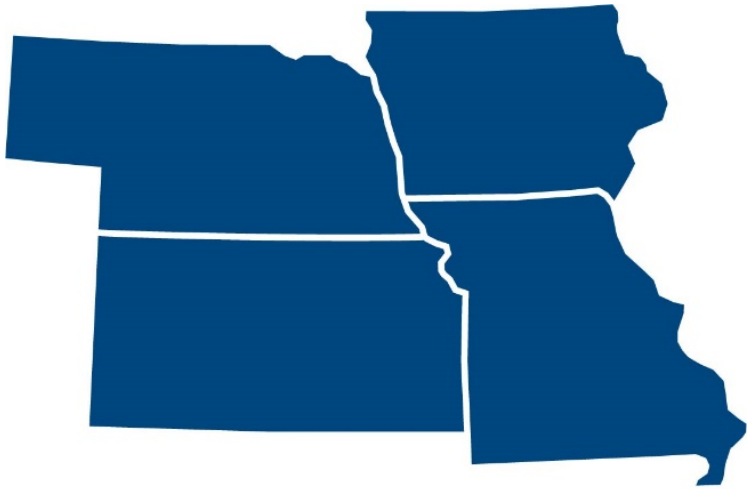
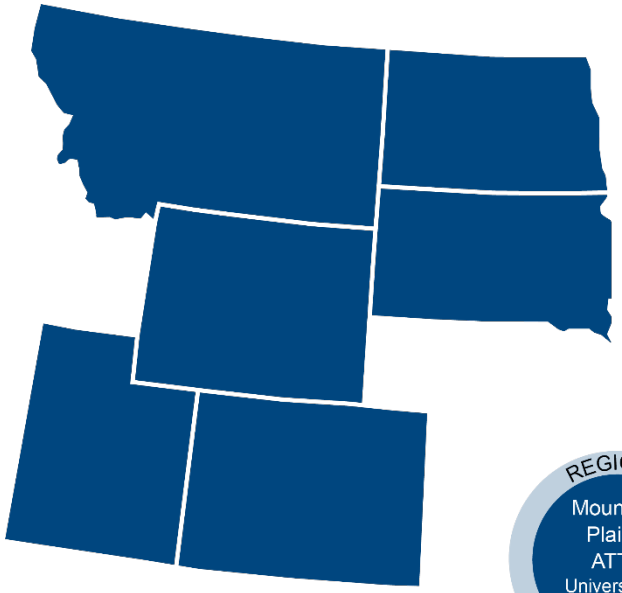
The Complex Problem of Substance Use Disorder and Traumatic Brain Injury

Presented by
Frank R. Sparadeo, Ph.D.
April 28, 2021
Clinical Neuropsychologist
Consultant to the Massachusetts Rehabilitation Commission

Mid-America ATTC & Mountain Plains ATTC



REGION 7
Mid-America
ATTC
Truman
Medical Center

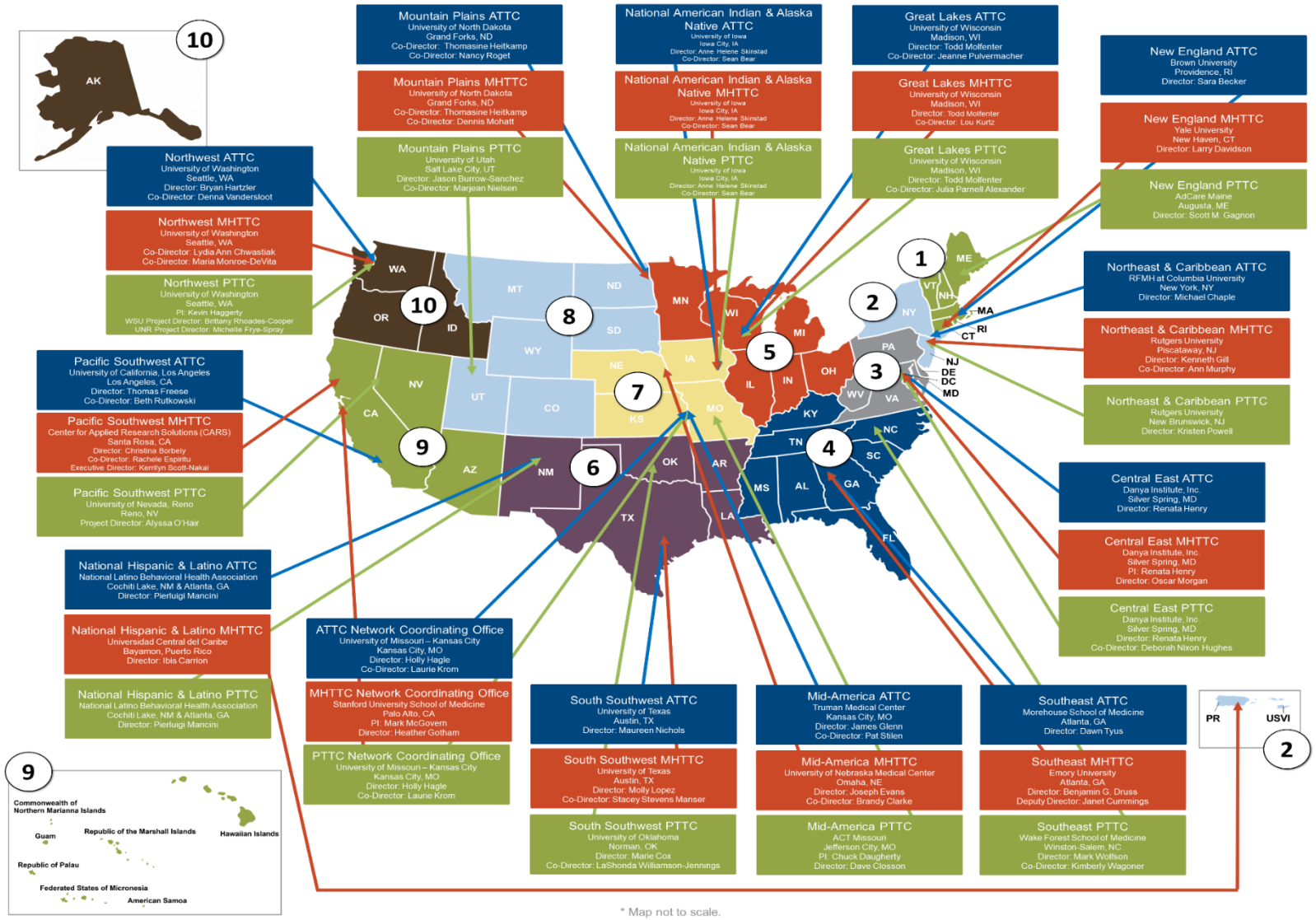


REGION 8
Mountain
Plains
ATTC
University of
North Dakota

Mid-America (HHS Region 7)
ATTC
Addiction Technology Transfer Center Network
Funded by Substance Abuse and Mental Health Services Administration

Mountain Plains (HHS Region 8)
ATTC
Addiction Technology Transfer Center Network
Funded by Substance Abuse and Mental Health Services Administration

Technology Transfer Center Network



Disclaimer and Funding Statement

At the time of this presentation, Tom Coderre served as SAMHSA Acting Assistant Secretary. The opinions expressed herein do not reflect the official position of the Department of Health and Human Services (DHHS), or SAMHSA. No official support or endorsement of DHHS, SAMHSA, for the opinions described in this presentation is intended or should be inferred.

The work of the Mid-America ATTC is supported by grant 1H79TI080208-01 the Mountain Plains ATTC is supported by grant TI080200_01. Funded by the Department of Health and Human Services, Substance Abuse and Mental Health Services Administration.

Disclosures

Successful Completion

This live webinar offers 1.5 contact hours. To receive contact hours, participants must complete the activity in its entirety and complete the Evaluation/Request for Credit Form. CHES and NAADAC certificates as well as Certificates of Completion will be emailed within four to six weeks after submission of the Evaluation/Request for Credit form.

Commercial Support/Sponsorship

There is no commercial support for this training.

Non-Endorsement of Products

The University of Missouri-Kansas City School of Nursing and Health Studies, Mid-America Addiction Technology Transfer Center, NCHEC and NAADAC do not approve or endorse any commercial products associated with this activity.

Accreditation Statements

NAADAC

This course has been submitted to NAADAC for approval by the Addiction Technology Transfer Center (ATTC) Network Coordinating Office, as a NAADAC Approved Education Provider, for # 1.5 CE(s). NAADAC Provider #64973, Addiction Technology Transfer Center (ATTC) Network Coordinating Office, is responsible for all aspects of its programing."

Certified and Master Certified Heath Education Specialists (CHES & MCHES)

Sponsored by the University of Missouri-Kansas City School of Nursing and Health Studies, a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. This webinar is designated for Certified Health Education Specialists (CHES) to receive up to 1.5 total Category I continuing education contact hours.

Other CEs

- Iowa Board of Certification
- Missouri Credentialing Board
- Kansas Behavioral Sciences Regulatory Board
- Nebraska (deemed alcohol and drug specific – accepted for continuing education for licenses alcohol and drug counselors in NE)
- NASW
- CRC

Housekeeping Items

- All attendees are muted and attendees cannot share video during this session.
- Remember to ask questions using the Q&A feature
- How to access training materials

Objectives

At the end of this activity participants will be able to…

1. Describe the scope of the SUD and TBI problems in the US

2. Acquire general understanding of neurobiological systems relevant to working with people experiencing TBI and SUD

3. Identify assessment procedures, treatment planning components, and typical treatment modifications needed in treating TBI and SUD

Dr. Frank R. Sparadeo

- Practiced as a neuropsychologist for over 34 years
- Experience evaluating and treating people with people experiencing brain disorders or chronic pain, and/or addiction.
- Most recently involved in the formation of a special program in the combined problem of chronic pain and addiction
- Experience in switching patients from pain meds to suboxone
- Closely involved in the treatment of chronic pain utilizing a new theoretical approach that relies on information theory to reduce pain responses. The neuromatrix theory of pain is the basis of this treatment.

Scope of the Problem in the US

TBI is among the most common of serious, disabling neurological disorders.

It is a significant problem in all societies

In the U.S. at least 1.4 million TBIs occur every year, and there are 5.3 million people living with disability from TBI.

TBI is largely a younger and older person's disorder.

Individuals younger than 30, mostly males, make up the largest proportion of those affected.

People older than 60 also make up a sizeable proportion of the TBI population.

Scope of the Problem Re. TBI #1

Older persons present particular problems related to aging including co-morbidities, slower and less complete recovery and vulnerability to complications of injury and treatment.

TBI commonly affects people with preexisting problems such as substance use disorder, psychiatric disorders, learning disabilities, ADHD and behavioral disorders

The most important and consistent effects of TBI involve cognitive, emotional and behavioral functioning.

Motor and sensory perceptual problems also occur in varying amounts, more likely in those with more severe injuries.

Scope of the Problem Re. TBI #2

TBI is a disorder with a wide variety of pathophysiological effects, a range of severities and a multitude of problems that may occur as the result of injury.

Persons with TBI, particularly less severe injuries, may not have any obvious physical markers of the injury, though there may be profound effects of the individual's ability to function, largely resulting from cognitive or behavioral dysfunction.

TBI especially more severe injuries, can have a relatively extended natural history and lifelong effects.

Scope of the Problem Re. TBI #3

TBI is a disorder with a wide variety of pathophysiological effects, a range of severities and a multitude of problems that may occur as the result of injury.

Persons with apparently similar injuries may have significant variation in their presentation, course of recovery, response to interventions and ability to return to function.

Scope of the Problem Re. SUD #1

Mental illnesses, nervous system diseases, brain tumors and head trauma are the first things that come to mind when most people consider pathological conditions of the human brain; but in reality substance use disorder (SUD) and addiction are more prevalent than other brain diseases and have a much greater impact on the fabric of society.

Among people age 19-54, the one-year prevalence rate of:

- Anxiety Disorder is about 18.1%
- Mood disorders is about 9.5%
- Schizophrenia is about 1.1%
- Any mental disorder is about 26.2%

Scope of the Problem Re. SUD #2

This compares with:

- Illicit-drug use by 8.7% of the US population age 12+ in the past month
- Underage alcohol use (12-20) ranged from a low of 17.5% in Utah to a high of 40% in North Dakota
- Nicotine addiction in 29.1% of the population over 12
- Gambling addiction that affects 2% to 6% of adults.

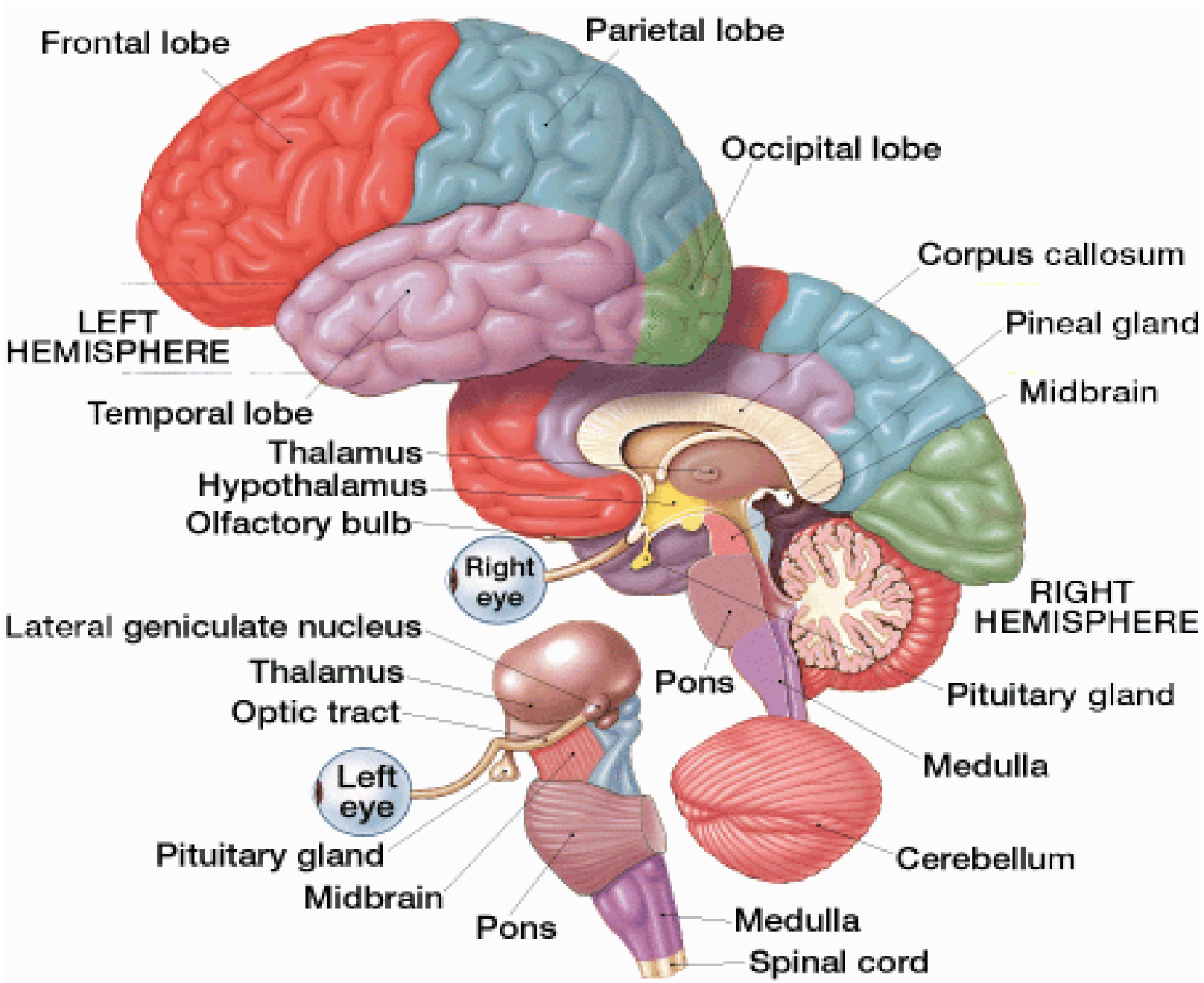
Scope of the Problem Re. SUD #3

- SUD may also be the number one continuing public health problem in the US.
- More than 443,000 Americans die prematurely every year due to nicotine addiction and another 53,000 die from secondhand smoke.
- Another 80,000 die prematurely from SUD, abuse, overdose or associated diseases.
- 6,000 to 10,000 die of cocaine, heroine and methamphetamine overdose or dependence
- Each day 1,740 Americans die from SUD or related causes; that is more than one death every minute.

Neuroanatomical Correlates

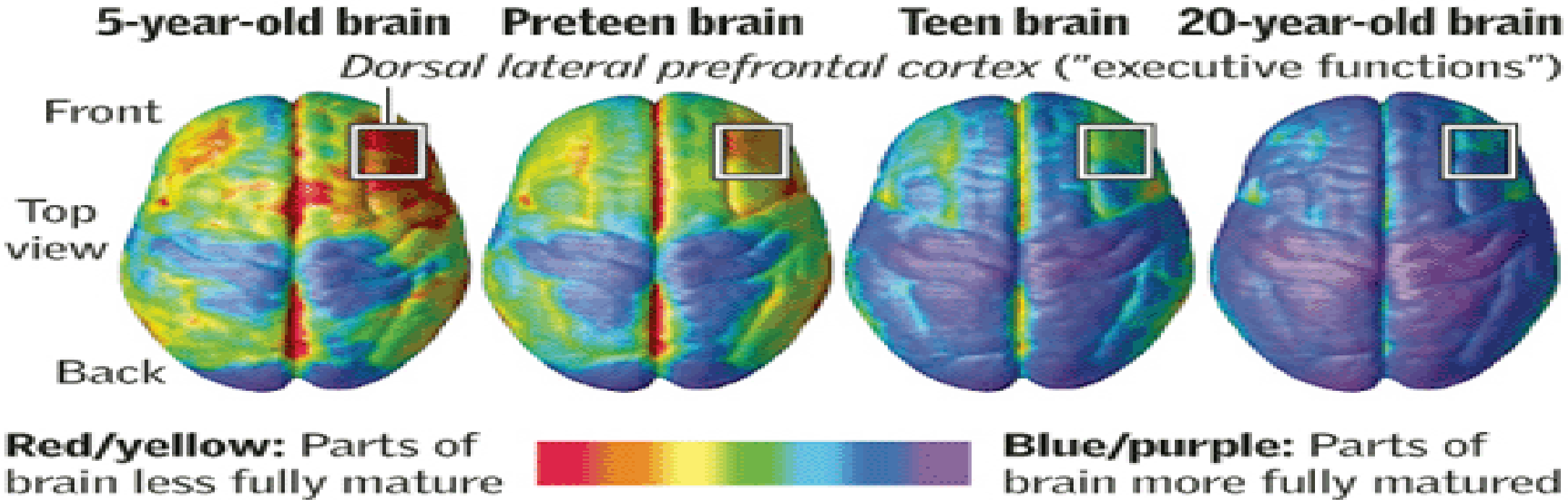
- The history of TBI or other acquired brain disorder (e.g. tumor, CVA, anoxia) can result in either or both focal lesions or diffuse lesioning.
- Key structures pertaining to cognitive functions include: white matter, cortex, basal ganglia, frontal systems, cerebellum, diencephalon
- In TBI, injury to the frontal lobes is most common

Brain Organization



Judgement Last to Develop

The area of the brain that controls "executive functions" — including weighing long-term consequences and controlling impulses — is among the last to fully mature. Brain development from childhood to adulthood:



National Institute of Mental Health, Paul Thompson, UCLA Laboratory of Neuro Imaging
Source: The Denver Post [date unknown]

The Teenage Brain #1

The teenage brain is a whirlwind of change

Although parents of teenagers sometimes wonder if aliens have taken over their children's brains, the truth is that their behavior is more likely a result of a sensitive neuroplastic period.

The teenage brain undergoes disorganization and reorganization from the onset of puberty into the early 20's.

The Teenage Brain

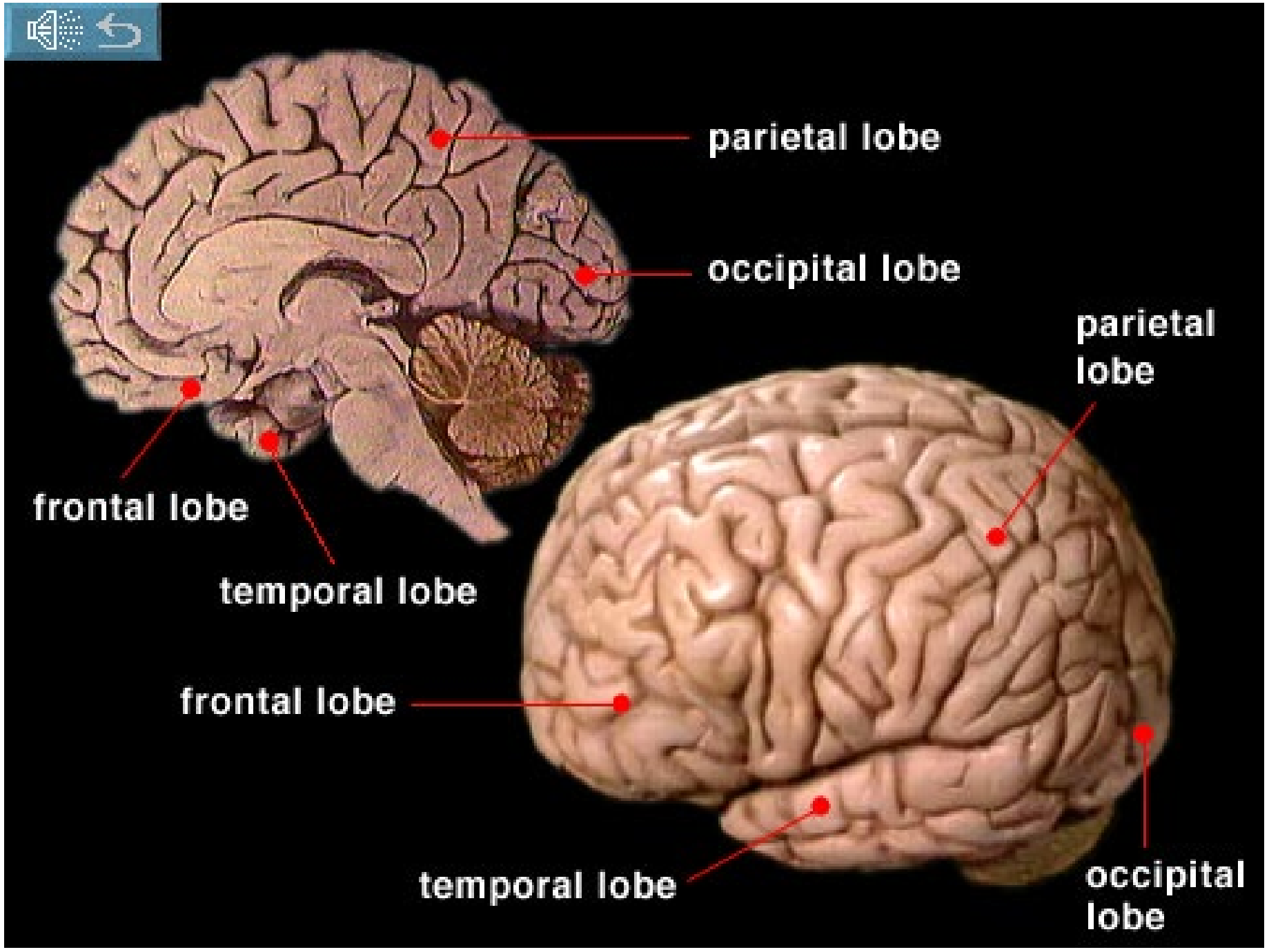
The discovery of reorganization of the adolescent brain supports the notion that natural developmental milestones and life challenges coincide with sensitive periods of neural development and enhanced plasticity.

All in all, neuroplasticity offers a slightly more reasonable explanation than extraterrestrials.

The kinds of changes discovered in the adolescent brain (roughly 12-18 years of age) show a loss of the overall number of neurons (gray matter) with an increase in the number of myelinated fibers (white matter) connecting functional neural networks.

These changes represent a process of selection and reorganization of neural networks with a goal of faster and more efficient information processing.

Cerebral Cortex #1



Cerebral Cortex #2

Occipital Lobe—visual processing

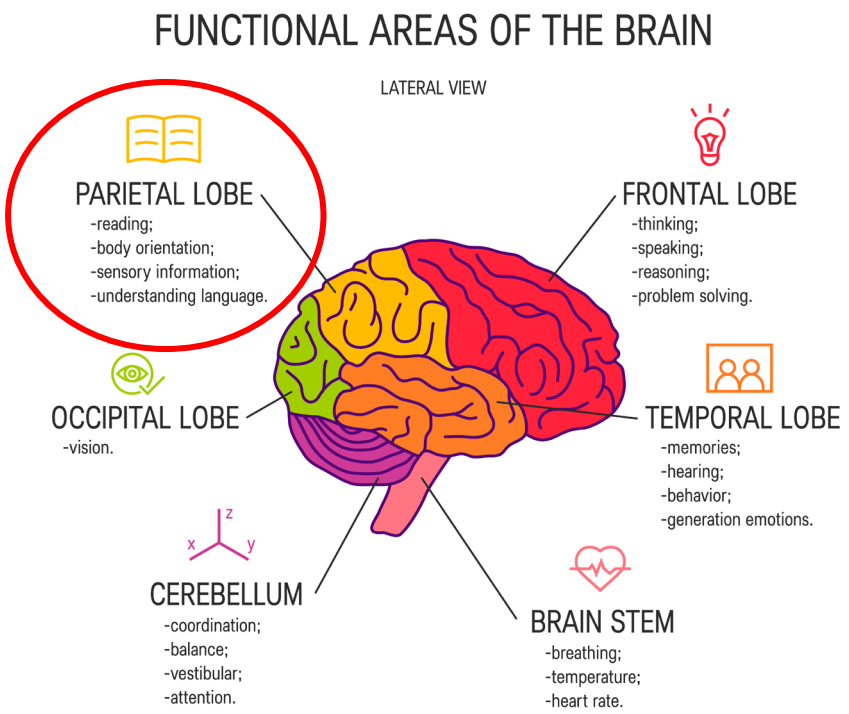
Temporal Lobe—auditory processing, receptive language, and memory functions

Parietal Lobe—Links the senses with motor abilities and creates the experience of a sense of our body in space

Insula and Cingulate—integrate limbic processing and link it to cortical networks

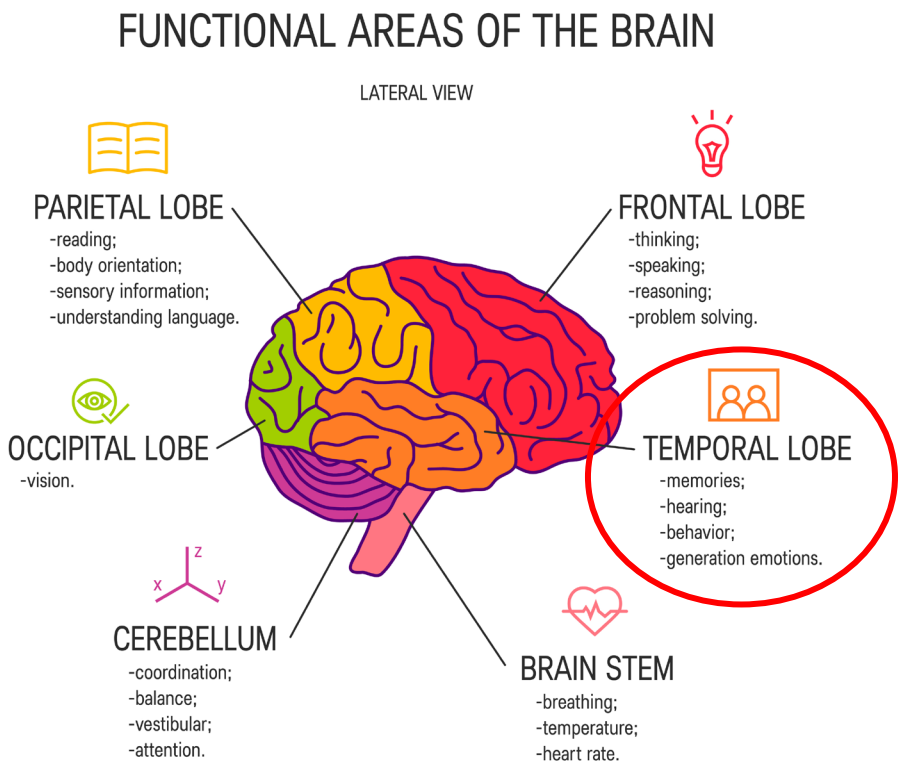
Frontal Lobes—regulate motor behavior, language, executive functioning, abstract reasoning and directed attention

Parietal Lobe



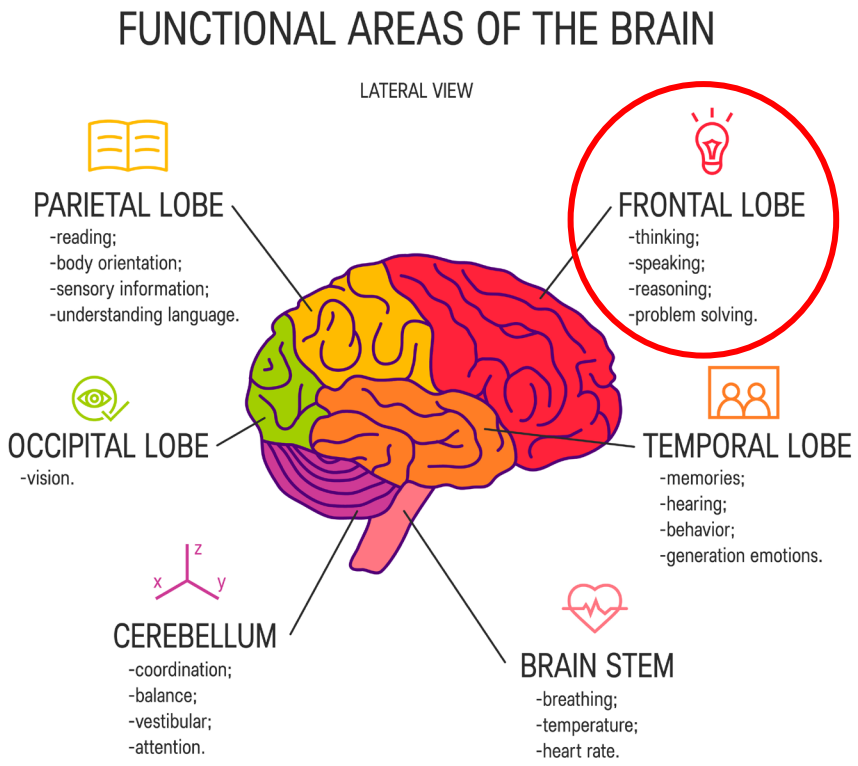
- Somatosensory cortex
- Sensations
    - Perceptions of peripheral sensations
- Spatial orientation
- Spatial awareness of limbs

Temporal Lobe



- Perception of smell, taste & hearing
- Structures affecting memory, mood & personality
- Wernicke's area in dominant hemisphere
- Recognition & understanding of spoken words
- Formulates response & conveys to Broca's area to be executed

Frontal Lobe Functions: Motor Control



Control of skeletal muscles

Voluntary motor activities

- Gross motor control
- Fine motor control

Outputs

- Motor neurons in ventral horn of spinal cord
- Other coordinating regions like the cerebellum & basal ganglia

Frontal Lobe (The Output System)

- Memory
- Personality
- Planning & judgment
- Executive function
- Motor Cortex
- Dominant hemisphere contains Broca’s area
- concerned with language expression

Frontal Lobe Functions #1

- Will
- Volition
- Choice
- Self control / behavioral inhibition
- Some emotional content
  - Mood
  - Empathy

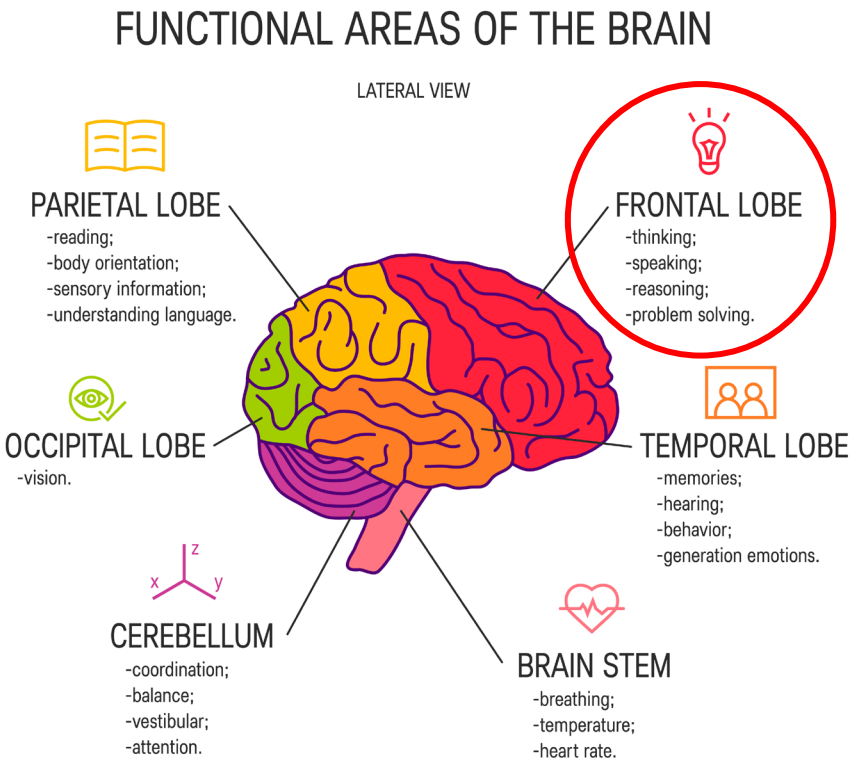
Frontal Lobe Functions #2

- Thinking
- Reasoning
- Problem solving
- Memory
  - Association
  - Long term potentiation (LTP)

Frontal Lobe Damage

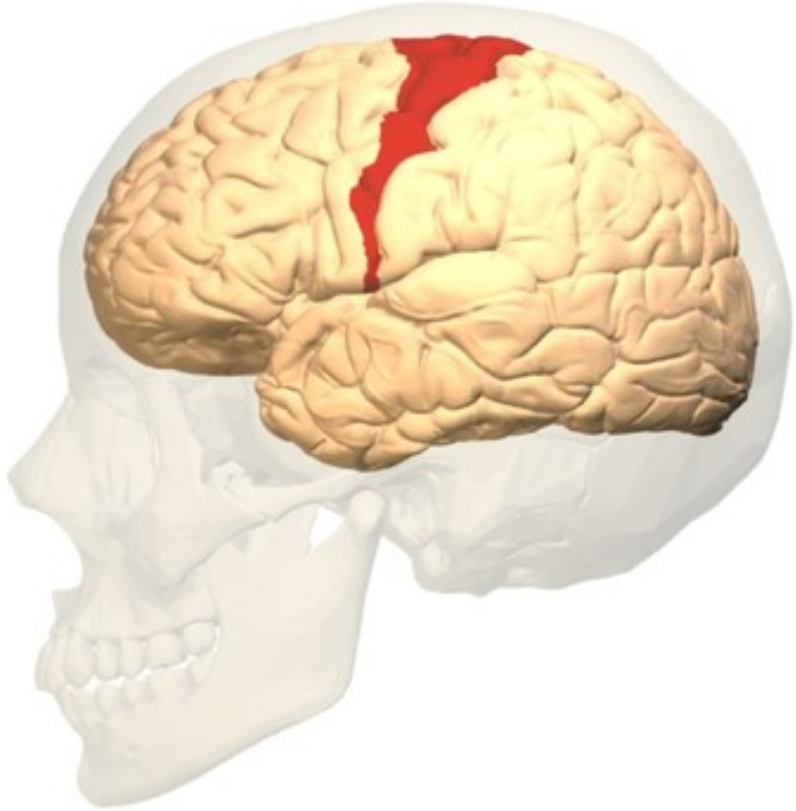
Damage to prefrontal area:

- Future planning
- Initiating action
- Judgment
- Importance of stimuli
- Memory
- Mood disorders (depression & anxiety)
- Attention disorders



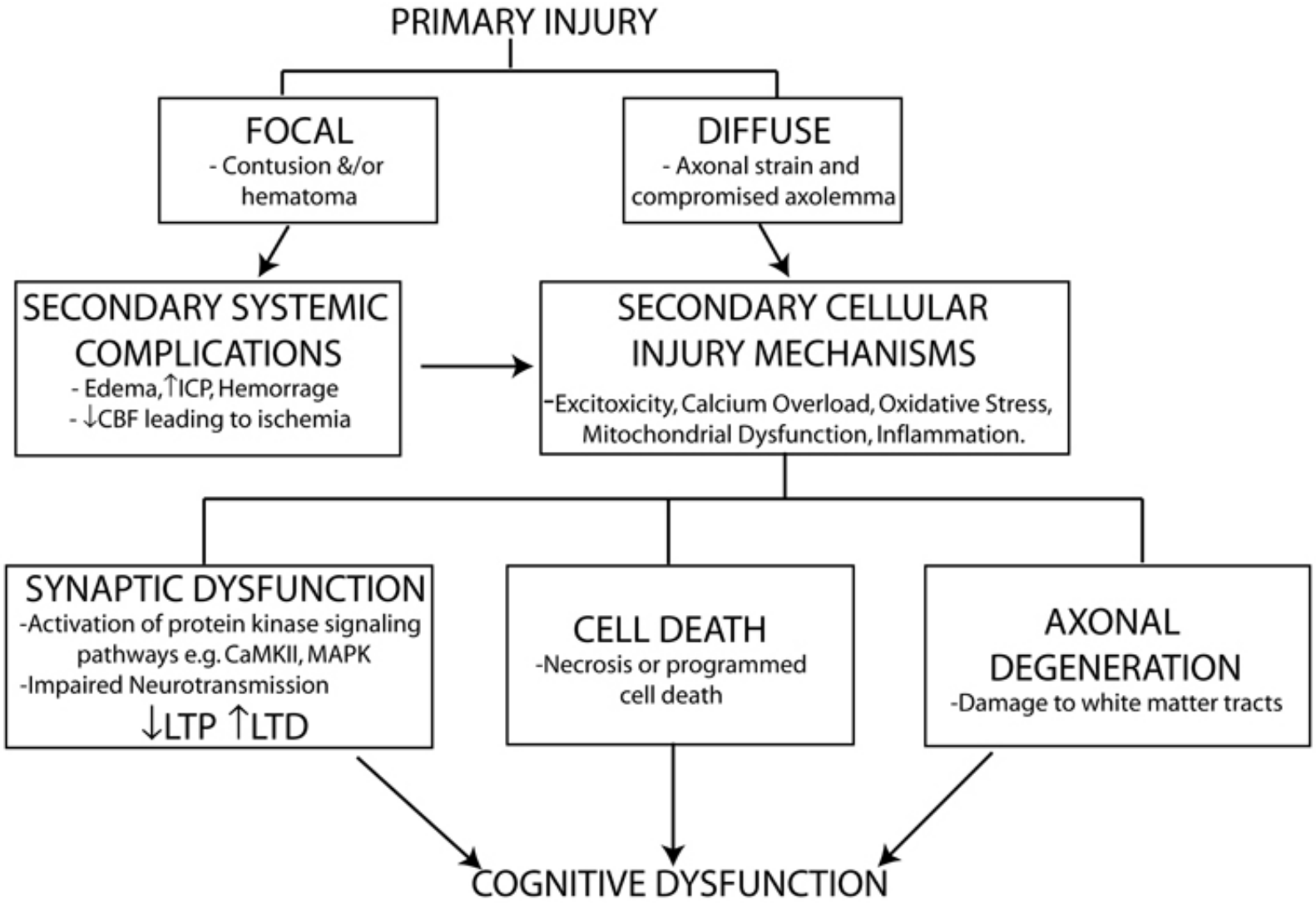
Motor Cortex

- Damage causes loss of muscle control
- Mainly affects opposite side of the body
- Rigid paralysis on opposite side of body
- Depending on the specific area affected, can affect the ability to speak

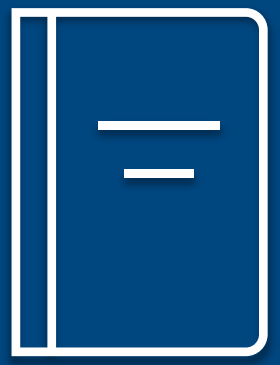


https://musicalbrainwaves.weebly.com/uploads/5/1/1/5/51154897/358404073.jpg?381

Traumatic Brain Injury



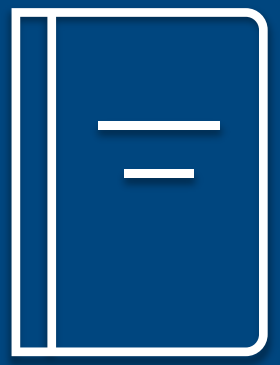
Implications of Drug Use on Brain Function



The use of drugs of abuse have significant implications for the functioning of the brain both at the time of use and also after the use of the substance has remitted.

Each drug of abuse has unique impact on brain structure and chemistry as well as function.

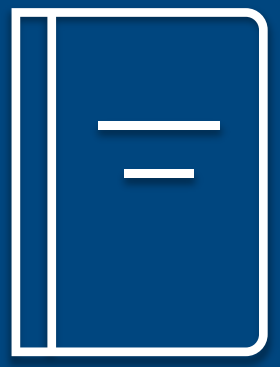
Cognitive Aspects of Sobriety



Staying sober or drug free requires a number of "executive" functions:

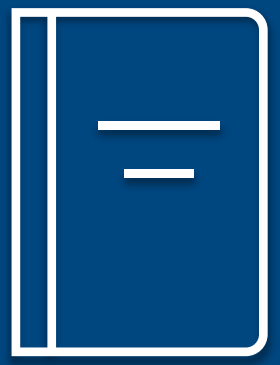
- Self-monitoring/self-guidance
- Use of knowledge to guide behavior
- Impulse Control
- Learning from negative feedback
- Reflection/Empathy

Cognitive Impairments Associated with TBI



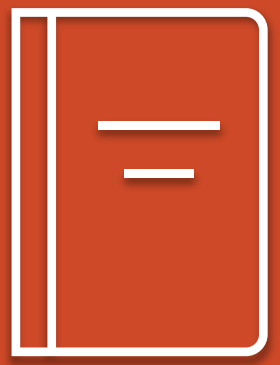
- Information processing and attention impairment
- Unawareness of Deficits (Anosognosia)
- Memory Impairment
- Visuospatial Deficits
- Executive Functioning Deficits
- Intellectual Difficulty

Information Processing and Attention



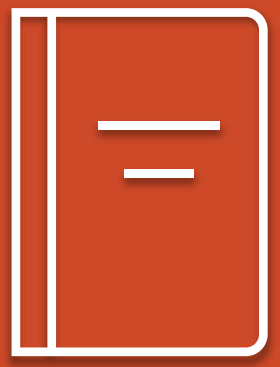
- Information processing measures are designed to determine the speed and accuracy of sensory-perception and perceptual motor responses.
- These processes are mediated by diverse brain structures that include multiple cortical regions, subcortical nuclei and many white matter connections
- Information processing is affected in several ways. One example: similar to ADHD those with mild TBI were impaired on reaction time however were worse than ADHD on reaction time tasks that require making choices

Anosognosia



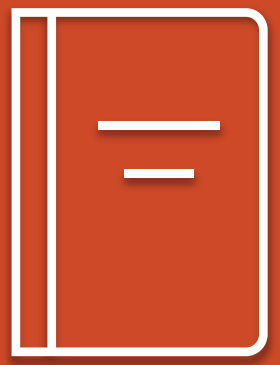
- A common behavioral disorder occurring in people with TBI.
- The symptoms of which the patient is unaware may vary, and include memory and cognitive deficits, hemiparesis, visual and other sensory disturbances, gait disorder and deficits of naturalistic action/limb apraxia
- Patient's inability to recognize functionally-relevant problems is known to be a major barrier to rehabilitation.
- Denial of neurological impairment can take several forms, including lack of emotional concern for acknowledged deficits (anosodiaphoria), verbal denial of deficits that are implicitly acknowledged (person claims he can walk but never attempts to) and a combination of explicit and implicit acknowledgment

Memory



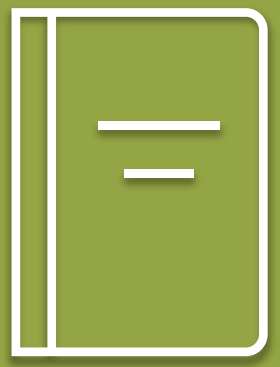
- Subjective complaints of memory impairment after TBI are quite common even among those with mTBI
- Reliable correlation has been found between memory performance and brain volumetric measures in the temporal lobe of adults with mild TBI.
- Specifically, decreased hippocampal and temporal white matter volumes were significantly related to memory impairment
- Functional imaging suggests brain activity patterns of people with TBI become altered during tasks of memory retrieval.

Neurological Implications of Brain Injury



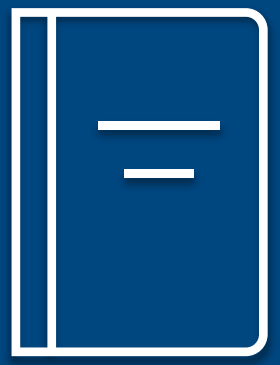
- Post-traumatic seizures
- Impairment of movement
- Balance and Dizziness
- Visual processing difficulty
- Fatigue
- Sleep Difficulty

Substance Use Disorder



- Estimates indicate that 18.9 million adults in the U.S. were diagnosed with substance abuse or dependence in 2011, or approximately 8% of the adult population
- Approximately 23.5 million Americans age 12 and older required intervention for substance use.
- It is projected that disability caused by substance use disorders will surpass that caused by any other physical disease worldwide by 2020.

Neurochemistry and Physiology of Addiction



Addiction appears to represent anomalies of 4 areas of the addiction pathway:

- A survival/reinforcement circuit involving an overactive “go” switch in the “old” brain
- A damaged or underactive “Stop” switch in the control circuit in the “new” brain
- Impaired communication between these two key circuits
- Damaged, stay-stopped brain areas that make recovery extremely difficult for some addicts.

Reward System

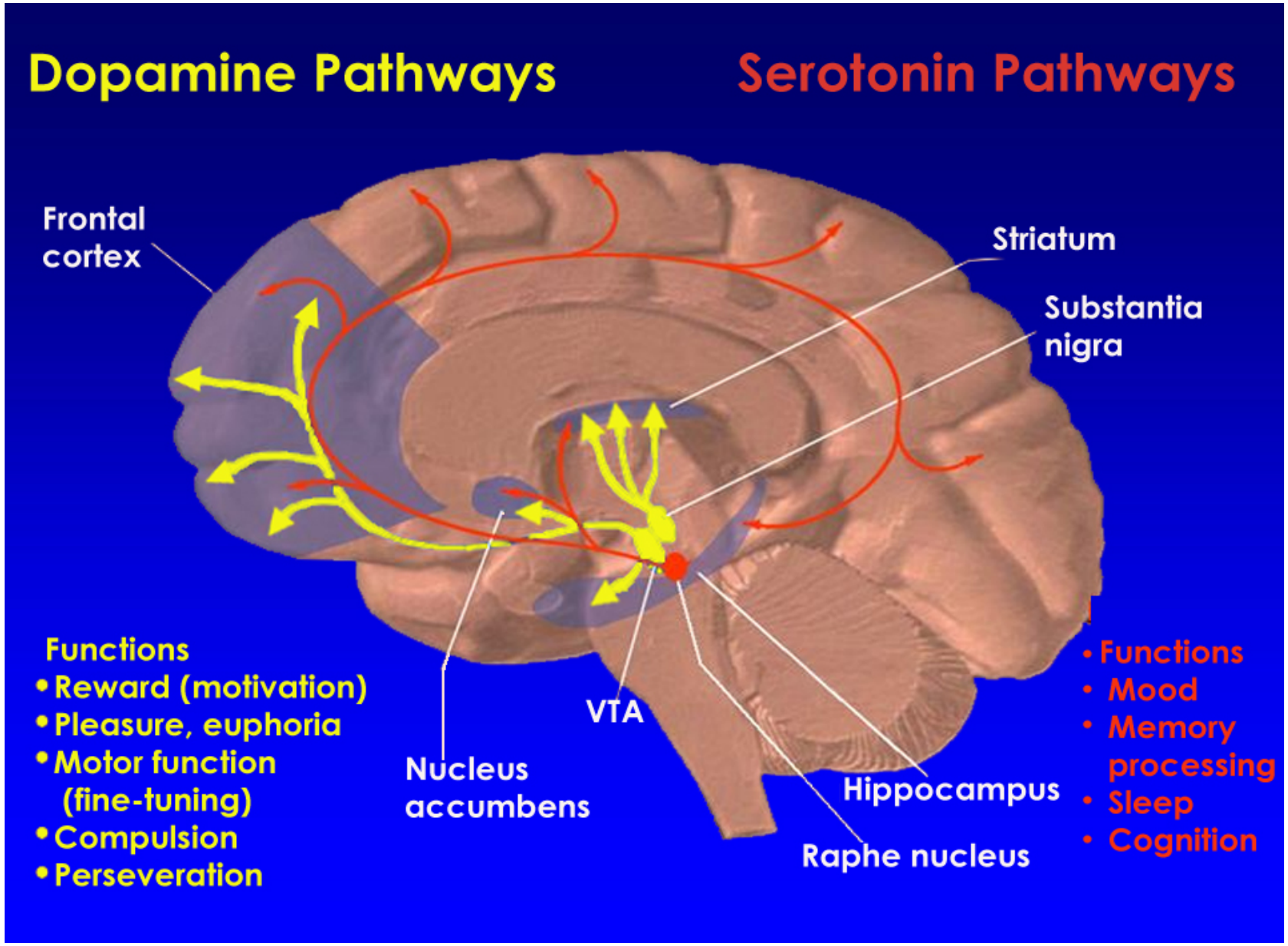
REWARD, **COMFORT, AND PLEASURE** from ordinary activities; and a degree of calming to fight off unwanted stress.

Genetics and environment greatly affect this cascade; and unfortunately, some of our genes come with variations called polymorphisms.

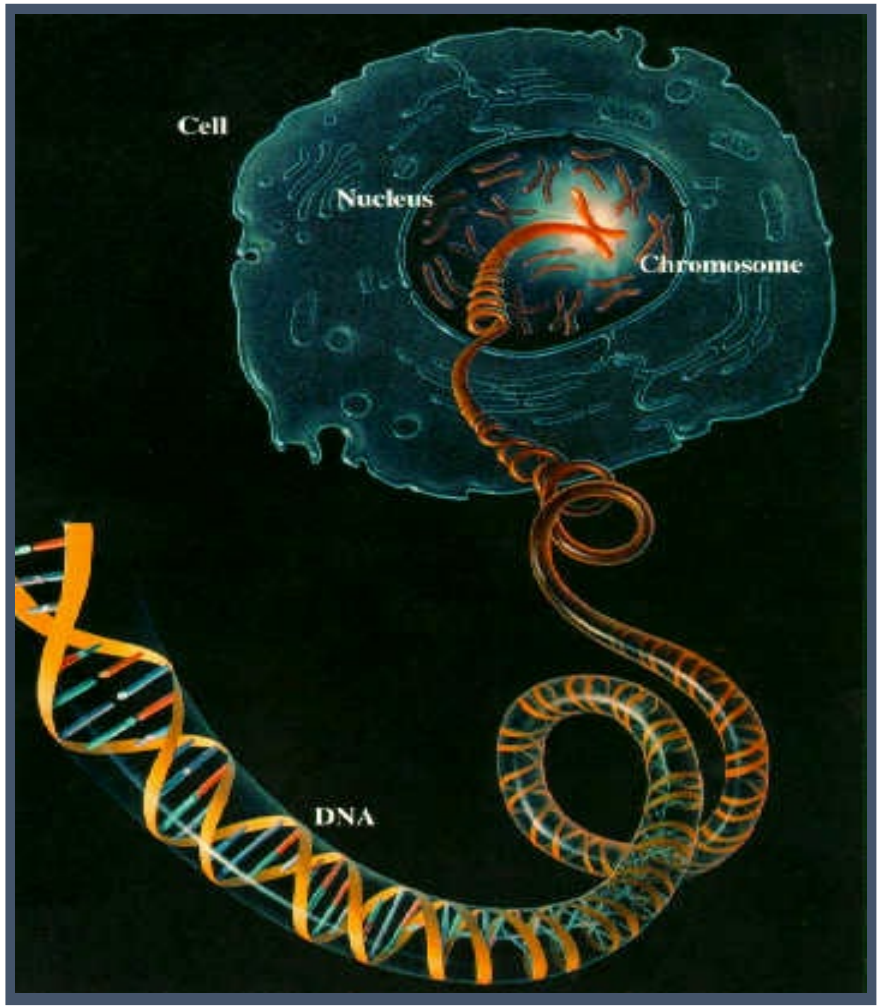
Polymorphisms change the way the gene expresses itself. Most people will call this a predisposition.

These polymorphism's can alter their intended genetic function.

Neurochemistry



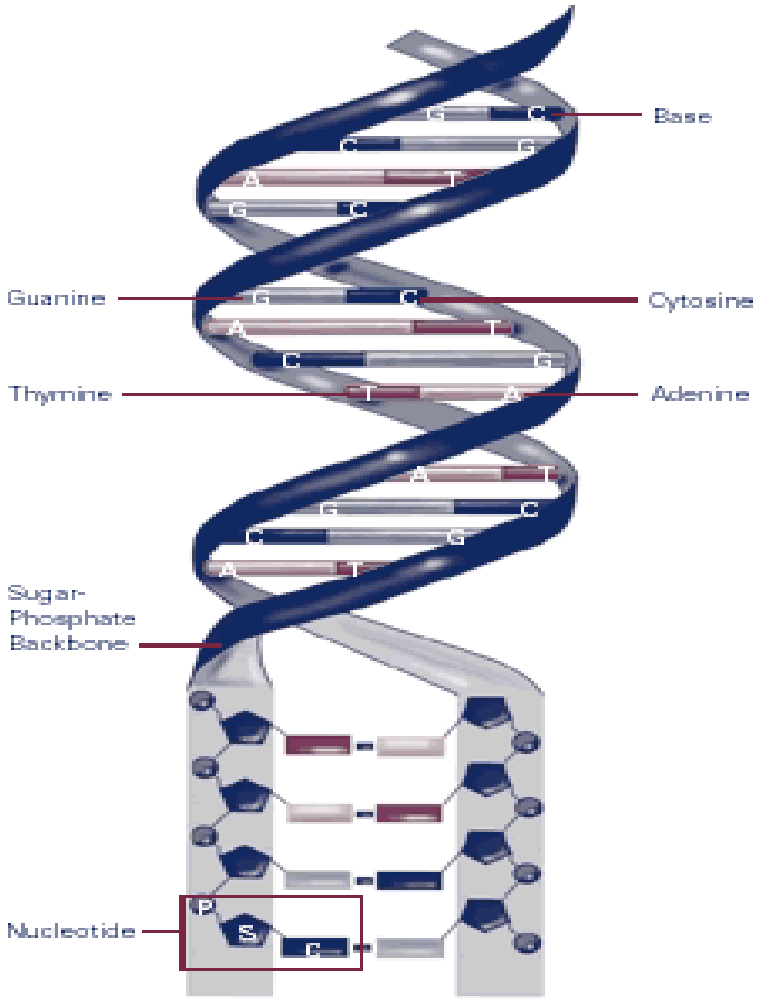
The Human Genome



- **In the human genome, there are ~3 billion bases (nucleotides)**
- **In humans, there are estimated to be ~30,000 genes (many but not all identified and annotated)**
- **Each gene is a sequence of bases or nucleotides**

Kreek (Rockefeller University) & Hassin (Columbia P&S), 2004

Single Nucleotide Polymorphisms (SNPs) in Genes: Definitions



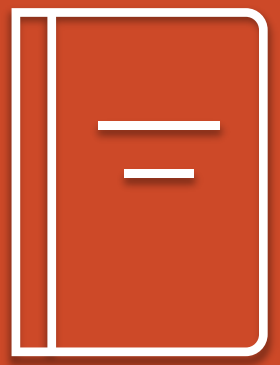
SNP — a single nucleotide polymorphism, that is, one nucleotide or base of any base pair

Allelic Frequency:

<1%	low or rare
1–5%	intermediate
>5%	high, frequent

Kreek (Rockefeller University) & Hassin (Columbia P&S), 2004

All Roads Lead to Dopamine



- Important for the motivation and reinforcement of actions.
- Drugs that interfere with dopamine transmission interfere with reinforcement learning, while manipulations that enhance dopamine transmission, such as brain stimulation and addictive drugs, often act as reinforcers.
- Dopamine transmission is crucial for creating a state of motivation to seek rewards.

“Go” and “Stop” Switches #1

- The area of the brain that encourages a human (or any mammal) to perform or repeat an action that promotes survival is called the survival/reinforcement circuit. Its normal function is to reinforce an action that promotes survival (e.g. eating, drinking, having sex). It is also the part of the brain most affected by psychoactive drugs.

- Technically, this circuit is referred to as the mesolimbic dopaminergic reward pathway which is located in the old brain.

“Go” and “Stop” Switches #2

- This survival/reinforcement circuit, located in the old brain, acts as a “go” or “more” switch. At the heart of the circuit is the Nucleus Accumbens Septi (NAc).
- The Ventral Tegmental Area, lateral hypothalamus and amygdala also play important roles.
- The control circuit, located mostly in the new brain, acts as a “stop” switch and is driven by the left orbital prefrontal cortex. The stop switch works in conjunction with the fasciculus retroflexus and the lateral habenula, which connect and communicate from the “Stop” switch to the “go” switch.

Role of the Pre-Frontal Cortical Regions in Drug Addiction

- Pre-frontal cortical areas work in tandem with striatal regions via corticostriatal networks that are modulated by DA

- These include the dorsolateral PFC, which is involved in higher cognitive operations and decision-making; the OFC, which is involved in salience attribution and goal-directed behaviors; and the anterior cingulate cortex, involved in inhibitory control and awareness in addicted subjects could underlie the enhanced incentive motivational value of drugs and the user's loss of control over drug intake.

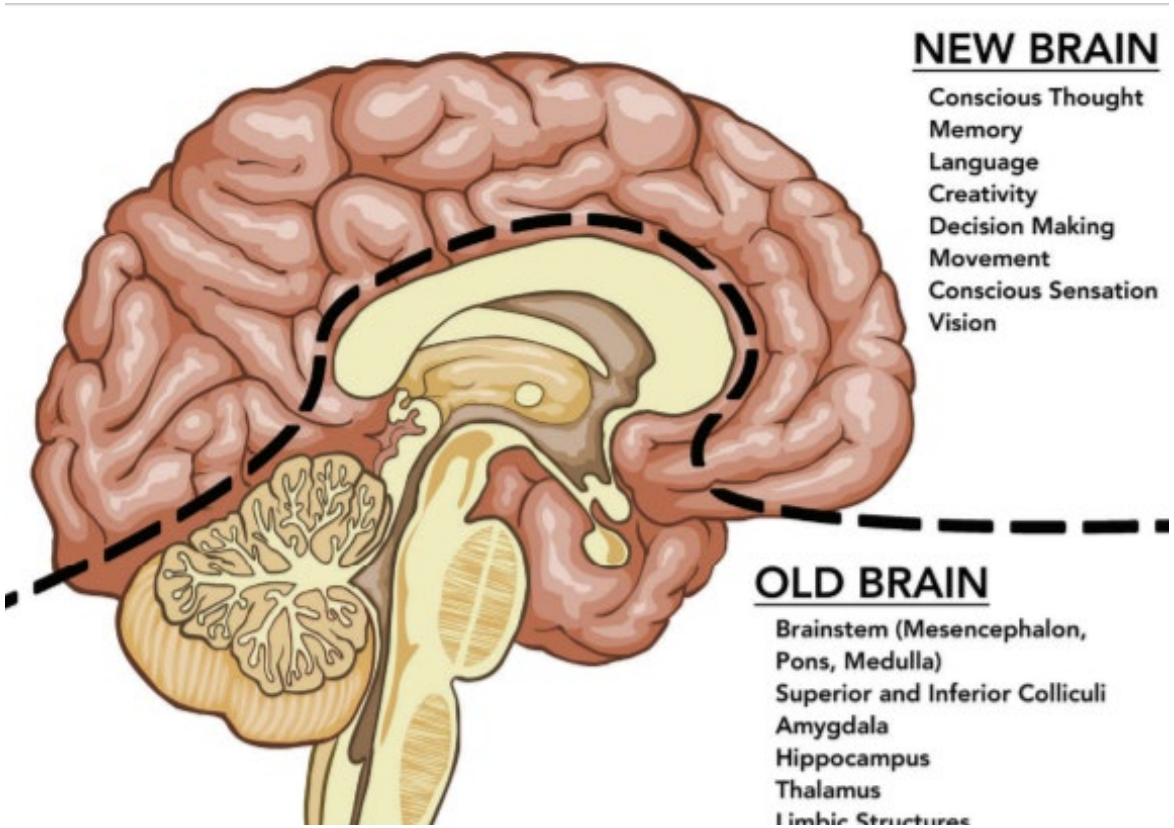
Memory, Psychoactive Drugs and Euphoric Recall

- When people use psychoactive drugs, memories of the experience are imprinted on the brain: where they got the drug, the reason they used it, and what feelings (emotional and physical) resulted.

- The stronger the drug, the more rapid the growth and proliferation of memory “footprints” (dendritic spines) and therefore the more deeply imprinted the memory.

- The earlier in life a person begins using drugs or practices addictive behaviors, the longer and stronger the memories remain in the brain and the more likely the brain is to use the information from those memories to deal with events later in life.

Old Brain – New Brain Distinction



- The old brain consists of: brainstem, cerebellum and mesocortex (mid brain), which contain the limbic system (the emotional center).
- Regulating physiological functions of the body.
- Experiencing basic emotions and cravings (e.g. anger, fear, hunger, thirst, lust, pain and pleasure)
- Imprinting survival memories (e.g. that green plant tastes good, this bad odor signifies danger)

Image: https://university.zhealtheducation.com/wp-content/uploads/2019/09/Neurology_Foundations_StudyGuide.pdf

Old Brain – New Brain #1

- The old brain responds to internal changes and memories as well as to sensory inputs from external influences.

- When a person uses a psychoactive drug, most often it is the old brain that remembers the experience and how it felt.

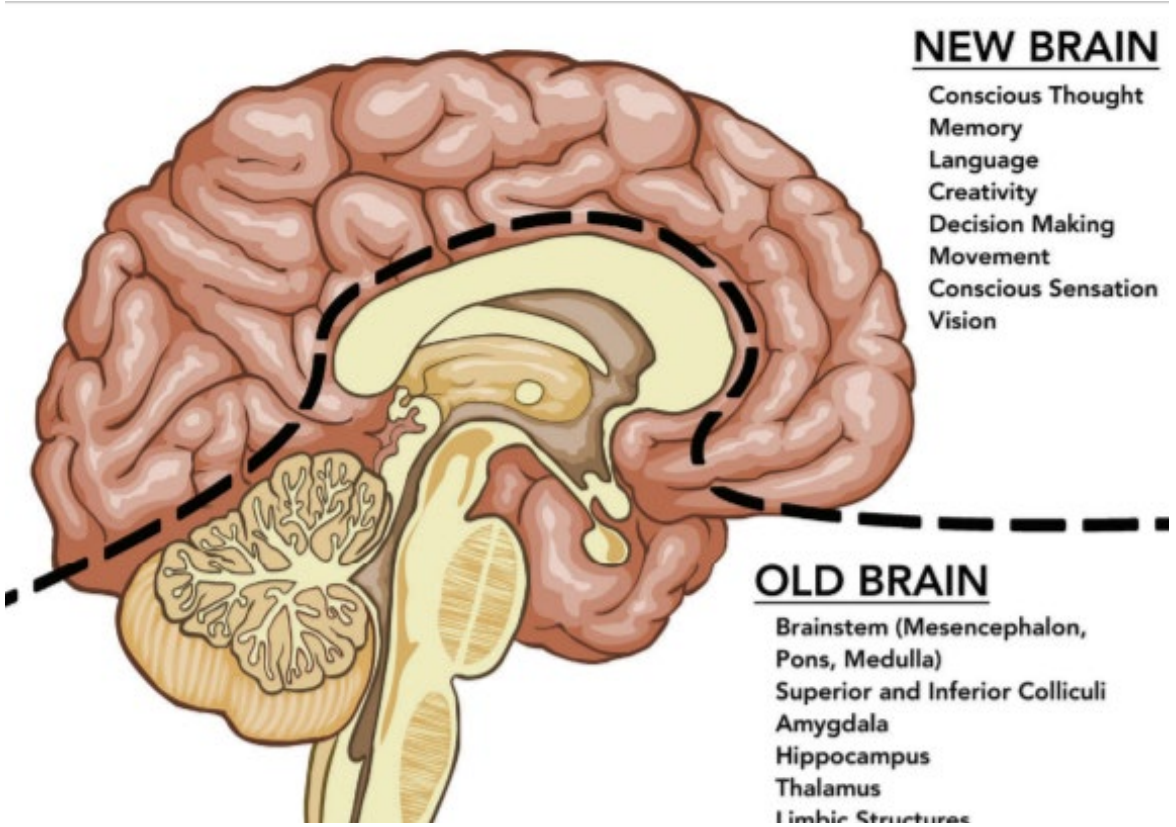
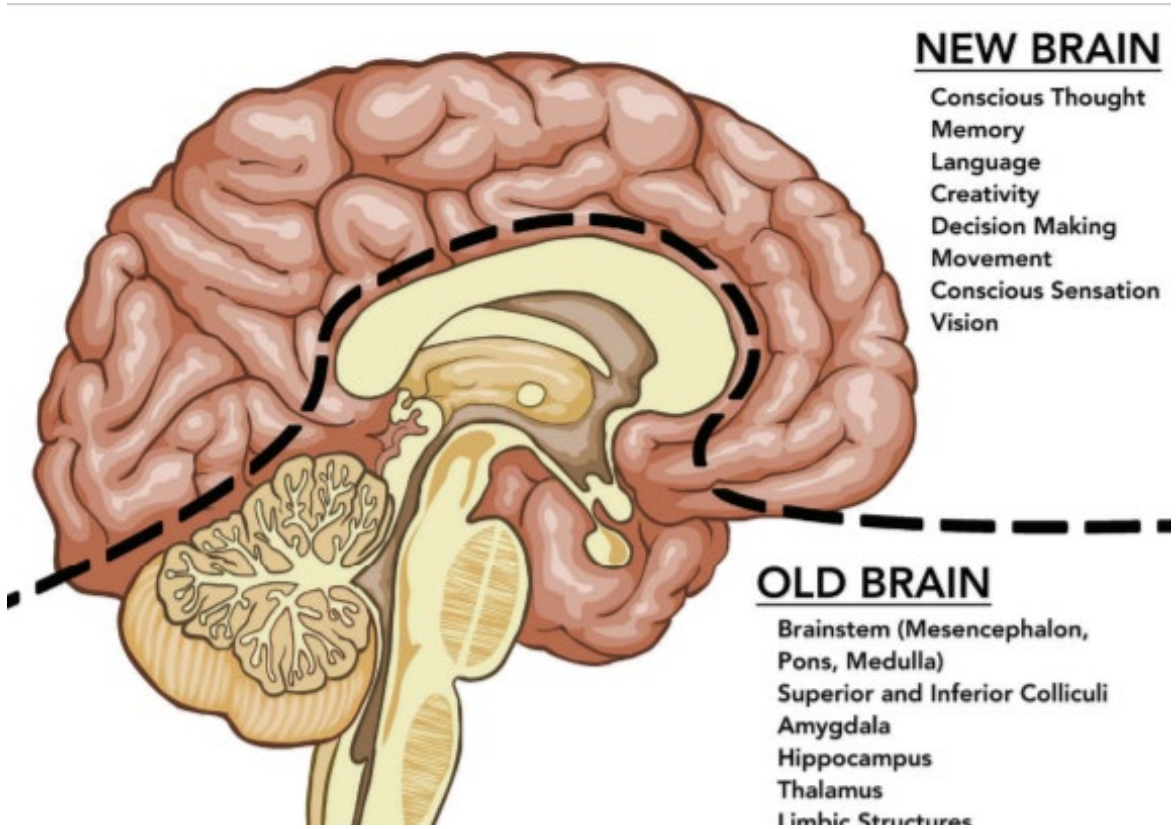


Image: https://university.zhealtheducation.com/wp-content/uploads/2019/09/Neurology_Foundations_StudyGuide.pdf

Old Brain – New Brain #2



- The old brain is the senior partner, and the new brain is the young upstart.
- Whenever the two brains are challenged by a crisis such as fear or anger, there is an automatic tendency to revert to the more established old-brain function.
- Because the craving to use a psychoactive drug almost always resides in the old brain, the desire for the pleasure, pain relief and excitement that drugs promise can be very powerful.

Image: https://university.zhealtheducation.com/wp-content/uploads/2019/09/Neurology_Foundations_StudyGuide.pdf

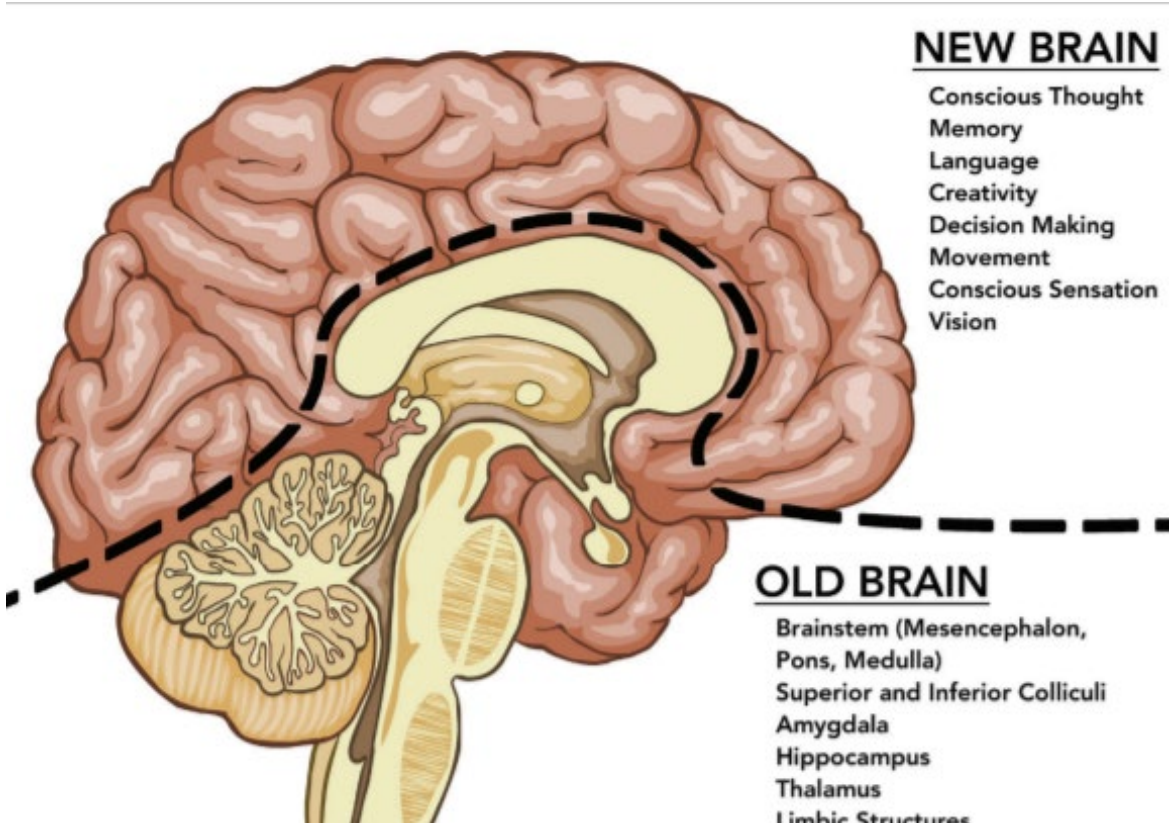
Old Brain – New Brain #3

- The new brain (neocortex) processes information from the old brain, from different areas of the new brain, and from the senses via the peripheral nervous system.

- The new brain allows us to speak, reason, create, remember, make decisions and then act. The old brain simply reacts.

Image: https://university.zhealtheducation.com/wp-content/uploads/2019/09/Neurology_Foundations_StudyGuide.pdf

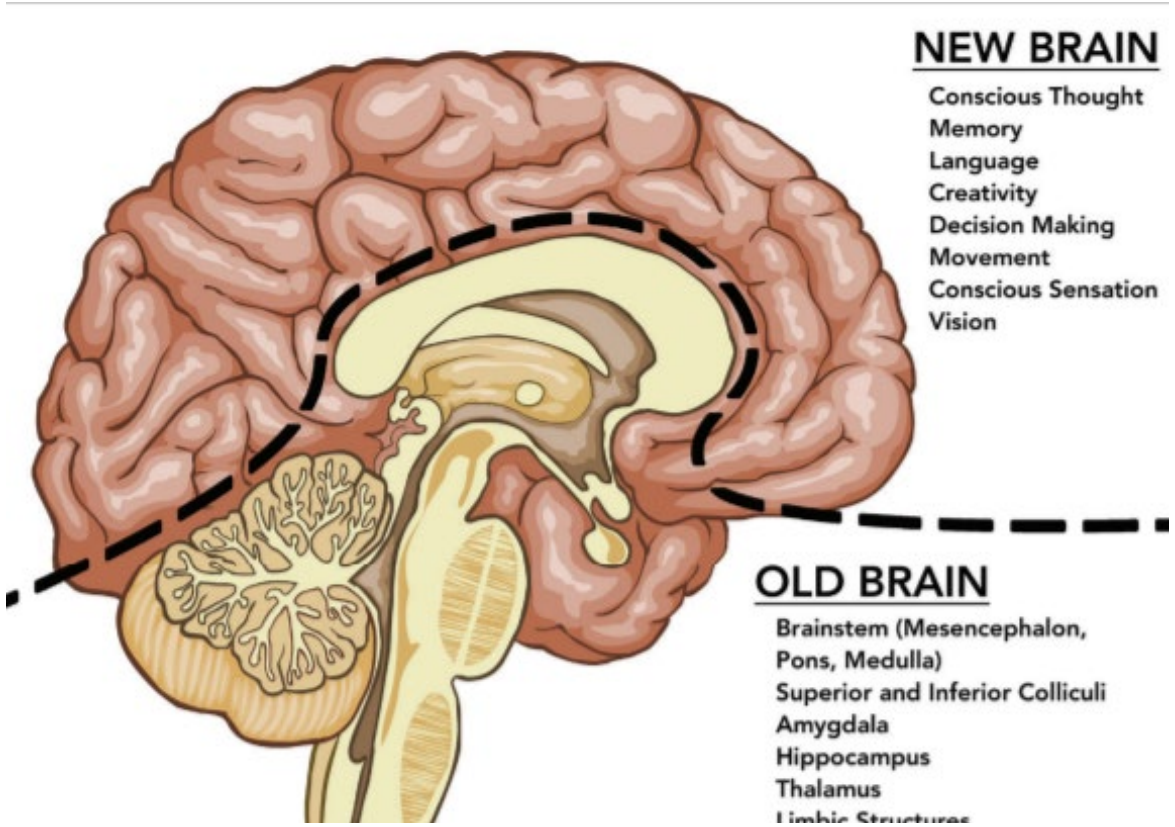
Old Brain – New Brain #4



- Craving can override the new brain's rational arguments of "too expensive" or "bad consequences" or "there's a midterm tomorrow so don't party tonight."
- The old brain acts four or five times more rapidly than the new brain, so an action is usually well under way before common sense kicks in.
- During intentional abstinence from a drug cravings are evoked by memory and emotions and a virtual tug of war between the old brain and the new brain occurs. There is a conscious desire to remain drug-free but the old brain seeks to resume drug use, mistaking the craving as a survival need.

Image: https://university.zhealtheducation.com/wp-content/uploads/2019/09/Neurology_Foundations_StudyGuide.pdf

Old Brain – New Brain #5



- The old brain and the new brain carry out their functions by creating, storing and utilizing memories. Even emotions and cravings depend on memories. Some memories are stored on a conscious level (explicit memory) and some are stored on an unconscious level (implicit memory).

- Storage, activation, and use of memories are at the heart of the obsession to use drugs, which is one-half of the addictive process.

Image: https://university.zhealtheducation.com/wp-content/uploads/2019/09/Neurology_Foundations_StudyGuide.pdf

Development

- As humans develop, they continue to learn to integrate the drives of the old brain and the common sense of the new brain. Some people, however, lose some of this ability due to genetic learning abnormalities, a chaotic or abusive childhood, brain injury, the use of psychoactive drugs and the practice of compulsive behaviors.

- Psychoactive drugs subvert the survival mechanism from common sense integration of the new and old brains, resulting in the irrational behavior of addiction, which relies on the “wants” of the old brain rather than the rational “needs” of the new brain.

Orbital Frontal Cortex (OFC)

The OFC has been shown to participate in outcomes related to primary reinforcers in both nonhuman and human studies.

These neurons encode details concerning the sensory properties of rewards, such as visual, olfactory and gustatory aspects, and the size or timing of past or future rewards, as well as the magnitude of more abstract rewards and penalties.

OFC and Anterior Cingulate Cortex (ACC)

- Impairments of the OFC and ACC are associated with compulsive behaviors and impulsivity, and it has also been postulated that impaired modulation of these regions by DA might underlie the compulsive and impulsive aspects of drug-taking and abuse.

- Impaired self-control plays a fundamental role in drug-taking behaviors in addiction. Successful self-regulation functions require top-down control from the PFC to the striatal and limbic regions involved with rewards and emotions.

OFC and ACC (Self-Regulation)

Impaired self-control in addicted people is believed to reflect disrupted prefrontal regulation of striatal regions. The level of impairment is influenced by the emotional state (negative mood increases impairment) and the context (exposure to unexpected cues can also impair it).

Damage to the OFC also interferes with the inhibition of responding to formerly rewarding cues that are no longer reinforcing, thus favoring the emergence of perseverative behaviors even when these are no longer reinforcing

OFC and ACC: Underlying Issues

- Dysregulated activity of the OFC could underlie both the impulsive choices for immediate rewards and compulsive drug taking when the drug-induced DA increases may be profoundly attenuated in addicted people.

- This loss of control might continue even when drug-taking has become less rewarding or when adverse consequences far outweigh the psychological or physiological benefits of drug-taking.

SUD and TBI: Research Findings

History of TBI is frequent among individuals receiving treatment for alcohol and substance use disorders

The relationship between alcohol abuse and TBI is complex and probably circular

Adolescents who drink regularly were twice as likely to sustain a TBI compared with adolescents who had never used alcohol.

Initial alcohol-related TBI sustained after age 12 were associated with a four-fold increased risk of repeat TBI by age 34

SUD and TBI: Misuse and Intoxication

There is strong evidence that intoxication at the time of injury is related to acute complications, longer hospital stays, and poorer discharge status.

Alcohol abuse prior to TBI has consistently been found to mediate outcome from TBI.

A history of substance misuse is related to a wide range of outcomes, including higher mortality rates, poorer neuropsychological functioning, increased chance of repeated injury, late deterioration and worse functional outcome.

SUD and TBI: More Facts

Intoxication and a history of premorbid alcohol use are related to worsening injury severity indicators and early medical outcomes.

Patient's with +BALs on hospital admission have lower levels of consciousness when admitted, longer duration of coma, and longer lengths of hospitalization.

Post-traumatic amnesia and loss of consciousness were significantly longer in groups of patients with pre-injury alcohol abuse.

SUD and TBI: Alcohol Use History

Pre-injury history of alcohol abuse also appears to exacerbate the effects of TBI on brain structure and function.

TBI patients with a history of alcohol abuse demonstrated greater volumes of intracranial hemorrhage.

TBI patients with a history of alcohol abuse also have more pronounced local brain atrophy over time compared to non-drinkers.

SUD and TBI: Implications after Injury

TBI sustained in people with a history of alcohol intoxication at the time of the injury demonstrated worse cognitive outcomes than those with negative toxicology screens, with particular difficulty on tests of verbal intelligence, verbal memory, attention and concentration.

Harmful or hazardous alcohol use in the 12 months prior to TBI was associated with poorer verbal learning and memory and slowed processing speed.

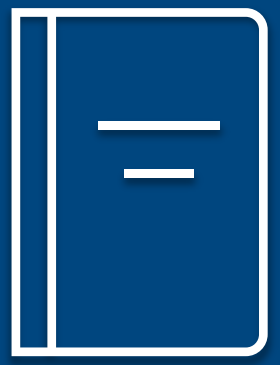
Previous alcohol abuse increases the risk for development of mood disorders following TBI

SUD and TBI: Complications

Substance use or SUD may complicate issues of TBI recovery by:

- Lowering seizure threshold
- Increasing risk for additional TBIs
- Contributing to brain damage
- After TBI, alcohol and other drugs may have a more powerful effect

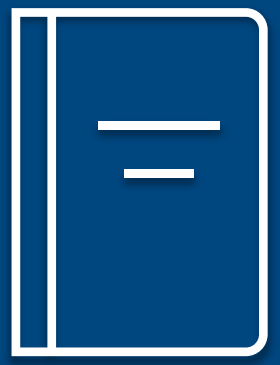
SUD and TBI: TBI Symptoms and SUD Treatment



The following TBI-related symptoms may hinder treatment for substance abuse:

- Cognitive limitations
- Increased irritability or emotional distress
- Problems with inhibition
- Treatment of pain with medications

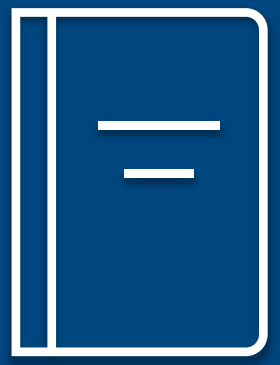
SUD and TBI: Implications for TBI Recovery



Negative consequences of ongoing SUD following TBI include:

- Interference with the natural healing process of the brain
- Increased risk for seizures
- Exacerbations of TBI-related physical and psychological symptoms (e.g., balance difficulties, depression)
- Magnifications of TBI-related cognitive difficulties (e.g., judgment, decision-making)
- Heightened risk for suicide attempts, particularly when depression is also present
- Increased risk for legal difficulties/criminal misconduct
- Difficulties distinguishing whether cognitive difficulties (e.g., problems with memory) are due to TBI or substance abuse
- Increased risk for future TBIs

Substance Use Disorder and TBI Risk Factors

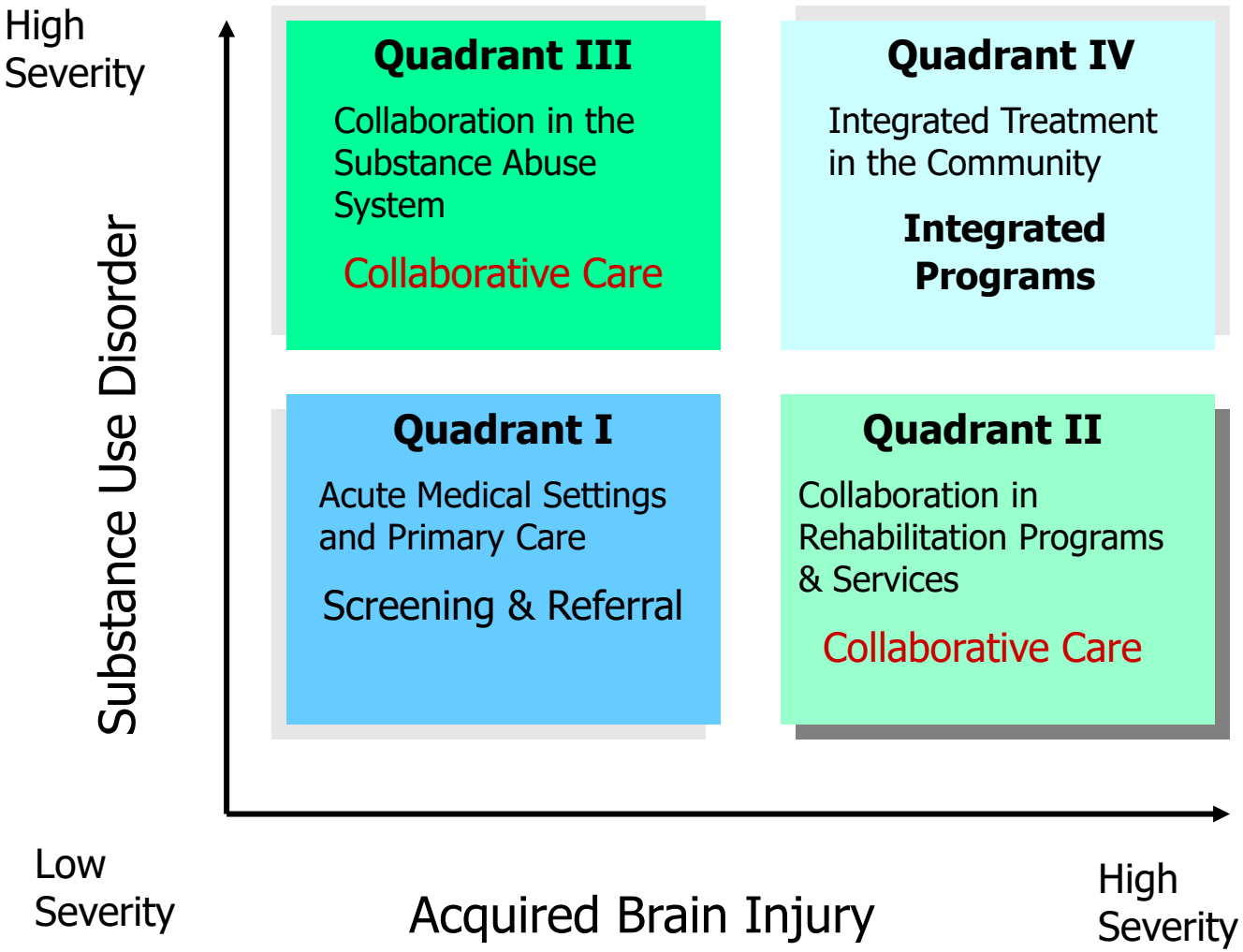


Risk factors for substance abuse following TBI include:

- Pre-TBI substance abuse
- Post-TBI depression
- Male gender
- Younger age
- Unmarried
- Having Medicaid or no health insurance

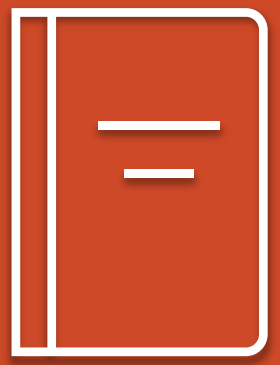
Integrated Treatment of Traumatic Brain Injury and Substance Use Disorder

4 Quadrant Model of Services



doi:10.1097/TA.0b013e3181e904cc

Assessment of Medical Status



- History of medical illnesses
- Implications of various medical illnesses on behavior (e.g. thyroid disease)
- Current medical status
- Most recent medical evaluation
- Current medications and implications for behavior and cognitive functioning

Assessment of Psychological Status and Psychiatric Illness

- Mental status examination
- History of psychiatric illness
- Use of “state” tests (e.g. BDI-II, BAI, DAPS)
- Use of “trait” tests (e.g. MMPI, PAI)
- Impact of psychological status on motivation
- Implications of a history of such psychiatric illnesses as recurrent depression, bipolar disorder, psychotic episodes, PTSD

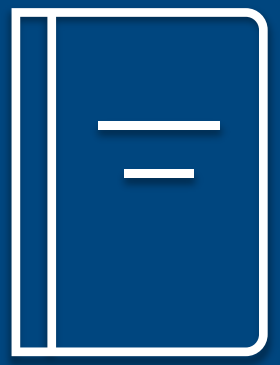
Substance Use Assessment

- Assessing substance abuse
- History taking
- Medical Issues
- Specifics regarding substance use (preferred drugs)
- Understanding the implications of specific drugs (e.g. MDMA) on brain function
- Implications of specific use patterns in reference to brain neurochemistry
- Understanding the circumstances of use
- Use of standardized measures

Assessment of Cognitive Status

- History—A significant number of people seeking treatment for SUD have histories of head trauma from multiple concussions to severe TBI.
- Not uncommon for people being seen in TBI programs to have had histories of substance use/abuse.
- At the time of treatment initiation, the use of standardized cognitive screening tools can be applied but the limits of these tools must be understood
- In addition to the detection of cognitive impairment due to TBI many people with SUD have cognitive impairment due to the drugs that have been taken and some have cognitive deficits due to developmental problems (e.g. LD, ADHD, toxic exposure)

Neuropsychological Assessment



- Assessing Neuropsychological Status
- Attention impairment
- Retrieval Difficulty/Memory impairment
- Executive Function Difficulty
- Language impairment/word finding difficulty
- Visuoperceptual impairment

Treatment Planning

- Taking neuropsychological status into account when planning treatment
- Cognitive demands of treatment
- Cognitive demands of living sober/drug free
- Modifying standard treatment techniques when initiating treatment for SUD
- Cognitive rehabilitation

Continuing Education Credit - Please Note!

- You must miss no more than 10 minutes AND complete the CE Evaluation Survey in order to be eligible for continuing education
- You will receive an email at the email address you used to register that will contain a link to a required survey
- You will have 72 hours from the date/time you receive the email to complete the survey
- You will receive your CE Certificate in approximately 4-6 weeks

GPRA Evaluation

ttc-gpra.org/GPRAOnline/SG?e=524112