there is less resistance to the flow of an electrical impulse. This means that the wave of depolarisation is increased. Key idea: if the size of the stimulus is insufficient, depolarisation will not occur because of the threshold figure (action potential graph) | Combination of knowledge from Lesson 1: structure of a neurone & Lesson 3: action potential detail including the graph with labels for specific values. | *Node of Ranvier**Saltatory conduction* |
| **Lesson 5:** **Synapses** | The tiny gap between two cells at a synapse is the synaptic cleft. The presynaptic neurone has a swelling called the presynaptic knob. The gap between this structure and the next cell is the cleft. The presynaptic knob produces vesicles containing neurotransmitters. These are stimulated to leave the neuron via exocytosis and diffuse across the cleft. The are bound to complementary protein molecules embedded in the postsynaptic membrane. This results in an action potential in the neighbouring neuron. The receptors are only located on the post-synaptic membrane and hence the impulse maintains its unidirectional flow. To prevent an undesirable build-up of neurotransmitter in the cleft, an enzyme is released to breakdown the used/unwanted neurotransmitter. Example of neurotransmitter is acetylcholine.Process: an action potential arrives at the presynaptic knob. It triggers voltage-gated channel proteins to open. Calcium ions diffuse into the knob. These stimulate the movement of vesicles containing the neurotransmitter towards the terminal membrane. The vesicles fuse with the presynaptic membrane. The neurotransmitter is released into the cleft. When the neurotransmitter binds to the receptors on the post-synaptic membrane, sodium channels open and sodium ions diffuse into the new neuron starting a new action potential.  | GCSE Biology: a synapse is the gap between two adjacent neurones/a neuron and an effector. A simple synapse sequence includes the words neurotransmitter, pre-synaptic knob, post-synaptic knob, receptors, vesicles and diffusion.Y12 topic 2: exocytosis | *Cleft**Acetylcholine**Voltage-gated channel proteins**Acetylcholinesterase**READ Synapses* |

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| **Lesson 6:** **Gross and fine structure of muscles** | *Muscles are effectors (The reflex arc)**Skeletal Muscle is known as striated/striped or voluntary muscle. They are attached to bones by tendons and help to move the skeleton. Pairs of skeletal muscles work together to effect the movement of bones. The contracting muscle is the agonist and the relaxing muscle is the antagonist.**Skeletal muscle is made up of fibres that can be stimulated to contract by an impulse.**Muscle fibres are made of large bundles of long cells called fibres. The cell membrane is the sarcolemma. The sarcolemma is not smooth, it has folds that stick into the sarcoplasm. These folds are called T tubule.**T tubules help to ensure that electrical impulses reach all areas within the sarcoplasm/muscle fibre.**A network of internal membranes are extensive throughout the sarcoplasm. These store Ca2+ ions needed for muscle contraction.**Muscle fibres also have lots of mitochondria. This generates ATP needed for contraction (see later)**Myofibrils are organelles within a muscle fibre that have long molecules of proteins specific to muscle function.* | *Muscles are effectors.**Skeletal muscle acts in antagonistic pair.s* | *Antagonistic**Striated**Voluntary**Sarcolemma**Sarcoplasm**T tubules**Myofibril**Sarcoplasmic reticulum**READ Structure of muscles – skeletal* |
| **Lesson 7:****Sliding Filament Theory -****The role of actin and mysosin** | Myofibrils have long chain molecules of protein called actin and myosin. Myosin appears as a thick ‘dark’ band and actin appears as a thin ‘light’ band. A myofibril can be divided into regions (striations) known as sarcomeres. The ends of a sarcomere are known as Z bands. In the middle of a sarcomere is the M line. The myosin filaments are fixed to this line at the middle. The two proteins are able to slide past each other. This is contraction. Detail: actin molecules have binding sites for myosin molecules. Actin molecules are bound by another long chain molecule called tropomyosin. This conceals the binding sites temporarily. This prevents movement of the two types of molecule. For contraction to olccur, the tropomyosin must move away from the binding sites in order that actin-myosin molecules bond temporarily. This occurs when Ca2+ ions are present. Once the two main protein molecules are bonded together, Ca2+ ions cause the myosin head to change shape and effectively ‘row/ratchet’ the actin molecule along. Energy from ATP is required to make this myosin head change shape. Note: this occurs in many sites at the same time to ensure that the two molecules move past one another. Contraction stops when Ca2+ ions are not available. At this point, the tropomyosin is no longer exposing the troponin bonding sites. Ca2+ ions return to the sarcoplasmic reticulum for storage. The actin slides back to its original position and the muscle is relaxed. | Structure of a sarcomere.Y12 Topic 1: protein structure and function; ATP structure and function; enzymes structure and function. | *Myofibril**Actin**Myosin**Troponin**Tropomoysin**Sarcomere**Z band**A band**I band**Actin-myosin crossbridge**READ Sliding Filament Theory* |
| **Lesson 8:****Neuro-muscular junction** | A NMJ is a synapse that occurs between a motor neurone and a muscle cell. NMJ only use the neurotransmitter: acetylcholine (ACh). This molecule binds to receptors on the postsynaptic membrane (sarcolemma) known as cholinergic receptors. The process is very similar to way that a synapse functions (see L5). Key differences: the postsynaptic membrane is highly folded. These folds are called clefts. They store the enzyme acetylcholinesterase which breaks down excess neurotransmitter. There are more receptors on this membrane than in a ‘usual synapse’. This junction is always excitatory ie the response to neurotransmitters is positive – an action potential from the motor neurone WILL trigger a muscle to contract.Drugs affect action of neurotransmitters at NMJ: some will block neurotransmitter function by binding to the receptors that would usually trigger contraction. Others may mimic the action of a neurotransmitter or prevent the removal of the acetylcholinesterase enzyme and hence stimulate more contraction than is actually required/healthy eg., nerve gasses used in warfare. Some prevent release of neurotransmitters and this then causes relaxation eg., alcohol.  | The role of neurotransmitters in a typical cholinergic synapse. *(Refer to L5: synapses)*  | *NMJ**Cholinergic receptor**Acetylcholine**Motor end plate (post-synaptic membrane)**READ: NMJ and drugs* |
| **Lesson 9:****Consolidation of the elements of this topic** | Use of the elements of this unit in combination to illustrate how a stimulus results in a response. Outcomes from L1 – 8 integrated fluently to explain the molecular basis of these physical changes in the body. | Target language and sequences of knowledge from L1 – 8. | *As above* |
| **Lesson 10:****Marking and feedback point 3**  | Target language and sequences of knowledge from L1 – 8 and exemplification / application activities covered in L9. | All content from Units 1 – 6; key focus for application is this unit. |  |