Protein Adsorption & Surface Interactions

1. First event after material contacts blood

Protein adsorption happens within seconds and defines downstream reactions (platelets, cells, inflammation).

2. Driving forces for adsorption

• Hydrophobicity ↑ → adsorption ↑ → often irreversible

• Electrostatic interactions depend on surface charge & protein pI

• High surface energy → more proteins stick

• Proteins may unfold on the surface → stronger binding

Rule: Hydrophobic + charged + high-energy → more adsorption.

3. Vroman Effect

Arrival order ≠ final layer.

Albumin arrives first; fibrinogen replaces it and dominates.

Fibrinogen ↑ → platelet adhesion ↑ → thrombosis risk.

4. Surface type comparison

• Hydrophobic (silicone, PET, PU): strong adsorption + unfolding

• Hydrophilic (PEG/PEO): reduced fouling

• Charged surfaces: varies with pI

• Metals (high energy): multilayer adsorption

pH < pI →(+)

pH > pI →(-)

Hydrophobic > Hydrophilic in protein binding strength.

5. Device relevance

• Catheters: protein → bacteria → biofilm → infection

• Stents: fibrinogen → platelets → restenosis

• Heart valves: some adsorption remains → thrombosis risk

6. Reducing adsorption

• Hydrophilic coatings (PEG, zwitterions)

• Charge neutral surfaces

• Nano-engineered surfaces

• Drug coatings

7. Quick takeaways

• Adsorption is immediate

• Final layer = high-affinity proteins

• Determines thrombosis/inflammation

• Tunable via chemistry & structure