Effector mechanism of humoral immunity

- Behring & Kitasato
- Ab + serum by diphtheria antitoxin serum

Humoral immunity - ultimate result of immune responses (Ab + memory cell)

Target by:
- Extracellular microbe + toxin
- Fungi
- Viruses

Effect of vaccine = Ab response

- Extra Ab
- vs distant site from production.
  - From lymph node, spleen, bone marrow
  - To blood, across mucosa to lumen, Peyer's patch in SI, across placenta (IgG)

Activation:
- Naive B-cell → Plasma cell
- Memory B-cell → Memory plasma cell

Plasma cell derived early (short lived) in B1, S marginal zone
- Late (long lived) for years. Most IgG in serum

- In exposed again (2)
  1. Ab in serum → Immediate response
  2. Memory cell activation

Function of Ab isotype

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<td>Neonatal immunity</td>
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Isotype Switching

1. B-lymphocyte
   - IgM
   - IgG
   - IgA
   - IgE

2. Early plasma cell → late plasma cell

Major stimuli:
- T cell → cytokine
- TH-cell → CD40

Causes:
1. Virus + bacteria (TH1 response, IgG isotype)
2. Helminth (TH2, IgE Ab)

Function of Ab:
1. Neutralization of microbe/sti/s and toxin
2. Opsonization of phagocytosis by receptor
   - Fc gamma receptor
   - Affinity
   - Inhibitory, stimulate NK-cell
   - bind IgG 1/3
   - Induced by IFN-γ

Ab-dependent cell-mediated cytoxicity (ADCC)
- IgG
- NK-cell by bind to Fc (III) → then secrete (IFN-γ + killing molecule)
Ab clearance helminths by $\gamma_2$

- IgE & IgA → coat helminth bind to Fc receptor on eosinophil.

- IgE → little effect.
  - induce mast cell degeneration → mortality GI → bronchoconstriction → clearance.

- Mast cell attract eosinophil by cytokine & chemokine (IL-5)

* Vaccine induced humoral immunity *

- Active immunity (via inflammation)

  - Normal vs. Not normal

  - Vaccine

  - Polio vaccine
  - DTP
  - Hepatitis A & B
  - Hemophilia

* B-cell activation & Ab production *

- In secondary lymphoid organ: Concentrated + B-lymphocyte

  - B naive B-cell
  - Ag binds in (lymph node)

  - Follicular dendritic cell

- Secondary lymphoid organ trap Ag:
  - Blood borne Ag → in spleen.
  - Afferent lymphatic Ag → in lymph node.
  - Mucosal Ag → in MALT → Peyer patch (tonsil adenoid in naso-ph)
protection, protein peptide.

T-cell processing APC

MHC-II

B-cell

Ag

T-cell activation

paracortex in lymph node

periaortic lymph sheath in spleen

B-T collaboration

Thymus-dependent Ag

T-cell

B-cell-T-cell collaboration

Ag

AFC

AFC development

B-cell

cytokine

TH-cell

CD8 + DC

137-1

co-stimulates

B-cell

T-cell

para cortex plasma cell

memory

proliferation, affinity maturation, mutation (somatic hypermutation)

early activation

late activation

plasma cell

class switching

1st dendritic cell

B-cell

Ag

affinity

( + selection)
1. Polyvalent → polysaccharide has multiple epitopes.
   Example: \( \beta(1,2\alpha + 6\beta) \)
   - Epitope → BCR vs. \( \text{Ag} \)
   - Cross linking BCR → Activation of B-cell

2. Alternative pathway (C3b → C3c) → Activation of B-cell by complement receptor on B-cell

- Characteristic of T1-Ag:
  1. Low affinity
  2. X affinity maturation
  3. Limit isotype switching to IgG
  4. High affinity
  5. Most polyvalent

- In B1 & B-margined zone → Macrophage in it is particularly efficient in trapping polysaccharide Ag.

- Secretory Ab
- X spleen → P infection with encapsulated bacteria:
  - H-influenzae, S-pneumonia, meningitis