If ATP is being broken down, it is usually a primary active transporter

OST alpha beta - facilitated diffusion

NA⁺K⁺ATPase - primary active ATPase you can identify it is primary as ATP is the source of energy.

-((Remember glucose cellular transport as an example if you will memorise a transport chain))

How LDLs are taken up

1. LDLs bind to receptor protein
2. Conformational change occurs
3. Adapter proteins bind and this attracts clathrin
4. stimulates vesicle production

A mutation can cause hypercholesterolemia - the deletion of the clathrin interaction domain. This increases the risk of the patient suffering from coronary heart disease and atherosclerosis.

Cystic fibrosis transmembrane regulator

Cystic fibrosis;

- Increases fluidity
- Mutation at position 508 causes it, which impairs folding
- Also caused by glycine being replaced by aspartic acid at position 551 so channel doesn’t open frequently, if it does it opens a bit.

Drugs can treat this;
1. Correctors - effective against mutation at position 508
   - Suberoylanilide hydroxamic acid, SAHA switches on folding chaperones that allows it fold correctly
   - Drug approved as a HDAC inhibitor for the treatment of T cell lymphoma
2. Potentiators - effective against mutation at position 551
   - Bind to transport protein (channel) to increase its ability to open
   - Drug approved by the FDA