inactive precursor "Pro-protein"

25 proteins

Complements

Adaptive → Effector arm of Response

innate → Activated early in infections

IGG1.IGG3 IGA .IGM (viruses or bacteria) → one IgMemory

Classical

Ag Ab ① Recognition Fc Region

C_1 [C1q, C1r, C1s]

② activated & Proteolytic Cascade.

C1S → C4 ③ → a (inactive) / B (opsonization)

C1S ↓ ④ C2 → B + a (inactive)

⑤ → C4B + C2B = C3 Convertase

Opsonization

Alternative

LPS

① C3 → a + B

② C3b → Post Conformational change → FB ③

④ FD Cleavage FB → FBa + FBb

FBb Remain with C3b ⑤ → C3 Convertase ⑥

lectin

Ab

Pathogen — Mannose binding ①

MBL ② go to C4 / C2

C4 → a + B

C2 → B + a

C3 Convertase

Regulate humoral immunity

↑↑ signaling

during B activation

BCR Ag C3d CR2 CD21 B D

C3 Convertase

B

binds to ① → glycoprotein

Micro D

opsonin

② Receptor for C3b

3 4

on Macrophage & neutrophils

a

Released into fluid

Baso Mast E

Vasoactive

Anaphylatoxins

MAC

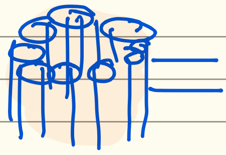
C5 Convertase — C3 Convertase + C3b

(1) ↳ C5 —(2)→ a → Chemoattractant Factor, Anaphylatoxin

(3) ↳ b ↳

Anchor Assembly of single C6, 7, 8

(4) ↳ polymerization of 18 C9



Receptors :-

(1) R1 (C3b, C4b)

(2) CR2 (C3d)

(3) R3, 4 → on phagocyte

Regulation of complement activity

- Factor H & DAC — Remove Bb from alternative C3 Convertase
- C1 INH — bind to (C1s, C1r) inactivate them
- CD59 — on normal tissue inhibite C9 —X→ C5b-8
- → Factor I — inactivate C3b

Disorder

- ← inherited deficiency in C3 — Predispose "pateints" to Frequent pob of infection [- gram]
- → C1, C2, C3, C4 — ↓C2 → lupus SLE erythrematous لبرس (AB- Ag)
- ← C9 → إذا قلة not problem
- → C1 INH (heredity angioedema) — massive Release of (C3a, C5a) — dangerous swelling [Edema] skin / intestine
- ← CD59 ↓ — inadequate Control of Mac — intra vascular Red Cell lysis — Paroxysmal nocturnal haemoglobinuria