

“MINI” PSYCHOPHARMACOLOGY & DRUG ADDICTION (p.1)

1. Basic Principles of Drug Action

a. **Drug Administration**

Ingestion (oral route)

Injection (SC, IM, IV)

Inhalation

Absorption through mucous membranes

Transdermal

b. **Drug Distribution**

Must enter bloodstream

Must pass blood-brain barrier (non-ionized, lipid soluble molecules)

Distributes to all the body cells

c. **Drug Metabolism** (liver enzymes)

d. **Drug Elimination** (kidney, urine; other routes)

e. **Drug “half-life”**

f. **Mechanisms of Drug Action** (in NS is synapse, receptor sites)

g. **Drug Tolerance**

Shifts dose-response curve to the right

Takes more of drug to get same result as before

Not all drugs exhibit tolerance

Can develop tolerance to some, but not all, of a drug’s effect

Mechanisms: e.g. liver enzyme induction, changes in RSs

h. **Drug Withdrawal Effects & Physical Dependence**

Exposure to a drug produces compensatory changes in NS that offset the drug’s effects and produce tolerance

Rapid/abrupt D/C of drug vs. gradual tapering off of drug

Is *not* the same as addiction (cravings, concentrated focus, reward)

“Conditioned” tolerance (conditioned stimuli --- compensatory bodily changes), role in drug ODs

2. Drug Addiction (p.2)

a. **Biopsychological Theories of Addiction**

“older” physical-dependence theories

do not adequately describe the data on addiction

addicts that do not exhibit withdrawal effects still crave

relapse occurs without dependence/withdrawal

addiction does not occur even with dependence/withdrawal

“newer” **positive-incentive theories of addiction**

addict uses drug in order to re-experience the positive incentive

(“pleasure”, “release” from tension effects)

to stop the “cravings” from the drug

“pleasure” vs. “intense focus/attention on” experiencing the drug

b. **Brain areas involved in “pleasure/focus”**

intracranial self-stimulation studies (septal/lateral hypothalamus)

mesotelencephalic **dopamine** system

cell bodies in midbrain (substantia nigra & **ventral tegmental area**)

axons project to telencephalon (forebrain), including prefrontal ctx,

limbic ctx (*cingulated gyrus*), *olfactory bulb*, *amygdala*, *septum*,

dorsal striatum (caudate nucl. & putamen),

& *nucleus Accumbens*

(2 pathways using dopamine, one for motor control via the

basal ganglia, and one for “reward” via forebrain & limbic

structures)

“**mesocorticolimbic pathway**”

dopamine agonists are likely to be very addictive

e.g. cocaine, methamphetamines, nicotine

drugs that are dopamine antagonists or that have no effect on dopamine

are not addictive

nucleus Accumbens may not actually mediate the reward/pleasure

experience per se; but does attach “meaning” to a stimulus (e.g. a

drug) that signals **that “reward” is imminent**, stimulus becomes the

focus of attention, S will try to get the stimulus, will “crave” it, will

seek it to the exclusion of all other stimuli... “addiction”