### Immune response to infections

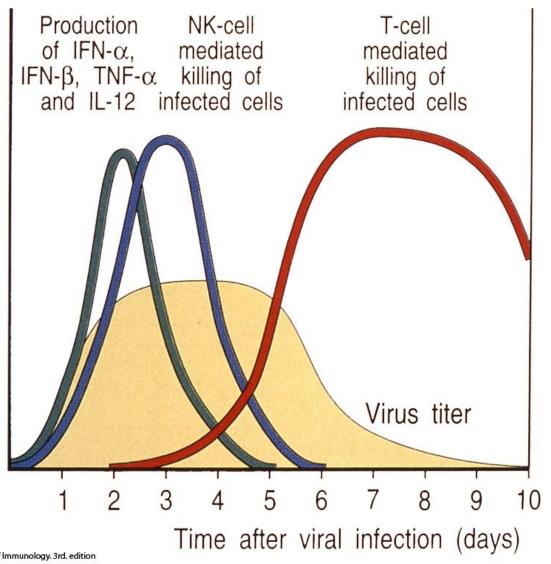
## Factors influencing the extent and severity of infection

- Pathogen factors
  - Dose
  - Virulence of organism
  - Route of entry
- Host factors
  - Integrity of non-specific defences
  - Competence of the immune system
  - Genetic influences
  - Previous exposure to antigen
  - Existence of co-infection

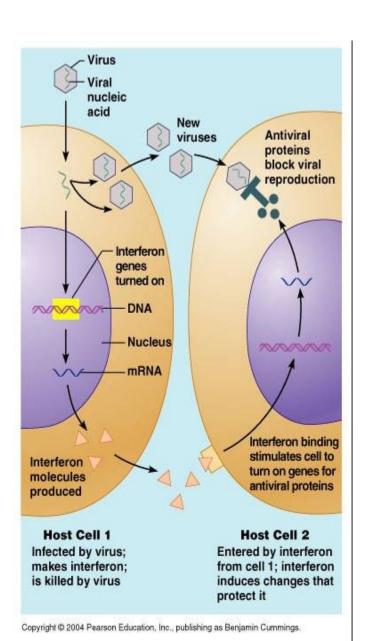
### Immune response to viral infections

- Non-speciphic immunity
  - Interferons (  $\alpha$  and  $\beta$ )
  - Natural killer cells (NK cells)
  - Receptor-like molecules in various secretions
- Speciphic immunity
  - Antibodies neutralization of extracellular viruses
  - Tc lymphocytes elimination of virus-infected cells

#### Mechanisms of antiviral immunity

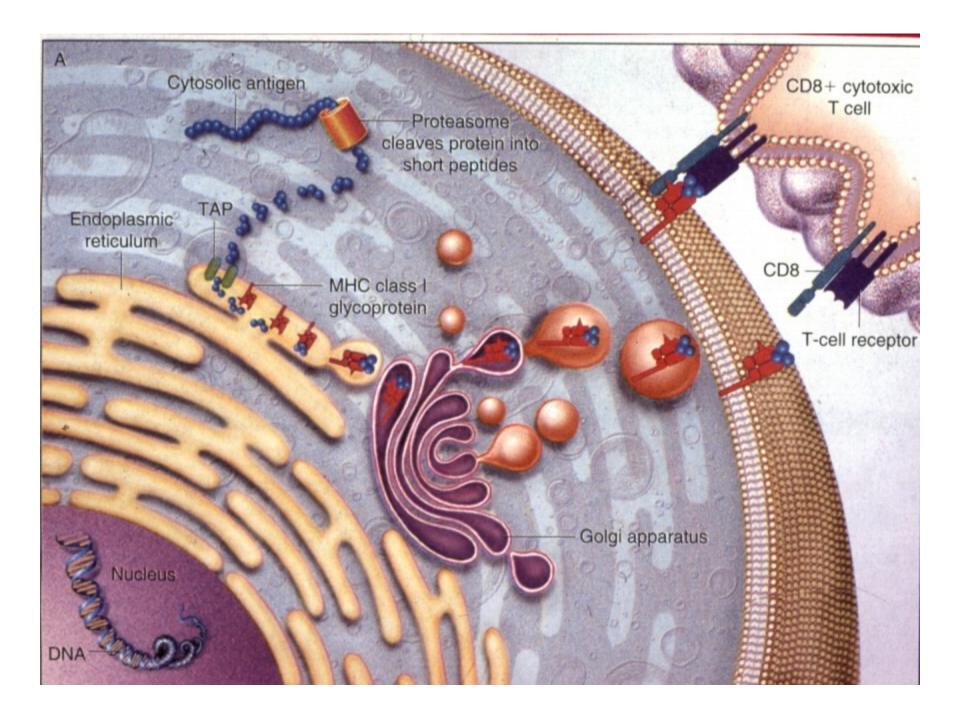


#### The action of interferon (IFN)



#### Natural killers (NK cells)

- Originate in non-T non-B lymphocyte lineage.
- Morphologically: large granulated lymphocytes (LGL).
- Recognition of target cells in antigen nonspeciphic.
- Virus infected and tumor cells are killed.
- Target cells are recognisd mainly by decreased HLA-I expression.
- Cytotoxic mechanisms are similar to Tc cells: perforin and induction of apoptosis.



### Viral strategies to evade the immune response

- Antigenic variations
  - antigenic drift minor changes
  - antigenic shift major changes
- Long-term survival in a host
  - Viral persistence
  - Viral latency
  - Oncogenic transformation
- Immunosuppressive effect of viruses

### Immunosuppressive effects of viruses

- Suppression of T-cells: HIV, morbilli, CMV,
- Inhibition of MHC antigens expression:
  CVM (binds β-2 microglobulin),
  Adenoviruses, RSV decreased expression of HLA antigens
- Production of inhibitory cytokies: EBV (IL-10 - like factor)

### Damage of a host caused by anti-viral immune response

- Autoimmune diseases: hemolytic anemia after EBV infection, autoimmune hepatitis induced by hepatitis-B virus
- Immune complex diseases: arthritis in hepatitis B, vasculitis
- <u>Tc meadited diseases</u>: rash in exanthematic viral diseases, myocarditis caused by coxackie virus

#### Immune responses to bacterial infections

- Non-specific immunity
  - Mechanical barriers
  - Phagocytosis
  - Complement system
- Specific immunity
  - Antibodies opsonisation, complementactivation, neutralisation of toxins, binding to receptors
  - T-lymphocytes against intracellular parasites

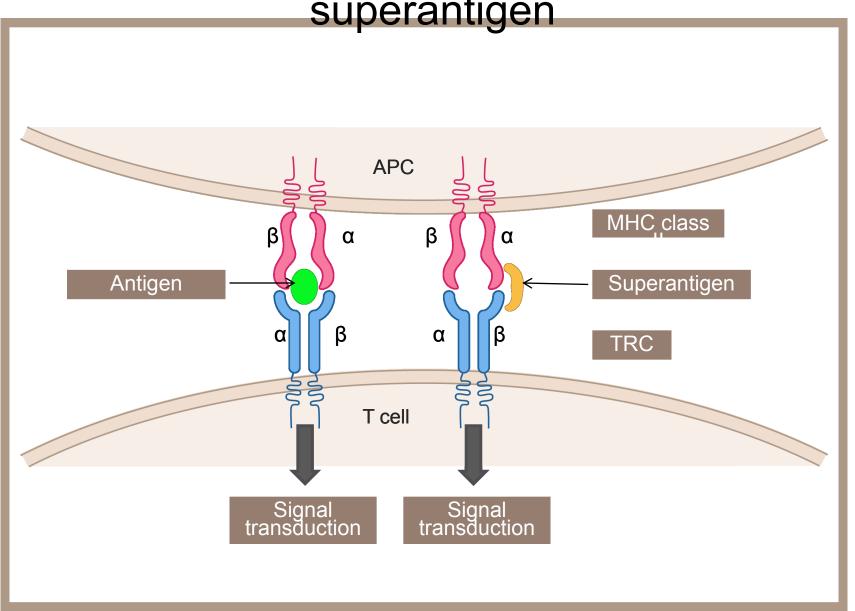
#### Bacterial evasions of immune defences

- Antiphagocytic machanisms: toxins, capsular polysaccharides
- Inhibition of the complement system: Str. pyogenes, E. coli, N. meningitidis
- Antigenic variations: Borrelia recurrentis
- Proteases lysing IgA Neisseria,
  Haemophilus
- Sequestration in avascular regions-Salmonella typhi in the gall bladder and urinary tract
- Intracellular parasitism

#### Macroorganism damage caused by the immune response to bacterial infection

- Autoimmune diseases
  - Cross-reactivity of bacterial and corporal antigens rheumatic fever
  - Type-II hypersensitivity autoimmune hemolytic anemia caused by *Mycoplasma* infection
  - Heat shock proteins
  - Superantigens (streptococcal, staphylococcal)
- Immunocomplex diseases
- Type IV hypersensitivity- cavitatoin in pulmonary tuberculosis

Activation of TCR by antigen and superantigen



# Mechanisms of anti-fungal resistance

- Normal bacterial flora
- Phagocytic cells
- T-lymphocytes -probably most important
- Antibodies usually present, but no protective effect

#### Imunity against parasites

Different mechanisms involved against different parasites.

 Immune response frequently leads to <u>premunity</u> – the situation when the parazite perzists, however it does not lead to dissemination and new infections.

IgE and esinophils play a crucial role.



#### Role of IgE and mastocytes in protection against parasites

