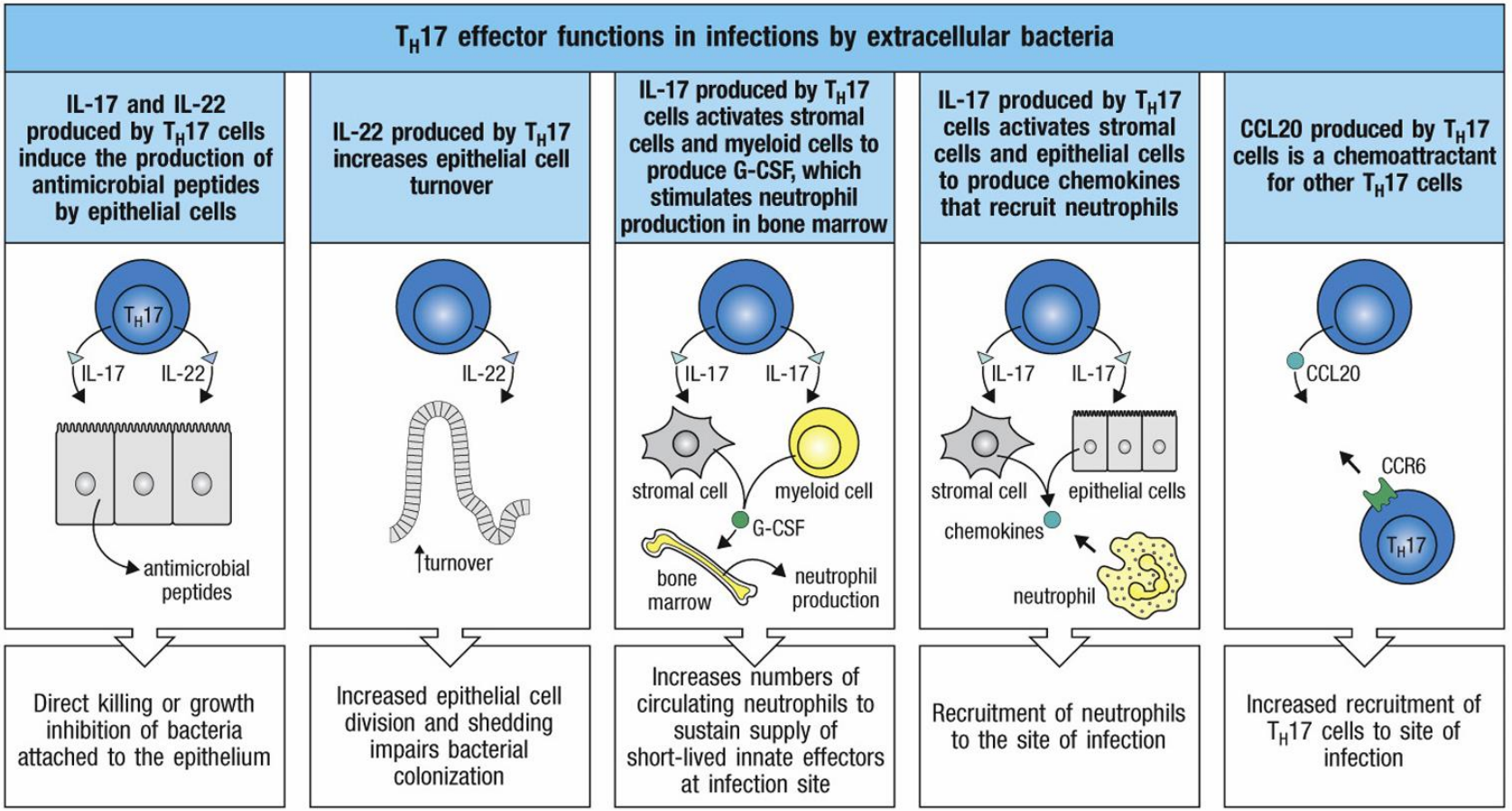
Which cell type is the primary driver of adaptive immunity to extracellular bacteria?

- A) Th1
- B) Th17
- C) Th2
- D) CTLs
- E) NK cells

Answer

- Correct answer: B) Th17



What is the main function of Yersinia pestis YOPs?

A) Form membrane pores
B) Block complement deposition
C) Inhibit phagocytosis by disrupting the host actin cytoskeleton
D) Destroy IgA
E) Activate neutrophils

Answer

Correct answer: C) Inhibit phagocytosis by disrupting the host actin cytoskeleton

Explanation: YOPs are injected via a type III secretion system and inhibit actin rearrangement, preventing phagocytosis

Which cytokine produced by Th17 cells promotes epithelial repair and antimicrobial peptide production?

A) IL-11
B) IL-17F
C) IL-22
D) IL-4
E) IL-12

Answer

- Correct answer: C) IL-22
- Explanation: IL-22 alongside IL-17 targets epithelial cells, enhancing regeneration and antimicrobial activity.

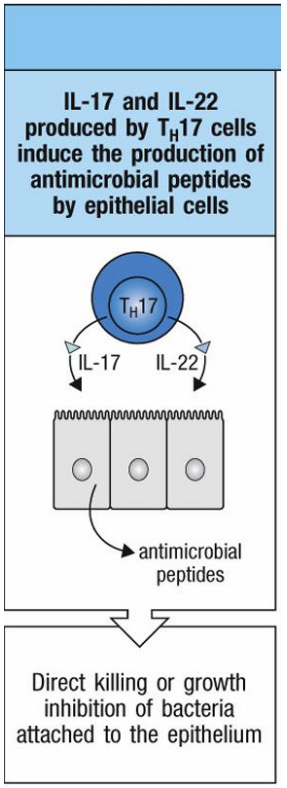


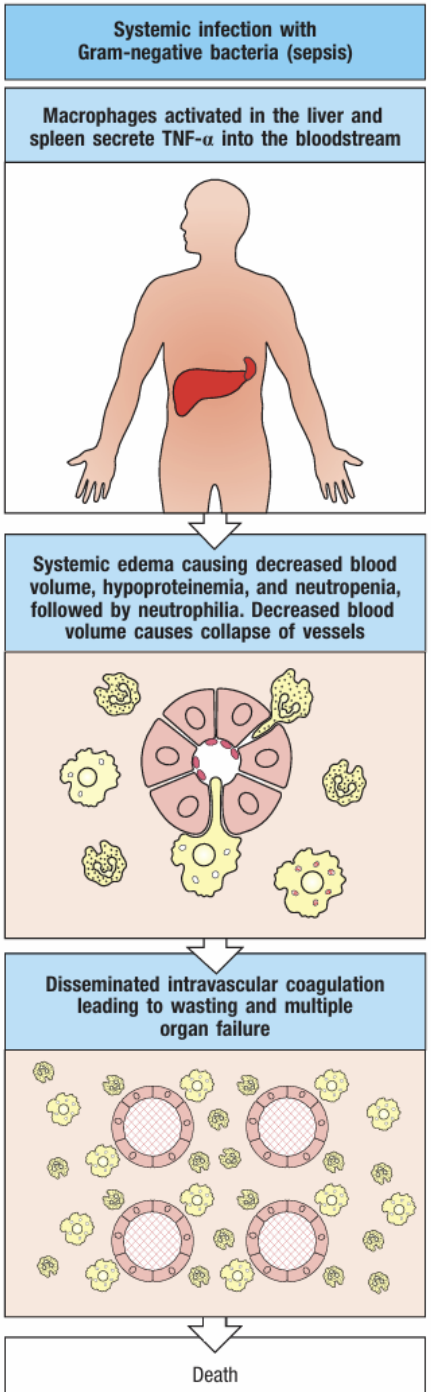
Figure 11.16 Janeway's Immunobiolog

What is sepsis?

- A) Localized bacterial infection
- B) Uncontrolled antiviral cytokine release
- C) Fungal dissemination
- D) Autoimmune tissue destruction
- E) viable bacteria are found in blood

Answer

- Correct answer: E) viable bacteria are found in blood.



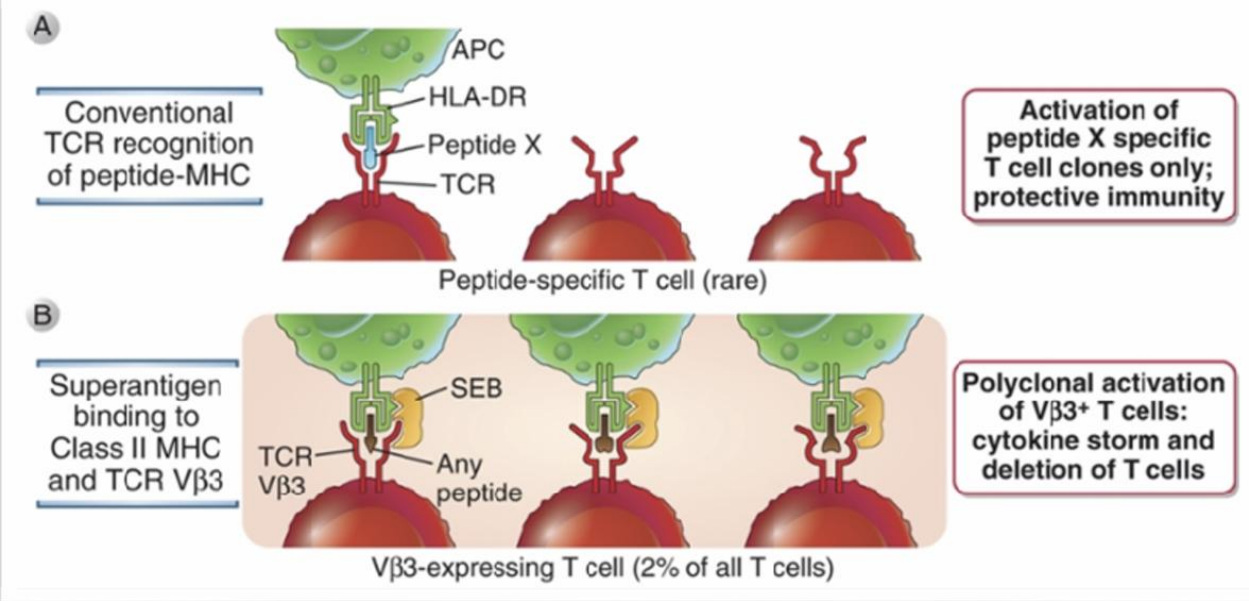
Superantigens activate T cells by:

A) Binding only to CD8
B) Crosslinking MHC II and TCR Vβ region independently of peptide
C) Requiring antigen processing
D) Binding only high-affinity TCRs
E) Inducing receptor internalization

Answer

Correct Answer: B) Crosslinking MHC II and TCR Vβ region independently of peptide

Explanation: Superantigens are bacterial toxins that bind to TCR and also II MHC molecules but not to the peptide-binding cleft, thus activating many more T cell clones than conventional antigens

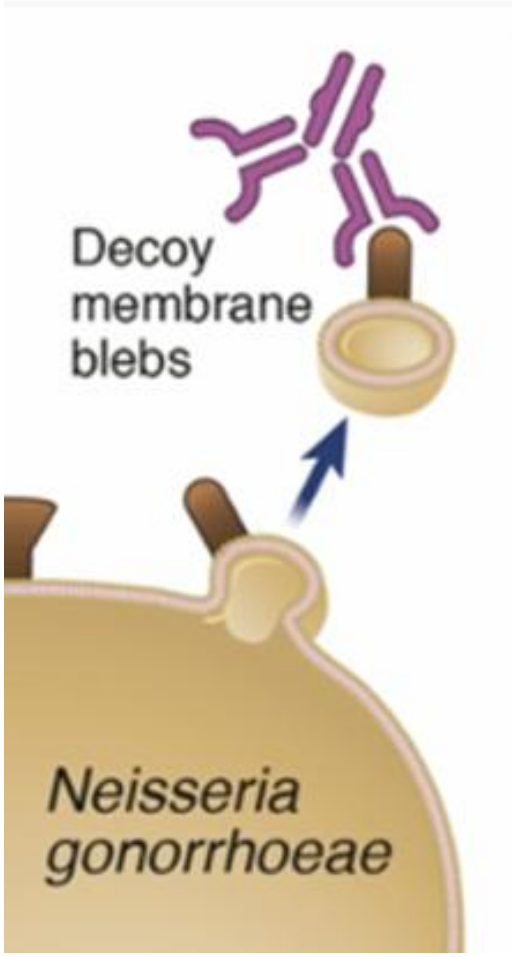


What is a “decoy bleb”?

- A) Viral inclusion body
  B) Dendritic cell pseudopod
  C) Host-derived apoptotic vesicle
  D) Bacterial membrane vesicle that absorbs antibodies/complement
  E) Macrophage phagosome

Answer

- Correct answer: D) Bacterial membrane vesicle that absorbs antibodies/complement
- Explanation: Some bacteria release membrane “blebs” that mimic their surface. These vesicles bind antibodies and complement, diverting immune attack away from the real bacterium and helping it evade clearance.

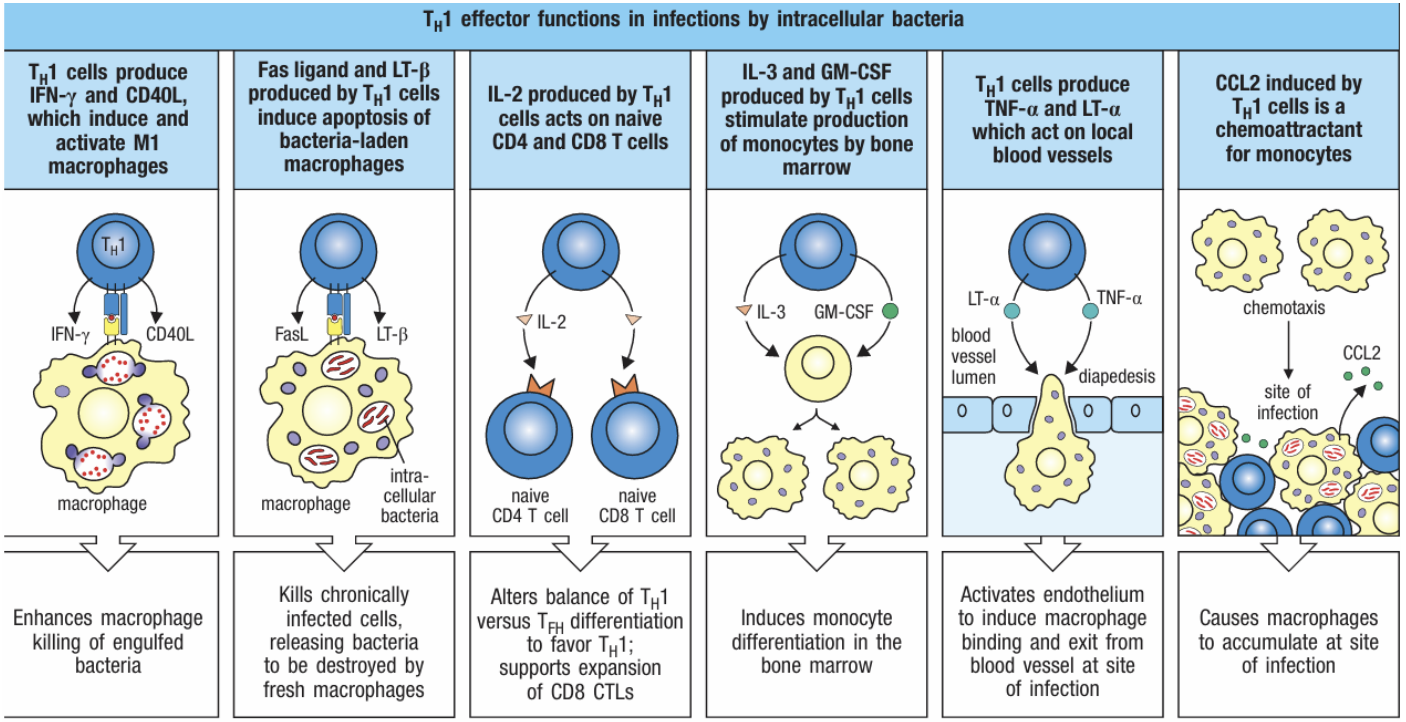


Immunity to intracellular bacteria is primarily mediated by:

- A) Antibodies
- B) Th2 cells
- C) Th1 and activated macrophages
- D) Th17 cells
- E) Eosinophils

Answer

- Correct answer: C) Th1 and activated macrophages

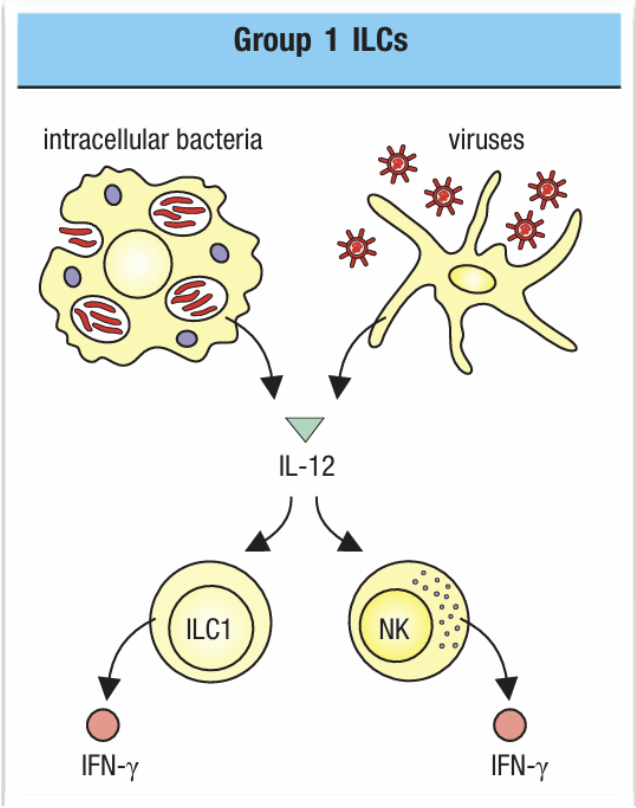


What is a major early role of NK cells in intracellular bacterial infection?

A) Produce IL-4
B) Produce IFN-γ to activate macrophages
C) Kill neutrophils
D) Promote antibody class switching
E) Block complement activation

Answer

- Correct answer: B) Produce IFN-γ to activate macrophages

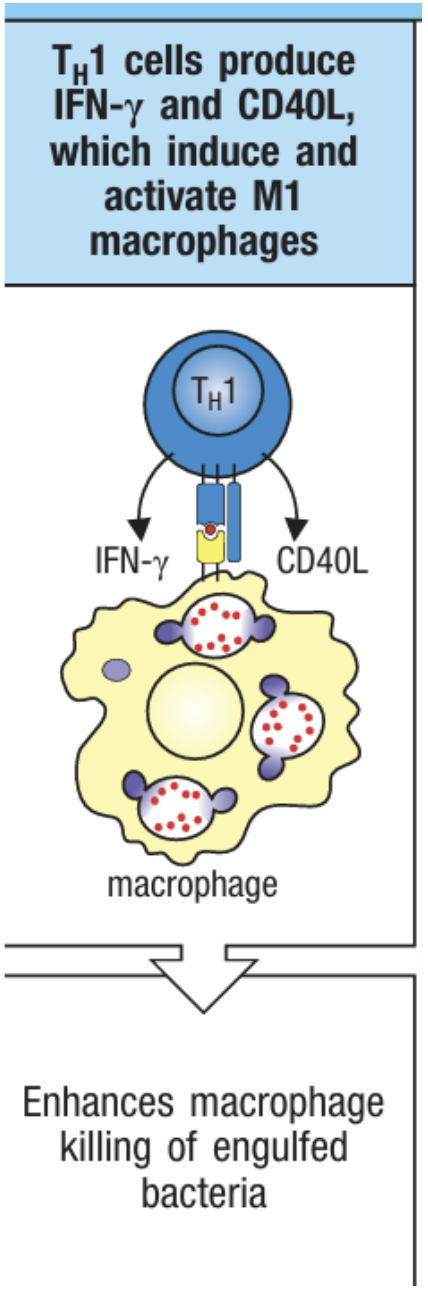


What is the role of CD40–CD40L in intracellular bacterial immunity?

- A) Activates neutrophils
- B) promotes sepsis
- C) Inhibits phagolysosomal fusion
- D) Generates IgE
- E) Activates macrophages via Th1 cell help

Answer

- Correct answer: E) Activates macrophages via T cell help
- Explanation: CD40L on Th1 cells binds CD40 on macrophages, enabling full activation.

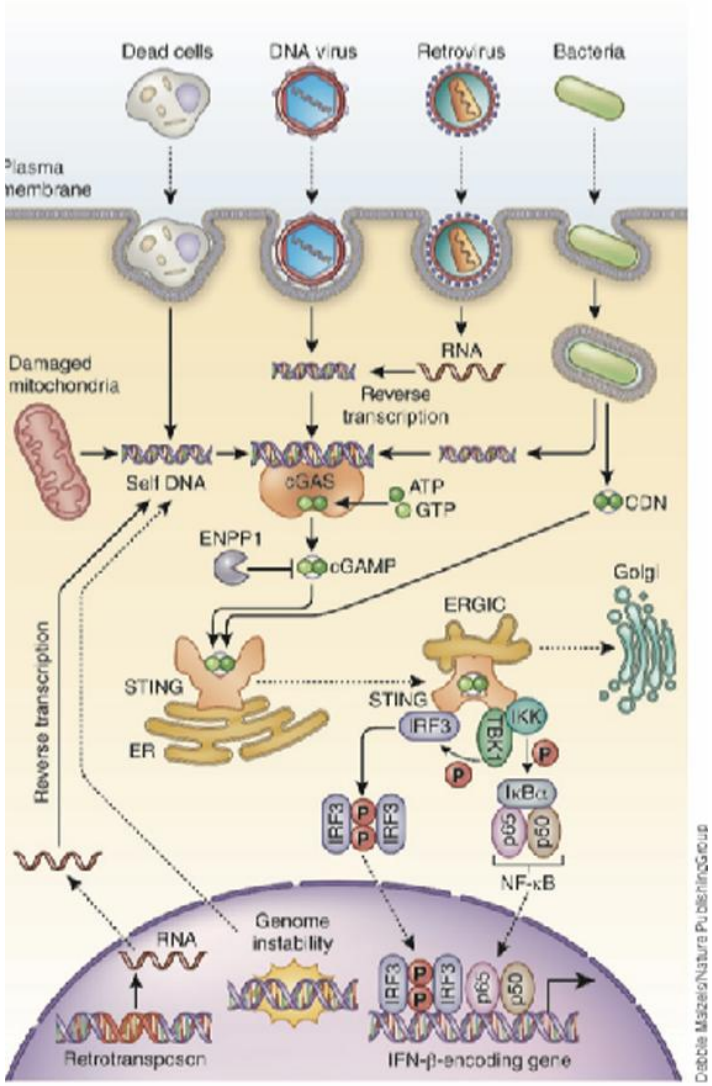


What does cGAS detect?

- A) Bacterial lipoproteins
- B) Cytosolic dsDNA
- C) Viral RNA in endosomes
- D) Complement fragments
- E) Fungal mannan

Answer

- Correct answer: B) Cytosolic dsDNA
- Explanation: cGAS senses cytosolic double-stranded DNA and produces cGAMP, which activates STING.
  STING signaling then activates IRF3, leading to the production of type I interferon (IFN-β).

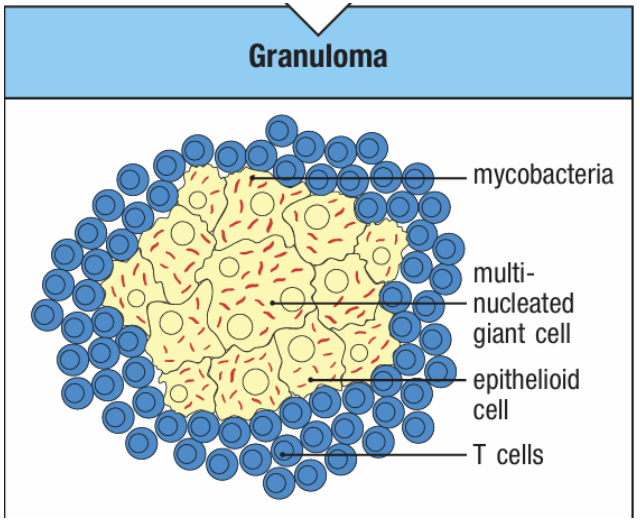


Granulomas form because:

- A) Macrophages fail to recognize pathogens
  B) Microbes die too quickly
  C) Persistent intracellular pathogens cannot be eradicated
  D) Excess complement activation
  E) Mast cell chronic activation

Answer

- Correct answer: C) Persistent intracellular pathogens cannot be eradicated
- Explanation: Granulomas develop when macrophages cannot eliminate persistent intracellular microbes.
  To control the infection, the immune system walls off the pathogen with activated macrophages and T cells, forming a contained, organized structure.



IFN-λ is primarily important for immunity at which location?

- A) Mucosal epithelium
- B) Liver
- C) Bone Marrow
- D) CNS
- E) Spleen

Answer

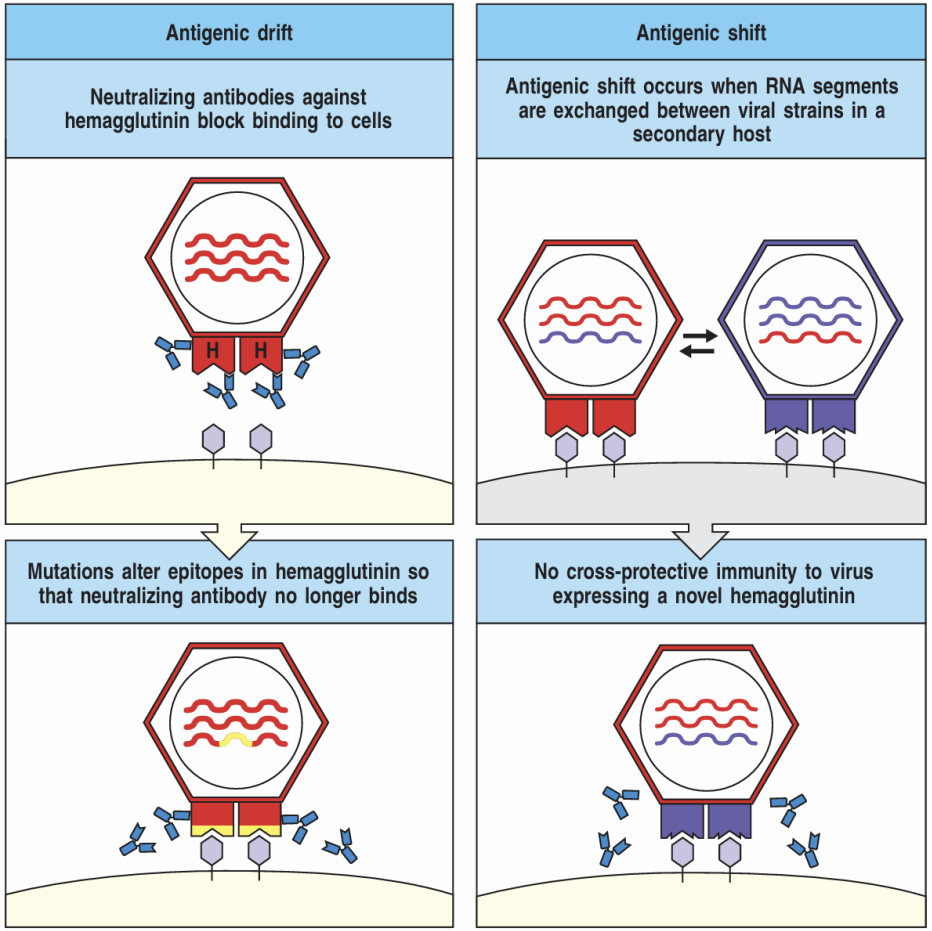
- Correct answer: A) Mucosal epithelium
- Explanation: IFN-λ mediates critical antiviral effects - non redundant to type I IFNs - at mucosal epithelial barriers

Antigenic drift is caused by:

A) RNA recombination between two viruses
B) Accumulation of point mutations in viral surface proteins
C) Switching host species
D) Integration into the host genome
E) Changes in Ig gene expression

Answer

- Correct answer: B) Accumulation of point mutations in viral surface proteins

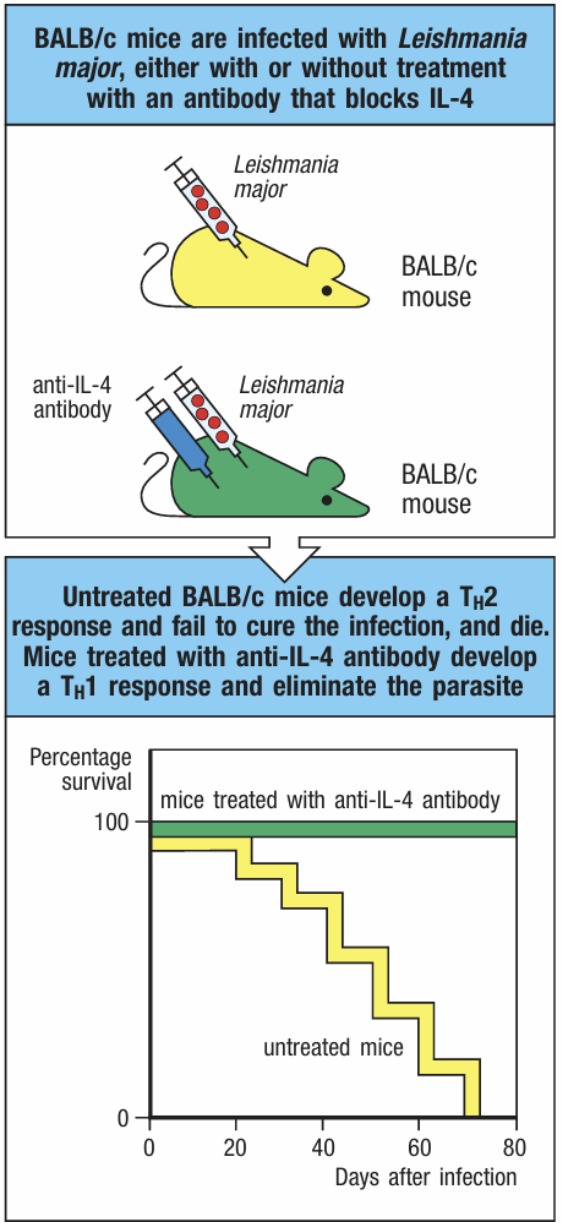


Administration of anti-IL-4 antibody in BALB/c mice infected with *Leishmania major* causes:

- A) Increased susceptibility
  B) Complement inhibition
  C) Loss of granuloma formation
  D) Eosinophil depletion
  E) Shift toward a protective Th1 response

Answer

- Correct answer: E) Shift toward a protective Th1 response
- Explanation: Blocking IL-4 stops the Th2 response in BALB/c mice and allows a protective Th1 (IFN-γ) response to develop, improving control of *Leishmania major*.



Which cytokine from Th2 cells promotes mucus production and epithelial repair in helminth infection?

- A) IL-17
- B) IL-22
- C) IL-21
- D) IL-13
- E) TNF

Answer

- Correct answer: D) IL-13
- Explanation: IL-13 increases mucus, smooth muscle contraction, and epithelial turnover for parasite expulsion.

What structure protects helminths against host immunity?

- A) Tegument or cyst wall
- B) Peptidoglycan
- C) LPS
- D) Capsule
- E) Flagella

Answer

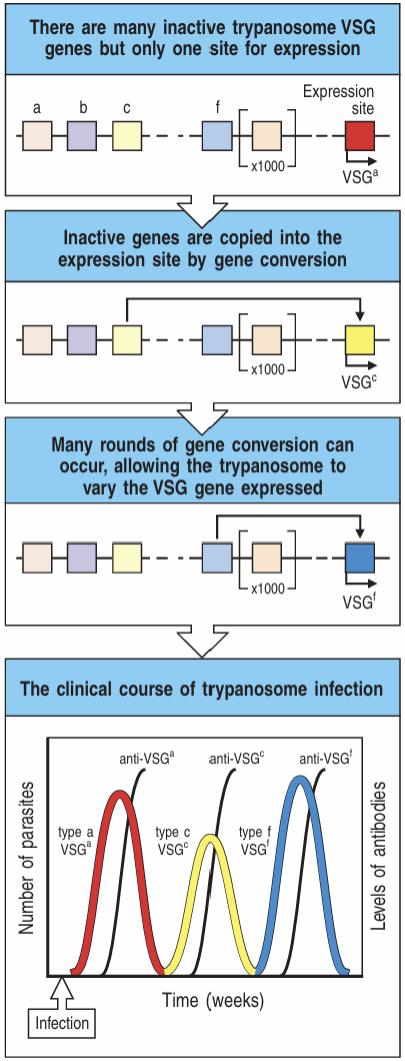
- Correct answer: C) Tegument or cyst wall
- Explanation: Thick external layers shield helminths from immune attack.

Antigenic variation in Trypanosoma brucei occurs through changes in:

- A) VSG surface glycoproteins
- B) Capsular polysaccharides
- C) Flagellar structure
- D) Ribosomal RNA
- E) MHC-like molecules

Answer

- Correct answer: A) VSG surface glycoproteins
- Explanation: Trypanosomes switch VSG genes to evade antibody responses.



Immunological tolerance refers to:

- A) Immunosuppression of all lymphocytes
  B) Global inhibition of cytokine production
  C) Inability of naïve T cells to leave the thymus
  D) The failure of B cells to undergo class switching
  E) Unresponsiveness to an antigen induced by prior exposure

Answer

- Correct answer: E) Unresponsiveness to an antigen induced by prior exposure
- Explanation: Tolerance is antigen-specific unresponsiveness induced by prior exposure; not a global shutdown like immunosuppression.

Central tolerance occurs:

- A) In peripheral tissues
  B) During germinal center reactions
  C) Only after infection
  D) During lymphocyte development in thymus/bone marrow
  E) During antigen presentation in lymph nodes

Answer

Correct answer: D) During lymphocyte development in thymus/bone marrow
Explanation: Central tolerance eliminates or edits self-reactive T and B cells during development.

Peripheral tolerance mainly involves:

A) Negative selection in thymus
B) Receptor editing
C) Anergy, suppression, and apoptosis in mature lymphocytes
D) Somatic hypermutation
E) Only Treg activation

Answer

- Correct answer: C) Anergy, suppression, and apoptosis in mature lymphocytes
- Explanation: Peripheral tolerance prevents self-reactive mature lymphocytes from causing damage by inducing anergy, Treg-mediated suppression, or apoptosis..

AIRE is:

A) A transcriptional regulator that enhances chromatin accessibility for antigen presentation
B) A guidance molecule directing developing T cells toward the medulla
C) A co-regulator involved in B7–CD28 costimulatory signaling
D) A cytokine-induced nuclear factor released during thymocyte maturation
E) A transcription factor enabling thymic expression of tissue-restricted antigens

Answer

- Correct answer: E) A transcription factor enabling thymic expression of tissue-restricted antigens
- Explanation: AIRE is expressed in medullary thymic epithelial cells and drives the expression of peripheral tissue antigens in the thymus. This exposes developing T cells to a wide range of self-antigens, allowing deletion of strongly self-reactive clones and ensuring central tolerance.

APS-1 results from:

A) PD-1 deficiency
B) AIRE mutations
C) CTLA-4 overexpression
D) IgA deficiency
E) Loss of TGF-β

Answer

- Correct answer: B) B) AIRE mutations
- Explanation: AIRE deficiency impairs central tolerance, leading to autoimmune polyendocrine syndrome type 1 (APS-1), a rare disorder characterized by autoimmune attack against multiple endocrine organs, most commonly causing hypoparathyroidism, adrenal insufficiency, and chronic mucocutaneous candidiasis.

T cell anergy occurs when a T cell recognizes antigen:

- A) with strong costimulation
- B) in presence of IL-2
- C) during infection
- D) without costimulation
- E) only in lymph nodes

Answer

- Correct answer: D) without costimulation
- Explanation: Antigen recognition without costimulation induces long-lived functional unresponsiveness.

Which inhibitory receptor competes with CD28 for B7 with much higher affinity?

- A) PD-1
- B) LAG-3
- C) CTLA-4
- D) TIM-3
- E) OX40

Answer

- Correct answer: C) CTLA-4
- Explanation: CTLA-4 binds B7 molecules 10–20× more strongly than CD28, limiting T cell activation.

PD-1 activation leads to:

- A) Inactivation of T cells
- B) Enhanced TCR signaling
- C) Increased IL-2 production
- D) Increased co-stimulation
- E) B cell receptor editing

Answer

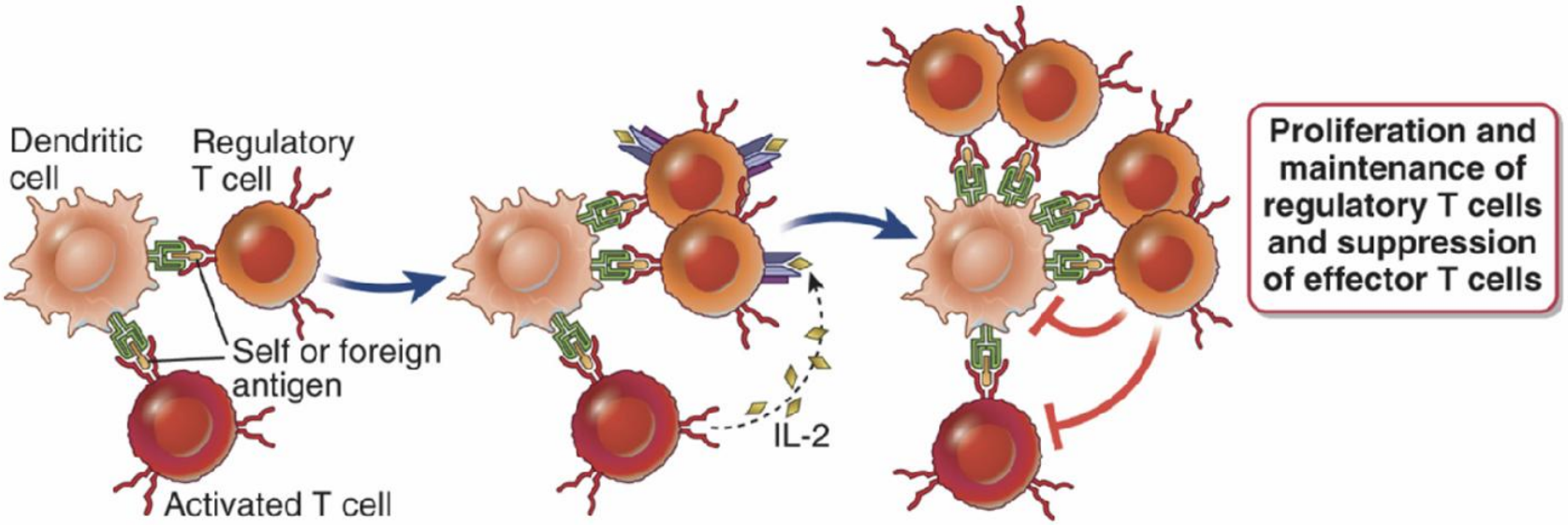
- Correct answer: A) Inactivation of T cells
- Explanation: PD-1 inhibits TCR signaling and dampens T cell responses.

Treg generation critically depends on:

A) IL-4
B) IL-10
C) IL-12
D) IL-1β
E) IL-2

Answer

- Correct answer: E)IL-2
- Explanation: IL-2 is essential for Treg development, expansion, and survival.



Treg cells are characterized by expression of:

A) CD40 and RORγt
B) CD25 and FoxP3
C) T-bet and CD8
D) PD-1 and CD28
E) CD19 and CD20

Answer

- Correct answer: B) CD25 and FoxP3
- Explanation: Regulatory T cells characteristically express CD25, the high-affinity IL-2 receptor α-chain, which allows them to efficiently capture IL-2 needed for their survival.
  They also express FoxP3, a transcription factor essential for their development and suppressive function.

Which cytokine produced by Tregs inhibits macrophage and dendritic cell activation?

- A) IL-12
- B) IL-6
- C) IL-10
- D) IL-17
- E) TNF

Answer

- Correct answer: C) IL-10
- Explanation: IL-10 suppresses DC and macrophage activation and limits inflammation.

TGF-β contributes to tolerance through:

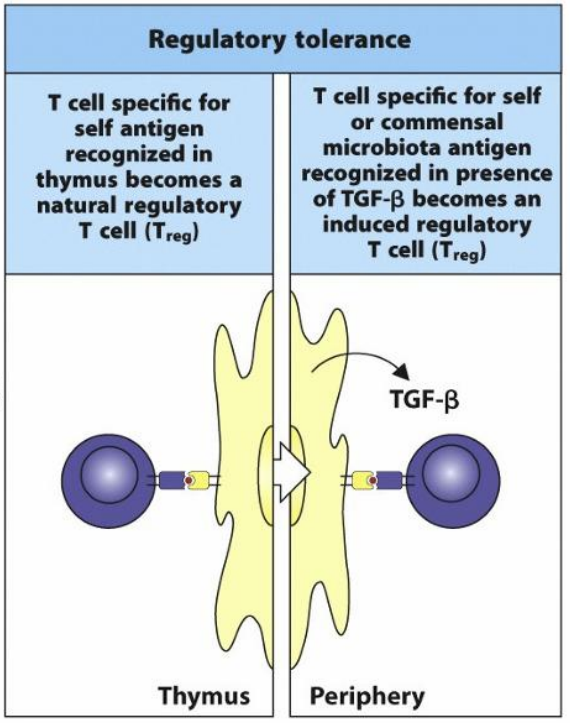
- A) Promoting Th1 cell differentiation
- B) Enhancing B7 expression
- C) Inhibiting T cell proliferation and promoting Treg generation
- D) Activating NK cells
- E) Inducing IL-2 secretion

Answer

- Correct answer: C) Inhibiting T cell proliferation and promoting Treg generation

<u>Inhibitory cytokines produced by Tregs:</u>

- TGF-β
  - inhibits the proliferation of T cells and the activation of macrophages
  - regulates the differentiation of induced Treg cells and Th17 cells
  - promotes tissue repair

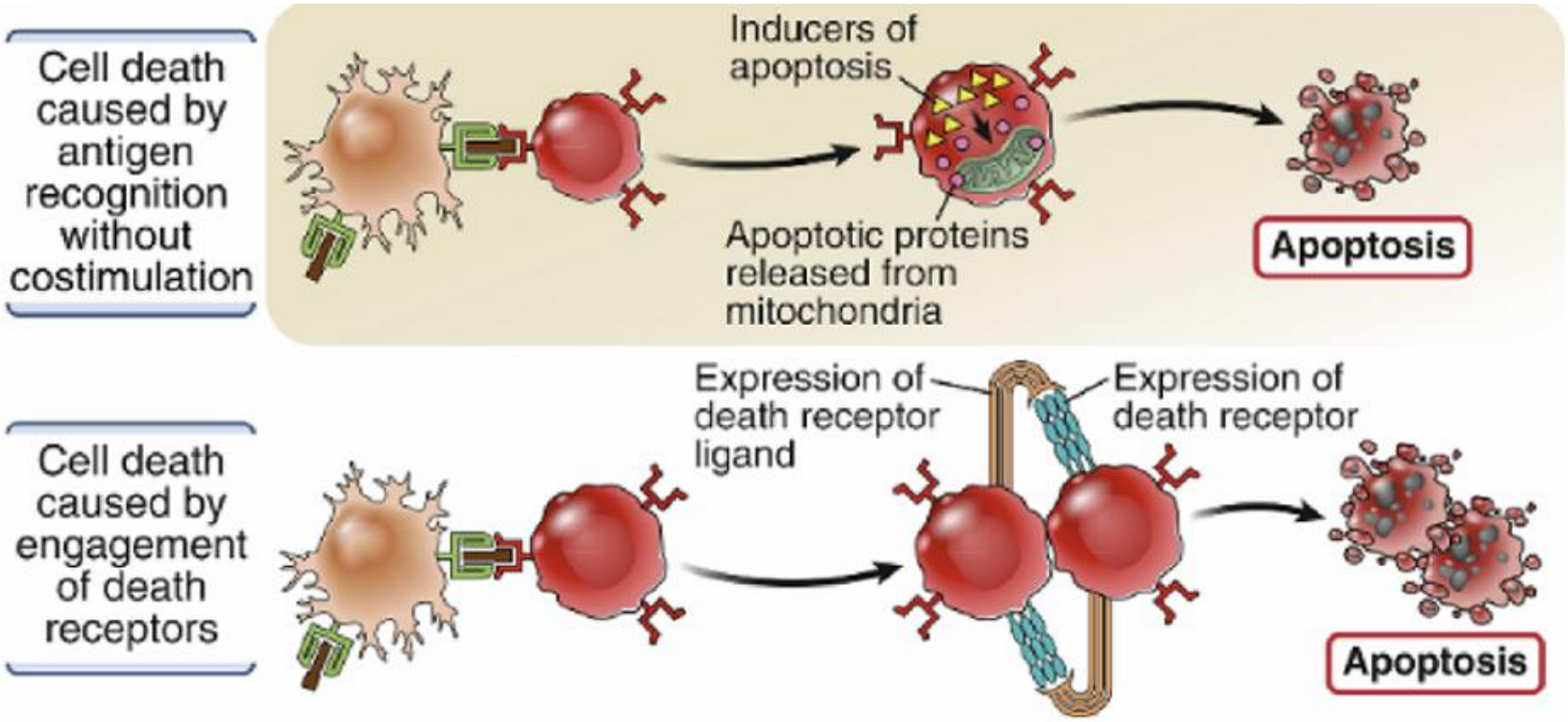


Apoptosis of self-reactive T cells in peripheral tolerance is often triggered by:

A) Lack of IL-10
B) Fas–FasL interactions
C) BCR signaling
D) IgE crosslinking
E) Increased IL-4

Answer

- Correct answer: B) Fas–FasL interactions



The strongest genetic link to autoimmune disease is due to:

A) TLR polymorphisms
B) Variants in cytokine receptors
C) MHC (HLA) alleles that influence which self-peptides are presented
D) IgE allotypes
E) Fc receptor mutations

Answer

- Correct answer: C) MHC (HLA) alleles that influence which self-peptides are presented

Hypotheses for explaining the MHC allele associations:

- Differences of antigen-presenting capability of MHC molecules
- Differences in shaping TCR repertoire (e.g., poor binding of certain autoantigens in the thymus)
- Induction of negative selection of pathogenic T cells for alleles that are protective
- Promoting of Treg development for protective MHC alleles

An environmental trigger for autoimmunity often involves:

- A) UV light in SLE
- B) Excess vitamin D
- C) High IL-10 levels
- D) Increased NK activity
- E) Removal of commensal microbes

Answer

- Correct answer: A) UV light in SLE
- Explanation: UV light increases apoptotic cell debris, exposing nuclear antigens that drive autoantibody formation in SLE.

Infections can promote autoimmunity by:

A) Blocking antigen presentation
B) Increasing IgE
C) Blocking MHC expression
D) Decreasing IL-1
E) Activating APCs and inducing costimulation (bystander activation)

Answer

- Correct answer: E) Activating APCs and inducing costimulation (bystander activation)
- Explanation:Infections activate APCs through innate signals, increasing costimulatory molecules and cytokines.
  This inflammatory environment can inadvertently activate nearby self-reactive T cells, even if they are not specific for the pathogen.
  This is called bystander activation, and it can initiate or worsen autoimmunity.

Molecular mimicry refers to:

- A) T cells mimicking B cells
  B) Self-antigens resembling microbial antigens
  C) NK cells mimicking cytokines
  D) Pathogens blocking MHC
  E) Antibodies mimicking complemen

Answer

- Correct answer: B) Self-antigens resembling microbial antigens
- Explanation: Molecular mimicry occurs when microbial antigens resemble host antigens. Immune responses generated against the pathogen can then cross-react with similar self antigens, leading to autoimmune tissue damage.