

# Table 3.5: Principal Mediators of Inflammation

\* Source 8

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

Leukocytes → Prostaglandins, 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, Prostaglandins, Platelet-AF, Complement, Kinins (mast cell stimulation)

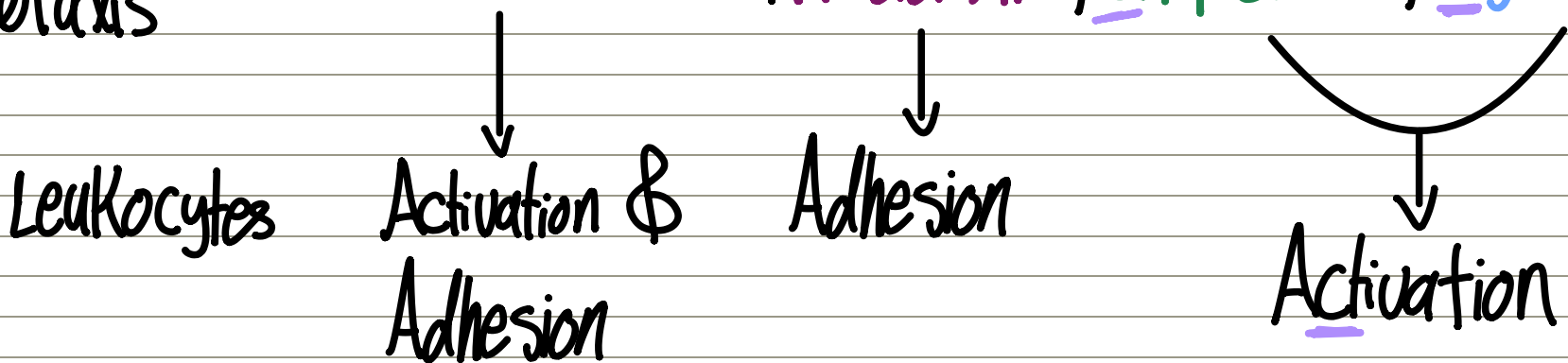
↑ vascular permeability → Histamine, Leukotrienes, Platelet-AF, Kinins

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

Pain & Fever → Prostaglandins

↳ Kinins    ↳ Cytokines

Chemotaxis → Leukotrienes, Platelet-AF, Complement, Cytokines



<b>Cytokines</b> (TNF, IL-1, IL-6)	Macrophages, endothelial cells, mast cells	Local: endothelial activation (expression of adhesion molecules). Systemic: fever, metabolic abnormalities, hypotension (shock)
<b>Platelet-activating factor</b>	Leukocytes, mast cells	Vasodilation, increased vascular permeability, leukocyte adhesion, chemotaxis, degranulation, oxidative burst
<b>Complement</b>	Plasma (produced in liver)	Leukocyte chemotaxis and activation, direct target killing (membrane attack complex), vasodilation (mast cell stimulation)
<b>Kinins</b>	Plasma (produced in liver)	Increased vascular permeability, smooth muscle contraction, vasodilation, pain