ALCOHOL AND OTHER DRUG USE RELATED BRAIN CHANGES

- How do alcohol and other drugs work in the brain?

1. Neuro –transmitters
2. Pleasure centre: upregulated with AOD; down regulated when AOD withdrawn
3. Other brain effects: long term use causes disruptions to neuro –T. balance, axonal functioning and structure – changes are reversible to a degree.
4. State dependant learning
5. Indirect effects of AOD: nutrition, strokes, liver damage

ALCOHOL RELATED BRAIN INJURY

What is ARBI

- Physical injury that can be directly and indirectly attributed to the effects of alcohol
- Differs from dementia
- reversible

How does alcohol cause brain injury

- Alters brain cell physiology
- Acetaldehyde?

- Decreases absorption of thiamine
- Thiamine used in metabolism
- Poor nutrition
- Increased probability of significant BI if intoxicated
- Liver damage

Assessment

- ARBI effects occur on a continuum
- Damage occurs in many parts of the brain – including gray matter
- Obvious signs occur after many years – neurological signs can occur much earlier.

ASSESSMENT

- Psychometric screening should be after at least 6 weeks post-detox – assuming abstinence, thiamine, multi –V. and proper nutrition
- Screen for factors: nutrition, pattern of use, age, reports of cog deficits by others, Werneke's E.
- Mini-mental not used
- CT scan findings

ARBI

- More comprehensive assessment be conducted by a psychologist –Tests used must be culture sensitive/relevant
- Test results not necessarily indicative of day to day functioning –OT assessment may be more valid indicator.
- Verbal based test may not be valid in certain populations e.g. where English is not the primary language.
- More specific cognitive assessment protocols/tools for aboriginal and Torres strait islanders needed. Focus on functionality/non-verbal abilities

ASSESSMENT

- Assess probability of ARBI from history:
- 40+ years of age
- 10 year regular alcohol use at harmful levels (≥ 80 grams)
- Poor nutrition
- At least 1 diagnosed episode of Wernicke's Encephalopathy
- Frequent medication assisted detoxification
- Significant liver disease (including encephalopathy)

ARBI effects

- Found across all cultures/races.
- COGNITIVE:
- Frontal lobes/executive functions:

Planning, organizing, learning new info., flexibility in thinking, impulse control, initiative, processing information, word finding

Cognitive manifestations

- Difficulties in:

Formulating/operationalizing plans
Thinking/planning ahead
Solving novel problems
Decision making
Goal setting
Reacting to environmental changes
Learning new info
Learning from mistakes
Thinking through consequences (‘gambling paradigm’)
Ability to perceive other’s view
Visual-spatial abilities
Perceptual-motor integration

Memory problems

- Immediate: not usually affected if concentration ok
- short term/working: (hold and manipulate info) most affected
- long term: not usually affected, however can experience:

chronological confusion, confabulation, lack of insight into memory probs.

Memory problems related to retrieval

Behavioural manifestations of ARBI

- Disorientation –get easily lost in a new environment e.g. rural to city
- Routines/repetitive behaviour
- Lack of spontaneity
- Poor social skills

Emotional manifestations of ARBI

- Blunted affect
- Amotivational
- Low frustration threshold
- Decreased ability to control anger expression
- Uncharacteristic aggression
- Not understanding how their behaviour affects others.

Non verbal manifestations of ARBI

- Slowed reaction time
- Decreased visual-motor coordination
- Decreased psychomotor speed
- Cerebellar ataxia

Less affected abilities

- Well learned behaviours
- Long term memory
- Well learned vocabulary/verbal skills

Other factors to be considered

- Liver functioning
- Nutrition
- Head injuries
- Medication
- Other drug use
- Pre-morbid functioning/disorders

Treatment

- Early warning signs
- Facilitate understanding and acceptance of limitations
- Memory aids
- Memory retraining
- Basic behavioural strategies (routines/structure)
- Avoid ‘talking therapies’
- Involvement of significant culturally relevant others/ agencies/community (community reinforcement approach)
- Environmental context of treatment – especially for aboriginal people in rural areas.

CANNABIS

- -Endogenous receptors in the hippocampus, amygdala , cerebellum, basal ganglia and to lesser degree in regions of the cortex (and testes)
- Increased THC concentration in current strains
- No firm evidence of permanent brain changes in early studies however improved scanning suggest changes in the hippocampus and amygdala. However no correlation with cognitive deficits
- No firm evidence of amotivational syndrome

CANNABIS

- Dose dependent relationship re: induced mental health problems
- A long term study findings indicated significant adverse effects on developing brain in adolescents
- Can facilitate expression of genetic predisposition of mental health problems

STIMULANTS

- Various illicit forms of amphetamines -powder, ‘ice’, paste. Operate on the dopaminergic system in the striatal and other areas.
- Dexamphetamine used in ADHD
- Other stimulants: Ritalin, caffeine, nicotine
- Various methods of administration
- ACUTE EFFECTS:
- Alertness, autonomic arousal, enhanced sexual arousal, decreased appetite

STIMULANTS

BRAIN CHANGES WITH LONG TERM USE (based mainly on animal studies)

- Frontal lobe damage
- Increased probability of CVA
- Depletion of dopamine, norepinephrine, serotonin
- Reduction in the size of the hypothalamus/Corpus Collosum; damage in left orbito-frontal region
- Altered receptor structure
- Decreased white matter
- Shearing of axons
- Altered brain metabolism
- Oxidative stress

STIMULANTS

MANIFESTATIONS:

- Shortened attention span
- Short term memory deficits
- Deficits in learning new information
- Slowed processing speed
- Lowered frustration threshold
- Poor decision making –'gambling task'
- Anhedonia
- Psychosis (sleep deprivation?)

SOLVENTS

- Many forms
- Administration: sniffing, huffing, bagging

ACUTE EFFECTS: Speech, ataxia, disorientation, visual hallucinations – can progress to coma, seizures and death

Chronic use: heart, liver and renal problems

BRAIN CHANGES: cerebral and cerebella atrophy, white matter degeneration which can manifest as:

Attention deficit, psychomotor dysfunction, insomnia, depression, parkinsonism, decrease IQ

HOWEVER evidence suggests these aspects are reversible if there is minimal cell death.

Note: Lead petrol can cause permanent disability due to the lead.

ADOLESCENT BRAIN DEVELOPMENT

- Popular beliefs related to brain development

- Stages of adolescence:

10-14 yrs.: start of sexual development; concrete thinking; info. processing deficits; mimic adult behaviours, peer group influence

15-17 yrs.: significant sexual development; peer group influence increases; increase in family conflicts; death an abstraction; increased risk taking

18-21 yrs: developing sense of self; career/life goal come into focus; increase in abstract thought; increasing awareness of risks

Resilience factors: positive adult role model; peer group acceptance; good impulse control; sense of positive future; cultural connectedness/identity; good communication skills

ADOLESCENT BRAIN DEVELOPMENT

- Significant brain development between 11-25 years;

Proliferation of new cells influenced by genetics, hormones and environment;

gray matter peaks at age 11 in girls and 12 in boys;

selected stimuli may increase the number and type of cells

Pruning of cells (also in first 18 months after birth);

Myelinization (nutrition important) This can decrease the brain's ability to recover from injury as well as the ability to learn novel concepts

ADOLESCENT BRAIN DEVELOPMENT

- STAGES OF SEQUENTIAL DEVELOPMENT:

Cerebellum

Amygdala

Basal ganglia

Corpus collosum

Prefrontal cortex

- Consequences of lag in development of prefrontal cortex
- Hormones can influence maturational process
- Development of pleasure centre

ADOLESCENT BRAIN DEVELOPMENT

- Developing brain is sensitive to chemicals in the brain (ie drugs):

Drug use disrupts neurochemistry, physiology

Drug use related lifestyle influences the environment, personality development, social interactions which in turn can influence brain development.

ADOLESCENT BRAIN DEVELOPMENT

- Cognitive, emotional and behavioural manifestations in brain development:

Balancing reward/approach and harm/avoidance system;
Focus on immediate short term relevant feedback;
Difficulties in response inhibition;
Ability to select appropriate stimuli/ideas;
Decision making process still developing;
Sensation seeking (evolutionary basis);
Reduced melatonin;
Greater probability of physiological disruption;
Increase in dopamine/decrease in serotonin;
Stress sensitivity heightened;
Don't purposely seek health or other risks – vulnerable to risk taking when situation arises;

ADOLESCENT BRAIN DEVELOPMENT

- IMPLICATIONS FOR ‘TREATMENT’:

Importance of family involvement where possible

Avoid ‘deep and meaningful’

Conditioning factors not as important? – brain developing/no ‘stable’ drug use memories?

Focus on practical aspects

Problems in emotional communication/empathy –be patient!

Implications for Motivational Interviewing

Foetal Alcohol Syndrome

The sequelae of alcohol use and related lifestyle, including other drug use and poor nutrition

- Incidence: varies according to method of detection/assessment :

Up to 1.7 per 1,000 live births; 1.87 -4.7 per 1000 births in aboriginal population ; (Burns et al, 2013, cited in Aust. Inst. Family Stud. Dec 2014)

- 1% of live births in Europe ?(Br. J. of Special education, 2011)
- Higher incidence in Aboriginal population?

Foetal Alcohol Spectrum Disorder

- Spectrum of severity of signs and symptoms in a group of related diagnoses.

FAS – facial abnormalities; reduced birth size and growth; behaviour problems; cognitive problems; (significant alcohol use by mother)

pFAS: significant structural, neurological and/or functional abnormalities of the CNS but not all the facial/growth problems of FAS (confirmed history of prenatal exposure to

Foetal Alcohol Spectrum Disorder

Alcohol related neurodevelopmental disorder(ARND) –severe CNS dysfunction in absence of facial anomalies in context of confirmed prenatal exposure to alcohol and:

Clinically significant structural (head/brain structure) or neurological abnormality (e.g. seizure disorder) AND/OR

Severe dysfunction in 3 or more of cognition, language, memory, executive function, attention/ activity, social/adaptive skills

Foetal Alcohol Spectrum Disorder

- Behavioural/adaptive problems manifest as:

Disrupted school experience

Forensic problems

Confinement (police, rehab) –police can be the main agency in their life

Inappropriate sexual behaviours

Mental health problems

Alcohol and other drug related problems.

The probability of manifesting above is decreased if diagnosed at an early age and/or being reared in a stable environment

Foetal Alcohol Spectrum Disorder

- Psychological consequences:

Below average intellect

- Problems in :

Problem solving, Planning, concept formation, inhibitory control, working memory, verbal learning; non-verbal learning and memory, visual-spatial abilities, fine/gross motor ability, transfer of learning, sensory integration

- hyperactivity and poor attention;

Foetal Alcohol Spectrum Disorder

- Alcohol related physical birth defects:

Malformations/abnormal growth associated with prenatal alcohol exposure – heart, kidney, skeletal, renal, ocular, auditory and others.

Some physical defects can change with physical development, however the cognitive problems tend to persist – can get worse without appropriate intervention

Foetal Alcohol Spectrum Disorder

Factors:

- How much alcohol?
- How often? Binge pattern can be more damaging than regular use?
- Timing of exposure – 1st and 3rd trimester worse?
- Mother's physiological response to alcohol (e.g. metabolism)
- Nutrition (often poor in women with alcohol misuse)
- Falls and knocks (when intoxicated)
- Age
- Socio –economic status (related to prenatal care)
- Education re: effects of alcohol on the foetus.

Foetal Alcohol Spectrum Disorder

- Maternal risk factors (U.S. Study, NIH):

Poor health; 25+ years; had 3 children prior when FAS child was born; use of other drugs; low SES; unemployment; transience; early age onset of drinking; low self esteem, depression, sexual dysfunction; family history of alcohol misuse; partner with alcohol use related issues.

Foetal Alcohol Spectrum Disorder

- Minimising/preventing/treating FASD:

Early diagnosis –relevant health professionals need to be trained to detect and discuss with the mother/family

Targeted long term support/intervention strategies for the individual and family – including cultural aspects

Use of government support agencies – cooperation not conflict driven

Foetal Alcohol Spectrum Disorder

- Psycho-education –providing support to the pregnant mother rather than confrontation. Knowledge of the effects of alcohol on foetus not necessarily enough to induce behaviour change.
- Treat the mother holistically not focus on AOD use –include partner. Many women in lower SES are wary of health agencies – e.g. fear of being reported to child welfare related agency
- Minimise harm to mother and foetus.
- Assertive follow up especially in high risk situations

Foetal Alcohol Spectrum Disorder

- Destimagtization –especially re: mother drinking unaware of pregnancy
- Long term stable, supportive living environment.
- Limited use of usual pharmacotherapies in pregnancy present a challenge
- Government policy