EUNOIA JUNIOR COLLEGE
JC2 Preliminary Examinations 2024
General Certificate of Education Advanced Level
Higher 2

CANDIDATE NAME	**ANSWERS**

CIVICS GROUP	2	3	-			REGISTRATION NUMBER		

H2 Biology 9744/03

Paper 3 Long Structured and Free-response Questions **16 September 2024**

2 hours

Additional Materials: 12-page Answer Booklet

READ THESE INSTRUCTIONS FIRST

Write your name, civics group and registration number on all the work you hand in.
Write your answers in dark blue or black pen.
You may use an HB pencil for any diagrams or graphs.
Do not use paper clips, highlighters, glue, or correction fluid/tape.

Section A
Answer **all** questions on the **Question Paper**.

Section B
Answer **one** question on the **12-page Answer Booklet** provided**.**

Write your answer to each part of the question on a fresh sheet of paper.

The use of an approved scientific calculator is expected, where appropriate.

The number of marks is given in brackets [ ] at the end of each question or part question.

At the end of the examination, ensure that you submit both the Question Paper and Answer Booklet.

For Examiner's Use	
Section A	
1	
2	
3	
Section B	
4 OR 5	
Total	/75

This document consists of **14** printed pages and **2** blank pages.

Section A

Answer **all** questions on the Question Paper.

1 The development of a mouse from a fertilised egg into an adult is regulated by variations in DNA methylation.

Fig. 1.1 shows the developmental stages of a mouse with corresponding levels of DNA methylation.

R, **S** and **T** represent the zygote, blastocyst and embryo respectively.

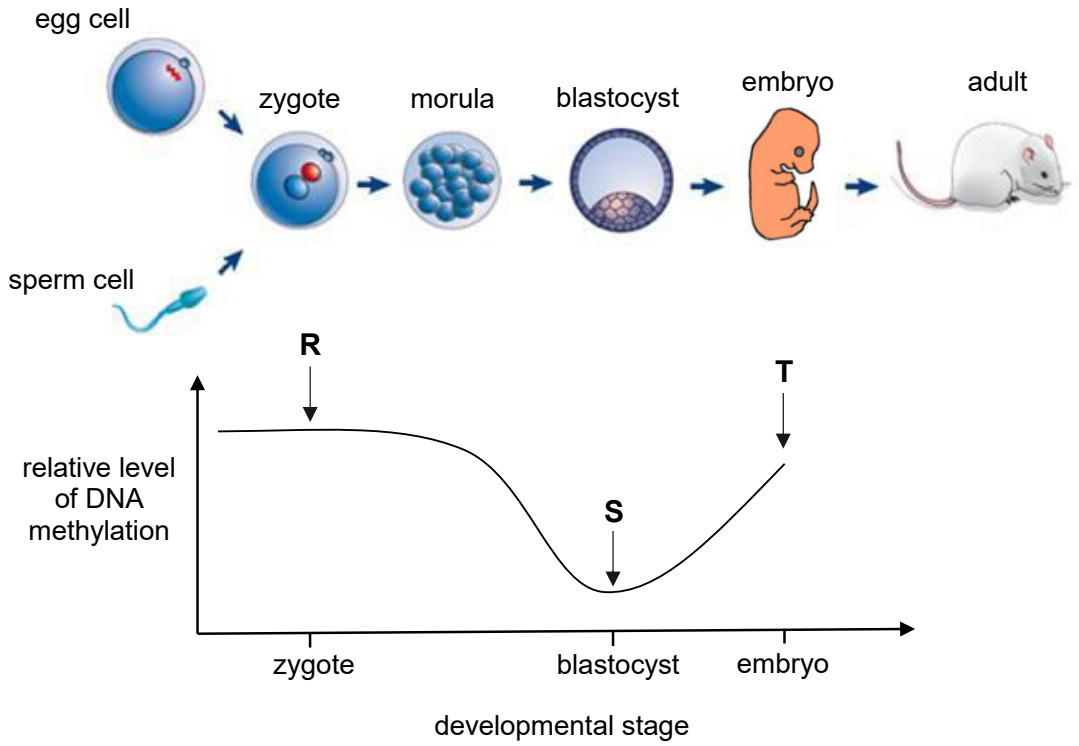

Fig. 1.1

(a) (i) Compare the features of a cell derived from the zygote with that from the inner cell mass of the blastocyst. [3]

3m = 2 similarities + 1 difference OR 1 similarity + 2 differences

[Similarities]

1. Both cells are **unspecialized and undifferentiated**; (any one)
2. Both cells can be **differentiated into specialized** cells;
3. Both cells are **capable of self-renewal and extensive proliferation**;

[Differences]

4. Cell derived from the zygote are **totipotent** while cell derived from inner cell mass of blastocyst is **pluripotent**; (A: former higher potency than latter);
5. Cell derived from the zygote has **higher level of methylation** while cell derived from inner cell mass of blastocyst has **lower level of methylation**;

R: Cell derived from the zygote is a zygotic stem cell while cell derived from the inner cell mass of the blastocyst is an embryonic stem cell [Identity; not a feature difference]

(ii) Explain how changes to DNA methylation from **R** to **S** bring about differentiation. [4]

1. Relative level of **DNA methylation decreases from R to S**
2. DNA methylation involves **addition** of **methyl groups** to CpG islands / selected cytosine nucleotides in promoters of totipotency genes, catalysed by **DNA methyltransferase**;
3. It **prevents transcription of genes necessary for maintaining totipotency** (OWTTE)
4. **Blocking binding of** (general) **transcription factors** and hence, **preventing the assembly of transcription initiation complex** at **promoter** and/or
5. **Recruiting chromatin remodeling complexes / histone deacetylases** (at least 1) to **condense chromatin** / cause DNA to be more tightly wound around histones

(iii) At different developmental stages of the mouse, the control of the telomerase gene expression is crucial.

Suggest if the telomerase gene in cells is likely to be methylated from **T** to an adult mouse. [1]

Not likely to be methylated because **telomerase** is **required** even in **adult stem cells**
OR
Likely to be methylated because **telomerase** is **inactivated** in terminally **differentiated / specialized cells**.

(iv) Active telomerase can be found in some cell types in a mouse.

Using a named example, explain the role of telomerase. [3]

1. Telomerase **elongate / extend telomeres** that would otherwise be **shortened after each cell division** (idea of compensating for end-replication problem), to allow for **maintenance of telomere length**.
2. and allows for **stem cell / cancer cell (reference to specific cell)** to continue **dividing indefinitely**;
3. Stem cell: **maintain pool of stem cells / differentiate to form more specialized cells over time**
   OR
   Cancer cell: **supports sustained tumour growth / accumulate genetic mutations over time**

In an experiment, chromatin from various tissues were isolated and treated with DNase, an enzyme that degrades naked double-stranded DNA. After digestion, the enzyme was removed. Any remaining intact DNA was extracted and mixed with radioactively labelled DNA probes specific for certain genes, under conditions that favoured nucleic acid hybridisation.

The levels of binding of the labelled DNA probes were measured. Some of the results are shown in Table 1.1 below.

Table 1.1

Sample	Tissue source of chromatin	Gene radioactive DNA probe is specific to	Percentage binding of radioactive DNA probe
1	Skeletal muscles	Myosin gene	25
2	Pancreas	Myosin gene	91
3	Skeletal muscles	Chymotrypsin gene	93

(b) (i) Outline the steps between extraction of intact DNA and mixing with radioactively labelled DNA probes for nucleic acid hybridization. [3]

1. **Gel electrophoresis** is carried out to **separate DNA fragments** by molecular **size**;
2. **Alkaline solution** to **denature double-stranded DNA** into single-stranded DNA;
3. **Single-stranded DNA** are **transferred from agarose gel slab to nitrocellulose membrane** (A: nylon membrane);

(ii) State two similarities between the probes used in the experiment and primers used in Polymerase Chain Reaction (PCR). [2]

1. Both are **complementary** in **sequence** to **target gene** (A: undergo complementary base-pairing with target gene);
2. Both are **single-stranded** polynucleotide chains;
3. Both are made of **DNA**;
4. AVP e.g. both are **artificially synthesized**;

(iii) Suggest how DNA in the chromatin are protected from digestion by DNase. [2]

1. **Negatively-charged DNA** are wound around **positively-charged histone octamers (A: histones)** to form nucleosomes, held together by **electrostatic interactions** (A: ionic bonds);
2. DNase can only degrade naked double-stranded DNA and are **not able to access and digest the DNA** when they are wound around histone octamers;

(iv) With reference to Table 1.1, explain which gene is more actively transcribed in skeletal muscle cells. [2]

1. **Myosin gene**;
2. The **percentage binding of radioactive DNA probe** to **myosin gene** is **25%**, which is **lower** than **93%** for **chymotrypsin gene**; (A: difference)

(v) Explain one type of protein modification that supports your answer to **(b)(iv)**. [2]

1. **Histone acetylation** (OR **histone de-methylation**);
2. Myosin gene is **less tightly wound around histones**; (A: chromatin decondense)
3. Hence, its **promoter** is **more accessible to transcription factors / RNA polymerase**;

Dystrophin is a cytoskeletal protein found in human muscles. The gene which encodes

dystrophin has 79 exons. Mutations in this gene leads to Duchenne muscular dystrophy (DMD), which causes progressive muscle impairment in children.

One of the most common mutations in the dystrophin gene, which occurs in exon 44, results in a non-functional protein. The asterisks (*) in Fig. 1.3 show the positions where deletions were detected in exon 44 and the reading frame is indicated by the boxes.

To treat DMD, scientists modified the dystrophin gene by removing exon 44, producing a partially functional protein.

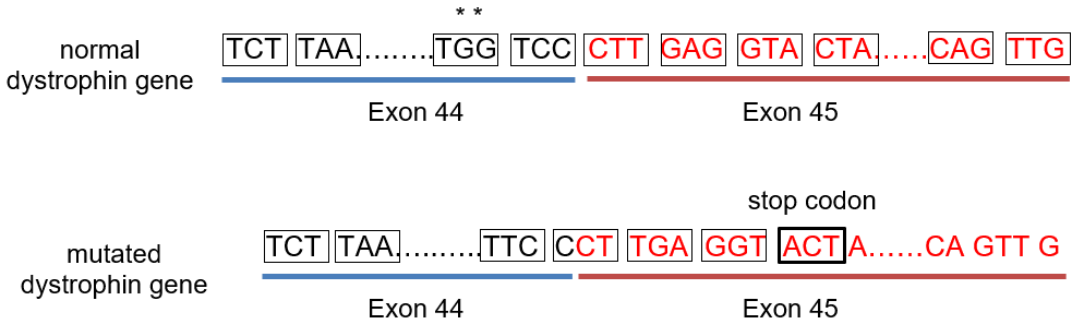

Fig. 1.3

(c) Explain how removal of exon 44 could result in production of a partially functional dystrophin protein. [3]

1. **Reading frame is restored**;
2. **Stop codon in exon 45 is removed**, thus **preventing premature termination of translation**;
3. A **shorter polypeptide** is **synthesised** as it **lacks the amino acid sequence coded for by exon 44**;
4. However, as it **folds into similar conformation**, it is partially functional;

Duchenne muscular dystrophy (DMD) is a X-linked recessive disorder. Mary and John are a normal, healthy couple without DMD who gave birth to their first-born son with DMD. They went for genetic testing and discovered that Mary is a carrier.

(d) (i) Using suitable symbols, identify the genotypes of the following individuals. [2]

Mary: X^DX^d
John: X^DY
First-born son with DMD: X^dY

2 correct: 1m
All correct: 2m

(ii) Calculate the probability that their second child is also a son with DMD. [1]

P (second child is son with DMD) = ½ x ½ = ¼

(e) Rare females with DMD have a translocation that disrupts the dystrophin gene on one X chromosome and causes non-random inactivation of the normal X chromosome, resulting in

the expression of the disease.

Molecular characterization of the translocation junctions revealed reciprocal translocation between the X chromosome and an autosome, with both deletion and addition of nucleotides at the junction.

Suggest how reciprocal translocation and non-random X-chromosome inactivation lead to expression of the diseased phenotype. [2]

1. **Addition and deletion of nucleotides** (A: change in nucleotide sequences) caused by **reciprocal translocation** in one X chromosome result in **production of non-functional dystrophin protein**;
2. **Functional dystrophin protein cannot be expressed** due to **X-chromosome inactivation** of the other normal chromosome;

[Total: 30]

2 Cyanobacteria are a group of bacteria that obtains their energy through photosynthesis. They carry an operon known as phycocyanin operon which controls the expression of phycocyanin. Phycocyanin is a protein complex which serves as accessory pigment to chlorophyll in cyanobacteria. Without phycocyanin, light harvesting process is halted. The amount of phycocyanin increases from very low level to high level in the presence of light.

Fig. 2.1 below shows the structure of phycocyanin operon in the cyanobacterium *Anacystis nidulans*.

P	O	*CPCB1*	*CPCA1*	Intergenic region	P	O	*CPCB2*	*CPCA2*

Legend:

P	:	**promoter**
O	:	**operator**
CPCB1* and *CPCA1	:	**structural genes coding for β – subunit of phycocyanin**
CPCB2* and *CPCA2	:	**structural genes coding for α – subunit of phycocyanin**

Fig. 2.1

(a) Compare the lac operon to the phycocyanin operon. [3]

Features	lac operon	phycocyanin operon
Similarity:		
Type of operon	**Inducible** operon	**Inducible** operon
Differences:		
Number of promoter	**1 promoter** controlling gene expression	**2 promoters** controlling gene expression
Number and type of structural genes	**3 structural genes; *lacZ*, *lacY*, and *lacA***	**2 structural genes** for **β-subunit** of phycocyanin and **2 structural genes** for **α-subunit** of phycocyanin.
Arrangement and type of structural genes	OR 3 **structural genes lacZ, lacY and lacA adjacent to each other**.	OR **structural genes** of **β-subunit** and **α-subunit** are **separated** by **intergenic region**
Inducer	**Allolactose**	**Light**

2 max for differences

Cyanobacterium *Anacystis nidulans* has thylakoid which contain the same photosystems as the thylakoid of plant cells. Fig. 2.2 shows a hybrid phycocyanin operon. The *trpR* gene is upstream of the hybrid phycocyanin operon.

trp PO	*CPCB1*	*CPCA1*	Intergenic region	trp PO	*CPCB2*	*CPCA2*

Legend:

trp PO	:	**trp promoter + operator**

Fig. 2.2

(b) Explain the rate of production of oxygen in the cyanobacterium *Anacystis nidulans* carrying the hybrid operon when light is present and tryptophan is present. [3]

1. The rate of production of **oxygen** is **very low** or **negligible**;
2. the hybrid operon is under the control of **trpPO** (R: if only mention either one) which **phycocyanin genes are not expressed when tryptophan is present.** (A: description of effect of tryptophan on repressor and subsequent blocking of RNA polymerase)
3. **photoactivation halts**, therefore, **photolysis cannot occur** to produce oxygen.

Fig. 2.3 shows the outer layers of cyanobacteria and bacteria **Y**.

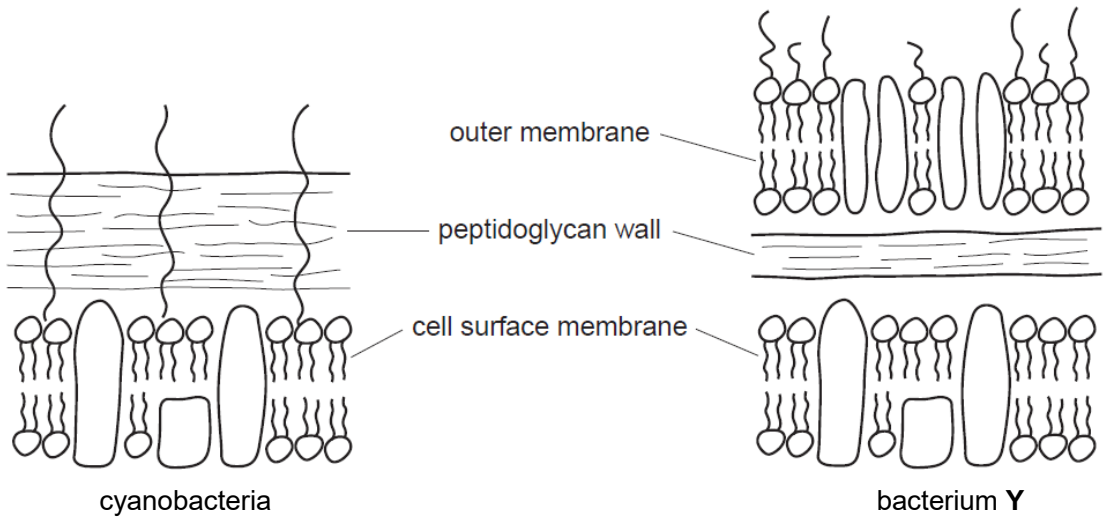

Fig. 2.3

(c) Describe how the outer layers of bacterium **Y** differ from those of cyanobacteria. [2]

1. Y has a **thinner peptidoglycan wall** compared to X;
2. X has the **peptidoglycan wall exposed** on the surface while Y has an **outer membrane exposed** on the surface. AW describing location of peptidoglycan wall;
3. X has **one phospholipid bilayer** (A: only has cell surface membrane) while Y has **two phospholipid bilayers** (A: cell surface membrane and outer membrane).
4. Y has **channel proteins** / an **outer membrane** which are / is absent in X;

Many bacteria that have a similar outer layer structure to that of bacterium **Y** are known pathogens that can cause diseases. To treat such diseases, doctors sometimes prescribe antibiotics. Gramicidin A is an example of an antibiotic.

Gramicidin A folds into a 3-dimensional configuration that inserts itself into the bacterium's cell surface membrane. It allows non-specific movement of ions which eventually cause the bacterial cell to die. Fig. 2.4 shows the interaction of Gramicidin A with the bacterium's cell surface membrane.

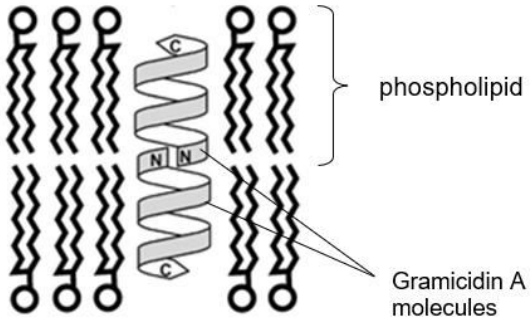

Fig. 2.4

(d) Using the information provided and Fig. 2.4, explain how Gramicidin A kills the bacterium. [2]

1. Gramicidin A forms a **hydrophilic channel**, which allows non-specific movement of ions in and out of the bacteria via **facilitated diffusion.**
2. **Disrupting the ion concentrations** in the bacterial cell which can **disrupt metabolic functions** leading to cell death;

(R: osmotic lysis as cell wall are not weakened)

[Total: 10]

3 Yeasts are unicellular organisms from the kingdom Fungi. *Saccharomyces cerevisiae* is one species of yeast that can carry out either asexual reproduction by mitosis or sexual reproduction by meiosis.

Budding in *S. cerevisiae* is a process where a small daughter cell forms as a bud on the parent cell. The bud contains a copy of the parent cell nucleus and it eventually separates from the parent cell to form a new cell.

S. cerevisiae can exist in two forms: haploid cells or diploid cells.

- Haploid cells can be one of two different mating types: **a** and α
- Haploid cells can only mate with other haploid cells of the opposite mating type.

Fig. 3.1 shows the life cycle of *S. cerevisiae* with its asexual and sexual reproductive stages.

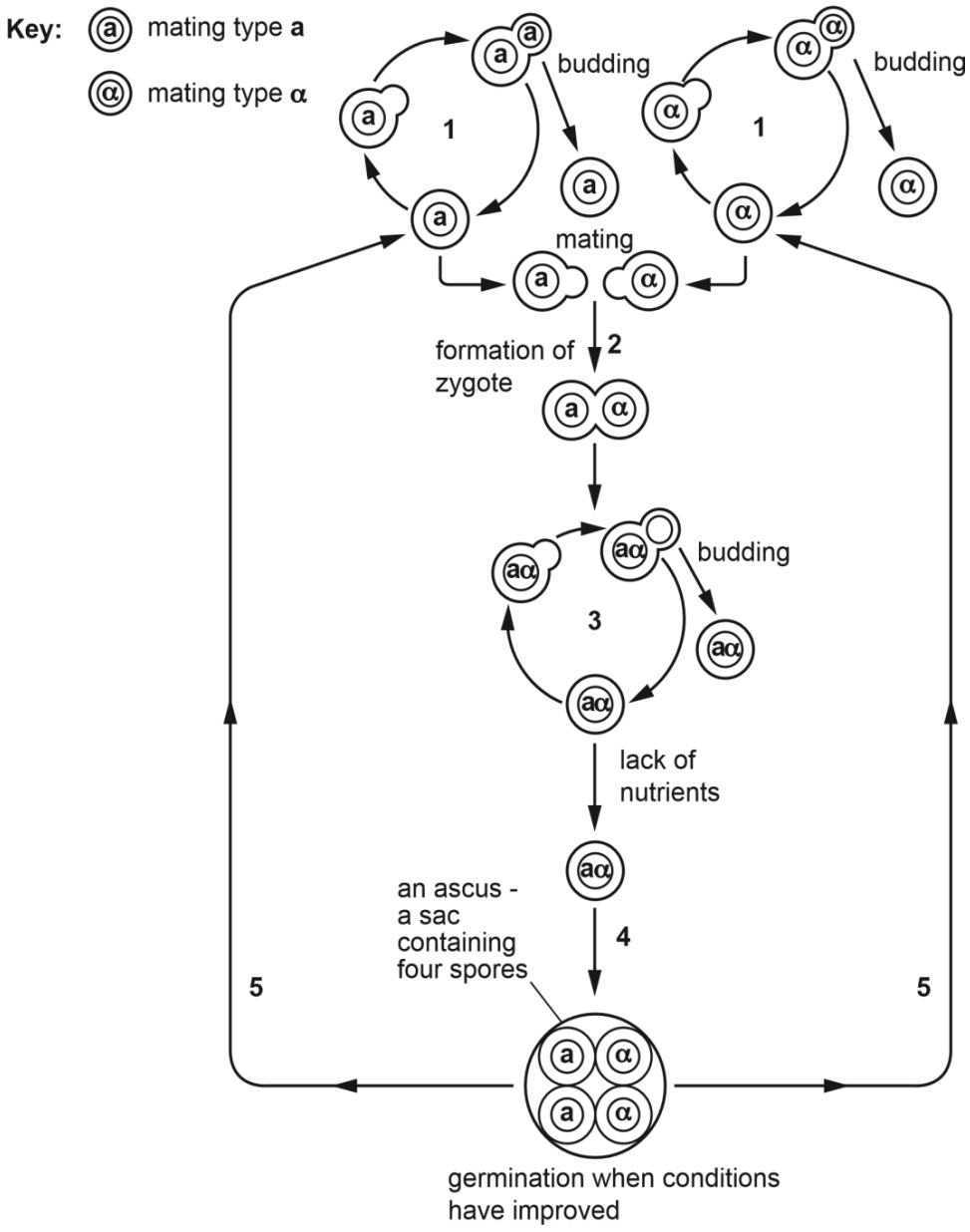

Fig. 3.1

(a) With reference to Fig. 3.1, state **one** number of the stages **1**–**5** that:

involve mitosis .....

involve meiosis .....

produces new genetic variation .....

shows only haploid cells .....

shows only diploid cells .....

involve mitosis: **1 / 3**
involve meiosis: **4**
produces new genetic variation: **2 / 4**
shows only haploid cells: **1 / 5** (I: 2)
shows only diploid cells: **3** (I: 2)
All correct: 3 marks; 3 – 4 correct: 2 marks; 1 – 2 correct: 1 mark; 0 correct: 0 mark

(b) When there is a lack of nutrients, cells made in stage **3** will carry out stage **4** to make spores, which germinate only when conditions improve.

Suggest and explain how stage **4** is advantageous for *S. cerevisiae* in a changing environment. [4]

1. Result in **genetic variation**;

Any three from points 2 to 7
2. due to **crossing over** and **independent assortment**;
3. some will, be **adapted** (to changing environment) / **survive** / **avoids whole population being wiped out**;
4. allows for **asexual reproduction** to occur; (A: stage 1 / 3)
5. allows **random mating** / **random fertilisation** / **random fusion of gametes**;
6. some have **advantageous combinations of alleles**;
7. AVP **(**e.g. *ref. to* dormancy)

(c) Haploid and diploid cells of *S. cerevisiae* can carry out **asexual** reproduction.

Suggest why a new harmful recessive mutation may **not** have a damaging effect on:
- an asexually reproducing population of haploid cells of *S. cerevisiae*
- an asexually reproducing population of diploid cells of *S. cerevisiae*. [3]

haploid
1. **only cells with harmful mutation will be affected / die**
2. **rest of population unaffected** (OWTTE)

diploid
3. **recessive allele for harmful mutation**, will be **masked** by dominant (normal) allele / not expressed in heterozygote
4. AVP (e.g. **haploid removes mutated allele from population**)

[Any three]
[Total: 10]

Section B

Answer **one** question in this section.

Write your answers on the 12-page Answer Booklet 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 parts **(a)** and **(b)**, as indicated in the question.

4 **(a)** Describe how variation arises due to mutations, and how recessive alleles are

preserved in a population. [13]

1. **Gene mutation** involves a **change in nucleotide sequence;**
2. any one example: **substitution, deletion or insertion of a nucleotide**;
3. in **coding region** that changes triplet code, hence **codon** of mRNA and then amino acid hence **3D conformation and charge of polypeptide / protein;**
4. in **non-coding regions** such as **e.g. promoter/enhancer/silencer** that can **increase/decrease rate of transcription;**
5. and hence changes **phenotype** of organism;

6. **chromosomal mutations/aberrations** which involve a **change in number and structure** of chromosomes resulting in a change of phenotype of organism;
7. change in number of chromosomes results in **non-disjunction** resulting in **polyploidy/aneuploidy;**
8. change in structure such as:
   deletion - when a segment of a chromosome is **removed**
   OR **duplication** - when an **extra segment** of a chromosome is present
   OR **inversion** - when a chromosome segment is **detached, flipped around 180 degrees and reattached** to the **same** chromosome
   OR **translocation** - when a segment from one chromosome is **detached and reattached to a different chromosome**;

9. **Heterozygote protection/diploidy** occurs in diploid organism with **2 different alleles at 1 gene locus**
10. where **dominant allele** determines **organism's phenotype/ recessive allele** remains **hidden/masked;**
11. **dominant phenotype selected for and heterozygotes survive and reproduce**
12. and pass on **recessive allele to offspring, maintaining recessive allele in population;**
13. e.g. heterozygous condition hides recessive Hb^S allele that is less favourable from natural selection which only acts on sickle cell anaemia phenotypes
    or any relevant example with details (e.g. cystic fibrosis);

14. **balancing selection** where natural selection **maintains two or more alleles at a gene locus** (such as in heterozygote advantage and frequency dependent selection);

15. **heterozygote advantage** when individuals who are **heterozygous** at a particular gene locus have **greater fitness/ selective advantage** over **both homozygotes;**
16. **Heterozygote is selected for with named e.g.** in malaria prone regions, Hb^AHb^S do not suffer from negative effects/do not die of sickle cell anemia or more resistant to malaria;
17. thus **heterozygotes pass on recessive allele (Hb^S) to offspring** when heterozygotes propagate/interbreed, **maintaining recessive allele in population**;
18. **Both homozygotes are selected against with named e.g. Hb^SHb^S** individuals will be disadvantaged due to **serious effect of sickle-cell anaemia** and **Hb^A Hb^A will be susceptible to malaria;**

19. **frequency dependent selection** is where **fitness/selective advantage of phenotype depends on how common it is;**
20. **the frequency of each phenotype oscillates over time but is kept close to 50%, thus maintaining both alleles;**
21. e.g. in Lake Tanganyika in Africa, there are two forms of scale-eating fish i.e. left-mouthed and right-mouthed. Prey of scale-eating fish guards itself against attack from whatever phenotype of scale-eating fish is most common in the lake. **So from year to year, selection favours whichever mouth phenotype is least common;**

QWC (1m): covers at least 1 example of how variation arises and 1 example of how recessive alleles are preserved

(b) Describe how anatomical and molecular homology support Darwin's theory of descent with modification.
[12]

1. homology refers to **similar characteristics found in different species due to inheritance from a common ancestor**;

2. organisms with **anatomical homology** have **anatomical structures** such as bones, organs and gross structural features that they **share with a common ancestor** and thus supports Darwin's theory of descent with modification;
3. named e.g. **pentadactyl limb** structure in forelimbs;
4. of all **tetrapods/humans, cats, whales (any 2 examples);**
5. Forelimbs have **same arrangement of bones but have different functions** and superficially look different;
6. e.g. **legs for walking in cats, flippers for swimming in whales (any two e.g.);**
7. 5 digit pentadactyl limb structure in common ancestor was **altered by natural selection/different selection pressures** in different organisms to **adapt to specialised functions/environments**, **resulting in variations** of the pentadactyl limb structure;

8. anatomically homologous structures that are **greatly reduced in size / have little to no function** as they were **selected against/ confer a selective disadvantage;**
9. are called **vestigial structures;**
10. organisms with vestigial structures **share common ancestry with organisms in which the structure is still functional** and thus supports Darwin's theory of descent with modification.
11. named e.g. **Hind limbs (femur) / hips (pelvic bones) in whales** are reduced to small bones (i.e. vestigial structures) as they are no longer beneficial to whales which swim. **However, their presence in whales suggest common ancestry with tetrapods;**
    OR **Appendix in humans** is also a vestigial structure as it is reduced from cecum of its primate ancestors which was involved in digestion of plant material. Thus, presence of appendix in humans, **suggests common ancestry with primates;**

12. Organisms with **molecular homology** have **similar DNA, RNA & amino acid* sequences** as they **share a common ancestor** that had these molecules;
13. **named e.g. cytochrome C/ p53 / haemoglobin** are homologous genes;
14. Homologous genes share **significant sequence homology** and when expressed produce **proteins that have same function** in all organisms that possess them and thus supports Darwin's theory of descent with modification;
15. **Nucleotide sequences** in ancestral genes were **modified due to accumulation of mutations** that occurred over **many generations that were selected for;**
16. The **greater the sequence similarity** between homologous genes, the **more closely related** the 2 species are;

QWC (1m): discuss 1 scenario of anatomical homology and 1 scenario of molecular homology

5 (a) Explain how genetic stability in eukaryotes is maintained at the molecular and cellular levels. [13]

Molecular level (max 8m)

M1. Genetic stability at molecular level means **maintaining same DNA sequence in daughter cells and parental cells**;
M2. In order to have identical copies of DNA as parent cell before nuclear division, **DNA** is first **replicated** during **S phase** of interphase via **semi-conservative replication**;
M3. **Each parental strand** of **double stranded DNA** act as **template** to **synthesise daughter strand**;
M4. following **complementary base pairing** where complementary nitrogenous bases **form hydrogen bonds** with each other;
M5. **adenine with thymine**, **guanine** with **cytosine**;
M6. Each **DNA molecule** formed consists of **one parental strand** and **one daughter strand/newly synthesized strand**;
M7. **Double stranded** so that **one of the strands** can be a **backup** to be used as a **template for repair**;
M8. **DNA polymerase I proof-reads** newly-synthesised DNA. If there is an **wrong deoxyribonucleotide added**, it will remove and **replace** with **correct nucleotide**. This is to ensure fidelity of DNA sequence;
M9. **Numerous hydrogen bonds** of DNA increases **stability** of the molecule;
M10. **Telomeres** at the ends of chromosomes **prevent the loss of genetic information** due to shortening of DNA after **each round of DNA replication**;

Cellular level (max 8m)

C1. Genetic stability at cellular level means **maintaining same number and type of chromosomes in daughter and parental cells**;
C2. During **prophase**, identical **DNA molecules condense to form genetically identical sister chromatids** to **allow for separation**;
C3. During **metaphase**, **chromosomes arrange/align** singly **along metaphase plate / at cell equator**; (R: middle of cell)
C4. During **anaphase**, genetically identical **sister chromatids** are **separated** and **daughter chromosomes** are pulled apart to opposite **poles** of the cell; (R: opposite ends)
C5. Mitosis thus **ensures equal distribution of identical sister chromatids to daughter cells**;
C6. **Cell cycle** is **regulated** at **checkpoints**. to **ensure the previous stage has occurred correctly before moving onto the next stage**;
C7. The main checkpoints are at **G_1, G_2 and M phase**;
C8. When there is **DNA damage**, the ***p53* gene** is **activated** to produce **p53 proteins** to **upregulate the expression of some genes**;
C9. **Cell cycle is halted** so that there is enough time for cell to **repair its damaged DNA;**
C10. When DNA damage is **beyond repair**, **apoptosis** is initiated to **stop the production of mutant daughter cells**;
C11. **Length of telomeres limit the number of cell divisions** (Cells with telomeres at critical length do not go through any further cell divisions) to prevent loss of genetic information

AVP: any mention of genetically identical.

Max 12 points

QWC (1m): attempt to define genetic stability + reference to both molecular and cellular levels

(b) Describe how genetic variation increases over many generations in prokaryotes. [12]

"Genetic variation increases" via:
[Conjugation]

1. **Conjugation** where **F plasmid is transferred** from **donor cell** to **recipient cell**
2. **Sex pilus** of **F+ cell makes contact with a F^- cell** and **retracts** to bring the F- cell closer;
3. **forming** a temporary **mating bridge** between the two cells;
4. **One** of the 2 strands of the **F plasmid** in F+ cell is **nicked** and **transferred from the F+ cell to the F- cell** through **mating bridge** as the other DNA strand is used as a template for elongation via rolling circle mechanism
5. The single-stranded F plasmid DNA **circularises** in **F- cell** and is **used as a template** to **synthesise** a **complementary strand** for a **double-stranded** F plasmid DNA resulting in **F+ cell**
   (Mark for F+ cell and F- cell only once in point 2)

[Transformation]

6. **Transformation** where a **competent bacterial cell take up fragments of naked foreign DNA** (A: foreign DNA?)
7. The foreign DNA is **incorporated into the bacterial chromosome / DNA** via **homologous recombination**
8. If the foreign DNA contains **a different allele that is now expressed** in the bacterial cell, the bacterial cell has **transformed**;

[Transduction]

9. **Transduction** where **bacteriophages/phages transfer bacterial genes** from a **donor host cell** to a **recipient host cell**;

10. A **lytic phage infects** host **bacterium** carrying a new/different allele and **hydrolyses/degrades the bacteria chromosome**;
11. Fragment of bacteria chromosome containing the **new/different allele** may be **randomly / accidentally packaged into a capsid head** during the **assembly** of new phages

12. A **temperate phage infects** a host **bacterium** carrying a new/different allele, **injecting** its **viral DNA genome** into the **bacterium**, and the **viral DNA is integrated into bacterial chromosome** forming a **prophage**
13. During induction event, a **fragment containing the new/different allele** may be **improperly excised along with the prophage** and is packaged into a **capsid head** during the **assembly** of new phages;

14. Upon **bacterial cell lysis**, the defective phage will **infect another bacterium** and inject the fragment from the previous host cell into the new bacterium;
15. The new/different alelle is **incorporated into the bacterial chromosome / DNA** via **homologous recombination**

[Mutation]

16. **Mutation(s)** that result in formation of **new allele(s)**;
17. Due to **environmental factors e.g. ionizing radiation, carcinogenic chemicals**;
18. or **DNA replication errors as DNA polymerase lacks proof-reading mechanism**;

"over generations" via:

19. **Bacteria pass on genetic variation to the next generation via binary fission**;
20. Bacterial chromosome undergoes **DNA replication** at origin of replication (*ori*) to **produce two daughter DNA molecules / daughter chromosomes**;
21. Two daughter DNA molecules / daughter chromosomes **move to opposite ends of the bacterial cell**, and **each daughter cell inherits a daughter DNA molecules**;

Any 11 points

QWC (1m): At least three correct processes (among conjugation, transformation, transduction and mutation) that generate genetic variation + mention of binary fission as process to pass on genetic variation over generations;