RIVER VALLEY HIGH SCHOOL
YEAR 6
PRELIMINARY EXAMINATION II

CANDIDATE NAME									
CENTRE NUMBER	S					INDEX NUMBER				

H2 BIOLOGY **9648/02**

Paper 2 Core Paper **15 Sep 2016**

2 hours

Additional Materials: Answer Paper

READ THESE INSTRUCTIONS FIRST

Write your index number and name on all the work you hand in.
Write in dark blue or black pen.
You may use an HB pencil for any diagrams or graphs.
Do not use staples, paper clips, glue or correction fluid.
DO **NOT** WRITE IN ANY BARCODES.

Section A
Answer **all** questions in the spaces provided on the question paper.

Section B
Answer any **one** question on the answer paper provided.
Circle the question attempted on the cover page.

The use of an approved scientific calculator is expected, where appropriate. You may lose marks if you do not show your working or if you do not use appropriate units.

At the end of the examination, fasten all your work securely together. The number of marks is given in brackets [ ] at the end of each question or part question.

For Examiner's Use	
Section A	
1	**/ 10**
2	**/ 10**
3	**/ 9**
4	**/ 10**
5	**/ 10**
6	**/ 11**
7	**/ 10**
8	**/ 10**
Section B	
9 or 10*	**/ 20**
Total	**/ 100**

This Question Paper consists of **23** printed pages.

Section A (80 marks)

Answer **all** the questions in this section.

1 **Fig. 1.1** shows photomicrographs of a zebrafish cell undergoing mitotic cell division.

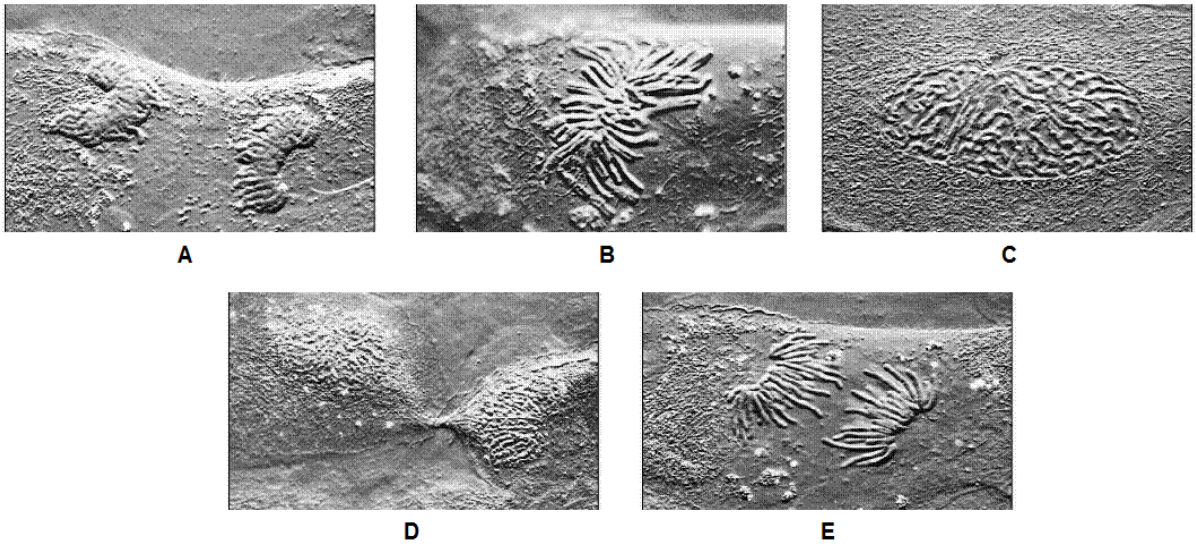

Fig. 1.1

(a) Explain the significance of mitosis in the development of zebrafish. [2]

1. **Produces genetically identical cells;**
2. **resulting in genetic stability in a zebrafish;**
3. **Allows growth;**
4. **into a multicellular zebrafish;**
5. **Allows replacement of worn-out / damaged tissues;**

Reject: asexual reproduction as context given is zebrafish

(b) **(i)** Identify stage **A**. [1]

Telophase;;

(ii) With reference to **Fig. 1.1**, describe two visible features that support the identification in **1(b)(i)**. [2]

1. **Chromosomes de-condense into chromatins;;**
2. **Chromatin are gathered at opposite poles of the cell;;**

(iii) Explain the significance of stage **D** in cell division. [2]

1. **Cytokinesis;**
2. **Separation of cytoplasmic materials;**
3. **and chromosomes;**
4. **into <u>two</u> daughter cells;**

(c) Explain why sister chromatids are genetically identical. [3]

1. **Sister chromatids are formed by DNA replication (in S phase);**
2. **which is semi conservative;**
3. **The two parental strands separate;**
4. **Each parental strand act as a template;**
5. **for the synthesis of a daughter strand;**
6. **via complementary base pairing;**

[Total: 10]

2 Human Immunodeficiency Virus (HIV) is a retrovirus which infects immune cells expressing CD4 receptor on its cell surface membrane.

(a) **(i)** Explain the term retrovirus. [2]

1. **Obligate parasite;**
2. **with single stranded RNA/RNA genome (positive sense);**
3. **Contains enzyme <u>reverse transcriptase</u>;**
4. **which can reverse transcribe <u>RNA to cDNA</u>;**
5. **which can be <u>integrated</u> into host chromosome;**

In 2012, the United States Food and Drug Administration (FDA) approved the OraQuick In-Home HIV Test, which is the first test kit which can be bought at pharmacies. If an individual had been infected by HIV for at least a month, there is a low probability of a false-negative result, whereby the kit incorrectly reports a negative result.

The test kit relies on the presence of antibodies against gp120 in blood. Antibodies are produced by immune cells in response to exposure to foreign particles.

Fig 2.1 shows the changes in concentration of HIV RNA and antibodies against gp120 in the blood stream after HIV infection.

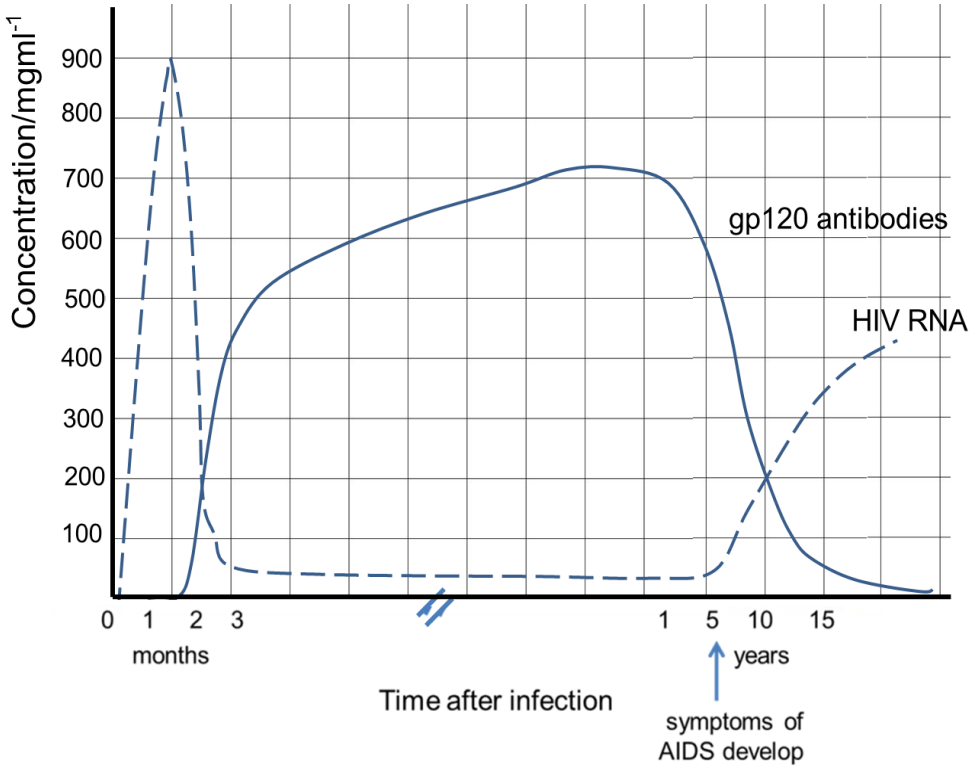

Fig 2.1

Adapted from Hunt, 2016, Virology, Microbiology and Immunology On-line.

http://www.microbiologybook.org/lecture/hiv3.htm

(b) With reference to **Fig 2.1**,

(i) describe how the concentration of gp120 antibodies in blood changes in relation to the concentration of HIV RNA in the first 3 months after infection; [2]

1. **From 0 to 1.5 month after infection, concentration of HIV RNA increases from 0 to 900 mgml^{-1} while concentration of gp120 antibodies remains at 0 mgml^{-1};;**
2. **From 1 to 3 months after infection, concentration of HIV RNA decreases from 900 mgml^{-1} to 50 mgml^{-1}while concentration of gp120 antibodies increases from 0 mgml^{-1} to 410 mgml^{-1};;**

(ii) explain how HIV RNA concentration increases in the first month after infection; [1]

1. **HIV RNA synthesised using HIV cDNA;**
2. **as a template;**
3. **using host cell machinery;**

(iii) explain why presence of gp120 antibodies is used as a basis for the detection of HIV infection. [2]

1. **A month after infection, gp120 antibodies concentration in blood increases;**
2. **and remains high throughout dormant period;**

or

1. **HIV RNA concentration is low after 3 months/during dormancy;**
2. **May not be accurately detected by kit (leading to false negative test results)**

Enveloped viruses like HIV leave the host cell via budding, but T4 bacteriophages use a different mechanism for release.

(c) Explain why release of HIV differs from the release of bacteriophages. [3]

1. **Budding from host cell allows HIV to acquire glycoproteins gp120 and gp41;;**
2. **which are embedded on host cell surface membrane;**
3. **While T4 bacteriophages exit the host cell via lysis of host cell;**
4. **If HIV is released via lysis of host cell, it will not be enclosed by viral envelope/lysis does not allow for virus to acquire viral envelope;;**

[Total: 10]

3 **Part I**

An *in-vitro* transcription system allows a DNA segment from yeast to be successfully transcribed under the control of a eukaryotic promoter. Transcription of this DNA segment occurs when purified components (RNA polymerase II and general transcription factors) are added.

However, this *in-vitro* transcription system using purified components occurs at low efficiency, as compared to that using nuclear extract. This suggests that an important gene regulatory protein present in the nuclear extract is missing from the purified components.

(a) **(i)** State a possible identity of the missing gene regulatory protein. [1]

Activator protein;;

(ii) Describe how the gene regulatory protein identified in **3(a)(i)** could result in higher efficiency of transcription. [1]

1. **Binds to enhancer;**
2. **Increases the probability of (general) transcription factors binding / RNA polymerase (II) binding / forming transcription initiation complex;**

To search for the DNA sequence to which this gene regulatory protein binds, five segments located upstream of the transcription start site (+1) is each deleted in various experimental set-ups. The TATA box is located 15 base-pairs upstream of the transcription start site. Each deleted template is incubated with a non-deleted template, which serves as a control. The four deletion sites are shown in **Fig. 3.1**.

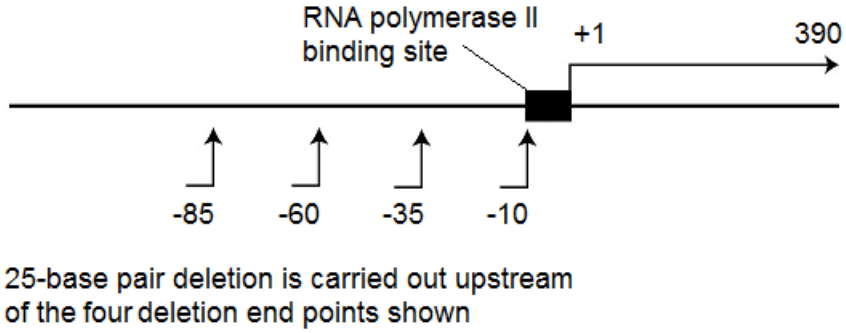

Fig. 3.1

The transcription activity of these deletions in the transcription system using nuclear extract is shown in **Table 3.1**.

Table 3.1

Deletion end point	-10	-35	-60	-85
Activity in deleted template / a.u.	0	10	24	23
Activity in non-deleted template / a.u.	23	24	24	23

(b) **(i)** With reference to **Fig. 3.1** and **Table 3.1**, describe the extent of change in transcription activity caused by different deletions. [2]

1. **Deletion end point of -10 resulted in no transcription;**

Accept: quoting of drop from 23 a.u to 0 a.u.

2. **Deletion end point of -35 reduces transcription activity by 14 a.u.;**

Accept: quoting of drop from 24 a.u to 10 a.u.

3. **Deletion end points of -60 has no impact on transcription activity;**
4. **Deletion end points of -85 has no impact on transcription activity;**

(ii) Suggest a reason for the transcription activity at -10 deletion end point. [2]

1. **Deletion of the TATA box;**
2. **results in (general) transcription factors unable to bind;**
3. **RNA polymerase (II) could not bind / transcription initiation complex could not form;**
4. **Transcription not initiated;**

(ii) Deduce the binding site of the gene regulatory protein. [1]

35 to 59 base pairs upstream of start site;;

Accept: 35 to 60 base pairs

Part II

In a separate experiment that studies the effect of starvation on yeast cells, it was observed that the cells upregulate the synthesis of GCN4 protein when deprived of purine. The α subunit of eukaryotic initiation factor 2 (eIF2) was found to be phosphorylated, and this leads to increased translation of mRNA encoding GCN4.

(c) **(i)** State the level of gene regulation employed for GCN4. [1]

Translation control;;

(ii) Explain why this level of gene regulation may be advantageous to the survival of yeast cells. [1]

1. **Allows for yeast cells to respond quickly to environment changes;**
2. **gaining selective advantage;**

[Total: 9]

4 In pigeon, pigment distribution is controlled by two genes. In the presence of the dominant allele for spread pigmentation, no pattern is observed. **Fig 4.1** shows the appearance of pigeon with spread and patterned pigmentation. In a farm, pure-breeding pigeon with spread pigmentation and pure-breeding pigeon with barless pattern pigmentation were bred, all the pigeons in the F1 generation have spread pigmentation.

Patterned pigmentation

Spread pigmentation

Fig. 4.1

When the F_1 pigeons were allowed to interbreed, the phenotype and number of offspring were recorded.

Pigeon with spread pigmentation	86
Pigeon with barless pattern pigmentation	8
Pigeon with bar pattern pigmentation	23

Use the following symbols to represent the alleles:

S – Spread **s** – no spread

B – Bar pattern **b** – barless

(a) Draw a genetic diagram in the space below to explain the cross described. [4]

F1 phenotype: Spread pigment x Spread pigment

Interbreeding between F_1: **SsBb;** x **SsBb;**

F1 gametes;; **SB** **Sb** **sB** **sb** x **SB** **Sb** **sB** **sb**

Random fertilization of F1 gametes:

	SB	**Sb**	**sB**	**sb**
SB	SSBB Spread	SsBb Spread	SsBB Spread	SsBb Spread
Sb	SsBb Spread	SSbb Spread	SsBb Spread	Ssbb Spread
sB	SsBB Spread	SsBb Spread	ssBB bar	ssBb bar
sb	SsBb Spread	Ssbb Spread	ssBb bar	ssbb barless

Genotype;;

Phenotype;;

F2 phenotypic ratio 12 spread: 3 bar : 1 barless

(b) State the name for this type of interaction between gene loci. [1]

Dominant epistasis;;

(c) Explain how different genotypes give rise to spread pigmentation in pigeons [2]

1. **Genotypes S_ _ _, result in spread pigmentation;**
2. **Dominant allele S is epistatic over gene locus B/b;**
3. **Encodes for sufficient amount of inhibitor;**
4. **To prevent formation of patterned pigmentation;**

The χ^2 distribution table and equation to calculate χ^2 is shown below. Using the formula shown below, the calculated χ^2 value for the cross is 4.3.

$$\chi^2 = \Sigma \frac{(O-E)^2}{E}$$

Table 4.1

Degree of freedom	Probability, p				
	0.10	0.05	0.02	0.01	0.001
1	2.71	3.84	5.41	6.64	10.83
2	4.61	5.99	7.82	9.21	13.82
3	6.25	7.82	9.84	11.35	16.27
4	7.78	9.49	11.67	13.28	18.47

(d) Using the calculated χ^2 value and **Table 4.1,** explain what conclusion can be drawn from the recorded data. [2]

1. **At v=2, calculated χ^2 value corresponds to p-value more than 0.10;**
2. **Which is more than 0.05;**
3. **Do not reject H_0 in favour of H_1;**
4. **There is no significant difference between the observed and expected values;**

(e) Suggest one reason why the conclusion in **(d)** may not be valid. [1]

Small sample size;;

[Total: 10]

5 **Fig 5.1A** and **Fig 5.1B** are electron micrographs of the same plant cell.

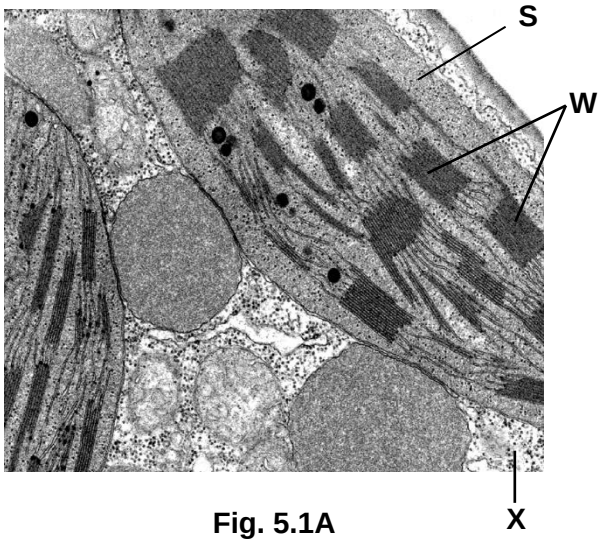

Fig. 5.1A

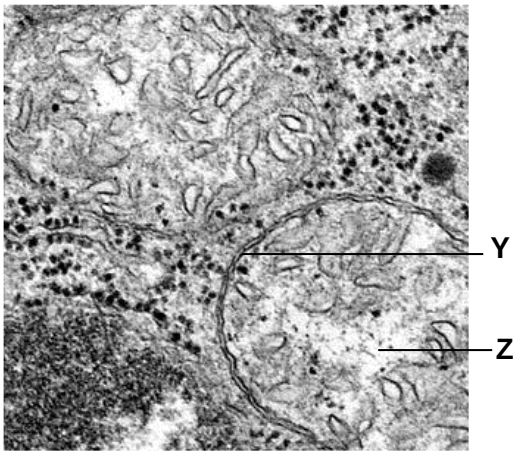

Fig. 5.1B

Source: http://botit.botany.wisc.edu/Resources/Botany/

(a) State in which labelled component(s) will there be the highest concentration of [2]

RuBP carboxylase	**S;**
ATP synthase	**W, Y;**
pyruvate decarboxylase	**X;**
acetyl-coA	**Z;**

The optimum pH for the activity of RuBP carboxylase is pH8.

(b) Explain why the illumination of chloroplasts leads to optimum pH condition for RuBP carboxylase. [3]

1. **RuBP carboxylase function in the stroma;**
2. **Where protons are present from photolysis of water;**
3. **Light results in high energy electrons displaced from special chlorophyll a/photosystem;**
4. **Pass along the electron transport chain;**
5. **Protons pumped from stroma to thylakoid lumen;**
6. **Protons leaving the stroma increases the pH;**

(c) Herbicide **X** binds irreversibly to RuBP carboxylase. Explain how herbicide **X** kills weeds. [3]

1. **RuBP and CO_2;**
2. **cannot bind to active site of RuBP carboxylase;**
3. **Carbon fixation cannot take place;;**
4. **Triose phosphate not produced;**
5. **Weeds die because cells cannot synthesis glucose for aerobic respiration/cannot synthesised amino acids for translation;**

(d) Describe two ways in which the reactions of the Calvin cycle differs from Kreb's cycle. [2]

Factor	Calvin cycle	Kreb's cycle
Type of reactions;;	Reduction reactions	Oxidation reactions
Coenzyme;;	NADPH/H+	NAD+
Role of ATP;;	ATP hydrolysed to provide energy for phosphorylation of GP / regeneration of RuBP	ATP produced from substrate level phosphorylation
Role of CO_2;;	Reduced to form triose phosphate	Released as by-product during oxidative decarboxylation

1 mark per comparison;;

Reject comparison relating to location

[Total: 10]

6 ATP-binding cassette (ABC) transporters are transmembrane proteins that utilises the energy from ATP binding and hydrolysis to transport various substances across cellular membranes. They exhibit the ability to switch between two states upon hydrolysis of ATP. Most eukaryotic ABC transporters function as part of the efflux system, removing substances out of cells.

The human ABC-B1 transporter is responsible for multiple drug resistance observed in patients, rendering a variety of structurally unrelated drugs ineffective in treatment of diseases. **Fig. 6.1** shows the structure of a ABC-B1 transporter.

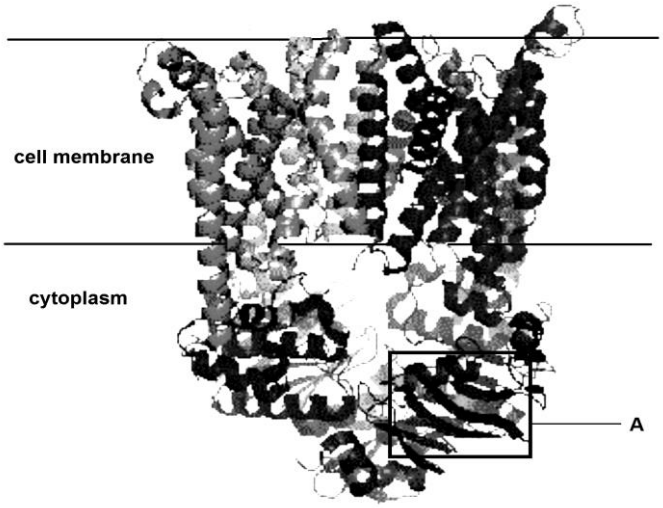

Fig. 6.1

(a) **(i)** Describe how structure **A** is folded. [2]

1. **Hydrogen bonds;**
2. **form between CO and NH groups;**
3. **of adjacent regions of a polypeptide chain;**
4. **that lie parallel to each other;**

(ii) Explain how ABC-B1 transporter is held in the cell membrane. [3]

1. **Hydrophobic interaction;**
2. **between non-polar R groups of ABC-B1 transporter;**
3. **and non-polar hydrocarbon tails of phospholipid molecules;**
4. **H bonds;**
5. **between polar R groups of ABC-B1 transporter;**
6. **and polar phosphate head of phospholipid molecules;**

Accept: ionic bonds between oppositely charged R groups and phosphate heads

Digoxin is drug derived from the leaves of a plant, and is used in treatment of congestive heart failure. Digoxin is polar in nature, thus is retained in cells by the cell membrane to exert its effect. However, patients are observed to develop resistance to digoxin due to the increased number of ABC-B1 transporters removing digoxin out of cells.

(b) Explain how ABC-B1 transporter removes digoxin out of cells. [3]

1. **Via active transport;**
2. **Digoxin binds to (cytoplasmic) binding site of ABC-B1 transporter;**
3. **triggering change in three dimensional conformation of ABC-B1 transporter;**
4. **with the binding / hydrolysis / investment of ATP;**
5. **This allows digoxin to move through its hydrophilic channel;**
6. **against its concentration gradient;**
7. **shielded from hydrophobic core of membrane;**

To overcome the drug-resistance, patients may be prescribed with verapamil, an inhibitor of ABC-B1 transporter.

In a clinical trial to determine the effectiveness of verapamil, fluorescent-tagged verapamil was administered to a patient with overexpression of ABC-B1 transporter proteins.

At various time intervals, the relative fluorescence of the target cells was measured and the results are recorded in **Table 6.1**.

Table 6.1

Time after administration of verapamil / h	Relative fluorescence / rfu
20	15.7
40	9.8
60	5.4
80	2.3

(c) **(i)** State a feature of verapamil that allows for it to carry out its function. [1]

Complementary shape to binding site on ABC-B1 transporter protein;;

Permanent / stable / irreversible binding to ABC-B1 transporter protein;;

Reject: structurally different from digoxin, as this does not help in its function.

(ii) Describe the results shown in **Table 6.1**. Suggest a reason for the observation. [2]

Describe

The relative fluorescence of the target cells decreases from 15.7 rfu 20h after administration of verapamil to 2.3 rfu 80h after administration of verapamil;;

Reason

Verapamil is broken down / transported out of the cell;;

Binding of verapamil is not stable / permanent;;

Reject: metabolised with no reference to anabolised / catabolised.

[Total: 11]

7 **(a)** Explain the role of calcium ions in synaptic transmission. [3]

1. **Influx of Ca^{2+};**
2. **into synaptic knob / pre-synaptic membrane;**
3. **causing membrane of vesicles to fuse with pre-synaptic membrane;**
4. **releasing acetylcholine;**
5. **into synaptic cleft;**
6. **via exocytosis;**

GABA is a neurotransmitter which inhibits the production of action potential. **Fig. 7.1** and **Fig. 7.2** shows how the release of GABA from a pre-synaptic neurone affects the membrane potential of a post-synaptic membrane.

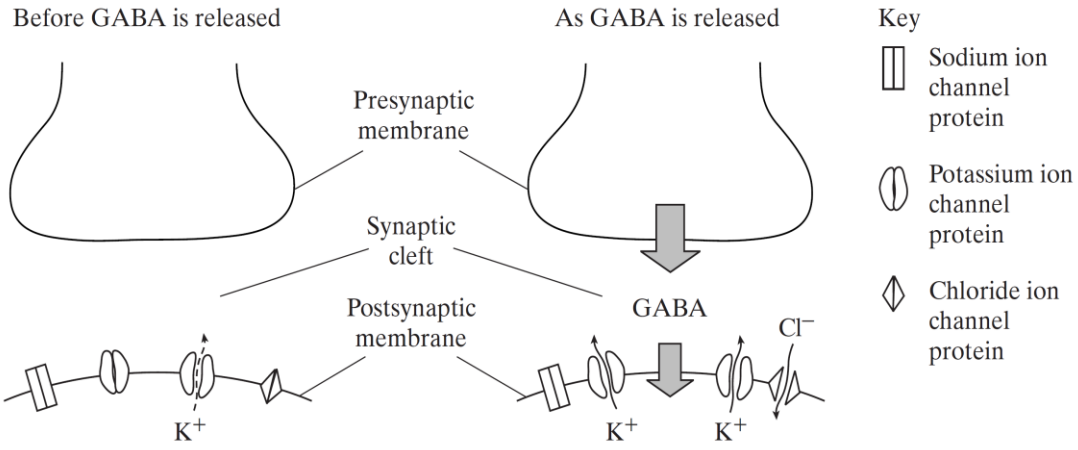

Fig. 7.1

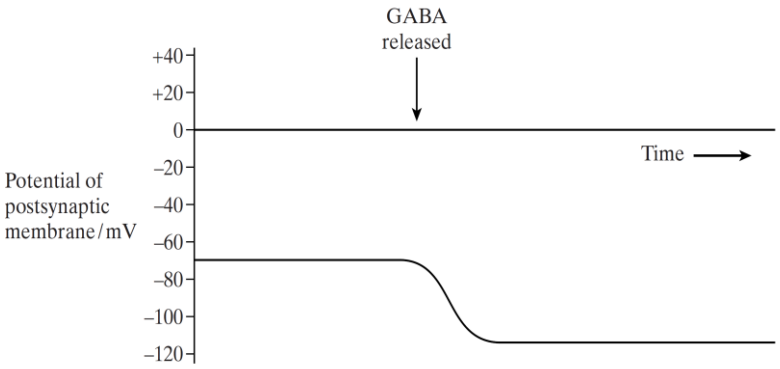

Fig. 7.2

(b) When the post-synaptic membrane is stimulated by acetylcholine, an action potential is less likely to occur if GABA is released. Explain why. [3]

1. **GABA opens ligand-gated K^+ and Cl^- channels (in post-synaptic membrane);**
2. **Allowing K^+ to diffuse out of post-synaptic neurone;**
3. **and Cl^- to diffuse into post-synaptic neurone;**
4. **Causing hyperpolarisation;**

Accept: membrane potential below resting potential

5. **Stimulation greater than normal / more Na+ influx is required;**
6. **to reach threshold potential (to trigger action potential);**

Epilepsy is a neuronal disorder which causes recurrent, unprovoked seizures. This may result when there is increased neuronal activity in the brain.

One form of epilepsy is due to insufficient GABA. GABA is broken down on the post-synaptic membrane by the enzyme transaminase. Vigabatrin is a new drug used to treat this form of epilepsy. The drug has a similar molecular structure to GABA.

(c) Suggest how Vigabatrin may be effective in treating this form of epilepsy. [2]

1. **Vigabatrin binds to GABA transaminase;**
2. **via complementary shape;**
3. **Prevents breakdown of GABA / more GABA available;**
4. **Reduces neuronal activity / frequency of action potential;**

Fig. 7.3 shows the relationship between diameter of the axon and the speed of conduction of nerve impulses in the myelinated axons of a cat.

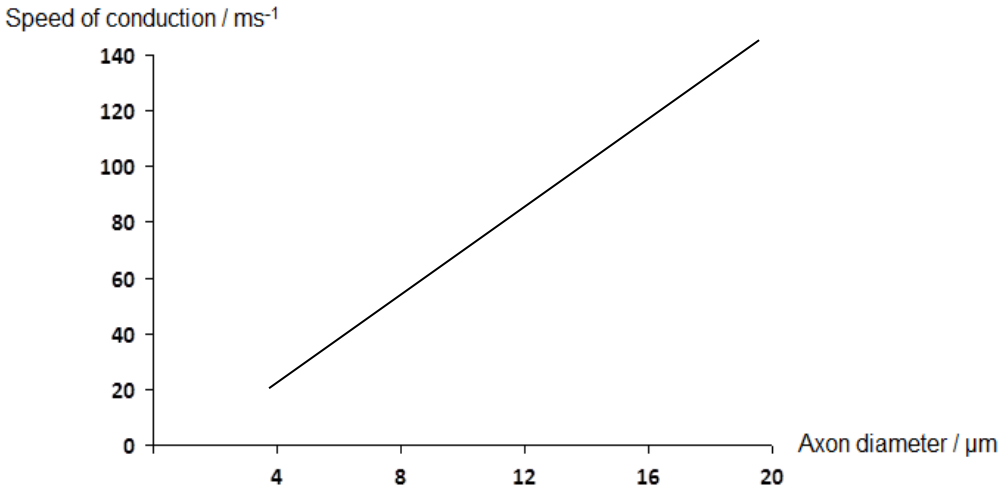

Fig. 7.3

(d) As the diameter of the axon increases, the length of myelination between the nodes increases.

Explain how this resulted in the speed of conduction shown in **Fig 7.3**. [2]

1. **As diameter of axon increases from 4µm to 20µm, length of myelination between nodes increases, thus speed of conduction increases from 20ms^{-1} to 140ms^{-1};;**
2. **Fewer depolarisations/action potentials to travel the length of the axon;**
3. **when impulse transmits from node to node / travel by saltatory conduction;**

[Total: 10]

8 The Canary Islands form an archipelago of seven volcanic islands just west of the African continent. Lanzarote is the oldest island of about 24.0 million years old in the island chain while Hierro is the youngest island of about 0.8 million years old. The distribution of three species of lizards of the genus Gallotia in the Canary Islands is investigated.

Fig 8.1 shows the distribution of the lizard species in these islands and the maximum age of the island. **Fig 8.2** shows the relative body size of the lizards found in these islands.

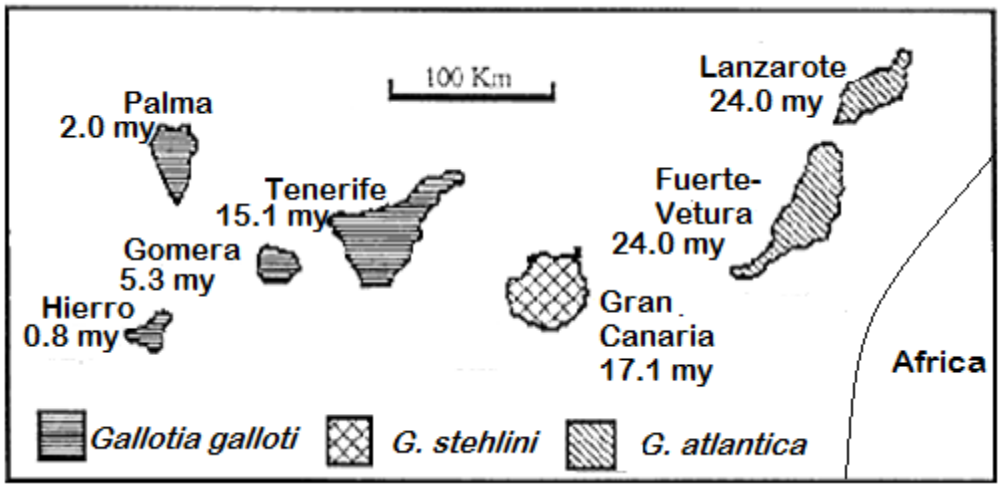

Fig. 8.1

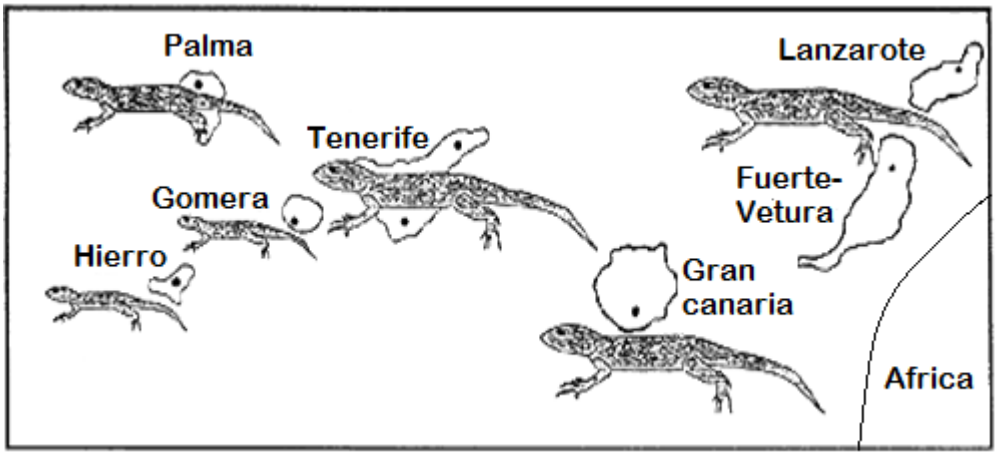

Fig. 8.2

(a) **(i)** Explain how the distinct phenotypic differences between the lizard populations may have arisen. [5]

1. **Geographical isolation as a result of separated islands;**
2. **No gene flow between populations on different islands;**
3. **Mutation;**
4. **Leads to variation in body size in a population;**
5. **Different environment on different islands acts as selection pressure;**
6. **Natural Selection occurs;**

7. **Lizards with body size <u>best suited</u> for the environment are selected for;**
8. **Survive and <u>reproduce</u>**
9. **Pass on favourable alleles to offspring;**
10. **Change in <u>allelic frequency</u> over time;**
11. **Genetic drift (such as founder's effect);**

(a) (ii) Suggest why the lizard populations on Tenerife, Palma, Gomera and Hierro are classified as a single species. [1]

1. **Individuals from different population able to interbreed;**
2. **To produce fertile offspring;**

The cytochrome b gene from the different populations of lizard is sequenced. The cytochrome b gene sequences were then compared and the difference in number of base pairs is summarised in the table below.

Table 8.1

G. stehlini	*G. stehlini*					
G. atlantica	36	*G. atlantica*				
G. galloti Palma	41	25	*G. galloti* Palma			
G. galloti N. Tenerife	40	23	8	*G. galloti* N. Tenerife		
G. galloti S. Tenerife	40	19	10	6	*G. galloti* S. Tenerife	
G. galloti Gomera	45	24	19	19	15	*G. galloti* Gomera
G. galloti Hierro	49	28	19	21	17	4

N. Tenerife - North of Tenerife island
S. Tenerife - South of Tenerife island

G. galloti Palma — species name / location

(b) Describe how these changes in DNA sequences and dates of island formation can help taxonomist to classify the lizards on the Canary Islands accurately. [4]

1. **Neutral mutations are accumulated at a relatively constant rate;;**
2. **By determining the number of mutations/differences in DNA bases between lizards of different populations;**
3. **And by using the age of the island for calibration;**
4. **Can be used to track mutations accumulated over a period of time / calculate rate of mutation;**
5. **via a molecular clock;;**
6. **Can calculate time since divergence;**

[Total: 10]

Section B (20 marks)

Answer **one** question.

Write your answers on the separate answer paper provided.
Your answers should be illustrated by large, clearly labelled diagrams, where appropriate.
Your answers must be in continuous prose, where appropriate.
Your answers must be set out in sections **(a)**, **(b)** etc., as indicated in the question.

A **NIL** return is necessary if you have not attempted this section.

9. **(a)** Outline how the structure of membranes in the endomembrane system facilitates their function. [10]

1. **The endomembrane system comprises the (outer nuclear membrane), rough endoplasmic reticulum, Golgi apparatus, (associated vesicles) and cell surface membrane;;**
2. **Membranes in the endomembrane system forms a continuous network for protein synthesis;;**
3. **Membranes of endomembrane system have similar composition allowing transfer of membrane (as vesicles) from one organelle to the next organelle;;**
4. **Fluid nature of phospholipid bilayer allows for fusion and budding of vesicles with membrane;;**
5. **Nuclear pores of nuclear membrane allows export of mRNA for protein synthesis at RER;;**
6. **Endoplasmic reticulum membrane allows for ribosomes to be embedded for protein synthesis;;**
7. **Endoplasmic reticulum membrane enclose lumen for folding of protein;;**
8. **RER membrane forms transport vesicle through budding for**

(intracellular) transport;;

9. **Membrane on the cis face of Golgi apparatus allows for fusion with transport vesicle membrane/for proteins can enter;;**
10. **Golgi apparatus membrane enclose lumen for modification/sorting;;**
11. **Membrane on the trans face of Golgi Apparatus allows for Golgi vesicle to bud off;**
12. **Membrane of secretory vesicle fuse with cell surface membrane, leading to exocytosis;;**
13. **AVP;;**

(b) Distinguish between tropocollagen and amylose. [10]

Differences

Factor	Tropocollagen	Amylose
1. Element present;;	C, H, O, N, S	C, H, O
2. Monomer;;	Amino acids	α-glucose
3. Types of monomers;;	20	1
4. Bonds between monomers;;	Peptide bond	α(1-4) glycosidic bonds
5. Inter-chain association;;	Yes (hydrogen bonds)	No
6. Number of chains;;	3 polypeptide chains	1 polysaccharide chain
7. Shape;;	Linear	Helical
8. Function;;	Structural role	Energy store
9. Further assembly;;	Can further assemble to form collagen fibres	Do not further assemble
10. Tensile strength;;	High tensile strength	Low tensile strength
11. Site of synthesis;;	Ribosomes/RER	Golgi apapatus;;
12. Occurrence;;	Animal cells	Plant cells

[Total: 20]

10. (a) Using a named example, describe how mutation may result in a disease and its associated symptoms. [11]

1. **Sickle cell anemia;;**

2. **Base pair substitution;**
3. **thymine replaced by adenine;**
4. **changes nucleotide sequence;**
5. **of β globin gene;**

6. **This changes codon 6 on mRNA;**
7. **to GUA;**
8. **resulting in missense mutation;**
9. **that changes glutamic acid;**
10. **to valine;**
11. **resulting in a neutral amino acid;**

Accept: hydrophobic amino acid

12. **This changes the folding / three dimensional conformation of haemoglobin / β globin;**
13. **generating a sticky patch;**
14. **on the surface of haemoglobin;**

15. **The deoxygenated form of mutant haemoglobin;**
16. **is insoluble in red blood cells;**
17. **forming crystalline arrays;**
18. **This causes red blood cells to form a sickle shape;**

19. **Sickle-shaped red blood cells are rigid;**
20. **and are often trapped in blood capillaries;**
21. **causing pain;**

22. **reduced oxygen supply to tissues;**
23. **causes cell death / tissue damage / organ failure;**
24. **haemolyse readily**
25. **accumulate in spleen / abnormal enlargement of spleen**

(b) Explain the role of nuclear membrane in regulating eukaryotic gene expression. [9]

1. **Nuclear membrane forms a barrier;**
2. **between nucleus and cytoplasm;**

3. **protect DNA / mRNA from degradation;**
4. **by cytoplasmic enzymes;**
5. **maintaining integrity of <u>template</u> for transcription / translation;**

6. **prevents mixing of intermediates;**
7. **of transcription and translation;**

8. **allows for post-transcriptional modification;**
9. **such as 5' capping / 3' polyadenylation / mRNA splicing;**

10. **Nuclear membrane is made up of phospholipid bilayer;**
11. **comprising a hydrophobic core;**
12. **rendering it selectively permeable;**
13. **Nuclear pores;**

14. **regulate the movement of mRNA out of the nucleus;**
15. **control availability of <u>template</u> for translation;**

16. **regulates the movement of ribosomal proteins into the nucleus;**
17. **and ribosomal subunits out of nucleus;**
18. **controlling synthesis of ribosomes;**
19. **for initiation of translation;**

20. **concentrates the enzymes / proteins;**
21. **such as RNA polymerase / transcription factors;**
22. **to increase frequency of transcription;**

[Total: 20]