SUPPLEMENTARY MATERIAL

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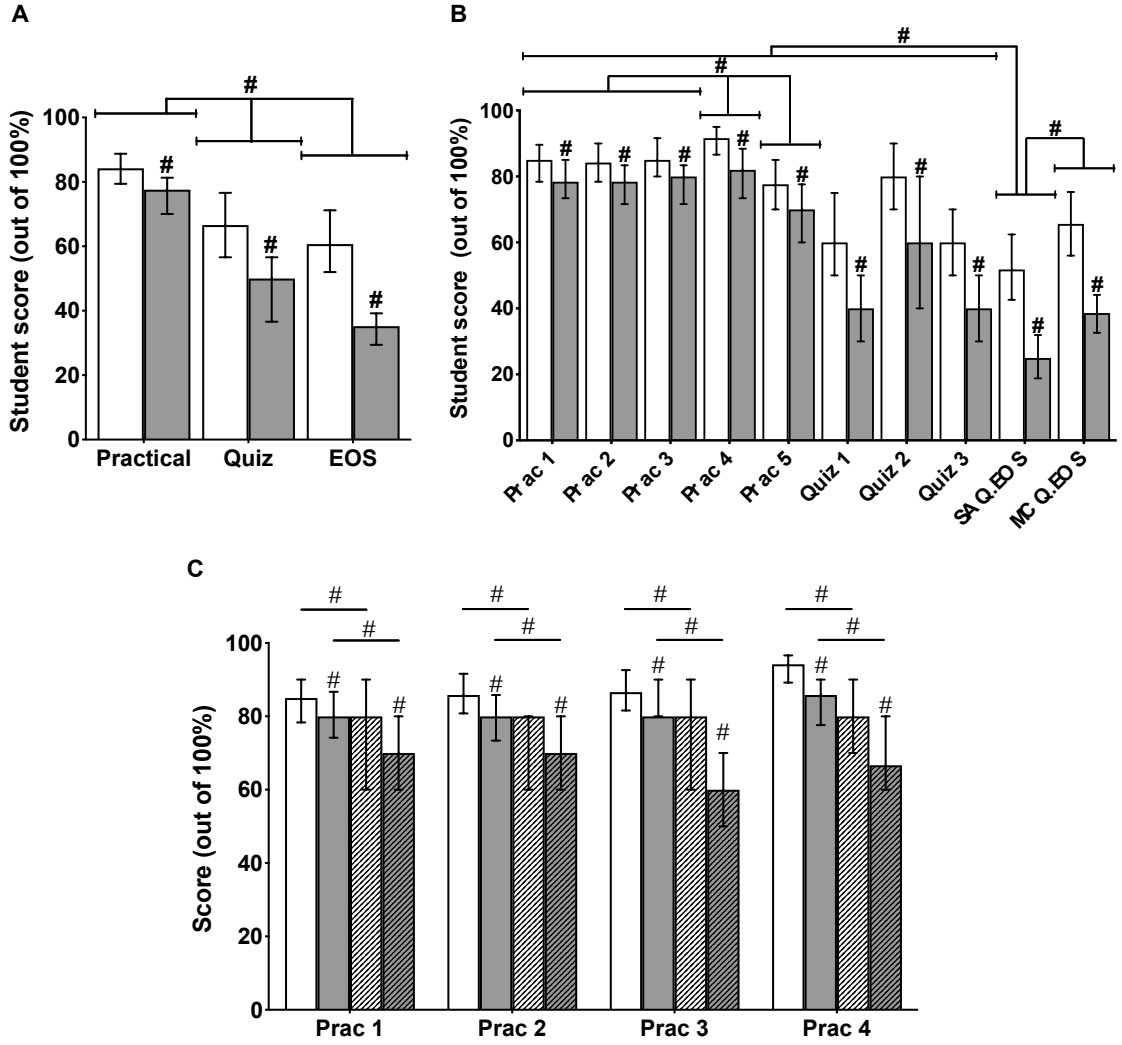
Appendix A

Analysis of student assessment performance in the 2016 cohort to inform intervention design (I) and construction of predictive models that was implemented in the 2017 iteration (II).

(I)



Student performance in the different course assessment tasks. Performance of students who passed the course in 2016 (white bars) and those who failed (shaded bars) in each assessment component, and the individual tasks within, are presented in A and B respectively. C present student performance in the different criteria used to evaluate their performance in each practical report of the course. Student performance in criteria which evaluates their understanding of the concepts covered in the laboratory practicals are presented as bars with stripes. Student performance in the remaining criteria are presented as bars without stripes. Only students who completed the practical assessment were included in the analysis. # on shaded bars were used to denote significant differences between student groups for each assessment component (A), task (B) and within each knowledge/non-knowledge criteria groups (C). The Kruskal-Wallis test with Dunn's post-hoc analysis was used to test for significant relationships. Data is presented as median with IQR. N(pass) = 430, N(fail) = 159, N(pass) and N(fail) in Practical tasks (C) ranged from 427 to 429 and 154 to 159 respectively. # $p<0.001$

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(II)

Summary of predictive models used to identify those at-risk of failing the course in 2017. A squared transformation was applied to Quiz 1, denoted by a superscript 2. Avg prac variable was constructed from the average score across Prac 1 and 2 for each student.

Variables	β	Std.Error	exp(β) / OR	p-value
Model with pre-university data (N=505)				
Intercept	14.516	2.297	2.015x10^6	<0.001
Chem Hs	-0.584	0.2862	0.558	<0.05
ATAR	-0.115	0.0275	0.891	<0.001
Avg prac	-1.635	0.4289	0.195	<0.001
Quiz 1^2	-0.267	0.1155	0.766	<0.05
Quiz 1	-0.928	0.2004	0.395	<0.001
Quiz 2	-0.538	0.1266	0.584	<0.001
Model without pre-university data (N=446)				
Intercept	6.762	1.504	864.369	<0.001
Nationality	0.966	0.485	2.627	<0.05
Avg prac	-1.662	0.377	0.190	<0.001
Quiz 1^2	-0.228	0.102	0.796	<0.05
Quiz 1	-1.102	0.194	0.332	<0.001
Quiz 2	-0.596	0.118	0.551	<0.001

Predictive model summary

	Without pre-university data	With pre-university data
Out-of-sample model accuracy	**Value**	**Value**
Brier score	0.132	0.131
AUCROC	0.806	0.849

Prac 1, a variable that did not contribute significantly to the outcome (β(SE) = -0.49(0.323), Z_{wald} = -1.51, p>0.05) was kept in the model by using the average student score across Prac 1 and 2 (i.e., Avg prac) as a variable. Prac 1 was not removed because student performance in this task is used to calculate their final grade and thus affect their course outcome.

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Appendix B

Announcement on course LMS site, inviting everyone in the cohort to participate in the interventional activity:

Dear Students,

The lectures for the remainder of this course will be based on cardiovascular, respiratory and endocrine systems, which will be given by *'instructor name'* and *'instructor name'*. Along with thinking about this new content, it is also important to revisit the concepts that are already covered in this course so far.

Remember that the Short Answer Questions in the final exam of this course are "integrative" in nature, combining concepts learnt from several lecture modules; and we know from the past that this is particularly challenging for students. Hence, my first lecture next week (Monday 8th May at 12noon) will be converted into an active learning workshop (still at the usual lecture theatre where you have this lecture though!). In this workshop, you will be able to answer some sample questions, following which we will provide some guidelines on what makes a good answer in our viewpoint. While this is not part of course assessment (and hence doesn't count towards your course grade!), it would be good if you give it your best shot by revising the topics that the workshop will be based on. These will be (i) Cell membrane structure and function, and (ii) the Nervous System module.

Regards, *'instructor name'*.

Email sent to students who were at-risk of failing the course, inviting them to participate in the interventional activity:

Dear Student,

You may have seen the Blackboard announcement regarding a workshop on 8th May at the usual lecture time (12noon) at the usual lecture venue. After reviewing the results that you have obtained so far in '*course code'*, we highly recommend that you attend this session. Specifically, this workshop is designed to assist you in the construction of complex, yet specific, responses to the Short Answer Questions of the End of Semester exam. The workshop would be extremely beneficial to you as it would highlight the key points that are needed to construct complex answers, which students find challenging in exams, especially in their first year.

Regards,
'Instructor name' and the *'course code'* team.

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Appendix C

The worksheet used in the interventional activity. The five sections of the worksheet in this section is presented in the order in which students receive them in the activity.

Section I

GUIDE TO CONSTRUCTING SAQs

Your friend, Georgia, has just hit her finger with a hammer and is screaming from the pain. While you are trying to calm her down, you are thinking about the course lectures that you were studying until her screams disturbed the peace. You think about the importance of materials passing across cell membranes in the processes involved in neurotransmission that resulted in Georgia feeling the pain in her finger.

Q1 Compare and contrast the processes of facilitated diffusion and active transport of substances across cell membranes. You may wish to include a diagram in your answer. (Score 3)

Q2 Explain how the passage of ions across cell membranes is involved in:
(i) generation of an action potential at the axon hillock and
(ii) direct synaptic transmission generating EPSPs and IPSPs at synapses.
Include the specific types of integral membrane proteins involved. Again, you may wish to include diagrams in your answer.
(Score 6)

[Continue your answer to b) on the other side]

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Section II

What mark would you assign to your answer for Q(a) (0 – 3; use half scores)?
Score:

What mark would you assign to your answer for Q(b) (0 – 6; no half scores)?
Score:

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Section III

Record your marks for the alternative answers to each question here

Q(a)

Answer Serial Number	Score (Out of 3; use half scores)
1	
2	
3	
4	
5	
6	

Q(b)

Answer Serial Number	Score (Out of 6; no half scores)
1	
2	
3	
4	
5	
6	

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Section IV – Instructor sample answers for Q1 and Q2 with instructor score filled in for each answer and answer ordered based on instructor score.

Answer		Instructor Score
1	Active transport in neurotransmission is when Na^+/K^+-ATPase pumps 3 Na^+ out of the cell while moving $2K^+$ ions into the cell using ATP to provide energy. This is to establish chemical and electrical gradients at resting membrane potential. Facilitated diffusion is when Na^+ and K^+ ions leave and enter the cells via their respective voltage-gated ion channels during an action potential.	0.5
2	Facilitated diffusion involves the transport of polar molecules across cell membrane through channel proteins. Polar molecules are transported down the concentration gradient. Active transport involves the transport of polar molecules across the cell membrane through transport proteins. These molecules are transported against the concentration gradient.	1
3	• Facilitated diffusion and active transport are critical to permit movement of polar and charged substances, such as ions, that cannot freely diffuse through the membrane. • This is because the cell membrane exhibits selective permeability due to phospholipid bilayer structure. • For example, both are involved in the generation of an action potential which a key component of neurotransmission. • First, Na^+/K^+-ATPase pumps 3 Na^+ out of the cell while moving $2K^+$ ions into the cell. This requires energy as ions are moved against their concentration gradients to establish the resting membrane potential of -70mV. • When a stimulus of sufficient strength depolarises the membrane potential to -55mV, Na^+ channels open and allows Na^+ ions to enter the cell down its concentration gradient. • As the membrane potential approaches +30 mV, K^+ ion channels open to allow K^+ ions to leave the cell down its concentration gradient to repolarise the membrane, while Na^+ channels inactivate. K^+ and Na^+ channels then close at the end of the action potential. Resting membrane potential is restored through the action of Na^+/K^+-ATPase.	1.5

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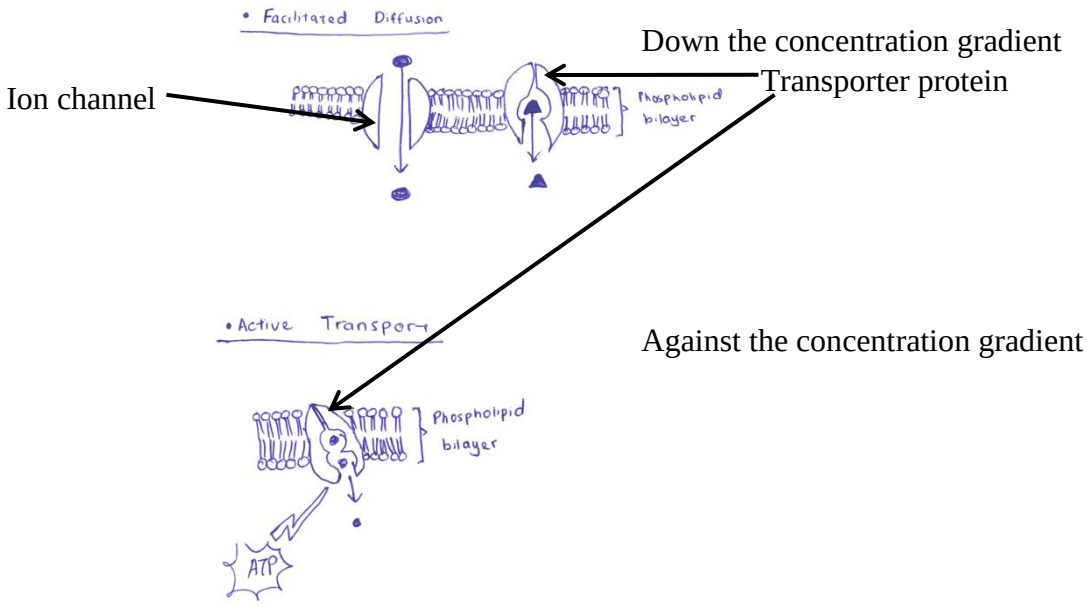
4		2
5	• The cell membrane exhibits selective permeability due to its phospholipid bilayer structure, which allows non-polar and non-ionic molecules to pass through by passive diffusion, but not ions or polar molecules. • Polar molecules and ions require other means to pass through the membrane. • Both facilitated diffusion and active transport permit the transport of polar molecules/ions through the bilayer via proteins embedded in the cell membrane. • These molecules/ions are facilitated to pass through the membrane passively, i.e. down the concentration gradient in facilitated diffusion. • In contrast, active transport involves moving these molecules against the concentration gradient, using a transporter protein with ATP as a source of energy.	2.5

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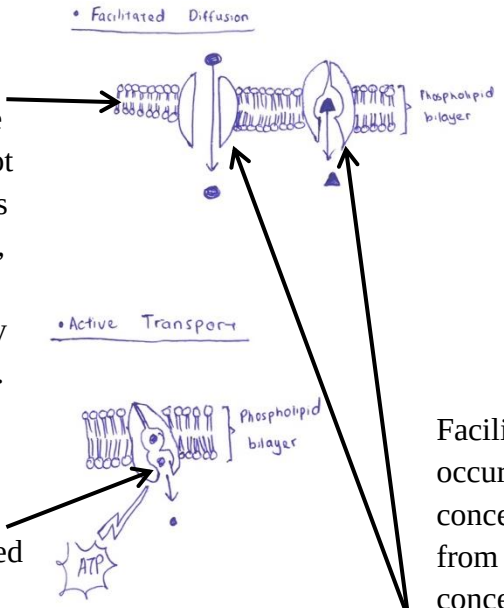
6	Phospholipid bilayer allows non-polar molecules to pass across the cell membrane by passive diffusion, but does not allow polar molecules or ions to pass across, hence showing the selective permeability of the cell membrane. Active transport differs from facilitated diffusion in using transporters to transport ions or polar molecules AGAINST a concentration gradient by using energy from ATP (e.g. Na^+, K^+-ATPase).  Facilitated diffusion and active transport both utilise integral membrane proteins to allow ions and polar molecules to pass across the cell membrane, as shown in the diagrams. Facilitated diffusion occurs along a concentration gradient from higher to lower concentration, either by the use of ion channels (e.g. for Na^+, K^+, etc) or water channels (aquaporins) OR by the use of transporters (e.g. glucose transporters) – without requiring energy.	3

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These are potential answers for Q(b). Without adjusting your answer, what score (out of 6; using full scores 1-6 and not repeating any score) would you assign each of these model answers?

	Answer	**Instructor score**
1	• Both action potential and direct synaptic transmission is involved in muscle contraction to withdraw Georgia's hand in response to the hammer hitting her hand. • Pain receptors detect pain, which results in signals being sent to Georgia's arm muscles to contract and pull hand away. • Signal to contract is in the form of action potentials. • Action potentials are generated through the movement of Na^+ and K^+ ions down their concentration gradient, across the cell membrane. This action potential is propagated along neurons to the relevant muscle groups. • Once the signal reaches the neuromuscular junction, acetylcholine is released onto the synapse. This binds to the acetylcholine receptors on the muscle fibre membrane. • Binding of neurotransmitters onto receptors will either result in an EPSP or an IPSP. • For acetylcholine, binding will trigger action potentials, which travels along the t-tubules until it reaches the sarcoplasmic reticulum. • Action potentials then trigger the release of Ca^{2+} ions into the cytosol • Ca^{2+} ions bind to the thin filament and exposes the myosin binding sites. • Myosin heads hydrolyses ATP and then binds to actin via the binding site to form cross bridges. ADP and inorganic phosphate is released by the myosin head, which then slides the actin towards the centre of the sarcomere. ATP then binds to myosin to release myosin binding to actin which then repeats the cycle until the signals from acetylcholine stops.	1
2	*(i) Action potential* Initially, Na^+ ions will have to pass through the membrane via ions channels. This increases the membrane potential until it becomes positive, as the resting membrane potential is negative. Then, K^+ ion channels allow K^+ to pass through the membrane out of the cell to decrease the membrane potential back to negative. However, it does not return to resting potential immediately due to an undershoot happening. *(ii) EPSP/IPSP* EPSP refers to excitatory postsynaptic potential. It is excitatory because it promotes the generation of an action potential in the post-synaptic neuron. This happens when a neurotransmitter binds to its receptor in the postsynaptic neuron. IPSP refers to inhibitory postsynaptic potential. This happens when a neurotransmitter binds to its receptor in the postsynaptic neuron. This binding prevents the generation of an action potential in the post-synaptic neuron.	2

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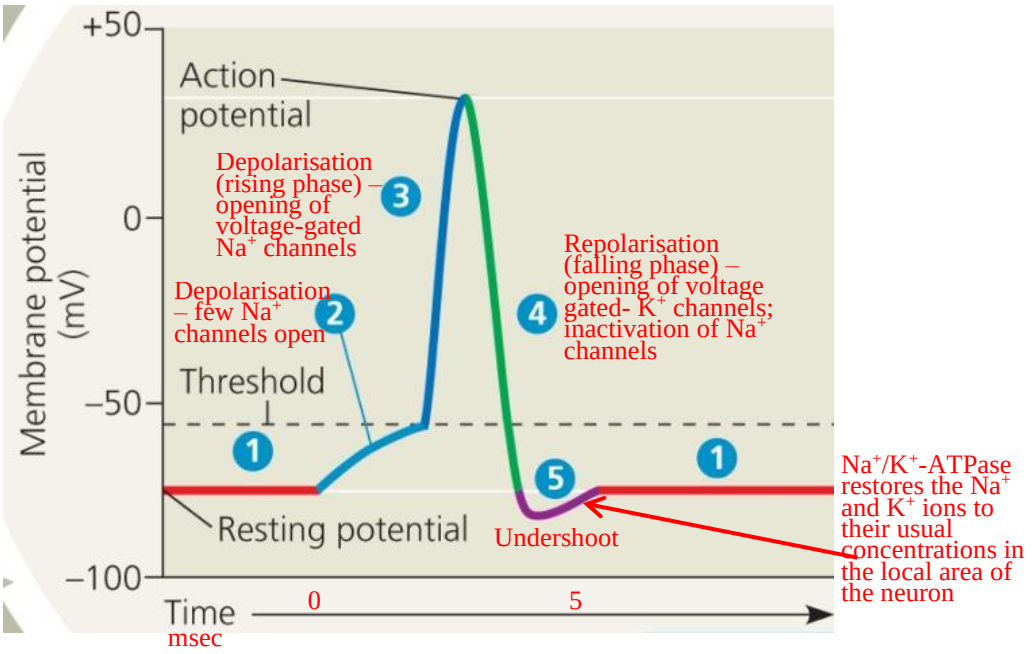
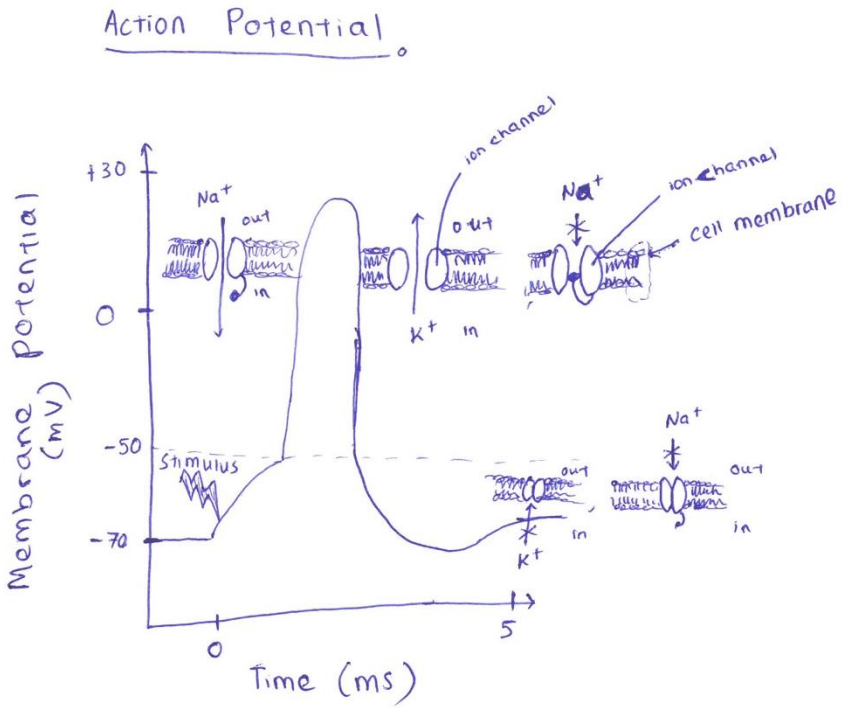
3		3
4		4

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	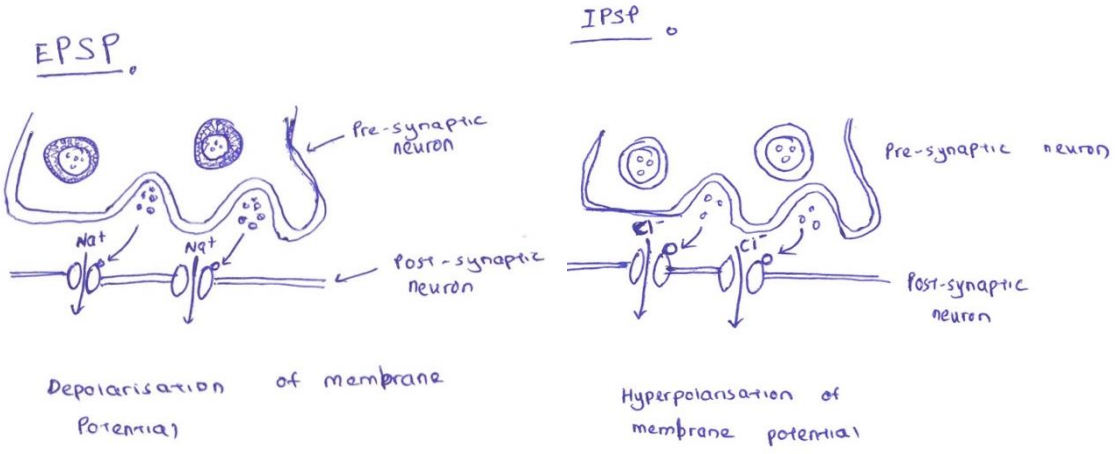	
5	A. Action Potential generation • Ions are charged molecules and require ion channels to pass through the cell membrane. • Na^+/K^+ pump utilises ATP to pump 3 Na^+ ions out of the cell and $2K^+$ ions into the cell, against their concentration gradient to establish resting membrane potential. • Stimulus of sufficient strength → hits threshold (-50 mV) → voltage-gated Na^+ channels open → depolarisation up to (+30 mV) → voltage-gated K^+ channels start to open (because they open quickly when the membrane potential reaches at least +20 mV), Na^+ channels close → hyperpolarisation (<-70 mV). B. Neurotransmitter release at synaptic vesicles • Action potential arrives at the synaptic terminal • Activates voltage-gated Ca^{2+} channels • Stimulates release of vesicle-bound neurotransmitter into synaptic cleft • Released when membrane of vesicles fuses with the cell membrane, which is a process called exocytosis • Since this is an example of direct synaptic transmission, neurotransmitters then travel across the synaptic cleft to bind with the G protein-coupled receptors (GPCRs) on the post-synaptic neuron leading to the production of 2nd messengers that determine whether an EPSP or IPSP is generated. C. EPSP/IPSP The probability of the post-synaptic neuron firing an action potential is partially dependent on the type of second messenger generates after neurotransmitter binding to the GPCRs. • EPSP will be generated if binding of the neurotransmitter onto its receptor causes depolarisation of the membrane potential of the post-synaptic neuron, e.g. if binding of the neurotransmitter onto the receptor causes an increase in a cation, such as Na^+, influx into the cell. • IPSP will occur if binding of the neurotransmitter onto its receptor causes hyperpolarisation of the membrane potential of the post-synaptic neuron, e.g. if	5

	binding of the neurotransmitter onto the receptor causes an increase in anion, such as Cl^-, influx into the cell. The strength of the signal is the other factor that would influence the probability of action potential generation in the post-synaptic neuron. • EPSP and IPSP effects can be amplified through two processes called spatial and temporal summation. Spatial summation occurs when two or more pre-synaptic neurons signal to the post-synaptic neuron while temporal summation occurs when one pre-synaptic neuron successively fires signals to the post-synaptic neuron. A post-synaptic action potential is generated if the signal strength of the EPSP is sufficient to reach the membrane potential threshold, resulting in the opening of Na^+ ion channels to repeat point A. For example, Georgia would withdraw her finger from the hammer when EPSP of sufficient strength signals the muscles in her arm and forearm to contract.	
6	(i) Action potential generation • Ions are charged and require ion channels to pass through the hydrophobic central region of the phospholipid bilayer of the cell membrane. • Stimulus of sufficient strength → membrane potential reaches threshold (-50 mV) due to opening of a few voltage-gated Na^+ channels → all voltage-gated Na^+ channels open and Na^+ ions move down their concentration gradient into the neuron → depolarisation of the neuronal membrane up to (+30 mV) → voltage-gated K^+ channels start to open (after responding slowly to the depolarising signal) so K^+ ions move down their concentration gradient out of the cell, and Na^+ channels become inactivated and gradually close preventing any further loss of Na^+ ions → there is some overshoot with a small hyperpolarisation (<-70 mV) → finally, Na^+/K^+-ATPase restores the local concentrations of Na^+ and K^+ ions so that the neuron can again be stimulated. (ii) Direct synaptic transmission generating EPSPs and IPSPs • When the action potential reaches the synaptic terminal, voltage-gated Ca^{2+} channels open due to the depolarising signal, and the increase in free Ca^{2+} stimulates release of vesicle-bound neurotransmitter into the synaptic cleft • Neurotransmitter then binds with the ligand-gated ion channels (which is another term for ion channel linked receptors) on the post-synaptic neuron • EPSPs will be generated if binding of the neurotransmitter onto its receptor causes depolarisation of the membrane potential of the post-synaptic neuron, e.g. if binding of the neurotransmitter onto the receptor causes an increase in influx of a cation, such as Na^+, into the cell. IPSPs will occur if binding of the neurotransmitter onto its receptor causes hyperpolarisation of the membrane potential of the post-synaptic neuron, e.g. if binding of the neurotransmitter onto the receptor causes an increase in anion influx, such as Cl-, into the cell or an increase in movement of cation, such as K^+, out of the cell.	6

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Section V

What mark would you now assign to your answer for Q(a)?
(0 – 3; use half scores)
Score:

What mark would you now assign to your answer for Q(b)?
(0 – 6; no half scores)
Score:

What did you think of the activity that you have just completed?
Do you consider it useful for your learning and exam preparation?
Explain in a few lines.