Neurohumoral Transmission in CNS

Types of Neurotransmitters (NT)

Excitatory - Glutamate, Aspartate
Inhibitory - GABA, Glycine
Others - Noradrenaline, dopamine, 5-HT, Ach, histamine, Adenosine Endocannabinoids

NT	Location	Mechanism	Nature
GABA	All location of CNS	Ligand gated Cl^- ion channel opens	Inhibitory
Glycine	Brain stem & spinal cord	Ligand gated ion channel	Inhibitory
Glutamate	All level of CNS	"	Excitatory
Ach	Ganglia NMJ, Spinal cord	"	Excitatory

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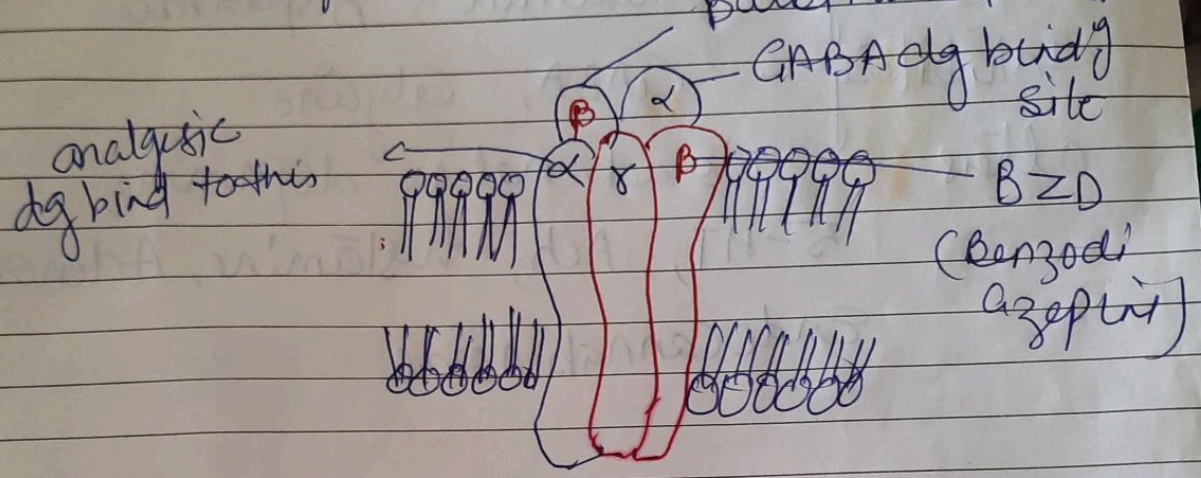
Dopamine	Basalganglia frontal cortex	» Inhibitory
Histamine	Ventral Posterior Hypothalamus	" Excitatory



GABA Rx

Note

GABA Rx is very important. Most of the dg acting on CNS act through this GABA Rx

There are 5 Subunit in GABA Rx, 2 α, 2 β, 1 γ. (Pantameric Structure)

These subunits are binding site of respective dgs. dgs binding occur on specific site (Subunit) & open the Cl⁻ channel and Cl⁻ ions move from Extracellular to intracellular.

EPSP = Excitatory post synaptic → secretion, depolarization, hyperpolarization

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Mechanism of Action

1) They Block nerve Conduction by ↓ entry of Na^+ ions
2) Because there is no entry of Na^+ Rate of action potential ↑ & depolarization become ↓.
3) Conduction, slow and ultimately block.
4) LA enteract with Rv and ↑ threshold of Na^+ Channel opening.
5) Due to this Na^+ permeability fails to ↑ in response to an impulse.
6) ~~Action is better at i) Active Rv, ii) inactive Na^+ channel state but not as resting~~

Action is better at i) Active Rv
ii) inactive Na^+ channel
but not an resting memb. potential

7) Binding of LA to Rv results in ↑ed inactive state of Na^+ channel. This will lead to longer time to recover.

Main site for action of LA

Sensory nerve ending, nerve trunk

neuromuscular Jn, Synapses, Rx.

·) They also ↓ release of Acetylcholine

·) Automatic fibres are more sensitive to LA than somatic fibres.

·) LA failed to reduced at inflammed tissue.

LA produces Anesthesia by ~~inhibiting~~ inhibiting excitation of nerve ending or by blocking conduction in peripheral nerves. This is achieved by anaesthetics reversibly binding to and inactivating sodium channels. Sodium influx through these channels is necessary for the depolarization of nerve cell membranes & subsequent propagation of impulses along the course of the nerve.