RAMAKRISHNA MISSION VIDYAMANDIRA

(Residential Autonomous College under University of Calcutta)

B.A./B.Sc. FIFTH SEMESTER EXAMINATION, DECEMBER 2014

THIRD YEAR

Date : 24/12/2014 **MICROBIOLOGY (Honours)**

Time : 11 am – 1 pm **Paper :** V (Gr. B) Full Marks : 50

Answer **any two** questions from **Q.No. 1-4** : [2×7·5]

1. a) What are the modifications present in T4 phage DNA? [2]
   b) How viruses enter the plant cells. [2]
   c) Differentiate between λ – induction and lysogenic conversion. [2]
   d) What is phage typing? [1·5]

2. a) What should be the probability of the bacteria not being attacked by the virus if the MOI is 2? [2]
   b) How the repressor protein in Lambda phage regulates its own synthesis in the lysogenic state? [2·5]
   c) State the role of Q protein in establishing lytic cycle of Lambda phage. [2]
   d) What are helper viruses? [1]

3. a) What will happen when bacteria are infected with—
      i) λCII^- and $\lambda CIII^-$ phage
      ii) Normal CII with mutant $\lambda CIII$. [2]
   b) Design an experiment to prove that Herpes simplex virus carries direct repeats at its ends. [2]
   c) Design an experiment to prove that genes of bacteriophage T_4 are circularly permutated. [2]
   d) What are satellite viruses? [1·5]

4. a) What do you mean by P^{32} suicidal rate? [2]
   b) Explain Frankel and Conrat's experiment to establish that RNA serves as the genetic material of Tobacco Mosaic Virus? [2·5]
   c) How bacteriophage T4 takes control of the host cell? [3]

Answer **any two** questions from **Q.No. 5-8** : [2×7·5]

5. a) Write down the role of *Propionibacter acnes* in the establishment of acne vulgaris? [2·5]
   b) "Each and every person of the population residing in an area does not suffer from the same disease at the same time." Why is it so, explain? [2]
   c) When do the normal flora cause disease? [1]
   d) What is antibiotic associated colitis? [2]

6. a) How can the pathogens avoid the contact with the phagocytes present in the host body? [2]
   b) When does bacteremia progress to septicemia? [1·5]
   c) What all properties should a microrganism have to be a successful pathogen? [2]
   d) Differentiate primary infection from secondary infection. [2]

7. a) Distinguish between exotoxin and endotoxin [2]
   b) How does superantigen cause disease? [1·5]
   c) Explain the difference between toxigenecity, pathogenecity and virulence. [3]
   d) What is toxoid? [1]

8. a) Write down the structure activity of cholera toxin. [3]
   b) Why is diphtheria considered as a toxemia? [2·5]
   c) How does quorum sensing regulate bioluminescence? [2]

Answer **any two** questions from **Q.No. 9-12** : [2×10]

9. a) Immunogens are antigens but all antigens are not immunogens. Justify the statement. [2]

b) I_gM binds antigen more strongly than I_gG though the antigen affinity of I_gM is weaker than I_gG — Explain. [2]

c) A typical antigen in-vivo induces a polyclonal response —Explain [3]

d) How and why do the primary and secondary humoral responses against the same immunogen differ? [3]

10. a) What do you mean by the term "Antigenically Committed"? [2·5]

b) Which types of MHC response does a capsulated virus primarily induce? [3]

c) What is the reason for swelling of lymph nodes in an infection? [2·5]

d) What are cytokines? [2]

11. a) What are the causes that an antibody could be sequenced? [2]

b) What are the effect functions mediated by antibodies? [3]

c) How does an adjuvant stimulate immunity? [3]

d) What is the use of antigen–antibody ternary complex? [2]

12. a) Define monoclonal antibody. How is it prepared? [1+3]

b) Define "Immunoglobulin fold". Where do you find it. [2+1]

c) How does a cytotoxic T cell kill an infected cell? [3]

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