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Item 12 of 26 Question Id: 20817

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Immunoglobulins (antibodies) are the principal component of the humoral immune system. They are effector proteins that bind to specific **epitopes of antigens** based on the unique group of 110-130 amino acids present in the **hypervariable region** of the immunoglobulin light and heavy chain. Because individual immunoglobulins can identify molecular targets with a high degree of specificity, immunoglobulin therapy (**immunotherapy**) has been developed to target specific ligands, cytokines, receptors, growth factors, and other proteins that contribute to the pathogenesis or progression of inflammatory and neoplastic conditions.

Immunotherapy is **monoclonal** because all the immunoglobulin components in the medication have the same hypervariable region (produced from the same B-cell clone). However, most immunotherapy regimens use a **fragment** of the immunoglobulin with 1 valence (binding) site rather than the full immunoglobulin with 2 valence sites, because fragments are significantly **smaller** than the full immunoglobulin, which improves **tissue/tumor penetration** and medication pharmacokinetics.

Common types of immunoglobulin fragments include the following:

- **Antigen binding fragments** (Fab) contain a variable domain and the first constant region of a heavy and light chain. Because Fab fragments do not contain an Fc region, they do not activate complement or trigger phagocytosis via the Fc receptor on macrophages (**Choices C and D**). Therefore, Fab fragments generally are not used in applications that require cell death (eg, cancer immunotherapy).
- Single-chain variable fragments (scFv) contain a light chain and heavy chain variable region linked together by a peptide.
- Single-domain antibody (sdAb) has only a light chain variable region or a heavy chain variable region.

(Choice A) Antibody fragments pass through the glomerular basement membrane into the collecting system more easily than full immunoglobulins due to their small size; therefore, antibody fragments typically are excreted

penetration and medication pharmacokinetics.

Common types of immunoglobulin fragments include the following:

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- Single-domain antibody (sdAb) has only a light chain variable region or a heavy chain variable region.

(Choice A) Antibody fragments pass through the glomerular basement membrane into the collecting system more easily than full immunoglobulins due to their small size; therefore, antibody fragments typically are excreted more (not less) quickly.

(Choice E) Because the hypervariable region in a Fab fragment is the same as that in the full immunoglobulin, the fragment and full immunoglobulin bind the antigen with an equivalent affinity.

Educational objective:

Immunotherapy medications often utilize fragments of a monoclonal immunoglobulin rather than the full immunoglobulin; because fragments are smaller, they typically have better tissue penetration and pharmacokinetics. Fab fragments contain a variable domain and the first constant region from a heavy and light chain; because they do not contain an Fc receptor, Fab fragments cannot trigger cell killing via complement or phagocytosis.



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A 37-year-old man comes to the emergency department because of increasing pain and tenderness in his right forearm. During a bar brawl 6 days earlier, he sustained a 4-cm laceration through the skin and subcutaneous tissue of his forearm. Treatment at the time of injury included cleaning and dressing the wound. Physical examination shows erythema surrounding the wound site and expression of yellow pus when pressure is applied adjacent to the wound. Which of the following molecules is most likely responsible for causing accumulation of pus over this patient's wound?

- A. Bradykinin (1%)
- B. C3a (15%)
- C. IL-3 (3%)
- D. IL-8 (65%)
- E. IL-10 (5%)
- F. Leukotriene C₄ (8%)

Omitted
Correct answer
D

65%
Answered correctly

02 secs
Time Spent

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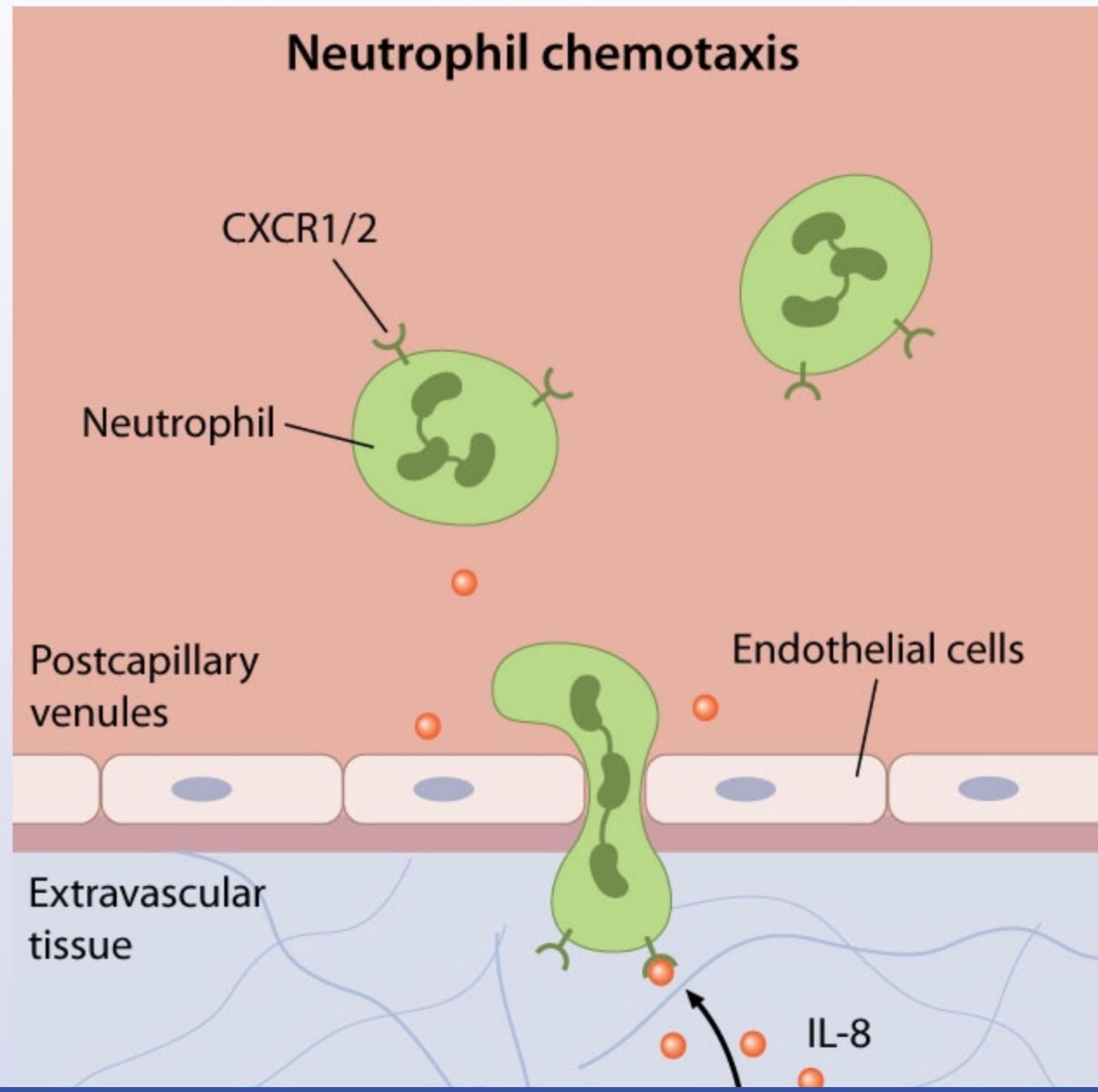
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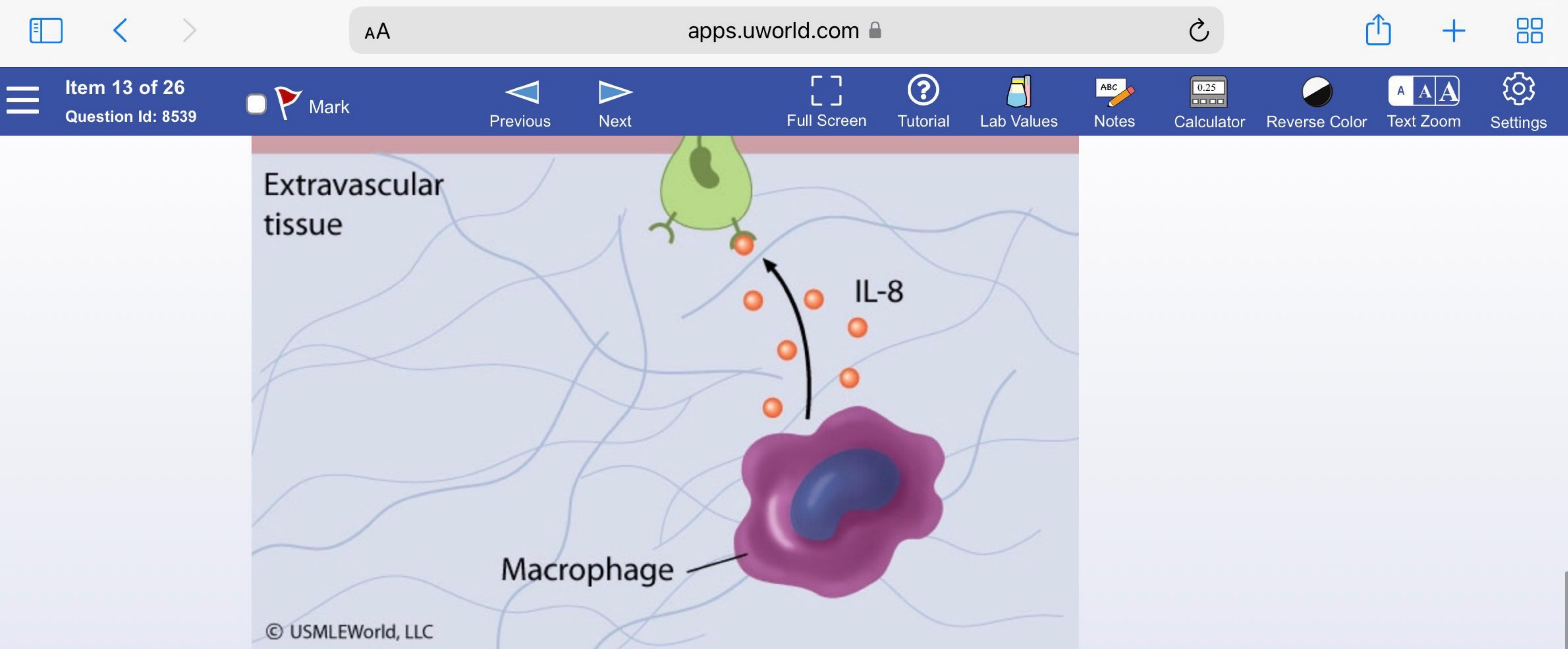
Neutrophil chemotaxis

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Explanation





Pus consists of a thin, protein-rich fluid, known as liquor puris, and dead leukocytes, primarily neutrophils.

During infection, macrophages and surrounding endothelial cells release **cytokines** such as **interleukin-8 (IL-8)** that trigger **neutrophils** to enter the site of infection via **chemotaxis**. IL-8 also induces phagocytosis in neutrophils once they have arrived.

(Choice A) Bradykinin is a component of the kinin system. It causes vasodilation, increases vascular permeability, stimulates nonvascular smooth muscle contraction, and mediates pain.

(Choice B) C3a is a component of the complement system. C3a, C4a, and C5a are inflammatory anaphylatoxins that trigger histamine release from mast cells, resulting in vasodilation and enhanced vascular permeability. C5a also recruits and activates neutrophils, monocytes, eosinophils, and basophils. In contrast,



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(Choice C) IL-3 is a cytokine produced by activated T cells. It stimulates the growth and differentiation of stem cells in the bone marrow.

(Choice E) IL-10 is an anti-inflammatory cytokine produced by T_H2 cells and macrophages. IL-10 limits the production of pro-inflammatory cytokines (eg, gamma interferon, IL-2, IL-3, and TNF- α).

(Choice F) Leukotriene C₄ (and its relatives, leukotriene D₄ and E₄) triggers intense vasoconstriction, increased vascular permeability, and bronchospasm. Leukotriene B₄ and the leukotriene precursor 5-HETE stimulate neutrophil migration to the site of inflammation (but not leukotriene C₄).

Educational objective:

Interleukin-8 is a chemokine produced by macrophages that induces chemotaxis and phagocytosis in neutrophils. Other significant chemotactic agents include leukotriene B₄, 5-HETE (the leukotriene precursor), and complement component C5a.

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An 18-month-old boy is brought to the rural health clinic by his mother to establish care. The patient got some routine childhood vaccinations from previous providers but has not had a pneumococcal vaccination. Vital signs are normal for age. A pneumococcal conjugate vaccine is ordered, but the clinic manager advises the physician that the vaccine is not available due to delays in shipment from the supplier; however, the pneumococcal polysaccharide vaccine is available. Which of the following statements is true regarding the difference between these vaccine types?

- A. The conjugate vaccine causes local site reactions less often than the polysaccharide vaccine
- B. The conjugate vaccine induces a more robust immune response through B and T cell activation
- C. The conjugate vaccine is inactivated, and the polysaccharide vaccine is live attenuated
- D. The conjugate vaccine protects against meningitis, but the polysaccharide vaccine does not
- E. The conjugate vaccine protects against more pneumococcal strains than the polysaccharide vaccine

Omitted
Correct answer
B

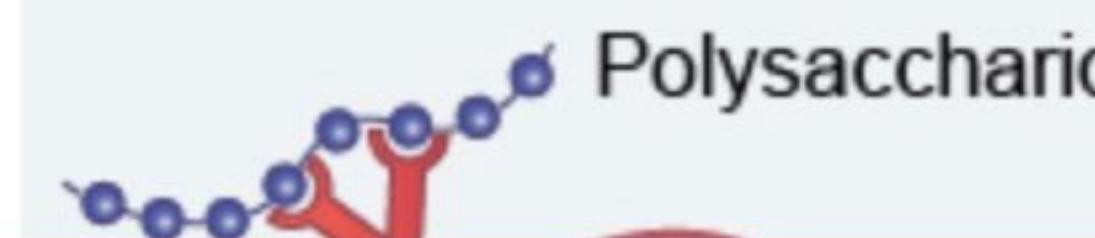
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Explanation

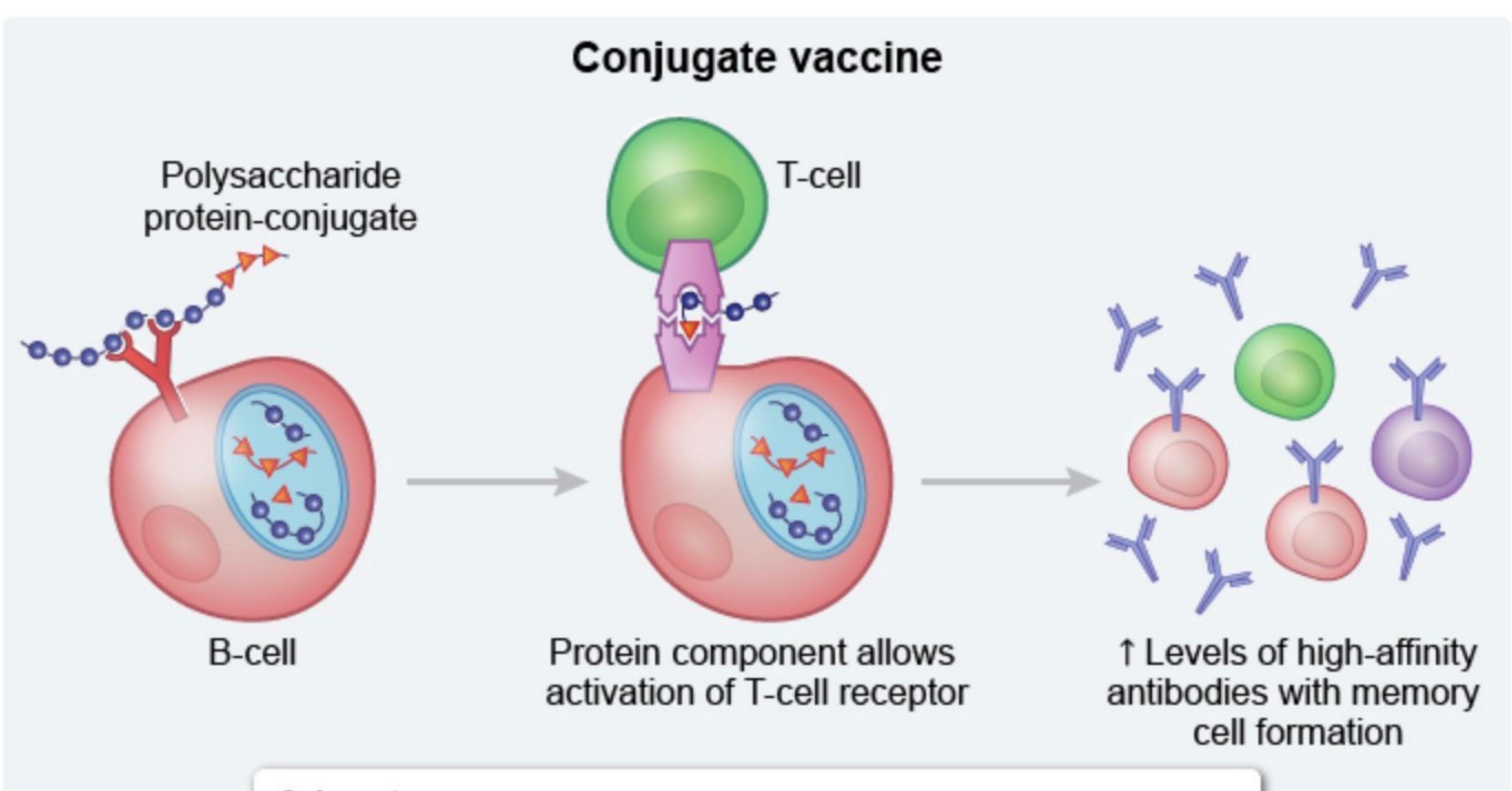
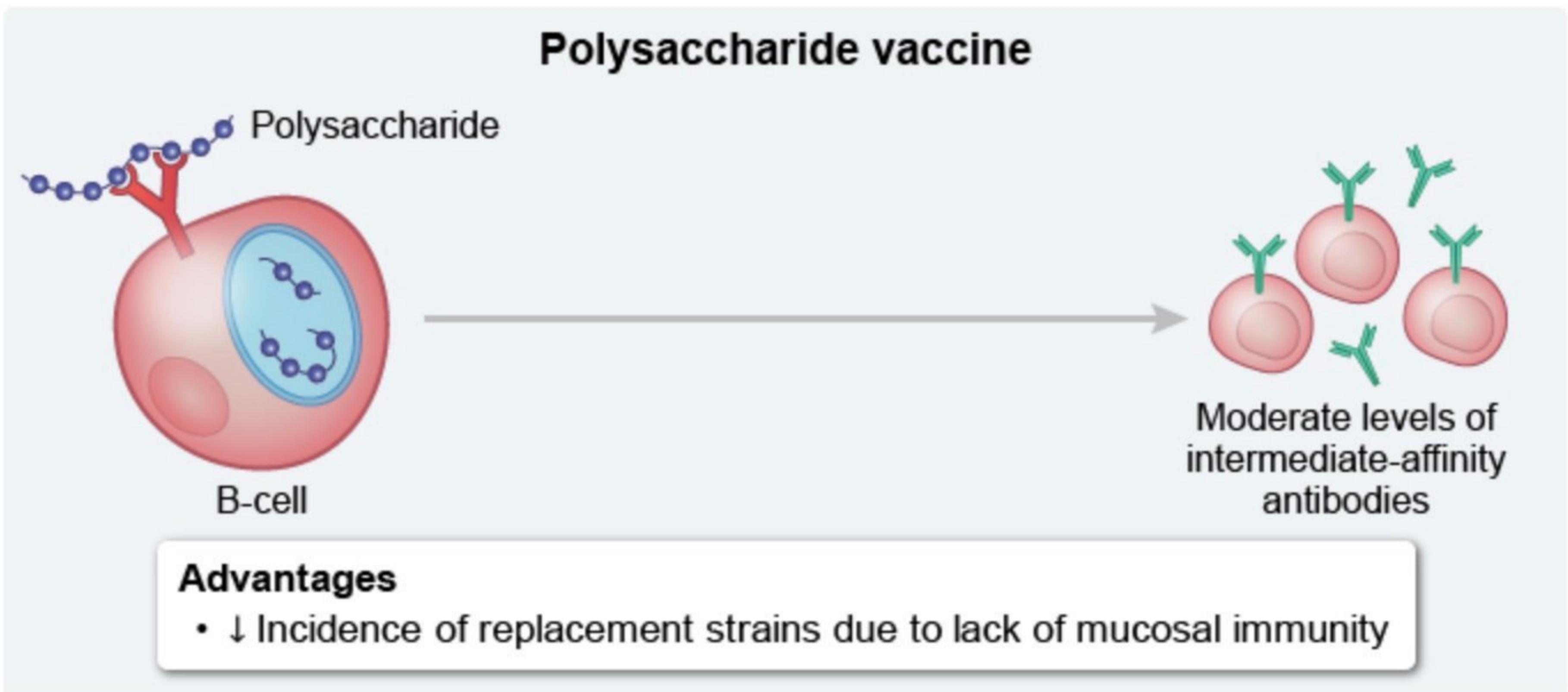
Polysaccharide vaccine



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Advantages

- ↑ Efficacy in the elderly & children <2 years
- ↑ Mucosal immunity reduces colonization (herd protection)
- ↑ Immunogenic memory

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Streptococcus pneumoniae is a common cause of invasive infection (eg, meningitis, pneumonia) in infants and children, particularly those age <2 years and those with chronic conditions (eg, heart/lung disease, immunosuppression). Pneumococcal strains are distinguished by the **polysaccharide components** of their capsule; different polysaccharides are included in vaccines to provide strain-specific immunity.

There are 2 types of pneumococcal vaccine: conjugate and polysaccharide. In **pneumococcal conjugate vaccines** (eg, PCV13), the polysaccharide is bound to a nontoxic **carrier protein** (eg, mutated diphtheria toxin) that allows the antigen complex to be presented on MHC molecules, triggering activation of T cells. T cell recruitment stimulates the production of high-affinity, long-lasting antibodies (T cell-dependent B cell activation), making the conjugate vaccines **strongly immunogenic**. In addition, conjugate vaccines are better at inducing **mucosal immunity**, which helps suppress the prevalence of vaccine strains in the community (ie, herd immunity); over time, the strains covered by the vaccine may be replaced by strains not included in the vaccine (ie, replacement strains).

In contrast, the **pneumococcal polysaccharide vaccine** (ie, PPSV23) uses unconjugated polysaccharide antigens. The polysaccharide binds to B cells without triggering T cell activation, leading to a weaker humoral response (eg, modest levels of intermediate-affinity antibodies). PPSV23 is **not immunogenic** in infants and children **age <2** years due to a limited capacity for T cell-independent B cell activation by the immature immune system. However, PPSV23 protects against a wider range of serotypes (**Choice E**), and the absence of mucosal immunity is less likely to lead to emergence of replacement strains.

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(Choice A) The rate of local site reactions (~40%-50%) is similar between pneumococcal conjugate and polysaccharide vaccines. Most local site reactions are minor (eg, transient pain, redness, swelling).

(Choice C) PPSV23 is a polysaccharide capsular subunit vaccine, not a live attenuated vaccine. Examples of live attenuated vaccines include those for rotavirus, varicella, and measles-mumps-rubella.

(Choice D) Both polysaccharide and conjugate vaccines provide strain-specific protection against invasive disease, including meningitis and sepsis.

Educational objective:

Pneumococcal conjugate vaccines are strongly immunogenic in infancy due to both B and T cell recruitment. They provide higher-affinity, longer-lasting antibody responses than the pneumococcal polysaccharide vaccine. The pneumococcal polysaccharide vaccine is poorly immunogenic in infants and children age <2 years due to a limited capacity for T cell-independent B cell activation.

References

- A review of the evidence to inform pneumococcal vaccine recommendations for risk groups aged 2 years and

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A 2-year-old boy is brought to the emergency department due to wheezing and difficulty breathing. The patient had been trick-or-treating with his parents and ate several packs of candy containing peanuts. After he receives an intramuscular epinephrine injection, his symptoms resolve. At a follow up appointment, an allergy specialist places droplets of various allergens on the patient's skin and punctures the epidermis at each site. After 15 minutes, the skin at the site with peanut extract is erythematous with a raised, itchy bump that improves by the time the family leaves the office. Three hours later, the parents notice increased swelling at the puncture site. Which of the following is most likely involved in this secondary reaction?

- A. Cell lysis following IgG autoantibody binding (5%)
- B. Complement activation by immune complexes (13%)
- C. Epithelial damage by major basic protein (27%)
- D. IgE-mediated histamine release from mast cells (28%)
- E. Interferon gamma release from CD4 T cells (25%)

Omitted
Correct answer
C

27%
Answered correctly

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Explanation

Cutaneous type 1 hypersensitivity reactions

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Cutaneous type 1 hypersensitivity reactions

Initial exposure

The diagram shows an allergen interacting with an antigen-presenting cell. The antigen-presenting cell presents the allergen to a Th2 cell. The Th2 cell secretes IL-5, leading to eosinophil recruitment. It also secretes IL-4 and IL-13, which lead to B-cell activation and IgE production. Simultaneously, the allergen binds to IgE antibodies on the surface of a mast cell, leading to mast cell priming.

Repeat exposure

Early phase (immediate): IgE cross-linking on the mast cell triggers the release of histamine and leukotrienes. This leads to vasodilation and increased vascular permeability, resulting in a wheal and flare.

Late phase (hours later): The reaction continues with eosinophil recruitment and infiltration. Major basic protein from the eosinophils causes tissue damage, leading to induration.

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Following skin prick testing, this patient developed an erythematous, edematous welt that was followed 3 hours later by an indurated skin lesion. These findings are consistent with the early and late phases of a [type I hypersensitivity](#) reaction.

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After first exposure to an allergen (eg, peanuts), antigen specific IgE is produced by B-cells and binds to the surface of mast cells. If repeat exposure occurs, the bound IgE can cross-link and stimulate the release of preformed histamine and leukotrienes that cause vasodilation and increased capillary permeability. The result is a rapid (eg, minutes after exposure) **early-phase** type I hypersensitivity response characterized by superficial dermal edema and erythema (eg, [wheal and flare reaction](#)) that can progress to a more systemic response (eg, anaphylaxis) (**Choice D**).

IgE also initiates the **late phase** of a type I hypersensitivity reaction by stimulating type 2 helper T cells to release cytokines (eg, IL-5) that activate **eosinophils**. Cationic proteins (eg, **major basic protein**, eosinophil peroxidase) released from eosinophils cause **tissue damage**, which usually manifests as a palpable, **indurated lesion** 2-10 hours following the early-phase reaction.

(Choice A) Binding of preformed IgG antibodies to cell surface antigens initiates cell lysis mediated by complement and/or natural killer cells ([type II hypersensitivity](#) reactions). These reactions can develop hours to days after exposure to an antigen (eg, medications, transfused red blood cells), resulting in destruction of erythrocytes (causing fatigue, pallor), platelets (petechial bleeding), or leukocytes (fever, sepsis).

(Choice B) Immune complex-mediated complement activation typically causes tissue damage in sites where immune complexes tend to deposit (eg, skin, joints, kidneys) days to weeks after initial antigen exposure in [type III hypersensitivity](#) reactions. Although an Arthus reaction can cause local swelling within hours of an exposure, it is usually associated with intradermal injection of a vaccine.

(Choice E) In [type IV \(delayed\) hypersensitivity](#) reactions, CD4⁺ T cells release cytokines (eg, interferon gamma) that promote T cell- and macrophage-mediated tissue damage. Although these reactions can also present as an indurated skin lesion (eg, tuberculosis skin testing), type IV hypersensitivity reactions develop over days (rather than hours) because of the time needed for cellular amplification.

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Educational objective:

The late phase of dermatologic type I hypersensitivity reactions manifests as an indurated skin lesion hours after exposure to the allergen due to local tissue damage caused by major basic protein released from eosinophils. In contrast, type IV hypersensitivity reactions develop over days because of the time needed to produce a cell-mediated immune response.

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A 58-year-old woman comes to the office for a health checkup prior to starting volunteer work at a hospital. She has a history of hypothyroidism and takes levothyroxine. The patient feels well, and review of systems is negative. She does not use tobacco, alcohol, or illicit drugs. Her examination findings are unremarkable. During a laboratory test, her white blood cells are incubated with mycobacterial antigens. Compared to the control, a large amount of interferon-gamma is detected in her blood sample. Which of the following cell types is most directly responsible for this finding?

- A. B lymphocytes (4%)
- B. Monocytes (19%)
- C. Neutrophils (5%)
- D. T lymphocytes (70%)

Omitted
Correct answer
D

 70%
Answered correctly

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Time Spent

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Explanation

Interferon-gamma (IFN- γ) activates macrophages, increases major histocompatibility complex expression, and promotes T helper 1 lymphocyte (Th1) differentiation. It is produced primarily by activated **T lymphocytes** and natural killer cells and is critical for immunity against viral and intracellular bacterial infections. IFN- γ release assays (IGRAs) test for **latent tuberculosis infection** (LTBI) by measuring the response of T lymphocytes when exposed to antigens unique to *Mycobacterium tuberculosis*. Similar to tuberculin skin tests (eg. purified protein

Explanation

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IGRAs have comparable sensitivity and specificity to tuberculin skin tests, but advantages include their lack of cross-reactivity to the Bacille Calmette-Guérin (BCG) vaccine and that a follow-up visit is not required. Neither skin tests nor IGRAs can be used to [distinguish active tuberculosis from LTBI](#).

(Choice A) B lymphocytes are the main cell type involved in the humoral immune system and the production of circulating antibodies.

(Choice B) Antigen-presenting cells such as monocyte-derived macrophages interact with T cells to control the immune response but are not directly responsible for IFN- γ release.

(Choice C) Neutrophils are involved in the phagocytosis of bacteria and other pathogens.

Educational objective:

Interferon-gamma (IFN- γ) release assays test for latent tuberculosis infection by measuring the amount of IFN- γ released by T lymphocytes when exposed to antigens unique to *Mycobacterium tuberculosis*.

References

- [Gamma interferon release assays for detection of *Mycobacterium tuberculosis* infection.](#)

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Mark

Explanation

Exhibit Display

Latent TB infection & active TB disease		
	Latent TB infection	Active pulmonary TB disease
Clinical manifestations	<ul style="list-style-type: none">Asymptomatic	<ul style="list-style-type: none">CoughConstitutional symptoms<ul style="list-style-type: none">Fever/chills, malaiseWeight loss, night sweatsAnorexia, fatigue
TB transmission	<ul style="list-style-type: none">No	<ul style="list-style-type: none">Yes
Diagnostic tests	<ul style="list-style-type: none">Positive tuberculin skin test & interferon-γ release assayNormal chest x-rayNegative sputum smear/culture	<ul style="list-style-type: none">Positive tuberculin skin test & interferon-γ release assayAbnormal chest x-rayPositive sputum smear/culture

TB = tuberculosis.

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References

- Gamma interferon release assays for detection of *Mycobacterium tuberculosis* infection.

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A 4-year-old male is exposed to latex gloves during a minor surgical procedure and is subsequently found to produce anti-latex IgM antibodies. Several months later he develops a severe allergic reaction to latex and is found to have a high level of serum anti-latex IgE antibodies. Which of the following cytokines is most likely responsible for this anti-latex antibody isotype change?

- A. IL-1 (1%)
- B. IL-2 (5%)
- C. IL-3 (3%)
- D. IL-4 (82%)
- E. IL-10 (3%)
- F. IL-12 (3%)

Omitted

Correct answer

D



82%

Answered correctly



02 secs

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Explanation

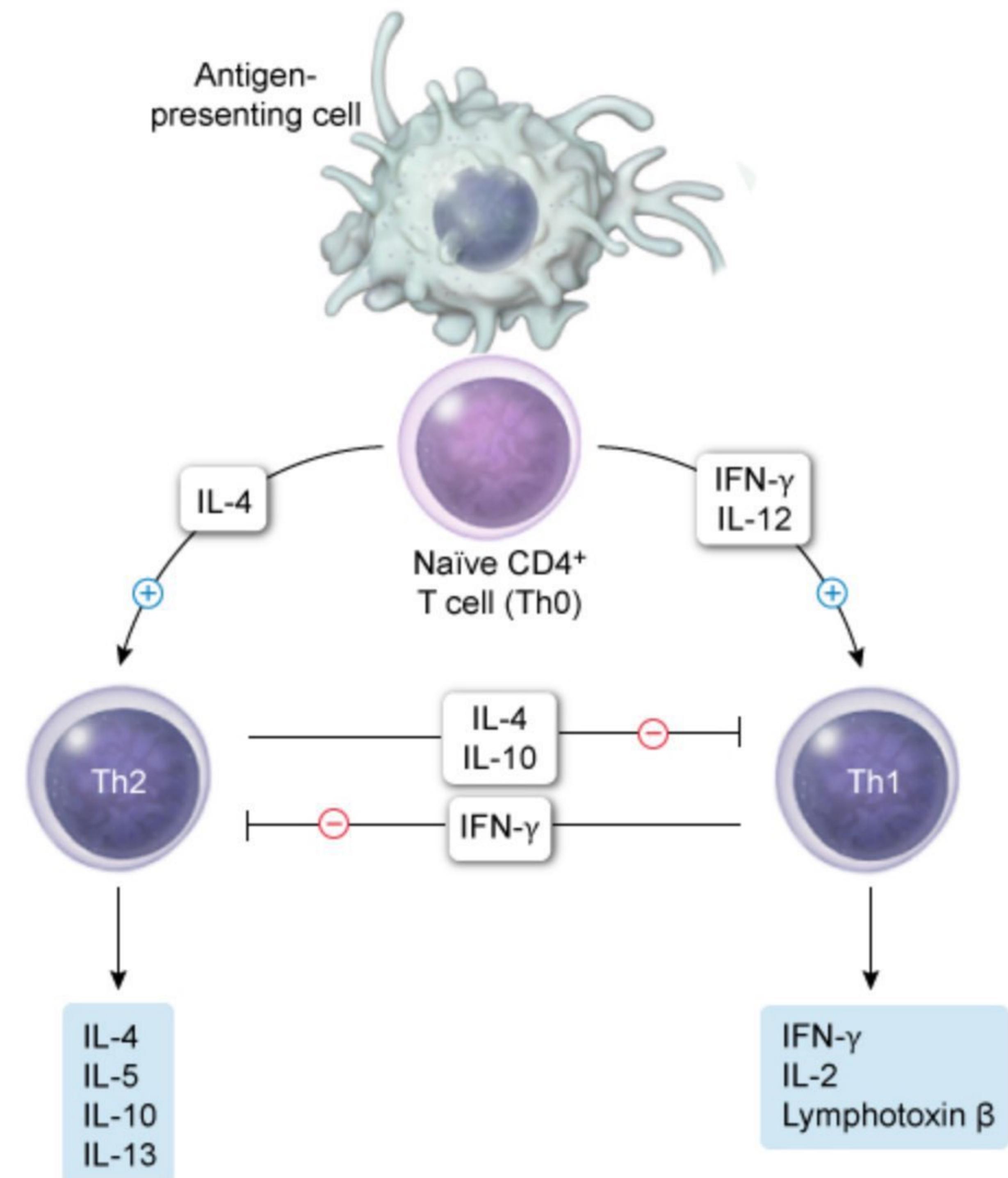
This patient has a **severe allergic reaction** to latex several months after initial sensitization. During the first encounter with an antigen (allergen), antigen-presenting cells (eg, macrophages, B cells, and dendritic cells) take up the antigen and display it with MHC class II molecules. CD4+ T cells are activated when they detect the MHC class II-associated antigen and differentiate into either T₁ or T₂ subsets. T₂ lymphocytes promote the

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Factors in T helper cell differentiation



- Initiation of antibody response
- Immunoglobulin class switching
- Immediate (IgE-mediated) hypersensitivity

- Activation of macrophages and CD8⁺ T cells
- Delayed-type hypersensitivity

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IL-4 stimulates the **proliferation and differentiation** of Th0 (naïve) T cells into Th2 lymphocytes, thus increasing the Th2 subpopulation and the stimulus for the primary humoral immune response (eg, IgM). It also induces B cell proliferation and immunoglobulin **class switching to IgE** which is responsible for the **type I hypersensitivity** reaction following repeated exposure to an allergen. IL-5 contributes to B cell differentiation in addition to its role in stimulating IgA production and eosinophil activity (eg, host defense against parasitic infections).

(Choice A) IL-1 is produced by macrophages. It activates naïve Th0 lymphocytes and promotes their differentiation into Th1 and Th2 subpopulations. IL-1 is also an endogenous pyrogen.

(Choice B) IL-2 is the first interleukin produced by T cells after contact with antigen. It is secreted mainly by Th1 cells and stimulates development of CD4+ T helper cells, CD8+ cytotoxic cells, and B cells.

(Choice C) IL-3 stimulates growth and differentiation of bone marrow stem cells and is produced by T helper cells.

(Choice E) IL-10 helps regulate the balance between the Th1 and Th2 subpopulations of T helper cells. It is produced by Th2 lymphocytes and inhibits synthesis of interferon- γ leading to a decrease in the Th1 subpopulation. IL-10 also plays a part in class switching from IgM to IgG antibodies.

(Choice F) IL-12 is synthesized by macrophages and stimulates growth and development of the Th1



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IL-4 stimulates the proliferation and differentiation of T_H0 (naïve) T cells into T_H2 lymphocytes, thus increasing

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(Choice C) IL-3 stimulates growth and differentiation of bone marrow stem cells and is produced by T helper cells.

(Choice E) IL-10 helps regulate the balance between the T_H1 and T_H2 subpopulations of T helper cells. It is produced by T_H2 lymphocytes and inhibits synthesis of interferon-γ leading to a decrease in the T_H1 subpopulation. IL-10 also plays a part in class switching from IgM to IgG antibodies.

(Choice F) IL-12 is synthesized by macrophages and stimulates growth and development of the T_H1 subpopulation of T helper cells.

Educational Objective:

IL-4 is produced by the T_H2 subset of T helper cells. It facilitates proliferation of B cells and T_H2 lymphocytes and stimulates antibody isotype switching to IgE which mediates type I hypersensitivity (allergic) reactions.

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A 24-year-old, previously healthy woman is evaluated for skin rash, joint pains, and renal failure. She is found to have decreased C3 and C4 levels and a normal factor B level. Which of the following most likely triggered the complement system activation in this patient?

- A. Antigens binding to IgA (4%)
- B. Autoactivation of C3 component (22%)
- C. C1 components binding to C1 inhibitor (8%)
- D. C9-lipid membrane complex formation (1%)
- E. IgG-antigen complex formation (62%)

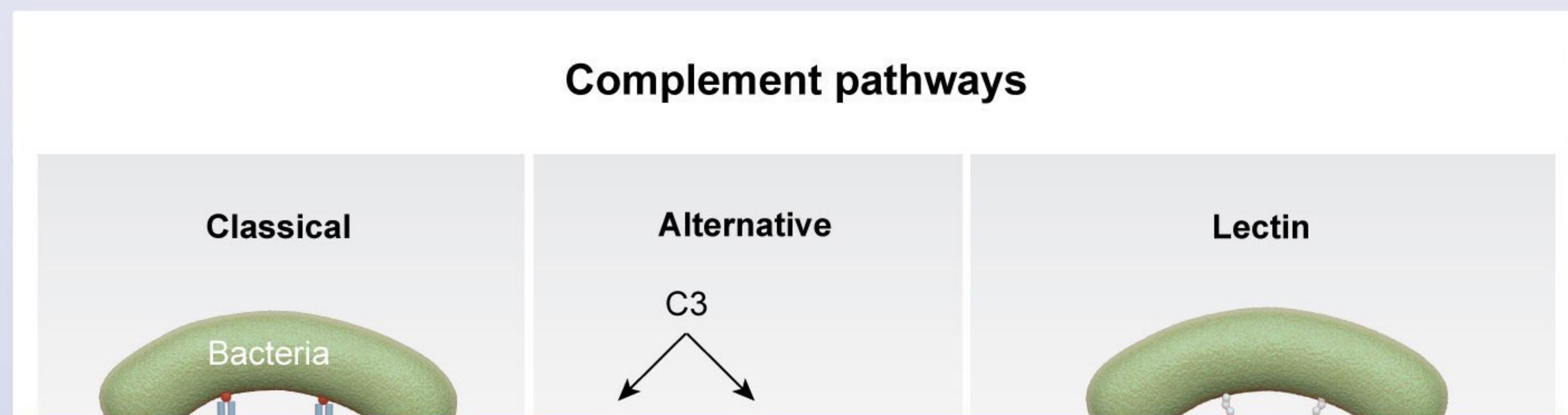
Omitted
Correct answer
E

62%
Answered correctly

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Explanation

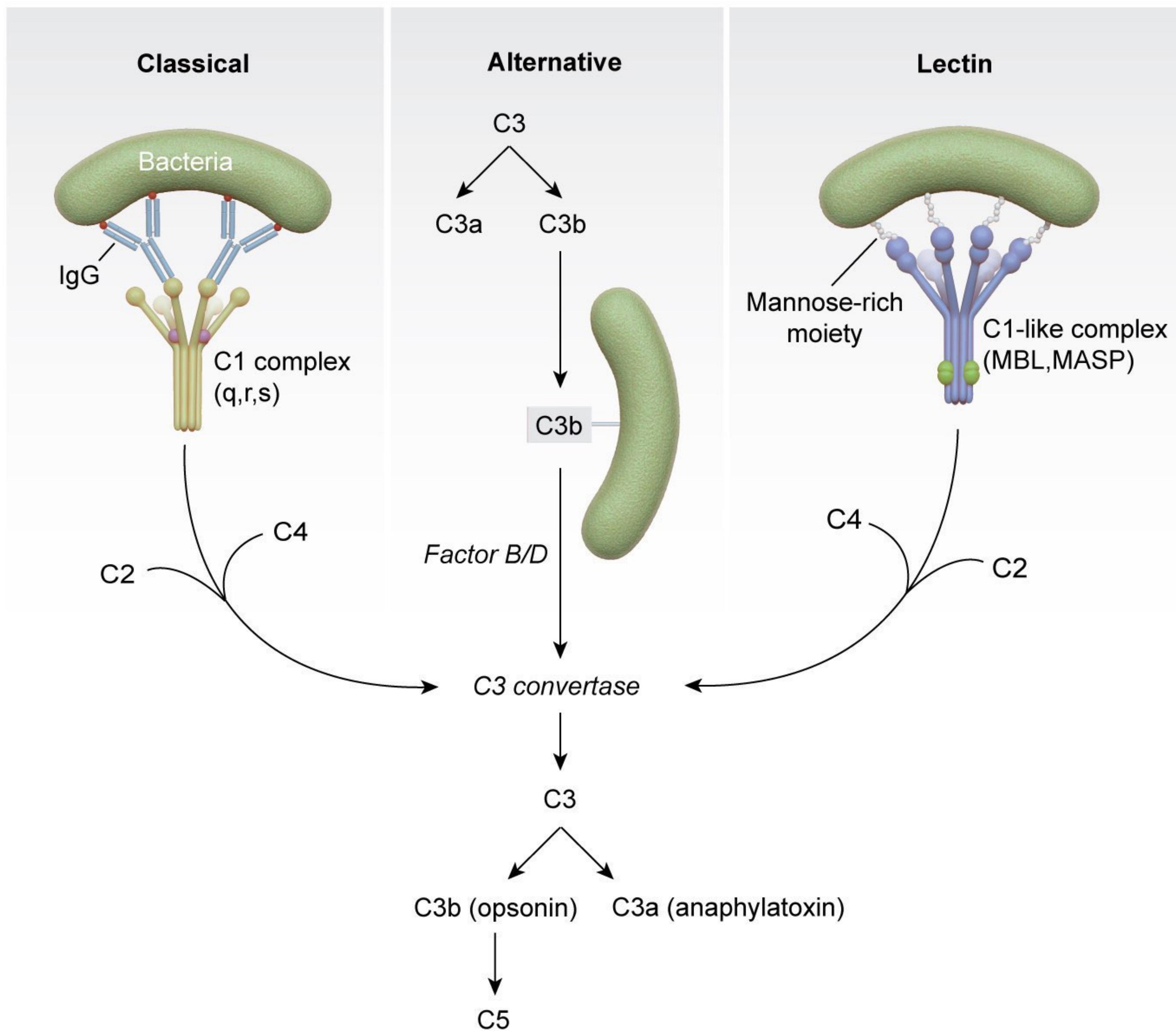


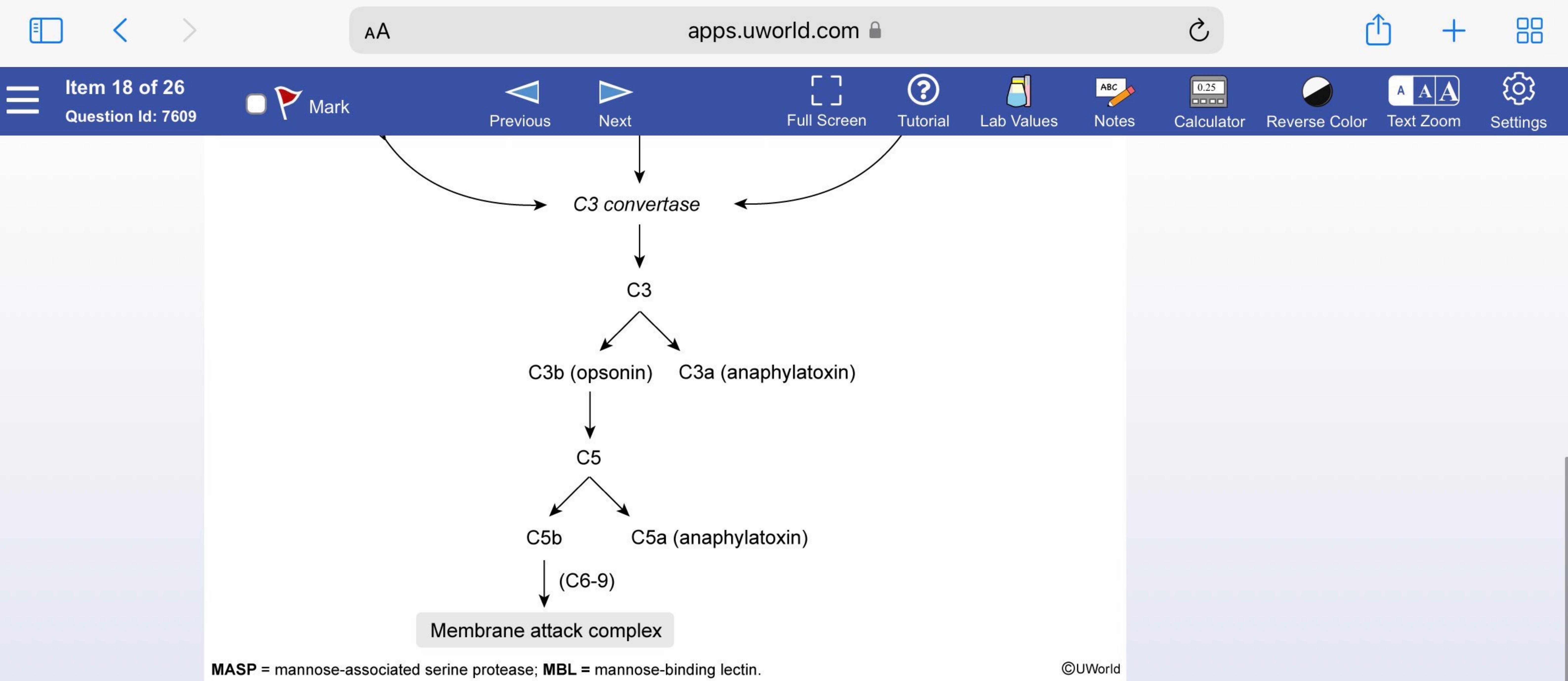
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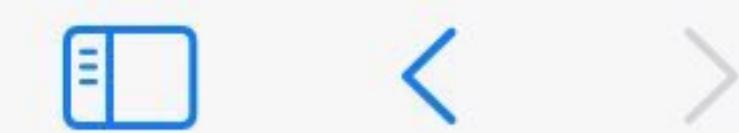
Complement pathways





The complement cascade is an ancient proteolytic defense mechanism that plays a major role in both the innate and adaptive immune responses. It is activated by 3 major inciting events, all of which terminate in the generation of **C3 convertase** as follows:

- **Antibody-antigen binding** (classical pathway): The C1 complex (C1q/r/s) forms on the Fc portion of an IgM or IgG antibody that is bound to an antigen; the C1 complex then cleaves C4 and C2 into C3 convertase.
- **Lectin pattern recognition receptor binding** (lectin pathway): Host pattern recognition receptors bind to carbohydrates that are produced only by foreign pathogens; binding generates proteases that cleave C4



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The complement cascade is an ancient proteolytic defense mechanism that plays a major role in both the innate and adaptive immune responses. It is activated by 3 major inciting events, all of which terminate in the generation of **C3 convertase** as follows:

- **Antibody-antigen binding** (classical pathway): The C1 complex (C1q/r/s) forms on the Fc portion of an IgM or IgG antibody that is bound to an antigen; the C1 complex then cleaves C4 and C2 into C3 convertase.
- **Lectin pattern recognition receptor binding** (lectin pathway): Host pattern recognition receptors bind to carbohydrates that are produced only by foreign pathogens; binding generates proteases that cleave C4 and C2 into C3 convertase without requiring the C1 complex.
- **C3b binding** (alternative pathway): A small amount of autoactivated C3b continually forms in the intravascular space and is rapidly inactivated by healthy cells. However, the presence of microbes or damaged cells amplifies the production of C3b, which then engages with **factor B** and factor D and generates C3 convertase.

C3 convertase catalyzes the formation of proteins that opsonize pathogens, promote inflammation, and lead to the generation of membrane attack complexes.

The most common cause of complement deficiency is **autoantibodies**, which activate the classical complement system after binding host antigens. Classical complement pathway activation is marked by **low C4 and C3 levels and normal factor B levels**; CH50, a measure of functional activity of the entire classical pathway (eg, sufficient C1-C9), will also be low. This pattern is common in **systemic lupus erythematosus**, particularly in the setting of active **renal, skin, and joint disease**.

(Choice A) The Fc portions of IgA, IgE, and IgD cannot activate the complement system. Therefore, IgA-

intravascular space and is rapidly inactivated by healthy cells. However, the presence of microbes or damaged cells amplifies the production of C3b, which then engages with **factor B** and factor D and generates C3 convertase.

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(Choice A) The Fc portions of IgA, IgE, and IgD cannot activate the complement system. Therefore, IgA-antigen binding does not reduce C3 and C4 levels.

(Choice B) Autoactivation of C3b triggers the alternative complement pathway, which is marked by normal C4, low C3, and low factor B levels; AH50, a measure of functional activity of the alternative pathway, will also be low.

(Choice C) C1 inhibitors remove C1r/s from the Fc portion of immunoglobulin (classical pathway) and block the activation of C2/C4 by lectin pattern recognition receptors (lectin pathway). Therefore, C1 inhibitors prevent activation of the complement cascade and increase (not decrease) complement levels.

(Choice D) The complement cascade culminates with the generation of a membrane attack complex using C9 multimers in combination with C5-C8, leading to cell lysis.

Educational objective:

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Educational objective:

The binding of autoantibodies to host antigens can trigger the classical complement cascade, leading to low C4 and C3 levels. Because autoantibodies do not activate the alternative complement cascade, factor B levels remain normal. This pattern is frequently seen in rheumatologic diseases such as systemic lupus erythematosus.

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A 75-year-old man is hospitalized due to respiratory distress. The patient developed fever, cough, and muscle aches 4 days prior to admission. He is otherwise healthy and has no chronic medical conditions. The patient has received all recommended vaccinations, including a yearly flu vaccine. Temperature is 39 C (102.2 F), blood pressure is 110/65 mm Hg, pulse is 115/min, and respirations are 29/min. Chest x-ray shows bilateral infiltrates. Reverse transcriptase PCR of a specimen from a nasopharyngeal swab reveals a strain of influenza A virus that was included in the seasonal trivalent flu vaccine. The patient lives with his 50-year-old son, who received the same vaccine but did not develop the infection. Which of the following factors most likely increased this patient's risk of vaccine failure compared with that of his son?

- A. Decreased overall quality of antibodies (15%)
- B. Decreased production of naive B lymphocytes (37%)
- C. Diminished levels of memory T lymphocytes (45%)
- D. Increased apoptosis induced by neutrophils (0%)
- E. Increased phagocytosis by macrophages (0%)

Omitted

Correct answer

B



37%

Answered correctly



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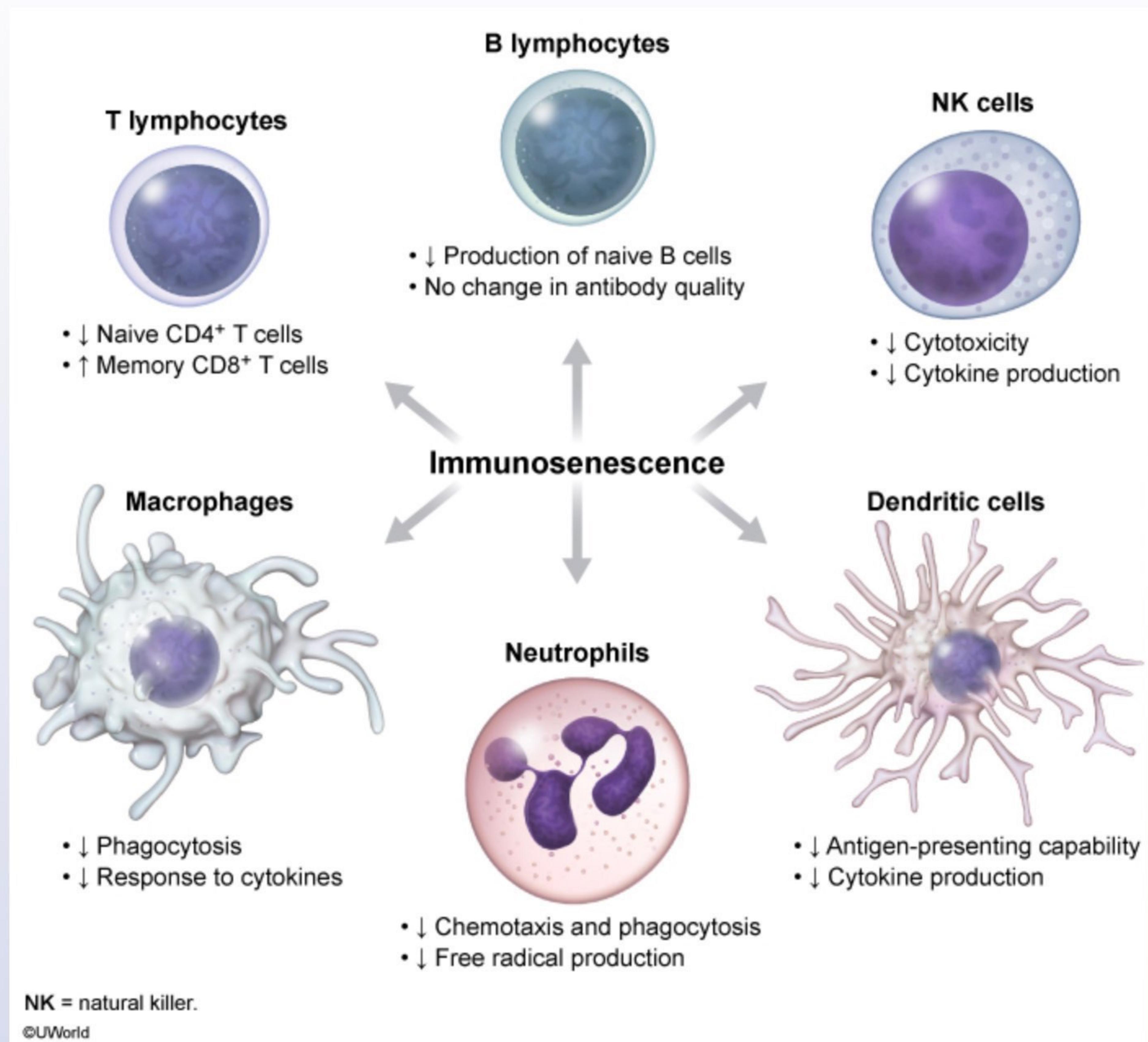
Explanation

B lymphocytes

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Most of the protection provided by vaccines depends on antibodies generated by an immune response to pathogen-specific antigens. These antibodies can bind to the pathogen and directly neutralize it or facilitate elimination of the pathogen through phagocytosis, complement fixation, and/or antibody-dependent cytotoxicity. The risk of primary **vaccine failure** is increased in patients with altered immune function, including those with atopic disorders (eg, asthma, eczema), steroid use, or age-related immune decline (eg, **immunosenescence**).

The loss of telomere length during normal aging particularly affects rapidly dividing immune cells (eg, bone marrow stem cells, lymphocyte precursors), resulting in **decreased** production of **naive B and T lymphocytes**. Aging is also associated with chronic low-grade inflammation that causes much of the remaining naive lymphocyte pool to differentiate into memory lymphocytes against previously encountered antigens. These changes **impair** the adaptive immune **response to novel antigens** (eg, pathogens, vaccinations) and predispose these patients to vaccine failure and increased susceptibility to infection.

(Choices A and C) Antibody quality (ie, avidity for the target antigen) and levels of memory B and T lymphocytes are preserved with age, allowing most aging individuals to mount an effective immune response to previously encountered antigens.

(Choice D) Neutrophil-induced apoptosis is reduced in aging individuals, increasing susceptibility for nonhealing wounds/infections. However, a change in neutrophil function would not significantly impact the vaccine response.

(Choice E) Phagocytosis and antigen presentation by dendritic cells and macrophages decline with age, further impairing the immune response to novel antigens.

Educational objective:

Immunosenescence is the normal age-related decline that impairs most aspects of immune function, including the production of naive B and T cells. This results in a diminished antibody-based immune response to novel

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Educational objective:

Immunosenescence is the normal age-related decline that impairs most aspects of immune function, including the production of naive B and T cells. This results in a diminished antibody-based immune response to novel antigens (eg, infections, vaccinations). The immune response to previously experienced pathogens is typically intact due to normal or increased levels of memory B and T cells and preserved antibody quality.

References

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Item 20 of 26 Question Id: 20698

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A 26-year-old man returns to the emergency department after developing a fever and skin rash. The patient was discharged from the hospital 10 days ago after treatment for a copperhead snake bite to his left leg. He received multiple doses of polyvalent Fab antivenom therapy and other supportive care during hospitalization. The patient's bite site pain, swelling, and ecchymosis have resolved; however, he has developed fever, pain in multiple extremity joints, and pruritic rash over the past 2 days. He has no chronic medical conditions.

Temperature is 38.5 C (101.3 F), blood pressure is 128/70 mm Hg, pulse is 98/min, and respirations are 17/min. Physical examination shows a diffuse urticarial rash. No mucous membrane lesions are present. There is tenderness to palpation of the bilateral metacarpophalangeal joints, wrists, and ankles with no redness or swelling. Blood cell counts, serum chemistry studies, and coagulation parameters are within normal limits.

Which of the following is the most likely underlying mechanism of this patient's current condition?

- A. IgE-mediated hypersensitivity reaction to the antivenom (9%)
- B. Polyclonal T-cell activation by the antivenom (17%)
- C. Receptor-mediated phagocytosis of unbound antivenom (1%)
- D. Snake venom-induced diffuse mast cell degranulation (4%)
- E. Tissue deposition of host antibodies and antivenom complexes (67%)

Omitted
Correct answer
E

67%
Answered correctly

02 secs
Time Spent

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Explanation

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Polyclonal fab antivenom is a collection of immunoglobulin fragments from the plasma of venom-inoculated animals (eg, horses). Because the antivenom contains **foreign proteins**, exposure triggers the **adaptive immune response** to form high-affinity IgG antibodies against the foreign components of the antivenom. This typically takes **1-2 weeks** due to time lag between antigen processing, antigen presentation, CD4 T-lymphocyte activation, and T-cell-mediated B-cell activation/differentiation.

Once formed, IgG then binds to the free-circulating antivenom, which creates **immune complexes** (ICs). The Fc portion of the IgG triggers clearance of the ICs by activating the classical complement system and by directly binding to the Fc receptor on mononuclear phagocytes in the reticuloendothelial system. Clearance of ICs generally proceeds without issue when there is minimal antigen because the phagocytic system has a large capacity.

However, the administration of multiple antivenom doses can overwhelm the phagocytic system, leading to the aggregation of ICs in the bloodstream. Aggregated ICs then **deposit in tissue** (eg, skin, joints), activate the complement cascade, and cause a **type III hypersensitivity** reaction called **serum sickness**. Serum sickness generally presents with **fever, urticarial rash, and arthralgia** 1-2 weeks after exposure to nonhuman proteins in antivenom, antitoxins, monoclonal antibodies, or vaccinations. Most cases resolve spontaneously over several days as the ICs are cleared.

(Choices A and D) Antivenom administration can cause IgE antibodies to form against the nonhuman portions of the protein, which then triggers a **type I hypersensitivity** reaction upon reexposure. Although these reactions often cause urticaria due to mast cell degranulation, symptoms typically arise within seconds or minutes of reexposure, not a week or two after initial exposure.

(Choice B) Superantigens directly activate polyclonal populations of T cells, leading to acute-onset fever, hypotension, sunburn-like rash, and organ failure. Fab fragments in antivenom are unlikely to activate T cells.

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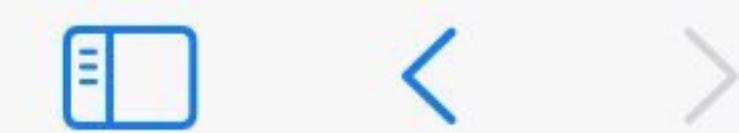
(Choice B) Superantigens directly activate polyclonal populations of T cells, leading to acute-onset fever, hypotension, sunburn-like rash, and organ failure. Fab fragments in antivenom are unlikely to activate T cells directly; they are processed by antigen-presenting cells and lead to the generation of antibodies.

(Choice C) The phagocytic Fc receptor can only bind antivenom that has bound IgG or IgM antibody. Unbound antivenom cannot attach to phagocytic receptors.

Educational objective:

Serum sickness is an immune complex-mediated type III hypersensitivity reaction that occurs 1-2 weeks after exposure to nonhuman protein in antitoxins (eg, antivenom), monoclonal antibodies (eg, rituximab), or vaccines (eg, rabies antigens). Deposition of immune complexes in tissue leads to complement activation and subsequent self-limited fever, arthralgia, and urticarial rash.

References



AA

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Question Id: 21420



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A 2-week-old infant is brought to the emergency department due to fever, lethargy, grunting, and poor feeding. The patient was born at full term after an uneventful pregnancy and has had no prior medical issues. Blood samples are obtained for culture, and the patient is hospitalized for broad-spectrum antibiotic therapy. Cultures grow *Escherichia coli*. The patient's condition developed in part due to exposure to bacterial lipopolysaccharide, which stimulates NF- κ B-induced transcription of inflammatory cytokines such as TNF-alpha, IL-1, and IL-6. This bacterial component most likely interacted with the patient's immune cells via which of the following?

- A. Beta-2 integrin (1%)
- B. Fc receptor (6%)
- C. L selectin (1%)
- D. MHC class II molecule (18%)
- E. Mannose-binding lectin (9%)
- F. Toll-like receptor (61%)

Omitted
Correct answer
F

61%
Answered correctly

02 secs
Time Spent

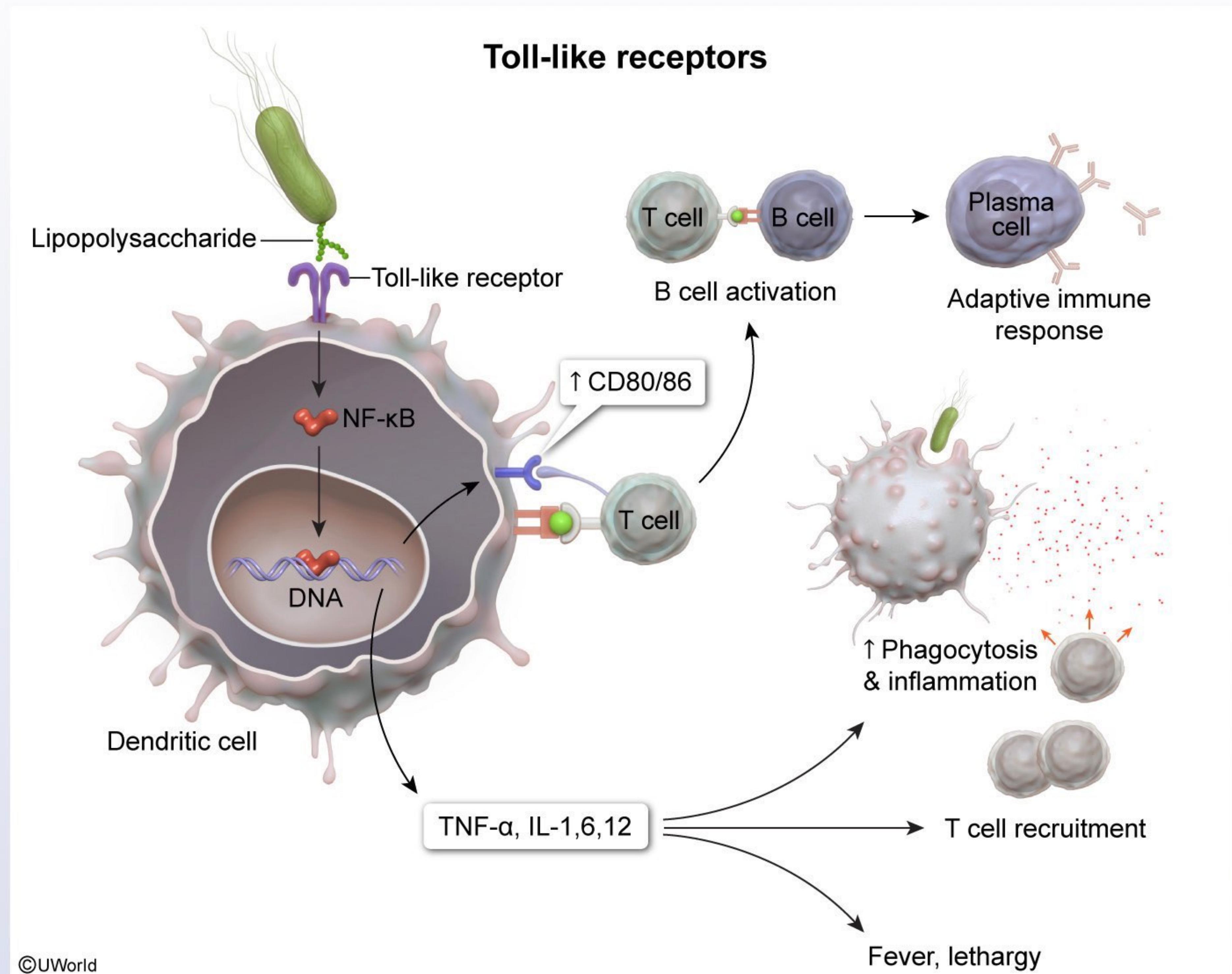
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Explanation

Toll-like receptors

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Dendritic cells and **macrophages** are phagocytic antigen-presenting cells that have an important role in both the innate and adaptive immune responses. As part of their innate immune function, they express **pattern recognition receptors** (PRRs) on their surfaces that recognize 2 major categories of ligands:

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- Damage-associated molecular patterns (**DAMPs**) are intracellular components (proteins, phospholipids, DNA) released by host cells when they are damaged by inflammation or infection.
- Pathogen-associated molecular patterns (**PAMPs**) are microbial components conserved across numerous species that are generally required for microbial survival.

One of the most common PAMPs is **lipopolysaccharide**, a component of the outer membrane of all **gram-negative bacteria** (eg, *Escherichia coli*). Lipopolysaccharide binds to a type of PRR called a **toll-like receptor**, which contains an extracellular ligand-binding domain, a transmembrane domain, and a cytoplasmic domain that conducts **transcription signals** to the nucleus via **NF- κ B**.

NF- κ B signaling promotes transcription of **proinflammatory cytokines** (eg, TNF-alpha, IL-1, IL-6, IL-12), leading to local inflammation, immune cell recruitment, and systemic effects (eg, fever, malaise, lethargy, poor feeding). It also stimulates antigen-presenting cells to increase phagocytosis, antigen display, and expression of costimulatory molecules (eg, CD80/86) for T- and B-cell activation, thereby triggering a strong adaptive immune response.

(Choice A) Beta-2 integrin (CD18) helps leukocytes migrate from the bloodstream to tissue by binding to intercellular adhesion molecule-1 on the extracellular matrix of the endothelium. It also helps macrophages, neutrophils, and natural killer cells generate complement receptors, which recognize and phagocytose foreign peptides.

(Choice B) The **Fc** receptor (CD16) on phagocytic cells binds to opsonized (eg, IgG-bound) foreign pathogens,

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(Choice B) The [Fc receptor](#) (CD16) on phagocytic cells binds to opsonized (eg, IgG-bound) foreign pathogens, leading to phagocytic destruction. The Fc receptor on natural killer cells mediates their ability to destroy infected or cancerous cells by antibody-dependent cellular cytotoxicity.

(Choice C) L selectin (CD62L) is a lymphocyte adhesion molecule that binds to a ligand on the venule endothelium, which allows lymphocytes to leave the bloodstream and enter secondary lymphoid tissue.

(Choice D) Major histocompatibility complex class II molecules are on antigen-presenting cells; they present antigens to the T cell receptor on CD4 cells.

(Choice E) Mannose-binding lectin is a PRR that recognizes microbial carbohydrates and activates the [lectin complement pathway](#).

Educational objective:

Pattern recognition receptors (PRRs) are part of the innate immune response; they recognize damaged host proteins or conserved microbial molecules and trigger inflammation. Toll-like receptors, a type of PRR on macrophages and dendritic cells, recognize lipopolysaccharide and promote the release of inflammatory cytokines (eg, IL-1/6/12, TNF-alpha) via NF- κ B signaling.

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NF-kB signal transmits transcription signals to the nucleus via NF-kB.

to local inflammatory response.

It also stimulates costimulatory molecules.

(Choice A) intercellular adhesion molecules, neutrophils, and cytokine peptides.

(Choice B) leading to apoptosis or cancerous transformation.

(Choice C) endothelial progenitor cells.

(Choice D) antigens to T cells.

(Choice E) complements.

Exhibit Display

Opsonization & phagocytosis

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Educational objective:

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