

NEUROTRANSMITTERS

	Neurotransmitter	Function	Receptor	Action	Related drugs/illnesses
	Acetylcholine	Excitatory NT for many post ganglionic parasympathetics	Nicotinic ACh-R (N1-neuromusc, N2-ANS) Muscarinic ACh-R (M1-CNS, GI; M2-heart)	Ionotropic channels → incr gNa, gK, gCa Metabotropic channels → 2 nd mssrs, phos of channels, conductance changes	Curare (N1), hexamethonium (N2) Atropine (M)
BIOGENIC AMINES	Norepinephrine	(many postgang symp) α ₁ - contracts sm musc, bv α ₂ - autorec of NE (inhibits release) β ₁ - incr HR, contractility β ₂ - relax GI sm musc, some vasc sm	α ₁ α ₂ β ₁ β ₂	Tyrosine → Dopa → dopamine → NE	Lack of NE may cause manic-depression: Tricyclics (ie desipramine) inhibit reuptake (by autoreceptors); MAO (monoamine oxidase) inhibitors increase presynaptic concentration (prevent NE degradation)
	Dopamine	Pleasure and reward Involved in memory	D1 D2	→Gs→incr cAMP→phosphor. →Gi→decr cAMP	Cocaine, amphetamines – inhibit reuptake (can have long-term effect-dcr receptors) Parkinson's-decr in DA→ treat with DOPA Schizophrenia: excess D4 → treat with blockers of receptors (ie phenothiazines)
	Serotonin	(pleasure)	All GPCR except 5-HT ₃ (ionotropic)	Made from Trp-5-HT	SSRIs = selective serotonin reuptake inhibitors ie Prozac (fluoxetine) – targets Na-coupled transport – treat depression, esp anxiety OCDs – use Paxil (SSRI) LSD – activates 5-HD2 receptor MDMA (ecstasy)- elev 5-HT, then decr
	Histamine	Mediates allergic response; arousal	H1-H3= GPCRs in hypothalamus		Benadryl (antihistamine) →drowsiness -Can block H1 to decr motion sickness (blocks vestibular function) -Block H2 (gastric acid synthesis) to control peptic ulcers
AMINO ACIDS	Glutamate (and Aspartate)	Main excitatory NT in the brain Synaptic plasticity – learning *mGluR4 = basis of umami	11 metabotropic receptors Ionotropic: kainite, AMPA, NMDA	*NMDA is ligand- and voltage-gated: Mg blocking pore is only moved when V > -60mV but necessary for Ca influx	if Glu → too much Ca → kills cell Excitoxin achieves this
	GABA (and glycine)	Main inhibitory NT in the brain (20% of CNS synapses)	GABA _A -ionotropic GABA _B -metabotrop	GABA _A incr gCl – opposes depol GABA _B → Gi→ decr gCa, inc gK Gly – incr gCl mainly in SC Activation by GABA alone → hyperpol; GABA+enhancer → more sustained inhibitory response	Barbiturates and benzodiazepines (ie Valium) – enhance effects of GABA on gCl →reduce anxiety, muscle relaxants Strychnine – blocks Gly receptors→ convulsions, hyperactivity – used by athletes to boost performance

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PEPTIDES	Oxytocin and vasopressin	Uterine contractions, nursing; water reabsorption in kidney			
	Substance P	Mediates pain (found in intestine)			Can block in depressed patients who don't respond to SSRIs
	Vasoactive intestinal peptide (VIP) and cholecystinin	GI function			
	Opioid peptides	analgesics	Same as opiates	POMC (pro-opiomelanocortin) → β -endorphin and enkephalin (analgesics)	May be involved in athletic "high" Receptors can also bind morphine, codeine
GASES	NO	Relaxes smooth muscle Interneuronal messenger in brain	--	NO → SGC → synth cGMP → phos smooth musc protein	cGMP hydrolyzed by PDE – block with Viagra etc.
	CO		--	Made from heme-oxygenase	
	H ₂ S	Cardioprotective effect during ischemia	--		