ADDICTIVE DISORDERS

SUBSTANCE USE

DSM-V SYMPTOMS

1. Taking substance in larger amounts or for longer than intended
2. Unable to reduce use despite intending to
3. Spending a lot of time getting, using, or recovering from use of substance
4. Cravings and urges
5. Impaired social, occupational functioning
6. Impaired interpersonal relationships due to substance use
7. Giving up important social, occupational, or recreational activities
8. Using substances even in harmful situations
9. Continuing to use, even with physical or psychological problem that could have been caused/made worse
10. Needing more of the substance to get the same effect (tolerance)
11. Development of withdrawal symptoms

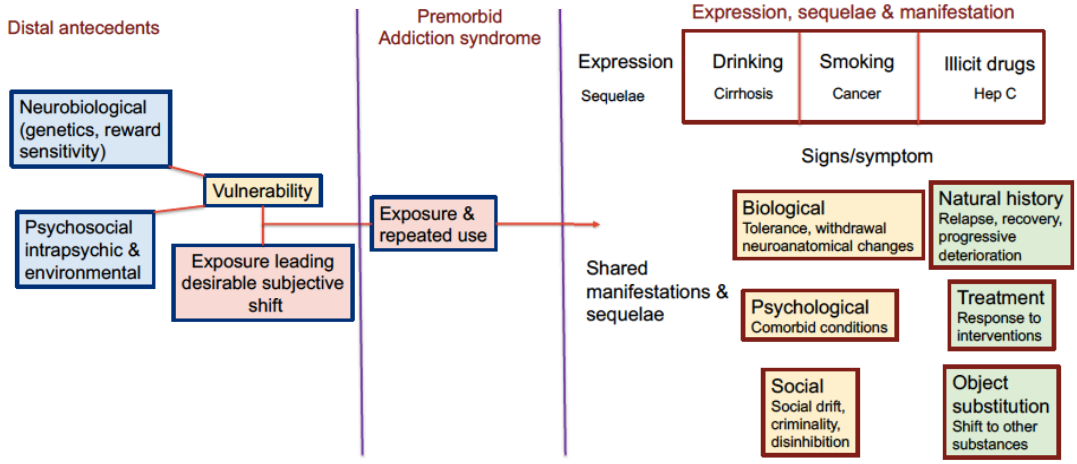
ADDICTION VS DEPENDENCE

- Definition of addiction
    - Primary, chronic disease of brain
    - Involves reward, motivation, memory and related circuitry
    - Potential for relapse and recovery
    - Loss of control over reward-seeking behaviour w/ use in harmful situations i.e. medical model
    - Syndrome w/ combination of signs + symptoms (may differ between cases) rather than unitary disorder
- Components of addiction:
    1. Salience: Decreases salience of other alternatives, with focus on consuming and acquiring drugs
    2. Mood modification: Drugs used to reduce -ve moods, and acquire +ve moods
    3. Tolerance: Need increased dose of drug for same effect
    4. Withdrawal: Cravings + other –ve symptoms experienced when drug not consumed
    5. Conflict: Knowledge of -ve consequences conflicts with desire for drug
    6. Relapse: Environmental cues, memories and other emotions may trigger relapse
- Physical + psychological dependence:
    - Physical: Dependence not due to motivation/reward systems e.g. child of parent w/ addiction to heroin is physically dependent
    - Psychological: Dependence not based on physical systems e.g. dependence on placebo, believing Xanax/drug prevents panic attack
    - *Neuroadaptation: Physiological changes of body to compensate for effects of drugs e.g. increased receptors → tolerance, with withdrawal symptoms experienced when drug not taken
        - Needed in transition to addiction

- Not necessary in deciding whether addiction is present

BRAIN DISEASE VS BIOPSYCHOSOCIAL MODEL

- Addiction as brain disease: Focus on disease of brain rather than effects of social + environmental stressors e.g. loneliness, poverty, violence
  - Deflects responsibility and ability to change
  - Conceptualises urge as irresistible rather than just a compulsive drive
- **Biopsychosocial** framework: **Dominant** framework – Considers biology, social and psychological factors which drive addiction



  - Distal antecedents: Cause increased vulnerability to exposure to drug + consumption
    - Neurobiological: Genetics, reward sensitivity (more sensitive to +ve effects of drug)
    - Intrapsychic: Mental disorders w/ -ve affect e.g. anxiety, depression, PTSD
    - Environmental/psychosocial: Poverty, peer pressure in work + social environments, vicarious learning
    - → Exposure leads to desirable subjective shift e.g. +ve mood, decreased -ve affect
  - Premorbid addiction syndrome: Exposure + repeated use
  - Expression of addictive behaviours → consequences (some shared + some unique w/ diff addictions)
    - Unique consequences: Drinking → cirrhosis, smoking → cancer, illicit drugs → hepatitis C
    - Shared signs + symptoms:
      - Biological: Tolerance, withdrawal, neuroanatomical changes
      - Psychological: Comorbid conditions
      - Social: Social drift (associate w/ diff class), criminality, disinhibition
      - Object substitution: Use of other substances
    - Shared course of addiction:
      - Natural history: Relapse, recovery, deterioration
      - Treatment: Response to interventions

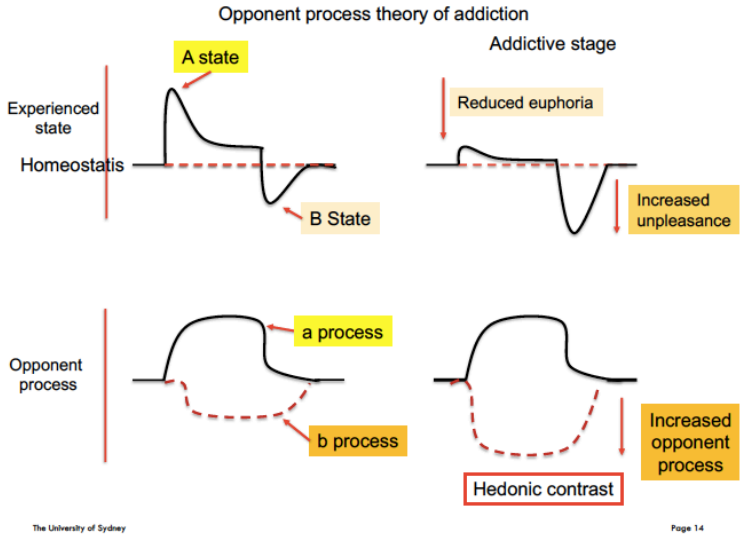
MEDICAL VS RATIONAL CHOICE MODELS

- Medical/disease model: Impaired control over urges/cravings

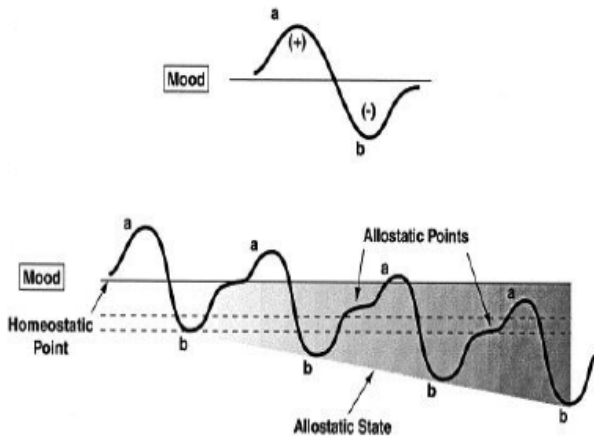
- Rational choice model: Voluntary behaviour which is under control; but drive → compulsive use of drug

MEDICAL/DISEASE MODEL

- Drug affects brains' reward systems: Dopaminergic system + endogenous opioid system
    - Increases dopamine levels w/ drugs blocking reuptake of dopamine → excess in synapses → pleasure + euphoria
    - E.g. cocaine → decrease in receptor levels due to tolerance
- Tolerance explained via opponent process theory:

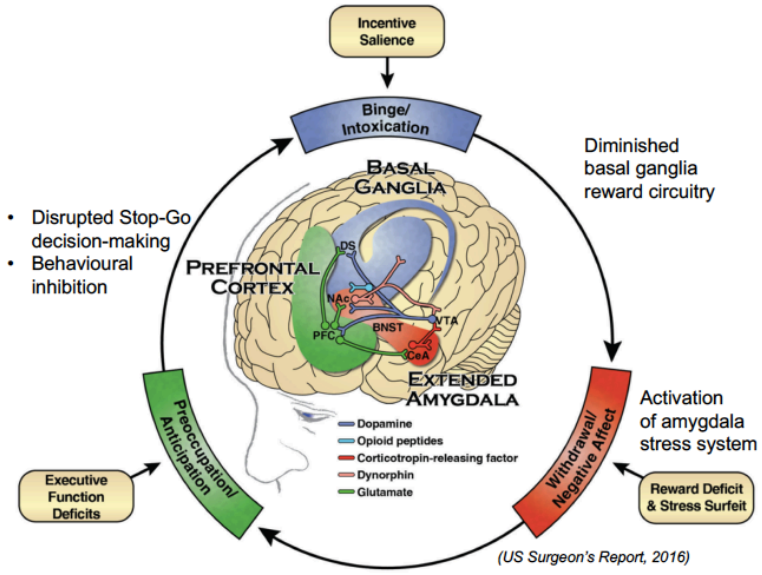


    - Opponent processes a and b occur in response to drug consumption
        - A process = high from drug
        - B process = homeostatic response of body, begins and ends later than a process
    - Opponent processes sum to form experienced state
        - 'a state' = drug high
        - 'b state' = rebound from drug e.g. hangover after alcohol
    - During addiction, 'b process' increases → lower 'a state' and 'b state' i.e. less euphoria, increased unpleasantness of drug
    - With repeated drug use, the body returns to a lower allostatic state (steady state)



(Koob & Le Moal, 2001)

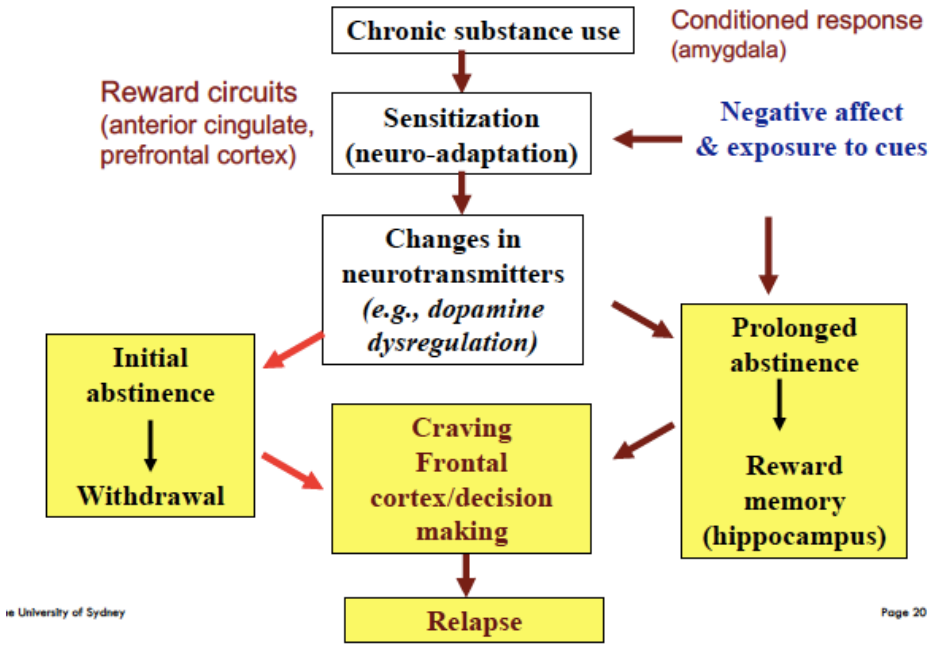
- Impaired response inhibition and salience attribution model (I-RISA) (Goldstein + Volkow)
    - Drug addiction → functional and structural changes in dopamine circuits of brain
        - Mesolimbic: Amygdala, hippocampus, NAcc
            - Amygdala = emotion-related memories
            - Hippocampus = associative learning (contextual cues etc.)
            - Nucleus accumbens = pleasure centre
            - Responsible for acute reinforcing effects (NAcc), memories + conditioning of drug cues (hippocampus), emotion and motivational changes during withdrawal (amygdala)
        - Meso-cortical: PFC, OFC, ACC
            - PFC: Attention, inhibitory control
            - ACC: Monitoring behavior
            - OFC: Cravings
            - Involved in conscious experience of euphoria, salience of drugs, expectations, cravings and inhibitory control
    - 4 clusters of behaviour:
        1. Intoxication/excitement: Higher extracellular dopamine (released by VTA) in limbic circuits (NAcc) + frontal lobe
        2. Craving: Cues → desire for drug; classical + operant conditioning of cues w/ pleasure and memories stored in amygdala + hippocampus; thalamo-orbitofrontal circuit responsible for experience of craving
        3. Withdrawal: Dysphoria, anhedonia, irritability during withdrawal (mesolimbic circuits), w/ involvement of frontal cortical circuits (for inhibition + control, and relapse)
        4. Compulsive use: Continued use even when no longer pleasurable
    - Factors contributing to addictive behaviour:



        1. Incentive salience of drug (expect +ve effects)→ binge/intoxication w/ dopamine released into VTA (part of basal ganglia) (acute reinforcing effects)
        2. → Diminished basal ganglia reward circuitry (neuroadaptation → tolerance + withdrawal effects)
        3. Increased stress w/o drug → withdrawal/-ve affect w/ activation of amygdala stress systems

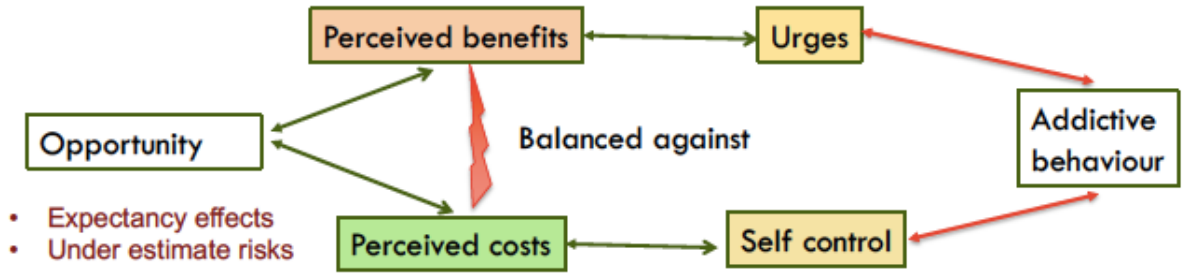
4. **Executive function deficits → preoccupation/ anticipation** of drug involving PFC
5. → Disrupted behavioural inhibition using PFC → binge

- Biological model:
  1. Chronic substance use → neuroadaptation/sensitization of reward circuits (ACC, PFC) i.e. changes in NT + downregulation in dopamine receptors
  2. Initial abstinence: Withdrawal effects → cravings → relapse w/ frontal cortex inhibited
  3. Prolonged abstinence: Negative affect and exposure to cues → memory of reward (hippocampus) → cravings → relapse w/ frontal cortex inhibited



ADDICTION AS CHOICE

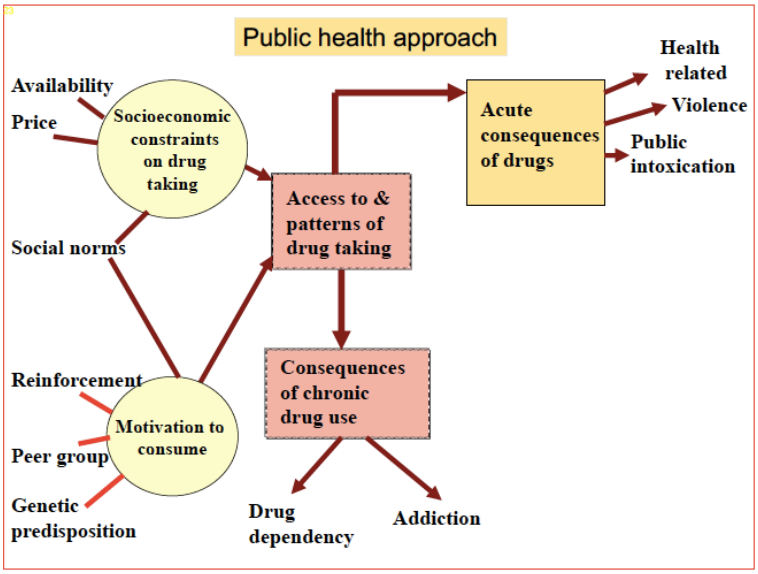
- Focus on compulsion i.e. inability to resist acting on desire; behaviour is choice rather than loss of capacity for self-control (medical model)
- Different models:
  - Rational informed stable choice (Vuchinich, Heather)
  - Theory of rational addiction (Becker + Murphy)
  - Self-medication model (Gelkopf)
  - Choice theory (Skog)
- Addiction as choice (West, 2006)



- Addictive behaviour involves balancing perceived benefits w/ perceived costs → choice
  - Perceived benefits → urges; perceived costs → self-control
  - Behaviour has utility: Subjective short-term benefits outweigh long term costs w/ underestimation of risks → addictive behaviour
  - Unstable preferences i.e. failure to cease consumption despite perceived costs

PUBLIC HEALTH APPROACH TO TREATMENT

- Need to intervene at multiple levels which influence behaviour
  1. Socioeconomic constraints on drug taking: Availability, price, social norms
     a. Can reduce availability and increase price
  2. Motivation to consume: Reinforcement (reduce –ve affect etc.), peer group, genetic disposition (e.g. impulsivity), social norms
     a. Attempt to alter social norms
- *Drug taking behaviour → drug dependency + addiction, health-related problems, violence, etc.



CLINICAL INTERVENTION AND CHALLENGES

- Barriers:
  1. Psychiatric comorbidity: Need to treat all e.g. PTSD, depression which may → -ve affect + drug consumption
  2. Acute/chronic cognitive deficits: Inhibitory problems
  3. Medical problems: May need to consume drug for medical problem
  4. Social stressors: Predispose people to consume drugs to alleviate -ve affect → need to address e.g. poverty
  5. Lack of social resources: Little support available to treat addiction
  6. Stigma: Stigma to seeking help
- Principles of effective treatment:
  1. Treatment needs to be readily available and accessible
  2. No single treatment is sufficient – need multiple

3. Need to address psychological, medical, social needs e.g. use CBT, naloxone, peer support
4. Need to treat comorbid conditions
5. Can use mandated treatment e.g. treatment is condition for parole, early release
    a. Have found more +ve results
6. Need multiple episodes of treatment to prevent relapse

- Learning-based treatment: Target maladaptive behaviour, motivation + cognitive barriers to change, skills deficits
    1. Identify high risk situations + events which can → relapse (people, places, internal cues), and reduce likelihood they are encountered e.g. alternative activities
    2. Rehearse non-drug alternative behaviours to cues + -ve affect e.g. go for a walk rather than consume alcohol when stressed, garner social support
    3. Target cognitions enhancing drug use + why they give up e.g. 'one drink won't hurt', 'this won't work'
    4. Improve emotion regulation + distress intolerance
    5. Motivational interviewing i.e. why does patient want to be treated