312 Psychopharmacology Hand-out (1)

Cholinergic Drugs (Ach):

Name	Site of Action	Mode of Action	Effect/Use
d-tubocurarine	NMJ	competitive antagonism	paralysis; immobilization
Gallamine	NMJ	competitive antagonism	paralysis; immobilization
Decamethonium	NMJ	depolarization blockade	paralysis; immobilization
Succinylcholine	NMJ	depolarization blockade	paralysis; immobilization
Atropine & Scopalamine	Cholinergic parasympathetic synapses	competitive antagonism	makes pupils dilate; used in surgery to dry out lungs to prevent fluid accumulation
Botulinis Toxin	ALL Ach synapses	Prevents release of Ach from presynaptic terminals	can cause death; used in research
Neostigmine	Cholinergic synapses, esp. NMJ	incomplete anticholinesterase (excitatory)	used in the diagnosis and treatment of Myasthenia Gravis
Eserine	Cholinergic synapses, esp. NMJ	Completely stops acetylcholinesterase (inhibitory)	research inhibitor
Edrophonium	same	same	research inhibitor
Nicotine	parasympathetic ganglia	depolarization blockade	research inhibitor
Carbachol	same	facilitates depolarization	used in research
Hemicholinium	All Ach synapses	prevents reuptake	research
DFP: Di-isopropylflorophosphate	All Ach synapses	complete anticholinesterase	research inhibitor

Catecholaminergic Drugs (Epi, NE, DA): (2)

Name	Site of Action	Mode of Action	Effect/Use
AMPT (alpha methyl para tyrosine)	all catecholaminergic synapses	inhibits synthesis of catecholamine transmitters (inhibitory)	research
Alpha-methyl-dopa	Localized to sympathetic post-ganglionic neurons only	competitive antagonism	research; also previously used as a depressant
6-hydroxydopamine	All cat. synapses	Attacks presynaptic terminals, causing cat. transmitter to leak out and become depleted	research inhibitor (when used with desmethyl imipramine, effect is restricted to dopaminergic neurons)
reserpine (see also 5-HT drug effects below)	all adrenergic synapses	Causes synaptic vesicles to rupture and prevents reuptake, causing depletion	Used as early depressant until side effects were discovered
Propanalol	Beta adrenergic synapses	"Beta blocker" – blocks β-adrenergic receptors	Used to treat hypertension
phentolamine	Alpha adrenergic synapses	Blocks alpha adrenergic receptors	research
dopamine beta hydroxylase	all adrenergic synapses	facilitates adrenergic transmission	research
cocaine	most adrenergic synapses	1. slows recapture of NE (excitatory), 2. prevents sodium from entering axons, thereby preventing spikes	1. Antidepressant 2. Local anesthetic
imipramine	All adrenergic synapses	slows recapture of NE (excitatory)	antidepressant
Amphetamine	All catacholaminergic synapses	1. promotes release of NE and DA 2. slows recapture of NE (maybe DA too)	Antidepressant; stimulant; but can produce psychosis in large doses

Catecholaminergic Drugs (Epi, NE, DA): -continued (3)

Name	Site of Action	Mode of Action	Effect/Use
MAO inhibitors (e.g. pargyline)	all adrenergic synapses	inhibits MAO, slowing breakdown of adrenergic transmitters (excitatory)	antidepressant
Lithium	adrenergic synapses	Increases reuptake of NE (inhibitory)	Used to treat manic psychosis
L-dopa	Dopaminergic synapses	DA precursor, increases DA synthesis (excitatory)	Used to treat Parkinson's disease
Haloperidol	All DA synapses, especially afferent collaterals in the RF	Blocks DA receptors	Antipsychotic
Phenothiazines (chlorpromazine, thorazine)	same	same	Antipsychotic, used in treatment of schizophrenia
apomorphine	all DA synapses	stimulates DA receptors	Research
benzotropine	all DA synapses	slows DA reuptake (excitatory)	Research
Ritalin	same	same	A.D.D. therapy

Serotonergic Drugs (5-HT):

Name	Site of Action	Mode of Action	Effect/Use
PCPA	all 5-HT synapses	completely inhibits synthesis by tryptophan hydroxylase (inhibitory)	research inhibition
Cinanserin	same	competitive antagonism	research
5-HTP (5-hydroxy-typtophan)	same	5-HT precursor, increases synthesis (excitatory)	research
amytryptaline	same	slows reuptake (excitatory)	research
Iproniazid	same	Anti-MAO	research
LSD	same	inhibitory	hallucinogenic
mescaline	same	?	hallucinogenic
[SSRI], Paxil, Prozac	same	enhances duration of serotonin in synapse	antidepressant

GABA-ergic drugs: (4)

Name	Site of Action	Mode of Action	Effect/Use
Tetanus toxin	all inhibitory synapses	Prevents release of GABA and glycine	Lockjaw, research
strychnine	vertebrate inhibitory synapses (GABA and Glycine)	competitive antagonism	rat poison, research
muscimol	GABA synapses	Stimulates GABA receptors	research
picrotoxin, bicuculline	invertebrate inhibitory synapses	competitive antagonism	research
Benzodiazepines (Valium, Librium)	GABA synapses	GABA receptor agonists	Anxiolytic muscle relaxer, mild anesthesia
Barbiturates (Pentobarbital, Nembutol)	same	same	General anesthesia antiepileptic

Psychopharmacology terms/concepts:

competitive antagonism: Drug competes with neurotransmitter to bond with receptor site.

depolarization blockade: Drug causes depolarization of cell, but does not allow it to return to equilibrium potential, thereby inhibiting it.

reuptake/breakdown enzyme inhibitors: If a drug slows or partially inhibits reuptake, the effect is that the transmitter remains in the synapse longer, thus producing an excitatory effect. If a drug completely blocks reuptake, however, the neurotransmitter supply will be depleted, causing an inhibitory effect. Likewise, drugs which partially block breakdown enzymes (e.g. MAO inhibitors), have an excitatory effect, but drugs which completely inhibit these enzymes have inhibitory effects.