Table 3.5 : Principal Mediators of Inflammation

* Source :

Mast cells → Histamine, Prostglandins, Leukotrienes, Cytokines, Platelet-AF

Leukocytes → Prostglandins, Leukotrienes, Platelet-AF

Macrophage → Cytokines, Chemokines (activated macrophage)

Plasma (in liver) → Complement, Kinins

Endothelial cells → Cytokines

Basophils Platelet → Histamine

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* Action :

Vasodilation → Histamine, Prostglandins, Platelet-AF, Complement (mast cell stimulation), Kinins

↑ vascular permeability → Histamine, Leukotrienes, Platelet-AF, Kinins

Endothelial activation → Histamine, Cytokines (expression of adhesion molecule)

Pain & Fever → Prostglandins

↳ Kinins ↳ Cytokines

Chemotaxis → Leukotrienes, Platelet-AF, Complement, Cytokines

Leukocytes → Activation & Adhesion

Adhesion

Activation

Cytokines (TNF, IL-1, IL-6)	Macrophages, endothelial cells, mast cells	Local: endothelial activation (expression of adhesion molecules). Systemic: fever, metabolic abnormalities, hypotension (shock)

Platelet-activating factor	Leukocytes, mast cells	Vasodilation, increased vascular permeability, leukocyte adhesion, chemotaxis, degranulation, oxidative burst
Complement	Plasma (produced in liver)	Leukocyte chemotaxis and activation, direct target killing (membrane attack complex), vasodilation (mast cell stimulation)
Kinins	Plasma (produced in liver)	Increased vascular permeability, smooth muscle contraction, vasodilation, pain