H

NATIONAL JUNIOR COLLEGE, SINGAPORE
Senior High 2
Preliminary Examination
Higher 2

CANDIDATE NAME			
BIOLOGY CLASS	2bi2_____	REGISTRATION NUMBER	

Biology

9744/03

Paper 3 Long Structured and Free-response Questions

13 September 2024

2 hours

Candidates answer on the Question Paper.

Additional Materials: Answer Booklet

READ THESE INSTRUCTIONS FIRST

Write your name, Biology class and registration number on all the work you hand in.
Write in dark blue or black pen.
You may use an HB for any diagrams or graphs.
Do not use staples, paper clips, glue or correction fluid.

Section A
Answer **all** questions in the spaces provided on the Question Paper.

Section B
Answer any **one** question in the separate Answer Booklet.

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

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

For Examiner's Use	
Section A	
1	**/30**
2	**/10**
3	**/10**
Section B	
4 or 5	**/25**
Total	**/75**

This document consists of **15** printed pages and **1** blank page.

Section A

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

1 Collagen is a key structural protein found in various tissues throughout the body. Its measurements can vary significantly depending on the type and function of the collagen. Type I collagen is the most abundant form.

Fig. 1.1 shows the structure of type 1 collagen fibrils.

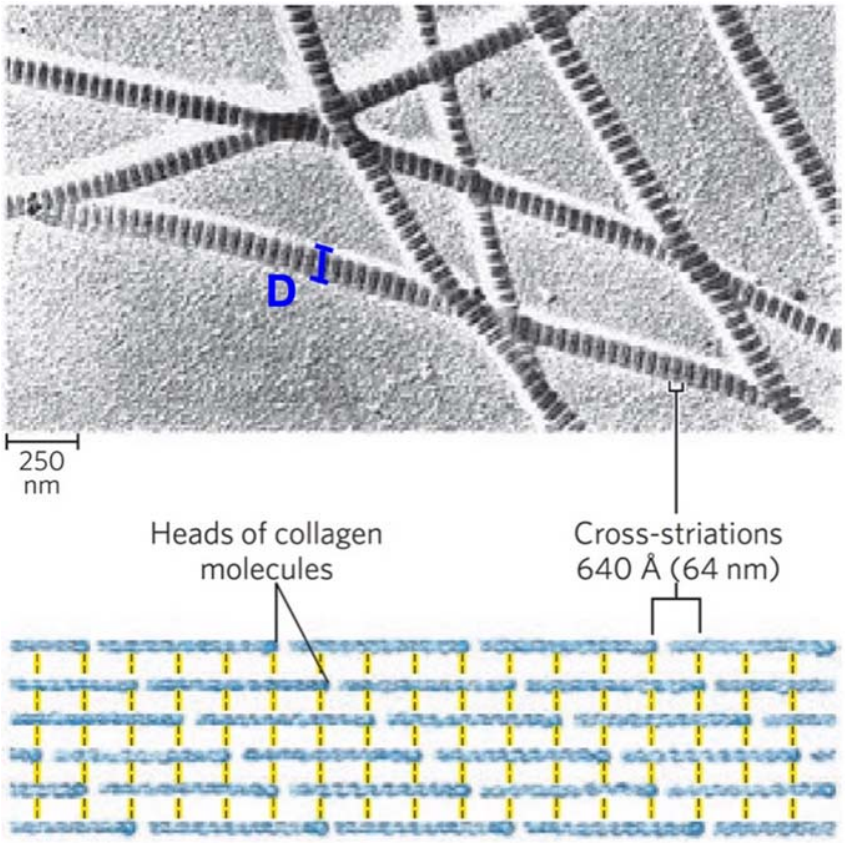

Fig. 1.1

(a) (i) Describe the molecular structure of collagen and explain how its structure relates to its function.

.....

LO 1(o) [5]

Answer:

Describe

D1. Ref to repeated **Gly–X–Y** motif + X is often **proline**, Y is often **hydroxyproline**;
D2. Ref to **1000** amino acids forming **left-handed helix**;
D3. Ref to **three polypeptide chains** supercoiling to form **tropocollagen**;

Explain

.....

E1. Ref to glycine allowing tight-packing of helix + increasing strength of tropocollagen;
E2. Ref to proline/ hydroxyproline's bulky structure + contributing to rigidity (limit rotation of peptide chain) and hence stability of tropocollagen;
E3. Ref to extensive hydrogen bonding between –NH and –C=O of peptide bonds in adjacent polypeptide chains/ adjacent tropocollagen molecules + increasing tensile strength of tendons/ cartilages;
E4. Ref to covalent cross-linking between lysine at the ends of tropocollagen molecules + increasing tensile strength of/ distributing pressure across tendons/ cartilages;
E5. Ref to staggered arrangement + minimising points of weaknesses;
E6. Ref to microfibrils/fibrils forming fibres + increasing tensile strength increasing tensile strength of tendons/ cartilages;
E7. Ref to hierarchical organisation of tropocollagen into fibrils into fibres + allowing body to form biomechanical structures of different size and nature;
E8. Ref to insolubility + providing permanent scaffold for cell adhesion, etc

(ii) Use Fig. 1.1 to calculate the number of rows of collagen molecules found in the diameter of collagen fibril, **D**.

Assume that:

- the diameter of a collagen molecule is 1.5nm
- the length of hydrogen bond between two rows of collagen molecules is 3.0 Å (angstroms).

Show your working clearly. Give your answer to the nearest whole number.

number of rows of collagen molecules in one collagen fibril = ...

Working involved: [3]

1 – shows clearly how D is determined;

250nm scale bar 0.8–0.9cm

D 0.3-0.4cm, therefore D = (0.3–0.4) / (0.8–0.9) x 250 = 83.3–125nm

2 – shows logical method of determining number of rows of collagen molecules;

Length of hydrogen bond = 3.0/10 = 0.3nm

M1: Number of rows of collagen molecules = D/(1.5 + 0.3)

M2: Number of rows of collagen molecules = (D – 1.5) /(1.5 + 0.3) +1

3 – gives final answer in whole number;

Number of rows = 46–69

(b) Osteogenesis imperfecta (OI) is a heritable disorder of connective tissues caused by abnormal synthesis or dysfunctional type I collagen. Each type 1 collagen molecule contains two COL1A1 polypeptides and one COL1A2 polypeptide.

A study was carried out to examine the mutations in *COL1A1* and *COL1A2* genes in patients with OI. PCR was carried out to amplify the genes in segments, and the resulting PCR product was used for DNA sequencing to identify the nucleotide sequence.

Fig. 1.2 shows a segment of *COL1A1* gene, with the 5' end starting at position 1.

```
  1 tttgcccagg ctggagtgca atggtgtgat ctcggttcac tgcaaccccc gcctcctggg
 61 ttcaagtgat tctcctgcct cagcctccca agtagctggt actacaggcc catgccgcca
121 tgccgggcta atttttgtat ttttagtaga gatggagttt caccatgttg gctaggctgg
181 ggtctcaaac tctcgacctc aggtgatccg actgcctcag cctcccaaaa tgttgggatt
```

Fig. 1.2

(i) Describe the principle and procedure of PCR.

.....

LO 2(k) [4]

Answer:

Principle:

1. ref to using temperature change to regulate separation of DNA strands, binding of primers, elongation;
2. ref to repeated 20–30 cycles producing 2–fold increase in number of products per cycle;

Procedure:

3. ref to Taq polymerase + synthetic oligonucleotides/ primers + dNTPS;
4. ref to denaturation + 92 – 95 °C + 15 seconds;
5. ref to annealing + 50 – 60 °C + 30 seconds;
6. ref to extension + 70 – 72 °C + 90 seconds;
7. ref to breaking of hydrogen bond during denaturation + formation of hydrogen bond between complementary base pairs during primer annealing;
8. ref to Taq polymerase catalysing formation of phosphodiester bond between 3'OH end of primer/ elongating DNA and dNTP;

.....

(ii) Use Fig. 1.2 to propose the sequence of the pair of primers used in PCR.

Forward primer: 5' 3'

Reverse primer: 5' 3' [2]

LO 2(k)

Answer: accept any correct primer between 15–30 nucleotides long

Forward primer: 5' TTT GCC CAG GCT GGA 3' (shortest) – 5' TTT GCC CAG GCT GGA GTG CAA TGG TGT GAT 3' (longest)

Reverse primer: 5' AAT CCC AAC ATT TTG 3' (shortest) – 5' AAT CCC AAC ATT TTG GGA GGC TGA GGC AGT 3' (longest)

.....

Fig. 1.3 shows the gene maps of mutations in *COL1A1* and *COL1A2* genes. The numbered box represents the numbered exon while each line between the boxes represents the intron between two exons. The types of mutations are represented by symbols “del”, “dup” and “>”.

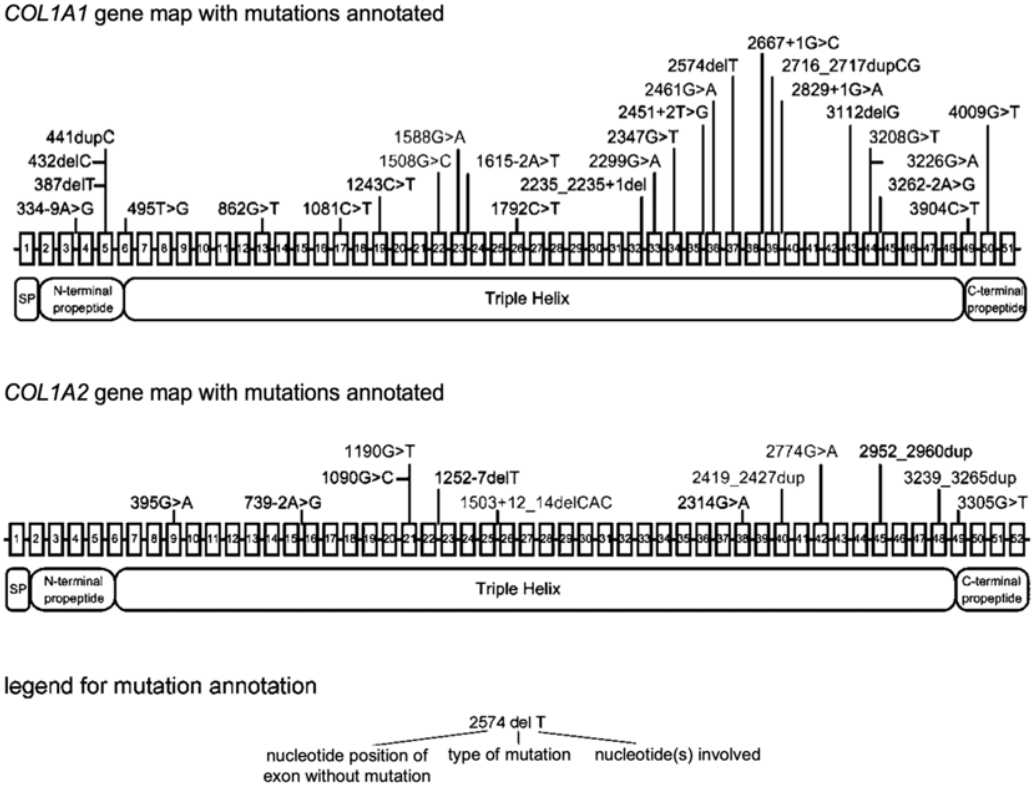

Fig. 1.3

(iii) With reference to Fig. 1.3, explain the effect of the mutations in exons and introns on type I collagen protein.

Answer: [5]

1. ref to relevant example with “>” + **nucleotide substitution**;
2. ref to relevant example with “del” + **nucleotide deletion** / “dup” + **nucleotide insertion**;
3. ref to “>” exon mutations + **silent** mutation / **missense** mutation;
4. ref to impact on bonding within triple helix/ between triple helices;
5. ref to impact on post-translation modification, such as hydroxylation of proline and lysine residues in ER;
6. ref to impact on post-translation modification, such as cleavage of terminal peptide/ aggregation into collage fibrils;
7. ref to “dup”/ ‘del” exon mutations + **frameshift** / **nonsense** mutation;
8. ref to impact on synthesis of functional collagen;
9. ref to “>” exon mutations + silent mutation effect;
10. ref to any intron mutations (*COL1A1: 334-9A>G, 1615-2A>T, 2451+2T>G, 2667+1G>C, 2829+1G>A, 3262-2A>G. COL1A2: 739-2A>G,* 1252-7delT) + affect splice sites;

11. ref to additional amino acids due to translation of intronic sequence;
12. ref to intron mutation 1503+12_14delCAC + three nucleotide deletion + no impact on protein;
13. ref to no impact because introns are not translated;

Source: Ohata (2019)

Osteogenesis imperfecta (OI) can also arise from *de novo* mutations (DNM), which refer to sequence alterations not found in parents.

Fig. 1.4 shows the pedigree of a family affected with OI. Patient 716 was diagnosed with OI at the age of 3 days. Her parents, individuals 710 and 711, are healthy without history of chronic or clinically significant diseases and are free of mutations known to cause OI. Her younger brother, individual 715, is normal and does not carry any mutation known to cause OI.

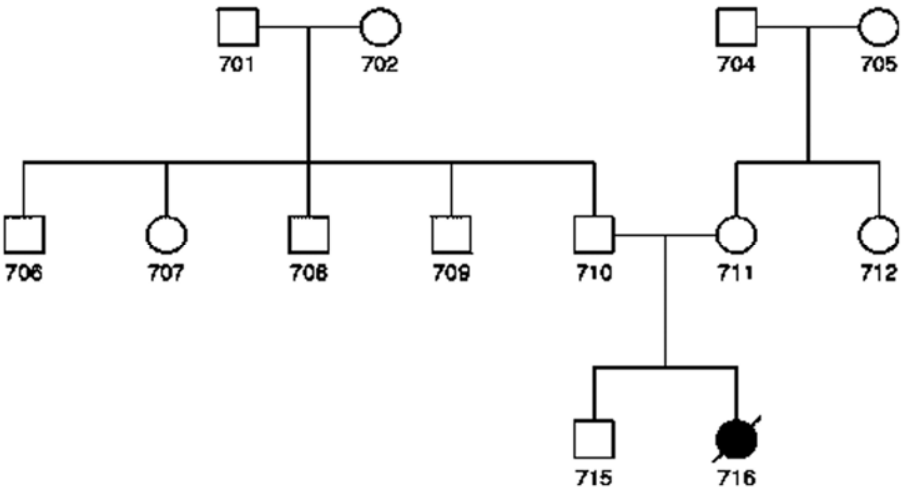

Fig. 1.4

(iv) Describe how genetic variation is produced in sperms under normal conditions.

LO 2(s, t) [3]

Answer:

1. ref to crossing over at prophase I + exchange of genetic material between non-sister chromatids;
2. resulting in different combination of alleles on the chromosomes;
3. ref to independent assortment of homologous chromosomes at metaphase/anaphase I;
4. ref to independent assortment of non-identical sister chromatids at metaphase/anaphase II;
5. resulting in different combination of paternal and maternal chromosomes/ homologues in daughter cells;

Fig. 1.5 shows how DNMs may be affected by gender and age.

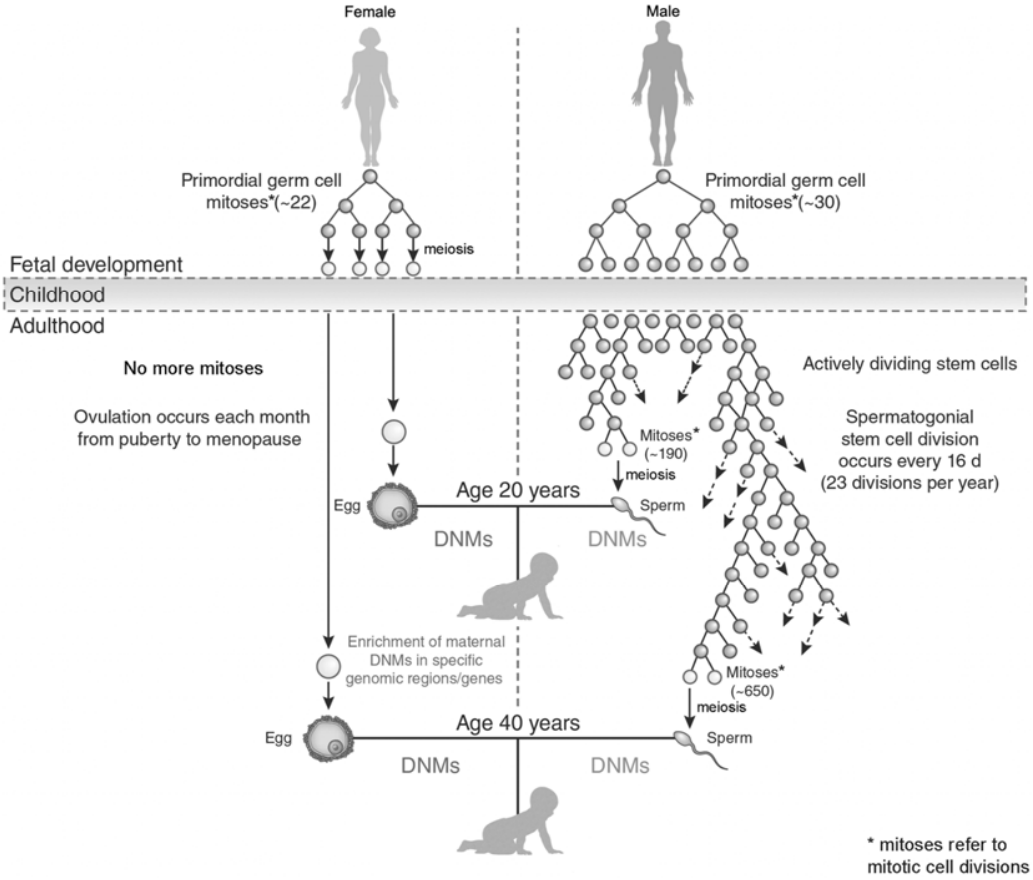

Fig. 1.5

(v) With reference to Fig. 1.4 and Fig. 1.5, explain how the OI condition is present in individual 716 but absent in individual 715 and their parents.

Answer: [3]

1. ref to spontaneous errors in replication by DNA polymerase in **actively dividing stem cells**;
2. ref to rapid **spermatogonial stem cell division** every 16 day + inefficient DNA repair mechanisms/ increased chance of accumulating mutations;
3. ref to mutation occurring in **primordial germ cells** in mother or father/ **actively diving stem cells** in father;
4. ref to individual 715 developing from sperm and egg cells from a separate lineage from individual 716;
5. ref to lack of OI–causing mutations (no DNM, no inherited mutation) in the sperm and egg cells that formed genetic makeup of parents of individual(s) 715 &/ 716;

A. AVP: eg: mutation occurring after sperm and egg fused to form individual 716;

(c) Type I collagen is the most abundant protein in the human body. It is degraded slowly and its replacement synthesis is low. However, during wound healing, the cells can increase the production of type I collagen by several hundred-fold.

Describe how the expression of type 1 collagen may be upregulated during wound healing.

LO 2(j) [5]

Answer:

	Elaboration
1. ref to transcriptional level: activators binding to enhancers + increase transcription; (ora with repressors)	E1. Activators bind to mediator, triggering assembly of RNA polymerase and general transcription factors;
2. ref to post-transcriptional level: recognition of alternative polyadenylation signal/ activation of polyA polymerase + synthesis of longer polyA tail;	E2. Longer polyA tail delay of degradation of the coding sequence by 3'exonucleases, so more collagen can be synthesised per mRNA;
3. ref to translational level: 3'UTR binding to proteins that decrease rate of polyA shortening/ 3'UTR binding to proteins that block recognition by endonucleases ;	E3. longer polyA tail increases half-life of mRNA, allowing it to be used a template to synthesise collagen for a longer time;
4. ref to translational level: blocking of small interfering RNA (siRNA) / microRNA (miRNA) that cause mRNA to be cleaved and degraded;	E4.
5. ref to translational level: inactivation of translational repressors + increased formation of translational initiation complex;	E5.
6. ref to translational level: activation of translation initiation factors + increased formation of translational initiation complex;	E6. Translation initiation factors may be activated through phosphorylation/ dephosphorylation;

7. ref to chromatin level: histone acetylation/ DNA demethylation + allowing general transcription factors, RNA polymerase to access promoter;	E7. Histone acetyltransferase catalyses addition of acetyl to lysine side chain, disrupting electrostatic attraction between histone protein and DNA backbone;

[Total: 30]

2 Sahiwal cattle and Holstein Friesian cattle are known for their high milk yield.

Milk yield is affected by heat stress due to higher temperatures which results in protein misfolding within cells.

Cattle have several *hsp* genes that code for heat shock proteins (HSPs). The expression of HSPs increases in response to heat stress to help in refolding of proteins to their normal conformations.

Fig. 2.1 shows the relative expression of HSPs in the Sahiwal cattle and Holstein Friesian cattle during summer (S) and winter (W) seasons.

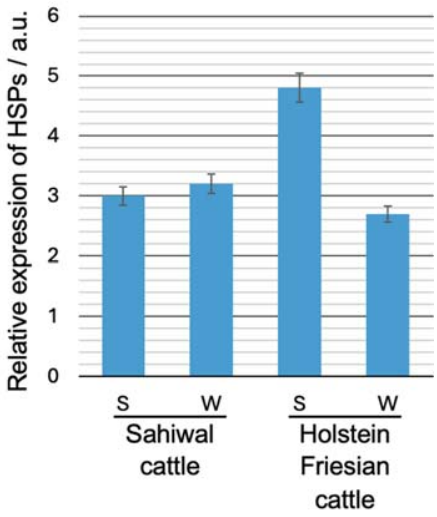

Fig. 2.1

(a) With reference to Fig. 2.1, describe the differences in the relative expression of HSPs in Sahiwal cattle and Holstein Friesian cattle.

1. In **summer**, Holstein Friesian cattle express **1.8a.u. relatively more** HSPs than Sahiwal cattle ; OR
   Holstein Friesian cattle express **higher HSPs at 4.8a.u.** while Sahiwal cattle express **lower HSPs at 3.0a.u.** (accept 3a.u. since y-axis is whole number);

2. In **winter**, Holstein Friesian cattle express 0.5a.u. **relatively less** HSPs than Sahiwal cattle ; OR
   Holstein Friesian cattle express **lower HSPs at 2.7a.u.** while Sahiwal cattle express **higher HSPs at 3.2a.u.**

3. From **summer to winter**, relative expression levels of HSPs in Sahiwal cattle **increased slightly by 0.2a.u.** / remained **relatively similar**, while that of Holstein Friesian cattle **decreased greatly by 2.1a.u.**;

Reject: comparison of total expression levels (S+W) [3]

Holstein Friesian cattle are less heat tolerant than Sahiwal cattle and hence their milk yield are more affected by changing temperatures.

Fig. 2.2 shows the mean daily air temperature and the mean monthly milk yield per cow for Holstein Friesian cattle in Central Europe.

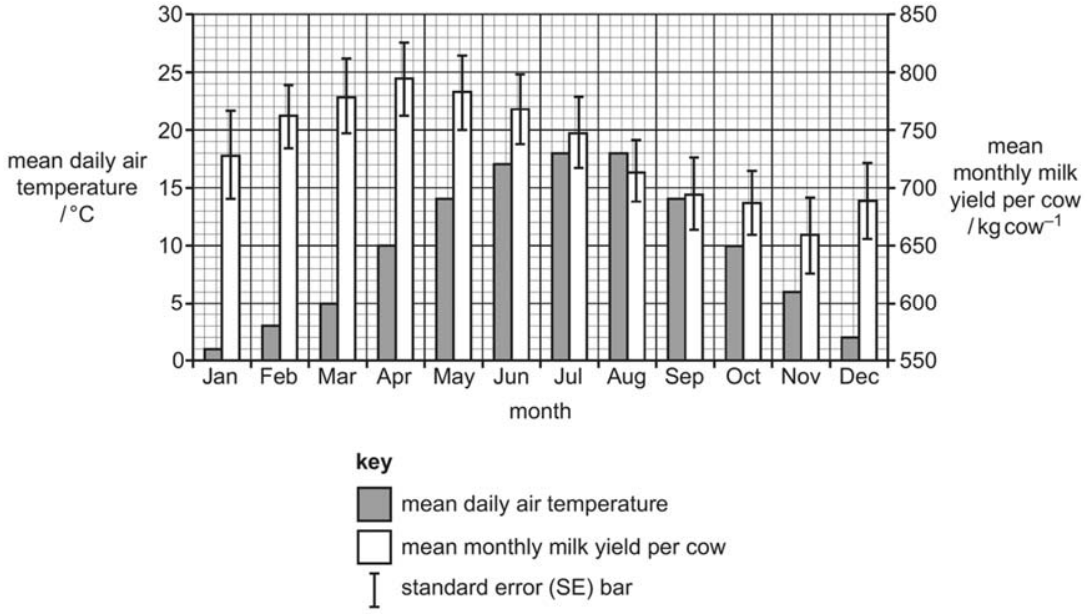

Fig. 2.2

(b) With reference to Fig. 2.2, describe the trends in air temperature and milk yield during summer from April to August.

Air temperature increases + quote values (10°C to 18°C);

milk yield decreases + quote values (795±3 kg cow^{-1} to 715±3 kg cow^{-1})

[2]

(c) Suggest reasons for the decrease in milk yield by Holstein Friesian cattle during summer.

Heat stress caused **more proteins to be misfolded** due to **increased kinetic energy / thermal agitation, disrupting weak bonds such as hydrogen bonds, ionic bonds, and hydrophobic interactions (at least 1 bond);**

Misfolding enzymes involved in milk synthesis / milk proteins results in decrease milk yield;

Reallocation of energy/resources from milk production to maintenance functions, such as production of HSPs to refold proteins, activating heat dissipation mechanisms, etc.;

Fig. 2.1 shows higher expression of HSPs in response to heat stress to refold proteins, however as many proteins are misfolded, refolding of proteins essential for survival are prioritised over non-essential proteins like enzymes involved in milk production / milk proteins OR higher expression of HSPs is insufficient to refold proteins like enzymes involved in milk production / milk proteins OR rate of milk proteins refolding by HSPs is lower than milk protein misfolding;

To cope with increased heat, Holstein Friesians **reduce their feed intake**, leading to a **decrease in the availability of essential nutrients required for milk synthesis**;

AVP;

[2]

(d) Holstein Friesian cattle are the most common dairy cattle breed worldwide. However, there are rising concerns about the decreasing genetic diversity of the Holstein Friesian cattle populations due to intense directional selection practised in the last century.

Explain why low genetic diversity may decrease the long-term survival of Holstein Friesian cattle.

Less variation for natural selection to act on;

Less beneficial alleles / fewer alleles that can confer selective advantage;

Holstein Friesian cattle population is less able to adapt/evolve;

All/most could be killed by same disease / selection pressure;

[3]

[Total: 10]

3 HIV-1 is the most common type of HIV. HIV-1 binds to CCR5 receptor on helper T lymphocytes.

Current treatment for HIV-1 involves the use of daily antiretroviral therapy (ART) to stop viral replication. Only 59% of HIV-positive individuals have access to ART.

Scientists found that two HIV-1-positive patients **P** and **Q** have no detectable HIV-1 after blood stem cell transplant (BSCT).

- Patient **P** was given two rounds of BSCTs, while patient **Q** was given one round of BSCT.
- All BSCTs came from a donor with helper T lymphocytes without the CCR5 receptor.
- In addition to BSCT, patient **P** had radiotherapy, while patient **Q** had chemotherapy. Both treatments are toxic.
- Both patients **P** and **Q** stopped receiving ART 16 months after BSCT.

18 months after stopping ART, both patients had no HIV-1 RNA in their plasma, no HIV-1 DNA in their helper T lymphocytes and no CCR5 on their helper T lymphocytes.

(a) Using the information provided, discuss the effectiveness of the use of BSCT to treat HIV-1 infections.

Effective (max 3m)

1. No virus/ HIV(-1)/RNA/DNA 18 months after stopping ART, so it could be a cure;

2. No CCR5/receptor, so not get HIV(-1) in the future OR No CCR5/receptor, so nothing for HIV(-1) to bind to;

3. Only one transplant/BSCT needed (shown by patient Q);

4. Would not need (daily) ART (16 months after BSCT);

Not effective (max 3m)

5. Don't know if chemotherapy/radiotherapy is needed OR Do not know if BSCT alone would be effective OR Do not know which treatment is having the effect OR Could be due to chemotherapy/radiotherapy;

6. Don't know if it would work in all people OR Only worked/tried in 2 cases;

7. Might not be long term OR Only 18 months;

8. HIV-1 may mutate and be able to bind to a different receptor (on helper T cells);

9. Might be a lack of (suitable stem cell/BSCT) donors;

[5]

Currently, scientists are developing mRNA vaccines to prevent HIV infections.

To develop the vaccines, mRNAs coding for specific HIV proteins are introduced into the cells. The mRNAs used for vaccines must be stable so that they are not degraded before the proteins are produced.

Scientists modified the 5' cap of mRNAs to make them more stable than those with normal GTP cap.

To test the effect of the modified caps, they introduced the same amount of each mRNA to different groups of cells. The mRNA half-life and the total amount of protein translated from the mRNAs.

Table 3.1 shows the results.

Table 3.1

5' cap structure	mRNA half-life / hours after introduction into cells	total amount of protein translated from mRNA relative to amount with normal cap
no cap	1.41	0.011
normal cap	16.10	1.000
modified cap I	15.50	4.777
modified cap II	27.00	13.094
modified cap III	18.09	6.570

(b) **(i)** Identify the 5' cap structure that is most effective in stabilising the mRNA.

Modified cap II [1]

(ii) After examining the results in Table 3.1, a student hypothesised that mRNA with modified cap I was translated more frequently than mRNA with the normal GTP cap.

Evaluate the validity of the student's hypothesis.

The hypothesis is valid / the data support the hypothesis;

because the half-lives of the two mRNAs are similar (modified cap I is slightly lower), but the amount of protein produced from the mRNA with modified cap I is more than (4.777 times more) that produced from the mRNA with the normal cap;

[2]

(iii) Scientists can introduce either mRNA or DNA into cells to produce foreign proteins.

Explain why the introduction of mRNA is more likely to produce foreign proteins than the introduction of DNA.

mRNA (with appropriate modifications) can be **directly translated** to produce proteins, but DNA needs to be **transcribed** into mRNA (processed into mature mRNA) then **translated** into proteins;

Transcription of DNA may not occur as DNA may not be transported into nucleus;

Transcription of DNA requires transcription factors which may not be available / may not bind to the DNA introduced / repressors might bind and prevent transcription;

DNA needs to be integrated/inserted into the genome in the region where genes are frequently expressed for transcription to occur;

mRNA transcribed from the DNA depends on proper post-transcriptional modifications such as 5' capping, splicing and polyadenylation in the cell which are not needed for the mature mRNA that is introduced;

Ref to more level of gene regulation for DNA compared to mRNA, increasing the chances of repressing gene expression;

AVP;

[2]

[Total: 10]

Section B

Answer **all** parts of the question in this section.

Write your answers on the separate 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 section (a), (b) etc., as indicated in the question.

4 (a) Describe the various roles of RNA in eukaryotes. [13]

messenger RNA (mRNA)

M1. role in transferring genetic information from nucleus to cytoplasm / serves to carry the information or "message" that is encoded in genes to the sites of protein synthesis in the cell;

M2. DNA triplet codes are carried in the form of codons in mRNA;

M3. Each codon corresponds to one amino acid / used as template to direct synthesis of a polypeptide chain;

ribosomal RNA (rRNA)

R1. role in forming ribosome for translation / provides the structural and functional foundation for ribosomes;

R2. aligns tRNA and mRNA in ribosome;

R3. makes up peptidyl transferase that catalyses the formation of peptide bond between adjacent amino acids;

transfer RNA (tRNA)

T1. 3' end binds to corresponding amino acid via covalent bond / attached to amino acid by aminoacyl-tRNA synthetase / possess a 3' terminal nucleotide sequence that reads –CCA, and the amino acid is covalently linked to the 3'–OH of the terminal A residues;

T2. role in carrying the corresponding amino acid to ribosome to match with the codon in translation / carry amino acids to ribosomes for use in protein synthesis;

T3. contains anti-codon that is complementary to codon on mRNA for translation;

small nuclear RNA (snRNA)

S1. role in forming small nuclear ribonucleoprotein particles (snRNPs);

S2. mediates the splicing of eukaryotic gene transcripts to form mature mRNA;

small nucleolar RNA (snoRNA)

N1. role in alternative splicing;

N2. binding to a splicing enhancer / silencer leads to activation / skipping of individual splice sites;

microRNA (miRNA)

C1. role in the control of gene expression;

C2. silences gene expression by repressing translation / accelerating target mRNA degradation;

C3. causes histone modification and DNA methylation of promoter sites, which affects the expression of target genes;

RNA primer

P1. role in allowing DNA replication by DNA polymerase;

P2. Provides 3'OH group for the addition of complementary deoxyribonucleotide to the growing DNA strand;

RNA template in telomerase

L1. role in lengthening telomere / the synthesis of telomeres at the ends of linear chromosomes;

L2. used as template in telomerase to direct synthesis of telomeric DNA repeats;

QWC: at least 3 different roles of RNA;

(b) Explain the advantages of regulating gene expression at different levels in eukaryotes and suggest why prokaryotes have less complex gene regulation. [12]

Advantages (maximum 9 marks)

1. **Chromatin level**
   a. longer term switching genes on and off to restrict expression of genes;
   b. allows for specialisation / differentiation of cells;
   c. more efficient / less wasteful of resources as only genes required for cellular functions are expressed;

2. **Transcriptional level**
   a. Rate of transcription can be regulated to meet shorter term requirement of the cell;
   b. Combinatorial control allows flexibility in regulation of transcription in response to changes in signals or stimuli spatially and temporally when the appropriate combination of specific transcription factors are present;
   c. Coordinate control allows simultaneous transcription of genes with related functions / involved in the same metabolic pathway in the presence of the activators;

3. **Post-transcriptional level**

a. Alternative splicing allows for the production of different proteins from a single gene;
b. Degradation of unprocessed or incompletely processed mRNAs prevents wastage of resources (e.g. ribonucleotides) in translation of these mRNAs;

4. Translational level

a. Half-life of mRNA will affect how long translation of the mRNA can occur and hence the amount of protein produced, preventing continuous translation of mRNA and production of proteins that may not be needed;

5. Post-translational level

a. allows for rapid production of functional protein from stored precursor by phosphorylation / cleavage for immediate responsiveness to cell conditions / signals OR able to convert between active and inactive form quickly in response to signals;
b. allows for activation of protein where it is needed, ensuring safe transport / storage of inactive form of protein;
c. allows recycling of amino acids from proteins that are no longer required for cellular functions;

Why less complex gene regulation in prokaryotes

S1. Prokaryotes are unicellular or colonial / do not organise into tissues, organs and systems, hence they do not specialise in any particular function, and there is no need for long-term switching on and off of genes controlled at the chromatin level;

S2. Prokaryotes do not have histone proteins bound to DNA, hence gene regulation at the chromatin level via histone modification (e.g. acetylation / deacetylation) is not possible;

S3. Operon systems in prokaryotes enable coordinate control of related genes by clustering and regulating them under a single promoter, unlike in eukaryotes where related genes are scattered and usually on different chromosomes, hence their transcriptional level of control is less complex;

S4. Prokaryotes lack introns, hence unable to have (alternative) splicing;

S5. Prokaryotes lack nuclear envelope / nucleus, hence transcription and translation occur simultaneously and they cannot have post-transcriptional control and translational control is limited;

S6. Prokaryotes lack membrane-bound organelles required for many post-translational modifications, hence they have limited post-translational control;

QWC: at least 1 advantage linked to the correct level + at least 1 suggestion;

[Total: 25]

5 (a) Describe the various roles of ATP in eukaryotes. [13]

General

G1. ref to ATP as the universal energy currency / carrier in cells of living organisms (as it is small and water soluble, hence can be transported within the cell easily / a lot of chemical energy is stored in the bonds of ATP / hydrolysis of the third phosphate group releases large amount of energy);

Respiration

R1. Activation of glucose to glucose-6-phosphate via phosphorylation using ATP during the energy investment phase of glycolysis. This process is catalysed by hexokinase;

R2. ATP is required for the phosphorylation of fructose-6-phosphate to fructose-1,6-bisphosphate catalysed by phosphofructokinase;

R3. ATP also serves as an allosteric inhibitor to the phosphofructokinase enzyme;

Photosynthesis

P1. Energy from hydrolysis of ATP during Calvin cycle of the light-independent stage of photosynthesis in stroma of chloroplasts in plant cell is required to convert glycerate-3-phosphate (GP) to glyceraldehyde-3-phosphate (G3P / GALP / triose phosphate);

P2. Energy from hydrolysis of ATP is also needed to regenerate ribulose bisphosphate (RuBP) in Calvin cycle;

P3. allows the continuation of Calvin cycle to produce glucose in photosynthesis;

Cell Signalling

S1. ATP is required as a substrate for the adenylyl cyclase enzyme, which upon activation by the activated GTP-bound G-protein, catalyses the conversion of ATP to cAMP, which serves as a second messenger in the signal transduction pathway;

S2. ATP is also the substrate for the kinases in the phosphorylation cascade, which catalyses the addition of phosphate groups from the ATP molecules to the subsequent kinases in the cascade;

S3. ATP is required for the activation of receptor tyrosine kinase (RTK) through autophosphorylation;

DNA Replication

D1. ATP is required for the synthesis of nucleotides;

D2. ATP is required as an energy source for DNA replication in unwinding and unzipping of DNA helix to separate the parental strands by DNA helicase / ref to energy required to break hydrogen bonds between two DNA strands so that they can each act as templates for replication;

Gene Expression

E1. ATP is required for transcription initiation;

E2. ATP is a ribonucleotide, which is a substrate of RNA polymerase during transcription / RNA synthesis;

E3. ATP required for amino acid activation prior to translation for the covalent attachment of the amino acid to the 3' acceptor stem of the corresponding tRNA, catalysed by aminoacyl-tRNA synthetase;

E4. ATP (used to form GTP) is required to complete the assembly of translation initiation complex;

Transport

T1. Active transport of molecules against concentration gradient across the cell surface membrane via the action of a specific carrier protein called "pump", which uses ATP to change its conformation. E.g. sodium-potassium pump;

T2. Endocytosis requires ATP for the uptake of macromolecules, particulate substances, and, in specialised cases, even other cells. E.g. ingestion of pathogens via phagocytosis by phagocytes;

T3. Exocytosis / Movement of secretory vesicles from the Golgi body to the cell surface membrane along the cytoskeleton requires energy from the hydrolysis of ATP. E.g. secretion of insulin by beta cells / secretion of antibodies by plasma cells;

AVP (max 2)

A1. ATP is required for macromolecule synthesis;

A2. ATP is required for organelle synthesis;

A3. ATP is required for assembly of microtubules / arrangement of chromosomes during cell division;

A4. ATP is required for reestablishment of ion concentration gradient in neurones;

A5. ATP is required for sperm movement;

A6. ATP is required for muscle contraction to allow for movement of the animals;

Reject: ATP is converted to cAMP, which then binds to CAP to increase the rate of transcription in bacteria

QWC: at least 3 different categories of roles (excluding general and AVP) of ATP;

(b) Explain the advantages of having cyclic processes in eukaryotes and suggest why some processes need to be non-cyclic.

[12]

Advantages (maximum 9 marks)

1. **Allow molecules to be regenerated / reused / minimising need to re-synthesise molecules; max 4**
   a. ref to switching between active and inactive states of proteins, proteins can be reactivated easily without a need to re-synthesise the proteins e.g. cell signalling / phosphorylation cascade, cyclin-dependent kinase (cdk);
   b. ref to ATP-ADP cycling where ATP is constantly generated from ADP during respiration, and then broken down to ADP when energy is required, without a need to synthesise new adenosine nucleosides;
   c. ref to cycling between NADH (reduced form) and NAD^+ (oxidised form) in respiration (e.g. fermentation);
   d. ref to cycling between NADPH (reduced form) and $NADP^+$ (oxidised form) in photosynthesis (e.g., photophosphorylation and Calvin cycle);
   e. ref to regeneration of oxaloacetate in Krebs cycle;
   f. ref to regeneration of RuBP in Calvin cycle;
   g. ref to recycling transport proteins e.g. GLUT for regulation of blood glucose;
   h. ref to reusing enzymes to catalyse a reaction;

2. **Macromolecules can be synthesised and then broken down to allow the recycling of monomers;**

a. ref to proteins synthesised by translation and unwanted proteins are degraded by proteasomes / lysosomal enzymes so that amino acids can be recycled for the synthesis of other proteins;
b. ref to mRNA synthesised by transcription and degraded by exonucleases which give ribonucleotides that can be recycled for the synthesis of other RNAs;
c. ref to depolymerisation of microtubules which give tubulins that can be recycled for polymerisation of microtubules;

Reject carbohydrates / fats (Breakdown of glycogen releases glucose, which is used for respiration, hence the glucose is not recycled. Similar for fats.)

3. Cyclic Photophosphorylation / Electron Flow (Photosynthesis)

a. ref to cyclic photophosphorylation / electron flow repeating to produce more ATP but no NADPH so as to meet the higher ATP requirement by the Calvin cycle more efficiently;

4. Mitotic cell cycle

a. The mitotic cell cycle ensures that DNA is copied correctly by semi-conservative replication during the S phase and then evenly distributed during mitosis to produce genetically identical daughter cells after cytokinesis;
b. ref to mitotic cell cycle producing new cells for growth / repair;
c. ref to cyclical changes of cyclin;

5. **Homeostasis**: E.g. blood glucose level maintained by cyclical secretion of insulin / glucagon (antagonistic hormones)

Why some processes need to be non-cyclic

S1. ref to one named example of a non-cyclic process (e.g. glycolysis, non-cyclic photophosphorylation, cell signalling pathway, gene expression) whose end-product is required for other cellular process; **max 4**

S2. ref to one named example of a non-cyclic process (e.g. exocytosis) whose end-product is in another location that does not allow it to be used in the same process again;

S3. ref to the significance of end-product inhibition that contributes to cellular efficiency by preventing the wasteful overproduction of substances;

S4. ref to the need to remove the unwanted / toxic substance and not allowing it to exist perpetually in the cell;

S5. ref to the products of non-cyclic processes only required at certain times;

QWC: at least 1 example for advantage + at least 1 example for suggestion;

[Total: 25]