# LEC 4 PATHO INFLAMMATIONS Q:

1. **What is the primary focus of the "4th R" in inflammation?**
   A) Recruitment of white blood cells
   B) Recognizing the offending agent
   C) Removing harmful agents via phagocytosis
   D) Regulating the inflammatory response
2. **Which of the following is NOT a side effect of prolonged inflammation?**
   A) Tissue damage
   B) Pain
   C) Fever
   D) Increased vascular permeability
3. **What does the mediator TGF-β primarily do in inflammation?**
   A) Stimulate leukocyte recruitment
   B) Inhibit and degrade other inflammatory mediators
   C) Promote vascular dilation
   D) Trigger fever and metabolic abnormalities
4. **Which of the following inflammatory cells has the shortest lifespan?**
   A) Neutrophils (PMNs)
   B) Macrophages
   C) Mast cells
   D) Dendritic cells
5. **Which mediator is primarily responsible for inhibiting platelet aggregation?**
   A) Thromboxane A2
   B) Prostaglandin E2 (PGE2)
   C) Prostacyclin (PGI2)
   D) Leukotrienes
6. **Which of the following inflammatory mediators is synthesized through the 5-Lipoxygenase pathway?**
   A) Prostaglandins
   B) Thromboxane A2
   C) Leukotrienes
   D) Cytokines
7. **Which of the following is an effect of cyclooxygenase inhibitors such as aspirin?**
   A) Inhibition of phospholipase
   B) Reduction of prostaglandin production
   C) Inhibition of leukotriene synthesis
   D) Increased synthesis of arachidonic acid
8. **What is the major function of chemokines in inflammation?**
   A) Stimulating vasodilation
   B) Recruiting leukocytes to the site of injury
   C) Inhibiting the release of pro-inflammatory mediators
   D) Enhancing platelet aggregation
9. **Which of the following is a key function of IL-6 in acute inflammation?**
   A) Inducing endothelial adhesion molecule expression
   B) Stimulating fever and acute-phase protein production

C) Recruiting neutrophils to the site of injury
D) Promoting apoptosis in neutrophils

10. **Which of the following is a potential pathological effect of excessive TNF in systemic inflammation?**
    A) Increased WBC count
    B) Decreased cardiac output leading to heart failure
    C) Inhibition of leukocyte migration
    D) Reduction in vascular permeability
11. **What is the role of the complement system in inflammation?**
    A) Directly kill pathogens via membrane attack complex (MAC)
    B) Inhibit phagocytosis
    C) Degrade cytokines
    D) Promote leukocyte apoptosis
12. **What is the primary action of C3b in the complement system?**
    A) Trigger vasodilation
    B) Opsonize pathogens to enhance phagocytosis
    C) Activate neutrophils
    D) Produce cytokines
13. **Which complement protein is considered the "gatekeeper" in the activation of the complement cascade?**
    A) C1
    B) C3
    C) C9
    D) C5
14. **What is the main clinical relevance of measuring C3 levels in the complement system?**
    A) To assess bacterial infection
    B) To evaluate the degree of inflammation and immune function
    C) To track cytokine storm effects
    D) To determine the balance between PGI2 and TXA2
15. **Which of the following cytokines is most associated with the systemic effects of fever and acute-phase responses?**
    A) TNF
    B) IL-1
    C) IL-6
    D) IL-17

## Answers:

1. D
2. D
3. B
4. A
5. C
6. C
7. B
8. B
9. B

10. A
11. B
12. B
13. B
14. B
15. C

**16. Which of the following is a key mediator for terminating the inflammation response?**
A) TNF
B) IL-1
C) IL-10
D) Prostaglandins

**17. Which of the following is NOT a characteristic of inflammatory mediators?**
A) Produced in rapid bursts
B) Released continuously throughout the response
C) Stimulus-dependent release
D) Short half-life

**18. Which mediator is known for inhibiting TNF and other inflammatory mediators during the repair phase?**
A) IL-17
B) TGF-ß
C) IL-6
D) IL-1

**19. Which inflammatory mediator is primarily produced by platelets and causes vasoconstriction and platelet aggregation?**
A) Thromboxane A2 (TXA2)
B) Prostacyclin (PGI2)
C) PGE2
D) IL-6

**20. Which of the following is the main function of neutrophils (PMNs) in acute inflammation?**
A) Release cytokines to amplify inflammation
B) Perform phagocytosis of pathogens
C) Repair damaged tissue
D) Produce antibodies

**21. Which mediator is specifically involved in leukocyte chemotaxis and the recruitment of immune cells to inflammation sites?**
A) IL-1
B) Chemokines
C) Thromboxane A2
D) PGE2

**22. What is the role of corticosteroids (e.g., cortisone) in inflammation?**
A) Inhibit COX enzymes to block prostaglandin production
B) Inhibit phospholipase to block arachidonic acid production
C) Enhance leukotriene production
D) Promote the synthesis of IL-6

**23. In the context of arachidonic acid metabolism, which of the following is an anti-inflammatory drug that inhibits the COX pathway?**
A) Aspirin
B) Cortisone
C) Leukotriene inhibitors
D) Anti-IL-6 drugs

**24. Which of the following cytokines is primarily responsible for inducing fever during inflammation?**
A) TNF
B) IL-1
C) IL-6
D) IL-17

**25. How does the complement system aid in the immune response?**
A) By directly inducing apoptosis of infected cells
B) By initiating an inflammatory response through cytokine release
C) By tagging pathogens for phagocytosis
D) By stimulating antibody production

**26. What is the main action of C3b in the complement system?**
A) Induce vasodilation
B) Enhance phagocytosis
C) Promote platelet aggregation
D) Increase vascular permeability

**27. Which inflammatory mediator plays a key role in increasing vascular permeability and smooth muscle contraction in the bronchi during an asthma attack?**
A) Prostaglandins
B) Leukotrienes
C) Histamine
D) IL-10

**28. What is the primary function of chemokines in inflammation?**
A) Induce fever
B) Promote smooth muscle contraction
C) Recruit and activate leukocytes
D) Cause tissue damage via necrosis

**29. What is the effect of excessive TNF levels in acute inflammation?**
A) Stimulates vasodilation and decreases vascular permeability
B) Increases cardiac output and prevents thrombosis

C) Reduces immune cell migration
D) Decreases cardiac output, potentially leading to heart failure

**30. Which of the following is a systemic effect caused by cytokines in inflammation?**
A) Inducing apoptosis in infected cells
B) Stimulating CRP production by the liver
C) Blocking the release of leukotrienes
D) Promoting the contraction of smooth muscles in the uterus

## Answers:

16. C
17. B
18. B
19. A
20. B
21. B
22. B
23. A
24. B
25. C
26. B
27. B
28. C
29. D
30. B

**31. Which of the following best describes the role of Transforming Growth Factor-Beta (TGF-ß) in inflammation resolution?**
A) It amplifies the inflammatory response by increasing cytokine production.
B) It triggers apoptosis of inflammatory cells to prevent prolonged inflammation.
C) It inhibits the production and action of inflammatory mediators during the repair phase.
D) It promotes the activation of complement proteins during the immune response.

**32. What mechanism primarily ensures the resolution of inflammation by limiting the lifespan of neutrophils (PMNs)?**
A) Phagocytosis of apoptotic neutrophils by macrophages
B) Production of anti-inflammatory cytokines like IL-10
C) Release of TGF-ß, which induces neutrophil apoptosis
D) Enzymatic degradation of mediators released by neutrophils

**33. In the context of acute inflammation, which of the following is NOT a consequence of excessive production of IL-1 and TNF?**
A) Endothelial activation leading to increased leukocyte adhesion
B) Systemic fever and acute-phase protein production
C) Inhibition of vascular permeability and reduced edema
D) Worsening tissue damage and increased risk of organ failure

**34. Which of the following cytokines is directly responsible for sustaining chronic inflammation by continuously recruiting neutrophils and monocytes?**
A) IL-12
B) IL-17
C) IL-6
D) TNF

**35. In the complement system, which complement protein is considered the "gatekeeper" and is essential for the activation of all three complement pathways?**
A) C3
B) C1
C) C5
C) C9

**36. Which of the following enzymes plays a critical role in releasing arachidonic acid from membrane phospholipids during the inflammatory response?**
A) Cyclooxygenase (COX)
B) Phospholipase A2
C) Lipoxygenase
D) 5-lipoxygenase

**37. What is the primary clinical effect of inhibiting both COX-1 and COX-2 enzymes in acute inflammation?**
A) Increased production of leukotrienes
B) Reduction in pain, fever, and vasodilation
C) Enhanced neutrophil migration to the site of injury
D) Increased vascular permeability and edema

**38. Which cytokine is primarily responsible for stimulating the liver to produce acute-phase proteins like C-reactive protein (CRP) during inflammation?**
A) IL-10
B) IL-6
C) TNF
D) IL-1

**39. What is the role of the complement protein C5a in the inflammatory response?**
A) It acts as a potent opsonin to enhance phagocytosis.
B) It induces vasodilation and increases vascular permeability.
C) It forms the membrane attack complex (MAC) to directly kill pathogens.
D) It triggers the release of histamine from mast cells.

**40. Which of the following describes the function of prostacyclin (PGI2) in the inflammatory response?**
A) It acts as a vasoconstrictor and promotes platelet aggregation.
B) It inhibits platelet aggregation and promotes vasodilation.
C) It increases vascular permeability and recruits neutrophils.
D) It stimulates the release of TNF from macrophages.

**41. In chronic inflammation, which of the following cytokines is crucial for macrophage activation and the formation of granulomas?**
A) IFN-γ
B) IL-10
C) IL-1
D) IL-4

**42. What is the mechanism by which corticosteroids (e.g., cortisone) reduce inflammation at the molecular level?**
A) They inhibit the activation of C3 in the complement system.
B) They block the synthesis of arachidonic acid by inhibiting phospholipase.
C) They block the release of IL-6 from activated macrophages.
D) They stimulate the production of anti-inflammatory cytokines like IL-10.

**43. Which of the following is a major mediator responsible for the increased vascular permeability observed in inflammation?**
A) Histamine
B) Prostaglandin E2 (PGE2)
C) Complement C3b
D) Leukotrienes (C4, D4, E4)

**44. In an acute inflammatory response, which of the following is the main source of IL-6 that induces fever and stimulates the liver to produce acute-phase proteins?**
A) Endothelial cells
B) Mast cells
C) Macrophages
D) T lymphocytes

**45. What is the primary effect of the imbalance between prostacyclin (PGI2) and thromboxane A2 (TXA2) in conditions like ischemic heart disease?**
A) Enhanced platelet aggregation and vasoconstriction, contributing to thrombosis
B) Increased vasodilation and reduced vascular permeability
C) Enhanced bacterial clearance and resolution of inflammation
D) Decreased neutrophil activation and recruitment to the site of injury

## Answers:

31. C
32. A
33. C
34. B
35. A
36. B
37. B
38. B
39. B

40. B
41. A
42. B
43. D
44. C
45. A

**46. Which of the following molecules is primarily involved in the acute-phase response, stimulating the liver to produce proteins such as C-reactive protein (CRP)?**
A) IL-1
B) IL-6
C) TNF
D) IFN-γ

**47. During chronic inflammation, which of the following cells plays a central role in the formation of granulomas and the persistence of inflammation?**
A) B lymphocytes
B) T lymphocytes
C) Macrophages
D) Neutrophils

**48. What is the primary function of the membrane attack complex (MAC) in the complement system?**
A) To neutralize bacterial toxins
B) To form pores in the membranes of pathogens, causing cell lysis
C) To enhance the production of IL-6 and TNF
D) To bind to immune complexes and remove them from circulation

**49. In the resolution phase of inflammation, what role does the enzyme 15-lipoxygenase play in the body's response?**
A) It inhibits the production of cytokines like IL-6 and TNF.
B) It produces lipoxins that promote the resolution of inflammation.
C) It increases the synthesis of prostaglandins to prolong inflammation.
D) It enhances the production of chemotactic factors to attract immune cells.

**50. What is the primary purpose of the acute-phase response triggered by pro-inflammatory cytokines?**
A) To decrease the synthesis of acute-phase proteins by the liver
B) To activate the adaptive immune system and initiate antibody production
C) To enhance tissue repair and restore homeostasis
D) To inhibit the recruitment of immune cells to the site of injury

**51. Which of the following is a direct consequence of complement activation via the classical pathway?**
A) The formation of the membrane attack complex (MAC)
B) The release of anti-inflammatory cytokines
C) The secretion of histamine from mast cells
D) The inhibition of IL-1 and TNF production

**52. In the context of autoimmune diseases, what is the role of antibodies against complement proteins?**
A) They increase the clearance of immune complexes.
B) They enhance complement activation, exacerbating inflammation.
C) They inhibit the formation of the membrane attack complex (MAC).
D) They promote the resolution of inflammation.

**53. Which of the following is the primary function of the cytokine IL-10 during the resolution phase of inflammation?**
A) It enhances the production of pro-inflammatory cytokines.
B) It activates neutrophils and macrophages to clear pathogens.
C) It suppresses the production of pro-inflammatory cytokines to limit tissue damage.
D) It induces the synthesis of prostaglandins to prolong the inflammatory response.

**54. How does TNF contribute to the pathophysiology of sepsis during systemic inflammation?**
A) By increasing vascular permeability and promoting edema
B) By enhancing the clearance of pathogens from tissues
C) By suppressing the activation of complement proteins
D) By decreasing neutrophil migration to the infection site

**55. Which of the following best describes the function of leukotrienes in the inflammatory response?**
A) They inhibit the formation of prostaglandins and thromboxanes.
B) They act as powerful vasodilators and promote edema.
C) They promote the recruitment of leukocytes to the site of injury.
D) They decrease vascular permeability and inhibit neutrophil migration.

**56. Which of the following is an important characteristic of chronic inflammation compared to acute inflammation?**
A) The presence of a predominant neutrophil infiltrate
B) The involvement of adaptive immunity and tissue remodeling
C) The rapid resolution of inflammation and tissue repair
D) The absence of macrophages and T lymphocytes

**57. Which of the following cytokines is most closely associated with the "Th1" inflammatory response, characterized by the activation of macrophages and cytotoxic T cells?**
A) IL-4
B) IL-2
C) IFN-γ
D) IL-17

**58. What is the primary mechanism by which corticosteroids inhibit inflammation?**
A) By blocking histamine release from mast cells
B) By inhibiting cyclooxygenase (COX) activity
C) By suppressing the transcription of pro-inflammatory cytokines
D) By promoting the resolution of inflammation via lipid mediators

**59. Which of the following is a primary outcome of the activation of the NLRP3 inflammasome in macrophages during infection or injury?**
A) Enhanced production of IL-10 and suppression of inflammation
B) Induction of pyroptosis and the release of IL-1β
C) Activation of T-helper cells to produce IFN-γ
D) Increased production of reactive oxygen species (ROS) to kill pathogens

**60. Which of the following molecules primarily functions as an "opsonin" to enhance the phagocytosis of pathogens during the inflammatory response?**
A) C3b
B) Histamine
C) Interleukin-6 (IL-6)
D) Tumor necrosis factor (TNF)

## Answers:

46. B
47. C
48. B
49. B
50. C
51. A
52. C
53. C
54. A
55. C
56. B
57. C
58. C
59. B
60. A

**61. Which of the following is the most potent mediator of vasodilation in the early stages of inflammation?**

A) Prostaglandin E2 (PGE2)
B) Histamine
C) Nitric oxide (NO)
D) Leukotriene B4 (LTB4)

**62. In a patient with a bacterial infection, what is the primary function of IL-12 in the immune response?**

A) Induces the differentiation of Th1 cells and enhances the production of IFN-γ
B) Stimulates the production of acute-phase proteins from the liver

C) Suppresses the activity of Treg cells to promote inflammation
D) Inhibits the activation of macrophages and neutrophils

**63. Which of the following best describes the role of TGF-β in the resolution phase of inflammation?**

A) Stimulates the production of pro-inflammatory cytokines and chemokines
B) Induces the apoptosis of inflammatory cells and promotes tissue repair
C) Enhances the activation of macrophages to clear pathogens
D) Promotes the production of prostaglandins and leukotrienes

**64. What is the key difference between the classical and alternative pathways of complement activation?**

A) The classical pathway is antibody-dependent, whereas the alternative pathway is antibody-independent
B) The classical pathway requires the activation of C3b, whereas the alternative pathway does not
C) The alternative pathway generates the membrane attack complex (MAC) more rapidly than the classical pathway
D) The classical pathway uses C5 convertase, whereas the alternative pathway does not activate C5

**65. Which of the following is a consequence of excessive TNF-α production during systemic inflammation?**

A) Activation of anti-inflammatory pathways and reduction in immune cell activation
B) Suppression of macrophage activity and reduced neutrophil recruitment
C) Systemic vasodilation leading to shock and organ dysfunction
D) Inhibition of the acute-phase response and cytokine production

**66. In response to infection, which of the following is primarily responsible for the fever associated with inflammation?**

A) IL-6
B) Prostaglandin E2 (PGE2)
C) Nitric oxide (NO)
D) C-reactive protein (CRP)

**67. What is the role of interferons (IFN-α and IFN-β) in the antiviral immune response?**

A) They activate neutrophils to phagocytose virus-infected cells
B) They enhance the production of IL-6 and TNF to promote inflammation
C) They inhibit viral replication and enhance the antiviral state in surrounding cells
D) They promote the differentiation of T helper cells into Th17 cells

**68. During inflammation, which of the following molecules is primarily responsible for recruiting neutrophils to the site of injury or infection?**

A) Interleukin-1 (IL-1)
B) Interleukin-8 (IL-8)
C) Tumor necrosis factor-alpha (TNF-α)
D) C3b

**69. What is the effect of lipoxins during the resolution of inflammation?**

A) They enhance neutrophil recruitment to the site of infection
B) They suppress the activation of macrophages to limit tissue damage
C) They inhibit the formation of the membrane attack complex (MAC)
D) They increase the production of prostaglandins to prolong inflammation

**70. Which of the following is the most important action of the acute-phase reactant C-reactive protein (CRP)?**

A) It binds to damaged tissues and promotes the clearance of dead cells.
B) It stimulates the production of pro-inflammatory cytokines by macrophages.
C) It enhances the phagocytosis of pathogens by opsonizing them.
D) It activates the alternative complement pathway to kill pathogens.

**71. What is the main function of the enzyme cyclooxygenase (COX-2) during inflammation?**

A) To inhibit the synthesis of leukotrienes and limit inflammation
B) To degrade inflammatory cytokines such as TNF-α
C) To catalyze the production of prostaglandins, particularly PGE2, to mediate vasodilation and fever
D) To enhance the resolution of inflammation by producing lipoxins

**72. Which of the following cytokines is primarily involved in the recruitment of neutrophils during acute inflammation?**

A) IL-10
B) IL-6
C) IL-8
D) IFN-γ

**73. How do corticosteroids inhibit inflammation at the molecular level?**

A) By blocking the activity of neutrophils and T cells
B) By inhibiting the transcription of pro-inflammatory genes, including cytokines like TNF-α and IL-1
C) By enhancing the activity of anti-inflammatory cytokines like IL-10
D) By increasing the production of reactive oxygen species (ROS) in immune cells

**74. Which of the following is a key feature of granulomatous inflammation?**

A) The presence of eosinophils and neutrophils at the site of infection
B) The accumulation of activated macrophages that form multinucleated giant cells
C) The predominant presence of B lymphocytes and plasma cells
D) The absence of fibrosis or scarring in the affected tissue

**75. Which complement component is most directly responsible for the generation of anaphylatoxins that contribute to vasodilation and inflammation?**

A) C3b
B) C4b
C) C5a
D) C1q

## Answers:

61. B
62. A
63. B
64. A
65. C
66. B
67. C
68. B
69. B
70. C
71. C
72. C
73. B
74. B
75. C

**76. Which of the following is the primary mechanism by which neutrophils clear pathogens in the acute phase of inflammation?**

A) Phagocytosis and subsequent intracellular killing via reactive oxygen species (ROS)
B) Secretion of cytokines that recruit additional immune cells
C) Formation of extracellular traps (NETs) to capture and neutralize pathogens
D) Activation of the complement system to directly kill pathogens

**77. Which of the following statements accurately describes the role of dendritic cells in initiating adaptive immune responses during inflammation?**

A) Dendritic cells are primarily involved in the phagocytosis of pathogens but do not play a role in antigen presentation.

B) Dendritic cells activate T cells by presenting processed antigen on MHC II molecules to naive T cells, initiating the adaptive immune response.
C) Dendritic cells inhibit the function of T-helper cells and promote regulatory T cell differentiation to suppress inflammation.
D) Dendritic cells are the primary source of pro-inflammatory cytokines during inflammation but do not influence the development of memory T cells.

**78. Which of the following cytokines is involved in the induction of acute-phase proteins during inflammation?**

A) IL-1
B) TNF-α
C) IL-6
D) IL-10

**79. What is the key difference between the effects of IL-1 and TNF-α in acute inflammation?**

A) IL-1 primarily induces fever and endothelial activation, while TNF-α leads to increased vascular permeability and recruitment of neutrophils.
B) IL-1 activates macrophages, whereas TNF-α suppresses macrophage function and limits inflammation.
C) IL-1 induces a Th2 response, while TNF-α induces a Th1 response.
D) IL-1 is responsible for resolving inflammation, while TNF-α exacerbates inflammation.

**80. In chronic inflammation, which of the following cell types is most responsible for the persistence of tissue damage and fibrosis?**

A) Neutrophils
B) Macrophages
C) Eosinophils
D) T lymphocytes

**81. Which of the following molecules is involved in the resolution of inflammation by promoting the clearance of apoptotic cells?**

A) TGF-β
B) IL-10
C) Annexin A1
D) C3b

**82. Which of the following is the primary action of the complement protein C5a during inflammation?**

A) It acts as an opsonin to promote phagocytosis.
B) It induces neutrophil chemotaxis and activation.
C) It directly lyses pathogens through the formation of the membrane attack complex (MAC).
D) It inhibits the activation of macrophages and neutrophils.

**83. How does IL-17 contribute to the inflammatory response in autoimmune diseases?**

A) It inhibits the differentiation of Th1 and Th2 cells to prevent excessive inflammation.
B) It stimulates the production of pro-inflammatory cytokines and chemokines from various cells, including macrophages and epithelial cells.
C) It suppresses the activation of Treg cells, promoting immune tolerance.
D) It induces the differentiation of naïve T cells into Th2 cells.

**84. Which of the following is the primary role of nitric oxide (NO) in the inflammatory response?**

A) NO enhances the function of neutrophils by promoting the formation of neutrophil extracellular traps (NETs).
B) NO causes vasoconstriction and limits the delivery of immune cells to the site of infection.
C) NO has antimicrobial effects and also helps modulate vascular tone by causing vasodilation.
D) NO promotes the activation of complement proteins to enhance opsonization and pathogen clearance.

**85. What is the mechanism by which acute-phase reactants like C-reactive protein (CRP) enhance immune function during inflammation?**

A) By binding to bacterial surfaces and enhancing the phagocytosis of pathogens through opsonization.
B) By activating the alternative complement pathway, directly leading to pathogen lysis.
C) By stimulating the production of TNF-α and IL-6 to increase the inflammatory response.
D) By inhibiting the activity of neutrophils and macrophages to prevent tissue damage.

**86. Which of the following best describes the function of matrix metalloproteinases (MMPs) during inflammation?**

A) MMPs break down extracellular matrix components to facilitate tissue repair and remodeling after inflammation resolution.
B) MMPs degrade microbial components to limit pathogen survival during infection.
C) MMPs promote the formation of granulomas by activating macrophages and T cells.
D) MMPs inhibit the proliferation of fibroblasts to prevent fibrosis during chronic inflammation.

**87. What is the role of the inflammasome in the regulation of inflammation?**

A) The inflammasome promotes the release of IL-10 to limit inflammation.
B) The inflammasome activates caspase-1, leading to the cleavage and activation of IL-1β and IL-18, potent pro-inflammatory cytokines.
C) The inflammasome suppresses the release of IL-6 and TNF-α to resolve inflammation.
D) The inflammasome induces apoptosis of macrophages to prevent excessive tissue damage.

**88. Which of the following is a direct effect of prostaglandin E2 (PGE2) during inflammation?**

A) It promotes the recruitment of neutrophils to the site of infection.
B) It stimulates the formation of antibodies by B cells.
C) It induces vasodilation and sensitizes pain receptors, contributing to fever and pain.
D) It inhibits the activity of natural killer (NK) cells to reduce inflammation.

**89. Which of the following is the major mediator responsible for the tissue damage seen in autoimmune diseases, such as rheumatoid arthritis?**

A) Autoantibodies targeting self-antigens
B) Neutrophils producing reactive oxygen species (ROS)
C) Activated T cells releasing pro-inflammatory cytokines like TNF-α and IL-1
D) Macrophages phagocytosing normal tissue

**90. What is the role of IFN-γ in the immune response to intracellular pathogens?**

A) IFN-γ enhances the differentiation of naive CD4+ T cells into Th2 cells.
B) IFN-γ activates macrophages, increasing their ability to kill intracellular pathogens through increased oxidative burst.
C) IFN-γ inhibits the activation of CD8+ T cells and suppresses cytotoxic T cell responses.
D) IFN-γ stimulates B cells to produce antibodies against extracellular pathogens.

## Answers:

76. A
77. B
78. C
79. A
80. B
81. C
82. B
83. B
84. C
85. A
86. A
87. B
88. C
89. C
90. B

**Case 1: a 35-year-old woman, presents with fever, pain, redness, and swelling at a wound site after stepping on a nail. On physical examination, there is erythema, increased warmth, and local tenderness at the injury site. Laboratory tests show an elevated white blood cell (WBC) count and increased C-reactive protein (CRP).**

**Question 1:**

**Which of the following is most likely to be activated as part of the innate immune response in this patient's condition?**
A) Adaptive immune response (T and B lymphocytes)
B) Complement system, specifically the classical pathway
C) TLRs (Toll-like receptors) recognizing pathogen-associated molecular patterns (PAMPs)
D) Th2 cells leading to eosinophil activation

**Question 2:**

**Which of the following cytokines is most likely responsible for the fever and systemic symptoms in this patient?**
A) IL-1β
B) IL-6
C) TNF-α
D) IL-10

**Question 3:**

**Which of the following molecules is primarily responsible for the increased vascular permeability and edema seen at the injury site in this patient?**
A) Histamine
B) Prostaglandin E2 (PGE2)
C) Leukotriene B4 (LTB4)
D) C3b

**Case 2: a 50-year-old male with a history of rheumatoid arthritis (RA), presents with joint pain, morning stiffness, and swelling in his hands and knees. On examination, his joints are swollen and tender. His laboratory results reveal an elevated ESR, CRP, and positive rheumatoid factor.**

**Question 4:**

**In this patient with rheumatoid arthritis, which of the following is the most likely mediator of the chronic inflammation and tissue damage in the joints?**
A) IL-4
B) TNF-α
C) IL-10
D) IFN-γ

**Question 5:**

**Which immune cells are most likely involved in the chronic inflammatory response in the joints of this patient?**
A) Neutrophils
B) T-helper 17 (Th17) cells
C) Natural killer (NK) cells
D) Eosinophils

**Question 6:**

**Which of the following cytokines is primarily responsible for the recruitment and activation of macrophages in the inflamed joints of this patient?**
A) IL-12
B) IL-6
C) IL-1β
D) TGF-β

**Case 3: a 60-year-old woman, is admitted to the hospital with signs of sepsis following a urinary tract infection. She is hypotensive, febrile, and has an elevated heart rate. Laboratory tests show an elevated white blood cell count and significantly increased procalcitonin levels. Blood cultures grow Escherichia coli.**

**Question 7:**

**Which of the following best describes the primary mechanism underlying the systemic symptoms in this patient with sepsis?**
A) The release of bacterial endotoxins triggering excessive activation of the complement system.
B) The activation of the adaptive immune response and subsequent antibody production.
C) The excessive release of pro-inflammatory cytokines like TNF-α, IL-1β, and IL-6.
D) The suppression of the innate immune response leading to decreased pathogen clearance.

**Question 8:**

**Which of the following cytokines is most likely responsible for the hypotension and vascular changes (e.g., vasodilation) observed in this patient?**
A) IL-1β
B) IL-6
C) TNF-α
D) IFN-γ

**Question 9:**

**Which of the following is the most likely outcome if the inflammatory response is not appropriately controlled in this patient?**
A) Sepsis-induced organ dysfunction and multi-organ failure
B) Resolution of inflammation with complete recovery
C) Inhibition of leukocyte recruitment and pathogen clearance
D) Formation of protective granulomas to isolate the pathogen

**Case 4: a 28-year-old female with a history of asthma, presents to the emergency department with wheezing, difficulty breathing, and increased mucus production. She reports exposure to dust mites at home. On examination, she has a prolonged expiratory phase and wheezing on auscultation. Her spirometry shows reduced forced expiratory volume (FEV1).**

**Question 10:**

**Which of the following immune responses is most likely driving the airway inflammation in this patient's asthma exacerbation?**
A) Th1-mediated response with macrophage activation
B) Th2-mediated response with eosinophil activation and IgE production
C) Activation of the complement system and neutrophil recruitment
D) A B cell-mediated response producing high levels of antibodies against viral pathogens

**Question 11:**

**Which of the following is the most likely mediator responsible for bronchoconstriction in this patient's asthma exacerbation?**
A) Histamine
B) Leukotriene D4 (LTD4)
C) Prostaglandin E2 (PGE2)
D) Interferon-gamma (IFN-γ)

**Question 12:**

**Which of the following is most likely to be elevated in the serum of this patient during an acute asthma exacerbation?**
A) C-reactive protein (CRP)
B) Interleukin-4 (IL-4)
C) Neutrophil elastase
D) Interleukin-17 (IL-17)

## Answers:

**Case 1:**

1. C
2. A
3. A

**Case 2:**

4. B
5. B
6. C

**Case 3: Systemic Inflammation in Sepsis**

7. C
8. C
9. A

**Case 4:**

10. B
11. B
12. B

**Done By: Khaled Ghanayem**