# Nov 2013 H2 Bio Paper 2

## N13P2Q1

**(a)** Describe the processes that occur in the Golgi body. [3]

1. ***Glycosylation**** where oligosaccharides/short sugar chains are added to proteins and lipids to form glycoproteins and glycolipids respectively;
2. Modification of existing glycoproteins and glycolipids made in the endoplasmic reticulum by modifying the existing oligosaccharide / cleaving sugar molecule(s) from the oligosaccharide;
3. In plant cells, Golgi body is the site for synthesis of polysaccharides such as pectin (a component in plant cell wall) and then transported in vesicles to the cell membrane;
4. ***Sorting**** of proteins into different kinds of vesicles to target the proteins to different parts of the cell or for secretion;

**(b)** Describe the role of vesicles that fuse with the forming face of the Golgi body. [3]

1. Vesicles that bud off from rough endoplasmic reticulum (RER) carry proteins;
2. and bud off from smooth endoplasmic reticulum (SER) carry lipids;
3. These vesicles are targeted to fuse to cis-face of Golgi by microtubules of the cytoskeleton/proteins;

**(c)** Outline **two** roles for the vesicles that are formed at the maturing face of the Golgi body. [4]

1. Lysosome / lysosomal vesicles;
2. fuses with vesicles/vacuoles formed by endocytosis to digest the contents (e.g. foreign particles) within the vesicles/vacuoles; (pt 1 & 2 = 2 marks)
3. Exocytic vesicles;
4. fuses with plasma membrane to release enzymes outside of cell by exocytosis to breakdown extracellular content / fuses with plasma membrane to release contents outside of cell by exocytosis; (pt 3 & 4 = 2 marks)
5. Secretory vesicles;
6. fuses with plasma membrane to release contents upon receiving signal; (pt 5 & 6 = 2 marks)

Any 2 pairs of points

## N13P2Q2

**(a)** State what happens if a cell loses control of the cell cycle. [1]

1. Uncontrolled cell proliferation;

**(b)** With reference to Fig. 2.1, suggest how the dysregulation of checkpoints of cell division may occur. [3]

1. Point mutation of S-cyclin gene to cause change in amino acid sequence of S-cyclin protein, thus S-cyclin is resistant to degradation / Point mutation of M-cyclin gene to cause change in amino acid sequence of M-cyclin protein, thus M-cyclin is resistant to degradation;
2. Prolonged activation of M-CDK and S-CDK to cause more rounds of cell division thus excessive cell proliferation;
3. Gene amplification of CDK gene to increase number of copies of gene resulting in increase amount of CDK protein;
4. Increase number of binding sites for S-cyclin/M-cyclin to form increase amount of active CDK;

**(c)** **(i)** Name **one** causative agent of cancer. [1]

1. Viruses e.g. HPV, avian sarcoma virus;
2. Ultraviolet light / Ionising radiation (mention of radiation along is insufficient);
3. Carcinogens e.g. tar in cigarette smoke, asbestos, benzene, formaldehyde, ethidium bromide;

Any one

**(ii)** Outline the development of cancer including the effects of this causative agent. [5]

1. The causative agent increase chances of DNA damage and mutations in the genes which control regulatory checkpoints of the cell cycle in a single cell;
2. Loss-of-function mutation of tumor suppressor genes will result in inability to inhibit cell cycle, repair damaged DNA and promote apoptosis;
3. Gain-in-function mutation of proto-oncogenes to form oncogenes will result in overexpression of proteins/growth factors OR production of hyperactive/degradation resistant proteins/growth factors;
4. leading to excessive cell proliferation/division to form tumour;
5. Loss of contact inhibition enables cells to grow into a tumour;
6. Activation of genes coding for telomerase so that cells can divide indefinitely;
7. Angiogenesis must occur within the tumour so that the blood vessels formed can transport oxygen and nutrients for its growth;
8. Resulting in the formation of a maglinant tumour capable of metastasizing to other parts of body to form secondary tumours;

**N13P2Q3**

Fig.3.1 shows the mechanism of DNA replication originally proposed by Watson and Crick.

**(a)** State the name given to this mechanism of DNA replication. [1]

***Semi-conservative DNA replication****

**(b)** Describe how these results provided evidence for Watson and Crick's proposed mechanism. [3]

1. In generation 0 only the heavy $^{15}N$-$^{15}N$ DNA molecules were present which appeared as the lowest band in the caesium chloride solution.
2. During semi-conservative replication, the original $^{15}N$-$^{15}N$ DNA strands ***unzipped**** and served as ***templates**** for the formation of the new strands. Since only $^{14}N$ DNA was present in the medium, the resulting DNA molecules in generation 1 were ***hybrid**** DNA molecules where each molecule was made up of one original $^{15}N$ strand and one new $^{14}N$ strand. This hybrid DNA molecule thus appeared as the intermediate band in the caesium chloride solution.
3. Each hybrid DNA molecule from generation 1 unzipped and were used as templates for DNA replication. Hence 50% of the DNA in generation 2 was made up of hybrid DNA (i.e. $^{15}N$-$^{14}N$) which appeared as the intermediate band and 50% was made up of light DNA (i.e. $^{14}N$-$^{14}N$) which appeared as the uppermost band in the caesium chloride solution.

Thus the appearance of the various bands at the various generations is consistent with semi-conservative replication.

**(c)** List three ways in which transcription is different from DNA replication.[3]

| **Feature** | **DNA replication** | **Transcription** |
|---|---|---|
| **Template** | In replication, both DNA strands serve as template. | In transcription, only one strand acts as template. |
| **Product** | 2 double-stranded DNA molecules are synthesised. | 1 single stranded RNA molecule is synthesised. |
| **Polymerase involved** | *DNA polymerase** catalyses the reaction. | DNA-dependent *RNA polymerase** catalyses the reaction. |
| **Process associated with** | The process is associated with cell and nuclear division. | The process is associated with protein synthesis. |
| **Base that pairs with Adenine** | Thymine is used during the process. | Uracil is used during the process. |
| **Process** | Both DNA strands are used as template in the synthesis of DNA. | One DNA strand is used as template for the synthesis of mRNA. |
| **Nucleotides used** | Deoxyribonucleotides and ribonucleotides (for RNA primer) are used | Ribonucleotides are used. |

*Comments: The main differences between transcription and replication were well known to most candidates and many provided full responses.*

**(d)** Explain how the information to synthesise polypeptides is coded for by DNA.[3]

1. A specific sequence of nucleotides on a template DNA strand is transcribed by RNA polymerase to form mRNA by ***complementary base pairing****;
2. The mRNA contains triplet base codes known as ***codons**** that each codes for a specific amino acid;
3. When the mRNA containing a specific sequence of codons which code for a specific amino acid sequence,is translated at ribosomes, polypeptides are formed.

**N13P2Q4**

Fig.4.1 shows an outline of the first three stages of aerobic respiration

**(a)** Name the stages **A**, **B** and **C** and state precisely where in a eukaryotic cell these stages occur.[3]

**A:** ***Glycolysis*** occurs in the ***cytosol***
**B:** ***Link reaction*** occurs in the ***matrix of the mitochondrion***
**C:** **Krebs cycle** occurs in the ***matrix of the mitochondrion***

*Comments: Candidates demonstrated sound knowledge and understanding of aerobic respiration. Most candidates were able to identify where the stages occurred. Some candidates were not precise in their responses.*

**(b)** For each glucose molecule, state the total number of ATP formed as a result of stages A and C, including any ATP produced through oxidative phosphorylation of the products.[2]

A: **7** (2 ATP + 2NADH where each NADH produces 2.5ATP = 2 +5 =7)
C: **20** (2 ATP + 6NADH where each NADH produces 2.5ATP + 2 FADH where each FADH produced 1.5ATP = 2 +15 + 3 =20)

Also accept: A:8 and C:24 (as students may use 1 NADH produced 3ATP and 1 FADH produced 2 ATP)

Oxidative phosphorylation regenerates FAD and NAD from the reduced FAD and reduced NAD.

**(c)** Outline how this results in the production of ATP.[4]

1. Reduced NAD and FAD transfer their high energy electrons to the electron acceptors of the electron transport chain, and get re-oxidised in the process;
2. As electrons are passed down electron carriers of increasing electronegativity, the energy release is coupled to the pumping of $H^+$ into the intermembrane space to generate a proton motive force/proton gradient;
3. As $H^+$ ions flow through the ATP synthase back into the matrix down the proton gradient, ATP is produced from ADP and inorganic phosphate via chemiosmosis;
4. Reoxidation of reduced NAD and FAD allows the regeneration of the coenzyme NAD and FAD, allows them to pick up more protons and electrons from Krebs cycle, link reaction and glycolysis, so that these reactions can continue;

**N13P2Q5**

**(a)** Suggest why the $I^A$ and $I^B$ alleles are dominant over the i allele. [3]

1. The expression of the dominant $I^A$ or $I^B$ allele masks the effect of the recessive i allele;
2. The presence of $I^A$ or $I^B$ allele leads to the production of A antigens/glycoproteins or B antigens/glycoproteins respectively on the surface of the red blood cells;
3. Therefore heterozygous individuals, with $I^Ai$ or $I^Bi$ genotypes, have A antigens or B antigens, respectively, on their red blood cells. This suggests that $I^A$ and $I^B$ alleles are dominant over the i allele;

**(b)**

**(i)** The ABO blood type gene is located on chromosome nine. Suggest where the Rhesus (Rh) factor gene may be found. Explain the reason for your answer. [2]

1. Not found on chromosome 9
   OR
   Found on another chromosome apart from chromosome 9;

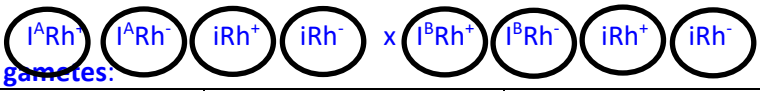
2. As the alleles coding for the Rhesus factor are inherited independently, they are not linked to the alleles of the ABO blood types. Thus, they are not found on the same chromosome;

**(ii)** A type A Rhesus positive mother and a type B Rhesus positive father have a type O Rhesus negative child.

Draw a genetic diagram in the space below to show how this is occurred.

Use the symbols given above and show all the possible genotypes and phenotypes for the offspring of these parents.

**Parental phenotype**: Type A, Rhesus positive x Type B, Rhesus positive
**Parental genotype**: $I^AiRh^+Rh^-$ x $I^BiRh^+Rh^-$
**Meiosis**
**Gametes:**




**Random fusion of gametes:**

| | $I^ARh^+$ | $I^ARh^-$ | $iRh^+$ | $iRh^-$ |
|---|---|---|---|---|
| $I^BRh^+$ | $I^AI^BRh^+Rh^+$<br>Type AB, Rhesus positive | $I^AI^BRh^+Rh^-$<br>Type AB, Rhesus positive | $I^BiRh^+Rh^+$<br>Type B, Rhesus positive | $I^BiRh^+Rh^-$<br>Type B, Rhesus positive |
| $I^BRh^-$ | $I^AI^BRh^+Rh^-$<br>Type AB, Rhesus positive | $I^AI^BRh^-Rh^-$<br>Type AB, Rhesus negative | $I^BiRh^+Rh^-$<br>Type B, Rhesus positive | $I^BiRh^-Rh^-$<br>Type B, Rhesus negative |
| $iRh^+$ | $I^AiRh^+Rh^+$<br>Type A, Rhesus positive | $I^AiRh^+Rh^-$<br>Type A, Rhesus positive | $iiRh^+Rh^+$<br>Type O, Rhesus positive | $iiRh^+Rh^-$<br>Type O, Rhesus positive |
| $iRh^-$ | $I^AiRh^+Rh^-$<br>Type A, Rhesus positive | $I^AiRh^-Rh^-$<br>Type A, Rhesus negative | $iiRh^+Rh^-$<br>Type O, Rhesus positive | $iiRh^-Rh^-$<br>Type O, Rhesus negative |

**Offspring genotype:**

| | $I^AI^BRh^+Rh^+$, $I^AI^BRh^+Rh^-$ | $I^AI^BRh^-Rh^-$ | $I^BiRh^+Rh^+$, $I^BiRh^+Rh^-$ | $I^BiRh^-Rh^-$ | $I^AiRh^+Rh^+$, $I^AiRh^+Rh^-$ | $I^AiRh^-Rh^-$ | $iiRh^+Rh^+$, $iiRh^+Rh^-$ | $iiRh^-Rh^-$ |
|---|---|---|---|---|---|---|---|---|
| **Phenotype** | Type AB, Rhesus positive | Type AB, Rhesus negative | Type B, Rhesus positive | Type B, Rhesus negative | Type A, Rhesus positive | Type A, Rhesus negative | Type O, Rhesus positive | Type O, Rhesus negative |

**Offspring phenotypic ratio:**
Type AB, Rhesus positive : Type AB, Rhesus negative: Type B, Rhesus positive: Type B, Rhesus negative: Type A, Rhesus positive: Type A, Rhesus negative: Type O, Rhesus positive: Type O, Rhesus negative

3:1:3:1:3:1:3:1
Therefore, there is a **1 in 16 chance** of the child having a Type O, Rhesus negative phenotype

[5]

## N13P2Q6

Fig. 6.1 represents the behaviour of one pair of chromosomes during meiosis.

**(a)** Name the structures A, B and C. [3]

A: Homologous chromosomes/ Bivalents
B: Centromere
C: Sister chromatid

**(b)** **(i)** Explain how structure C is similar to D. [2]

1) C & D are non-identical sister chromatids of homologous chromosomes;
2) They have the same sequence of genes (e.g. genes coding of X,Y and Z) at the same loci;
3) They are also joined to their own identical sister chromatid at the same centromere position;

**(ii)** Explain how the structure of C is different from structure D. [2]

1) Structures C & D carry different alleles at the same gene loci (e.g. C carries dominant allele Y while D carries recessive allele y);
2) This is due to each homologue being inherited from each of the parents (i.e. maternal and paternal chromosomes);

**(c)** Describe what is occurring during stage 2 and explain its effects on the products of meiosis. [4]

1) Following synapsis to form bivalents (stage 1), ***crossing over**** occurs between non-identical sister chromatids of homologous chromosomes;
2) During crossing over, bivalents are seen as tetrads;
3) The sites at which non-sister chromatids of homologous chromosomes break and rejoin with each other are known as chiasmata;
4) This process leads to different allelic combinations being found on the sister chromatids, which eventually separate, following meiosis II, to become individual chromosomes in gametes (stage 3);
5) Thus, this increases genetic variation within a population. Allelic combinations which confer advantageous phenotypes to individuals are selected for due to natural selection and increase in frequency over time;

## N13P2Q7

**(a)** Describe the properties of the phospholipid bilayer and the aquaporin channels in relation to the movement of water across the cell surface membrane. [4]

1. Phospholipids have ***hydrophilic phosphate heads**** which face outwards and interact with the aqueous environment of the cell interior or exterior;
2. whilst the ***hydrophobic hydrocarbon tails**** face inwards, away from the water giving rise to a lipid bilayer;
3. Phospholipids have a hydrophobic core region that prevents movement of polar water molecules across the bilayer;
4. Aquaporin channels are protein channels which are made up of amino acids with hydrophobic R groups that are able to interact with the hydrophobic core region of the phospholipid bilayer;
5. The interior of the channel is made up of amino acids with hydrophilic R groups which allow movement of the polar water molecules through the channel pore;

**(b)** Explain what has happened to the treated cells after 3 minutes. [4]

1. The treated cell appeared to increase in size and eventually lyse after 3 minutes as compared to the control cell which did not change in size within the same duration;
2. The genes coding for the aquaporin channels injected into the treated cells were expressed;
3. resulting in more aquaporin channels in the cell surface membrane compared to the control cells;
4. More water molecules were able to enter the treated cells by osmosis causing it to increase in volume and eventually rupture;

**(c)** Outline the differences between osmosis and facilitated diffusion. [2]

1. Osmosis involves the diffusion of water molecules from a region of higher water potential (less negative water potential) to a region of lower water potential (more negative water potential). Facilitated diffusion involves the diffusion of polar (or charged) molecules or ions, unable to diffuse through the hydrophobic core of membrane, from a region of higher concentration to a region of lower concentration;
2. Osmosis involves movement of water molecules through a partially permeable membrane. Facilitated diffusion involves movement of polar (or charged) molecules or ions across the membrane via channel or carrier proteins;

## N13P2Q8

**(a)** Explain the significance to the alpha and beta cells of their blood supply. [3]

1. Alpha and beta cells produce and secrete hormones glucagon and insulin respectively;
2. A good blood supply ensures that the hormones produced by these cells can be secreted directly into the bloodstream;
3. The hormones will also be able to travel quickly to their target organs where they will perform their function; [3]

**(b)** With reference to Fig. 8.2, outline the advantages of such a cell signalling pathway. [3]

1. Facilitate signal amplification
   - only a small number of glucagon molecules needed to solicit a large response from cell
2. Provides multiple checkpoints for regulation
   - Several steps in the signalling pathway can be regulated and controlled. E.g. activation/ inactivation of G protein, activation of adenyl cyclase and amount of cAMP produced. This will eventually regulate the cellular response of the pathway.
3. Multiple responses to 1 glucagon molecule (ligand)
   - 1 glucagon molecule will result in the production of several secondary messengers, e.g. cAMP.
   - cAMP can then trigger multiple signal transduction pathways to elicit different responses;
4. Ensure specificity
   - glucagon has a specific conformation that is complementary to the ligand binding site of GPCR.
   - binding of a specific ligand (glucagon) to a specific receptor (GPCR) will elicit specific reaction via specific pathway in each cell type;

**(c)** Describe how cAMP also increases blood glucose concentration. [3]

1. cAMP is the secondary messenger in the glucagon signalling pathway;
2. Amplification of the signal transduction pathway begins with adenyl cyclase converting many ATP to cAMP.
3. Numerous cAMPs which are small, non-protein water-soluble molecules are able to diffuse quickly throughout the cytosol to
4. Activate many other relay molecules/protein kinase A thus leading to the activation of many enzymes involved in break down glycogen to high glucose production in the liver cells;
5. The glucose molecules are then released into the bloodstream causing the blood glucose concentration to increase;

**(d)** State how the cell signalling pathway for insulin differs from that for glucagon as shown in Fig. 8.2. [1]

The cell signalling pathway for insulin makes use of a receptor tyrosine kinase (RTK) system while that for glucagon makes use of a G-protein coupled receptor (GPCR) system.

**N13P2Q9**

**(a)** Describe the binomial nomenclature of a species and the basis of hierarchical classification of species into taxonomic groups. [7]

1. In binomial nomenclature, each species is assigned a 2-part latin name with the genus followed by the species;
2. e.g. Homo sapiens is the binomial name of humans with Homo being the genus while sapiens is the species. The genus is capitalized while the species is not. Both names are underlined separately or italicized;
3. Linnaeus came up with this precise naming system because it avoids ambiguity that may arise if common names are used instead. e.g. there are many “cats” and there must be a better way to distinguish the different cats;
4. Being systematic, a newly discovered species can be easily categorized and named;
5. Closely related organisms are grouped together in the same taxon, a group of related organisms at a particular level;
6. Traditional Linnean classification determines relatedness based on observable morphological similarities but increasingly, evolutionary relationships are taken into account as well;
7. The organisms are grouped into a hierarchy of increasingly inclusive taxons/categories;
8. Related species are grouped in the same genus, related genera in the same family and so on;
9. The hierarchical taxons are species, genus, family, order, class, phylum, kingdom, domain with the species being the most exclusive group and the domain being the most inclusive group;

**(b)** Explain the biological concept of the species. [7]

1. The biological concept of a species defines a species as is group of organisms capable of interbreeding and producing fertile, viable offspring;
2. A species is thus reproductively isolated from other populations and no longer share a common gene pool with them;
3. The basis of this concept is that two populations have accumulated sufficient genetic differences in both allele frequencies and unique mutations that they develop both prezygotic and postzygotic mechanisms that prevent mating and/or fertilization;
4. This definition cannot be applied to asexually reproducing organisms;
5. This concept cannot be applied to extinct species whose breeding behavior cannot be observed;
6. For ring species, like the salamanders in California, the adjacent subspecies can interbreed but the non-adjacent subspecies can’t. Where is the line drawn?;
7. There are some organisms that may physically and physiologically be capable of mating but, for various reasons, do not normally do so in the wild. e.g. different mating call. It is disputed whether they are indeed a different species;

**(c)** Outline the advantages of molecular methods in classifying organisms. [6]

1. They can be used to compare and categorise species so morphologically indistinguishable due to convergent evolution or are very closely related;
2. On the other hand, remotely related organisms such as bacteria, humans and sunflower can also be compared because they share some proteins such as cytochrome c and all known life is based on nucleic acids. This forms a basis for comparison.;
3. They are objective. Molecular character states are unambiguous as A, C, G and T are easily recognisable and cannot be confused with another whereas some morphological characters, such as those based on the shape of a structure, can be less easy to distinguish because of overlaps between different character states;
4. They are quantitative. Molecular data are easily converted to numerical form and hence are amenable to mathematical and statistical analysis and hence computation. The degree of relatedness can be inferred and quantified by calculating the nucleotide differences between species;
5. Some molecular differences may not be reflected as a difference in the morphological character and hence may not be picked up by morphological analysis. e.g. a nucleotide difference in the third base of a codon, or intron.
6. While small genetic differences, may result in major phenotypic differences. In such instances, vast differences in morphology can exaggerate the evolutionary distance between two species but not in the case of molecular methods. e.g. in snakes, the loss of forelimbs and elongation of the body, both radical changes in body form, are due to mutations in several Hox genes that affect the expression of body patterns and limb formation.
7. Offers a large set of characters to be studied relatively quickly. Each nucleotide position can be considered a character with 4 character states, A, C, G and T, and organisms have millions - billions of nucleotide positions. There are limited morphological characters that can be studied, e.g number of segments, number of legs, shape of thorax etc;
8. Amino acid sequences for many proteins and nucleotide sequences for a rapidly increasing number of genomes can be accessed from electronic databases and used for comparative study and classification. No physical specimen needs to be preserved;
9. Both living and dead tissue may be used so long as the DNA or protein remains intact and you do not even need an entire specimen. This increases the accessibility of studying elusive or rare species;

***Teacher's comments:***

*Students should not simply lift everything from the notes because this is specific to classification and not about establishing phylogenetic relationships.*

**N13P2Q10**

**(a)** Describe how infection by HIV causes disease. [7]

1. The HIV attaches to specific target CD4 cells such as ***T helper lymphocytes/cells**** and macrophages via its gp120 glycoproteins;
2. Upon entering the cell via fusion, ***reverse transcriptase**** will convert the ssRNA genome into ds DNA and with the help of ***integrase**** , integrates the viral DNA into the host genome. Host is now at the latency stage and no symptoms are visible;
3. Integration is random and this may result in the activation of a protooncogene or inactivation of a tumour suppressor gene which may result in cancer (e.g. Karposi sarcoma, a rare skin cancer more prevalent in AIDs victims);
4. During the activation of HIV, synthesis of viral glycoproteins gp 120 and 41 occurs and these will become incorporated into the host cell membrane;
5. allowing the immune system to recognize such cells as infected and become a target of antibodies causing the host cells to lyse;
6. Budding off causes the progressive loss of host plasma membrane and the cells eventually die;
7. Or the host cell transcription and translation mechanism has been taken over by the virus producing viral proteins, compromising the vital functions of the cell and the cell

eventually dies;
8. AVP regarding how infection kills the host cell directly
9. As the target CD4 cells play a central role in the host immune system, destroying these cells will result in host immune system being compromised/suppressed; (connection must be made between CD4 cells and its role in immune system)
10. The host cannot fight off infections from bacteria, viruses and fungi effectively and will suffer from ***secondary/opportunistic infections**** and may even die from them; (effect of being immune compromised)

***Teacher's comments:***

*Students should not focus on the viral life cycle and instead focus on how infection causes disease. This requires students to draw on their knowledge of retroviruses from the topic of gene therapy (pt 3) and make inferences within the topic of viruses to come up with pt 5.Students cannot always expect straightforward "describe" questions.*

**(b)** Explain why viruses may be regarded as non-living organisms. [7]

1. Viruses are ***obligate parasites**** which requires a living host to support many of their functions;
2. They lack the ability to reproduce on their own independently.
3. They do not have a cellular organization such as a cell membrane enclosing cytoplasm with all the various organelles;
4. They do not show metabolic activity outside the host cell. They do not produce their own source of energy but instead acquires it from the host cell and this energy is used to maintain metabolic processes while still inside the host cell;
5. They require host cells to make products such as their coat proteins and their nucleic acids which they can't on their own;
6. They do not grow and undergo developmental changes in its life cycle. Once a virus particle is formed and is released, it is largely inert until a new infection cycle begins.
7. They do not respond to stimuli when outside the host cell. Organisms have specialized receptors that detect environmental stimuli to allow their cells to adjust metabolism in response. Viruses do not.

**(c)** Outline the structure and function of viral nucleic acid. [6]

1. Viral nucleic acid is very diverse. A virus can have either RNA or DNA but never both; (type)
2. and it can be a double or single stranded, circular or linear, single or multiple/segmented; (organization)
3. The viral nucleic acid encodes the genetic information that when transcribed and translated will result in the formation of;
4. Important viral components such as capsid proteins, glycoproteins and viral enzymes;
5. These products are important for the assembly of the viral particles;
6. As well as to allow for the replication of the virus;
7. e.g. RNA dependent RNA polymerase for synthesis of mRNA and genome in influenza, integrase for integration of viral DNA into the host cell genome in HIV, protease to cleave the polyprotein of HIV into functional proteins and enzymes;