• Depending on the shape of the amphiphilic molecules, this can happen in either of the 2 ways: they can either form spherical micelles or they can form bilayers.



- This force that enables phospholipids to form bilayers also provides the lipid bilayer a self-healing ability.
- A small tear in the lipid bilayer causes the edges of the intrabrane to get exposed.
- This is energetically unfavorable
- Therefore, the phospholipids spontaneously agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the free elige 900 Prevention of the phospholipids agregate with one another to eliminate the phospholipids agregate with one another to eliminate



- This architecture is observed in porin proteins.
- 30 % of an organism's proteins are transmembrane proteins.
- The strong drive to maximize hydrogen-bonding in the absence of water means that a polypeptide chain that enters the lipid bilayer is likely to pass entirely through it before changing direction, since chain bending requires a loss of regular hydrogen-bonding interactions.
- But multi-pass transmembrane proteins can also contain regions that fold into the membrane from either side, squeezing into spaces between transmembrane α helices without contacting the hydrophobic core of the lipid bilayer.
- Because such regions interact only with other polypeptide regions, they do not need to maximize hydrogen-bonding; they can therefore have a variety of secondary structures, including helices that extend only part way across the lipid bilayer.
- Such regions are important for the function of some membrane proteins, including water channel and ion channel proteins, in which the regions contribute to the walls of the pores traversing the membrane and confer substrate specificity on the channels.
- Many single-pass membrane proteins form homo- or heterodimers that we held together by noncovalent, but strong and highly specific, interactions between the two transmembrane α helices.
- The sequence of the hydrophobic **unp** acids of these helices contains the information that directs cleptotein-protein interaction.
- Similarly, the characteristic α helices D multi-pass membrane proteins occupy occide positions in the Q and protein structure that are determined by interactions between the neighboring helices.
- These interactions are crucial for the structure and function of the many channels and transporters that move molecules across cell membranes.
- In these proteins, neighboring transmembrane helices in the folded structure of the protein shield many of the other transmembrane helices from the membrane lipids.
- Multi-pass membrane proteins that have their transmembrane segments arranged as β barrels rather than as α helices are comparatively rigid and therefore tend to form crystals readily when isolated.
- A number of β -Strands ranging from 8-22 come together to form β -Barrels.
- These proteins are abundantly present in the membranes of bacteria, mitochondria and chloroplasts.
- Some of the pore forming proteins such as porins are β -barrel proteins.
- Many porin barrels are formed from a 16-strand, antiparallel β sheet rolled up into a cylindrical structure.

• A rare type of anemia called paroxysmal nocturnal hemoglobinuria, results from a deficiency in GPI synthesis that makes red blood cells susceptible to lysis.



The complex and multifaceted prothrombotic state in paroxysmal nocturnal hemoglobinuria (PNH)



- Although the structures of bacteriorhodopsins and GPCRs are strikingly similar, they show no sequence similarity and thus probably belong to two evolutionarily distant branches of an ancient protein family.
- A related class of membrane proteins, the channel rhodopsins that green algae use
- to detect light, form ion channels when they absorb a photon.

16. LARGE PROTEIN COMPLEXES

- Many membrane proteins function as part of multicomponent complexes, several of which have been studied by x-ray crystallography.
- One is a bacterial photosynthetic reaction center, which was the first membrane protein complex to be crystallized and analyzed by x-ray diffraction.
- Many of the membrane protein complexes involved in photosynthesis, proton pumping, and electron transport are even larger than the photosynthetic reaction center.
- The enormous photosystem II complex from cyanobacteria, for example, contains 19 protein subunits and well over 60 transmembrane helices.
- Membrane proteins are often arranged in large complexes, not only for largesting various forms of energy, but also for transducing e gracelular signals into intracellular ones.

17. DIFFUSION OF MEMBRANE PLOTEINS

- Like most in orbitale lipids, menobrane proteins do not tumble (flip-flop) across inelipid bilayer, but they do not at about an axis perpendicular to the plane of the
- bilayer (rotational diffusion).
- In addition, many membrane proteins are able to move laterally within the membrane (lateral diffusion).

18. PROTEIN AND LIPID DOMAINS WITHIN THE PLASMA MEMBRANE

- Most cells confine membrane proteins to specific regions in a continuous lipid bilayer.
- Large aggregates like the bacteriorhodopsin and ATP synthase diffuse very slowly.
- In epithelial cells, such as those that line the gut or the tubules of the kidney, certain plasma membrane enzymes and transport proteins are confined to the apical surface of the cells, whereas others are confined to the basal and lateral surfaces.
- This asymmetric distribution of membrane proteins is often essential for the function of the epithelium.

- If the peripheral proteins are removed from erythrocyte ghosts, the membrane becomes fragmented into small vesicles, indicating that the inner protein network is required to maintain the integrity of the membrane.
- Erythrocytes are circulating cells that are squeezed under pressure through microscopic capillaries whose diameter is considerably less than that of the erythrocytes themselves.
- To traverse these narrow passageways, and to do so day after day, the red blood cell must be highly deformable, durable, and capable of withstanding shearing forces that tend to pull it apart.
- The spectrin–actin network gives the cell the strength, elasticity, and pliability necessary to carry out its demanding function.
- When first discovered, the membrane skeleton of the erythrocyte was thought to be a unique structure suited to the unique shape and mechanical needs of this cell type.
- However, as other cells were examined, similar types of membrane skeletons containing members of the spectrin and ankyrin families have been revealed, indicating that inner membrane skeletons are widespread.
- Dystrophin, for example, is a member of the spectrin acity that is found in the membrane skeleton of muscle cells.
- Mutations in dystrophin areatespansible for causing muscular dystrophy, a devastating disease that emples and kill children.
- The motion ditating mutation are ones that lead to a complete absence of the protein in the cell.
- The plasma membranes of muscle cells lacking dystrophin are apparently destroyed as a consequence of the mechanical stress exerted on them as the muscle contracts.
- As a result, the muscle cells die and eventually are no longer replaced.