PAPER 2

QUESTION 1

(a)(i) Identify the class of carbohydrate molecule of which agarose is an example. [1]

polysaccharide

(ii) Plants contain a carbohydrate called amylose. Amylose does not contain galactose. Describe one similarity and another difference in structure between agarose and amylose. [2]

similarity:

1. monosaccharides of both agarose and amylose are held together by glycosidic bonds
2. both agarose and amylose are unbranched

difference:

1a. coiled/helical structures in amylose
1b. but linear/straight structures in agarose

2a. alternate glucose is not inverted/ same orientation in amylose
2b. but alternate galactose is inverted 180^{o} in agarose

3a. α(1,4) glycosidic bond is present in amylose
3b. different types of glycosidic bonds are present in agarose

(iii) Suggest why bacteria are unable to metabolise agarose. [2]

1. lack of bacterial enzyme
2. to hydrolyse the type of glycosidic bonds present in agarose
3. substrate not complementary to active site of enzyme

(b) (i) Describe how the structure of domain **A** of the cholera toxin is maintained. [3]

1. primary structure folds into secondary structures
2. maintained by hydrogen bonds formed between C=O and NH groups
3. further folding
4. into a unique 3D conformation
5. maintained by hydrogen bonds, ionic bonds, disulfide bonds and hydrophobic interactions between R-groups of amino acid residues

(ii) Explain how a globular protein like cholera toxin differs from a fibrous protein, such as collagen. [2]

1a. cholera toxin being a globular protein is soluble due to the presence of hydrophilic amino acids on the surface of the protein
1b. collagen being a fibrous protein is insoluble due to the presence of mainly hydrophobic amino acids in the protein

2a. cholera toxin being a globular protein is more compact/ spherical in shape
2b. collagen being a fibrous protein is elongated in shape/forms multimolecular parallel filament to strands/ collagen fibrils and collagen fibres

3a. cholera toxin being a globular protein is made up of fixed, non-repetitive specific sequence of amino acids
3b. collagen being a fibrous protein is made up of repetitive sequence of amino acids

4a. cholera toxin being a globular protein has a relatively unstable structure ref. to weak non-covalent bonds
4b. collagen being a fibrous protein has a stable structure ref. to extensive intra- and inter-molecular hydrogen bonds/ covalent crosslinks

5a. cholera toxin being a globular protein performs metabolic functions since they are soluble
5b. collagen being a fibrous protein performs structural functions since they have high tensile strength

(c) Suggest how the cholera toxin enters epithelial cells. [2]

1. receptor binding domain B of cholera toxin is complementary to receptors on intestinal epithelial cells
2. binds/attach to receptors on cell membrane
3. resulting in a conformational change in receptor
4. cholera toxin enters by receptor mediated endocytosis
5. invagination/ infolding of epithelial cell membrane
6. formation of a vesicle enclosing cholera toxin

[Total: 12 marks]

QUESTION 2

(a) (i) Name structures **X** and **Y**. [2]

X: Heterochromatin
Y: Euchromatin

(ii) Account for the difference in the structures **X** and **Y**. [2]

1. Region X is more electron dense / darkly stained than region Y
2. Thus heterochromatin is more condensed / tightly packed / coiled than euchromatin
3. Heterochromatin is transcriptionally inactive as compared to euchromatin
4. Heterochromatin is wound around deacetylated histones

Fig. 2.2 shows part of a DNA molecule.

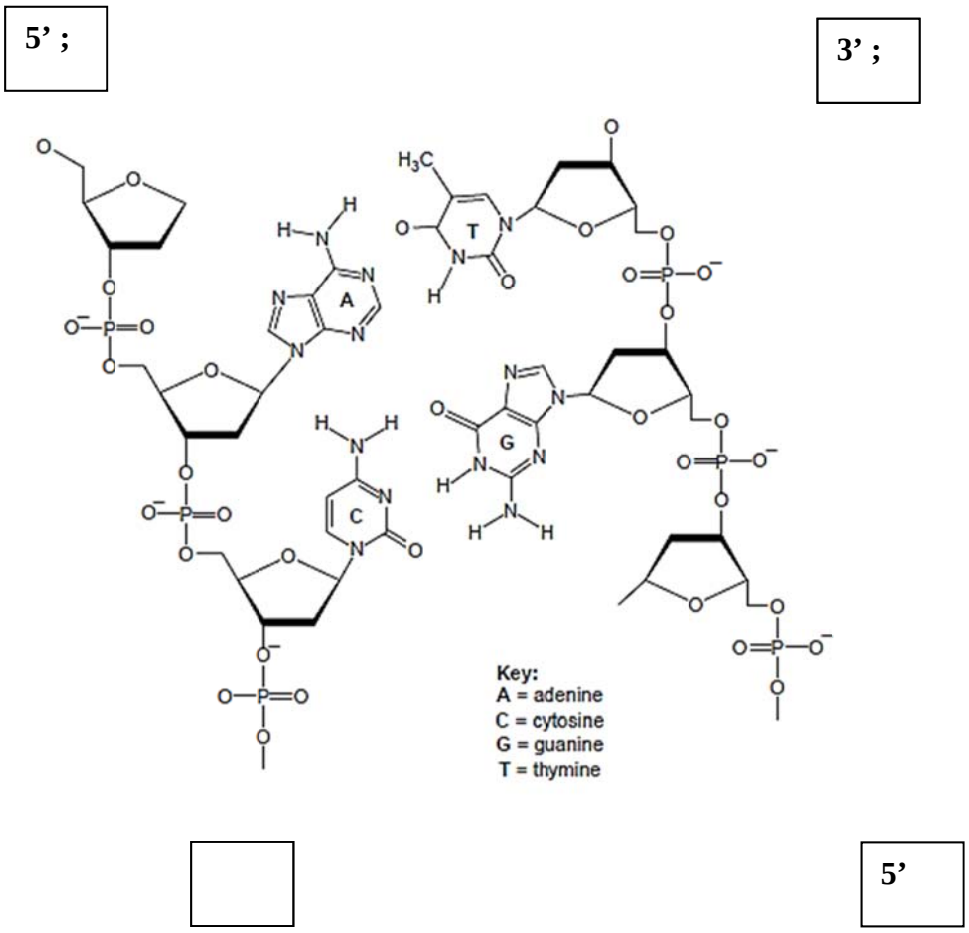


Fig. 2b

(b) (i) Complete Fig. 2.2 by indicating the polarity of the DNA molecule in the boxes provided. [1]

(ii) State the importance of hydrogen bonds in DNA structure. [2]

1. Hydrogen bonds allow for the formation of double stranded DNA / a double helix

2. Hydrogen bonds hold the two polynucleotide strands together
3. Hydrogen bonds hold the complementary nucleotides / bases together
4. Many hydrogen bonds give stability to DNA molecule
5. Hydrogen bonds can be broken for transcription / DNA replication to occur

(c) Explain how the data in Table 2.1 helps to confirm the arrangement of bases in DNA. [3]

1. In all the organisms, percentage of adenine to thymine and percentage of guanine to cytosine are approximately 1:1 ratio / equal / similar
2. For example, in yeast, percentage of adenine is 31.3% which is very similar to thymine with percentage at 32.9% and percentage of guanine is 18.7% which is very similar to cytosine with percentage at 17.1%.

Accept any one example

3 . This shows that adenine and guanine base pair with thymine and cytosine respectively

(d) (i) State how the result for the virus differs from those for all the organisms given in table 2.1. [1]

1. The percentage of adenine to thymine and guanine to cytosine are not similar / not 1:1

(ii) Give a reason for your answer to d(i). [1]

1. Virus is made up of single-stranded DNA / DNA that is not a double helix

[Total: 12 marks]

QUESTION 3

(a) Describe the role of lactose in the regulation of *lac* operon. [3]

1. Lactose / allolactose acts as an inducer that binds the lac repressor
2. Lac repressor is inactivated and dissociates from the operator
3. RNA polymerase can bind to the promoter, resulting in transcription of structural genes

(b) Identify the location of the point mutation in the operon of strain A. Give a reason for your answer. [2]

1. The mutation occurred in the promoter / operator
2. RNA polymerase cannot recognise / bind to mutant promoter and hence, transcription initiation cannot occur or mutant operator is permanently bound to lac repressor and hence RNA polymerase cannot recognise and bind promoter
3. Plasmids used in rescue experiments bear functional promoter and operator and hence, RNA polymerase can bind to this functional promoter and initiate transcription

(c) Suggest why strain B cannot be “rescued” at all. [2]

1. Mutation occurred in the *lacI* gene
2. Resulting in expression of a constitutively active / hyperactive repressor / constitutively active repressor cannot be inactivated by allolactose
3. Hence, even if the genes are inserted under the control of the functional *lac* promoter and operator, the constitutively active repressor will always bind to *lac* operator and prevent RNA polymerase from binding to the promoter / continues to inhibit the transcription of the new genes

(d) Describe how the formation of separate mature proteins from these polycistronic mRNAs in HIV maturation and in bacteria differs. [3]

Bacteria	HIV maturation
1. The polycistronic mRNA in bacteria has individual start and stop codons for the coding region for each polypeptide	1. The polycistronic mRNA in HIV only has one start and stop codon shared by the coding regions of all polypeptides
2. Coding regions in the polycistronic mRNAs are translated separately	2. Coding regions in the polycistronic mRNAs are translated as a whole / to give one polyprotein
3. There is no need for cleavage / post-translational modifications of the polypeptide	3. Each polyprotein is cleaved by the protease to give separate proteins
4. Polypeptides are translated separately	4. Each polyprotein is cleaved by the protease to give separate proteins

[Total: 10]

QUESTION 4

(a) Describe how an individual can inherit a recessive disorder like albinism or LDHA deficiency from his parents. [2]

1. Ref to both parents having at least 1 recessive allele each
2. Ref to individual receiving 2 recessive alleles

(b) (i) Using the symbols provided, draw a genetic diagram in the space provided below to show the cross between the couple. For each of their children, state their genotypes. [5]

Let **A** represent the dominant allele for normal LDHA production
a represent the recessive allele for LDHA deficiency
N represent the dominant allele for normal skin pigment
n represent the recessive allele for no skin pigment

Parental phenotypes: Woman with normal skin pigment and LDHA production x albino man with LDHA deficiency

Gametes: $\frac{AN}{an}$ → A N, a n, A n, a N; $\frac{an}{an}$ → a n

Random fertilization

		Female gametes			
		A N	a n	A n	a N
Male gametes	a n	$\frac{AN}{an}$	$\frac{an}{an}$	$\frac{An}{an}$	$\frac{aN}{an}$

Child **I** $\frac{AN}{an}$ Child **II** $\frac{AN}{an}$

Child **III** $\frac{aN}{an}$ Child **IV** $\frac{an}{an}$

(ii) Suggest how the genotype of Child **III** has arisen. [2]

1. Ref to incomplete linkage
2. Ref to crossing over

(c) (i) Define the term *chromosomal aberrations*. [1]

Ref to changes in number or structure of chromosomes

(ii) With reference to Fig. 4.1, identify the parent who have the abnormal karyotype and suggest how the abnormality arises. [2]

1. Mother
2. Ref to chromosomal translocation

[Total: 12 marks]

QUESTION 5

(a) (i) Describe the effect of flashing light on ATP synthesis in the absence of venturicidin. [2]

1. As the number of light flashes increases from 1 – 3 flashes, the concentration of ATP increases from 25nM – 190 nM
2. which eventually plateaus

(ii) Explain your answer in **a (i)** [4]

1. Increasing the number of flashes of light result in an increase in the number of excited electrons

2. and results in more excited electrons travelling down the electron transport chain
3. The energy released is coupled to pump/ actively transport H^+
4. from the stroma into the thylakoid space/ thylakoid lumen
5. resulting in the formation of a proton motive force/ electrochemical gradient (OWTTE)
6. H^+ then diffuses down the concentration gradient from the thylakoid space to the stroma
7. via the ATP synthase complex
8. releasing more free energy which is coupled for synthesise more ATP

(b) Describe and explain the effect of venturicidin on the plant and its ability to produce sugars. [4]

1. In the presence of ventruricidin, ATP concentration remains at close to 0nM even when 3 flashes of light are used
2. Since ATP synthase is inhibited, H^+ cannot diffuse down the electrochemical gradient
3. from thylakoid space to stroma
4. thus there is no supply of energy to drive ATP synthesis/ no energy released for formation of ATP
5. This prevents the progression of the Calvin's cycle / no G3P/ no hexose sugar being produced (OWTTE)
6. since ATP is not available for conversion of GP to 1,3-bisphosphoglycerate / regeneration of RuBP

(c) Distinguish the role of ATP synthesis in mitochondria and chloroplasts [1]

1. In mitochondria, ATP synthesised is transported into the cytosol and used for various cellular functions/ active transport/ protein synthesis/ @ref to specific process
2. While ATP synthesised in chloroplasts is transported into the stroma for use in the light-independent reaction / Calvin cycle for synthesis of glucose

R: Photosynthesis/ light dependent reaction

[Total: 11 marks]

QUESTION 6

(a) State what is meant by an *intracellular receptor*. [1]

1. Receptor proteins that are located in the cytoplasm / cytosol / nucleus / cell
2. Ligands, e.g. estrogens, that binds such receptors are hydrophobic / lipid soluble and small enough to diffuse across the hydrophobic core of the cell membrane

(b) Describe stages **A**, **B** and **C**. [5]

Stage A:

1a. Estrogen binds estrogen receptor / ER
1b. via complementary binding (accept: at specific binding site)
1c. resulting in a change in conformation / shape or resulting in ER activation
2. ER bound with estrogens dimerises *(essential for full credit)*
3. and enters the nucleus

Stage B:

4. Estrogen-ER complex binds estrogen response element (ERE)
Accept: reference to ER as a specific transcription factor / activator that binds ERE
Reject: ER activates ERE

Stage C:

5a. Binding of ER complex to ERE causes DNA to bend
5ai activator/ERE is brought close to the promoter
5b. recruitment of mediators / coactivators / M1 and M2
5c. recruitment of general transcription factors / G

6. RNA polymerase is able to bind to the promoter

7. resulting in the formation of stable transcription initiation complex at the promoter

8. to initiate transcription/rate of transcription increases

(c) With reference to Table 6.1,

(i) explain how SERMs can be used in the treatment of breast cancer. [2]

1a. SERMs are structurally similar to estrogen
1b compete with estrogens for binding with ER

2a. SERMs recruit M1 but not M2 / prevent complete recruitment / assembly of mediators
2b. Formation of stable transcription initiation complex is reduced / prevented
Accept: positioning / stability of TIC

3.* This reduces rate of oncogene/cancer critical gene expression OR reduce rate of transcription of gene that result in excessive cell division/rate of cell division exceeding rate of cell death
* Point 3 is essential to achieve full credit because it specifically address 'treatment of cancer'.

(ii) suggest why ERDRs might be more effective than SERMs. [2]

1. ERDRs prevent movement of ERDR-ER complexes to the nucleus
2. ER remaining in the cytoplasm are degraded
3. This completely prevents up-regulation of oncogene expression / transcription (mark not awarded if concept in point 1 is wrong)
4. SERMs still allows movement of ER into the nucleus/enables recruitment of M1 and general transcription factors / transcription initiation complex to be formed for transcription/gene expression

[Total: 10 marks]

QUESTION 7

(a) Discuss how cladograms can be used in classification of organisms. [5]

1. A cladogram is used in classification to represent evolutionary relationship /descent with modification among organisms
2. A cladogram is made up of nested hierarchies
3. Each clade consists of an ancestral species and all its descendants
4. Organisms within a clade are grouped based on synapomorphies/shared derived characters/homology
5. Shared derived characters originate from a recent common ancestor and are present in all its descendants
6. Within a cladogram, living organism may be classified based on molecular homologies
7. Therefore, classification based on cladograms may correspond to traditional classification where each clade may correspond to a genus, family or some broader taxon

(b) State what is meant by transient polymorphism in moths. [1]

1. The existence of two phenotypes / phenotypically distinct forms (melanic and non-melanic) of moths in a particular species

(c) Explain the binomial nomenclature of *Biston betularia*. [2]

1. *Biston betularia* is the unique two-part name of a particular moth species, recognised internationally
2. *Biston* indicates the genus and *betularia* is the specific epithet

(d) Explain for the trends observed for the three moths species from 1987 to 2002. [3]

1. From 1987 to 2002, the % melanic *B. betularia* declined from 95% to 5% as compared to the % melanic *A. crenata* which ranged between ≈ 63% to 60% and % melanic *O. bidentata* declined from 60% to 32%
2. There was a change from polluted (industrial) to clean (post-industrial) environment leading to growth of pale coloured lichens on the bark of trees
3. *B. betularia* are most vulnerable to in the day as they lay on the branches
4. *O. bidentata* are hidden under the leaves or within cracks less likely to be vulnerable

5. Grass grows quickly / colour not influenced by pollution so least change in / less selection pressure on *A. crenata*
6. AVP

(e) Account for the type of speciation that has led to the existence of the three different moth species in Manchester. [2]

1. Sympatric speciation
2. The three moth species occupy different ecological niches

[Total: 13 marks]

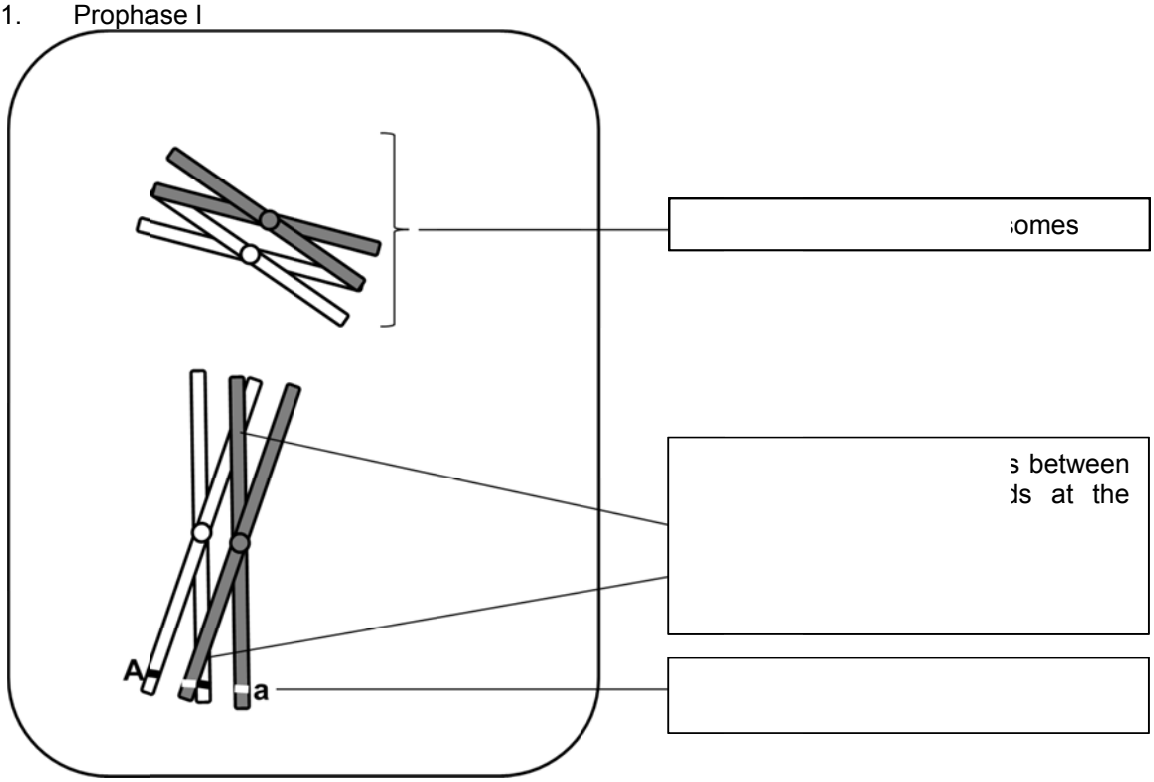
QUESTION 8

(a) Describe the structure of a chromosome prior to the start of mitosis. [5]

1. Prior to the start of mitosis, each chromosome contains two identical DNA molecules joined at the centromeres
2. This is due to DNA replication during S phase of interphase
3. The chromosome exists in the form of uncondensed chromatin and discrete chromosomes are not observed in the cell
4. Each DNA molecule is tightly coiled around the positively charged histone octamer
5. This forms the 10-nm chromatin fibre/ nucleosome fibre/ "beads-on-a-string"
6. DNA is further coiled to give a 30-nm chromatin fibre / solenoid
7. The chromatin within a nucleus prior to mitosis can be organized into diffused euchromatin, or highly condensed heterochromatin.

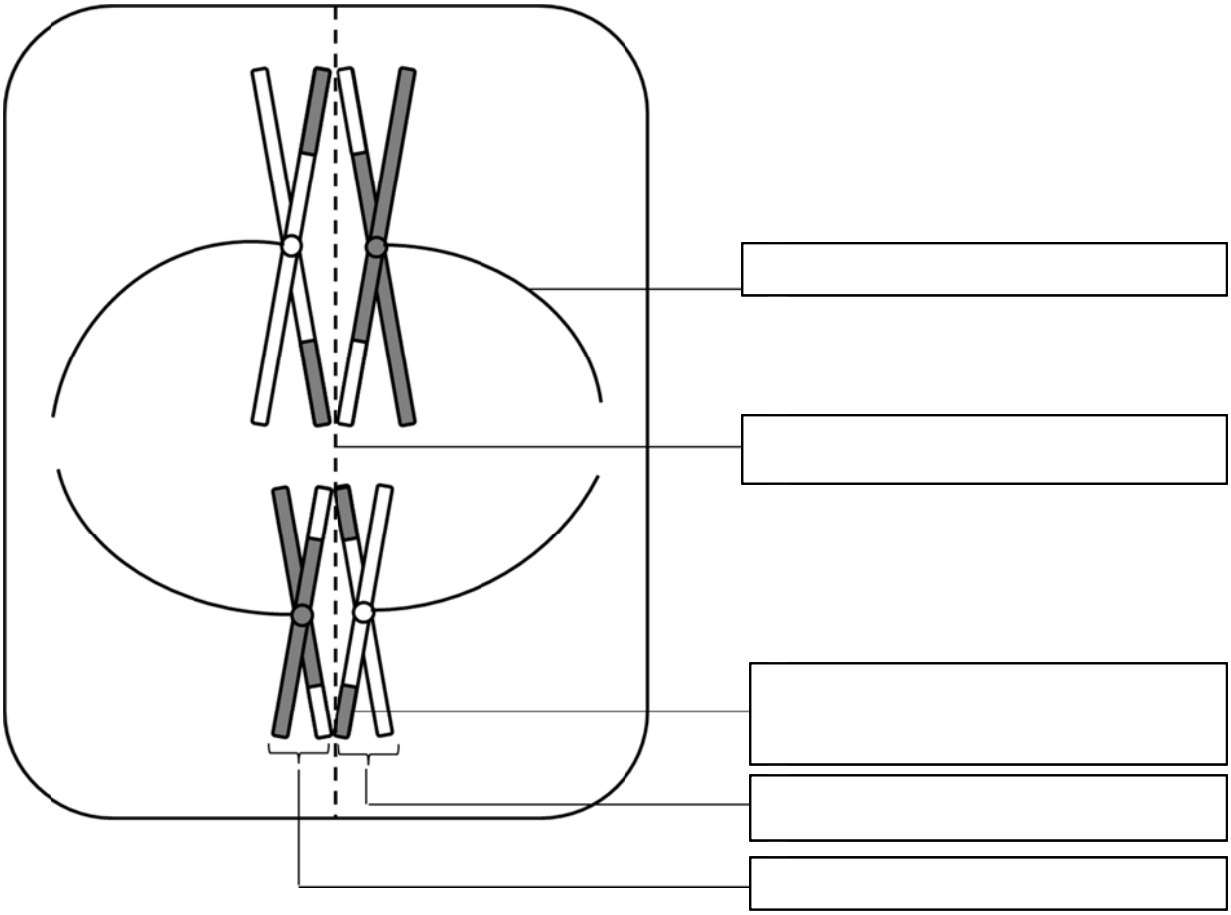
(b) With the aid of large and clearly labeled diagrams, explain the significance of the behaviour of chromosomes during meiosis, which can lead to genetic variation in plants. [10]

PL1. Prophase I



PD1. Homologous chromosomes pair up
PD2. Homologous chromosomes have the same size, shape and centromere location, but different alleles at the same gene loci
PD3. Crossing over
PS1. Exchange of genetic material (alleles) between homologous sections of non-sister chromatids
PS2. Give rise to new combination of paternal and maternal alleles
PS3. Gametes will have chromosomes that are different from parental cells
PS4. AVP

ML1. Metaphase I



MD1. Pairs of homologous chromosomes align at the metaphase plate
MD2. Each homologue on one side of the metaphase plate
MD3. Two or more pairs of homologous chromosomes having different orientations of paternal and maternal recombinant chromosomes towards the poles

MS1. Independent assortment
MS2. There is random distribution of paternal and maternal chromosomes in each daughter cell
MS3. Number of possible combination of gametes due to independent assortment is 2^n for a diploid organism, where n=number of sets of homologous chromosomes
MS4. Daughter cells have different combination of chromosomes compared to parental cells
MS5. AVP

(c) Discuss how the dysregulation of checkpoints of cell division can result in cancer.

[5]

1. Cell cycle checkpoints control/ regulate the cell cycle progression / cell division
2. Mutations in genes E.g., cyclins/ cdks/ Rb/ E2F/ p21/p53/ AVP controlling cell cycle may lead to cell cycle progression
3. Dysregulation in cell cycle signalling pathways may also lead to cell cycle progression
4. Dysregulation of G_1 checkpoint may result in replication of damaged/ defective DNA
5. Dysregulation of G_2 checkpoint may result in cell cycle progression despite DNA damage during DNA replication/ cells escaping apoptosis even if there is irreparable damage
6. Dysregulation of M checkpoint may result in cell cycle progression even if there is non-disjunction
7. Therefore, resulting in more copies of protooncognes /absence of tumour suppressor genes in the daughter cell
8. Gene mutations/ chromosomal aberrations are passed on to daughter cells as the cells do not stop dividing to repair damage
9. Successive rounds of cell division allow accumulation of mutations in other cancer critical genes such as Ras/ p53 in a single cell lineage
10. Eventually there is uncontrolled cell division that the rate of cell division is greater than that of cell death, leading to cancer

QUESTION 9

(a) Discuss the principles of homeostasis. [5]

1. Homeostasis is the maintenance of a constant internal environment of an organism providing it with a degree of independence from the external environment
2. This involves the constant monitoring of multiple physiological conditions or variables within the body to maintain the steady state value / range of values about a specific set point
3. Any fluctuations / deviation above or below the set point serve as the stimulus, which is detected by a receptor
4. This information is integrated at the integrating centre to initiate a response in the form of signals and will act upon target / effector cells
5. Homeostatic control systems usually operate via negative feedback mechanisms where the response carried out returns the variable to the set point

(b) Explain the roles of insulin and glucagon in the regulation of blood glucose. [10]

1. The control of blood glucose concentration operates by a negative feedback system via insulin and glucagon, which are antagonistic hormones
2. When blood glucose concentration is higher than the set point of 90 mg/100ml of blood, the change is detected by the β-cells of the islets of Langerhans of pancreas and insulin is released to decrease blood glucose concentration towards the set point
3. Insulin binds to insulin receptors on target cells and accelerates rate of glucose uptake
4. In muscle, liver and adipose cells, the number of glucose transporters increases in cell membrane
5. In liver and skeletal muscle cells, glucose utilisation and storage increases such that there is an increased rate of glycolysis for production of ATP, glycogenesis stimulated by activating glycogen synthase and glycogenolysis inhibited
6. In skeletal muscle cells, amino acid absorption and protein synthesis stimulated and gluconeogenesis inhibited
7. In adipose tissues, lipogenesis is stimulated
8. When blood glucose concentration is lower than the set point of 90 mg/100ml of blood, change is detected by the α-cells of the islets of Langerhans of pancreas, glucagon is released to increase blood glucose concentration towards the set point
9. Glucagon binds to glucagon receptors on target cells and stimulates glycogenolysis in liver and muscle cells
10. Gluconeogenesis stimulated in liver cells and lipolysis in adipose tissues

(c) Describe how two named membrane proteins are involved in impulse transmission along the axon of a motor neuron. [5]

L1. Sodium and potassium leak channels
L2. There are more K^+ leak channels than Na^+ leak channels, leading to more diffusion of K^+ than Na^+ down their respective concentration gradient via facilitated diffusion
L3. This results in a net loss of positive charge
L4. Resting membrane potential of neurons restored / maintained at about -70mV, so that it can be excited to produce action potential when there is a stimulus
P1. Sodium-potassium pumps
P2. Three sodium ions are pumped out of the neurone while two potassium ions are pumped into the neuron via active transport against their respective concentration gradient
P3 It restores the original ionic gradients and results in a net loss of positive charges
P4. Resting membrane potential of neurons restored / maintained at about -70mV, so that it can be excited to produce action potential when there is a stimulus
S1. Voltage-gated sodium channels
S2. A stimulus causes some voltage-gated Na^+ channels to open, which allows influx of Na^+,
S3. and results in depolarisation of membrane
S4. If the depolarisation reaches threshold potential of -55mV, an action potential is generated, so that more/ all voltage-gated Na^+ channels will open
S5. depolarising the membrane potential to +40mV
K1. Voltage-gated potassium channels
K2. At the peak of an action potential, voltage-gated K^+ channels opens, which allows efflux of K^+
K3. and hence repolarisation
K4. Voltage-gated K^+ channels are slow to close, so that excessive K^+ efflux causes the membrane potential to hyperpolarised