

2024 JC1 DECEMBER REVISION-ANSWERS

NAME:

Class:

Topic: CELLULAR STRUCTURES AND FUNCTION

Question 1 [6 marks] TJC Prelims 2009

(a) With reference to Fig. 1.1, suggest the principal function of the pancreatic acinar cell. Give two reasons to explain your answer. [3]

Function

- **secretion** of pancreatic / digestive enzymes or secretion of hormones

Reasons

- presence of large amount of RER, which is the site where pancreatic enzymes are synthesized
- presence of SER, which is the site where steroid hormones / lipids are synthesized
- presence of large number of secretory vesicles for release of pancreatic enzymes/ hormones outside the cell
- presence of mitochondria which synthesizes ATP for
 - Activation of amino acids OR
 - Formation of peptide bonds (at ribosomes) OR
 - Transport / Movement of vesicles towards cell surface membrane for release of pancreatic enzymes/ hormones

(b) (i) Describe two ways in which A is similar to B. [2]

- Both are bound by a **single membrane / single membrane-bound** structures.
- Both consist of membranous sacs called **cisternae**, in which newly synthesized proteins undergo processing.
- Both give rise to membrane-bound **vesicles** which are involved in **transport** of newly synthesized proteins within the cell.

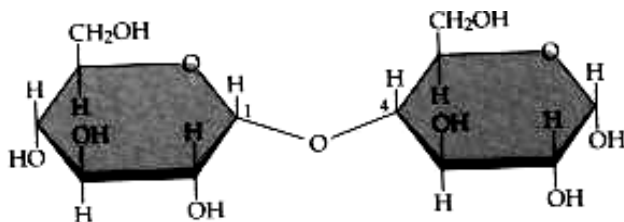
(ii) The products of B are usually in the inactive form. Suggest the significance of this. [1]

- to prevent the enzymes (of lysosomes) from digesting proteins / contents within the cells in which they are synthesized

Topic: BIOMOLECULES AND CELL MEMBRANE

Question 2 [11 marks]

(a) Draw the resulting maltose molecule arising from a dehydration reaction. [1]



(b) Label the resulting bond. [1]

- Alpha (1→4) glycosidic bonds

Starch, a storage molecule in plants, is a polymer made of glucose molecules joined together. It consists of two components.

(c) Name these two components. [2]

- amylose
- amylopectin

(d) (i) Give one similarity between these two components. [1]

- contain α -glucose as monomers
- coiled into a helix
- no cross-linking

(ii) Give two differences between these two components. [2]

- The amylose component is unbranched whilst the amylopectin component is branched.
- The glucose residues in the amylose component are linked by $\alpha(1\rightarrow4)$ glycosidic bonds, whilst the glucose residues in the amylopectin component are linked by both $\alpha(1\rightarrow4)$ glycosidic bonds and $\alpha(1\rightarrow6)$ glycosidic bonds (at the branchpoints)

(f) The following figure shows a section of a cell surface membrane.

(i) Explain why membranes are described as fluid mosaic in structure. [2]

'Fluid' (any 1)

- phospholipids **and** proteins free to **move laterally** within the membrane
- phospholipids able to move **transversely / flip flop**

'Mosaic'

- **Different** proteins **randomly** scattered embedded/ ref. to position

(ii) Some molecules are able to pass through the plasma membrane without the facilitation of transport proteins. Describe the unique properties of these molecules. [1]

- small / non-polar / hydrophobic

(iii) Outline two other roles of membrane proteins apart from their role in transport. [2]

- Enzymatic activity: Protein has its active site exposed to substances in the adjacent solution.
→ Enables the cell membrane to be involved in certain metabolic pathway
- Signal transduction: Protein function as receptors for extracellular signaling molecules
→ allow cells to sense changes in the external environment and respond to them
- Glycoproteins serve as cell identity markers → Enable cells to recognize other cells
- Membrane proteins of adjacent cells may be attached → Enables cell-to-cell adhesion
- A protein built into the membrane may be an enzymes with is active site exposed to substances in the adjacent solution.
- Membrane proteins may be bonded to components of the cytoskeleton → maintains cell shape

Topic: ENZYMES

Question 3 [7 marks] JJC Prelims 2009

(a) Suggest how the protein may be held in the active site of a papain while the reaction occurs. [2]

- Protein substrate has shape complementary to the shape of the active site of the enzyme
- enzyme interacts with the substrate via hydrophobic interactions, hydrogen bonds and ionic bonds → temporary enzyme-substrate complex

(b) Use your knowledge of enzymes to explain:

(i) Why the rate at which protein is digested increases as the meat warms up in the early stages of cooking. [2]

- In early stages of cooking, heating increases the kinetic energy of both papain and protein molecules in the muscle
- rate of successful collisions / number of collisions per unit time between papain and protein molecules increase; → increase rate of formation enzyme-substrate complexes/ number of E-S complexes per unit time ;

(ii) Why protein digestion stops once the meat has been heated to 90°C. [3]

- At 90°C, large amount of heat present resulting in denaturation of papain;
- bonds (give 2 examples: hydrogen bonds, hydrophobic interactions, ionic bonds) maintaining the 3D conformation of enzyme are broken
- causing loss of active site / change in shape of active site → shape of active site no longer complementary to substrate

Question 4 [6 marks]

(i) Using the information in Fig 4.1, describe and explain the results obtained for test tubes **1** and **4**. [3]

- Tube 1– from 0min to 50min, percentage transmission increases from 20% to 70% and then levels off/ remains constant at 70% for the next 10mins
- (increases) as more substrate had been converted/ [substrate] decreases. Levels off as all substrate has been converted
- Tube 4 – remains constant/ remains at 20% (Reject – no transmission) as pectinase **denatured**

(ii) Explain why test-tube **2** was included in the experiment. [2]

Control;

To show that enzyme/ pectinase is the only factor responsible for cloudiness clearing/ increase in transmission

(iii) Sketch on the graph the results you would expect for test-tube **3**. [1]

Horizontal line somewhere between 70 and 100% transmission

Topic: CELL AND NUCLEAR DIVISION

Question 5 [10 marks]

(a)(i) Identify stages A to D. [2]

A Metaphase I

B Anaphase I

C Telophase I

D Anaphase II

(ii) State the type of nuclear division shown in Fig. 5.1 [1]

Meiosis

(iii) Explain your answer to part (ii). [1]

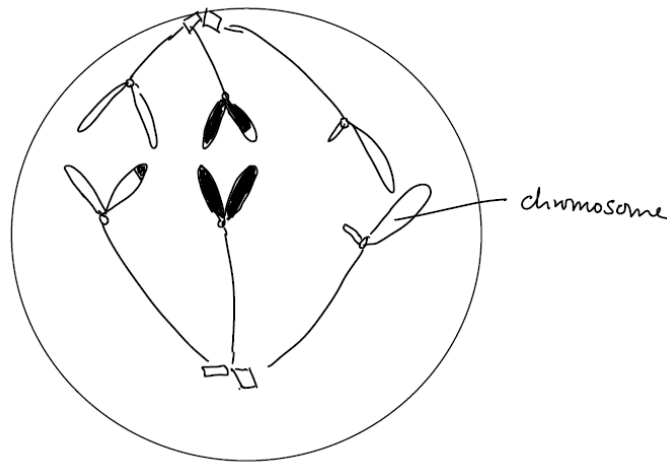
- 2 nuclear divisions resulting in 4 nuclei/cells formed from 1 cell

(b) The graph below shows the relative amounts of DNA per cell during two successive cell divisions in a plant.

(i) Describe the events occurring at X. [3]

- cytokinesis occurred where amount of DNA in the cell dropped from 4.8 units to 2.4 units and then becomes a constant
- DNA replication occurred: Amount of DNA then doubled from 2.4 units to 4.8 units

c) Draw the behaviour of the chromosomes undergoing anaphase II during nuclear division in the cell given below where ($2n = 6$). [3]



- Number of replicated chromosomes = 3
- Separation of sister chromatids
- At least 1 pair of sister chromatids must be non-identical

Topic: DNA REPLICATION

Question 6 [12 marks] ACJC Prelims 2010

- (a) Describe the normal function of nuclear pores. [2]
- regulates the **movement** of substances into and out of the nucleus
 - Entry of histones, gene regulatory proteins, DNA and RNA polymerase, ribosomal proteins (at least 1 example)
 - Exit of mRNA, ribosomal subunits (at least 1 example)
- (b) Explain the importance of DNA replication prior to mitosis. [2]
- DNA/genome is replicated to form 2 **identical** copies/sets / **doubling** of DNA/genetic material
 - Each daughter cell will receive a **complete** set of/the same genetic information / ref. to each daughter cell will be **genetically identical**
- (c) Describe the importance of complementary base pairing in DNA replication. [2]
- Allows **accurate** replication of DNA, since daughter strand is complementary to parental strand
 - Ref. to using one strand as a **template**
 - Ref. to A always pairs with T and C always pairs with G

- (d) Explain why synthesis of DNA occurs both continuously and discontinuously during DNA replication. [2]
- DNA polymerase only adds **DNA nucleotides** to the **free 3' end** of the newly synthesized strand/ DNA polymerase synthesizes DNA in the 5' to 3' direction
 - The two **parental/ template DNA strands** are **anti-parallel**
- (e) Acyclovir is an anti-viral drug which is used to treat herpes. It prevents the complete replication of viral DNA by viral DNA polymerase. The structure of acyclovir is shown below in Fig. 9.1.
- (i) With reference to Fig. 9.1, suggest how the incorporation of the phosphorylated acyclovir prevents further elongation of the DNA strand. [2]
- ref. to **OH group not in correct orientation / missing 3' OH group**
 - **DNA pol** cannot catalyse formation of **phosphodiester bond** (between acyclovir and new nucleotide)
- (ii) Some strains of herpes simplex virus are now resistant to acyclovir. Suggest how the virus has gained resistance to this anti-viral drug. [2]
- **Mutation affecting thymidine kinase viral / viral DNA polymerase**
 - **Thymidine kinase cannot phosphorylate acyclovir / viral DNA polymerase cannot add acyclovir**

Accept Any valid points

Topic: PROTEIN SYNTHESIS

Question 7 [10 marks]

- (a) Label processes A and B. [1]

A: Transcription, B: Translation

- (b) In order for process A to occur, the DNA double helix must first be unzipped by enzymes. In vitro, DNA strands can also be separated by heat – denaturation of DNA.
- (i) Explain how DNA is denatured by heat. [1]
- **High heat breaks the hydrogen bonds between the nitrogenous bases**

(ii) Two DNA segments are shown below.

CTAGCTGAACGCGCCCGATT
GATCGACTTGCGCGGGCTAA

Segment 1

TAAGCTTAACGCGACTGATA
ATTCGAATTGCGCTGACTAT

Segment 2

Identify the segment that would require a higher temperature for denaturation of the DNA. Explain your answer. [2]

- Segment 1
- Segment 1 has 52 hydrogen bonds versus 48 hydrogen bonds in segment 2/ segment 1 has 4 more hydrogen bonds than segment 2/ segment 1 has 4 more G-C pairs than segment 2 hence **more (heat) energy** required to break bonds

(c) Process A and DNA replication require different enzymes and substrates. Describe two other differences between these two processes. [2]

Features	Transcription	Replication
Monomers used (REJECT)	▪ Ribonucleoside triphosphates / RNA nucleotides / ribonucleotides	▪ deoxyribonucleoside triphosphates / DNA nucleotides / deoxyribonucleotides
Enzyme involved in forming bonds between monomers (REJECT)	▪ RNA polymerase	▪ DNA polymerase
Template	• Only 1 DNA strand is used as template	▪ Both DNA strands are used as template
Extent of replication/ transcription	Only a certain region (gene) of the DNA molecule is transcribed in a single process.	The whole DNA molecule is replicated in a single process.
Products	▪ RNA ▪ Product transported out of nucleus (REJECT)	▪ DNA ▪ Product remains in nucleus (REJECT)

(d) In all cells, there are many types of structure C. State the minimum number of types that should be present and explain your answer. [2]

- 20
- As there are 20 different types of a.a. each requiring a specific type of tRNA

(e) Based on Fig. 10.1, explain how protein synthesis in *Agrobacterium tumefaciens* would differ from that of a plant. [2]

- Transcription and translation occur together in the same location / simultaneously
- Because there is no nuclear membrane in bacteria

Topic : CONTROL OF EUKARYOTIC GENE EXPRESSION

Question 8 [5 marks] NJC Prelims 2009

(a) With reference to Fig. 11.1, state two ways in which transcription in eukaryotes differs from that in prokaryotes. [2]

- In eukaryotes, transcription begins when **general transcription factors (TFIID and TFIIA)** and **RNA polymerase II holoenzyme complex** bind to the promoter / TATA box while in prokaryotes, transcription begins when RNA polymerase binds to the promoter
- In eukaryotes, DNA bending has to occur for activator to be brought near to promoter while in prokaryotes, there is no DNA bending
- In eukaryotes, RNA polymerase / GTFs / binds to the TATA box while in prokaryotes, RNA polymerase binds to the Pribnow box / there is no TATA box

(b) With reference to Fig. 11.1, explain the mechanism behind the enhancement of the rate of transcription. [3]

- **Activators** bound to **enhancer** causes **DNA bending**, bringing the bound activators close to the promoter.
- Activator's **activation domain** bind to **mediator protein** of the **RNA polymerase II holoenzyme complex**
- Promoting the assembly of the **RNA polymerase II holoenzyme complex** with **general transcriptions (TFIID and TFIIA)** → **accelerate** and **stabilise** formation of **transcriptional initiation complex** on the **promoter**

Question 9 [12 marks]

If the cDNA probe finds sequences to bind to, then the gene concerned is said to be 'protected' from DNase I digestion by chromatin proteins.

(c) (i) Name one such chromatin protein. [1]

- Histone(s) / Any of histone H1, H2A, H2B, H3 and H4

(ii) Explain how these proteins protect the gene concerned from DNase I digestion. [2]

1. DNA wound/wrapped around histones
2. Gene or DNA not exposed / accessible to DNase I enzyme / DNA double helix cannot fit into active site of DNase I;

(d) With reference to Table 9.1,

(i) describe the difference in the levels of binding of the two types of cDNA probe for the developing RBC samples. [1]

- Lower % binding of globin cDNA probe (quote value 25%) compared to % binding of ovalbumin cDNA probe (quote value 93%)

(ii) state which gene is more actively transcribed in developing red blood cells. [1]

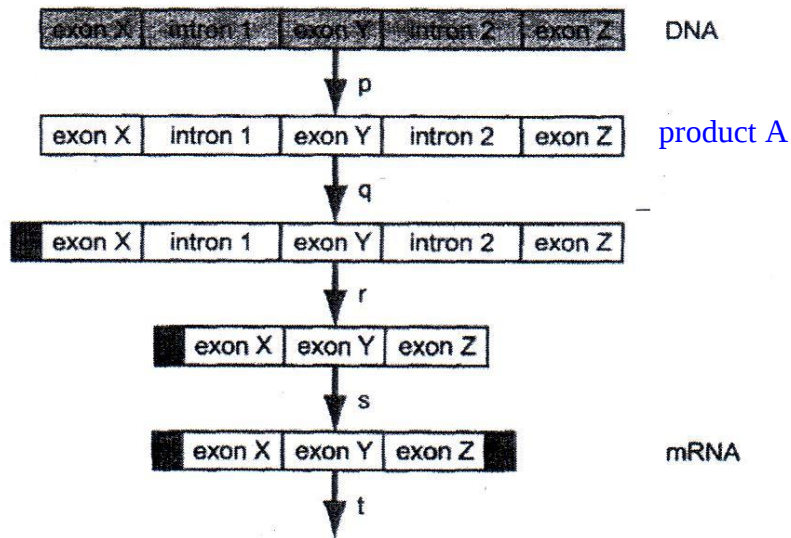
- Globin gene;

(iii) explain your answer to **(b)(ii)**. [2]

Reject answers that relate function of globin gene to rbc e.g. globin gene encodes for part of Hb and Hb is found in rbc to help transport oxygen around the body

1. globin gene DNA was relatively less protected against DNase I digestion
Or has larger extent of DNase I digestion;
2. Globin gene DNA less tightly coiled (around histones)
OR Globin gene found within euchromatin / loosely coiled chromatin whereas ovalbumin gene found within heterochromatin;
3. So that **promoter** region accessible to **transcriptional factors and RNA polymerase** to form **transcription initiation complex**

Fig 9.1 below represents the processing of pre-messenger RNA. PJC Prelims 2008



Name process p to t

Identify product A

Fig 9.1

(c) Describe events r to t. [5]

Event r

- introns 1 and 2 are **removed/ excised** from **pre-mRNA** (by spliceosome)
- exons X,Y and Z are **spliced / joined** together
- to form a mRNA with a **continuous coding sequence**

Event s:

- Addition of **adenine nucleotides** (by poly-A polymerase) to **3' end** of mRNA
→ **poly-A tail**

Event t:

- **functional/mature** mRNA leaves the nucleus via nuclear pore to cytoplasm for **translation**

Topic : CELL SIGNALING

Question 10

(a) With reference to Fig. 6.1,

(i) describe how EGFR is activated, [3]

- EGF binds at EGFR binding site via complementary shape
- triggers dimerisation of EGFR subunits
- This cause the subunits to undergo cross phosphorylation at their tyrosine residues

(ii) describe how the signal from the activated receptor is transduced to Raf. [3]

- Activated receptor binds to GAB1-Grb2-Shp2 complex
- phosphorylating / activating the complex, Complex dissociates from the EGFR
- binds and activates Ras which dissociate from the cell membrane
- binds to and activate Raf

(b) Describe and explain the results of the test for the diabetic patient in the first 100 minutes. [3]

- From 0 min to 100 min, blood glucose concentration increases from 8 mM/L to 15.5 mM/L
- Glucose is absorbed into the blood stream
- and is not taken up by liver / muscle cells
- Insulin is not secreted by β cells of Islet of Langerhans of the pancreas

(c) Suggest a reason for the drop in blood glucose concentration for the diabetic patient. [1]
Glucose used for cellular respiration

Question 11 CJC 2015 Prelims P2 Q2

(a)

(i) Suggest why the hormones, referred to in the passage, are unable to enter the cell. [2]

- too large to cross membrane ;
- hydrophilic/water soluble ; Accept: not, hydrophobic/lipid soluble
unable to pass through hydrophobic core / AW of phospholipid bilayer ;

(ii) Use the information in the passage to outline the process of cell signalling. [3]

- hormones combine with cell surface receptors ;
- on target cells / cells where transcription is triggered ;
- action of kinases and phosphatases (within the cell) lead to (specific) response
- specific response = transcription / production of mRNA ;

(a) With reference to Fig. 18.1, state how the levels of *ras* and p53 proteins correlate with the development of cancer. [2]

- In the cancerous tissue, there are elevated / higher levels of *ras* protein as compared to the normal tissue.
- Also, the p53 protein level is diminished / lower in cancer tissue than in the normal tissue

- (b) State the role of the *ras* gene in relation to the development of cancer. [2]
- Ras gene is a proto-oncogene.
 - High levels of expression ras protein constantly stimulates cell division
- (c) Explain how mutations in the *p53* gene may lead to uncontrolled cell division. [4]
- *p53* is a tumor suppressor gene involved in the inhibition of normal cell division
 - Mutation in both alleles of the *p53* gene leads to a loss of function mutation
 - When there is DNA damage, the mutated *p53* protein is unable to repair the DNA damage / there is not stimulation for the cell to undergo apoptosis
 - cells with mutation continue to divide, resulting in increased in frequency of oncogenes and mutated tumour suppressor genes accumulating in cells lacking p53

Topic: STEM CELL

Question 12 [6 marks]

(a)

- (i) In terms of differentiation potential, explain how an adult bone stem cell is different from a fertilized egg of a human. [2]

The zygote/fertilized egg is totipotent i.e. it can give rise to all cells types in the body including extra embryonic tissue

but the adult bone stem cell is multipotent i.e. it can only give rise to a limited range of cell types

- (ii) Explain how the adult bone stem cells may increase bone growth in children with osteogenesis imperfecta. [2]

Stem cells can divide by mitosis

and in the presence of the appropriate signals can also differentiate into new bone cells;

This thus increases the number and type of bone cells present which contribute towards bone growth

(b)

- (i) With reference to Table 3, give evidence that the cells from the stem tips are totipotent. [2]

Quote any values that produce plantlets, For example, at $10 \mu\text{mol dm}^{-3}$ auxin and $50 \mu\text{mol dm}^{-3}$ cytokinin, whole plantlets (with leaves and roots) are produced from the stem tips

The stem tip cells are totipotent and hence able to differentiate into all specialized cell types to form a whole plant

Topic: CANCER

Question 13 [8 marks] YJC Prelims 2009

- (a) With reference to Fig. 18.1, state how the levels of *ras* and *p53* proteins correlate with the development of cancer. [2]
- In the cancerous tissue, there are elevated / higher levels of *ras* protein as compared to the normal tissue.
 - Also, the *p53* protein level is diminished / lower in cancer tissue than in the normal tissue
- (b) State the role of the *ras* gene in relation to the development of cancer. [2]
- Ras* gene is a proto-oncogene.
 - High levels of expression *ras* protein constantly stimulates cell division
- (c) Explain how mutations in the *p53* gene may lead to uncontrolled cell division. [4]
- p53* is a tumor suppressor gene involved in the inhibition of normal cell division
 - Mutation in both alleles of the *p53* gene leads to a loss of function mutation
 - When there is DNA damage, the mutated *p53* protein is unable to repair the DNA damage / there is not stimulation for the cell to undergo apoptosis
 - cells with mutation continue to divide, resulting in increased in frequency of oncogenes and mutated tumour suppressor genes accumulating in cells lacking *p53*

Question 14 [13 marks] HCI Prelims 2008

- (a) Account for the difference between Group B and Group C mice. [3]
- Group B mice have one functional copy of *p53* gene while Group C mice have both copies of the *p53* gene mutated
 - Therefore, Group B mice still can produce sufficient functional *p53* while Group C mice cannot produce any *p53* protein.
 - Since *p53* is a tumour suppressor protein / describe function of *p53*
 - (compulsory pt) leading to lower rate of survival (quote values comparing both groups: values related to age age(days) , % survival and comparative terms must be present: higher/lower/ faster/ slower)
- (b) Contrast the possible genetic changes that may lead to tumour formation in the *p53* gene and in a proto-oncogene. [3]

	Possible changes to <i>p53</i> wild type allele	Possible changes to proto-oncogene
Point mutation in gene itself leads to:	<u>Point mutation leads to loss in function of tumour suppressor protein, decrease in activity or less resistance to degradation.</u>	<u>Point mutation leads to gain in function to give rise to oncogene increase in activity of protein or increased resistance to degradation.</u>
	<i>Must have a point for point comparison loss of fn – gain in fn etc</i>	
Point mutation in control element leads to:	<u>Point mutation in control element must lead to less transcription or no transcription / expression</u>	<u>Point mutation in control element leads to increased transcription / expression</u>
Gene amplification:	<u>Gene amplification will not cause tumours to arise</u>	<u>Gene amplification will cause tumours to arise</u>
Translocation of gene to:	<u>Translocation, gene moved to be controlled by a less active / inactive promoter may cause tumours</u>	<u>Translocation to an active promoter may result in tumours</u>

- (c) (i) Describe the significant changes in a mouse cell from the point of acquiring of the mutant p53 alleles to the cell becoming cancerous. [4]
1. With mutant p53 alleles, there is a **defective** transcription factor / **no** transcription factor is produced / no activation of transcription of genes
 2. (max 2) coding for proteins that inhibit the cell cycle / no activation of transcription of genes
damaged DNA is not repaired. / cell which has damaged DNA do not undergo apoptosis
 3. **accumulation** of mutations in cells through mutation occurring in other genes and **tumour** leading to **uncontrolled cell division**.
 4. Example of mutations occurring in other genes (any 1) : inactivation of **proto-oncogenes** into oncogenes / activation of genes that signal **angiogenesis**/ **inactivation of** genes involved in **cell-to-cell adhesion**/ activation of **telomerase** gene
- (ii) Explain how can one cancerous cell eventually lead to the death of the mouse. [3]
1. Cancerous cell divides to give more cancerous cells → forming a tumour;
 2. Tumour / cancerous cells can metastasize / travel in the blood stream to other tissues;
 3. Disruption of function of the organ / tissue where the tumour is located
 4. Diversion of nutrients from normal tissue cells to tumour cells causing normal cells to die

Topic: GENETICS OF VIRUSES

Question 15 [14 marks] NJC Prelim 2013 and RVHS Prelim 2013

- (a) Name structures A and B. [2]
- A: gp120
 - B: CD4 receptor
- (b) Describe the events occurring in stage C leading to formation of structure A on the host cell's plasma membrane. [3]
- Polypeptides translated by ribosomes enter the lumen of rough endoplasmic reticulum for proper folding.
 - Proteins are packaged in transport vesicles which bud off RER and fuse with cis face of golgi apparatus in which glycosylation occurs.
 - Glycoprotein is inserted into the membrane of secretory vesicles when they bud off the trans face of golgi apparatus and fuse with the plasma membrane. The glycoprotein/structure A will be incorporated in the plasma membrane.
- (c) After contraction of HIV, the initial symptoms are followed by a stage called clinical latency when no symptoms are observed for a few years. With reference to the reproductive features of HIV, explain the cause of clinical latency. [2]
- The reverse transcribed HIV DNA is integrated into the host chromosome as provirus.
 - Provirus remains silent/ transcriptionally inactive for years, causing no symptoms.

(d) Explain how lamivudine prevents HIV from multiplying in the body. [2]

- Prevents viral cDNA synthesis
- Reverse transcriptase incorporates
- lamivudine instead of viral nucleotide
- Terminating DNA chain elongation due to absence of 3' OH group

(e) Explain how the genomic variation shown in Fig. 3.3 affects the structure of gp120 on the two strains of HIV. [3]

- Base pair substitution mutation at 145th base pair, changes adenine to cytosine
- results in change in corresponding codon on mRNA
- thus changes the amino acid encoded in the resultant polypeptide chain
- This changes the property of amino acid residue / folding of polypeptide
- *Reject: protein*
- resulting each HIV having gp120 with different three-dimensional conformation

(f) Flu is caused by influenza viruses. Distinguish the genome replication between HIV and influenza virus. [2]

Feature	HIV	Influenza virus
<i>Starting material</i>	<i>Single stranded Positive sense RNA</i>	<i>Single stranded negative sense RNA</i>
<i>Viral enzymes involved</i>	• <i>Reverse transcriptase synthesize double stranded cDNA from ss RNA;</i> • <i>Integrase incorporate the viral cDNA as provirus into host chromosome</i>	• <i>RNA-dependent RNA polymerase synthesizes the complementary sense strand followed by anti-sense strand</i> • <i>The viral genome is not integrated into the host chromosome</i>
<i>Time taken to replicate the genome</i>	<i>Provirus remains silent for years. It will be transcribed to form the positive ssRNA and mRNA when provirus is activated to produce new HIV virion.</i>	<i>Fast and immediate.</i>