[This question paper contains 6 printed pages.]

999 *Your Roll No.* .....

B.Sc. (Hons.) / III **C**

BIOCHEMISTRY – Paper XV

(Immunology)

(Admissions of 2000 and onwards)

Time : 3 hours *Maximum Marks : 60*

(Write your Roll No. on the top immediately on receipt of this question paper.)

Attempt **five** *questions in all, including Question No.* **1**, *which is compulsory.*

1. (a) Indicate whether each of the following statements is **true or false.** Defend your choice (Be brief).

(i) T-cell epitopes tend to be accessible amino acid residues that can interact with the TCR.

(ii) All lymphoid cells express antigen specific receptors on their membranes.

(iii) Self-/non-self discrimination of the immune system is perfect.

P.T.O.

(iv) MHC genes play a major role in determining the degree of immune responsiveness to an antigen.

(v) The Igα/Igβ heterodimer and CD3 polypeptides serve analogous functions in the BCR and TCR, respectively. (5)

(b) Explain why ?

(i) Only IgG can cross the placental barrier.

(ii) IgM can be detected at low levels along with IgA in mucosal secretions.

(iii) Human skin is resistant to colonization by *E. coli* despite constant exposure to it.

(iv) Naive lymphocytes continuously re circulate through the peripheral lymphoid tissues.

(v) A deficiency of C3 has more severe clinical manifestations than a deficiency in C1 protein.

(vi) All T-cells in the vicinity of an activated T cell do not proliferate in response to IL-2. (9)

(c) Give one significant contribution of each of the following investigators :

(i) Jules Bordet

(ii) A. Tiselius and E.A. Kabat

(iii) W. Dreyer and J. Bennett

(iv) Paul Ehrlich (2)

2. (a) Explain the biological consequences of complement activation. (4)

(b) Name any three proteins that regulate the complement cascade and at what step do they act ? (3)

(c) A single molecule of IgM, in spite of being pentameric fails to activate the classical pathway of complement in the absence of antigen, why ? (2)

(d) The alternate pathway of complement provides an amplification loop to all three pathways of complement activation Explain how ? (2)

3. (a) Explain how hematopoiesis in the bone marrow is induced by tissue injury or local infection. (4)

(b) As adaptive immunity evolved in vertebrates, the more ancient system of innate immunity was retained. Can you think of any disadvantages to having a dual system of immunity ? Would you argue that either system is more beneficial ? (2)

(c) Explain how antigen and naïve lymphocytes enter the lymph nodes. (5)

4. (a) What is the role of the following proteins/enzymes during B cell development ?

(i) Recombination activation genes (RAG-1 & 2)

(ii) λ5 and VpreB.

(iii) Terminal deoxy nucleotidyl transferase (Tdt)

(iv) IL-7 receptor.

(v) Activation induced deaminase (AID).

(vi) Bruton's tyrosine kinase (BtK). (6)

(b) How do T-helper cells find the right B cell to guide their activation ? (5)

5. (a) Mice with the gene for the TCR α-chain knocked out thrive much better than those with a knockout of the CD3 ζ-chain. Explain why ? (2)

(b) What led Zinkernagel and Doherty to conclude that TCR recognition requires both self MHC and antigen molecules ? (2)

(c) Explain how TH_1 cells coordinate the host response to intracellular pathogens. (3)

(d) Compare MHC Class I and class II proteins. (4)

6. (a) What is the source and role(s) of the secretary component associated with sIgA ? (2)

(b) (i) How will you make rabbit antiserum specific for mouse IgG ?

(ii) What type of antibodies will be induced in rabbit when it is injected with human IgG or rabbit IgG ? (3)

(c) Explain why ?

(i) Fewer B cells are lost during light rearrangement than at the stage of heavy chain gene construction.

(ii) Unlike Tc cells, natural killer (NK) cells can kill IgG coated targets.

(iii) A V_H segment cannot join directly to a J_H segment during H chain gene rearrangement. (2×3)

7. (a) Serum sickness can result when an individual is given a large dose of antiserum such as mouse antitoxin to snake venom. How could you take advantage of recent technological advances to produce an antitoxin that would not produce serum sickness in patients who receive it ? (2)

(b) Describe the type II hypersensitivity reaction that can occur in an Rh^+ infant of an Rh^- mother.

OR

Describe the difference between the early and late phases of asthma. (4)

(c) Graft Vs host disease frequently develops after certain types of transplantations.

(i) Briefly outline the mechanisms involved in GVHD.

(ii) Some researchers have found that GVHD can be diminished by prior treatment of the graft with monoclonal antibody plus complement or with monoclonal antibody conjugated with toxins.

List at least two surface antigens to which monoclonal antibodies could be prepared and give the rationale for your choices. (5)

3. Compare the following pairs :

(i) Carrier and adjuvant. (2)

(ii) Immature and mature dendritic cell. (3)

(iii) Tc cell and NK cells. (3)

(iv) Primary and secondary immune response. (3)