- 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))

<u>How LDLs are taken up</u>

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.

<u>Cystic fibrosis transmembrane regulator</u>

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 lumphoma
  2. Potentiators- effective against mutation at position 551
     - Bind to transport protein (channel) to increase its ability to open
     - Drug approved by the FDA