THE SCIENCE OF ADDICTION AND RECOVERY

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THE SCIENCE OF RECOVERY

• OVERVIEW
  • NEUROBIOLOGY OF ADDICTIVE DISORDERS
  • GENETICS
  • THE REWARD PATHWAY
  • REVERSAL LEARNING
  • PSYCHOLOGICAL ASPECTS OF RECOVERY
    • ADDICTION AND AFFECT REGULATION
    • ADDICTION AND COGNITIVE FUNCTION
    • ADDICTION AND ATTACHMENT
    • ADDICTION AND SLEEP
  • APPENDIX

NEUROBIOLOGY OF ADDICTIVE DISORDERS

• The addiction process involves a three-stage cycle
  • BINGE/INTOXICATION
  • WITHDRAWAL/NEGATIVE AFFECT
  • PREOCCUPATION/ANTICIPATION
• Disruptions in three areas of the brain are particularly important in the onset, development and maintenance of addictive disorders
  • BASAL GANGLIA
  • EXTENDED AMYGDALA
  • PREFRONTAL CORTEX

NEUROBIOLOGY OF ADDICTIVE DISORDERS

• These disruptions…
  • Enable substance-associated cues to trigger drug seeking (increase incentive salience)
  • Reduce sensitivity of brain systems involved in pleasure or reward and heighten activation of brain stress systems
  • Reduce functioning of brain executive control systems in the prefrontal cortex which are involved in the ability to make decisions and regulate one's actions, emotions and impulses
  • These changes persist long after substance use stops
NEUROBIOLOGY OF ADDICTIVE DISORDERS

THREE AREAS OF THE BRAIN

• BASAL GANGLIA
  - Controls the rewarding effects of substance abuse and responsible for habit formation
  - Two important sub-regions in substance use disorders
    - NUCLEUS ACCUMBENS
      - Motivation and the experience of reward
    - DORSAL STRIATUM
      - Habit formation and other routine behaviors

• EXTENDED AMYGDALA
  - Involved in stress including behavioral responses such as “fight or flight”
  - Feelings of unease, anxiety and irritability that typically accompany withdrawal
  - Interacts with the hypothalamus with controls glandular activity and in the right hemisphere has top down control of the autonomic nervous system
  - Hypothalamic-pituitary-adrenal axis controls response to stress

• PREFRONTAL CORTEX
  - Involved in executive function (i.e. the ability to organize thoughts and activities, prioritize tasks, manage time and make decisions) including exerting control over substance taking
  - These three brain areas and their associated networks are critical to survival and are “hijacked” by addictive substances
NEUROBIOLOGY OF ADDICTIVE DISORDERS

• THE ADDICTION CYCLE
  • A repeating cycle with three stages
    • BINGE/INTOXICATION
      • The stage in which an individual consumes a mind-altering substance and experiences a rewarding or pleasurable effect
    • WITHDRAWAL/NEGATIVE AFFECT
      • The stage at which an individual experiences a negative emotional state in the absence of the mind-altering substance
    • PREOCCUPATION/ANTICIPATION
      • The stage at which one seeks the mind-altering substance again after a period of abstinence
  • The stages are linked to and feed off of each other

NEUROBIOLOGY OF ADDICTIVE DISORDERS

• THE ADDICTION CYCLE
  • A person may go through the three-stage cycle over the course of weeks or months or progress through it several times in a day
  • Tends to intensify over time, leading to greater physical and psychological harm

• FOUR BEHAVIORS CENTRAL TO THE ADDICTION CYCLE
  • IMPULSIVITY
    • POSITIVE REINFORCEMENT (diminish over time leading to tolerance)
    • NEGATIVE REINFORCEMENT (removal of a stimulus such as negative emotions increases the probability of repeated use)
  • COMPULSIVITY (reduces tension, stress or anxiety)
NEUROBIOLOGY OF ADDICTIVE DISORDERS

• BINGE/INTOXICATION STAGE: BASAL GANGLIA
  • Heavily involves the basal ganglia and two sub-regions called the Nucleus Accumbens (NAc) and dorsal striatum
  • The rewarding effects involve activation of the nucleus accumbens and includes activation of the dopamine and opioid signaling system
  • Over time the brains reward system is “hijacked”

NEUROBIOLOGY OF ADDICTIVE DISORDERS

• Despite diverse initial actions, addictive substances produce some common effects on the Ventral Tegmental Area (VTA) and NAc
  • Stimulants directly increase dopamine transmission in the NAc. Opioids, alcohol and inhalants do the same indirectly
  • Alcohol also activates the release of opioid peptides
  • Opioids directly activate opioid peptide receptors
  • Nicotine activates dopamine neurons in the VTA
  • Cannabinoids may act in the VTA to activate dopamine neurons and also act on NAc neurons, as well as, the endogenous cannabinoid system

NEUROBIOLOGY OF ADDICTIVE DISORDERS

• Over time stimuli (People, Places and Things) can activate the dopamine system and/or trigger powerful urges to take the substance
  • The “wanting” urges are called incentive salience
  • Researchers give an animal a stimulant drug along with a previously neutral stimulus (light, sound, etc.)
  • The neutral stimulus can now cause the animal to engage in drug seeking behavior
  • In recording the electrical activity of DA-transmitting neurons that had been exposed multiple times to a neutral stimulus followed by the drug
  • At first, the neurons responded only when exposed to the drug
  • Over time the neurons stopped firing when exposed to drugs and only responded to the neutral stimulus (i.e. brain starts to release DA due to People, Places and Things)
NEUROBIOLOGY OF ADDICTIVE DISORDERS

• BINGE/INTOXICATION STAGE: BASAL GANGLIA
  - A second sub-region of the basal ganglia, the dorsal striatum involved in habit formation
  - The release of dopamine (along with activation of the brain opioid systems) and the release of glutamate trigger changes in the dorsal striatum
  - This strengthens drug-seeking contributing to compulsive use and intense craving

• SUMMARY:
  - The “reward circuitry” of the basal ganglia (i.e., the nucleus accumbens), along with dopamine and naturally occurring opioids, play a key role in the rewarding effects of alcohol and other substances and the ability of stimuli, or cues, associated with that substance use to trigger craving, substance seeking, and use.
  - As alcohol or substance use progresses, repeated activation of the “habit circuitry” of the basal ganglia (i.e., the dorsal striatum) contributes to the compulsive substance seeking and taking that are associated with addiction.
  - The involvement of these reward and habit neurocircuits helps explain the intense desire for the substance (craving) and the compulsive substance seeking that occurs when actively or previously addicted individuals are exposed to alcohol and/or drug cues in their surroundings.

NEUROBIOLOGY OF ADDICTIVE DISORDERS

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    - As alcohol or substance use progresses, repeated activation of the “habit circuitry” of the basal ganglia (i.e., the dorsal striatum) contributes to the compulsive substance seeking and taking that are associated with addiction.
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NEUROBIOLOGY OF ADDICTIVE DISORDERS

• WITHDRAWAL/NEGATIVE AFFECT STAGE: EXTENDED AMYGDALA
  - When stop taking the drug negative emotions and symptoms of physical illness appear
  - This generally leads to binge/intoxication
  - The negative feelings are thought to come from two sources diminished activation of the reward circuitry of the basal ganglia and activation of the brain stress systems in the extended amygdala
WITHDRAWAL/NEGATIVE AFFECT STAGE: EXTENDED AMYGDALA

- Imaging studies of addicts show a long-lasting decrease on DAD2 receptors (see next slide)
- Creates a reduction in the sensitivity of the brain's reward system both to addictive substances and to natural reinforcers
- This can lead to a compulsive escalation of use in an attempt to regain the pleasurable feelings the reward system once provided

A second process occurs during the withdrawal stage: activation of the stress neurotransmitters (including corticotropin releasing factor-CRH, norepinephrine and dynorphin—acts primarily at kappa opioid receptors and is generally associated with negative emotional states) in the extended amygdala (see next slide).

- These NT’s play a role in the negative feelings and in stress-triggered substance abuse
- Contributes to drug-seeking
- Neuroadaptations in the endogenous cannabinoid system within the extended amygdala also contribute to the negative feelings and enhanced stress-reactivity
- Negative reinforcement leading to a vicious cycle—take drug to lessen symptoms only to have worse symptoms when you next stop using
NEUROBIOLOGY OF ADDICTIVE DISORDERS:

- WITHDRAWAL/NEGATIVE AFFECT STAGE: EXTENDED AMYGDALA
- SUMMARY:
  - This stage of addiction involves a decrease in the function of the brain reward systems and an activation of stress neurotransmitters, such as CRF and dynorphin, in the extended amygdala. Together, these phenomena provide a powerful neurochemical basis for the negative emotional state associated with withdrawal. The drive to alleviate these negative feelings negatively reinforces alcohol or drug use and drives compulsive substance taking.

NEUROBIOLOGY OF ADDICTIVE DISORDERS:

- PREOCCUPATION/ANTICIPATION STAGE: PREFRONTAL CORTEX
- SUMMARY:
  - When addict begins to drug-seek
  - In severe addiction this can be very short-minutes to hours
  - Preoccupied which is commonly called “craving”
  - This involves the prefrontal cortex (PFC)
  - Prefrontal controls executive functions: the ability to organize thoughts and activities, prioritize tasks, manage time, make decisions and regulate one's actions, emotions and impulses
    - Whether or not to use and to override strong urges
NEUROBIOLOGY OF ADDICTIVE DISORDERS

• **PREOCCUPATION/ANTICIPATION STAGE: PREFRONTAL CORTEX**
  - Think of the prefrontal as having a "Go System" and an opposing "Stop System"
  - "Go System":
    - Helps people make decisions that require significant attention and planning
    - Use when begin behaviors that help reach goals
    - Research shows that when substance-seeking behavior is triggered by "cues", activity in the "Go" circuit increases dramatically
    - The increased activity stimulates the nucleus accumbens to release glutamate promoting incentive salience which creates a powerful "craving"

• **PREOCCUPATION/ANTICIPATION STAGE: PREFRONTAL CORTEX**
  - The "Stop" system inhibits the activity of the "Go" system
  - This system controls the dorsal striatum and nucleus accumbens the areas of the basal ganglia involved in the binge/intoxication stage
  - It seems to control habit responses driven by the dorsal striatum (i.e. it inhibits incentive salience, reduces the ability of "cues" to trigger relapse)
NEUROBIOLOGY OF ADDICTIVE DISORDERS

• PREOCCUPATION/ANTICIPATION STAGE: PREFRONTAL CORTEX
  • “Stop” system also controls the stress and emotional systems and plays an important role in relapse triggered by stressful life events or circumstances
  • Stress-induced relapse is driven by activation of CRF, dynorphin and norepinephrine in the extended amygdala
  • Lower activity in the “Stop” component of the PFC equals increased activity of the stress circuitry involving the extended amygdala and this increased activity drives substance-taking behavior and relapse

SUMMARY:
• This stage of the addiction cycle is characterized by a disruption of executive function caused by a compromised prefrontal cortex. The activity of the neurotransmitter glutamate is increased, which drives substance use habits associated with craving, and disrupts how dopamine influences the frontal cortex. The over-activation of the Go system in the prefrontal cortex promotes habit-like substance seeking, and the under-activation of the Stop system of the prefrontal cortex promotes impulsive and compulsive substance seeking.

PROMINENT IN PERSONS WITH PTSD
• APPROACH BY STRENGTHENING THE PFC

*NEUROBIOLOGY OF ADDICTIVE DISORDERS*
NEUROBIOLOGY OF ADDICTIVE DISORDERS
PRIMARY BRAIN SYSTEMS AND NEUROTRANSMITTERS

• **OPIOIDS**
  • Attach to opioid receptors leading to release of dopamine
  • Causes euphoria, drowsiness, slowed breathing and reduction in pain

• **ALCOHOL**
  • Interacts with several neurotransmitter systems including GABA, glutamate and endorphins
  • Cause euphoria, sedation, motor impairment and anxiety reduction

• **STIMULANTS**
  • Cause increase of dopamine either by directly stimulating the release of dopamine or by inhibiting the removal from the synapse
  • Boost dopamine in areas important for attention and focus
  • Also cause release of norepinephrine increasing autonomic functions

• **CANNABIS**
  • Leads to increased dopamine in the basal ganglia
  • Interacts with a wide variety of other systems and circuits that contain cannabinoid receptors

• **SYNTHETICS**
  • Cathinones ("bath salts") release dopamine similar to stimulants
  • Cannabinoids mimic effect of marijuana but much stronger
  • MDMA and LSD act on serotonin to produce changes in perception
VARIATION IN GENE FOR MU RECEPTOR CAN INFLUENCE RESPONSE

• A single nucleotide polymorphism (SNP) in the messenger RNA of the µ-opioid receptor gene was associated with patients’ responses to methadone treatment for opioid dependence.
• The association may occur because one variant of the SNP produces fewer µ-opioid receptors than the alternate form.


ENDOCANNABINOID REGULATES COCAINE REWARD

• The firing of dopaminergic neurons that are situated in the ventral tegmental area underlies feelings of reward. **Dopaminergic-neuron firing is promoted by synaptic glutamate activating glutamate receptors, and inhibited by synaptic gamma-aminobutyric acid (GABA) activating GABAB receptors.** A third neurotransmitter, norepinephrine, which can indirectly promote dopaminergic firing by activating α1 receptors, generally is inactive because it is taken back up into its releasing cell via the norepinephrine transporter.
ENDOCANNABINOID REGULATES COCAINE REWARD

- Cocaine blocks the norepinephrine transporter from drawing the neurotransmitter back into its releasing cell. Consequently, norepinephrine builds up in the synapse. Higher concentrations of synaptic norepinephrine stimulate G-protein-coupled α1 receptors to:
  - Increase biochemical processes that release 2-AG, and
  - Increase dopamine neuron activity, further increasing 2-AG, which;
  - Enhances activation of cannabinoid-1 (CB1) receptors on GABA neurons, which;
  - Lowers GABA neurons’ release of GABA into the synapse, which;
  - Tilts the balance of glutamate stimulation and GABA inhibition of dopaminergic neuron firing in favor of stimulation, which;
  - Increases the neurons’ release of dopamine into the nucleus accumbens, which;
  - Enhances feelings of reward.

THE SCIENCE OF ADDICTION AND RECOVERY: GENETICS

- Regardless of the drug involved about 50% of the risk is genetic within a range of about 40-60%
- Certain variations of certain genes can increase the risk of developing addiction
  - A gene is a giant molecule that carries information
  - A gene is a section of a chromosome with a recipe for a certain molecule

GENETICS

- Genes can increase or reduce risk
- However there is another very important part of the formula.
  - Environment, upbringing and exposure to stress are some of the factors that can trump genetics
- Epigenetics is the science of understanding what increases or decreases the activities of certain genes
  - Epigenetics control whether risk factors become disorders
A signaling pathway associated with alcoholism is regulated by a gene, called neurofibromatosis type 1 (Nf1), which scientists found is linked with excessive drinking in mice. The new research shows Nf1 regulates gamma-aminobutyric acid (GABA), a neurotransmitter that lowers anxiety and increases feelings of relaxation.

Variations in the human version of the Nf1 gene are linked to alcohol-dependence risk and severity in patients.

Nf1 is one of those rare risk genes, but the researchers weren’t sure exactly how Nf1 affected the brain. The research team suspected that Nf1 might be relevant to alcohol-related GABA activity in an area of the brain called the central amygdala, which is important in decision-making and stress- and addiction-related processes.

They found that mice with functional Nf1 genes steadily increased their ethanol intake starting after just one episode of withdrawal. Conversely, mice with a partially deleted Nf1 gene showed no increase in alcohol consumption.

The researchers found that in mice with partially deleted Nf1 genes, alcohol consumption did not further increase GABA release in the central amygdala. In contrast, in mice with functional Nf1 genes, alcohol consumption resulted in an increase in central amygdala GABA.

In the second part of the study, a collaboration with a distinguished group of geneticists at various U.S. institutions, the team analyzed data on human variations of the Nf1 gene from about 9,000 people. The results showed an association between the gene and alcohol-dependence risk and severity.

A1 Allele of the dopamine D2 receptor gene
• Found in one-third of the population
• Reduced density of D2DA receptors in the striatum
• THE MORE SEVERE THE ALCOHOLISM THE HIGHER THE ASSOCIATION WITH THE A1 ALLELE

LOW CNS DA TONE

A shortage of D2 receptors, some researchers surmise, could predispose a person to addiction.
• Nora Volkow, NIDA Director, led two studies that involved artificially increasing the number of D2 receptors in rats by administering adenoviral vectors directly into their brains. Viral vectors transmit their genetic material and makeup into foreign cells, in this case increasing the number of D2 receptors in the new cells to match their own.

In one study involving rats and alcohol, the increased number of D2 receptors led the rodents to consume less alcohol, compared with their baseline intake.
• In the other study, the D2-receptor increase caused rats to significantly reduce their intake of cocaine.
Michael Nader, a researcher at Wake Forest School of Medicine, is investigating ways to raise D2-receptor levels naturally. One experiment he helped conduct focused on five separate groups of four monkeys. Each had been self-administering cocaine to the point of habit and were then deprived of the drug for an eight-month period. To create a picture of D2-receptor availability, the monkeys were given a radioactive tracer that competes with dopamine for receptors.

The monkeys were then randomly put in social groups of four and given the opportunity to self-administer the drug again. Positron emission tomography (PET) imaging of the monkeys over time showed fluctuations in dopamine levels, which allowed the researchers to estimate the changing numbers of available D2 receptors. After only three months, the socially dominant monkeys in each group had naturally increased their numbers of D2 receptors.

There was no increase in the subordinate monkeys. Further, the subordinate monkeys reverted to using cocaine at much higher levels than the dominant monkeys. "There is an interesting relationship between D2-receptor numbers and vulnerability to drug addiction," Nader said. "It appears that individuals with low D2 measures are more vulnerable compared to individuals with high D2-receptor numbers."
**GENETICS**

- Why did the socially dominant monkeys show D2-receptor increases? “One hypothesis,” Nader said, “is *environmental enrichment.*” For the monkeys, it seems, being dominant was the enriching trigger.
- One physiological consequence of involvement in 12-step meetings, therefore, could be an increase in the natural production of D2 receptors.

**GENETICS**

- Anandamide is specifically responsible for regulating anxiety
- It produces calmness and relaxation when released
- If you make less of it you will probably be prone to anxiety
- In fact many who smoke marijuana (specifically THC) do so to reduce anxiety

**GENETICS**

- FAAH stands for fatty acid amide hydrolase
- FAAH is an enzyme that deactivates anandamide
- FAAH is a protein and like every protein the instructions on how to make it are encoded on a specific gene
- There is a variation of the FAAH gene that weakens it (less FAAH is available)
- Less FAAH equals more anandamide
- People with this variant are less prone to anxiety and actually have an aversion to marijuana

**REWARD PATHWAY**

- The beginning of the brain's addiction pathway involves the Ventral Tegmental Area (VTA) releasing dopamine that binds to the Nucleus Accumbens (NAc) activating survival instincts
- For those vulnerable to addiction, the initial activation of this pathway results in much greater NAc activation encouraging the activity mistaken to be of survival value
**REWARD PATHWAY**

- Excessive dopamine release leads to depletion resulting in craving and obsession.
- Another major dopamine brain circuit runs from the prefrontal cortex (PFC) to the reward center.
- The feedback mechanism does not work properly and the control circuitry is not activated by the dopamine sent from the NAc or there is a poor connection from the PFC to NAc. The “stop” message is mediated by glutamate.

**REWARD PATHWAY**

- Other neurotransmitters involved in the addiction pathway include norepinephrine, endorphins and GABA.
- *Endorphins* facilitate saliency providing greater influence to brain addiction related processes as opposed to other processes.
- GABA is involved in withdrawal and craving.
REWARD PATHWAY

• Pain can produce a reward deficiency syndrome, as does chronic opioid self-administration
• Learning how to increase endogenous rewarding mechanisms is an important area of research
• An example of this is how to increase endorphin levels (SEE APPENDIX)

REVERSAL LEARNING

Addiction as impairment in reversal learning
IN ADDICTION...

"WHEN I USE DRUGS I FEEL GOOD"

“WHEN I USE DRUGS BAD THINGS HAPPEN”

NEW RULE BUT CANNOT ADAPT

REVERSAL LEARNING

• Changes in brain glutamate signaling induced by chronic drug exposure causes a wide variety of neurological effects instrumental in the transition from abuse to addiction (Kavalis, 2009)
• These neural alterations limit the ability to adapt to new information (to stop taking drugs in spite of adverse consequences) and strengthens the power of drug learned associations

REVERSAL LEARNING

• Addicts can learn a new rule but run into problems when the rule changes
  • Cocaine and alcohol abusers were asked to press key each time they saw a green rectangle on the screen
  • After 500 repetitions told not to press key when viewing the green rectangle
  • Controls easily adapted while addicts kept pushing the key when the green rectangle appeared even after given feedback

IMPAIRED REVERSAL LEARNING DUE TO DRUG USE AND NOT GENETICS
THE SCIENCE OF ADDICTION AND RECOVERY: PSYCHOLOGICAL

First consideration:
Is the patient a candidate for rehabilitation or do they need habilitation?

PSYCHOLOGICAL

If Habilitation
Emphasis is on:
Neuroplastic development of the prefrontal cortex
Staff as healthy family
Discharge planning
“Wrap around” services

PSYCHOLOGICAL

• PREFRONTAL TAKES AROUND 25 YEARS TO DEVELOP IN A GOOD ENVIRONMENT

0-5 YEARS OLD- NURTUREANCE
10-20 YEARS OLD- SUPERVISION
PSYCHOLOGICAL

Second consideration:
Where is the patient in regard to their desire to change?

PSYCHOLOGICAL

• If patient is in precontemplation or contemplation (Stages of Change Model), the use of Motivational Interviewing techniques are warranted
• Education and assessment can serve as confrontational tools
  • To create cognitive dissonance

PSYCHOLOGICAL

Third consideration:
Is there presence or absence of a recovery-oriented environment and/or support system?

RECOVERY SAFETY PLAN

A recovery safety plan involves 3 parts. The first part helps each client identify how they will know they need to activate their safety plan. The second part dictates a place of physical/psychological safety to which they will go as quickly as is possible. The third is a list of who they will contact as soon as they can.
RECOVERY SAFETY PLAN

PART I. I will know I am in trouble and need to activate my recovery safety plan if any of the following occur:
1. ____________________________________
2. ____________________________________
3. ____________________________________
4. ____________________________________
5. ____________________________________

PART II. If I experience any of the above, I will go to one of the following places as soon as I can get there.
1. ____________________________________
2. ____________________________________
3. ____________________________________

PART III. If I experience any of the above, I will contact the following people as soon as I possibly can.
1. ____________________________________
2. ____________________________________
3. ____________________________________

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<th>NAME</th>
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PSYCHOLOGICAL

Fourth consideration:
Understanding and treating the patients that struggle
History of maltreatment with or without self-injurious behavior
Earlier onset of use and other self-destructive behaviors
Personality immaturity
High levels of distress (Neuroticism)
Nonenriched environment
Lack of recovery capital

MALTREATMENT

• Maltreatment is associated with an earlier age of alcohol/drug initiation, a greater likelihood of engagement in risky sexual behaviors, a greater risk for recent incarceration, greater ratings of psychological distress, and a greater risk for comorbid personality disorders. Physical maltreatment appears to be a particularly salient risk factor for the development of substance abuse and progression to injection drug use.

MALTREATMENT

• There appear to be sensitive periods when these regions are maximally susceptible to the effects of stress. The hippocampus seems to be maximally susceptible to maltreatment in women exposed between the ages of 3 and 5 years. However, when maltreatment occurred at ages 9–10, the midportion of the corpus callosum was maximally susceptible, and at ages 14–16, the prefrontal cortex was affected. Thus, there appear to be specific windows of vulnerability in development that determine the negative effects of exposure.

MALTREATMENT

• The effects of maltreatment on brain functioning may not appear immediately after exposure. Several studies have reported reductions in the gray matter volume of the hippocampus in adults with a history of maltreatment but not in maltreated children. This pattern of results is consistent with translational studies showing that effects of early stress on the hippocampus first emerge during the transition between puberty and adulthood.

MALTREATMENT

- Maltreatment also appears to affect the development of sensory systems and pathways that process and convey the adverse experience. For example, parental verbal abuse is associated with decreased fractional anisotropy in the arcuate fasciculus, which interconnects Wernicke's and Broca's areas, and with alterations in witnessing domestic violence is associated with a reduction in gray matter volume in the primary and secondary visual cortex and with decreased fractional anisotropy in the inferior longitudinal fasciculus, which interconnects the visual cortex and the limbic system to shape our emotional and memory response to things that we see.


- The longer depression persists, the more likely the subjects interviewed are to recount having undergone sexual abuse, which no doubt means that they have been exposed to severe stress on many occasions in early life.

- In addition to the usual psychological trauma, it has been demonstrated that this stress modifies the neurochemistry and structure of the brain, making it more vulnerable to depression.

ENRICHED ENVIRONMENT

A GOOD PARENT SETS GOOD LIMITS

FAIR
CONSISTENT
AVAILABLE
ENRICHED ENVIRONMENT

SETTING LIMITS

Overly Strict

Very Loose

PSYCHOLOGICAL

• ADDICTION AND AFFECT REGULATION
• ADDICTION AND COGNITIVE FUNCTIONING
• ATTACHMENT
• ADDICTION AND SLEEP

DEVELOPING THE PREFRONTAL CORTEX

• There are three areas that make up the prefrontal cortex (PFC) and its link to the limbic system
  • ORBITOFRONTAL CORTEX (VENTROMEDIAL PFC)
    • AFFECT CONTROL
    • WEIGHING DECISIONS
  • DORSOLATERAL PREFRONTAL CORTEX
    • EXECUTIVE FUNCTIONS
    • MORAL JUDGMENTS
  • ANTERIOR CINGULATE GYRUS
    • RELATIONAL
    • ATTENTION AND FOCUS

DEVELOPING THE PREFRONTAL CORTEX

• Research tells us...
  • We can impact the areas of relationships, affective control and cognitive abilities
  • RELATIONSHIPS (ATTACHMENT) = ANTERIOR CINGULATE CORTEX
  • AFFECTIVE CONTROL = ORBITOFRONTAL CORTEX
  • COGNITION = DORSOLATERAL PREFRONTAL CORTEX

THIS IS A SIMPLISTIC MODEL AS MANY DIFFERENT AREAS ARE INVOLVED
EMOTIONAL DEVELOPMENT

• The core features of emotional development include the ability to identify and understand one’s own feelings, to accurately read and comprehend emotional states in others, to manage strong emotions and their expression in a constructive manner, to regulate one’s own behavior, to develop empathy for others, and to establish and maintain relationships.

EMOTIONAL DEVELOPMENT

• The motor control, meaning the myelination of the motor pathways, occurs around 15 on average. Then the dorsolateral prefrontal cortex, responsible for cognitive control and executive function, is pretty much myelinated by 25.

• The immense impact of emotions on cognitive control.
EMOTIONAL DEVELOPMENT

• Emotions can change how much control you have. So, when you look at the medial and orbital surfaces of the frontal lobe, which some call the 'social' brain, the mean age of myelination of those connections between the limbic system and those frontal areas is about 32.


EMOTIONAL DEVELOPMENT

• Slow development of emotion regulation paralleled by slow development of neurobiological substrates (mPFC and amygdala)

• Supporting regions are anti-correlated
  • Increased mPFC=Reduced amygdala activity

• Top down regulation with excitatory/inhibitory balance

EMOTIONAL DEVELOPMENT

• Amygdala to mPFC connection develops earlier than the inhibitory connection from mPFC to amygdala

• During early life the caregiver can serve as an external social regulator when an immature mPFC exists

• Children depend on adults or survival

EMOTIONAL DEVELOPMENT

• Caregiver as an external regulator of emotion…

• Social referencing
  • Intergenerational transmission of emotional knowledge

• Modulation of stress reactivity and fear learning

• Parental buffering

• Access to parental cues provide external means of regulation
EMOTIONAL DEVELOPMENT

- Modulation of stress reactivity...(continued)
  - A regulated parent can modulate a physiological response to threat
  - Parental cues can dampen elevation in the stress hormone cortisol
  - Parental cues can reduce activity of amygdala
  - Prevents threat learning
  - When parent exhibits defensive behavior can heighten negative affect


EMOTIONAL DEVELOPMENT

- Research since the 1970’s has found one factor consistently associated with positive outcome in therapy—the therapeutic relationship (strength of the relationship between client and therapist) (Butler and Strupp, 1986; Horvath and Symonds, 1991; Martin, Garske & Davis, 2000)
  - The therapeutic conditions consisted of increased levels of empathy, unconditional regard and acceptance (Rogers, 1961)
  - Largely fostered through nonverbal communication of respect, acceptance and affective attunement (ability to be present to, and with, another's expression of their experience) (D’Elia, 2001)

EMOTIONAL DEVELOPMENT

- Therapist as a secondary attachment figure promoting healthy neurological functioning and development of an adaptive stress response system
- Nonenriched (stressful) environments induce the adrenal-medullary system (SAM) to secrete epinephrine and norepinephrine (rapid and short-lived preparation of body to meet the challenge) and hypothalamic-pituitary-adrenal (HPA) axis to release cortisol (also prepares body but slower in onset and longer acting)

EMOTIONAL DEVELOPMENT

- Altered activity of HPA associated with both affective and disruptive behavior disorders (Van Goozen, et. al., 2000)
- Some clients have difficulty managing response to stressful situations while others have problems inhibiting aggressive and reactive tendencies (McEwen, 2002a)
- The secondary attachment figure can help correct these negative neurological effects associated with nonenriched environments (Hertzman, 1999)
- Enriched environments can downregulate the stress system
EMOTIONAL DEVELOPMENT

• Neurological effects of secure attachment impact right Orbitofrontal Cortex (rOFC) which can regulate the Autonomic Nervous System (ANS) by pushing down the emotional “clutch” that disengages the sympathetic “accelerator” while activating the parasympathetic “brakes” (Siegel, 1999)

• An attuned relationship can influence neurobiology by releasing oxytocin which also down regulates and soothes the stress system (Panksepp, 1998). This effect can last for several days (McEwen, 2002b)

EMOTIONAL DEVELOPMENT

• The rOFC can serve the function of what has been theoretically referred to as an “internalized object”

• The internalization of the therapist serves as a biological regulator of the client’s ANS

• Implicit representations of the supportive experience allow for future self-soothing and are stored as memories (Cozolino, 2002)

• Over time the client via plasticity manufactures their own functional connections allowing for self-soothing

EMOTIONAL DEVELOPMENT

• A “selfobject” represents an experience of another as part of the self (Kohut, 1977)

• A “selfobject” provides basic psychological needs of soothing, support and acknowledgment

• Rooted in empathic attunement or resonance between client and therapist where therapist utilizes nonverbal attending skills

• By development of the rOFC the client internalizes the functions of the “selfobject”

EMOTIONAL DEVELOPMENT

• The ability to monitor and control affect, or “emotion regulation,” refers to the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions. Impairments in emotion regulation contribute to substance use disorder (substance disorder) development, persistence, and severity
EMOTIONAL DEVELOPMENT

• Individuals with substance disorders reliably demonstrate weakened strength of resting state connectivity between the amygdala/insula and regulatory regions (Rostral Anterior Cingulate Cortex and Ventromedial Prefrontal Cortex), consistent with observations in individuals with disorders associated with negative affect.

ADDICTION AND AFFECT REGULATION

• Individuals who use substances to relieve negative affect develop addictive patterns of drug use more quickly, and emotion regulation difficulties are associated with greater substance use severity in individuals in whom a substance disorder has already developed.

• Impaired emotion regulation would render an individual with a substance disorder more vulnerable to cue-induced cravings or impulsive responding.

• Impaired emotion regulation predicts poor response to treatment and accentuates the risk of relapse during negative affect.

ADDICTION AND AFFECT REGULATION

• Well-established pharmacologic treatments for anxiety disorders, depressive disorders, and other disorders associated with impaired emotion regulation have been tested in substance disorders, most show little or no effect on substance use. (Wilens CE, Bogenschutz MP. Psychopharmacology for addictions, in Addictions: A Comprehensive Guidebook, 2nd ed. Edited by McCrady BS, Epstein EE. New York, Oxford University Press, 2013)

ADDICTION AND AFFECT REGULATION

• Individuals with substance disorders reliably demonstrate weakened strength of resting state connectivity between the amygdala/insula and regulatory regions, consistent with observations in individuals with disorders associated with negative affect.

• Decreased resting state functional connectivity is also generally observed between and within regulatory regions in substance disorders relative to controls. This weakening of functional connectivity strength may be caused by impairment in the integrity of white matter tracts.
ADDICTION AND AFFECT REGULATION

- Hypoactivation of the rostral anterior cingulate cortex/ventromedial prefrontal cortex (rACC/vmPFC) is the most consistent finding across studies, dimensions, and clinical populations (individuals with and without substance disorders).
- The same pattern is evident for regions in the cognitive control network (anterior cingulate and dorsal and ventrolateral prefrontal cortices) during cognitive modulation and behavioral control.

ADDICTION AND AFFECT REGULATION

- These congruent findings are possibly related to attenuated functional and/or structural connectivity between the amygdala and insula and between the rACC/vmPFC and cognitive control network.
- Although increased amygdala and insula activation is associated with impaired emotion regulation in individuals without substance disorders, it is not consistently observed in substance disorders.

ADDICTION AND AFFECT REGULATION

- Emotion regulation disturbances in substance disorders may therefore stem from impairments in prefrontal functioning, rather than excessive reactivity to emotional stimuli.
- Treatments for emotion regulation in individuals without substance disorders that normalize prefrontal functioning may offer greater efficacy for substance disorders than treatments that dampen reactivity.

ADDICTION AND AFFECT REGULATION

- Individuals with substance disorders reliably demonstrate weakened strength of resting state connectivity between the amygdala/insula and regulatory regions, consistent with observations in individuals with disorders associated with negative affect.
- Decreased resting state functional connectivity is also generally observed between and within regulatory regions in substance disorders relative to controls. This weakening of functional connectivity strength may be caused by impairment in the integrity of white matter tracts.
ADDICTION AND AFFECT REGULATION

• Emotion regulation disturbances in substance disorders may stem primarily from impairments in PFC activation, as a direct result of disrupted neural functioning, rather than from excessive reactivity to negatively charged affective stimuli.


ADDICTION AND AFFECT REGULATION


DEVELOPING THE PREFRONTAL CORTEX

• Role play
  • How to manage potential relapse and craving situations
  • People, Places and Things (PPT) group

• Anger management
  • How when I get angry I give up control to the person I claim is making me angry

• Spiritual
  • Each day a patient reads from a chosen passage from a spiritual text (AA, NA, Bible, Koran, Bhagavad Gita, Tao, etc. purchased as library)
  • Utilizing the right hemisphere to be in the present and to appreciate beauty
DEVELOPING THE PREFRONTAL CORTEX

• AFFECTIVE CONTROL
  • Introduction to meditation
    • Opportunity to experience various approaches to spending time in the right hemisphere
  • Develop a personalized SAFETY PLAN
    • Developed on a 3x5 index card
    • Qualifying-writing and reading your story-experience, hope and expectations

SEE APPENDIX FOR TECHNIQUES USEFUL IN MANAGING NEGATIVE AFFECT

ADDICTION AND COGNITIVE FUNCTION

• Alcohol exposure during adolescence, before the brain is fully developed, can result in cellular and synaptic abnormalities that have enduring, detrimental effects on behavior.
• If drink heavily during this period of development, there could be changes occurring that have a lasting impact on memory and other cognitive functions

ADDICTION AND COGNITIVE FUNCTION

• Adolescent animals exposed to alcohol grow into adults that are much less adept at memory tasks than normal animals -- even with no further alcohol exposure.
• What has not been known is how these impairments manifest at the cellular level in the region of the brain known as the hippocampus, where memory and learning are controlled.
ADDICTION AND COGNITIVE FUNCTION

• Something happens during adolescent alcohol exposure that changes the way the hippocampus and other regions of the brain function and how the cells actually look—for example, the dendritic spines have an immature appearance in adulthood.

• This immature quality of the brain cells might be associated with behavioral immaturity. In addition to spine changes in the hippocampus, which affects learning, structural changes in other brain regions that control impulsiveness and emotionality are impacted.

ADDICTION AND COGNITIVE FUNCTION

• It is possible that alcohol disrupts the maturation process, which can affect these cognitive function later on.


ADDICTION AND COGNITIVE FUNCTION

• Addiction is a disorder of altered cognition.

• Addiction impacts…
  • LEARNING
  • MEMORY
  • ATTENTION
  • REASON
  • IMPULSE CONTROL

• Effects are particularly disruptive when exposed during brain development and in the co-occurring population.

ADDICTION AND COGNITIVE FUNCTION

• Drugs impact on cognition include the areas…
  • STRIATUM
  • PREFRONTAL CORTEX
  • AMYGDALA
  • HIPPOCAMPUS

• These regions underlie declarative memory—the memories that define an individual and generate and maintain a concept of self.
ADDICTION AND COGNITIVE FUNCTION

• Cognitive deficits in chronic drug abuse
  • Withdrawal produces cognitive symptoms
  • Cocaine-deficits in cognitive flexibility
  • Amphetamine-deficits in attention and impulse control
  • Opioids-deficits in cognitive flexibility
  • Ethanol-deficits in working memory and attention
  • Cannabis-deficits in cognitive flexibility and attention
  • Nicotine-deficits in working memory and declarative learning

ADDICTION AND COGNITIVE FUNCTION

• Cognitive deficits in chronic drug abuse (continued)
  • In general, there is a problem with learning new patterns of thought and behavior
  • Methamphetamine—at 6 months still problems with motor functions, memory of spoken words; when retested at 12-17 months both of the above approached normal
  • MDMA—continued to score poorly on tests of immediate and delayed recall of spoken word even after 2.5 years of abstinence

ADDICTION AND COGNITIVE FUNCTION

• Paying attention, making decisions, inhibiting responses, and detecting and controlling emotions all depend on the anterior cingulate cortex
• Patients in the early stages of treatment for stimulant abuse may require help to stay focused on recovery when they make decisions

*Drug and Alcohol Dependence 113(2-3):113-138, 2011*
ADDICTION AND COGNITIVE FUNCTION

- Drugs of abuse in the developing brain
  - Prenatal
    - Fetal Alcohol Spectrum Disorders are leading cause of mental retardation in US
    - Fetal alcohol exposure increases susceptibility to later substance abuse problems
    - 5 year olds whose mother used cocaine, alcohol and/or opioids had problems with language skills, impulse control and visual attention
    - Methamphetamine-cognitive deficits and altered brain structure (shortened attention span with reduced volume in hippocampus and basal ganglia)
    - Mothers who smoked had 3 times incidence of ADHD in offspring

- Adolescent exposure
  - Smoking-working memory, verbal comprehension, oral arithmetic and auditory memory
  - Resolves with cessation except for working memory and arithmetic performance
  - Smoking is associated with later onset of depression


- Multiple assessment points to investigate the effects of cigarette smoking on cognitive recovery over the first eight months of abstinence from alcohol. We chose to examine measures of processing speed, learning and memory, and working memory because these abilities have been shown to be adversely affected by alcohol use disorders as well as chronic cigarette smoking.

ADDICTION AND COGNITIVE FUNCTION

- The ALC as a group showed the greatest rate of recovery on most abilities during the first month of abstinence. Over eight months of sustained abstinence from alcohol, active-smoking ALC showed poorer recovery than never-smoking ALC on measures of learning, and both former-smoking ALC and active-smoking ALC recovered less than never-smoking ALC on processing speed measures. In addition, after eight months of abstinence, active-smoking ALC performed worse than both controls and never-smoking ALC on most measures, former-smoking ALC performed worse than never-smoking ALC on several tests, but never-smoking ALC were not different from controls on any measure.
ADDICTION AND COGNITIVE FUNCTION

- Overall, the findings indicated never-smoking ALC showed full recovery on all measures after 8 months of sustained abstinence from alcohol.

Timothy C. Durazzo et al. Effects of Cigarette Smoking History on Neuropsychological Recovery Over 8 Months of Abstinence in Alcohol-Dependent Individuals. Alcoholism: Clinical & Experimental Research, October 2014 DOI: 10.1111/acer.12552

DEVELOPING THE PREFRONTAL CORTEX

- EXECUTIVE FUNCTIONING
  - Write a job resume and have the group give feedback
  - Practice interviewing for a job
    - The interviewer will use a standard set of questions which the patient will answer in front of the group
  - Group feedback encouraged
  - Skills Development
    - Experimenting with several opportunities to develop an interest or hobby
      - Photography, cooking, painting, drawing using multiple mediums

DEVELOPING THE PREFRONTAL CORTEX

- EXECUTIVE FUNCTIONING
  
- Jig saw puzzles, cross word puzzles, etc.
- Certain computer games that are nonviolent but demand attention and delayed gratification such as SimCity
- The puzzles and computer games can be competitive in nature leading to a discussion on winning and losing or maybe the losing team waits on the winning team at dinner, etc.

<table>
<thead>
<tr>
<th>EVENT OR SITUATION</th>
<th>AUTOMATIC THOUGHTS</th>
<th>EMOTIONS</th>
<th>LOGICAL THOUGHTS</th>
<th>OUTCOME</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actual event</td>
<td>Write AT’s</td>
<td>Specify</td>
<td>Id thinking</td>
<td>Rate feeling 0-100</td>
</tr>
<tr>
<td></td>
<td>Rate belief in AT 0-100</td>
<td>Feelings</td>
<td>distortion</td>
<td>作家 realistic healthier thought Rate belief 0-100</td>
</tr>
<tr>
<td></td>
<td>Memories</td>
<td></td>
<td></td>
<td>Describe changes in how you could handle situation</td>
</tr>
</tbody>
</table>
DEVELOPING THE PREFRONTAL CORTEX

• EXECUTIVE FUNCTIONING
  • EDUCATION
    • Why give an alcoholic or addict a 60 minute didactic or video?
    • A new format
      • 15-20 minute simple didactic
      • How to participate in treatment
      • 10 minute questionnaire
      • 30 minute discussion group

DEVELOPING THE PREFRONTAL CORTEX

I THINK...........

I FEEL............

I LEARNED......

MY FUTURE BEHAVIOR WILL CHANGE...

DEVELOPING THE PREFRONTAL CORTEX

THE NUCLEUS BASALIS IS...

NOVELTY

THE MODULATORY CONTROL CENTER FOR PLASTICITY
DEVELOPING THE PREFRONTAL CORTEX

YOU ARE NEUROPLASTICIANS!

WHAT ENHANCES PLASTICITY?

• Novelty
• Therapeutic Relationships
• Physical Exercise
• Mindfulness

ATTACHMENT

• Attachment refers the particular way in which you relate to other people. Your style of attachment was formed at the very beginning of your life, during your first two years. Once established, it is a style that stays with you and plays out today in how you relate in relationships and how you parent your children.

ATTACHMENT-EARLY ATTACHMENT PATTERNS

• Young children need to develop a relationship with at least one primary caregiver in order for their social and emotional development to occur normally
• During the first two years, how the parents or caregivers respond to their infants establishes the types of patterns of attachment their children form. These patterns will go on to guide the child's feelings, thoughts and expectations as an adult in future relationships.

SECURE ATTACHMENT

• Ideally, from the time infants are six months to two years of age, they form an emotional attachment to an adult who is attuned to them, that is, who is sensitive and responsive in their interactions with them. It is vital that this attachment figure remain a consistent caregiver throughout this period in a child's life. During the second year, children begin to use the adult as a secure base from which to explore the world and become more independent. A child in this type of relationship is securely attached.
ATTACHMENT-EARLY ATTACHMENT PATTERNS

<table>
<thead>
<tr>
<th>Attachment Style</th>
<th>Parental Style</th>
<th>Resulting Adult Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secure</td>
<td>Attached with the child; in tune with the child's emotions</td>
<td>Able to create meaningful relationships; empathetic; able to set appropriate boundaries</td>
</tr>
<tr>
<td>Avoidant</td>
<td>Unavailable or rejecting</td>
<td>Avoids closeness or emotional connection; distant, critical, rigid, intolerant</td>
</tr>
<tr>
<td>Ambivalent</td>
<td>Inconsistent and sometimes intrusive parent communication</td>
<td>Anxious and insecure; controlling; blaming; erratic; unpredictable; sometimes charming</td>
</tr>
<tr>
<td>Disorganized</td>
<td>Ignored or didn't see child's needs; parental behavior was frightening/taking away</td>
<td>Chaotic, insensitive; explosive, abusive; unstable even while craving security</td>
</tr>
<tr>
<td>Reactive</td>
<td>Extremely unattached or malfunctioning</td>
<td>Cannot establish positive relationships; often misdiagnosed</td>
</tr>
</tbody>
</table>

DISMISSIVE PERSONALITY

- Those who had avoidant attachments in childhood most likely have dismissive attachment patterns as adults. These people tend to be loners; they regard relationships and emotions as being relatively unimportant. They are cerebral and suppress their feelings. Their typical response to conflict and stressful situations is to avoid them by distancing themselves. These people’s lives are not balanced: they are inward and isolated, and emotionally removed from themselves and others.

PREOCCUPIED PERSONALITY

- Children who have an ambivalent/anxious attachment often grow up to have preoccupied attachment patterns. As adults, they are self-critical and insecure. They seek approval and reassurance from others, yet this never relieves their self-doubt. In their relationships, deep-seated feelings that they are going to be rejected make them worried and not trusting. This drives them to act clingy and overly dependent with their partner. These people’s lives are not balanced: their insecurity leaves them turned against themselves and emotionally desperate in their relationships.

SECURE PERSONALITY

- People who formed secure attachments in childhood have secure attachment patterns in adulthood. They have a strong sense of themselves and they desire close associations with others. They basically have a positive view of themselves, their partners and their relationships. Their lives are balanced: they are both secure in their independence and in their close relationships.
FEAR-AVOIDANT PERSONALITY

- People who grew up with disorganized attachments often develop fearful-avoidant patterns of attachment. Since, as children, they detached from their feelings during times of trauma, as adults, they continue to be somewhat detached from themselves. They desire relationships and are comfortable in them until they develop emotionally close. At this point, the feelings that were repressed in childhood begin to resurface and, with no awareness of them being from the past, they are experienced in the present. The person is no longer in life today but rather, is suddenly re-living an old trauma. These people's lives are not balanced: they do not have a coherent sense of themselves nor do they have a clear connection with others.

DEVELOPING THE PREFRONTAL CORTEX

- ENHANCING RELATIONAL ABILITIES (continued)
  - Music therapy
    - Examine their interpretation of their favorite lyric of their favorite song and discuss in group
    - Have the group make their own instruments and write their own recovery song to be performed at community meeting or graduation

ADDICTION AND SLEEP

- Sleep problems are commonly associated with drug and alcohol use. Nearly 70% of patients admitted for detoxification report sleep problems prior to admission, and 80% of those who report sleep problems relate them to their substance use. The association between substance use and sleep problems appears to be bidirectional, with sleep problems increasing risk for developing substance use disorders, and acute and chronic substance use leading to acute and chronic problems with sleep. Evidence also indicates that long-term abstinence from chronic substance use can reverse some sleep problems.
ADDICTION AND SLEEP-ALCOHOL

• Alcohol is widely used as a sleep-promoting agent. However, as the consumption of alcohol becomes chronic, alcohol has less of an hypnotic effect.
• Sleep complaints associated with alcohol use disorders are one of the most refractory problems to resolve, and insomnia is the most frequent complaint among alcoholics after they stop drinking.
• Congruent with increased sleep latency, total sleep time is reduced in persons with alcohol use disorders during periods of drinking, acute withdrawal, and post-acute withdrawal.

ADDICTION AND SLEEP-ALCOHOL

• Acute alcohol use has been shown to reverse the chronic slow wave sleep deficits observed in chronic alcohol users. Given the widespread importance of slow-wave sleep in factors including sleep continuity, learning, and memory, as well as other types of cognitive performance, the deficits associated with chronic use (and their reversal with acute use of alcohol) suggests the particular importance of slow-wave sleep in alcohol use disorders. More specifically, as the brain processes that underlie the generation of these slow waves appear to be chronically altered by chronic alcohol use, and to be temporarily restored by acute use, this chronic alteration is implicated as a potential factor in relapse.

ADDICTION AND SLEEP-CANNABIS

• Cannabis may improve subjective sleep complaints, particularly when used over short periods of time.
• Chronic cannabis use is associated with negative subjective effects on sleep that are manifested most prominently during withdrawal.
• Symptoms reported include sleep difficulties such as strange dreams, insomnia, and poor sleep quality. Such symptoms occur in anywhere from 32% to 76% of persons experiencing withdrawal.

ADDICTION AND SLEEP-CANNABIS

• Among the problems with sleep in chronic cannabis users is the presence of strange dreams. Such dreams typically begin 1–3 days after cannabis discontinuation—when sleep quality is particularly poor, peak after 2–6 days, and last 4–14 days, coincident with other subjective sleep complaints. However, large studies have found sleep difficulties lasting for longer periods, such as 43 days, and strange dreams in particular lasting for as long as 45 days. Returning to cannabis use (or using alcohol or other sedatives) to promote sleep is commonly observed.
ADDICTION AND SLEEP-COCAIN

- The first several days to 1 week after cocaine cessation are characterized by sleep disturbances, hypersomnia, bad dreams, depressed mood, psychomotor agitation and retardation, fatigue, and increased appetite. With continued abstinence, however, there is subjective improvement of sleep as well as improvements in other cocaine withdrawal measures, with apparent normalization of subjective sleep over the course of several weeks.
- Although self-reported sleep improves following the initial withdrawal from cocaine, polysomnographic findings have consistently shown deterioration in sleep to insomnia-like levels in the same period.

ADDICTION AND SLEEP-OPIATES

- Dizziness and sleepiness are common side effects of opioid pain medications. With a stable dose, tolerance to the subjective, sedative effects of opioids develops within 2-3 days and some studies find that cognition normalizes after that, supporting the notion of tolerance to the sedative effects. However, there is also evidence that unpleasant sedative effects, decreased alertness and increased reaction time in a variety of cognitive tasks continue to be experienced by some patients on a stable dose of narcotic medication.
- Insomnia, hypersomnia, increased sleep latency, and reduced sleep duration in individuals with opioid use disorder after 3 weeks of abstinence.

ADDICTION AND SLEEP-OPIATES

- Several studies have shown that acute use of various opioids results in increased REM latency, decreased REM sleep time, increased stage 1 and stage 2 sleep, and decreased slow-wave sleep. Acute use of opioids also leads to increased sleep latency, increased wakefulness after sleep onset (WASO), and concomitant decreases in total sleep time (TST) and sleep efficiency (SE).
- During the first 3 weeks of abstinence, prolonged sleep latency, decreased sleep efficiency, decreased TST, increased arousal index, increased stage 1 and 2, and decreased slow-wave sleep (SWS) were prominent compared to healthy sleepers. After 6 weeks and up to 6 months of abstinence from methadone, there is a rebound increase in SWS and REM time to a higher level than baseline.
ADDICTION AND SLEEP-OPIATES

• Chronic opioid use has been associated with several abnormalities including nocturnal oxygen desaturations, abnormal breathing patterns, and Biot's respiration pattern which ultimately may lead to hypercapnia and hypoxia. Chronic opioid treatment, particularly with extended release preparations is associated with increased risk of central and obstructive sleep apneas.

• Several studies have indicated that chronic opioid use is an independent risk factor for irregular breathing, central apneas, and hypopneas.

ADDICTION AND SLEEP-SUMMARY

• The effects of chronic use on sleep are similar among both CNS stimulants and depressants. Decreased sleep time, increased sleep latency and wake time after sleep onset, and deficiency in slow-wave sleep generation appear to be common to chronic use of alcohol, cocaine, cannabis, and opiates. REM sleep is also affected by acute and chronic use.

• Also linking these abnormalities are connections with ongoing use and relapse. However, treatment with typical sleep promoting agents that increase sleep time or efficiency by increasing light sleep may be counterproductive.

SEE APPENDIX

APPENDIX

• HOW TO INCREASE ENDOPHIN LEVELS
• MANAGING NEGATIVE FEELING STATES
• ATTACHMENT
• SLEEP
• ASK, ADMIT AND TAKE ACTION SELF TEST

HOW TO INCREASE ENDOPHIN LEVELS

• Dark Chocolate
  • Phenylethylamine works by raising endorphin levels

• Spicy Foods
  • When you eat hot peppers, your brain perceives the heat they cause as pain
  • This causes endorphins to rush in to put out the fire.
  • This endorphin-stimulating quality makes capsaicin a useful treatment for the pain of arthritis, neuropathy, and severe itching.
HOW TO INCREASE ENDORPHIN LEVELS

• Laugh Yourself Happy
  • One way laughter works to reduce pain, boost your immune system, and increase happiness is by boosting levels of endorphins

• Physical Exercise
  • Taking an exercise class or having an exercise partner increases endorphins and tolerance to pain better than exercising alone

• Service Work
  • A study done at the US National Institutes of Health found that helping others activates regions of the brain associated with pleasure, social connection, and trust.
  • This flood of endorphins caused by being generous has been coined “helper's high.”

• Gossip
  • Apparently, gossiping is a primitive need that’s essential for our social and psychological well-being.
  • And for better or for worse, electronic devices, the internet, and social media make it easier than ever to share gossip.

• Fall in Love
  • Phenylethylamine, the same endorphin-boosting compound found in chocolate, is also produced by our brains when we fall in love.
  • It’s not possible to be “in love” all of the time, but having sex will do!
  • You’ll get a burst of endorphins when you have an orgasm — whether you are with a partner or “flying solo.”
HOW TO INCREASE ENDORPHIN LEVELS

• Dance
  • Listening to music, especially music that makes you feel joyful, increases endorphins.
  • Interestingly, one study found that country music was the best for endorphin boosting while heavy metal rock was the worst since it tended to leave listeners anxious.
  • Playing an instrument, singing, and dancing all stimulate endorphin release even more than passive listening.

• Catch Some Rays
  • The best known benefit of sun exposure is essential vitamin D formation, but the sun's ultraviolet radiation also boosts endorphin production.

• Make Time to Play
  • Spend time with a child or pet. They haven't forgotten how to play and can be the best teachers.

• Meditation
  • A regular meditation practice reduces the stress hormone cortisol, while increasing endorphins to quell chronic inflammation, an underlying cause of many diseases and mental disorders, including possibly depression.

• Massage
  • The healing touch of massage can reduce stress hormone levels while increasing levels of endorphins and serotonin.

• Acupuncture
  • It's now known that acupuncture causes the brain to release endorphins.

• Breathing
  • When you breathe at the rate of six breaths per minute your heart and breath become synchronized.
  • This sends a signal to the brain to release the feel-good neurotransmitters endorphins, serotonin, and dopamine.
MANAGING NEGATIVE FEELING STATES

• TWO MINUTES OF SILENCE
  • The auditory cortex has a separate network of neurons that fires when silence begins
  • Two hours of silence per day prompted cell development in the hippocampus
  • Silence helps newly generated cells to differentiate into neurons and integrate into the system
  • Creates a state of “environmental enrichment”
  • Two minutes of silence allows the “default mode”-situated in the prefrontal cortex- to activate

• TWO MINUTES OF SILENCE (continued)
  • The “default mode” gathers and evaluates information. Focused attention curtails this scanning activity
  • It is observed most closely during the psychological task of reflecting on one’s personality and characteristics (self-reflection)
  • It integrates external and internal information (Joseph Moran, Frontiers in Human Neuroscience, 2013)

Gross, DA. “This is Your Brain on Silence.” Brain in the News. September 2016, pgs. 5-6.

MANAGING NEGATIVE FEELING STATES

• DANCE
  • By yourself put on music that makes you feel like moving
  • Let your body lead
  • When you start to tire gradually slow down
  • Take two minutes in silence to appreciate the changes that have occurred in your brain's emotional system

• BREATHING FROM YOUR HEART (HEARTMATH)
  • Focus on the area of your chest that houses your heart
  • Breath in and out from the heart

• BREATHING FROM YOUR HEART (HEARTMATH) (continued)
  • The breathes should be a little deeper than usual
    • 5-6 seconds on the in-breath and 5-6 seconds on the out-breath
  • Appreciate the differences in your feeling state

• APPRECIATION BREATHE
  • 2-3 times per day
  • Bring into your mind something or someone you appreciate
MANAGING NEGATIVE FEELING STATES

• **APPRECIATION BREATHE** (continued)
  • Might consider using a nice note or email from a friend that you carry with you
  • Could be a prayer or Bible verse
  • Discern the changes you experience as you read or recall the positive experience

• **BE OF SERVICE**
  • “I’VE LEARNED THAT PEOPLE WILL FORGET WHAT YOU SAID, PEOPLE WILL FORGET WHAT YOU DID, BUT PEOPLE WILL NOT FORGET HOW YOU MAKE THEM FEEL.” ~ Maya Angelou

MANAGING NEGATIVE FEELING STATES

• **LETTING GO**
  • Letting go is a mechanism of the mind and causes a sense of relief and lightness
    • Example: I was with a friend and we were talking about all of the problems we had to deal with on a work project. We both broke out in laughter. The problems still existed but they were no longer our problems (i.e. some deficit in us)
  • Technique: Letting go consciously and frequently at will
    • NO LONGER THE VICTIM

MANAGING NEGATIVE FEELING STATES

• **BE OF SERVICE** (continued)
  • Focus on your environment
  • Remember everything is God
  • Kindly act to be of service to people, places and things
  • Notice how your feelings change as you direct love outward
  • The more you give of yourself the more we are filled with Love

MANAGING NEGATIVE FEELING STATES

• **WELCOMING PRAYER**
  • Become aware of a feeling (sensation) without labeling the sensation, venting, resisting, moralizing and judging
  • Ignore all thoughts as they are just excuses and get us nowhere
  • Let the sensation in and just stay with it
  • Let it run its course without trying to make it different; just let the energy run out
MANAGING NEGATIVE FEELING STATES

• WELCOMING PRAYER (continued)
  • If stay with anger, hatred, resentments and self-pity…
  • All have secondary gains (THE VICTIM)
  • It is our ego-mean, competitive, cheap, mistrusting, vindictive, judgmental, guilty, ashamed, vain (little energy) and resentful

MANAGING NEGATIVE FEELING STATES

• OTHER APPROACHES
  • Grounding
  • Taking a walk in nature
  • Playing with dogs, cats and small children
  • Taking a shower
  • Writing in a journal
  • Listening to certain music such as classical, improvisational jazz, Tibetan Incantations, Gregorian Chant, etc.
  • Physical exercise

SLEEP

• Consider using the program called Conquering Insomnia which can be found at CBTforINSOMNIA.com or teach basic sleep hygiene

• Evidence-based program developed by Dr. Greg Jacob at Harvard Medical School and funded by a NIH grant

• In a study conducted at Harvard was found to be more effective than Ambien

SLEEP

• INSOMNIA
  • 5 session interactive program
    • SESSION 1: BASIC FACTS ABOUT SLEEP
    • SESSION 2: SLEEP SCHEDULING AND STIMULUS CONTROL
    • SESSION 3: COGNITIVE RESTRUCTURING AND SLEEP MEDICATION TAPERING TECHNIQUES
    • SESSION 4: DAYTIME RELAXATION TECHNIQUES
    • SESSION 5: BEDTIME RELAXATION TECHNIQUES
SLEEP

• The journal SLEEP demonstrated online CBT program for insomnia effective for improving sleep in 80% of patients
• The interactive version in a study by NIH showed it was comparable to the results garnered from face-to-face CBT
• Wake time after sleep onset was reduced from over an hour to less than 30 minutes per night
• Sleep onset latency decreased from over 30 minutes to less that 20 minutes per night
• Total sleep time increased about an hour

SLEEP HYGIENE

• Go to bed and get up at the same times each day.
  ■ Use natural light (that comes through a window) to remind yourself of when it’s time to be asleep and awake. This can help you set a healthy sleep–wake cycle.
  ■ Exercise regularly.
  ■ If you take naps, keep them short and before 5 p.m.
  ■ Don’t eat or drink too much when it is close to bedtime.

SLEEP HYGIENE

• Avoid caffeine (in coffee, tea, chocolate, cola, and some pain relievers) and nicotine for several hours before bedtime.
  ■ Wind down before going to bed (e.g., take a warm bath, do light reading, practice relaxation exercises).
  ■ Keep the bedroom a relaxing place—avoid working or paying bills in bed.
  ■ Sleep in a dark, quiet room that isn’t too hot or too cold.
  ■ Don’t lie in bed awake. If you can’t fall asleep within 20 minutes get up

SLEEP

• PHARMACOTHERAPY
  • Melatonin—a metabolite of serotonin is a hormone secreted by the pineal gland; plays a role in maintenance of sleep-wake cycle (suprachiasmatic nucleus)
  • Valerian (could damage the liver)
  • Tryptophan—precursor amino acid to serotonin
  • Antidepressants—Trazodone is a popular choice although not backed by formal clinical studies
  • Quetiapine (Seroquel) and gabapentin (mixed results)
SLEEP

- **Transcranial Electrical Stimulators**
  - Stimulation of the brain to produce serotonin and melatonin while reducing cortisol (the stress hormone) and calming the brain's Default Mode Network. The device is effective in treating the following types of insomnia:
    - Chronic Insomnia
    - Onset Insomnia
    - Comorbid Insomnia
    - Maintenance Insomnia: Difficulty staying asleep through the night (waking up often or waking up too early).

ATTACHMENT-EARLY ATTACHMENT PATTERNS

- Young children need to develop a relationship with at least one primary caregiver in order for their social and emotional development to occur normally.
- During the first two years, how the parents or caregivers respond to their infants establishes the types of patterns of attachment their children form. These patterns will go on to guide the child's feelings, thoughts and expectations as an adult in future relationships.

ATTACHMENT-EARLY ATTACHMENT PATTERNS

- **SECURE ATTACHMENT**
- **AVOIDANT ATTACHMENT**
- **AMBIVALENT/ANXIOUS ATTACHMENT**
- **DISORGANIZED ATTACHMENT**

SECURE ATTACHMENT

- Ideally, from the time infants are six months to two years of age, they form an emotional attachment to an adult who is attuned to them, that is, who is sensitive and responsive in their interactions with them. It is vital that this attachment figure remain a consistent caregiver throughout this period in a child's life. During the second year, children begin to use the adult as a secure base from which to explore the world and become more independent. A child in this type of relationship is securely attached.
AVOIDANT ATTACHMENT

• There are adults who are emotionally unavailable and, as a result, they are insensitive to and unaware of the needs of their children. They have little or no response when a child is hurting or distressed. These parents discourage crying and encourage independence. Often their children quickly develop into “little adults” who take care of themselves. These children pull away from needing anything from anyone else and are self-contained. They have formed an *avoidant* attachment with a misattuned parent.

<table>
<thead>
<tr>
<th>Characteristics of Avoidant Attachment</th>
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</thead>
<tbody>
<tr>
<td><strong>As Children:</strong></td>
</tr>
<tr>
<td>1. May avoid parents.</td>
</tr>
<tr>
<td>2. Does not seek much comfort or contact from parents.</td>
</tr>
<tr>
<td>3. Shows little or no preference between parent and stranger.</td>
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<tr>
<td><strong>As Adults:</strong></td>
</tr>
<tr>
<td>1. May have problems with intimacy.</td>
</tr>
<tr>
<td>2. Invest little emotion in social and romantic relationships.</td>
</tr>
<tr>
<td>3. Unable or unwilling to share thoughts and feelings with others.</td>
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AVOIDANT ATTACHMENT

• Do not feel the need for love and reassurance
• This lowers the need for intimacy
• Reluctant to trust and rely on another
• *Fear intimacy*

AMBIVALENT/ANXIOUS ATTACHMENT

• Some adults are inconsistently attuned to their children. At times their responses are appropriate and nurturing but at other times they are intrusive and insensitive. Children with this kind of parenting are confused and insecure, not knowing what type of treatment to expect. They often feel suspicious and distrustful of their parent but at the same time they act clingy and desperate. These children have an *ambivalent/anxious attachment* with their unpredictable parent.
AMBIVALENT/ANXIOUS ATTACHMENT

• Need for love and reassurance but fear rejection
• Project their own flirtatiousness and sexual interest onto another based upon hope he/she will reciprocate

DISORGANIZED ATTACHMENT

• When a parent or caregiver is abusive to a child, the child experiences the physical and emotional cruelty and frightening behavior as being life-threatening. This child is caught in a terrible dilemma: her survival instincts are telling her to flee to safety but safety is the very person who is terrifying her. The attachment figure is the source of the child’s distress. In these situations, children typically disassociate from their selves. They detach from what is happening to them and what they are experiencing is blocked from their consciousness. Children in this conflicted state have disorganized attachments with their fearsome parental figures.

ADULT ATTACHMENT STYLES

• SECURE PERSONALITY
• DISMISSIVE PERSONALITY
• PREOCCUPIED PERSONALITY
• FEAR-AVOIDANT PERSONALITY

<table>
<thead>
<tr>
<th>Positive</th>
<th>Thoughts of Self</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>Secure</td>
<td>Preoccupied</td>
</tr>
<tr>
<td></td>
<td>Comfortable with intimacy and autonomy</td>
<td>Preoccupied with relationships</td>
</tr>
<tr>
<td>Negative</td>
<td>Dissociative</td>
<td>Fearful</td>
</tr>
<tr>
<td></td>
<td>Demanding of intimacy</td>
<td>Fearful of intimacy</td>
</tr>
<tr>
<td></td>
<td>Struggling with independence</td>
<td>Socially isolated</td>
</tr>
</tbody>
</table>
**SKILL SET:**
**TWELVE STEPS OF AA: CYCLE ONE**

- **1st Step:** We admitted we were powerless over alcohol—that our lives had become unmanageable. (ADMIT)
- **2nd Step:** Came to believe that a Power greater than ourselves could restore us to sanity. (ASK)
- **3rd Step:** Made a decision to turn our will and our lives over to the care of God as we understood Him. (ACT)
- **4th Step:** Made a searching and fearless moral inventory of ourselves. (ACT)

**SKILL SET:**
**TWELVE STEPS OF AA: CYCLE ONE**

- **5th Step:** Admitted to God, to ourselves, and to another human being the exact nature of our wrongs. (ADMIT)
- **6th Step:** Were entirely ready to have God remove all these defects of character. (ASK)
- **7th Step:** Humbly asked Him to remove our shortcomings. (ASK)
- **8th Step:** Made a list of all persons we had harmed, and became willing to make amends to them all. (ACT)
- **9th Step:** Made direct amends to such people wherever possible, except when to do so would injure them or others. (ACT)
SKILL SET: TWELVE STEPS OF AA

CYCLE THREE

• 10th Step - Continued to take personal inventory and when we were wrong promptly admitted it. (ADMIT)

• 11th Step - Sought through prayer and meditation to improve our conscious contact with God as we understood Him, praying only for knowledge of His will for us and the power to carry that out. (ASK)

• 12th Step - Having had a spiritual awakening as the result of these steps, we tried to carry this message to alcoholics, and to practice these principles in all of our affairs. (ACT)

SKILL SET: TWELVE STEPS OF AA

FIRST: Admit, getting in touch with feelings and attitudes and learn how to acknowledge them

SECOND: Learn how to ask for assistance when needed; how to trust more

THIRD: Learn how to take prompt action

SKILL SET: TWELVE STEPS OF AA

WHEN IN PAIN...

• There are those who simply will not admit it

• There are those who will admit something is wrong but will not ask for help

• There are those that can admit and ask but refuse to act on corrective measures

SKILL SET: TWELVE STEPS OF AA: PROFILE

Use the following scale to express the extent of your agreement or disagreement with the statement:

• 5=This statement applies to me all the time

• 4=This statement applies to me much of the time

• 3=This statement sometimes applies to me

• 2=This statement rarely applies to me

• 1=This statement never applies to me

TWELVE STEPS OF AA: PROFILE

Put your response (one number from the scale) beside each statement...

1. I am a procrastinator; I put things off. (___)
2. I admit when I am wrong. (___)
3. I prefer to do things without help. (___)
4. When I know what needs to be done, I do it. (___)
5. I am open to suggestions. (___)
6. When I am not happy, I don't talk about it. (___)
7. It is hard for me to ask for things. (___)
8. Friends say I am a “doer.” (___)

9. I do not like keeping secrets. (___)
10. I am more of a thinker than a doer. (___)
11. It takes a lot of evidence before I see something about myself I hadn't seen before. (___)
12. I am willing to do things differently than I usually do them. (___)
13. I am enjoying life. (___)

TWELVE STEPS OF AA: SCORING THE PROFILE

A. Add the numbers recorded for items 2 and 9. (___)
B. Add the numbers recorded for items 6 and 11. (___)
C. Subtract B from A (may be a negative number). (___)
D. Add the numbers recorded for items 5 and 12. (___)
E. Add the numbers recorded for items 3 and 7. (___)
F. Subtract E from D (may be a negative number). (___)
G. Add the numbers recorded for items 4 and 8. (___)
H. Add the numbers recorded for items 1 and 10. (___)
I. Subtract H for G (may be a negative number). (___)

SKILL SET:

TWELVE STEPS OF AA: PROFILE

Put your response (one number from the scale) beside each statement...

The admit index is C: (___)
The ask index is F: (___)
The act index is I: (___)
The largest number (C, F or I) is the persons strongest element. (_______)
The smallest number (C, F or I) is the persons weakest element. (_______)

An index lower than -3 indicates an area for great concern.
REFERENCES