TABLE 86-2 CYTOKINES

CYTOKINE	CELLULAR SOURCE	TARGETS	FUNCTION	RECEPTOR
IL-1α	Epi, fibroblasts, damaged or dying cells	Wide variety	"Dual function" cytokine involved in initiating inflammatory response and modifying gene expression	CD121a or CD121b
IL-1β	M, B	T, B, M, End, other	Leukocyte activation, increases endothelium adhesion	CD121a or CD121b
IL-2	T	TB, NK, M, oligo	T cell proliferation, regulation	CD122/CD25
IL-3	T,* Mas, Eos, NK, End	Ery, G	Proliferation and differentiation of hematopoietic precursors	CD123/CDw131
IL-4	Mas, T, M	B, T, End	Differentiation of T_H2 and B cells	CD124/CD132
IL-5	Mas, T, Eos	Eos, B	Growth differentiation of B cells and eosinophils	CD125/CDw131
IL-6	T, B, M, astrocytes, End	T, B, others	Hematopoiesis, differentiation, inflammation	CD126/CD130
IL-7	Bone marrow and thymic stroma	pB, pT	Pre/pro-B proliferation, T, upregulation of proinflammatory cytokines	CD127/CD132
IL-8	M, L, others	PMN, Bas, L	Chemoattractant	CD128
IL-9	T_H2*	T, B	Potentiates production of IgM, IgG, IgE	
IL-10	$CD8^+$ T,* T_H2, (B),† M	T, B, Mas, M	Inhibits IFN-γ,TNF-β, IL-2 by T_H1 cells, DTH, stimulates T_H2	CD210
IL-11		Bone marrow stroma	Osteoclast formation	
IL-12	DC, B, T	T, NK	Potentiates IFN-γ and TNF-α production by T and NK, downregulates IL-10	CD212
IL-13	T_H2,* Mas, NK	T_H2, B, M	T_H2 modulator, downregulated IL-1, IL-6, IL-8, IL-10, IL-12	
IL-14	T	B*	Stimulates proliferation, inhibits Ig secretion	
IL-15	M, Epi	T, B*	Proliferation	
IL-16	Eos, $CD8^+$ T*	$CD4^+$ T*	CD4* chemoattractant	
IL-17	(T)	Epi, End, others	Osteoclastogenesis, angiogenesis	
IL-18	M	T_H1, NK	Induces IFN-γ production, enhances NK activity	
IL-32	Tn, NK, Epi	Wide variety	Proinflammatory	
TGF-β	Eos, others	Many cell types	Anti-inflammatory, promotes wound healing	
TNF-α	M,* PMN, T, B, NK	M, PMN, T, End, others	Mediator of inflammatory reactions	CD120a and CD120b
TNF-β	L	Wide variety	Mediator of inflammatory reactions	CD120a and CD120b
IFN-α	L, Epi, fibroblasts	Wide variety	Upregulates MHC class I, inhibits viral proliferation	
IFN-β	Epi, fibroblasts	Wide variety	Upregulates MHC class I, inhibits viral proliferation	
IFN-γ	$CD8^+$,* ($CD4^+$*), NK	T, B, M, NK, End	Antiviral, antiparasitic, inhibits proliferation, enhances MHC class I and II expression	CD119
M-CSF	L, M, G, End, Epi, others	M	Growth and differentiation of Ms	CD115
G-CSF	T,* M, End	G	Growth and differentiation of Gs	
GM-CSF	T, M, End, Mas	pG, pMye	Stimulates growth and differentiation of Gs and Mye lineage cells	CD116
MIF	M	M	Antiapoptotic activity for macrophages, promotes M survival	

From Doan T, Melvold R, Viselli S, Waltenbaugh C: Immunology, ed 2, Philadelphia, 2012, Lippincott, Williams & Wilkins.

B, B cells; Bas, basophils; CSF, colony-stimulating factor; DC, dendritic cells; DTH, delayed-type hypersensitivity; End, endothelium; Eos eosinophil; Epi, epithelium; Ery, erythrocytes; G, granulocytes; IFN, interferon; IL, interleukin; L, lymphocytes; M, macrophage; Mas, mast cells; MHC, major histocompatibility complex; MIF, macrophage migration inhibitory factor; Mye, myeloid; NK, natural killer cells; p, precursor; PMN, neutrophils; oligo, oligodendrocytes; T, T cell; TGF, transforming growth factor; T_H, helper T cell subset; TNF, tumor necrosis factor.

*Activated cells.

†Parentheses indicate that only a subset of the designated cell types produce the cytokine.

TABLE 86-3 TOLL-LIKE RECEPTORS

PRR	PAMP	PATHOGEN	PRR EXPRESSION
TLR2	Peptidoglycan	Gram-positive bacteria	mDC
TLR3	Double-stranded RNA	Viruses	mDC
TLR4	Lipopolysaccharide	Gram-negative bacteria	mDC
TLR7	Single-stranded RNA	Viruses	pDC
TLR9	Double-stranded DNA	Viruses	pDC

From Kumar P, Clark M, editors: Kumar and Clark's clinical medicine, ed 8, London, 2012, Elsevier.

mDC, Mature dendritic cell; PAMP, pathogen-associated molecular pattern; pDC, precursor dendritic cell; PRR, pattern recognition receptor; TLR, toll-like receptor.

injury, or dysfunctional adaptive immunity (e.g., autoantibodies). The response includes inflammatory molecules as previously described and tissue and migrating leukocytes. Neutrophils are central to the clinical manifestations of inflammation in tissue, and patients with neutropenia often lack the signs of inflammation at the site of serious infection.

Neutrophils are bone marrow–derived phagocytes whose production is greatly stimulated by infection through the action of macrophage-produced growth factors, including granulocyte colony-stimulating factor (G-CSF) and granulocyte-macrophage colony-stimulating factor (GM-CSF). Neutrophils circulate in blood, are attracted to sites of inflammation, and are activated