OUDPC 2019

Acquired Brain Injury & Substance Use Disorder

Faculty/Presenter Bio

Faculty: Dr. Peter Selby, MBBS, CCFP, FCFP, MHSc, DipABAM, DFASAM

Chief, Medicine in Psychiatry Division, Centre for Addiction and Mental Health (CAMH)
Clinician Scientist, Addictions Division, Centre for Addiction and Mental Health (CAMH)
Professor, Departments of Family and Community Medicine and Psychiatry, and Dalla Lana School of Public Health, University of Toronto
peter.selby@camh.ca (416) 535-8501 ext. 36859

Peter Selby is the Chief of Medicine in Psychiatry Division and a Clinician Scientist at the Centre for Addiction and Mental Health (CAMH). He is a Professor in the Departments of Family and Community Medicine, Psychiatry, and the Dalla Lana School of Public Health at the University of Toronto. He is also a Clinician Scientist in the Department of Family and Community Medicine. His research focus is on innovative methods to understand and treat addictive behaviours and their comorbidities. He also uses technology to combine clinical medicine and public health methods to scale up and test health interventions. His cohort of 240,000 treated smokers in Ontario is an example.

He has received grant funding totaling over 85 million dollars from CIHR, NIH, and Ministry of Health and has published 150 peer reviewed publications. He has published 6 books (including 5 edited), is the author of 31 book chapters, and 38 research reports prepared for the government. He is the Chair of the Medical Education Council for the American Society of Addiction Medicine. Dr. Selby mentors Fellows in Addiction Medicine and Addiction Psychiatry, junior investigators and medical students. The use of innovative methods to communicate messages makes Dr. Selby a sought after speaker for various topics including addictive disorders, motivational interviewing, and health behavior change at individual and system levels.

Disclosures

- **Grants/Research Support:**
  - CAMH, Health Canada, OMOH, CIHR, CCSA, PHAC, Pfizer Inc./Canada, OLA, Medical Psychiatry Alliance, ECHO, CCSRI, CCO, OICR, Ontario Brain Institute, McLaughlin Centre, AHSC/AFP, WSIB, NIH, AFMC, Shoppers Drug Mart, Bhasin Consulting Fund Inc., Patient-Centered Outcomes Research Institute

- **Speaking Engagements (Content not subject to sponsors approval)/Honoraria:**
  - Pfizer Canada Inc., ABBVie, Bristol-Myers Squibb

- **Consulting Fees**:
  - Pfizer Inc./Canada, Evidera Inc., Johnson & Johnson Group of Companies, Medcan Clinic, Inflexxion Inc., V-CC Systems Inc., MedPlan Communications, Kataka Medical Communications, Miller Medical Communications, Nvision Insight Group, Sun Life Financial

- **Other: (Received drugs free/discounted for study through open tender process)**
  - Johnson & Johnson, Novartis, Pfizer Inc.

- **NO TOBACCO or VAPING or CANNABIS or ALCOHOL or FOOD INDUSTRY FUNDING**

Mitigating Potential Bias

- In order to mitigate the potential for bias, all the material presented herein is based explicitly on evidence-based research

Learning Objectives

1

Examine how cognitive impairments may alter the approach to the treatment of substance use

2

Explore adaptations of mainstream treatment using a case-based approach

3

Develop a cessation treatment using a case-based approach

1

Examine how cognitive impairments may alter the approach to the treatment of substance use

Relevance

- There is evidence that having sustained a brain injury increases the likelihood of smoking…(kids)
- As many as 70% of concurrent disorders patients also report a history of brain injury with loss of consciousness
- In individuals who are cognitively impaired, smoking may impede access to housing and health care.

What's the Problem?

- Low Motivation
- Low Ability

What do these people have in common?

- Derek Boogard
- Rick Rypien
- Wade Belak
- Steve Montador
- Bob Probert
- Todd Ewen

Rest in Peace

- Derek Boogard 28
- Rick Rypien 27
- Wade Belak 35
- Steve Montador 35
- Bob Probert 45
- Todd Ewen 49

How Brain Injury May Affect Smoking Cessation

- Emotional Dysregulation
  - Increased likelihood of concurrent mental health issues
  - Increased likelihood of substance misuse/substance use disorders
  - Increased impulsivity
- Cognitive Impairment
  - Memory Impairment
  - Attention (focusing and shifting)
  - Executive Dysfunction
    - Planning
    - Self-monitoring (insight and awareness)
    - Problem-solving
    - Initiating
  - Communication

Acquired Brain Injury in Canada

Incidence of ABI greater than multiple sclerosis, HIV, spinal cord injury and breast cancer combined

~500 000 Canadians will endure a brain injury each year

TBI is the leading cause of death and disability in Canadians under age 40

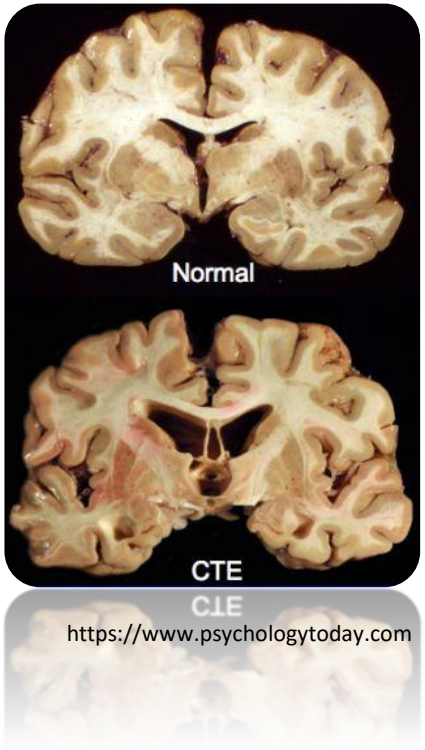
Annual cost of ABI in Ontario roughly **$2-3 billion**

Chronic Traumatic Encephalopathy

Progressive neurodegenerative disease from multiple concussions

Initial symptoms, impairment in

- Mood
- Behaviour
- Cognition and memory
- Executive function
- Motor coordination
- Chronic headache



https://www.psychologytoday.com

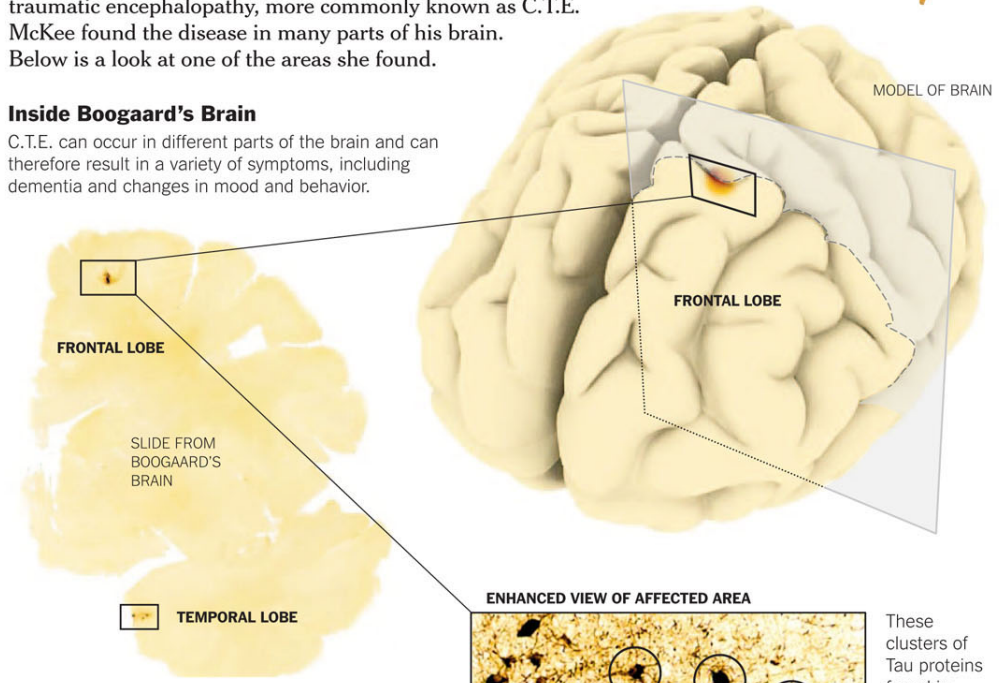
Symptoms worsen over time and can lead to Parkinson's and dementia

The Signs and Science of C.T.E.

Dr. Ann McKee, a neuropathologist, received Derek Boogaard's brain within days of his death and began testing it for chronic traumatic encephalopathy, more commonly known as C.T.E. McKee found the disease in many parts of his brain. Below is a look at one of the areas she found.

Inside Boogaard's Brain

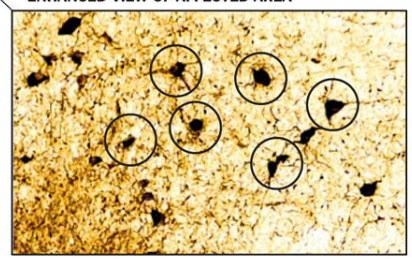
C.T.E. can occur in different parts of the brain and can therefore result in a variety of symptoms, including dementia and changes in mood and behavior.



This slide from Boogaard's brain shows two of the many areas of dying cells, including in his frontal lobe, top, which controls personality and judgment.

Sources: Dr. Ann McKee, director of Neuropathology, Bedford V.A. Medical Center and co-director of the Boston University Center for the Study of Traumatic Encephalopathy; Department of Health and Human Services

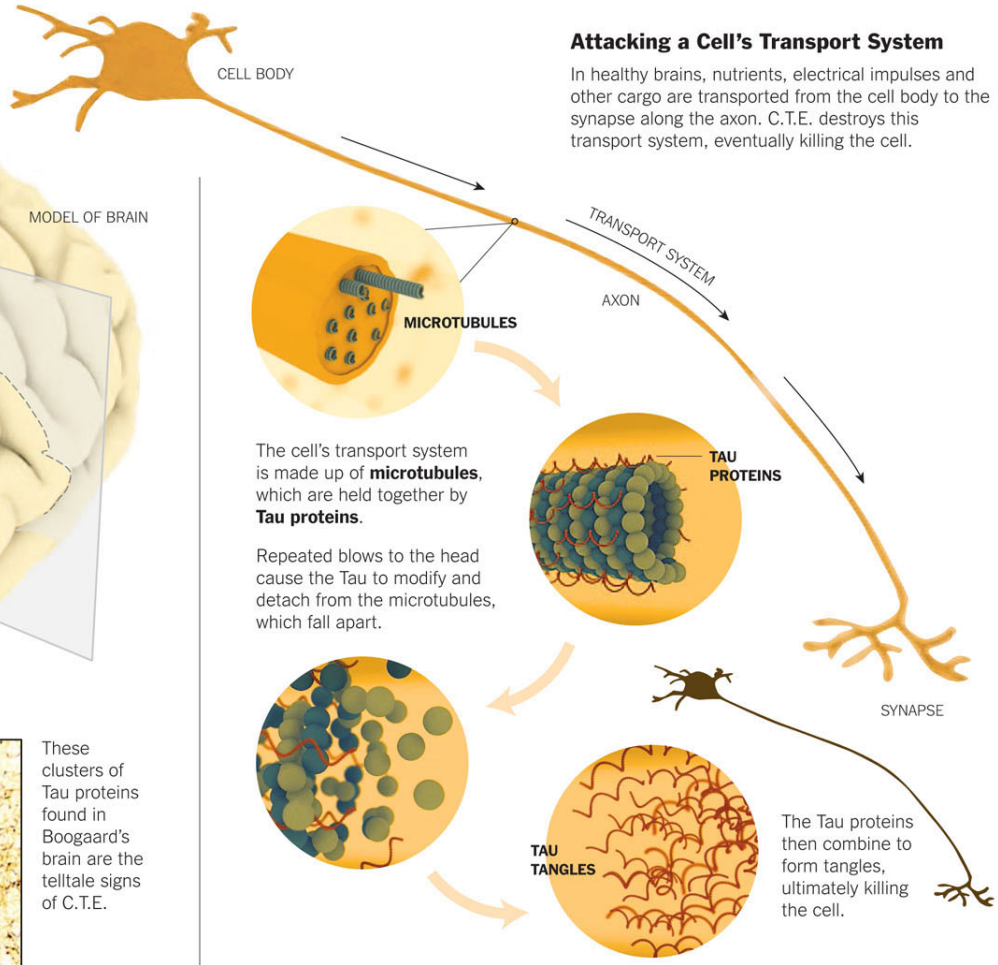
ENHANCED VIEW OF AFFECTED AREA



These clusters of Tau proteins found in Boogaard's brain are the telltale signs of C.T.E.

Attacking a Cell's Transport System

In healthy brains, nutrients, electrical impulses and other cargo are transported from the cell body to the synapse along the axon. C.T.E. destroys this transport system, eventually killing the cell.



The cell's transport system is made up of **microtubules**, which are held together by **Tau proteins**.

Repeated blows to the head cause the Tau to modify and detach from the microtubules, which fall apart.

The Tau proteins then combine to form tangles, ultimately killing the cell.

JOE WARD AND GRAHAM ROBERTS/THE NEW YORK TIMES

Substance Use as a Risk Factor for TBI?

- **36-73%** of individuals are intoxicated during time of TBI
- Among individuals that have experienced TBI

31 – 79% history of alcohol misuse

21 – 44% history of drug use

8 – 13% history of drug "abuse"

Intoxication at Time of Injury

40-60% of TBIs presenting to ER are associated with substance use (Graham & Cardon, 2009; Bjork & Grant, 2009; IOM, 2009)

The higher the blood alcohol content, the more likely an injury includes a TBI
(Savola, Niemela & Hillbom, 2005)

How Does TBI Trigger Addiction?

Pathophysiology

- Cellular mechanisms involved in brain injury result in structural damage that increase susceptibility to addiction

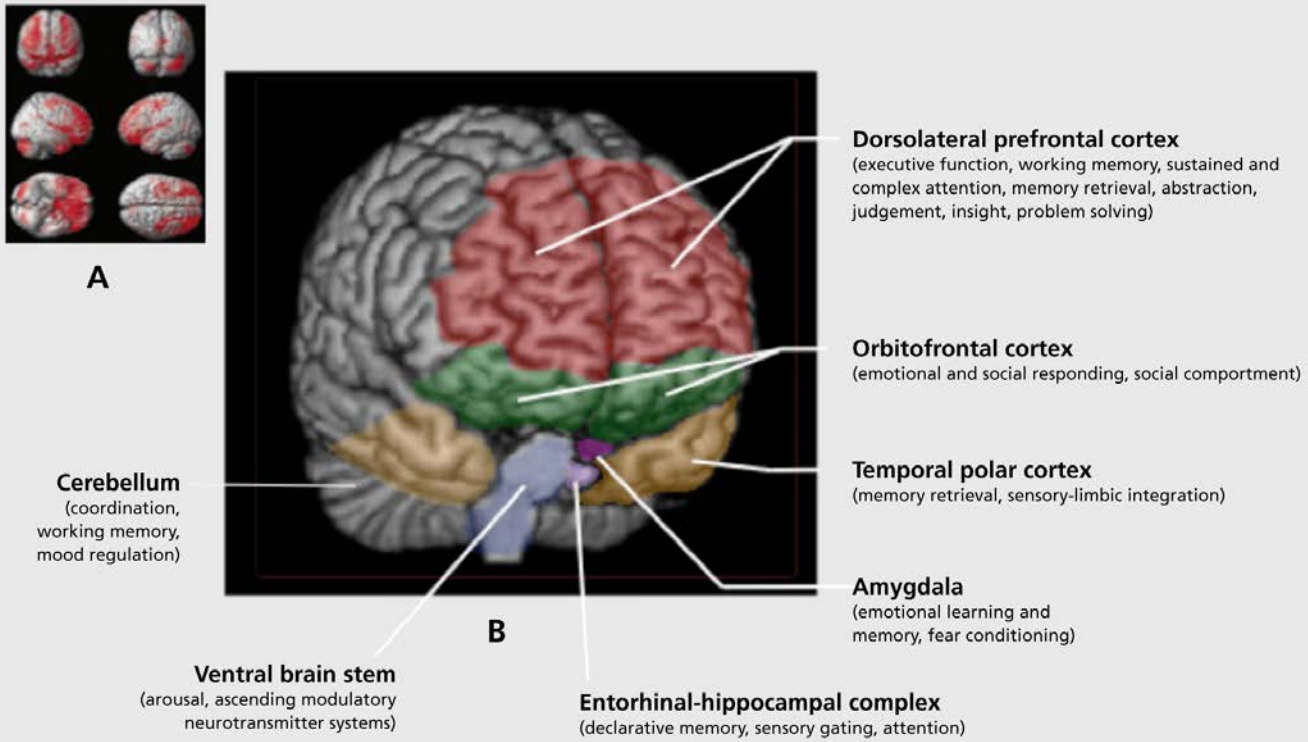
Neurobehaviour

- Damage to brain regions involved in executive function, emotional processing and judgement increase likelihood of risky/addictive behaviour

Development

- Early life TBI can increase risk of substance abuse and addiction later in life

Brain regions vulnerable to TBI and relationship to neurobehavioural sequelae

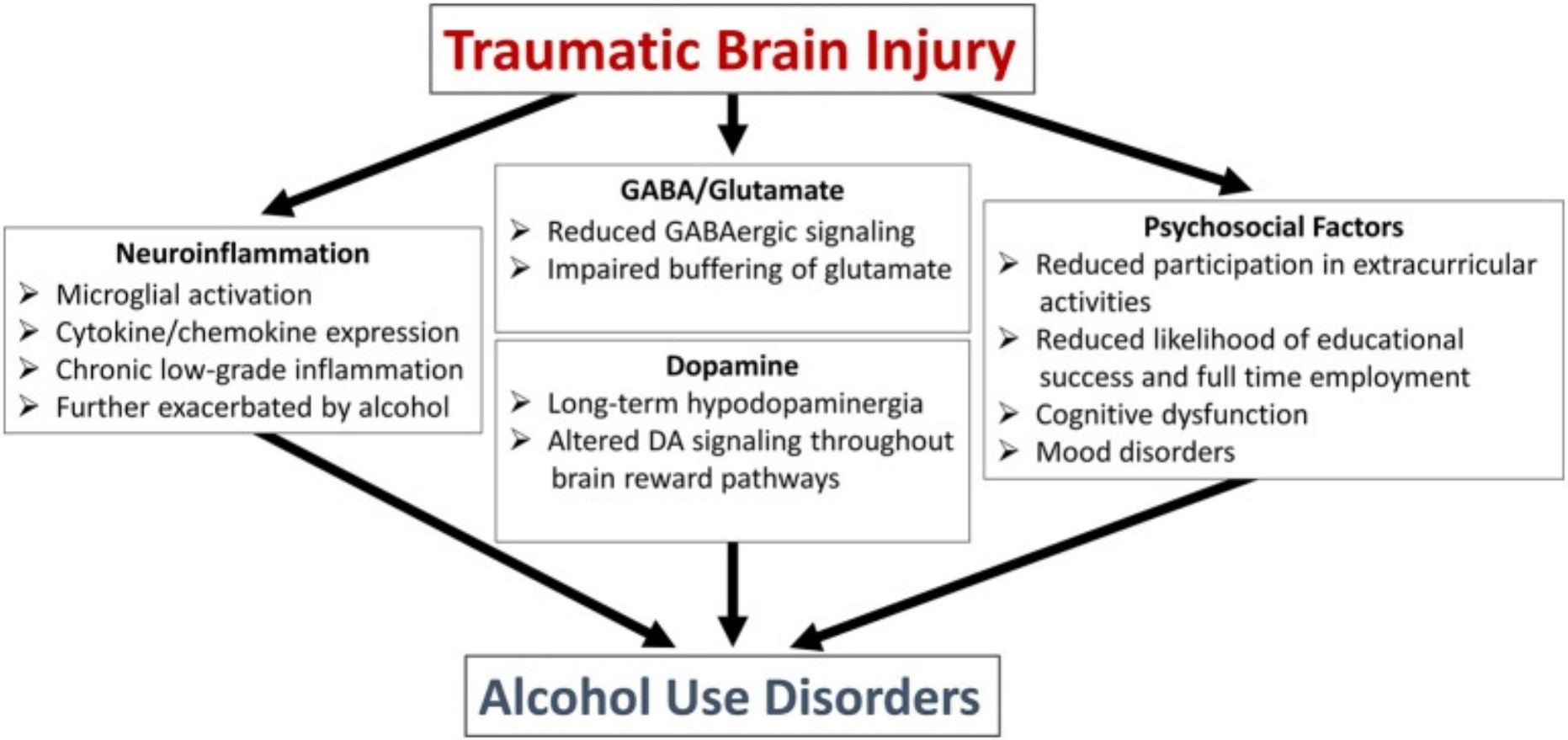


A) Adapted from ref 112: Bigler E. Structural imaging In: Silver J, McAllister T, Yudofsky S, eds. *Textbook of Traumatic Brain Injury.* Washington DC: American Psychiatric Press; 2005:87. Copyright © American Psychiatric Press, 2005.
(B) Adapted from ref 111: Arciniegas DB, Beresford TP. *Neuropsychiatry: an Introductory Approach.* Cambridge, UK: Cambridge University Press; 2001:58. Copyright © Cambridge University Press, 2001

TBI and the Role of Dopamine

- TBI causes reduced dopamine transporter (DAT) expression in brain
- May alter dopamine neurotransmission
- Increased risk of:
  - Depression
  - Anxiety
  - Substance abuse
    - Increased stimulation of reward pathways from psychostimulants

Proposed Mechanisms for Substance Use after TBI



2

Explore adaptations of mainstream treatment using a case-based approach

Case Study

- 18 year-old student in Grade 10
- ATV accident resulting in Scattered SAH
- Pre-morbid a daily ETOH and THC user
- Smoked 7-10 cigarettes per day (some more as poppers)
- Pre-morbid diagnoses included: PTSD/ADHD/ODD/FAS/anxiety and depression
- Has fought in fight clubs
- Polite and cooperative arriving on the unit, participated in therapy for the most part
- Poor memory
- AMA four days after admission

Work Flow

- Pharmacist started him on Nicotine Patch
- OT started wayfinding assessment, repeated many times over the week:
  - hard time remembering the route back to the unit
  - ++distractible outdoors
  - didn’t seem to recall the conditions associated with leaving the unit
- Family/friends took him out to smoke for first four days, then needed to return home (lived too far away)
- Discussion with interdisciplinary team
- Practicing of skills for wayfinding as this was patient goal by all disciplines (took precedence over other assessments)

Simultaneous Occurrences

- Statement was made that if he was still here in a month and he had no access to cigarettes, he was going to punch someone
- OT decided to upgrade to independent off-unit as he was aware and accepting of risks, had strategy
- Discussion with Physician and Pharmacist regarding break-through craving management
- BT added in strategy for if lost (business card for help to return) and script regarding risks and craving management
- Patient removed his patch saying he didn't like using it

Outcomes

- Patient made it clear he was unwilling now to remain and was going AMA (locked unit and lack of access to any of his coping strategies too overwhelming)
- Refused to access lawyer for private therapy
- Couch surfing at friends
- Sister attempting to access supports
- Patient kept calm demeanor despite cravings and frustrations

Take-Away

- Quicker to look at alternatives (how can we facilitate a modified version of success if capable and wanting to take on risk)
  - Safety plan for risks?
- Break-through in addition to standing NRT at start for high risk cases?
- Quicker explanation of optional modalities for therapy?

2

Explore the connection between ABI and Substance Abuse

Early Life Brain Injury and Substance Abuse

- Age of first brain injury associated with earlier age of substance abuse (Fishbein et al., 2016; Weil and Karelina, 2017)
- Children < 5 years old experiencing TBI 3.6x more likely to develop substance abuse during teens than those without TBI (McKinlay et al., 2014)
- Brain injury during early life associated with later development of AUD (Weil et al., 2016a; Merkel et al., 2017a)
- Students in high school experiencing TBI are twice as likely to develop AUD (Ilie et al., 2015)
- Individuals in inpatient rehabilitation for TBI ~2x more likely to meet diagnostic criteria for substance abuse if suffered prior brain injury before age 16 (Corrigan et al., 2013)

Longitudinal Studies

- **New Zealand** (McKinlay et al, 2002, 2008, 2013, 2014)
  - First TBI before age 6 → by age 25
    - 300% increased risk of alcohol
    - 269% increased risk of drug dependence
  - First TBI age 16-21
    - 300% increased risk of drug dependence
    - TBI before 21 highly associated with likelihood of arrest

- **Denmark** (Orlovska et al., 2014)
  - 65% increase in risk of schizophrenia
  - 59% increase in risk of depression
  - 28% increase in risk of bi-polar disorder
  - Those injured between age 11-15 at highest risk for depression and schizophrenia
  - Added risk not increased by family psychiatric history

Natural Hx of TBI and Substance Use

- Substance use may decrease during first year post-injury due to hospitalization and rehabilitation
- Substance use following TBI can increase risk of neurological impairments and reduce recovery rate
- Research by Pagulayan et al. (2016) revealed significant increase in alcohol consumption 1-6 months after TBI
  - Intervention during this critical window may improve clinical outcome

Pagulayan et al., 2016; Beaulieu-Bonneau et al., 2017

Substance Use Disorder Treatment in Clients with Brain Injury

- First used at a younger age
- Have more severe SUD (worse use and more prior treatments)
- Have more co-occurring mental health problems
- Have poorer prognosis for successful treatment outcome (more so earlier the age at first TBI?)

Corrigan & Mysiw, 2012

Prevalence of TBI among individuals with Mental Illness and SUD

- N = 295 individuals with concurrent mental illness and SUD
  - 80% screen positive for at least 1 TBI
  - 25% reported at least 1 TBI (moderate or severe)
- TBI associated with greater:
  - Current alcohol use
  - Psychiatric symptomatology
  - Lifetime institutionalization
  - Homelessness
- Higher prevalence of TBI among:
  - Individuals with PTSD, borderline personality disorder and antisocial personality disorder
  - Individuals with psychotic disorders vs mood disorders
  - Men vs Women
  - Individuals that experienced early age TBI with loss of consciousness

McHugo et al., 2017

TBI and Subsequent Psychiatric Disorder: meta-analysis

Association between prior TBI and subsequent psychiatric diagnosis in adults ≥ 18 years old

	# of Studies (N)	Odds Ratio (95% CI)	What does this mean?
All Psychiatric Disorders of interest	**20**	**2.00** (1.50 – 2.66)**	Previous TBI is associated with an **increased odds** of developing a psychiatric disorder ≥12 months after injury
Depression	10	2.14** (1.65 – 2.77)	
Mixed Affective Disorder	2	1.84** (1.50 – 2.66)	
Bipolar Disorder	3	1.85* (1.17 – 2.94)	

*$p < 0.01$; **$p < 0.0001$

Perry et al., 2016

Potential Long-Term Effects of Sport-Related Concussion: Systematic Review

Self-reported mental health issues among former collegiate athletes enduring ≥ 1 concussion

- Depression = 10.4%
- Anxiety = 16.2%
- Alcohol dependence = 5.8%
- Substance Use = 2.9%
- Cognitive Problems = 3.8%

Potential Long-Term Effects of Sport Related Concussions: Systematic Review

- Rate of depression **(PHQ-9 ≥ 10)** among former collegiate athletes (N= 797)
  - No history of concussion = 2.8%
  - One = 5.6%
  - Two = 10.4%
  - Three or more = 8.9%
- **Self-reported** depression among retired NFL players (N= 1044)
  - No history of concussion = 3.0%
  - 1 – 2 = 8.2%
  - 3 – 4 = 13.7%
  - 5 – 9 = 19.3%
  - 10 or more = 26.7%

Manley et al., 2017

3

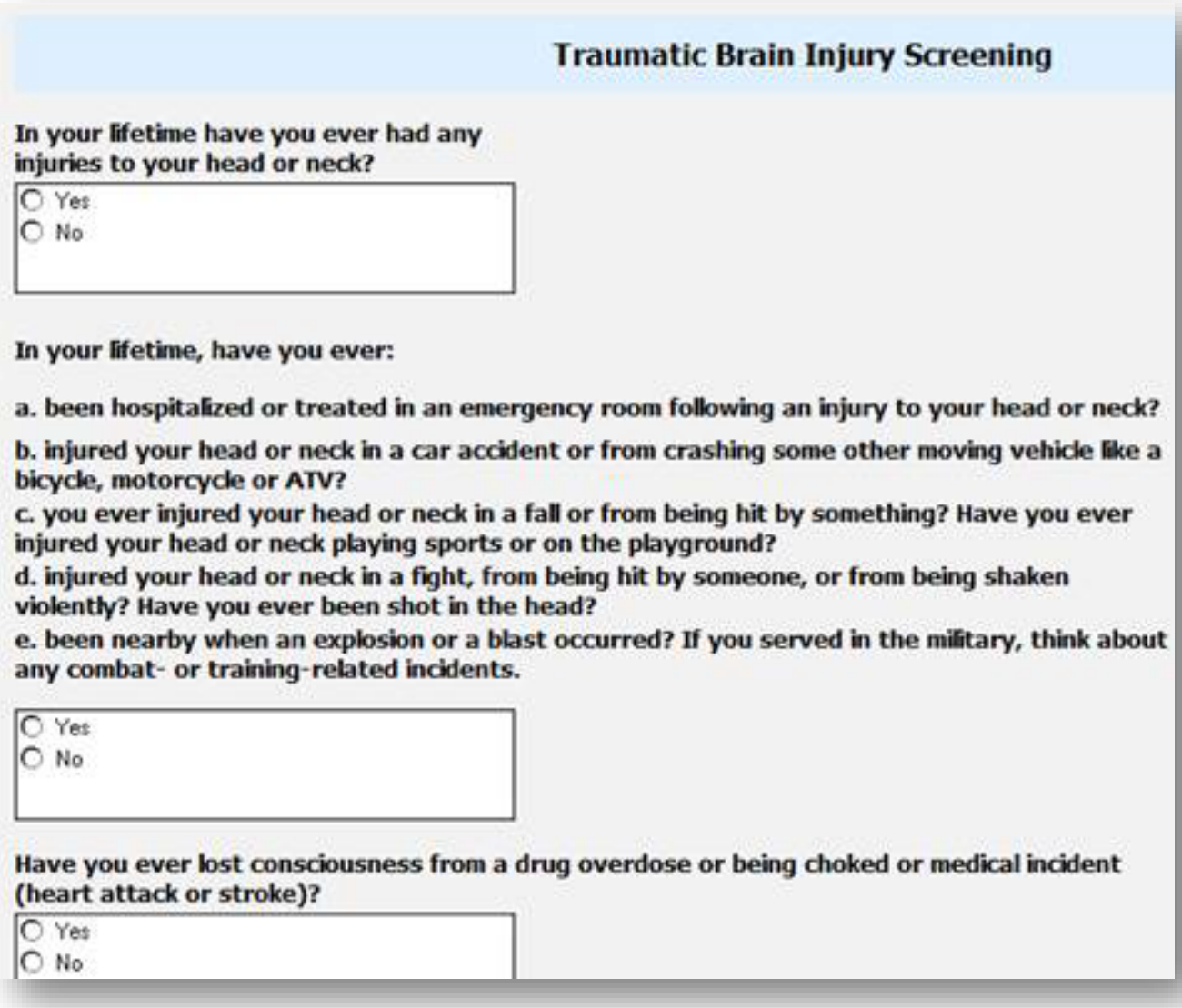
Develop a cessation treatment using a case-based approach

CAMH Screening Project

First Canadian Data

The Screener

- Adaptation of the Ohio Valley Brain Injury Identification Method
- Integrated into current assessment protocol at 60 White Squirrel Way and in ICARE



Traumatic Brain Injury Screening

In your lifetime have you ever had any injuries to your head or neck?

- ○ Yes
- ○ No

In your lifetime, have you ever:

a. been hospitalized or treated in an emergency room following an injury to your head or neck?

b. injured your head or neck in a car accident or from crashing some other moving vehicle like a bicycle, motorcycle or ATV?

c. you ever injured your head or neck in a fall or from being hit by something? Have you ever injured your head or neck playing sports or on the playground?

d. injured your head or neck in a fight, from being hit by someone, or from being shaken violently? Have you ever been shot in the head?

e. been nearby when an explosion or a blast occurred? If you served in the military, think about any combat- or training-related incidents.

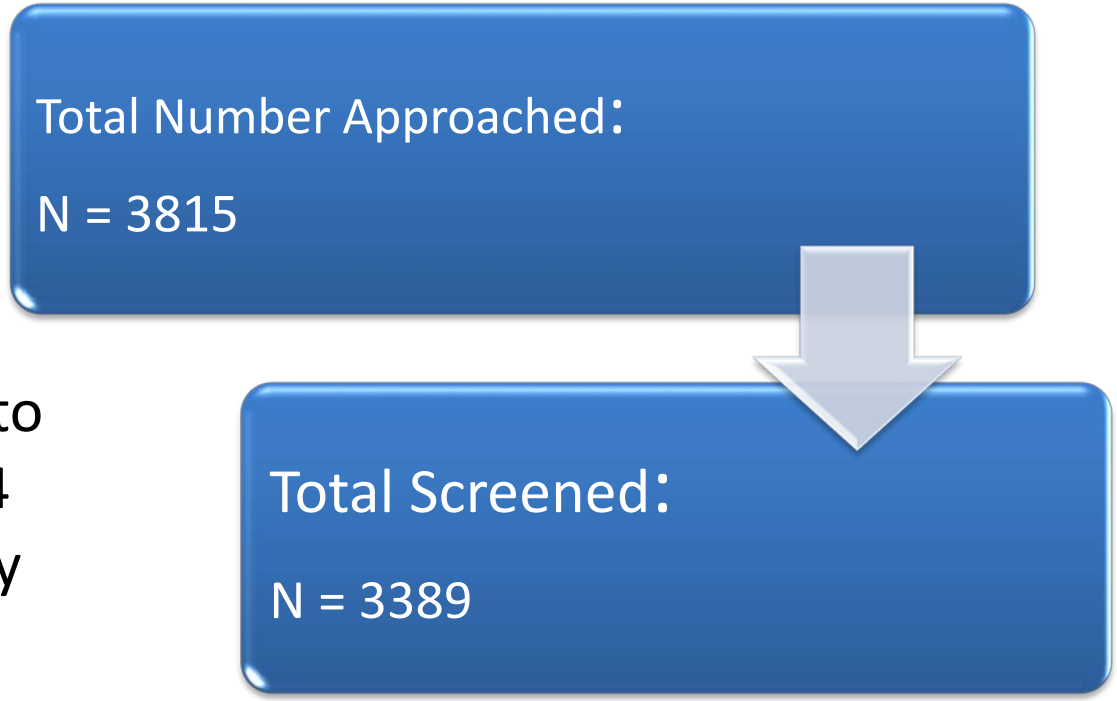
- ○ Yes
- ○ No

Have you ever lost consciousness from a drug overdose or being choked or medical incident (heart attack or stroke)?

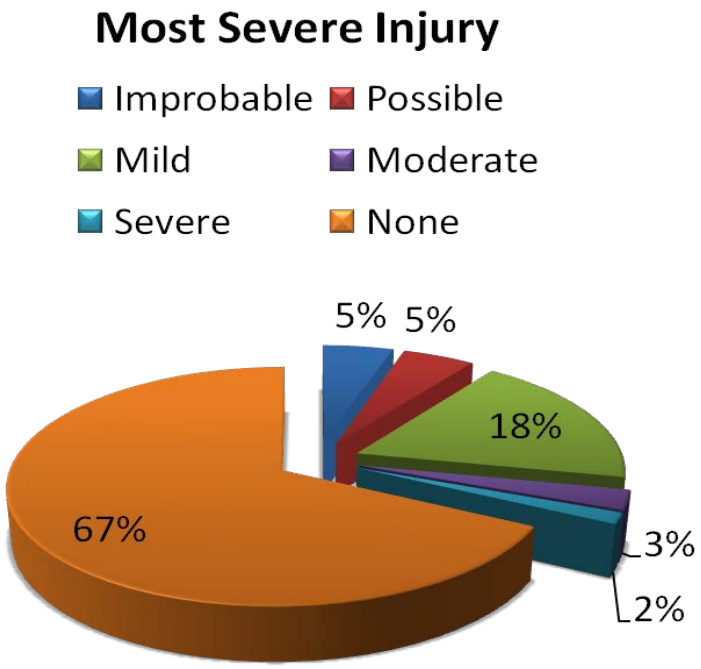
- ○ Yes
- ○ No

Screening Project

- July 2011 to ongoing
- Partial data up to December 2014 presented today



From All Addictions Programs at CAMH



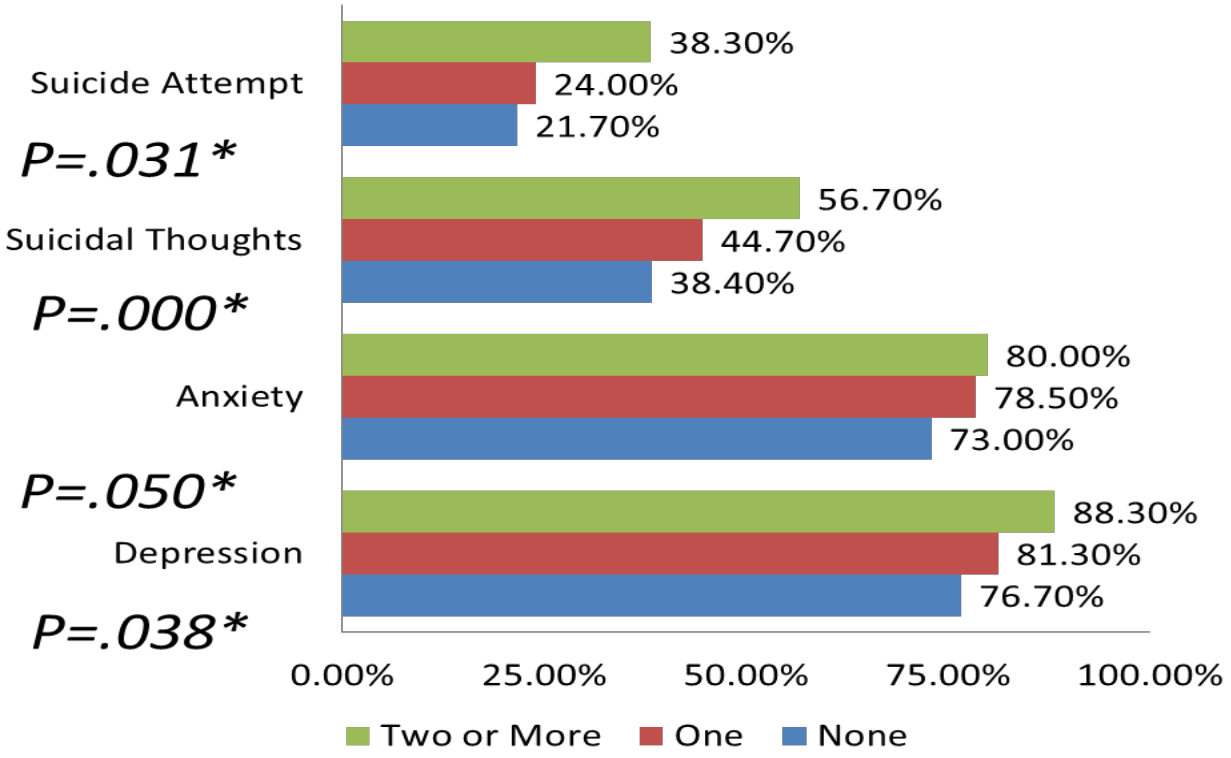
N= 3389

28% of mild to severe injuries occurred before the age of 15

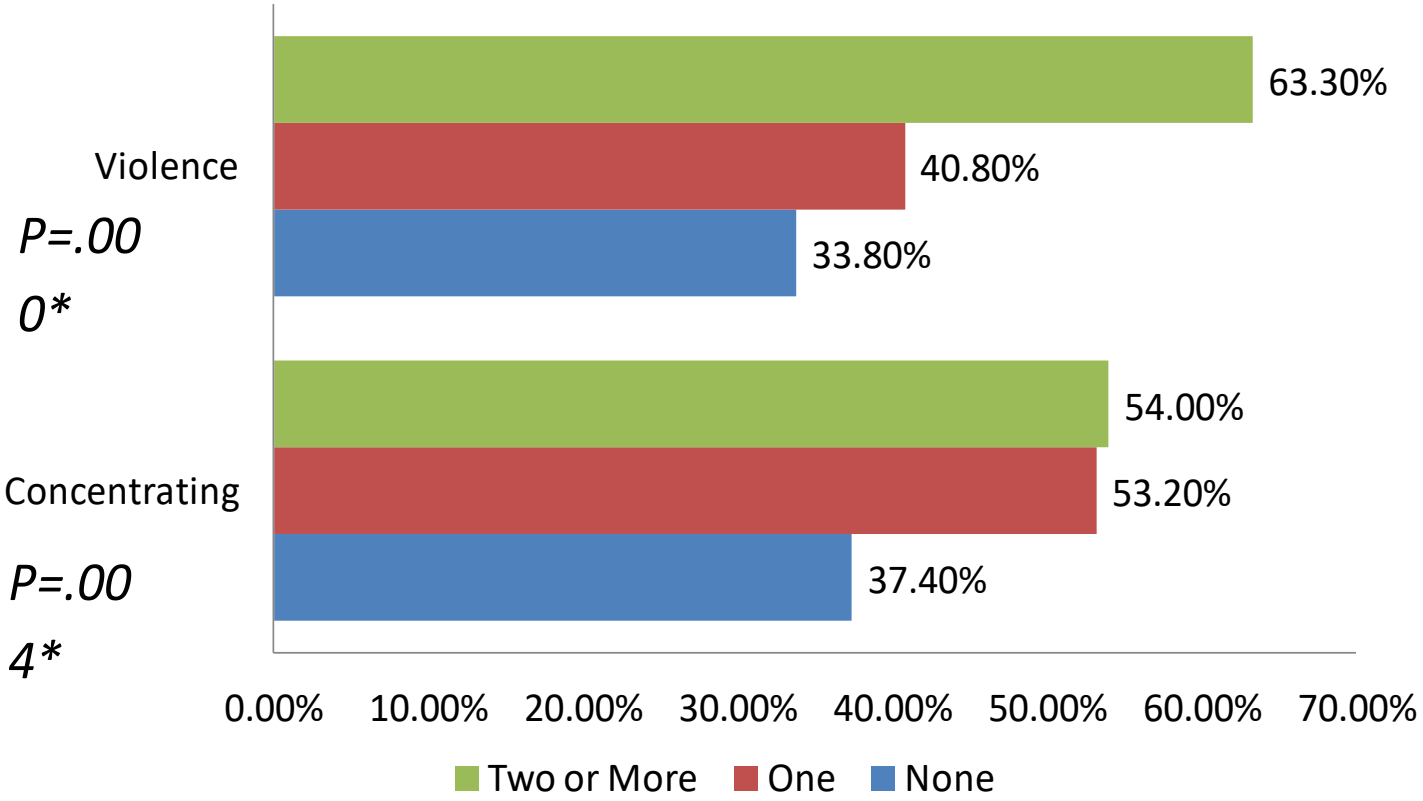
Lifetime substance use (years)

	No ABI with LOC N=1180	1 ABI with LOC N=286	2 or more ABI with LOC N=75	F*
Alcohol	19.09 (12.49)	22.57 (11.82)	22.68 (11.57)	p=.000
Alcohol to intoxication	12.36 (12.18)	14.66 (12.29)	17.69 (13.03)	p=.000
Cocaine	5.02 (7.95)	7.27 (8.90)	6.66 (8.68)	p=.000
Cannabis	8.57 (10.39)	11.00 (12.33)	11.17 (14.81)	p=.005
Lifetime DTs	.513 (4.18)	1.62 (9.10)	1.95 (6.42)	p=.027
* Welch robust test of equality of means				

Mood Symptoms Lifetime



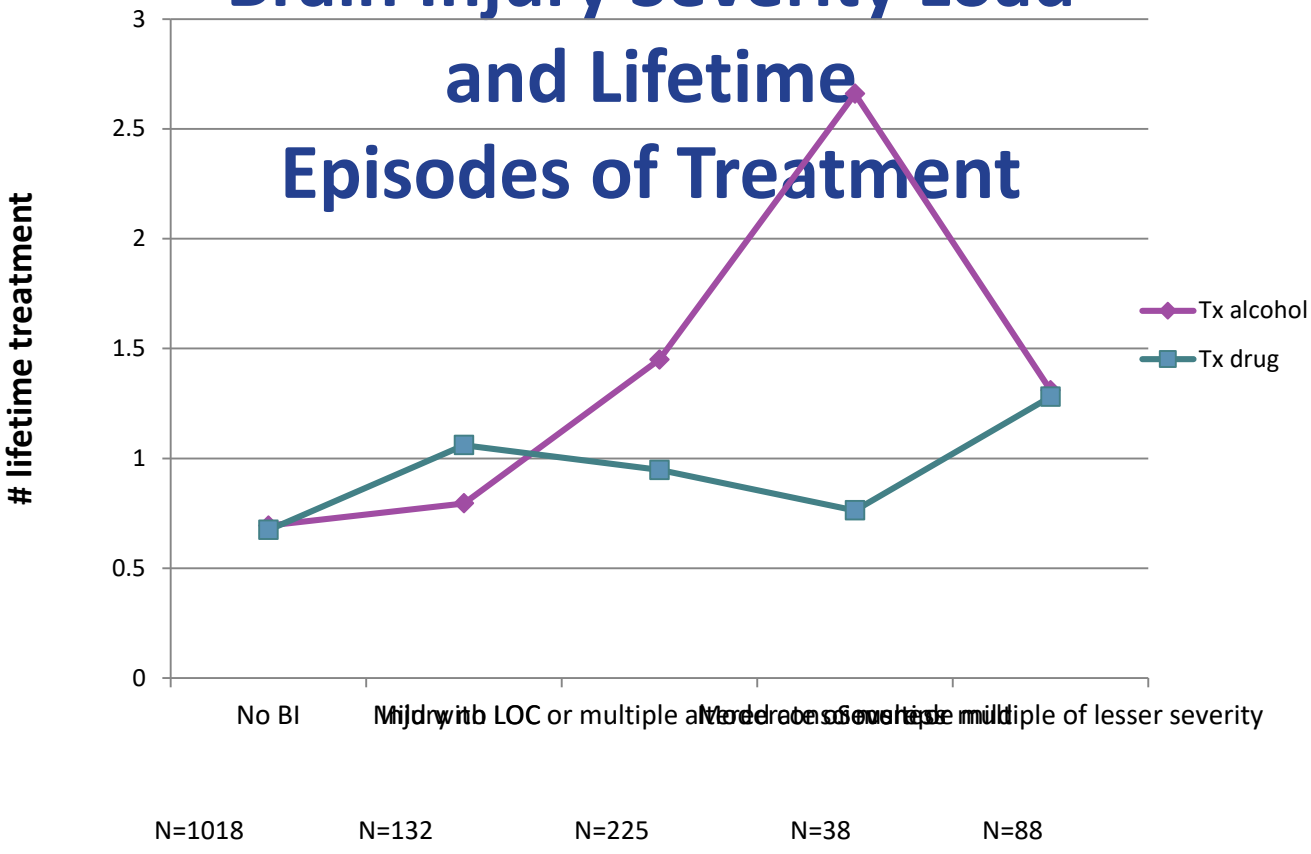
Other Symptoms Lifetime



Number of previous episodes of inpatient treatment

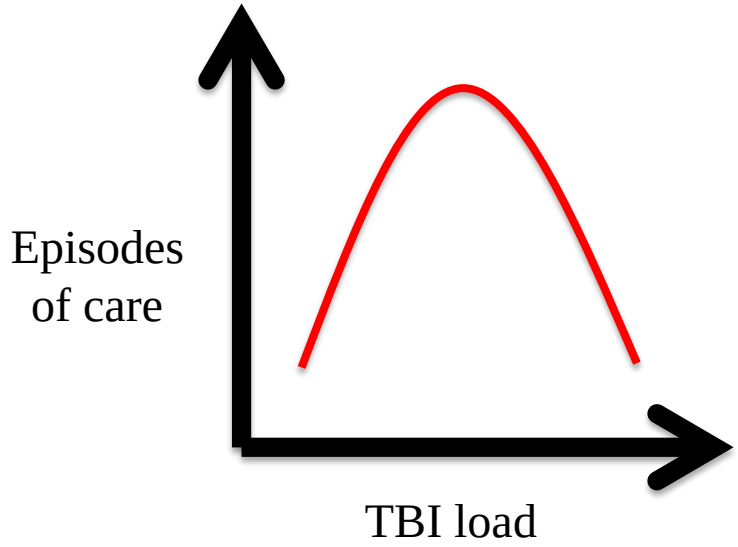
	No ABI with LOC N=1180	1 ABI with LOC N=286	2 or more ABI with LOC N=75	F*
Alcohol Treatment	.794 (3.55)	1.25 (2.97)	1.63 (3.97)	p=.029
Drug Treatment	.747 (2.28)	1.02 (2.44)	1.24 (2.15)	p=.051
* Welch robust test of equality of means				

Brain Injury Severity Load and Lifetime Episodes of Treatment



Reflections on CAMH Data

Consistent with other findings



- Walking wounded
  - What you don't measure, you don't see
- 2 years post-injury
  - Fall off wheel
  - Less support

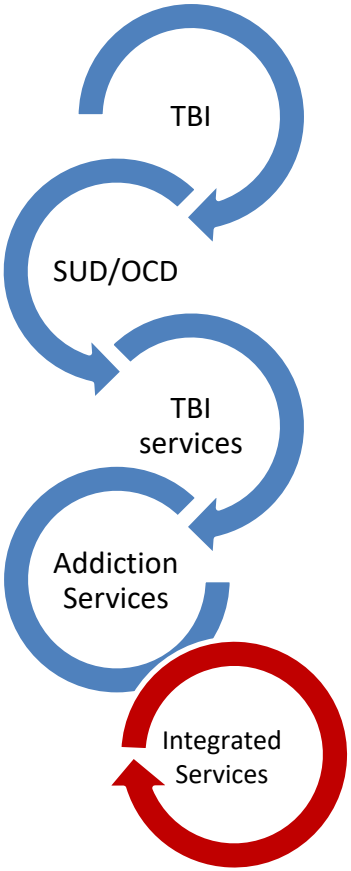
Neurocognitive Impairment from all Causes

Affects the majority of clients served

- Getting there
- Staying there
- Benefiting
- Maintaining Gains

Moderate TBI

- Male, Aged 52
- Severe TBI with 2 weeks coma as the result of an MVA
- Social alcohol use prior to injury (in religious household with limited use)
- No Marijuana use
- No Mental Health Issues
- Post injury SUD, OCD and Clinical Depression



Common Presentation

- Attention Deficits (mental control)
  - Distractibility
  - Stimulus binding (unable to resist distraction)
- New-learning and memory
- Executive Functioning
  - Impulsivity (not seeing consequences)
  - Organization (planning and sequencing)
- Emotional Dysregulation
- Abulia (impaired initiation/indecision)
- Awareness of Impairment

Case Example 2: DJ

Substance use beginning at age 15

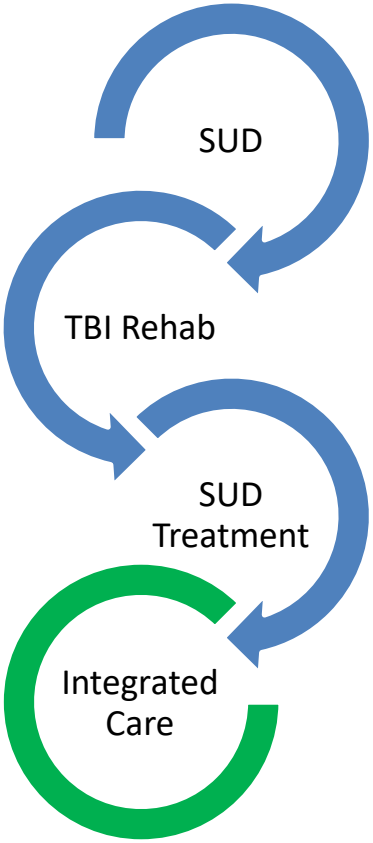
Poly substance use disorder emerging in adulthood

Severe brain injury at age 30: DUI

CAMH 21 day inpatient 7 years post injury

Day Treatment 10 years post-injury

Referred to CHIRS 18 years post injury CAMH



Presenting Issues

- Difficulty managing schedule of groups
- Seemed disorganized
- Ongoing marijuana use, 2 years crack-free, 10 years alcohol free
- Hoarding (housing in jeopardy)
- Limited engagement outside of CAMH

CHIRS Services

- Neuropsychological assessment
- Case management
  - Budgeting
  - Housing
  - Family Education
  - Support to attend addictions services
  - Behavioural intervention to address hoarding, impulsivity, etc.

Case Example 3: AC

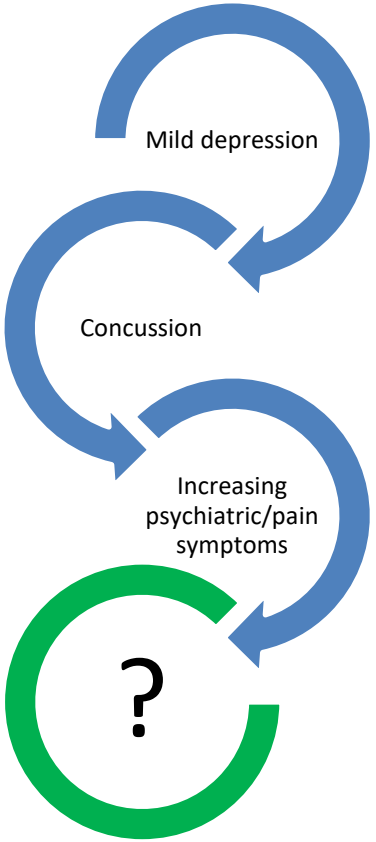
Married ICU Nurse, mother of one with a concussion at age 36 resulting from MVA 4years ago. 20 minutes LOC

No previous psychiatric history, but reportedly always struggled a bit with anxiety and depressed mood.

Struggled with return to work, now part-time

Recurrent Major depressive episodes with anxiety

Headache and developing codeine dependence.



Possible services

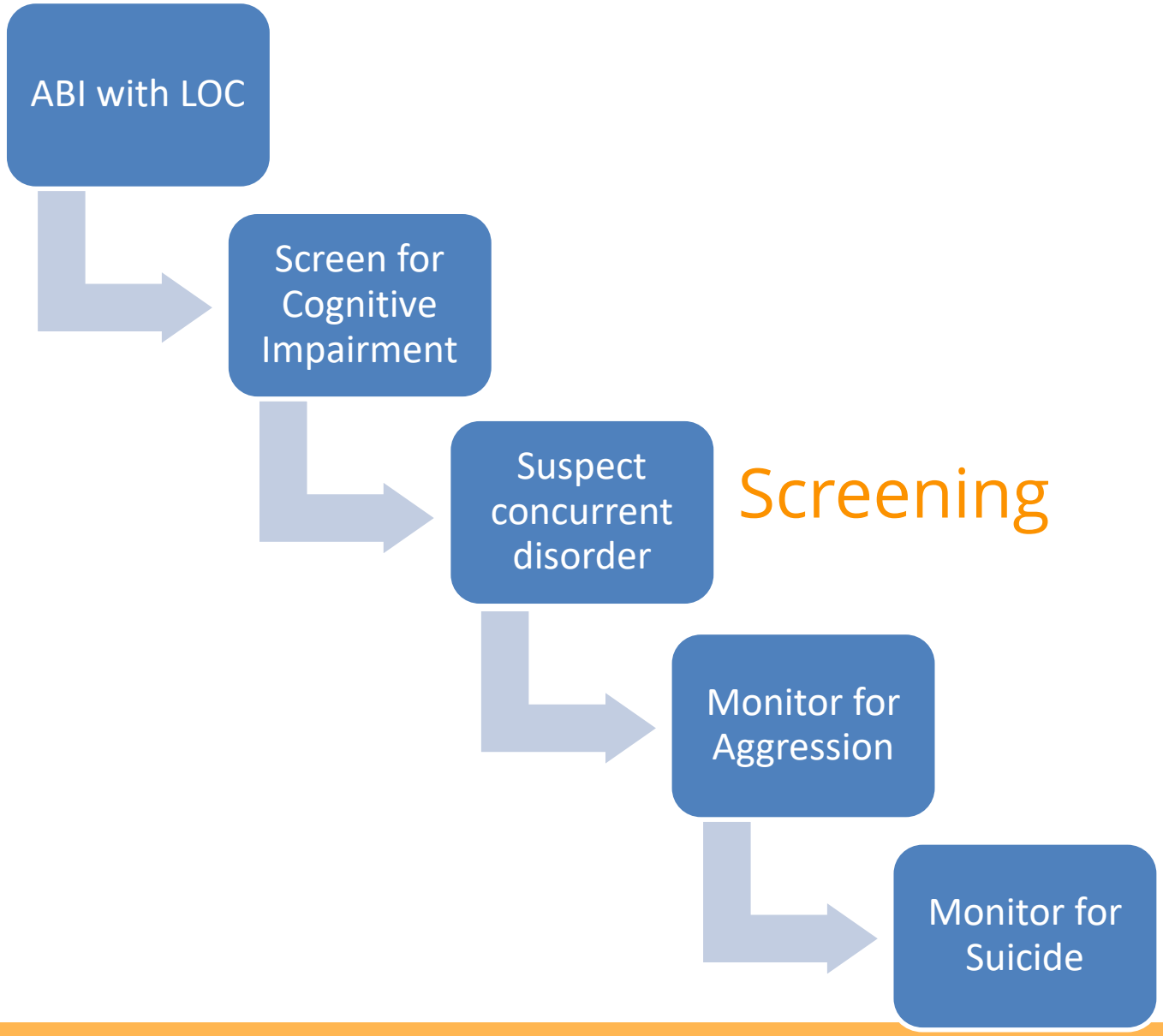
- CAMH- Pain and Addiction Clinic
- CHIRS
- TWH- Psychiatric consultation

What can be done here and now?

- Screen for ABI
- Learn to recognize and accommodate cognitive impairment
- Refer complex cases (the trifecta) to neuro-behavioural case conferences
- Consider expansion of case management services for those with less obvious impairment.

Access to Care

- Support in getting to treatment is crucial
  - Case management to organize
  - Reminders
  - Flexible policies during engagement
- Use of treatment incentives
- Programming that competes with substance use
- Psycho-social rehabilitation paradigms (CRA)
- Design of programming to accommodate problems in attention and memory and disinhibition (most common impairments)


ABI with LOC
Screen for Cognitive Impairment
Suspect concurrent disorder
Monitor for Aggression
Monitor for Suicide

Screening

Future: Invest in Community Care Paradigms

Cost of inpatient care

- $1350 per client per day (2013 estimate for inpatient addiction service).
- $37,800 for a 28 day cycle

Cost of outreach services

$100/hour (including travel, supervision and administration)

1 year of 7 hours per week of direct 1 one on 1 support in the community.

Prevention of readmissions, ALC days, and improvement of QOL

	Calculation	Total Cost	Potential Savings to Health System when using the proposed model of care *
978 Days of care of ALC Hospital Services	$1,500/day	$1,467,000	$0.00
978 days of Community Support*	$100/hr. Includes admin, travel and clinical supervision	$78,240-$156,480 (Range of 4-8 hrs/wk)	$1,388,760 - $1,310,520 every two years
978 days of specialized ABI residential support	Based on current actual care cost at CHIRS	$342,300	$1,124,700 Every two years

Questions & Discussion

References

- Beaulieu-Bonneau, S., St-Onge, F., Blackburn, M.C., Banville, A., Paradis-Giroux, A.A., Ouellet, M.C. (2017). Alcohol and drug use before and after during the first year after traumatic brain injury. J Head Trauma Rehabil. 1-10.
- Chen, Y., Yi-Kung Huang, E., Kua, T., Miller, J., Chiang, Y., Hoffer, B.J. (2017). Impact of traumatic brain injury on dopaminergic transmission. Cell Transplantation. 26(7): 1156-1168.
- Corrigan, J. D., Bogner, J., Mellick, D., Bushnik, T., Dams-O'Connor, K., Hammond, F. M., et al. (2013). Prior history of traumatic brain injury among persons in the traumatic brain injury model systems national database. Arch. Phys. Med. Rehabil. 94, 1940–1950.
- Fishbein, D., Dariotis, J. K., Ferguson, P. L., and Pickelsimer, E. E. (2016). Relationships between traumatic brain injury and illicit drug use and their association with aggression in inmates. Int J Offender Ther Comp Criminal. 60: 575-597.
- Ilie, G., Mann, R. E., Hamilton, H., Adlaf, E. M., Boak, A., Asbridge, M., et al. (2015). Substance use and related harms among adolescents with and without traumatic brain injury. J. Head Trauma Rehabil. 30, 293–301.
- Manley, G., Gardner, A.J., Schneider, K.J., Guskiewicz, K.M., Bailes, J., Cantu, R.C., Castellani, R.J., Turner, M., Jordan, B.D., Randolph, C., Dvořák, J., Hayden, K.A., Tator, C.H., McCrory, P., Iverson, G.L. (2017). A systematic review of potential long-term effects of sport-related concussion. J Sports Med. 51: 969-977.

References

- McAllister, T.W. (2011). Neurobiological consequences of traumatic brain injury. Dialogues in Clin Neurosci. 13(3): 287-300.
- McHugo GJ, Krassenbaum S, Donley S, Corrigan JD, Bogner J, Drake RE. (2017). The prevalence of traumatic brain injury among people with co-occuring mental health and substance use disorders. J Head Trauma Rehabil. 32(3): E65-E74.
- McKinlay, A., Corrigan, J., Horwood, L. J., and Fergusson, D.M. (2014). Substance abuse and criminal activities following traumatic brain injury in childhood, adolescence, and early adulthood. J. Head Trauma Rehabil. 29, 498–506.
- Merkel, S. F., Cannella, L. A., Razmpour, R., Lutton, E., Raghupathi, R., Rawls, S. M., et al. (2017a). Factors affecting increased risk for substance use disorders following traumatic brain injury: what we can learn from animal models. Neurosci. Biobehav. Rev. 77, 209–218
- Pagulayan, K.F., Temkin, N.R., Machamer, J.E., Dikmen, S.S. (2016). Patterns of alcohol use after traumatic brain injury. J Neurotrauma. 33: 1390-1396.
- Perry, D., Sturm, V.E., Peterson, M.J., Pieper, C.F., Bullock, T., Boeve, B.F., Miller, B.L., Guskiewicz, K.M., Berger, M.S., Kramer, J.H., Welsh-Bohmer, K.A. (2016). Traumatic brain injury is associated with subsequent neurologic and psychiatric disease: a meta-analysis. J Neurosurg. 124(2): 511-526.

References

- The Brain Injury Society of Toronto. (2017). *Acquired Brain Injury*. Retrieved from http://www.bist.ca/facts-about-abi/#
- Weil, Z.M., Corrigan, J. D., and Karelina, K. (2016a). Alcohol abuse after traumatic brain injury: experimental and clinical evidence. Neurosci. Biobehav. Rev. 62, 89–99.
- Weil, Z.M., Karelina, K. (2017). Traumatic brain injuries during development: implications for alcohol abuse. Front Behav Neurosci. 11(135): 1-8.
- Yi, J., Padalino D.J., Chin, L.S., Montenegro, P., Cantu, R.C. (2013). Chronic traumatic encephalopathy. Curr Sports Med Rep. 12(1): 28-32.