

***Title of Lecture: Pharmacodynamics***

***Date of Lecture:***

***Sheet no: 7***

***Refer to slide no. : 1-28***

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**☺ Note: the doctor didn't talk much outside the slides so I will write the extra notes of each slide by referring to its number**

**🡪Slide 3:**   
1- Non-receptor mechanisms: like local anesthetics

2- Osmotic diuretics: drugs which will increase urine output … mechanism: they will increase the osmotic pressure in the renal tubules so that will drag water and salt toward these tubules to be excreted

3- Osmotic cathartics: laxatives "in cases of constipation" which enhance feces excretion by increasing the osmotic pressure in the intestines and that will drag water toward intestines and the feces will be excreted easily

**🡪Slide 4:**

1- antacids: to neutralize the action of acid by giving an alkali like aluminum hydroxide or magnesium hydroxide

2- Disinfectants kill bacteria which contain enzymes (many people consider enzymes as receptors but actually they are not)

**🡪Slide 6:**

- NSAID’s: non steroidal anti inflammatory drugs inhibits Cyclooxygenase which is important in prostaglandin synthesis

**🡪Slide 7:**

1-drugs may have receptors all over the body but selective drugs drugs have receptors in specific organs not all over the body and that may decrease the side effects of these drugs

2- No. of receptors should be reasonable in order to produce the therapeutic effect

3- Changes in a drug’s chemical structure can alter the effect of this drug ( any change in the structure of an agonist can convert it to an antagonist )

**🡪Slide 11:**

- Lock and key theory is the best theory that describes the binding of drugs with their receptors: the structure of the drug is complementary to that of the receptor

**🡪Slide 14:**

- Sites of loss: sites where some of the drug is lost by binding to something other than the site of action

**🡪Slide 16:**

- Hydrogen bonding requires complementary structures between the drug and the receptor

**🡪Slide 19:**

- Affinity expresses the strength of binding between the drug and the receptor…

**🡪Slide 20:**

- toxic effects are sometimes receptor mediated

**🡪Slide 22:**

- Ligand-gated ion channels: the drug binds to the receptor and this complex will activate or deactivate ion channels (according to the response)

**🡪Slide 23:**

- G protein-coupled receptors: when the drug binds to its receptor, it activates membrane bound enzymes like: tyrosine kinase which will mediate the phosphorylation of certain proteins inside the cell that will mediate the effect of the drug

**🡪Slide 25:**

1- Intracellular ( nuclear) receptors like thyroid and steroid hormones' receptors … these can affect the translation and the transduction of DNA which will affect the resulted proteins that elicit the effect of the drug  
2-the ligand should be lipid soluble in order to penetrate the cell membranes ( plasma membrane and the strong nuclear membrane)  
3- first messanger : the ligand (the drug) while the second messanger is resulted molecules (e.g. cyclic AMP, cyclic GMP, IP3, DAG, calcium)

⮊QUESTION: How can we prove that this drug elicits its action through cyclic AMP for example not through other second messanger?

we get many target cells from an animal and put them in Petri dishes .. We put the drug on one sample and we treat the others with different second messangers >> the cell (treated with a second messanger) that will act as the cell treated with the drug will determine the second messanger that this drug is working through

**🡪Slide 28:**

1- increasing the dose will increase the response until we reach appoint where any further increase in the dose will not result in an increased response ( V max ) and that happens because all the receptors are now saturated and in order to have a response again, the attached drugs should be metabolized to become detached …. This process continues until the end of duration of action of the drug

2 - V max is a measure of efficiency (intrinsic activity or maximal response) and it's the most important parameter in dynamics and kinetics