PHRM20001: Drug Names per lecture

SEM 2 2016

Lecture 1 - nothing

Lecture 2- Drug targets- receptors, enzymes, other proteins

Ion channels (ionotropic) - Nifedipine: blocks Ca channels - hypertension	Carrier molecules: - Fluoxetine: block serotonin reuptake by blocking High Affinity Uptake 1 - Used for depression
Enzymes: a. Prodrug - L-DOPA which is turned into dopamine through DOPA decarboxylase b. False substrates - Fluorouracil instead of uracil to stop DNA synthesis c. Enzyme inactivation - Aspirin—inhibiting COX-1 and COX-2 - SARIN—inactivating AChE so it wont break down ACh	Receptors a. Ion channel receptors (ionotropic) - ACh for nicotinic receptors b. Tyrosine Kinase - Growth Factors c. GPCR's (metabotropic) - 7 transmembrane receptor - ACh for muscarinic - NA and Adrenaline for adrenoceptors - Gs: beta 1 and 2—adenylate cyclase stimulates production of cAMP, and leads to phosphorylation events, - Gq: alpha 1—Phospholipase C (PLC) leading to Gi: alpha 2. d. Nuclear receptors

Lecture 3-nothing

Lecture 4

Allosteric modulation	Benzodiazepines (positive, onto $GABA_A$ receptors on alpha 1 and gamma 1 subunits)
Pathway inhibition	Verapamil (Phenyl alkylamines) blocks ca2+ channels to lower BP (antihypertensive)
Functional antagonism	On heart: PSNS and SNS (SA node) - PSNS: M2 receptors using ACh to decrease HR - SNS: beta1 adrenoceptors using NA to increase HR

Lecture 5-nothing

Lecture 6

Cocaine	Blocks High Affinity Neuronal Uptake 1 Blocking neuronal reuptake therefore more NA in synaptic cleft
MAO-inhibitor	Blocks metabolism of catecholamines (NA< dopamine, serotonin) Effective with IAS because IAS replaces NA in vesicles, send them down conc gradient, reuptaken into neurons but cannot be broken down!
Indirectly acting Sympathomimetics	Replace NA in vesicles, goes up by High Affinity Uptake 1 like NA. Isnt broken down by MAO -tyramine (vegemite) and amphetamine Cheese effect: headaches, hypertensive effects