T Cell Immunity

Medical Immunology 662
Immunology and Host Defense

Lectures 14-16
24, 27 March, 2006

Questions

What is the genetic process by which antigen-binding receptors are formed?

1. Insertional mutagenesis
2. Gene rearrangement
3. RNA editing
4. Somatic hypermutation
5. RAG recombinase

What are important attributes of Class I and Class II MHC loci and molecules?

1. Peptide binding
2. Polymorphism
3. Selection for non-self peptides
4. Antigen recognition
5. Binding of pathogens

Which classes of MHC molecules are important in T cell recognition processes?

1. Class I
2. Class II
3. Class III
4. Class IV
5. All classes

What does “self” mean in terms of the peptide antigens that T cells recognize?

1. All endogenous peptide antigens
2. Persistently encountered peptides
3. Only the epitopes of empty MHC molecules
4. Only endogenous peptides that bind Class II MHC molecules
5. Endogenous peptides that bind any MHC molecule

Where within their life cycle are T lymphocytes subject to selective cell death?

1. Within the cortex of the thymus
2. In the medulla of the thymus
3. Only within the thymus
4. Only in the periphery
5. In the periphery

What is not an important attribute of Class I and Class II MHC genes and gene products?

1. Peptide binding
2. Polymorphism
3. Recombination suppression
4. Recognition of foreign antigen
5. Multigenism

What is the functionally significant role of CD95 in T cell biology?

1. Attenuation of T cell responsiveness
2. Establishment of central tolerance
3. Immune privilege
4. T cell antigen
5. Cell death of pathogens

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What is the biological significance of superantigen-mediated polyclonal T cell activation for a pathogen?

1. Triggering of potent pathogen-specific immunity
2. Enhancement of pathogen spread
3. Elimination of all T cell responders via peripheral deletion
4. Generation of susceptible targets for infection
5. Escape from innate immunity

T_H1 cells characteristically produce which factor?

1. IL-1
2. IL-2
3. IL-4
4. IL-5
5. IFN-β

T_H2 cells characteristically produce which factor?

1. IL-1
2. IL-2
3. IL-4
4. IL-5
5. IFN-β

Which cells are not cytotoxic?

1. CTL
2. T_H1
3. T_H2
4. Macrophages
5. None of the above

Which molecules are not involved in T cell homing?

1. TCR
2. Integrin
3. LFA
4. VLA-4
5. L-selectin

Which of the following is characteristic of the process of target cell death triggered by CTL?

1. Antibody dependence
2. Osmotic lysis
3. Bystander toxicity
4. Caspase dependence
5. Inflammation

What aspect of T lymphocyte biology is most interesting?

1. Activation
2. Effector function
3. Cell death
4. Antigen recognition
5. Clearance of bacterial pathogens

Complement

Medical Immunology 662
Immunology and Host Defense

Lecture 4
15 March, 2006

Questions

How does a Complementologist count?

a. 1, 2, 3, 4, 5, 6, 7, 8, 9, 10
b. 9, 8, 7, 6, 5, 4, 3, 2, 1
c. 1, 4, 2, 3, 5, 6, 7, 8, 9
d. 1, 4, 2, 5, 3, 6, 9, 8, 7, 10
e. 3, B, 3, 5, 6, 7, 8, 9

What is the MOST critical event in Complement effector function?

1. Activation of $\overline{C1s}$
2. Synthesis of Membrane Attack Complex components
3. Cleavage of $\overline{C4b}$
4. Activation of C3 convertase
5. Inactivation of $C2a$ by Decay Accelerating Factor

Functions of the Complement system include:

1. Participation in inflammatory processes
2. Amplification of the effects of specific antibodies
3. Participation in lysis of bacteria and erythrocytes
4. All of the above
5. None of the above

Which Complement activity is NOT physiologically significant?

1. Lysis of pathogens
2. Triggering of inflammatory responses
3. Lysis of virally-infected host cells
4. Targeting pathogens for phagocytosis
5. Clearing of immune complexes

Which of the following most likely is responsible for the chemotactic accumulation of inflammatory cells that occurs at the sites where immune complexes are deposited?

1. C5a
2. IgA
3. IgD
4. IgE
5. Sensitized lymphocytes

Why might dialysis lead to Complement activation?

1. Proteolytic fragment activation by mechanical shearing
2. Spontaneous activation in concentrated serum
3. Sustained activation due to dialysis of inhibitory factors
4. C3 convertase activation on membrane via thioesterification
5. Activation of C1 by aggregated immune complexes

What is the most likely cause of shock during infection with gram-negative bacteria?

1. Exotoxin
2. Endotoxin
3. Complement fixation and anaphylatoxin release
4. Capsular antigen
5. Flagellar antigen

What is the consequence of a deficiency in C1 Inhibitor?

1. Multiple myeloma
2. Systemic lupus erythematosis
3. Angioneurotic edema
4. Rheumatoid arthritis
5. Paroxysmal nocturnal hemoglobinuria

I will think about Complement:

1. as a profound mode of self / non-self discrimination.
2. as a biological response amplifier.
3. as complementing anticipatory immunity by enhancing immune responsiveness.
4. in its evolutionarily primordial role in self / non-self discrimination.
5. as more interesting than it might at first appear to be.