

TABLE 3.5 Principal Mediators of Inflammation

Mediator	Source	Action
Histamine	Mast cells, basophils, platelets	<u>Vasodilation, increased vascular permeability, endothelial activation</u> (recall initial vascular Phase)
Prostaglandins	Mast cells, leukocytes	Vasodilation, pain, fever <i>why we take aspirin?</i>
<u>Leukotrienes</u>	Mast cells, leukocytes	<u>Increased vascular permeability, chemotaxis, leukocyte adhesion, and activation</u> <i>تسبب الالتهاب</i>
Cytokines (TNF, IL-1, IL-6)	Macrophages, endothelial cells, mast cells	<u>Local: endothelial activation</u> (expression of adhesion molecules). <u>Systemic: fever, metabolic abnormalities, hypotension (shock)</u>
Chemokines	Leukocytes, <u>activated macrophages</u>	<u>Chemotaxis, leukocyte activation</u> <i>تسبب الهجرة</i>
Platelet-activating factor	<u>Leukocytes, mast cells</u>	<u>Vasodilation, increased vascular permeability, leukocyte adhesion, chemotaxis, degranulation, oxidative burst</u> <i>تسبب الالتهاب</i>
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

