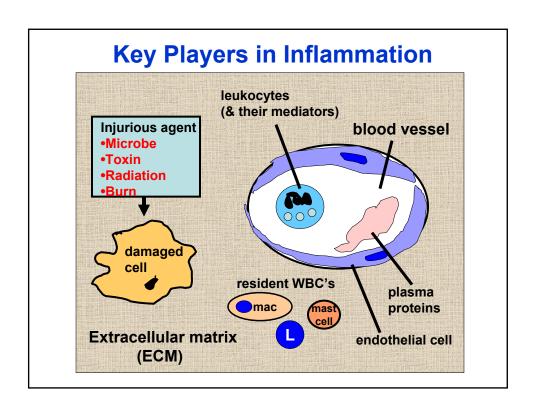
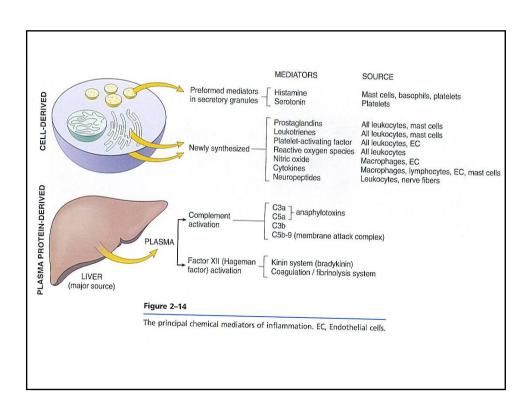
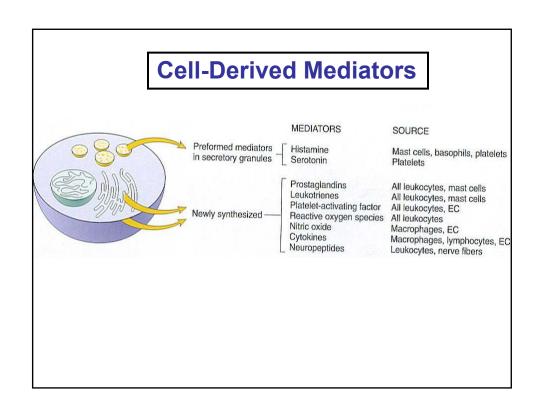
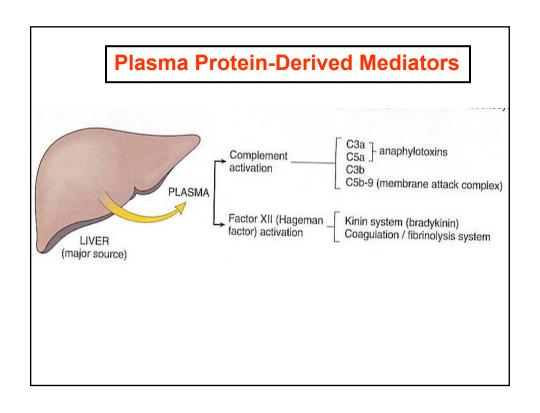
Inflammation 2:

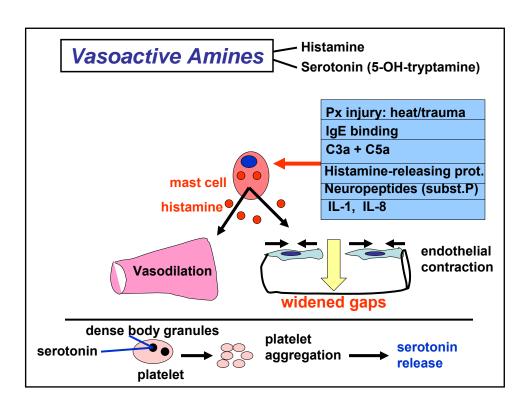
- 1. Chemical mediators
- 2. Systemic effects
- 3. Chronic inflammation

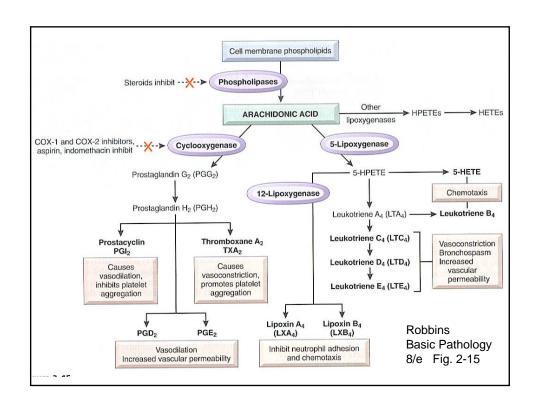


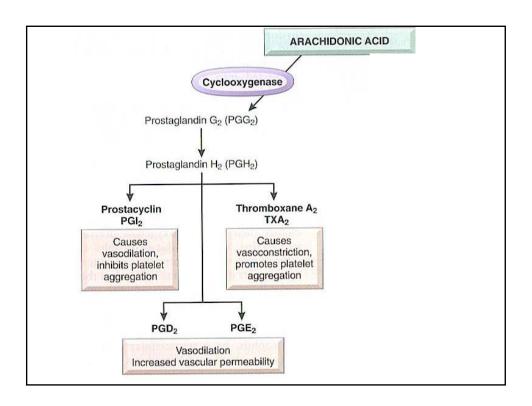


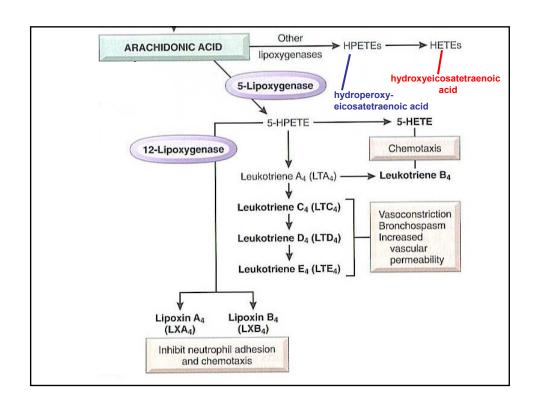


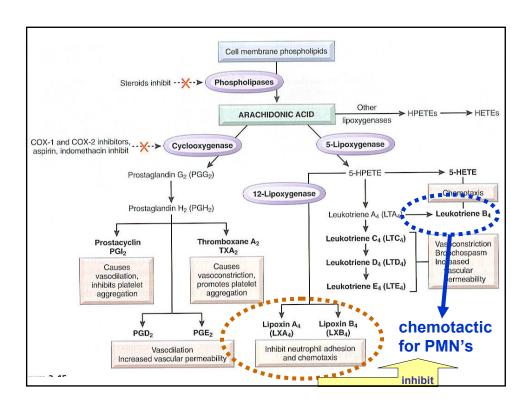


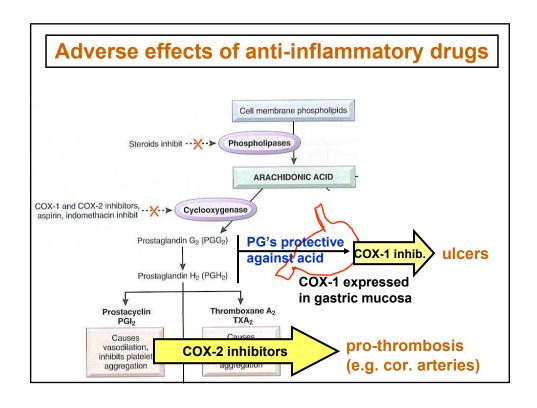


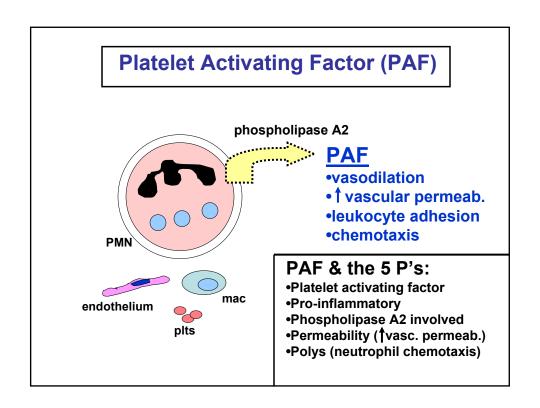


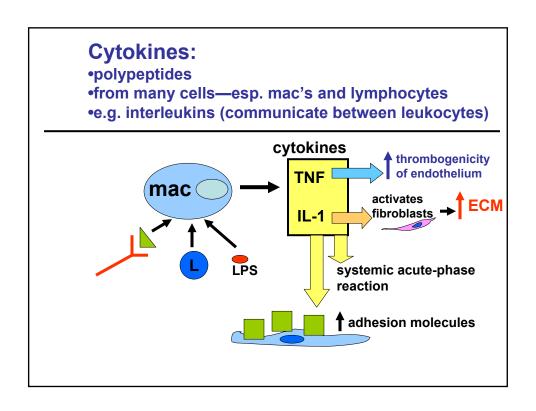


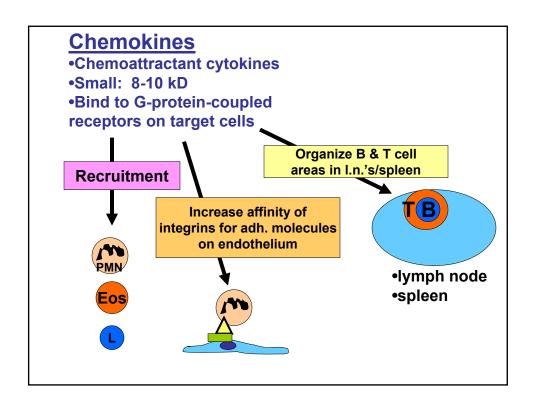


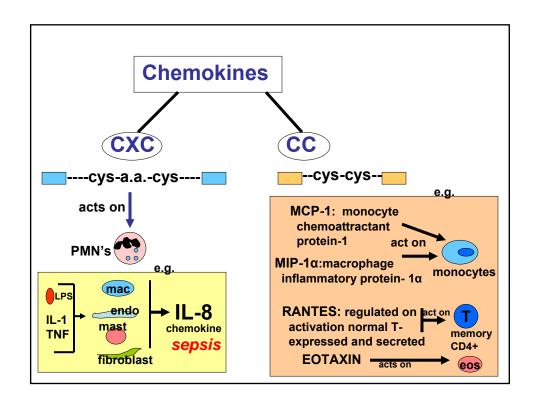


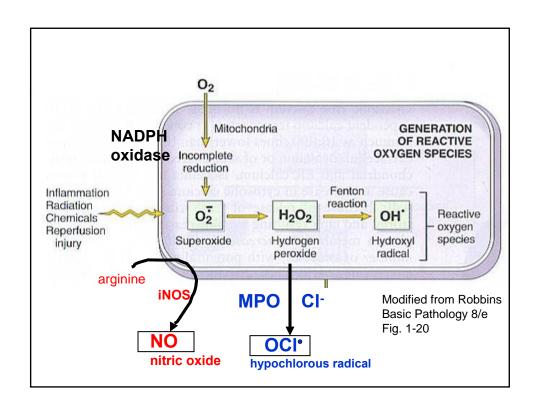


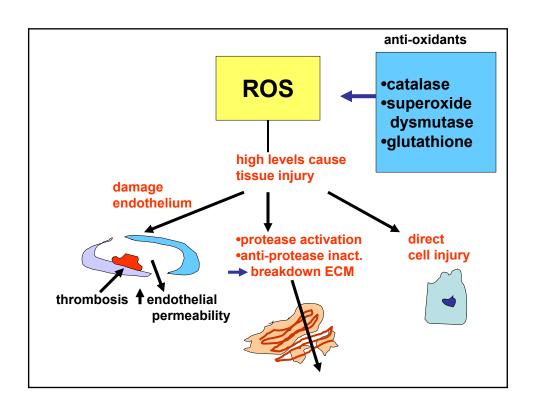


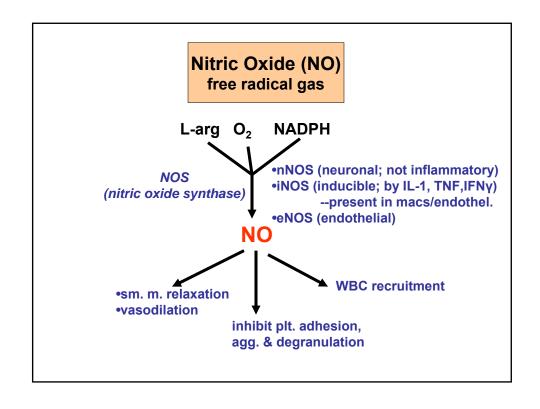


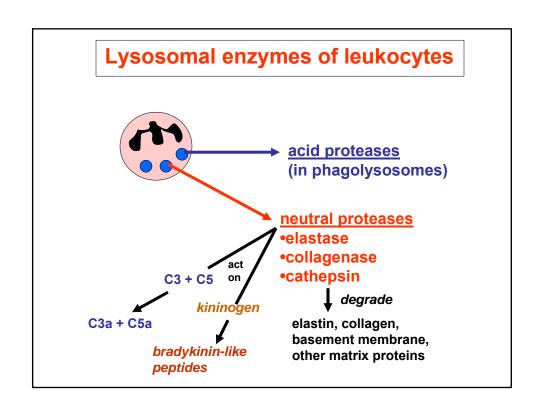


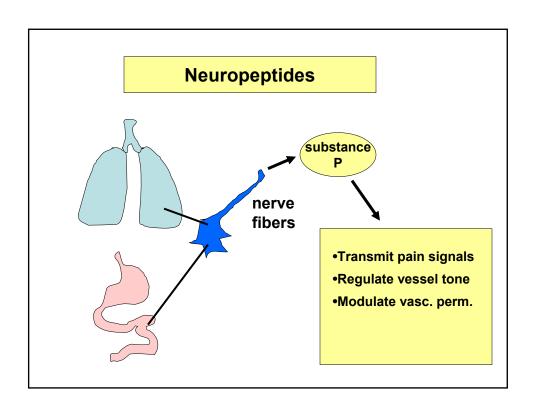


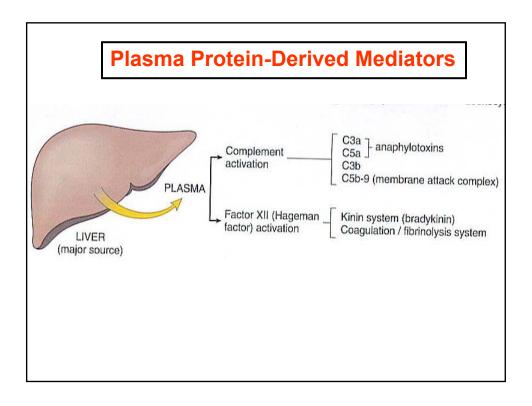






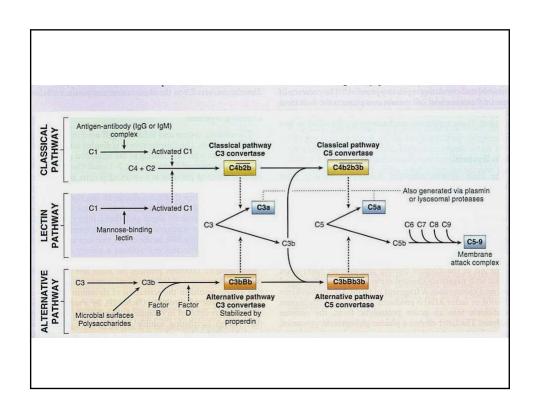


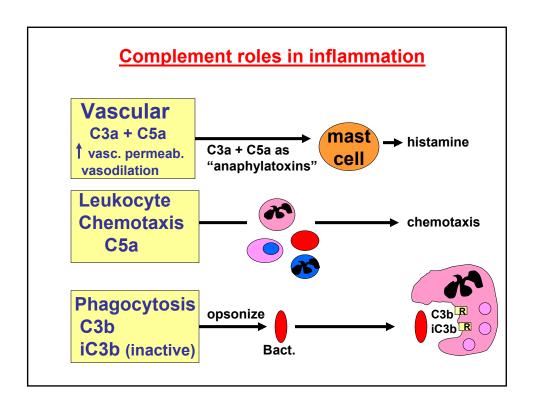


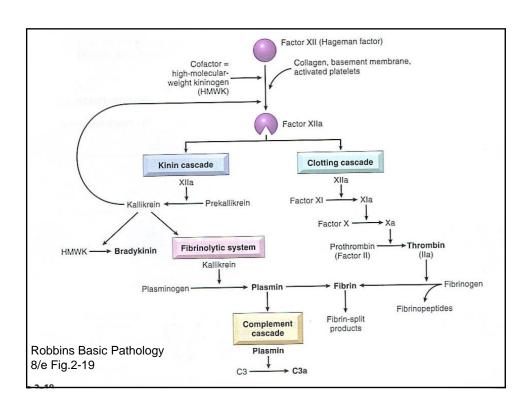


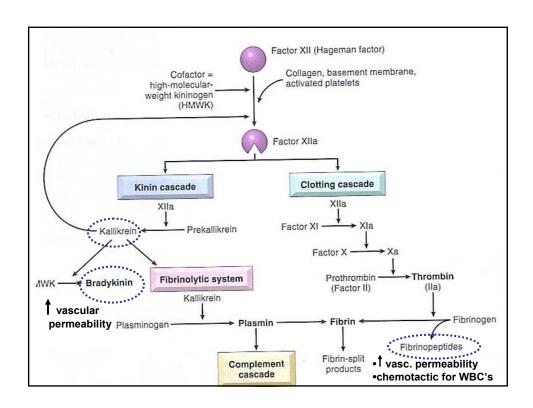
Complement

- •Present in plasma as 9 inactive proteins C1 C9
- •Progressive conversions from inactive to active forms (C1→C1a, C3→C3a + C3b,etc.)
- •Membrane attack complex: C5-9 forms channel in lipid membranes→ entry of fluid & ions, cell lysis
- •Activation pathways:
 - 1. Classical (Ag-Ab complexes; IgG/IgM)
 - 2. Alternative (bacterial polysaccharides, e.g., endotoxin, cell wall components)
 - 3. Lectin (plasma mannose-binding lectin binds to mannose residues on microbes)

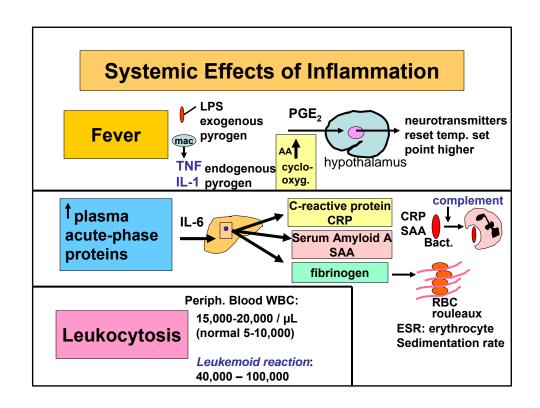








Vasodilation	Prostaglandins Nitric oxide Histamine
Increased vascular permeability	Vasoactive amines C3a and C5a (through liberating amines) Bradykinin Leukotrienes C4, D4, E4 PAF Substance P
Chemotaxis, leukocyte recruitment and activation	C5a Leukotriene B ₄ Chemokines IL-1, TNF Bacterial products
Fever	IL-1, TNF Prostaglandins
Pain	Prostaglandins Bradykinin
Tissue damage	Neutrophil and macrophage lysosomal enzymes Oxygen metabolites Nitric oxide



Chronic Inflammation

