體內的三道防衛線

1. 體表的障礙：非專一性目標
2. 非專一性的反應（非專一性目標）：發炎反應
3. 免疫反應（專一性目標）
In certain immune responses, complement proteins become activated when they bind to proteins called antibodies (here, the Y-shaped molecules). These antibodies have already become bound to a pathogen.

Complement proteins also are activated when they bind directly to a bacterial surface.

Cascading reactions produce huge numbers of different complement proteins. These become assembled into many molecules, which form many attack complexes.

The attack complexes become inserted into the plasma membrane or lipid envelope of the pathogen. Each forms a large pore through it.

The pores result in lysis, and the pathogen dies as a result of the severe disruption of its structure.
MHC marker designating "self" (present only at the surface of the body's own cells)

T cells and B cells ignore this.

Processed antigen bound to MIC marker at surface of an antigen-presenting cell

T cell recognition initiates immune response.

Antigen (any unprocessed foreign or abnormal molecular configuration that lymphocytes recognize as "nonself")

B cell recognition initiates immune response.
ANTIGEN-PRESENTING CELL
A tumor cell or body cell infected by an intracellular pathogen synthesizes antigen, then displays it as part of antigen-MHC complexes at the cell surface. Cytotoxic T cells recognize antigen synthesized in a body cell:

VIRGIN CYTOTOXIC T CELLS
Responsive virgin T cells that encounter these particular antigen-MHC complexes bind with them and thereby are stimulated to enter mitosis. Repeated cell divisions and differentiations produce large subpopulations of:

EFFECOR CYTOTOXIC T CELLS
Cytotoxic T cell and helper T cell subpopulations set aside for secondary (future) responses

MEMORY T CELLS

EFFECOR HELPER T CELLS
Effector cells secrete interleukins, which stimulate T and B cells to divide and differentiate

MAIN TARGETS: Cells bearing antigen-MHC complexes at surface. These include body cells already infected by intracellular pathogens (such as viruses that stay hidden inside host cell), tumor cells, and cells of organ transplants.
OUTCOME: Target cells are directly and rapidly destroyed through a touch-killing mechanism.

IMMUNE RESPONSES MEDIATED BY CYTOTOXIC CELLS

VIRGIN B CELL
Antigen of extracellular pathogen or freely circulating toxin binds to virgin B cell receptors (i.e., antibody molecules projecting from its surface), moves into cell by endocytosis. Antigen fragments processed, displayed at surface as antigen-MHC complexes:

ANTIGEN-PRESENTING B CELL
Antigen-presenting B cell is activated when a virgin helper T cell binds to its antigen-MHC complexes. The cells disengage. Interleukins from helper T cell and other signals move the B cell to mitosis. Large subpopulations of its descendants differentiate into:

MEMORY B CELLS
Memory B cells set aside for secondary (future) response

EFFECOR B CELLS
Effector B cells secrete:

ANTIBODIES

MAIN TARGETS: Extracellular pathogens and toxins circulating in body tissues.
OUTCOMES: Target is directly inactivated, inflammation is promoted, complement activation contributes to defense activities.

ANTIBODY-MEDIATED IMMUNE RESPONSES
antigen-binding site

variable region of light chain

constant region of light chain

hinge region (flexible)

constant region of heavy chain

antigen on surface of bacterial cell

binding site on one kind of antibody molecule for this specific antigen

antigen on surface of a virus particle

binding site on a different antibody molecule for this specific antigen
a. Gene segments that undergo rearrangement while B cells mature:

b. Segments recombined into finished gene sequence:

c. The finished sequence is transcribed into pre-mRNA:

d. Transcript processing yields mature mRNA transcript (e.g., with introns snipped out, exons spliced together):

e. The mature mRNA is translated into a polypeptide chain (which in this case is the light chain of an antibody molecule):
antigen binds only to those B cells having receptors specific for that antigen

clonal population of B cells

secreted antibodies
First exposure to an antigen provokes a primary immune response:

- virgin B cell or T cell
  - effector cells
  - memory cells

Subsequent exposure to the same antigen provokes a secondary immune response:

- effector cells
- memory cells
The graph illustrates the relative concentration of antibody over time following exposure to an antigen.

- **First exposure to antigen**: A significant increase in antibody concentration occurs, peaking around 5 weeks.
- **Subsequent exposure to the same antigen**: Another increase in antibody concentration is observed, but at a lower level than the first exposure.

The x-axis represents response time in weeks, ranging from 0 to 10, while the y-axis indicates the relative concentration of antibody, with a logarithmic scale ranging from $10^{-1}$ to $10^4$. The graph shows a steady decline in antibody concentration after the peak.

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gp120 (red) at surface of HIV particle

One CD4 receptor (green) projecting above the surface of a helper T cell