Receptor Families		
Ligand-Gated Ion Channels (Ionotropic)	extracellular receptor in plasma membrane e.g. NicR, ACh R	- fastest response time (milliseconds) - change in ion concentration triggers cellular responses - e.g. in NicR, ACh binds to alpha subunits (x2), which opens Na+ channel, Na+ entry stimulates contraction (depolarisation)
G-Protein Coupled Receptors (Metabotropic)	extracellular receptor in plasma membrane e.g. MusR, ACh R consists 7 transmembrane segments (serpentine receptor)	- fast response time (seconds) - largest family - agonist binds to the 7 transmembrane domain at extracellular membrane, which causes a conformational change in the receptor. This allows the G protein to interact with the receptor. A number of events occur which allows the G protein to either: 1. diffuse through the lipid membrane to interact with the ion channel causing ion influx or efflux, producing cellular effects; 2. produce 2nd messengers which produces a number of cellular effects in the enzyme - linked to an effector protein by a G protein (ion channel or enzyme) - alpha-1 AR: Gq protein (coupled to phospholipase C, which increases IP3 & DAG) - alpha-2 AR: Gi protein (inhibits Adenylate Cyclase, which decreases cAMP) - beta-1 and 2 AR: Gs protein (stimulates Adenylate Cyclase, which increases cAMP)

Kinase-linked Receptors	extracellular receptor on cell surface e.g. cytokine receptor (i.e. insulin, leptin)	- fast response time (minutes) - have intrinsic enzymatic activity or activates enzyme - e.g. in growth factor receptors, agonist binding causes receptor dimerisation activation of tyrosine kinase in the cytoplasmic domain. This causes phosphorylation of the tyrosine residues in the intracellular domains in the receptors (protein phosphorylation). The proteins that are phosphorylated (esp. transcription factors) are then translocated into the nucleus and causes modulation or alteration on gene transcription which can cause change in protein synthesis back in the cytoplasm.
Nuclear Receptors	intracellular receptor in cytoplasm e.g. oestrogen receptor, glucocorticoid receptor	- slow response time (hours/days) - produces changes in gene transcription - drug needs to be lipid soluble to cross cell membrane - e.g. glucocorticoid receptor activation inhibits COX synthesis (glucocorticoids treat chronic inflammatory disease. Glucocorticoids are lipid soluble and crosses the cell membrane. It then binds to glucocorticoid receptor and results in dimerisation. They are then translocated into the nucleus and binds to DNA to alter protein synthesis in the cytoplasm (reduce or repress gene.)

Drug Targets	Drug	Effect	Clinical Usage
Ion Channels	**Nifedepine**	blocks Ca2+ channel	Hypertension
	Verapamil	low Ca2+ = reduced contraction = reduced TPR = lower BP	