Acquired Immunity

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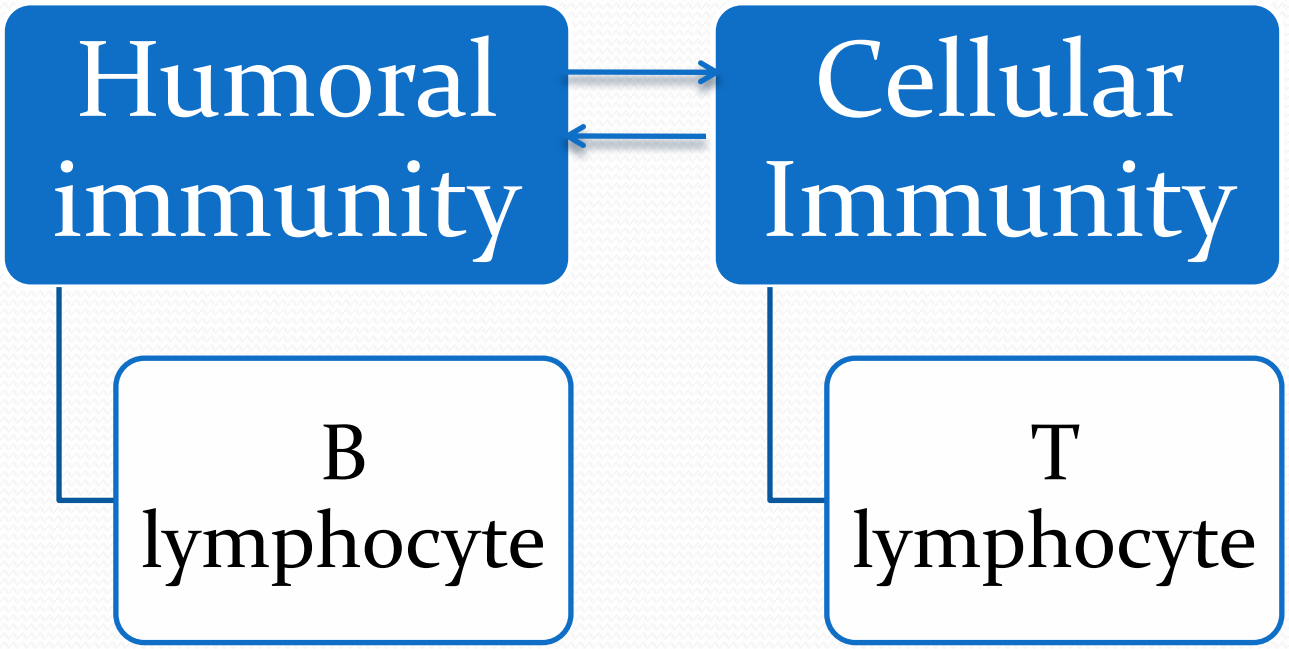
Adaptive (Acquired) Immunity

- Adaptive immunity occurs after exposure to an agent , improved upon repeated exposure
- Specific
- Mediated by antibodies produced by B lymphocyte and by 2 types of T. lymphocyte (T helper,T cytotoxic)
- Have long term memory for specific antigen
- Macrophage and dendritic cell
- Play an important role in both → Innate Immunity
  Adaptive (acquired)

Adaptive (Acquired) Immunity



Features :

- **1. RECOGNITION:** Microbial antigens are recognized by specific T-cell or B- cell receptor .

- **2. SPECIFICITY :** Specific response to each microbe (Humoral or Cellular).

- **3. MEMORY :** Immunological memory is the most important consequence of adaptive immunity .

Humoral immunity

- Humoral immunity is directed primarily against :

1. Exotoxin mediated diseases e.g tetanus
2. Infection in which virulence is related to polysaccharide capsule (pneumococci)
3. Certain viral infection

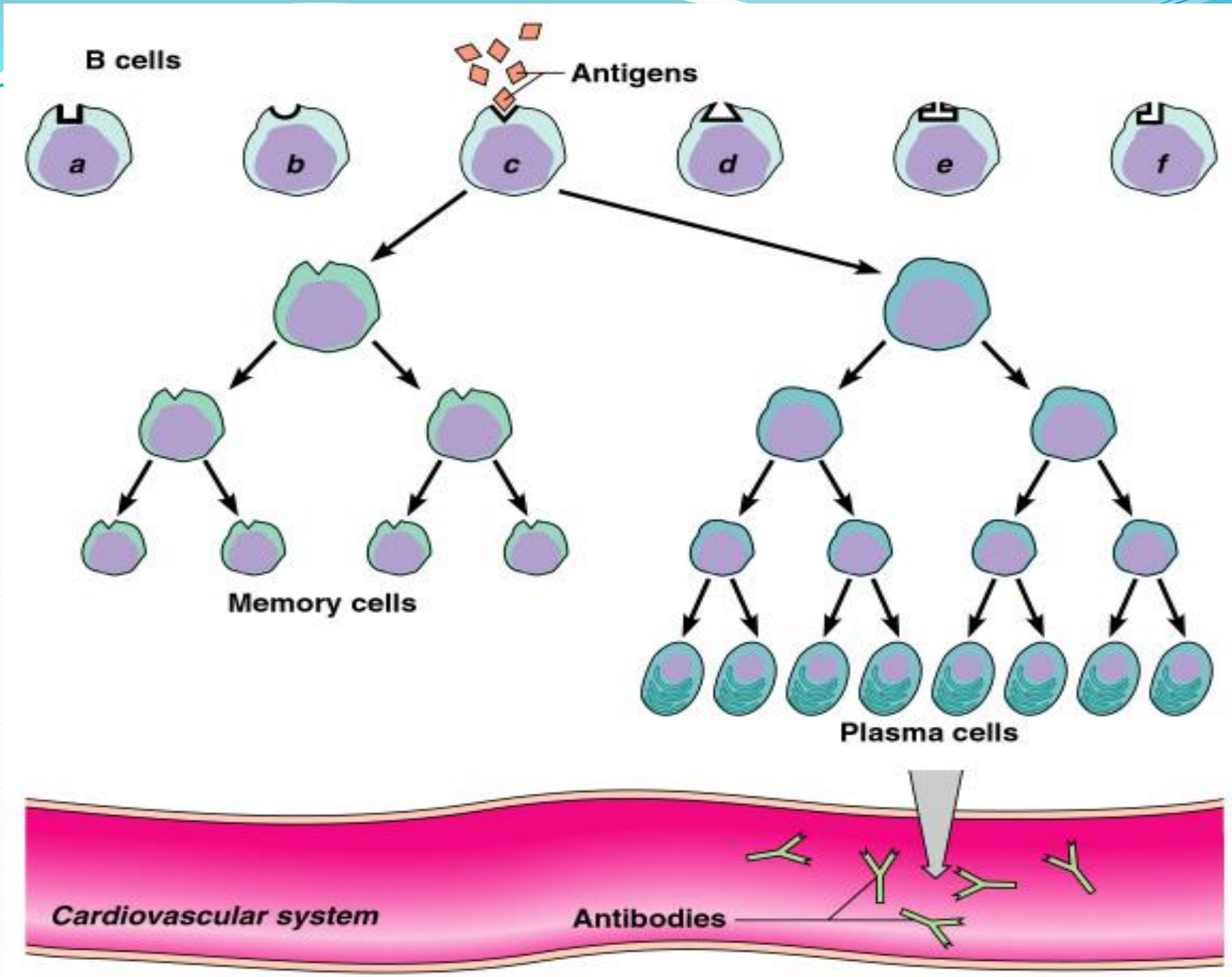
ANTIBODIES MEDIATE :

Humoral immunity .

- (Antibody –mediated immunity)
- (Destroy extracellular pathogens .)

MEMORY B-CELLS.

INDUCE : Secondary immune responses .


B cells
Antigens
a
b
c
d
e
f
Memory cells
Plasma cells
Cardiovascular system
Antibodies

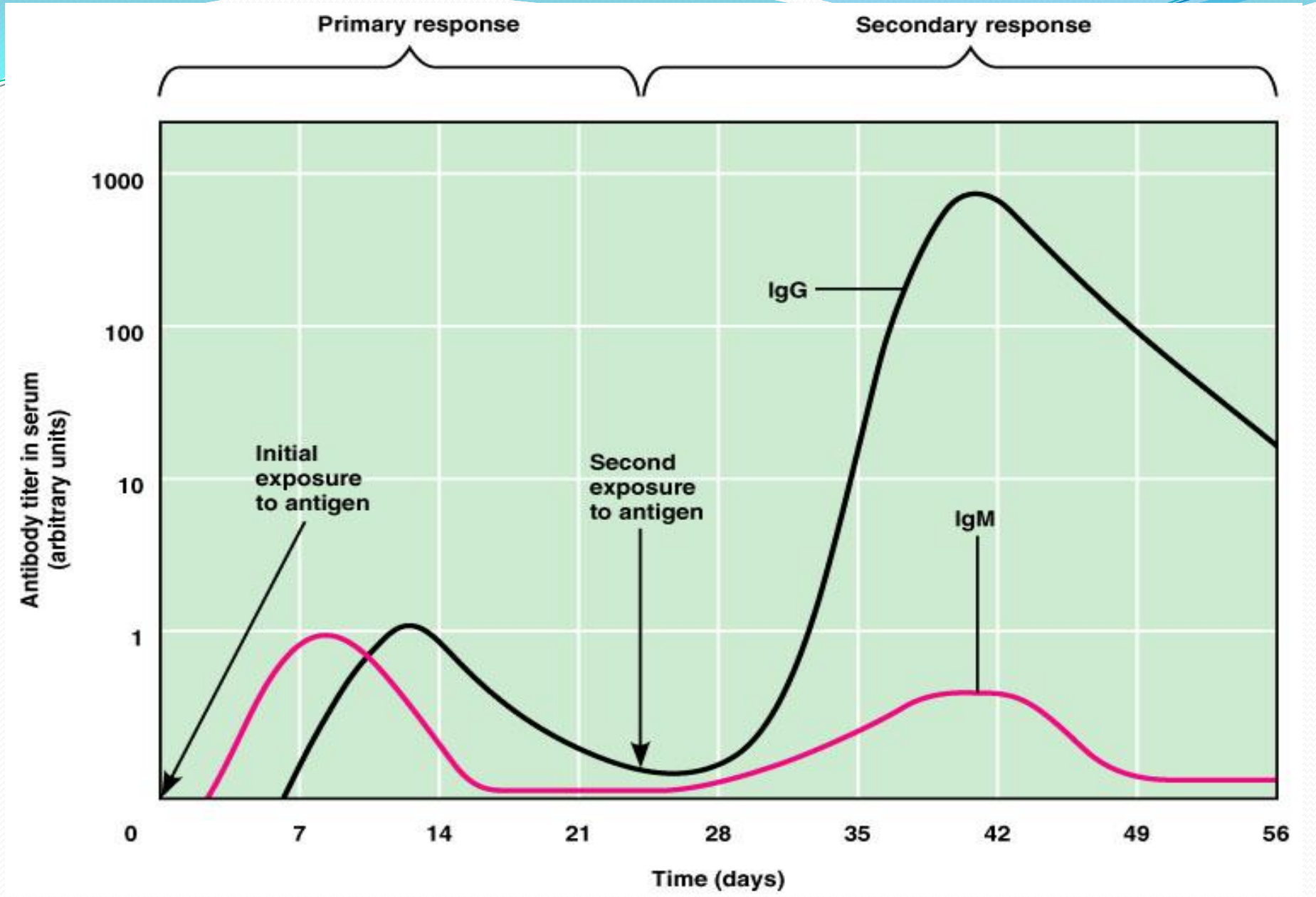
Primary and secondary immune responses:

- FIRST ENCOUNTER WITH A MICROBIAL ANTIGEN GENERATES:
  A PRIMARY IMMUNE RESONSE .

  4 PHASES :

  1. LAG. 3-4 DAYS.
  2. LOG. 4-7 DAYS.
  3. PLATEU. 7-10 DAYS.
  4. DECLINE.

(Primary I.R. may take few days to several weeks ,)


Primary response
Secondary response
1000
100
10
1
Antibody titer in serum (arbitrary units)
IgG
IgM
Initial exposure to antigen
Second exposure to antigen
0
7
14
21
28
35
42
49
56
Time (days)

Features of primary immune responses :

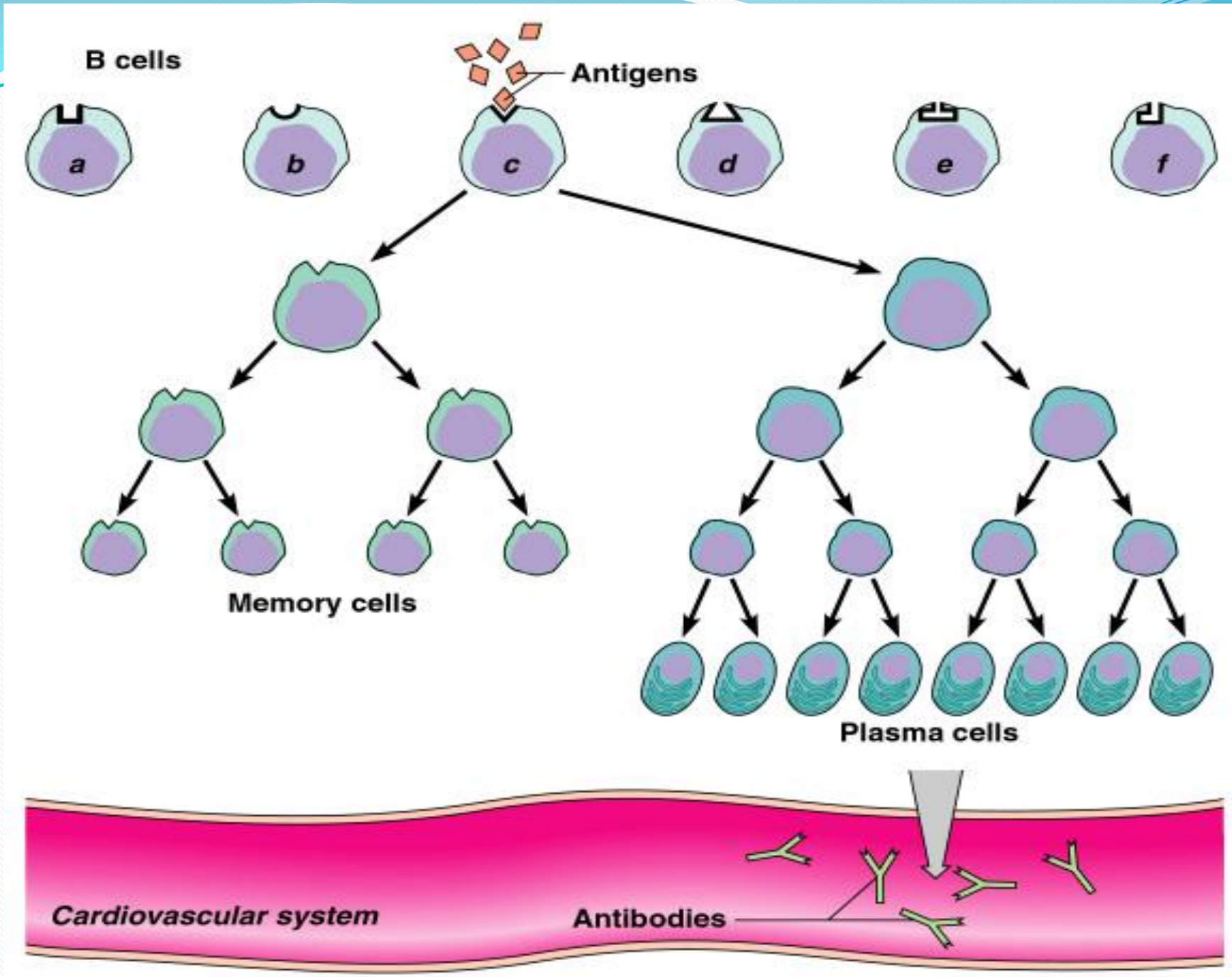
1. Takes longer (4 phases)
2. IgM predominate .
3. Memory cells generated .

ACTIVATED B-CELLS :

- UNDERGO :
  1. Clonal expansion.
  2. Proliferation .
  3. Differentiation into :

(PLASMA CELLS)

* Synthesize and secrete antibodies into the blood .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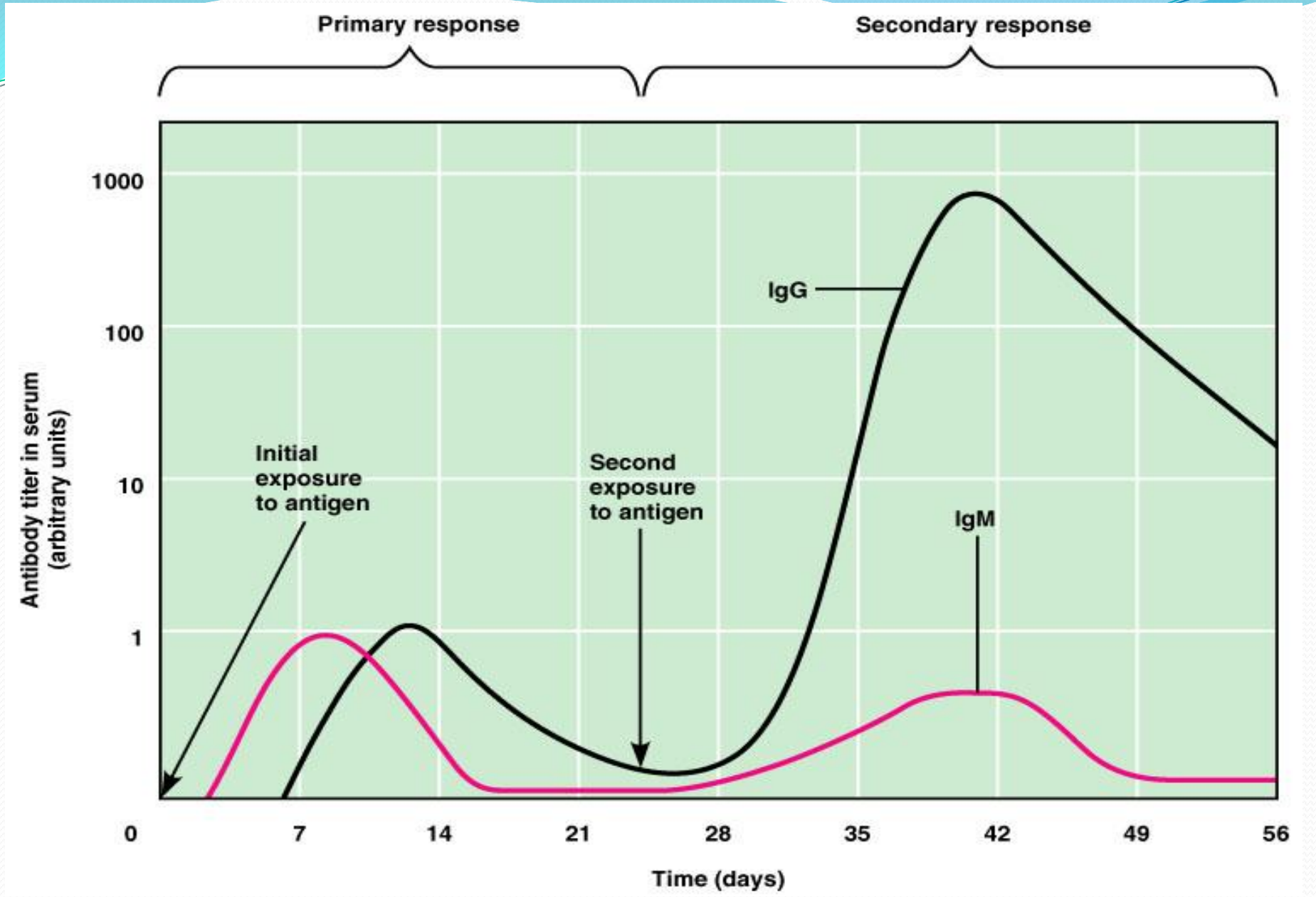
Features of secondary immune responses :

1. Fast response (memory cells)
2. IgG predominate .
3. High concentration of antibody or cells.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FACTORS INFLUENCING I.R.

- **1. Nature of microbial antigen (Epitope)**
  - * T- dependant (TD).
  - * T- independent (TI).
  - * protein , CHO., lipopolysaccarhide, lipid.

2. Dose of antigen.

- high , optimum , low .
  - *Immunological paralysis.

FACTORS cont.

3.Route of entry:

A. Blood-borne antigens – spleen .

B. Skin & tissues – draining lymph nodes.

C. Mucosal surfaces - MALT.

D. Intranasal & inhaled - palatine tonsils & adenoids.

E. Ingested –micro fold (M-cells), Peyers patches.

CONTROL OF IMMUNE RESPONSES.

After control of infections and elimination of the pathogens :

The immune response down regulate and return to a near basal level .

several mechanisms are involved.

CONTROL MEHANISMS.

1. Antigen concentration gradually decrease as the infecting microbe is eliminated .

2. Antibody exert a negative feed -back that switch off responses. (antibody & BCR linked by immune- complexes that contain the relevant antigen .)

3. Antibodies bind and form idiotypic networks.

4.Cytokine - mediated regulation.

5. Interaction of the immune system with endocrine and nervous system . This involve cytokines , hormones and neurotransmitters.

HELPER T- CELLS :

1. Synthesize a membrane bound molecule (CD 40 ligand)

   * Bind on CD -40 on B-cells .

2. Secrete B – cell stimulatory factors:

   IL-4 , IL-5 , IL-6 .

   * These cytokines act on receptors on the B-cell & the cell become activated .

T dependent (TD) & T independent (TI) Antigens

- **Naïve mature B - cells are free to leave BM & migrate to the periphery**

- **If they do not encounter Ag, they die within few weeks by apoptosis**

- **If however , encounter the specific Ag , they undergo activation, proliferation & differentiation leading to generation of plasma cells (AFCs) & memory B - cells (Bm)**

- **Immune response to most Ags depends on T & B cells recognizing Ags in a linked fashion which is called T- dependent (TD) antigens . However , a small number of Ags can activate B cells without MHC class II restricted T - cell help & they are referred as T- independent (TI) antigens that include [*Lipopolysaccharides (LPS) , Ficoll & Dextran*] and characterized by induction of poor memory response**

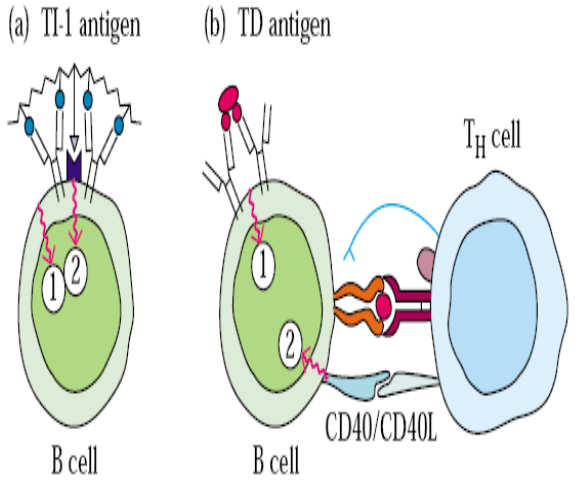

FIGURE 11-6 An effective signal for B-cell activation involves two distinct signals induced by membrane events. Binding of a type 1 thymus-independent (TI-1) antigen to a B cell provides both signals. A thymus-dependent (TD) antigen provides signal 1 by crosslinking mIg, but a separate interaction between CD40 on the B cell and CD40L on an activated T_H cell is required to generate signal 2.