RAMAKRISHNA MISSION VIDYAMANDIRA

(Residential Autonomous College affiliated to University of Calcutta)

B.A./B.Sc. SIXTH SEMESTER EXAMINATION, MAY 2019

THIRD YEAR [BATCH 2016-19]

Date : 03/05/2019 **MICROBIOLOGY (Honours)**

Time : 11 am – 1 pm **Paper : VII [Gr-A]** Full Marks : 50

Answer **any five** of the following questions: [5×10]

1. a) How does natural selection work?
   b) What is 'industrial melanism'?
   c) How does directional selection differ from stabilizing selection?
   d) Define genetic drift and genetic shift?
   e) Explain 'descent with modification' according to Darwin's theory.
   f) What is disruptive selection? What do you mean by co-evolution?

   1+1.5+1.5+2+2+(1+1)

2. a) How does UV-irradiation help in lysogenic to lytic switching?
   b) Cro and CI proteins are essential for the conversion between lysogenic to lytic modes – Explain.
   c) What do you mean by temperate phage? 3+4+3

3. a) Explain Hamilton's rule with an example.
   b) What is kin selection?
   c) Define reciprocal altruism.
   d) Explain game theory with the help of 'hawk-dove' model.
   e) 'A male spider allows a female fertilized by him to eat him' - Explain this peculiar behaviour.

   2+2+2+2+2

4. a) The peacock carries its tail as an advertisement of its fitness. Explain.
   b) What is 'Red queen' hypothesis?
   c) Describe different types of speciation.
   d) Write a short note on adaptation. 2+2+4+2

5. a) Name the genes those are involved in Uvr ABC endonuclease repair palhway. Mention the function of those gene products.
   b) How does lexA protein control SOS response?
   c) Design a simple experiment to isolate auxotrophs. 3+3+4

6. a) What is Ts mutant? Illustrate with example.

   b) What is deletion mutant? What is the probable cause of deletion mutation?

   c) Which of the following DNA polymerases has maximum proofreading activity?

      i) DNA polymerase I?

      ii) Taq polymerase

   d) Why is Taq polymerase so named?

   e) "Tobaco is a potent carcinogen"— Explain. 2+(1+2)+1+2+2

7. a) What is IS element? What do you understand by 'composite' transposon?

   b) Mention the functions of integrase and resolvase.

   c) What is photoreactivation? Briefly state the mechanism. (1+2)+2+(2+3)

8. a) By way of transformation a F' *lac*$^{-}$*(lacZ*$^{+}$ *lacY*$^{-}$*)* was incorporated into a *lac*$^{-}$*(lacZ*$^{-}$ *lacY*$^{+}$*) E. coli* cells, but showed no complementation.

   However, continued growth after transfer of a F' *lac*$^{+}$ to gal^{-} (polar) *lacZ*$^{-}$ cells yielded a few cells with gal^{+} *lacZ*$^{-}$(polar) phenotype.

   How can you explain this result?

   b) How does keto-enol tautomerism exert a differential effect on induced mutation if the cells are treated with the mutagen, 5-Bromouracil, a thymin analogue?

   c) Cytosine is spontaneously converted to uracil by means of oxidative deamination and as a result a mismatched base pair is formed. How do the repair enzymes repair the mismatch? 3+4+3

9. a) The ability of certain people to taste a chemical phenylthiocarbamide (PTC) Is governed by a dominant allele **T**, and the inability to taste PTC by its recessive allele **t**. If 24% of a population is homozygous taster and 40% is heterozygous taster, then what is the gene frequency of **t**?

   b) What are meant by dominant gain-of -function and recessive-loss - of function mutation to explain the genetic causes of cancer ?

   c) You intend to change a specific amino acid in a polypeptide chain because the changed polypeptide bears economic importance. This polypeptide is encoded by its corresponding gene in the cell. How can you achieve this?

   d) What are homoeotic genes? Cite examples. 2+2+4+2

10. a) What are the molecular mechanisms by which proto-oncogenes are converted to oncogenes.

    b) "Introduction of hyperactive '*ras*' mutant into cell leads to cancer" - Explain.

    c) How do p53 and RB suppress the progression of tumour? 4+2+(2+2)

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