Answer **all** questions.

1 Fig. 1.1 is an electronmicrograph showing parts of two liver cells.

(a) Name the structures labelled A, B and C in Fig. 1.1.

A Mitochondrion

B Nucleus

C Cell surface membrane or plasma membrane

[3]

(b) Membranes are used within cells to create separate compartments.

Explain the advantages to cells of having separate compartments. [3]

1. Within a cell, compartmentalization allows the formation of unique environments optimal for specialized activities;
2. such as enzyme reactions where enzyme and substrate can accumulate at high concentration to increase the rate of enzyme-catalyzed reaction;
3. Enzymes and substrate involved in related metabolic reactions can be localized in the same compartment to increase the rate of enzyme-catalyzed reaction;
4. e.g Enzymes and substrates involved in Krebs cycle in mitochondria matrix or Calvins cycle in stroma of chloroplast;
5. Maintenance of optimal pH for enzyme action;
6. e.g. acidic pH for lysosomal enzymes in lysosomes;
7. For the establishment of proton gradients within specialized organelles such as mitochondria and chloroplasts for chemiosmosis and formation of ATP;
8. Allows storage of molecules within a separate environment to prevent degradation by other enzymes;
9. e.g. storage of starch in amyloplast;

Any 3 of the above

Table 1.1 shows the percentage of the total area of all membranes of a liver cell contributed by each type of membrane.

Table 1.1

type of membrane	percentage of total cell membrane area
cell surface membrane	2.0
outer mitochondrial membrane	7.0
inner mitochondrial membrane	32.0
membrane of rough endoplasmic reticulum	34.0
membranes of smooth endoplasmic reticulum and Golgi body combined	24.0
membranes of nucleus (nuclear envelope)	0.4
membrane of lysosomes	0.6

Table 1.2 shows the percentage of the total volume of a liver cell contributed by each type of membrane-bound compartment.

Table 1.2

type of membrane-bound compartment	percentage of total cell volume
cytosol	57
mitochondria	20
rough endoplasmic reticulum	10
smooth endoplasmic reticulum and Golgi body combined	6
nucleus	6
lysosomes	1

(c) Explain the difference between the percentage of total cell membrane area contributed by the inner mitochondrial membrane and the percentage of total cell membrane area contributed by the outer mitochondrial membrane, as shown in Table 1.1. [3]

1. Inner mitochondria membrane take up 25% more total cell membrane area than the outer mitochondria membrane;
2. Inner mitochondria membrane/Cristae is highly folded to increase surface area;
3. Allowing more ***electron carriers**** of electron transport chain and ***ATP synthase**** to be embedded in it;
4. To increase the rate of oxidative phosphorylation/***ATP**** production;
5. Outer mitochondria membrane is not folded, hence smaller total cell membrane area;

(d) The ratio of the percentage of total cell membrane area to percentage of total cell volume for the nucleus is approximately 0.07 : 1.

(Note to student: 0.4:6 = 4:60 = 0.07:1) (see * on tables 1.1 and 1.2)

(i) Use Table 1.1 and Table 1.2 to calculate the ratio of the percentage of total cell membrane area to percentage of total cell volume for the smooth endoplasmic reticulum and Golgi body combined.

ratio = ... [1]

24 : 6, then reduce to lowest term = 4:1 (see * * on tables 1.1 and 1.2)

(ii) State how the structure of the smooth endoplasmic reticulum and Golgi body accounts for the ratio calculated in **(d)(i).** [2]

1. Smooth ER consist of tubular network of membranes, cisternae and the Golgi body consist of a stacks of flattened sac of membranes which contributes to their high surface area;
2. Both have small cisternal space that contain small volume resulting in a higher surface area to volume ratio;

[Total: 11]

2 Fig. 2.1 shows the main structural features of the human immunodeficiency virus (HIV).

(a) Name the structures labelled D, E and F in Fig. 2.1.[3]

D viral envelope

E capsid protein

F viral RNA genome

(b) Describe how an enveloped virus such as HIV enters a host cell.[4]

Attachment

1. The viral ***glycoprotein****, ***gp120**** recognizes and binds to the ***CD4 receptor**** on the membrane of the host cell e.g. helper T cells or macrophages;
2. that it is ***complementary**** in shape/conformation and charge to;

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3. ***gp41**** binds to co receptor;
4. and facilitiates ***fusion**** of the viral envelope with plasma membrane of the host cell to release the capsid into the cell leaving the envelope behind;

(c) Anti-retroviral therapy (ART) uses multiple anti-retroviral drugs to control HIV in individuals who are infected with HIV (HIV positive individuals).

Fig. 2.2 shows the number of copies of HIV RNA per cm^3 plasma and the number of copies of HIV DNA per 106 T lymphocytes in an HIV positive individual over a period of 20 years. The start of anti-retroviral therapy (ART) is shown with an arrow.

(i) Describe the effect of anti-retroviral therapy (ART) on the number of copies of HIV RNA per cm^3 plasma in an HIV positive individual, as shown in Fig. 2.2.[2]

1. When ART started, there was a steep decrease in number of copies of HIV RNA per cm^3 plasma from 100,000 copies per cm^3 to 0.6 copies per cm^3 within 1 year from year 2 to year 3; (compulsory point)
2. Thereafter remained low between 0.6 copies per cm^3 to and 300 copies per cm^3 from year 2 to year 20;
   A: Thereafter there was a gradual increase with some fluctuation up to 300 copies per cm^3 until year 14, then a gradual decrease to about 12 copies per cm^3 at year 20;

(ii) Suggest why HIV RNA is measured in the plasma and HIV DNA is measured in the T lymphocytes.[2]

1. HIV has RNA genome and the amount of RNA is a measure of HIV viruses in the plasma;
2. The amount of DNA in T lymphocytes is a measure of HIV-infected cells as T lymphocytes are the host cell of the virus and upon successful infection, HIV RNA genome is reverse transcribed to form DNA which is integrated into host cell genome as a ***provirus***** during the latent phase;

[Total: 11]

3 Proteins are one constituent of cell membranes. Fig. 3.1 shows the molecular structure of three other constituent biomolecules of cell membranes, **J**, **K** and **L**.

(a) Name biomolecules **J**, **K** and **L**.

J Phospholipid;

K Glycolipid;

L Cholesterol;

(b) Describe the formation of the bonds labelled **M** in Fig. 3.1.[2]

1. An ***ester*** * linkage is formed between a ***hydroxyl* group*** (-OH) of a glycerol molecule and a ***carboxyl* group*** (-COOH) of a fatty acid chain;
2. via a ***condensation reaction**** whereby a molecule of ***water**** is removed;

(c) Explain the roles of biomolecules **K** and **L** in the cell surface membrane.[3]

1. **K** - glycolipid acts as markers for ***cell-cell recognition**** to distinguish cells as 'self'/'non-self' as the basis of the immune system;
   Or
   Cell-cell recognition* also result in cell adhesion allowing cells to be attached to one another to form tissues and organs;
2. Acts as ***receptor**** for certain pathogens and toxins to bind to host cells and gain entry into cell;

3. **L** - Cholesterol ***regulates membrane fluidity**** by preventing the membrane from being overly fluid at warmer temperatures as cholesterol's rigidity restricts phospholipids' lateral movement;
4. The membrane is prevented from being overly firm at lower temperatures as cholesterol prevents the close packing of phospholipids and hence prevents its solidification/ crystallization;

[Total: 8]

4 Sickle cell anaemia is a genetic disease caused by a recessive mutant allele of the β-globin gene.

Scientists investigated the inheritance of the disease in one family. Fig. 4.1 shows the inheritance of sickle cell anaemia in this family.

(a) Describe how PCR is carried out to amplify only the region of DNA that includes the β-globin gene and **not** other regions of the DNA. [2]

1. Design and use ***primers**** with nucleotide sequences ***complementary**** to regions flanking the ***β-globin**** gene;
2. Ensure that the nucleotide sequence on the ***primer**** is not ***complementary**** to other regions of the DNA;
3. After denaturation of the DNA double helix, cool the PCR mixture to ***64℃****, in the presence of excess primer.

(b) State and explain which probe, **X**, **Y** and **Z**, was used to produce the results shown in Fig. 4.2. [3]

1. Probe Z;
2. The band pattern in Fig. 4.2 reflects only the ***376bp**** and ***201bp**** fragments but not the ***175bp**** fragment which can be produced upon restriction digest.
3. This is because the probe is ***complementary in sequence/complementary base pairs**** only to regions that are found on the ***201bp**** fragment.

*Point 1 is a compulsory point.

	(c)	Using the information provided in Fig. 4.3, describe, as precisely as possible, the mutation that causes sickle cell anaemia. [2]
		1. Sickle-cell anaemia is an autosomal recessive genetic disease which is caused by a ***substitution**** mutation in DNA coding for ***β-globin**** chain of haemoglobin; 2. The mutation is located within a ***DdeI restriction site**** in the disease-causing allele (HbS), such that second restriction site is now no longer recognised by the ***DdeI*** enzyme/absent in the mutant allele.
	(d)	Explain why family members **P**, **Q**, **T** and **U** each have two bands showing on Fig. 4.2.[2]
		1. Individuals P, Q, T and U are carriers for sickle cell disease and are heterozygotes/ carry both the normal and disease allele; 2. Restriction digest of a normal allele will give 2 fragments with only the ***201bp**** fragment hybridizing to the probe and appearing as the upper band in the band pattern; 3. Restriction digest of a disease allele gives a ***376bp**** fragment which appears as the lower band seen in the band pattern.
		[Total: 9]
5	**(a)**	Describe the packing of DNA in eukaryote chromosomes.[3]
		1. DNA is highly coiled/packed in eukaryotic chromosomes; 2. The ***negatively-charged DNA**** double helix is wound around ***positively-charged**** ***histone**** octamers twice to form ***nucleosomes**** with ***linker DNA**** joining adjacent nucleosomes; forming a 10nm fibre/chromatin; 3. The 10nm fibre coils around itself to form 30nm chromatin fibre/solenoid; 4. The 30nm fibre forms ***looped domains**** when associated with scaffold proteins, forming 300nm fibre; 5. The 300nm fibre supercoils/further coils and folds to form a characteristic metaphase ***chromosome****.
	(b)	Explain how DNA methylation and histone modification alter gene expression.[4]
		DNA methylation: 1. Addition of methyl group to selected ***cytosine**** nucleotides in on DNA (e.g. a CG sequence); 2. This leads to recruitment of ***histone deacetylase**/ **chromatin remodeling complexes**** to condense chromatin (decrease accessibility of promoter to general transcription factors and RNA polymerase); 3. Reduces the accessibility and binding of ***general transcription factors**** and ***RNA polymerase**** to ***promoter****, preventing formation of ***transcription initiation complex****. *Point 3 is a compulsory point. *Histone modification:* 1. ***Histone acetyltransferase**** adds ***acetyl groups**** to lysine residues on ***histones****; 2. This removes the positive charge from histones thus reducing electrostatic interaction between negatively-charged DNA and histone, causing DNA to wind less tightly around histones; 3. This allows ***RNA Polymerase**** and ***general transcription factors**** to bind the ***promoter**** to form the ***transcription initiation complex**** to initiate transcription; *Accept reverse argument i.e. removal of acetyl groups by histone deacetylase.

(c) During the production of human induced pluripotent stem cells (iPSCs), changes in DNA methylation and histone modification are crucial for the re-establishment of gene expression.

Discuss how using human induced pluripotent stem cells (iPSCs) in medical treatments avoids some of the ethical problems of using other pluripotent stem cells. [3]

1. As iPS cells are not from an embryo, there will be no destruction of embryos and hence not regarded as killing a life due to some views that embryos are viable living organisms;
2. As iPS cells are not from an embryo, it will not be seen as treating embryos as just a source of spare parts;
3. One ethical concern is that other pluripotent stem cells denies human status to embryos, and that this precedent may extend to other categories of human beings such as the profoundly disabled or elderly infirm. This concern could be avoided if iPS cells are used.

[Total: 10]

6 Fig. 6.1 is an electronmicrograph showing the complete set of chromosomes (karyotype) of a person.

(a) Identify, with reasons, **two** phenotypic features of this person that can be deduced from the karyotype shown in Fig. 6.1.[4]

1. the karyotype shows 3 copies of/trisomy of/an extra chromosome 21;
2. the individual has ***Down syndrome**** and should have characteristic facial features, short stature, heart defects, susceptibility to respiratory infection and mental retardation (any 1);
3. the karyotype also shows 2 homologous chromosome 23/ 2 X chromosomes;
4. the individual is a ***female**** with female reproductive structures such as ovaries;

(b) With reference to Fig. 6.2, describe how the age of a woman when she gives birth to a child affects the probability that the child will have a chromosomal aberration.[3]

1. When the mother is younger, that is, below 20 and up to the age of 29 years, the probability of chromosomal aberration is low at 0.0008;
2. When the mother is older, at 30 years and above, the probability increases steeply, 0.0014 at age 30-34, 0.0044 at age 35-39 and 0.0136 when the age is above 39 (quote any one);

   or

   probability increased by 16 times by age 40/within the next 10 years;

(c) In a human female, the process of meiosis that eventually leads to the formation of mature ova (egg cells) begins early in her development, at the fetal stage.

While she is still a fetus, meiosis is then suspended part way through. Meiosis does not resume until she reaches puberty. For any individual ovum, an entire meiotic division is only completed after fertilisation of the ovum by a sperm.

Use this information to suggest reasons for the relationship described in your answer to **(b)**.[3]

1. In females who give birth to a child/ when fertilization events occur at a more advanced age, meiotic divisions also completes at a more advanced age;
2. this allows an accumulation of mutations with time;
3. that can cause alteration to chromosome structure due to ***deletion**, **duplication*** or ***translocation**** of a segment of the chromosome (any 1 example);
4. age can also lead to a higher likelihood of ***non-disjunction**** events during meiosis;
5. leading to chromosomal aberrations such as aneuploidy where there is an extra or one less chromosome;

[Total: 10]

7 **(a)** Explain the need for the tight control of the mitotic cell cycle. [3]

1. The cell cycle is regulated for normal growth and development.
2. Cell cycle is regulated at ***checkpoints**** at G1, G2 and M, which determines if the cell cycle can proceed
3. Dysregulation of checkpoints cause the cell to escape cell cycle control mechanism
4. Leading to ***uncontrolled division**** of cells and cancer.

Additional information in double boxed section of lecture notes.

- G1 check point can proceed if DNA is not damaged
- G2 check point can proceed if DNA is not damaged/chromosome has replicated/sufficient cyclin can bind with Cdks to form MPF
- M Check point can proceed if all chromosome are attached to spindle fibres from both poles

Cyclins are proteins that control the cell cycle by activating cyclin-dependent kinase enzymes (Cdk). These enzymes allow checkpoints to be passed. Fig. 7.1 shows changes as the cell cycle progresses from the G_2 phase of interphase to mitosis (M phase).

(b) Use Fig. 7.1 to explain how mitosis is initiated.[3]

1. From 11h to 14h, the concentration of M-cyclin increases steadily;
2. And reach maximum concentration/peak at 14h
3. Resulting in increased in M-cyclin binding to and activation of Cdk, passing checkpoint;
4. When M-cyclin bind to Cdk to form MPF;
5. that phosphorylates and activates a range proteins, promoting mitosis;

	(c)	Suggest why cyclin-dependent kinase enzymes are considered potential targets for anti-cancer drugs and suggest how such drugs might work. [3]
		Drugs may be 1. ***Competitive inhibitors**** of CDK; 2. That are ***complementary in shape**** to the ***active site**** of CDK; 3. thus bind to the active site of CDK; 4. preventing it from phosphorylating/activation of proteins that promotes mitosis and uncontrolled cell division; or 5. Similar in shape and charge to M-cyclin; 6. Binds to M-cyclin binding site on CDK; 7. Prevent CDK from binding to M-cyclin to form MPF; 8. preventing it from phosphorylating/activation of proteins that promotes mitosis and uncontrolled cell division;
		[Total: 9]
8	**(a)**	Outline how light energy is converted to chemical energy during the light-dependent reactions of photosynthesis. [4]
		1. When a photon of ***light**** strikes a chlorophyll molecule, one of its electrons is excited to a higher energy state; 2. Energy is relayed from pigment to pigment, via resonance transfer of energy, until it reaches one of the two specialized ***chlorophyll a**** (P680/700) in the ***reaction center**** of ***photosystem*** II/I. 3. Excited electron emitted from chlorophyll a (P680/P700) is captured by the ***primary electron acceptor****, in the ***reaction centre****. 4. As the excited electrons flow down a chain of carriers of ETC with progressively lower energy levels 5. energy released is coupled to **pumping H^+ ions** from stroma across the thylakoid membrane into the thylakoid space establishing a ***proton-motive force****. 6. As protons / H^+ diffuse down the concentration gradient back into the stroma via the ***ATP synthase****. 7. ***ADP**** is phosphorylated to form ***ATP**** in the process via ***chemiosmosis****. 8. Electrons from PS I are passed down an electron transport chain and are finally transferred to NADP which combines together with H^+ to form ***reduced NADP**** in stroma;
	(b)	Outline the **three** phases of the Calvin cycle. [3]
		Phase 1 – Carbon fixation 1. During carbon fixation, ***carbon dioxide**** combines with ***RuBP**** Which is catalyzed by enzyme, ***RuBP carboxylase/oxygenase**** (Rubisco); Phase 2 – Carbon reduction 2. NADPH is the reducing power used to ***reduce**** glycerate phosphate (GP) to glyceraldehyde-3-phosphate (G3P); And ATP is the source of energy required for the reaction; Phase 3 – Regeneration of RuBP 3. Some G3P is used to ***regenerate**** RuBP so that the cycle of carbon dioxide fixation can continue while some G3P leaves the cycle to make sugars etc.

(c) Fig. 8.1 shows the action spectrum for photosynthesis of a particular land plant.

Explain why the rate of photosynthesis for this plant was low at wavelengths of light between 525 nm and 575 nm. [2]

1. At wavelengths of light between 525 nm and 575 nm, the frequency of light absorption by these pigments is low;
2. Hence, there will be less ***photoactivation**** of electrons at the reaction centres and less ATP and NADPH generated during the light dependent reaction;
3. As such less G3P is produced during the light independent reaction which leads to a lower rate of photosynthesis;

[Total: 9]

9 Terrestrial ecosystems are dominated by flowering plants and the herbivores that feed on them. Herbivores are very diverse and include many species of mammals and insects, such as beetles. Some beetles feed on the dung of herbivorous mammals and are known as dung beetles

(a) With reference to Fig. 9.1, suggest **and** explain how changes in the diversity of flowering plants and changes in the diversity of mammals have affected the diversification of beetles.[3]

1. As the diversity of flowering plants increased steeply from 135 million years ago (mya) to 75 mya, this created new niches for habitats e.g and food types for herbivorous mammals;
2. This led to the gradual linear increase of diversity of mammals from 100 mya to 5 mya, as there is increase in food source for herbivores, insectivores and carnivores;
3. With the increase in mammals, the types of dung and location where they are excreted increased. As dung is a food source for dung beetles, this led to the further divergence and evolution of dung beetles evolve from 100 mya;

(b) Fig. 9.2 shows the relationship between horn length and thorax width in male and female dung beetles of the species *Trypoxyus dichotomus*. Fig. 9.3 shows how horn length and thorax width are measured in this species.

Complete Table 9.1 to describe **four** differences in horn length and thorax width between male and female dung beetles of this species, as shown in Fig. 9.2. [4]

Table 9.1

feature	male	female
Range of width of thorax	Wider range of 1.325cm to 2.650cm within the species	Narrower range of 1.400cm to 1.875cm within the species
Range of length of horn	Very wide range of 0.55cm to 3.10cm within the species	Very narrow range of between 0.20cm to 0.35cm within the species
Correlation between thorax width and horn length	As the thorax width increases from 1.325cm to 2.650cm, the horn length increases significantly from 0.55cm to 3.10cm	As the thorax width increases, the horn length does not increase significantly and remains between 0.20cm to 0.35cm.
Horn length	Males have much longer (0.55 to 3.10cm) horn lengths than females.	Females have a much shorter horn length (0.20cm to 0.35cm) than males

.

(c) Describe the biological, ecological and morphological concepts of a species.[6]

1. Biological: A species is a group of organisms capable of interbreeding and producing fertile, viable offspring**;**
2. Members of the same species are reproductively isolated from other species;
3. They share a common gene pool and have the same chromosome number;
4. and usually have similar morphological, physiological and behavioural features; (note: pts 1and 2 are more important)

5. Ecological: A species is a group of organisms **sharing the same ecological niche***.
6. The differences between species are due to the differences in the ecological resources that they depend on. This means that if a species can no longer occupy a particular niche, it would be considered a new species;

7. Morphological: A species is a group of organisms sharing similar **body shape, size** and other **structural features;**
8. definition can be applied to all organisms (i.e. sexually and asexually reproducing organisms);

[Total: 13]

10 Fig. 10.1 shows how temperature affects the relative fitness of insects living in temperate regions and Fig. 10.2 shows how temperature affects the relative fitness of insects living in tropical regions.

Relative fitness is a measure of the reproductive success of a species.

(a) Using Fig. 10.1 and Fig. 10.2, compare the effect of temperature on the relative fitness of insects living in temperate regions and insects living in tropical regions.[2]

1. The relative fitness of temperate insects reaches at optimum of 1.00 at 25°C while the relative fitness of tropical insects reaches an optimum of 1.00 at 30°C;

2. The relative fitness of temperate insects increases more gradually from 0.00 to 1.00 from -10°C to 25°C respectively compared to the relative fitness of tropical insects which increase from 0.00 to 1.00 more steeply from -10°C to 25°C respectively;

3. The relative fitness of temperate insects decreases more gradually 1.00 to 0.00 from 25°C to 34°C respectively compared to the relative fitness of the tropical insects that decrease more sharply from 1.00 to 0.00 form 30°C to 34°C respectively;

4. The tropical insects survive within a narrower range of temperatures of 16°C to 34°C while the temperate insects survive within a broader range of temperatures of -10°C to 34°C;

5. Similarites: Both tropical and temperate insects have an optimum temperature when maximum relative fitness is 1.00.

(b) Fig. 10.3 shows how the relative fitness of insect species at different latitudes is predicted to change by the year 2100, as a result of global warming.

Using Fig. 10.1, Fig. 10.2 and Fig. 10.3, describe **and** explain the predicted effect of global warming on the relative fitness of insects in temperate and tropical regions.[3]

1. Increase in temperatures due to global warming in tropical regions 20°S and 20°N of equator will decrease relative fitness of tropical insects from 0.0 to -0.1;

2. as they have small ***thermal safety margins**** as their environments are already close to their physiological optimum and so they will approach their physiological optimum temperature faster and risk extinction;

3. Increase in temperatures due to global warming in temperate regions 20°S to 60 °S of the equator and 20°N to 60°N of equator will increase relative fitness of temperate insects from 0.0 to +0.12 and 0.0 to +0.09 respectively,

4. as they have larger ***thermal safety margins**** as their environments are on average cooler than their physiological optimal, and thus have broader thermal tolerance;

Note: The difference between an organism's thermal optimum and its current climate temperature is known as the **thermal safety margin**.
For insect species, **thermal safety margins increase sharply with latitude**. [Total: 5]

11	Fig. 11.1 shows the main steps in antigen presentation by an antigen-presenting cell.
	Describe the main stages in antigen presentation by an antigen-presenting cell, as shown in Fig. 11.1. [5]
	1. Antigen presenting cell (macrophages) take up bacterial cell by ***phagocytosis**** where ; 2. ***pseudopodia**** are formed and extended outwards to engulf the bacteria; 3. When the ends of the pseudopodia ***fuse****, a vesicle / vacuole containing the bacteria is pinched off and enters into the cytoplasm as a ***phagosome****; 4. ***Phagosome**** fuses with ***lysosomes****, forming phagolysosomes; 5. Hydrolytic enzymes from lysosome breaks down bacterial antigen into short peptides; 6. And form ***peptide:MHC complex****, which is transported via vesicles and presented on the cell surface membrane;