"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 Addition
   "older" physical-dependence theories
do not adequately describe the data on addition
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”